THE STATE OF NEW HAMPSHIRE
SUPREME COURT

DOCKET NO. 2020-0058

PLYMOUTH VILLAGE WATER & SEWER DISTRICT, RESOURCES MANAGEMENT, INC., CHARLES G. HANSON, AND 3M COMPANY

V.

ROBERT R. SCOTT AS COMMISSIONER OF THE NEW HAMPSHIRE DEPARTMENT OF ENVIRONMENTAL SERVICES

APPENDIX TO BRIEF OF AMICI CURIAE NATURAL RESOURCES DEFENSE COUNCIL, INC. AND CONSERVATION LAW FOUNDATION, INC. IN SUPPORT OF DEFENDANT

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April 29, 2020

TABLE OF CONTENTS

THE STATE OF NEW HAMPSHIRE
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PLYMOUTH VILLAGE WATER & SEWER DISTRICT, ET AL.

V.

ROBERT R. SCOTT AS COMMISSIONER OF THE NEW HAMPSHIRE DEPARTMENT OF ENVIRONMENTAL SERVICES

AFFIDAVIT OF SEAN MAHONEY

On oath, I, Sean Mahoney, say and depose as follows:

1. My name is Sean Mahoney, and I am Executive Vice President at Conservation Law Foundation ("CLF").
2. CLF is a non-profit environmental advocacy organization with offices in Massachusetts, Maine, New Hampshire, Rhode Island, and Vermont. CLF works across the region (including in Connecticut) with the mission to protect New England's environment for the benefit of all people, using the law, science, and the market to create solutions that preserve our natural resources, build healthy communities, and sustain a vibrant economy. CLF has approximately 5,000 members, including approximately 665 members in New Hampshire.
3. CLF's work over the years has included advocacy to better protect the public health from toxic substances. Such advocacy, for example, has included efforts across the region, including in New Hampshire, to protect the public from the health threats associated with lead. In recent years, as the pollution and health threats associated with per- and polyfluorinated alkyl substances ("PFAS") increasingly have come to light, CLF has

engaged in advocacy before state agencies and state legislatures to address the environmental and public health threats associated with this class of highly persistent chemicals that are toxic at extremely low concentrations.

4. In New Hampshire, the problem of PFAS contamination has arisen in several locations. In Merrimack, in 2016, public and private wells were found to be contaminated with PFAS associated with air emissions from the Saint-Gobain Performance Plastics facility, leading to an investigation by the N.H. Department of Environmental Services ("DES") that has been reported as the largest groundwater contamination investigation in the state's history.[1] PFAS contamination also has been found at Pease Tradeport, requiring measures to treat one of the City of Portsmouth's public wells, and at the Coakley landfill in North Hampton. PFAS contamination has been found in other locations across the state as a result of NHDES's investigation.[2]

5. CLF has engaged in advocacy in New Hampshire with DES and others on a site-specific basis (related to the Coakley landfill and Pease Tradeport) and by advocating for protective standards to protect the environment and public health from PFAS. CLF submitted extensive comments on DES's rulemaking to establish drinking water standards (maximum contaminant levels, or "MCLs") and ambient groundwater quality standards for four PFAS (PFOA, PFOS, PFNA, and PFHxS) to ensure protection of the public health, including those most vulnerable to PFAS exposure. CLF also has engaged

[1] *See* NH Public Radio, "NHDES to prove PFAS update in Merrimack," (Oct. 9, 2008) at https://www.nhpr.org/post/nh-des-provide-pfas-update-merrimack#stream/0. *See also* NHDES's PFAS Investigation website at https://www4.des.state.nh.us/nh-pfas-investigation/.

[2] *See* NHDES PFAS Sampling Map at http://nhdes.maps.arcgis.com/apps/View/index.html?appid=66770bef141c43a98a445c54a17720e2&extent=-73.5743,42.5413,-69.6852,45.4489.

in advocacy before the New Hampshire General Court, including advocacy in support of the bipartisan legislation requiring DES to proceed with the rulemaking at issue in this case.

6. Among CLF's members is the group Merrimack Citizens for Clean Water, which has been active on PFAS-related matters for years as a result of the widespread PFAS problems in Merrimack (including public and private wells that have been polluted by PFAS), and which participated in the DES rulemaking process. *See* Exhibit A.

Further, the Affiant saith not.

Date: April 28, 2019

/s/ Sean Mahoney
Sean Mahoney

STATE OF MAINE
COUNTY OF CUMBERLAND

Personally appeared the above named Sean Mahoney and made oath that the statements by him are true and accurate to the best of his knowledge, information, and belief.

Before me: Phelps Turner, Esq.

Date: April 28, 2020

/s/ Phelps Turner
Phelps Turner
Attorney at Law, ME Bar No. 5945

Exhibit A

March 4, 2019

NHDES PFAS MCL Merrimack Public Comment Hearing Statement

Three years ago, we became aware that Merrimack and several surrounding communities were identified by the EPA as a PFAS contamination site that we now know is at least 65 square miles in proximity. Fifteen years ago, prior to our awareness, NHDES archives show memos of concern with PFAS chemicals released from Saint Gobain Performance Plastics via air emissions that we now know have contaminated, and continue to contaminate groundwater, waterways, aquifers, soil, wells, wastewater -generated biosolids and compost, and no doubt our local food chain. We are a PFAS- impacted community with long-term exposure to what we now know is a bio-accumulative class of chemicals that do not break down and have been linked to thousands of health studies showing their harm. Dr Ben Chan of NHDHHS clarified last year in public comment that our blood serum in Merrimack will not decrease until we can stop exposure to these chemicals. That was received by our citizen group as a powerful statement.

We, in Merrimack and surrounding communities, bear a disproportionate burden of harm as compared to the rest of New Hampshire, and the time to act is now, to not only protect us from harm, but to give us rights to stop the accumulation of contamination and place the cost of remediation on the polluters whose actions put these chemicals in our drinking water.

NHDES was thankfully legislated to review health science and incorporate research that pertained to the most sensitive endpoints and most vulnerable population, infants, into maximum contaminant level recommendations for four PFAS compounds. The proposed levels of 38 ppt PFOA, 70ppt PFOS, 23ppt PFNA and 85 ppt PFHXS do not incorporate the full body of research for the most critical endpoints and cannot, in good conscience, become law. Additionally, these compounds are only four of the many PFAS we are exposed to in this community on a daily basis with active and direct source-identified pollution.

Over the last three years, I have dedicated as much time to advocating for PFAS impacts in exposed communities as I have my social work practice. Why? Because despite being told we have had a relatively low PFOA alone exposure level as compared to the current EPA health advisory of 70 parts per trillion, which we know is not protective, I have come to believe that health disruptions that I have seen not only in my entire family but in many other households in this town are associated with long-term PFAS exposure. We know from blood tests taken this year in Hoosick Falls, NY where residents have had exposure to the same chemical class and formulations via the same polluter, that babies are being born with high levels of PFAS in their

blood serum despite their parents having stopped their exposure to PFAS in drinking water four years ago.

I have reviewed all of the information NHDES considered to formulate MCLs and their methodology, and was surprised to see that the most sensitive endpoints that other states such as New Jersey, Vermont, New York and Pennsylvania have previously utilized in their MCL formulations and recommendations were not chosen. Our residents have heard the state rationale that only the most definitive science was chosen. This rationale does not serve our citizens, and we do not accept this approach. I will leave the scientific discussion of which endpoints, reference doses and uncertainty factors are truly protective to the scientists who specialize in this environmental health area as I know they will comment will full references cited in the NH MCL process.

On behalf of our citizen group, I will close by saying that it is unacceptable to exclude categories of studies that other states have concluded support more sensitive endpoints with the greatest potential for harm. These studies include mammary tissue and delayed development impacts for PFOA, and immune response and immune suppression impacts for PFOS. These disruptions in development in the formative years are likely to play out across the lifetime. Additionally, using a precautionary uncertainty factor is crucial as we know in this area we have had both long term and ongoing exposure to an entire class of PFAS, the majority of which are unrecognized in this process.

Laurene Allen, LICSW
Co-founder, Merrimack Citizens for Clean Water

THE STATE OF NEW HAMPSHIRE
SUPREME COURT

Docket No. 2020-0058

The Plymouth Village Water & Sewer District, Resources Management, Inc., Charles G. Hanson, and 3M Company,

Plaintiffs,

v.

Robert R. Scott as Commissioner of the New Hampshire Department of Environmental Services,

Defendant

AFFIDAVIT OF ERIK D. OLSON

I, Erik D. Olson, declare as follows:

1. I am the Senior Strategic Director for Health & Food at the Natural Resources Defense Council (NRDC) in the Healthy People and Thriving Communities Program. My current duties at NRDC include helping to guide the organization's work on issues such as drinking water, toxic chemicals in food, and pesticides.

2. This declaration is based on my personal knowledge and review of NRDC documents.

3. From 1991 to 2006, I worked at NRDC as a senior attorney, health program director, and director of the advocacy center. I then served as

General Counsel and Deputy Staff Director for the U.S. Senate Committee on Environment and Public Works and later as Deputy Director of the Pew Health Group at The Pew Charitable Trusts before rejoining NRDC in 2013. I previously worked as an attorney in the U.S. Environmental Protection Agency's Office of General Counsel.

4. NRDC currently has more than 350,000 members nationwide, including members in all fifty states and the District of Columbia. NRDC has more than 2,500 members in New Hampshire.

5. NRDC was founded in 1970. For the last fifty years, the organization has fought to protect the rights of all Americans to clean air and clean water, and to protect the wilderness and environment more broadly from pollution and degradation.

6. NRDC's purpose, as set forth in its mission statement, is "to safeguard the Earth: its people, its plants and animals, and the natural systems on which all life depends." NRDC's mission includes the protection of safe drinking water for all Americans and the prevention of exposure to health risks from drinking water contaminated with microbes or toxic chemicals such as lead and PFAS.

7. NRDC has long played a leadership role in the public interest community in the efforts to strengthen drinking water protection. My colleagues and I have issued over a dozen reports on drinking water quality, testified numerous times before the U.S. Senate and House of Representatives on drinking water issues, and are frequently sought out and quoted as experts on drinking water quality in major media outlets.

8. I and my colleague Mae Wu have both served during separate terms as appointed members of the National Drinking Water Advisory Council,

which Congress established to advise the U.S. Environmental Protection Agency (EPA) on drinking water issues. Additionally, Mae Wu and I have both served separately on Federal Advisory Committees advising the EPA on drinking water issues including establishing rules for disinfection byproducts, filtration for microbial contaminants, disinfectants, and the total coliform rule.

9. Through our Healthy People and Thriving Communities program, NRDC continues to pursue federal and state policies to improve aging infrastructure, protect drinking-water sources from pollution, and defend access to safe drinking water for every community in the United States, especially low-income communities and communities of color which are disproportionately at risk of exposure to polluted water.

10. Protecting NRDC members and others from the health risks of PFAS-contaminated drinking water is central to NRDC's mission. In recent years, NRDC has urged states to set up enforceable and health-protective drinking water standards for PFAS and to regulate the use of PFAS in consumer and industrial products.

11. For example, NRDC worked with local partners and filed extensive scientific and legal comments in several states including Massachusetts, Michigan, New Hampshire, New Jersey, and New York, urging that they adopt stringent, health-protective drinking water standards and expanded monitoring for PFAS. NRDC has also been actively involved in the efforts in California to have the state adopt health-protective drinking water standards for PFAS, and to initiate additional monitoring for PFAS in drinking water source wells across the state. In addition, we have worked with partners in other states to provide advice on their comments on state

PFAS drinking water standards.

12. NRDC filed extensive scientific comments on the draft Toxicological Profile for Perfluoroalkyls (a category of PFAS substances) proposed by the Centers for Disease Control and Prevention's (CDC) Agency for Toxic Substances Disease Registry (ATSDR), issued in June 2018.

13. NRDC experts have spoken at numerous national and state meetings, hearings, and forums on PFAS. For example, I was the sole representative of a non-profit environmental organization invited to speak at the EPA's May 2018 PFAS National Leadership Summit convened by the Administrator of the EPA and featuring senior state, federal, and local officials, and industry representatives. In addition, my colleague Mae Wu spoke at the 2019 Per- and Polyfluoroalkyl Substances: Second National Conference which brought together non-profit environmental organizations, community organizations, academia, and government officials to discuss emerging topics in public health and regulation of PFAS.

14. I was the only national environmental organization witness invited to testify at the hearings on PFAS held by the U.S. House of Representatives, Committee on Energy and Commerce in September 2018 and May 2019. Additionally, I was the only non-profit environmental representative to speak at the symposium on PFAS at the Toxicology Forum held in January 2020. The Toxicology Forum is an international organization that provides a large formal venue at which "views are exchanged among experts from domestic and international government regulatory and health agencies, industry, academia, 'political policymakers', and public interest groups." *See* https://dialogue.toxforum.org.

15. My colleague NRDC Scientist Dr. Anna Reade testified at a California State Water Resources Control Board hearing on PFAS in March 2019 regarding PFAS in California, core scientific principles, and policy recommendations. Dr. Reade also spoke recently at the February 2020 symposium on PFAS that brought together contributors from academia, industry, government, and NGOs to share information on the science and policy, convened by the Green Science Policy Institute.

16. On April 12, 2019, NRDC submitted comments to the New Hampshire Department of Environmental Services (DES) regarding the agency's PFAS rulemaking. In its comments, NRDC recommended that DES set strong drinking water standards to ensure that all residents of New Hampshire, regardless of life stage or vulnerability, would be protected from the harmful effects of PFAS exposure. NRDC's comments are attached as **Exhibit A**.

17. NRDC's April 2019 comments to DES referenced and attached a report prepared by NRDC and outside scientists. NRDC's report is attached as **Exhibit B**.

18. On April 16, 2020, I used the DES public data portal, OneStop, to calculate the number of active non-transient non-community water systems, the number of New Hampshire residents served by those systems, and the number of systems that serve daycares, schools, and senior housing units.[1]

19. To calculate the number of active non-transient non-community water systems using OneStop, I selected "Public Water Systems," "Status:

[1] DES OneStop, available at https://www4.des.state.nh.us/DESOnestop/BasicSearch.aspx (last visited April 16, 2020).

Active," "Non-Transient Non-Community,"and then "Enter." On the page that was generated, I clicked "Save Results," and then "Public Water Systems," and "Start Download." This generated a spreadsheet listing, in the first tab, all active non-transient non-community water systems. **Exhibit C** presents the data in the first tab of the spreadsheet; I added the "Total Population Served" row at the bottom to present the sum described in the next paragraph.

20. By summing up the figures in the "Population Served" column for each system, I calculated a total of 84,706 people served. By counting the total number of systems, I calculated a total of 478 non-transient non-community systems.

21. To calculate the number of senior housing units served by public water systems using OneStop, I selected "Public Water Systems," "Status: Active," "Senior Housing," and then "Enter." On the page that was generated, I clicked "Save Results," and then "Public Water Systems," and "Start Download." This generated a spreadsheet listing, in the first tab, senior housing units served by public water systems. **Exhibit D** presents the data in the first tab of the spreadsheet; I added the "Total Population Served" row at the bottom to present the sum described in the next paragraph.

22. By summing up the figures in the "Population Served" column for each senior housing unit, I calculated a total of 3,668 people served. By counting the total number of senior housing units, I calculated a total of 46 senior housing units served by public water systems. I recognize that this is only a subset of senior housing units in the state, as only senior housing units with their own public water systems would be listed, not those that get

their water from another public water system.

23. To calculate the number of day cares served by public water systems using OneStop, I selected "Public Water Systems," "Status: Active," "Day cares," and then "Enter." On the page that was generated, I clicked "Save Results," and then "Public Water Systems," and "Start Download." This generated a spreadsheet listing, in the first tab, day cares served by public water systems. **Exhibit E** presents the data in the first tab of the spreadsheet; I added the "Total Population Served" row at the bottom to present the sum described in the next paragraph.

24. By summing up the figures in the "Population Served" column for each day care, I calculated a total of 6,678 people served. By counting the total number of day cares, I calculated a total of 102 day cares served by public water systems. I recognize that this is only a subset of day cares in the state, as only day cares with their own public water systems would be listed, not those that get their water from another public water system.

25. To calculate the number of schools served by public water systems using OneStop, I selected "Public Water Systems," "Status: Active," "Schools (Public, Private, Day Schools)," and then "Enter." On the page that was generated, I clicked "Save Results," and then "Public Water Systems," and "Start Download." This generated a spreadsheet listing, in the first tab, all schools served by public water systems. **Exhibit F** presents the data in the first tab of the spreadsheet; I added the "Total Population Served" row at the bottom to present the sum described in the next paragraph.

26. By summing up the figures in the "Population Served" column for each school, I calculated a total of 56,680 people served. By counting the

total number of schools, I calculated a total of 171 schools served by public water systems. I recognize that this is only a subset of schools in the state, as only schools with their own public water system would be listed, not those that get their water from another public water system.

27. On April 16, 2020, I visited https://www.census.gov/quickfacts/NH to obtain the U.S. Census Bureau's most recent population estimates for demographic groups in New Hampshire. According to this data, the estimated total population of New Hampshire as of July 1, 2019 is 1,359,711. In addition, 4.7 percent of the New Hampshire population is 5 years or younger and 18.1 percent of the New Hampshire population is 65 years or older. The U.S. Census Bureau population estimates are attached as **Exhibit G**.

28. To calculate the estimated percentage of the New Hampshire population that was born in the last year, I used the number of births in New Hampshire in 2018, as reported in the Centers for Disease Control and Prevention (CDC) November 2019 National Vital Statistics Report, attached as **Exhibit H**, and divided that number (11,995) by the estimated population of New Hampshire (1,359,711), as reported by the U.S. Census Bureau (see Exhibit G).

I declare under penalty of perjury that the foregoing is true and correct.

_____ April 28, 2020

Erik Olson Date

STATE OF NEW HAMPSHIRE

COUNTY OF MERRIMACK

The above named Erik D. Olson signed or attested before me that the statements by him are true and accurate to the best of his knowledge, information, and belief.

Before me: Nicole Manteau

Date: April 28, 2020

/s/ Nicole Manteau, Notary
My commission expires April 20, 2021.

EXHIBIT A

April 11, 2019

Robert S. Scott
Commissioner
New Hampshire Department of Environmental Services
29 Hazen Drive
PO Box 95
Concord, NH 03302-0095

Michael J. Wimsatt
Director
New Hampshire Department of Environmental Services
29 Hazen Drive
PO Box 95
Concord, NH 03302-0095

Re: Comments on New Hampshire's Proposed Maximum Contaminant Levels for Perfluorooctanoic Acid (PFOA), Perfluorooctanesulfonic Acid (PFOS), Perfluorononononanoic Acid (PFNA), Perfluorohexane Sulfonic Acid (PFHxS), and Total Per- and Polyfluoroalkyl (PFAS) Chemicals in Drinking Water

Dear Commissioner Scott and Director Wimsatt,

On behalf of the more than 17,400 New Hampshire members and activists of the Natural Resources Defense Council (NRDC), thank you for the opportunity to comment on New Hampshire's proposed rulemaking establishing Maximum Contaminant Levels for certain per- and polyfluoroalkyl (PFAS) substances in drinking water. We write to express concern that the Maximum Contaminant Levels proposed by the New Hampshire Department of Environmental Services (NHDES) are too high and fail to consider available evidence establishing the toxicity of these contaminants at extremely low doses.

The Natural Resources Defense Council (NRDC) is an international nonprofit environmental organization with more than 3 million members and online activists. Since 1970, NRDC has worked to protect Americans from toxic contaminants in their drinking water. NRDC led efforts to strengthen the federal Safe Drinking Water Act in the 1986 and 1996 Amendments to the Act. NRDC has also spearheaded national campaigns for more protective drinking water rules for microbial contaminants and toxic metals and chemicals.

Perfluorooctanoic acid (PFOA), perfluorooctanesulfonic acid (PFOS), perfluorononononanoic acid (PFNA), and perfluorohexane sulfonic acid (PFHxS) belong to a class of similarly-structured chemicals, PFAS, which are synthetic fluorinated compounds,

prevalent in consumer products and industrial settings, and increasingly detected in drinking water. The toxicity, persistence, and mobility of these contaminants is well-established.

While New Hampshire has taken important steps towards evaluating the prevalence of certain PFAS chemicals in drinking water and their associated health risks, the state's proposed standards – 38 parts per trillion (ppt) for PFOA; 70 ppt for PFOS; 85 ppt for PFHxS; and 23 ppt for PFNA – fall short of reaching a health-protective threshold.[1] In light of the prevalence, persistence and toxicity of these dangerous chemicals at very low levels, NRDC recommends that NHDES promptly establish an enforceable final Maximum Contaminant Level for PFOA, PFOS, PFNA, and PFHxS at a combined concentration of 2 ppt. And when these MCLs have been finalized, NHDES should, within two years, set a Maximum Contaminant Level Goal of zero for the entire PFAS class and a Treatment Technique for total PFAS based on the best available detection and treatment technologies. If NHDES declines to regulate total PFAS, the agency should set a Maximum Contaminant Level of 5 ppt for the PFAS substance GenX, and set additional health-protective Maximum Contaminant Levels for other PFAS contaminants detected in the state's drinking water.

In the absence of adequate federal safeguards, New Hampshire must act to protect drinking water, reduce risks to the public, and remediate contaminated drinking water sources. Clear and mounting evidence demonstrates the link between low dose-exposures to these chemicals and serious human health risks, including cancer and adverse immunological, developmental and reproductive effects. Further, while there is limited toxicity information for PFAS outside the more-studied contaminants listed above, a growing body of scientific research indicates that the class collectively poses similar threats to human health and the environment.

Over the past two years, NRDC has conducted a detailed review the health risks associated with PFAS exposure in drinking water. As part of this effort, NRDC has released a report, authored by NRDC staff scientist Anna Reade, Ph.D.,[2] engineer Tracy Quinn, P.E.,[3] and our expert consultant, Judith Schreiber, Ph.D.,[4] that makes recommendations

[1] *See* N.H. Rev. Stat. § 485:3, 1(b); *see also* N.H. Rev. Stat. § 541-A.

[2] Anna Reade, Ph.D., is a scientist with the Natural Resources Defense Council. She previously worked in the California State Senate with the California Council on Science and Technology.

[3] Tracy Quinn, P.E., is Director of California Water Conservation and Efficiency with the Natural Resources Defense Council. She holds a M.E. in civil engineering.

[4] Judith Schreiber, Ph.D., leads Schreiber Scientific LLC and is a former Chief Scientist at the Environmental Protection Bureau of the New York State Office of the Attorney General and former Section Chief of Environmental Research at the New York State Department of Health.

regarding health-protective Maximum Contaminant Levels and Maximum Contaminant Level Goals for PFAS in states. The full report is attached to this letter for your review.

The reasoning behind NRDC's recommendation that New Hampshire set a Maximum Contaminant Level for PFOA, PFOS, PFNA, and PFHxS at a combined concentration of 2 ppt, and within two years set a Maximum Contaminant Level Goal of zero for the PFAS class and a Treatment Technique for total PFAS – or alternatively set an additional Maximum Contaminant Level of 5 ppt for GenX and Maximum Contaminant Levels for other detected PFAS contaminants at health-protective levels – is explained thoroughly in the attached report and summarized for your review below.

I. Statutory Framework

A. New Hampshire Safe Drinking Water Act

The New Hampshire Safe Drinking Water Act, as amended, provides that NHDES must establish Maximum Contaminant Levels for PFOA, PFOS, PFNA and PFHxS.[5] We further urge NHDES to, within two years, set a Maximum Contaminant Level Goal of zero for the entire PFAS class and a Treatment Technique for total PFAS based on the best available detection and treatment technologies. If NHDES declines to regulate total PFAS, the agency should set a Maximum Contaminant Level of 5 ppt for the PFAS substance GenX, and set additional health-protective Maximum Contaminant Levels for other PFAS contaminants detected in the state's drinking water. Under the New Hampshire Safe Drinking Water Act, these standards shall be established at levels "acceptable in water for human consumption" after considering "the extent to which the contaminant is found in New Hampshire, the ability to detect the contaminant in public water systems, the ability to remove the contaminant from drinking water, and the costs and benefits to affected parties."[6] Because New Hampshire law provides little guidance on the procedure to follow in determining health-protective regulatory standards for contaminants in drinking water, the federal framework for promulgating health-protective Maximum Contaminant Level Goals and Maximum Contaminant Levels is instructive. This framework is set out in the federal Safe Drinking Water Act,[7] and its implementing regulations.

[5] N.H. Rev. Stat. § 485:16-e.

[6] N.H. Rev. Stat. § 485:3, 1(b).

[7] 40 U.S.C. § 300f *et seq.*

B. Federal Safe Drinking Water Act

Under the federal framework for regulating contaminants in drinking water, a Maximum Contaminant Level Goal is the maximum level of a contaminant in drinking water at which no known or anticipated adverse effect on the health of persons would occur, allowing an adequate margin of safety.[8] When determining a Maximum Contaminant Level Goal, the agency considers adverse health risk to sensitive subpopulations, such as infants, children, the elderly, those with compromised immune systems and chronic diseases. Maximum Contaminant Level Goals are non-enforceable public health goals and consider only public health and not the limits of detection and treatment technology effectiveness. Therefore, they sometimes are set at levels which water systems cannot meet because of technological limitations.

A Maximum Contaminant Level Goal is derived by first considering the carcinogenic potential of the contaminant, or suite of contaminants. For "known or probable" carcinogens, a Maximum Contaminant Level Goal of zero is set for the contaminant, or for the contaminant class.[9] This is because it is assumed that, in the absence of other data, there is no known threshold at which no adverse health effects would occur.

For chemical contaminants that are non-carcinogens and exhibit a threshold for their non-cancer health effects, the Maximum Contaminant Level Goal is based on the "reference dose."[10] The "reference dose" is an estimate of the amount of a chemical that the human population can be exposed to on a daily basis without an appreciable risk of adverse health effects during a lifetime. This estimate depends on the "most sensitive endpoint." The "most sensitive endpoint" is the adverse health effect associated with the lowest level of exposure in scientific studies. The "reference dose" is then adjusted by selecting and applying "uncertainty factors" in order to establish an appropriate margin of safety.[11]

An enforceable Maximum Contaminant Level is to be established at a level as close to the Maximum Contaminant Level Goal as is "feasible;" the term "feasible" is defined to mean "feasible with the use of the best technology, treatment techniques and other means which the Administrator finds, after examination for efficacy under field conditions and not

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[8] *See* 42 U.S.C. § 300g-1.

[9] *See 56 Fed. Reg. 20, 3532* (Jan. 30, 1991); U.S. DEP'T OF COMMERCE, NAT. TECH. INFO. SERVICE, Development of Maximum Contaminant Levels under the Safe Drinking Water Act, Report Prepared for U.S. Envtl. Prot. Agency (1988);

[10] *See 56 Fed. Reg. 20, 3531-3532* (Jan. 30, 1991).

[11] *Id.*

solely under laboratory conditions, are available (taking cost into consideration)."[12] With respect to removal of synthetic organic chemicals such as PFAS, the Act explicitly states that "granular activated carbon is feasible for the control of synthetic organic chemicals, and any technology, treatment technique, or other means found to be the best available for the control of synthetic organic chemicals must be at least as effective in controlling synthetic organic chemicals as granular activated carbon."[13] When there is no reliable method that is economically or technically feasible to measure a contaminant at concentrations to indicate there is not a public health concern, a "Treatment Technique"[14] rather than a Maximum Contaminant Level is set. A Treatment Technique is an enforceable procedure or level of technological performance that public water systems must follow to ensure control of a contaminant or class of contaminants in drinking water.

II. Per- and Polyfluoroalkyl Substances

Per- and polyfluoroalkyl substances (PFAS) are a group of synthetic chemicals that include perfluorooctanoic acid (PFOA), perfluorooctanesulfonic acid (PFOS), perfluorononononanoic acid (PFNA), perfluorohexane sulfonic acid (PFHxS), and GenX. Since the 1940s, these chemicals have been widely used in industrial settings and consumer products, including nonstick cookware (e.g., Teflon), stain-resistant repellents used on carpets and fabric (e.g., Scotchgard and Stainmaster), paper and cardboard food packaging (e.g., fast food wrappers),[15] firefighting foam, textiles (e.g., Gore-Tex), toothpaste, shampoos, cosmetics, polishes and waxes, pesticides and herbicides, windshield wipers, and many products for the aerospace, automotive, construction, and electronic industries.[16]

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[12] 42 U.S.C. § 300g-1(b)(4)(D)..

[13] 42 U.S.C. § 300g-1(b)(4)(D).

[14] 42 U.S.C. § 300g-1(b)(3)(C)(ii).

[15] *See* Amy Martyn, *Anti-grease Chemicals Used in Fast Food Wrappers Can Accumulate in Organs, Study Finds*, CONSUMER AFFAIRS (Mar. 30, 2017), https://www.consumeraffairs.com/news/anti-grease-chemicals-used-in-fast-food-wrappers-can-accumulate-inorgans-study-finds-033017.html.

[16] *See* INTERSTATE TECH. & REG. COUNCIL, History and Use of Per- and Polyfluoroalkyl Substances (PFAS) (Nov. 2017), https://pfas-1.itrcweb.org/wp-content/uploads/2017/11/pfas_fact_sheet_history_and_use__11_13_17.pdf; U.S. ENVTL. PROT. AGENCY, Drinking Water Health Advisory for Perfluorooctanoic Acid (PFOA), EPA DOC. NO. 822-R-16-005, at 24 (May 2016), https://www.epa.gov/sites/production/files/2016-05/documents/pfoa_health_advisory_final_508.pdf; U.S. ENVTL. PROT. AGENCY, Drinking Water Health Advisory for Perfluorooctane Sulfonate (PFOS), EPA DOC. NO. 822-R-16-004, at 24-25 (May 2016), https://www.epa.gov/sites/production/files/2016-05/documents/pfos_health_advisory_final_508.pdf.

A. PFAS Contaminates Drinking Water Sources Across the United States and is Highly Prevalent in New Hampshire

While PFAS do not occur naturally in the environment, due to widespread use, PFAS are now ubiquitous across the planet – present in rivers, soil, air, house dust, food and drinking water from surface and groundwater sources. PFAS are extremely persistent in the environment, meaning they are resistant to environmental degradation.[17] They are also highly mobile in the environment and can thus move through the soil and into groundwater and remain there for many years.[18]

Elevated PFAS levels have been detected in drinking water supplies across the country, in at least 33 states, 3 territories, and one indigenous community, contaminating the water supplies of millions of people.[19] Exceedances of the U.S. Environmental Protection Agency's (EPA's) lifetime health advisory limit have been detected not only in New Hampshire, but also Alaska, Arizona, California, Colorado, Florida, Illinois, Indiana, Kentucky, Massachusetts, New Jersey, New York, Ohio, Pennsylvania, Texas, and Vermont, among other states.[20] Elevated levels of PFAS in drinking water are strongly

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[17] U.S. ENVTL. PROT. AGENCY, Technical Factsheet for Perfluorooctane Sulfonate (PFOS) and Perfluorooctanoic Acid (PFOA) (Nov. 2017), https://www.epa.gov/sites/production/files/2017-12/documents/ffrrofactsheet_contaminants_pfos_pfoa_11-20-17_508_0.pdf.

[18] *See* U.S. ENVTL. PROT. AGENCY, Technical Factsheet for Perfluorooctane Sulfonate (PFOS) and Perfluorooctanoic Acid (PFOA) (Nov. 2017), https://www.epa.gov/sites/production/files/2017-12/documents/ffrrofactsheet_contaminants_pfos_pfoa_11-20-17_508_0.pdf.

[19] *See* Xindi C. Hu *et al.*, *Detection of Poly- and Perfluoroalkyl Substances (PFASs) in U.S. Drinking Water Linked to Industrial Sites, Military Fire Training Areas, and Wastewater Treatment Plants*, 3 ENVTL. SCI. & TECH. LETTERS 344 - 346, fig.1 (2016) (using data from EPA's third Unregulated Contaminant Monitoring Rule in order to create maps to display where PFOS and PFOA have been found in water supplies).

[20] *Id.*; *see also* Tim Ellis, *'Dire Health Effects': Local Group Seeks Strict Regulation of Firefighting-foam Chemicals,* KUAC (Nov. 28, 2017), http://fm.kuac.org/post/dire-health-effects-local-group-seeks-strict-regulation-firefighting-foam-chemicals (Alaska); Bruce Finley, *Air Force Sends First $400,000 Filter to Fountain to Scrub PFC Contamination from Ground Water*, DENVER POST, http://www.denverpost.com/2017/06/29/air-force-filter-fountain-colorado-contaminated-water/ (last updated Jul. 3, 2017) (Colorado); Jess Mancini, *No Surprises in C8 Report: Study Finds Higher Concentrations*, PARKERSBURG NEWS & SENTINEL (May 26, 2017), http://www.newsandsentinel.com/news/local-news/2017/05/researcher-no-surprises-in-c8-report (Indiana); Garret Ellison, *PFAS Found In Drinking Water Wells In Unexpected Places*, MLIVE (Nov. 8, 2017), http://www.mlive.com/news/grand-rapids/index.ssf/2017/11/pfas_private_well_test_results.html (Michigan); *Officials: Elevated Levels of*

associated with proximity to major industrial sites, civilian airports, and military fire training areas.[21]

In New Hampshire, NHDES has documented some levels of PFOA, PFOS, PFNA, and/or PFHxS in 17 percent, or 272 non-transient public water systems;[22] of these, 59 contained PFAS levels above 10 ppt.[23]

B. Several States Have Taken Affirmative Action to Fill the Regulatory Gap on PFAS in Drinking Water

In the absence of robust federal regulation, several states have established or put forth draft (and in one case a final) Maximum Contaminant Levels or taken other steps to fill the regulatory gap. For example, New Jersey, in November 2017, recommended – and is now poised to adopt – Maximum Contaminant Levels for PFOA at 14 ppt and PFOS at 13 ppt.[24] New Jersey recently also formally adopted a Maximum Contaminant Level for PFNA at 13 ppt.[25] Vermont has established a drinking water health advisory and enforceable

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PFOA Measured near Landfill, WASH. TIMES (Jun. 4, 2016), http://www.washingtontimes.com/news/2016/jun/4/officials-elevated-levels-of-pfoa-measured-near-la/ (New Hampshire); Jeff Hirsh, *Drinking Water Safety Concerns: New Historical Evidence of "PFOA" in Ohio River*, LOCAL12.COM (May 25, 2017), http://local12.com/news/local/drinking-water-safety-concerns-new-historical-evidence-of-pfoa-in-ohio-river (Ohio); TEXAS MILITARY DEPARTMENT, DRINKING WATER SAMPLING RESULTS NOTIFICATION (2017), *available at* https://tmd.texas.gov/Data/Sites/1/media/press-releases/2017/may/18may/tmd-pfos-pfoa-results-notification-fact-sheet-17-may.pdf (Texas); Brad Evans & Renee Wunderlich, *'Stop Drinking the Water': Pownal Municipal Samples Test Positive for PFOA*, NBC5 (Mar. 24, 2016, 6:25 PM), http://www.mynbc5.com/article/stop-drinking-the-water-pownal-municipal-samples-test-positive-for-pfoa/3326716 (Vermont).

[21] Hu *et al.*, *Detection of PFAS*, *supra* note 26, at 345.

[22] N.H. Dep't of Envtl. Servs., Summary Report on the New Hampshire Department of Environmental Services Development of Maximum Contaminant Levels and Ambient Groundwater Quality Standards for Perfluorooctanesulfonic Acid (PFOS), Perfluorooctanoic Acid (PFOA), perfluorononononanoic acid (PFNA), and perfluorohexane sulfonic acid (PFHxS), 10 (Jan. 4, 2019).

[23] *Id.*

[24] Katie Jennings, *DEP Adopts Tough Limits for PFOA Contamination in Drinking Water*, POLITICO N.J. PRO (Nov. 1, 2017), https://www.politicopro.com/states/new-jersey/story/2017/11/01/dep-adopts-tough-limits-forpfoa-contamination-in-drinking-water-115413.

[25] Scott Fallon, New Jersey Becomes First State to Regulate Dangerous Chemical PFNA in Drinking Water, NJ.com (Sept. 6, 2018),

groundwater cleanup level for combined concentrations of PFOA, PFOS, PFHxS, PFNA and PFHpA at 20 ppt.[26] In January 2019, Vermont announced it will initiate the process of adopting its health advisory for these five PFAS as an enforceable MCL.[27] Minnesota has published groundwater guidance levels for PFOA and PFOS at 35 ppt and 27 ppt, respectively.[28] California has recommended an interim notification level of 14 ppt for PFOA, and 13 ppt for PFOS in drinking water.[29] Connecticut has adopted an action level for combined levels of PFOA, PFOS, PFNA, PFHxS and PFHpA of 70 ppt.[30] New York state has additionally recommended Maximum Contaminant Levels of 10 ppt for PFOA and 10 ppt for PFOS.[31] Michigan recently established recommended Public Health Drinking

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https://www.northjersey.com/story/news/environment/2018/09/06/new-jersey-first-state-regulate-dangerous-chemical-pfna-pfoa/1210328002/.

[26] Vt. Dep't of Health, Memorandum from Mark A. Levine, Commissioner, to Emily Boedecker, Commissioner, Drinking Water Health Advisory for Five PFAS (per- and polyfluorinated alkyl substances) (July 10, 2018), http://www.healthvermont.gov/sites/default/files/documents/pdf/ENV_DW_PFAS_HealthAdvisory.pdf; Vt. Nat. Res. Agency, Dep't of Envtl. Conservation, Chapter 12 of the Environmental Protection Rules: Groundwater Protection Rule and Strategy, Emergency Rule (Jan. 8, 2019), https://dec.vermont.gov/sites/dec/files/documents/GWPR%26S%20Clean%20Version.pdf.

[27] Vt. Nat. Res. Agency, Agency of Natural Resources Initiates Rulemaking Process to Adopt Maximum Contaminant Level for PFAS Compounds, https://anr.vermont.gov/content/agency-natural-resources-initiates-rulemaking-process-adopt-maximum-contaminant-level-pfas (last accessed Jan. 19, 2019).

[28] Minn. Dep't of Health, Toxicological Summary for: Perfluorooctanoate (Aug. 2018), http://www.health.state.mn.us/divs/eh/risk/guidance/gw/pfoa.pdf; Minn. Dep't of Health, Toxicological Summary for: Perfluorooctane Sulfonate (May 2017), http://www.health.state.mn.us/divs/eh/risk/guidance/gw/pfoa.pdf.

[29] Cal. Office of Envtl. Health Hazard Assessment, Memorandum from Lauren Zelise, Director, to Darrin Polhemus, Deputy Director. Div. of Drinking Water State Water Resources Control Board, Recommendation for Interim Notification Levels for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS) (June 26, 2018), https://www.waterboards.ca.gov/drinking_water/certlic/drinkingwater/documents/pfos_and_pfoa/OEHHA_Recommended_Int_NL_Jun_26_2018.pdf.

[30] Ct. Dep't of Health, Drinking Water Action Level for Perfluorinated Alkyl Substances (PFAS), Environmental and Occupational Health Assessment (Dec. 12, 2016), https://www.asdwa.org/wp-content/uploads/2018/01/CT-PFASActionLevel.pdf.

[31] N.Y. Dep't of Health, Drinking Water Quality Council Recommends Nation's Most Protective Maximum Contaminant Levels for Three Unregulated Contaminants in Drinking Water (Dec. 18, 2018), https://www.health.ny.gov/press/releases/2018/2018-12-18_drinking_water_quality_council_recommendations.htm.

Water Screening Levels of 9 ppt for PFOA, 8 ppt for PFOS, 9 ppt for PFNA, 84 ppt for PFHxS, and 1000 ppt for PFBS.[32]

C. New Hampshire's Response to PFAS Contamination

In the absence of federal action to regulate PFAS, New Hampshire has taken some steps to protect residents on its own. In 2018, the state legislature passed a law, amending the New Hampshire Safe Drinking Water Act, requiring the state Department of Environmental Services, with consultation with the New Hampshire Department of Health and Human Services, to develop drinking water and groundwater standards for four types of PFAS: PFOA, PFOS, PFNH, and PFHxS.[33]

On January 2, 2019, NHDES published its proposed standards for drinking water. NHDES proposed the following Maximum Contaminant Levels for four PFAS: 38 parts per trillion (ppt) for PFOA; 70 ppt for PFOS; 85 ppt for PFHxS; and 23 ppt for PFNA.

D. Health Effects of PFAS

PFAS Accumulate in the Human BodyMany PFAS are also bioaccumulative, meaning the body retains these chemicals long after exposure to these chemicals ends.[34] PFOA, PFOS, PFNA, PFHxS, and related PFAS are known to bioaccumulate in the body of people of all ages, even before birth.

PFAS are detected in over 98 percent of Americans' bodies.[35] Because the manufacturing of PFOA and PFOS has largely been phased out in the United States, some

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[32] Mich. Dep't of Health and Hum. Servs, Division of Environmental Health Michigan PFAS Action Response Team Human Health Workgroup, Public Health Drinking Water Screening Levels for PFAS, Feb. 22, 2019, available online at https://www.michigan.gov/documents/pfasresponse/MDHHS_Public_Health_Drinking_Water_Screening_Levels_for_PFAS_651683_7.pdf.

[33] N.H. Rev. Stat. § 485:16-e (July 10, 2018).

[34] U.S. DEP'T HEALTH & HUM. SERVS., AGENCY FOR TOXIC SUBSTANCE & DISEASE REGISTRY, An Overview of Perfluoroalkyl and Polyfluoroalkyl Substances and Interim Guidance for Clinicians Responding to Patient Exposure Concerns, at 2 (July 7, 2017), https://www.atsdr.cdc.gov/pfc/docs/pfas_clinician_fact_sheet_508.pdf.

[35] Antonia M. Calafat *et al.*, *Polyfluoroalkyl Chemicals in the U.S. Population: Data from the National Health and Nutrition Examination Survey (NHANES) 2003–2004 and Comparisons with NHANES 1999–2000*, 115 ENVTL. HEALTH PERSP. 11, 1596-1602 (2007); *see also* U.S. CTRS. FOR DISEASE CONTROL & PREVENTION, National Biomonitoring Program: Biomonitoring Summary, Perfluorochemicals,

PFOA and PFOS levels in blood serum have started to decrease in recent years.[36] However, PFOA and PFOS may still enter the country through imported goods, and many other PFAS, such as GenX, are still used as PFOA and PFOS substitutes within the United States. Because certain PFAS bioaccumulate and are not readily excreted by the body, and because certain PFAS are resistant to degradation and persist in water systems absent filtration, PFAS will continue to be present in the general population for many years in the future.

1. Wherever PFAS are Present at Detectable Levels in Drinking Water, Drinking Water Is the Dominant Source of Exposure to PFAS Over Any Other Source

Once PFAS levels are found in drinking water, drinking water becomes the overwhelming source of exposure to PFAS. Drinking water PFAS concentrations of 100 ppt and 400 ppt, for example, are predicted to contribute 71 percent and 91 percent, respectively, of total exposure; and are estimated to increase PFAS blood serum levels, on average, by 250 percent and 1,000 percent, respectively.[37] Indeed, PFAS concentrations in drinking water are associated with even higher levels of PFAS in blood serum. For example, chronic exposure to PFOA in drinking water results in blood serum PFOA levels approximately 100 times greater than the PFOA concentration in drinking water.[38] In order to reduce levels of PFAS in the blood serum of New Hampshire residents, it is of utmost importance to remove PFAS from the drinking water to non-detectable levels.

2. Fetuses, Infants and Children Are Particularly Vulnerable to PFAS Exposure Through Drinking Water

In setting an MCL for PFOA, PFOS, PFNH, PFHxS, and in setting any future MCL for GenX, NHDES should take into account the effect of exposure on the state's most sensitive populations—fetuses, infants, and children. By the time we are children, nearly all of us have some level of PFAS in our bodies. This is because PFAS exposure begins in

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https://www.cdc.gov/biomonitoring/PFAS_BiomonitoringSummary.html (last visited Sept. 3, 2018).

[36] U.S. DEP'T HEALTH & HUM. SERVS., AGENCY FOR TOXIC SUBSTANCE & DISEASE REGISTRY, Perfluoroalkyl and Polyfluoroalkyl Substances (PFAS) in the U.S. Population (Aug. 21, 2017), https://www.atsdr.cdc.gov/pfc/docs/PFAS_in_People.pdf.

[37] Robin Vestergren & Ian T. Cousins, Tracking the Pathways of Human Exposure to Perfluorocarboxylates, 43 ENVTL. SCI. TECH. 15, 5565-5575 (2009).

[38] G.B. Post *et al.*, Perfluoroocantoic acid (PFOA), An Emerging Drinking Water Contaminant: A Critical Review of Recent Literature, 116 ENVTL. RESEARCH, 93-117 (2012).

utero through our mothers.[39] The blood serum PFAS level for fetuses and infants is therefore determined in part by the mothers' past PFAS exposures. For infants, PFAS exposure can be further elevated through the ingestion of contaminated breastmilk (a result of the mothers' ingestion of contaminated water, and other sources of PFAS) or infant formula prepared with contaminated drinking water.[40] Infants and children are especially vulnerable to PFAS in drinking water, as they consume a disproportionate volume of drinking water for their body weight.[41] Fetuses, infants, and children and are also more sensitive to these toxic contaminants.[42]

3. Even Low-Dose Exposures to PFOA, PFOS, PFNA, PFHxS, GenX and the PFAS Class Are Associated With Adverse Health Effects

A robust body of scientific evidence demonstrates the link between low dose-exposures of PFOA, PFOS, PFNA, PFHxS, and GenX and serious human health risks, including cancer and adverse immunological, developmental, and reproductive effects. In humans, elevated levels of PFAS have been linked to, among other things, testicular and kidney cancer; thyroid disease; pregnancy-induced hypertension/pre-eclampsia; liver damage; increases in serum lipids, particularly total cholesterol and low-density lipoprotein; immunological effects such as decreased antibody response to vaccines; increased risk of asthma diagnosis; increased risk of decreased fertility; and small decreases in birth weight. In animals, PFAS has also been linked to developmental toxicity such as delayed development, decreases in litter size and survival, effects on neurodevelopment, and skeletal alterations; reproductive toxicity such as delays or defects in reproductive organ development; effects on blood; and cancer.

E. There is Strong Evidence of the Adverse Health Effects of PFOA, PFOS, PFNA, PFHxS, GenX, and the PFAS Class at Low-Dose Exposures

The attached report provides the scientific basis for NRDC's recommendations regarding the proposed Maximum Contaminant Levels and Maximum Contaminant Level Goals for PFAS in New Hampshire. While the report's findings are summarized below, we

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[39] U.S. DEP'T HEALTH & HUM. SERVS., AGENCY FOR TOXIC SUBSTANCE & DISEASE REGISTRY, Toxicological Profile for Perfluoroalkyls, Draft for Public Comment (June 2018).

[40] M. Llorca *et al.*, Infant Exposure of Perfluorinated Compounds: Levels in Breast Milk and Commercial Baby Food, 36 ENVTL. INT. 6, 584-592 (2010).

[41] *See* Anna Reade, Tracy Quinn, and Judith Schreiber, Scientific and Policy Assessment for Addressing Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water (2019).

[42] *Id.*

encourage the State to review the attached report in its entirety for a more complete analysis of the health effects associated with these chemicals.

III. Recommendations

As explained in more detail in the attached report, we believe New Hampshire should set maximum contaminant level goals (MCLGs) and enforceable maximum contaminant levels (MCLs) as follows:

RECOMMENDED STANDARDS

CONTAMINANT	MCLG	MCL OR TREATMENT TECHNIQUE
PFOA	0	2 ppt
PFOS	0	2 ppt
PFNA	0	2 ppt
PFHxS	0	2 ppt
GenX	0	5 ppt
TOTAL PFAS	0	Treatment Technique: Reverse Osmosis or Equivalent

A. PFAS' Demonstrated Carcinogenic Potential Compels a Maximum Contaminant Level Goal of Zero for PFOA, PFOS, PFNA, PFHxS, GenX, and the PFAS Class

Numerous toxicological studies in humans and animals have found associations between exposure to PFOA and PFOS and increased cancer risk. Several authoritative bodies have made findings on the chemicals' carcinogenic potential. PFOA, for example, has been identified as a probable human carcinogen by the C8 Science Panel.[43] Based on epidemiologic and other data, the C8 Science Panel concluded that there is a probable link between PFOA exposure and testicular cancer and kidney cancer, in addition to an array of other adverse health effects, including high cholesterol, thyroid disease and pregnancy-induced hypertension.

[43] *See* C8 Science Panel, The Science Panel Website (last updated Jan. 4, 2017), http://www.c8sciencepanel.org/index.html.

PFOA has also been classified as a possible human carcinogen by the World Health Organization's International Agency for Research on Cancer.[44] Human studies considered by the International Agency for Research on Cancer found an exposure relationship between PFOA and kidney and testicular cancer. Additionally, the EPA Office of Water and the EPA Science Advisory Board has determined that PFOA and PFOS demonstrate suggestive and likely evidence of carcinogenic potential, respectively.[45]

While PFNA, PFHxS, and GenX are less studied, existing data and the chemical similarity between those three chemicals to PFOA and PFOS, and the limited existing data, suggests that all five contaminants contribute to increased cancer risk. Indeed, EPA employed parallel reasoning in finding that the entire class of PCB compounds demonstrated cancer risk potential, based on limited data showing statistically significant evidence of carcinogenicity only in PCBs that were 60 percent chlorinated, given the structural complexity of the compounds, and the incomplete data available regarding toxicity of the isomers in PCB compounds.[46] On that basis, EPA established an MCLG of zero the entire class of PCB compounds.[47]

There is, therefore, no known safe threshold of exposure with an adequate margin of safety for exposure to PFOA, PFOS, PFNA, PFHxS, and GenX. Consistent with the U.S. Environmental Protection Agency's (EPA) approach of setting the Maximum Contaminant

[44] Int'l Agency for Research on Cancer, Monograph: Perfluorooctanoic Acid (updated Dec. 22, 2016), https://monographs.iarc.fr/iarc-monographs-on-the-evaluation-of-carcinogenic-risks-to-humans-6/.

[45] U.S. ENVTL. PROT. AGENCY, Drinking Water Health Advisory for Perfluorooctanoic Acid (PFOA), EPA DOC. NO. 822-R-16-005, at 24 (May 2016), https://www.epa.gov/sites/production/files/2016-05/documents/pfoa_health_advisory_final_508.pdf; U.S. ENVTL. PROT. AGENCY, Drinking Water Health Advisory for Perfluorooctane Sulfonate (PFOS), EPA DOC. NO. 822-R-16-004, at 24-25 (May 2016), https://www.epa.gov/sites/production/files/2016-05/documents/pfos_health_advisory_final_508.pdf.

[46] Fed. Reg. Vol. 56 No. 20, 1991.

[47] 40 C.F.R. § 141.50(a)(16) (PCB MCLG of zero); *id.* § 141.61(c)(15) (MCL for total PCBs is 500 ppt).

Level Goal[48] at zero for chemicals that are known or probable human carcinogens,[49] NRDC recommends a combined Maximum Contaminant Level Goal of zero for PFOA, PFOS, PFNA, PFHxS, and GenX.

B. Even Without Considering Cancer Effects, the Maximum Contaminant Level Goal for PFOA, PFOS and PFNA Should Be Set to Below 1 ppt; a Maximum Contaminant Level Goal for PFHxS and GenX Should Be Set at 2 ppt.

Even if NHDES declined to recognize the strong link between PFAS exposure and cancer effects, reliance on a "reference dose," based on the "most sensitive endpoint" at which adverse health effects linked to exposure occur would still result in extremely low Maximum Contaminant Level Goals for PFOA, PFOS, and PFNA (below 1 ppt) and for PFHxS and GenX (2 ppt).

Differences in the selection of critical endpoints, the application of uncertainty factors, and selection of drinking water exposure assumptions have led to the generation of different health thresholds for PFOA, PFOS, PFNA, PFHxS, and GenX chemicals by states and other bodies. Evidence shows that PFAS exposure poses a high risk to fetuses, infants, children and pregnant women. Sensitive members of the population face particular risk from chemicals of such persistence, and which demonstrate clear adverse effects at very low levels of exposure. Were NHDES to decline to recognize persuasive evidence of the carcinogenic potential of PFAS, NHDES should develop a health threshold most 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.[50]

NRDC's scientific analysis – as explained in greater detail in the attached report – shows that a risk assessment based on the most sensitive health endpoint, the full acknowledgement of uncertainty, including the application of an additional uncertainty factor of 10 to protect fetuses, infants and children as recommended by the National

[48] Under section 1412 of the Safe Drinking Water Act, EPA regulates drinking water contaminants by first setting a Maximum Contaminant Level Goal based on health effects data. The Maximum Contaminant Level Goal is the maximum level of a contaminant in drinking water at which no known or anticipated adverse effect on the health of persons would occur, allowing an adequate margin of safety.

[49] *See* 56 Fed. Reg. 20, 3532-33 (Jan. 30, 1991).

[50] Landrigan P and Goldman L, 2011. Children's Vulnerability to Toxic Chemicals: A Challenge and Opportunity to Strengthen Health and Environmental Policy. *Health Affairs* 30(5):842-850

Academy of Sciences[51] and as required in the Food Quality Protection Act,[52] and the selection of exposure parameters that protect breastfeeding or formula-fed infants, would result in a Maximum Contaminant Level Goal well below 1 ppt for PFOA, based on altered mammary gland development;[53] well below 1 ppt for PFOS, based on immunotoxicity;[54] below 1 ppt for PFNA, based on decreased body weight and delayed development;[55] approximately 2 ppt for PFHxS, based on evidence of thyroid toxicity;[56] and approximately 2 ppt for GenX, based on evidence of liver toxicity.[57]

C. Based on Limits in Detection Sensitivity, NHDES Should Set a Maximum Contaminant Level of 2 ppt for PFOA, PFOS, PFNA, PFHxS, and GenX

NRDC recommends a Maximum Contaminant Level of 2 ppt for combined concentrations of PFOA, PFOS, PFNA, and PFHxS consistent with the federal framework for promulgating Maximum Contaminant Levels at a level as close as possible to the Maximum Contaminant Level Goal.

A review of the best technologies available for detection and treatment of PFOA, PFOS, PFNA, PFHxS, GenX and related PFAS, establishes that a detection sensitivity of below 1 ppt and a reporting limit of 2 ppt are achievable[58] with EPA Method 537.1.[59] A statutorily-recognized[60] filtration technique, granular activated carbon (GAC), has been demonstrated, with sufficient regeneration frequency, to remove PFOA, PFOS, PFNA,

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[51] Nat. Acad. of Sci., Nat. Res. Council, Pesticides in the Diets of Infants and Children (1993).

[52] 21 U.S.C. § 346a(b)(2)(C)(ii)(II).

[53] *See* Reade, Quinn & Schreiber, *supra* note 41.

[54] *See* Reade, Quinn & Schreiber, *supra* note 41.

[55] *See* Reade, Quinn & Schreiber, *supra* note 41.

[56] *See* Reade, Quinn & Schreiber, *supra* note 41.

[57] *See* Reade, Quinn & Schreiber, *supra* note 41.

[58] For PFOA, PFOS, PFNA and PFHxS *see* http://greensciencepolicy.org/wp-content/uploads/2017/12/Andy_Eaton_UCMR3_PFAS_data.pdf.

[59] *See* Envtl. Prot. Agency, EPA Drinking Water Research Methods (updated Nov. 27, 2018), https://www.epa.gov/water-research/epa-drinking-water-research-methods.

[60] The Safe Drinking Water Act states that "granular activated carbon is feasible for the control of synthetic organic chemicals, and any technology, treatment technique, or other means found to be the best available for the control of synthetic organic chemicals must be at least as effective in controlling synthetic organic chemicals as granular activated carbon." 42 U.S.C. §300g-1.

PFHxS, and GenX to below detection levels, in addition to other techniques such as reverse osmosis. As such, NRDC recommends a Maximum Contaminant Level of 2 ppt for combined concentrations of PFOA, PFOS, PFNA, and PFHxS (based on reliable quantifiable detection levels for these compounds), consistent with the federal framework for promulgating Maximum Contaminant Levels at a level as close as possible to the Maximum Contaminant Level Goal. Maximum Contaminant Levels of 2 ppt for PFOA, PFOS, PFNA, and PFHxS, and 5 ppt for GenX, would also be the standards "acceptable in water for human consumption," under New Hampshire state law.[61]

D. Because of the Growing Scientific Evidence that PFAS as a Class Collectively Pose Similar Threats to Human Health and the Environment, NHDES Should Set a Maximum Contaminant Level Goal of Zero and a Treatment Technique for Total PFAS

There is growing evidence that PFAS as a class collectively pose similar threats to human health and the environment as PFOA, PFOS, PFNA, PFHxS, and GenX. The PFAS class of chemicals is characterized by extreme persistence, high mobility, and is associated with a multitude of different types of toxicity at very low levels of exposure. The 2014 Helsingør and 2015 Madrid Statements, founded on extensive reviews of the scientific literature, provided consensus from more than 200 scientists on the potential for harm associated with the entire class of PFAS.[62] Several adverse health outcomes have been reported for other PFAS in both animal and human studies. These include increased serum lipids (PFDeA), decreased antibody response (PFDeA, PFUA and PFDoA), liver and/or kidney damage (PFBS, PFHxA, and PFUA), decreased body weight (PFDoA, PFDeA, and PFUA), endocrine disruption (PFDeA, PFBS, and PFBA), developmental toxicity (PFDeA, PFHxA, PFUA, PFDoA, PFBS, and PFBA), reproductive toxicity (PFBS), and effects on blood (PFUA, PFBS, and PFBA), similar to findings for PFOA, PFOS, PFNA, and PFHxS.

Because the PFAS class is characterized by extreme persistence, high mobility, and is associated with a multitude of different types of toxicity at very low levels of exposure, it poses a threat to human health and the environment. A Maximum Contaminant Level Goal of zero should be set for the class to protect public health and avoid a "whack a mole" problem whereby dangerous PFAS are swiftly replaced by one another and regulatory action fails to keep pace. Many complex PFAS have the potential to break down into less

[61] *See* N.H. Rev. Stat. § 485:3, 1(b).

[62] M. Scheringer *et al.*, *Helsingør Statement on Poly- and Perfluorinated Alkyl Substances (PFASs)*, 114 CHEMOSPHERE, 337-339 (2014); A Blum *et al.*, *The Madrid Statement on Poly- and Perfluoroalkyl Substances (PFASs)*, 123 ENVTL. HEALTH PERSP. 5, A107-A111 (2015).

complex perfluoroalkyls (PFAAs), a subgroup of PFAS for which there are substantial known health risks, including PFOA and PFOS. These problems are compounded by the fact that the production of certain PFAS, such as fluoropolymers, requires the use of PFAAs in their manufacture. This use increases total PFAA contamination and exposure through industrial discharge, as well as through impurities in PFAS-containing products.

Regulation of PFAS as a class would not represent the first time that EPA or NHDES has regulated a class of toxic chemicals with a single standard for the entire class. As mentioned above, EPA found that the class of PCB compounds demonstrated cancer risk potential, based on limited data showing statistically significant evidence of carcinogenicity only in PCBs that were 60 percent chlorinated, given the structural complexity of the compounds, and the incomplete data available.[63] Thus, EPA established, and New Hampshire has adopted, a Maximum Contaminant Level of 500 parts per trillion (ppt) for total PCBs.[64]

Therefore, a Treatment Technique for the class should be set within two years, based on the best detection and treatment technologies available. At present, there is no single methodology for isolating, identifying, and quantifying all PFAS in drinking water. NRDC recommends that the state explore an analytical method, or combination of methods, that can be used as a surrogate for total PFAS. In particular, NRDC recommends that NHDES evaluate alternative detection methodologies, such as the total oxidizable precursor assay (TOPA), to measure the concentration of non-discrete and difficult to measure PFAS compounds that are not determined by conventional analytical methods. NRDC additionally recommends reverse osmosis as the treatment technique for public water supplies, or another treatment method that has been demonstrated to be at least as effective as reverse osmosis for removing all identified PFAS chemicals.

IV. Conclusion

Regulating PFAS in drinking water in New Hampshire is long overdue, and NRDC commends the state for taking steps towards regulating certain chemicals within the PFAS family. The serious adverse effects of PFAS exposure and the confirmed highly elevated drinking water concentrations compel NHDES to, under the New Hampshire Safe Drinking Water Act[65] and the New Hampshire Administrative Procedure Act,[66] promptly issue a final

[63] Fed. Reg. Vol. 56 No. 20, 1991.

[64] 40 C.F.R. § 141.61(c)(15) (MCL for total PCBs); *see also* N.H. Env-Ws 327.54 (governing monitoring of PCBs in New Hampshire).

[65] N.H. Rev. Stat. § 485:16-e; *id.* § 485:3, 1(b).

[66] N.H. Rev. Stat. § 541-A.

rule adopting a protective combined Maximum Contaminant Level and Maximum Contaminant Level Goal for PFOA, PFOS, PFNA, and PFHxS at 2 ppt and zero respectively. We further recommend that on a separate track, NHDES establish, within two years, a Maximum Contaminant Level Goal of zero for the entire PFAS class and a Treatment Technique for total PFAS based on the best available detection and treatment technologies. If NHDES declines to regulate total PFAS, the agency should set a Maximum Contaminant Level of 5 ppt for the PFAS substance GenX, and set additional health-protective Maximum Contaminant Levels for other PFAS contaminants detected in the state's

Please do not hesitate to contact us if you wish to discuss these recommendations further.

Sincerely,

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EXHIBIT B

PFAS in Drinking Water 2019

Scientific and Policy Assessment for Addressing Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water

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April 12, 2019

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

LIST OF DISCUSSION BOXES

EXECUTIVE SUMMARY

Over the past few decades per- and poly-fluoroalkyl substances (PFAS) contamination has grown into a serious global health threat. PFAS are a large class of several thousand chemically-related synthetic chemicals that are widely used for their water- and oil-repellant properties in a variety of industrial processes and consumer goods. A defining feature of PFAS is their carbon-fluorine bonds, which impart high thermal stability and resistance to degradation. PFAS are also highly mobile in the environment and many have been found to bioaccumulate, or build up, in humans and animals. People are concurrently exposed to dozens of PFAS chemicals daily through their drinking water, food, air, indoor dust, carpets, furniture, personal care products, and clothing. As a result, PFAS are now present throughout our environment and in the bodies of virtually all Americans.

PFAS are associated with many serious health effects such as cancer, hormone disruption, liver and kidney damage, developmental and reproductive harm, changes in serum lipid levels, and immune system toxicity - some of which occur at extremely low levels of exposure. Additionally, because PFAS are chemically related, they may have additive or synergistic effects on target biological systems within our bodies.

Despite the known health impacts and known contamination in people's homes and in the environment, no enforceable national drinking water standards have been set. The few, mostly non-enforceable, advisories or guidelines that do exist at the federal and state levels are mainly for perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS). PFOA and PFOS are the most extensively studied PFAS to-date and, as such, their toxicity has been well characterized in humans and animal models. Although the database for other PFAS is not as robust as for PFOA and PFOS, evidence is growing quickly that indicates they collectively pose similar threats to human health and the environment, often at exceedingly low doses. These toxicity data, combined with concerns over their similar environmental mobility and persistence and widespread human and environmental exposure, have led independent scientists and other health professionals from around the globe to express concern about the continued and increasing production and release of PFAS.

The purpose of this report is to provide relevant scientific information which will help states make informed decisions about how to protect its citizens. This report discusses the most critical health effects known to be associated with PFAS, the risk of additive/synergistic effects from concurrent exposure to multiple PFAS, existing or proposed standards and advisories, and detection and treatment technologies available. Special attention has been given to comparing and analyzing existing or proposed standards and advisories, from which our recommendations arise. For this analysis, we focused on PFOA and PFOS, and two additional PFAS, perfluorononanoic acid (PNFA), and perfluorohexane sulfonic acid (PFHxS), because the Agency for Toxic Substances and Disease Registry has generated minimal risk levels for all four. GenX chemicals, used as a replacement for PFOA, were also analyzed in this report, as their toxicity was recently assessed by the US Environmental Protection Agency (EPA).

Our analysis of current literature and standards/advisories for PFOA, PFOS, PFNA, PFHxS, and GenX show that existing standards and advisories are not health protective. For example, Michigan's PFAS Science Advisory Panel concluded that, "*the research supports the potential for health effects resulting from long term exposure to drinking water with concentrations below 70 ppt*" (the EPA's lifetime health advisory for PFOA and PFOS). If toxicity assessments were based on the most sensitive health effect, protective of the most vulnerable population, and fully acknowledged uncertainties in the toxicity assessment process, maximum contaminant level goals (MCLGs)[a], which are to be set at a level fully protective of human health, would range from 0 to 2 ppt for drinking water. As technology for detection and water treatment do not currently allow for the complete removal of PFAS from drinking water, maximum contaminant levels (MCLs)[b] for PFOA, PFOS, PFNA, PFHxS, and GenX should be based on the best detection and treatment technologies available. Our review of detection and treatment capabilities suggests, a combined MCL of 2 ppt is feasible for PFOA, PFOS, PFNA, and PFHxS, with a separate MCL of 5 ppt for GenX.

However, we conclude that setting a MCLG of zero for the class is needed to provide an adequate margin of safety to protect public health from a class of chemicals that is characterized by extreme persistence, high mobility, and is associated with a multitude of different types of toxicity at very low levels of exposure. If only a handful of PFAS are regulated, there will be swift regrettable substitution with other, similarly toxic PFAS - creating an ongoing problem where addressing one chemical at a time incentivizes the use of other toxic chemicals and we fail to establish effective safeguards to limit this growing class of dangerous chemicals.

The problems with PFAS as a class are highlighted by the fact that many complex PFAS have the potential to break down into less complex perfluoroalkyl acids (PFAAs), a subgroup of PFAS that includes PFOA and PFOS, for which there are substantial known health risks. These problems are compounded by the fact that the production of certain PFAS, such as fluoropolymers, requires the use of PFAAs in their manufacture. This use increases total PFAA contamination and exposure through industrial discharge, as was seen with the production of Teflon®, as well as through impurities in PFAS-containing products.

At present, there is no single methodology for isolating, identifying, and quantifying all PFAS compounds in drinking water. We recommend that the state explore an analytical method, such as total oxidizable precursor assay (TOPA)[c], or combination of methods, that can be used as a surrogate for total PFAS. Until a comprehensive analytical method has been approved to

[a] An MCLG is the maximum level of a contaminant in drinking water at which no known or anticipated adverse effect on the health of persons would occur, allowing an adequate margin of safety. MCLGs are non-enforceable health goals and consider only public health and not the limits of detection and treatment technology effectiveness.
[b] An MCL is the legal threshold of the amount of a chemical that is allowed in public water systems under the Safe Drinking Water Act. An MCL is based on the concentration established by its corresponding MCLG, but may be adjusted up for feasibility reasons, reflecting difficulties in measuring small quantities of a contaminant, or a lack of available, adequate treatment technologies.
[c] TOPA estimates the full array of potential polyfluoroalkyl acid (PFAA) precursors in a sample. TOPA replicates what micro-organisms in the environment would achieve after many years by rapidly converting precursors into PFAAs such as PFOA, using a hydroxyl radical-based chemical oxidation method.

quantify PFAS compounds as a class, we recommend reverse osmosis, or other treatment method at least as effective as reverse osmosis, as a treatment technique – an enforceable treatment procedure to ensure contamination control - for public water supplies. Reverse osmosis is the preferred treatment technology because it has been demonstrated to effectively remove a broad range of PFAS compounds, it is the most robust technology for protecting against unidentified contaminants, and it does not require frequent change out of treatment media or release elevated concentrations of pollutants after media is spent. We recommend the evaluation of the safest disposal method for high-strength waste streams and spent/used membranes, and that disposal require full destruction of PFAS compounds before entering the environment.

In summary, this report finds that the current available scientific evidence supports the need for:

1) comprehensive testing of drinking water;

2) a maximum contaminant level goal of zero for total PFAS;

3) a combined maximum contaminant level of 2 parts per trillion (ppt) for PFOA, PFOS, PFNA, and PFHxS, and a maximum contaminant level of 5 ppt for GenX; and

4) the setting of a Treatment Technique – an enforceable treatment procedure to ensure contamination control – for the PFAS class based on the best available detection and treatment technologies.

INTRODUCTION

Per- and poly-fluoroalkyl substances (PFAS) are synthetic chemicals that are widely used in a variety of industrial processes and consumer goods. The carbon-fluorine bonds in PFAS impart high thermal stability and resistance to degradation. While useful chemicals, PFAS are highly resistant to environmental degradation and persist in the environment. As a result, PFAS are now present throughout our environment and in the bodies of virtually all people.

PFAS have been associated with a wide variety of adverse health effects including cancer, hormone disruption, liver damage, developmental harm, and immune system toxicity - some of which occur at extremely low levels of exposure. PFAS are widely prevalent in drinking water sources across the country. Consequently, there is an urgent need to take action to address this growing health threat. Yet, there are still no enforceable regulations for PFAS in drinking water at the federal level, and very few regulations addressing PFAS in drinking water at the state level.

In response to a national PFAS contamination crisis in drinking water, this report provides a summary of relevant scientific information on PFAS, including information on PFAS exposure, their effects on human health, and how existing or proposed standards and advisories have been developed. Based on this information, we make recommendations on how states can protect the health of their citizens by addressing PFAS contamination in its drinking water.

This report is organized into six parts: Part I is an introduction to the PFAS class of chemicals. Part II provides an overview of the widespread presence of PFAS in drinking water and in people. Part III discusses the health risks associated with PFAS exposure. Part IV compares and analyzes existing health thresholds set or recommended for levels of certain PFAS (PFOA, PFOS, PFNA, PFHxS and GenX chemicals[d]). Part V provides an overview of detection/analytical methods and treatment technologies for PFAS removal from water. Part VI offers conclusions and recommendations on how PFAS contamination in drinking water can be addressed.

PART I: WHAT ARE PFAS

PFAS are a large class of synthetic fluorochemicals that are widely used for their water- and oil-repellant properties. PFAS can be found in consumer products such as non-stick cookware, clothing, leather, upholstery, and carpets; in paints, adhesives, waxes and polishes; in aqueous

[d] As explained by the U.S. Environmental Protection Agency, "GenX is a trade name for a processing aid technology developed by DuPont (now Chemours). In 2008, EPA received new chemical notices under the Toxic Substance Control Act from DuPont (which is now Chemours) for two chemical substances that are part of the GenX process (Hexafluoropropylene oxide (HFPO) dimer acid and the ammonium salt of HFPO dimer acid)." See EPA, GenX Chemicals Studies, available online at https://www.epa.gov/pfas/genx-chemicals-studies, visited December 4, 2018.

fire-fighting foams; and industrially as surfactants, emulsifiers, wetting agents, additives and coatings.[1,2,3]

A defining feature of PFAS are their carbon-fluorine bonds, which impart high thermal stability and resistance to degradation.[4,5] As a result, PFAS are highly resistant to environmental degradation and persist in the environment. They are relatively water-soluble and have been detected in drinking water sources and in finished (treated) drinking water. Due to their water solubility, after exposure by any route, these chemicals are found in human blood serum rather than in body fat where fat-soluble persistent organic pollutants such as PCBs reside. With half-lives of years, PFAS persist in humans and are found in the blood serum of almost all US residents and populations worldwide.[2,6] PFAS are commonly found together in samples from contaminated water[7] and are identified as co-contaminants in blood serum.[6]

The two most well-known PFAS, perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS), were manufactured between the 1940s and mid-2010 when they were voluntarily phased out from U.S. manufacturing due to health concerns.[8] However, PFOA and PFOS are still manufactured and used internationally and may enter the U.S. through imported goods.[9] There is widespread contamination of PFOA and PFOS in the environment and their toxicity has been well characterized in humans and animal models.[5] PFOA and PFOS are the most extensively studied PFAS to-date, and as such, they are often the only PFAS chemicals with exposure guidelines in drinking water or other environmental media.

However, issues related to the entire PFAS class, which has now grown to an estimated 4,700 chemicals, have been of increasing concern for researchers and health authorities.[10,11,12] Although there is not a robust toxicity database for the suite of PFAS, it is generally recognized that these chemicals are structurally similar, and it is reported that the health risks associated with one PFAS are expected for other PFAS as well.[2,10,13,14] Moreover, as discussed below, many PFAS have the potential to convert into perfluoroalkyl acids (PFAAs), a subgroup of PFAS that includes PFOA and PFOS, for which there are substantial known health risks. Health risks of PFAS include cancer, immune system disfunction, liver damage, hormone disruption, low birth weight and other developmental effects, changes in serum lipid levels, and reproductive harm.[5] While some scientific uncertainties exist, the weight of scientific evidence is substantial: in experimental animals, in exposed residential populations drinking contaminated water, and in occupational studies, PFOA, PFOS, and related PFAS cause adverse health effects, particularly on the young, and increase cancer risks[15] in exposed populations (discussed further in Part III).

PFAS Classification

PFAS can be classified into various subgroups (see Figure 1 below for a simplified classification diagram).[10] The PFAS subgroup with the most toxicological information is perfluoroalkyl acids (PFAAs), which includes PFOA and PFOS.[5] Another PFAS subgroup is PFAA precursors, which consists of PFAS that can be converted into PFAAs.[16,17] PFAA precursors include fluorotelomer-based substances and PASF (perfluoroalkane sulfonyl fluoride)-based substances.

In a recent review of the global distribution of PFAS, authors concluded that PFAA precursors should be given attention in addition to PFOA, PFOS and other PFAAs.[18] For example, one PFAA precursor subgroup, polyfluorinated phosphate esters (PAPs), are not routinely measured or widely investigated, however recent studies show that they are present in house dust, sometimes at extremely high levels that exceed other PFAS subgroups.[19] Additionally, PAPs were found to be incorporated into produce, such as pumpkin, grown on contaminated soils.[20] PFAA precursors can pose health risks associated with their precursor form and when broken down into PFAAs. Germany and Sweden have proposed a restriction under REACH (a 2006 European regulation that addresses the registration and production of chemical substances) to cover six PFAS and any substance that can degrade into one of the six. The Swedish Chemicals Agency estimates that the restriction will cover a group of about 200 PFAS.[21]

Figure 1: Simplified Classification of PFAS Class

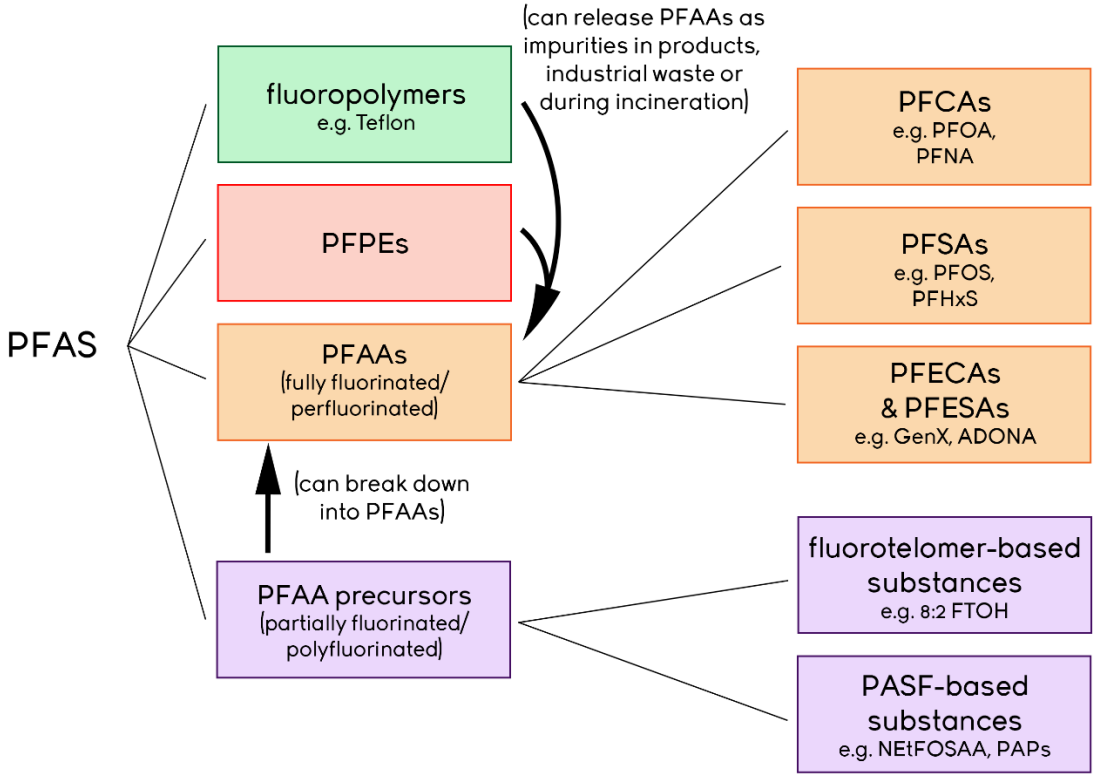

Figure 1 shows the relationship between various subgroups within the PFAS class. This classification scheme is not inclusive of all PFAS subgroups. PFAS (per- and polyfluoroalkyl substances), PFPEs (perfluoropolyethers), PFAAs (perfluoroalkyl acids), PFCAs (perfluoroalkyl carboxylic acids), PFSAs (perfluoroalkyl sulfonic acids), PFECAs (perfluoroether carboxylic acids), PFESAs (perfluoroether sulfonic acids), PASF (perfluoroalkane sulfonyl fluoride).

Perfluoropolyethers (PFPEs) are large molecular sized PFAS with ether linkages and fluoropolymers are composed of multiple repeating units of PFAS.[10,17] While neither are known to actively degrade into PFAAs, they are highly persistent and PFAAs are used in their manufacture, can occur as impurities in the final product, and can be formed when the polymers are heated or incinerated. A well-known fluoropolymer is polytetrafluoroethylene, also known as Teflon. The use of PFAAs such as PFOA and GenX chemicals in the manufacture of perfluoropolyethers and fluoropolymers has resulted in severe environmental contamination around manufacturing and processing plants.[22]

There is concern that simply substituting one PFAS that has been shown to be toxic for another, often less studied PFAS, will result in a regrettable substitution that is not protective of public health. Regrettable substitutions of certain PFAS compounds with others demonstrating similar toxicological characteristics have already occurred. For example, GenX is a replacement technology for PFOA and perfluorobutane sulfonic acid (PFBS) is a replacement for PFOS. The US Environmental Protection Agency (EPA) released draft toxicity assessments in November of 2018 on two GenX chemicals (hexafluoropropylene oxide (HFPO) dimer acid and its ammonium salt) and PFBS confirming that GenX chemicals are associated with liver and pancreatic cancers and adverse effects on the kidneys, blood, liver, immune system, and development.[23] In addition, PFBS is associated with thyroid and kidney effects and reproductive and developmental toxicity.[24]

Table 1: Replacements for PFOA and PFOS are Associated with Similar Health Effects

	Cancer	Immune	Liver or Kidney	Developmental & Reproductive	Endocrine
PFOA	●	●	●	●	●
GenX	●	●	●	●	
PFOS	●	●	●	●	●
PFBS		○	●	●	●

Table 1 compares several health effects associated with exposure to PFOA and its replacement GenX, and PFOS and its replacement PFBS. Based on human and animal evidence (not inclusive of all associated health effects).[e,f,g]

Indeed the EPA, in an evaluation of alternative PFAS to PFOA and PFOS, stated that there is, *"concern that these … substances will persist in the environment, could bioaccumulate, and be toxic ("PBT") to people, wild mammals, and birds."*[25] The Michigan PFAS Science Advisory

[e] ATSDR, 2018. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Perfluoroalkyls. Draft for Public Comment, June 2018.
[f] U.S. Environmental Protection Agency, 2018. Toxicity Assessment: Human Health Toxicity Values for Hexafluoropropylene Oxide (HFPO) Dimer Acid and Its Ammonium Salt (CASRN 13252-13-6 and CASRN 62037-80-3). November 2018. EPA 823-P-18-001.
[g] U.S. Environmental Protection Agency, 2018. Toxicity Assessment: Human Health Toxicity Values for Perfluorobutane Sulfonic Acid (CASRN 375-73-5) and Related Compound Potassium Perfluorobutane Sulfonate (CASRN 29420-49-3). November 2018. EPA 823-R-18-0307.

Panel has recommended that, although there is limited data on PFAS other than PFOA and PFOS, Michigan should "consider setting advisory limits for these additional PFAS in light of their similar chemical structures and toxicity."[26] Vermont is in the process of setting a combined standard for drinking water for 5 PFAS based on their structural and chemical similarity. Furthermore, the 2014 Helsingør[11] and 2015 Madrid Statements,[12] founded on extensive reviews of the scientific literature, provide consensus from more than 200 scientists on the potential for harm associated with the entire class of PFAS.

PART II: HOW ARE PEOPLE EXPOSED TO PFAS

Almost all Americans tested have one or more PFAS in their bodies.[6,27] Widespread use of PFAS has resulted in the ubiquitous presence of these chemicals in the environment including in rivers, soil, air, house dust, food and drinking water from surface water and groundwater sources. We are exposed to PFAS by inhaling house dust contaminated with PFAS due to their use in consumer products, such as treated upholstery and carpet, and from ingesting small amounts in drinking water, food and food packaging.

PFAS in People

Persistent, bioaccumulative chemicals such as those in the PFAS family are characterized by long periods during which the body retains these chemicals after exposure ceases.[3,5,28] PFOA, PFOS, PFNA, PFHxS, and related PFAS are known to bioaccumulate in the bodies of people of all ages, even before birth. Government agencies estimate the human adult half-life (the time it takes to reduce the concentration of a chemical by half) of various PFAS to be on the order of years. Half-life estimates for the PFAS discussed in this report are: 2.3 to 3.8 years for PFOA; 5.4 years for PFOS, 8.5 years for PFHxS, and 2.5 to 4.3 years for PFNA.

The use of PFOA and PFOS in manufacturing has been phased out in the United States, and levels in blood serum have started to decrease as reported in national surveys.[6] However, PFOA and PFOS bioaccumulate and do not degrade in the environment, therefore they will persist in the environment and continue to be a source of exposure for many years in the future.

Blood serum can be used as a long-term measure of exposure for some PFAS and can indicate an increase in risk of disease at the population level. Blood serum concentrations of several PFAS have been evaluated in a large representative sample of the US populations age 12 and older by the National Health and Nutrition Examination Survey (NHANES).[6] The table below (Table 2) summarizes the geometric mean blood serum concentration in ng/L, or parts per trillion (ppt), of different PFAS measured by NHANES since 1999. Note that blood serum concentration is usually expressed in ppb (ug/L or ng/mL) but was converted to ppt in this report to facilitate comparisons to drinking water levels, usually reported in ppt for PFAS.

Table 2: Results of NHANES Biomonitoring Data

Survey Year	PFBS	PFDA	PFDoA	PFHpA	PFHxS	PFNA
1999-2000	NA	*	*	*	2130	551
2003-04	*	*	*	*	1930	966
2005-06	*	355	*	*	1670	1090
2007-08	*	286	*	*	1950	1220
2009-10	*	279	*	*	1660	1260
2011-12	*	199	*	*	1280	881
2013-14	*	185	*	*	1350	675
Survey Year	**PFOA**	**PFOS**	**PFOSA**	**EtFOSAA**	**MeFOSAA**	**PFUA**
1999-2000	5210	30400	355	642	846	*
2003-04	3950	20700	*	*	*	*
2005-06	3920	17100	*	*	410	*
2007-08	4120	13200	*	*	303	*
2009-10	3070	9320	*	*	198	172
2011-12	2080	6310	*	*	*	*
2013-14	1940	4990	NA	NA	*	*

Table 2 shows the geometric mean levels in blood serum in ng/L (ppt) from NHANES biomonitoring data. "" indicates mean was not calculated, proportion of results below limit of detection was too high to provide a valid result. "NA" indicates the PFAS was not measured in that round of NHANES.*

State and regional biomonitoring trends, as well as trends among different age groups and sexes can differ from the national trends represented in NHANES. For example, one study found that children 2 to 5 years old and adults over 60 had a higher blood serum PFOA (median 600 ppb) in the Little Hocking Water Association district compared with residents in all other age groups (median 321 ppb).[29] The authors note that infants and children proportionally drink more water per unit of body weight than adults, and children and the elderly tend to spend more time at home with exclusive use of residential water than other age groups. Additionally, NHANES biomonitoring measures a limited number of PFAS and is likely not reflective of current exposures to PFAS. Alternative methods for detecting PFAS in blood serum are showing an increasing trend of unidentified organofluorine in blood serum samples, which suggest that people are being exposed to new and unidentified PFAS.[30,31]

Fetal and Infant Exposure to PFAS

Fetuses, infants and children are particularly susceptible to the impacts of exposure to toxic chemicals due to their rapidly growing and developing bodies. As such, they are at increased risk of harmful health effects due to PFAS exposure (discussed in further detail in Part II of this

report). Almost all fetuses and infants will have some degree of exposure to PFAS, including fetal exposure during pregnancy through placental transfer.[2,5] For infants, PFAS exposure may be further elevated due to ingestion of contaminated breast milk (a result of the mother's ingestion of contaminated water, and other sources) or infant formula contaminated by PFAS-containing food packaging and/or prepared with contaminated drinking water.[32,33] Fetuses and nursing infants' exposures are influenced by the mother's past exposures or "body burden," as measured by blood serum concentrations.

PFAS have been detected in virtually all umbilical cord blood tested, indicating that PFAS can cross the placental barrier, exposing fetuses *in utero*.[5] Researchers have studied the transfer of PFAS during pregnancy and found a positive correlation between maternal plasma and serum with cord serum levels, concluding that either maternal plasma or serum could be used to estimate fetal exposure to PFAS.[34]

Infant formula can be contaminated with PFAS through the use of PFAS-contaminated water when reconstituting powdered formula. PFAS has also been detected in infant formula itself. For example, one study detected PFAS in all infant milk formulas and baby cereals tested, with the highest levels coming from PFOA, PFOS, PFNA, and PFDA.[33] Contamination of infant formula and cereal could be due to migration from food packaging and/or from containers during production.[35]

ATSDR summarizes reports on breast milk concentrations of PFAS found in the general population.[5] Numerous PFAS, including PFOS, PFOA, PFBS, PFHxS, PFNA, perfluorodecanoic acid (PFDeA), perfluorododecanoic acid (PFDoA), perfluoroundecanoic acid (PFUA), and perfluorooctanesulfonamide (PFOSA), have been detected in breast milk samples in women in China, Korea, Japan, Malaysia, Cambodia, India, Korea, Vietnam, Indonesia, Norway, Philippines, Sweden, and the United States.

PFAS levels in breast milk are higher than what is typically found in drinking water, due to the mothers' past accumulated exposures and transfer to breast milk. For example, in biomonitoring studies average concentrations of PFOA in breast milk range from 2.5%[36] to 9%[37] of the concentration of PFOA in mothers' blood serum. Therefore, breast milk concentrations can be up to an order of magnitude higher than drinking water concentrations because PFOA maternal blood serum levels are approximately 100 times greater than the drinking water she ingested over time.

PFAS in Drinking Water

Drinking water is the dominant source of exposure to PFAS for people living in communities with drinking water highly contaminated with these chemicals, far exceeding exposure from other sources.[38] Even relatively low PFAS concentrations in drinking water can be associated with substantial increases in blood serum levels. For example, since the clearance of PFOA is slow and because it accumulates in blood, after a long period of exposure, a person's blood

serum PFOA level will be about 100 times greater than the PFOA concentration ingested via drinking water.[2]

In 2009, researchers evaluated the contribution of water, diet, air and other sources for various exposure scenarios to PFOA.[38] They found that when drinking water concentrations of PFOA are low, dietary exposure is the dominant source of exposure. However, as drinking water concentrations increase, the ingestion of contaminated water becomes the predominant source of exposure. Drinking water concentrations of 100 ppt and 400 ppt are predicted to contribute 71% and 91%, respectively, of total exposure; and are estimated to increase blood serum levels, on average, by 250% and 1000%, respectively.[2]

Analysis of EPA's Unregulated Contaminant Monitoring Rule (UCMR3) data shows that about 4% of tested public water supplies in the U.S. (about 200 of 5,000 public water supplies studied), serving 16.5 million Americans in 33 states, 3 territories and an American Indian community, have levels of PFAS above the EPA-specified reporting limits[h] for UCMR3.[7] Sixty-six tested public water supplies, serving six million Americans, had at least one sample above EPA's 2016 PFOA and PFOS non-enforceable lifetime health advisory of 70 ppt.[3,28] PFOA was the most frequently detected PFAS in drinking water, followed by PFOS. Exceedances of the EPA's health advisory have been detected in California, New Jersey, North Carolina, Alabama, Florida, Pennsylvania, Ohio, New York, Georgia, Minnesota, Arizona, Massachusetts and Illinois. High levels of PFAS in drinking water were strongly associated with proximity to major PFAS industrial sites, civilian airports, and military fire training areas.

As concerning as the UCMR3 data are, they significantly underestimate how many drinking water sources are contaminated by PFAS. This is in part because the lowest levels of PFAS that are required to be reported to EPA, sometimes referred to as the "Minimum Reporting Levels" or "Method Reporting Levels" under the UCMR3 were very high, meaning that even if PFAS were detected at levels below these cutoffs, they are not required to be reported to EPA. Indeed, these cutoffs are significantly higher than the limit of quantitation reported in most published studies and by a prominent laboratory using the same method, which completed about one-third of the PFAS monitoring under the UCMR3.[39] The UCMR3's overall limitations have been well described:

> *"The [Minimum Reporting Levels] (10−90 ng/L) in the UCMR3 database are up to 2 orders of magnitude higher than the limit of quantitation in most published studies, and more than 10 times higher than the drinking water limit (1 ng/L) suggested by human and animal studies. Because PFASs are detectable in virtually all parts of the environment, we infer that the large fraction of samples below reporting limits is driven in part by high [Minimum Reporting Levels]."* [7]

Moreover, the UCMR3 only required testing for 6 PFAS out of the several thousand PFAS that have been cleared for use in the United States.[40] The UCMR3 data are further limited by the

[h] Reporting limits for UCMR3 were: PFOA - 20 ppt, PFOS - 40 ppt, PFHxS - 30 ppt, PFNA - 20 ppt, perfluorohepatanoic acid (PFHpA) - 10 ppt, and perfluorobutane sulfonic acid (PFBS) - 90 ppt

inclusion of only 0.5 % of the nation's small public water supplies and no testing results for private wells.

PART III: HEALTH RISKS ASSOCIATED WITH EXPOSURE TO PFAS

There is a sufficiently robust body of scientific research to evaluate the adverse health effects of several PFAS, with the most highly studied being PFOA, PFOS, PFNA and PFHxS. Both human studies and animal studies should be used to evaluate adverse effects of chemical exposures (see Box 8 for further discussion). Animal and human studies show similar adverse effects and cancer risks.

Due to the structural similarity and the co-occurrence of PFOA and PFOS in the environment and in people, public health protection and guidance usually address both PFOA and PFOS. In June 2018, minimal risk levels were also generated by the Agency for Toxic Substances and Disease Registry (ATSDR) for PFNA and PFHxS, which are chemically related and often co-occur with PFOA and PFOS.[5] In November of 2018, the EPA released human health toxicity values (reference doses) for PFBS and hexafluoropropylene oxide (HFPO) dimer acid and its ammonium salt, also known as GenX chemicals.[23,24] PFBS is a replacement chemical for PFOS and GenX is a replacement technology for PFOA, and both were found to be associated with a variety of adverse health effects. Considerably less information is available for the larger group of PFAS, however, as stated above, due to the structural similarity of these contaminants, it is expected that many PFAS will have similar health effects. [2,13,14]

Several reviews of the scientific literature on the health effects associated with PFAS exposure have recently been published.[1,2,5,14,15,41,42,43] ATSDR has performed the most recent and comprehensive review. This review is summarized below, as an overview of health effects associated with PFAS exposure. This summary is followed by sections that discuss in further detail cancer risk and two of the most common and sensitive health effects for PFAS, development harm and immunotoxicity. Understanding these health effects is particularly important to determining how to best protect the public from PFAS contamination.

ATSDR Draft Toxicological Profile for Perfluoroalkyls

ATSDR performs risk assessment and evaluation of chemicals as part of the U.S. Centers for Disease Control and Prevention (CDC). ATSDR released a draft Toxicological Profile for Perfluoroalkyls in June 2018.[5] The toxicological profile on perfluoroalkyl compounds included the suite of chemicals in that group that have been measured in the blood serum collected as part of the NHANES 2003-2004 survey, and other monitoring studies. The 14 perfluoroalkyl compounds included in the toxicological profile are:

Perfluorobutyric acid (PFBA, CAS 375-22-4)
Perfluorohexanoic acid (PFHxA, CAS 307-24-4)

Perfluoroheptanoic acid (PFHpA, CAS 375-85-9)
Perfluorooctanoic acid (PFOA, CAS 335-67-1)
Perfluorononanoic acid (PFNA, CAS 375-95-1)
Perfluorodecanoic acid (PFDeA, CAS 335-76-2)
Perfluoroundecanoic acid (PFUA, CAS 2058-94-8)
Perfluorododecanoic acid (PFDoA, CAS 307-55-1)
Perfluorobutane sulfonic acid (PFBS, CAS 375-73-5)
Perfluorohexane sulfonic acid (PFHxS, CAS 355-46-4)
Perfluorooctane sulfonic acid (PFOS, CAS 1763-23-1)
Perfluorooctane sulfonamide (PFOSA, CAS 754-91-6)
2-(N-Methyl-perfluorooctane sulfonamide) acetic acid (Me-PFOSA-AcOH, CAS 2355-31)
2-(N-Ethyl-perfluorooctane sulfonamide) acetic acid (Et-PFOSA-AcOH, CAS 2991-50-6)

ATSDR provided an exhaustive assessment of these 14 PFAS in their Toxicological Profile for Perfluoroalkyls. Their assessment found that there is consistent association between PFAS exposure and several health outcomes. The table (Table 3) below summarizes health effects ATSDR found linked to the 14 PFAS reviewed in the profile.

Table 3: Summary of ATSDR's Findings on Health Effects from PFAS Exposure

	Immune e.g. decreased antibody response, decreased response to vaccines, increased risk of asthma diagnosis	**Developmental & Reproductive** e.g. pregnancy-induced hypertension/pre-eclampsia, decreased fertility, small decreases in birth weight, developmental toxicity	**Lipids** e.g. increases in serum lipids, particularly total cholesterol and low-density lipoprotein	**Liver** e.g. increases in serum enzymes and decreases in serum bilirubin levels	**Endocrine** e.g. increased risk of thyroid disease, endocrine disruption	**Body Weight** e.g. decreased body weight	**Blood** e.g. decreased red blood cell count, decreased hemoglobin and hematocrit levels
PFOA	✖	✖	✖	✖	✖	✖	✖
PFOS	✖	✖	✖	✖	✖	✖	✖
PFHxS	✖			✖			✖
PFNA	✖		✖			✖	
PFDeA	✖	✖	✖	✖	✖	✖	
PFDoA	✖	✖				✖	
PFUA	✖	✖				✖	✖
PFHxA		✖					✖
PFBA		✖		✖	✖		✖
PFBS				✖			✖

Table 3 summarizes ATSDR's findings on the associations between PFAS exposure and health outcomes in human and animal studies (not an exhaustive list of health outcomes).

ATSDR determined that there was sufficient data to support generating minimal risk levels for PFOA, PFOS, PFNA, and PFHxS. Our maximum contaminant level recommendations are, in part, based on these minimal risk levels, which is discussed in Part III of this report.

Cancer Risks from PFOA, PFOS, PFNA, PFHxS, and GenX Exposure

Chemical exposures that contribute to an increase in cancer risk have a significant impact on public health. As the National Cancer Institute states, "*the years of life lost due to premature deaths, the economic burden due to lost productivity and the costs associated with illness and therapy, and the long-term effects of cancer and its treatment on the quality of life of survivors take a toll at a population level*."[44]

Toxicological studies in humans and animals have found associations between increased cancer risk and PFOA and PFOS exposure, and several authoritative bodies have made findings on their carcinogenic potential. PFNA, PFHxS, and GenX are less well studied, however, their chemical similarity to PFOA and PFOS and the data that is available suggests that there is reason to be concerned about increased cancer risk.

PFOA and PFOS

Carcinogens are chemicals that cause cancer. The C8 Science Panel[i] has identified PFOA as a probable carcinogen[15], and the International Agency for Research on Cancer (IARC) has classified PFOA as a possible[45] carcinogen. The EPA Science Advisory Board and the EPA Office of Water have concluded that PFOA and PFOS demonstrate likely[46] or suggestive[3] evidence of carcinogenic potential, respectively.

From 2005-2013 the C8 Science Panel determined blood levels and collected health information from communities in the Mid-Ohio Valley that had been potentially affected by the release of PFOA emitted from a DuPont plant since the 1950s.[15,47,48] They then assessed the links between PFOA exposure and a number of diseases. Based on epidemiologic and other data available to the C8 Science Panel, they concluded that there is a probable link between exposure to PFOA and testicular and kidney cancer (as well as high cholesterol, ulcerative colitis, thyroid disease and pregnancy-induced hypertension). Because these studies relied largely on a survivor cohort, results regarding associations with PFOA may be biased toward the null (i.e. a greater chance of failing to identify an association) for highly aggressive cancers like pancreatic, lung and kidney cancers, which should not be ruled out based on this study.

[i] The C8 Science Panel was established as a result of a class action lawsuit against DuPont and charged with assessing probable links between PFOA (also called C8) exposure and disease in communities near the DuPont Washington Works plant in Parkersburg, West Virginia.

IARC, the specialized cancer agency of the World Health Organization, has classified PFOA as "possibly carcinogenic to humans" (Group 2B) based on limited evidence that PFOA causes testicular and renal cancer, and limited evidence in experimental animals."[45] IARC considers human, animal, and mechanistic data in making its determinations of evidence for cancer risk to humans. The human data considered by IARC in making this determination included increases in cancer among highly exposed members of the C8 Health Project study population[47,48] discussed above, and among workers in the DuPont Washington Work plant in Parkersburg, WV.[49] Researchers studied the mortality of 5,791 workers at the DuPont chemical plant in Parkersburg, West Virginia from 1952-2008. The authors found exposure-response relationships with PFOA for chronic renal disease, both malignant and non-malignant.[49]

The EPA Office of Water concluded that there is suggestive evidence of carcinogenic potential of PFOA in humans.[3] This conclusion was based on Leydig cell testicular tumors in rats, and the reported probable link to testicular and renal tumors among the members of the C8 Health Project. EPA also concluded that there is suggestive evidence of carcinogenic potential of PFOS in humans based on liver and thyroid adenomas observed in a chronic rat bioassay.[28,50]

Cancers other than kidney and testicular cancer have also shown positive associations in studies of occupational exposure, though they have not reached statistical significance. One study reported a non-significant positive association between PFOA and prostate cancer in employees of DuPont in West Virginia.[51] Another study reported modestly elevated risk of prostate and bladder cancer in employees of 3M in Minnesota.[52]

Two small studies of the Inuit population in Greenland found significantly increased risk of breast cancer associated with certain PFAS, including PFOA and PFOS,[53] and a greater elevated odds ratio for breast cancer in women with both high PFAS levels and specific genetic variations that affect levels of hormones such as estrogens.[54] A later, larger study evaluated the association between PFAS serum levels in pregnant Danish women and the risk of premenopausal breast cancer.[55] This study did not find convincing evidence establishing a causal link between PFAS exposures and increased risk of breast cancer 10 to 15 years later. These data suggest the need for further research on this topic, especially considering the effects PFAS exposure can have on mammary gland development (see Box 6).

While there have been some studies that do not support a relationship between PFAS exposure and cancer, those studies have notable limitations. For example, New York State Department of Health (NYSDOH) conducted an evaluation of cancer occurrence in the Hoosick Falls population where residents' blood serum median levels were 23,500 ppt.[56] In that study, no relationship was found between PFOA exposure and testicular, kidney, prostate or bladder cancer. However, studies of community exposures have inherent limitations and are difficult to evaluate in low number populations. As noted by NYSDOH, limitations of this study include small population and incomplete inclusion of the potentially exposed populations.

PFNA, PFHxS, and GenX

PFNA and PFHxS have been studied to a lesser degree than PFOA and PFOS. One study reported a significantly higher risk for prostate cancer among subjects with a hereditary risk and blood serum PFHxS levels above the median, finding a significant odds ratio of 4.4 (1.7-12).[57] An increased, though non-significant, odds ratio of 2.1 (1.2-6.0) was also reported among subjects with a hereditary risk for prostate cancer and blood serum PFNA levels above the median.

Researchers evaluated participants in the C8 Health studies for associations between PFNA and PFHxS and elevated serum levels of prostate-specific antigen, a biomarker that can be used to screen for prostate cancer.[58,59] Their findings were non-significant, however, one limitation with this study is that changes in prostate-specific antigen levels are not exclusively due to cancer but can also be attributed to other factors such as prostate inflammation, urinary retention, local trauma and increase in age.

In EPA's draft toxicity assessment of GenX, the EPA determined that "*there is Suggestive Evidence of Carcinogenic Potential of oral exposure to GenX chemicals in humans, based on the female hepatocellular adenomas and hepatocellular carcinomas and male combined pancreatic acinar adenomas and carcinomas [in rats]*."[23] The EPA also notes that evidence suggest that mice are more sensitive to the effects of GenX than rats, and that a lack of data evaluating cancer in mice is a database deficiency. There are currently no studies evaluating cancer risk from GenX exposure in humans.

Further research is needed to understand the relationship between PFOA and PFOS exposure and various cancers other than kidney and testicular cancer, such as prostate, bladder, ovarian and breast cancer, which have limited, but suggestive evidence for association with PFAS exposure. Additionally, more research is needed to understand the carcinogenic potential of other PFAS, which, due to similar chemical characteristics to PFOA and PFOS, are likely to also increase the risk for certain cancers.

Risks to Fetal Development and the Young

Developing infants and children are particularly susceptible to the impacts of exposure to toxic chemicals. The impacts of PFAS exposure on fetal development and the young have been studied in both humans and animals. These studies find similar and profound adverse health effects.

Since infants and children consume more water per body weight than adults, their exposures may be higher than adults in communities with PFAS in drinking water. In addition, the young may also be more sensitive to the effects of PFAS due to their immature developing immune system, and rapid body growth during development.[1,5,60,61,62] Exposure to PFAS before birth or in early childhood may result in decreased birth weight, decreased immune responses, and hormonal effects later in life.

Recent literature has identified developmental effects of significance from exposure to PFAS. For a review of effects on children from PFAS exposure, sixty-four studies were evaluated for six categories of health outcome: immunity, infection, asthma, cardio-metabolic, neurodevelopmental/attention, thyroid, renal, and puberty onset.[62] The review found evidence of later age at menarche (menstruation), effects on renal function and lipid serum levels, and immunotoxicity (asthma and altered vaccine response).

A particularly significant developmental effect linked to PFAS exposure is alterations to mammary gland development. Prenatal exposure of mice to PFOA results in delays in mammary gland development in offspring of treated females, including reduced ductal elongation and branching, delays in timing and density of terminal end buds (developmental structures important for forming proper mammary gland ductal structure), and decreases in mammary epithelial growth.[63,64,65] These studies found that PFOA-induced effects on mammary tissue occur at extremely low doses - much lower than effects on liver weight. Due to the low-dose sensitivity of mammary glands to PFOA in mice, a no-observable adverse effect level for mammary gland developmental delays could not be determined. In other words, the studies found that all dose levels were associated with effects on mammary gland development. (see Box 6 for a discussion on the biological relevance of altered mammary gland development)

Risk to Immune System Function

Evidence from both animal and human studies suggest that the immune system is also highly sensitive to PFAS exposure. For instance, immunotoxicity is currently the most sensitive health endpoint identified for PFOS exposure and occurs at doses at least an order of magnitude less than other health endpoints. As documented in the ATSDR profile, both animal and epidemiology studies provide strong evidence linking PFAS exposure to immunotoxic effects.[5]

The strongest evidence of the PFAS-associated immunotoxicity in humans comes from epidemiology studies finding associations evaluating the antibody response to vaccines.[5] Associations have been found for PFOA, PFOS, PFHxS, and PFDeA; with limited evidence for PFNA, PFUA, and PFDoA. Increases in asthma diagnosis and effects on autoimmunity, specifically ulcerative colitis, have also been linked to PFAS exposure. Animal studies suggest the immune system is a highly sensitive target of PFAS-induced toxicity; observed effects include impaired responses to T-cell dependent antigens, impaired response to infectious disease, decreases in spleen and thymus weights, and in the number of thymic and splenic lymphocytes.[5,23]

The immunotoxic effects of PFAS could have significant detrimental impacts on public health. For example, PFAS is associated with reduced antibody titer rise in response to vaccines,[5,66] resulting in increased risk of not attaining the antibody level needed to provide long-term protection from serious diseases such as measles, mumps, rubella, tetanus and diphtheria. PFAS can also be transferred to fetuses *in utero*, and to infants via breast milk[67] or PFAS-contaminated infant formula, which presents a particular hazard to the adaptive immune system during this critical window of development. As noted by the Michigan PFAS Science Advisory Panel, "*the developing immune system is especially sensitive to environmental stressors… Disruption 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.*"[26]

Box 1: Immunotoxicity of PFOA, PFOS

In 2016, the National Toxicology Program conducted a systematic review to evaluate immunotoxicity data on PFOA and PFOS. It concluded that both 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 additional evidence linking PFOA exposure to reduced infectious disease resistance, increased hypersensitivity-related outcomes, and increased autoimmune disease incidence (human studies), and PFOS exposure to suppressed disease resistance and lowered immune cell activity (animal studies).[66]

In 2018, the Michigan PFAS Science Advisory Panel recommended adding immunologic effects to ATSDR's list of health conditions of concern, "*particularly those that arise during prenatal exposure and childhood…based on strong toxicologic findings and supporting epidemiologic evidence*."[26]

Short-chain PFAS

Short-chain PFAS (less than six or seven carbons, depending on the PFAS subclass) have been introduced as 'safer' alternatives due to their supposed shorter half-lives in humans, but little research is publicly available on the toxic effects related to exposure, retention, and persistence. The evidence that does exist suggests short-chain PFAS are associated with similar adverse health effects as the long-chain, legacy PFAS that they have replaced.[68,69] Importantly, short-chain PFAS are still highly persistent and are even more mobile in the environment than long-chain PFAS.[70]

Some short-chain PFAS are not detected frequently or detected at low levels in human blood; therefore, some industry groups have claimed that short-chain PFAS are readily eliminated from the body. However, recent research does not support this conclusion. Short-chain PFAS are found to accumulate in

interior organs, some at concentrations that are higher than long-chain PFAS, such as PFOA and PFOS.[77] As Dr. Philippe Grandjean pointed out in his testimony to the Michigan State Legislature, "*Given the inability to assess organ concentrations in clinical studies, our understanding of the health risks associated with the short-chained compounds is extremely limited*." Biomonitoring programs are currently exploring other forms of media, such as urine, as more appropriate measures of short-chain PFAS exposure and retention.

Additionally, developing science on short-chain PFAS metabolism indicates, "*that some fluorinated alternatives have similar or higher toxic potency than their predecessors when correcting for differences in toxicokinetics* [rate a chemical enters the body, is metabolized, and excreted]".[69] The rate a chemical will enter the body and the process of excretion and metabolism in the body may in fact be an inadequate measure of health threats to humans from chemicals with chronic exposure. The widespread use of short-chain PFAS in commerce and their persistence in the environment could lead to chronic exposures in people. Researchers find:

> "*Considering that the exposure to short-chain PFAAs is unlikely to be stopped shortly, there will be increasing continuous and poorly reversible environmental background concentrations of short-chain PFAAs. Consequently, organisms and humans will be permanently exposed to short-chain PFAAs, resulting in continuous and poorly reversible internal concentrations. The poorly reversible internal concentrations in organisms are caused by the persistence of short-chain PFAAs and their continuous presence in the environment. Therefore, the organismal elimination efficiencies are of secondary relevance*."[68]

Finally, it is important to acknowledge that exposure to short-chain and other replacement PFAS, is happening on top of a pre-existing health burden from historically used, long-chain PFAS, as discussed further in the following section.

Box 2: Persistence, Mobility, and Toxicity

The German Environment Agency has shifted the classification of emissions, registered under REACH, to specific intrinsic properties that indicate a hazard to sources of drinking water.[71] These properties include persistence (P) in the environment, mobility (M) in the aquatic environment, and toxicity (T) (PMT). Substances that are considered very persistent in the environment (vP) and very mobile in the aquatic environment (vM), regardless of their toxicity, must also be considered, due to their increased probability of reaching and accumulating in sources of drinking water.[72] Because very short chain PFAS are volatile and can be dispersed far from areas of direct exposure,[73,74] recent efforts have shifted the focus toward mobility as a key chemical parameter of concern, moving from the established criteria persistent (P), bioaccumulative (B), and toxic (T) (PBT) toward PMT.[71,75] This new criteria has prompted the designation of PFAS substances as posing an "equivalent level of concern" under REACH, thereby prompting the need for a new paradigm for chemical assessment and authorization.[76]

Additive and Synergistic Effects of Exposure to Multiple PFAS

Importantly, exposures to PFAS do no occur in isolation. Biomonitoring studies demonstrate that Americans have chronic exposure to multiple PFAS chemicals throughout their lifetimes. CDC's national biomonitoring studies, NHANES, reveal that nearly every American has PFOS, PFOA, PFHxS and PFNA detected in their blood stream, including young children.[6] At least eight other PFAS are detected in blood serum by NHANES studies: MeFOSAA, PFDeA, PFUA, PFHpA, PFBS, FOSA, EtFOSAA, PFDoA, and PFHpA.[6] Most other PFAS chemicals are not routinely included in biomonitoring studies. As mentioned previously, alternative methods in biomonitoring suggest that humans are being exposed to new and unidentified PFAS.[30,31]

Multiple PFAS are found in drinking water, food, dust, personal care products and a variety of different environmental media. In drinking water PFOA, PFOS, PFNA, PFHxS, PFBS, PFHpA (measured in UCMR3), and other PFAS are often found in conjunction.[7] Food contact materials and packaging in the United States has shown detectable levels of PFOA, PFHxS, PFDA, PFHpA, PFDoA, PFHxA, PFBA, PFPeA, PFUA, PFOS and 8:2 FTOH,[78] and likely contain other unknown PFAS. A single consumer product such as carpet, clothing, outdoor gear, or dental floss can contain up to nine different identifiable PFAS compounds[79] along with other undetermined PFAS. Samples of dust collected throughout homes and offices have shown high concentrations of 8:2 FTOH, PFDA, PFHpA, PFNA, 10:2 FTOH, PFDoA and PFTeDA with detection frequencies over 70%.[80]

Figure 2: Possible Sources of PFAS Exposure

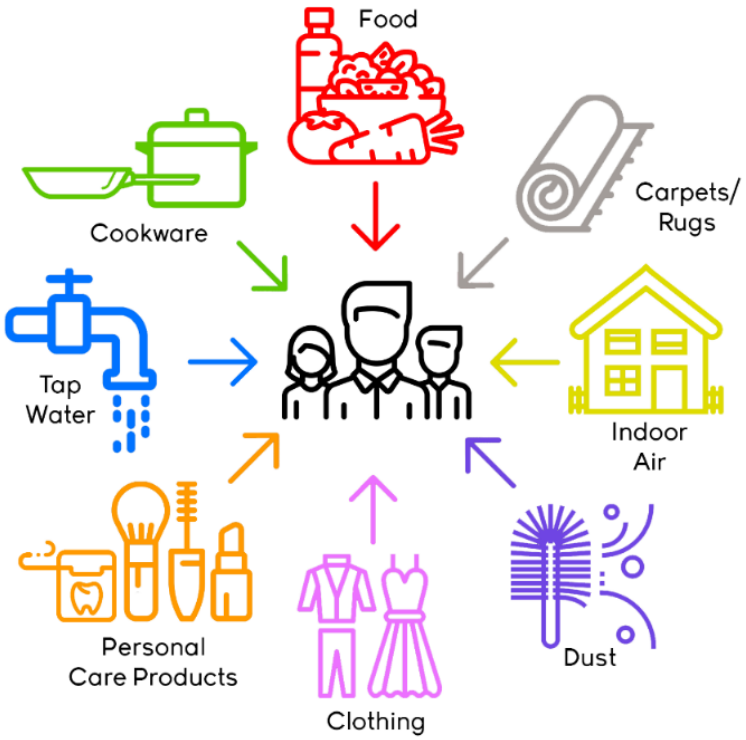

Figure 2 shows the most common pathways of PFAS exposure for humans. PFAS can be found in people's bodies as a result of exposure from multiple environmental sources. [j,k]

Therefore, risk and safety assessments cannot assume that exposures occur in isolation. A person is concurrently exposed to dozens of PFAS chemicals daily, and their exposures extend throughout their lifetimes. Health evaluations should consider the impacts of multiple PFAS chemicals that target the same body systems regardless of detailed knowledge of the underlying mechanism of action. Because PFAS are chemically related, they may have additive or synergistic effects on target systems. An additive effect is when the combined effect of multiple chemicals is the sum of each of the chemicals' effects alone. A synergistic effect is caused when concurrent exposure to multiple chemicals results in effects that are greater than the sum of each of the chemicals' effects alone. For example, many PFAS have been associated with immunological effects. Exposure to a mixture of PFAS could result in adverse effects on the immune system that represents the total dose of all PFAS in the mixture or even greater adverse effects than predicted by summing the dose of all PFAS in the mixture.

PART IV: COMPARISON AND ANALYSIS OF EXISTING HEALTH THRESHOLDS

A number of regulatory and non-regulatory health-based thresholds have been developed for PFAS (mainly PFOA and PFOS) by both federal and state agencies. The data used, and decisions made by these agencies are discussed in this section.

Health advisories issued by the EPA are non-enforceable and non-regulatory. Health advisories provide technical information to state agencies and other public health officials on health effects, analytical methodologies, and treatment technologies associated with drinking water contamination.

Guidance values are state-specific values – used, for example, by the Minnesota Department of Health to evaluate potential human health risks from exposures to chemicals in groundwater – that are non-enforceable goals, benchmarks, or indicators of potential concern. There are three types of guidance values used by Minnesota, health risk limits which are guidance values that have been adopted, and health-based values and risk assessment advice which provide technical guidance but have not yet been formally adopted. In Minnesota, the state develops guidance values by considering health impacts to the most sensitive and most exposed populations across all stages of human development.

Notification levels are state-specific values. California's Division of Drinking Water, for example, has established advisory levels for chemicals in drinking water that lack maximum

[j] ATSDR, 2018. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Perfluoroalkyls. Draft for Public Comment, June 2018.
[k] Guo, Z, et al., 2009. Perfluorocarboxylic acid content in 116 articles of commerce. *Research Triangle Park, NC: US Environmental Protection Agency*

contaminant levels (MCLs, see below). When these chemicals are detected at concentrations greater than their notification levels, state actions include consumer notification and, for larger exceedances, removal of the source water from the drinking water supply.

EPA defines a **Reference dose (RfD)** as "*an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfD is generally expressed in units of milligrams per kilogram of bodyweight per day (mg/kg/day)*."[81]

A **minimal risk level (MRL)** is an estimate made by ATSDR of the daily human total exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified route, including routes other than drinking water exposure, and a specified duration of exposure. MRLs serve as screening tools to help public officials decide where to look more closely and identify contaminants of concern at hazardous waste sites. Like EPA's health advisories, MRLs do not carry regulatory weight by requiring agency-initiated cleanup or setting of action or maximum contaminant levels. MRLs are based on noncancer effects only. These MRLs can be used, similar to reference doses, to generate maximum contaminant level goals for drinking water.

A **maximum contaminant level goal (MCLG)** is the maximum level of a contaminant in drinking water at which no known or anticipated adverse effect on the health of persons would occur, allowing an adequate margin of safety. When determining a MCLG under the federal Safe Drinking Water Act, the EPA considers adverse health risk to sensitive subpopulations, such as infants, children, the elderly, those with compromised immune systems and chronic diseases. MCLGs are non-enforceable health goals and consider only public health and not the limits of detection and treatment technology effectiveness. Therefore, they sometimes are set at levels which water systems cannot meet because of technological limitations.

A **maximum contaminant level (MCL)** is the legal threshold of the amount of a chemical that is allowed in public water systems under the federal Safe Drinking Water Act. A MCL is based on the concentration established by its corresponding MCLG but may be adjusted for feasibility reasons, reflecting difficulties in measuring small quantities of a contaminant, or a lack of available, adequate treatment technologies. The MCL is an enforceable standard and exceedance of the MCL requires water systems to take certain steps, including providing public education, notifying consumers, and adjusting treatment or making structural changes or repairs to come into compliance with the standard for public health protection.

Current or proposed state and federal health thresholds for PFOA and PFOS in drinking water range from 10 ppt to 70 ppt and higher. Although the health thresholds for PFOA and PFOS in drinking water vary, the thresholds cluster at low ppt levels, orders of magnitude lower than thresholds set for many other environmental contaminants. The thresholds are based on adverse health effects, such as developmental effects and cancer risks, and health authorities uniformly acknowledge the serious concerns related to exposure from consuming PFOA and/or PFOS contaminated drinking water. The selection of critical endpoints to use, uncertainty factors to

apply, and estimates of exposure parameters are the major determinants for the variation in the concentrations developed as thresholds. However, none of the federal and state assessments dispute that very serious adverse health effects are associated with exposure to PFOA and PFOS at very low levels of exposure.

The generation of health thresholds by various agencies for PFOA, PFOS, PFNA, PFHxS, and GenX chemicals are **summarized and compared in Tables 4-7** and described in further detail below. Notably, advisories have become more stringent over time as more information becomes available on the exposure to and toxicity of these chemicals.

Table 4: Selected Thresholds for Drinking Water and/or Groundwater- PFOA

Author	Threshold type	Threshold (ppt)	Critical Dose includes UFs (mg/kg/day)	Total UFs	Study Endpoint 2	Drinking water exposure assumptions	Notes
				PFOA			
USEPA	health advisory	70	2×10^{-5}	300	Developmental effects on bone growth and male puberty (Lau, 2006)	0.054 L/kg/day, 90th percentile for lactating women, RSC = 20%	combined with PFOS
Minnesota	guidance value	35	2×10^{-5}	300	Developmental effects on bone growth and male puberty, increased liver weights (Lau, 2006)	modeled for breast- or formula-fed infants, including fetal exposure, RSC = 50%	adopted guidance value - health risk limit - for groundwater
Vermont	health advisory	20	2×10^{-5}	n/a	based on EPA	0.175 L/kg/day for a infants less than 1 year of age, RSC = 20%	combined with PFOS, PFNA, PFHxS, PFHpA (also a ground water enforcement standard); to be adopted as a combined MCL
New Jersey	MCL	14	2×10^{-6}	300	Increased liver weights (Loveless, 2006) + UF for mammary gland effects	0.029 L/kg/day, default adult assumptions, RSC = 20%	proposed; groundwater criteria also proposed at 10 ppt
California	notification level	14	n/a	n/a	Developmental, immunotoxicity, liver toxicity, and cancer	n/a	interim notification levels based on NJ & ATSDR values
ATSDR	environmental media evaluation guide	21	3×10^{-6}	300	Developmental: altered activity, skeletal alterations (Onishchenko, 2011; Koskela, 2016)	0.143 L/kg/day for a infant, RSC = 100%	minimal details provided on calculation of drinking water concentrations from MRL
ATSDR - more protective	estimated MCL	3*	3×10^{-6}	300	Developmental: altered activity, skeletal alterations (Onishchenko, 2011; Koskela, 2016)	**0.175 L/kg/day for a infants less than 1 year of age, RSC = 20%**	*threshold for water based on ATSDR's minimal risk level (for total exposure)
NJ - more protective	estimated MCL	0.1	1×10^{-7}	30	**altered mammary gland development**	**0.175 L/kg/day for a infants less than 1 year of age, RSC = 20%**	using RfD calculated by New Jersey
Protective choices combined	**MCLG (goal)**	0.01	1×10^{-8}	300**	**altered mammary gland development**	**0.175 L/kg/day for a infants less than 1 year of age, RSC = 20%**	****an additional UF of 10, to protect fetuses, infants, children added**

**An additional uncertainty factor of 10 to protect fetuses, infants and children is recommended by the National Academy of Sciences (NAS 1993) for pesticides and as required in the Food Quality Protection Act. 21 U.S.C. §346a(b)(2)(C)(ii)(II).

More protective choices highlighted in bold

Table 5: Selected Thresholds for Drinking Water and/or Groundwater - PFOS

Author	Threshold type	Threshold (ppt)	Critical Dose includes UFs (mg/kg/day)	Total UFs	Study Endpoint 2	Drinking water exposure assumptions	Notes
USEPA	health advisory	70	2×10^{-5}	30	Developmental: decreased pup weight (Leubker, 2005)	0.054 L/kg/day, 90th percentile for lactating women, RSC = 20%	combined with PFOA
Minnesota	guidance value	27	5×10^{-6}	100	Developmental: decreased pup weight (Leubker, 2005)	modeled for breast- or formula-fed infants, including fetal exposure, RSC = 50%	health-based value, provides technical guidance for groundwater
Vermont	health advisory	20	2×10^{-5}	n/a	based on EPA	0.175 L/kg/day for a infants less than 1 year of age, RSC = 20%	combined with PFOS, PFNA, PFHxS, PFHpA (also a ground water enforcement standard); to be adopted as a combined MCL
New Jersey	MCL	13	2×10^{-6}	30	Immunotoxicity: decreased plaque forming response (Dong, 2009)	0.029 L/kg/day, default adult assumptions, RSC = 20%	proposed; groundwater criteria also proposed at 10 ppt
California	notification level	13	n/a	n/a	Developmental, immunotoxicity, liver toxicity, and cancer	n/a	interim notification levels based on NJ & ATSDR values
ATSDR	environmental media evaluation guide	14	2×10^{-6}	300	Developmental: delayed eye opening, decreased pup weight (Leubker, 2005) + UF for immunotoxicity	0.143 L/kg/day for a infant, RSC = 100%	minimal details provided on calculation of drinking water concentrations from MRL
ATSDR - more protective	estimated MCL	2*	2×10^{-6}	30	Developmental: delayed eye opening, decreased pup weight (Leubker, 2005) + UF for immunotoxicity	**0.175 L/kg/day for a infants less than 1 year of age, RSC = 20%**	*threshold for water based on ATSDR's minimal risk level (for total exposure)
NJ - more protective	estimated MCL	2	2×10^{-6}	30	Immunotoxicity (Dong, 2009)	**0.175 L/kg/day for a infants less than 1 year of age, RSC = 20%**	
ATSDR - more protective	estimated MCL	0.02	2×10^{-8}***	30	**Immunotoxicity (Peden-Adams, 2008)**	**0.175 L/kg/day for a infants less than 1 year of age, RSC = 20%**	***critical dose estimated by ATSDR's MRL method
Protective choices combined	**MCLG (goal)**	0.002	2×10^{-9}	300**	**Immunotoxicity**	**0.175 L/kg/day for a infants less than 1 year of age, RSC = 20%**	**an additional UF of 10, to protect fetuses, infants, children added**

**An additional uncertainty factor of 10 to protect fetuses, infants and children is recommended by the National Academy of Sciences (NAS 1993) for pesticides and as required in the Food Quality Protection Act. 21 U.S.C. §346a(b)(2)(C)(ii)(II).

More protective choices highlighted in bold

Table 6: Selected Thresholds for Drinking Water and/or Groundwater - PFNA

Author	Threshold type	Threshold (ppt)	Critical Dose includes UFs (mg/kg/day)	Total UFs	Study Endpoint 2	Drinking water exposure assumptions	Notes
Vermont	health advisory	20	n/a	n/a	based on class similarity to PFOA/PFOS, added to original PFOA/PFOS combined MCL	n/a	combined with PFOS, PFNA, PFHxS, PFHpA (also a ground water enforcement standard); to be adopted as a combined MCL
New Jersey	maximum contaminant level (MCL)	13	5 ng/mL ^	1000	Increased liver weights (Das, 2015)	RSC of 50% for 95th percentile general population	adopted; ^ internal serum level, not external dose
ATSDR	environmental media evaluation guide	21	3×10^{-6}	300	Developmental delays, decreased body weight (Das, 2015)	0.143 L/kg/day for a infant, RSC = 100%	minimal details provided on calculation of drinking water concentrations from MRL
ATSDR - more protective	estimated MCL	3*	3×10^{-6}	300	Developmental delays, decreased body weight (Das, 2015)	**0.175 L/kg/day for a infants less than 1 year of age, RSC = 20%**	*threshold for water based on ATSDR's minimal risk level (for total exposure)
ATSDR - more protective	estimated MCL	2*	2×10^{-6} #	300	Developmental delays, decreased body weight (Das, 2015)	**0.175 L/kg/day for a infants less than 1 year of age, RSC = 20%**	**# Using longer, more representative (men and older women) half-life estimate than ATSDR used (young women)**
Protective choices combined	**MCLG (goal)**	0.2	2×10^{-7}	3000**	Develomental toxicity	**0.175 L/kg/day for a infants less than 1 year of age, RSC = 20%**	****an additional UF of 10, to protect fetuses, infants, children added**

**An additional uncertainty factor of 10 to protect fetuses, infants and children is recommended by the National Academy of Sciences (NAS 1993) for pesticides and as required in the Food Quality Protection Act. 21 U.S.C. §346a(b)(2)(C)(ii)(II).

More protective choices highlighted in bold

Table 7: Selected Thresholds for Drinking Water and/or Groundwater - PFHxS

Author	Threshold type	Threshold (ppt)	Critical Dose includes UFs (mg/kg/day)	Total UFs	Study Endpoint 2	Drinking water exposure assumptions	Notes
ATSDR	environmental media evaluation guide	140	2×10^{-5}	300	Thyroid follicular cell damage (Butenhoff, 2009; Hoberman & York, 2003)	0.143 L/kg/day for a infant, RSC = 100%	minimal details provided on calculation of drinking water concentrations from MRL
Minnesota	guidance value	27	n/a	n/a	based on class similarity to PFOS	n/a	risk assessment advice - for ground water; use PFOS as surrogate for PFHxS until more data is available
ATSDR - more protective	estimated MCL	23*	2×10^{-5}	300	Thyroid follicular cell damage (Butenhoff, 2009; Hoberman & York, 2003)	**0.175 L/kg/day for a infants less than 1 year of age, RSC = 20%**	*threshold for water based on ATSDR's minimal risk level (for total exposure)
Vermont	health advisory	20	n/a	n/a	based on class similarity to PFOA/PFOS, added to original PFOA/PFOS combined MCL	n/a	combined with PFOS, PFNA, PFHxS, PFHpA (also a ground water enforcement standard); to be adopted as a combined MCL
Protective choices combined	**MCLG (goal)**	2	2×10^{-6}	3000**	developmental and thyroid toxicity	**0.175 L/kg/day for a infants less than 1 year of age, RSC = 20%**	****an additional UF of 10, to protect fetuses, infants, children added**
**An additional uncertainty factor of 10 to protect fetuses, infants and children is recommended by the National Academy of Sciences (NAS 1993) for pesticides and as required in the Food Quality Protection Act. 21 U.S.C. §346a(b)(2)(C)(ii)(II).							

More protective choices highlighted in bold

PFOA

Comparison

In May 2016, the EPA issued a drinking water health advisory for PFOA of 70 ppt.[3] In the case of co-occurrence of PFOA and PFOS, the sum of the concentrations is not to exceed 70 ppt. The EPA applied a combined uncertainty factor of 300 (10 for human variability, 3 for animal to human toxicodynamic differences, 10 for use of a lowest-observed-adverse-effect-level (LOAEL) instead of a no-observed-adverse-effect-level (NOAEL)) on a LOAEL for decreased bone development in the fore and hind limbs, in pup mice (both sexes) and accelerated puberty in male mice[85] to generate a reference dose of 2×10^{-5} mg/kg/day.

The EPA used drinking water intake and body weight parameters for lactating women in the calculation of their lifetime health advisory due to the potential increased susceptibility during this time window. EPA assumed a drinking water ingestion rate of 0.054 L/kg-day, which represents the 90th percentile water ingestion estimate for a lactating woman, based on direct and indirect water intake of community water supply consumers.[86] The EPA also concluded that there are significant sources of PFOA and PFOS exposure other than drinking water ingestion. As information is not available to quantitatively characterize exposure from all of these different sources, the EPA used a default relative source contribution (RSC, discussed in Box 3) of 20% of daily exposure coming from drinking water and 80% from other sources.

Box 3: Uncertainty Factors

The use of uncertainty factors (UFs) has a long history in developing regulatory standards and guidance for chemicals. Uncertainty refers to our inability to know all the adverse effects related to a chemical, often due to incomplete data. When assessing the potential for risks to people, toxicology studies often involve exposing test animals (generally rats and mice) which are used as a surrogate for humans.[82] A thorough review of the development and use of science-based uncertainty factors is provided by the EPA and National Academy of Sciences.[82,83,84]

Risk assessment for public health protection must account not only for what is known about a chemical's adverse effects, but also what is not known about differences between toxic effects in animals compared to humans; children compared to adults; differences in absorption, metabolism and excretion; and other unknown factors. The selection of uncertainty factors is designed to account for the incomplete understanding or availability of studies upon which toxicity is appraised.

The EPA typically uses factors of 1, 3 (an approximation of $\sqrt{10}$), or 10, depending on the level of uncertainty for each factor.

In June 2016, Vermont published a health advisory for combined exposure to PFOA and PFOS not to exceed 20 ppt based on EPA's selected developmental effects.[87] It also applied combined uncertainty factors of 300 using EPA's rationale, however generated a lower health advisory due to selection of drinking water exposure parameters for a breastfeeding or formula-fed infant. Breastfeeding and formula-fed infants is a population that drinks the largest volume per body

weight and is the most vulnerable to the toxic effects of exposure to PFAS. The 95th \percentile Body Weight Adjusted Water Intake Rate for the first year of life based on combined direct and indirect water intake from community water supplies for consumers only is 0.175 L/kg-day.[86,89] Vermont also used a relative source contribution from drinking water of 20%.

In August 2018, Minnesota adopted a guidance value (health risk limit) of 35 ppt for PFOA in groundwater based the same critical health effect as the EPA.[90] Minnesota applied a combined uncertainty factor of 300 including: 10 for human variability, 3 for animal to human toxicodynamic differences, 3 for use of a LOAEL instead of a NOAEL, and 3 for database uncertainty. Like Vermont, Minnesota's more protective guidance values are due to the use of drinking water exposure estimates based on infants, but also the accounting of a pre-existing body burden through placental transfer (Minnesota calculated a placental transfer factor of 87% based on average cord to maternal serum concentration ratios). Minnesota estimated breastmilk concentrations by applying a breast milk transfer factor of 5.2%, which is an estimate of the amount of PFOA that is transferred from a mother's serum to her breastmilk. Minnesota published this transgenerational toxicokinetic model for PFOA in January 2019.[91] As serum levels for PFOA are approximately 100 times the concentration in a person's drinking water, a breast milk transfer factor of 5.2% would result in breast milk concentrations approximately 5 times higher than in the drinking water. However, Minnesota also used a less conservative relative source contribution of 50%, resulting in drinking water values approximately half of EPA's.

Box 4: Relative Source Contribution

One important factor that should be considered when generating a health-protective drinking water limit for a contaminant is the percentage of the total allowable dose (RfD or MRL) that comes from water, versus other exposure routes. The portion of a total daily dose that comes from a specific exposure route (such as drinking water) is represented by a relative source contribution (RSC).

EPA suggest RSC's for drinking water range from 0.2 to 0.8 (20% to 80% coming from drinking water). In the absence of complete data, the EPA's default RSC value is 0.2.

- Studies demonstrate that there are many other sources of PFAS exposure, including food and consumer products, though the relative contribution from each source is still poorly understood.
- For children, researchers estimated exposure to PFOA and PFOS from hand-to-mouth transfer from treated carpets to be 40–60% of the total uptake in infants, toddlers, and children.[88]
- Therefore, the RSC from drinking water for this vulnerable population should not exceed 0.4 (40%). Importantly, as we do not understand all the exposure sources for this population, the default value of 0.2 is the most protective and recommended.

In March 2017, New Jersey Drinking Water Quality Institute derived a recommended MCL in water for PFOA of 14 ppt based on increased liver weight in rodent studies.[92] Previously in 2007, New Jersey issued a preliminary drinking water guidance level for PFOA of 40 ppt, which was

revised in 2016 to a more stringent level of 14 ppt based on chronic exposure from drinking water for cancer and non-cancer

endpoints. Non-cancer endpoints were derived based on increased liver weight with applied uncertainty factors of 300 (10 for human variability, 3 for animal to human toxicodynamic differences, and 10 to protect against more sensitive toxicological effects). The more protective health threshold is mainly due to the use of an additional uncertainty factor of 10 to protect against more sensitive toxicological effects (delayed mammary gland development), which is explained by New Jersey in the following excerpt:

> *"Delayed mammary gland development from perinatal exposure is the most sensitive systemic endpoint for PFOA with data appropriate for dose-response modeling. 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.*
>
> *To the knowledge of the Health Effects Subcommittee, an RfD 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. Because the use of this endpoint as the basis for human health criteria is a currently developing topic, the Health Effects Subcommittee decided not to recommend a Health-based MCL with the RfD for delayed mammary gland development as its primary basis. However, the occurrence of this and other effects 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."*[92]

The recommended MCL based on cancer endpoints was derived from testicular tumor data from chronic dietary exposure in rats and also resulted in a MCL of 14 ppt. New Jersey used values for adult drinking water exposure (0.029 L/kg-day) and a relative source contribution of 20%. In January 2019, New Jersey announced a proposed specific ground water quality criteria based on the same reasoning for its proposed MCL, however, since interim ground water criteria are rounded to one significant figure in New Jersey, the proposed criteria for PFOA is 10 ppt (0.01 µg/L).[93] In April 2019, New Jersey announced a rule proposal to adopt the New Jersey Drinking Water Quality Institute's recommended MCL of 14 ppt.[94]

In June 2018, ATSDR generated a MRL for PFOA.[5] A MRL exposure scenario of 3×10^{-6} mg/kg/day was based on a LOAEL of 0.000821 mg/kg/day for neurodevelopmental and skeletal effects in mice[95,96] with an uncertainty factor of 300 (10 for use of a LOAEL instead of a NOAEL, 3 for extrapolation from animals to humans with dosimetry adjustments, and 10 for human variability). A MCLG based on ATSDR's MRL for PFOA would be 11 ppt, using the same assumptions and parameters the EPA used for calculating their health advisory (based on lactating mothers), or 3 ppt, using drinking water exposure assumptions based on breastfeeding and formula-fed infants (see Appendix C for MCLG calculations).

Box 5: ATSDR's Environmental Media Evaluation Guides

In November 2018 ATSDR posted on its website a webpage entitled "ATSDR's Minimal Risk Levels (MRLs) and Environmental Media Evaluation Guides (EMEGs) for PFAS."[97] ATSDR provides the body weights and drinking water intake rates it would use for an average adult or child (under one year) and lists what the corresponding drinking water concentrations would be if converted from ATSDR's proposed MRLs: for an adult 78 ppt for PFOA, 52 ppt for PFOS, 517 ppt for PFHxS, and 78 ppt for PFNA; and for a child, 21 ppt for PFOA, 14 ppt for PFOS, 140 ppt for PFHxS, and 21 ppt for PFNA. ATSDR does not provide any details as to how it derived the values presented on the webpage. However, based on the information ATSDR did provide, drinking water values, body weight and intake rates, we were able to calculate the relative source contribution used by ATSDR. According to our calculations, ATSDR used a relative source contribution of 1, which assumes that 100% of a person's exposure comes from drinking water, not 20% or 50%, as all other agencies have adopted (see Appendix E for calculations).

Studies demonstrate that there are many other sources of PFAS exposure, including food and consumer products. For example, NHANES demonstrates that greater than 95 percent of Americans have detectable PFAS in their bodies, however many of these Americans do not have detectable PFAS in their drinking water. Therefore, the assumption that a person would be only exposed to PFAS from drinking water is not supported by the scientific literature.

In June 2018, at the request of the California State Water Resources Control Board, the California Office of Environmental Health Hazard Assessment (OEHHA) recommended an interim notification level of 14 ppt for PFOA in drinking water.[98] The notification level is based on developmental toxicity, immunotoxicity, liver toxicity, and cancer. OEHHA reviewed currently available health-based advisory levels and standards, including the documents and process used by New Jersey to derive its water advisory levels. OEHHA found New Jersey's process to be both rigorous and sufficient for establishing an interim notification level for PFOA. They note that this level is similar to that derived by ATSDR, whose minimal risk level equates to a drinking water advisory level of 13 ppt for PFOA, as calculated by OEHHA. OEHHA is currently completing its own derivation of a recommended drinking water notification level for PFOA.

In December 2018, the New York Drinking Water Quality Council recommended that the New York Department of Health adopt MCLs of 10 ppt each for PFOA and PFOS.[99] Although no supporting documentation is currently available in relation to this recommendation, the council notes that these levels "take into consideration the national adult population's "body burden," or the fact that all adults already have some level of exposure to these and other related chemicals."

Analysis

Although altered mammary gland development is the most sensitive endpoint for PFOA exposure,[63,64,65] both the EPA and ATSDR did not consider altered mammary gland development as the critical effect in their toxicity assessment of PFOA.

The EPA excluded the results of the mammary gland findings based on the agency's view that the effects were of "unknown biological significance," concern for variability in the sensitivity for these effects amongst mice strains,[65] the fact that the mode of action for these effects are unknown, and that mammary gland effects had not been previously used for risk assessment.[3] Similarly, ATSDR classified altered mammary gland development as not adverse due to uncertainty around the effect's biological significance.

However, experts in the field have concluded that changes in mammary gland growth and differentiation, including changes in developmental timing, are a health concern.[100] Studies have shown a relationship between altered breast development, lactational deficits and breast cancer (discussed further in Box 6). Therefore, unless it can be shown that this relationship does not exist for PFOA, altered mammary gland growth and differentiation should be considered an adverse health effect of PFOA exposure and the critical endpoint for PFOA.

Box 6: "Is altered mammary development an adverse effect?"

Both the EPA and ATSDR did not consider altered mammary gland development as the critical effect in their toxicity assessment of PFOA. However, in a 2009 a workshop of experts in mammary gland biology and risk assessment came to the consensus that changes in mammary gland growth and differentiation, including changes in developmental timing, are a health concern.[100] Altered mammary gland development may lead to difficulty in breastfeeding and/or an increase in susceptibility to breast cancer later in life.[101]

Only one animal study has assessed the effects of PFOA exposure on mammary gland growth and differentiation for multiple generations.[64] The authors saw striking morphological abnormalities in the lactating glands of dams (mothers) chronically exposed to environmentally relevant levels of PFOA; however, no effects on body weight of their pups were seen. It is possible that compensatory behavior, such as increased number of nursing events per day or longer nursing duration per event masked a decreased potential in milk production by the dams, however the authors did not evaluate these endpoints in the study. It is also possible that PFOA exposure could increase time to peak milk output through the reduction in number and density of alveoli available to produce milk.

For human mothers, low-level functional effects on lactation that cause even a short delay in substantial milk output might result in cessation in breastfeeding before the recommended time-frame. This is supported by a cohort study that found an inverse correlation between levels of maternal serum PFOA and duration of breastfeeding.[102]

Early life exposures to factors that disrupt development may influence susceptibility to carcinogens later in life. For example, hormone disruption is an important determinant of breast cancer susceptibility in humans and rodents.[103] Proliferating and undifferentiated

structures, such as terminal end buds, display elevated DNA synthesis compared to other mammary gland structures; which is why terminal end buds are considered the most vulnerable mammary gland target structure of carcinogen exposure.[104] Delays in mammary gland development would result in a prolonged window of increased vulnerability to carcinogens. In humans, perturbations to the timing of menarche is linked to breast cancer.[105] This further raises the concern that changes in patterns of breast development in U.S. girls could be contributing to an increased risk of breast cancer or other adult diseases later in life.[106] However, an increase in susceptibility to breast cancer later in life was not explored in the multigeneration mammary gland development study.[64]

In general, "*developmental delay can reflect an overall detrimental effect of chemical exposure that lead to growth and developmental deficit in the offspring*."[26]

New Jersey did classify delayed mammary gland development as adverse, though, it stopped short of using it to generate their MCL for PFOA. However, New Jersey did calculate a reference dose, 1.1×10^{-7} mg/kg/day, based on delayed mammary gland development. If this more protective reference dose were used, the MCLG for PFOA would be less than 1 ppt, regardless of which population the drinking water parameters are based on (see Appendix D for calculation). The MCLG would be lowered even further below 1 ppt if an additional uncertainty factor of 10 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 (see Box 7).

PFOS

Comparison

In May 2016, the EPA issued a drinking water health advisory for PFOS of 70 ppt,[28] with the sum of PFOA and PFOS concentrations not to exceed 70 ppt. The EPA applied combined uncertainty factors of 30 (10 for human variability, 3 for animal to human toxicodynamic differences) on a NOAEL of decreased pup weight in a two-generation rat study.[107] As with PFOA, the EPA used drinking water intake and body weight parameters for lactating women and a relative source contribution of 20%.

As mentioned above, in June 2016 Vermont published a health advisory for total concentrations of PFOA and PFOS in drinking water at 20 ppt based on EPA's selected developmental effects and drinking water exposure parameters for breastfeeding or formula-fed infants.[87]

In May 2017, Minnesota proposed a groundwater guidance value (health-based value) of 27 ppt for PFOS based the same critical endpoints as the EPA.[108] However, Minnesota applied a larger combined uncertainty factor than the EPA. Minnesota applied a total uncertainty factor of 100

including: 3 for animal to human toxicodynamic differences, 10 for human variability and an additional 3 for database uncertainty (based on the need for additional immunotoxicity data). Minnesota accounted for a pre-existing body burden through a placental transfer factor of 46%, used drinking water exposure estimates based on infants with an estimated breast milk transfer factor of 1.3%, and used a relative source contribution of 50%.

In June 2018, New Jersey derived a recommended MCL in water for PFOS of 13 ppt for chronic exposure from drinking water based on immune suppression in mice,[110] an endpoint that is significantly more sensitive than the endpoint used by EPA.[111] New Jersey applied a combined uncertainty factor of 30 (10 for human variability and 3 for animal to human toxicodynamic differences) to an internal NOAEL of 674 ng/ml of PFOS in animal serum to generate an human serum target level. This target level was then multiplied by a clearance factor to arrive at a reference dose of 1.8×10^{-6} mg/kg/day. New Jersey used values for adult drinking water exposure and a relative source contribution of 20%. Like for PFOA, in January 2019, New Jersey announced a proposed specific ground water quality criteria based on the same reasoning for its proposed MCL, however, since interim ground water criteria are rounded to one significant figure in New Jersey, the proposed criteria for PFOS is 10 ppt (0.01 µg/L).[112] In April 2019, New Jersey announced a rule proposal to adopt the New Jersey Drinking Water Quality Institute's recommended MCL of 13 ppt.[94]

Box 7: Additional Protection for Fetuses, Infants, and Children

The National Academy of Sciences has recommended the use of an additional uncertainty factor of 10 to ensure protection of fetuses, infants and children who often are not sufficiently protected from toxic chemicals such as pesticides by the traditional intraspecies (human variability) uncertainty factor.[109] Congress adopted this requirement in the Food Quality Protection Act for pesticides in foods. 21 U.S.C. 346a(b)(2)(C)(ii)(II)

Considering the many health effects linked to PFAS that affect this vulnerable population and the substantial data gaps on exposure and toxicity of these compounds in complex mixtures, we recommend the use of this uncertainty factor when deriving health-protective thresholds for PFAS.

In June 2018, ATSDR generated a MRL for PFOS based on delayed eye opening and decreased pup weight[107] in rats.[5] A MRL exposure scenario of 2×10^{-6} mg/kg/day was based on a NOAEL of 0.000515 mg/kg/day using an uncertainty factor of 300 (10 for concern that immunotoxicity may be a more sensitive endpoint than developmental toxicity, 3 for extrapolation from animals to humans with dosimetry adjustments, and 10 for human variability). A MCLG based on ATSDR's MRL for PFOS would be 7 ppt, using EPA's drinking water exposure assumptions, or 2 ppt, using drinking water exposure assumptions based on breastfeeding and formula-fed infants (see Appendix C for MCLG calculations).

In June 2018, at the request of the California State Water Resources Control Board, OEHHA recommended an interim notification level of 13 ppt for PFOS in drinking water.[98] The notification level is based on the same analysis performed for PFOA, described above. OEHHA

notes that this level is similar to that derived by ATSDR, whose minimal risk level equates to a drinking water advisory level of 9 ppt for PFOS, as calculated by OEHHA. OEHHA is currently completing its own derivation of recommended drinking water notification levels for PFOS.

As noted above, a MCL of 10 ppt each for PFOA and PFOS were recommended by the New York Drinking Water Quality Council.[99]

Analysis

Immunotoxicity is currently the most sensitive health endpoint known for PFOS exposure. As documented in the ATSDR's profile, both animal and epidemiology studies provide strong evidence linking PFOS exposure to immunotoxic effects (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 reviewed the immunotoxicity data on PFOA and PFOS in 2016 and concluded that both are presumed to constitute immune hazards to humans[66] (discussed further in Box 1).

Again, although immunotoxicity is the most sensitive endpoint for PFOS exposure, the EPA excluded immune system effects based on uncertainties related to mode of action, variation in dose effects between studies, differences in sensitivity between males and females, and lack of a *"demonstrated clinically recognizable increased risk of infectious diseases as a consequence of a diminished vaccine response."*[28]

ATSDR states concern that immunotoxicity is a more sensitive endpoint than developmental toxicity; however, it stops short of deriving a MRL from this endpoint. Instead, ATSDR posits that an additional modifying, or uncertainty factor of 10 is sufficient to address the doses where immunotoxic effects have been observed. However, this value is only consistent with the immunotoxicity study with the highest LOAEL.[113] The other immunotoxicity studies all result in MRLs approximately 2.5-100 times lower than those currently calculated (see Appendix A for MRL derivations). If a MCLG were generated from the most sensitive health endpoint (immunotoxicity) and from the study with the lowest LOAEL, as is normally done by ATSDR, it would be less than 1 ppt (see Appendix C for MCLG calculations). The MCLG would be lowered even further below 1 ppt if an additional uncertainty factor of 10 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. Additionally, a MCLG based on benchmark dose calculations for immunotoxicity in children would also be approximately 1 ppt.[114]

New Jersey did select immunotoxicity as its critical health effect, resulting in the lowest generated reference dose for PFOS. However, the use of adult drinking water assumptions results

in a higher proposed MCL than what we have calculated using estimated MRLs based on immunotoxicity (see Appendix A and C).[l]

PFNA

Comparison

In July 2015, New Jersey proposed a MCL for PFNA of 13 ppt for chronic exposure from drinking water based on increased liver weight in rodents[115] with a total uncertainty factor of 1000 (10 for human variability and 3 for animal to human toxicodynamic differences, 10 for less than chronic exposure duration, and 3 for database uncertainty).[116] Extrapolation from animal to human dose levels were made on the basis of internal serum levels rather than administered dose and were based on an estimated 200:1 ratio between PFNA serum levels and drinking water concentration in humans. A chemical-specific relative source contribution of 50% was developed using the "subtraction" approach. A subtraction approach is used when other sources of exposure (air, food, consumer product, etc.) can be considered background, and can thus be subtracted from the total dose to arrive at the allowable limit or dose from drinking water.[117] New Jersey based their calculations on the 2011-12 NHANES biomonitoring data for the 95th percentile PFNA serum level in the U.S. general population. This MCL was adopted into law in September 2018.[118] As of January 2019, this is the only finalized, enforceable drinking water limit for a PFAS chemical. New Jersey also has a specific ground water quality criteria for PFNA set at 13 ppt, based on its MCL for PFNA.

In July 2018, Vermont updated its drinking water health advisory level to include (based on class similarity) PFOA, PFOS, PFHxS, PFHpA, and PFNA for a combined total not to exceed 20 ppt.[119] Based on its health advisory, Vermont updated its enforceable groundwater standard to include all 5 PFAS at a combined 20 ppt.[120] In January 2019, Vermont announced it will initiate the process of adopting its health advisory for these five PFAS as an enforceable MCL.[121]

For PFNA, ATSDR based its assessment on decreased body weight and developmental delays in mice pups.[5,115] A MRL exposure scenario of 3×10^{-6} mg/kg/day was based on a NOAEL of 0.001 mg/kg/day using an uncertainty factor of 300 (10 for database limitations, 3 for extrapolation from animals to humans with dosimetry adjustments, and 10 for human variability).[5] A MCLG based on ATSDR's MRL for PFNA would be 11 ppt, using EPA's drinking water exposure assumptions for PFOA and PFOS, or 3 ppt, using drinking water exposure assumptions based on breastfeeding and formula-fed infants (see Appendix C for MCLG calculations).

Analysis

[l] Additionally, there are a couple of differences between New Jersey's and ATSDR's approach to generating a RfD/MRL, including the use of slightly different clearance factors and ATSDR's use of the trapezoid rule to estimate a time weighted average serum concentration for the animal point of departure.

Importantly, ATSDR underestimated the half-life of PFNA in humans. In the paper used to estimate the half-life of PFNA,[122] two different half-life values were derived: one of 900 days for young women and one of 1,570 days for everyone else. Younger women of childbearing age have additional excretion pathways for PFAS than other populations, including through breastmilk and menstruation. ATSDR provided no rationale for why the shorter half-life was selected. The longer half-life represents a larger population with minimal excretion pathways for PFNA and would result in a more protective MRL value. Importantly, New Jersey's 200:1 estimated ratio between PFNA serum levels and drinking water concentration in humans is based on the longer, more representative half-life of 1,570 days.[116] When the longer half-life is used, the resulting MRL is 2 x 10^{-6} mg/kg/day (see Appendix B for MRL calculations). A MCLG based on this more protective MRL for PFNA would be 7 ppt, using EPA's drinking water exposure assumptions for PFOA and PFOS, or 2 ppt, using drinking water exposure assumptions based on breastfeeding and formula-fed infants (see Appendix C for MCLG calculations). The MCLG would be below 1 ppt if an additional uncertainty factor of 10 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.

PFHxS

Comparison

As mentioned above, Vermont's drinking water health advisory and its groundwater standard now includes PFOA, PFOS, PFHxS, PFHpA, and PFNA for a combined total not to exceed 20 ppt and Vermont is now in the process of adopting the advisory as a MCL. [119,121]

Minnesota recently recommended using PFOS as surrogate for PFHxS until more data is available, setting a guidance value (risk assessment advice) of 27 ppt for PFHxS.[123]

For PFHxS, ATSDR based its assessment on thyroid follicular cell damage in rats.[124,125] A MRL exposure scenario of 2 x 10^{-5} mg/kg/day was based on a NOAEL of 0.0047 mg/kg/day using an uncertainty factor of 300 (10 for database limitations, 3 for extrapolation from animals to humans with dosimetry adjustments, and 10 for human variability).[5] A MCLG based on ATSDR's MRL for PFHxS would be 74 ppt, using EPA's drinking water exposure assumptions for PFOA and PFOS, or 23 ppt, using drinking water exposure assumptions based on breastfeeding and formula-fed infants (see Appendix C for MCLG calculations). The MCLG would be lowered to 2 ppt if an additional uncertainty factor of 10 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.

GenX

Comparison

In 2017, North Carolina set a non-enforceable health goal for the GenX chemical, HFPO dimer acid, to 140 ppt in drinking water.[126] The health goal was based on a reference dose of 1×10^{-4} mg/kg/day, generated from a NOAEL for liver toxicity in mice (single-cell necrosis in hepatocytes and correlative increases in liver enzymes) with combined uncertainty factor of 1000 (10 for human variability, 10 for animal to human toxicodynamic differences, 10 for extrapolating from subchronic to chronic exposure duration). According to North Carolina Department of Human Health Services, their health goal for GenX is for "the most vulnerable population – i.e. bottle-fed infants, the population that drinks the largest volume of water per body weight."[126] The state used drinking water exposure assumptions based on bottle-fed infants (0.141 L/kg/day) and a relative source contribution of 20%.

In November 2018, the EPA proposed a chronic reference dose of 8×10^{-5} mg/kg/day for two GenX chemicals, HFPO dimer acid and its ammonium salt.[23] The EPA applied a combined uncertainty factor of 300 (10 for human variability, 3 for animal to human toxicodynamic differences, 3 for database limitations, and 3 for extrapolation from subchronic to chronic exposure duration) on a NOAEL for single-cell necrosis in livers of male mice from a DuPont study.[127] The EPA did not provide drinking water values in their toxicity assessment of GenX chemicals, however, using EPA's drinking water exposure assumptions for PFOA and PFOS, a MCLG would be 296 ppt, or 91 ppt using drinking water exposure assumptions based on breastfeeding and formula-fed infants (see Appendix F for calculations).

Analysis

The EPA notes that there are the following database deficiencies for GenX chemicals: no human data from epidemiological studies, limited testing for developmental toxicity and immunological responses, lack of a full two-generational reproductive toxicity study, and lack of a chronic study in mice (which appear to be more sensitive to GenX than rats). Additionally, of the studies considered for the development of the reference dose, only two were published in a peer-reviewed journal. These are significant limitations in the toxicity data available for GenX, and as such, an uncertainty factor of 3 is unlikely to be sufficient. Importantly, North Carolina does not apply an uncertainty factor for database limitations at all. In comparison, ATSDR used an uncertainty factor of 10 for database limitations for PFNA and PFHxS due to a lack of or limited testing of developmental and immunological effects, which ATSDR states are two of the most sensitive PFAS endpoints.[5]

To extrapolate from animal to human dose, the EPA used the Body Weight$^{3/4}$ allometric scaling approach, which is based on body surface area and basal metabolic rate in adults. This approach does not account for differences in toxicokinetics between animals and humans, which for PFAS are often vastly different. The Netherland's National Institute for Public Health and the Environment (RIVM) determined that although the elimination rates for GenX are faster than PFOA in animal models, without data in humans, it is not possible to make assumptions on the toxicokinetics of GenX chemicals in humans.[128] Due to the uncertainty from lack of human toxicokinetic data on GenX chemicals, RIVM calculated and applied an additional uncertainty factor to account for the potential kinetic difference between animals and humans.

This additional toxicokinetic factor used by RIVM is based on the difference in half-lives between cynomolgus monkeys and humans for PFOA. A half-life ratio was calculated using a half-life of 1378 days in humans[129] and of 20.9 days in male cynomolgus monkeys[130] resulting in an additional toxicokinetic factor of 66 (1378 / 20.9). This additional uncertainty factor to account for the potential kinetic difference between animals and humans is an example of an alternative approach to extrapolating animal doses to human doses for PFAS like GenX that do not yet have human toxicokinetic data. Considering the limitations of EPA's scaling approach, an uncertainty factor of 3 to account for interspecies toxicokinetic differences is likely to be insufficient.

Finally, North Carolina used an uncertainty factor of 10 to extrapolate from subchronic to chronic exposure duration, compared to the EPA's use of an uncertainty factor of 3. The EPA states that effects for the subchronic study it selected (performed in mice) are consistent with effects seen for the single chronic study available. However, the chronic study is in rats, a species that the EPA acknowledges is much less sensitive to the effects of GenX than mice. Therefore, this logic is not supported by the EPA's own findings.

If uncertainty factors that properly reflected the deficiencies in toxicity data (database, sub-chronic to chronic, children's vulnerability, human variability, animal to human differences) were used, the combined uncertainty factor could be as high as 100,000, which would result in a MCLG of less than 1 ppt for GenX chemicals (see Appendix F for calculations). This highlights the current considerable level of uncertainty in determining a safe level of exposure for GenX chemicals.

Box 8: Epidemiological Data in Risk Assessment

To generate accurate and relevant health thresholds, all toxicological information available should be evaluated. Epidemiological studies provide direct information on effects of chemical exposures in people. However, epidemiological data from human health studies are not always utilized. Human studies should be used in conjunction with animal studies to best inform risk assessment.

Use of epidemiology data in risk assessment is not a new approach, for example, epidemiological data was used quantitatively in an EPA evaluation of risk for methylmercury, as recommended by the National Academy of Sciences.[131] The EPA based the oral reference dose on lasting neurological effects in children exposed during early life.[132] In 2018, the European Food Safety Authority (EFSA) derived health-based guidance values for PFOA and PFOS based on epidemiological studies.[133] EFSA used benchmark modelling of serum levels to generate daily tolerable intakes (similar to a reference dose, a daily or weekly tolerable intake is an estimate of the amount of a substance in food or drinking water which can be consumed over a lifetime without presenting an appreciable risk to health) of 0.8 ng/kg/bw for PFOA based on increased serum cholesterol in adults and 1.8 ng/kg/bw for PFOS based on increased serum cholesterol in adults and decrease in antibody response at vaccination in

children. These values are approximately 10-20 times stricter than the reference dose generated by the EPA, 20 ng/kg/bw.

Another powerful way of using epidemiological data is demonstrated by the Michigan PFAS Science Advisory Panel's use of epidemiology data to evaluate the EPA's health advisory level of 70 ppt for PFOA and PFOS.[26] The Panel estimated that drinking water with 70 ppt of PFOA over several years would result in serum concentrations around 10,000 ppt in adults and 16,500 ppt among those with higher consumption (such as nursing mother and infants). For adults, the Panel used a model[134] to estimate that 8,000 ppt would result from drinking water that contained 70 ppt PFOA, which is in addition to 2,000 ppt from background exposures (as estimated from NHANES national biomonitoring data).

A PFOA serum concentration of 10,000 ppt would represent the first quartile in the C8 study (contaminated community) and the top bracket in epidemiology studies of the general population. Many health effects have been seen in epidemiology studies at these blood serum concentrations. The Panel concludes, ***"…this evaluation places those with chronic exposure to 70 ppt or higher levels of PFOA in their drinking water well within the range at which credible associations with health effects were found by the C8 Science Panel studies."***[26] In other words, human data shows that the EPA's health advisory for PFOA and PFOS is not health protective.

Conclusions

Differences in the selection of critical endpoints and the application of uncertainty factors have led to the generation of different health thresholds for PFOA, PFOS, PFNA, PFHxS and GenX chemicals. Another source of variation in health thresholds comes from differences in exposure assumptions, such as drinking water intake rate, body weight and relative source contribution from drinking water. For example, the exposure levels of an average male adult versus a lactating mother versus a breastfeeding or formula-fed infant vary greatly. For an in-depth discussion of the main sources of variation in current health thresholds for PFOA and PFOS, including "*managing scientific uncertainty, technical decisions and capacity, and social, political, and economic influences from involved stakeholders*," see recently published article by researchers from Whitman College, Silent Spring Institute, and Northeastern University.[135]

Evidence shows that PFAS exposure poses a high risk to fetuses, infants, children and pregnant women. There is particular risk for sensitive members of the population from chemicals of such persistence and clear adverse effects at very low levels of exposure. Decisions made when developing a health threshold, such as evaluation of data gaps, the selection of uncertainty factors, and the choice of exposure parameters to use, should be made to be protective of the most vulnerable populations, particularly developing fetuses, infants, and children.[136]

Taking into consideration the above information, for risk assessment we recommend: 1) the use of the most sensitive health endpoint, regardless of whether the endpoint has been used in a risk assessment previously; 2) the use of drinking water exposure parameters that protect vulnerable populations, particularly breastfeeding or formula-fed infants; 3) the use of an additional uncertainty factor of 10 to protect fetuses, infants and children as recommended by the National Academy of Sciences[109] and as required in the Food Quality Protection Act (see Box 7); 4) the use of both human and animal data when assessing the toxicity of a chemical, or group of chemicals (see Box 8); and 5) the examination of possible additive or synergistic effects from exposure to mixtures of similar chemicals that target the same biological systems (see Box 9).

Box 9: Real-World Exposures

Fundamentally, exposures to PFAS occur as mixtures. With individual PFAS targeting many of the same biological systems, concurrent exposures to multiple PFAS likely have additive or synergistic effects. Therefore, traditional toxicity assessments that assume exposures to a chemical occur in isolation could be significantly underestimating the real-world effects of PFAS.

PART V: DETECTION/ANALYTICAL METHODS AND TREATMENT TECHNOLOGIES

As discussed in this section, PFOA, PFOS, PFNA, PFHxS, and GenX chemicals can be reliably quantified and treated to low levels, therefore, it is feasible for the state to establish strict MCLs for such PFAS. At present, there is no single methodology for isolating, identifying, and quantifying all PFAS in drinking water. Until total PFAS can be reliably quantified, the state should establish a treatment technique for the class of PFAS chemicals.

Analytical Methods for Detecting and Measuring Concentrations of PFAS

When a laboratory measures an chemical, the laboratory often reports the method detection limit (MDL) and the method reporting limit (also sometimes called the minimum reporting limit or limit of quantification).[137] The MDL is the minimum concentration of a substance that can be measured and reported with 99% confidence that the chemical is present in a concentration greater than zero; any concentration measured below the minimum detection limit is considered non-detect. The method reporting limit is the lowest chemical concentration that meets data quality objectives that are developed based on the intended use of this method; concentrations

above this limit are considered quantified with statistical rigor. A laboratory may also report the single laboratory lowest concentration minimum reporting limit (LCMRL), a value between the method detection and reporting limits, which is the "lowest true concentration for which the future recovery is predicted to fall, with high confidence (99%), between 50 and 150% recovery."[137] Action levels, such as a MCL, should be set at or above the method reporting limit.

Figure 3: Detection, Quantification and Reporting Limits

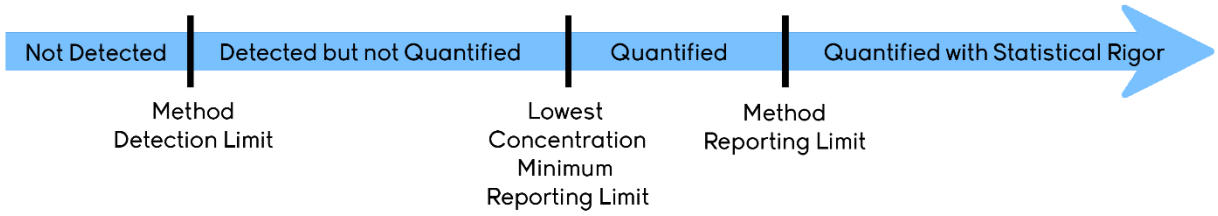

Figure 3 shows the relationship between the types of detection and quantification limits for laboratory testing. The method detection limit (MDL) is the lowest concentration that can be detected. The lowest concentration minimum reporting limit (LCMRL) is the lowest concentration that can be quantified and the method reporting limit, also known as the limit of quantification (LOQ), is the lowest concentration that can be reliably quantified and meets data quality objectives.[m]

The detection sensitivity of PFAS varies depending on the method of analysis used to quantify the results and the laboratory conducting the analysis. Historically, laboratories have used a liquid chromatography-tandem mass spectrometry method such as EPA Method 537, or a modified version,[138] with quantified reporting limits in the low single-digit ppt range. EPA Method 537, updated in November 2018 and referred to as Method 537.1, now includes detection limits ranging from 0.53 to 2.8 ppt for the 18 PFAS compounds included in the updated testing method.[139] In studies where an alternative method is used, researchers were able to achieve reporting limits below 1 ppt for PFOS, PFNA, and PFHxS. In Europe and Australia, reporting limits of less than 1 ppt for PFOA have been achieved.[140] Prominent laboratories that provide analytical detection services for PFAS have already established reporting limits of 2 ppt for at least 17 PFAS compounds including PFOA, PFOS, PFNA, and PFHxS, and a reporting limit of 5 ppt for GenX, using EPA Method 537 or Method 537.1; and one company confirms a 2 ppt reporting limit for the additional PFAS compounds in the updated EPA Method 537.1 will be achievable, except for GenX, which would typically be reported at 5 ppt, but can be lowered to a 2 ppt with an alternative analytical method.[141]

EPA Method 537.1

EPA Method 537.1 is a solid phase extraction (SPE) liquid chromatography/tandem mass spectrometry (LC/MS/MS) method for the determination of selected PFAS in drinking water.[139] This method can be used to quantify 18 PFAS compounds including PFOA, PFOS, PFNA,

[m] Adapted from https://acwi.gov/monitoring/webinars/mpsl_qa_services_intro_rls_012517.pdf

PFHxS, and a GenX chemical, HFPO dimer acid. The EPA states that detection limits range from 0.53 to 1.9 ppt and single laboratory LCMRLs range from 0.53 – 2.7 ppt for PFOA, PFOS, PFNA, PFHxS, and HFPO-DA. We recommend that, at minimum, the state require the use EPA Method 537.1 with method reporting limits of 2 ppt, 5 ppt for GenX, when testing for PFAS in drinking water.

Table 8: Method Reporting Limits from three sources that use EPA Method 537 and/or 537.1

Contaminant	CAS Registry Number	Method Reporting Limits (ppt)			
		EPA 537.1[n]	UCMR3[o]	Eaton Analytics[p]	Vista Analytical[q]
PFOS	1763-23-1	2.7	40	2	2
PFOA	335-67-1	0.82	20	2	2
PFNA	375-95-1	0.83	20	2	2
PFHxS	355-46-4	2.4	30	2	2
HFPO-DA	13252-13-6	4.3	Not available	5	Not available

Table 8 shows the method reporting limits documented for the new EPA Method 537.1, the method reporting limits under the unregulated contaminant monitoring rule 3 (UCMR3) for EPA Method 537, and the method reporting limits reported by two laboratories that conduct testing of PFAS compounds, Eaton Analytical and Vista Analytical.

Alternative Analytical Methods

A Water Research Foundation report published in 2016[142] evaluated the ability of a wide spectrum of full-scale water treatment techniques to remove PFASs from contaminated raw water or potable reuse sources. One of the studies in the report was conducted at Southern Nevada Water Authority's Research and Development laboratory where researchers used a methodology that was able to achieve reporting limits below 1 ppt for several PFAS compounds, including PFOS, PFNA and PFHxS. The method used by researchers in this study is described as "*an analysis…via liquid-chromatography tandem mass-spectrometry (LC-MS/MS) using a previously reported method,[143] adapted and expanded to include all analytes of interest"*. This method achieved minimum reporting limits below 1 ppt for PFOS, PFNA, and PFHxS.

[n] LCMR from https://cfpub.epa.gov/si/si_public_file_download.cfm?p_download_id=537290&Lab=NERL

[o] https://www.epa.gov/dwucmr/third-unregulated-contaminant-monitoring-rule

[p] http://greensciencepolicy.org/wp-content/uploads/2017/12/Andy_Eaton_UCMR3_PFAS_data.pdf

[q] http://www.vista-analytical.com/documents/Vista-PFAS-rev3.pdf

Table 9: Minimum Reporting Levels Using Southern Nevada Water Authority Method

Contaminant	CAS Registry Number	Minimum Reporting Level (ppt)
PFOS	1763-23-1	0.25
PFOA	335-67-1	5
PFNA	375-95-1	0.5
PFHxS	355-46-4	0.25

Table 9 shows the minimum reporting levels achieved by the Southern Nevada Water Authority's analytical method for detecting selected PFAS.[r]

International Analytical Methods

A study conducted in Catalonia, Spain analyzed the concentrations of 13 perfluorinated compounds (PFBS, PFHxS, PFOS, THPFOS, PFHxA, PFHpA, PFOA, PFNA, PFDA, PFUA, PFDoA, PFTeA, and PFOSA) in municipal drinking water samples collected at 40 different locations.[140] Detection limits ranged between 0.02 ppt (PFHxS) and 0.85 ppt (PFOA). Analysis was performed "*using an Acquity UPLC coupled to a Quattro Premier XE tandem mass spectrometer (Waters Corporation, Milford, CT, USA) with an atmospheric electrospray interface operating in the negative ion mode (ES-MS/MS)*". Reporting limits or limits of quantification were not reported for this study.

Another study, conducted in Germany, was aimed at determining concentrations of PFAS in various sources of water intended for human consumption.[144] The study analyzed up to 19 PFAS compounds, including PFOS, PFOA, PFNA, and PFHxS, and the limits of quantification, or reporting limits, for all 19 compounds were 1 ppt. The researchers note that the water samples were measured *"using UPLC-MS/MS (Aquity with a TQ-detector, both from Waters, Eschborn, Germany) on a Kinetex column (2.6 µm, C18, 100A, 100 × 2.1 mm; Phenomenex, Aschaffenburg, Germany).*"

A third study conducted in Australia evaluated the fate of perfluorinated sulfonates (PFSAs) and carboxylic acids (PFCAs) in two water reclamation plants.[145] For this study, instrumental detection limits ranged from 0.2–0.7 ppt and reporting limits were set at double this, ranging from 0.4–1.5 ppt. Authors describe the analysis as "*using a QTRAP 4000 MS/MS (AB/Sciex, Concord, Ontario, Canada) coupled with a Shimadzu prominence HPLC system (Shimadzu, Kyoto Japan) using a gradient flow of mobile phase of methanol/water with 5 mM ammonium acetate. A Gemini C18 column (50 mm _ 2 mm i.d. 3 lm 110 Å) (Phenomenex, Torrance, CA) was used for separation, and an additional column (Altima, C18, 150 mm _ 2 mm i.d. 5 lm, 100 Å)(Grace Davison, Deerfield, IL) was installed between the solvent reservoirs and sample injector to separate peaks consistently present in the system from those in the samples (e.g. small*

[r] Dickenson ERV and Higgins C, 2016. Treatment Mitigation Strategies for Poly- and Perfluoroalkyl Substances. Water Research Foundation, Web Report #4322 http://www.waterrf.org/PublicReportLibrary/4322.pdf

peaks for PFDoDA (C12 PFCA), and for PFOA present in the mobile phase, and/or from fluoropolymer components in the LC system)."

Table 10: Detection and Reporting Limits for PFOA, PFOS, PFNA, PFHxS Internationally

Contaminant	Detection Limit (ppt)[s]	Reporting Limit (ppt)[t]
PFOS	0.12	1
PFOA	0.85	1
PFNA	0.15	1
PFHxS	0.02	1

Table 10 provides examples of detection and reporting limits achieved by two different international studies for PFOA, PFOS, PFNA, and PFHxS.

Comprehensive PFAS Assessment Techniques

At present, there is no single methodology for isolating, identifying, and quantifying all PFAS in drinking water. Current commercial laboratory methodologies are typically able to quantify between 14 and 31 PFAS compounds and only a very small number of PFAA precursors can be quantitatively analyzed by commercial laboratories.[146] For instance, N-ethyl perfluorooctanesulfonamidoacetic acid and N-methyl perfluorooctanesulfonamidoacetic acid are the only two precursors included in EPA Method 537.1. For classes other than PFCAs between 4-14 carbons long and PFSAs that are 4, 6, or 8 carbons long, methodologies are generally not available outside academic settings.[26] The Michigan PFAS Science Advisory Panel summarizes the advantages and disadvantages of some available analytical methodologies to quantify PFAS as a class. These are included in Table 11 below (with additional information as cited).[26]

We recommend states determine an analytical method, or combination of methods, that can be used as a surrogate for total PFAS. In particular, we recommend the evaluation of alternative detection methodologies, particularly TOPA, to measure the concentration of non-discrete and difficult to measure PFAS compounds that are not determined by conventional analytical methods.

[s] Ericson I, et al., 2009. Levels of Perfluorinated Chemicals in Municipal Drinking Water from Catalonia, Spain: Public Health Implications. *Arch Environ Contam Toxicol* 57:631–638

[t] Gellrich V, et al., 2013. Perfluoroalkyl and polyfluoroalkyl substances (PFASs) in mineral water and tap water. *J Environ Sci Health* 48:129–135

Table 11: Comparison of Various Analytical Approaches to Quantifying PFAS

Method	Advantages	Limitations
Method 537 V 1.1 Liquid Chromatography-Tandem Mass Spectrometry LC- MS/MS	• commercially available • QA/QC extensive • UCMR3/Method 537/SW-846 8327&8328/ASTM based on instrument • Differentiates branched/linear • Suited for analysis of ionic compounds[u]	• expensive • approved for a limited number of PFAS (18 in drinking water)[v] • value for forensics depends on number of PFAS evaluated
Total Oxidizable Precursor (TOP) assay	• commercially available • QA/QC improving • some chain length & branched and linear isomer information • reveals presence of significant precursors in AFFF-contaminated water, sediment, soil, and wastewater • data sets obtained by this methodology are comparable between sites and across states	• twice as expensive • no information on individual PFAS • conservative (lower estimate) • limited comparative data at this time • results treated with caution, especially for health and ecological risk assessments[w] • limited value for forensics
Suspect screening (LC-HRMS)	• unlimited number of PFAS • stored data can be searched in future • value as a forensics tool • a reference standard is not needed, the exact mass and isotopic pattern calculated from the molecular formula is used to screen for substances[x]	• instruments available but PFAS analysis by LC-HRMS not commercially available in US (research tool) • expensive • no standards for the other PFAS • data are 'screening' level or semi-quantitative • limited comparable data - data obtained on different instruments, ratioing to various internal standards may not be comparable between sites and across states (generates lab- specific data until standardized)
Particle Induced Gamma Ray Emission (PIGE)	• quantifies fluorine • currently captures anionic PFAS, currently being adapted for cationic/zwitterionic PFAS • less expensive • availability through academic institutions	• only quantifies total fluorine (the atom) • no information on individual PFAS • small database (few comparative data) • cannot analyze different isotopes[y] • limited value for forensics • detection limits are in the µg/L range, regulatory standards are now increasingly at ng/L levels[z]

[u] https://pfas-1.itrcweb.org/wp-content/uploads/2018/03/pfas_fact_sheet_site_characterization_3_15_18.pdf
[v] https://www.epa.gov/water-research/epa-drinking-water-research-methods
[w] https://www.alsglobal.com/-/media/als/resources/services-and-products/environmental/data-sheets-canada/pfas-by-top-assay.pdf
[x] https://link.springer.com/article/10.1007/s00216-018-1028-4
[y] https://www.sciencedirect.com/science/article/pii/0168583X86903812
[z] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5895726/

Total adsorbable organic fluorine (AOF)	• quantifies total fluorine • captures broad spectrum of PFAS • can be compared to individual PFAS analysis to determine presence of other PFAS (e.g., precursors)	• measures total fluorine (the atom) • no information on individual PFAS • not commercially available in US (or elsewhere) • must convert total fluorine in units of molar F to equivalents, assuming a specific PFAS to compare measurements • few comparable data • detection limits are in the µg/L range, regulatory standards are now increasingly at ng/L levels[aa]

Table 11 summarizes advantages and limitations of various analytical approaches to quantifying PFAS.[bb]

Treatment

There are a number of treatment options available to public water systems to address PFAS contamination.

On August 23, 2018, EPA published the results of its efforts to study a variety of technologies used to remove PFAS from drinking water.[147] The EPA's treatability analysis for PFAS compounds demonstrates that current treatment technologies can reduce concentrations of PFOA, PFOS, PFNA, and PFHxS to concentrations below 2 ppt. Full-scale treatment facilities in the U.S., Europe, and Australia have demonstrated effective removal of PFAS compounds through a variety of treatment technologies, most successfully with activated carbon or membrane filtration. The EPA's treatability analysis did not include data on the treatment of GenX, but pilot studies conducted in North Carolina have demonstrated reductions of GenX to below 2 ppt. [148]

Under federal law, standards for synthetic organic contaminants such as PFAS must be "feasible," and that term is defined to be a level that is at least as stringent as the level that can be achieved by Granular Activated Carbon (GAC). Specifically, the Safe Drinking Water Act provides, *"granular activated carbon is feasible for the control of synthetic organic chemicals, and any technology, treatment technique, or other means found to be the best available for the control of synthetic organic chemicals must be at least as effective in controlling synthetic organic chemicals as granular activated carbon."* Safe Drinking Water Act §1412(b)(4)(D). Therefore, states should establish MCLs for PFAS at levels at least as stringent as can be achieved by GAC.

In this report, we recommend MCLs for PFOS, PFOA, PFNA, PFHxS, and GenX that have been demonstrated to be achievable with GAC. However, for total PFAS, greater protections can be

[aa] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5895726/

[bb] Michigan PFAS Science Advisory Panel, 2018. Scientific Evidence and Recommendations for Managing PFAS Contamination in Michigan. December 7, 2018.

achieved with reverse osmosis than GAC (discusses below), therefore we recommend a treatment technique of reverse osmosis, or other treatment method that has been demonstrated to be at least as effective as reverse osmosis for removing all identified PFAS chemicals.

Granular Activated Carbon (GAC) Treatment

According to the EPA, "*Activated carbon treatment is the most studied treatment for PFAS removal. Activated carbon is commonly used to adsorb natural organic compounds, taste and odor compounds, and synthetic organic chemicals in drinking water treatment systems. Adsorption is both the physical and chemical process of accumulating a substance, such as PFAS, at the interface between liquid and solids phases. Activated carbon is an effective adsorbent because it is a highly porous material and provides a large surface area to which contaminants may adsorb*."[147] Activated carbon is made from organic materials with high carbon contents and is often used in granular form called granular activated carbon but can also be used in a powdered form called powdered activated carbon.

Granulated active carbon has been used for more than 15 years to remove PFOA and PFOS from water. The most common carbonaceous materials include raw coal, coconut, and wood. According to the Rapid Scale Small Column Testing Summary Report by Calgon Carbon, *"bench scale studies have shown that reagglomerated bituminous coal-based GAC significantly out performs other GAC materials including direct activated coconut GAC.*"[149]

While the EPA notes that, "*GAC has been shown to effectively remove PFAS from drinking water when it is used in a flow through filter mode after particulates have already been removed*,"[147] it should be noted that GAC has only been demonstrated to be effective for a certain PFAS chemicals. Factors impacting the effectiveness of GAC treatment include:

- the type of carbon used,
- the depth of the bed of carbon,
- flow rate of the water,
- the specific PFAS to be removed,
- temperature, and
- the degree and type of organic matter as well as other contaminants, or constituents, in the water.

A report reviewing the effectiveness of emerging technologies for treatment of PFAS chemicals noted that "*GAC is a widely used water treatment technology for the removal of PFOS and PFOA, and, to a lesser extent, other PFAAs from water…It is an established technology that can be deployed at scales between municipal water treatment and domestic point of entry systems, either as a standalone technology or part of a treatment train*."[150] And while GAC can consistently remove PFOS at parts per billion concentrations with an efficiency of more than 90 percent, it can be inefficient at removing PFOA[151] and becomes progressively less effective for

removing shorter chain PFCAs such as PFHxA, PFPeA, PFBS, and PFBA as the chain length diminishes.[152,153]

There are several examples of full-scale treatment systems using GAC to remove PFAS from drinking water sources. A report prepared for the New Jersey Department of Environmental Protection[154] included several case studies, two of which are included below.

Amsterdam, Netherlands - A study of the removal of a number of PFAS from several steps in the treatment process from raw water to finished water found that longer chain PFAA were readily removed by the GAC treatment step.[155] In this study, a final GAC adsorber was able to reduce both PFOS and PFNA measured in the raw samples at values of 6.7 to 10 ppt and 0.5 to 0.8 ppt, respectively to levels measured below the limits of quantitation (0.23 ppt and 0.24 ppt, respectively). PFOA concentrations in the influent ranged between 3.8 to 5.1 ppt and in the final GAC adsorber ranged between 3.6 to 6.7 ppt. GAC adsorption for this study was done in two stages with adsorbers operated in series, each with a 20-minute empty bed contact time. The GAC in the lag adsorber is placed in the lead position after 15 months of operation and replaced with fresh GAC. The GAC used in this study was Norit ROW 0.8S.

New Jersey American Water, Logan System Birch Creek - Water samples from the Logan System Birch Creek had detectable levels of PFNA (18 – 72 ppt) and of PFOA (33 – 60 ppt), in addition to three other PFAS.[154] GAC treatment removed all detectable PFAS below the reporting level of 5 ppt. GAC adsorbers were operated with an empty-bed contact time of approximately 15 minutes. The GAC used in this study was Calgon F-400.

Additionally, on-going pilot studies being conducted by engineering firm CDM demonstrates effective GAC treatment for GenX and other PFAS with reductions below detection limits of 2 ppt.[148] According to an April 2018 report by CDM for Brunswick County Public Utilities, long-term effective treatment with GAC requires media changeout to avoid breakthrough of compounds and the study indicates approximately 8,000 bed volumes (approximately 4 months at 20-minute contact time) is the appropriate frequency of media changeout for GenX and most PFAS.

GAC treatment can produce contaminated spent carbon or, if regenerated, contaminated air emissions, which require safe disposal. The Michigan PFAS Science Advisory Panel notes that, "*When regenerating PFAS-loaded activated carbon, the off-gases should be treated by high temperature incineration to capture and destroy any PFAS in the stack gases and to prevent the release of PFAS and/or partially oxidized byproducts to the atmosphere."*[26] For example, for complete destruction of PFOS, researchers recommend that incineration be performed at temperatures over 1,000ºC.[156] If an incinerator operates at temperatures below 1,000ºC, it will likely result in incomplete destruction and the formation of byproducts, and therefore require stack treatment to prevent PFAS release.

In sum, use of GAC by multiple water utilities at scale have achieved reductions of greater than 90 percent to below detection limits for certain PFAS chemicals, including PFOS, PFOA, PFNA,

PFHxS, and GenX. GAC has not been demonstrated to be effective for removing other PFAS chemicals, particularly short-chain PFAS.

Ion Exchange (IX) Treatment

Ion exchange resins essentially act as "magnets," attracting the contaminated materials as it passes through the water system.[147] Ion exchange resins can be cationic or anionic; positively charged anion exchange resins (AER) are effective for removing negatively charged contaminants, like PFAS. Ion exchange resins are made up of highly porous, polymeric hydrocarbon materials that are acid, base, and water insoluble.

As summarized by the EPA,

> *"AER has shown to have a high capacity for many PFAS; however, it is typically more expensive than GAC. Of the different types of AER resins, perhaps the most promising is an AER in a single use mode followed by incineration of the resin. One benefit of this treatment technology is that there is no need for resin regeneration so there is no contaminant waste stream to handle, treat, or dispose. Like GAC, AER removes 100 percent of the PFAS for a time that is dictated by the choice of resin, bed depth, flow rate, which PFAS need to be removed, and the degree and type of background organic matter and other contaminants of constituents*."[147]

Reverse Osmosis Treatment

According to the EPA, high-pressure membranes, such as nanofiltration or reverse osmosis (RO), have been effective at removing a broad array of PFAS compounds.[147] High-pressure membranes can be more than 90 percent effective at removing a wide range of PFAS, including shorter chain PFAS.

In a 2011 paper, researchers examined the fate of PFAS in two water reclamation plants in Australia.[145] The authors found that:

> "*Both facilities take treated water directly from wastewater treatment plants (WWTPs) and treat it further to produce high quality recycled water. The first plant utilizes adsorption and filtration methods alongside ozonation, whilst the second uses membrane processes and advanced oxidation to produce purified recycled water. At both facilities perfluorooctane sulfonate (PFOS), perfluorohexane sulfonate (PFHxS), perfluorohexanoic acid (PFHxA) and perfluorooctanoic acid (PFOA) were the most frequently detected PFCs [perfluorinated compounds]. At the second plant, influent concentrations of PFOS and PFOA ranged up to 39 and 29 ppt. All PFCs present were removed from the finished water by reverse osmosis (RO) to concentrations below detection and reporting limits (0.4–1.5 ppt).*"[145]

Preliminary results of an on-going pilot study at Northwest Water Treatment Plant in North Carolina indicate that RO is expected to provide high level of removal (90 percent or greater) for the PFAS compounds, including GenX.[148] The RO membranes being proposed for this project and being tested in the pilot study are standard commercially available brackish water RO membranes rated for 99.3 percent rejection of a standard 2000 mg/L sodium chloride salt solution; this is considered a high rejection, broad spectrum RO membrane. The study also evaluated GAC, IX, and advanced treatment trains and concluded that low-pressure reverse osmosis was the preferred alternative for both removal efficiency and cost-effectiveness. The CDM report states:

> "*RO is recommended over the other options for the following reasons:*
>
> - *RO is the Best Technology for Removal of PFAS. Some PFAS, such as GenX, PFMOAA and PFO2HxA would require very frequent change-out of GAC and IX for removal.*
> - *GAC and IX would likely result in higher finished water concentrations of GenX, PFMOAA, and PFO2HxA than RO (technologies are not equal).*
> - *RO has the lowest net present worth costs for removing 90% or more of the Target Contaminants.*
> - *RO is the most robust technology for protecting against unidentified contaminants.*
> - *RO treated water concentrations will not vary as much with influent concentrations as with GAC and IX. RO treated water quality does not rely on frequent media change-out to protect from the spills and contaminants in the Cape Fear River.*
> - *RO does not release elevated concentrations after bed life is spent as can happen with GAC and IX if feed concentration drops."*[148]

Like GAC, RO treatment technology generates contaminated waste material including liquid concentrate and spent/used membranes. We recommend states evaluate the safest disposal method for contaminated waste, and that disposal require full destruction of PFAS compounds before entering the environment.

Furthermore, the EPA also suggests,

> "*Because reverse osmosis removes contaminants so effectively, it can significantly lower the alkalinity of the product water. This can cause decreased pH and increased corrosivity of the product water. The product water may need to have corrosion inhibitors added or to have the pH and alkalinity adjusted upwards by the addition of alkalinity. These actions may avoid simultaneous compliance issues in the distribution system such as elevated levels of lead and copper*."[157]

Treatment Trains

A treatment train is a sequence of multiple treatment techniques designed to meet specific water quality parameters. According to the Water Research Foundation, when evaluating treatment trains,

> *"Quiñones and Snyder (2009) saw the best removal of PFOA, PFOS, PFNA, and PFHxS using an integrated membrane treatment consisting of microfiltration (MF) and RO and ultraviolet (UV) (medium pressure) followed by SAT [soil aquifer treatment]. This treatment train caused concentrations to drop from the low ng/L [ppt] range to below detection levels. Their success in removing these substances was most likely due to the use of RO. Takagi (2008) looked at the effectiveness of rapid sand filtration followed by GAC and then chlorination on PFOA and PFOS and measured a drop from 92 ng/L to 4.1 ng/L and 4.5 ng/L to <0.1 ng/L, respectively. GAC was most likely responsible for the majority of the removal. Snyder et al. (2014) detected >90% removal of PFOA and >95% removal of PFOS using a treatment train (70 MGD) consisting of MF/RO/UV-advanced oxidation process (AOP)/direct injection (DI). Again, their success was likely due to the RO membrane step using Hydranautics EPSA2 RO dismembranes."*[142]

Although there is still additional research that can be done, removal rates of greater than 90 percent and effluent concentrations of less than 2 ppt for PFOA, PFOS, PFNA, PFHxS, and GenX can be achieved currently with a combination of treatment technologies, along with careful monitoring.

Innovative Technologies

This section describes promising innovative technologies that are designed to treat and/or destroy PFAS chemicals.

- Diamond Technology – According to researchers at Michigan State University-Fraunhofer USA, Inc. Center for Coatings and Diamond Technologies (MSU-Fraunhofer), "*the MSU-Fraunhofer team has a viable solution to treat PFAS-contaminated wastewater that's ready for a pilot-scale investigation. The electrochemical oxidation system uses boron-doped diamond electrodes. The process breaks down the contaminants' formidable molecular bonds, cleaning the water while systematically destroying the hazardous compounds*."[158] While this treatment technology has been developed to treat wastewater, further research may demonstrate effectiveness for removing PFAS from drinking water or waste streams produced by membrane filtration as well.

- AECOM DE-FLUORO Technology – This technology was designed to destroy PFAS compounds concentrated on spent media after treatment.[159] According to AECOM's informational sheet:

> "*Mass transfer technologies (e.g., granular activated carbon, ion exchange resin, reverse osmosis) do not destroy PFAS but concentrate PFAS on the spent media. The spent media may require off-site incineration or regeneration for filtration media reuse that will produce regenerant wastes requiring further management and treatment ... As of today, electrochemical oxidation is one of the most documented PFAS destruction technologies. AECOM has successfully used a proprietary electrode to complete mineralization of C4 ~C8 perfluoroalkyl acids (PFAAs) with evidence of complete defluorination and desulfurization. PFAS are destructed via direct electron transfer on "nonactive" anodes under room temperature and atmospheric pressure with relatively low energy consumption. AECOM has also successfully used this proprietary electrode to treat PFAS in ion-exchange regenerant waste and other PFAS-impacted wastewater*."[159]
>
> In the information sheet, AECOM notes that this technology may also be effective for treating drinking water.

The available research demonstrates that both GAC and IX can be effective treatment techniques for certain PFAS compounds that have been studied, including PFOA, PFOS, PFNA, PFHxS, and GenX, when there is appropriate design, operation, and maintenance. RO has been demonstrated to be an effective treatment technology for removing all PFAS that have been studied and is the most effective treatment technique for effectively removing unknown contaminants. Due to the nature of GAC and IX treatment, water suppliers run the risk of releasing PFAS compounds back into the finished water after GAC bed life is spent or if IX feed concentration drops. Additionally, frequent changeout of GAC or IX to maintain removal efficiency can make the lifecycle costs more expensive than alternatives, such as RO. While GAC, IX, or RO can be effective at removing certain PFAS, RO is advantageous for treating total PFAS because it is the most robust technology for protecting against unidentified contaminants and provides greater protection from future unidentified PFAS. Potential considerations for RO are that it often has a higher capital cost, it can require a 10 to 20 percent higher treatment capacity because it produces a reject stream, and it requires safe disposal of the reject water which will have higher concentrations of contaminants than the source water.

PART VI: CONCLUSIONS AND RECOMMENDATIONS

Taking into consideration the information provided in this report, the following actions are recommended to address PFAS contamination in drinking water:

1. Comprehensive Monitoring of Drinking Water

Understanding the extent of PFAS contamination in drinking water is an important step in protecting people from exposure to these toxic chemicals. Based on national monitoring 4 years ago, there are approximately 16 million people drinking PFAS contaminated water. However, due to limitations in the national survey, including high reporting limits, a focus on large public

water systems, and a limited number of PFAS chemicals tested, the actual numbers are likely much larger, suggesting that there could be significantly more people drinking PFAS contaminated water.

For reference, when expanded testing was carried out by Michigan, the estimates of affected population went from less than 200,000 people to approximately 1.5 million people. The national survey resulted in 3 detections in Michigan. However, once Michigan became aware that they had a PFAS contamination problem, they performed their own site investigations for sites deemed at risk and tested all of their public water systems serving over 25 people. Furthermore, Michigan tested for between 14-24 PFAS at lower health-relevant reporting limits (2 ppt). With this improved testing, they found over 40 contamination sites and over 100 of their public water systems were contaminated with PFAS. Importantly, there are sites of contamination that are not reflected in their public water system survey, and vice versa, public water system contamination not fully predicted through site investigation. The comparison of these two surveys highlights how important comprehensive testing is for understanding the extent of PFAS contamination of drinking water.

Therefore, states should perform both site investigations for at risk sites and a comprehensive statewide survey of public water systems. States should also offer testing of private water systems and private wells serving residences that are near known or suspected PFAS contamination sites, or as requested by a private well user. Priority for testing and monitoring should be sites near former PFAS manufacturing or processing facilities; near fire-fighting stations where PFAS was or continues to be used for training; near military bases and airports which may still use PFAS; and near landfills.

Periodic rounds of PFAS testing should be performed to account for testing variability, to ensure no additional discharges of PFAS are occurring, and to evaluate treatment effectiveness. The analyses should be conducted using the most sensitive detection methods for a comprehensive assessment, which at minimum should now include the expanded EPA 537.1 list at reporting limits of 2 ppt for all PFAS covered by the method, except for GenX, whose reporting limit should be no greater than 5 ppt. We also recommend that states evaluate newer methodologies, particularly the total oxidizable precursor assay, as an analytical technique to help measure the concentration of non-discrete and difficult to measure PFAS compounds that are not determinable by conventional analytical methods.

Data on PFAS in drinking water supplies should be provided to residents served by the tested water supplies, researchers, and the public. Where both biomonitoring data and water testing data are available, that information should be provided to individuals participating in the biomonitoring program so that participants are informed of their own body burden and drinking water exposures. Biomonitoring data and water testing data should also be provided to researchers (in matched pairs, if possible, and with identifying information removed to protect the confidentiality of participants) so that the contribution of PFAS-contaminated drinking water to total PFAS exposure can be studied further. Additionally, unique values for all detected levels of individual PFAS compounds should be publicly reported. All data should be provided in a timely manner and in a common format on a publicly-available database.

2. Set a MCLG of Zero for Total PFAS.

PFAS share similar structure and properties, including extreme persistence and high mobility in the environment. Many PFAS are also associated with similar health endpoints, some at extremely low levels of exposure. There is additionally potential for additive or synergistic toxicity among PFAS. Given the similarity among chemicals of the PFAS class and the known risk of the well-studied PFAS, there is reason to believe that other members of the PFAS class pose similar risk. Therefore, health-protective standards for PFAS should be based on the known adverse effects of the well-studied members of the PFAS class.

First, there is sufficient evidence to classify PFOA as a known or probable carcinogen. Therefore, a MCLG of zero should be promulgated for PFOA, consistent with EPA's approach to regulating known or probable carcinogens (see Box 10). Both IARC's and EPA's findings on PFOA's carcinogenic potential are based heavily on the C8 study, whose Science Panel determined that PFOA is a probable carcinogen. There is also significant additional animal and human evidence for an association between PFOA exposure and cancer, particularly kidney and testicular cancer.

Box 10: Maximum Contaminant Level Goals for Carcinogens

The EPA derives a MCLG under the Federal Safe Drinking Water Act by first considering the carcinogenic potential of the contaminant, or suite of contaminants. For known or probable carcinogens, EPA sets a MCLG of zero for the contaminant, or for the contaminant class, under the federal framework. This is because EPA assumes that, in the absence of other data, there is no known threshold at which no adverse health effects would occur. For chemicals suspected as carcinogens, the agency considers the weight of evidence, including animal bioassays and epidemiological studies. Information that provides indirect evidence, such as mutagenicity and other short-term test results, is also considered by the agency. Known human carcinogens, under EPA's classification scheme, are chemicals for which there exists sufficient evidence of carcinogenicity from epidemiological studies. Probable human carcinogens demonstrate either limited evidence of carcinogenicity in humans or sufficient evidence in animals without corresponding human data, under this classification scheme. See *56 Fed. Reg. 20, 3532* (Jan. 30, 1991).

In addition to being a carcinogen, PFOA causes adverse non-cancer health effects at exceedingly low doses. A MCLG based on altered mammary gland development would be well below 1 ppt for PFOA, further supporting our recommendation of zero for a MCLG (see Table 12 below).

Although the evidence of carcinogenic potential for PFOS is not as well established as PFOA, given the similarities in structure and toxicity of PFOS to PFOA, we recommend a MCLG of zero for PFOS as well. The weight of evidence indicates that PFOS also causes adverse non-cancer health effects at exceedingly low doses. A MCLG based on immunotoxicity would be

well below 1 ppt for PFOS, further supporting our recommendation of zero for a MCLG (see Table 12 below).

There is less information on the carcinogenic potential of PFNA, PFHxS, and GenX, however, given the similarities in structure and toxicity of these PFAS to PFOA and PFOS, their potential for the carcinogenicity cannot be ruled out. Other shared health effects that occur at extremely low levels, such as immunotoxicity, developmental harm, and liver damage, along with their co-occurrence in our environment, must also be considered in setting a health protective MCLG for PFNA, PFHxS, and GenX.

A MCLG for PFNA based on developmental toxicity is below 1 ppt, approximately 2 ppt for PFHxS based on thyroid toxicity, and below 1 ppt for GenX based on liver toxicity (see Table 12 below).

Please see Appendices A, B, C, D and F for more detailed calculations.

Table 12: NRDC Recommended MCLGs for PFOA, PFOS, PFNA, PFHxS, and GenX

Threshold (ppt)	Threshold type	Study Endpoint	Total UFs	Critical Dose includes UFs (mg/kg/day)	Drinking water exposure assumptions	Notes
PFOA						
0	proposed MCLG (goal)	cancer and altered mammary gland development				
0.01		altered mammary gland development	300**	1×10^{-8}	0.175 L/kg/day for a infants, RSC = 20%	**additional UF of 10, to protect fetuses, infants, children
PFOS						
0	proposed MCLG (goal)	class similarity to PFOA (supported by immunotoxicity)				
0.002		Immunotoxicity	300**	2×10^{-9}	0.175 L/kg/day for a infants, RSC = 20%	**additional UF of 10, to protect fetuses, infants, children
PFNA						
0	proposed MCLG (goal)	class similarity to PFOA (supported by develomental toxicity)				
0.3		Develomental toxicity	3000**	3×10^{-7}	0.175 L/kg/day for a infants, RSC = 20%	**additional UF of 10, to protect fetuses, infants, children
PFHxS						
0	proposed MCLG (goal)	class similarity to PFOA (supported by developmental and thyroid toxicity)				
2		developmental and thyroid toxicity	3000**	2×10^{-6}	0.175 L/kg/day for a infants, RSC = 20%	**additional UF of 10, to protect fetuses, infants, children
GenX						
0	proposed MCLG (goal)	class similarity to PFOA (supported by liver toxicity)				
0.2		liver toxicity	100000#	2×10^{-6}	0.175 L/kg/day for a infants, RSC = 20%	# due to data limitations, uncertainty could be up to 100,000

**An additional uncertainty factor of 10 to protect fetuses, infants and children is recommended by the National Academy of Sciences (NAS 1993) for pesticides and as required in the Food Quality Protection Act. 21 U.S.C. §346a(b)(2)(C)(ii)(II).

PFOA, PFOS, PFNA, PFHxS, and GenX share similar structure and properties and are associated with similar health endpoints, many at extremely low levels of exposure, across animal and epidemiological studies. Thus, because they often co-occur in our environment, there is potential for additive toxicity among these PFAS. New Jersey noted that the modes of action and health effects are generally similar for PFAS and acknowledged the possibility that the effects may be additive.[92] Given the above information we recommend a combined MCLG of zero for PFOA, PFOS, PFNA, PFHxS, and GenX.

However, this reasoning should be applied to the PFAS class as a well. Information on and lessons learned from these more extensively studied PFAS need to be used to guide regulations and ensure actions taken are adequately protective of human health in the long term. While there is limited toxicity data on many of the newer short-chain or other alternative PFAS replacing long-chain PFAS in various applications, evidence suggests that they collectively pose similar threats to human health and the environment. The rise in use of alternative PFAS and concerns with the environmental fate and persistence of these alternative PFAS have led to a call from independent scientists from around the globe to address PFAS as a class both in terms of their impacts and in limiting their uses.[12]

The structure of the fluorine-carbon bond and the impacts documented on the studied PFAS already available support concern over the health impacts of the entire class. This is supported by the constant exposure to short-chain chemicals, even if they have a relatively short presence in the body, as well as the fact that in many cases the use of these chemicals may be much higher than their long-chain cousins. Furthermore, many PFAS can convert into PFAAs (a PFAS subgroup, which includes PFOA and PFOS, that is linked to many adverse health effects) or PFAAs are used in their manufacture and can be contaminants in their final product.

Box 11: Regulating Classes in Tap Water - The PCB Precedent

There is precedent for regulating a group of chemicals as a class. For example, polychlorinated biphenyls (PCBs) are a class hundreds of man-made chlorinated hydrocarbons that are persistent in the environment, can bioaccumulate, and have a range of toxicity, including cancer and disruption of the immune, reproductive, endocrine, and nervous systems.[160] Drinking water standards and regulations regarding their clean up, disposal and storage apply to the class and are not set separately for each PCB in use.

In promulgating drinking water regulations for the large class of PCBs, EPA found that although statistically significant evidence of carcinogenicity had been demonstrated only in PCBs that were 60 percent chlorinated, the evidence justified regulation of the whole class of PCB compounds, given the structural complexity of the compounds, and the incomplete data regarding toxicity of the isomers in PCB compounds. EPA, 56 Fed. Reg. 3526, at 3546 (January 30, 1991)[161]

Setting a MCLG of zero for the class is needed to provide an adequate margin of safety to protect public health from a class of chemicals that is characterized by extreme persistence, high mobility, and is associated with a multitude of different types of toxicity at very low levels of exposure. If we regulate only a handful of PFAS, there will be swift regrettable substitution with other, similarly toxic PFAS - creating an ongoing problem where addressing one chemical at a time incentivizes the use of other toxic chemicals and we fail to ever establish effective safeguards to limit this growing class of dangerous chemicals.

3. Immediately Set a Combined MCL of 2 ppt for PFOA, PFOS, PFNA, and PFHxS, and a MCL of 5 ppt for GenX

As discussed in our second recommendation, NRDC's review of the toxicity studies for five PFAS compounds finds evidence that they are linked to cancer and other serious adverse health effects. Following conventional risk assessment protocols, we determine that the goal for PFOA, PFOS, PFNA, PFHxS and GenX should be zero exposure to these chemicals in drinking water.

As technologies for detection and water treatment do not currently allow for the complete removal of PFAS from drinking water, a MCL for PFOA, PFOS, PFNA, PFHxS, and GenX should be based on the best detection and treatment technologies available. Our review suggests a combined MCL of 2 ppt is feasible for PFOA, PFOS, PFNA, and PFHxS, with a separate MCL of 5 ppt for GenX.

Laboratory methods support a reporting limit of 2 ppt with EPA Method 537.1 (5 ppt for GenX), and therefore all water testing should be required to achieve this limit for the PFAS chemicals detectable with this method. Further, the removal of PFOA, PFOS, PFNA, PFHxS, and GenX has been demonstrated to be effective with technologies such as GAC and RO to below detection levels, supporting our determination that the MCL meets technological feasibility.

Residents who rely on private wells for drinking water depend on the safety of their state's groundwater, therefore a groundwater cleanup standard should also be set to 2 ppt for PFOA, PFOS, PFNA and PFHxS and to 5 ppt for GenX, consistent with the recommended MCL for public water systems.

4. Develop a Treatment Technique Requirement for the PFAS Class Within Two Years

As discussed in our second recommendation, setting a MCLG of zero for the class is needed to protect public health and the environment from all types of PFAS that share common negative qualities including extreme persistence, high mobility, and the association with a multitude of different types of toxicity at very low levels of exposure. The replacement of PFOA with GenX is a perfect example of regrettable substitution where a well-studied, toxic PFAS was replaced by a poorly-studied but structurally similar PFAS.

Technology for detection and treatment cannot achieve a MCLG of zero for total PFAS. In the absence of a reliable method that is economically and technically feasible to measure a contaminant at concentrations to indicate there is not a public health concern, the state should establish a treatment technique. A treatment technique is a minimum treatment requirement or a necessary methodology or technology that a public water supply must follow to ensure control of a contaminant.

At present, there is no single methodology for isolating, identifying, and quantifying all PFAS in drinking water. We recommend that states explore an analytical method, or combination of methods, that can be used as a surrogate for total PFAS. In particular, we recommend that states evaluate alternative detection methodologies, such as the total oxidizable precursor assay, to measure the concentration of non-discrete and difficult to measure PFAS compounds that are not determined by conventional analytical methods.

Furthermore, we recommend reverse osmosis, or other treatment method that has been demonstrated to be at least as effective as reverse osmosis for removing all identified PFAS chemicals, as the treatment technique for public water supplies. Reverse osmosis is currently the preferred treatment technology for the following reasons:

- Reverse osmosis has been demonstrated to effectively remove a broad range of PFAS compounds.[148]
- Reverse osmosis is the most robust technology for protecting against unidentified contaminants.[148]
- Reverse osmosis would likely result in lower finished water concentrations of GenX and other PFAS compounds such as PFMOAA and PFO2HxA.[148]
- Reverse osmosis does not require frequent change out of treatment media and does not release elevated concentrations after granular activated carbon bed life is spent or ion exchange feed concentration drops.[148]

Reverse osmosis requires considerations for the safe disposal of high-strength waste streams and spent/used membranes. We recommend states evaluate the safest disposal method for contaminated waste, and that disposal require full destruction of PFAS compounds before entering the environment.

UNITS AND DEFINITIONS

AER - anion exchange resins

ATSDR – Agency for Toxic Substances and Disease Registry

C8 - PFOA

CDC - Centers for Disease Control and Prevention

EPA – U.S. Environmental Protection Agency

EtFOSAA - 2-N-Ethyl-perfluorooctane sulfonamide

FOSE – perfluorooctane sulfonamide ethanol

FTOH - fluorotelomer alcohol

GAC – granular activated carbon

GenX – HFPO dimer acid and its ammonium salt

HFPO - hexafluoropropylene oxide

IARC – International Agency for Research on Cancer

IX - strong base anion exchange resin

LCMRL - lowest concentration minimum reporting limit

LC/MS/MS - liquid chromatography/tandem mass spectrometry

LOAEL – lowest-observable-adverse-effect-level

LOQ – limit of quantitation

MCL - maximum contaminant level

MCLG – maximum contaminant level goal

MDL – minimum detection level

MeFOSAA - 2-N-Methyl-perfluorooctane sulfonamide

MRL - minimal risk level

NAS – National Academy of Sciences

NHANES – National Health and Nutrition Examination Survey

NOAEL – no-observable-adverse-effect-level

OEHHA – California Office of Environmental Health Hazard Assessment

PBT – persistent bioaccumulative toxic

PFAA – perfluoroalkyl acid

PFAS – per- and polyfluoroalkyl substances

PFBS - perfluorobutane sulfonic acid, also known as PFBuS

PFCA – perfluorocarboxylic acid

PFDeA - perfluorodecanoic acid, also known as PFDeDA

PFDoA - perfluorododecanoic acid, also known as PFDoDA

PFHpA - perfluoroheptanoic acid

PFHxS - perfluorohexane sulfonic acid

PFNA - perfluorononanoic acid

PFOA - perfluorooctanoic acid

PFOS - perfluorooctane sulfonic acid

PFOSA - perfluorooctane sulfonamide

PFSA – perfluorosulfonic acid

PFTeA – perfluorotetradecanoic acid, also known as PFTDA

PFUA - perfluoroundecanoic acid, also known as PFUnDA or PFUnA

PMT – persistent mobile toxic

ppt - parts per trillion = nanograms per liter (ng/L) (usually used to express water concentration)

ppb - parts per billion = micrograms per liter (ug/L) (usually used to express blood serum concentration)

PWS – public water system

RfD - reference dose

RO – reverse osmosis

RSC – relative source contribution

THPFOS - 1H,1H,2H,2H-perfluorooctanesulfonic acid

TOP or TOPA – total oxidizable precursor assay

UCMR3 – EPA's Unregulated Contaminant Monitoring Rule 3

UF - uncertainty factor

APPENDIX A - MRL CALCULATIONS FOR PFOS USING IMMUNOTOXICITY ENDPOINT

Based on information from: https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf

Immunotoxicity is currently the most sensitive health endpoint for PFOS exposure. Although ATSDR states concern that immunotoxicity is a more sensitive endpoint than developmental toxicity, it stops short of deriving a MRL from this endpoint. Instead, ATSDR claims that a modifying factor of 10 is sufficient to address the doses where immunotoxic effects have been observed. This statement is based on ATSDR calculating a candidate MRL for one of the four immunotoxicity studies in rodents identified by ATSDR, Dong et al., 2011, but not the other studies (ATSDR, 2018, see page A-43 of Appendix A).

However, Dong et al. 2011 is the immunotoxicity study with the highest LOAEL, which is not consistent with ATSDR's practice of choosing the study with the lowest LOAEL when selecting the principle study for MRL derivation. The other immunotoxicity studies all result in MRLs approximately 2.5-100 times lower than the MRL proposed by ATSDR (Table 1, calculations to follow, performed as described in ATSDR, 2018, Appendix A).

Table 13: Comparison of candidate MRLs for PFOS

Source	Year	Critical Endpoint	Minimal Risk Level (mg/kg/day)
ASTDR	2018	Developmental toxicity (delayed eye opening, decreased pup weight) + Modifying Factor	2 x 10-6 MRL
Dong et al.	2011	Immunotoxicity (impaired response to sRBC)	2.7 x 10-6 Estimated MRL[a]
Dong et al.	2009	Immunotoxicity (impaired response to sRBC)	7.8 x 10-7 Estimated MRL[a]
Guruge et al.	2009	Immunotoxicity (decreased resistance to influenza virus)	2.2 x 10-7 Estimated MRL[a]
Peden-Adams et al.	2008	Immunotoxicity (impaired response to sRBC)	2.1 x 10-8 Estimated MRL[a]

a – Calculated using the derivation method described on pg. A43 of the ATSDR profile

In equation A-6 from Appendix A, ATDSR defines an expression relating the external steady-state dosage and steady-state serum concentration:

$$D_{SS} = (C_{SS} \times k_e \times V_d) / AF$$

Where:

D_{SS} = steady-state absorbed dosage (mg/kg/day)

C_{SS} = steady-state serum concentration in humans (mg/L)

k_e = elimination rate constant (day-1)

V_d = assumed apparent volume of distribution (L/kg)

AF = gastrointestinal absorption fraction

ATSDR provided the following First Order One-Compartment Model Parameters for PFOS in Table A-4:

$K_e = 3.47 \times 10^{-4}$

$V_d = 0.2$

AF=1

ATSDR made the assumption that "humans would have similar effects as the laboratory animal at a given serum concentration." Therefore, the time weighted average serum levels from animal studies (C_{TWA}) are used to back-calculate D_{SS} by imputing C_{TWA} as C_{SS} in equation A-6.

The immunotoxicity studies, are the most sensitive endpoints, having NOAELs 6-625 times lower than the NOAEL for the developmental endpoint chosen for deriving the MRL. Though they did report serum levels, the immunotoxicity studies were performed in different strains/species of animals than those used for the pharmacokinetic modeling completed by Wambaugh et al. As such, they were not chosen for calculation of an MRL, though the ATSDR used other methods to calculate TWA concentrations for PFHxS and PFNA (the trapezoid rule) which were also lacking pharmacokinetic modeling.

From ATSDR (Appendix A, pg. A-43):

"A candidate MRL was calculated using the NOAEL of 0.0167 mg/kg/day identified in the Dong et al. (2011)...A TWA concentration was estimated using a similar approach described for

PFHxS and PFNA in the MRL approach section. The estimated TWA concentration was 1.2 µg/mL for the 0.0167 mg/kg/day; this estimated TWA concentration was used to calculate a human equivalent dose (HED) of 0.000083 mg/kg/day. A candidate MRL of 3x10-6 was calculated using an uncertainty factor of 30 (3 for extrapolation from animals to humans using dosimetric adjustments and 10 for human variability)."

Following this logic:

The time weighted average (TWA) serum levels for the other immunotoxicity studies can be predicted by using the trapezoid rule, as was done for PFNA, PFHxS, and the candidate PFOS MRL based on Dong et al., 2011.

Dong et al. 2009:

Measured serum level at NOAEL dose of 0.0083 mg/kg/day: 0.674 ug/mL

Estimated TWA = (0.674 ug/mL - 0 ug/mL) / 2 = 0.337 ug/mL = 0.337 mg/L

Guruge et al. 2009:

Measured serum level at NOAEL dose of 0.005 mg/kg/day: 0.189 ug/mL

Estimated TWA = (0.189 ug/mL - 0 ug/mL) / 2 = 0.0945 ug/mL = 0.0945 mg/L

Peden-Adams et al. 2008:

Measured serum level at NOAEL dose of 0.00016 mg/kg/day: 0.0178 ug/mL

Estimated TWA = (0.0178 ug/mL - 0 ug/mL) / 2 = 0.0089 ug/mL = 0.0089 mg/L

These estimated TWA serum levels can then be inputted into equation A6 as the steady state serum concentration, C_{SS}, using the same values used by ATSDR for the other parameters to generate candidate MRLs for these immunotoxicity studies.

$$D_{SS} = (C_{SS} \times 0.000347\ day^{-1} \times 0.2\ L/kg) / 1$$

Dong et al. 2009:

D_{SS} = (0.337 mg/L x 0.000347 day-1 x 0.2 L/kg) / 1 = 2.34 x 10^{-5} mg/kg/day

Then, divide by UF of 30

MRL = 7.8 x 10^{-7} mg/kg/day

Guruge et al. 2009:

D_{SS} = (0.0945 mg/L x 0.000347 day-1 x 0.2 L/kg) / 1 = 6.56 x 10^{-6} mg/kg/day

Then, divide by UF of 30

MRL = 2.2 x 10^{-7} mg/kg/day

Peden-Adams et al. 2008:

D_{SS} = (0.0089 ug/mL x 0.000347 day-1 x 0.2 L/kg) / 1 = 6.2 x 10^{-7} mg/kg/day

Then, divide by UF of 30

MRL = 2.1 x 10^{-8} mg/kg/day

APPENDIX B - MRL CALCULATIONS FOR PFNA USING LONGER HALF-LIFE

Based on information from: https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf

In equation A-6 from Appendix A, ATDSR defines an expression relating the external steady-state dosage and steady-state serum concentration:

$$D_{SS} = (C_{SS} \times k_e \times V_d) / AF$$

Where:

D_{SS} = steady-state absorbed dosage (mg/kg/day)

C_{SS} = steady-state serum concentration in humans (mg/L)

k_e = elimination rate constant (day-1)

V_d = assumed apparent volume of distribution (L/kg)

AF = gastrointestinal absorption fraction

ATSDR provided the following First Order One-Compartment Model Parameters for PFNA in Table A-4:

$k_e = 7.59 \times 10^{-4}$

V_d=0.2

AF=1

The $k_e = 7.59 \times 10^{-4}$ is based on a half-life estimate of 900 days for young women. Based on Eq. A-5, a half-life of 1570 days for all other adults would result in a k_e of 4.4×10^{-4} (k_e = ln(2) / half-life).

Thus, if the k_e representing the longer, more representative half-life for PFNA was used, along with ATSDR's estimated C_{SS} of 6.8 mg/L:

$D_{SS} = (6.8 \text{ mg/L} \times 0.000441 \text{ day-1} \times 0.2 \text{ L/kg}) / 1 = 6 \times 10^{-4}$ mg/kg/day

Then, divide by UF of 300

MRL = 2 x 10^{-6} mg/kg/day

APPENDIX C - MCLG CALCULATIONS

From EPA's Drinking Water Health Advisory for PFOA and PFOS (EPA, 2016 a and b)

The EPA used drinking water intake and body weight parameters for lactating women in the calculation of a lifetime health advisory for PFOA and PFOS. EPA used the rate of 54 mL/kg-day representing the consumers only estimate of combined direct and indirect community water ingestion at the 90th percentile for lactating women (see Table 3-81 in EPA 2011).

First, a Drinking Water Equivalent Level (DWEL) is derived from the reference dose (RfD) and assumes that 100% of the exposure comes from drinking water. The RfD is multiplied by body weight and divided by daily water consumption to provide a DWEL.

$$DWEL = (RfD \times bw) / DWI = RfD / (DWI/bw)$$

Where:

RfD = critical dose (mg/kg/day)

bw = body weight (kg)

DWI = drinking water intake (L/day)

DWI/bw = 0.054 L/kg-day

Then, the DWEL is multiplied by the relative source contribution (RSC). The RSC is the percentage of total drinking water exposure, after considering other exposure routes (for example, food, inhalation). Following EPA's Exposure Decision Tree in its 2000 methodology (EPA, 2000), significant potential sources other than drinking water ingestion exist; however, information is not available to quantitatively characterize exposure from all of these different sources (Box 8B in the Decision Tree). Therefore, EPA recommends a RSC of 20% (0.20) for PFOA and PFOS.

Thus, the lifetime health advisory (HA) is calculated after application of a 20% RSC as follows:

$$HA = DWEL \times RSC$$

The two above equations can be combined to generate:

HA = (RfD / (DWI/bw)) x RSC

For these purposes, we can assume that ATSDR's MRL is equivalent to a RfD, and an HA equivalent to a MCLG.

MCLG = (MRL / (DWI/bw)) x RSC

The EPA used estimated drinking water parameters for lactating mothers, making the equation:

MCLG = (MRL / 0.054 L/kg-day) x 0.2

*NOTE:

DWI/bw for average adult = 0.029 L/kg-day, used by New Jersey;

DWI/bw for lactating mother = 0.054 L/kg-day, used by EPA; and

DWI/bw for breastfeeding or formula-fed infant = 0.175 L/kg-day, used by Vermont

This equation can be applied to proposed and candidate MRLs from ATSDR (final values are rounded):

Using ATSDR's proposed MRLs and drinking water assumptions for lactating women:

PFOA

MCLG = (3×10^{-6} mg/kg/day / 0.054 L/kg-day) x 0.2 = 1.11×10^{-5} mg/L = 11 ng/L or ppt

PFOS

MCLG = (2×10^{-6} mg/kg/day / 0.054 L/kg-day) x 0.2 = 7.41×10^{-6} mg/L = 7 ng/L or ppt

PFNA

MCLG = (3×10^{-6} mg/kg/day / 0.054 L/kg-day) x 0.2 = 1.11×10^{-5} mg/L = 11 ng/L or ppt

PFHxS

MCLG = (2 x 10^{-5} mg/kg/day / 0.054 L/kg-day) x 0.2 = 7.41 x 10^{-5} mg/L = 74 ng/L or ppt

Using NRDC's estimated MRLs for immunotoxicity studies and drinking water assumptions for lactating women:

In Appendix A we noted that ATSDR did not choose to use the most sensitive endpoint for PFOS. Here we show the MCLGs that would result if the studies with most sensitive endpoints were to be chosen for calculation of MRL as in Appendix A and translated to MCLGs using the drinking water assumptions for lactating women.

Dong et al. 2011

MCLG = (3 x 10^{-6} mg/kg/day / 0.054 L/kg-day) x 0.2 = 1.11 x 10^{-5} mg/L = 11 ng/L or ppt

Dong et al. 2009

MCLG = (8 x 10^{-7} mg/kg/day / 0.054 L/kg-day) x 0.2 = 2.96 x 10^{-6} mg/L = 3 ng/L or ppt

Guruge et al. 2009

MCLG = (2 x 10^{-7} mg/kg/day / 0.054 L/kg-day) x 0.2 = 7.41 x 10^{-7} mg/L, **0.7 ng/L (< 1 ppt)**

Peden-Adams et al. 2008

MCLG = (2 x 10^{-8} mg/kg/day / 0.054 L/kg-day) x 0.2 = 7.41 x 10^{-8} mg/L, **0.07 ng/L (< 1 ppt)**

In Appendix B we noted that ATSDR did not use the half-life for PFNA that was the most representative. Here we show the MCLG that would result if the longer, more representative half-life were to be chosen for calculation of the MRL as in Appendix B and translated to a MCLG using drinking water assumptions for lactating women.

MCLG = (2 x 10^{-6} mg/kg/day / 0.054 L/kg-day) x 0.2 = 7.41 x 10^{-6} mg/L = 7 ng/L or ppt

Using ATSDR's proposed MRLs and drinking water assumptions for infants:

Vermont used the drinking water assumptions for breastfeeding or formula-fed infants of 0.175 L/kg-day. If this value is used, the equation becomes:

MCLG = (MRL / 0.175 L/kg-day) x 0.2

This equation can be applied to proposed and candidate MRLs from ATSDR (final values are rounded):

PFOA

MCLG = (3×10^{-6} mg/kg/day / 0.175 L/kg-day) x 0.2 = 3.43×10^{-6} mg/L = 3 ng/L or ppt

PFOS

MCLG = (2×10^{-6} mg/kg/day / 0.175 L/kg-day) x 0.2 = 2.29×10^{-6} mg/L = 2 ng/L or ppt

PFNA

MCLG = (3×10^{-6} mg/kg/day / 0.175 L/kg-day) x 0.2 = 3.43×10^{-6} mg/L = 3 ng/L or ppt

PFHxS

MCLG = (2×10^{-5} mg/kg/day / 0.175 L/kg-day) x 0.2 = 2.29×10^{-5} mg/L = 23 ng/L or ppt

Using NRDC's estimated MRLs for immunotoxicity studies and drinking water assumptions for infants:

Candidate MRL's (rounded) for immunotoxicity studies identified by ATSDR, calculated in Appendix B:

Dong et al. 2011

MCLG = (3×10^{-6} mg/kg/day / 0.175 L/kg-day) x 0.2 = 3.43×10^{-6} mg/L = 3 ng/L or ppt

Dong et al. 2009

MCLG = (8×10^{-7} mg/kg/day / 0.175 L/kg-day) x 0.2 = 9.14×10^{-7} mg/L, **0.9 ng/L (< 1 ppt)**

Guruge et al. 2009

MCLG = (2×10^{-7} mg/kg/day / 0.175 L/kg-day) x 0.2 = 2.28×10^{-7} mg/L, **0.2 ng/L (< 1 ppt)**

Peden-Adams et al. 2008

MCLG = (2×10^{-8} mg/kg/day / 0.175 L/kg-day) x 0.2 = 2.28×10^{-8} mg/L, **0.02 ng/L (< 1 ppt)**

Candidate MRL's (rounded) for PFNA using longer half-life estimate, calculated in Appendix C:

MCLG = (2 x 10^{-6} mg/kg/day / 0.175 L/kg-day) x 0.2 = 2.28 x 10^{-6} mg/L = **2 ng/L or ppt**

****ALSO NOTE:** All estimated MCLGs presented here would be an order of magnitude lower/stricter if an additional UF of 10 was applied to the RfD or MRL to protect fetuses, infants and children as recommended by the National Academy of Sciences (NAS, 1993) for pesticides and as required in the Food Quality Protection Act. 21 U.S.C. §346a(b)(2)(C)(ii)(II).

APPENDIX D - MCLG CALCULATIONS FOR PFOA BASED ON REFERENCE DOSE CALCULATED BY NEW JERSEY FOR ALTERED MAMMARY GLAND DEVELOPMENT

Based on information from Gleason et al., 2017, found at:
https://www.nj.gov/dep/watersupply/pdf/pfoa-appendixa.pdf

Selected Study

The New Jersey Drinking Water Quality Institute selected the late gestational exposure study conducted by Macon et al. 2011[63] because it was the only developmental exposure study of mammary gland development that provides serum PFOA data from the end of the dosing period (PND 1) that can be used for dose-response modeling.

Determination of Point of Departure (POD)

EPA Benchmark Dose Modeling Software 2.1.2 was used to perform Benchmark Dose (BMD) modeling of the data for two endpoints, mammary gland developmental score and number of terminal endbuds, at PND 21 from Macon et al. 2011[63], using serum PFOA data from PND 1 as the dose. Continuous response models were used to obtain the BMD and the Benchmark Dose Lower (BMDL) for a 10% change from the mean for the two endpoints. The lowest significant BMDL, for decreased number of terminal endbuds, of 22.9 ng/ml in serum was used as the POD for reference dose (RfD) development.

Target Human Serum Level

Uncertainty factors (UFs) were applied to the POD to obtain the Target Human Serum Level. The Target Human Serum Level (ng/ml in serum) is analogous to a RfD but is expressed in terms of internal dose rather than administered dose. The total of the uncertainty factors (UFs) applied to the POD serum level was 30 (10 for human variation and 3 for animal-to-human extrapolation).

The target human serum level is: (22.9 ng/ml) / 30 = 0.8 ng/ml (800 ng/L).

Reference Dose (RfD)

EPA used a pharmacokinetic modeling approach to develop a species-independent clearance factor, 1.4×10^{-4} L/kg/day that relates serum PFOA level (μg/L) to human PFOA dose (μg/kg/day). The clearance factor can be used to calculate the RfD, as follows:

RfD = Target Human Serum Level x Clearance factor

RfD = 800 ng/L x 1.4 x 10^{-4} L/kg/day = 0.11 ng/kg/day

Where:

Target Human Serum Level = 800 ng/L

Clearance factor = 1.4 x 10-4 L/kg/day

RfD = Reference Dose = 0.11 ng/kg/day

Maximum Contaminant Level Goal (MCLG) for Drinking Water

Default relative source contribution (RSC) of 20% is used to develop the Health-based MCLG.

To calculate a Health-based MCLG based on mammary gland effects instead of hepatic effects:

MCLG = (RfD x bw x RSC) / DWI

MCLG = (0.11 ng/kg/day x 70 kg x 0.2) / (2 L/day) = **0.77 ng/L (< 1 ppt)**

Where:

RfD = Reference Dose for altered mammary gland development = 0.11 ng/kg/day

bw = assumed adult body weight = 70 kg

RSC = Relative Source Contribution from drinking water = 0.2

DWI = assumed adult daily drinking water intake = 2 L/day

***NOTE:** A MCLG based on mammary gland effects using EPA's drinking water exposure assumptions (for a lactating mother) or Vermont's drinking water exposure assumptions (breastfeeding infant) would result in an even lower MCLG than calculated above. (See Appendix C)

For example, if the drinking water exposure parameters for lactating mothers (EPA) is used:

MCLG = (0.11 ng/kg/day / 0.054 L/kg-day) x 0.2 = **0.41 ng/L (<1 ppt)**

If drinking water exposure parameters for infants under 1 year of age is used (as was done in Vermont):

MCLG = (0.11 ng/kg/day / 0.175 L/kg-day) x 0.2 = **0.13 ng/L (<1 ppt)**

APPENDIX E – APPROXIMATION OF RSC USED BY ATSDR FOR DRINKING WATER ENVIRONMENTAL MEDIA EVALUATION GUIDES

In November 2018 ATSDR published the webpage https://www.atsdr.cdc.gov/pfas/mrl_pfas.html, which stated:

"When ATSDR uses an average adult's or child's weight and water intake to convert these MRLs into drinking water concentrations, the individual PFOA, PFOS, PFHxS, and PFNA concentrations are

- PFOA: 78 ppt (adult) and 21 ppt (child)
- PFOS: 52 ppt (adult) and 14 ppt (child)
- PFHxS: 517 ppt (adult) and 140 ppt (child)
- PFNA: 78 ppt (adult) and 21 ppt (child)"

In posting this webpage, ATSDR provided minimal information as to how the proposed drinking water values were calculated and what assumptions were made and used in their derivation. According to ATSDR, their calculations were based on,

"…the guidelines published in the Public Health Assessment Guidance Manual, and the EPA 2011 Exposure Factors Handbook External. For example, for an estimate of a child's drinking water exposure, ATSDR bases this calculation on an infant (age birth to one year old) weighing 7.8 kg and an intake rate of 1.113 liters per day. For an adult's drinking water exposure, ATSDR bases this calculation on a body weight of 80 kg and an intake rate of 3.092 liters per day. Scientists may use different assumptions when calculating concentrations from dosages."

In this Appendix we back calculate to derive the missing information, namely the relative source contribution (RSC).

From Appendix C:

$$MCLG = (MRL / (DWI/bw)) \times RSC$$

Where (values provided by ATSDR on website):

DWI for adults = 3.092 L/day

and

bw for adults = 80 kg

thus,

DWI/bw for adults = 0.0387 L/kg/day

DWI for children = 1.113 L/day

and

bw for children = 7.8 kg

thus,

DWI/bw for children = 0.142 L/kg/day

So, for adults:

MCLG = (MRL / (0.039 L/kg/day)) x RSC*

And for children:

MCLG = (MRL / (0.142 L/kg/day)) x RSC*

*RSC not provided by ATSDR, however, drinking water values provided by ATSDR can be used with these equations to solve for the RSC used by ATSDR. For example, for PFOA:

Adults:

RSC = (MCLG x DWI/bw) / MRL

RSC = (78 ng/L x 0.0387 L/kg/day) / 3 ng/kg/day

RSC = 1

Children:

RSC = (MCLG x DWI/bw) / MRL

RSC = (21 ng/L x 0.142 L/kg/day) / 3 ng/kg/day

RSC = 1

APPENDIX F – RFD AND MCLG CALCULATIONS FOR GENX

From EPA's Draft Toxicity Assessment of GenX chemicals:

https://www.epa.gov/sites/production/files/2018-11/documents/genx_public_comment_draft_toxicity_assessment_nov2018-508.pdf

"…POD human equivalent dose is 0.023 mg/kg/day. UF applied include a 10 for intraspecies variability, 3 for interspecies differences, and 3 for database deficiencies, including immune effects and additional developmental studies, to yield a subchronic RfD of 0.0002 mg/kg/day. In addition to those above, a UF of 3 was also applied for extrapolation from a subchronic to a chronic duration in the derivation of the chronic RfD of 0.00008 mg/kg/day."

If uncertainty factors that properly reflected the deficiencies in toxicity data (database, sub-chronic/chronic, children's vulnerability, inter/intra species) were used, the combined uncertainty factor could be as high as 100,000 (see Part IV, section GenX).

From pg. 58 of EPA's Draft Toxicity Assessment of GenX chemicals:

RfD = POD/total UF

With NRDC recommended UFs:

RfD = (0.023 mg/kg/day)/100,000 = 2.3×10^{-7} mg/kd/day

Where:

POD = Point of departure human equivalent dose

Total UF = 10 for intraspecies variability, 10 for interspecies differences, 10 for database limitations, 10 for extrapolation from subchronic to chronic duration, and 10 to protect fetuses, infants and children.

From Appendix C:

MCLG = (RfD / (DWI/bw)) x RSC

Using drinking water exposure parameters for lactating mothers, DWI/bw = 0.054 L/kg-day, the MCLG based on liver toxicity would be (rounded):

MCLG = (2 x 10^{-7} mg/kd/day / 0.054 L/kg-day) x (0.2 RSC) = 7.41 x 10^{-7} mg/L = **0.7 ppt**

Using drinking water exposure parameters for an infant under 1 year, DWI/bw = 0.175 L/kg-day, the MCLG based on liver toxicity would be (rounded):

MCLG = (2 x 10^{-7} mg/kd/day / 0.175 L/kg-day) x (0.2 RSC) = 2.29 x 10^{-7} mg/L = **0.2 ppt**

*NOTE: A MCLG based on EPA's proposed RfD for GenX based on liver toxicity would be (rounded):

Using drinking water exposure parameters for lactating mothers

MCLG = (8 x 10^{-5} mg/kd/day / 0.054 L/kg-day) x (0.2 RSC) = 2.96 x 10^{-4} mg/L = 296 ppt

Using drinking water exposure parameters for an infant under 1 year

MCLG = (8 x 10^{-5} mg/kd/day / 0.175 L/kg-day) x (0.2 RSC) = 9.14 x 10^{-5} mg/L = 91 ppt

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ACKNOWLEDGEMENTS

The authors are enormously grateful to Dr. Katie Pelch, Senior Scientist at The Endocrine Disruption Exchange, Dr. Sonya Lunder, Senior Toxics Advisor for the Gender, Equity & Environment Program at Sierra Club, and Dr. Christina Swanson, Director of the Science Center at NRDC, for their careful review and thoughtful comments on this report. The authors also acknowledge the invaluable contributions of: Erik Olson, Mae Wu, Mekela Panditharatne, Cyndi Roper, Joan Leary Matthews, Kim Ong, Miriam Rotkin-Ellman, and Alex Franco.

REFERENCES

[1] Ballesteros V, et al., 2017. Exposure to perfluoroalkyl substances and thyroid function in pregnant women and children: A systematic review of epidemiologic studies. *Environ Int* 99:15-28.

[2] Post GB, et al., 2012. Perfluoroocantoic acid (PFOA), an emerging drinking water contaminant: A critical review of recent literature. *Env Research* 116(2012) 93-117.

[3] U.S. Environmental Protection Agency, 2016a. Drinking Water Health Advisory for perfluorooctanoic acid (PFOA). May 2016. EPA 822-R-16-005. U.S. Environmental Protection Agency, Office of Water. Washington, DC.

[4] Schultz MM, et al., 2003. Fluorinated alkyl surfactants. *Environmental Engineering Science*, *20*(5), 487-501.

[5] Agency for Toxic Substances and Disease Registry, 2018. Toxicological Profile for Perfluoroalkyls. Draft for Public Comment, June 2018.

[6] Centers for Disease Control and Prevention, 2018. Fourth National Report on Human Exposure to Environmental Chemicals. Department of Health and Human Services. Updated Tables, March 2018. https://www.cdc.gov/exposurereport/pdf/FourthReport_UpdatedTables_Volume1_Mar2018.pdf

[7] Hu XC, et al., 2016. Detection of PFASs in US drinking water linked to industrial sites, military fire training areas, and waste water treatment plants. *Env Sci and Tech Letters* 3(10):344–350

[8] U.S. Environmental Protection Agency, PFOA Stewardship Program, https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/risk-management-and-polyfluoroalkyl-substances-pfass#tab-3

[9] U.S. Environmental Protection Agency notes that "Although PFOA and PFOS are no longer manufactured in the United States, they are still produced internationally and can be imported into the United States in consumer goods such as carpet, leather and apparel, textiles, paper and packaging, coatings, rubber and plastics." U.S. Environmental Protection Agency, Basic Information on PFAS, Accessed on February 6, 2019 https://www.epa.gov/pfas/basic-information-pfas

[10] Wang Z, et al., 2017. A never-ending story of per- and polyfluoroalkyl substances (PFASs)? *Environ Sci Technol* 51(5):2508-2518

[11] Scheringer M, et al., 2014. Helsingør statement on poly- and perfluorinated alkyl substances (PFASs). *Chemosphere* 114:337-339

[12] Blum A, et al., 2015. The Madrid Statement on Poly- and Perfluoroalkyl Substances (PFASs). *Environ Health Perspect* 123(5):A107-A111

[13] Lau C, et al., 2007. Perfluoroalkyl Acids: A Review of Monitoring and Toxicological Findings. *Toxicol Sci* 99(2):366-394.

[14] Lilienthal H, et al. 2017. Recent experimental results of perfluoroalkyl substances in laboratory animals in relation to current regulations and guidance values. *Int J Hyg Environ Health* 220(4):766-775.

[15] C8 Science Panel Report, 2017 (and related sub-sections). Accessed October 2018. http://www.c8sciencepanel.org/

[16] D'Eon JC and Mabury SA, 2007. Production of perfluorinated carboxylic acids (PFCAs) from the biotransformation of polyfluoroalkyl phosphate surfactants (PAPS): exploring routes of human contamination. Environ Sci Technol 41(13):4799–4805; doi:10.1021/es070126x

[17] Safer Consumer Products, 2018. *Product-Chemical Profile for Perfluoroalkyl and Polylfluoroalkyl Substances (PFASs) in Carpets and Rugs*. Retrieved from https://www.dtsc.ca.gov/SCP/upload/Product-Chemical-Profile-PFAS-Carpets-and-Rugs.PDF

[18] Jian J, et al., 2017. Global distribution of perfluorochemicals (PFCs) in potential human exposure - A review. *Environ Int* 108: 51-62

[19] Eriksson U and Kärrman A, 2015. World-wide indoor exposure to polyfluoroalkyl phosphate esters (PAPs) and other PFASs in household dust. *Environmental science & technology*, *49*(24), 14503-14511.

[20] Lee H, et al., 2013. Fate of polyfluoroalkyl phosphate diesters and their metabolites in biosolids-applied soil: biodegradation and plant uptake in greenhouse and field experiments. *Environmental science & technology*, *48*(1), 340-349.

[21] KEMI, 2017. Proposal for a ban on 200 highly fluorinated substances. December 20, 2017. https://www.kemi.se/nyheter-fran-kemikalieinspektionen/2017/forslag-om-forbud-mot-200-hogfluorerade-amnen/

[22] Fromme H, et al., 2009. Perfluorinated compounds - Exposure assessment for the general population in western countries. Int J Hyg Environ Health 212(3):239-270 doi:10.1016/j.ijheh.2008.04.007

[23] U.S. Environmental Protection Agency, 2018b. Toxicity Assessment: Human Health Toxicity Values for Hexafluoropropylene Oxide (HFPO) Dimer Acid and Its Ammonium Salt (CASRN 13252-13-6 and CASRN 62037-80-3). November 2018. EPA 823-P-18-001. U.S. Environmental Protection Agency, Office of Water. Washington, DC.

[24] U.S. Environmental Protection Agency, 2018c. Toxicity Assessment: Human Health Toxicity Values for Perfluorobutane Sulfonic Acid (CASRN 375-73-5) and Related Compound Potassium Perfluorobutane Sulfonate (CASRN 29420-49-3). November 2018. EPA 823-R-18-0307. U.S. Environmental Protection Agency, Office of Water. Washington, DC.

[25] U.S. Environmental Protection Agency, 2009b. In the matter of: Premanufacture Notice Numbers: Dupont Company, April 9, 2009. https://assets.documentcloud.org/documents/2746607/Sanitized-Consent-Order-P08-0508-and-P08-0509.pdf

[26] Michigan PFAS Science Advisory Panel, 2018. Scientific Evidence and Recommendations for Managing PFAS Contamination in Michigan. December 7, 2018.

[27] Kato K, et al., 2011. Trends in exposure to polyfluoroalkyl chemicals in the US population: 1999-2008. *Environ Sci & Tech* 45:8037-8045.

[28] U.S. Environmental Protection Agency, 2016b. Drinking Water Health Advisory for perfluorooctanesulfonate (PFOS). May 2016. EPA 822-R-16-004. U.S. Environmental Protection Agency, Office of Water. Washington, DC.

[29] Emmett EA, et al., 2006. Community Exposure to Perfluorooctanoate: Relationships Between Serum Concentrations and Exposure Sources. *J Occup Environ Med* 48(8): 759-770.

[30] Yeung LWY, et al., 2008. Perfluorinated compounds and total and extractable organic fluorine in human blood samples from China. Environ. Sci. Technol 42(21): 8140-8145.

[31] Yeung LW and Mabury SA, 2016. Are humans exposed to increasing amounts of unidentified organofluorine. *Environ. Chem*, *13*(1), 102-110.

[32] Gyllenhammar K, et al., 2018. Perfluoroalkyl Acids (PFAAs) in serum from 2-4-month-old infants: Influence of maternal serum concentration, gestational age, breast-feeding, and contaminated drinking water. *Environ Sci Technol* 52:7101-7110

[33] Llorca M, et al., 2010. Infant exposure of perfluorinated compounds: levels in breast milk and commercial baby food. *Environ Int* 36(6): 584-592

[34] Manzano-Salgado CB, et al. 2015. Transfer of perfluoroalkyl substances from mother to fetus in a Spanish birth cohort. *Environ Res* 142:471-478. 10.1016/j.envres.2015.07.020

[35] Begley TH, et al., 2008. Migration of fluorochemical-paper additives from food-contact paper into foods and food simulants. Food Addit Contam Part A Chem Anal Control Expo Risk Assess 25(3):384–390; doi:10.1080/02652030701513784

[36] Kim SK, et al., 2011. Distribution of perfluorochemicals between sera and milk from the same mothers and implications for prenatal and postnatal exposures. *Environ Pollut* 159(1):169-174.

[37] Liu J, et al., 2011. Comparison on gestation and lactation exposure of perfluorinated compounds for newborns. *Environ Int* 37(7):1206-1212.

[38] Vestergren R and Cousins IT, 2009. Tracking the pathways of human exposure to perfluorocarboxylates. *Environ Sci Technol* 43:5565-5575

[39] Eaton A, 2017. A Further Examination of a Subset Of UCMR 3 PFAS Data Demonstrates Wider Occurrence. Accessed in September 2018. http://greensciencepolicy.org/wp-content/uploads/2017/12/Andy_Eaton_UCMR3_PFAS_data.pdf

[40] U.S. Environmental Protection Agency, Unregulated Contaminant Monitoring Rule 3, 77 Fed. Reg. 26071-26101 (May 2, 2012), summarized at https://www.epa.gov/dwucmr/third-unregulated-contaminant-monitoring-rule (last visited February 8, 2019).

[41] Dong Z, et al., 2017. Issues raised by the reference doses for PFOS and PFOA. *Environ Int* 105:86-94.

[42] Winkens K, et al., 2017. Early life exposure to per- and polyfluoroalkyl substances (PFASs): A critical review. *Emerging Contaminants* 3(2):55-68

[43] Chang E, et al., 2016. A critical review of perfluorooctanoate and perfluorooctansulfonate exposure and immunological health conditions in humans. *Crit Rev Toxicol* 46(4):279-331.

[44] Public Health Research and Cancer, 2018. Accessed November 2018. https://www.cancer.gov/research/areas/public-health

[45] Benbrahim-Tallaa L, et al., 2014. Carcinogenicity of perfluorooctanoic acid, tetrafluoroethylene, dichloromethane, 1,2-dichloropropane, and 1,3-propane sultone. *Lancet Oncol* 15(9):924-925

[46] EPA Science Advisory Board, 2006. SAB Review of EPA's Draft Risk Assessment of Potential Human Health Effects Associated with PFOA and Its Salts. EPA-SAB-06-006, May 30, 2006.

[47] Barry V, et al., 2013. PFOA exposures and incident cancers among adults living near a chemical plant. *Environ Health Perspect* 121 (11-12):1313-1318

[48] Vieira VM, et al., 2013. Perfluorooctanoic acid exposure and cancer outcomes in a contaminated community: a geographic analysis. *Environmental health perspectives*, *121*(3), 318.

[49] Steenland K and Woskie S, 2012. A cohort mortality study of workers exposed to perfluorooctanoic acid. *Am J Epidemiol* 176(10):909-917.

[50] Thomford PJ, 2002. 104-Week Dietary Chronic Toxicity and Carcinogenicity Study with Perfluorooctane Sulfonic Acid Potassium Salt (PFOS; T-6295) in Rats. Final Report, 3M T-6295 (Covance Study No. 6329-183), Vol. I-IX, 4068 pages, January 2, 2002. 3M, St. Paul, MN.

[51] Steenland K, et al., 2015. A cohort incidence study of workers exposed to perfluorooctanoic acid (PFOA). *Occup Environ Med* 72(5):373-380.

[52] Raleigh KK, et al., 2014. Mortality and cancer incidence in ammonium perfluorooctanoate production workers. *Occup Environ Med* 71(7):500-506.

[53] Bonefeld-Jorgensen EC, et al., 2011. Perfluorinated compounds are related to breast cancer risk in Greenlandic Inuit: a case control study. *Environ Health* 10:88

[54] Ghisari M, et al., 2014. Polymorphisms in phase I and phase II genes and breast cancer risk and relations to persistent organic pollutant exposure: a case–control study in Inuit women. *Environ Health* 13(1):19

[55] Bonefeld-Jorgensen EC, et al., 2014. Breast cancer risk after exposure to perfluorinated compounds in Danish women: A case control study nested in the Danish National Birth Cohort. *Cancer Causes Control* 25(11):1439-1448.

[56] New York State Department of Health, 2017a. Cancer Incidence Investigation 1995-2014, Village of Hoosick Falls, Rensselaer County, New York. May 2017.

[57] Hardell E, et al., 2014. Case-control study on perfluorinated alkyl acids (PFAAs) and the risk of prostate cancer. *Environ Int* 63:35-39

[58] Ducatman A, et al., 2015a. Letter to the editor, commenting on: Prostate-specific antigen and perfluoroalkyl acids in the C8 health study population. *J Occup Environ Med* 57(6): e61.

[59] Ducatman A, et al., 2015b. Prostate-specific antigen and perfluoroalkyl acids in the C8 health study population. *J Occup Environ Med* 57(1): 111-114.

[60] Apelberg B, et al., 2007. Cord serum concentrations of perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA) in relation to weight and size at birth. *Environ Health Perspectives*, 115 (11):1670-1676

[61] Johnson P, et al., 2014. The Navigation Guide - Evidence of medicine meets environmental health: Systematic review of human evidence for PFOA effects on fetal growth. *Environ Health Perspectives* 122(10):1028-1039

[62] Rappazzo K, et al., 2017. Exposure to perfluorinated alkyl substances and health outcomes in children: A systematic review of the epidemiologic literature. *Int J Environ Res Public Health* 14(7):691.

[63] Macon MB, et al., 2011. Prenatal perfluoroocyanoic acid exposure in CD-1 mice: low dose developmental effects and internal dosimetry. *Toxicol Sci* 122(1):131-145.

[64] White SS, et al., 2011. Gestational and chronic low-dose PFOA exposures and mammary gland growth and differentiation in three generations of CD-1 mice. *Environ Health Perspect* 119(8):1070-1076

[65] Tucker DK, et al., 2015. The mammary gland is a sensitive pubertal target in CD-1 and C57Bl/6 mice following perinatal perfluorooctanoic acid (PFOA) exposure. *Reprod Toxicol* 54:26-36.

[66] National Toxicology Program, 2016. NTP Monograph on Immunotoxicity Associated with Exposure to Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS). September 2016. Office of Health Assessment and Translation, Division of the National Toxicology Program, U.S. Department of Health and Human Services.

[67] Mondal D, et al., 2014. Breastfeeding: a potential excretion route for mothers and implications for infant exposure to perfluoroalkyl acids. *Environ Health Perspect* 122(2):187-192

[68] Brendel S, et al., 2018. Short-chain perfluoroalkyl acids: environmental concerns and a regulatory strategy under REACH. *Environ Sci Eur* 30(1):9

[69] Gomis MI, et al., 2018. Comparing the toxic potency in vivo of long-chain perfluoroalkyl acids and fluorinated alternatives. *Environ Int* 113:1–9.

[70] Wang Z, et al., 2015. Hazard assessment of fluorinated alternatives to long-chain perfluoroalkyl acids (PFAAs) and their precursors: Status quo, ongoing challenges and possible solutions. *Environ Int* 75:172-179

[71] Neumann M & Schliebner I, 2017. Protecting the sources of our drinking water. *German Environment Agency (UBA)*, 20.

[72] SGS, 2017. EU Regulates PFOA and Related Substances under REACH. June 23, 2017. Accessed January, 2019 https://www.sgs.com/en/news/2017/06/safeguards-09717-eu-regulates-pfoa-and-related-substances-under-reach

[73] Hu X, et al., 2013. Determination of gaseous and particulate trifluoroacetic acid in atmosphere environmental samples by gas chromatography-mass spectrometry. *Chin J Anal Chem* 41, 1140–1146. doi: 10.1016/S1872-2040(13)60676-3

[74] Scheurer M, et al., 2017. Small, mobile, persistent: Trifluoroacetate in the water cycle - Overlooked sources, pathways, and consequences for drinking water supply. *Water Res* 126, 460–471. doi: 10.1016/j.watres.2017.09.045

[75] Arp HPH, et al., 2017. Ranking REACH registered neutral, ionizable and ionic organic chemicals based on their aquatic persistency and mobility. *Environ Sci Process Impacts* 19, 939–955. doi: 10.1039/C7EM00158D

[76] Henry BJ, et al., 2018. A critical review of the application of polymer of low concern and regulatory criteria to fluoropolymers: Fluoropolymers PLC. *Integr Environ Assess Manag*, *14*(3), 316–334.

[77] Pérez F, et al., 2013. Accumulation of perfluoroalkyl substances in human tissues. *Environ Int, 59*, 354-362.

[78] Liu X, et al., 2014. Concentrations and trends of perfluorinated chemicals in potential indoor sources from 2007 through 2011 in the US. *Chemosphere* 98:51-57.

[79] Guo, Z, et al., 2009. Perfluorocarboxylic acid content in 116 articles of commerce. *Research Triangle Park, NC: US Environmental Protection Agency*

[80] Fraser AJ, et al., 2013. Polyfluorinated compounds in dust from homes, offices, and vehicles as predictors of concentrations in office workers' serum. *Environ Int* 60:128-136

[81] U.S. Environmental Protection Agency, 1993. Reference Dose (RfD): Description and Use in Health Risk Assessments. Background Document 1A. March 15, 1993 https://www.epa.gov/iris/reference-dose-rfd-description-and-use-health-risk-assessments

[82] U.S. Environmental Protection Agency, 2018a. About Risk Assessment. Accessed September 2018. https://www.epa.gov/risk/about-risk-assessment

[83] National Academy of Sciences, 2013a. Risk Assessment and Uncertainty, Chapter 2. https://www.ncbi.nlm.nih.gov/books/NBK200844/

[84] National Academy of Sciences, 2013b. Science and Decisions: Advancing Risk Assessment. National Research Council. National Academies Press

[85] Lau C, et al., 2006. Effects of perfluorooctanoic acid exposure during pregnancy in the mouse. *Toxicol Sci* 90:510–518.

[86] U.S. Environmental Protection Agency, 2011. Exposure Factors Handbook: 2011 Edition (Final). EPA/600/R-09/052F. U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment. Washington, DC.

[87] Vermont Department of Health, 2016. Perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS) Vermont Water Health Advisory. Memo dated June 22, 2016.

[88] Trudel D, et al., 2008. Estimating consumer exposure to PFOS and PFOA. *Risk Anal, 28*(2), 251-269.

[89] U.S. Environmental Protection Agency, 2008. Child-Specific Exposure Factors Handbook. EPA/600/R-06/096F. Washington, D.C. U.S. EPA, Office of Research and Development, National Center for Environmental Assessment.

[90] Minnesota Department of Health, 2018a. Toxicological Summary for: Perfluorooctanoate. August 2018.

[91] Goaden HM, et al., 2019. A transgenerational toxicokinetic model and its use in derivation of Minnesota PFOA water guidance. *J Expo Sci & Environ Epi* 29:1833-195

[92] New Jersey Drinking Water Quality Institute, 2017. Health-based maximum contaminant level support document: Perfluorooctanoic acid (PFOA). February 2017.

[93] New Jersey Department of Environmental Protection, Division of Science, Research & Environmental Health, 2019. Technical support document: interim specific ground water criterion for perfluorooctanoic acid (PFOA, C8) (CAS #: 335-67-1; Chemical Structure:

CF3(CF2)6COOH)
https://www.nj.gov/dep/dsr/Technical%20Support%20Document%20Draft%20ISGWQC%20for%20PFOA.pdf

[94] NJ Department of Environmental Protection, 2019. Notice of Rule Proposal https://www.nj.gov/dep/rules/notices/20190401a.html

[95] Koskela A, et al., 2016. Effects of developmental exposure to perfluorooctanoic acid (PFOA) on long bone morphology and bone cell differentiation. *Toxicol Appl Pharmacol* 301:14-21.

[96] Onishchenko N, et al., 2011. Prenatal exposure to PFOS or PFOA alters motor function in mice in a sex-related manner. *Neurotox Res* 19(3):452-461

[97] ATSDR, Accessed November 2018. https://www.atsdr.cdc.gov/pfas/mrl_pfas.html

[98] OEHHA, 2018. California Office of Environmental Health Hazard Assessment. Memo dated June 26, 2018: Recommendation for Interim Notification Levels for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)
https://www.waterboards.ca.gov/drinking_water/certlic/drinkingwater/documents/pfos_and_pfoa/OEHHA_Recommended_Int_NL_Jun_26_2018.pdf

[99] New York State Department of Health. December 18, 2018.
https://www.health.ny.gov/press/releases/2018/2018-12-18_drinking_water_quality_council_recommendations.htm

[100] Rudel RA, et al., 2011. Environmental exposures and mammary gland development: State of the science, public health implications, and research recommendations. *Environ Health Perspect* 119(8):1053-1061

[101] Macon MB and Fenton SE, 2013. Endocrine disruptors and the breast: Early life effects and later life disease. *J Mammary Gland Biol Neoplasia* 18(1):43-61.

[102] Romano M, et al., 2016. Maternal serum perfluoroalkyl substances during pregnancy and duration of breastfeeding. *Environ Res* 149:239-246.

[103] Russo J and Russo IH, 2004. Molecular Basis of Breast Cancer: Prevention and Treatment. New York:Springer

[104] Medina D, 2007. Chemical carcinogenesis of rat and mouse mammary glands. *Breast Dis* 28:63-68

[105] Kelsey JL, et al., 1993. Reproductive factors and breast cancer. *Epidemiol Rev* 15(1):36-47

[106] Euling SY, et al., 2008. Role of environmental factors in the timing of puberty. *Pediatrics* 121(suppl 3):S167-S171

[107] Luebker D, et al., 2005. Two-generation reproduction and cross-foster studies of PFOS in rats. *Toxicology* 215(1-2):126-148.

[108] Minnesota Department of Health, 2017. Toxicological Summary for: Perfluorooctane Sulfonate. May 2017.

[109] National Academy of Sciences, 1993. Pesticides in the Diets of Infants and Children. National Research Council. National Academies Press

[110] Dong GH, et al., 2009. Chronic effects of perfluorooctanesulfonate exposure on immunotoxicity in adult male C57BL/6 mice. *Arch Toxicol* 83(9):805-815

[111] New Jersey Drinking Water Quality Institute, 2018. Health-based maximum contaminant level support document: Perfluorooctane Sulfonate (PFOS). June 2018.

[112] New Jersey Department of Environmental Protection, Division of Science, Research & Environmental Health, 2019. Technical support document: interim specific ground water criterion for perfluorooctane sulfonate (PFOS) (CAS #: 1763-23-1; Chemical Formula: C8HF17O3S)
https://www.nj.gov/dep/dsr/Technical%20Support%20Document%20ISGQWC%20for%20PFOS.pdf

[113] Dong GH, et al., 2011. Sub-chronic effect of perfluorooctanesulfonate (PFOS) on the balance of type 1 and type 2 cytokine in adult C57BL6 mice. *Arch Toxicol* 85(10):1235-1244

[114] Grandjean P and Budtz-Jorgensen E, 2013. Immunotoxicity of Perfluorinated alkylates: calculation of benchmark doses based on serum concentration in children. *Environ Health* 12 (1):35.

[115] Das KP, et al., 2015. Developmental toxicity of perfluorononanoic acid in mice. *Reprod Toxicol* 51:133-44

[116] New Jersey Drinking Water Quality Institute, 2015a. Health-based maximum contaminant level support document: Perfluoronanoci acid (PFNA). June 2015.

[117] U.S. Environmental Protection Agency, 2000. Methodology for Deriving Ambient Water Quality Criteria for the Protection of
Human Health. Office of Science and Technology. Office of Water. Washington, DC. EPA 822-B-00-004. October 2000.

http://water.epa.gov/scitech/swguidance/standards/upload/2005_05_06_criteria_humanhealth_method_complete.pdf

[118] New Jersey Department of Environmental Protection, 2018. Federal and New Jersey State Primary and Secondary Drinking Water Standards as of September 2018. https://www.state.nj.us/dep/watersupply/pdf/dw-standards.pdf

[119] Vermont Department of Health, 2018. Drinking Water Health Advisory for Five PFAS (per- and polyfluorinated alkyl substances). Memo dated July 10, 2018

[120] Vermont Natural Resources Agency. ANR Adopting Emergency PFAS Rules. July 2018 Update. https://dec.vermont.gov/news/PFAS-emergency-rule

[121] Vermont Natural Resources Agency. Accessed January 2019. https://anr.vermont.gov/content/agency-natural-resources-initiates-rulemaking-process-adopt-maximum-contaminant-level-pfas

[122] Zhang Y, et al., 2013. Biomonitoring of perfluoroalkyl acids in human urine and estimates of biological half-life. Environ Sci Technol 47(18):10619-10627

[123] Minnesota Department of Health, 2018b. Perfluoroalkyl Substances (PFAS) and Health. May 2018.

[124] Butenhoff J, et al., 2009. Evaluation of potential reproductive and developmental toxicity of potassium perfluorohexanesulfonate in Sprague Dawley rats. *Reprod Toxicol* 27(3-4):331-41.

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

[126] North Carolina Department of Human Health Services, 2017. Questions and Answers Regarding North Carolina Department of Health and Human Services Updated Risk Assessment for GenX (Perfluoro-2-propoxypropanoic acid), July 2017. https://ncdenr.s3.amazonaws.com/s3fs-public/GenX/NC%20DHHS%20Risk%20Assessment%20FAQ%20Final%20Clean%20071417%20PM.pdf

[127] Dupont Chem C. 2010. Dupont-18405-1037: An oral (gavage) reproduction/developmental toxicity screening study of h-28548 in mice. Ashland, Ohio

[128] RIVM, 2016. Evaluation of substances used in the Genx technology by Chemours, Dordrecht. RIVM Letter report 2016-0174. The Netherlands: National Institute for Public Health and the Environment.

[129] Olsen GW, et al., 2007. Half-life of serum elimination of perfluorooctanesulfonate,perfluorohexanesulfonate, and perfluorooctanoate in retired fluorochemical production workers. *Environ Health Perspect* 115:1298-1305.

[130] Buttenhoff JL, et al., 2004. Pharmacokinetics of perfluorooctanoate in cynomolgus monkeys. *Toxicol Sci*, 82:394-406.

[131] National Research Council, 2010. EPA's Methylmercury Guideline is Scientifically Justifiable for Protecting Most Americans, But Some May Be at Risk. *The National Academy of Sciences Press.* Press release - July 11, 2010. http://www8.nationalacademies.org/onpinews/newsitem.aspx?RecordID=9899.

[132] Integrated Risk Information System, 2001. Chemical Risk Assessment Summary for Methylmercury. U.S. Environmental Protection Agency. https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0073_summary.pdf

[133] European Food Safety Authority, 2018. Risk to human health related to the presence of perfluorooctane sulfonic acid and perfluorooctanoic acid in food. Scientific Opinion. *EFSA Journal* 16(12):5194

[134] Bartell SM, et al., 2017. Bayesian analysis of silica exposure and lung cancer using human and animal studies. *Epidemiology* 28:281-287 (2017). PMID: 27922537.

[135] Cordner A, et al., 2019. Guideline levels for PFOA and PFOS in drinking water: the role of scientific uncertainty, risk assessment decisions, and social factors. *J Expo Sci & Environ Epi* 29:157-171

[136] Landrigan P and Goldman L, 2011. Children's Vulnerability to Toxic Chemicals: A Challenge and Opportunity to Strengthen Health and Environmental Policy. *Health Affairs* 30(5):842-850

[137] Shoemaker J, et al., 2009. Method 537: Determination of selected perfluorinated alkyl acids in drinking water by solid phase extraction and liquid chromatography/tandem mass spectrometry (LC/MS/MS). Retrieved from https://cfpub.epa.gov/si/si_public_file_download.cfm?p_download_id=525468

[138] Shoemaker JA, et al., 2009. Development of a US EPA drinking water method for the analysis of selected perfluoroalkyl acids by solid-phase extraction and LC-MS-MS. *J Chromatogr Sci* 47(1):3-11

[139] Shoemaker J & Tettenhorst D, 2018. Method 537.1: Determination of Selected Per- and Polyfluorinated Alkyl Substances in Drinking Water by Solid Phase Extraction and Liquid

Chromatography/Tandem Mass Spectrometry (LC/MS/MS). Retrieved from https://cfpub.epa.gov/si/si_public_record_Report.cfm?dirEntryId=343042&Lab=NERL

[140] Ericson I, et al., 2009. Levels of Perfluorinated Chemicals in Municipal Drinking Water from Catalonia, Spain: Public Health Implications. *Arch Environ Contam Toxicol* 57:631–638

[141] Personal communication with Eaton Eurofins

[142] Dickenson ERV and Higgins C, 2016. Treatment Mitigation Strategies for Poly- and Perfluoroalkyl Substances. Water Research Foundation, Web Report #4322 http://www.waterrf.org/PublicReportLibrary/4322.pdf

[143] Quiñones O and Snyder S, (2009). Occurrence of perfluoroalkyl carboxylates and sulfonates in drinking water utilities and related waters from the United States. *Environ Sci Technol* 43(24): 9089-9095

[144] Gellrich V, et al., 2013. Perfluoroalkyl and polyfluoroalkyl substances (PFASs) in mineral water and tap water. *J Environ Sci Health* 48:129–135

[145] Thompson J, et al., 2011. Removal of PFOS, PFOA and other perfluoroalkyl acids at water reclamation plants in South East Queensland Australia. *Chemosphere* 82:9-17

[146] Casson R and Chaing SY, 2018. Integrating total oxidizable precursor assay data to evaluate fate and transport of PFASs. *Remediation* 28(2):71-87

[147] U.S. Environmental Protection Agency, 2018d. Reducing PFAS in Drinking Water with Treatment Technologies. Published August 23, 2018. https://www.epa.gov/sciencematters/reducing-pfas-drinking-water-treatment-technologies

[148] CDM Smith, Inc., 2018. Advanced Treatment Options for the Northwest Water Treatment Plant. Final Report Prepared for Brunswick County Public Utilities, April 2018. http://www.brunswickcountync.gov/wp-content/uploads/2018/04/CDM-Smith-Brunswick-Final-Report-April-2018.pdf

[149] RSSCT Summary Report, 2017. Removal of Short Chain PFAS Compounds via GAC. Calgon Carbon. https://www.calgoncarbon.com/app/uploads/removal_short_chain_PFAS_compounds_via_GAC_summary_report_10-3-2017.pdf

[150] Ross I, et al., 2018. A review of emerging technologies for remediation of PFASs. *Remediation* 28(2):101-126.

[151] Oliaei F, et al., 2013. PFOS and PFC releases and associated pollution from a PFC production plant in Minnesota (USA). *Environ Sci Pollut Res Int* 20(4):1977–1992.

[152] Inyang M & Dickenson ERV, 2017. The use of carbon adsorbents for the removal of perfluoroalkyl acids from potable reuse systems. *Chemosphere,* 184:168–175.

[153] McCleaf P, et al., 2017. Removal efficiency of multiple poly- and perfluoroalkyl substances (PFASs) in drinking water using granular activated carbon (GAC) and anion exchange (AE) column tests. *Water Research* 12:77–87.

[154] New Jersey Drinking Water Quality Institute, 2015b. Recommendation on Perfluorinated Compound Treatment Options for Drinking Water. June 2015.

[155] Eschauzier C, et al., 2012. Impact of treatment processes on the removal of perfluoroalkyl acids from the drinking water production chain. *Environ Sci Tech*, *46*(3), 1708-1715.

[156] Concawe Soil and Groundwater Taskforce (STF/33), 2016. Environmental fate and effects of poly- and perfluoroalkyl substances (PFAS). Brussels: Concawe. https://www.concawe.eu/wp-content/uploads/2016/06/Rpt_16-8.pdf

[157] U.S. Environmental Protection Agency, 2015. Reverse Osmosis. Retrieved January 30, 2019, from https://cfpub.epa.gov/safewater/radionuclides/radionuclides.cfm?action=Rad_Reverse%20Osmosis

[158] Michigan State University, College of Engineering. Fraunhofer Center for Coatings and Diamond Technologies (CCD). Accessed October 2018: https://www.egr.msu.edu/fraunhofer-ccd/projects/diamond-technology-cleaning-pfas-contaminated-wastewater

[159] AECOM, 2018. AECOM's Promising New PFAS Treatment Technology DE-FLUOROTM Shows Complete Destruction of PFAS. Retrieved from https://www.aecom.com/wp-content/uploads/2018/10/PFAS-Info-Sheet.pdf

[160] ATSDR, 2014. Polychlorinated Biphenyls (PCBs) Toxicity. What Are Adverse Health Effects of PCB Exposure? ATSDR Case Studies in Environmental Medicine. https://www.atsdr.cdc.gov/csem/csem.asp?csem=30&po=10

[161] U.S. Environmental Protection Agency, "National Primary Drinking Water Regulations—Synthetic Organic Chemicals and Inorganic Chemicals; Monitoring for Unregulated Contaminants; National Primary Drinking Water Regulations Implementation; National Secondary Drinking Water Regulations,", 56 Fed. Reg. 3526, at 3546 (January 30, 1991).

EXHIBIT C

	A	B	C	D	E	F	G	H	I	J	K	L	M
1	PWS_ID	SYSTEM_NAME	ADDRESS_1	ADDRESS_2	TOWN	ACTIVITY_STATUS	STARTUP_DATE	SYSTEM_TYPE	SYSTEM_CATEGORY	POPULATION_SERVED	CONNECTIONS	SEASON	
2	0015010	ACWORTH PRIMARY SCH	TURKEY SHOOT RD	ACWORTH RD	ACWORTH	ACTIVE	01-1932	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	32	1	Open: SEPTEMBER Close: JUNE	
3	0025010	WHITE MOUNTAIN WALDORF SCH	1371 RTE 16		ALBANY	ACTIVE	09-2008	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	110	6	Open: SEPTEMBER Close: JUNE	
4	0055010	ALSTEAD VILAS SCH	82 MECHANIC ST	RTE 123	ALSTEAD	ACTIVE	01-1934	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	190	2	Open: SEPTEMBER Close: JUNE	
5	0055020	ALSTEAD PRIMARY SCH	58 MECHANIC ST	RTE 123	ALSTEAD	ACTIVE	06-1991	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	177	1	Open: SEPTEMBER Close: JUNE	
6	0055030	ORCHARD SCHOOL	114 OLD SETTLERS RD	E ALSTEAD	ALSTEAD	ACTIVE	09-1994	NON-TRANSIENT NON-COMMUI	DAY CARE	45	1	Open: JANUARY Close: DECEMBER	
7	0059010	MOLE HILL THEATRE/LEAF CHARTER	789 GILSUM MINE RD		ALSTEAD	ACTIVE	05-2013	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	60	2	Open: JANUARY Close: DECEMBER	
8	0065020	PROSPECT MOUNTAIN HIGH SCH	242 SUNCOOK VALLEY RD	RTE 28	ALTON	ACTIVE	09-2004	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	500	1	Open: JANUARY Close: DECEMBER	
9	0075060	AMHERST MEDICAL CENTER	8 LIMBO LANE		AMHERST	ACTIVE	11-2015	NON-TRANSIENT NON-COMMUI	HOSPITAL, MEDICAL FACILITY	32	1	Open: JANUARY Close: DECEMBER	
10	0075070	CHRISTS CHURCH OF AMHERST	58 MERRIMACK RD		AMHERST	ACTIVE	01-2020	NON-TRANSIENT NON-COMMUI	DAY CARE	28	1	Open: JANUARY Close: DECEMBER	
11	0076040	SALZBURG SQUARE	292 HORACE GREELEY HWY	292 RTE 101	AMHERST	ACTIVE	04-1982	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	25	9	Open: JANUARY Close: DECEMBER	
12	0076070	MC SQUARE	135 RTE 101A		AMHERST	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	45	4	Open: JANUARY Close: DECEMBER	
13	0076130	COLONIAL PARK	7 NASHUA RD	7 RTE 101A	AMHERST	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	45	1	Open: JANUARY Close: DECEMBER	
14	0078090	MEETING PLACE	199 HORACE GREELEY HWY	199 RTE 101	AMHERST	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	76	12	Open: JANUARY Close: DECEMBER	
15	0086030	BELLETETES ANDOVER	18 TEN PENNY LN		ANDOVER	ACTIVE	01-2017	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	35	2	Open: JANUARY Close: DECEMBER	
16	0095010	OVERSEAS UNITED EDUC FNDTN	100 OLD NORTH BRANCH RD/RTE 9	HAWTHORNE AC/	ANTRIM	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	25	4	Open: JANUARY Close: DECEMBER	
17	0106010	ROCHESTER SHOE TREE	18 CEDAR LN	RTE 3N	ASHLAND	ACTIVE	01-1980	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	85	2	Open: JANUARY Close: DECEMBER	
18	0115010	ATKINSON ACADEMY SCH	17 ACADEMY AVE		ATKINSON	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	429	1	Open: SEPTEMBER Close: JUNE	
19	0115060	LEARNING PATH CHILD CARE CTR	72 RTE 111		ATKINSON	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	112	1	Open: JANUARY Close: DECEMBER	
20	0116010	PEU/PIONEER COMMERCE PARK	2 COMMERCE DR	RTE 111	ATKINSON	ACTIVE	01-1987	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	400	4	Open: JANUARY Close: DECEMBER	
21	0116020	GEX	2 INDUSTRIAL WAY		ATKINSON	ACTIVE	03-1998	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	62	1	Open: JANUARY Close: DECEMBER	
22	0116040	PALMER GAS	13 HALL FARM RD		ATKINSON	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	65	1	Open: JANUARY Close: DECEMBER	
23	0135010	AUBURN VILLAGE SCH	11 EATON HILL RD		AUBURN	ACTIVE	03-1960	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	766	1	Open: JANUARY Close: DECEMBER	
24	0135020	AUBURN CHILDRENS HOUSE	78 ROCKINGHAM RD		AUBURN	ACTIVE	08-1990	NON-TRANSIENT NON-COMMUI	DAY CARE	30	1	Open: JANUARY Close: DECEMBER	
25	0135030	ITS A CHILDS WORLD	32 HOOKSETT RD		AUBURN	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	51	1	Open: JANUARY Close: DECEMBER	
26	0145010	BARNSTEAD ELEMENTARY SCH	91 MAPLE ST		BARNSTEAD	ACTIVE	09-1957	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	646	1	Open: JANUARY Close: DECEMBER	
27	0146040	BARNSTEAD BUSINESS PARK	27 DEPOT RD		BARNSTEAD	ACTIVE	09-2017	NON-TRANSIENT NON-COMMUI	DAY CARE	52	2	Open: JANUARY Close: DECEMBER	
28	0155020	EARLY CHILDHOOD LEARNING CTR	77 RAMSDELL LN		BARRINGTON	ACTIVE	09-1975	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	179	1	Open: SEPTEMBER Close: JUNE	
29	0155050	BARRINGTON ELEMENTARY SCH	570 CALEF HWY		BARRINGTON	ACTIVE	01-1990	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	488	2	Open: SEPTEMBER Close: JUNE	
30	0155070	FRISBIE HEALTH SERVICES	426 CALEF HWY		BARRINGTON	ACTIVE	10-2001	NON-TRANSIENT NON-COMMUI	HOSPITAL, MEDICAL FACILITY	150	1	Open: JANUARY Close: DECEMBER	
31	0155080	BARRINGTON MIDDLE SCH	51 HALEY DR	85 RTE 9	BARRINGTON	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	510	1	Open: JANUARY Close: DECEMBER	
32	0155090	STORYBOOK HOLLOW ELP	41 COMMERCE WAY		BARRINGTON	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	30	1	Open: JANUARY Close: DECEMBER	
33	0155100	BARRINGTON VLG ENRICHMENT CTR	45 COMMERCE WAY		BARRINGTON	ACTIVE	01-2013	NON-TRANSIENT NON-COMMUI	DAY CARE	58	1	Open: JANUARY Close: DECEMBER	
34	0156020	COMPUTER RESOURCES	1037 CALEF HWY		BARRINGTON	ACTIVE		NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	27	1	Open: JANUARY Close: DECEMBER	
35	0156030	ASSOCIATED BUYERS	50 COMMERCE WAY		BARRINGTON	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	84	1	Open: JANUARY Close: DECEMBER	
36	0156040	TURBOCAM/PHASE I	607 CALEF HWY		BARRINGTON	ACTIVE		NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	375	1	Open: JANUARY Close: DECEMBER	
37	0156060	MIDPOINT PROPERTIES	219 OLD CONCORD TPKE		BARRINGTON	ACTIVE	07-2014	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	25	2	Open: JANUARY Close: DECEMBER	
38	0156070	TURBOCAM B2	38 REDEMPTION RD		BARRINGTON	ACTIVE	07-2014	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	100	1	Open: JANUARY Close: DECEMBER	
39	0167010	MT ATTITASH SKI AREA/LODGE	775 RTE 302		BARTLETT	ACTIVE		NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	1000	1	Open: JANUARY Close: DECEMBER	
40	0168460	BEAR PEAK AT ATTITASH	GRAND SUMMITT RD	OFF RTE 302	BARTLETT	ACTIVE	12-1995	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	800	2	Open: JANUARY Close: DECEMBER	
41	0195010	MONTESSORI SCH OF BEDFORD	24 TIRRELL HILL RD		BEDFORD	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	40	1	Open: SEPTEMBER Close: JUNE	
42	0195090	BETHANY COVENANT CHURCH	1 COVENANT WAY		BEDFORD	ACTIVE	06-1991	NON-TRANSIENT NON-COMMUI	DAY CARE	79	1	Open: JANUARY Close: DECEMBER	
43	0195100	BEDFORD VILLAGE MORNING SCH	19 MINISTERIAL RD		BEDFORD	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	60	2	Open: SEPTEMBER Close: JUNE	
44	0196020	101 PLAZA	209 RTE 101		BEDFORD	ACTIVE	01-1972	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	75	5	Open: JANUARY Close: DECEMBER	
45	0196170	PINE TREE PLACE CONDOS	360 RTE 101W		BEDFORD	ACTIVE	04-1987	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	35	14	Open: JANUARY Close: DECEMBER	
46	0196190	IRONSIDES	18 CONSTITUTION DR		BEDFORD	ACTIVE	01-1987	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	50	1	Open: JANUARY Close: DECEMBER	
47	0196210	288 ROUTE 101	288 RTE 101		BEDFORD	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	25	1	Open: JANUARY Close: DECEMBER	
48	0196270	EVERSOURCE ENERGY BEDFORD AW	12 BELLEMORE DR		BEDFORD	ACTIVE	04-2001	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	30	1	Open: JANUARY Close: DECEMBER	
49	0196280	292 ROUTE 101	292 RTE 101		BEDFORD	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	25	1	Open: JANUARY Close: DECEMBER	
50	0196310	FRENCH ATWOOD MARKETPLACE	256 WALLACE RD		BEDFORD	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	33	4	Open: JANUARY Close: DECEMBER	
51	0196320	FAMILY MEDICAL CENTER	188 RTE 101		BEDFORD	ACTIVE	12-2011	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	35	2	Open: JANUARY Close: DECEMBER	
52	0196330	124 BEDFORD CENTER RD	124 BEDFORD CTR RD		BEDFORD	ACTIVE	04-2012	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	50	2	Open: JANUARY Close: DECEMBER	
53	0196340	BEDFORD HILLS	1 COOPER LN		BEDFORD	ACTIVE	01-2014	NON-TRANSIENT NON-COMMUI	PRIVATELY OWNED REDISTRIBUTION SYSTEI	200	2	Open: JANUARY Close: DECEMBER	
54	0205010	LAKES REGION DAYCARE	24 EASTGATE PARK DR		BELMONT	ACTIVE	04-2015	NON-TRANSIENT NON-COMMUI	DAY CARE	67	1	Open: JANUARY Close: DECEMBER	
55	0206020	BELMONT BUSINESS PARK	RTE 106		BELMONT	ACTIVE	05-1985	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	80	3	Open: JANUARY Close: DECEMBER	
56	0206040	AFL TELECOMMUNICATIONS LLC	16 EASTGATE PARK RD	RTE 106	BELMONT	ACTIVE	01-1975	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	35	1	Open: JANUARY Close: DECEMBER	
57	0206050	EASTGATE PARK	3 EASTGATE PARK RD	RTE 106	BELMONT	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	40	1	Open: JANUARY Close: DECEMBER	
58	0209040	BELMONT MEDICAL CENTER	8 CORPORATE DR		BELMONT	ACTIVE	06-2007	NON-TRANSIENT NON-COMMUI	HOSPITAL, MEDICAL FACILITY	28	1	Open: JANUARY Close: DECEMBER	
59	0245010	PROFILE HIGH SCH	691 PROFILE RD	JCT I93/302/18/11(	BETHLEHEM	ACTIVE	01-1976	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	284	1	Open: JANUARY Close: DECEMBER	
60	0265010	BOW MEMORIAL SCHOOL	20 BOW CENTER RD		BOW	ACTIVE	09-1964	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	615	1	Open: SEPTEMBER Close: JUNE	
61	0265020	BOW ELEMENTARY SCHOOL	22 BOW CENTER RD		BOW	ACTIVE	09-1979	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	522	1	Open: SEPTEMBER Close: JUNE	
62	0265030	BOW HIGH SCHOOL	32 WHITE ROCK HILL RD	THREE POND RD	BOW	ACTIVE	09-1997	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	645	1	Open: SEPTEMBER Close: JUNE	
63	0265040	JOYFUL NOISE PRESCH	6 BRANCH LONDONDERRY TPKE EAST		BOW	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	72	1	Open: JANUARY Close: DECEMBER	
64	0265050	BOW YOUTH CENTER	21 BOW CTR RD		BOW	ACTIVE	08-2008	NON-TRANSIENT NON-COMMUI	DAY CARE	150	1	Open: JANUARY Close: DECEMBER	
65	0265060	MEETING HOUSE MONTESSORI	28 LOGGING HILL RD		BOW	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	58	1	Open: JANUARY Close: DECEMBER	
66	0266010	GSP/MERRIMACK STATION	431 RIVER RD		BOW	ACTIVE		NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	75	1	Open: JANUARY Close: DECEMBER	
67	0266020	BOVIE SCREEN PROCESS PRINTING	4 NORTHEAST AVE		BOW	ACTIVE	01-1962	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	32	1	Open: JANUARY Close: DECEMBER	

	A	B	C	D	E	F	G	H	I	J	K	L	M
68	0266050	GRAPPONE FORD COMPLEX	516 RTE 3A		BOW	ACTIVE	06-1971	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	100	4	Open: JANUARY Close: DECEMBER	
69	0266090	PITCO FRIALATOR	552/553 RTE 3A	JCT I89	BOW	ACTIVE	01-1961	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	326	2	Open: JANUARY Close: DECEMBER	
70	0266110	BOW TECHNOLOGIES CENTER	3 ROBINSON RD		BOW	ACTIVE	07-1986	NON-TRANSIENT NON-COMMUI	DAY CARE	34	2	Open: JANUARY Close: DECEMBER	
71	0266130	RUGGLES III OFFICE BLDG	553 RTE 3A	783 RTE 3A	BOW	ACTIVE	07-1989	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	35	2	Open: JANUARY Close: DECEMBER	
72	0266140	NH AUTO DEALERS ASSN	507 SOUTH ST		BOW	ACTIVE	06-1986	NON-TRANSIENT NON-COMMUI	DAY CARE	104	1	Open: JANUARY Close: DECEMBER	
73	0266150	CONCORD GROUP	504 SOUTH ST		BOW	ACTIVE	01-1989	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	64	4	Open: JANUARY Close: DECEMBER	
74	0266200	GRAPPONE TOYOTA	594 RTE 3A		BOW	ACTIVE	01-1972	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	119	1	Open: JANUARY Close: DECEMBER	
75	0266220	501 SOUTH STREET	501 SOUTH ST		BOW	ACTIVE	11-1989	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	25	1	Open: JANUARY Close: DECEMBER	
76	0266240	GRAPPONE HONDA	519 RTE 3A		BOW	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	100	1	Open: JANUARY Close: DECEMBER	
77	0266250	BOW SAFETY BLDG	7 KNOX RD		BOW	ACTIVE	03-2019	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	40	1	Open: JANUARY Close: DECEMBER	
78	0269010	BOW MUNICIPAL BUILDING	10 GRANDVIEW RD		BOW	ACTIVE	01-1927	NON-TRANSIENT NON-COMMUI	DAY CARE	70	1	Open: JANUARY Close: DECEMBER	
79	0269040	JOYFUL NOISE LEARNING CENTER	8 BRANCH LONDONDERRY TPKE EAST		BOW	ACTIVE	07-2018	NON-TRANSIENT NON-COMMUI	DAY CARE	25	1	Open: JANUARY Close: DECEMBER	
80	0275070	KEARSARGE REG ELEM SCH/BRADFR	163 OLD WARNER RD	OFF RTE 103	BRADFORD	ACTIVE	09-1987	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	231	1	Open: SEPTEMBER Close: JUNE	
81	0275080	NFI NORTH TRNS HSING	2552 RTE 103		BRADFORD	ACTIVE	06-2009	NON-TRANSIENT NON-COMMUI	INSTITUTION, REHAB FACILITIES	50	5	Open: JANUARY Close: DECEMBER	
82	0275090	CHILDRENS CTR FOR CREATVE LRNG	57 W MAIN ST		BRADFORD	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	40	1	Open: JANUARY Close: DECEMBER	
83	0285010	SWASEY CENTRAL SCH	355 MIDDLE RD	RTE 111A	BRENTWOOD	ACTIVE	01-1949	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	392	1	Open: SEPTEMBER Close: JUNE	
84	0285040	A PLACE TO GROW	436 RTE 125		BRENTWOOD	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	45	1	Open: JANUARY Close: DECEMBER	
85	0286060	ROCKINGHAM COUNTY COURTHOUSE	10 RTE 125		BRENTWOOD	ACTIVE	01-1998	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	116	1	Open: JANUARY Close: DECEMBER	
86	0295010	BRIDGEWATER/HEBRON VIL SCH	25 SCHOOLHOUSE RD	RTE 3A	BRIDGEWATEI	ACTIVE	07-1999	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	200	1	Open: JANUARY Close: DECEMBER	
87	0305010	NEWFOUND REGIONAL HS	150 NEWFOUND RD	RTE 104	BRISTOL	ACTIVE	01-1988	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	500	1	Open: JANUARY Close: DECEMBER	
88	0305020	PB AND JS FAMILY CHILDCARE	1420 PEAKED HILL RD		BRISTOL	ACTIVE	08-2017	NON-TRANSIENT NON-COMMUI	DAY CARE	40	2	Open: JANUARY Close: DECEMBER	
89	0325010	RICHARD MAGHAKIAN MEMORIAL SCI	22 MILFORD ST		BROOKLINE	ACTIVE	04-1987	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	327	1	Open: SEPTEMBER Close: JUNE	
90	0325050	CAPT SAMUEL DOUGLASS ACADEMY	24 TOWNSEND HILL RD	RTE 13	BROOKLINE	ACTIVE	09-2000	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	358	1	Open: SEPTEMBER Close: JUNE	
91	0325060	STONEY LEDGE PLAZA	181 RTE 13		BROOKLINE	ACTIVE	12-2019	NON-TRANSIENT NON-COMMUI	DAY CARE	70	2	Open: JANUARY Close: DECEMBER	
92	0326020	DECCO	31 SOUTH ST	31 RTE 13	BROOKLINE	ACTIVE	12-1998	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	50	1	Open: JANUARY Close: DECEMBER	
93	0346030	USFS WMNF ADMINISTRATIVE CMPLX	71 WHITE MOUNTAIN DR	DG EXT/LF OF R	CAMPTON	ACTIVE	10-2009	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	110	3	Open: JANUARY Close: DECEMBER	
94	0355060	INDIAN RIVER SCH	45 ROYAL RD	RTE 4	CANAAN	ACTIVE	09-1987	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	799	2	Open: JANUARY Close: DECEMBER	
95	0355070	MASCOMA COMMUNITY HEALTH CARI	18 ROBERTS RD		CANAAN	ACTIVE	04-2017	NON-TRANSIENT NON-COMMUI	HOSPITAL, MEDICAL FACILITY	40	1	Open: JANUARY Close: DECEMBER	
96	0356020	BARKER STEEL	450 RTE 4		CANAAN	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	30	1	Open: JANUARY Close: DECEMBER	
97	0365010	HENRY W MOORE SCH	12 DEERFIELD RD	RTE 43	CANDIA	ACTIVE	01-1938	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	352	1	Open: SEPTEMBER Close: JUNE	
98	0365020	M AND C CHILDRENS LEARNING PL	205 MAIN ST	RTE 43 AND RTE	CANDIA	ACTIVE	08-1984	NON-TRANSIENT NON-COMMUI	DAY CARE	75	1	Open: JANUARY Close: DECEMBER	
99	0365040	REMINGTON EDUCATION CENTER	15 STEVENS LN		CANDIA	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	62	2	Open: SEPTEMBER Close: JUNE	
100	0366040	BIRCHWOOD PLAZA	45 RAYMOND RD		CANDIA	ACTIVE	01-1988	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	80	10	Open: JANUARY Close: DECEMBER	
101	0375010	CANTERBURY ELEMENTARY SCH	15 BAPTIST RD		CANTERBURY	ACTIVE	01-1987	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	156	1	Open: JANUARY Close: DECEMBER	
102	0396020	SENTERS MARKET CONDOS	SENTERS MARKETPLACE	ROUTE 25B	CENTER HARE	ACTIVE	01-1989	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	35	4	Open: JANUARY Close: DECEMBER	
103	0419010	LIFE FLLWSHP FOURSQUARE CHURC	85 WHEELER RAND RD		CHARLESTOW	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	70	2	Open: JANUARY Close: DECEMBER	
104	0435060	CHESTER ACADEMY	22 MURPHY DR	RTE 102	CHESTER	ACTIVE	09-1999	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	645	1	Open: SEPTEMBER Close: JUNE	
105	0435070	PLAY LAUGH N GROW	184 RAYMOND RD		CHESTER	ACTIVE	01-2015	NON-TRANSIENT NON-COMMUI	DAY CARE	40	1	Open: JANUARY Close: DECEMBER	
106	0435080	CHESTERBROOK SCH OF NTRL LRNG	232 FREMONT RD		CHESTER	ACTIVE	09-2018	NON-TRANSIENT NON-COMMUI	DAY CARE	28	2	Open: JANUARY Close: DECEMBER	
107	0437020	WASON POND COMMUNITY CENTER	RTE 102	603 RAYMOND RI	CHESTER	ACTIVE		NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	25	1	Open: JANUARY Close: DECEMBER	
108	0445010	CHESTERFIELD CENTRAL SCH	535 OLD CHESTERFIELD RD	RTE 63	CHESTERFIEL	ACTIVE	01-1950	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	322	1	Open: SEPTEMBER Close: JUNE	
109	0446020	UNITED NATURAL FOODS	71 STOW DR		CHESTERFIEL	ACTIVE	09-1988	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	600	4	Open: JANUARY Close: DECEMBER	
110	0449040	FEDEX FACILITY	40 COACHMAN RD		CHESTERFIEL	ACTIVE	09-2007	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	29	1	Open: JANUARY Close: DECEMBER	
111	0455010	CHICHESTER CENTRAL SCH	219 MAIN ST		CHICHESTER	ACTIVE	01-1949	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	261	1	Open: SEPTEMBER Close: JUNE	
112	0455030	KELLEY CORNER SCHOOL	67 KELLEYS CORNER RD		CHICHESTER	ACTIVE	11-2019	NON-TRANSIENT NON-COMMUI	DAY CARE	28	1	Open: JANUARY Close: DECEMBER	
113	0456010	CAMPING WORLD OF NEW HAMPSHIR	165 DOVER RD		CHICHESTER	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	50	1	Open: JANUARY Close: DECEMBER	
114	0505010	SHAKER RD SCH & CHILDCARE CTR	95 SHAKER RD		CONCORD	ACTIVE	01-1979	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	355	6	Open: JANUARY Close: DECEMBER	
115	0515020	NEW PINE TREE SCH	183 MILL ST	RTE 302E	CONWAY	ACTIVE	09-1990	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	328	1	Open: SEPTEMBER Close: JUNE	
116	0515040	LITTLE HANDS BIG DREAMS	90 ODELL HILL RD	CENTER CONWA	CONWAY	ACTIVE	09-2015	NON-TRANSIENT NON-COMMUI	DAY CARE	42	1	Open: JANUARY Close: DECEMBER	
117	0516020	NORTHERN HUMAN SVCS	626 EASTMAN RD	RTE 302	CONWAY	ACTIVE	01-1980	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	50	1	Open: JANUARY Close: DECEMBER	
118	0516030	CONWAY MUNICIPAL CENTER	EAST CONWAY RD	RTE 302E	CONWAY	ACTIVE	10-1989	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	36	2	Open: JANUARY Close: DECEMBER	
119	0516040	LUPINE	E CONWAY RD		CONWAY	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	58	1	Open: JANUARY Close: DECEMBER	
120	0519020	CONWAY TOWN HALL	MAIN ST	RTE 113	CONWAY	ACTIVE	01-1940	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	45	8	Open: JANUARY Close: DECEMBER	
121	0525010	CORNISH ELEMENTARY SCH	274 TOWNHOUSE RD	OFF RTE 120	CORNISH	ACTIVE	01-1950	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	140	2	Open: SEPTEMBER Close: JUNE	
122	0545010	CROYDON VILLAGE SCH	889 RTE 10		CROYDON	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	33	4	Open: SEPTEMBER Close: JUNE	
123	0575010	DANBURY ELEMENTARY SCH	20 DAFFODIL LN	RTE 104E	DANBURY	ACTIVE	09-1979	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	85	1	Open: JANUARY Close: DECEMBER	
124	0585010	DANVILLE ELEMENTARY	23 SCHOOL ST	MAIN ST/RTE 111	DANVILLE	ACTIVE	01-1962	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	340	1	Open: SEPTEMBER Close: JUNE	
125	0595010	GEORGE B WHITE BLDG	8 RAYMOND RD		DEERFIELD	ACTIVE		NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	118	1	Open: JANUARY Close: DECEMBER	
126	0595020	DEERFIELD COMMUNITY SCH	66 NORTH RD	RTE 107	DEERFIELD	ACTIVE	08-1988	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	630	1	Open: SEPTEMBER Close: JUNE	
127	0595040	LONGVIEW SCH	55 RESERVATION RD		DEERFIELD	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	31	1	Open: JANUARY Close: DECEMBER	
128	0605010	ROBIN HILL FARM/BLUE HOUSE	55 DONOVAN RD		DEERING	ACTIVE	01-1983	NON-TRANSIENT NON-COMMUI	HOSPITAL, MEDICAL FACILITY	25	1	Open: JANUARY Close: DECEMBER	
129	0607030	HIS MANSION DINING HALL	395 WOLF HILL RD	RTE 149	DEERING	ACTIVE	01-1953	NON-TRANSIENT NON-COMMUI	INSTITUTION, REHAB FACILITIES	105	13	Open: JANUARY Close: DECEMBER	
130	0609010	HIS MANSION NEW BEGINNINGS	395 WOLF HILL RD	RTE 149	DEERING	ACTIVE	01-1984	NON-TRANSIENT NON-COMMUI	INSTITUTION, REHAB FACILITIES	62	3	Open: JANUARY Close: DECEMBER	
131	0615060	E DERRY MEMORIAL ELEM SCH	20 DUBEAU DR	OLESEN RD	DERRY	ACTIVE	07-1986	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	488	1	Open: SEPTEMBER Close: JUNE	
132	0615070	OVER THE RAINBOW PRESCH	223 ROCKINGHAM RD	RTE 28S	DERRY	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	78	1	Open: SEPTEMBER Close: JUNE	
133	0616040	BROOKSTONE PARK/EAST	16 RTE 111		DERRY	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	143	2	Open: JANUARY Close: DECEMBER	
134	0618150	COWBELL CORNERS	418 ISLAND POND RD		DERRY	ACTIVE	07-2016	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	27	3	Open: JANUARY Close: DECEMBER	
135	0619030	BROOKSTONE PARK/WEST	12 RTE 111		DERRY	ACTIVE		TRANSIENT NON-COMMUNITY	MEDICAL OFFICES (DOCTOR/DENTIST)	35	1	Open: JANUARY Close: DECEMBER	
136	0655030	MISS PATTYS CHILDCARE	49 PISCATAQUA RD		DOVER	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	36	1	Open: JANUARY Close: DECEMBER	

	A	B	C	D	E	F	G	H	I	J	K	L	M
137	0664010	DUBLIN CHRISTIAN ACADEMY	106 PAGE RD		DUBLIN	ACTIVE	01-1964	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	155	7	Open: JANUARY Close: DECEMBER	
138	0664020	DUBLIN SCHOOL	18 LEHMANN WAY	DUBLIN RD	DUBLIN	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	215	21	Open: JANUARY Close: DECEMBER	
139	0665010	DUBLIN CONSOLIDATED SCH	1177 MAIN ST	RTE 101	DUBLIN	ACTIVE	01-1938	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	80	1	Open: SEPTEMBER Close: JUNE	
140	0665030	PHOENIX HOUSE DUBLIN CENTER	3 PIERCE RD		DUBLIN	ACTIVE	01-1967	NON-TRANSIENT NON-COMMUI	INSTITUTION, REHAB FACILITIES	65	5	Open: JANUARY Close: DECEMBER	
141	0665050	MOUNTAIN SHADOWS SCH	149 VALLEY RD	.DE PETERBROU	DUBLIN	ACTIVE	01-1971	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	72	2	Open: SEPTEMBER Close: JUNE	
142	0666010	YANKEE PUBLISHING	1121 MAIN ST		DUBLIN	ACTIVE	01-1955	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	60	2	Open: JANUARY Close: DECEMBER	
143	0666020	HIGH STANDARD INC	MAIN ST		DUBLIN	INACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	0	0	Open: Close:	
144	0666030	DUBLIN VILLAGE PARK	1281 / 1283 MAIN ST		DUBLIN	ACTIVE	07-2007	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	44	2	Open: JANUARY Close: DECEMBER	
145	0685010	DUNBARTON ELEMENTARY SCH	20 ROBERT ROGERS RD		DUNBARTON	ACTIVE	01-1972	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	245	1	Open: SEPTEMBER Close: JUNE	
146	0705020	E KINGSTON ELEMENTARY SCH	5 ANDREWS LN	SOUTH RD	EAST KINGSTO	ACTIVE	09-1971	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	197	1	Open: JANUARY Close: DECEMBER	
147	0708040	POWWOW JUNCTION	14 POWWOW RIVER RD		EAST KINGSTO	ACTIVE	07-2013	NON-TRANSIENT NON-COMMUI	DAY CARE	63	2	Open: JANUARY Close: DECEMBER	
148	0732020	GREEN MOUNTAIN TREATMENT CTR	244 HIGHWATCH RD		EFFINGHAM	ACTIVE	01-1985	NON-TRANSIENT NON-COMMUI	INSTITUTION, REHAB FACILITIES	150	14	Open: JANUARY Close: DECEMBER	
149	0735030	EFFINGHAM ELEMENTARY SCH	6 PARTRIDGE COVE RD		EFFINGHAM	ACTIVE	09-2003	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	128	1	Open: SEPTEMBER Close: JUNE	
150	0775020	EPSOM MEDICAL CENTER	1990 DOVER RD		EPSOM	ACTIVE	06-2009	NON-TRANSIENT NON-COMMUI	HOSPITAL, MEDICAL FACILITY	25	1	Open: JANUARY Close: DECEMBER	
151	0805010	BUILDING BLOCK COMMONS	125 KINGSTON RD	RTE 111	EXETER	ACTIVE	10-1979	NON-TRANSIENT NON-COMMUI	DAY CARE	40	1	Open: SEPTEMBER Close: JUNE	
152	0805040	EXETER HIGH SCH	1 BLUE HAWK DR		EXETER	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	1890	1	Open: JANUARY Close: DECEMBER	
153	0825010	GEORGE S EMERSON ELEM SCH	27 RHODODENDRON RD	RTE 119W	FITZWILLIAM	ACTIVE	01-1950	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	182	1	Open: SEPTEMBER Close: JUNE	
154	0835010	FRANCESTOWN ELEMENTARY SCH	325 SECOND NH TPKE SOUTH		FRANCESTOW	ACTIVE	09-1997	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	69	1	Open: SEPTEMBER Close: JUNE	
155	0875010	ELLIS SCHOOL	432 MAIN ST		FREMONT	ACTIVE	01-1947	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	460	1	Open: SEPTEMBER Close: JUNE	
156	0875030	COUNTRY CLUB FOR KIDS	50 MAIN ST	RTE 107	FREMONT	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	61	2	Open: JANUARY Close: DECEMBER	
157	0875040	BARNYARD BUDDIES	83 CHESTER RD		FREMONT	ACTIVE	09-2002	NON-TRANSIENT NON-COMMUI	DAY CARE	40	1	Open: JANUARY Close: DECEMBER	
158	0876030	COOPERS CORNER/EAST	25 SPAULDING RD		FREMONT	ACTIVE	05-2018	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	40	1	Open: JANUARY Close: DECEMBER	
159	0885010	GILFORD ELEMENTARY SCH	76 BELKNAP MOUNTAIN RD	RTE 11A	GILFORD	ACTIVE	09-1972	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	422	1	Open: SEPTEMBER Close: JUNE	
160	0885020	GILFORD MIDDLE AND HIGH SCH	72/88 ALVAH WILSON RD	RTE 11A	GILFORD	ACTIVE	06-1974	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	958	1	Open: JANUARY Close: DECEMBER	
161	0885050	GILFORD PROFESSIONAL PARK	401 GILFORD AVE		GILFORD	ACTIVE	07-2018	NON-TRANSIENT NON-COMMUI	DAY CARE	135	3	Open: JANUARY Close: DECEMBER	
162	0886040	UNDER HIS WINGS PRESCHOOL	2 AIRPORT RD		GILFORD	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	110	1	Open: JANUARY Close: DECEMBER	
163	0886120	VILLAGE WEST I	25 COUNTRY CLUB RD	RTE 11A	GILFORD	ACTIVE	01-1983	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	113	45	Open: JANUARY Close: DECEMBER	
164	0886150	GILFORD MUNICIPAL BLDG COMPLEX	55 CHERRY VALLEY RD		GILFORD	ACTIVE	06-1988	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	90	3	Open: JANUARY Close: DECEMBER	
165	0886170	VILLAGE WEST II	36 COUNTRY CLUB RD	RTE 11A	GILFORD	ACTIVE	11-1990	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	30	12	Open: JANUARY Close: DECEMBER	
166	0886190	NHDOS MARINE PATROL BLDG	31 DOCK RD		GILFORD	ACTIVE	11-2016	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	25	2	Open: JANUARY Close: DECEMBER	
167	0887050	GUNSTOCK AREA	719 CHERRY VALLEY RD		GILFORD	ACTIVE		NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	152	13	Open: JANUARY Close: DECEMBER	
168	0895010	GILMANTON ELEMENTARY SCH	1386 RTE 140		GILMANTON	ACTIVE	01-1965	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	494	1	Open: JANUARY Close: DECEMBER	
169	0905010	GILSUM ELEMENTARY SCH	640 RTE 10		GILSUM	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	62	1	Open: SEPTEMBER Close: JUNE	
170	0906010	WS BADGER CO	768 RTE 10		GILSUM	ACTIVE	11-2011	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	80	1	Open: JANUARY Close: DECEMBER	
171	0915020	LEARN AS WE PLAY CHLD CARE CTR	276 GOFFSTOWN BACK RD		GOFFSTOWN	ACTIVE	05-1997	NON-TRANSIENT NON-COMMUI	DAY CARE	94	1	Open: JANUARY Close: DECEMBER	
172	0926010	ANDROSCOGGIN RANGER STATION	300 GLEN RD	RTE 16	GORHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	30	1	Open: JANUARY Close: DECEMBER	
173	0955010	GRANTHAM VILLAGE SCH	75 LEARNING DR	10/DUNBAR HILI	GRANTHAM	ACTIVE	01-1978	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	303	1	Open: JANUARY Close: DECEMBER	
174	0956040	GRANTHAM GREENWAY	151 RTE 10 N		GRANTHAM	ACTIVE	07-2016	NON-TRANSIENT NON-COMMUI	DAY CARE	200	2	Open: JANUARY Close: DECEMBER	
175	0975020	GREENFIELD ELEMENTARY SCH	860 FOREST RD		GREENFIELD	ACTIVE	12-1999	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	109	1	Open: AUGUST Close: JUNE	
176	1034010	HAMPSTEAD HOSPITAL	218 EAST RD		HAMPSTEAD	ACTIVE		NON-TRANSIENT NON-COMMUI	INSTITUTION, REHAB FACILITIES	200	1	Open: JANUARY Close: DECEMBER	
177	1035020	AGES AND STAGES HAMPSTEAD	499 MAIN ST		HAMPSTEAD	ACTIVE	08-1984	NON-TRANSIENT NON-COMMUI	DAY CARE	99	1	Open: JANUARY Close: DECEMBER	
178	1035040	HAMPSTEAD MIDDLE SCH	28 SCHOOL ST		HAMPSTEAD	ACTIVE	01-1978	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	552	1	Open: SEPTEMBER Close: JUNE	
179	1035050	HAMPSTEAD ACADEMY	320 EAST RD	RTE 111	HAMPSTEAD	ACTIVE	08-1987	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	92	3	Open: SEPTEMBER Close: JUNE	
180	1035110	HAZEL DRIVE KIDS	35 HAZEL DR		HAMPSTEAD	ACTIVE	11-2011	NON-TRANSIENT NON-COMMUI	DAY CARE	34	1	Open: JANUARY Close: DECEMBER	
181	1036010	HAMPSTEAD SHOPPERS VILLAGE	45 DANVILLE RD	RTE 111E	HAMPSTEAD	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	50	3	Open: JANUARY Close: DECEMBER	
182	1036040	STAGE ROAD JUNCTION	213 STAGE RD	JCT RTE 111/121	HAMPSTEAD	ACTIVE	09-1988	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	55	3	Open: JANUARY Close: DECEMBER	
183	1036080	RAM PRINTING	5 COMMERCE PARK DR	RTE 111	HAMPSTEAD	ACTIVE	10-1984	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	42	1	Open: JANUARY Close: DECEMBER	
184	1036090	THUNDERLINE Z	11 HAZEL DR		HAMPSTEAD	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	25	1	Open: JANUARY Close: DECEMBER	
185	1045010	LINCOLN AKERMAN SCH	8 EXETER RD	RTE 88	HAMPTON FAL	ACTIVE	04-1974	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	281	1	Open: SEPTEMBER Close: JUNE	
186	1045040	HERONFIELD ACADEMY	356 EXETER RD		HAMPTON FAL	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	114	2	Open: JANUARY Close: DECEMBER	
187	1046050	CRN REALTY	105 LAFAYETTE RD		HAMPTON FAL	ACTIVE	12-1986	NON-TRANSIENT NON-COMMUI	DAY CARE	190	7	Open: JANUARY Close: DECEMBER	
188	1075010	HAMPSHIRE COOP NURSERY SCH	104 LYME RD	RTE 10	HANOVER	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	32	2	Open: JANUARY Close: DECEMBER	
189	1075020	MONTESSORI CHILDRENS SCH	67 TRESCOTT RD		HANOVER	ACTIVE	03-1999	NON-TRANSIENT NON-COMMUI	DAY CARE	51	1	Open: JANUARY Close: DECEMBER	
190	1085010	WELLS MEMORIAL SCH	235 CHESHAM RD	CHESHAM	HARRISVILLE	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	60	1	Open: SEPTEMBER Close: JUNE	
191	1085020	CHESHIRE MILLS BOARDING HOUSE	66 MAIN ST		HARRISVILLE	ACTIVE	01-1953	NON-TRANSIENT NON-COMMUI	DAY CARE	44	1	Open: JANUARY Close: DECEMBER	
192	1086010	HISTORIC HARRISVILLE	CHESHIRE MILLS COMPLEX	MAIN ST	HARRISVILLE	ACTIVE		NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	30	3	Open: JANUARY Close: DECEMBER	
193	1105010	OLIVERIAN EAST CAMPUS	2634 MT MOOSILAUKE HWY	RTE 25/PIKE	HAVERHILL	ACTIVE	07-2011	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	30	5	Open: JANUARY Close: DECEMBER	
194	1105020	BECKET HOUSE AT HALL FARM	1977 MT MOOSILAUKE HWY		HAVERHILL	ACTIVE		NON-TRANSIENT NON-COMMUI	INSTITUTION, REHAB FACILITIES	46	5	Open: JANUARY Close: DECEMBER	
195	1126030	HHP	14 BUXTON INDUSTRIAL DR		HENNIKER	ACTIVE		NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	70	3	Open: JANUARY Close: DECEMBER	
196	1127030	PATS PEAK SKI AREA	686 FLANDERS RD		HENNIKER	ACTIVE	01-1962	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	47	19	Open: JANUARY Close: DECEMBER	
197	1145010	ROSEWALD FARM	213 CENTER RD		HILLSBOROUG	ACTIVE		NON-TRANSIENT NON-COMMUI	INSTITUTION, REHAB FACILITIES	100	6	Open: JANUARY Close: DECEMBER	
198	1145020	HILLSBORO BAPTIST CHURCH	337 2ND NH TPKE		HILLSBOROUG	ACTIVE	08-2019	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	28	5	Open: JANUARY Close: DECEMBER	
199	1156010	GEORGES FIELD WATER SYSTEM	18 GEORGES FIELD RD		HINSDALE	ACTIVE	02-1993	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	99	6	Open: JANUARY Close: DECEMBER	
200	1165010	WHITE MOUNTAIN MONTESSORI SCH	133 MT PROSPECT RD		HOLDERNESS	ACTIVE	05-2015	NON-TRANSIENT NON-COMMUI	DAY CARE	30	1	Open: JANUARY Close: DECEMBER	
201	1165020	HOLDERNESS CENTRAL SCH	3 SCHOOL ST	RTE 175	HOLDERNESS	ACTIVE	01-1953	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	185	1	Open: SEPTEMBER Close: JUNE	
202	1167190	SLNSC BLUE HERON SCH	25 SCIENCE CENTER DR	RTE 113	HOLDERNESS	ACTIVE	05-2010	NON-TRANSIENT NON-COMMUI	DAY CARE	41	2	Open: JANUARY Close: DECEMBER	
203	1175030	HOLLIS SCH DIST	39 ROCKY POND RD		HOLLIS	ACTIVE	01-1970	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	2000	11	Open: JANUARY Close: DECEMBER	
204	1175050	HOLLIS BROOKLINE HS	24 CAVALIER CT		HOLLIS	ACTIVE	09-1997	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	945	1	Open: JANUARY Close: DECEMBER	
205	1175060	HOLLIS MONTESSORI SCHOOL	9 S MERRIMACK RD		HOLLIS	ACTIVE	04-2013	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	145	3	Open: SEPTEMBER Close: JUNE	

	A	B	C	D	E	F	G	H	I	J	K	L	M
206	1176020	26 CLINTON DRIVE BLDG	26 CLINTON DR		HOLLIS	ACTIVE	07-1987	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	150	11	Open: JANUARY Close: DECEMBER	
207	1176060	DIAMOND CASTING AND MACHINE	95 PROCTOR HILL RD	RTE 130	HOLLIS	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	60	1	Open: JANUARY Close: DECEMBER	
208	1176070	ULTRASOURCE	22 CLINTON DR		HOLLIS	ACTIVE	06-1984	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	68	1	Open: JANUARY Close: DECEMBER	
209	1176080	PURITAN PRESS	95 RUNNELLS BRIDGE RD		HOLLIS	ACTIVE		NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	80	1	Open: JANUARY Close: DECEMBER	
210	1176090	FARM DESIGN	27 PROCTOR HILL RD		HOLLIS	ACTIVE	04-2015	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	45	1	Open: JANUARY Close: DECEMBER	
211	1176100	18 CLINTON DR BLDG	18 CLINTON DR		HOLLIS	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	75	7	Open: JANUARY Close: DECEMBER	
212	1185010	PLACES YOULL GROW	167 LONDONDERRY TPKE RD		HOOKSETT	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	30	2	Open: JANUARY Close: DECEMBER	
213	1186010	198 LONDONDERRY TURNPIKE	198 LONDONDERRY TPKE		HOOKSETT	ACTIVE	06-1986	NON-TRANSIENT NON-COMMUI	DAY CARE	98	1	Open: JANUARY Close: DECEMBER	
214	1186040	POULTRY PRODUCTS	11 BEMIS SAVOIE RD	OFF 3A	HOOKSETT	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	40	1	Open: JANUARY Close: DECEMBER	
215	1189030	NH DOT BUREAU TURNPIKE OFFICES	36 HACKETT HILL RD		HOOKSETT	ACTIVE		NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	105	4	Open: JANUARY Close: DECEMBER	
216	1195040	BEECH HILL SCHOOL	20 BEECH HILL RD		HOPKINTON	ACTIVE	06-1999	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	40	1	Open: JANUARY Close: DECEMBER	
217	1196030	MCLANE NORTHEAST	932 MAPLE ST	CONTOOCOOK	HOPKINTON	ACTIVE	01-1975	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	300	1	Open: JANUARY Close: DECEMBER	
218	1196050	YANKEE BOOK PEDDLER	999 MAPLE ST	EXIT 6 RTE 89	HOPKINTON	ACTIVE		NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	240	1	Open: JANUARY Close: DECEMBER	
219	1205060	KIDDIE KONNECTION	301 DERRY RD	RTE 102	HUDSON	ACTIVE	04-1995	NON-TRANSIENT NON-COMMUI	DAY CARE	191	1	Open: JANUARY Close: DECEMBER	
220	1205070	EARLY START LEARNING ACADEMY	141 KIMBALL HILL RD		HUDSON	ACTIVE	08-1999	NON-TRANSIENT NON-COMMUI	DAY CARE	79	1	Open: JANUARY Close: DECEMBER	
221	1212110	EAGLE MOUNTAIN HOUSE	179 CARTER NOTCH RD	RTE 16B	JACKSON	ACTIVE	01-1975	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	125	2	Open: JANUARY Close: DECEMBER	
222	1245010	MONADNOCK WALDORF SCH	424 OLD WALPOLE RD		KEENE	ACTIVE	02-2003	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	48	1	Open: JANUARY Close: DECEMBER	
223	1255010	KENSINGTON ELEMENTARY SCH	122 AMESBURY RD	RTE 150	KENSINGTON	ACTIVE	01-1952	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	147	1	Open: SEPTEMBER Close: JUNE	
224	1256010	UNITIL ENERGY SYS	114 DRINKWATER RD		KENSINGTON	ACTIVE	01-1954	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	40	1	Open: JANUARY Close: DECEMBER	
225	1275010	DANIEL J BAKIE SCH	179 MAIN ST		KINGSTON	ACTIVE	01-1962	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	440	1	Open: SEPTEMBER Close: JUNE	
226	1275060	SANBORN REGIONAL HIGH SCH	17 DANVILLE RD		KINGSTON	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	950	1	Open: JANUARY Close: DECEMBER	
227	1276020	266 ROUTE 125	266 RTE 125N		KINGSTON	ACTIVE	06-1994	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	80	1	Open: JANUARY Close: DECEMBER	
228	1278060	CARRIAGE TOWNE PLAZA	53 CHURCH ST		KINGSTON	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	43	17	Open: JANUARY Close: DECEMBER	
229	1279010	KINGSTON CHILDREN CENTER	12 CHURCH ST		KINGSTON	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	71	1	Open: JANUARY Close: DECEMBER	
230	1285010	LACONIA CHRISTIAN SCH	1386 MEREDITH CENTER RD		LACONIA	ACTIVE	01-1979	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	108	4	Open: SEPTEMBER Close: JUNE	
231	1289010	PRESCOTT FARM ENVIRON ED CTR	928 WHITE OAKS RD		LACONIA	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	50	1	Open: JANUARY Close: DECEMBER	
232	1296010	EASTER SEALS YOUTH RESDNCE SVS	525 PROSPECT ST		LANCASTER	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	48	1	Open: JANUARY Close: DECEMBER	
233	1305020	LANDAFF BLUE SCH	813 MILLBROOK RD		LANDAFF	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	25	1	Open: SEPTEMBER Close: JUNE	
234	1315010	FALL MOUNTAIN REGIONAL HS	134 FALL MT REG HS RD		LANGDON	ACTIVE	01-1966	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	850	5	Open: JANUARY Close: DECEMBER	
235	1315020	SARAH PORTER SCH	111 VILLAGE RD	RTE 12A N	LANGDON	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	43	2	Open: SEPTEMBER Close: JUNE	
236	1335010	MAST WAY ELEMENTARY SCH	23 MAST RD	RTE 155	LEE	ACTIVE	01-1960	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	392	1	Open: SEPTEMBER Close: JUNE	
237	1335020	LIVE AND LEARN DAY CARE	114 MAST RD	RTE 155	LEE	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	45	1	Open: JANUARY Close: DECEMBER	
238	1335030	GROWING PLACES	56 PINKHAM RD		LEE	ACTIVE	01-1965	NON-TRANSIENT NON-COMMUI	DAY CARE	47	2	Open: JANUARY Close: DECEMBER	
239	1336020	LEE MARKETPLACE	54 CALEF RD	LEE TRAFFIC CIR	LEE	ACTIVE	06-1993	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	25	2	Open: JANUARY Close: DECEMBER	
240	1336030	BENUCK AND RAINEY	25 CONCORD RD		LEE	ACTIVE	10-2017	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	40	1	Open: JANUARY Close: DECEMBER	
241	1339010	LEE CONGREGATIONAL CHURCH	17 MAST RD	RTE 155	LEE	ACTIVE	01-1980	NON-TRANSIENT NON-COMMUI	DAY CARE	34	2	Open: JANUARY Close: DECEMBER	
242	1339030	WENTWORTH DOUGLASS MED OFF	65 CALEF HWY	RTE 125	LEE	ACTIVE	01-2009	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	379	1	Open: JANUARY Close: DECEMBER	
243	1345010	LEMPSTER COMMUNITY SCH	29 SCHOOL ST	RTE 10	LEMPSTER	ACTIVE	01-1956	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	150	1	Open: JANUARY Close: DECEMBER	
244	1378070	LOBSTER BOAT PLZ	273 DERRY RD		LITCHFIELD	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	70	9	Open: JANUARY Close: DECEMBER	
245	1379010	TABERNACLE CHRISTIAN SCH	242 DERRY RD	RTE 102	LITCHFIELD	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	125	1	Open: JANUARY Close: DECEMBER	
246	1386010	PEAK THREE	RTE 135	ORTH LITTLETOI	LITTLETON	ACTIVE	09-1996	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	40	1	Open: JANUARY Close: DECEMBER	
247	1395090	CREATIVE LITTLE ANGELS	40 MAMMOTH RD	RTE 128	LONDONDERR	ACTIVE	01-1973	NON-TRANSIENT NON-COMMUI	DAY CARE	167	1	Open: JANUARY Close: DECEMBER	
248	1395110	28 BUTTRICK RD PROPERTY	28 BUTTRICK RD		LONDONDERR	ACTIVE	06-1986	NON-TRANSIENT NON-COMMUI	DAY CARE	83	1	Open: JANUARY Close: DECEMBER	
249	1395140	VICTORY BAPTIST SCH	78 LITCHFIELD RD		LONDONDERR	ACTIVE	08-2003	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	79	1	Open: AUGUST Close: JUNE	
250	1396060	ZOHL WPS INC	562 MAMMOTH RD		LONDONDERR	ACTIVE		NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	40	2	Open: JANUARY Close: DECEMBER	
251	1396070	TOWNE SQUARE PROF CONDOS	12 PARMENTER RD		LONDONDERR	ACTIVE	01-1984	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	75	26	Open: JANUARY Close: DECEMBER	
252	1396110	REEDS FERRY SMALL BLDGS	3 TRACY LN		LONDONDERR	ACTIVE	04-2019	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	27	1	Open: JANUARY Close: DECEMBER	
253	1405010	LOUDON ELEMENTARY SCH	7039 SCHOOL ST		LOUDON	ACTIVE	01-1970	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	321	1	Open: SEPTEMBER Close: JUNE	
254	1407060	NH MOTOR SPEEDWAY/MAIN OFFICE	1122 RTE 106 NORTH		LOUDON	ACTIVE	06-1990	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	45	4	Open: JANUARY Close: DECEMBER	
255	1435030	CROSSROADS ACADEMY	95 DARTMOUTH COLLEGE HWY		LYME	ACTIVE	09-1991	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	162	3	Open: JANUARY Close: DECEMBER	
256	1435040	CROSSROADS ACADEMY/NORTH	95 DARTMOUTH COLLEGE HWY		LYME	ACTIVE	08-2006	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	180	1	Open: JANUARY Close: DECEMBER	
257	1435050	LYME NURSERY SCHL	155 DARTMOUTH COLLEGE HWY		LYME	ACTIVE	06-2005	NON-TRANSIENT NON-COMMUI	DAY CARE	36	1	Open: JANUARY Close: DECEMBER	
258	1436020	PATHWAYS ORFORD ROAD RESIDEN(	RTE 10		LYME	ACTIVE	08-1985	NON-TRANSIENT NON-COMMUI	INSTITUTION, REHAB FACILITIES	40	1	Open: JANUARY Close: DECEMBER	
259	1445010	LYNDEBOROUGH CENTRAL SCH	192 FOREST RD	192 RTE 31	LYNDEBOROU	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	74	1	Open: JANUARY Close: DECEMBER	
260	1455010	MOHARIMET SCH	11 LEE RD	RTE 155	MADBURY	ACTIVE	09-1988	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	427	1	Open: SEPTEMBER Close: JUNE	
261	1456030	CARRIAGE HILL ASSISTED LIVING	306 KNOX MARSH RD		MADBURY	ACTIVE	11-2015	NON-TRANSIENT NON-COMMUI	INSTITUTION, REHAB FACILITIES	31	1	Open: JANUARY Close: DECEMBER	
262	1458010	LITTLE TREE ED CENTER	316 RTE 108		MADBURY	ACTIVE	09-1997	NON-TRANSIENT NON-COMMUI	DAY CARE	60	1	Open: JANUARY Close: DECEMBER	
263	1465010	MADISON ELEMENTARY SCH	2069 VILLAGE RD	RTE 113	MADISON	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	170	2	Open: SEPTEMBER Close: JUNE	
264	1466020	MADISON LUMBER MILL	71 MARCELLA DR	OFF RTE 41	MADISON	ACTIVE	01-1984	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	50	5	Open: JANUARY Close: DECEMBER	
265	1466050	MACLEAN PRECISION MACHINE	1928 VILLAGE RD	1928 RTE 113	MADISON	ACTIVE	01-1993	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	30	1	Open: JANUARY Close: DECEMBER	
266	1475010	VA MEDICAL CENTER MANCHESTER	718 SMYTH RD		MANCHESTER	ACTIVE		NON-TRANSIENT NON-COMMUI	PRIVATELY OWNED REDISTRIBUTION SYSTEI	1700	1	Open: JANUARY Close: DECEMBER	
267	1495010	JOHN D PERKINS SR ELEM SCH	919 RTE 10		MARLOW	ACTIVE	09-1973	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	41	1	Open: SEPTEMBER Close: JUNE	
268	1515010	MASON PUBLIC SCH	13 DARLING HILL RD		MASON	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	122	2	Open: SEPTEMBER Close: JUNE	
269	1525010	INTER LAKES HIGH SCH	1 LAKER LN	RTE 25	MEREDITH	ACTIVE	09-1956	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	556	1	Open: JANUARY Close: DECEMBER	
270	1525020	INTER LAKES ELEMENTARY SCH	21 LAKER LN	RTE 25	MEREDITH	ACTIVE	09-1972	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	587	1	Open: JANUARY Close: DECEMBER	
271	1525030	LAKELAND SCH	40 MEREDITH CTR RD	OFF RTE 104	MEREDITH	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	80	1	Open: SEPTEMBER Close: JUNE	
272	1545010	MIDDLETON ELEMENTARY SCHOOL	116 KINGS HWY		MIDDLETON	ACTIVE	06-2016	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	174	1	Open: JANUARY Close: DECEMBER	
273	1555010	MILAN VILLAGE SCH	11 BRIDGE ST		MILAN	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	151	3	Open: SEPTEMBER Close: JUNE	
274	1565010	LITTLE ARROWS CHILD CARE SVS	365 SOUTH ST	RTE 13	MILFORD	ACTIVE	07-1990	NON-TRANSIENT NON-COMMUI	DAY CARE	52	1	Open: JANUARY Close: DECEMBER	

	A	B	C	D	E	F	G	H	I	J	K	L	M
275	1566020	MILFORD TECHNOLOGY CENTER	528 RTE 13S		MILFORD	ACTIVE	01-1980	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	160	1	Open: JANUARY Close: DECEMBER	
276	1566040	115/119 EMERSON RD	115/119 EMERSON RD		MILFORD	ACTIVE	02-2014	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	32	3	Open: JANUARY Close: DECEMBER	
277	1582010	SHORTRIDGE ACADEMY	619 GOVERNORS RD		MILTON	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	64	6	Open: JANUARY Close: DECEMBER	
278	1585030	MILTON CHILDRENS CENTER	55 INDUSTRIAL WAY		MILTON	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	25	1	Open: JANUARY Close: DECEMBER	
279	1586040	INDEX PACKAGING	1055 WHITE MOUNTAIN HWY	RTE 125	MILTON	ACTIVE		NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	165	5	Open: JANUARY Close: DECEMBER	
280	1605010	MONT VERNON VILLAGE SCH	1 KITTREDGE RD		MONT VERNOI	ACTIVE	01-1969	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	216	1	Open: JANUARY Close: DECEMBER	
281	1615010	MOULTONBOROUGH CENTRAL SCH	916 WHITTIER HWY	RTE 25	MOULTONBOR	ACTIVE	01-1974	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	330	1	Open: SEPTEMBER Close: JUNE	
282	1615020	MOULTONBOROUGH ACADEMY	25 BLAKE RD	RTE 25	MOULTONBOR	ACTIVE	09-1980	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	345	1	Open: SEPTEMBER Close: JUNE	
283	1615030	A CHILDS PLACE	903 WHITTIER HWY	RTE 25	MOULTONBOR	ACTIVE	09-1991	NON-TRANSIENT NON-COMMUI	DAY CARE	59	1	Open: JANUARY Close: DECEMBER	
284	1616010	HARBOR PINES ON LAKE SHORE DR	LAKE SHORE DR		MOULTONBOR	ACTIVE	07-1991	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	58	12	Open: JANUARY Close: DECEMBER	
285	1616040	CRUCON CRUISE OUTLET	81 WHITTIER HWY		MOULTONBOR	ACTIVE	04-2018	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	130	1	Open: JANUARY Close: DECEMBER	
286	1618240	HARBOR SQUARE SHOPPING CENTEI	60 WHITTIER HWY	TR HARBOR TOV	MOULTONBOR	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	43	17	Open: JANUARY Close: DECEMBER	
287	1625020	SECOND NATURE ACADEMY	10 GROTON RD		NASHUA	ACTIVE	01-2009	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	212	2	Open: JANUARY Close: DECEMBER	
288	1635010	NELSON ELEMENTARY SCH	441 GRANITE LAKE RD	RTE 9	NELSON	ACTIVE	01-1950	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	60	1	Open: SEPTEMBER Close: JUNE	
289	1645010	NEW BOSTON ELEMENTARY SCH	15 CENTRAL SCHOOL RD	RTE 13	NEW BOSTON	ACTIVE	01-1954	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	613	3	Open: JANUARY Close: DECEMBER	
290	1645020	STRONG FOUNDATIONS	643 NORTH MAST RD	RTE 114	NEW BOSTON	ACTIVE	01-1982	NON-TRANSIENT NON-COMMUI	DAY CARE	139	1	Open: JANUARY Close: DECEMBER	
291	1645030	CHESTNUT CHRISTIAN PRESCHOOL	219 CHESTNUT HILL RD		NEW BOSTON	ACTIVE	12-2011	NON-TRANSIENT NON-COMMUI	DAY CARE	34	1	Open: JANUARY Close: DECEMBER	
292	1645040	ROSE MEADOW ACRES	539 OLD COACH RD		NEW BOSTON	ACTIVE	09-2015	NON-TRANSIENT NON-COMMUI	HOSPITAL, MEDICAL FACILITY	31	1	Open: JANUARY Close: DECEMBER	
293	1646020	USAF NH SATELLITE TRACKING STN	317 CHESTNUT HILL RD	BLDG 107	NEW BOSTON	ACTIVE	01-1961	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	123	13	Open: JANUARY Close: DECEMBER	
294	1656010	NEWBURY HARBOR PLAZA	RTE 103	WBURY HEIGHTS	NEWBURY	ACTIVE	01-1985	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	25	14	Open: JANUARY Close: DECEMBER	
295	1675010	NEW DURHAM ELEMENTARY SCH	7 OLD BAY RD		NEW DURHAM	ACTIVE	09-1963	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	180	1	Open: SEPTEMBER Close: JUNE	
296	1675020	7 DEPOT RD/BEING COOL	7 DEPOT RD		NEW DURHAM	ACTIVE	02-2020	NON-TRANSIENT NON-COMMUI	DAY CARE	45	1	Open: JANUARY Close: DECEMBER	
297	1715020	MASCENIC REGIONAL HIGH SCH	175 TURNPIKE RD		NEW IPSWICH	ACTIVE	01-1978	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	350	1	Open: SEPTEMBER Close: JUNE	
298	1715040	BOYNTON MIDDLE SCH	500 TURNPIKE RD	RTE 124	NEW IPSWICH	ACTIVE	08-1989	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	410	1	Open: SEPTEMBER Close: JUNE	
299	1715060	HIGHBRIDGE HILL ELEM SCH	171 TURNPIKE RD		NEW IPSWICH	ACTIVE	08-2010	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	465	1	Open: JANUARY Close: DECEMBER	
300	1716050	VANGUARD MANUFACTURING	90 TEMPLE RD		NEW IPSWICH	ACTIVE	01-1970	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	30	1	Open: JANUARY Close: DECEMBER	
301	1716060	APPLETON BUSINESS CENTER	30 TRICNIT RD		NEW IPSWICH	ACTIVE	01-1963	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	32	16	Open: JANUARY Close: DECEMBER	
302	1716080	WARWICK MILLS	301 TURNPIKE RD		NEW IPSWICH	ACTIVE		NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	110	1	Open: JANUARY Close: DECEMBER	
303	1746020	LATVA MACHINE	744 JOHN STARK HWY		NEWPORT	ACTIVE	01-1991	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	45	1	Open: JANUARY Close: DECEMBER	
304	1749010	KIDS WORLD ACADEMY OF NH	660 JOHN STARK HWY		NEWPORT	ACTIVE	06-2014	NON-TRANSIENT NON-COMMUI	DAY CARE	50	2	Open: JANUARY Close: DECEMBER	
305	1755010	SANBORN REGIONAL MIDDLE SCH	31A WEST MAIN ST	ER ST EXT/NEWT	NEWTON	ACTIVE	01-1978	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	703	2	Open: SEPTEMBER Close: JUNE	
306	1755050	NEWTON LEARNING CENTER	31 S MAIN ST	RTE 108	NEWTON	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	82	1	Open: JANUARY Close: DECEMBER	
307	1756040	TEREX ENVIRONMENTAL EQUIPMENT	22 WHITTIER ST		NEWTON	ACTIVE		NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	50	1	Open: JANUARY Close: DECEMBER	
308	1756050	RAVENSBURGER USA	ONE PUZZLE LN	153 S MAIN ST	NEWTON	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	40	1	Open: JANUARY Close: DECEMBER	
309	1764010	SPAULDING YOUTH CENTER	72 SPAULDING RD		NORTHFIELD	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	300	11	Open: JANUARY Close: DECEMBER	
310	1766030	YOUNG ENTERPRISES NH	200 TILTON RD	RTE 140	NORTHFIELD	ACTIVE	11-1988	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	33	1	Open: JANUARY Close: DECEMBER	
311	1795020	COE BROWN ACADEMY	907 FIRST NH TPKE	RTE 4	NORTHWOOD	ACTIVE	01-1980	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	796	4	Open: SEPTEMBER Close: JUNE	
312	1795040	COE BROWN SMITH HALL	902 FIRST NH TPKE		NORTHWOOD	ACTIVE	11-1994	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	789	1	Open: SEPTEMBER Close: JUNE	
313	1796010	HANNAFORD NORTHWOOD 8160	178 FIRST NH TPKE		NORTHWOOD	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	30	1	Open: JANUARY Close: DECEMBER	
314	1798030	JOHNSONS SEAFOOD AND STEAK	1334 FIRST NH TPKE		NORTHWOOD	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	25	1	Open: JANUARY Close: DECEMBER	
315	1805050	NOTTINGHAM COMMUNITY SCH	245 STAGE RD	RTE 152	NOTTINGHAM	ACTIVE	07-1994	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	609	1	Open: JANUARY Close: DECEMBER	
316	1805060	ALL ABOARD PRESCH AND CHLDCAR	249 STAGE RD	RTE 152	NOTTINGHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	86	2	Open: JANUARY Close: DECEMBER	
317	1809020	NOTTINGHAM COMMUNITY CHURCH	106 CHURCH ST		NOTTINGHAM	ACTIVE	04-2016	NON-TRANSIENT NON-COMMUI	DAY CARE	28	1	Open: JANUARY Close: DECEMBER	
318	1835010	RIVENDELL INTERSTATE SCH	2972 RTE 25A	HOOL DR /ADMIN	ORFORD	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	280	2	Open: SEPTEMBER Close: JUNE	
319	1842020	INDIAN MOUND SHOPPING CENTER	OSSIPEE LAKE DR	RTE 16 BYPASS	OSSIPEE	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	40	9	Open: JANUARY Close: DECEMBER	
320	1845020	CORNERSTONE CHRISTIAN ACADEMY	129 RTE 28		OSSIPEE	ACTIVE	09-1997	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	72	3	Open: JANUARY Close: DECEMBER	
321	1846010	OCEAN STATE JOB LOT	JCT RTE 16 AND RTE 28		OSSIPEE	ACTIVE	01-1981	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	45	1	Open: JANUARY Close: DECEMBER	
322	1846090	HANNAFORD OSSIPEE 8159	JCT RTE 16 AND RTE 28		OSSIPEE	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	30	1	Open: JANUARY Close: DECEMBER	
323	1855060	PERFECT PLACE FOR CHILDREN	125 MAIN ST UNIT A	OFF RTE 38N	PELHAM	ACTIVE	01-1950	NON-TRANSIENT NON-COMMUI	DAY CARE	54	5	Open: JANUARY Close: DECEMBER	
324	1855070	DYNAMIC FOUNDATION FOR CHLDRE	90 BRIDGE ST		PELHAM	ACTIVE	11-2001	NON-TRANSIENT NON-COMMUI	DAY CARE	60	1	Open: JANUARY Close: DECEMBER	
325	1855080	COLONIAL WAY PLAZA	43 BRIDGE ST		PELHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	28	1	Open: JANUARY Close: DECEMBER	
326	1855090	KINGS KIDS CHILD CARE CENTER	955 BRIDGE ST	RTE 38	PELHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	100	1	Open: JANUARY Close: DECEMBER	
327	1856010	PELHAM PLAZA	150 BRIDGE ST	RTE 38	PELHAM	ACTIVE	09-1966	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	100	2	Open: JANUARY Close: DECEMBER	
328	1856040	HANNAFORD PELHAM 8015	150 BRIDGE ST	RTE 38	PELHAM	ACTIVE	01-1965	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	90	1	Open: JANUARY Close: DECEMBER	
329	1856050	WAKEFIELD THERMAL SOLUTIONS	33 BRIDGE ST	RTE 38	PELHAM	ACTIVE	04-1986	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	200	1	Open: JANUARY Close: DECEMBER	
330	1856070	PELHAM INDUSTRIAL PARK I	1 INDUSTRIAL PARK DR	RTE 128	PELHAM	ACTIVE	01-1986	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	100	28	Open: JANUARY Close: DECEMBER	
331	1856080	VICTORY SUPERMARKET	150 BRIDGE ST		PELHAM	INACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	102	1	Open: Close:	
332	1859040	CROSSROADS BAPTIST CHURCH	43 ATWOOD RD		PELHAM	ACTIVE	08-1978	NON-TRANSIENT NON-COMMUI	DAY CARE	50	1	Open: JANUARY Close: DECEMBER	
333	1875010	HAPPY VALLEY SCHOOL	130 GULF RD		PETERBOROU	ACTIVE	09-1970	NON-TRANSIENT NON-COMMUI	DAY CARE	40	1	Open: JANUARY Close: DECEMBER	
334	1875020	WELL SCHOOL	360 MIDDLE HANCOCK RD		PETERBOROU	ACTIVE	01-1965	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	160	5	Open: JANUARY Close: DECEMBER	
335	1875030	MONADNOCK COMM EARLY LNG CTR	5 COMMUNITY LN		PETERBOROU	ACTIVE	11-1980	NON-TRANSIENT NON-COMMUI	DAY CARE	80	1	Open: JANUARY Close: DECEMBER	
336	1885010	PIERMONT VILLAGE SCH	131 RTE 10		PIERMONT	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	88	1	Open: JANUARY Close: DECEMBER	
337	1897020	AMC PINKHAM NOTCH CAMP	361 RTE 16		PINKHAMS GR	ACTIVE	01-1965	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	60	7	Open: JANUARY Close: DECEMBER	
338	1926010	TOWNLINE EQUIPMENT	1474 RTE 12A		PLAINFIELD	ACTIVE	08-2014	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	35	1	Open: JANUARY Close: DECEMBER	
339	1935010	POLLARD ELEMENTARY SCH	120 MAIN ST		PLAISTOW	ACTIVE	01-1952	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	574	2	Open: SEPTEMBER Close: JUNE	
340	1935020	TIMBERLANE MIDDLE SCH	44 GREENOUGH RD		PLAISTOW	ACTIVE	01-1968	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	984	1	Open: SEPTEMBER Close: JUNE	
341	1935030	TIMBERLANE REGIONAL HS	36 GREENOUGH RD		PLAISTOW	ACTIVE	01-1964	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	1295	2	Open: SEPTEMBER Close: JUNE	
342	1935070	LITTLE EXPLORERS	3 BLOSSOM RD		PLAISTOW	ACTIVE	08-1997	NON-TRANSIENT NON-COMMUI	DAY CARE	44	1	Open: JANUARY Close: DECEMBER	
343	1936070	144 MAIN STREET	144 MAIN ST		PLAISTOW	ACTIVE	11-2016	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	95	1	Open: JANUARY Close: DECEMBER	

	A	B	C	D	E	F	G	H	I	J	K	L	M
344	1936100	PENTUCKET SHOPPING CENTER	HOME DEPOT USA	LAISTOW RD /RT	PLAISTOW	ACTIVE	01-1985	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	200	6	Open: JANUARY Close: DECEMBER	
345	1936110	SCANDIA PLASTICS	55 WESTVILLE RD	55 PLAISTOW RD	PLAISTOW	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	35	1	Open: JANUARY Close: DECEMBER	
346	1936130	PLAISTOW COMMONS	160 RTE 125		PLAISTOW	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	200	17	Open: JANUARY Close: DECEMBER	
347	1936150	MARKET BASKET DEMOULAS 25	34 PLAISTOW RD	RTE 125	PLAISTOW	ACTIVE	01-1979	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	395	5	Open: JANUARY Close: DECEMBER	
348	1936230	3 / 11 PLAISTOW RD PLAZA	RTE 125	9 PLAISTOW RD	PLAISTOW	ACTIVE	08-1989	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	500	39	Open: JANUARY Close: DECEMBER	
349	1936260	MARKET BASKET/BRADLEES	34 PLAISTOW RD	RTE 125	PLAISTOW	INACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	65	1	Open: Close:	
350	1936270	FIELDSTONE INDUSTRIAL PARK	2 WILDER DR		PLAISTOW	ACTIVE		NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	75	2	Open: JANUARY Close: DECEMBER	
351	1936280	SPARTON BECKWOOD	27 HALE SPRING RD		PLAISTOW	ACTIVE	05-1985	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	35	1	Open: JANUARY Close: DECEMBER	
352	1936300	BARONS CONDOS	95A PLAISTOW RD		PLAISTOW	ACTIVE		NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	60	1	Open: JANUARY Close: DECEMBER	
353	1936310	ETHAN ALLEN PLAZA	24/26 PLAISTOW RD		PLAISTOW	ACTIVE	01-1973	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	32	5	Open: JANUARY Close: DECEMBER	
354	1938090	BRICKYARD I PLAZA	95 PLAISTOW RD	RTE 125	PLAISTOW	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	80	19	Open: JANUARY Close: DECEMBER	
355	1938190	GREAT ELM PLAZA	37 PLAISTOW RD		PLAISTOW	ACTIVE	01-1991	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	30	11	Open: JANUARY Close: DECEMBER	
356	1938240	PLAISTOW COMMUNITY YMCA	175 PLAISTOW RD		PLAISTOW	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	99	1	Open: JANUARY Close: DECEMBER	
357	1945010	MOUNTAIN VILLAGE SCHOOL	13 RTE 25		PLYMOUTH	ACTIVE	08-2014	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	62	1	Open: JANUARY Close: DECEMBER	
358	1949030	PLYMOUTH COMMERCE PARK	12 YEATON RD		PLYMOUTH	ACTIVE	01-2007	NON-TRANSIENT NON-COMMUI	DAY CARE	43	2	Open: JANUARY Close: DECEMBER	
359	1985010	ST BENEDICT CENTER MONASTERY	95 FAY MARTIN RD	OFF RTE 119	RICHMOND	ACTIVE	08-1989	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	200	5	Open: JANUARY Close: DECEMBER	
360	1994010	FRANKLIN PIERCE UNIVERSITY	40 UNIVERSITY DR		RINDGE	ACTIVE	08-1962	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	1638	37	Open: JANUARY Close: DECEMBER	
361	1995010	RINDGE MEMORIAL SCH	45 SCHOOL ST		RINDGE	ACTIVE	09-1950	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	450	2	Open: JANUARY Close: DECEMBER	
362	1995030	HAMPSHIRE COUNTRY SCHOOL	28 PATEY CIR		RINDGE	ACTIVE	01-1901	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	40	5	Open: JANUARY Close: DECEMBER	
363	1995050	HERITAGE CHRISTIAN SCHOOL	13 NORTH ST		RINDGE	ACTIVE	09-1996	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	64	1	Open: SEPTEMBER Close: JUNE	
364	1996010	CHESHIRE MARKET PLACE	497 RTE 202S		RINDGE	ACTIVE	01-1991	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	200	7	Open: JANUARY Close: DECEMBER	
365	1996020	WAL MART STORE 2057	750 RTE 202		RINDGE	ACTIVE	01-1994	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	40	1	Open: JANUARY Close: DECEMBER	
366	1996040	HANNAFORD RINDGE 8180	RTE 202		RINDGE	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	275	1	Open: JANUARY Close: DECEMBER	
367	1996060	PHASE 10	31 SONJA DR		RINDGE	ACTIVE	11-2005	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	69	1	Open: JANUARY Close: DECEMBER	
368	2035010	RUSSELL ELEMENTARY SCH	195 SCHOOL ST	RTE 25	RUMNEY	ACTIVE	01-1957	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	157	1	Open: JANUARY Close: DECEMBER	
369	2036010	STONEWALL CABLE	126 HAWKENSEN DR	RTE 25	RUMNEY	ACTIVE		NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	64	3	Open: JANUARY Close: DECEMBER	
370	2055010	NORTH SALEM ELEMENTARY SCH	140 ZION HILL RD	MILLVILLE RD	SALEM	ACTIVE	09-1968	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	427	1	Open: SEPTEMBER Close: JUNE	
371	2055020	MERRIMACK VALLEY MONTESSOR SC	111 LOWELL RD		SALEM	ACTIVE	09-1967	NON-TRANSIENT NON-COMMUI	DAY CARE	65	1	Open: SEPTEMBER Close: JUNE	
372	2056010	KLEIN MARINE SYSTEMS	11 KLEIN DR	TE 111 /HAVERH	SALEM	ACTIVE	07-1985	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	47	2	Open: JANUARY Close: DECEMBER	
373	2056040	NORTH SALEM VILLAGE	15 ERMER RD	RTE 111	SALEM	ACTIVE	01-1987	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	50	1	Open: JANUARY Close: DECEMBER	
374	2065010	SALISBURY ELEMENTARY SCH	6 WHITTEMORE RD	RTE 4	SALISBURY	ACTIVE	01-1959	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	101	1	Open: SEPTEMBER Close: JUNE	
375	2075010	SANT BANI SCHOOL UPPER BLDG	221A OSGOOD RD	127S /PRESCOT	SANBORNTON	ACTIVE	01-1973	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	60	2	Open: SEPTEMBER Close: JUNE	
376	2075020	SANBORNTON CENTRAL SCH	16 HUNKINS POND RD	RTE 132	SANBORNTON	ACTIVE	01-1949	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	244	1	Open: SEPTEMBER Close: JUNE	
377	2075030	SANT BANI SCHOOL MIDDLE BLDG	WEEKS OSGOOD RD	127S /PRESCOT	SANBORNTON	ACTIVE	01-1988	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	120	1	Open: SEPTEMBER Close: JUNE	
378	2075070	SAPLINGS PRESCHOOL	266 UPPER BAY RD		SANBORNTON	ACTIVE	10-2019	NON-TRANSIENT NON-COMMUI	DAY CARE	28	2	Open: JANUARY Close: DECEMBER	
379	2078010	STEELE HILL RESORT	UPPER BAY RD	6 STEELE HILL R	SANBORNTON	ACTIVE	01-1942	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	60	200	Open: JANUARY Close: DECEMBER	
380	2085010	SANDOWN CENTRAL SCH	295 MAIN ST		SANDOWN	ACTIVE	07-1971	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	170	1	Open: SEPTEMBER Close: JUNE	
381	2085020	PLAYMATES LEARNING CENTER	56 NORTH RD	OFF RTE 121A	SANDOWN	ACTIVE	01-1976	NON-TRANSIENT NON-COMMUI	DAY CARE	56	1	Open: JANUARY Close: DECEMBER	
382	2085040	SANDOWN NORTH ELEMENTARY SCH	23 STAGECOACH DR		SANDOWN	ACTIVE	09-2001	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	417	1	Open: SEPTEMBER Close: JUNE	
383	2095020	SANDWICH CENTRAL SCH	28 SQUAM LAKE RD		SANDWICH	ACTIVE	01-1950	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	87	1	Open: SEPTEMBER Close: JUNE	
384	2095030	SANDWICH CHILDRENS CENTER	54 MAPLE ST		SANDWICH	ACTIVE	01-1985	NON-TRANSIENT NON-COMMUI	DAY CARE	48	1	Open: JANUARY Close: DECEMBER	
385	2165020	BARNARD SCH	219 MAIN AVE	AKA RTE 107A	SOUTH HAMPT	ACTIVE	01-1998	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	107	2	Open: SEPTEMBER Close: JUNE	
386	2185010	STARK VILLAGE SCH	1192 STARK HWY	RTE 110	STARK	ACTIVE	01-1976	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	30	1	Open: SEPTEMBER Close: JUNE	
387	2195020	STEWARTSTOWN COMMUNITY SCH	60 SCHOOL ST	3/W STEWARTST	STEWARTSTO	ACTIVE	05-1998	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	93	1	Open: SEPTEMBER Close: JUNE	
388	2205010	JAMES FAULKNER MEMORIAL SCH	200 SCHOOL ST	OFF RTE 123	STODDARD	ACTIVE	01-1979	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	96	1	Open: SEPTEMBER Close: JUNE	
389	2206010	CARLISLE WIDE PLANK FLOORS	1676 RTE 9		STODDARD	ACTIVE	03-1996	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	40	1	Open: JANUARY Close: DECEMBER	
390	2215010	STRAFFORD SCH	22 ROLLER COASTER RD	RTE 202A	STRAFFORD	ACTIVE	09-1960	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	481	4	Open: JANUARY Close: DECEMBER	
391	2215040	WHITEHOUSE EARLY LEARNING CTR	352 PROVINCE RD		STRAFFORD	ACTIVE	06-2019	NON-TRANSIENT NON-COMMUI	DAY CARE	28	1	Open: JANUARY Close: DECEMBER	
392	2235010	ACORN SCH	136 WINNICUTT RD		STRATHAM	ACTIVE	01-1975	NON-TRANSIENT NON-COMMUI	DAY CARE	54	1	Open: SEPTEMBER Close: JUNE	
393	2235050	STRATHAM MEMORIAL SCH	39 GIFFORD FARM RD	ELL RD /OFF RTE	STRATHAM	ACTIVE	09-1989	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	705	1	Open: SEPTEMBER Close: JUNE	
394	2235060	CORNERSTONE SCHOOL	146 HIGH ST		STRATHAM	ACTIVE	09-1988	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	210	1	Open: JANUARY Close: DECEMBER	
395	2236010	KINGS HIGHWAY PLAZA	28 PORTSMOUTH AVE	E 101 AND RTE 1	STRATHAM	ACTIVE	01-1974	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	40	14	Open: JANUARY Close: DECEMBER	
396	2236020	MARKET BASKET	27 PORTSMOUTH AVE	RTE 101	STRATHAM	INACTIVE	07-1971	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	250	1	Open: Close:	
397	2236040	BELL AND FLYNN	69 BUNKER HILL AVE		STRATHAM	ACTIVE	01-1956	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	26	3	Open: JANUARY Close: DECEMBER	
398	2236050	STRATHAM PLAZA	72 PORTSMOUTH PLZ	TE 108 AND RTE	STRATHAM	ACTIVE	01-1987	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	50	19	Open: JANUARY Close: DECEMBER	
399	2236070	NP STRATHAM	20 PORTSMOUTH AVE		STRATHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	100	9	Open: JANUARY Close: DECEMBER	
400	2236090	RCN CONDOS	4 WEST RD		STRATHAM	ACTIVE	03-1989	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	75	2	Open: JANUARY Close: DECEMBER	
401	2236100	PIPERS LANDING	142 PORTSMOUTH AVENUE		STRATHAM	ACTIVE	12-1986	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	45	1	Open: JANUARY Close: DECEMBER	
402	2236120	MILLBROOK OFFICE PARK	118 PORTSMOUTH AVE		STRATHAM	ACTIVE	09-1998	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	55	2	Open: JANUARY Close: DECEMBER	
403	2236130	STRATHAM CENTRAL CONDOS	157 PORTSMOUTH AVE		STRATHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	50	3	Open: JANUARY Close: DECEMBER	
404	2236140	STRATHAM CROSSING 7621	100 SHAWS LN		STRATHAM	ACTIVE	11-2004	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	56	3	Open: JANUARY Close: DECEMBER	
405	2236150	BMW OF STRATHAM	71 PORTSMOUTH AVE		STRATHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	49	1	Open: JANUARY Close: DECEMBER	
406	2236160	LINDT AND SPRUNGLI USA BLD B/C	1 FINE CHOCOLATE PL		STRATHAM	INACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	100	1	Open: JANUARY Close: DECEMBER	
407	2236170	LINDT AND SPRUNGLI USA/BLDG D	1 FINE CHOCOLATE PL		STRATHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	50	1	Open: JANUARY Close: DECEMBER	
408	2236180	LINDT AND SPRUNGLI USA/BLDG E	1 FINE CHOCOLATE PL		STRATHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	25	1	Open: JANUARY Close: DECEMBER	
409	2236190	149/151 PORTSMOUTH AVE	149 PORTSMOUTH AVE		STRATHAM	ACTIVE	12-2018	NON-TRANSIENT NON-COMMUI	WORKPLACE (NOT COMMERCIAL OR INDUST	25	3	Open: JANUARY Close: DECEMBER	
410	2238060	STRATHAM PLZ/MARKET BASKET	27 PORTSMOUTH AVE		STRATHAM	ACTIVE	01-1985	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	410	3	Open: JANUARY Close: DECEMBER	
411	2239010	STRATHAM COMMUNITY CHURCH	6 EMERY LN		STRATHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	55	1	Open: JANUARY Close: DECEMBER	
412	2275020	MOUNT ROYAL ACADEMY	26 SEVEN HEARTHS LN	RTE 11	SUNAPEE	ACTIVE	09-1998	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	217	3	Open: SEPTEMBER Close: JUNE	

	A	B	C	D	E	F	G	H	I	J	K	L	M
413	2285010	SURRY VILLAGE CHARTER SCH	449 RTE 12A		SURRY	ACTIVE	01-1978	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	83	1	Open: SEPTEMBER Close: JUNE	
414	2295010	KEARSARGE REGIONAL HIGH SCH	457 NORTH RD	I89 EXIT 10	SUTTON	ACTIVE	09-1969	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	650	1	Open: SEPTEMBER Close: JUNE	
415	2295020	SUTTON CENTRAL SCH	28 NEWBURY RD	SUTTON MILLS	SUTTON	ACTIVE	06-1954	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	118	1	Open: SEPTEMBER Close: JUNE	
416	2295030	KEARSARGE REGIONAL MIDDLE SCH	32 GILE POND RD	114/NORTH SUT	SUTTON	ACTIVE	03-2008	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	525	1	Open: JANUARY Close: DECEMBER	
417	2296010	LABSPHERE	231 SHAKER ST		SUTTON	ACTIVE		NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	86	1	Open: JANUARY Close: DECEMBER	
418	2305050	MONADNOCK REGIONAL HIGH SCH	580 OLD HOMESTEAD HWY	E 32/CTR SWANZ	SWANZEY	ACTIVE	09-1962	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	1202	3	Open: JANUARY Close: DECEMBER	
419	2306050	HONDA OF KEENE	567 MONADNOCK HWY	RTE 12	SWANZEY	ACTIVE	09-2002	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	40	1	Open: JANUARY Close: DECEMBER	
420	2306060	TOYOTA OF KEENE	591 MONADNOCK HWY	RTE 12	SWANZEY	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	85	2	Open: JANUARY Close: DECEMBER	
421	2315010	KENNETH A BRETT SCH	881 TAMWORTH RD	RTE 113	TAMWORTH	ACTIVE	09-1955	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	230	1	Open: SEPTEMBER Close: JUNE	
422	2315020	TAMWORTH PRESCHOOL	27 DURRELL RD	RTE 113	TAMWORTH	ACTIVE	10-1987	NON-TRANSIENT NON-COMMUI	DAY CARE	100	1	Open: JANUARY Close: DECEMBER	
423	2315030	COMMUNITY SCH	1164 BUNKER HILL RD	113W/S TAMWO	TAMWORTH	ACTIVE	09-1992	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	45	1	Open: JANUARY Close: DECEMBER	
424	2345010	THORNTON CENTRAL SCH	1886 RTE 175		THORNTON	ACTIVE	01-1955	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	275	2	Open: SEPTEMBER Close: JUNE	
425	2356010	3M TILTON	1 PAPER TRAIL	MAINVILLE RD	TILTON	ACTIVE	01-1947	NON-TRANSIENT NON-COMMUI	INDUSTRIAL FACILITY	45	1	Open: JANUARY Close: DECEMBER	
426	2356020	TOWN LINE PLAZA	630 W MAIN ST	RTE 3	TILTON	ACTIVE	01-1975	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	86	9	Open: JANUARY Close: DECEMBER	
427	2356060	TANGER OUTLET CENTER	RTE 3		TILTON	ACTIVE	07-1994	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	400	57	Open: JANUARY Close: DECEMBER	
428	2356080	BJS WHOLESALE CLUB 309	RTE 3 11	119 LACONIA RD	TILTON	ACTIVE	05-1996	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	50	1	Open: JANUARY Close: DECEMBER	
429	2356100	AUTOSERV OF TILTON	40 E MAIN ST		TILTON	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	120	1	Open: JANUARY Close: DECEMBER	
430	2356110	EVERSOURCE ENERGY TILTON AWC	64 BUSINESS PARK RD		TILTON	ACTIVE	12-2004	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	52	1	Open: JANUARY Close: DECEMBER	
431	2375010	TUFTONBORO CENTRAL SCH	205 MIDDLE RD	RTE 109A	TUFTONBORO	ACTIVE	01-1938	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	150	1	Open: SEPTEMBER Close: JUNE	
432	2385010	UNITY ELEMENTARY SCH	864 2ND NH TURNPIKE		UNITY	ACTIVE	01-1954	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	150	1	Open: SEPTEMBER Close: JUNE	
433	2395020	LEARNING WITH LOVE	16 WINDY HOLLOW RD		WAKEFIELD	ACTIVE	02-2017	NON-TRANSIENT NON-COMMUI	DAY CARE	25	1	Open: JANUARY Close: DECEMBER	
434	2395030	PINE RIVER POND CNTRY DAYCARE	556 PINE RIVER POND RD		WAKEFIELD	ACTIVE	05-2019	NON-TRANSIENT NON-COMMUI	DAY CARE	25	1	Open: JANUARY Close: DECEMBER	
435	2405010	DREWSVILLE CARRIAGE HOUSE	4 COMMON RD		WALPOLE	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	31	6	Open: JANUARY Close: DECEMBER	
436	2406010	BENSON WOODWORKING	6 BLACKJACK CROSSING RD		WALPOLE	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	80	2	Open: JANUARY Close: DECEMBER	
437	2415010	NORTHEAST CATHOLIC COLLEGE	511 KEARSARGE MOUNTAIN RD		WARNER	ACTIVE	03-1991	NON-TRANSIENT NON-COMMUI	INSTITUTION, REHAB FACILITIES	105	7	Open: JANUARY Close: DECEMBER	
438	2435020	WASHINGTON ELEMENTARY SCH	337 MILLEN POND RD	RTE 31	WASHINGTON	ACTIVE	01-1992	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	125	3	Open: JANUARY Close: DECEMBER	
439	2455020	WEARE MIDDLE SCHOOL	RTE 114		WEARE	INACTIVE	01-1948	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	675	2	Open: Close:	
440	2455040	JOHN STARK REG HIGH SCH	618 N STARK HWY	RTE 114	WEARE	ACTIVE	10-1986	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	825	1	Open: JANUARY Close: DECEMBER	
441	2455060	CENTER WOOD ELEMENTARY SCH	14 CENTER RD	RTE 114N	WEARE	ACTIVE	04-1990	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	611	1	Open: SEPTEMBER Close: JUNE	
442	2455080	WEARE MIDDLE SCH	16 EAST ST		WEARE	ACTIVE	08-2007	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	500	1	Open: JANUARY Close: DECEMBER	
443	2455100	WEARE VILLAGE KIDZ ACADEMY	92 WOODBURY RD		WEARE	ACTIVE	01-2020	NON-TRANSIENT NON-COMMUI	DAY CARE	76	1	Open: JANUARY Close: DECEMBER	
444	2456010	GRANITE STATE TELEPHONE	600 S STARK HWY		WEARE	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	50	2	Open: JANUARY Close: DECEMBER	
445	2458070	LANCTOTS CENTER	RTE 114		WEARE	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	64	7	Open: JANUARY Close: DECEMBER	
446	2459010	TOWN OFFICES	15 FLANDERS MEMORIAL DR		WEARE	ACTIVE	01-1930	NON-TRANSIENT NON-COMMUI	DAY CARE	65	2	Open: JANUARY Close: DECEMBER	
447	2465010	WEBSTER ELEMENTARY SCH	936 BATTLE ST		WEBSTER	ACTIVE	01-1987	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	100	1	Open: JANUARY Close: DECEMBER	
448	2475010	WENTWORTH ELEMENTARY SCH	1247 MOUNT MOOSILAUKE H\	RTE 25	WENTWORTH	ACTIVE	01-1971	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	83	1	Open: SEPTEMBER Close: JUNE	
449	2495030	WESTMORELAND ELEMENTARY SCH	40 GLEBE RD	RTE 63	WESTMORELA	ACTIVE	08-1960	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	167	1	Open: SEPTEMBER Close: JUNE	
450	2505010	WHITE MTN REGIONAL HS	127 REGIONAL RD	RTE 3	WHITEFIELD	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	453	1	Open: SEPTEMBER Close: JUNE	
451	2525010	HIGH MOWING SCH	222 ISAAC FRYE HWY	RTE 101/ABBOT	WILTON	ACTIVE	01-1943	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	163	9	Open: JANUARY Close: DECEMBER	
452	2525020	PINE HILL WALDORF SCH	77 PINE HILL DR	ABBOTT HILL RD	WILTON	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	150	1	Open: SEPTEMBER Close: JUNE	
453	2525030	PINE HILL WALDORF CHLD VILLAGE	77 PINE HILL RD		WILTON	ACTIVE	01-2013	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	40	2	Open: JANUARY Close: DECEMBER	
454	2526040	KIMBALL PHYSICS	311 KIMBALL HILL RD		WILTON	ACTIVE	11-2016	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	60	3	Open: JANUARY Close: DECEMBER	
455	2545010	WINDHAM CENTER SCH	2 LOWELL RD		WINDHAM	ACTIVE	09-1960	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	535	1	Open: SEPTEMBER Close: JUNE	
456	2545020	GOLDEN BROOK SCH	112B LOWELL RD		WINDHAM	ACTIVE	09-1971	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	1175	1	Open: SEPTEMBER Close: JUNE	
457	2545030	WINDHAM MIDDLE SCH	112A LOWELL RD		WINDHAM	ACTIVE	09-1986	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	620	1	Open: SEPTEMBER Close: JUNE	
458	2545040	WINDHAM CROSSING LEARNING CTR	125 N LOWELL RD		WINDHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	77	2	Open: JANUARY Close: DECEMBER	
459	2545080	KIDDIE ACADEMY	156 HAVERHILL RD		WINDHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	138	1	Open: JANUARY Close: DECEMBER	
460	2545090	WINDHAM COOPTVE KINDERGARTEN	12 INDUSTRIAL WAY		WINDHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	90	1	Open: JANUARY Close: DECEMBER	
461	2545100	WINDHAM HIGH SCH	64 LONDON BRIDGE RD		WINDHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	1076	1	Open: JANUARY Close: DECEMBER	
462	2545110	WEE CARE LEARNING CTR	21 ROULSTON RD		WINDHAM	ACTIVE	09-2019	NON-TRANSIENT NON-COMMUI	DAY CARE	85	1	Open: JANUARY Close: DECEMBER	
463	2546020	AP DAILEY	6 LEDGE RD		WINDHAM	ACTIVE	09-2017	NON-TRANSIENT NON-COMMUI	INSTITUTION, REHAB FACILITIES	45	1	Open: JANUARY Close: DECEMBER	
464	2546030	WINDHAM PLAZA	4 COBBETTS POND RD		WINDHAM	ACTIVE	01-1966	NON-TRANSIENT NON-COMMUI	DAY CARE	112	16	Open: JANUARY Close: DECEMBER	
465	2546070	WINDHAM ACADEMY	1 INDUSTRIAL DR		WINDHAM	ACTIVE	05-1983	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	30	1	Open: JANUARY Close: DECEMBER	
466	2546080	CASTLE COMMONS	49 RANGE RD	RTE 111	WINDHAM	ACTIVE	01-1981	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	125	2	Open: JANUARY Close: DECEMBER	
467	2546110	WINDHAM VILLAGE GREEN	33 INDIAN ROCK RD	RTE 111	WINDHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	DAY CARE	150	17	Open: JANUARY Close: DECEMBER	
468	2546150	COBBETTS PROFESSIONAL PARK	31 LOWELL RD	W COBBETT PON	WINDHAM	ACTIVE	01-1988	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	49	2	Open: JANUARY Close: DECEMBER	
469	2546160	COMMONS AT WINDHAM 2/EAST BLD	25 INDIAN ROCK RD	RTE 111	WINDHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	50	7	Open: JANUARY Close: DECEMBER	
470	2546170	CAPITAL TRANSPORTATION	7 WALL ST		WINDHAM	ACTIVE	07-2001	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	45	5	Open: JANUARY Close: DECEMBER	
471	2546180	KAHUNA	10 INDUSTRIAL DR		WINDHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	30	2	Open: JANUARY Close: DECEMBER	
472	2546190	SHAWS SUPERMARKET 686	43 INDIAN ROCK RD		WINDHAM	ACTIVE		NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	100	1	Open: JANUARY Close: DECEMBER	
473	2546200	CYR LUMBER	39 ROCKINGHAM RD		WINDHAM	ACTIVE	06-2002	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	60	1	Open: JANUARY Close: DECEMBER	
474	2546220	FIVE INDUSTRIAL DR	5 INDUSTRIAL DR		WINDHAM	ACTIVE	03-2011	NON-TRANSIENT NON-COMMUI	DAY CARE	49	1	Open: JANUARY Close: DECEMBER	
475	2546230	MEDICUS OFFICES	22 ROULSTON RD		WINDHAM	ACTIVE	12-2012	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	300	3	Open: JANUARY Close: DECEMBER	
476	2546240	183 ROCKINGHAM ROAD	183 ROCKINGHAM RD		WINDHAM	ACTIVE	06-2015	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	40	1	Open: JANUARY Close: DECEMBER	
477	2546250	GATEWAY PARK	55 RANGE RD		WINDHAM	ACTIVE	04-2019	NON-TRANSIENT NON-COMMUI	COMMERCIAL PROPERTY	10	1	Open: JANUARY Close: DECEMBER	
478	2547060	WINDHAM WOODS SCHOOL	39 ROULSTON RD		WINDHAM	ACTIVE	05-2014	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	99	1	Open: JANUARY Close: DECEMBER	
479	2557060	WEDIKO KITCHEN	167 BLACK POND RD	RTE 31 N	WINDSOR	ACTIVE	06-1990	NON-TRANSIENT NON-COMMUI	SCHOOLS (PUBLIC, PRIVATE DAY SCHOOLS)	125	5	Open: JANUARY Close: DECEMBER	
480													
481									**Total Population Served**	84706			

EXHIBIT D

	A	B	C	D	E	F	G	H	I	J	K	L
1	PWS_ID	SYSTEM_NAME	ADDRESS_1	ADDRESS_2	TOWN	ACTIVITY_STATUS	STARTUP_DATE	SYSTEM_TYPE	SYSTEM_CATEGORY	POPULATION_SERVED	CONNECTIONS	SEASON
2	0052010	PAPERMILL VILLAGE	49 PLEASANT ST		ALSTEAD	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	24	20	
3	0072090	CONNOR COURT	1 SMITH LN		AMHERST	ACTIVE	04-2017	COMMUNITY SYSTEM	SENIOR HOUSING	51	28	
4	0262040	COTTAGES AT WINDCHIMES	10 ALBIN RD		BOW	ACTIVE	06-2002	COMMUNITY SYSTEM	SENIOR HOUSING	75	30	
5	0262050	PEU/WHITE ROCK SENIOR LIVING	4 BOW CENTER RD		BOW	ACTIVE	12-2002	COMMUNITY SYSTEM	SENIOR HOUSING	300	43	
6	0262060	PEU/STONE SLED FARM	STONE SLED LN		BOW	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	82	41	
7	0282010	MILL POND CROSSING	403 SOUTH RD		BRENTWOOD	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	45	48	
8	0583020	IRON WHEEL MHP	52 IRON WHEEL		DANVILLE	ACTIVE	01-1983	COMMUNITY SYSTEM	SENIOR HOUSING	124	81	
9	0594030	SHERBURN WOODS	1 UPHAM DR	CHURCH ST	DEERFIELD	ACTIVE	06-2003	COMMUNITY SYSTEM	SENIOR HOUSING	50	20	
10	0771020	KINGS GRANT	MAPLE ST	RTE 28	EPSOM	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	71	47	
11	0774030	MEADOW BROOK	464 SUNCOOK VALLEY HWY	RTE 28	EPSOM	ACTIVE	02-1999	COMMUNITY SYSTEM	SENIOR HOUSING	75	50	
12	0872010	GOVERNORS FOREST	90 MAIN ST		FREMONT	ACTIVE	09-2004	COMMUNITY SYSTEM	SENIOR HOUSING	69	37	
13	0872020	BLACKROCKS VILLAGE	16 HOYT WAY		FREMONT	ACTIVE	04-2011	COMMUNITY SYSTEM	SENIOR HOUSING	226	113	
14	1042010	MEADOWS AT GRAPEVINE RUN	27 BROWN RD		HAMPTON FALLS	ACTIVE	09-2017	COMMUNITY SYSTEM	SENIOR HOUSING	36	24	
15	1172020	RUNNELLS LANDING	RTE 111	RUNNELLS BRIDGE RD	HOLLIS	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	74	49	
16	1272010	KINGSTON PINES ELDERLY HOUSING	11 SCOTLAND RD		KINGSTON	ACTIVE	01-1981	COMMUNITY SYSTEM	SENIOR HOUSING	125	50	
17	1272030	LAMPLIGHTER ESTATES	2 LANTURN LN	OFF OF SCOTLAND RD	KINGSTON	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	140	56	
18	1272040	ROWELL ESTATES	ASH DR		KINGSTON	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	76	40	
19	1272070	KINGS LANDING	2 MONARCH WAY		KINGSTON	ACTIVE	11-2015	COMMUNITY SYSTEM	SENIOR HOUSING	65	43	
20	1272080	ALL AMERICAN ASSISTED LIVING	193 MAIN ST		KINGSTON	ACTIVE	06-2019	COMMUNITY SYSTEM	SENIOR HOUSING	112	58	
21	1402020	VILLAGES AT LOUDON	20 IRIS LN		LOUDON	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	250	100	
22	1402030	VOA/NNE SENIOR HOUSING	142 S VILLAGE RD		LOUDON	ACTIVE	04-2008	COMMUNITY SYSTEM	SENIOR HOUSING	50	33	
23	1462040	SILVER LAKE LANDING SR HOUSING	1420 VILLAGE RD		MADISON	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	25	20	
25	1652050	NEWBURY ELDERLY HOUSING	NEWBURY HEIGHTS RD		NEWBURY	ACTIVE	10-2014	COMMUNITY SYSTEM	SENIOR HOUSING	51	34	
26	1692030	MANSFIELD WOODS	MANSFIELD WOODS WAY		NEW HAMPTON	ACTIVE	07-2012	COMMUNITY SYSTEM	SENIOR HOUSING	66	44	
27	1752070	SARGENT WOODS	26 SMITH CORNER RD		NEWTON	ACTIVE	05-2010	COMMUNITY SYSTEM	SENIOR HOUSING	230	116	
28	1792040	VILLAGE AT MEAD FIELD	617 FIRST NH TPKE		NORTHWOOD	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	20	13	
29	1792050	THE MEADOW AT NORTHWOOD	243 BOW ST		NORTHWOOD	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	31	1	
30	1852100	BOULDER HILLS	31 HILLCREST LN		PELHAM	ACTIVE	03-2011	COMMUNITY SYSTEM	SENIOR HOUSING	39	24	
31	1852110	PARADISE ESTATES CONDOMINIUM	5 JUNIPER LN		PELHAM	ACTIVE	09-2013	COMMUNITY SYSTEM	SENIOR HOUSING	47	31	
32	1852140	LONG POND WOODS	14 SAGEWOOD DR		PELHAM	ACTIVE	06-2016	COMMUNITY SYSTEM	SENIOR HOUSING	45	30	
33	1932240	THE RESERVE AT SNOWS BROOK	24 AUGUSTA DR		PLAISTOW	ACTIVE	11-2014	COMMUNITY SYSTEM	SENIOR HOUSING	85	35	
34	1932250	CHANDLER PLACE APTS	18 CHANDLER AVE		PLAISTOW	ACTIVE	02-2017	COMMUNITY SYSTEM	SENIOR HOUSING	44	39	
35	1972070	PEU/CLEARWATER ESTATES	263 RTE 27		RAYMOND	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	78	52	
36	1972080	BLACKSTONE RESERVE	61 LANE RD		RAYMOND	ACTIVE	12-2019	COMMUNITY SYSTEM	SENIOR HOUSING	10	6	
37	1992070	PAYSON VILLAGE	60 PAYSON HILL RD		RINDGE	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	54	24	
38	2004010	INN AT SECRETARIAT ESTATES	118 SECRETRIAT WAY	WASHINGTON ST/RTE 202	ROCHESTER	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	54	33	
39	2082070	MILL PINE VILLAGE	WOODBURY RD	NORTH RD	SANDOWN	ACTIVE	03-2003	COMMUNITY SYSTEM	SENIOR HOUSING	157	92	
40	2082100	AUTUMN HILLS	1 AUTUMN LN		SANDOWN	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	8	4	
41	2232190	VINEYARDS	2 CHANCELLOR DRIVE		STRATHAM	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	111	76	
42	2232210	ROLLINS HILL	2 KIRKWALL DR		STRATHAM	ACTIVE	07-2019	COMMUNITY SYSTEM	SENIOR HOUSING	23	15	
43	2312050	REMICK ACRES	RTE 113		TAMWORTH	ACTIVE	01-1981	COMMUNITY SYSTEM	SENIOR HOUSING	60	24	
44	2352040	WINNISQUAM VILLAGE CONDOS	CARDIGAN CT	OFF RTE 3	TILTON	ACTIVE	09-2007	COMMUNITY SYSTEM	SENIOR HOUSING	33	22	
45	2542130	MCAULEY COMMONS	37 SEARLES RD		WINDHAM	ACTIVE	03-2001	COMMUNITY SYSTEM	SENIOR HOUSING	25	24	
46	2542160	HADLEIGH WOODS	22 HADLEIGH RD		WINDHAM	ACTIVE		COMMUNITY SYSTEM	SENIOR HOUSING	93	62	
47	2563010	BIRCH HILL ESTATES	RTE 28		WOLFEBORO	ACTIVE	01-1985	COMMUNITY SYSTEM	SENIOR HOUSING	159	106	
48												
49									Total Population Served	3668		

EXHIBIT E

	A	B	C	D	E	F	G	H	I	J	K	L	M	N
1	PWS_ID	SYSTEM_NAME	ADDRESS_1	ADDRESS_2	TOWN	ACTIVITY_STATUS	STARTUP_DATE	SYSTEM_TYPE	SYSTEM_ CATEGORY	POPULATION_SERVED	CONNECTIONS	SEASON		
2	0055030	ORCHARD SCHOOL	114 OLD SETTLERS RD	E ALSTEAD	ALSTEAD	ACTIVE	09-1994	NON-TRANSIENT NON-COMMU	DAY CARE	45	1	Open: JANUARY Close: DECEMBER		
3	0075070	CHRISTS CHURCH OF AMHERST	58 MERRIMACK RD		AMHERST	ACTIVE	01-2020	NON-TRANSIENT NON-COMMU	DAY CARE	28	1	Open: JANUARY Close: DECEMBER		
4	0115060	LEARNING PATH CHILD CARE CTR	72 RTE 111		ATKINSON	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	112	1	Open: JANUARY Close: DECEMBER		
5	0135020	AUBURN CHILDRENS HOUSE	78 ROCKINGHAM RD		AUBURN	ACTIVE	08-1990	NON-TRANSIENT NON-COMMU	DAY CARE	30	1	Open: JANUARY Close: DECEMBER		
6	0135030	ITS A CHILDS WORLD	32 HOOKSETT RD		AUBURN	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	51	1	Open: JANUARY Close: DECEMBER		
7	0146040	BARNSTEAD BUSINESS PARK	27 DEPOT RD		BARNSTEAD	ACTIVE	09-2017	NON-TRANSIENT NON-COMMU	DAY CARE	52	2	Open: JANUARY Close: DECEMBER		
8	0155090	STORYBOOK HOLLOW ELP	41 COMMERCE WAY		BARRINGTON	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	30	1	Open: JANUARY Close: DECEMBER		
9	0155100	BARRINGTON VLG ENRICHMENT CTR	45 COMMERCE WAY		BARRINGTON	ACTIVE	01-2013	NON-TRANSIENT NON-COMMU	DAY CARE	58	1	Open: JANUARY Close: DECEMBER		
10	0195010	MONTESSORI SCH OF BEDFORD	24 TIRRELL HILL RD		BEDFORD	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	40	1	Open: SEPTEMBER Close: JUNE		
11	0195090	BETHANY COVENANT CHURCH	1 COVENANT WAY		BEDFORD	ACTIVE	06-1991	NON-TRANSIENT NON-COMMU	DAY CARE	79	1	Open: JANUARY Close: DECEMBER		
12	0195100	BEDFORD VILLAGE MORNING SCH	19 MINISTERIAL RD		BEDFORD	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	60	2	Open: SEPTEMBER Close: JUNE		
13	0196280	292 ROUTE 101	292 RTE 101		BEDFORD	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	25	1	Open: JANUARY Close: DECEMBER		
14	0205010	LAKES REGION DAYCARE	24 EASTGATE PARK DR		BELMONT	ACTIVE	04-2015	NON-TRANSIENT NON-COMMU	DAY CARE	67	1	Open: JANUARY Close: DECEMBER		
15	0265040	JOYFUL NOISE PRESCH	6 BRANCH LONDONDERRY TPKE EAST		BOW	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	72	1	Open: JANUARY Close: DECEMBER		
16	0265050	BOW YOUTH CENTER	21 BOW CTR RD		BOW	ACTIVE	08-2008	NON-TRANSIENT NON-COMMU	DAY CARE	150	1	Open: JANUARY Close: DECEMBER		
17	0266110	BOW TECHNOLOGIES CENTER	3 ROBINSON RD		BOW	ACTIVE	07-1986	NON-TRANSIENT NON-COMMU	DAY CARE	34	2	Open: JANUARY Close: DECEMBER		
18	0266140	NH AUTO DEALERS ASSN	507 SOUTH ST		BOW	ACTIVE	06-1986	NON-TRANSIENT NON-COMMU	DAY CARE	104	1	Open: JANUARY Close: DECEMBER		
19	0269010	BOW MUNICIPAL BUILDING	10 GRANDVIEW RD		BOW	ACTIVE	01-1927	NON-TRANSIENT NON-COMMU	DAY CARE	70	1	Open: JANUARY Close: DECEMBER		
20	0269040	JOYFUL NOISE LEARNING CENTER	8 BRANCH LONDONDERRY TPKE EAST		BOW	ACTIVE	07-2018	NON-TRANSIENT NON-COMMU	DAY CARE	25	1	Open: JANUARY Close: DECEMBER		
21	0275090	CHILDRENS CTR FOR CREATVE LRNG	57 W MAIN ST		BRADFORD	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	40	1	Open: JANUARY Close: DECEMBER		
22	0285040	A PLACE TO GROW	436 RTE 125		BRENTWOOD	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	45	1	Open: JANUARY Close: DECEMBER		
23	0305020	PB AND JS FAMILY CHILDCARE	1420 PEAKED HILL RD		BRISTOL	ACTIVE	08-2017	NON-TRANSIENT NON-COMMU	DAY CARE	40	2	Open: JANUARY Close: DECEMBER		
24	0325060	STONEY LEDGE PLAZA	181 RTE 13		BROOKLINE	ACTIVE	12-2019	NON-TRANSIENT NON-COMMU	DAY CARE	70	2	Open: JANUARY Close: DECEMBER		
25	0365020	M AND C CHILDRENS LEARNING PL	205 MAIN ST	OFF RTE 43 AND RTE 101	CANDIA	ACTIVE	08-1984	NON-TRANSIENT NON-COMMU	DAY CARE	75	1	Open: JANUARY Close: DECEMBER		
26	0435070	PLAY LAUGH N GROW	184 RAYMOND RD		CHESTER	ACTIVE	01-2015	NON-TRANSIENT NON-COMMU	DAY CARE	40	1	Open: JANUARY Close: DECEMBER		
27	0435080	CHESTERBROOK SCH OF NTRL LRNG	232 FREMONT RD		CHESTER	ACTIVE	09-2018	NON-TRANSIENT NON-COMMU	DAY CARE	28	2	Open: JANUARY Close: DECEMBER		
28	0455030	KELLEY CORNER SCHOOL	67 KELLEYS CORNER RD		CHICHESTER	ACTIVE	11-2019	NON-TRANSIENT NON-COMMU	DAY CARE	28	1	Open: JANUARY Close: DECEMBER		
29	0515040	LITTLE HANDS BIG DREAMS	90 ODELL HILL RD	CENTER CONWAY	CONWAY	ACTIVE	09-2015	NON-TRANSIENT NON-COMMU	DAY CARE	42	1	Open: JANUARY Close: DECEMBER		
30	0615070	OVER THE RAINBOW PRESCH	223 ROCKINGHAM RD	RTE 28S	DERRY	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	78	1	Open: SEPTEMBER Close: JUNE		
31	0655030	MISS PATTYS CHILDCARE	49 PISCATAQUA RD		DOVER	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	36	1	Open: JANUARY Close: DECEMBER		
32	0708040	POWWOW JUNCTION	14 POWWOW RIVER RD		EAST KINGSTON	ACTIVE	07-2013	NON-TRANSIENT NON-COMMU	DAY CARE	63	2	Open: JANUARY Close: DECEMBER		
33	0805010	BUILDING BLOCK COMMONS	125 KINGSTON RD	RTE 111	EXETER	ACTIVE	10-1979	NON-TRANSIENT NON-COMMU	DAY CARE	40	1	Open: SEPTEMBER Close: JUNE		
34	0875030	COUNTRY CLUB FOR KIDS	50 MAIN ST	RTE 107	FREMONT	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	61	2	Open: JANUARY Close: DECEMBER		
35	0875040	BARNYARD BUDDIES	83 CHESTER RD		FREMONT	ACTIVE	09-2002	NON-TRANSIENT NON-COMMU	DAY CARE	40	1	Open: JANUARY Close: DECEMBER		
36	0885050	GILFORD PROFESSIONAL PARK	401 GILFORD AVE		GILFORD	ACTIVE	07-2018	NON-TRANSIENT NON-COMMU	DAY CARE	135	3	Open: JANUARY Close: DECEMBER		
37	0886040	UNDER HIS WINGS PRESCHOOL	2 AIRPORT RD		GILFORD	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	110	1	Open: JANUARY Close: DECEMBER		
38	0915020	LEARN AS WE PLAY CHLD CARE CTR	276 GOFFSTOWN BACK RD		GOFFSTOWN	ACTIVE	05-1997	NON-TRANSIENT NON-COMMU	DAY CARE	94	1	Open: JANUARY Close: DECEMBER		
39	0956040	GRANTHAM GREENWAY	151 RTE 10 N		GRANTHAM	ACTIVE	07-2016	NON-TRANSIENT NON-COMMU	DAY CARE	200	2	Open: JANUARY Close: DECEMBER		
40	1035020	AGES AND STAGES HAMPSTEAD	499 MAIN ST		HAMPSTEAD	ACTIVE	08-1984	NON-TRANSIENT NON-COMMU	DAY CARE	99	1	Open: JANUARY Close: DECEMBER		
41	1035110	HAZEL DRIVE KIDS	35 HAZEL DR		HAMPSTEAD	ACTIVE	11-2011	NON-TRANSIENT NON-COMMU	DAY CARE	34	1	Open: JANUARY Close: DECEMBER		
42	1046050	CRN REALTY	105 LAFAYETTE RD		HAMPTON FALLS	ACTIVE	12-1986	NON-TRANSIENT NON-COMMU	DAY CARE	190	7	Open: JANUARY Close: DECEMBER		
43	1075010	HAMPSHIRE COOP NURSERY SCH	104 LYME RD	RTE 10	HANOVER	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	32	2	Open: JANUARY Close: DECEMBER		
44	1075020	MONTESSORI CHILDRENS SCH	67 TRESCOTT RD		HANOVER	ACTIVE	03-1999	NON-TRANSIENT NON-COMMU	DAY CARE	51	1	Open: JANUARY Close: DECEMBER		
45	1085020	CHESHIRE MILLS BOARDING HOUSE	66 MAIN ST		HARRISVILLE	ACTIVE	01-1953	NON-TRANSIENT NON-COMMU	DAY CARE	44	1	Open: JANUARY Close: DECEMBER		
46	1165010	WHITE MOUNTAIN MONTESSORI SCH	133 MT PROSPECT RD		HOLDERNESS	ACTIVE	05-2015	NON-TRANSIENT NON-COMMU	DAY CARE	30	1	Open: JANUARY Close: DECEMBER		
47	1167190	SLNSC BLUE HERON SCH	25 SCIENCE CENTER DR	RTE 113	HOLDERNESS	ACTIVE	05-2010	NON-TRANSIENT NON-COMMU	DAY CARE	41	2	Open: JANUARY Close: DECEMBER		
48	1185010	PLACES YOULL GROW	167 LONDONDERRY TPKE RD		HOOKSETT	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	30	2	Open: JANUARY Close: DECEMBER		
49	1186010	198 LONDONDERRY TURNPIKE	198 LONDONDERRY TPKE		HOOKSETT	ACTIVE	06-1986	NON-TRANSIENT NON-COMMU	DAY CARE	98	1	Open: JANUARY Close: DECEMBER		
50	1205060	KIDDIE KONNECTION	301 DERRY RD	RTE 102	HUDSON	ACTIVE	04-1995	NON-TRANSIENT NON-COMMU	DAY CARE	191	1	Open: JANUARY Close: DECEMBER		
51	1205070	EARLY START LEARNING ACADEMY	141 KIMBALL HILL RD		HUDSON	ACTIVE	08-1999	NON-TRANSIENT NON-COMMU	DAY CARE	79	1	Open: JANUARY Close: DECEMBER		
52	1279010	KINGSTON CHILDREN CENTER	12 CHURCH ST		KINGSTON	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	71	1	Open: JANUARY Close: DECEMBER		
53	1289010	PRESCOTT FARM ENVIRON ED CTR	928 WHITE OAKS RD		LACONIA	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	50	1	Open: JANUARY Close: DECEMBER		
54	1335020	LIVE AND LEARN DAY CARE	114 MAST RD	RTE 155	LEE	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	45	1	Open: JANUARY Close: DECEMBER		
55	1335030	GROWING PLACES	56 PINKHAM RD		LEE	ACTIVE	01-1965	NON-TRANSIENT NON-COMMU	DAY CARE	47	2	Open: JANUARY Close: DECEMBER		
56	1339010	LEE CONGREGATIONAL CHURCH	17 MAST RD	RTE 155	LEE	ACTIVE	01-1980	NON-TRANSIENT NON-COMMU	DAY CARE	34	2	Open: JANUARY Close: DECEMBER		
57	1378070	LOBSTER BOAT PLZ	273 DERRY RD		LITCHFIELD	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	70	9	Open: JANUARY Close: DECEMBER		
58	1395090	CREATIVE LITTLE ANGELS	40 MAMMOTH RD	RTE 128	LONDONDERRY	ACTIVE	01-1973	NON-TRANSIENT NON-COMMU	DAY CARE	167	1	Open: JANUARY Close: DECEMBER		
59	1395110	28 BUTTRICK RD PROPERTY	28 BUTTRICK RD		LONDONDERRY	ACTIVE	06-1986	NON-TRANSIENT NON-COMMU	DAY CARE	83	1	Open: JANUARY Close: DECEMBER		
60	1435050	LYME NURSERY SCHL	155 DARTMOUTH COLLEGE HWY		LYME	ACTIVE	06-2005	NON-TRANSIENT NON-COMMU	DAY CARE	36	1	Open: JANUARY Close: DECEMBER		

	A	B	C	D	E	F	G	H	I	J	K	L	M	N
61	1458010	LITTLE TREE ED CENTER	316 RTE 108		MADBURY	ACTIVE	09-1997	NON-TRANSIENT NON-COMMU	DAY CARE	60	1	Open: JANUARY Close: DECEMBER		
62	1565010	LITTLE ARROWS CHILD CARE SVS	365 SOUTH ST	RTE 13	MILFORD	ACTIVE	07-1990	NON-TRANSIENT NON-COMMU	DAY CARE	52	1	Open: JANUARY Close: DECEMBER		
63	1585030	MILTON CHILDRENS CENTER	55 INDUSTRIAL WAY		MILTON	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	25	1	Open: JANUARY Close: DECEMBER		
64	1615030	A CHILDS PLACE	903 WHITTIER HWY	RTE 25	MOULTONBOROU	ACTIVE	09-1991	NON-TRANSIENT NON-COMMU	DAY CARE	59	1	Open: JANUARY Close: DECEMBER		
65	1645020	STRONG FOUNDATIONS	643 NORTH MAST RD	RTE 114	NEW BOSTON	ACTIVE	01-1982	NON-TRANSIENT NON-COMMU	DAY CARE	139	1	Open: JANUARY Close: DECEMBER		
66	1645030	CHESTNUT CHRISTIAN PRESCHOOL	219 CHESTNUT HILL RD		NEW BOSTON	ACTIVE	12-2011	NON-TRANSIENT NON-COMMU	DAY CARE	34	1	Open: JANUARY Close: DECEMBER		
67	1675020	7 DEPOT RD/BEING COOL	7 DEPOT RD		NEW DURHAM	ACTIVE	02-2020	NON-TRANSIENT NON-COMMU	DAY CARE	45	1	Open: JANUARY Close: DECEMBER		
68	1749010	KIDS WORLD ACADEMY OF NH	660 JOHN STARK HWY		NEWPORT	ACTIVE	06-2014	NON-TRANSIENT NON-COMMU	DAY CARE	50	2	Open: JANUARY Close: DECEMBER		
69	1755050	NEWTON LEARNING CENTER	31 S MAIN ST	RTE 108	NEWTON	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	82	1	Open: JANUARY Close: DECEMBER		
70	1805060	ALL ABOARD PRESCH AND CHLDCAR	249 STAGE RD	RTE 152	NOTTINGHAM	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	86	2	Open: JANUARY Close: DECEMBER		
71	1809020	NOTTINGHAM COMMUNITY CHURCH	106 CHURCH ST		NOTTINGHAM	ACTIVE	04-2016	NON-TRANSIENT NON-COMMU	DAY CARE	28	1	Open: JANUARY Close: DECEMBER		
72	1855060	PERFECT PLACE FOR CHILDREN	125 MAIN ST UNIT A	OFF RTE 38N	PELHAM	ACTIVE	01-1950	NON-TRANSIENT NON-COMMU	DAY CARE	54	5	Open: JANUARY Close: DECEMBER		
73	1855070	DYNAMIC FOUNDATION FOR CHLDRE	90 BRIDGE ST		PELHAM	ACTIVE	11-2001	NON-TRANSIENT NON-COMMU	DAY CARE	60	1	Open: JANUARY Close: DECEMBER		
74	1855080	COLONIAL WAY PLAZA	43 BRIDGE ST		PELHAM	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	28	1	Open: JANUARY Close: DECEMBER		
75	1855090	KINGS KIDS CHILD CARE CENTER	955 BRIDGE ST	RTE 38	PELHAM	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	100	1	Open: JANUARY Close: DECEMBER		
76	1859040	CROSSROADS BAPTIST CHURCH	43 ATWOOD RD		PELHAM	ACTIVE	08-1978	NON-TRANSIENT NON-COMMU	DAY CARE	50	1	Open: JANUARY Close: DECEMBER		
77	1875010	HAPPY VALLEY SCHOOL	130 GULF RD		PETERBOROUGH	ACTIVE	09-1970	NON-TRANSIENT NON-COMMU	DAY CARE	40	1	Open: JANUARY Close: DECEMBER		
78	1875030	MONADNOCK COMM EARLY LNG CTR	5 COMMUNITY LN		PETERBOROUGH	ACTIVE	11-1980	NON-TRANSIENT NON-COMMU	DAY CARE	80	1	Open: JANUARY Close: DECEMBER		
79	1935070	LITTLE EXPLORERS	3 BLOSSOM RD		PLAISTOW	ACTIVE	08-1997	NON-TRANSIENT NON-COMMU	DAY CARE	44	1	Open: JANUARY Close: DECEMBER		
80	1938090	BRICKYARD I PLAZA	95 PLAISTOW RD	RTE 125	PLAISTOW	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	80	19	Open: JANUARY Close: DECEMBER		
81	1938240	PLAISTOW COMMUNITY YMCA	175 PLAISTOW RD		PLAISTOW	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	99	1	Open: JANUARY Close: DECEMBER		
82	1949030	PLYMOUTH COMMERCE PARK	12 YEATON RD		PLYMOUTH	ACTIVE	01-2007	NON-TRANSIENT NON-COMMU	DAY CARE	43	2	Open: JANUARY Close: DECEMBER		
83	2055020	MERRIMACK VALLEY MONTESSOR SC	111 LOWELL RD		SALEM	ACTIVE	09-1967	NON-TRANSIENT NON-COMMU	DAY CARE	65	1	Open: SEPTEMBER Close: JUNE		
84	2075070	SAPLINGS PRESCHOOL	266 UPPER BAY RD		SANBORNTON	ACTIVE	10-2019	NON-TRANSIENT NON-COMMU	DAY CARE	28	2	Open: JANUARY Close: DECEMBER		
85	2085020	PLAYMATES LEARNING CENTER	56 NORTH RD	OFF RTE 121A	SANDOWN	ACTIVE	01-1976	NON-TRANSIENT NON-COMMU	DAY CARE	56	1	Open: JANUARY Close: DECEMBER		
86	2095030	SANDWICH CHILDRENS CENTER	54 MAPLE ST		SANDWICH	ACTIVE	01-1985	NON-TRANSIENT NON-COMMU	DAY CARE	48	1	Open: JANUARY Close: DECEMBER		
87	2215040	WHITEHOUSE EARLY LEARNING CTR	352 PROVINCE RD		STRAFFORD	ACTIVE	06-2019	NON-TRANSIENT NON-COMMU	DAY CARE	28	1	Open: JANUARY Close: DECEMBER		
88	2235010	ACORN SCH	136 WINNICUTT RD		STRATHAM	ACTIVE	01-1975	NON-TRANSIENT NON-COMMU	DAY CARE	54	1	Open: SEPTEMBER Close: JUNE		
89	2239010	STRATHAM COMMUNITY CHURCH	6 EMERY LN		STRATHAM	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	55	1	Open: JANUARY Close: DECEMBER		
90	2315020	TAMWORTH PRESCHOOL	27 DURRELL RD	RTE 113	TAMWORTH	ACTIVE	10-1987	NON-TRANSIENT NON-COMMU	DAY CARE	100	1	Open: JANUARY Close: DECEMBER		
91	2395020	LEARNING WITH LOVE	16 WINDY HOLLOW RD		WAKEFIELD	ACTIVE	02-2017	NON-TRANSIENT NON-COMMU	DAY CARE	25	1	Open: JANUARY Close: DECEMBER		
92	2395030	PINE RIVER POND CNTRY DAYCARE	556 PINE RIVER POND RD		WAKEFIELD	ACTIVE	05-2019	NON-TRANSIENT NON-COMMU	DAY CARE	25	1	Open: JANUARY Close: DECEMBER		
93	2405010	DREWSVILLE CARRIAGE HOUSE	4 COMMON RD		WALPOLE	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	31	6	Open: JANUARY Close: DECEMBER		
94	2455100	WEARE VILLAGE KIDZ ACADEMY	92 WOODBURY RD		WEARE	ACTIVE	01-2020	NON-TRANSIENT NON-COMMU	DAY CARE	76	1	Open: JANUARY Close: DECEMBER		
95	2458070	LANCTOTS CENTER	RTE 114		WEARE	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	64	7	Open: JANUARY Close: DECEMBER		
96	2459010	TOWN OFFICES	15 FLANDERS MEMORIAL DR		WEARE	ACTIVE	01-1930	NON-TRANSIENT NON-COMMU	DAY CARE	65	2	Open: JANUARY Close: DECEMBER		
97	2545040	WINDHAM CROSSING LEARNING CTR	125 N LOWELL RD		WINDHAM	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	77	2	Open: JANUARY Close: DECEMBER		
98	2545080	KIDDIE ACADEMY	156 HAVERHILL RD		WINDHAM	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	138	1	Open: JANUARY Close: DECEMBER		
99	2545090	WINDHAM COOPTVE KINDERGARTEN	12 INDUSTRIAL WAY		WINDHAM	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	90	1	Open: JANUARY Close: DECEMBER		
100	2545110	WEE CARE LEARNING CTR	21 ROULSTON RD		WINDHAM	ACTIVE	09-2019	NON-TRANSIENT NON-COMMU	DAY CARE	85	1	Open: JANUARY Close: DECEMBER		
101	2546030	WINDHAM PLAZA	4 COBBETTS POND RD		WINDHAM	ACTIVE	01-1966	NON-TRANSIENT NON-COMMU	DAY CARE	112	16	Open: JANUARY Close: DECEMBER		
102	2546110	WINDHAM VILLAGE GREEN	33 INDIAN ROCK RD	RTE 111	WINDHAM	ACTIVE		NON-TRANSIENT NON-COMMU	DAY CARE	150	17	Open: JANUARY Close: DECEMBER		
103	2546220	FIVE INDUSTRIAL DR	5 INDUSTRIAL DR		WINDHAM	ACTIVE	03-2011	NON-TRANSIENT NON-COMMU	DAY CARE	49	1	Open: JANUARY Close: DECEMBER		
104														
105									**Total Population Served**	6678				

EXHIBIT F

	A	B	C	D	E	F	G	H	I	J	K	L
1	PWS_ID	SYSTEM_NAME	ADDRESS_1	ADDRESS_2	TOWN	ACTIVITY_STATUS	STARTUP_DATE	SYSTEM_TYPE	SYSTEM_CATEGORY	POPULATION_SERVED	CONNECTIONS	SEASON
2	0015010	ACWORTH PRIMARY SCH	TURKEY SHOOT RD	ACWORTH RD	ACWORTH	ACTIVE	01-1932	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	32	1	Open: SEPTEMBER Close: JUNE
3	0025010	WHITE MOUNTAIN WALDORF SCH	1371 RTE 16		ALBANY	ACTIVE	09-2008	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	110	6	Open: SEPTEMBER Close: JUNE
4	0055010	ALSTEAD VILAS SCH	82 MECHANIC ST	RTE 123	ALSTEAD	ACTIVE	01-1934	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	190	2	Open: SEPTEMBER Close: JUNE
5	0055020	ALSTEAD PRIMARY SCH	58 MECHANIC ST	RTE 123	ALSTEAD	ACTIVE	06-1991	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	177	1	Open: SEPTEMBER Close: JUNE
6	0059010	MOLE HILL THEATRE/LEAF CHARTER	789 GILSUM MINE RD		ALSTEAD	ACTIVE	05-2013	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	60	2	Open: JANUARY Close: DECEMBER
7	0065020	PROSPECT MOUNTAIN HIGH SCH	242 SUNCOOK VALLEY RD	RTE 28	ALTON	ACTIVE	09-2004	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	500	1	Open: JANUARY Close: DECEMBER
8	0095010	OVERSEAS UNITED EDUC FNDTN	100 OLD NORTH BRANCH RD/RTE 9	D/B/A HAWTHORNE ACADEMY	ANTRIM	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	25	4	Open: JANUARY Close: DECEMBER
9	0115010	ATKINSON ACADEMY SCH	17 ACADEMY AVE		ATKINSON	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	429	1	Open: SEPTEMBER Close: JUNE
10	0135010	AUBURN VILLAGE SCH	11 EATON HILL RD		AUBURN	ACTIVE	03-1960	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	766	1	Open: JANUARY Close: DECEMBER
11	0145010	BARNSTEAD ELEMENTARY SCH	91 MAPLE ST		BARNSTEAD	ACTIVE	09-1957	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	646	1	Open: JANUARY Close: DECEMBER
12	0155020	EARLY CHILDHOOD LEARNING CTR	77 RAMSDELL LN		BARRINGTON	ACTIVE	09-1975	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	179	1	Open: SEPTEMBER Close: JUNE
13	0155050	BARRINGTON ELEMENTARY SCH	570 CALEF HWY		BARRINGTON	ACTIVE	01-1990	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	488	2	Open: SEPTEMBER Close: JUNE
14	0155080	BARRINGTON MIDDLE SCH	51 HALEY DR	85 RTE 9	BARRINGTON	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	510	1	Open: JANUARY Close: DECEMBER
15	0245010	PROFILE HIGH SCH	691 PROFILE RD	JCT I93/302/18/116	BETHLEHEM	ACTIVE	01-1976	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	284	1	Open: JANUARY Close: DECEMBER
16	0265010	BOW MEMORIAL SCHOOL	20 BOW CENTER RD		BOW	ACTIVE	09-1964	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	615	1	Open: SEPTEMBER Close: JUNE
17	0265020	BOW ELEMENTARY SCHOOL	22 BOW CENTER RD		BOW	ACTIVE	09-1979	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	522	1	Open: SEPTEMBER Close: JUNE
18	0265030	BOW HIGH SCHOOL	32 WHITE ROCK HILL RD	THREE POND RD	BOW	ACTIVE	09-1997	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	645	1	Open: SEPTEMBER Close: JUNE
19	0265060	MEETING HOUSE MONTESSORI	28 LOGGING HILL RD		BOW	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	58	1	Open: JANUARY Close: DECEMBER
20	0275070	KEARSARGE REG ELEM SCH/BRADFRD	163 OLD WARNER RD	OFF RTE 103	BRADFORD	ACTIVE	09-1987	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	231	1	Open: SEPTEMBER Close: JUNE
21	0285010	SWASEY CENTRAL SCH	355 MIDDLE RD	RTE 111A	BRENTWOOD	ACTIVE	01-1949	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	392	1	Open: SEPTEMBER Close: JUNE
22	0295010	BRIDGEWATER/HEBRON VIL SCH	25 SCHOOLHOUSE RD	RTE 3A	BRIDGEWATER	ACTIVE	07-1999	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	200	1	Open: JANUARY Close: DECEMBER
23	0305010	NEWFOUND REGIONAL HS	150 NEWFOUND RD	RTE 104	BRISTOL	ACTIVE	01-1988	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	500	1	Open: JANUARY Close: DECEMBER
24	0325010	RICHARD MAGHAKIAN MEMORIAL SCH	22 MILFORD ST		BROOKLINE	ACTIVE	04-1987	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	327	1	Open: SEPTEMBER Close: JUNE
25	0325050	CAPT SAMUEL DOUGLASS ACADEMY	24 TOWNSEND HILL RD	RTE 13	BROOKLINE	ACTIVE	09-2000	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	358	1	Open: SEPTEMBER Close: JUNE
26	0355060	INDIAN RIVER SCH	45 ROYAL RD	RTE 4	CANAAN	ACTIVE	09-1987	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	799	2	Open: JANUARY Close: DECEMBER
27	0365010	HENRY W MOORE SCH	12 DEERFIELD RD	RTE 43	CANDIA	ACTIVE	01-1938	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	352	1	Open: SEPTEMBER Close: JUNE
28	0365040	REMINGTON EDUCATION CENTER	15 STEVENS LN		CANDIA	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	62	2	Open: SEPTEMBER Close: JUNE
29	0375010	CANTERBURY ELEMENTARY SCH	15 BAPTIST RD		CANTERBURY	ACTIVE	01-1987	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	156	1	Open: JANUARY Close: DECEMBER
30	0419010	LIFE FLLWSHP FOURSQUARE CHURCH	85 WHEELER RAND RD		CHARLESTOWN	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	70	2	Open: JANUARY Close: DECEMBER
31	0435060	CHESTER ACADEMY	22 MURPHY DR	RTE 102	CHESTER	ACTIVE	09-1999	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	645	1	Open: SEPTEMBER Close: JUNE
32	0445010	CHESTERFIELD CENTRAL SCH	535 OLD CHESTERFIELD RD	RTE 63	CHESTERFIELD	ACTIVE	01-1950	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	322	1	Open: SEPTEMBER Close: JUNE
33	0455010	CHICHESTER CENTRAL SCH	219 MAIN ST		CHICHESTER	ACTIVE	01-1949	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	261	1	Open: SEPTEMBER Close: JUNE
34	0505010	SHAKER RD SCH & CHILDCARE CTR	95 SHAKER RD		CONCORD	ACTIVE	01-1979	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	355	6	Open: JANUARY Close: DECEMBER
35	0515020	NEW PINE TREE SCH	183 MILL ST	RTE 302E	CONWAY	ACTIVE	09-1990	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	328	1	Open: SEPTEMBER Close: JUNE
36	0525010	CORNISH ELEMENTARY SCH	274 TOWNHOUSE RD	OFF RTE 120	CORNISH	ACTIVE	01-1950	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	140	2	Open: SEPTEMBER Close: JUNE
37	0545010	CROYDON VILLAGE SCH	889 RTE 10		CROYDON	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	33	4	Open: SEPTEMBER Close: JUNE
38	0575010	DANBURY ELEMENTARY SCH	20 DAFFODIL LN	RTE 104E	DANBURY	ACTIVE	09-1979	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	85	1	Open: JANUARY Close: DECEMBER
39	0585010	DANVILLE ELEMENTARY	23 SCHOOL ST	MAIN ST/RTE 111A	DANVILLE	ACTIVE	01-1962	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	340	1	Open: SEPTEMBER Close: JUNE
40	0595020	DEERFIELD COMMUNITY SCH	66 NORTH RD	RTE 107	DEERFIELD	ACTIVE	08-1988	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	630	1	Open: SEPTEMBER Close: JUNE
41	0595040	LONGVIEW SCH	55 RESERVATION RD		DEERFIELD	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	31	1	Open: JANUARY Close: DECEMBER
42	0615060	E DERRY MEMORIAL ELEM SCH	20 DUBEAU DR	OLESEN RD	DERRY	ACTIVE	07-1986	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	488	1	Open: SEPTEMBER Close: JUNE
43	0664010	DUBLIN CHRISTIAN ACADEMY	106 PAGE RD		DUBLIN	ACTIVE	01-1964	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	155	7	Open: JANUARY Close: DECEMBER
44	0664020	DUBLIN SCHOOL	18 LEHMANN WAY	DUBLIN RD	DUBLIN	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	215	21	Open: JANUARY Close: DECEMBER
45	0665010	DUBLIN CONSOLIDATED SCH	1177 MAIN ST	RTE 101	DUBLIN	ACTIVE	01-1938	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	80	1	Open: SEPTEMBER Close: JUNE
46	0665050	MOUNTAIN SHADOWS SCH	149 VALLEY RD	OLDE PETERBROUG	DUBLIN	ACTIVE	01-1971	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	72	2	Open: SEPTEMBER Close: JUNE
47	0685010	DUNBARTON ELEMENTARY SCH	20 ROBERT ROGERS RD		DUNBARTON	ACTIVE	01-1972	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	245	1	Open: SEPTEMBER Close: JUNE
48	0705020	E KINGSTON ELEMENTARY SCH	5 ANDREWS LN	SOUTH RD	EAST KINGSTON	ACTIVE	09-1971	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	197	1	Open: JANUARY Close: DECEMBER
49	0735030	EFFINGHAM ELEMENTARY SCH	6 PARTRIDGE COVE RD		EFFINGHAM	ACTIVE	09-2003	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	128	1	Open: SEPTEMBER Close: JUNE
50	0805040	EXETER HIGH SCH	1 BLUE HAWK DR		EXETER	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	1890	1	Open: JANUARY Close: DECEMBER
51	0825010	GEORGE S EMERSON ELEM SCH	27 RHODODENDRON RD	RTE 119W	FITZWILLIAM	ACTIVE	01-1950	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	182	1	Open: SEPTEMBER Close: JUNE
52	0835010	FRANCESTOWN ELEMENTARY SCH	325 SECOND NH TPKE SOUTH		FRANCESTOWN	ACTIVE	09-1997	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	69	1	Open: SEPTEMBER Close: JUNE
53	0875010	ELLIS SCHOOL	432 MAIN ST		FREMONT	ACTIVE	01-1947	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	460	1	Open: SEPTEMBER Close: JUNE
54	0885010	GILFORD ELEMENTARY SCH	76 BELKNAP MOUNTAIN RD	RTE 11A	GILFORD	ACTIVE	09-1972	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	422	1	Open: SEPTEMBER Close: JUNE
55	0885020	GILFORD MIDDLE AND HIGH SCH	72/88 ALVAH WILSON RD	RTE 11A	GILFORD	ACTIVE	06-1974	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	958	1	Open: JANUARY Close: DECEMBER
56	0895010	GILMANTON ELEMENTARY SCH	1386 RTE 140		GILMANTON	ACTIVE	01-1965	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	494	1	Open: JANUARY Close: DECEMBER
57	0905010	GILSUM ELEMENTARY SCH	640 RTE 10		GILSUM	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	62	1	Open: SEPTEMBER Close: JUNE
58	0955010	GRANTHAM VILLAGE SCH	75 LEARNING DR	E 10/DUNBAR HILL I	GRANTHAM	ACTIVE	01-1978	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	303	1	Open: JANUARY Close: DECEMBER
59	0975020	GREENFIELD ELEMENTARY SCH	860 FOREST RD		GREENFIELD	ACTIVE	12-1999	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	109	1	Open: AUGUST Close: JUNE
60	1035040	HAMPSTEAD MIDDLE SCH	28 SCHOOL ST		HAMPSTEAD	ACTIVE	01-1978	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	552	1	Open: SEPTEMBER Close: JUNE
61	1035050	HAMPSTEAD ACADEMY	320 EAST RD	RTE 111	HAMPSTEAD	ACTIVE	08-1987	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	92	3	Open: SEPTEMBER Close: JUNE
62	1045010	LINCOLN AKERMAN SCH	8 EXETER RD	RTE 88	HAMPTON FALLS	ACTIVE	04-1974	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	281	1	Open: SEPTEMBER Close: JUNE
63	1045040	HERONFIELD ACADEMY	356 EXETER RD		HAMPTON FALLS	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	114	2	Open: JANUARY Close: DECEMBER
64	1085010	WELLS MEMORIAL SCH	235 CHESHAM RD	CHESHAM	HARRISVILLE	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	60	1	Open: SEPTEMBER Close: JUNE

	A	B	C	D	E	F	G	H	I	J	K	L
65	1105010	OLIVERIAN EAST CAMPUS	2634 MT MOOSILAUKE HWY	RTE 25/PIKE	HAVERHILL	ACTIVE	07-2011	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	30	5	Open: JANUARY Close: DECEMBER
66	1145020	HILLSBORO BAPTIST CHURCH	337 2ND NH TPKE		HILLSBOROUGH	ACTIVE	08-2019	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	28	5	Open: JANUARY Close: DECEMBER
67	1165020	HOLDERNESS CENTRAL SCH	3 SCHOOL ST	RTE 175	HOLDERNESS	ACTIVE	01-1953	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	185	1	Open: SEPTEMBER Close: JUNE
68	1175030	HOLLIS SCH DIST	39 ROCKY POND RD		HOLLIS	ACTIVE	01-1970	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	2000	11	Open: JANUARY Close: DECEMBER
69	1175050	HOLLIS BROOKLINE HS	24 CAVALIER CT		HOLLIS	ACTIVE	09-1997	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	945	1	Open: JANUARY Close: DECEMBER
70	1175060	HOLLIS MONTESSORI SCHOOL	9 S MERRIMACK RD		HOLLIS	ACTIVE	04-2013	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	145	3	Open: SEPTEMBER Close: JUNE
71	1195040	BEECH HILL SCHOOL	20 BEECH HILL RD		HOPKINTON	ACTIVE	06-1999	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	40	1	Open: JANUARY Close: DECEMBER
72	1245010	MONADNOCK WALDORF SCH	424 OLD WALPOLE RD		KEENE	ACTIVE	02-2003	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	48	1	Open: JANUARY Close: DECEMBER
73	1255010	KENSINGTON ELEMENTARY SCH	122 AMESBURY RD	RTE 150	KENSINGTON	ACTIVE	01-1952	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	147	1	Open: SEPTEMBER Close: JUNE
74	1275010	DANIEL J BAKIE SCH	179 MAIN ST		KINGSTON	ACTIVE	01-1962	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	440	1	Open: SEPTEMBER Close: JUNE
75	1275060	SANBORN REGIONAL HIGH SCH	17 DANVILLE RD		KINGSTON	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	950	1	Open: JANUARY Close: DECEMBER
76	1285010	LACONIA CHRISTIAN SCH	1386 MEREDITH CENTER RD		LACONIA	ACTIVE	01-1979	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	108	4	Open: SEPTEMBER Close: JUNE
77	1296010	EASTER SEALS YOUTH RESDNCE SVS	525 PROSPECT ST		LANCASTER	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	48	1	Open: JANUARY Close: DECEMBER
78	1305020	LANDAFF BLUE SCH	813 MILLBROOK RD		LANDAFF	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	25	1	Open: SEPTEMBER Close: JUNE
79	1315010	FALL MOUNTAIN REGIONAL HS	134 FALL MT REG HS RD		LANGDON	ACTIVE	01-1966	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	850	5	Open: JANUARY Close: DECEMBER
80	1315020	SARAH PORTER SCH	111 VILLAGE RD	RTE 12A N	LANGDON	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	43	2	Open: SEPTEMBER Close: JUNE
81	1335010	MAST WAY ELEMENTARY SCH	23 MAST RD	RTE 155	LEE	ACTIVE	01-1960	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	392	1	Open: SEPTEMBER Close: JUNE
82	1345010	LEMPSTER COMMUNITY SCH	29 SCHOOL ST	RTE 10	LEMPSTER	ACTIVE	01-1956	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	150	1	Open: JANUARY Close: DECEMBER
83	1379010	TABERNACLE CHRISTIAN SCH	242 DERRY RD	RTE 102	LITCHFIELD	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	125	1	Open: JANUARY Close: DECEMBER
84	1395140	VICTORY BAPTIST SCH	78 LITCHFIELD RD		LONDONDERRY	ACTIVE	08-2003	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	79	1	Open: AUGUST Close: JUNE
85	1405010	LOUDON ELEMENTARY SCH	7039 SCHOOL ST		LOUDON	ACTIVE	01-1970	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	321	1	Open: SEPTEMBER Close: JUNE
86	1435030	CROSSROADS ACADEMY	95 DARTMOUTH COLLEGE HWY		LYME	ACTIVE	09-1991	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	162	3	Open: JANUARY Close: DECEMBER
87	1435040	CROSSROADS ACADEMY/NORTH	95 DARTMOUTH COLLEGE HWY		LYME	ACTIVE	08-2006	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	180	1	Open: JANUARY Close: DECEMBER
88	1445010	LYNDEBOROUGH CENTRAL SCH	192 FOREST RD	192 RTE 31	LYNDEBOROUGH	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	74	1	Open: JANUARY Close: DECEMBER
89	1455010	MOHARIMET SCH	11 LEE RD	RTE 155	MADBURY	ACTIVE	09-1988	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	427	1	Open: SEPTEMBER Close: JUNE
90	1465010	MADISON ELEMENTARY SCH	2069 VILLAGE RD	RTE 113	MADISON	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	170	2	Open: SEPTEMBER Close: JUNE
91	1495010	JOHN D PERKINS SR ELEM SCH	919 RTE 10		MARLOW	ACTIVE	09-1973	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	41	1	Open: SEPTEMBER Close: JUNE
92	1515010	MASON PUBLIC SCH	13 DARLING HILL RD		MASON	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	122	2	Open: SEPTEMBER Close: JUNE
93	1525010	INTER LAKES HIGH SCH	1 LAKER LN	RTE 25	MEREDITH	ACTIVE	09-1956	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	556	1	Open: JANUARY Close: DECEMBER
94	1525020	INTER LAKES ELEMENTARY SCH	21 LAKER LN	RTE 25	MEREDITH	ACTIVE	09-1972	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	587	1	Open: JANUARY Close: DECEMBER
95	1525030	LAKELAND SCH	40 MEREDITH CTR RD	OFF RTE 104	MEREDITH	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	80	1	Open: SEPTEMBER Close: JUNE
96	1545010	MIDDLETON ELEMENTARY SCHOOL	116 KINGS HWY		MIDDLETON	ACTIVE	06-2016	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	174	1	Open: JANUARY Close: DECEMBER
97	1555010	MILAN VILLAGE SCH	11 BRIDGE ST		MILAN	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	151	3	Open: SEPTEMBER Close: JUNE
98	1582010	SHORTRIDGE ACADEMY	619 GOVERNORS RD		MILTON	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	64	6	Open: JANUARY Close: DECEMBER
99	1605010	MONT VERNON VILLAGE SCH	1 KITTREDGE RD		MONT VERNON	ACTIVE	01-1969	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	216	1	Open: JANUARY Close: DECEMBER
100	1615010	MOULTONBOROUGH CENTRAL SCH	916 WHITTIER HWY	RTE 25	MOULTONBOROUGH	ACTIVE	01-1974	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	330	1	Open: SEPTEMBER Close: JUNE
101	1615020	MOULTONBOROUGH ACADEMY	25 BLAKE RD	RTE 25	MOULTONBOROUGH	ACTIVE	09-1980	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	345	1	Open: SEPTEMBER Close: JUNE
102	1625020	SECOND NATURE ACADEMY	10 GROTON RD		NASHUA	ACTIVE	01-2009	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	212	2	Open: JANUARY Close: DECEMBER
103	1635010	NELSON ELEMENTARY SCH	441 GRANITE LAKE RD	RTE 9	NELSON	ACTIVE	01-1950	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	60	1	Open: SEPTEMBER Close: JUNE
104	1645010	NEW BOSTON ELEMENTARY SCH	15 CENTRAL SCHOOL RD	RTE 13	NEW BOSTON	ACTIVE	01-1954	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	613	3	Open: JANUARY Close: DECEMBER
105	1675010	NEW DURHAM ELEMENTARY SCH	7 OLD BAY RD		NEW DURHAM	ACTIVE	09-1963	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	180	1	Open: SEPTEMBER Close: JUNE
106	1715020	MASCENIC REGIONAL HIGH SCH	175 TURNPIKE RD		NEW IPSWICH	ACTIVE	01-1978	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	350	1	Open: SEPTEMBER Close: JUNE
107	1715040	BOYNTON MIDDLE SCH	500 TURNPIKE RD	RTE 124	NEW IPSWICH	ACTIVE	08-1989	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	410	1	Open: SEPTEMBER Close: JUNE
108	1715060	HIGHBRIDGE HILL ELEM SCH	171 TURNPIKE RD		NEW IPSWICH	ACTIVE	08-2010	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	465	1	Open: JANUARY Close: DECEMBER
109	1755010	SANBORN REGIONAL MIDDLE SCH	31A WEST MAIN ST	TIER ST EXT/NEWTO	NEWTON	ACTIVE	01-1978	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	703	2	Open: SEPTEMBER Close: JUNE
110	1764010	SPAULDING YOUTH CENTER	72 SPAULDING RD		NORTHFIELD	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	300	11	Open: JANUARY Close: DECEMBER
111	1795020	COE BROWN ACADEMY	907 FIRST NH TPKE	RTE 4	NORTHWOOD	ACTIVE	01-1980	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	796	4	Open: SEPTEMBER Close: JUNE
112	1795040	COE BROWN SMITH HALL	902 FIRST NH TPKE		NORTHWOOD	ACTIVE	11-1994	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	789	1	Open: SEPTEMBER Close: JUNE
113	1805050	NOTTINGHAM COMMUNITY SCH	245 STAGE RD	RTE 152	NOTTINGHAM	ACTIVE	07-1994	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	609	1	Open: JANUARY Close: DECEMBER
114	1835010	RIVENDELL INTERSTATE SCH	2972 RTE 25A	CHOOL DR /ADMIN E	ORFORD	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	280	2	Open: SEPTEMBER Close: JUNE
115	1845020	CORNERSTONE CHRISTIAN ACADEMY	129 RTE 28		OSSIPEE	ACTIVE	09-1997	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	72	3	Open: JANUARY Close: DECEMBER
116	1875020	WELL SCHOOL	360 MIDDLE HANCOCK RD		PETERBOROUGH	ACTIVE	01-1965	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	160	5	Open: JANUARY Close: DECEMBER
117	1885010	PIERMONT VILLAGE SCH	131 RTE 10		PIERMONT	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	88	1	Open: JANUARY Close: DECEMBER
118	1935010	POLLARD ELEMENTARY SCH	120 MAIN ST		PLAISTOW	ACTIVE	01-1952	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	574	2	Open: SEPTEMBER Close: JUNE
119	1935020	TIMBERLANE MIDDLE SCH	44 GREENOUGH RD		PLAISTOW	ACTIVE	01-1968	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	984	1	Open: SEPTEMBER Close: JUNE
120	1935030	TIMBERLANE REGIONAL HS	36 GREENOUGH RD		PLAISTOW	ACTIVE	01-1964	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	1295	2	Open: SEPTEMBER Close: JUNE
121	1945010	MOUNTAIN VILLAGE SCHOOL	13 RTE 25		PLYMOUTH	ACTIVE	08-2014	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	62	1	Open: JANUARY Close: DECEMBER
122	1985010	ST BENEDICT CENTER MONASTERY	95 FAY MARTIN RD	OFF RTE 119	RICHMOND	ACTIVE	08-1989	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	200	5	Open: JANUARY Close: DECEMBER
123	1994010	FRANKLIN PIERCE UNIVERSITY	40 UNIVERSITY DR		RINDGE	ACTIVE	08-1962	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	1638	37	Open: JANUARY Close: DECEMBER
124	1995010	RINDGE MEMORIAL SCH	45 SCHOOL ST		RINDGE	ACTIVE	09-1950	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	450	2	Open: JANUARY Close: DECEMBER
125	1995030	HAMPSHIRE COUNTRY SCHOOL	28 PATEY CIR		RINDGE	ACTIVE	01-1901	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	40	5	Open: JANUARY Close: DECEMBER
126	1995050	HERITAGE CHRISTIAN SCHOOL	13 NORTH ST		RINDGE	ACTIVE	09-1996	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	64	1	Open: SEPTEMBER Close: JUNE
127	2035010	RUSSELL ELEMENTARY SCH	195 SCHOOL ST	RTE 25	RUMNEY	ACTIVE	01-1957	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	157	1	Open: JANUARY Close: DECEMBER
128	2055010	NORTH SALEM ELEMENTARY SCH	140 ZION HILL RD	MILLVILLE RD	SALEM	ACTIVE	09-1968	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	427	1	Open: SEPTEMBER Close: JUNE
129	2065010	SALISBURY ELEMENTARY SCH	6 WHITTEMORE RD	RTE 4	SALISBURY	ACTIVE	01-1959	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	101	1	Open: SEPTEMBER Close: JUNE
130	2075010	SANT BANI SCHOOL UPPER BLDG	221A OSGOOD RD	E 127S /PRESCOTT	SANBORNTON	ACTIVE	01-1973	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	60	2	Open: SEPTEMBER Close: JUNE
131	2075020	SANBORNTON CENTRAL SCH	16 HUNKINS POND RD	RTE 132	SANBORNTON	ACTIVE	01-1949	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	244	1	Open: SEPTEMBER Close: JUNE

	A	B	C	D	E	F	G	H	I	J	K	L
132	2075030	SANT BANI SCHOOL MIDDLE BLDG	WEEKS OSGOOD RD	E 127S /PRESCOTT	SANBORNTON	ACTIVE	01-1988	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	120	1	Open: SEPTEMBER Close: JUNE
133	2085010	SANDOWN CENTRAL SCH	295 MAIN ST		SANDOWN	ACTIVE	07-1971	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	170	1	Open: SEPTEMBER Close: JUNE
134	2085040	SANDOWN NORTH ELEMENTARY SCH	23 STAGECOACH DR		SANDOWN	ACTIVE	09-2001	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	417	1	Open: SEPTEMBER Close: JUNE
135	2095020	SANDWICH CENTRAL SCH	28 SQUAM LAKE RD		SANDWICH	ACTIVE	01-1950	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	87	1	Open: SEPTEMBER Close: JUNE
136	2165020	BARNARD SCH	219 MAIN AVE	AKA RTE 107A	SOUTH HAMPTON	ACTIVE	01-1998	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	107	2	Open: SEPTEMBER Close: JUNE
137	2185010	STARK VILLAGE SCH	1192 STARK HWY	RTE 110	STARK	ACTIVE	01-1976	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	30	1	Open: SEPTEMBER Close: JUNE
138	2195020	STEWARTSTOWN COMMUNITY SCH	60 SCHOOL ST	E 3/W STEWARTSTO	STEWARTSTOWN	ACTIVE	05-1998	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	93	1	Open: SEPTEMBER Close: JUNE
139	2205010	JAMES FAULKNER MEMORIAL SCH	200 SCHOOL ST	OFF RTE 123	STODDARD	ACTIVE	01-1979	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	96	1	Open: SEPTEMBER Close: JUNE
140	2215010	STRAFFORD SCH	22 ROLLER COASTER RD	RTE 202A	STRAFFORD	ACTIVE	09-1960	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	481	4	Open: JANUARY Close: DECEMBER
141	2235050	STRATHAM MEMORIAL SCH	39 GIFFORD FARM RD	VELL RD /OFF RTE 1	STRATHAM	ACTIVE	09-1989	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	705	1	Open: SEPTEMBER Close: JUNE
142	2235060	CORNERSTONE SCHOOL	146 HIGH ST		STRATHAM	ACTIVE	09-1988	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	210	1	Open: JANUARY Close: DECEMBER
143	2275020	MOUNT ROYAL ACADEMY	26 SEVEN HEARTHS LN	RTE 11	SUNAPEE	ACTIVE	09-1998	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	217	3	Open: SEPTEMBER Close: JUNE
144	2285010	SURRY VILLAGE CHARTER SCH	449 RTE 12A		SURRY	ACTIVE	01-1978	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	83	1	Open: SEPTEMBER Close: JUNE
145	2295010	KEARSARGE REGIONAL HIGH SCH	457 NORTH RD	I89 EXIT 10	SUTTON	ACTIVE	09-1969	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	650	1	Open: SEPTEMBER Close: JUNE
146	2295020	SUTTON CENTRAL SCH	28 NEWBURY RD	SUTTON MILLS	SUTTON	ACTIVE	06-1954	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	118	1	Open: SEPTEMBER Close: JUNE
147	2295030	KEARSARGE REGIONAL MIDDLE SCH	32 GILE POND RD	E 114/NORTH SUTT(	SUTTON	ACTIVE	03-2008	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	525	1	Open: JANUARY Close: DECEMBER
148	2305050	MONADNOCK REGIONAL HIGH SCH	580 OLD HOMESTEAD HWY	TE 32/CTR SWANZE	SWANZEY	ACTIVE	09-1962	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	1202	3	Open: JANUARY Close: DECEMBER
149	2315010	KENNETH A BRETT SCH	881 TAMWORTH RD	RTE 113	TAMWORTH	ACTIVE	09-1955	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	230	1	Open: SEPTEMBER Close: JUNE
150	2315030	COMMUNITY SCH	1164 BUNKER HILL RD	E 113W/S TAMWOR	TAMWORTH	ACTIVE	09-1992	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	45	1	Open: JANUARY Close: DECEMBER
151	2345010	THORNTON CENTRAL SCH	1886 RTE 175		THORNTON	ACTIVE	01-1955	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	275	2	Open: SEPTEMBER Close: JUNE
152	2375010	TUFTONBORO CENTRAL SCH	205 MIDDLE RD	RTE 109A	TUFTONBORO	ACTIVE	01-1938	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	150	1	Open: SEPTEMBER Close: JUNE
153	2385010	UNITY ELEMENTARY SCH	864 2ND NH TURNPIKE		UNITY	ACTIVE	01-1954	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	150	1	Open: SEPTEMBER Close: JUNE
154	2435020	WASHINGTON ELEMENTARY SCH	337 MILLEN POND RD	RTE 31	WASHINGTON	ACTIVE	01-1992	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	125	3	Open: JANUARY Close: DECEMBER
156	2455040	JOHN STARK REG HIGH SCH	618 N STARK HWY	RTE 114	WEARE	ACTIVE	10-1986	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	825	1	Open: JANUARY Close: DECEMBER
157	2455060	CENTER WOOD ELEMENTARY SCH	14 CENTER RD	RTE 114N	WEARE	ACTIVE	04-1990	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	611	1	Open: SEPTEMBER Close: JUNE
158	2455080	WEARE MIDDLE SCH	16 EAST ST		WEARE	ACTIVE	08-2007	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	500	1	Open: JANUARY Close: DECEMBER
159	2465010	WEBSTER ELEMENTARY SCH	936 BATTLE ST		WEBSTER	ACTIVE	01-1987	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	100	1	Open: JANUARY Close: DECEMBER
160	2475010	WENTWORTH ELEMENTARY SCH	1247 MOUNT MOOSILAUKE	RTE 25	WENTWORTH	ACTIVE	01-1971	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	83	1	Open: SEPTEMBER Close: JUNE
161	2495030	WESTMORELAND ELEMENTARY SCH	40 GLEBE RD	RTE 63	WESTMORELAND	ACTIVE	08-1960	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	167	1	Open: SEPTEMBER Close: JUNE
162	2505010	WHITE MTN REGIONAL HS	127 REGIONAL RD	RTE 3	WHITEFIELD	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	453	1	Open: SEPTEMBER Close: JUNE
163	2525010	HIGH MOWING SCH	222 ISAAC FRYE HWY	E RTE 101/ABBOTT I	WILTON	ACTIVE	01-1943	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	163	9	Open: JANUARY Close: DECEMBER
164	2525020	PINE HILL WALDORF SCH	77 PINE HILL DR	ABBOTT HILL RD	WILTON	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	150	1	Open: SEPTEMBER Close: JUNE
165	2525030	PINE HILL WALDORF CHLD VILLAGE	77 PINE HILL RD		WILTON	ACTIVE	01-2013	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	40	2	Open: JANUARY Close: DECEMBER
166	2545010	WINDHAM CENTER SCH	2 LOWELL RD		WINDHAM	ACTIVE	09-1960	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	535	1	Open: SEPTEMBER Close: JUNE
167	2545020	GOLDEN BROOK SCH	112B LOWELL RD		WINDHAM	ACTIVE	09-1971	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	1175	1	Open: SEPTEMBER Close: JUNE
168	2545030	WINDHAM MIDDLE SCH	112A LOWELL RD		WINDHAM	ACTIVE	09-1986	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	620	1	Open: SEPTEMBER Close: JUNE
169	2545100	WINDHAM HIGH SCH	64 LONDON BRIDGE RD		WINDHAM	ACTIVE		NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	1076	1	Open: JANUARY Close: DECEMBER
170	2546070	WINDHAM ACADEMY	1 INDUSTRIAL DR		WINDHAM	ACTIVE	05-1983	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	30	1	Open: JANUARY Close: DECEMBER
171	2547060	WINDHAM WOODS SCHOOL	39 ROULSTON RD		WINDHAM	ACTIVE	05-2014	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	99	1	Open: JANUARY Close: DECEMBER
172	2557060	WEDIKO KITCHEN	167 BLACK POND RD	RTE 31 N	WINDSOR	ACTIVE	06-1990	NON-TRANSIENT NON-COMMU	SCHOOLS (PUBLIC, PRIVATE D/	125	5	Open: JANUARY Close: DECEMBER
173												
174									**Total Population Served**	56680		

EXHIBIT G

QuickFacts

New Hampshire

QuickFacts provides statistics for all states and counties, and for cities and towns with a ***population of 5,000 or more***.

Table

All Topics	New Hampshire
Population estimates, July 1, 2019, (V2019)	**1,359,711**
PEOPLE	
Population	
Population estimates, July 1, 2019, (V2019)	**1,359,711**
Population estimates, July 1, 2018, (V2018)	1,356,458
Population estimates base, April 1, 2010, (V2019)	1,316,462
Population estimates base, April 1, 2010, (V2018)	1,316,464
Population, percent change - April 1, 2010 (estimates base) to July 1, 2019, (V2019)	3.3%
Population, percent change - April 1, 2010 (estimates base) to July 1, 2018, (V2018)	3.0%
Population, Census, April 1, 2010	1,316,470
Age and Sex	
Persons under 5 years, percent	⚠ 4.7%
Persons under 18 years, percent	⚠ 19.0%
Persons 65 years and over, percent	⚠ 18.1%
Female persons, percent	⚠ 50.4%
Race and Hispanic Origin	
White alone, percent	⚠ 93.2%
Black or African American alone, percent (a)	⚠ 1.7%
American Indian and Alaska Native alone, percent (a)	⚠ 0.3%
Asian alone, percent (a)	⚠ 3.0%
Native Hawaiian and Other Pacific Islander alone, percent (a)	⚠ Z
Two or More Races, percent	⚠ 1.8%
Hispanic or Latino, percent (b)	⚠ 3.9%
White alone, not Hispanic or Latino, percent	⚠ 90.0%
Population Characteristics	
Veterans, 2014-2018	97,644
Foreign born persons, percent, 2014-2018	6.0%
Housing	
Housing units, July 1, 2018, (V2018)	638,091
Owner-occupied housing unit rate, 2014-2018	71.0%
Median value of owner-occupied housing units, 2014-2018	$252,800
Median selected monthly owner costs -with a mortgage, 2014-2018	$1,917
Median selected monthly owner costs -without a mortgage, 2014-2018	$792
Median gross rent, 2014-2018	$1,077
Building permits, 2018	4,445
Families & Living Arrangements	
Households, 2014-2018	528,078
Persons per household, 2014-2018	2.46
Living in same house 1 year ago, percent of persons age 1 year+, 2014-2018	86.0%
Language other than English spoken at home, percent of persons age 5 years+, 2014-2018	7.9%
Computer and Internet Use	
Households with a computer, percent, 2014-2018	92.2%
Households with a broadband Internet subscription, percent, 2014-2018	86.2%
Education	

High school graduate or higher, percent of persons age 25 years+, 2014-2018	92.9%
Bachelor's degree or higher, percent of persons age 25 years+, 2014-2018	36.5%
Health	
With a disability, under age 65 years, percent, 2014-2018	8.8%
Persons without health insurance, under age 65 years, percent	▲ 6.9%
Economy	
In civilian labor force, total, percent of population age 16 years+, 2014-2018	67.7%
In civilian labor force, female, percent of population age 16 years+, 2014-2018	63.6%
Total accommodation and food services sales, 2012 ($1,000) (c)	2,942,278
Total health care and social assistance receipts/revenue, 2012 ($1,000) (c)	9,616,460
Total manufacturers shipments, 2012 ($1,000) (c)	18,895,624
Total merchant wholesaler sales, 2012 ($1,000) (c)	18,029,179
Total retail sales, 2012 ($1,000) (c)	26,018,201
Total retail sales per capita, 2012 (c)	$19,700
Transportation	
Mean travel time to work (minutes), workers age 16 years+, 2014-2018	27.3
Income & Poverty	
Median household income (in 2018 dollars), 2014-2018	$74,057
Per capita income in past 12 months (in 2018 dollars), 2014-2018	$38,548
Persons in poverty, percent	▲ 7.6%

BUSINESSES

Businesses	
Total employer establishments, 2017	38,371[1]
Total employment, 2017	603,923[1]
Total annual payroll, 2017 ($1,000)	30,634,092[1]
Total employment, percent change, 2016-2017	1.6%[1]
Total nonemployer establishments, 2017	107,083
All firms, 2012	131,638
Men-owned firms, 2012	76,716
Women-owned firms, 2012	38,525
Minority-owned firms, 2012	6,111
Nonminority-owned firms, 2012	121,297
Veteran-owned firms, 2012	16,119
Nonveteran-owned firms, 2012	108,817

GEOGRAPHY

Geography	
Population per square mile, 2010	147.0
Land area in square miles, 2010	8,952.65
FIPS Code	33

About datasets used in this table

Value Notes

1. Includes data not distributed by county.

⚠ Estimates are not comparable to other geographic levels due to methodology differences that may exist between different data sources.

Some estimates presented here come from sample data, and thus have sampling errors that may render some apparent differences between geographies statistically indistinguishable. Click the Q row in TABLE view to learn about sampling error.

The vintage year (e.g., V2019) refers to the final year of the series (2010 thru 2019). *Different vintage years of estimates are not comparable.*

Fact Notes

(a) Includes persons reporting only one race
(b) Hispanics may be of any race, so also are included in applicable race categories
(c) Economic Census - Puerto Rico data are not comparable to U.S. Economic Census data

Value Flags

- Either no or too few sample observations were available to compute an estimate, or a ratio of medians cannot be calculated because one or both of the median estimates falls in t open ended distribution.
D Suppressed to avoid disclosure of confidential information
F Fewer than 25 firms
FN Footnote on this item in place of data
N Data for this geographic area cannot be displayed because the number of sample cases is too small.
NA Not available
S Suppressed; does not meet publication standards
X Not applicable
Z Value greater than zero but less than half unit of measure shown

QuickFacts data are derived from: Population Estimates, American Community Survey, Census of Population and Housing, Current Population Survey, Small Area Health Insurance Estimates, Sm Estimates, State and County Housing Unit Estimates, County Business Patterns, Nonemployer Statistics, Economic Census, Survey of Business Owners, Building Permits.

EXHIBIT H

National Vital Statistics Reports

Volume 68, Number 13 November 27, 2019

Births: Final Data for 2018

by Joyce A. Martin, M.P.H., Brady E. Hamilton, Ph.D., Michelle J.K. Osterman, M.H.S., and Anne K. Driscoll, Ph.D.,
Division of Vital Statistics

Abstract

Objectives—This report presents 2018 data on U.S. births according to a wide variety of characteristics. Trends in fertility patterns and maternal and infant characteristics are described and interpreted.

Methods—Descriptive tabulations of data reported on the birth certificates of the 3.79 million births that occurred in 2018 are presented. Data are presented for maternal age, live-birth order, race and Hispanic origin, marital status, tobacco use, prenatal care, source of payment for the delivery, method of delivery, gestational age, birthweight, and plurality. Selected data by mother's state of residence and birth rates by age also are shown. Trend data for 2010 through 2018 are presented for selected items. Trend data by race and Hispanic origin are shown for 2016–2018.

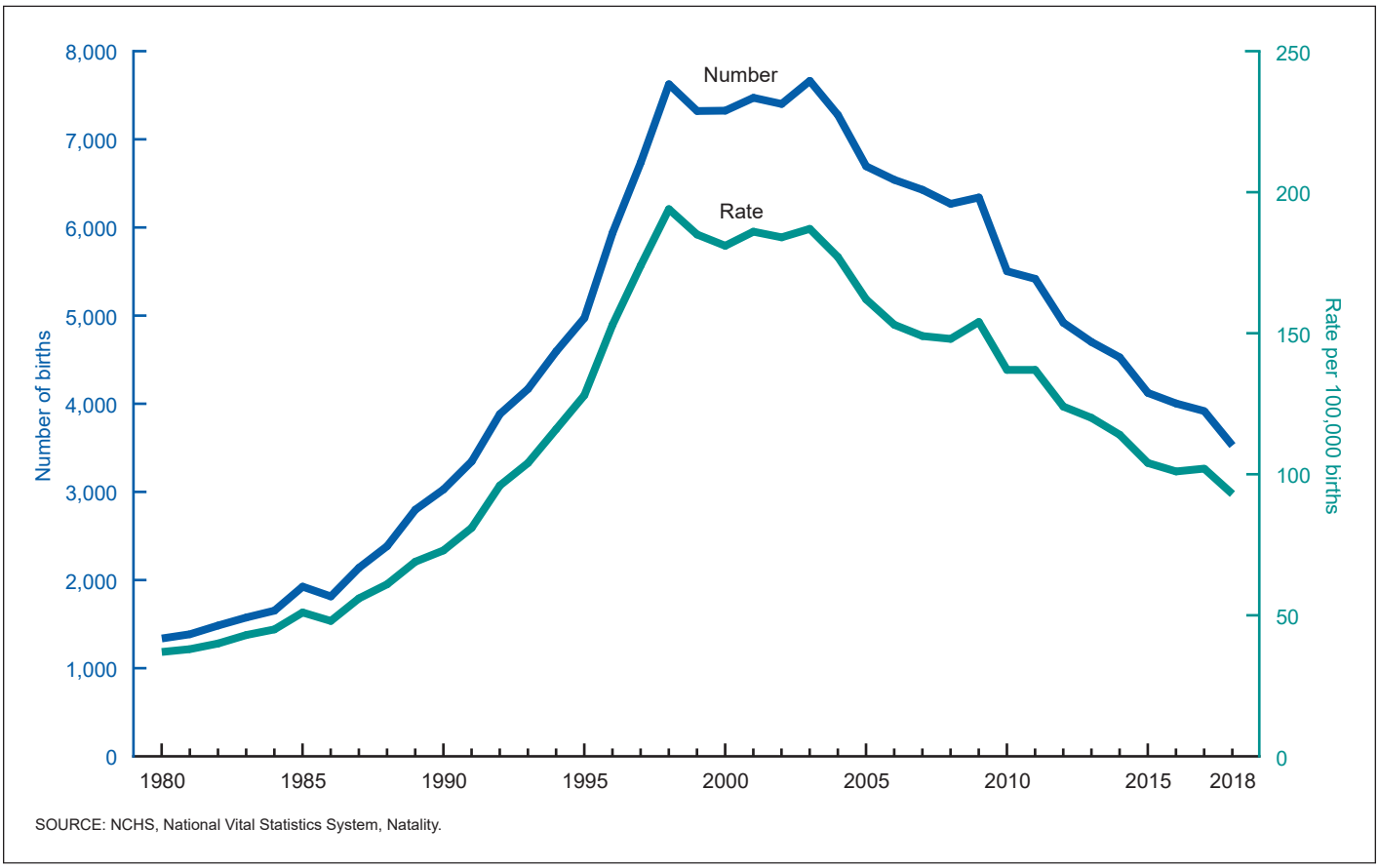


Figure 1. Number and rate of triplet and higher-order multiple births: United States, 1980–2018

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Centers for Disease Control and Prevention
National Center for Health Statistics
National Vital Statistics System

Results—3,791,712 births were registered in the United States in 2018, down 2% from 2017. Compared with rates in 2017, the general fertility rate declined to 59.1 births per 1,000 women aged 15–44. The birth rate for females aged 15–19 fell 7% in 2018. Birth rates declined for women aged 20–34 and increased for women aged 35–44. The total fertility rate declined to 1,729.5 births per 1,000 women in 2018. Birth rates for both married and unmarried women declined from 2017 to 2018. The percentage of women who began prenatal care in the first trimester of pregnancy rose to 77.5% in 2018; the percentage of all women who smoked during pregnancy declined to 6.5%. The cesarean delivery rate decreased to 31.9% in 2018 following an increase in 2017. Medicaid was the source of payment for 42.3% of all 2018 births, down 2% from 2017. The preterm birth rate rose for the fourth straight year to 10.02% in 2018; the rate of low birthweight was unchanged at 8.28%. Twin and triplet and higher-order multiple birth rates declined in 2018 (Figure 1).

Keywords: birth certificate • maternal and infant health • birth rates • maternal characteristics

Introduction

This report presents detailed data on numbers and characteristics of births in 2018, birth and fertility rates, maternal demographic and health characteristics, medical and health care utilization, source of payment for the delivery, and infant health characteristics. A report of provisional birth statistics for 2018 presented data on selected topics based on a sample of nearly all (99.73%) 2018 births (1); a National Center for Health Statistics Data Brief presented selected characteristics based on final data for 2018 births (2).

The 2018 report marks the third year for which data for all 50 states and the District of Columbia (D.C.) are based on the 2003 revision of the U.S. Certificate of Live Birth. Also presented for the third year are national data on race and Hispanic-origin categories based on the 1997 Office of Management and Budget (OMB) standards (3), allowing for the presentation of data by single race and Hispanic origin for 2016–2018. These race and Hispanic-origin groups—non-Hispanic single-race white, non-Hispanic single-race black or African American, non-Hispanic single-race American Indian or Alaska Native (AIAN), non-Hispanic single-race Asian, and non-Hispanic single-race Native Hawaiian or Other Pacific Islander (NHOPI)—differ from the bridged-race categories shown in most reports that are based on 2015 and earlier data. A comparison between bridged and single-race data by selected characteristics is shown in "Births: Final Data for 2016" (4).

In addition to the tabulations included in this report, more detailed analysis is possible by using the annual natality public-use file. The data file may be downloaded from: https://www.cdc.gov/nchs/data_access/Vitalstatsonline.htm (5). The public-use file does not include geographic detail, but a file with this information can be provided upon request (6). Birth data may also be accessed via the Centers for Disease Control and Prevention's (CDC) Wide-ranging Online Data for Epidemiologic Research (WONDER). This easy-to-use Web system makes CDC's information resources available to public health professionals and the public (7).

2003 revision of U.S. Standard Certificate of Live Birth

Starting in 2016, all 50 states, D.C., Puerto Rico, Guam, Commonwealth of the Northern Marianas, and U.S. Virgin Islands reported data based on the 2003 U.S. Certificate of Live Birth. American Samoa continued to report based on the 1989 birth certificate revision. Internet-only tables on a number of additional topics, such as births by attendant and place and delivery, mean age of mother by live-birth order, maternal prepregnancy body mass index, and infections during pregnancy and maternal morbidity, are also available; see List of Detailed Tables. For information on data quality, see reports that assessed the quality of selected medical and health data from the 2003 revised birth certificate and the "User Guide to the 2018 Natality Public Use File" (8–10).

Methods

Data shown in this report are based on 100% of the birth certificates registered in all states and D.C. More than 99% of births occurring in this country are registered (10). Tables showing data by state also provide separate information for Puerto Rico, Guam, and Northern Marianas. These areas, however, are not included in totals for the United States. Data for the U.S. Virgin Islands and American Samoa were not available for 2018.

The 2003 revision of the U.S. Standard Certificate of Live Birth allows the reporting of more than one race (multiple races) for each parent (11) in accordance with the revised standards issued by OMB in 1997 (3). Starting in 2016, all 50 states, D.C., Puerto Rico, Guam, Northern Marianas, and U.S. Virgin Islands reported race data in accordance with these 1997 OMB standards that allow for the reporting of a minimum of five race categories either by single race (i.e., reported alone) or in combination (i.e., more than one race or multiple races) (3). The race and Hispanic-origin groups shown in this report follow the 1997 standards and differ from the bridged-race categories shown in most previous reports that are based on data from 2015 and earlier (12). The new categories are: non-Hispanic single-race white, non-Hispanic single-race black or African American, non-Hispanic single-race AIAN, non-Hispanic single-race Asian, non-Hispanic single-race NHOPI, and Hispanic. For brevity, text references to non-Hispanic white or non-Hispanic black women omit the term "single-race." Because single-race data are not available for the entire United States prior to 2016, this report only makes comparisons by race for 2016–2018. For more information on differences between single- and bridged-race groups, see "Births: Final Data for 2016" (4).

Race and Hispanic origin are reported independently on the birth certificate. Most tables in this report show data for the categories of non-Hispanic single-race white, non-Hispanic single-race black, and Hispanic. Selected tables also include data for non-Hispanic single-race AIAN, non-Hispanic single-race Asian, and non-Hispanic single-race NHOPI. Data are also presented in some tables for specific Hispanic groups: Mexican, Puerto Rican, Cuban, Central and South American, and other and unknown Hispanic. Beginning with 2018, data are presented for an additional Hispanic group, Dominican. Data

for this subgroup had previously been included in the category "other and unknown Hispanic."

Trend tables included in the detailed tables for this report include the years 2010–2018; see List of Detailed Tables. Longer-term trends previously shown in this report series can be found in earlier-year reports (e.g., "Births: Final Data for 2015") (12).

In this report, the total number of births includes births to women up to age 64. In tables that include age of mother, the oldest age groups shown (40–54, 45–49, 45–54, or 50–54) include births to mothers up to age 64 (births to mothers 55–64 are recategorized as age group 50–54). For information on levels of incomplete reporting by state, see the User Guide (10). For information on the measurement of data items shown in this report, and the Internet tables, imputation techniques used, computation of derived statistics, and definitions of terms, see the User Guide (10).

Demographic Characteristics (Tables 1–12)

Births and birth rates

Number of births

In 2018, 3,791,712 births were registered in the United States, down 2% (or 63,788) from 2017 (Table 1, Figure 2). This is the fourth year that the number of births has declined following an increase in 2014. Before that year, the number of births declined steadily from 2007 through 2013 (12). Among the race and Hispanic-origin groups, the number of births declined 1% for Hispanic and 2% for non-Hispanic white and non-Hispanic black women, and 3% for non-Hispanic AIAN and non-Hispanic Asian women from 2017 to 2018; the number of births for non-Hispanic NHOPI women was essentially unchanged (Tables 1, 3, and 6). Among the specified Hispanic groups, births declined 3% for Mexican women in 2018, but rose 1% for Puerto Rican and Central and South American women (Tables 4 and 7). The number of births was essentially unchanged for Cuban women in 2018.

Fertility rate

The **general fertility rate** (GFR) for the United States in 2018 was 59.1 births per 1,000 females aged 15–44, down 2% from 2017 (60.3) and a record low rate for the nation (Table 1) (12,13). This is the fourth year that the rate has declined following an increase in 2014. From 2007 to 2013, the GFR declined steadily (12).

Among the race and Hispanic-origin groups, the GFR declined for nearly all groups in 2018, down 2% for non-Hispanic white and non-Hispanic black, 3% for non-Hispanic AIAN and Hispanic, and 4% for non-Hispanic Asian females. The rate for non-Hispanic NHOPI females was essentially unchanged from 2017 to 2018.

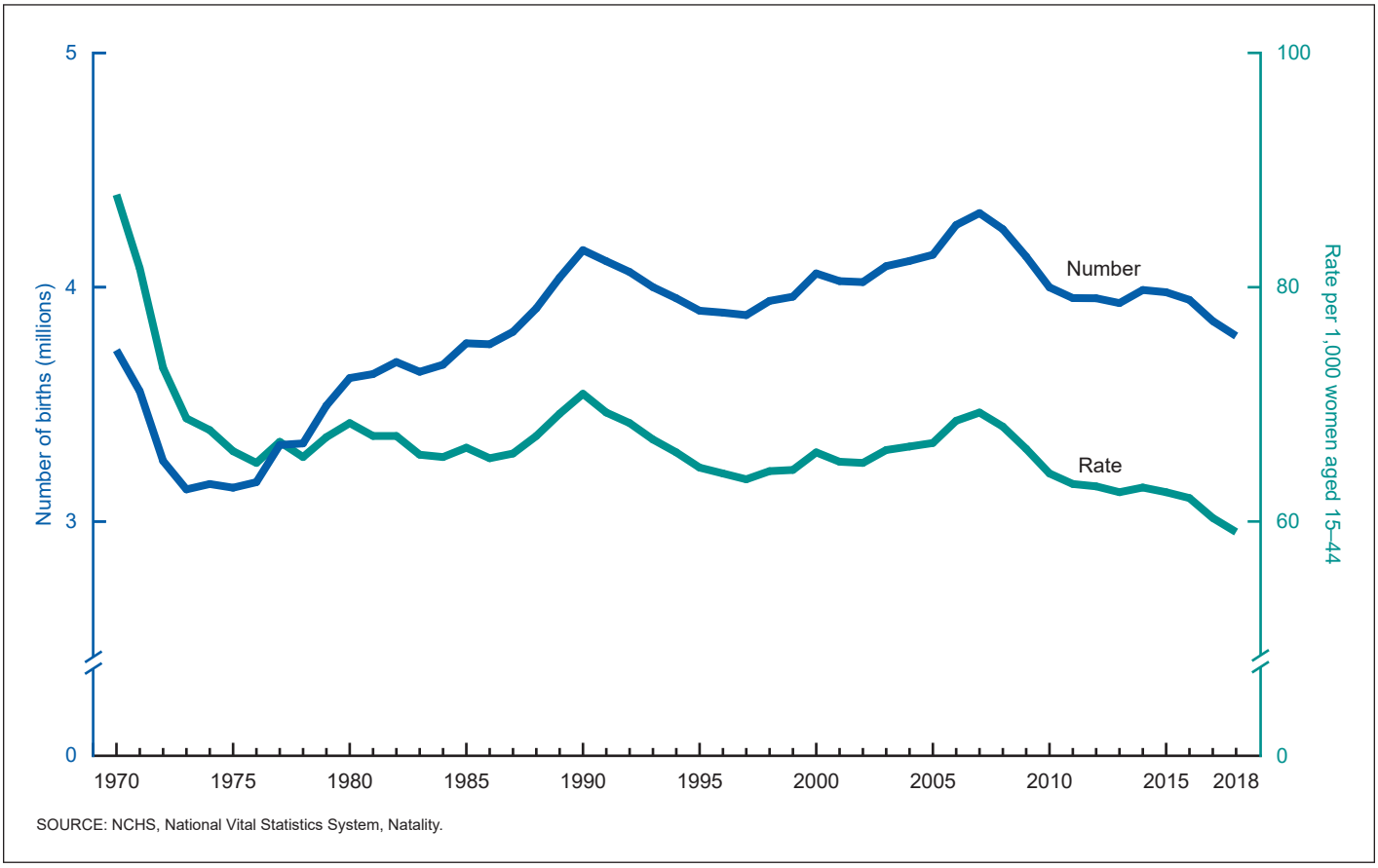


Figure 2. Live births and general fertility rates: United States, 1970–2018

Age of mother

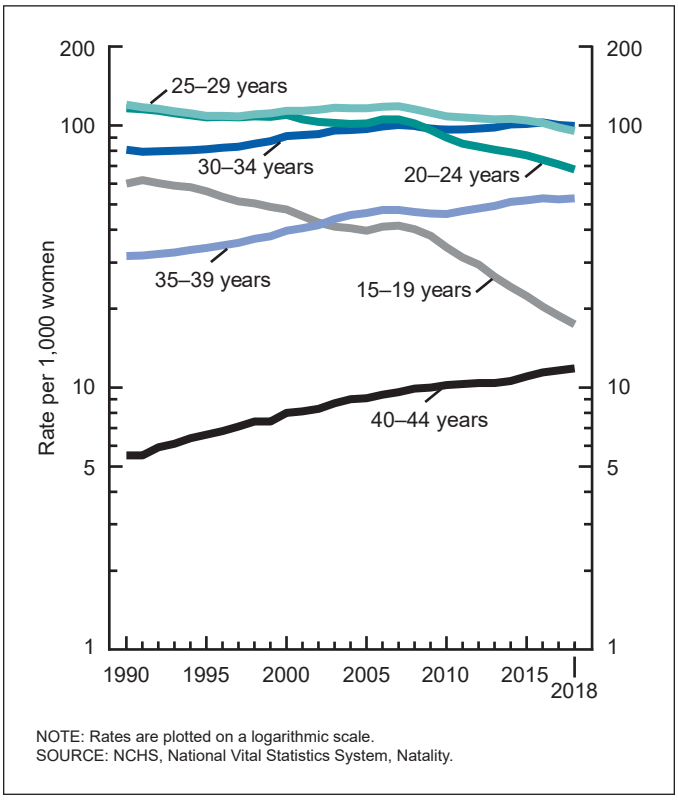
Birth rates decreased for females aged 15–34, increased for females aged 35–44, and were unchanged for females aged 10–14 and 45–49 from 2017 to 2018.

Teenagers—The birth rate for females aged 15–19 in the United States in 2018 was 17.4 births per 1,000, down 7% from 2017 (18.8) and another record low (Table 2, Figure 3) (12–14). Since 2009, the teen birth rate has fallen to a new low each year. The rate for this group has declined 58% since 2007 (41.5), the most recent high, and 72% since the 1991 high (61.8). The number of births to teenagers aged 15–19 was 179,871 in 2018, also down 7% from 2017 (194,377) (15).

The 2018 birth rates for teenagers aged 15–17 and 18–19 were 7.2 and 32.3 births per 1,000 females, respectively, down 9% and 8% from 2017, to record lows for both groups. Rates for these age groups have fallen 67% and 55%, respectively, since 2007, and by 81% and 66% since 1991. The birth rate for females aged 10–14 was unchanged in 2018 at 0.2 births per 1,000 females.

Among race and Hispanic-origin groups, the birth rates for teenagers aged 15–19 declined for nearly all groups in 2018: down 4% for non-Hispanic black, 8% for non-Hispanic white and Hispanic, 10% for non-Hispanic AIAN, and 15% for non-Hispanic Asian teenagers. The rate for non-Hispanic NHOPI teenagers was essentially unchanged from 2017 to 2018.

Women in their 20s—The birth rate for women aged 20–24 was 68.0 births per 1,000 women in 2018, down 4% from 2017 (71.0), and another record low (Table 2) (12,13). The rate for women in this age group has declined steadily since 2006. The number of births to women in their early 20s declined 5% from 2017 (15). The rate for women aged 25–29 was 95.3 births per 1,000 women, down 3% from 2017 (98.0). The rate for women in this age group has declined for all but 1 year since 2007 (Table 2). The number of births to women in their late 20s was down by 2% from 2017 to 2018 (see Tables 3 and 4 for 2018 data) (15).



NOTE: Rates are plotted on a logarithmic scale.
SOURCE: NCHS, National Vital Statistics System, Natality.

Figure 3. Birth rates, by age of mother: United States, 1990–2018

Women in their 30s—The birth rate for women aged 30–34 was 99.7 births per 1,000 women in 2018, down 1% from 2017 (100.3) and the second decline in the rate since 2010 (12,13). The 2018 rate for women aged 30–34 was higher than the rate for women aged 25–29 for the third year since reliable national records were available (1940) (12,13). The number of births to women aged 30–34 was essentially unchanged from 2017 to 2018. The birth rate for women aged 35–39 was 52.6 births per 1,000 women in 2018, up 1% from 2017 (52.3) after a brief decline in the rate in 2017, the first since 2010 (Table 2). The number of births to women aged 35–39 rose 2% in 2018 (see Tables 3 and 4 for 2018 data) (15).

Women in their 40s—The birth rate for women aged 40–44 was 11.8 births per 1,000 women in 2018, up 2% from 2017 (11.6) (Table 2); the rate for this group has risen almost continuously since 1985 (12,13). The number of births to women in their early 40s rose 2% from 2017 to 2018. The birth rate for women aged 45–49 (which includes births to women aged 50 and over) was 0.9 births per 1,000 women in 2018, unchanged from 2017. The number of births to women aged 45 and over was also unchanged from 2017 to 2018.

Women aged 50 and over—There were 959 births to women aged 50 and over in 2018, up from 840 in 2017 (Table 3) (15). The number of births to women in this age group has generally increased since 1997 (from 144 births), when data for women aged 50 and over became available again. The birth rate for women aged 50–54 rose to 0.9 births per 10,000 women in 2018, from 0.8 in 2017. Because of the small number of births to women in this age group, the birth rate for women aged 50–54 is expressed per 10,000 women. For rates shown elsewhere in this report, births to women aged 50 and over are included with births to women aged 45–49 when computing birth rates by age of mother (the denominator for the rate is women aged 45–49).

Live-birth order

The first birth rate for the United States was 22.4 births per 1,000 females aged 15–44 in 2018, down 2% from 2017 (22.9) (see Tables 3 and 4 for the number of births and Table 5 for birth rates). First birth rates declined for females in their teens (down 6% for females aged 15–19) and 20s (down 2% each for women aged 20–24 and 25–29), but rose for women in their 30s (up less than 1% for women aged 30–34 and 1% for women aged 35–39) and early 40s (up 4%). First birth rates for females aged 10–14 and 45–49 were unchanged in 2018 (15).

Mean age of mother

In 2018, the mean age of mothers at first birth was 26.9 years, an increase from 26.8 in 2017, and another record high

for the nation (Tables 11, 12, and I–6) (12,13). The mean age at first birth is the arithmetic average of the age of mothers at the time of birth and is computed directly from the frequency of first births by age of mother. The increase in the mean age in 2018 reflects, in part, the decline in first births to females in their teens and 20s, and the rise in first births to women in their 30s and early 40s (15).

Mean age at first birth increased for nearly all race and Hispanic-origin groups in 2018, rising to 23.5 years for non-Hispanic AIAN, 25.0 for Hispanic, 25.1 for non-Hispanic black, 27.7 for non-Hispanic white, and 30.5 for non-Hispanic Asian women (Tables 11, 12, and I–6). The average age at first birth for non-Hispanic NHOPI women was 24.7 in 2018, essentially unchanged from 2017. Among the specified Hispanic groups, average ages increased to 24.4 years for Mexican, 24.8 for Puerto Rican, and 27.7 for Cuban women, and were essentially unchanged for Central and South American women (26.5).

Total fertility rate

The **total fertility rate** (TFR) for the United States in 2018 was 1,729.5 births per 1,000 women, down 2% from 2017 (1,765.5) (Table 2). This is the fourth year that the TFR has declined following an increase in 2014. From 2007 to 2013, the rate declined steadily. The TFR estimates the number of births that a hypothetical group of 1,000 women would have over their lifetimes, based on age-specific birth rates in a given year. Because it is computed from age-specific birth rates, TFR is age-adjusted and can be compared for populations across time, population groups, and geographic areas.

Among the race and Hispanic-origin groups, the TFR declined for nearly all groups in 2018, down 2% for non-Hispanic white, non-Hispanic black, and Hispanic; 3% for non-Hispanic AIAN; and 5% for non-Hispanic Asian women. The rate for non-Hispanic NHOPI women was essentially unchanged from 2017 to 2018.

The TFR for the nation in 2018 remained below replacement, the level at which a given generation can exactly replace itself (generally considered to be 2,100 births per 1,000 women). The U.S. TFR has generally been below replacement since 1971 (12,13). The TFR was below replacement for all race and Hispanic-origin groups in 2018, except non-Hispanic NHOPI women (2,106.5) (Table 2).

Births and birth rates by state

The GFR, the number of births per 1,000 females aged 15–44, declined from 2017 to 2018 by 1% to 3% in 25 states, and by 4% to 6% in 10 states and D.C. (Arizona, Colorado, Idaho, Montana, Oregon, South Dakota, Utah, Vermont, Washington, and Wyoming), rose for 2 states (New York and New Jersey), and was essentially unchanged in 13 states (Connecticut, Delaware, Kansas, Maine, Maryland, Mississippi, Missouri, Nevada, New Hampshire, North Dakota, Oklahoma, Rhode Island, and West Virginia). Rates among the states ranged from 47.2 births per 1,000 females aged 15–44 in Vermont to 73.6 in South Dakota. (See Tables 6 and 7 for the number of births and Table 8 for birth rates.) Among the U.S. territories, the GFR declined in Puerto Rico, rose in Northern Marianas, and was essentially unchanged in Guam. For the number of births by state in 2018, see Tables 6 and 7.

Birth rates for teenagers by state

In 2018, the birth rate for teenagers aged 15–19 declined in 38 states, with declines ranging from 4% for Indiana, Michigan, and Missouri, to 19% for Montana. Rates were essentially unchanged in the remaining 12 states and D.C. (Connecticut, Delaware, Iowa, Maryland, New Hampshire, New Jersey, North Dakota, Rhode Island, South Carolina, South Dakota, Vermont, and West Virginia). Rates among the states ranged from 7.2 births per 1,000 in Massachusetts to 30.4 in Arkansas (Table 8). The wide range in state-specific teen rates is consistent with patterns observed in previous analyses (14,16). Teen birth rates declined in Puerto Rico, rose in Northern Marianas, and were essentially unchanged for Guam. Rates among the territories ranged from 19.3 births per 1,000 teenagers in Puerto Rico to 34.4 in Guam (Table 8).

Births to unmarried women

The birth rate for unmarried women was 40.1 births per 1,000 unmarried women aged 15–44 in 2018, down 2% from 2017 (41.0) (Table 10). The 2018 nonmarital birth rate was 23% lower than the peak of 51.8 in 2007 and 2008 (12). (See Technical Notes for description of the adjustments made to calculations of rates by marital status beginning with 2017.)

The birth rate for married women also declined in 2018, to 85.6 per 1,000 married women aged 15–44, from 87.4 in 2017. The marital birth rate declined 4% over the period 2005–2010 but rose 6% from 2010 through 2016 (12).

The percentage of all births to unmarried women was 39.6% in 2018, down from 2017 (39.8%) and the lowest level since 2007 (12). The percentage of all births to unmarried women peaked in 2009 at 41.0% (12).

In 2018, the percentage of nonmarital births decreased from 2017 for three race and Hispanic-origin groups: non-Hispanic white (28.2% in 2018), Hispanic (51.8%), and non-Hispanic AIAN women (68.2%). The percentage of nonmarital births increased for non-Hispanic NHOPI women (50.4%) and was unchanged for two groups: non-Hispanic black (69.4%) and non-Hispanic Asian women (11.7%). The number of nonmarital births decreased by 2% from 2017 (1,533,901) to 2018 (1,503,361) (15). Nonmarital birth rates declined from 2017 to 2018 for women in age groups under 30 and for women aged 35–39, with the rate for teenagers aged 15–19 dropping 6% (to 16.0 per 1,000 in 2018), and the rate for females aged 15–17 at another all-time low (7.1). Conversely, the nonmarital birth rate rose for women aged 30–34, to 59.5, and for women aged 40–44, reaching a historic peak of 10.6.

Compositional differences by race and Hispanic origin and maternal age among states are major contributing factors to the geographic variation in the percentage of births to unmarried mothers (Table I–7). In 2018, the percentages of unmarried births ranged from about one in five births in Utah (19.2%) to more than one-half of births in Louisiana (53.3%), Mississippi (54.1%), and New Mexico (51.2%).

Tobacco Use Before and During Pregnancy (Tables 13–15)

Of the women who gave birth in 2018, 6.5% reported smoking tobacco at some point while pregnant, a 6% decline from 2017 (Table 15) (15). Tobacco use was most common earlier in pregnancy: 6.3% of women smoked in their first trimester, 5.4% in their second, and 5.2% in their third. Of the 8.4% of women who reported smoking in the 3 months before becoming pregnant, 24.2% quit smoking before pregnancy (Table 15).

By race and Hispanic origin, 9.5% of non-Hispanic white women reported smoking at some point during pregnancy, down 6% from 2017; 5.2% of non-Hispanic black women reported smoking at some point during pregnancy, down 7%; and 1.7% of Hispanic women reported smoking during pregnancy, down 6% from 2017. See Tables 13 and 14 for smoking levels among other race and Hispanic-origin groups. Rates of quitting smoking before pregnancy also varied by race and Hispanic origin: 22.3% of non-Hispanic white, 25.8% of non-Hispanic black, and 38.7% of Hispanic women who smoked during the 3 months before pregnancy did not smoke while pregnant.

By maternal age, smoking rates during pregnancy were highest among women aged 20–24 (9.2%) and for women under age 20 (7.6%) and 25–29 (7.5%). Tobacco use during pregnancy was less common among older women, with 3.5% of women aged 40–54 and 4.3% of women aged 35–39 reporting smoking during pregnancy (Table 15).

Medical and Health Services Utilization and Source of Payment for the Delivery (Tables 13–19)

Use and timing of prenatal care

Among women giving birth in 2018, 77.5% began prenatal care in the first trimester of pregnancy, up from 77.3% in 2017. Late (beginning in the third trimester) or no prenatal care declined in 2018 to 6.2%, from 6.3% in 2017 (Table 16) (15).

By race and Hispanic origin, prenatal care beginning in the first trimester increased for non-Hispanic white (from 82.4% in 2017 to 82.5% in 2018), non-Hispanic black (66.6% to 67.1%), non-Hispanic Asian (81.1% to 81.8%), and Hispanic (72.3% to 72.7%) women. First trimester care declined in 2018 for non-Hispanic AIAN (63.4% to 62.6%) and non-Hispanic NHOPI (52.5% to 51.0%) women (Table 13) (15). Levels of late or no prenatal care declined for non-Hispanic black (10.2% to 9.9%) and non-Hispanic Asian (5.1% to 4.9%) women but remained essentially unchanged for other groups. Timing of prenatal care continued to vary by race and Hispanic origin in 2018. First trimester care ranged from 51.0% for non-Hispanic NHOPI women to 82.5% for non-Hispanic white women; late or no prenatal care ranged from 4.5% for non-Hispanic white women to 20.2% for non-Hispanic NHOPI women.

By age of mother, women aged 20–24 and 35–39 were more likely to receive first trimester prenatal care in 2018 (70.9% and 81.7%, respectively) than in 2017 (70.5% and 81.5%, respectively), whereas women aged 25–29 and 40 and over were less likely to receive first-trimester care (77.8% to 77.7% and 78.6% to 78.2%, respectively). Rates for other age groups were unchanged in 2018. Levels of late or no prenatal care increased for mothers under age 20 (11.4% to 11.7%) and declined for women aged 20–24 (8.2% to 8.1%) and 35–39 (5.0% to 4.9%), but were unchanged for other groups in 2018 (Table 16) (15).

Method of delivery

Cesarean delivery

In 2018, the cesarean delivery rate decreased to 31.9% from 32.0% in 2017 (Figure 4, Table 17). In 2017, the cesarean delivery rate had increased for the first time since 2009, when it peaked at 32.9% after increasing every year since 1996 (20.7%) (12,15).

Among the three largest race and Hispanic-origin groups, the cesarean delivery rate decreased for non-Hispanic white (30.9% to 30.8%) and Hispanic (31.8% to 31.6%) women from 2017 to 2018; rates for non-Hispanic black (36.1% in 2018) women were essentially unchanged (Table 17). Tables 13 and 14 show cesarean delivery rates for more detailed race and Hispanic-origin groups.

By maternal age, cesarean delivery decreased during 2017–2018 for women under age 35, but remained unchanged for older age groups (Table 18). Cesarean delivery continued to remain higher among older women compared with younger mothers; women aged 40 and over (48.0%) were more than twice as likely to deliver by cesarean as women under age 20 (19.8%).

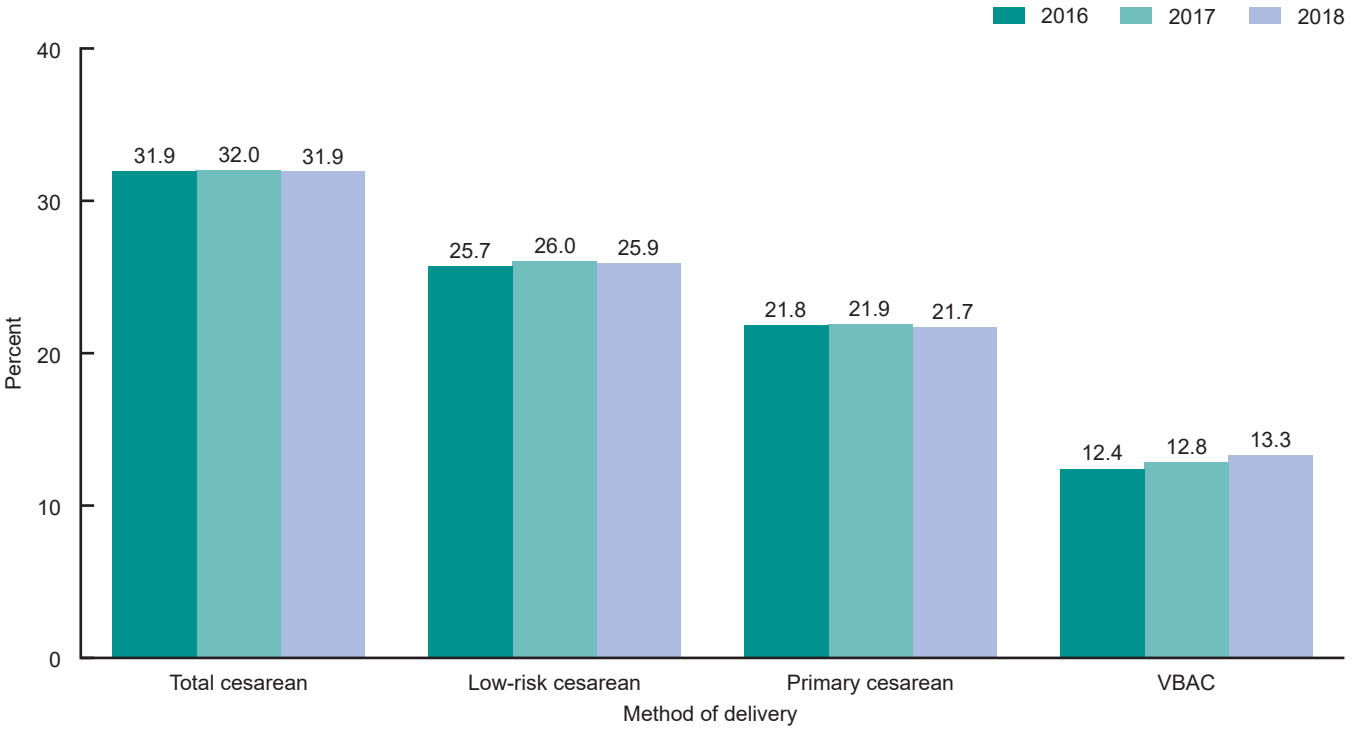
Low-risk cesarean delivery—The low-risk cesarean delivery rate also decreased in 2018, from 26.0% to 25.9% for 2017–2018. The low-risk cesarean delivery rate is cesarean delivery among nulliparous (first birth), term (37 or more completed weeks based on the obstetric estimate), singleton (one fetus), cephalic (head first) births. See Tables 13, 14, 17, and 18 for details by age and race and Hispanic origin of the mother.

Primary cesarean delivery—In 2018, the primary cesarean delivery rate, which measures cesarean deliveries among women who have not had a previous cesarean delivery, was 21.7%, down from 21.9% in 2017 (Tables 17 and 18.)

Vaginal birth after previous cesarean delivery (VBAC)—The VBAC rate measures vaginal births among women with a previous cesarean delivery. In 2018, 13.3% of women with a previous cesarean delivered vaginally, up 4% from 12.8% in 2017. See Tables 17 and 18 for details. Changes in VBAC overall and by race and Hispanic origin are presented in a recent report (2).

Source of payment for the delivery

The principal source of payment for the delivery of most births in 2018 continued to be either private insurance or Medicaid; however, the percentage of births covered by private insurance increased from 2017 (from 49.1% to 49.6%), and the percentage of births covered by Medicaid declined (from 43.0% to 42.3%). The category "self-pay," generally considered to indicate uninsured deliveries, accounted for 4.2% of 2018 births, up from 4.1% in 2017. The remainder of births were covered by other insurance (3.9%; up from 3.8% in 2017); other insurance



NOTES: Low-risk cesarean is cesarean delivery among nulliparous, term, singleton, and cephalic births. VBAC is vaginal birth after cesarean.
SOURCE: NCHS, National Vital Statistics System, Natality.

Figure 4. Method of delivery: United States, 2016–2018

includes Indian Health Service, CHAMPUS or TRICARE, other government (federal, state, or local), or charity (Table 19) (15).

Medicaid coverage for the delivery declined for each of the three largest race and Hispanic-origin groups during 2017–2018 (Table 19) (15). In 2018, the percentages of mothers with Medicaid-covered births ranged from 30.0% for non-Hispanic white women to 65.3% for non-Hispanic black women. The percentage of births covered by private insurance increased from 2017 to 2018 for non-Hispanic white (63.1% to 63.3%), non-Hispanic black (27.7% to 28.4%), and Hispanic (28.5% to 29.5%) women. See Tables 13 and 14 for information for additional race and Hispanic-origin groups.

By age of mother, Medicaid as the source of payment declined for women aged 20–24 (63.7% to 63.1%), 25–29 (45.0% to 44.8%), 30–34 (29.5% to 29.4%), and 35–39 (27.8% to 27.4%) (Table 19) (15). The percentages of births with Medicaid as the source of payment ranged from 27.4% for women aged 35–39 to 77.3% for women under age 20. Private insurance as the source of payment for the delivery increased in 2018 for women aged 20–24 (28.0% in 2017 to 28.2% in 2018), 25–29 (46.9% to 47.0%), and 35–39 (64.5% to 64.8%). Percentages with private insurance ranged from 15.1% for women under age 20 to 64.8% for women aged 35–39 (Table 19).

Infant Health Characteristics (Tables 20–25)

Period of gestation

The U.S. preterm birth rate rose to 10.02% in 2018, a 1% rise from 2017 (9.93%), and the fourth straight year of increases in this rate (9.57% in 2014). The preterm birth rate (percentage of all births delivered at less than 37 completed weeks of gestation) had declined steadily from 2007 (10.44%) (12) to 2014 (Table 20). (National data based on the obstetric estimate of gestation are available only from 2007 (17); see Technical Notes.)

All of the increase in the total preterm birth rate for 2017–2018 was among infants born late preterm (34–36 weeks), up from 7.17% to 7.28%. The early preterm birth rate (less than 34 weeks) was 2.75% in 2018, essentially unchanged since 2014, but down from 2.93% in 2007 (12,17).

Changes in the distribution of births delivered at term and later (37 weeks and higher) were also observed from 2017 to 2018. The percentage of infants born early term (37–38 weeks) rose by 2% in 2018, from 26.00% to 26.53% and the full term (39–40 weeks) birth rate declined, from 57.49% to 57.24%. From 2007 to 2014, in contrast, the early-term birth rate had generally been on the decline, and the full-term rate had been on the rise (12). Declines were also seen from 2017 to 2018 in late (41 weeks) and post-term (42 and higher) births (Table 20). Similar patterns for 2017–2018 were seen for the three largest race and Hispanic-origin groups (2).

The increase in the preterm birth rate among births to non-Hispanic white mothers between 2017 and 2018 (9.05% to 9.09%) was not statistically significant, but rates rose among births to non-Hispanic black (from 13.93% to 14.13%), and Hispanic mothers (9.62% to 9.73%) (Table 20). For 2018, preterm birth rates for the race and Hispanic-origin groups ranged from a high of 14.13% among births to non-Hispanic black mothers to a low of 8.57% among non-Hispanic Asian mothers (Table 13). Preterm levels for the Hispanic subgroups ranged from 9.19% (Cuban) to 10.87% (Puerto Rican) (Table 14).

It can also be important to analyze births in singleton deliveries separately from all births because of the shorter average gestations of multiple births and their accordant potential influence on preterm birth rates (also see "Multiple births" in this report). The 2018 preterm birth rate for singleton births was 8.24%, a 1% increase over the 2017 level of 8.13%, and a 5% rise from 2015 (7.82%). From 2007 through 2014, the singleton preterm rate had declined from 8.59% to 7.74% (Table) (12).

Increases in preterm birth rates were seen in nine states between 2017 and 2018: Alabama, Colorado, Illinois, Indiana, Iowa, Mississippi, Nebraska, North Dakota, and Texas. Rates declined in two states (Nevada and Oregon) for this period. See Table I–19 for 2018 data.

Birthweight

The percentage of infants born low birthweight (LBW) was unchanged for 2017–2018 at 8.28%. The LBW rate (the percentage of infants born at less than 2,500 grams or 5 pounds, 8 ounces) has risen 4% since the most recent low in 2014 (8.00%) and is the highest rate reported since the 2006 peak (8.26%) (12) (Tables 22 and 23). From 1990 to 2006, LBW levels rose nearly 20%, but then declined from 2007 to 2012 (7.99%) (12).

The very low birthweight (VLBW, less than 1,500 grams) rate declined to 1.38% in 2018 from 1.41% in 2017. The VLBW rate had been essentially stable at 1.40%–1.41% for 2013–2017, and is down from a high of 1.49% for 2005–2007 (10). The percentage of moderately low birthweight infants (1,500–2,499 grams) was 6.90% in 2018, a non-statistically significant increase from 2017 (6.87%); moderately low birthweight levels for 2016–2018 have matched or surpassed the peak reported for 2006 (6.77%) (Table 22) (12).

Between 2017 and 2018, LBW rates declined among births to non-Hispanic white women (from 7.00% to 6.91%), but rose for the second straight year for births to non-Hispanic black women (from 13.68% in 2016 to 13.89% in 2017, and 14.07% in 2018). The increase in LBW among births to Hispanic women (7.43% in 2017 to 7.49% in 2018) was not statistically significant, but the rate for this group is up from 7.32% in 2016. See Tables 13, 14, I–21, and I–22 for 2018 VLBW and LBW rates by race and Hispanic origin and by state.

The LBW rate among singleton births only rose from 6.56% to 6.60% from 2017 to 2018 (15); the increase was among infants born moderately low birthweight (see Table for 2018 data and "Multiple births"). It can be informative to examine births in singleton deliveries separately because multiple births tend to be born smaller than singletons, and changes in multiple-birth incidence can influence overall LBW levels.

Multiple births

The 2018 twin birth rate was 32.6 twins per 1,000 births, a 2% decline from the 2017 rate of 33.3. The twinning rate (births in twin deliveries per 1,000 total births) rose 76% from 1980 to 2009 (from 18.9 to 33.2 per 1,000), was generally stable from 2009 through 2012, and then rose for 2013 and 2014; the 2014 rate of 33.9 was the highest ever reported (Tables 24 and 25) (12).

The triplet and higher-order multiple birth rate (triplet/+) was 93.0 per 100,000 births for 2018, an 8% decline from 2017 (101.6) and down 52% from the 1998 peak (193.5) (12). The triplet/+ birth rate (number of triplets, quadruplets, and quintuplets and other higher-order multiples per 100,000 births), rose more than 400% from 1980 to 1998 (Tables 24 and 25; Figure 1).

There were 123,536 infants born in twin deliveries in 2018, a decline of 4% from the number in 2017 (Table 25). The number of triplet/+ births in 2018, 3,525, was the lowest number reported since 1991 and less than one-half of the highest number reported (7,663 triplet/+ births in 2003) (12). In 2018, triplet/+ births included 3,400 triplets, 115 quadruplets, and 10 quintuplets and higher-order multiple births. See Table for the number of births and percentages of preterm and LBW births by specified plurality.

Table. Gestational age and birthweight characteristics, by plurality: United States, 2018

Plurality	Number of births	Early preterm[1]	Preterm[2]	Very low birthweight[3]	Low birthweight[4]
		Percent			
All births	3,791,712	2.75	10.02	1.38	8.28
Singleton	3,664,651	2.12	8.24	1.09	6.60
Twin	123,536	19.52	60.32	9.07	55.62
Triplet	3,400	63.09	98.32	33.60	94.77
Quadruplet	115	82.61	97.39	50.44	98.23
Quintuplet and higher-order multiples	10	100.00	100.00	100.00	100.00

[1]Less than 34 completed weeks of gestation.
[2]Less than 37 completed weeks of gestation.
[3]Less than 1,500 grams.
[4]Less than 2,500 grams.

SOURCE: NCHS, National Vital Statistics System, Natality.

Twin birth rates declined among non-Hispanic white women by 3% (from 35.5 to 34.4 per 1,000), but were largely stable among non-Hispanic black (41.0 to 40.8) and Hispanic (24.5 to 24.4) women. Triplet/+ birth rates declined 13% among non-Hispanic white women (116.6 per 100,000 to 102.2) but did not decline significantly among either non-Hispanic black (119.7 to 119.2) or Hispanic women (68.3 to 64.7) (Table 24; see also Tables 13 and 14 for 2018 twin and triplet/+ rates for additional race and Hispanic-origin groups). For state-specific twin and triplet/+ rates, see Table I–23.

References

1. Hamilton BE, Martin JA, Osterman MJK, Rossen LM. Births: Provisional data for 2018. Vital Statistics Rapid Release; no 7. Hyattsville, MD: National Center for Health Statistics. May 2019. Available from: https://www.cdc.gov/nchs/data/vsrr/vsrr-007-508.pdf.
2. Martin JA, Hamilton BE, Osterman MJK. Births in the United States, 2018. NCHS Data Brief, no 346. Hyattsville, MD: National Center for Health Statistics. 2019.
3. Office of Management and Budget. Revisions to the standards for the classification of federal data on race and ethnicity. Fed Regist 62(210):58782–90. 1997.
4. Martin JA, Hamilton BE, Osterman MJK, Driscoll AK, Drake P. Births: Final data for 2016. National Vital Statistics Reports; vol 67 no 1. Hyattsville, MD: National Center for Health Statistics. 2018. Available from: https://www.cdc.gov/nchs/data/nvsr/nvsr67/nvsr67_01.pdf.
5. National Center for Health Statistics. Vital statistics data available online. Birth data files and CD-ROM. Hyattsville, MD: National Center for Health Statistics. Available from: https://www.cdc.gov/nchs/data_access/VitalStatsOnline.htm.
6. National Center for Health Statistics. NCHS data release and access policy for micro-data and compressed vital statistics files. Available from: https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.
7. Centers for Disease Control and Prevention. CDC WONDER. Natality information: Live births. Available from: https://wonder.cdc.gov/Natality.html.
8. Martin JA, Wilson EC, Osterman MJK, Saadi EW, Sutton SR, Hamilton BE. Assessing the quality of medical and health data from the 2003 birth certificate revision: Results from two states. National Vital Statistics Reports; vol 62 no 2. Hyattsville, MD: National Center for Health Statistics. 2013.
9. Gregory ECW, Martin JA, Argov EL, Osterman MJK. Assessing the quality of medical and health data from the 2003 birth certificate revision: Results from New York City. National Vital Statistics Reports; vol 68 no 8. Hyattsville, MD: National Center for Health Statistics. 2019.
10. National Center for Health Statistics. User guide to the 2018 natality public use file. Hyattsville, MD. Available from: https://www.cdc.gov/nchs/data_access/Vitalstatsonline.htm.
11. National Center for Health Statistics. 2003 revisions of the U.S. Standard Certificates of Live Birth, Death, and Fetal Death. Available from: https://www.cdc.gov/nchs/nvss/vital_certificate_revisions.htm.
12. Martin JA, Hamilton BE, Osterman MJK, Driscoll AK, Mathews TJ. Births: Final data for 2015. National Vital Statistics Reports; vol 66 no 1. Hyattsville, MD: National Center for Health Statistics. 2017. Available from: https://www.cdc.gov/nchs/data/nvsr/nvsr66/nvsr66_01.pdf.
13. National Center for Health Statistics. Vital statistics of the United States, 2003, volume I, natality. Available from: https://www.cdc.gov/nchs/products/vsus/vsus_1980_2003.htm.
14. Ventura SJ, Hamilton BE, Mathews TJ. National and state patterns of teen births in the United States, 1940–2013. National Vital Statistics Reports; vol 63 no 4. Hyattsville, MD: National Center for Health Statistics. 2014. Available from: https://www.cdc.gov/nchs/data/nvsr/nvsr63/nvsr63_04.pdf.
15. Martin JA, Hamilton BE, Osterman MJK, Driscoll AK, Drake P. Births: Final data for 2017. National Vital Statistics Reports; vol 67 no 8. Hyattsville, MD: National Center for Health Statistics. 2018. Available from: https://www.cdc.gov/nchs/data/nvsr/nvsr67/nvsr67_08-508.pdf.
16. Hamilton BE, Rossen LM, Branum AM. Teen birth rates for urban and rural areas in the United States, 2007–2015. NCHS Data Brief, no 264. Hyattsville, MD: National Center for Health Statistics. 2016. Available from: https://www.cdc.gov/nchs/data/databriefs/db264.pdf.
17. Martin JA, Osterman MJK, Kirmeyer SE, Gregory ECW. Measuring gestational age in vital statistics data: Transitioning to the obstetric estimate. National Vital Statistics Reports; vol 64 no 5. Hyattsville, MD: National Center for Health Statistics. 2015.
18. National Center for Health Statistics. Report of the Panel to Evaluate the U.S. Standard Certificates. 2000.
19. Ramirez RR, Ennis SR. Item nonresponse, allocation, and data editing of the question on Hispanic origin in the American Community Survey (ACS): 2000 to 2007. Population Division Working Paper No. 86. Washington, DC: U.S. Census Bureau. 2010.
20. Office of Management and Budget. Race and ethnic standards for federal statistics and administrative reporting. Statistical Policy Directive no. 15. 1977.
21. U.S. Census Bureau. 2018 population estimates. Annual state resident population estimates for 6 race groups (5 race alone groups and two or more races) by age, sex, and Hispanic origin: April 1, 2010 to July 1, 2018. 2019. Available from: https://www2.census.gov/programs-surveys/popest/datasets/2010-2018/state/asrh/sc-est2018-alldata6.csv.
22. U.S. Census Bureau. International data base. Population by single years of age and sex, 2018. 2019. Available from: https://www.census.gov/data-tools/demo/idb/informationGateway.php.
23. U.S. Census Bureau. 2018 population estimates. Annual estimates of the resident population by single year of age and sex for the United States, states, and Puerto Rico Commonwealth: April 1, 2010 to July 1, 2018. 2019. Available from: http://factfinder.census.gov/bkmk/table/1.0/en/PEP/2018/PEPSYASEX/0400000US72?#.
24. U.S. Census Bureau. The Data Web: DataFerrett. Current Population Survey. 2017 March Annual Social and Economic Supplement. 2018. Available from: https://dataferrett.census.gov/.
25. U.S. Census Bureau. The Data Web: DataFerrett. Current Population Survey. 2018 March Annual Social and Economic Supplement. 2019. Available from: https://dataferrett.census.gov/.
26. Ventura SJ, Bachrach CA. Nonmarital childbearing in the United States, 1940–99. National Vital Statistics Reports; vol 48 no 16. Hyattsville, MD: National Center for Health Statistics. 2000.

27. U.S. Census Bureau. American Community Survey (ACS), 2018 1-year estimates (st31002_2018_010_flags), by sex, age, nativity, and Hispanic origin. Population estimates for 2018 based on unpublished tabulations. [Forthcoming.]
28. Parker JD, Talih M, Malec DJ, Beresovsky V, Carroll M, Gonzalez Jr JF, et al. National Center for Health Statistics Data Presentation Standards for Proportions. National Center for Health Statistics. Vital Health Stat 2(175). 2017. Available from: https://www.cdc.gov/nchs/data/series/sr_02/sr02_175.pdf.
29. National Center for Health Statistics. Guide to completing the facility worksheets for the certificate of live birth and report of fetal death (2003 revision). 2016 update. Available from: https://www.cdc.gov/nchs/data/dvs/GuidetoCompleteFacilityWks.pdf.

List of Detailed Tables

Report tables

List of Internet Tables

(Available from: https://www.cdc.gov/nchs/data/nvsr/nvsr68/nvsr68_13_tables-508.pdf)

Table 1. Births and birth rates: United States, 2010–2018, and by race and Hispanic origin, United States, 2016–2018

[Birth rates are births per 1,000 population in specified group. Fertility rates are births per 1,000 women aged 15–44 years in specified group. Populations based on counts enumerated as of April 1 for census years and estimated as of July 1 for all other years]

Race and Hispanic origin and year	Number	Birth rate	Fertility rate
All races and origins[1]			
2018	3,791,712	11.6	59.1
2017	3,855,500	11.8	60.3
2016	3,945,875	12.2	62.0
2015	3,978,497	12.4	62.5
2014	3,988,076	12.5	62.9
2013	3,932,181	12.4	62.5
2012	3,952,841	12.6	63.0
2011	3,953,590	12.7	63.2
2010	3,999,386	13.0	64.1
Non-Hispanic, single race[2]			
White:			
2018	1,956,413	10.0	56.3
2017	1,992,461	10.2	57.2
2016	2,056,332	10.5	58.8
Black:			
2018	552,029	13.6	62.0
2017	560,715	13.9	63.1
2016	558,622	14.0	63.3
American Indian or Alaska Native:			
2018	29,092	12.2	57.7
2017	29,957	12.6	59.5
2016	31,452	13.3	62.7
Asian:			
2018	240,798	13.2	55.6
2017	249,250	13.8	58.0
2016	254,471	14.6	61.1
Native Hawaiian or Other Pacific Islander:			
2018	9,476	16.6	73.0
2017	9,426	16.7	72.8
2016	9,342	16.8	72.9
Hispanic[3]			
2018	886,210	14.8	65.9
2017	898,764	15.2	67.6
2016	918,447	16.0	70.6

[1]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[2]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes in this report. Single race is defined as only one race reported on the birth certificate.
[3]Includes all persons of Hispanic origin of any race; see Technical Notes.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 2. Birth rates, by age of mother: United States, 2010–2018, and by age and race and Hispanic origin of mother, United States, 2016–2018

[Total fertility rates are sums of birth rates for 5-year age groups multiplied by 5. Birth rates are births per 1,000 women in specified group. Populations based on counts enumerated as of April 1 for census years and estimated as of July 1 for all other years]

Year and race and Hispanic origin	Total fertility rate	Age of mother									
			15–19								
		10–14	Total	15–17	18–19	20–24	25–29	30–34	35–39	40–44	45–49[1]
All races and origins[2]											
2018	1,729.5	0.2	17.4	7.2	32.3	68.0	95.3	99.7	52.6	11.8	0.9
2017	1,765.5	0.2	18.8	7.9	35.1	71.0	98.0	100.3	52.3	11.6	0.9
2016	1,820.5	0.2	20.3	8.8	37.5	73.8	102.1	102.7	52.7	11.4	0.9
2015	1,843.5	0.2	22.3	9.9	40.7	76.8	104.3	101.5	51.8	11.0	0.8
2014	1,862.5	0.3	24.2	10.9	43.8	79.0	105.8	100.8	51.0	10.6	0.8
2013	1,857.5	0.3	26.5	12.3	47.1	80.7	105.5	98.0	49.3	10.4	0.8
2012	1,880.5	0.4	29.4	14.1	51.4	83.1	106.5	97.3	48.3	10.4	0.7
2011	1,894.5	0.4	31.3	15.4	54.1	85.3	107.2	96.5	47.2	10.3	0.7
2010	1,931.0	0.4	34.2	17.3	58.2	90.0	108.3	96.5	45.9	10.2	0.7
Non-Hispanic, single race[3]											
White:											
2018	1,640.0	0.1	12.1	4.1	23.6	57.8	92.9	103.1	51.1	10.2	0.7
2017	1,666.5	0.1	13.2	4.6	26.0	59.8	95.3	103.7	50.6	9.9	0.7
2016	1,719.0	0.1	14.3	5.2	27.7	62.4	99.3	106.2	51.1	9.7	0.7
Black:											
2018	1,792.0	0.4	26.3	11.9	46.9	90.0	97.1	84.1	47.1	12.3	1.1
2017	1,824.5	0.4	27.5	12.5	49.8	94.2	99.5	84.5	46.1	11.7	1.0
2016	1,832.5	0.5	29.3	13.7	52.5	95.8	100.8	83.0	45.0	11.1	1.0
American Indian or Alaska Native:											
2018	1,650.5	0.2	29.7	13.5	52.9	87.7	94.4	73.2	36.1	8.3	0.5
2017	1,702.0	0.5	32.9	15.7	58.1	93.6	96.4	72.1	36.3	8.2	0.4
2016	1,794.5	0.5	35.1	16.8	62.6	97.2	103.4	77.1	37.1	8.2	0.3
Asian:											
2018	1,525.0	*	2.8	1.0	5.4	24.1	73.0	116.5	70.7	16.1	1.8
2017	1,597.0	*	3.3	1.1	6.5	26.6	79.6	120.1	71.5	16.5	1.8
2016	1,690.5	*	3.9	1.4	7.5	28.9	87.0	127.4	72.8	16.4	1.7
Native Hawaiian or Other Pacific Islander:											
2018	2,106.5	*	26.5	8.2	53.7	118.5	118.9	91.9	49.2	15.4	*
2017	2,085.5	*	25.5	8.7	50.6	113.3	115.7	94.0	54.6	13.1	*
2016	2,076.5	*	28.6	11.0	55.3	109.5	115.5	96.0	49.9	14.7	*
Hispanic[4]											
2018	1,959.0	0.3	26.7	12.4	48.5	89.3	108.9	96.6	54.5	14.5	1.0
2017	2,006.5	0.3	28.9	13.6	52.7	93.8	111.8	96.4	54.6	14.5	1.0
2016	2,092.5	0.4	31.9	15.6	57.3	98.4	117.4	99.2	55.8	14.5	0.9

* Estimate does not meet NCHS standards of reliability.
[1]Birth rates computed by relating births to women aged 45 and over to women aged 45–49; see Technical Notes in this report.
[2]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[3]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes. Single race is defined as only one race reported on the birth certificate.
[4]Includes all persons of Hispanic origin of any race; see Technical Notes.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 3. Births, by age (years) of mother, live-birth order, and race and Hispanic origin of mother: United States, 2018

[Live-birth order refers to number of children born alive to mother]

Live-birth order and race and Hispanic origin of mother	All ages	Under 15	15–19						20–24	25–29	30–34	35–39	40–44	45–49	50–54
			Total	15	16	17	18	19							
All races and origins[1]	3,791,712	1,736	179,871	4,953	13,088	26,250	50,088	85,492	726,175	1,099,491	1,090,697	566,786	117,381	8,616	959
1st child	1,433,915	1,712	151,440	4,842	12,458	23,863	42,670	67,607	383,388	409,940	332,492	127,893	24,364	2,375	311
2nd child	1,208,762	16	24,506	91	586	2,196	6,535	15,098	231,051	362,863	370,337	185,345	32,257	2,154	233
3rd child	651,049	1	3,122	4	18	136	714	2,250	81,852	200,639	214,401	125,246	24,191	1,457	140
4th child	283,877	–	332	–	4	7	45	276	21,454	81,158	99,793	64,469	15,597	957	117
5th child	111,885	–	42	–	–	1	14	27	4,966	27,694	39,788	30,271	8,481	573	70
6th child	47,066	–	12	–	1	–	1	10	1,061	9,304	17,144	14,609	4,557	343	36
7th child	21,456	–	4	–	–	–	–	4	248	3,034	7,649	7,646	2,644	208	23
8th child and over	23,095	–	13	–	–	–	2	11	237	1,695	6,061	9,687	4,869	505	28
Not stated	10,607	7	400	16	21	47	107	209	1,918	3,164	3,032	1,620	421	44	1
Non-Hispanic, single race[2]															
White	1,956,413	337	64,917	1,244	3,555	8,237	17,848	34,033	326,575	576,811	624,015	304,062	55,417	3,875	404
1st child	768,672	330	55,599	1,224	3,433	7,619	15,560	27,763	179,304	238,135	205,709	74,987	13,255	1,203	150
2nd child	651,254	4	8,189	19	109	573	2,063	5,425	103,311	192,901	223,283	105,807	16,587	1,053	119
3rd child	320,395	–	880	1	4	28	172	675	33,444	94,689	115,044	65,194	10,494	596	54
4th child	125,198	–	82	–	–	4	9	69	7,643	34,360	47,303	29,496	5,981	300	33
5th child	45,494	–	17	–	–	1	7	9	1,441	10,600	17,486	12,635	3,094	201	20
6th child	18,970	–	2	–	–	–	–	2	274	3,105	7,456	6,294	1,709	117	13
7th child	9,173	–	1	–	–	–	–	1	61	904	3,380	3,583	1,146	89	9
8th child and over	11,599	–	3	–	–	–	1	2	94	524	2,555	5,186	2,943	288	6
Not stated	5,658	3	144	–	9	12	36	87	1,003	1,593	1,799	880	208	28	–
Black	552,029	554	37,715	1,276	3,013	5,709	10,523	17,194	137,974	166,802	124,206	67,268	15,988	1,348	174
1st child	194,089	546	31,512	1,234	2,856	5,172	8,918	13,332	70,246	48,551	27,559	12,438	2,832	337	68
2nd child	157,412	4	5,166	31	146	488	1,375	3,126	42,064	52,505	35,953	17,664	3,726	299	31
3rd child	101,714	–	816	1	5	36	184	590	17,514	35,129	28,655	15,835	3,503	238	24
4th child	52,115	–	109	–	–	1	18	90	5,666	17,876	16,225	9,566	2,490	167	16
5th child	23,576	–	9	–	–	–	3	6	1,606	7,477	7,858	5,172	1,354	90	10
6th child	10,948	–	3	–	–	–	1	2	396	3,024	3,916	2,739	796	68	6
7th child	5,139	–	2	–	–	–	–	2	87	1,099	1,931	1,508	460	43	9
8th child and over	5,504	–	5	–	–	–	–	5	67	664	1,746	2,140	772	100	10
Not stated	1,532	4	93	10	6	12	24	41	328	477	363	206	55	6	–
American Indian or Alaska Native	29,092	19	2,578	103	229	362	743	1,141	7,841	9,146	6,105	2,787	585	29	2
1st child	8,684	19	2,107	99	215	328	603	862	3,454	1,931	815	291	60	6	1
2nd child	7,575	–	406	4	12	31	126	233	2,678	2,605	1,318	481	79	8	–
3rd child	5,586	–	50	–	1	3	12	34	1,183	2,261	1,411	572	107	2	–
4th child	3,422	–	9	–	–	–	1	8	374	1,302	1,113	527	92	5	–
5th child	1,838	–	1	–	–	–	–	1	108	635	688	322	82	2	–
6th child	972	–	1	–	–	–	–	1	19	246	407	235	63	–	1
7th child	475	–	–	–	–	–	–	–	3	87	170	164	49	2	–
8th child and over	462	–	1	–	–	–	–	1	3	49	170	183	52	4	–
Not stated	78	–	3	–	1	–	1	1	19	30	13	12	1	–	–

See footnotes at end of table.

Table 3. Births, by age (years) of mother, live-birth order, and race and Hispanic origin of mother: United States, 2018—Con.

[Live-birth order refers to number of children born alive to mother]

Live-birth order and race and Hispanic origin of mother	All ages	Under 15	15–19						20–24	25–29	30–34	35–39	40–44	45–49	50–54
			Total	15	16	17	18	19							
Non-Hispanic, single race[2]—Con.															
Asian	240,798	18	1,444	34	92	182	393	743	14,876	57,810	96,385	57,136	11,951	1,055	123
1st child	109,429	16	1,220	34	84	165	329	608	9,807	34,162	43,369	17,113	3,319	383	40
2nd child	90,179	2	190	–	7	14	57	112	3,749	17,094	38,592	25,573	4,603	335	41
3rd child	28,123	–	26	–	–	2	5	19	1,015	4,552	10,088	9,844	2,413	171	14
4th child	8,367	–	5	–	1	–	1	3	211	1,328	2,836	2,925	958	91	13
5th child	2,595	–	–	–	–	–	–	–	60	398	820	937	346	28	6
6th child	994	–	–	–	–	–	–	–	10	134	342	349	133	21	5
7th child	378	–	–	–	–	–	–	–	4	32	119	143	68	10	2
8th child and over	448	–	–	–	–	–	–	–	3	28	116	188	95	16	2
Not stated	285	–	3	–	–	1	1	1	17	82	103	64	16	–	–
Native Hawaiian or Other Pacific Islander	9,476	3	516	10	23	62	159	262	2,482	2,887	2,189	1,097	290	12	–
1st child	2,791	3	424	10	22	57	135	200	1,151	712	336	124	39	2	–
2nd child	2,264	–	72	–	1	4	21	46	797	762	419	178	35	1	–
3rd child	1,773	–	14	–	–	–	3	11	349	666	488	207	47	2	–
4th child	1,196	–	1	–	–	–	–	1	124	426	394	193	56	2	–
5th child	672	–	1	–	–	–	–	1	30	190	272	137	41	1	–
6th child	364	–	–	–	–	–	–	–	10	84	136	109	25	–	–
7th child	180	–	–	–	–	–	–	–	–	31	80	50	17	2	–
8th child and over	184	–	–	–	–	–	–	–	–	11	50	91	30	2	–
Not stated	52	–	4	–	–	1	–	3	21	5	14	8	–	–	–
Hispanic[3]															
Total Hispanic	886,210	730	65,122	2,044	5,574	10,567	18,369	28,568	209,701	253,977	208,193	117,383	29,192	1,787	125
1st child	303,049	724	54,158	2,003	5,283	9,490	15,360	22,022	105,410	75,046	44,923	18,465	3,936	350	37
2nd child	264,775	5	9,498	35	278	1,000	2,635	5,550	70,061	86,821	61,610	30,255	6,164	341	20
3rd child	174,777	1	1,225	1	7	61	316	840	25,259	57,438	53,376	30,248	6,881	331	18
4th child	84,987	–	112	–	2	2	14	94	6,660	23,209	29,120	20,017	5,535	312	22
5th child	33,963	–	12	–	–	–	4	8	1,518	7,414	11,388	10,142	3,274	201	14
6th child	13,213	–	4	–	1	–	–	3	315	2,350	4,327	4,425	1,672	117	3
7th child	5,392	–	1	–	–	–	–	1	78	771	1,719	1,960	810	50	3
8th child and over	4,152	–	3	–	–	–	1	2	62	371	1,223	1,578	830	78	7
Not stated	1,902	–	109	5	3	14	39	48	338	557	507	293	90	7	1

– Quantity zero.

[1]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.

[2]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes in this report. Single race is defined as only one race reported on the birth certificate.

[3]Includes all persons of Hispanic origin of any race; see Technical Notes.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 4. Births, by age (years) of mother, live-birth order, and Hispanic origin of mother: United States, 2018

[Live-birth order refers to number of children born alive to mother. Includes births with stated origin of mother only]

Live-birth order and origin of mother	All ages	Under 15	15–19						20–24	25–29	30–34	35–39	40–44	45–49	50–54
			Total	15	16	17	18	19							
Hispanic															
Total	886,210	730	65,122	2,044	5,574	10,567	18,369	28,568	209,701	253,977	208,193	117,383	29,192	1,787	125
1st child	303,049	724	54,158	2,003	5,283	9,490	15,360	22,022	105,410	75,046	44,923	18,465	3,936	350	37
2nd child	264,775	5	9,498	35	278	1,000	2,635	5,550	70,061	86,821	61,610	30,255	6,164	341	20
3rd child	174,777	1	1,225	1	7	61	316	840	25,259	57,438	53,376	30,248	6,881	331	18
4th child	84,987	–	112	–	2	2	14	94	6,660	23,209	29,120	20,017	5,535	312	22
5th child	33,963	–	12	–	–	–	4	8	1,518	7,414	11,388	10,142	3,274	201	14
6th child	13,213	–	4	–	1	–	–	3	315	2,350	4,327	4,425	1,672	117	3
7th child	5,392	–	1	–	–	–	–	1	78	771	1,719	1,960	810	50	3
8th child and over	4,152	–	3	–	–	–	1	2	62	371	1,223	1,578	830	78	7
Not stated	1,902	–	109	5	3	14	39	48	338	557	507	293	90	7	1
Mexican	495,831	401	38,396	1,224	3,276	6,333	10,753	16,810	122,074	143,052	112,535	62,578	15,845	917	33
1st child	160,303	396	31,850	1,201	3,101	5,692	8,999	12,857	59,784	38,468	20,125	7,862	1,671	137	10
2nd child	141,295	5	5,679	20	171	598	1,556	3,334	41,533	47,795	30,176	13,345	2,616	139	7
3rd child	102,882	–	747	1	4	39	180	523	15,295	34,999	31,313	16,686	3,669	169	4
4th child	54,104	–	76	–	–	1	9	66	4,138	14,721	18,713	12,760	3,515	177	4
5th child	22,025	–	4	–	–	–	–	4	935	4,724	7,411	6,637	2,173	137	4
6th child	8,518	–	1	–	–	–	–	1	192	1,434	2,805	2,881	1,123	81	1
7th child	3,499	–	1	–	–	–	–	1	51	488	1,106	1,293	531	29	–
8th child and over	2,586	–	1	–	–	–	-	1	25	236	739	1,018	519	45	3
Not stated	619	–	37	2	–	3	9	23	121	187	147	96	28	3	–
Puerto Rican	71,614	51	5,511	136	419	785	1,534	2,637	19,577	21,578	15,055	7,910	1,824	91	17
1st child	27,045	51	4,636	134	400	718	1,288	2,096	10,207	6,553	3,697	1,542	331	22	6
2nd child	22,147	–	770	2	17	63	217	471	6,218	7,443	4,742	2,461	489	21	3
3rd child	12,705	–	86	–	–	2	21	63	2,286	4,653	3,467	1,794	400	17	2
4th child	5,553	–	3	–	1	–	–	2	623	1,864	1,734	1,038	265	20	6
5th child	2,265	–	3	–	–	–	3	–	149	651	771	533	154	4	–
6th child	954	–	1	–	1	–	–	–	36	242	325	273	75	2	–
7th child	394	–	–	–	–	–	–	–	8	79	141	123	43	–	–
8th child and over	361	–	–	–	–	–	–	–	9	32	135	122	58	5	–
Not stated	190	–	12	–	–	2	5	5	41	61	43	24	9	–	–
Cuban	23,471	2	725	11	48	101	207	358	3,878	7,331	7,426	3,280	770	56	3
1st child	10,976	2	643	11	47	97	187	301	2,623	3,624	2,920	953	186	23	2
2nd child	8,249	–	70	–	1	4	15	50	948	2,553	2,992	1,365	307	14	–
3rd child	2,789	–	9	–	–	–	3	6	216	761	1,022	606	167	8	–
4th child	801	–	–	–	–	–	–	–	46	221	274	205	49	6	–
5th child	260	–	–	–	–	–	–	–	15	62	82	78	22	1	–
6th child	102	–	–	–	–	–	–	–	3	19	39	28	13	–	–
7th child	50	–	–	–	–	–	–	–	–	13	18	12	7	–	–
8th child and over	54	–	–	–	–	–	–	–	4	7	19	11	11	2	–
Not stated	190	–	3	–	–	–	2	1	23	71	60	22	8	2	1

See footnotes at end of table.

Table 4. Births, by age (years) of mother, live-birth order, and Hispanic origin of mother: United States, 2018—Con.

[Live-birth order refers to number of children born alive to mother. Includes births with stated origin of mother only]

Live-birth order and origin of mother	All ages	Under 15	15–19						20–24	25–29	30–34	35–39	40–44	45–49	50–54
			Total	15	16	17	18	19							
Hispanic—Con.															
Dominican	32,072	9	1,655	52	127	248	470	758	6,891	9,990	7,837	4,512	1,073	94	11
1st child	12,726	9	1,459	50	121	229	419	640	4,338	3,931	1,998	791	172	25	3
2nd child	10,861	–	172	2	5	16	44	105	1,921	3,811	3,005	1,600	323	26	3
3rd child	5,826	–	23	–	1	3	7	12	505	1,689	1,896	1,360	327	24	2
4th child	1,794	–	–	–	–	–	–	–	96	391	666	475	150	15	1
5th child	512	–	–	–	–	–	–	–	14	100	157	171	67	3	–
6th child	159	–	–	–	–	–	–	–	5	27	49	60	18	–	–
7th child	63	–	–	–	–	–	–	–	1	13	16	27	6	–	–
8th child and over	51	–	–	–	–	–	–	–	–	4	18	18	8	1	2
Not stated	80	–	1	–	–	–	–	1	11	24	32	10	2	–	–
Central and South American	147,430	147	8,837	301	801	1,473	2,588	3,674	27,040	38,213	40,173	25,755	6,789	441	35
1st child	50,072	147	7,358	293	766	1,298	2,180	2,821	13,735	12,557	10,224	4,860	1,076	102	13
2nd child	47,566	–	1,286	6	33	161	358	728	9,343	13,978	13,290	7,786	1,787	91	5
3rd child	28,914	–	140	–	–	7	31	102	3,105	7,784	9,565	6,567	1,666	82	5
4th child	12,451	–	8	–	1	1	–	6	600	2,722	4,420	3,588	1,044	66	3
5th child	4,832	–	1	–	–	–	–	1	113	730	1,663	1,701	581	38	5
6th child	1,788	–	1	–	–	–	–	1	17	208	535	700	301	25	1
7th child	651	–	–	–	–	–	–	–	5	44	190	250	147	14	1
8th child and over	522	–	1	–	–	–	–	1	14	31	107	191	155	21	2
Not stated	634	–	42	2	1	6	19	14	108	159	179	112	32	2	–
Other and unknown Hispanic	115,792	120	9,998	320	903	1,627	2,817	4,331	30,241	33,813	25,167	13,348	2,891	188	26
1st child	41,927	119	8,212	314	848	1,456	2,287	3,307	14,723	9,913	5,959	2,457	500	41	3
2nd child	34,657	–	1,521	5	51	158	445	862	10,098	11,241	7,405	3,698	642	50	2
3rd child	21,661	1	220	–	2	10	74	134	3,852	7,552	6,113	3,235	652	31	5
4th child	10,284	–	25	–	–	–	5	20	1,157	3,290	3,313	1,951	512	28	8
5th child	4,069	–	4	–	–	–	1	3	292	1,147	1,304	1,022	277	18	5
6th child	1,692	–	1	–	–	–	–	1	62	420	574	483	142	9	1
7th child	735	–	–	–	–	–	–	–	13	134	248	255	76	7	2
8th child and over	578	–	1	–	–	–	1	–	10	61	205	218	79	4	–
Not stated	189	–	14	1	2	3	4	4	34	55	46	29	11	–	–

– Quantity zero.

NOTE: In this table, Hispanic women are classified only by place of origin; non–Hispanic women are not shown; see Technical Notes in this report.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 5. Birth rates, by age of mother, live-birth order, and race and Hispanic origin of mother: United States, 2018

[Rates are births per 1,000 women in specified age and race and Hispanic-origin group. Fertility rate computed by relating total births, regardless of age of mother, to women aged 15–44 years. Populations estimated as of July 1. Live-birth order refers to number of children born alive to mother. Figures for live-birth order not stated are distributed]

Live-birth order and race of mother	Fertility rate	Age of mother (years)									
			15–19								
		10–14	Total	15–17	18–19	20–24	25–29	30–34	35–39	40–44	45–49[1]
All races and origins[2]	59.1	0.2	17.4	7.2	32.3	68.0	95.3	99.7	52.6	11.8	0.9
1st child	22.4	0.2	14.7	6.7	26.4	36.0	35.6	30.5	11.9	2.5	0.3
2nd child	18.9	*	2.4	0.5	5.2	21.7	31.5	33.9	17.3	3.3	0.2
3rd child	10.2	*	0.3	0.0	0.7	7.7	17.4	19.6	11.7	2.4	0.2
4th child	4.4	*	0.0	*	0.1	2.0	7.1	9.1	6.0	1.6	0.1
5th child	1.7	*	0.0	*	0.0	0.5	2.4	3.6	2.8	0.9	0.1
6th and 7th child	1.1	*	*	*	*	0.1	1.1	2.3	2.1	0.7	0.1
8th child and over	0.4	*	*	*	*	0.0	0.1	0.6	0.9	0.5	0.1
Non-Hispanic, single race[3]											
White	56.3	0.1	12.1	4.1	23.6	57.8	92.9	103.1	51.1	10.2	0.7
1st child	22.2	0.1	10.4	3.9	19.7	31.8	38.5	34.1	12.7	2.4	0.2
2nd child	18.8	*	1.5	0.2	3.4	18.3	31.2	36.9	17.8	3.0	0.2
3rd child	9.2	*	0.2	0.0	0.4	5.9	15.3	19.0	11.0	1.9	0.1
4th child	3.6	*	0.0	*	0.0	1.4	5.6	7.8	5.0	1.1	0.1
5th child	1.3	*	*	*	*	0.3	1.7	2.9	2.1	0.6	0.0
6th and 7th child	0.8	*	*	*	*	0.1	0.7	1.8	1.7	0.5	0.0
8th child and over	0.3	*	*	*	*	0.0	0.1	0.4	0.9	0.5	0.0
Black	62.0	0.4	26.3	11.9	46.9	90.0	97.1	84.1	47.1	12.3	1.1
1st child	21.9	0.4	22.0	11.0	37.7	45.9	28.4	18.8	8.8	2.2	0.3
2nd child	17.7	*	3.6	0.8	7.6	27.5	30.7	24.4	12.4	2.9	0.2
3rd child	11.5	*	0.6	0.0	1.3	11.4	20.5	19.4	11.1	2.7	0.2
4th child	5.9	*	0.1	*	0.2	3.7	10.4	11.0	6.7	1.9	0.1
5th child	2.7	*	*	*	*	1.0	4.4	5.3	3.6	1.0	0.1
6th and 7th child	1.8	*	*	*	*	0.3	2.4	4.0	3.0	1.0	0.1
8th child and over	0.6	*	*	*	*	0.0	0.4	1.2	1.5	0.6	0.1
American Indian or Alaska Native	57.7	0.2	29.7	13.5	52.9	87.7	94.4	73.2	36.1	8.3	0.5
1st child	17.3	0.2	24.3	12.6	41.1	38.7	20.0	9.9	3.8	0.9	*
2nd child	15.1	*	4.7	0.9	10.2	30.0	27.0	15.8	6.2	1.1	*
3rd child	11.1	*	0.6	*	1.3	13.3	23.4	16.9	7.4	1.5	*
4th child	6.8	*	*	*	*	4.2	13.4	13.3	6.9	1.3	*
5th child	3.7	*	*	*	*	1.2	6.5	8.3	4.2	1.1	*
6th and 7th child	2.9	*	*	*	*	0.2	3.5	6.9	5.2	1.6	*
8th child and over	0.9	*	*	*	*	*	0.5	2.0	2.4	0.7	*

See footnotes at end of table.

Table 5. Birth rates, by age of mother, live-birth order, and race and Hispanic origin of mother: United States, 2018—Con.

[Rates are births per 1,000 women in specified age and race and Hispanic-origin group. Fertility rate computed by relating total births, regardless of age of mother, to women aged 15–44 years. Populations estimated as of July 1. Live-birth order refers to number of children born alive to mother. Figures for live-birth order not stated are distributed]

Live-birth order and race of mother	Fertility rate	Age of mother (years)									
			15–19								
		10–14	Total	15–17	18–19	20–24	25–29	30–34	35–39	40–44	45–49[1]
Non-Hispanic, single race[3]—Con.											
Asian	55.6	*	2.8	1.0	5.4	24.1	73.0	116.5	70.7	16.1	1.8
1st child	25.2	*	2.4	1.0	4.5	15.9	43.0	52.4	21.2	4.5	0.6
2nd child	20.8	*	0.4	0.1	0.8	6.1	21.6	46.6	31.5	6.1	0.6
3rd child	6.5	*	0.1	*	0.1	1.7	5.8	12.3	12.2	3.3	0.3
4th child	2.0	*	*	*	*	0.3	1.7	3.5	3.7	1.3	0.2
5th child	0.6	*	*	*	*	0.1	0.5	1.0	1.2	0.5	0.1
6th and 7th child	0.3	*	*	*	*	*	0.2	0.6	0.6	0.3	0.1
8th child and over	0.1	*	*	*	*	*	0.0	0.1	0.2	0.1	0.0
Native Hawaiian or Other Pacific Islander	73.0	*	26.5	8.2	53.7	118.5	118.9	91.9	49.2	15.4	*
1st child	21.7	*	21.9	7.8	42.9	55.4	29.7	14.3	5.8	2.1	*
2nd child	17.7	*	3.8	*	8.7	38.4	31.5	18.2	8.2	2.0	*
3rd child	13.7	*	*	*	*	16.8	27.5	20.6	9.5	2.4	*
4th child	9.2	*	*	*	*	6.0	17.4	16.6	8.5	3.0	*
5th child	5.2	*	*	*	*	1.4	7.7	11.3	6.1	2.2	*
6th and 7th child	4.2	*	*	*	*	*	4.7	8.8	7.1	2.2	*
8th child and over	1.4	*	*	*	*	*	*	2.1	4.1	1.6	*
Hispanic[4]											
Total	65.9	0.3	26.7	12.4	48.5	89.3	108.9	96.6	54.5	14.5	1.0
1st child	22.6	0.3	22.3	11.4	38.7	45.0	32.2	20.9	8.6	2.0	0.2
2nd child	19.7	*	3.9	0.9	8.5	29.9	37.3	28.6	14.1	3.1	0.2
3rd child	13.0	*	0.5	0.0	1.2	10.8	24.7	24.8	14.1	3.4	0.2
4th child	6.3	*	0.0	*	0.1	2.8	10.0	13.5	9.3	2.8	0.2
5th child	2.5	*	*	*	*	0.6	3.2	5.3	4.7	1.6	0.1
6th and 7th child	1.4	*	*	*	*	0.2	1.3	2.8	3.0	1.2	0.1
8th child and over	0.3	*	*	*	*	0.0	0.2	0.6	0.7	0.4	0.0

* Estimate does not meet NCHS standards of reliability.

0.0 Quantity more than zero but less than 0.05.

[1]Birth rates computed by relating births to women aged 45 years and over to women aged 45–49 years; see Technical Notes in this report.

[2]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.

[3]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes. Single race is defined as only one race reported on the birth certificate.

[4]Includes all persons of Hispanic origin of any race; see Technical Notes.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 6. Births, by race and Hispanic origin of mother: United States, each state and territory, 2018

[By place of residence]

Area	All races and origins[2]	Non-Hispanic, single race[1]					Hispanic[3]
		White	Black	American Indian or Alaska Native	Asian	Native Hawaiian or Other Pacific Islander	
United States[4]	3,791,712	1,956,413	552,029	29,092	240,798	9,476	886,210
Alabama	57,761	33,776	17,597	148	903	46	4,403
Alaska	10,086	5,057	280	1,873	641	299	807
Arizona	80,723	32,805	4,305	4,155	2,908	248	34,084
Arkansas	37,018	23,609	6,966	220	775	498	4,099
California	454,920	123,139	22,380	1,411	68,444	1,732	211,271
Colorado	62,885	36,466	3,032	352	2,496	155	17,817
Connecticut	34,725	18,488	4,423	38	2,232	5	8,762
Delaware	10,621	5,171	2,773	10	634	4	1,710
District of Columbia	9,212	3,040	4,252	15	444	2	1,296
Florida	221,542	95,868	48,174	261	6,996	152	67,201
Georgia	126,172	55,676	43,746	102	5,768	104	17,432
Hawaii	16,972	3,288	424	33	4,366	1,706	2,580
Idaho	21,403	16,574	233	220	348	65	3,549
Illinois	144,815	77,244	24,482	97	9,452	32	30,362
Indiana	81,646	59,520	10,242	73	2,382	59	7,867
Iowa	37,785	29,327	2,615	152	1,176	149	3,694
Kansas	36,261	25,323	2,575	151	1,228	66	5,977
Kentucky	53,922	43,317	4,950	68	1,144	79	3,226
Louisiana	59,615	30,458	22,119	299	1,156	32	4,717
Maine	12,311	11,022	546	96	202	3	224
Maryland	71,080	29,585	21,893	83	4,928	31	12,470
Massachusetts	69,109	39,663	6,826	53	6,183	23	13,810
Michigan	110,032	74,777	20,558	412	4,395	34	7,139
Minnesota	67,344	46,014	8,207	983	5,298	57	4,991
Mississippi	37,000	18,597	15,797	221	411	17	1,666
Missouri	73,269	53,697	10,589	140	1,698	199	4,409
Montana	11,513	9,224	58	1,162	112	15	558
Nebraska	25,488	17,645	1,739	318	925	24	4,155
Nevada	35,682	13,021	4,564	280	2,613	340	13,307
New Hampshire	11,995	10,317	241	7	472	6	745
New Jersey	101,223	45,500	13,886	40	11,452	27	27,597
New Mexico	23,039	6,450	387	2,590	409	13	12,783
New York	226,238	110,840	33,145	395	24,383	50	51,755
North Carolina	118,954	63,514	27,670	1,448	4,834	151	18,360
North Dakota	10,636	7,816	609	828	250	16	635
Ohio	135,134	97,423	22,201	96	4,285	73	7,432
Oklahoma	49,800	28,444	4,136	4,557	1,306	214	7,545
Oregon	42,188	28,265	959	388	2,260	309	7,993
Pennsylvania	135,673	90,862	17,779	74	6,207	54	15,826
Rhode Island	10,506	6,008	783	36	519	5	2,756
South Carolina	56,669	31,890	16,681	106	1,172	50	5,255
South Dakota	11,893	8,481	416	1,645	224	7	661
Tennessee	80,751	53,256	15,921	79	1,877	69	7,824
Texas	378,624	125,549	48,144	721	19,850	487	179,142
Utah	47,209	34,303	521	418	1,131	468	8,133
Vermont	5,432	4,934	118	11	152	1	121
Virginia	99,843	54,798	20,860	157	7,625	103	14,397
Washington	86,085	49,019	3,922	1,166	8,729	1,159	16,073
West Virginia	18,248	16,621	626	14	176	2	378
Wisconsin	64,098	45,654	6,622	678	3,155	29	6,365
Wyoming	6,562	5,078	57	212	72	7	851

See footnotes at end of table.

Table 6. Births, by race and Hispanic origin of mother: United States, each state and territory, 2018—Con.

[By place of residence]

Area	All races and origins[2]	Non-Hispanic, single race[1]					Hispanic[3]
		White	Black	American Indian or Alaska Native	Asian	Native Hawaiian or Other Pacific Islander	
Puerto Rico	21,424	480	49	3	30	–	20,837
Virgin Islands	---	---	---	---	---	---	---
Guam	3,165	170	23	6	719	2,096	25
American Samoa	---	---	---	---	---	---	---
Northern Marianas	566	13	–	–	162	377	–

– Quantity zero.
--- Data not available.
[1]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes in this report. Single race is defined as only one race reported on the birth certificate.
[2]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[3]Includes all persons of Hispanic origin of any race; see Technical Notes.
[4]Excludes data for the territories.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 7. Births, by Hispanic origin of mother: United States, each state and territory, 2018

[By place of residence]

Area	Total	Mexican	Puerto Rican	Cuban	Dominican	Central and South American	Other and unknown Hispanic
United States[1]	886,210	495,831	71,614	23,471	32,072	147,430	115,792
Alabama	4,403	2,517	243	50	38	1,440	115
Alaska	807	427	117	15	41	93	114
Arizona	34,084	29,499	639	218	68	1,389	2,271
Arkansas	4,099	2,893	148	31	17	669	341
California	211,271	155,578	1,951	795	160	17,538	35,249
Colorado	17,817	11,135	522	191	43	1,393	4,533
Connecticut	8,762	715	4,261	114	837	2,354	481
Delaware	1,710	605	441	18	62	494	90
District of Columbia	1,296	160	38	13	47	820	218
Florida	67,201	10,931	13,649	14,833	3,085	22,981	1,722
Georgia	17,432	10,082	1,296	337	325	4,540	852
Hawaii	2,580	753	871	25	19	174	738
Idaho	3,549	2,718	76	15	7	223	510
Illinois	30,362	24,549	2,219	255	123	2,541	675
Indiana	7,867	5,483	515	71	62	1,161	575
Iowa	3,694	2,726	137	44	15	612	160
Kansas	5,977	4,331	196	50	15	785	600
Kentucky	3,226	1,497	257	362	27	765	318
Louisiana	4,717	1,181	202	132	138	2,317	747
Maine	224	72	60	11	12	55	14
Maryland	12,470	1,688	721	126	400	8,535	1,000
Massachusetts	13,810	484	4,548	146	2,978	3,972	1,682
Michigan	7,139	4,121	526	127	106	820	1,439
Minnesota	4,991	3,319	219	60	30	1,105	258
Mississippi	1,666	861	115	19	26	513	132
Missouri	4,409	2,575	263	119	45	844	563
Montana	558	342	27	6	2	40	141
Nebraska	4,155	2,742	80	121	9	996	207
Nevada	13,307	9,849	418	457	62	1,578	943
New Hampshire	745	94	219	24	121	151	136
New Jersey	27,597	4,251	6,179	710	5,194	9,968	1,295
New Mexico	12,783	6,605	102	58	14	185	5,819
New York	51,755	7,020	11,090	667	12,786	16,534	3,658
North Carolina	18,360	9,637	1,734	352	506	5,114	1,017
North Dakota	635	400	69	12	6	68	80
Ohio	7,432	3,025	1,900	126	195	1,651	535
Oklahoma	7,545	5,726	249	54	19	849	648
Oregon	7,993	6,760	196	74	13	679	271
Pennsylvania	15,826	2,181	7,444	278	2,506	2,226	1,191
Rhode Island	2,756	135	691	19	943	829	139
South Carolina	5,255	2,499	605	122	121	1,518	390
South Dakota	661	354	58	12	9	151	77
Tennessee	7,824	4,188	448	142	76	2,448	522
Texas	179,142	124,114	2,725	1,571	375	16,160	34,197
Utah	8,133	5,033	174	35	66	1,552	1,273
Vermont	121	27	27	3	5	37	22
Virginia	14,397	3,024	1,276	223	210	4,439	5,225
Washington	16,073	12,045	521	143	45	1,459	1,860
West Virginia	378	149	63	12	8	80	66
Wisconsin	6,365	4,208	1,059	69	50	523	456
Wyoming	851	523	30	4	5	62	227
Puerto Rico	20,837	33	20,261	11	413	108	11
Virgin Islands	---	---	---	---	---	---	---
Guam	25	12	2	-	2	4	5
American Samoa	---	---	---	---	---	---	---
Northern Marianas	–	–	–	–	–	–	–

--- Data not available.
– Quantity zero.
[1]Excludes data for the territories.

NOTE: In this table, Hispanic women are classified only by place of origin; non-Hispanic women are not shown; see Technical Notes in this report.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 8. Birth rates, by age of mother: United States, each state and territory, 2018

[By place of residence. Fertility rates are births per 1,000 women aged 15-44 years; total fertility rates are sums of birth rates for 5-year age groups multiplied by 5; birth rates by age are births per 1,000 women in specified age group estimated in each area. Populations estimated as of July 1]

Area	Birth rate	Fertility rate	Total fertility rate	Age of mother (years)									
				10–14	15–19 years			20–24	25–29	30–34	35–39	40–44	45–49[1]
					Total	15–17	18–19						
United States[2]	11.6	59.1	1,729.5	0.2	17.4	7.2	32.3	68.0	95.3	99.7	52.6	11.8	0.9
Alabama	11.8	60.9	1,786.5	0.2	25.2	10.6	46.7	94.5	107.5	86.3	36.3	6.9	0.4
Alaska	13.7	69.3	1,965.5	*	19.3	5.6	43.9	96.4	109.9	101.7	52.7	12.5	*
Arizona	11.3	58.6	1,722.0	0.1	20.1	8.9	36.2	75.7	97.9	92.1	46.8	10.9	0.8
Arkansas	12.3	64.0	1,879.5	0.3	30.4	12.1	57.7	106.5	112.4	83.9	35.3	6.8	0.3
California	11.5	56.2	1,632.0	0.1	13.6	5.7	25.2	53.6	78.8	98.8	63.8	16.1	1.6
Colorado	11.0	54.1	1,557.0	*	14.3	6.1	26.4	58.2	81.1	93.5	52.1	11.3	0.8
Connecticut	9.7	51.8	1,570.5	*	8.3	3.1	15.2	37.1	82.3	109.1	62.4	13.9	0.9
Delaware	11.0	59.1	1,724.0	*	16.7	7.1	29.4	68.5	93.9	102.9	51.0	10.8	0.8
District of Columbia	13.1	48.8	1,346.5	*	19.3	16.0	21.5	43.0	40.9	71.3	69.6	21.7	3.1
Florida	10.4	57.2	1,674.0	0.2	16.7	6.7	31.8	66.6	92.2	95.1	51.3	11.9	0.8
Georgia	12.0	58.3	1,729.0	0.2	20.6	8.8	38.3	78.3	97.2	92.1	46.3	10.4	0.7
Hawaii	11.9	64.2	1,847.0	*	17.2	7.0	33.5	76.4	95.9	97.9	63.0	17.3	1.5
Idaho	12.2	63.4	1,899.0	*	16.0	5.6	32.5	93.3	120.4	96.3	44.5	8.6	0.6
Illinois	11.4	57.5	1,690.0	0.1	15.8	6.5	30.1	58.8	89.4	104.7	56.3	12.1	0.8
Indiana	12.2	62.8	1,853.5	0.2	21.8	8.3	41.6	84.0	115.8	98.9	41.0	8.5	0.5
Iowa	12.0	63.5	1,897.5	*	15.3	6.6	27.1	65.6	131.2	113.9	44.7	8.2	0.5
Kansas	12.5	64.7	1,915.0	*	20.0	8.0	37.7	77.0	122.1	108.5	45.9	8.9	0.5
Kentucky	12.1	63.5	1,871.5	0.2	27.3	10.6	51.9	97.5	110.5	92.8	38.8	6.8	0.4
Louisiana	12.8	64.3	1,862.5	0.4	27.5	11.2	52.8	99.6	108.5	87.6	40.2	8.3	0.4
Maine	9.2	53.3	1,577.5	*	11.1	4.3	20.9	62.2	94.5	94.9	43.5	8.8	*
Maryland	11.8	59.9	1,745.0	0.2	14.1	6.0	26.0	55.5	91.3	109.3	62.5	14.8	1.3
Massachusetts	10.0	49.5	1,449.5	*	7.2	3.0	12.1	30.4	64.1	105.1	67.0	15.1	0.9
Michigan	11.0	58.5	1,725.0	0.1	15.8	5.9	30.1	63.6	104.0	105.6	45.9	9.4	0.6
Minnesota	12.0	62.7	1,829.0	0.1	10.2	3.8	20.0	51.4	110.9	123.6	57.4	11.2	1.0
Mississippi	12.4	62.8	1,842.0	0.5	27.8	12.1	50.1	105.6	114.6	80.0	33.4	6.2	0.3
Missouri	12.0	62.4	1,818.5	0.2	21.6	8.6	41.0	81.9	110.2	97.7	42.8	8.8	0.5
Montana	10.8	59.6	1,733.0	*	17.2	6.7	33.5	70.3	106.1	99.9	44.1	8.4	*
Nebraska	13.2	68.4	2,031.0	*	16.7	6.7	31.3	68.4	133.6	123.8	52.6	10.3	0.7
Nevada	11.8	60.0	1,752.5	*	20.5	8.2	42.1	85.6	94.4	87.6	49.2	12.1	1.0
New Hampshire	8.8	49.1	1,465.5	*	8.0	2.8	14.8	38.8	84.4	101.9	50.0	9.2	0.7
New Jersey	11.4	60.1	1,784.5	0.1	10.3	4.2	20.2	47.5	87.4	122.6	71.1	16.6	1.3
New Mexico	11.0	58.0	1,700.0	*	25.3	11.1	46.7	83.3	101.0	80.2	40.4	9.0	0.5
New York	11.6	57.9	1,666.5	0.1	11.7	4.8	21.2	52.2	78.2	102.9	68.4	18.1	1.7
North Carolina	11.5	58.4	1,728.5	0.2	18.7	7.9	33.8	75.1	97.9	96.5	47.1	9.6	0.6
North Dakota	14.0	72.2	2,033.5	*	16.4	7.5	28.0	67.9	134.1	125.7	52.1	9.6	*
Ohio	11.6	61.1	1,791.5	0.2	18.9	7.5	35.7	77.6	105.8	101.9	44.7	8.6	0.6
Oklahoma	12.6	64.6	1,884.5	0.2	27.2	10.7	52.4	98.6	114.9	90.8	37.2	7.6	0.4
Oregon	10.1	51.4	1,490.0	*	13.3	5.0	25.7	56.7	81.2	87.8	47.1	11.1	0.7
Pennsylvania	10.6	56.9	1,665.0	0.2	14.1	6.4	24.3	59.3	93.7	103.5	51.3	10.2	0.7
Rhode Island	9.9	50.5	1,493.0	*	11.5	6.1	17.0	42.5	79.3	97.0	54.7	13.1	*
South Carolina	11.1	58.1	1,705.0	0.2	22.0	9.1	40.1	79.1	98.6	90.0	42.3	8.3	0.5
South Dakota	13.5	73.6	2,154.5	*	20.4	9.9	35.6	81.5	144.8	125.6	49.6	8.1	*
Tennessee	11.9	61.1	1,781.0	0.3	25.3	9.5	49.7	88.7	101.7	90.1	41.6	8.0	0.5
Texas	13.2	63.4	1,870.0	0.3	25.3	11.4	47.1	86.0	104.7	96.7	49.1	11.1	0.8
Utah	14.9	68.4	2,026.5	*	13.1	4.9	26.2	78.3	132.7	117.3	51.6	11.4	0.8
Vermont	8.7	47.2	1,443.5	*	8.8	2.5	15.2	37.8	85.5	96.9	49.6	9.4	*
Virginia	11.7	59.1	1,733.5	0.2	14.3	5.7	26.2	61.4	93.8	105.4	57.7	12.8	1.1
Washington	11.4	57.5	1,642.5	*	12.7	4.8	25.0	61.3	86.5	98.6	55.5	12.8	1.0
West Virginia	10.1	57.2	1,709.5	*	25.4	8.4	50.4	94.3	105.0	79.4	31.4	5.7	0.5
Wisconsin	11.0	58.9	1,757.0	0.1	13.0	5.0	24.4	56.1	109.4	113.7	49.2	9.4	0.5
Wyoming	11.4	61.0	1,787.5	*	20.8	6.3	43.6	83.8	113.7	91.5	37.8	9.1	*

See footnotes at end of table.

Table 8. Birth rates, by age of mother: United States, each state and territory, 2018—Con.

[By place of residence. Fertility rates are births per 1,000 women aged 15-44 years; total fertility rates are sums of birth rates for 5-year age groups multiplied by 5; birth rates by age are births per 1,000 women in specified age group estimated in each area. Populations estimated as of July 1]

Area	Birth rate	Fertility rate	Total fertility rate	Age of mother (years)									
				10–14	15–19 years			20–24	25–29	30–34	35–39	40–44	45–49[1]
					Total	15–17	18–19						
Puerto Rico	6.7	34.8	1,035.0	*	19.3	9.8	32.4	62.2	56.8	42.8	21.0	4.5	0.3
Virgin Islands	---	---	---	---	---	---	---	---	---	---	---	---	---
Guam	18.9	96.1	2,846.5	*	34.4	15.1	64.0	135.7	157.9	137.1	82.5	19.8	*
American Samoa	---	---	---	---	---	---	---	---	---	---	---	---	---
Northern Marianas	10.9	63.0	2,104.0	*	28.3	*	58.4	87.1	115.5	131.5	47.8	*	*

* Estimate does not meet NCHS standards of reliability.
--- Data not available.
[1]Birth rates computed by relating births to women aged 45 and over to women aged 45–49; see Technical Notes in this report.
[2]Excludes data for the territories.

NOTE: Population data for computing birth rates were provided by the U.S. Census Bureau. Rates by state may differ from rates computed on the basis of other population estimates.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 9. Births and percentage of births to unmarried women, by age and race and Hispanic origin of mother: United States, 2018

Age (years) of mother	All races and origins[2]	Non-Hispanic, single race[1]					Hispanic[3]
		White	Black	American Indian or Alaska Native	Asian	Native Hawaiian or Other Pacific Islander	
				Number			
All ages	1,503,361	551,217	383,364	19,853	28,191	4,776	459,242
Under 15	1,733	336	554	19	17	3	729
15–19	161,547	55,719	36,839	2,429	1,008	465	58,055
15–17	42,974	12,443	9,950	683	263	89	17,599
18–19	118,573	43,276	26,889	1,746	745	376	40,456
20–24	479,425	182,007	122,286	6,172	5,103	1,639	142,699
25–29	437,867	160,778	120,229	6,024	8,351	1,372	125,361
30–34	261,055	94,865	66,381	3,475	7,532	821	79,267
35–39	128,958	46,157	30,209	1,418	4,690	384	41,704
40 and over	32,776	11,355	6,866	316	1,490	92	11,427
				Percent			
All ages	39.6	28.2	69.4	68.2	11.7	50.4	51.8
Under 15	99.8	99.7	100.0	*	*	*	99.9
15–19	89.8	85.8	97.7	94.2	69.8	90.1	89.1
15–17	97.0	95.5	99.5	98.4	85.4	93.7	96.8
18–19	87.5	83.4	97.0	92.7	65.6	89.3	86.2
20–24	66.0	55.7	88.6	78.7	34.3	66.0	68.0
25–29	39.8	27.9	72.1	65.9	14.4	47.5	49.4
30–34	23.9	15.2	53.4	56.9	7.8	37.5	38.1
35–39	22.8	15.2	44.9	50.9	8.2	35.0	35.5
40 and over	25.8	19.0	39.2	51.3	11.3	30.5	36.7

* Estimate does not meet NCHS standards of reliability.
[1]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes in this report. Single race is defined as only one race reported on the birth certificate.
[2]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[3]Includes all persons of Hispanic origin of any race; see Technical Notes.

NOTE: For New York, mother's marital status is inferred; see reference 10 in this report.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 10. Birth rates for unmarried women: United States, 2010–2018, and by age and race and Hispanic origin of mother, United States, 2016–2018

[Rates are births to unmarried women per 1,000 unmarried women. Populations estimated as of July 1 for all years]

Year and race and Hispanic origin	Age of mother (years)								
		15–19							
	Fertility rate[1]	Total	15–17	18–19	20–24	25–29	30–34	35–39	40–44[2]
All races and origins[3]									
2018	40.1	16.0	7.1	29.2	52.2	62.8	59.5	35.2	10.6
2017	41.0	17.1	7.7	31.4	54.2	64.4	57.9	36.0	10.1
2016	42.4	18.5	8.6	33.5	56.6	65.8	59.2	35.6	10.0
2015	43.4	20.2	9.6	36.5	59.7	66.9	60.3	34.1	9.0
2014	43.9	22.0	10.6	39.4	61.6	67.6	58.1	33.4	8.5
2013	44.3	24.0	11.9	42.1	63.1	66.7	56.6	31.8	8.3
2012	45.3	26.7	13.7	45.8	64.7	67.2	56.3	30.9	8.5
2011	46.0	28.4	14.9	48.2	66.7	67.8	56.2	29.9	8.2
2010	47.5	31.1	16.8	52.0	70.0	69.2	56.3	29.6	8.0
Non-Hispanic, single race[4]									
White:									
2018	28.8	10.6	4.0	20.3	37.6	45.7	43.9	25.9	7.5
2017	29.2	11.5	4.4	22.1	38.9	46.2	41.9	25.3	7.2
2016	30.3	12.4	5.0	23.5	40.9	47.3	43.0	25.2	6.8
Black:									
2018	56.4	26.1	12.0	46.1	85.0	85.9	68.2	35.6	9.7
2017	57.5	27.3	12.6	48.7	88.8	89.3	66.2	35.1	8.9
2016	57.9	29.0	13.9	51.2	90.0	89.6	64.4	33.8	8.9
Hispanic[5]									
2018	59.5	24.4	12.2	43.4	74.6	95.7	96.1	57.4	20.0
2017	62.5	26.3	13.3	47.0	78.6	102.3	98.7	64.2	18.7
2016	66.0	29.2	15.1	51.2	83.1	106.4	103.6	65.0	19.3

[1]Rates computed by relating total births to unmarried mothers, regardless of age of mother, to unmarried women aged 15–44 years.
[2]Rates computed by relating births to unmarried women aged 40 years and over to unmarried women aged 40–44 years; see Technical Notes in this report.
[3]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[4]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes. Single race is defined as only one race reported on the birth certificate.
[5]Includes all persons of Hispanic origin of any race; see Technical Notes.

NOTE: For New York, mother's marital status is inferred; see reference 10 in this report.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 11. Selected demographic characteristics of births, by race and Hispanic origin of mother: United States, 2018

[Birth rates are births per 1,000 population. Fertility rates are computed by relating total births, regardless of age of mother, to women aged 15–44 years. Total fertility rates are sums of birth rates for 5-year age groups multiplied by 5. Unmarried rates are births to unmarried women per 1,000 unmarried women. Populations estimated as of July 1. Mean age at first birth is the arithmetic average of the age of mothers at the time of birth, computed directly from the frequency of first births by age of mother]

Characteristic	All races and origins[2]	Non-Hispanic, single race[1]					Hispanic[3]
		White	Black	American Indian or Alaska Native	Asian	Native Hawaiian or Other Pacific Islander	
				Number			
Births	3,791,712	1,956,413	552,029	29,092	240,798	9,476	886,210
				Rate			
Birth rate	11.6	10.0	13.6	12.2	13.2	16.6	14.8
Fertility rate	59.1	56.3	62.0	57.7	55.6	73.0	65.9
Total fertility rate	1,729.5	1,640.0	1,792.0	1,650.5	1,525.0	2,106.5	1,959.0
Unmarried rate	40.1	28.8	56.4	58.2	14.3	82.1	59.5
				Ratio			
Sex ratio[4]	1,046	1,052	1,029	1,036	1,063	1,022	1,036
All births				Percent			
Births to mothers under age 20	4.8	3.3	6.9	8.9	0.6	5.5	7.4
4th and higher-order births[5]	12.9	10.8	17.7	24.7	5.3	27.5	16.0
Births to unmarried mothers	39.6	28.2	69.4	68.2	11.7	50.4	51.8
Mothers born in the 50 states or District of Columbia	77.2	93.2	83.1	98.9	19.0	35.9	52.9
Educational attainment of mother:							
High school diploma or higher	87.3	93.0	86.8	79.5	93.6	77.3	73.6
Bachelor's degree or higher	33.0	42.5	17.8	9.0	65.1	8.8	14.4
				Mean			
Age (years) of mother at first birth	26.9	27.7	25.1	23.5	30.5	24.7	25.0

[1]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes in this report. Single race is defined as only one race reported on the birth certificate.
[2]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[3]Includes all persons of Hispanic origin of any race; see Technical Notes.
[4]Male births per 1,000 female births.
[5]Based on live-birth order.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 12. Selected demographic characteristics of births, by Hispanic origin of mother: United States, 2018

[Mean age at first birth is the arithmetic average of the age of mothers at the time of birth, computed directly from the frequency of first births by age of mother]

Characteristic	Total[1]	Mexican	Puerto Rican	Cuban	Dominican	Central and South American	Other and unknown Hispanic
				Number			
Births	886,210	495,831	71,614	23,471	32,072	147,430	115,792
				Ratio			
Sex ratio[2]	1,036	1,034	1,046	1,062	1,033	1,032	1,038
All births				Percent			
Births to mothers under age 20	7.4	7.8	7.8	3.1	5.2	6.1	8.7
4th and higher-order births[3]	16.0	18.3	13.3	5.4	8.1	13.8	15.0
Mothers born in the 50 states or District of Columbia	52.9	56.9	70.1	43.5	28.3	17.0	79.5
Educational attainment of mother:							
High school diploma or higher	73.6	72.5	84.1	91.8	83.1	61.9	80.1
Bachelor's degree or higher	14.4	11.2	17.0	29.8	20.8	19.1	15.2
				Mean			
Age (years) of mother at first birth	25.0	24.4	24.8	27.7	25.8	26.5	24.7

[1]Includes origin not stated.
[2]Male births per 1,000 female births.
[3]Based on live-birth order.

NOTES: In this table, Hispanic women are classified only by place of origin; non-Hispanic women are not shown; see Technical Notes in this report.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 13. Selected medical and health characteristics of births, by race and Hispanic origin of mother: United States, 2018

Characteristic	All races and origins[2]	Non-Hispanic, single race[1]					Hispanic[3]
		White	Black	American Indian or Alaska Native	Asian	Native Hawaiian or Other Pacific Islander	
Mother	Percent						
Prenatal care initiation:							
First trimester	77.5	82.5	67.1	62.6	81.8	51.0	72.7
Late or no care	6.2	4.5	9.9	13.1	4.9	20.2	7.7
Smoked during pregnancy	6.5	9.5	5.2	15.5	0.5	4.4	1.7
Pregnancy resulted from infertility treatment	1.9	2.7	0.7	0.4	3.4	0.5	0.7
Mother received WIC food for herself during this pregnancy[4]	36.0	23.6	54.6	55.3	20.3	42.1	55.5
Diabetes:							
Prepregnancy (diagnosis prior to this pregnancy)	0.9	0.8	1.2	2.4	1.0	1.8	1.1
Gestational (diagnosis in this pregnancy)	6.7	6.0	5.2	9.9	12.8	8.6	7.2
Overweight or obese (BMI of 25.0 or over)[5]	54.7	51.1	64.9	67.5	33.1	73.9	61.7
Induction of labor	27.1	30.3	25.2	29.0	21.7	18.5	22.8
CNM delivery[6]	9.4	10.0	8.0	20.1	7.5	9.7	9.0
Home birth	1.0	1.5	0.5	0.6	0.3	0.3	0.4
Cesarean delivery (total)	31.9	30.8	36.1	28.7	33.0	31.1	31.6
Low-risk[7]	25.9	24.9	30.3	22.3	27.6	26.5	25.4
Source of payment for the delivery:							
Medicaid	42.3	30.0	65.3	66.2	24.1	58.9	58.9
Private	49.6	63.3	28.4	20.4	67.0	27.0	29.5
Self-pay	4.2	3.2	2.9	1.9	5.9	6.2	6.8
Other[8]	3.9	3.5	3.3	11.6	3.0	7.9	4.7
Infant was being breastfed at discharge[9]	83.5	84.9	72.3	75.2	90.9	79.3	87.1
Infant							
Gestational age:							
Preterm[10]	10.02	9.09	14.13	11.52	8.57	11.79	9.73
Early preterm[11]	2.75	2.26	4.90	3.17	2.11	3.59	2.56
Late preterm[12]	7.28	6.83	9.23	8.35	6.46	8.20	7.17
Birthweight[13]:							
Very low birthweight[14]	1.38	1.02	2.92	1.35	1.11	1.48	1.24
Low birthweight[15]	8.28	6.91	14.07	8.00	8.58	8.97	7.49
Twin births[16]	32.6	34.4	40.8	26.1	28.2	26.4	24.4
Triplet or higher-order births[17]	93.0	102.0	119.2	20.6	59.0	158.3	64.7

[1]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes in this report. Single race is defined as only one race reported on the birth certificate.
[2]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[3]Includes all persons of Hispanic origin of any race; see Technical Notes.
[4]WIC is Special Supplemental Nutrition Program for Women, Infants, and Children.
[5]BMI is body mass index.
[6]Births delivered by certified nurse midwives.
[7]Low-risk cesarean rate is the number of singleton, term (37 or more weeks of gestation based on the obstetric estimate), cephalic, cesarean deliveries to women having a first birth per 100 women delivering singleton, term, cephalic births.
[8]Other includes Indian Health Service, CHAMPUS or TRICARE, other government (federal, state, or local), or charity.
[9]Excludes data for California which did not report if infant was being breastfed at discharge. Also excludes Michigan, for which item wording is not consistent with national standard.
[10]Born before 37 completed weeks of gestation based on the obstetric estimate; see Technical Notes.
[11]Born before 34 completed weeks of gestation based on the obstetric estimate; see Technical Notes.
[12]Born between 34 and 36 completed weeks of gestation based on the obstetric estimate; see Technical Notes.
[13]Equivalents of the gram weights in pounds are shown in the User Guide.
[14]Less than 1,500 grams (3 lb 4 oz).
[15]Less than 2,500 grams (5 lb 8 oz).
[16]Live births in twin deliveries per 1,000 live births.
[17]Live births in triplet and other higher-order multiple deliveries per 100,000 live births.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 14. Selected medical or health characteristics of births, by Hispanic origin of mother: United States, 2018

Characteristic	Total	Mexican	Puerto Rican	Cuban	Dominican	Central and South American	Other and unknown Hispanic
Mother				Percent			
Prenatal care initiation:							
First trimester	72.7	72.8	75.4	81.7	73.0	67.7	75.1
Late or no care	7.7	7.7	6.2	4.0	7.2	9.6	7.0
Smoked during pregnancy	1.7	1.4	5.3	1.8	0.9	0.5	2.3
Pregnancy resulted from infertility treatment	0.7	0.5	1.1	1.1	1.0	0.9	0.8
Mother received WIC food for herself during this pregnancy[1]	55.5	56.4	54.8	49.1	58.1	54.2	54.9
Diabetes:							
Prepregnancy (diagnosis prior to this pregnancy)	1.1	1.2	1.2	0.6	0.8	0.8	1.0
Gestational (diagnosis in this pregnancy)	7.2	7.7	7.1	6.5	6.5	6.7	6.2
Overweight or obese (BMI of 25.0 or over)[2]	61.7	63.9	61.0	51.8	56.5	56.2	62.8
Induction of labor	22.8	22.4	25.5	23.3	24.5	21.4	24.2
CNM delivery[3]	9.0	8.7	11.4	5.3	8.3	10.3	8.1
Home birth	0.4	0.3	0.5	0.5	0.3	0.4	0.5
Cesarean delivery (total)	31.6	30.2	33.6	45.7	41.1	31.2	31.6
Low-risk[4]	25.4	23.8	27.2	38.8	29.6	25.9	24.9
Source of payment for the delivery:							
Medicaid	58.9	59.9	60.3	51.2	66.1	54.1	59.9
Private	29.5	28.5	34.0	44.3	28.3	27.6	30.8
Self-pay	6.8	7.0	1.4	1.9	3.2	12.9	3.9
Other[5]	4.7	4.6	4.3	2.6	2.4	5.4	5.4
Infant was being breastfed at discharge[6]	87.1	87.2	82.0	90.0	88.8	89.5	85.7
Infant							
Gestational age:							
Preterm[7]	9.73	9.55	10.87	9.19	9.31	9.25	10.63
Early preterm[8]	2.56	2.45	3.20	2.60	2.79	2.32	2.82
Late preterm[9]	7.17	7.10	7.67	6.59	6.52	6.92	7.81
Birthweight[10]:							
Very low birthweight[11]	1.24	1.16	1.67	1.27	1.54	1.15	1.38
Low birthweight[12]	7.49	7.10	9.39	7.13	8.32	7.02	8.40
Twin births[13]	24.4	23.0	30.6	28.8	30.0	22.3	27.1
Triplet or higher-order births[14]	64.7	56.9	58.6	25.6	102.9	90.2	66.5

[1]WIC is Special Supplemental Nutrition Program for Women, Infants, and Children.
[2]BMI is body mass index.
[3]Births delivered by certified nurse midwives.
[4]Low-risk cesarean rate is the number of singleton, term (37 or more weeks of gestation based on the obstetric estimate), cephalic, cesarean deliveries to women having a first birth per 100 women delivering singleton, term, cephalic births.
[5]Other includes Indian Health Service, CHAMPUS or TRICARE, other government (federal, state, or local), or charity.
[6]Excludes data for California which did not report if infant was being breastfed at discharge. Also excludes Michigan, for which item wording is not consistent with national standard.
[7]Born before 37 completed weeks of gestation based on the obstetric estimate; see Technical Notes in this report.
[8]Born before 34 completed weeks of gestation based on the obstetric estimate; see Technical Notes.
[9]Born between 34 and 36 completed weeks of gestation based on the obstetric estimate; see Technical Notes.
[10]Equivalents of the gram weights in pounds are shown in the User Guide.
[11]Less than 1,500 grams (3 lb 4 oz).
[12]Less than 2,500 grams (5 lb 8 oz).
[13]Live births in twin deliveries per 1,000 live births.
[14]Live births in triplet and other higher-order multiple deliveries per 100,000 live births.

NOTES: Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, Hispanic women are classified only by place of origin; see Technical Notes.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 15. Mothers who smoked for the 3 months before and anytime during pregnancy and those who quit before and during pregnancy, by age (years) and race and Hispanic origin of mother: United States, 2018

Tobacco use and race and Hispanic origin	All ages	Under 20	20–24	25–29	30–34	35–39	40–54
All races and origins[1]				Percent			
3 months before	8.4	10.2	12.2	9.7	6.4	5.5	4.4
Anytime during pregnancy	6.5	7.6	9.2	7.5	5.0	4.3	3.5
First trimester	6.3	7.3	9.0	7.3	4.9	4.2	3.4
Second trimester	5.4	5.9	7.5	6.3	4.3	3.7	3.0
Third trimester	5.2	5.5	7.1	6.1	4.1	3.5	2.8
Quit before pregnancy[2]	24.2	27.5	25.4	23.5	23.2	23.4	22.0
Quit during pregnancy[3]	20.2	27.4	23.0	19.2	18.0	17.6	17.9
Non-Hispanic, single race[4]							
White:							
3 months before	12.1	21.6	20.4	13.6	8.2	7.4	6.4
Anytime during pregnancy	9.5	16.5	15.9	10.8	6.5	5.8	5.2
First trimester	9.3	16.0	15.6	10.6	6.4	5.7	5.1
Second trimester	8.1	13.1	13.3	9.3	5.6	5.1	4.5
Third trimester	7.7	12.3	12.6	8.9	5.4	4.9	4.3
Quit before pregnancy[2]	22.3	25.2	22.9	21.5	22.1	22.2	20.7
Quit during pregnancy[3]	18.4	25.3	20.8	17.2	16.3	15.9	16.4
Black:							
3 months before	6.8	3.4	6.4	8.0	7.2	6.3	4.7
Anytime during pregnancy	5.2	2.5	4.6	6.1	5.8	5.1	3.7
First trimester	5.0	2.3	4.5	5.9	5.6	5.0	3.7
Second trimester	4.2	1.8	3.5	4.9	4.8	4.2	3.2
Third trimester	3.9	1.6	3.2	4.6	4.5	4.0	3.1
Quit before pregnancy[2]	25.8	30.9	29.9	26.2	22.7	21.9	22.9
Quit during pregnancy[3]	24.6	32.8	29.5	24.7	21.3	21.0	18.3
Hispanic[5]							
3 months before	2.6	2.4	3.2	2.9	2.3	1.9	1.4
Anytime during pregnancy	1.7	1.4	1.9	1.9	1.6	1.3	1.0
First trimester	1.6	1.4	1.8	1.8	1.5	1.3	1.0
Second trimester	1.2	0.9	1.3	1.4	1.2	1.0	0.8
Third trimester	1.1	0.8	1.2	1.3	1.1	1.0	0.7
Quit before pregnancy[2]	38.7	42.9	42.1	38.3	35.6	35.0	30.5
Quit during pregnancy[3]	31.2	40.9	35.7	29.2	28.1	26.3	30.3

See footnotes at end of table.

Table 15. Mothers who smoked for the 3 months before and anytime during pregnancy and those who quit before and during pregnancy, by age (years) and race and Hispanic origin of mother: United States, 2018—Con.

Tobacco use and race and Hispanic origin	All ages	Under 20	20–24	25–29	30–34	35–39	40–54
All races and origins[1]				Number			
Total	3,791,712	181,607	726,175	1,099,491	1,090,697	566,786	126,956
Not stated 3 months before	18,048	1,122	3,982	5,457	4,389	2,492	606
Not stated during pregnancy	17,013	991	3,711	5,154	4,206	2,380	571
Quit before pregnancy[2]:							
Smokers	317,730	18,386	87,784	105,599	69,562	30,878	5,521
Unknown if quit	587	35	143	195	140	61	13
Quit during pregnancy[3]:							
Smokers	245,290	13,663	66,630	82,155	54,324	24,121	4,397
Unknown if quit	425	38	113	127	102	42	3
Non-Hispanic, single race[4]							
White:							
Total	1,956,413	65,254	326,575	576,811	624,015	304,062	59,696
Not stated 3 months before	7,417	382	1,581	2,180	1,969	1,079	226
Not stated during pregnancy	7,003	336	1,452	2,055	1,880	1,061	219
Quit before pregnancy[2]:							
Smokers	235,717	14,024	66,228	78,086	51,248	22,303	3,828
Unknown if quit	331	24	78	111	74	35	9
Quit during pregnancy[3]:							
Smokers	185,707	10,693	51,774	62,108	40,449	17,605	3,078
Unknown if quit	276	25	77	85	59	28	2
Black:							
Total	552,029	38,269	137,974	166,802	124,206	67,268	17,510
Not stated 3 months before	3,321	234	803	1,068	721	391	104
Not stated during pregnancy	3,209	225	780	1,029	708	368	99
Quit before pregnancy[2]:							
Smokers	37,188	1,277	8,727	13,222	8,925	4,221	816
Unknown if quit	94	5	21	27	29	10	2
Quit during pregnancy[3]:							
Smokers	28,549	933	6,341	10,102	7,109	3,415	649
Unknown if quit	72	4	12	23	27	6	–
Hispanic[5]							
Total	886,210	65,852	209,701	253,977	208,193	117,383	31,104
Not stated 3 months before	4,156	368	1,026	1,210	857	542	153
Not stated during pregnancy	3,718	298	913	1,099	791	483	134
Quit before pregnancy[2]:							
Smokers	23,214	1,590	6,613	7,442	4,860	2,269	440
Unknown if quit	79	6	19	30	16	8	–
Quit during pregnancy[3]:							
Smokers	14,740	944	3,961	4,751	3,233	1,528	323
Unknown if quit	28	4	9	9	5	1	–

– Quantity zero.
[1]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[2]Quit in the 3 months before pregnancy: births to women not reporting smoking during pregnancy per 100 women who smoked in the 3 months before pregnancy. See Technical Notes in this report.
[3]Quit during pregnancy: births to women not reporting smoking in the third trimester of pregnancy per 100 women who smoked in either the first or second trimester. See Technical Notes.
[4]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes. Single race is defined as only one race reported on the birth certificate.
[5]Includes all persons of Hispanic origin of any race; see Technical Notes.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 16. Trimester prenatal care began, by age (years) and race and Hispanic origin of mother: United States, 2018

Trimester care began and race and Hispanic origin of mother	All ages	Under 20	20–24	25–29	30–34	35–39	40–54
All races and origins[1]				Percent			
First trimester	77.5	61.2	70.9	77.7	82.1	81.7	78.2
Second trimester	16.3	27.1	21.0	16.2	13.0	13.4	16.0
Late or no care	6.2	11.7	8.1	6.1	4.9	4.9	5.8
Third trimester	4.5	8.4	5.9	4.4	3.5	3.6	4.2
No care	1.7	3.3	2.3	1.7	1.3	1.4	1.6
				Number			
Total	3,791,712	181,607	726,175	1,099,491	1,090,697	566,786	126,956
Not stated	95,362	5,343	19,977	27,209	25,633	13,934	3,266
Non-Hispanic, single race[2]				Percent			
White:							
First trimester	82.5	68.5	75.8	82.3	86.1	85.5	82.3
Second trimester	13.1	23.3	18.1	13.2	10.4	10.8	13.1
Late or no care	4.5	8.2	6.1	4.5	3.5	3.7	4.5
Third trimester	3.3	6.3	4.7	3.4	2.6	2.7	3.3
No care	1.1	1.9	1.4	1.1	0.9	1.0	1.2
				Number			
Total	1,956,413	65,254	326,575	576,811	624,015	304,062	59,696
Not stated	41,224	1,481	7,203	11,561	13,007	6,559	1,413
Black:				Percent			
First trimester	67.1	54.7	63.7	68.1	70.6	71.6	69.2
Second trimester	23.0	31.1	25.5	22.5	20.4	19.6	21.1
Late or no care	9.9	14.2	10.7	9.4	9.0	8.8	9.7
Third trimester	6.8	9.5	7.1	6.4	6.4	6.1	7.1
No care	3.1	4.7	3.6	3.0	2.7	2.6	2.6
				Number			
Total	552,029	38,269	137,974	166,802	124,206	67,268	17,510
Not stated	21,500	1,585	5,585	6,539	4,531	2,605	655
Hispanic[3]				Percent			
First trimester	72.7	58.3	68.7	74.4	76.8	76.5	73.9
Second trimester	19.6	28.3	22.1	18.6	17.0	17.4	19.5
Late or no care	7.7	13.4	9.2	7.1	6.2	6.1	6.5
Third trimester	5.4	9.5	6.6	5.0	4.3	4.2	4.7
No care	2.2	3.8	2.6	2.0	1.8	1.9	1.9
				Number			
Total	886,210	65,852	209,701	253,977	208,193	117,383	31,104
Not stated	22,096	1,895	5,449	6,175	4,955	2,880	742

[1]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[2]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes in this report. Single race is defined as only one race reported on the birth certificate.
[3]Includes all persons of Hispanic origin of any race; see Technical Notes.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 17. Births, by method of delivery: United States, 2010–2018, and by race and Hispanic origin of mother, United States, 2016–2018

Year and race and Hispanic origin	All births	Vaginal		Cesarean			Not stated	Cesarean			Vaginal birth after previous cesarean delivery[7]
		Total[1]	After previous cesarean	Total[2]	Primary	Low-risk[3]		Total[4]	Primary[5]	Low-risk[6]	
All races and origins[8]	Number							Percent			
2018	3,791,712	2,581,992	78,842	1,208,176	693,276	319,022	1,544	31.9	21.7	25.9	13.3
2017	3,855,500	2,621,010	76,301	1,232,339	710,963	325,086	2,151	32.0	21.9	26.0	12.8
2016	3,945,875	2,684,803	75,244	1,258,581	728,500	329,614	2,491	31.9	21.8	25.7	12.4
2015	3,978,497	2,703,504	---	1,272,503	---	331,982	2,490	32.0	---	25.8	---
2014	3,988,076	2,699,951	---	1,284,551	---	337,086	3,574	32.2	---	26.0	---
2013	3,932,181	2,642,892	---	1,284,339	---	344,405	4,950	32.7	---	26.8	---
2012	3,952,841	2,650,744	---	1,296,070	---	355,942	6,027	32.8	---	27.2	---
2011	3,953,590	2,651,428	---	1,293,267	---	359,669	8,895	32.8	---	27.2	---
2010	3,999,386	2,680,947	---	1,309,182	---	368,523	9,257	32.8	---	27.5	---
Non-Hispanic, single race[9]											
White:											
2018	1,956,413	1,353,424	38,345	602,361	356,796	164,087	628	30.8	21.4	24.9	13.5
2017	1,992,461	1,375,702	37,295	615,830	366,505	167,434	929	30.9	21.5	24.9	13.0
2016	2,056,332	1,419,788	37,442	635,588	379,240	172,006	956	30.9	21.5	24.7	12.8
Black:											
2018	552,029	352,750	12,702	199,117	114,150	49,481	162	36.1	25.2	30.3	13.0
2017	560,715	358,467	12,457	201,991	117,054	50,217	257	36.0	25.3	30.4	12.8
2016	558,622	357,859	11,763	200,460	117,410	50,287	303	35.9	25.4	30.3	12.4
Hispanic[10]											
2018	886,210	605,674	19,371	280,386	147,149	67,040	150	31.6	20.1	25.4	12.7
2017	898,764	613,101	18,423	285,379	150,592	67,860	284	31.8	20.2	25.6	12.0
2016	918,447	627,095	17,847	290,832	153,462	67,278	520	31.7	20.1	25.1	11.5

--- Comparable data were not available for the 50 states and the District of Columbia for 2010–2015 because not all reporting areas had adopted the 2003 U.S. Certificate of Live Birth.
[1]Includes unknown type of vaginal delivery; see Technical Notes in this report.
[2]Includes unknown type of cesarean delivery; see Technical Notes.
[3]Low-risk cesarean is defined as singleton, term (37 or more completed weeks of gestation based on the obstetric estimate), cephalic, cesarean deliveries to women having a first birth.
[4]Percentage of all live births delivered by cesarean.
[5]Primary cesarean rate is the number of births to women having a cesarean delivery per 100 births to women without a previous cesarean.
[6]Low-risk cesarean rate is the number of singleton, term (37 or more completed weeks of gestation based on the obstetric estimate), cephalic, cesarean deliveries to women having a first birth per 100 women delivering singleton, term, cephalic, first births.
[7]Vaginal birth after cesarean delivery rate is the number of births to women having a vaginal delivery per 100 births to women with a previous cesarean delivery.
[8]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[9]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes. Single race is defined as only one race reported on the birth certificate.
[10]Includes all persons of Hispanic origin of any race; see Technical Notes.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 18. Births, by method of delivery and by age and race and Hispanic origin of mother: United States, 2018

Race and Hispanic origin and age (years) of mother	All births	Vaginal		Cesarean			Not stated	Cesarean			Vaginal birth after previous cesarean delivery[7]
		Total[1]	After previous cesarean	Total[2]	Primary	Low-risk[3]		Total[4]	Primary[5]	Lowrisk[6]	
All races and origins[8]	Number							Percent			
All ages	3,791,712	2,581,992	78,842	1,208,176	693,276	319,022	1,544	31.9	21.7	25.9	13.3
Under 20	181,607	145,667	561	35,877	32,264	21,678	63	19.8	18.2	16.1	13.5
20–24	726,175	540,586	8,665	185,252	127,269	72,690	337	25.5	19.3	21.6	13.0
25–29	1,099,491	769,495	22,256	329,512	190,914	90,763	484	30.0	20.4	25.5	13.8
30–34	1,090,697	721,045	27,500	369,271	197,717	82,938	381	33.9	22.2	29.5	13.8
35–39	566,786	339,252	16,337	227,331	112,599	40,160	203	40.1	25.9	38.8	12.5
40–54	126,956	65,947	3,523	60,933	32,513	10,793	76	48.0	34.3	53.0	11.0
Non-Hispanic, single race[9]											
White, all ages	1,956,413	1,353,424	38,345	602,361	356,796	164,087	628	30.8	21.4	24.9	13.5
Under 20	65,254	52,167	136	13,058	11,886	7,789	29	20.0	18.6	15.9	10.4
20–24	326,575	245,152	3,048	81,289	57,174	31,790	134	24.9	19.1	20.2	11.2
25–29	576,811	411,907	9,921	164,709	101,506	48,801	195	28.6	20.2	23.6	13.6
30–34	624,015	424,241	14,753	199,603	111,895	47,858	171	32.0	21.5	27.5	14.4
35–39	304,062	187,866	8,733	116,125	59,158	22,268	71	38.2	24.8	36.6	13.3
40–54	59,696	32,091	1,754	27,577	15,177	5,581	28	46.2	33.4	50.3	12.4
Black, all ages	552,029	352,750	12,702	199,117	114,150	49,481	162	36.1	25.2	30.3	13.0
Under 20	38,269	29,707	140	8,552	7,692	5,181	10	22.4	20.7	18.8	14.0
20–24	137,974	97,638	1,990	40,300	27,891	15,649	36	29.2	22.6	25.9	13.8
25–29	166,802	107,994	4,357	58,760	32,119	13,624	48	35.2	23.7	33.3	14.1
30–34	124,206	73,762	3,746	50,407	25,420	8,914	37	40.6	26.7	39.9	13.0
35–39	67,268	35,593	1,984	31,651	15,928	4,704	24	47.1	32.2	49.4	11.2
40–54	17,510	8,056	485	9,447	5,100	1,409	7	54.0	40.3	61.9	10.0
Hispanic[10]											
All ages	886,210	605,674	19,371	280,386	147,149	67,040	150	31.6	20.1	25.4	12.7
Under 20	65,852	53,847	248	11,989	10,610	7,354	16	18.2	16.5	15.1	15.3
20–24	209,701	157,962	2,957	51,706	33,760	20,301	33	24.7	17.9	21.7	14.2
25–29	253,977	175,985	6,038	77,951	39,196	18,263	41	30.7	18.7	27.9	13.5
30–34	208,193	133,067	5,844	75,093	34,613	12,854	33	36.1	21.4	33.8	12.6
35–39	117,383	68,554	3,455	48,813	21,727	6,463	16	41.6	25.0	43.3	11.3
40–54	31,104	16,259	829	14,834	7,243	1,805	11	47.7	32.0	55.7	9.8

[1]Includes unknown type of vaginal delivery; see Technical Notes in this report.
[2]Includes unknown type of cesarean delivery; see Technical Notes.
[3]Low-risk cesarean is defined as singleton, term (37 or more completed weeks of gestation based on the obstetric estimate), cephalic, cesarean deliveries to women having a first birth.
[4]Percentage of all live births delivered by cesarean.
[5]Primary cesarean rate is the number of births to women having a cesarean delivery per 100 births to women without a previous cesarean.
[6]Low-risk cesarean rate is the number of singleton, term (37 or more completed weeks of gestation based on the obstetric estimate), cephalic, cesarean deliveries to women having a first birth per 100 women delivering singleton, term, cephalic, first births.
[7]Vaginal birth after cesarean delivery rate is the number of births to women having a vaginal delivery per 100 births to women with a previous cesarean delivery.
[8]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[9]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes. Single race is defined as only one race reported on the birth certificate.
[10]Includes all persons of Hispanic origin of any race; see Technical Notes.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 19. Principal source of payment for the delivery, by age (years) and race and Hispanic origin of mother: United States, 2018

[Percentages are number of live births with specified source of payment per 100 live births in specified group]

Source of payment and race and Hispanic origin of mother	All ages	Under 20	20–24	25–29	30–34	35–39	40–54
All races and origins[1]				Percent			
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Medicaid	42.3	77.3	63.1	44.8	29.4	27.4	29.9
Private insurance	49.6	15.1	28.2	47.0	63.0	64.8	61.2
Self-pay	4.2	3.8	3.8	4.0	4.2	4.6	5.7
Other[2]	3.9	3.8	4.9	4.1	3.4	3.2	3.1
				Number			
Total	3,791,712	181,607	726,175	1,099,491	1,090,697	566,786	126,956
Not stated	22,595	1,144	4,305	6,801	6,396	3,184	765
Non-Hispanic, single race[3]				Percent			
White:							
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Medicaid	30.0	72.3	53.5	32.4	18.6	16.8	18.3
Private insurance	63.3	22.2	38.6	60.4	75.4	77.2	74.7
Self-pay	3.2	2.0	3.2	3.3	3.1	3.4	4.4
Other[2]	3.5	3.5	4.8	3.9	3.0	2.6	2.6
				Number			
Total	1,956,413	65,254	326,575	576,811	624,015	304,062	59,696
Not stated	12,651	447	2,154	3,852	3,983	1,830	385
				Percent			
Black:							
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Medicaid	65.3	84.2	76.4	68.4	55.6	47.5	43.6
Private insurance	28.4	11.2	18.0	25.7	37.3	45.0	48.2
Self-pay	2.9	2.3	2.0	2.6	3.7	4.1	5.1
Other[2]	3.3	2.3	3.6	3.4	3.3	3.4	3.1
				Number			
Total	552,029	38,269	137,974	166,802	124,206	67,268	17,510
Not stated	2,832	187	615	841	705	367	117
Hispanic[4]				Percent			
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Medicaid	58.9	78.5	69.4	58.9	49.9	48.0	49.5
Private insurance	29.5	10.0	19.2	30.0	38.5	39.9	37.8
Self-pay	6.8	6.8	6.0	6.3	7.4	8.0	8.5
Other[2]	4.7	4.7	5.3	4.8	4.3	4.2	4.3
				Number			
Total	886,210	65,852	209,701	253,977	208,193	117,383	31,104
Not stated	4,177	384	1,030	1,160	904	539	160

[1]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, and Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[2]Other includes Indian Health Service, CHAMPUS or TRICARE, other government (federal, state, or local), or charity.
[3]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes in this report. Single race is defined as only one race reported on the birth certificate.
[4]Includes all persons of Hispanic origin of any race.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 20. Births, by gestational age (weeks): United States, 2010–2018, and by race and Hispanic origin of mother, United States, 2016–2018

Race and Hispanic origin of mother and year	All births	Preterm[1]						Term[1]				Post term[1]	
				Early			Late						
		Total under 37	Total under 34	Under 28	28–31	32–33	34–36	Total 37–41	Early 37–38	Full 39–40	Late 41	42 and over	Not stated
All races and origins[2]	Number	Percent											Number
2018	3,791,712	10.02	2.75	0.66	0.91	1.18	7.28	89.68	26.53	57.24	5.90	0.30	2,507
2017	3,855,500	9.93	2.76	0.67	0.92	1.17	7.17	89.74	26.00	57.49	6.25	0.33	2,759
2016	3,945,875	9.85	2.76	0.68	0.92	1.17	7.09	89.80	25.47	57.94	6.38	0.35	3,454
2015	3,978,497	9.63	2.76	0.68	0.91	1.17	6.87	89.98	24.99	58.47	6.51	0.40	2,926
2014	3,988,076	9.57	2.75	0.69	0.91	1.15	6.82	90.43	24.76	58.72	6.53	0.42	3,246
2013	3,932,181	9.62	2.79	0.70	0.92	1.17	6.83	90.38	24.81	58.85	6.31	0.41	7,467
2012	3,952,841	9.76	2.80	0.71	0.92	1.17	6.96	90.24	25.47	58.30	6.06	0.40	8,380
2011	3,953,590	9.81	2.81	0.70	0.93	1.18	6.99	90.19	26.09	57.51	6.16	0.43	9,290
2010	3,999,386	9.98	2.84	0.71	0.94	1.18	7.15	90.01	27.29	56.08	6.19	0.46	10,538
Non-Hispanic, single race[3]													
White:													
2018	1,956,413	9.09	2.26	0.44	0.76	1.07	6.83	90.53	24.68	59.16	6.70	0.37	924
2017	1,992,461	9.05	2.30	0.46	0.77	1.06	6.76	90.54	24.16	59.27	7.11	0.40	1,051
2016	2,056,332	9.04	2.33	0.48	0.78	1.07	6.72	90.53	23.64	59.61	7.28	0.43	1,409
Black:													
2018	552,029	14.13	4.90	1.51	1.60	1.79	9.23	85.62	29.64	51.34	4.64	0.25	384
2017	560,715	13.93	4.87	1.53	1.62	1.72	9.06	85.79	28.93	51.90	4.96	0.28	445
2016	558,622	13.77	4.93	1.56	1.61	1.75	8.84	85.94	28.48	52.42	5.04	0.30	506
Hispanic[4]													
2018	886,210	9.73	2.56	0.62	0.84	1.09	7.17	90.07	28.05	56.83	5.19	0.20	382
2017	898,764	9.62	2.56	0.62	0.85	1.09	7.05	90.16	27.58	57.15	5.43	0.23	455
2016	918,447	9.45	2.98	0.62	0.84	1.08	6.92	90.30	27.02	57.85	5.43	0.25	676
All races and origins[2]		Number											
2018	3,791,712	379,777	104,031	24,945	34,386	44,700	275,746	3,398,110	1,005,405	2,169,016	223,689	11,318	2,507
2017	3,855,500	382,726	106,417	25,913	35,476	45,028	276,309	3,457,455	1,001,601	2,214,983	240,871	12,560	2,759
2016	3,945,875	388,218	108,836	26,618	36,239	45,979	279,382	3,540,290	1,004,224	2,284,399	251,667	13,913	3,454
2015	3,978,497	382,786	109,660	26,996	36,149	46,515	273,126	3,577,072	993,599	2,324,474	258,999	15,713	2,926
2014	3,988,076	381,321	109,474	27,320	36,245	45,909	271,847	3,586,933	986,745	2,339,796	260,392	19,822	3,246
2013	3,932,181	377,655	109,435	27,550	36,096	45,789	268,220	3,283,457	973,569	2,309,888	247,476	16,126	7,467
2012	3,952,841	385,082	110,444	28,004	36,366	46,074	274,638	3,304,365	1,004,750	2,299,615	239,148	15,866	8,380
2011	3,953,590	386,855	111,002	27,737	36,758	46,507	275,853	3,297,649	1,029,157	2,268,492	242,999	16,797	9,290
2010	3,999,386	398,402	113,131	28,437	37,435	47,259	285,271	3,325,476	1,088,564	2,236,912	246,768	18,413	10,538
Non-Hispanic, single race[3]													
White:													
2018	1,956,413	177,842	44,287	8,665	14,764	20,858	133,555	1,770,366	482,550	1,156,775	131,041	7,281	924
2017	1,992,461	180,322	45,761	9,200	15,429	21,132	134,561	1,803,063	481,048	1,180,394	141,621	8,025	1,051
2016	2,056,332	185,854	47,823	9,766	16,101	21,956	138,031	1,860,243	485,846	1,224,872	149,525	8,826	1,409
Black:													
2018	552,029	77,939	27,008	8,348	8,804	9,856	50,931	472,316	163,530	283,202	25,584	1,390	384
2017	560,715	78,024	27,274	8,548	9,070	9,656	50,750	480,661	162,072	290,803	27,786	1,585	445
2016	558,622	76,834	27,501	8,729	8,987	9,785	49,333	479,630	158,937	292,539	28,154	1,652	506
Hispanic[4]													
2018	886,210	86,186	22,637	5,490	7,467	9,680	63,549	797,905	248,463	503,429	46,013	1,737	382
2017	898,764	86,393	23,025	5,605	7,635	9,785	63,368	809,882	247,788	513,342	48,752	2,034	455
2016	918,447	86,691	23,195	5,649	7,665	9,881	63,496	828,783	247,999	530,951	49,833	2,297	676

[1]Expressed in completed weeks based on the obstetric estimate of gestation; see Technical Notes in this report.
[2]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[3]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes. Single race is defined as only one race reported on the birth certificate.
[4]Includes all persons of Hispanic origin of any race; see Technical Notes.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 21. Births, by gestational age (weeks) and by age and race and Hispanic origin of mother: United States, 2018

Age (years) and race and Hispanic origin of mother	All births	Preterm[1]					Term[1]				Post term[1]	Not stated
		Total under 37	Early			Late	Total 37–41	Early 37–38	Full 39–40	Late 41	42 and over	
			Under 28	28–31	32–33	34–36						
All races and origins[2]	Number	Percent										Number
All ages	3,791,712	10.02	0.66	0.91	1.18	7.28	89.68	26.53	57.24	5.90	0.30	2,507
Under 15	1,736	15.40	1.33	1.74	1.51	10.83	84.42	27.97	50.32	6.14	0.17	9
15–19	179,871	10.41	0.90	1.01	1.23	7.27	89.37	26.40	56.31	6.65	0.22	157
20–24	726,175	9.65	0.70	0.90	1.11	6.95	90.10	26.65	57.43	6.01	0.26	524
25–29	1,099,491	9.38	0.63	0.84	1.06	6.85	90.34	25.95	58.26	6.12	0.28	805
30–34	1,090,697	9.62	0.59	0.85	1.16	7.03	90.04	25.84	57.90	6.31	0.34	580
35–39	566,786	11.39	0.67	1.03	1.39	8.30	88.27	27.83	55.43	5.01	0.33	334
40–44	117,381	13.80	0.83	1.25	1.70	10.02	85.91	31.10	51.89	2.92	0.29	90
45–54	9,575	22.12	1.33	2.54	3.44	14.81	77.58	33.18	42.09	2.31	0.30	8
Non-Hispanic, single race[3]												
White, all ages	1,956,413	9.09	0.44	0.76	1.07	6.83	90.53	24.68	59.16	6.70	0.37	924
Under 15	337	14.93	1.49	0.90	0.60	11.94	85.07	24.78	53.43	6.87	*	2
15–19	64,917	10.11	0.79	0.97	1.17	7.18	89.65	25.09	57.73	6.82	0.25	49
20–24	326,575	9.00	0.48	0.78	1.04	6.70	90.69	25.17	59.08	6.44	0.31	181
25–29	576,811	8.55	0.44	0.71	0.97	6.43	91.12	24.25	59.98	6.89	0.33	293
30–34	624,015	8.64	0.38	0.70	1.03	6.52	90.95	23.88	59.77	7.29	0.42	249
35–39	304,062	10.19	0.43	0.80	1.22	7.74	89.37	25.61	57.75	6.01	0.44	114
40–44	55,417	12.47	0.59	1.07	1.51	9.29	87.12	29.00	54.49	3.63	0.40	32
45–54	4,279	19.58	0.89	2.15	3.37	13.17	80.07	32.23	45.15	2.69	0.35	4
Black, all ages	552,029	14.13	1.51	1.60	1.79	9.23	85.62	29.64	51.34	4.64	0.25	384
Under 15	554	16.67	1.63	1.45	2.17	11.41	82.97	28.26	49.28	5.43	0.36	2
15–19	37,715	12.96	1.48	1.43	1.71	8.33	86.90	28.01	52.99	5.91	0.14	40
20–24	137,974	13.03	1.38	1.49	1.58	8.57	86.77	29.26	52.73	4.78	0.20	96
25–29	166,802	13.57	1.46	1.51	1.67	8.93	86.18	29.18	52.31	4.69	0.25	105
30–34	124,206	14.38	1.57	1.60	1.86	9.35	85.29	29.58	50.91	4.80	0.33	80
35–39	67,268	16.60	1.74	1.97	2.16	10.73	83.13	31.36	47.96	3.81	0.27	53
40–44	15,988	18.55	1.76	2.05	2.54	12.21	81.20	34.55	44.36	2.29	0.24	8
45–54	1,522	26.94	2.43	2.89	4.40	17.21	72.40	34.03	35.74	2.63	0.66	–
Hispanic[4]												
All ages	886,210	9.73	0.62	0.84	1.09	7.17	90.07	28.05	56.83	5.19	0.20	382
Under 15	730	14.62	0.69	2.34	1.24	10.34	85.24	28.00	50.90	6.34	0.14	5
15–19	65,122	9.23	0.68	0.81	1.00	6.74	90.54	26.77	56.94	6.83	0.23	43
20–24	209,701	8.47	0.58	0.71	0.93	6.25	91.33	27.10	58.17	6.05	0.20	107
25–29	253,977	8.86	0.56	0.75	0.93	6.63	90.94	27.32	58.21	5.41	0.20	92
30–34	208,193	10.15	0.62	0.87	1.18	7.48	89.66	28.24	56.57	4.85	0.19	69
35–39	117,383	12.05	0.73	1.13	1.46	8.74	87.77	30.40	53.65	3.72	0.18	51
40–44	29,192	14.08	0.81	1.18	1.68	10.41	85.76	32.72	50.90	2.14	0.16	14
45–54	1,912	23.02	1.31	2.93	2.83	15.96	76.87	35.95	39.46	1.47	0.10	1

* Estimate does not meet NCHS standards of reliability.
– Quantity zero.
[1]Expressed in completed weeks based on the obstetric estimate of gestation; see Technical Notes in this report.
[2]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[3]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes. Single race is defined as only one race reported on the birth certificate.
[4]Includes all persons of Hispanic origin of any race; see Technical Notes.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 22. Births, by birthweight (grams): United States, 2010–2018, and by age and race and Hispanic origin of mother, United States, 2016–2018

Year and race and Hispanic origin of mother	All births	Birthweight (grams)[1]													
		Low birthweight													
			Very low birthweight												
		Total under 2,500	Total under 1,500	Less than 500	500–999	1,000–1,499	1,500–1,999	2,000–2,499	2,500–2,999	3,000–3,499	3,500–3,999	4,000–4,499	4,500–4,999	5,000 or more	Not stated
All races and origins[2]	Number	Percent													Number
2018	3,791,712	8.28	1.38	0.14	0.50	0.74	1.62	5.28	18.75	38.76	26.44	6.72	0.94	0.11	2,932
2017	3,855,500	8.28	1.41	0.14	0.52	0.74	1.63	5.25	18.72	38.76	26.44	6.75	0.95	0.11	3,002
2016	3,945,875	8.17	1.40	0.14	0.52	0.74	1.59	5.17	18.54	38.76	26.60	6.85	0.97	0.11	4,518
2015	3,978,497	8.07	1.40	0.15	0.52	0.73	1.58	5.09	18.36	38.84	26.73	6.90	0.98	0.12	3,621
2014	3,988,076	8.00	1.40	0.15	0.52	0.74	1.56	5.04	18.27	38.80	26.88	6.94	0.99	0.12	3,270
2013	3,932,181	8.02	1.41	0.15	0.53	0.73	1.56	5.05	18.22	38.93	26.85	6.86	0.99	0.11	4,452
2012	3,952,841	7.99	1.42	0.15	0.54	0.73	1.56	5.01	18.28	39.00	26.81	6.83	0.97	0.12	4,008
2011	3,953,590	8.10	1.44	0.15	0.54	0.75	1.58	5.08	18.44	39.13	26.56	6.71	0.95	0.11	4,570
2010	3,999,386	8.15	1.45	0.15	0.55	0.75	1.59	5.11	18.63	39.21	26.41	6.58	0.92	0.11	3,964
Non-Hispanic, single race[3]															
White:															
2018	1,956,413	6.91	1.02	0.09	0.34	0.58	1.38	4.52	16.01	37.80	29.74	8.25	1.16	0.12	1,259
2017	1,992,461	7.00	1.05	0.09	0.36	0.60	1.40	4.54	16.05	37.68	29.70	8.28	1.17	0.12	1,211
2016	2,056,332	6.97	1.07	0.09	0.37	0.61	1.39	4.51	15.88	37.59	29.82	8.39	1.21	0.12	2,212
Black:															
2018	552,029	14.07	2.92	0.35	1.16	1.41	2.79	8.35	25.44	37.90	18.33	3.68	0.51	0.07	571
2017	560,715	13.89	2.95	0.36	1.18	1.41	2.72	8.22	25.44	37.96	18.37	3.76	0.51	0.07	642
2016	558,622	13.68	2.95	0.37	1.20	1.38	2.67	8.06	25.38	38.24	18.42	3.69	0.52	0.07	936
Hispanic[4]															
2018	886,210	7.49	1.24	0.12	0.46	0.66	1.43	4.81	19.01	40.64	25.90	6.02	0.83	0.11	381
2017	898,764	7.43	1.26	0.13	0.47	0.66	1.42	4.76	18.86	40.76	25.95	6.02	0.85	0.11	428
2016	918,447	7.32	1.24	0.13	0.46	0.65	1.41	4.68	18.76	40.79	26.06	6.10	0.85	0.12	556

See footnotes at end of table.

Table 22. Births, by birthweight (grams): United States, 2010–2018, and by age and race and Hispanic origin of mother, United States, 2016–2018—Con.

Year and race and Hispanic origin of mother	All births	Birthweight (grams)[1]													
		Low birthweight													
			Very low birthweight												
		Total under 2,500	Total under 1,500	Less than 500	500–999	1,000–1,499	1,500–1,999	2,000–2,499	2,500–2,999	3,000–3,499	3,500–3,999	4,000–4,499	4,500–4,999	5,000 or more	Not stated
All races and origins[2]								Number							
2018	3,791,712	313,752	52,171	5,303	18,970	27,898	61,509	200,072	710,444	1,468,639	1,001,803	254,704	35,435	4,003	2,932
2017	3,855,500	318,873	54,135	5,470	19,979	28,686	62,605	202,133	721,165	1,493,081	1,018,613	260,042	36,493	4,231	3,002
2016	3,945,875	321,839	55,110	5,710	20,323	29,077	62,863	203,866	730,710	1,527,707	1,048,476	269,865	38,264	4,496	4,518
2015	3,978,497	320,869	55,592	5,863	20,689	29,040	62,862	202,415	729,673	1,544,024	1,062,456	274,404	38,796	4,654	3,621
2014	3,988,076	318,847	55,947	5,936	20,721	29,290	61,992	200,908	727,987	1,546,274	1,071,007	276,592	39,353	4,746	3,270
2013	3,932,181	315,099	55,458	5,945	20,866	28,647	61,238	198,403	715,764	1,529,258	1,054,767	269,594	38,834	4,413	4,452
2012	3,952,841	315,709	56,252	5,947	21,432	28,873	61,499	197,958	721,840	1,540,161	1,058,604	269,581	38,288	4,650	4,008
2011	3,953,590	319,711	56,754	5,942	21,289	29,523	62,504	200,453	728,201	1,545,355	1,048,902	265,040	37,475	4,336	4,570
2010	3,999,386	325,563	57,841	5,980	22,015	29,846	63,427	204,295	744,181	1,566,755	1,055,004	262,997	36,706	4,216	3,964
Non-Hispanic, single race[3]															
White:															
2018	1,956,413	135,185	19,872	1,721	6,719	11,432	26,964	88,349	313,105	739,031	581,499	161,395	22,645	2,294	1,259
2017	1,992,461	139,358	20,981	1,752	7,233	11,996	27,968	90,409	319,523	750,331	591,376	164,898	23,299	2,465	1,211
2016	2,056,332	143,254	21,979	1,888	7,565	12,526	28,578	92,697	326,279	772,165	612,641	172,434	24,781	2,566	2,212
Black:															
2018	552,029	77,584	16,127	1,941	6,406	7,780	15,411	46,046	140,295	209,028	101,089	20,285	2,806	371	571
2017	560,715	77,815	16,544	2,024	6,597	7,923	15,228	46,043	142,459	212,599	102,878	21,065	2,854	403	642
2016	558,622	76,299	16,465	2,083	6,675	7,707	14,885	44,949	141,557	213,260	102,702	20,571	2,880	417	936
Hispanic[4]															
2018	886,210	66,310	11,006	1,084	4,040	5,882	12,706	42,598	168,418	359,966	229,450	53,359	7,358	968	381
2017	898,764	66,766	11,275	1,137	4,229	5,909	12,772	42,719	169,467	366,198	233,157	54,096	7,631	1,021	428
2016	918,447	67,210	11,378	1,175	4,237	5,966	12,910	42,922	172,171	374,434	239,210	55,983	7,770	1,113	556

[1]Equivalents of gram weights in pounds and ounces are shown in the User Guide (see reference 10 in this report).
[2]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[3]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes in this report. Single race is defined as only one race reported on the birth certificate.
[4]Includes all persons of Hispanic origin of any race; see Technical Notes.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 23. Births, by birthweight (grams) and by age and race and Hispanic origin of mother: United States, 2018

Age (years) and race and Hispanic origin of mother	All births	Birthweight (grams)[1]													
		Low birthweight													
			Very low birthweight												
		Total under 2,500	Total under 1,500	Less than 500	500–999	1,000–1,499	1,500–1,999	2,000–2,499	2,500–2,999	3,000–3,499	3,500–3,999	4,000–4,499	4,500–4,999	5,000 or more	Not stated
All races and origins[2]	Number	Percent													Number
All ages	3,791,712	8.28	1.38	0.14	0.50	0.74	1.62	5.28	18.75	38.76	26.44	6.72	0.94	0.11	2,932
Under 15	1,736	12.73	2.59	0.40	0.75	1.44	2.36	7.78	26.84	41.65	16.19	2.48	0.12	*	–
15–19	179,871	10.09	1.74	0.17	0.68	0.89	1.82	6.53	23.78	41.12	20.76	3.82	0.39	0.04	127
20–24	726,175	8.59	1.41	0.15	0.52	0.73	1.60	5.58	20.91	40.51	24.08	5.22	0.63	0.06	520
25–29	1,099,491	7.78	1.29	0.14	0.47	0.68	1.50	4.99	18.37	39.20	26.91	6.74	0.90	0.10	877
30–34	1,090,697	7.63	1.24	0.12	0.44	0.68	1.53	4.87	17.24	37.99	28.22	7.66	1.13	0.12	843
35–39	566,786	8.83	1.50	0.13	0.54	0.82	1.83	5.50	17.80	36.94	27.37	7.73	1.18	0.15	457
40–44	117,381	10.77	1.88	0.17	0.67	1.04	2.32	6.57	19.58	36.51	25.04	6.91	1.05	0.14	100
45–54	9,575	18.39	3.42	0.17	1.20	2.05	4.35	10.62	21.71	33.89	20.04	5.14	0.77	0.06	8
Non-Hispanic, single race[3]															
White, all ages	1,956,413	6.91	1.02	0.09	0.34	0.58	1.38	4.52	16.01	37.80	29.74	8.25	1.16	0.12	1,259
Under 15	337	10.39	2.37	0.30	1.19	0.89	0.89	7.12	21.96	41.54	21.36	4.75	*	*	–
15–19	64,917	8.97	1.57	0.12	0.63	0.82	1.60	5.79	20.14	40.43	24.67	5.15	0.59	0.04	44
20–24	326,575	7.21	1.08	0.09	0.38	0.61	1.36	4.77	18.01	39.81	27.49	6.59	0.81	0.08	193
25–29	576,811	6.52	0.98	0.09	0.33	0.56	1.28	4.27	15.71	38.30	30.07	8.18	1.09	0.11	347
30–34	624,015	6.40	0.90	0.08	0.29	0.53	1.31	4.19	14.85	37.06	31.15	9.07	1.33	0.13	417
35–39	304,062	7.39	1.05	0.09	0.35	0.61	1.54	4.80	15.59	36.03	30.22	9.21	1.40	0.16	211
40–44	55,417	9.27	1.45	0.12	0.46	0.88	1.99	5.83	17.67	35.84	27.58	8.22	1.25	0.17	43
45–54	4,279	15.98	2.78	0.12	0.80	1.87	3.53	9.66	19.53	34.69	22.13	6.60	0.96	0.12	4
Black, all ages	552,029	14.07	2.92	0.35	1.16	1.41	2.79	8.35	25.44	37.90	18.33	3.68	0.51	0.07	571
Under 15	554	14.62	3.25	0.54	0.90	1.81	2.71	8.66	31.05	39.17	13.36	1.62	0.18	*	–
15–19	37,715	14.91	2.84	0.35	1.13	1.36	2.85	9.22	30.48	38.79	13.86	1.77	0.16	0.02	34
20–24	137,974	14.06	2.71	0.33	1.05	1.33	2.68	8.67	28.02	39.02	16.01	2.55	0.30	0.03	136
25–29	166,802	13.54	2.75	0.37	1.08	1.30	2.66	8.14	25.28	38.54	18.53	3.61	0.44	0.06	173
30–34	124,206	13.49	2.96	0.34	1.20	1.42	2.74	7.79	23.17	37.11	20.65	4.76	0.70	0.11	146
35–39	67,268	15.13	3.51	0.37	1.43	1.71	3.15	8.47	22.37	35.84	20.65	5.03	0.88	0.10	66
40–44	15,988	16.63	3.77	0.38	1.51	1.87	3.63	9.23	23.44	35.11	19.34	4.63	0.74	0.11	13
45–54	1,522	24.16	5.00	0.26	2.11	2.63	6.85	12.31	23.90	30.35	17.12	3.88	0.59	*	3

See footnotes at end of table.

Table 23. Births, by birthweight (grams) and by age and race and Hispanic origin of mother: United States, 2018—Con.

Age (years) and race and Hispanic origin of mother	All births	Birthweight (grams)[1]													
		Low birthweight													
			Very low birthweight												
		Total under 2,500	Total under 1,500	Less than 500	500–999	1,000–1,499	1,500–1,999	2,000–2,499	2,500–2,999	3,000–3,499	3,500–3,999	4,000–4,499	4,500–4,999	5,000 or more	Not stated
Hispanic[4]	Number	Percent													Number
All ages	886,210	7.49	1.24	0.12	0.46	0.66	1.43	4.81	19.01	40.64	25.90	6.02	0.83	0.11	381
Under 15	730	12.19	1.78	0.41	0.14	1.23	2.60	7.81	25.48	43.56	16.85	1.78	0.14	*	–
15–19	65,122	8.40	1.28	0.11	0.48	0.69	1.43	5.69	23.53	43.36	20.80	3.57	0.31	0.04	35
20–24	209,701	7.13	1.11	0.13	0.41	0.57	1.25	4.77	20.46	42.54	24.35	4.89	0.57	0.06	76
25–29	253,977	6.78	1.11	0.11	0.41	0.58	1.28	4.40	18.28	40.96	26.82	6.22	0.83	0.11	106
30–34	208,193	7.32	1.27	0.13	0.45	0.69	1.44	4.61	17.49	39.43	27.60	6.97	1.06	0.14	93
35–39	117,383	8.55	1.56	0.12	0.58	0.86	1.84	5.16	18.08	37.99	26.91	7.17	1.12	0.18	57
40–44	29,192	10.19	1.69	0.15	0.66	0.88	2.30	6.20	19.24	37.67	24.94	6.77	1.08	0.12	13
45–54	1,912	18.32	3.72	0.26	1.41	2.04	3.35	11.25	22.29	34.59	19.73	4.19	0.84	0.05	1

* Estimate does not meet NCHS standards of reliability.
– Quantity zero.
[1]Equivalents of gram weights in pounds and ounces are shown in the User Guide (see reference 10 in this report).
[2]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[3]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes in this report. Single race is defined as only one race reported on the birth certificate.
[4]Includes all persons of Hispanic origin of any race; see Technical Notes.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 24. Twin and triplet and higher-order multiple births: United States, 2010–2018, and by race and Hispanic origin of mother: United States, 2016–2018

Race and Hispanic origin and year	Total births	Twin births	Triplet and higher-order births[1]	Multiple birth rate	Twin birth rate	Triplet and higher-order birth rate[1]
All races and origins[2]	Number			Per 1,000		Per 100,000
2018	3,791,712	123,536	3,525	33.5	32.6	93.0
2017	3,855,500	128,310	3,917	34.3	33.3	101.6
2016	3,945,875	131,723	4,003	34.4	33.4	101.4
2015	3,978,497	133,155	4,123	34.5	33.5	103.6
2014	3,988,076	135,336	4,526	35.1	33.9	113.5
2013	3,932,181	132,324	4,700	34.8	33.7	119.5
2012	3,952,841	131,024	4,919	34.4	33.1	124.4
2011	3,953,590	131,269	5,417	34.6	33.2	137.0
2010	3,999,386	132,562	5,503	34.5	33.1	137.6
Non-Hispanic, single race[3]						
White:						
2018	1,956,413	67,203	1,996	35.4	34.4	102.0
2017	1,992,461	70,704	2,324	36.7	35.5	116.6
2016	2,056,332	73,425	2,502	36.9	35.7	121.7
Black:						
2018	552,029	22,502	658	42.0	40.8	119.2
2017	560,715	22,982	671	42.2	41.0	119.7
2016	558,622	22,267	628	41.0	39.9	112.4
Hispanic[4]						
2018	886,210	21,654	573	25.1	24.4	64.7
2017	898,764	22,041	614	25.2	24.5	68.3
2016	918,447	22,625	538	25.2	24.6	58.6

[1]Triplet, quadruplet, quintuplet, and higher-order multiple deliveries.
[2]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[3]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes in this report. Single race is defined as only one race reported on the birth certificate.
[4]Includes all persons of Hispanic origin of any race; see Technical Notes.

SOURCE: NCHS, National Vital Statistics System, Natality.

Table 25. Births, by plurality and age and race and Hispanic origin of mother: United States, 2018

Race and Hispanic origin and age (years) of mother	Live births				Rates		
	Total	Singleton	Twin	Triplet and higher-order multiple[1]	Multiple birth	Twin birth	Triplet and higher-order multiple[1]
All races and origins[2]	Number				Per 1,000		Per 100,000
All ages	3,791,712	3,664,651	123,536	3,525	33.5	32.6	93.0
Under 15	1,736	1,715	21	–	12.1	12.1	0.0
15–19	179,871	176,756	3,084	31	17.3	17.1	17.2
15–17	44,291	43,658	624	9	14.3	14.1	20.3
18–19	135,580	133,098	2,460	22	18.3	18.1	16.2
20–24	726,175	709,046	16,835	294	23.6	23.2	40.5
25–29	1,099,491	1,065,264	33,394	833	31.1	30.4	75.8
30–34	1,090,697	1,050,006	39,441	1,250	37.3	36.2	114.6
35–39	566,786	541,686	24,268	832	44.3	42.8	146.8
40–44	117,381	111,902	5,269	210	46.7	44.9	178.9
45–54	9,575	8,276	1,224	75	135.7	127.8	783.3
Non-Hispanic, single race[3]							
White, all ages	1,956,413	1,887,214	67,203	1,996	35.4	34.4	102.0
Under 15	337	327	10	–	29.7	29.7	0.0
15–19	64,917	63,840	1,068	9	16.6	16.5	13.9
15–17	13,036	12,858	175	3	13.7	13.4	23.0
18–19	51,881	50,982	893	6	17.3	17.2	11.6
20–24	326,575	319,134	7,314	127	22.8	22.4	38.9
25–29	576,811	558,625	17,709	477	31.5	30.7	82.7
30–34	624,015	599,564	23,656	795	39.2	37.9	127.4
35–39	304,062	289,438	14,123	501	48.1	46.4	164.8
40–44	55,417	52,542	2,792	83	51.9	50.4	149.8
45–54	4,279	3,744	531	4	125.0	124.1	93.5
Black, all ages	552,029	528,869	22,502	658	42.0	40.8	119.2
Under 15	554	552	2	–	3.6	3.6	0.0
15–19	37,715	36,790	912	13	24.5	24.2	34.5
15–17	9,998	9,814	181	3	18.4	18.1	30.0
18–19	27,717	26,976	731	10	26.7	26.4	36.1
20–24	137,974	133,230	4,654	90	34.4	33.7	65.2
25–29	166,802	159,426	7,198	178	44.2	43.2	106.7
30–34	124,206	118,410	5,626	170	46.7	45.3	136.9
35–39	67,268	63,932	3,230	106	49.6	48.0	157.6
40–44	15,988	15,245	685	58	46.5	42.8	362.8
45–54	1,522	1,284	195	43	156.4	128.1	2,825.2
Hispanic[4]							
All ages	886,210	863,983	21,654	573	25.1	24.4	64.7
Under 15	730	723	7	–	9.6	9.6	0.0
15–19	65,122	64,226	890	6	13.8	13.7	9.2
15–17	18,185	17,968	214	3	11.9	11.8	16.5
18–19	46,937	46,258	676	3	14.5	14.4	6.4
20–24	209,701	205,889	3,752	60	18.2	17.9	28.6
25–29	253,977	247,961	5,891	125	23.7	23.2	49.2
30–34	208,193	201,987	6,001	205	29.8	28.8	98.5
35–39	117,383	113,358	3,906	119	34.3	33.3	101.4
40–44	29,192	28,164	985	43	35.2	33.7	147.3
45–54	1,912	1,675	222	15	124.0	116.1	784.5

– Quantity zero.
0.0 Equals zero events in the numerator or percentage of less than 0.05%.
[1]Triplet, quadruplet, quintuplet and higher-order multiple deliveries.
[2]Includes births to race and origin groups not shown separately, such as Hispanic single-race white, Hispanic single-race black, and non-Hispanic multiple-race women, and births with origin not stated.
[3]Race and Hispanic origin are reported separately on birth certificates; persons of Hispanic origin may be of any race. In this table, non-Hispanic women are classified by race. Race categories are consistent with the 1997 Office of Management and Budget standards; see Technical Notes in this report. Single race is defined as only one race reported on the birth certificate.
[4]Includes all persons of Hispanic origin of any race.

SOURCE: NCHS, National Vital Statistics System, Natality.

Technical Notes

Data source

Data shown in this report for 2018 are based on 100% of the birth certificates filed in all states and the District of Columbia (D.C.). The data are provided to the National Center for Health Statistics (NCHS) through the Vital Statistics Cooperative Program (VSCP). Information on the percentage of records with missing information for maternal and infant characteristics included in this report is shown by state in the User Guide (10); Methodological and measurement information for these characteristics is also available in the User Guide.

2003 revision of U.S. Standard Certificate of Live Birth

Data for 2016–2018 presented in this report are based on the 2003 revision of the U.S. Standard Certificate of Live Birth; data for 2010–2015 are based on both the 1989 and the 2003 birth certificate revisions. The 2003 revision is described in detail elsewhere (18).

Age of mother

Age of mother is computed in most cases from the mother's and infant's dates of birth as reported on the birth certificate. Since 2007, age of mother has been imputed for ages 8 and under and 65 and over. Mothers aged 9 are recoded as aged 10. A review and verification of unedited data for several years including 2007 showed that the vast majority of births reported as occurring to women aged 50 and over were to women aged 50–54. In this report, the final age group shown in the tables (45–49, 45–54, or 50–54) includes births to mothers up to age 64. For historical information on mother's age, see the User Guide (10).

Marital status

Due to state statutory restrictions, beginning in 2017, California no longer provides record-level data on the marital status of the mother for births occurring in California to California residents and nonresidents. Instead of record-level data, California provided counts of births by marital status category (married, unmarried, and unknown) by age and race and Hispanic origin of the mother to NCHS, according to the age and race and Hispanic origin categories shown in this report. For consistency with procedures for handling missing information for other jurisdictions, California's counts of birth by marital status were redistributed (proportionately) to the same maternal age, race, and marital status subgroups in the data set to obtain the national estimates by marital status. This approach is consistent with hot-deck procedures used for non-California records to impute for missing age, race, and marital status based on records for which these characteristics are known.

Hispanic origin and race

Hispanic origin

Hispanic origin and race are reported separately on the birth certificate. Data are shown in most cases for five specified Hispanic groups: Mexican, Puerto Rican, Cuban, Central and South American, and Other and unknown Hispanic. Starting with 2018, data are presented for an additional Hispanic group, Dominican, which was previously included in the category Other and unknown Hispanic. In tabulations of birth data by race and Hispanic origin, data for persons of Hispanic origin are not further classified by race because the vast majority of births to Hispanic women are reported as white.

Items asking for the Hispanic origin of the mother (and the father) have been included on the birth certificates of all states, D.C., U.S. Virgin Islands, and Guam since 1993, and on the birth certificates of Puerto Rico starting in 2005 and Commonwealth of the Northern Marianas starting in 2010 (10,12). American Samoa does not collect information on Hispanic origin.

The Hispanic origin question on the 2003 revision of the birth certificate asks respondents to select only one response. Occasionally, however, more than one Hispanic origin response is given (i.e., a specified Hispanic group [Mexican, Puerto Rican, Cuban, Dominican, or Central and South American] in combination with one or more other specified Hispanic group). From 2003 through 2012, respondents who selected more than one Hispanic origin on the birth certificate were classified as Other Hispanic. Beginning with the 2013 data year, respondents who select more than one Hispanic origin are randomly assigned to a single Hispanic origin. This change was implemented to be consistent with the coding methods of the American Community Survey (19), on which the rates for the specified Hispanic groups as of 2010 and are based (see "Population estimates for specific Hispanic groups").

Race

This report presents data on race and Hispanic origin based on the 1997 Office of Management and Budget (OMB) standards (3). The 2003 revision of the U.S. Standard Certificate of Live Birth allows the reporting of the five race categories either alone (i.e., single race) or in combination (i.e., more than one race or multiple races) for each parent (11), in accordance with OMB's 1997 revised standards (3). The five categories for race specified in the revised standards are: American Indian or Alaska Native (AIAN), Asian, Black or African American, Native Hawaiian or Other Pacific Islander (NHOPI), and White; see User Guide (10).

Beginning in 2016, all states and D.C., in addition to Puerto Rico, U.S. Virgin Islands, Guam, and Northern Marianas, were reporting race according to the 1997 revised OMB standards, with 2.7% of mothers in the U.S. reporting more than one race (10) in 2018. Data from American Samoa still follow the 1977 OMB standards (20). Before 2016, the multiple-race reporting states varied widely, increasing from 6 states in 2003 to the 50 states and D.C. and all territories except American Samoa in 2016.

Gestational age

Beginning with the 2014 data year, NCHS transitioned to a new standard for estimating the gestational age of the newborn. The new measure—the obstetric estimate of gestation at delivery (OE)—replaces the measure based on the date of the last menstrual period (LMP) (17). National data based on the OE are available only from data year 2007 forward. Gestational age estimates differ somewhat between the OE- and LMP-based measures. Accordingly, gestational age data in this report are based on the OE. Information and discussion of the reasons for the change, and a detailed comparison of the two measures, are presented elsewhere (17).

Computation of percentages, percent distributions, and means

For information and discussion on computations of percentages, percent distributions, and means, see the User Guide (10).

Population denominators

The birth and fertility rates for 2018 shown in Tables 1, 2, 5, 8, 10, 11, and I–2 are based on populations estimated from the 2010 Census as of July 1, 2018. These populations are shown in the User Guide (10). The population estimates have been provided by the U.S. Census Bureau (21) and are based on the 2010 Census counts by age, sex, and race. The race categories are consistent with the revised 1997 OMB standards (3).

The birth and fertility rates by state shown in Table 8 are based on state-level population counts, which are based on the 2010 census data provided by the U.S. Census Bureau (21). Birth and fertility rates for all territories except Puerto Rico shown in Table 8 are based on population estimates provided by the U.S. Census Bureau's International Data Base (22). Rates for Puerto Rico are based on population estimates from the 2010 Census as of July 1, 2018, and are provided by the U.S. Census Bureau (23).

Rates by state and territory shown in this report may differ from rates computed on the basis of other population estimates; rates for smaller population subgroups, such as those for teen mothers, may be particularly affected by differences in population estimates. Birth and fertility rates by month shown in Table I–2 are based on monthly population estimates for 2018, which are also based on 2010 Census estimates. For 2018, rates for unmarried women shown in Tables 10 and 11 are based on distributions of the population by marital status averaged over a 2-year period for 2017–2018. These distributions were reported by the U.S. Census Bureau in the March Current Population Survey for each year (24,25), and have been adjusted to July 1, 2018 (2010 Census), population levels (21) by NCHS' Division of Vital Statistics (26).

The population distributions by marital status are based on a 2-year average for 2014–2018 (12). For earlier years, rates for unmarried women are based on population distributions by marital status averaged over a 3-year period (26).

Population estimates for specific Hispanic groups

The 2018 population estimates for the specific Hispanic population groups were not available as of the preparation of this report (27). Accordingly, birth and fertility rates for these groups for 2018 are not shown in this report. Once available, birth and fertility rates for the specific Hispanic population groups will be added to Table 12. The 2018 special population estimates for Hispanic groups will be shown in the User Guide, once available (10).

Computation of rates and percentages

An asterisk (*) in the tables indicates that a rate or percentage does not meet NCHS standards of reliability or precision. For population-based rates, an asterisk is shown in place of a rate based on fewer than 20 births in the numerator. For percentages, new standards have been adopted by NCHS and implemented for natality data beginning with the 2017 data year. The new standard is based on denominator size and on the absolute or relative widths of the confidence interval of the proportion or percentage calculated using the Clopper–Pearson method. This compares with the previous standard for which, similar to the standard for populations-based rates, an asterisk was used for percentages based on fewer than 20 births in the numerator. For detailed information on the new standard, see "National Center for Health Statistics Data Presentation Standards for Proportions" and the User Guide (10,28).

Random variation and significance testing for natality data

For information and discussion on random variation and significance testing for natality data, see the 2010 User Guide (10).

For information and discussion on random variation and significance testing for birth and fertility rates for Mexican, Puerto Rican, Cuban, Dominican, Central and South American, and other Hispanic populations based on ACS population estimates, see the User Guide (10).

Definitions of medical terms

For definitions and discussion of medical and health items presented in this report, see "Guide to Completing the Facility Worksheets for the Certificate of Live Birth and Report of Fetal Death" (29).

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National Vital Statistics Reports, Vol. 68, No. 13, November 27, 2019

Contents

Acknowledgments

This report was prepared in the Division of Vital Statistics (DVS) under the general direction of DVS Director Steven Schwartz; Isabelle Horon, Chief, Reproductive Statistics Branch (RSB); and Joyce Martin, Team Leader, RSB Birth Team. Rajesh Virkar, Chief of the Systems, Programming, and Statistical Resources Branch (SPSRB), and Steve J. Steimel, Annie S. Liu, and Jasmine N. Mickens provided computer programming support and statistical tables. Steve J. Steimel and Annie S. Liu of SPSRB prepared the natality file. The Data Acquisition and Evaluation Branch staff of DVS evaluated the quality of and acceptance procedures for the state data files on which this report is based. This report was edited and produced by NCHS Office of Information Services, Information Design and Publishing Staff: Laura Drescher edited the report, and typesetting and graphics were produced by Odell D. Eldridge (contractor).

Suggested citation

Martin JA, Hamilton BE, Osterman MJK, Driscoll AK. Births: Final data for 2018. National Vital Statistics Reports; vol 68, no 13. Hyattsville, MD: National Center for Health Statistics. 2019.

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Rules Related to Per- and Polyfluoroalkyl Substances (PFAS):
FP 2019-14, Env-Wq 402 amendments
FP 2019-15, Env-Or 603.03 amendments
FP 2019-16, Env-Dw 700-800 amendments

Summary of Comments on Initial Proposals with NHDES Responses
June 28, 2019

Three sets of proposed rules and rule amendments relate to four per- and polyfluoroalkyl substances (PFAS), specifically perfluorohexane sulfonic acid (PFHxS), perfluorononanoic acid (PFNA), perfluorooctane sulfonic acid (PFOS), and perfluorooctanoic acid (PFOA). The three sets of rules are as follows:

Env-Dw 700 & 800 (FP 2019-16) establishes maximum contaminant levels (MCLs, the drinking water standards with which public water systems must comply) for the four PFAS in public drinking water and adds monitoring, compliance, reporting, and public notice requirements for the four PFAS;

Env-Or 603.03 (FP 2019-15) establishes ambient groundwater quality standards (AGQS), for the four PFAS, that are required by statute to be equivalent to the MCLs established in Env-Wq 700; and

Env-Wq 402 (FP 2019-14) establishes water quality standards and procedures for discharges to groundwater of wastewater containing any of the four PFAS.

The purpose of this document is to summarize the comments NHDES received from the public on all three proposed rules and to identify the changes made to the proposed rules in response to the comments or explain the reason(s) why NHDES did not make changes. Comments received that were unrelated to the proposed rules are not addressed in this document. To provide a foundation for the comments and responses, brief explanations of the purpose of the rules and of the rulemaking process are provided, as well as a summary of the main provisions of the rules and an explanation of how the currently proposed MCLs/AGQS were derived. A list of commenters on the rules and all written comments received concerning the rules as well as the transcripts for the three public hearings can be found on the NHDES website by searching on "PFAS".

OLS also provided written comments, which have been addressed.

Purpose of Proposed Rules

Env-Dw 700 & 800 establishes MCLs and monitoring, compliance, reporting and public notice requirements for the four health-related regulated PFAS ("health-regulated PFAS") that will apply to all non-transient public water systems, as required by RSA 485:16-e. The final proposed MCLs and AGQSs are:

Contaminant	Final Proposed MCL/AGQS (Part Per Trillion (ppt))
PFHxS	18 ppt
PFNA	11 ppt
PFOS	15 ppt
PFOA	12 ppt

The rules also eliminate the requirement for the owner or operator (O/O) of a laboratory that is seeking approval for an alternate analysis method to identify the specific PWS for which the alternate method would be used, meaning that once an alternate method is approved, it could be used for any PWS.

Env-Or 603.03 is being amended to change the existing AGQS for PFOA and PFOS and to add AGQS for PFNA and PFHxS. As required by RSA 485-C:6, those AGQS are identical to the MCLs that are proposed to be established under Env-Dw 700.

Env-Wq 402 is being amended to establish requirements for discharges to groundwater of wastewater containing any of the four PFAS. Those requirements reflect the proposed changes to the AGQS that would be established under Env-Or 603.03 and are intended to accommodate the lack of available technology to treat large quantities of wastewater that is contaminated with certain PFAS. Specifically, the rules would:

(1) Include residual PFOA, PFOS, PFNA, and PFHxS in the existing conditional exemption for meeting AGQS under certain circumstances;

(2) Establish a discharge limit for PFOA, PFOS, PFNA, and PFHxS in wastewater discharged to groundwater;

(3) Account for exceedances of the applicable limits for PFOA, PFOS, PFNA, and PFHxS; and

(4) Include PFOA, PFOS, PFNA, and PFHxS in the treatment/alternative response requirements established for 1,4-dioxane which includes identifying and eliminating contributing discharges to the wastewater stream.

Summary of Rule Development Process

Laws of 2018, Ch. 345 directed NHDES to initiate rulemaking related to PFOA, PFOS, PFHxS and PFNA by January 1, 2019, to:

(1) Establish MCLs for PFOA, PFOS, PFNA and PFHxS; and

(2) Re-evaluate the current AGQSs for PFOA and PFOS, which currently is 70 ppt combined, and to establish AGQSs for PFHxS and PFNA. AGQSs are clean-up standards for contaminated sites. Existing law (RSA 485-C:6) has always required an AGQS to be the same as any established MCL for a contaminant. The AGQS are also used to determine appropriate discharge limits for groundwater discharge permits.

The law provided funding for a toxicologist and health risk assessor position, which were filled in October of 2018. Also in October, NHDES held three technical sessions -- in Concord, Merrimack and Portsmouth (Pease Tradeport) -- to provide stakeholders with the opportunity to submit or identify studies and research pertinent to deriving health based standards and addressing other considerations required by law, including occurrence, ability to detect and treat as well as the anticipated costs and benefits. After careful review of appropriate studies and other states' approaches, NHDES began rulemaking by filing a Request for Fiscal Impact Statement with the Legislative Budget Assistant (*see* RSA 541-A:5) on December 31, 2018.

The initial proposal included the following levels for MCLs and AGQSs:

Contaminant	**Initial Proposed MCL/AGQS** (Part Per Trillion (ppt))
PFHxS	85 ppt
PFNA	23 ppt
PFOS	70 ppt
PFOA	38 ppt
PFOA & PFOS Combined	70 ppt

In conjunction with initiating the rulemaking, NHDES issued the "Summary Report on the New Hampshire Department of Environmental Services Development of Maximum Contaminant Levels and Ambient Groundwater Quality Standards for Perfluorooctane sulfonic Acid (PFOS), Perfluorooctanoic Acid (PFOA), Perfluorononanoic Acid (PFNA), and Perfluorohexane sulfonic Acid (PFHxS)" on January 4, 2019 ("January 2019 Report").

After filing the initial proposed rules and rulemaking notices, NHDES held public meetings in Merrimack and Portsmouth (Pease Tradeport) to explain how the proposed standards were derived. The public hearings on the

proposed rules required by RSA 541-A were held in early March 2019 in Merrimack, Portsmouth and Concord. In addition to soliciting comments on the initial proposal, participants were asked to comment on the use of a toxicokinetic model developed by the Minnesota Department of Health ("MN model") to assess blood serum levels of people exposed to PFOA, including breastfed and bottle-fed infants. In the press release announcing the public hearings, NHDES informed interested parties that a preliminary assessment indicated that using the model would likely lower the proposed standards.

The final proposed rules reflect further research and new studies, the use of the MN model, consideration of comments received, discussions with other state and academic toxicologists, and professional judgement on what health-based standards will be sufficiently protective of human health over all life stages. While NHDES is unable to quantify all the costs and benefits associated with these proposed rules due to the emerging nature of these contaminants and the science related to them, after considering what currently is known about costs and benefits NHDES believes that the benefit of adopting these rules is not outweighed by the costs of implementing the proposed health based standards.

Summary of Significant Differences Between Initial and Final Rulemaking Proposals

1. The proposed MCLs/AGQSs have been lowered, primarily due to using the MN model.

2. The term "per- and polyfluoroalkyl substances (PFAS)" has replaced the term "perfluorinated compounds (PFCs)" throughout the document. PFAS is the more inclusive term and was used in most of the comments received, even though the rules currently do not include any polyfluorinated compounds.

3. The implementation requirements for public water systems have changed to reduce initial sampling frequency to two quarters if both samples come back with non-detects and to limit the maximum time between performing sampling to three years.

Technical Explanation of Proposed Lower MCLs/AGQSs and Updated Costs and Benefit Information:

Attachment 1 is "New Hampshire Department of Environmental Services Technical Background for the June 2019 Proposed Maximum Contaminant Levels (MCLs) and Ambient Groundwater Quality Standards (AGQSs) for Perfluorooctane sulfonic Acid (PFOS), Perfluorooctanoic Acid (PFOA), Perfluorononanoic Acid (PFNA), and Perfluorohexane sulfonic Acid (PFHxS)" dated June 28, 2019 ("June 2019 Report"). It also includes findings of a peer review of NHDES's derivations conducted by Stephen M. Roberts, Ph.D.

Attachment 2 is an update on cost and benefit considerations.

Comments and Responses

General and technical comments concerning this rulemaking are categorized and listed below. Note that in addition to revisions discussed below, revisions have been made to each of the rules put the four compounds in alphabetical order.

General Comments Related to Proposed MCLs/AGQSs

Comments: The proposed MCLs and AGQS should be lower. A number of comments suggested the standards should be at 1 ppt combined. Others suggested that NHDES should adopt the lower advisory numbers adopted by other states or, in the case of New Jersey, its MCL for PFNA.

The proposed MCLs and AGQS should be higher. A few comments were received that urged NHDES to look at recently established health advisories in Canada and elsewhere that would increase the standards initially proposed.

Response: NHDES considered all of these comments and carefully reviewed all existing advisories and standards set elsewhere. However, the process used by NHDES incorporates long-established methodologies for setting standards that use the most current, defensible science and incorporates expert professional judgements. The resulting proposed standards are protective of human health at all life stages. Specific criticisms of factors used in the derivation of the standards are in the technical comments table.

Comment: NHDES did not have sufficient time, resources, or expertise to derive the standards and should collaborate with other state toxicologists and health risk assessment teams working on health advisories and standards.

Response: A full time toxicologist and a full-time and part-time health risk assessor along with contractor support and collaboration with academic, state, and federal agency health risk assessors and toxicologists provided the necessary expertise and effort to derive the standards for the final proposed rules. Their work and that of others at NHDES included routine meetings through state organizations such as Environmental Council of the States, Association of State Drinking Water Administrators, Northeast Waste Management Officials Organization, New England Interstate Water Pollution Control Commission, Interstate Technical and Regulatory Council, and the Federal-State Toxicology Risk Assessment Committee, all of which enhanced the agency's ability to meet the deadlines established by law. Because of the emerging nature of these contaminants, limitations are inherent in the amount of data and research available. NHDES made full use of available experts, science, and occurrence data in development of these proposed rules.

Comment: Laboratories cannot achieve a 2 part per trillion (ppt) reporting limit.

Response: NHDES has confirmed with the NH Environmental Laboratory Accreditation Program (NH ELAP) and the U.S. Environmental Protection Agency that a 2 ppt reporting limit is achievable.

Comment: NHDES should set a Maximum Contaminant Limit Goal (MCLG) for all PFAS at zero.

Response: NHDES agrees that there should be no man-made contaminants in New Hampshire's drinking water. However, these rules apply only to PFOA, PFOA, PFHxS, and PFNA, not the large class of chemicals to which they belong (*i.e.*, PFAS). The initial proposal included an MCLG of zero for each contaminant, which is consistent with the MCLG for other man-made chemicals and which is retained in the final proposed rules.

Comment: NHDES should review the science on PFAS every 2 years.

Response: Laws of 2018, Ch. 345 requires NHDES to review all AGQS every five years. Because of the evolving science related to PFAS, NHDES's health risk assessment team will monitor the science on an ongoing basis and will update the relevant rules as needed.

Comment: NHDES should have another public comment period if the standards change.

Response: NHDES solicited extensive public input and held three public hearings on the initial proposal, which resulted in 857 pages of comments on the rules. NHDES believes another public comment period will unduly delay adoption of the drinking water and ground water standards while providing few new perspectives that would alter the final proposed rules. Given the evolving nature of the science on these compounds, NHDES recognizes that revisions of the current rules to reflect new science may occur.

Comment: A Treatment Technique should be specified for these contaminants verses setting individual MCLs.

Response: A Treatment Technique is a tool under the state and federal Safe Drinking Water Acts used to lower the exposure to a contaminant through drinking water when an MCL cannot be set, which is not the case for these compounds. In addition, RSA 345 directs the NHDES to set an MCL for PFOA, PFOS, PFHxS, and PFNA.

General Comments Related to Costs and Benefits

Comment: The costs and benefits to affected parties that will result from establishing the new standards were not adequately quantified, did not follow federal requirements related to adopting MCLs, and did not identify the marginal costs and benefits at different MCL levels for each contaminant.

Response: Because NHDES was mandated by the Legislature to establish the MCLs and AGQS, any costs attributable to the standards are directly attributable to the law, not the rules. However, NHDES was able to estimate certain costs associated with standards for the four PFAS as explained in the January 2019 Report. These costs have been updated as shown in Attachment 2 for the final proposed MCLs and AGQS.

NHDES was not able to quantify the benefits (*e.g.*, avoided health care costs) in the initial proposal but was able to qualitatively explain the types of benefits that would result, and a future quantification may be possible (as explained in the January 4, 2019 report). In Attachment 2, NHDES has provided a summary of a recent report prepared by the Nordic Council of Ministers "The cost of Inaction: A socioeconomic analysis of environmental and health impacts linked to exposure to PFAS". This document provides further evidence of the benefits of setting health-based standards for these compounds that are protective of human health at all life stages, although NHDES could not directly estimate benefit for these four specific compounds for NH citizens using the report's methodologies. NHDES also provides information on a study that estimates costs related to low birthweight: "Perfluorooctanoic acid and low birth weight: Estimates of US attributable burden and economic costs from 2003 through 2014".

NHDES interprets the language in the statute regarding costs and benefits as a requirement to quantitatively estimate cost and benefit so far as the data is available to do so and to consider all that is known related to cost and benefit. Where needed data is lacking, NHDES has provided a qualitative description of what is known related to cost and benefit that was considered for this rule. The NH Department of Justice was consulted regarding the interpretation of some commenters regarding the lack of a comprehensive cost benefit analysis and identification of marginal costs consistent with federal procedures. The Office of the Attorney General found NHDES's interpretation of the requirement under RSA 485:3, I(b) to be reasonable and lawful (see Attachment 3). Because of the emerging nature and limitations of data for these chemicals and their impact to health, the quantification necessary to perform an analysis beyond what is currently provided for costs and benefits is not possible.

Comment: *Costs and benefits were not considered in establishing the proposed standards.*

Response: NHDES considered what is known about costs and benefits and determined that using the health-based numbers is appropriate, achievable, and necessary to protect human health at all life stages, as required by Laws of 2018, Ch. 345.

Comment: *Benefits can be calculated by assuming PFAS causes cancer.*

Response: The links between PFOA, PFOS, PFHxS, or PFNA and cancer are not sufficiently clear; it is not appropriate to base benefit on a health outcome that is still being studied.

Comment: *Costs are largely born by municipalities for landfill, fire station, wastewater residuals, and public drinking water system compliance with the new standards. The state should pay for these costs.*

Response: NHDES recognizes that there will be significant costs to municipalities resulting from the legislative directive to establish standards. Cost considerations are reflected in the proposed reduction in sampling required at public water systems to demonstrate that ongoing reduced sampling is appropriate. Also, the proposed provisions that will allow groundwater discharges containing PFAS above twice AGQS to occur in certain circumstances (*i.e.*, only if no impacted wells) provided that likely sources of PFAS are identified and eliminated, reflects the reality that municipalities need to economically discharge wastewater. There is currently no new source of state funding established to assist municipalities with the costs associated with the rules. Capital costs for public water system compliance with the new MCLs will be eligible for existing state and federal low interest loan and grant funds.

Comment: *Costs to small and rural public water systems with fewer customers will be significant.*

Response: NHDES agrees that Laws of 2018, Ch. 345 resulted in costs related to achieving compliance with the MCL for all public water systems, and that small systems have a smaller rate base to absorb cost increases. This has always been true for small systems, which under the federal and state Safe Drinking Water Acts must comply with all MCLs. Low interest loans and grants from the Drinking Water State Revolving Fund and other state and federal sources will continue to be available to small systems.

Comment: *NHDES should alter cost estimates for public water systems based on a study prepared for Merrimack Village District (MVD) and should make assumptions based on the potential use of more expensive technologies, variations in water quality, and the potential increases in costs to systems already*

treating rather than using the range of actual treatment and ongoing cost approach described in the January 4, 2019 report.

Response: NHDES reviewed the study prepared by Underwood Engineering for MVD and found the estimates consistent with those used to estimate costs in the January 2019 Report, as supplemented by the update in Attachment 2. NHDES considered all comments related to the assumption that the range of initial treatment and annual costs can be based on what actual costs have been incurred by public water systems. After doing so, NHDES continues to believe that this approach is the best way to quantify both initial treatment and ongoing costs. This approach includes both new technologies and granular activated carbon; NHDES believes those instances where more expensive treatment is selected is balanced by systems that will choose to blend, interconnect with another system, or take contaminated wells off line. Similarly, the annual cost estimate includes systems achieving water quality at lower levels than is required by the current AGQS and is potentially an overestimation for systems which may choose to blend, interconnect, or take a well off line rather than treat.

Comment: NHDES should have provided an order of magnitude or contingency cost for the potential sources of contamination for which they could not quantify costs due to insufficient data.

Response: Because of limited testing to date at a number of potential sources (*e.g.*, fire stations, oil remediation sites, biosolids/sludge/septage processing and application sites, air deposition sites, *etc*.), NHDES was unable to estimate the costs that could be associated with them. This same lack of information precluded the derivation of a possible contingency figure. Since that time NHDES has continued to investigate PFAS occurrence and has an improved data set for certain sources. For instance, while the initial report indicated that as many as one third of fire stations may have caused PFAS contamination in nearby wells through the use and storage of fire-fighting foams, more recent data indicates a much lower occurrence. Also, additional testing at oil remediation sites indicates little association of PFAS occurrence.

Comment: NHDES should have quantified costs that may occur due to establishing AGQSs and MCLs associated with residential septic tanks, residual management, leachate disposal, and landfill gas.

Response: NHDES does not have sufficient data to determine if these potential sources would result in a violation of the MCLs/AGQS being proposed, nor is there sufficient occurrence data to estimate costs.

Comment: NHDES should provide the present value of long-term monitoring on sites with a groundwater management permit that violate an AGQS for any of these compounds.

Response: Because of the persistent nature of these chemicals, costs associated with monitoring to ensure permit compliance is likely to be longer term than for more biodegradable contaminants. There is insufficient data to determine the length of time to be used in such a calculation, but NHDES acknowledges that the annual cost estimated will continue for many years.

Comment: The three rules create an unfunded mandate that is a violation of Article 28-a of New Hampshire's Constitution and RSA 541-A.

Response: The costs of implementing the rules are not attributable to the rules, but derive directly from the statutory mandate for NHDES to adopt standards. Because the costs are exclusively attributable to Laws of 2018, Ch. 345, the rules do not violate Part I, Article 28-a of the New Hampshire Constitution.

However, even if costs could be attributable to the rules, the costs are within the scope of modifications allowable under *City of Concord v. State of New Hampshire*, 164 N.H. 130 (N.H. 2012). In *City of Concord*, the Court reviewed all prior decisions on the same issue and concluded that:

> Collectively, these cases stand for the proposition that where a local subdivision has historically had responsibility for the subject matter of the mandate, some change in the scope of that responsibility does not result in a violation of Article 28-a.

City of Concord at 140 (footnote omitted). The Court further stated "Accordingly, we conclude that to constitute a new, expanded or modified 'responsibility,' the state action must impose some **substantive**

change to an underlying function, duty or activity performed or to be performed by local government." *Id.* at 141-142 (emphasis added).

Because municipalities and other political subdivisions historically have been required to test the drinking water supplied to the public for contaminants, the addition of the PFAS contaminants to the list of required testing does not violate Part I, Article 28-a.

For the same reasons, the rules do not violate RSA 541-A:25. RSA 541-A:25, I simply restates Part I, Article 28-a and then adds that programs covered include "those municipal functions which might be undertaken by a municipality or by a private entity and those functions which a municipality may legally choose not to undertake." RSA 541-A:25, III. The analysis in *City of Concord* does not depend on whether a political subdivision is legally required to undertake a program or responsibility, and so applies to RSA 541-A:25 as well.

Comments Related to Occurrence and Contamination

Comment: There is not sufficient occurrence data to determine the need for MCLs/AGQSs.

Response: NHDES and others have done extensive sampling throughout New Hampshire that includes public water systems, wells near many likely sources of PFAS contamination, and wells in areas that do not have likely sources of contamination. The occurrence data is described in the January 2019 Report. Since January, additional contamination at public water systems, hazardous waste sites, landfills, and other potential sources has been documented.

Comment: Contamination should be treated differently if from a diffuse source versus contamination related to industrial activity and the use of fire-fighting foams.

Response: NHDES statutes related to waste sites do not distinguish between sources of contamination.

Comment Related to Studies Received

Comment/Response: NHDES was provided with numerous studies for consideration in the derivation of the standards and a few references for establishing benefits. To the extent the health studies were relevant to PFOA, PFOS, PFNA, and PFHxS, they were reviewed by the health risk assessment team. The bibliography of health studies used in derivation of the standard can be found in the June 2019 Report.

Comments Related to MCL Implementation at Public Water Systems (Env-Dw 700 & 800)

Comment: The rules should align with initial monitoring precedents set in the NH Code of Administrative Rules for radionuclides and synthetic organic compounds (SOC), which allow the ongoing routine monitoring schedule to be determined after two quarters of non-detects versus four quarters.

Response: NHDES agrees with this comment and has revised the rule accordingly.

Comment: The proposed monitoring frequency is not protective of public health. Quarterly sampling should be required for any detection and annual sampling should occur at all public water systems.

Response: NHDES agrees that due to the ubiquitous nature of these four PFAS and the proposed lower MCL standards, the sampling frequency in the proposed initial rules may be insufficient. The rules have been changed to require quarterly sampling for systems with sample results above an MCL or systems with treatment to remove PFAS, annual sampling for systems with sample results greater than 50% of the MCL up to the MCL, and monitoring every three years for systems with sample results less than or equal to 50% of the MCL.

Comment: Env-Dw 712.23 (c) and (d) should be eliminated because they are too vague and unnecessarily complicate a determination of compliance.

Response: This is identical to language required for VOCs. However, the language has been eliminated as statistical variations of concern can be addressed under Env-Dw 708.01(d).

Comment: Tables 712-1 and 712-2 should contain consistent terminology.

Response: The two tables do not overlap, so it is unclear what terminology is not consistent.

Comment: Public Water Systems will need assistance with implementation and communication related to the new MCLs.

Response: NHDES intends to continue to work with public water systems and their trade organizations to understand what is required by the rules and to effectively communicate that with their customers about PFAS and the new rules.

Comments applicable to Groundwater Discharge Permit Rules (Env-Wq 402)

Comment: There should be no exception in the rules for discharges of wastewater containing PFAS to groundwater that result in exceedances beyond the groundwater management zone as is now allowed for 1,4 dioxane. Specifically, if no wells are impacted, the rule would allow the permittee to identify and eliminate the PFAS versus halting the discharge.

Response: Because of both the current inability of treating large quantities of wastewater and the need for wastewater disposal, NHDES believes that this provision is necessary and is in keeping with the pre-treatment requirements in the Clean Water Act.

Comment: Requiring the AGQS to be met in treated wastewater being discharged to groundwater eliminates the opportunity for the level to naturally decline prior to reaching the boundary.

Response: The intent of establishing the values in Table 402-2 for treated wastewater effluent being discharged to groundwater is to assess the likelihood of whether one or more facilities that are connected to a wastewater treatment facility are contributing substantially high concentrations PFAS discharges to its incoming wastewater stream that, in turn, result in high PFAS concentration in its effluent discharged to groundwater, which then results in a groundwater quality standard violation. Based on a limited dataset of PFAS results in influent and effluent at wastewater treatment facilities, establishing the threshold values in Table 402-2 at twice the revised proposed MCLs "weeds out" wastewater treatment facilities that have low concentrations of PFAS in their incoming wastewater stream that are likely related to domestic-consumer wastewater discharges only. Wastewater treatment facilities that are known to NHDES as having have individual connections to their sewer systems that contribute high PFAS loads have substantially higher PFAS concentrations in its treated wastewater effluent and will by captured by the revised values in table 402-2 (*i.e.*, twice the proposed standards).

Comment: NHDES' proposed rules related to the discharge to groundwater of wastewater containing PFAS fail to properly protect public health.

Response: The proposed rules specifically require that sources of drinking water be fully protected from potential contamination associated with groundwater discharges. The proposed groundwater discharge rules protect New Hampshire's groundwater by:

- Ensuring that permittees:
  (1) Monitor groundwater quality around permitted discharge sites;
  (2) Not cause any private or public drinking water supply sources to be contaminated by PFOA, PFOS, PFNA, or PFHxS at concentrations that exceed the proposed MCLs; and
  (3) Provide treatment or alternative drinking water when sources of water that have been contaminated at levels above the MCL due to the permittee's discharge.
- Requiring that permittees reduce the concentration of PFOA, PFOS, PFNA, and PFHxS in wastewater that is discharged to groundwater by reducing or eliminating discharges of these compounds into the wastewater system.
- Limiting the maximum amount of PFOA, PFOS, PFHxS, and PFNA that is allowed to be discharged to groundwater.

- Ensuring no groundwater discharge contributes to a violation of surface water quality standards. That is, should New Hampshire adopt a surface water quality standard for PFAS in the future, permitted groundwater discharges impacting surface water will be subject to these standards.

These actions, along with the reduction and/or phase-out of the use of these compounds in commerce, will help to ensure groundwater quality will be improved and protected at permitted discharge sites. NHDES does not agree that the proposed rules should require treatment based on the potential for the development of future technologies capable of treating large quantities of wastewater at a public wastewater treatment plant are not currently available.

General Comments Related to Health-Based Risk Assessment[1]

Comment: NHDES should have derived a health-protective drinking water value based on cancer effects in animal studies instead of non-cancer health effects.

Response: NHDES reviewed both human and animal studies investigating the cancer-causing potential for PFOA and PFOS. There are currently no peer-reviewed and published rodent model cancer studies for PFNA or PFHxS. There is limited evidence associating PFOA and PFOS with altered cancer risk, and the uncertainties of this were discussed in the January 2019 Report as well as other agencies (EPA 2006; EPA 2016ab; MDH 2017; ATSDR 2018). The U.S. EPA (EPA) has classified the carcinogenic potential of PFOA and PFOS as "suggestive", which is the lowest cancer classification category given the evidence for human cancer potential (EPA 2016ab).

EPA and the New Jersey Drinking Water Quality Institute (NJDWQI) have developed different numerical cancer guidelines for PFOA based on testicular cancer set at a one-in-one million cancer risk for a 70-year exposure from drinking water. In 2016, EPA determined a cancer value of 500 ng/L (EPA 2016a), while the following year NJDWQI calculated a different cancer value of 14 ng/L (NJDWQI 2017). The difference in calculated values is due to the limited quality of the available studies and variations in toxicokinetic adjustments. Regardless of which value is more accurate, the proposed PFOA MCL of 12 ng/L based on a non-cancer endpoint is below the more conservative of the aforementioned values (14 ng/L; NJDWQI 2017). The PFOS cancer evidence is even more uncertain than that of PFOA and not adequate for quantitative evaluation. Should federal agencies make new determinations about the carcinogenicity of these compounds, or should new studies arise that present clear evidence of carcinogenic potential in humans, NHDES will evaluate the new information and take such action as is appropriate.

Cancer is a complex and multifactorial group of diseases. Regional differences in cancer rates may be due to the interaction of multiple factors, including individual lifestyle choices, genetic susceptibility factors, and variations in exposure to physical, chemical, and biological agents in the environment. Based on the currently-available evidence, NHDES determined that a non-cancer health endpoint was more sensitive and more reliable for developing a health protective standard. NHDES agrees that additional research is needed to understand the broader health impacts of these contaminants on outcomes, including cancer.

Comment: The proposed MCLs should be protective across all human life stages, including but not limited to fetuses, neonates, infants and children.

Response: NHDES's adoption of the transgenerational model for the currently proposed MCLs is intended to be protective of all life stages. The exposure estimates used are from the 95th percentile water consumers, which is additionally protective for typical (average) water consumers. The use of the transgenerational model allows for determination of an MCL with a margin of safety across all life stages based on consideration of the health studies and toxicological reviews (*e.g.*, ATSDR 2018) evaluated by NHDES. The predicted contributions of drinking water to blood concentrations at the proposed MCLs are similar to background levels reported by the National Health and Nutrition Examination Survey (NHANES).

Additionally, NHDES selected critical health effects from animal studies based on sound evidence for human health relevance and were equally or more sensitive than developmental or teratogenic effects

[1] List of references begins on page 21.

observed in rodents. The human health relevance of many toxic responses observed in rodents is an ongoing area of research, and subject of debate amongst toxicologists because of a currently limited understanding of which species is more sensitive to PFAS at identical internal doses. Some developmental effects in rodents have been reported at remarkably lower doses of certain PFAS (*e.g.*, delayed mammary gland development in response to PFOA), and similar to NHDES, other agencies have declined to use these endpoints as the basis of their risk assessments and subsequent drinking water values (MDH 2017; NJDWQI 2017; EPA 2016a; ATSDR 2018; MIDHHS 2019). As concluded by other agencies, the cross-sectional or ecological studies of human health effects do not provide a sound basis for reference dose (RfD) determination, or demonstration of causality, and were therefore not used for direct calculation of RfDs. Such studies were used for evaluating the potential human health relevance of reported effects in animal studies.

Comment: *NHDES should be regulating all PFAS that are now in some people's drinking water.*

Response: In 2018, the Legislature decided that sufficient scientific information existed to determine whether the four PFAS covered by this rulemaking posed a health risk in drinking water, and mandated this rulemaking in Laws of 2018. Ch. 345. The Agency for Toxic Substances and Disease Registry (ATSDR) did not derive MRLs for other PFAS such as GenX, PFHpA, PFHxA, *etc*. NHDES is reviewing emerging studies to determine whether there is sufficient data to derive reference doses for other PFAS; this work includes consideration of draft toxicity assessments from EPA for PFBS and GenX. The work also includes consideration of future RfDs proposed by the EPA through the Integrated Risk Information System (IRIS) program for the following PFAS: PFBA, PFHxA, PFHxS, PFNA and PFDA.

Comment: *PFAS should be regulated as a "class" or "sub-class" and there should be a standard for total PFAS, or at least a combined standard for the four currently being regulated.*

Response: NHDES agrees that there is a need for an evidence-based class or subclass regulation of PFAS given the wide-spread occurrence and chemical diversity of this contaminant family. However, NHDES determined that differences in the most sensitive health effects, individual toxicokinetics and a lack of relative potency factors for PFAS do not support the assumption of identical (*i.e.*, 1-to-1) risks from exposure. Variation in the combinations of functional groups and carbon chain length appear to produce differences in biological activity (*e.g.* receptor and protein affinity) and the half-lives of individual PFAS. As discussed in the initial proposal (NHDES 2019), toxicity equivalency factors or other approaches have not been developed for this class of contaminants and highlights a critical research need. NHDES is aware that this is an active area of research and is therefore continuing to monitor publications on methods for this approach. Should a robust and scientifically-defensible approach to group regulation be developed, NHDES will consider its application in future development of drinking water standards for PFAS.

Comment: *The standards proposed by NHDES are different from the health advisory values, screening levels or MCLs developed by other states.*

Response: NHDES derived Maximum Contaminant Levels (MCLs) using standard EPA methodologies. Under the New Hampshire and federal Safe Drinking Water Acts, an MCL is the highest level of a contaminant that is allowed in drinking water delivered by public water systems. MCLs are enforceable standards (EPA 2018). To date, only New Jersey has established an MCL for any PFAS; for PFNA, at 13 ng/L (NJ DEP SRP 2019). Values developed by ATSDR (*e.g.*, Minimal Risk Levels (MRLs)) and other values derived in certain States (*e.g.*, Health Based Guidance Values (HBGVs)) are not enforceable and are largely intended to be used as guidance for site remediation and other public health responses.

NHDES understands the public's concerns regarding the initially proposed standards and the existing patchwork approach to regulatory standards for PFAS. This patchwork of regulatory standards underscores the need for action by EPA to harmonize standards for these wide-spread environmental contaminants.

The proposed final MCLs/AGQSs are similar to the standards set by other States, and are protective for the individual PFAS given the conservative exposure assumptions selected by NHDES. NHDES has collaborated and consulted with other states' health risk assessment teams that have been involved in deriving health advisories or are working towards setting MCLs. The collaborations included both formal

multistate conference calls and direct communications to discuss advances in PFAS toxicology and the rationale for each state's particular standard setting approach.

Comment: *NHDES should apply the precautionary principle in their health-based risk assessment.*

Response: The precautionary principle (PP) refers to a risk management strategy used by European Union countries when there is incomplete scientific knowledge of the risk to human health or the environment from chemicals/technologies. In the strictest interpretation, the PP recommends not using the substance or employing the technology at all until the risk is better understood. Like other U.S. state agencies, NHDES does not apply the PP as a default approach to health risk assessment of chemical contaminants.

NHDES did not apply the PP because application of the PP is inconsistent with risk assessments developed by other states and federal agencies (*e.g.*, US EPA and ATSDR). To date, no federal or state agencies have used the PP approach to develop PFAS drinking water criteria. Standard approaches used by federal and state agencies include weight-of-evidence considerations and the application of standard inputs for exposure considerations and uncertainty factors. The ubiquity of PFAS across environmental media makes application of the PP unreasonable. Furthermore, PFAS are already detected in the environment and a growing number of commercial and consumer products. NHDES's mandate is to use the best available scientific studies and data to determine concentrations in drinking water that will not present an appreciable health risk to water users throughout their lives. NHDES does not have the authority to ban PFAS from being used.

While NHDES did not conduct its assessment under the guidance of the precautionary principle, NHDES was conservative in its risk assessments of PFOA, PFOS, PFNA, and PFHxS. NHDES agrees that the proposed MCLs for PFAS should be based on exposure and effects in the most sensitive subpopulation to be protective of the broader population; that is the reason NHDES used the MN transgenerational toxicokinetic model to revise the initially proposed MCLs. In using the MN model, NHDES considered a protective reasonable exposure scenario of 12 months of exclusive breastfeeding. The 95th percentile ingestion rates were used for breastmilk consumption and water consumption across a lifetime. The newborn is the most exposed population due to placental transfer and subsequent exposure from breastfeeding or water-reconstituted formula at ingestion rates that are significantly higher for infants than for adults. Examples of upper level ingestion rate differences include: 1 to 3 months of age, water ingestion = 267 mL/kg-d; 1 to 3 months of age, breastmilk ingestion = 190 mL/kg-d; adult (21+ years), water ingestion = 44 mL/kg-d. Infants are also considered to be the most sensitive population to potential adverse health effects because of their rapidly developing bodies. Use of the MN transgenerational model to protect the most vulnerable population has significantly reduced the proposed MCLs and established a protective margin of exposure across a lifetime.

Comment: *NHDES's proposed reference doses and MCLs are different from the CDC Agency for Toxic Substances and Disease Registry's (ATSDR) minimal risk levels and drinking water screening values.*

Response: NHDES did not adopt the Agency for Toxic Substances and Disease Registry's (ATSDR) provisional minimal risk levels (MRLs) as the basis for its proposed maximum contaminant levels (MCLs) because: (1) MRLs are not synonymous with MCLs, (2) MRLs are developed by the CDC for use in screening impacted sites, and (3) NHDES determined different reference doses (RfDs) based on consideration of other sensitive health effects reported in animal studies. Additionally, the MRLs are currently only provisional, and are subject to change in response to public comments submitted on ATSDR's 2018 draft toxicological profile.

To the first point, an MRL is not developed to serve as an MCL or other actionable standard. As stated by the ATSDR:

> "These substance specific estimates, which are intended to serve as screening levels, are used by ATSDR health assessors and other responders to identify contaminants and potential health effects that may be of concern at hazardous waste sites. **It is important to note that MRLs are not intended to define clean up or action levels for ATSDR or other Agencies**."- (ATSDR, 2018, emphasis in original)

An ATSDR MRL is used for screening environmental media and to make decisions about additional surveillance and study planning at a site. Exposure at or above an MRL screening value does not mean that adverse health effects will occur (ATSDR, 2018). Thus, acknowledging the intention behind MRL development and application, NHDES did not use the provisional ATSDR MRLs for MCL development.

Using EPA methodology, RfDs are developed for calculating actionable drinking water standards. There are several chemical substances whose MRL value is not identical to the corresponding RfD as proposed in the Integrated Risk Information System (IRIS) Database. In some cases, such as PFAS, the ATSDR MRL is lower than the RfDs proposed by the USEPA IRIS Database (*e.g.*, PFOA, PFOS, and benzene). In other cases, the MRL is a higher value than the more protective RfD values proposed using EPA methodology (*e.g.*, 1,4-dioxane and nitrate). Such differences can arise from the determination of human health relevance, application of uncertainty factors, and other technical considerations used to translate findings from animal studies into estimates for protecting human health. Based on its evaluation of peer-reviewed studies as well as risk assessment work conducted by other state and federal agencies, NHDES derived RfDs for PFOA, PFOS, PFNA and PFHxS with its justifications detailed in Section III of the June 2019 Report.

Comment: *NHDES should consider the roles of biological plausibility and reverse causation in the reported associations between PFAS and human health outcomes.*

Response: In its initial proposal and re-evaluation of human health evidence (*i.e.*, epidemiological studies), NHDES considered the issues of confounding factors and reverse causation as they related to associations between PFAS and human health outcomes. NHDES disagrees with the statement of one commenter, who asserts "*confounding and/or reverse causation which (have) been shown the likely explanation for several reported epidemiological associations*". NHDES acknowledges there are confounding factors and limitations to some of the existing epidemiological studies on PFAS-associated health impacts. These limitations in the currently available epidemiological database make it difficult to demonstrate causality between PFAS and certain health outcomes (reviewed by ATSDR, 2018). However, this does not dismiss the fact that PFAS possess biologically-active properties in humans and therefore necessitates determination of acceptable levels of exposure from drinking water.

Confounding factors are variables other than the variable of interest (*e.g.*, a PFAS) that can influence the health outcome under investigation. One example from epidemiological studies of PFAS is co-exposure to other environmental contaminants and stressors. Many epidemiological studies are cross-sectional in design, which means they cannot account for historic exposures to other chemical or physical agents. Other environmental contaminants that possess dramatically shorter half-lives than these four PFAS are unlikely to be measured and are therefore unaccounted for in statistical analyses. Arguably, this could result in associations between health outcomes and PFAS due to their long physiological half-lives when other chemicals, that have been eliminated from the body, may have contributed to or caused the health outcome. Similarly, another confounding factor is the interplay of multiple PFAS aside from PFOA, PFOS, PFNA, and PFHxS. There is clear evidence that other PFAS are present in the blood of the U.S. population (reviewed by ATSDR, 2018), but the lack of any toxicity data for the majority of these compounds presents a major source of uncertainty for risk assessors and serious concern for the broader public.

Regarding PFAS, reverse causation would occur when certain health conditions elevate internal concentrations of PFAS. This could result from a certain health condition impairing the body's ability to eliminate PFAS, resulting in a correlation between markers of the disease and PFAS despite PFAS having no role in the origins of the disease. An example of this was discussed by Dhingra *et al.* (2017) and the Michigan Panel (2018), where negative associations of PFAS (*i.e.*, PFOS and PFOA) with uric acid levels and estimated glomerular filtration rates may be the result of reverse causation as impaired kidney function would result in elevated serum PFAS concentrations. NHDES selected health effects for the proposed MCLs after consideration of evidence from human epidemiological studies, as well as supporting evidence from controlled animal studies that are not as prone to the issue of reverse causality.

Evidence from studies of populations across different geographies (*e.g.*, C8 in Ohio, Frisbee *et al.*, 2009, Winquist *et al.*, 2013; and the Danish National Birth Cohort, Olsen *et al.*, 2001; Ernst *et al.*, 2019) support

the contention that PFAS are associated with health markers at exposure levels seen in background, community drinking water, and occupational settings. As with many epidemiological studies, these have limitations and further research is required to clarify the relationship between PFAS and human health outcomes. NHDES used the existing evidence to protect public health given the widespread occurrence of PFAS, the significance of exposure from drinking water, and the lack of toxicity data for these and other PFAS. There is sufficient consistency between epidemiological studies and animal models to indicate that PFAS elicit adverse biological activity from certain organ systems (*e.g.*, liver, immune, endocrine, reproductive). As the existing scientific literature regarding the health effects of PFAS has not kept pace with their widespread applications and dispersal into the environment, NHDES expects future studies will improve our understanding of health effects and acceptable levels of exposure. NHDES will continue to review emerging science for the re-assessment of the MCLs within 5 years of implementing the finalized values and will take such action as is appropriate.

Comment: *Certain references should be updated, or were omitted, from the initial proposal.*

Response: NHDES has updated their list of health impacts to include those referenced on pages 5-6 of the 2018 draft ATSDR Toxicological Profile for Perfluoroalkyls. This updated list is found in the Executive Summary of the June 2019 Report.

The reference for "PPARa activation in humans does not result in the same peroxisome proliferation effects but does induce changes in lipid metabolism and gene transcription." is: Tyagi S, *et al.* 2011. The peroxisome proliferator-activated receptor: A family of nuclear receptors role in various diseases. J. Adv. Pharm. Tech. Res., 2(4), 236-240.

The references for the human half-lives cited for PFOA (2.3-3.8 years) are (Olsen *et al.*, 2007; Bartell *et al.*, 2010); PFOS (5.4 years)(Olsen *et al.*, 2007; Bartell *et al.*, 2010); PFHxS (8.5 years)(Olsen *et al.*, 2007); PFNA (2.5 years)(Zhang *et al.*, 2013, ATSDR 2018). The reference for half-life data used in the calculations for PFOA, PFOS and PFHxS in the initial proposal is Li *et al.* 2018.

Comment: *In addition to these four PFAS NHDES needs to ban fluoride.*

Response: This rulemaking is not related to fluoride; it relates to regulatory standards for PFOA, PFOS, PFNA, and PFHxS. The four PFAS are organic compounds that contain fluorine. These organic compounds and their properties are distinctly different from fluoride (F^-), which is an anion or negatively charged element that is not synonymous with PFAS. Individual communities in NH determine their own drinking water fluoridation practices, and NHDES does not have authority over supplementation of fluoride into commercial personal care products.

Technical Comments Related to Application of the Minnesota Transgenerational Exposure Model (Goeden *et al.*, 2019)

Comment: *On February 21, 2019, NHDES solicited technical stakeholder input on the appropriateness of a toxicokinetic exposure model, or the Minnesota model (Goeden et al., 2019), for deriving the proposed MCLs. The majority of comments recommended its use based on technical merit, and a few commenters noting concerns with the model's limitations.*

Response: NHDES agreed with technical comments recommending the application of the transgenerational breastfeeding model developed by the Minnesota Department of Health (MN model). Details on the application of this model and factors applied by NHDES are found in the June 2019 Report.

After reviewing the MN model, NHDES concluded that this approach would be appropriately protective across all life stages after consideration of reasonable exposure scenarios. As discussed in the June 2019 Report, there are uncertainties and limitations with using this or any risk assessment tool for developing health-protective drinking water values. In spite of these uncertainties, NHDES has concluded that the extraordinary half-lives of these PFAS, combined with their transfer rates into breastmilk, merit consideration in the risk assessment supporting the proposed MCLs.

Some commenters urged the use of the unpublished version of this tool prior to its publication in January 2019 as the Minnesota Department of Health (MDH) had previously recommended non-MCL values for PFAS in drinking water. Scientific publications undergo a peer review process to ensure necessary feedback is garnered on methods, results, and conclusions, and the reviewers are tasked with assessing the quality of information in terms of both accuracy and validity. The document was undergoing the peer review and publication process at the time the initial MCLs were being developed for this rulemaking and did not follow traditional risk assessment methods. NHDES did not know what experts in modeling would have recommended or suggested based on their peer review of the model.

Until the current proposal by NHDES, this model has not been applied to determine protective health values for MCLs. NHDES acknowledges that this model, like other models, has existing data gaps (*i.e.*, it is a single compartment model). In a different model (Loccisano, *et al.*, 2013), several additional parameters were found to influence model predictions, including the liver:plasma partition coefficient, liver volume, maternal glomerular filtration rate, and the free fraction of PFOA in plasma. These limitations are discussed in further detail in the June 2019 Report. Incorporation of future studies on maternal transfer is expected to prove useful in refining this risk assessment tool, and NHDES will consider them when developing standards for PFAS in the future.

Other commenters have argued that this tool is not new nor "peer-reviewed" despite an informal review process (MDH 2017) conducted by MDH and subsequent peer-reviewed publication of the model (Goeden *et al.*, 2019). NHDES disagrees that this process does not constitute an adequate peer review of the model. After consideration of comments prepared by an external expert in physiological modeling, as well as consultation with MDH and other state risk assessment groups, NHDES concluded that this tool is appropriately vetted for use in developing health-protective drinking water standards.

Several critiques against the transgenerational model were essentially about the relative conservatism of the final drinking water value when considering the conservatism of the model variables and assumptions made in the RfD derivation. Similar to MDH, NHDES applied upper value estimates for the water ingestion rates of the mother and offspring, breastmilk ingestion rates, and duration of breastfeeding, all of which recommended a lower and more protective drinking water value. However, NHDES used central tendency values for the volume of distribution and half-life estimates, and limited the relative source contribution after consideration of the level of conservatism being applied to the exposure scenario. NHDES believes these considerations for the transgenerational model, and others detailed in the June 2019 Report, provide a sufficient level of protection without being hyper-conservative in its risk assessment.

Comment: *NHDES should reconsider whether its assumption that the water intake rate of lactating women is appropriately protective across a lifetime.*

Response: Several comments were submitted regarding the use of the 95th percentile water intake rate for lactating women as a part of the calculation of the MCL. The proposed MCLs no longer use the single fixed water ingestion rate of 0.055 L/kg-day, which is the estimated 95th percentile for a lactating woman (EPA, 2011). Given the use of the MN model, NHDES believes several of these comments have been addressed as the model incorporates different water ingestion rates (*e.g.*, infant, adolescent, and adult) over a lifetime instead of a single point estimate. To be consistent with its prior conservatism and fully protective of the entire population, NHDES applied upper value (95th percentile) breastmilk and drinking water ingestion rates within the transgenerational model.

As NHDES relied on the 2011 Exposure Factors Handbook in its prior recommendation, the new values for the drinking water ingestion rates from the 2019 Chapter 3 Update (EPA, 2019) were applied in place of the 2011 values (updated February 6, 2019). No update has been published for estimated breastmilk ingestion rates, so these were left unchanged in the transgenerational model. Table 3 of Section IV in the June 2019 Report lists these values as they were used in the model.

Because of the unique properties of PFAS and identified health impacts, NHDES applied the transgenerational model instead of the use of the standard 2 L/d assumption historically made by some state agencies. The highly bio-accumulative nature of PFAS requires consideration of age-specific drinking

water values as modeling clearly predicts prolonged elevations in blood concentrations of PFAS following early life exposure. The critical health effects from PFOA (liver damage), PFOS (immune suppression), PFNA (liver damage), and PFHxS (impaired female fertility) are considered to be chronic health effects in humans as a result of prolonged exposure. As NHDES is no longer using a developmental outcome (*e.g.*, for PFOS in the initial proposal), consideration of long-term serum levels as predicted by the MN model was deemed appropriate instead of relying on a single specific life stage.

Comment: *NHDES should select different serum half-life estimates for use in the Minnesota model and derivation of reference doses.*

Response: As a part of its re-evaluation of the proposed MCLs and consideration of scientifically-supported technical comments, NHDES revisited the physiological half-life estimates used for PFOA (now 2.3 years, Bartell *et al.*, 2010), PFOS (remained 3.4 years, Li *et al.*, 2018), PFNA (now 4.3 years, Zhang *et al.*, 2013) and PFHxS (now 4.7 years, Li *et al.*, 2018). The rationale behind these selections and their impact on the RfDs is detailed in Section III of the June 2019 Report.

The dosimetric adjustment factors that estimate external reference doses (RfDs) from internal serum levels use these half-lives to make chemical-specific estimates. The use of longer half-life values results in lower RfD values (see Section III of the June 2019 Report for mathematical operation, and Goeden *et al.*, 2019 for implications in the transgenerational model). This step accounts for the highly bio-accumulative nature of PFAS and has been used by other states (NJDWQI 2017, 2018; MDH 2017, 2019ab) and federal agencies (EPA 2016ab; ATSDR 2018) for estimating external doses of PFAS.

Certain commenters have asserted that this dosimetric adjustment factor approach is overly conservative, overestimating toxicity of PFAS by conflating bioaccumulation with toxicity in humans. NHDES disagrees. This step is necessary to account for the fact that low-level external exposures to these PFAS eventually result in chronic and elevated internal levels. Thus, this step is necessary to account for the unique and extraordinary half-lives of these PFAS reported in humans (Olsen *et al.*, 2007; Bartell *et al.*, 2010; Zhang *et al.*, 2013; Li *et al.*, 2018). If new methods are developed that can be applied to PFOA, PFOS, PFNA, and PFHxS, NHDES will consider these methods and take such action as is appropriate.

Comment: *NHDES should select a protective duration of exclusive breastfeeding for use in the Minnesota model.*

Response: NHDES assumed an exclusive breastfeeding duration of 12 months in its application of the MN model. This is a conservative assumption for the duration of exclusive breastfeeding based on recommendations of the American Academy of Pediatrics (AAP) and the World Health Organization (WHO). The U.S. Department of Health and Human Services, National Institute of Child Health and Human Development, notes that the AAP currently recommends:

> "…infants should be fed breast milk exclusively for the first 6 months after birth. Exclusive breastfeeding means that the infant does not receive any foods (except vitamin D) or fluid unless medically recommended. They further recommend that after the first 6 months and until the infant is 1-year-old, the mother continue breastfeeding while gradually introducing solid foods into the infant's diet." (AAP 2012; NIH 2018)

While experts recommend that infants transition from exclusive breastfeeding to a diet with complimentary foods after 6 months, NHDES determined that the assumption of a 12-month duration of exclusive breastfeeding in the model was conservative but appropriate given two considerations. The first is that NH-specific data from the CDC regarding breastfeeding duration indicates that a considerably higher proportion of NH infants are exclusively breastfed up to 6 months of age (30.2% of infants born in 2015; CDC 2018) when compared to the national average (24.9% of infants born in 2015). Additionally, there is an increasing trend of mothers who are or plan to breastfeed as indicated by the national data (CDC 2018). As infants are recommended to breastfeed up to 2 years of age, there is the possibility for additional exposure through breast milk which tends to contain higher concentrations of PFAS than the mother's drinking water. Secondly, the assumption of exclusive breastfeeding from 6 to 12 months of age is determined to be

appropriately protective given the mechanics of the model. Further discussion of this topic is found in Section IV of the June 2019 Report.

Comment: *NHDES should reconsider its selection of the relative source contribution (RSC) for each PFAS given available data from New Hampshire-specific and nationwide average blood concentrations of these four PFAS.*

Response: To derive the MCLs proposed in the final proposal, NHDES opted to apply a relative source contribution (RSC) of 50% for PFOA, PFOS, PFNA, and PFHxS (detailed explanation available in Section IV of the June 2019 Report). Based on the EPA Decision tree (EPA, 2000), NHDES capped the RSC from water at 50%, leaving up to 50% of the total safe exposure to come from non-drinking water sources. EPA recommends using average background concentrations for deriving RSCs, which in the case of PFAS can be estimated from the data collected by the National Health and Nutrition Examination Survey (NHANES). RSCs calculated using the average NHANES (2013-2014, as reported in Daly *et al.*, 2018) background serum levels for the ages 3 to 19 age group range from about 83 to 99% for the four PFAS, indicating background exposure only uses up 1 to 17% of the 50% allowed (See Table 4 in Section IV of the June 2019 Report). More recent data from NHANES suggest that the general background exposure rates are decreasing (CDC 2019). However, uncertainty about broader environmental contamination led NHDES to conclude that a 50% cap of the RSC was appropriate.

NHDES agrees that the use of New Hampshire-specific blood data potentially overestimates the background versus drinking water contributions of PFAS exposure. As these data were collected from communities with direct contamination of their drinking water supplies, their elevated serum levels likely have a significant portion that is due to drinking water or other potential sources (*e.g.*, dust deposition). Thus, NHDES used the NHANES estimates as calculations based on these populations potentially biases the resulting RSC estimate. However, these other environmental sources of exposure specific to these previously exposed populations underscores the necessity to cap the RSC at 50%.

Using an RSC of 50% for breastfed infants and the MN model, the predicted blood serum level for adult water consumers is approximately equal to or below 20% of the target serum threshold, or a 20% RSC for adults. See Section V of the June 2019 Report for the graphs of the estimated lifespan serum concentrations in relation to the RSC. These estimated serum levels are not predicted to result in a significant increase in serum PFAS levels relative to the national background levels. To achieve no increase above the national background levels would require setting standards at zero, which is inconsistent with standard setting procedures and at this time is not necessary to be adequately protective at all life stages.

Technical Comments Related to Health-Based Risk Assessment of PFOA

Comment: *NHDES did not select an appropriate critical health effect and principle study for deriving the PFOA reference dose, and subsequent MCL.*

Response: NHDES still recommends the use of hepatotoxicity (*i.e.*, liver enlargement and hypertrophy) as the critical health effect basis of the RfD for PFOA. This health effect endpoint is consistent with Health Canada (2016a) and the New Jersey Drinking Water Quality Institute (NJDWQI 2017). This is considered an adverse health outcome following chronic exposure to PFOA, and is relevant across all life stages and therefore appropriate for exposure modeling with the MN model. Additional information supporting this selection is detailed in the June 2019 Report.

NHDES disagrees with comments asserting that the hepatotoxic effects are irrelevant to human health based on the role of peroxisome proliferator-activated receptor α (PPARα) in rodent liver toxicity. As reviewed in the January 2019 Report and by other agencies (NHDES 2019; Health Canada 2016a; NJDWQI 2017; ATSDR 2018), there is evidence that the hepatic effects of PFOA are possibly mediated by PPARα-independent mechanisms and are therefore relevant to human health risk assessment. While humans are not susceptible to the same peroxisome proliferation observed in rodents, PPARα still plays a role in human lipid and energy metabolism, immune function and cell signaling (Issemann and Green, 1990; Lee *et al.*, 1995; Tyagi *et al.*, 2011).

NHDES does not agree that there is sufficient evidence to select the delayed mammary gland development in mice as the principal health effect for the PFOA RfD. Several comments criticized NHDES for not selecting this endpoint and assert that reports of any PFOA-related nuclear receptor activity (*e.g.*, PPARα, ERα or PR) from *in vitro* systems translates into human relevance of an effect from rodent models. NHDES considered the activations of PPARα and other nuclear receptors, and determined that there was insufficient information to rule out enhanced sensitivity in mice compared to humans as it relates to this specific outcome. As discussed in the January 2019 Report, this is due to interactions with nuclear receptor co-activators in mice (reviewed by Corton *et al.*, 2014) which have been shown to modulate PPARα-mediated effects on the development and function of mammary glands in mice (Qi *et al.*, 2004; Jia *et al.*, 2005). The functional significance remains unclear, as White *et al.* (2007) could not discern if effects on pups were due to changes in lactation or maternal toxicity other than the observed delays in mammary gland development. Direct investigation in a subsequent study failed to detect significant differences in treated mice (White *et al.*, 2011). Furthermore, no other state regulatory agency, to date, has adopted its use given uncertainty about its significance and the ATSDR which develops very conservative MRLs did not use this endpoint (ATSDR, 2018).

Epidemiological evidence associating this perinatal effect in mice to a human health outcome is limited to four studies. Three studies have suggested negative associations between certain PFAS (*i.e.*, PFOA and PFOS) to the duration of breastfeeding (Fei *et al.*, 2010; Romano *et al.*, 2016; Timmermann *et al.*, 2017), although two of these studies did not have information on prior breastfeeding durations which presents an important confounding factor (Fei *et al.*, 2010; Timmermann *et al.*, 2017). The most recent study accounting for prior breastfeeding, which several comments failed to reference, reported a positive association between PFAS and breastfeeding (Rosen *et al.*, 2018), although this outcome likely suggests an important role of PFAS toxicokinetics throughout pregnancy and breastfeeding. NHDES found that the epidemiological evidence for hepatotoxicity and altered lipid metabolism were more robust and deemed appropriate for use as the basis of an RfD at this time.

Conversely, other commenters criticized the selected critical health effect as being overly conservative given assessments made by another country (*i.e.*, Health Canada) and controlled studies of PFOA in humans. Health Canada (2016a) also selected hepatotoxicity as a critical effect for the basis of its RfD and concluded that increased liver weight at lower doses was relevant to human health. NHDES agreed with this judgement in critical effect selection. Health Canada opted for the no observed adverse effect level (NOAEL) for liver hypertrophy from Perkins *et al.* (2004) instead of Loveless *et al.* (2006). Health Canada (2016a) used a composite uncertainty factor of 25, whereas NHDES used 100 for PFOA. Health Canada uses values of 2.5 as partial and 10 for full uncertainty factors, whereas EPA methodology used 3 or 10, respectively. NHDES only differed from Health Canada in the more conservative application of a partial uncertainty factor for database uncertainty, which was not applied by Health Canada. Before the applications of uncertainty factors, the RfD proposed by NHDES is 610 ng/kg-d and the Health Canada value is 625 ng/kg-d. After uncertainty factors, the differences between the final drinking water values proposed by NHDES and Health Canada are therefore due to consideration of the relative source contribution (20% applied by Health Canada) and drinking water ingestion rate (*e.g.*, 1.5 L/d).

To the latter concern about over-conservatism from not deriving a RfD based on a recently-published clinical trial of PFOA (Convertino *et al.*, 2018), NHDES determined this study was not appropriate based on the population used. This study evaluated the direct effects of PFOA in late-stage cancer patients (n=49) and found negative associations with circulating cholesterol and free T_4 (Convertino *et al.*, 2018). Some commenters indicated that NHDES should re-evaluate this study and consider the effects observed in study participants who received a 6-week oral treatment of ammonium perfluorooctanoate. NHDES has serious reservations about relying on the results of such a study with a small sample size, restrictive inclusion criteria for participants, and the use of late-stage cancer patients whose metabolic function is not likely comparable to the general population. The age, health status, and limited information on population diversity of study participants raises several questions about confounding factors that were not addressed in the study's discussion.

Comment: NHDES did not select the appropriate uncertainty factors in its derivation of a reference dose for PFOA.

Response: NHDES applied uncertainty factors to each of the proposed RfDs after consideration of EPA methodology (EPA 2002) and RfD calculations made by other states agencies (NJDWQI 2017, 2018ab; MDH 2017, 2019ab; TCEQ 2016), the EPA (EPA 2016ab) and the ATSDR (2018). Section III of the June 2019 Report details each uncertainty factor applied for PFOA.

Evidence from gene knock-out (PPARα absent) studies indicates that other mechanisms of action are operating to cause liver toxicity besides those that are PPARα dependent. As the exact interaction of these mechanisms of toxicity with PPARα activation are still being studied, NHDES affirms that it is sound risk assessment policy to retain the partial uncertainty factor for animal-to-human toxicodynamic difference.

NHDES maintains the inclusion of the database uncertainty factor of 3 for immune and developmental effects is justified without being overly conservative. Per the National Toxicology Program (NTP)(2016), there is sufficient evidence for concern about PFOA's immunological effects as "PFOA is presumed to be an immune hazard to humans based on a high level of evidence that PFOA suppressed the antibody response from animal studies and a moderate level of evidence from studies in humans." This database uncertainty factor also accounts for other developmental effects (*e.g.*, delayed mammary gland development) that occur at lower doses in rodents but similar sensitivity in humans is currently suspect.

Technical Comments Related to Health-Based Risk Assessment of PFOS

Comment: NHDES did not select an appropriate critical health effect and principle study for deriving the PFOS reference dose, and subsequent MCL.

Response: NHDES agrees that in order to be more health protective the reference dose (RfD) calculation for PFOS should be based on immunosuppression. After review of available information, NHDES used the PFOS RfD recently proposed by MDH (2019a) and subsequent exposure assumptions, for immunosuppression as reported in Dong *et al.*, (2011).

As discussed in Section III of the June 2019 Report, NHDES selected the RfD developed by MDH (2019a) over the RfD for immunosuppression proposed by NJDWQI (2018a). MDH based the RfD for PFOS on reduced primary (IgM) antibody production in male mice following a 60-day oral exposure to PFOS (Dong *et al.*, 2011). Measurement of IgM is standard for immunotoxicity assays evaluating the T cell-dependent antibody response and, as a standard for regulatory toxicology (Ladics 2018, reviewed by DeWitt *et al.*, 2019), was deemed appropriate by NHDES. Results from this study were not amenable to benchmark dose modeling, so the NOAEL of 2,360 ng/mL (internal dose; Dong *et al.*, 2011) was used for RfD calculation. This RfD is on a similar order to others that have derived RfDs/MRLs for PFOS using immunosuppression as the base study or justification of additional uncertainty factors:

- ATSDR 2018 – 2.0 ng/kg-d (provisional, drinking water value varies)
- NJDWQI 2018a – 2.0 ng/kg-d (proposed MCL, 13 ng/L)
- MDH 2019a – 3.0 ng/kg-d (proposed health-based guidance value, 15 ng/L; recommended by NHDES)

As discussed by DeWitt *et al.* (2019), clinical classification of biomarkers of immune function plays a critical role in interpreting the existing epidemiological evidence. NHDES acknowledges some limitations of the human epidemiological data, as described by Chang *et al.* (2016), but determined that the growing body of evidence and consensus regarding the immunotoxicity of PFAS, including PFOS, merits use of immunosuppression in risk assessment. The National Toxicology Program (2016) concluded that PFOS is a presumed immunotoxin in humans, and emerging studies suggest that this is a relevant and sensitive endpoint for the protection of human health. More recently, ATSDR (2018) opted to apply additional uncertainty factors to arrive at an MRL that would be similar to an MRL or RfD based on immunosuppression.

Health Canada selected hepatotoxicity, similar to PFOA, as the critical health effect for PFOS (Health Canada, 2016). The proposed RfD based on liver toxicity (or hepatic hypertrophy) in rodents (Butenhoff *et*

al., 2012) was 60 ng/kg-d, after the application of a composite uncertainty factor of 25 (see previous PFOA RfD comment above). This was applied with a 20% relative source contribution and drinking water intake of 1.5 L/d to arrive at a drinking water value of 600 ng/L. Health Canada discussed the immunological studies on PFOS, but concluded that due to the nearly two-order of magnitude difference in lowest observed adverse effect levels (LOAELs) between various rodent studies this endpoint was not suitable for RfD development. NHDES concurs that the variation in LOAELs is a source of uncertainty, but given the significance of impaired immune function it is appropriate to use this endpoint to protect public health until more definitive scientific evidence quantifies the sensitivity of this outcome in humans.

Comment: *NHDES did not select the appropriate uncertainty factors in its derivation of a reference dose for PFOS.*

Response: NHDES applied uncertainty factors to each of the proposed RfDs after consideration of EPA methodology (EPA 2002) and RfD calculations made by other states agencies (NJDWQI 2017, 2018ab; MDH 2017, 2019ab; TCEQ 2016), the EPA (EPA 2016ab) and the ATSDR (2018). Section III of the June 2019 Report details each uncertainty factor applied for PFOS.

As the exact interaction of these mechanisms of immunotoxicity in rodents and humans is currently not understood, NHDES affirms that it is sound risk assessment policy to retain the partial uncertainty factor for animal-to-human toxicodynamic difference.

With respect to the database uncertainty factor, an additional partial database uncertainty factor of 3 was applied due to reports of thyroid disruption at early life stages (decreased T_4; as recommended by MDH 2019a). NHDES agrees with the approach taken by MDH, given the suggestive evidence for the human relevance of altered T_4 levels (reviewed by Ballesteros *et al.*, 2017 and ATSDR, 2018).

Technical Comments Related to Health-Based Risk Assessment of PFNA

Comment: *NHDES did not select an appropriate critical health effect and principle study for deriving the PFNA reference dose, and subsequent MCL.*

Response: As for the initial proposal, NHDES chose liver toxicity as the critical health effect basis of the RfD for PFNA. This used the benchmark dose model of Das *et al.* (2015) conducted by the NJDWQI (2018b). The LOAEL of this study was 12,400 ng/mL of serum PFNA (oral dose of 1 mg/kg-d), which was modeled down to 4,900 ng/mL as a basis for the RfD calculation. This study is the basis of the only other promulgated MCL, and NHDES determined there was sufficient evidence to support its application.

NHDES reviewed the recommended study on PFNA (Singh and Singh 2019). Singh and Singh (2019) evaluated the effects of PFNA on male Parkes mice following a 90-day exposure to either 0.2 or 0.5 mg/kg-d. For several of the evaluated outcomes, including reduced litter size, infertility, and histological changes in the testes of exposed mice, the no observed adverse effect level was 0.2 mg/kg-d.

Singh and Singh (2019) did not report internal serum doses for PFNA at any stage of the 90-day exposure, which makes direct comparisons to the internal doses reported by Das *et al.* (2015) unfeasible as there is limited toxicokinetic information on PFNA in this strain. Furthermore, this limits consideration of benchmark dose modeling for this endpoint given the importance of internal versus external doses. A single-dose (1 or 10 mg/kg) study using CD-1 mice suggests that the serum half-life of PFNA ranges from 34-69 days in males and 26-68 days in females (Tatum-Gibbs *et al.* 2011). This half-life is longer than the exposure and it is unclear what the internal steady-state levels would be in mice throughout the 90-day exposure.

One other study provides some estimate of internal serum levels at the NOAEL reported by Singh and Singh (2019). Using male Balb/c mice, Wang *et al.* (2015) measured serum levels of PFNA to be approximately 11,500 ng/mL at the LOAEL for hepatic hypertrophy following a 14-day exposure. The oral dose (0.2 mg/kg-d) for this LOAEL in Wang *et al.* (2015) was identical to the NOAEL for reduced litter size, infertility, and histological changes in the testes identified at the end of a 90-day exposure (Singh and Singh 2019). Given these dosing similarities between the two mouse studies (Wang *et al.*, 2015; Singh and Singh 2019) and the predicted serum levels in the proposed MCL, NHDES believes the present reference

dose combined with the exposure assumptions provide a protective margin of exposure for the aforementioned health effects.

Comment: *NHDES did not select the appropriate uncertainty factors in its derivation of a reference dose for PFNA.*

Response: NHDES applied uncertainty factors to each of the proposed RfDs after consideration of EPA methodology (EPA 2002) and RfD calculations made by other states agencies (NJDWQI 2017, 2018ab; MDH 2017, 2019ab; TCEQ 2016), the EPA (EPA 2016ab) and the ATSDR (2018). Section III of the June 2019 Report details each uncertainty factor applied for PFNA.

Similar to PFOA, evidence from gene knock-out (PPARα absent) studies has indicated that other mechanisms of action are operating to cause liver toxicity besides those that are PPARα dependent. As the exact interaction of these mechanisms of toxicity with PPARα activation are still being studied, NHDES affirms that it is sound risk assessment policy to retain the partial uncertainty factor for animal-to-human toxicodynamic difference.

As summarized in Section III of the June 2019 Report, NHDES did not agree with the additional application of uncertainty factors for duration of exposure. NHDES used the more conservative half-life estimate of PFNA derived from men and older women (4.3 years; Zhang *et al.*, 2013). Given the application of this more conservative half-life estimate, NHDES removed the associated partial database database uncertainty factor for PFNA. NHDES retained the partial database uncertainty factor of 3 to account for a lack of multigenerational rodent studies using PFNA, as well as concern for potential immunotoxic impacts seen with other PFAS, such as PFOA (NTP 2016; DeWitt *et al.*, 2012, 2019).

Technical Comments Related to Health-Based Risk Assessment of PFHxS

Comment: *NHDES did not select an appropriate critical health effect and principle study for deriving the PFHxS reference dose, and subsequent MCL.*

Response: NHDES disagrees with the comment that a different critical health effect should have been selected for PFHxS. Compared to PFOA, PFOS, and PFNA, there are significantly fewer studies available for understanding the health effects of PFHxS and its toxicity in rodent models. This is especially concerning given the dramatically longer half-life estimates for PFHxS despite the fact that it possesses a shorter carbon chain in comparison to PFNA, PFOA, and PFOS. Thus, there is significant concern for the health impacts of chronic exposure but an absence of long-term exposure studies in rodents. While liver toxicity and altered cholesterol metabolism are consistent with effects reported in association with other PFAS, the limited dataset for this compound merits consideration of any changes in an apical outcome such as reduced litter size. ATSDR did not review this study as a part of their 2018 draft toxicological profile for perfluoroalkyls (ATSDR 2018), but NHDES found that the statistically significant reduction in litter size, alteration in genital development in pups, and other observed toxicities merited consideration as mice may be better models than rats for evaluating PFHxS.

NHDES selected a reduced litter size as the critical health effect, based on results from mice orally-exposed to PFHxS for a sub-chronic duration prior to gestation (Chang *et al.*, 2018). Section III of the June 2019 Report provides additional information on this decision. A detailed review of background studies and RfD calculations based on this endpoint is currently under external peer-review for publication (Ali *et al.*, under review).

NHDES agreed that the volume of distribution should reflect the critical health effect in this case, and applied the female volume of distribution (213 mL/kg-d; Sundström *et al.*, 2012) for reference dose calculation. Details on its application are described in Section III of the June 2019 Report.

Comment: *NHDES should evaluate the use of benchmark dose modeling instead of the no-observed-adverse-effect-level (NOAEL) for the critical health effect of reduced litter size in mice.*

Response: In collaboration with faculty at the University of Florida, NHDES developed a RfD for PFHxS based on benchmark dose modeling of data reported in Chang *et al.* (2018). The supporting decisions and

methodology are currently under peer-review for publication, and the detailed methodology and numeric outputs will be made available after a decision is made regarding this publication.

Comment: *NHDES did not select the appropriate uncertainty factors in its derivation of a reference dose for PFHxS.*

Response: NHDES applied uncertainty factors to each of the proposed RfDs after consideration of EPA methodology (EPA 2002) and RfD calculations made by other states agencies (NJDWQI 2017, 2018ab; MDH 2017, 2019ab; TCEQ 2016), the EPA (EPA 2016ab) and the ATSDR (2018). Section III of the June 2019 Report details each uncertainty factor applied for PFHxS.

After review of this comment and applications of the database uncertainty factor, NHDES agreed that a partial database uncertainty factor of 3 was more appropriate. However, NHDES also identified studies suggesting that longer exposure durations would have been more appropriate for evaluating PFHxS given reproductive effects seen with PFOS (Feng *et al.*, 2015) and the considerably long half-life of PFHxS in humans (Olsen *et al.*, 2007; Li *et al.*, 2018). The rationale behind these decisions is detailed in Section III of the June 2019 Report.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2018. Toxicological Profile for Perfluoroalkyls – Draft for Public Comment, June 2018. Accessed online at: https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf.

Agency for Toxic Substances and Disease Registry (ATSDR). 2018a. Minimal Risk Levels (MRLs) – For Professionals. Retrieved from https://www.atsdr.cdc.gov/mrls/index.asp.

Agency for Toxic Substances and Disease Registry (ATSDR). 2019. Public Comment Version Per and Polyfluoroalkyl Substances (PFAS) in the Pease Tradeport Public Water System. EPA PWS ID: 1951020.

Ali JM, Roberts SM, Gordon DS, Stuchal LD. (under review) Derivation of a chronic reference dose for perfluorohexane sulfonate (PFHxS) for reproductive toxicity in mice.

American Academy of Pediatrics. (2012). Breastfeeding and the use of human milk. *Pediatrics, 129(3)*, e827–e841. Retrieved from http://pediatrics.aappublications.org/content/129/3/e827.full.pdf+html

Ballesteros V, Costa O, Iñiguez C, Fletcher T, Ballester F, Lopez-Espinosa MJ. 2017. Exposure to perfluoroalkyl substances and thyroid function in pregnant women and children: A systematic review of epidemiologic studies. Environ Int, 99:15-28. doi: 10.1016/j.envint.2016.10.015.

Bartell SM, Calafat AM, Lyu C, *et al.* 2010. Rate of decline in serum PFOA concentrations after granular activated carbon filtration at two public water systems in Ohio and West Virginia. Environ Health Perspect 118(2):222-228.

Butenhoff JL, Chang SC, Olsen GW, *et al.* 2012. Chronic dietary toxicity and carcinogenicity study with potassium perfluorooctanesulfonate in Sprague Dawley rats. Toxicology 293(1-3):1-15.

Centers for Disease Control and Prevention (CDC). (2018). Breastfeeding Report Card. Retrieved from https://www.cdc.gov/breastfeeding/data/reportcard.htm.

Centers for Disease Control and Prevention (CDC). (2019). Fourth National Report on Human Exposure to Environmental Chemicals, Updated Tables, January 2019, Volume One. Retrieved from https://www.cdc.gov/exposurereport/pdf/FourthReport_UpdatedTables_Volume1_Jan2019-508.pdf.

Chang ET, Adami HO, Boffetta P, Wedner HJ, Mandel JS. 2016. A critical review of perfluorooctanoate and perfluorooctanesulfonate exposure and immunological health conditions in humans. Crit Rev Toxicol., 46(4): 279-331.

Chang S, Butenhoff JL, Parker GA, Coder PS, Zitzow JD, Krisko RM, Bjork JA, Wallace KB, Seed JG. 2018. Reproductive and developmental toxicity of potassium perfluorohexanesulfonate in CD-1 mice. Reproductive Toxicology 78: 150-168.

Convertino M, *et al.* 2018. Stochastic Pharmacokinetic-Pharmacodynamic Modeling for Assessing the Systemic Health Risk of Perfluorooctanoate (PFOA). Toxicological Sciences 163(1): 293-306.

Corton JC, Cunningham ML, Hummer BT, *et al.* 2014. Mode of action framework analysis for receptor-mediated toxicity: The peroxisome proliferator-activated receptor alpha (PPARα) as a case study. Crit Rev Toxicol 4444(1):1-49. 10.3109/10408444.2013.835784.

Daly ER, Chan BP, Talbot EA, Nassif J, Bean C, Cavallo SJ, Metcalf E, Simone K, Woolf AD. 2018. Per- and polyfluoroalkyl substance (PFAS) exposure assessment in a community exposed to contaminated drinking water, New Hampshire, 2015. Int J Hyg Environ Health. 221(3):569-577. doi: 10.1016/j.ijheh.2018.02.007.

Das KP, Grey BE, Rosen MB, *et al.* 2015. Developmental toxicity of perfluorononanoic acid in mice. Reprod Toxicol 51:133-144. 10.1016/j.reprotox.2014.12.012.

DeWitt JC, Blossom SJ, Schaider LA. 2019. Exposure to per-fluoroalkyl and polyfluoroalkyl substances leads to immunotoxicity: epidemiological and toxicological evidence. J Expo Sci Environ Epidemiol. 29(2):148-156. doi: 10.1038/s41370-018-0097-y.

DeWitt JC, Peden-Adams MM, Keller JM, Germolec DR. 2012. Immunotoxicity of Perfluorinated Compounds: Recent Developments. Toxicologic Pathology, 40: 300-311.

Dhingra R, Winquist A, Darrow LA, Klein M, Steenland K. 2017. A study of reverse causation: examining the associations of perfluorooctanoic acid serum levels with two outcomes. Environ Health Perspect 125:416-421; https://ehp.niehs.nih.gov/doi/10.1289/EHP273.

Dong GH, Liu MM, Wang D, *et al.* 2011. Sub-chronic effect of perfluorooctanesulfonate (PFOS) on the balance of type 1 and type 2 cytokine in adult C57BL6 mice. Arch Toxicol 85(10):1235-1244.

Dong GH, Zhang YH, Zheng L, *et al.* 2009. Chronic effects of perfluorooctanesulfonate exposure on immunotoxicity in adult male C57BL/6 mice. Arch Toxicol 83(9):805-815.

Ernst A, Brix N, Lauridsen LLB, Olsen J, Parner ET, Liew Z, Olsen LH, Ramlau-Hansen CH. 2019. Exposure to Perfluoroalkyl Substances during Fetal Life and Pubertal Development in Boys and Girls from the Danish National Birth Cohort. Environ Health Perspect. 127(1):17004. doi: 10.1289/EHP3567.

Fei C, McLaughlin JK, Lipworth L, Olsen J. 2010. Maternal concentrations of perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA) and duration of breastfeeding. Scand J Work Environ Health. 36(5):413–21.

Feng X, Wang X, Cao X, Xia Y, Zhou R, Chen L. 2015. Chronic Exposure of Female Mice to an Environmental Level of Perfluorooctane Sulfonate Suppresses Estrogen Synthesis Through Reduced Histone H3K14 Acetylation of the StAR Promoter Leading to Deficits in Follicular Development and Ovulation. Toxicol Sci. 148(2):368-79. doi: 10.1093/toxsci/kfv197.

Frisbee SJ, Brooks AP Jr, Maher A, Flensborg P, Arnold S, Fletcher T, Steenland K, Shankar A, Knox SS, Pollard C, Halverson JA, Vieira VM, Jin C, Leyden KM, Ducatman AM. 2009. The C8 health project: design, methods, and participants. Environ Health Perspect. 117(12):1873-82. doi: 10.1289/ehp.0800379.

Goeden HM, Greene CW, Jacobus JA. 2019. A transgenerational toxicokinetic model and its use in derivation of Minnesota PFOA water guidance. J Expo Sci Environ Epidemiol. 29(2):183-195. doi: 10.1038/s41370-018-0110-5.

Health Canada. 2016a. Perfluorooctanoic acid (PFOA) in drinking water. Retrieved from https://www.canada.ca/content/dam/hc-sc/healthy-canadians/migration/health-system-systeme-sante/consultations/acide-perfluorooctanoic-acid/alt/perfluorooctanoic-eng.pdf.

Health Canada. 2016. Perflurooctane sulfonate (PFOS) in drinking water. Retrieved from https://www.canada.ca/content/dam/hc-sc/healthy-canadians/migration/health-system-systeme-sante/consultations/perfluorooctane-sulfonate/alt/perfluorooctane-sulfonate-eng.pdf.

Issemann I, Green S. 1990. Activation of a member of a steroid hormone receptor superfamily by peroxisome proliferators. Nature, 347:645-650.

Jia, Y., Qi, C., Zhang, Z., Zhu, Y. T., Rao, S. M., and Zhu, Y. J. (2005). Peroxisome proliferator-activated receptor-binding protein null mutation results in defective mammary gland development. J. Biol. Chem. 280, 10766–10773.

Ladics G.S. 2018. The Sheep Erythrocyte T-Dependent Antibody Response (TDAR). In: DeWitt J., Rockwell C., Bowman C. (eds) Immunotoxicity Testing. Methods in Molecular Biology, vol 1803. Humana Press, New York, NY.

Lee SS-T, Pineau T, Drago J, Lee EJ, Owens JW, Kroetz DL, Fernandez-Salguero PM, Westphal H, and Gonzalez FJ (1995) Targeted disruption of the alpha isoform of the peroxisome proliferator-activated receptor gene in mice results in abolishment of the pleiotropic effects of peroxisome proliferators. Mol Cell Biol 15:3012–3022.

Li Y, Fletcher T, Mucs D, *et al.* 2018. Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water. Occup Environ Med 75(1):46-51. 10.1136/oemed-2017-104651.

Loccisano AE, *et al.* 2013. Development of PBPK Models for PFOA and PFOS for Human Pregnancy and Lactation Life Stages. J Toxicol Environ Health A. 76(1): 25-57.

Loveless SE, Finlay C, Everds NE, *et al.* 2006. Comparative responses of rats and mice exposed to linear/branched, linear, or branched ammonium perfluorooctanoate (APFO). Toxicology 220:203-217.

Michigan Department of Health and Human Services (MIDHHS). 2019. Public health drinking water screening levels for PFAS. Retrieved from https://www.michigan.gov/documents/pfasresponse/MDHHS_Public_Health_Drinking_Water_Screening_Levels_for_PFAS_651683_7.pdf.

Michigan PFAS Science Advisory Panel Report. 2018. Scientific Evidence and Recommendations for Managing PFAS Contamination in Michigan. December 7, 2018. Retrieved from https://www.michigan.gov/documents/pfasresponse/Science_Advisory_Board_Report_641294_7.pdf.

Minnesota Department of Health (MDH). 2017a. Background Document. Toxicokinetic Model for Perfluorooctane Sulfonate (PFOS) and Perfluorooctanoic Acid (PFOA) and Its Use in the Derivation of Human Health-Based Water Guidance Values.

Minnesota Department of Health (MDH). 2017 - Toxicological Summary for: Perfluorooctanoate. Retrieved from http://www.health.state.mn.us/divs/eh/risk/guidance/gw/pfoa.pdf.

Minnesota Department of Health (MDH). 2019b - Toxicological Summary for: Perfluorohexane sulfonate. Retrieved from https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfhxs.pdf.

Minnesota Department of Health (MDH). 2019a - Toxicological Summary for: Perfluorooctane sulfonate. Retrieved from https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfos.pdf.

National Institutes of Health (NIH). (2018). What are the recommendations for breastfeeding? Retrieved from https://www.nichd.nih.gov/health/topics/breastfeeding/conditioninfo/recommendations.

National Toxicology Program (NTP). 2016. NTP Monograph: Immunotoxicity Associated with Exposure to Perfluorooctanoic Acid or Perfluorooctane Sulfonate. September 2016. Retrieved from https://ntp.niehs.nih.gov/ntp/ohat/pfoa_pfos/pfoa_pfosmonograph_508.pdf.

New Jersey Drinking Water Quality Institute (NJDWQI). 2017. Health-Based Maximum Contaminant Level Support Document: Perfluorooctanoic Acid (PFOA). February 15, 2017. Retrieved from https://www.state.nj.us/dep/watersupply/pdf/pfoa-appendixa.pdf.

New Jersey Drinking Water Quality Institute (NJDWQI). 2018a. Health-Based Maximum Contaminant Level Support Document: Perfluorooctane Sulfonate (PFOS). June 5, 2018. Retrieved from https://www.state.nj.us/dep/watersupply/pdf/pfos-recommendation-appendix-a.pdf.

New Jersey Drinking Water Quality Institute (NJDWQI). 2018b. Health-Based Maximum Contaminant Level Support Document: Perfluorononanoic Acid (PFNA). Retrieved from https://www.state.nj.us/dep/watersupply/pdf/pfna-health-effects.pdf.

NHDES. 2019. January 2019 Report. Retrieved from https://www.des.nh.gov/organization/commissioner/pip/publications/documents/r-wd-19-01.pdf.

NJ DEP SRP. 2019. New Jersey Department of Environmental Protection Site Remediation Program – Contaminants of Emerging Concern. Retrieved from https://www.nj.gov/dep/srp/emerging-contaminants/.

Olsen GW, Burris JM, Ehresman DJ, Froehlich JW, Seacat AM, Butenhoff JL, Zobel LR. 2007. Half-life of serum elimination of perfluorooctanesulfonate, perfluorohexanesulfonate, and perfluorooctanoate in retired fluorochemical production workers. Environ Health Perspect 115:1298-1305.

Olsen J, Melbye M, Olsen SF, Sorensen TI, Aaby P, Andersen AM, *et al.* 2001. The Danish National Birth Cohort–its background, structure and aim. Scand J Public Health 29(4):300–307, PMID: 11775787, 10.1177/14034948010290040201.

Perkins RG, Butenhoff JL, Kennedy GL, *et al.* 2004. 13-Week dietary toxicity study of ammonium perfluorooctanoate (APFO) in male rats. Drug Chem Toxicol 27(4):361-378.

Qi, C., Kashireddy, P., Zhu, Y. T., Rao, S. M., and Zhu, Y. J. (2004). Null mutation of peroxisome proliferator-activated receptor-interacting protein in mammary glands causes defective mammopoiesis. J. Biol. Chem. 279, 33696–33701.

Romano ME, Xu Y, Calafat AM, Yolton K, Chen A, Webster GM, Eliot MN, Howard CR, Lanphear BP, Braun JM. 2016. Maternal serum perfluoroalkyl substances during pregnancy and duration of breastfeeding. Environ Res. 149:239–46.

Rosen EM, Brantsæter AL, Carroll R, Haug L, Singer AB, Zhao S, Ferguson KK. 2018. Maternal Plasma Concentrations of Per- and polyfluoroalkyl Substances and Breastfeeding Duration in the Norwegian Mother and Child Cohort. Environ Epidemiol. 2(3). pii: e027. doi: 10.1097/EE9.0000000000000027.

Singh S, Singh SK. 2019. Chronic exposure to perfluorononanoic acid impairs spermatogenesis, steroidogenesis and fertility in male mice. J Appl Toxicol. 39(3):420-431. doi: 10.1002/jat.3733.

Sundström M, Chang SC, Noker PE, Gorman GS, Hart JA, Ehresman DJ, Bergman Å, Butenhoff JL. 2012. Comparative pharmacokinetics of perfluorohexanesulfonate (PFHxS) in rats, mice, and monkeys. Reproductive Toxicology 33(4):441-451.

Tatum-Gibbs K, Wambaugh JF, Das KP, *et al.* 2011. Comparative pharmacokinetics of perfluorononanoic acid in rat and mouse. Toxicology 281(1-3):48-55. 10.1016/j.tox.2011.01.003.

Texas Commission on Environmental Quality (TCEQ) technical Document on Perfluoro Compounds (PFCs). 2016. Retrieved from: https://www.tceq.texas.gov/assets/public/implementation/tox/evaluations/pfcs.pdf.

Timmermann CAG, Budtz-Jorgensen E, Petersen MS, Weihe P, Steuerwald U, Nielsen F, Jensen TK, Grandjean P. 2017. Shorter duration of breastfeeding at elevated exposures to perfluoroalkyl substances. Reproductive Toxicology. 68:164–70. Epub 2016/07/17. DOI: 10.1016/j.reprotox.2016.07.010.

Tyagi S, Gupta P, Saini AS, Kaushal C, and Sharma S. 2011. The peroxisome proliferator-activated receptor: A family of nuclear receptors role in various diseases. J Adv Pharm Technol Res. 2(4): 236–240.

U.S. Environmental Protection Agency (EPA). 2000. Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000) Documents. Accessed April 2019. Retrieved from https://www.epa.gov/wqc/methodology-deriving-ambient-water-quality-criteria-protection-human-health-2000-documents.

U.S. Environmental Protection Agency (EPA). 2002. A Review of the Reference Dose and Reference Concentration Processes. EPA/630/P-02/0002F. Risk Assessment Forum, Washington, DC. Retrieved from https://www.epa.gov/osa/review-reference-dose-and-reference-concentrationprocesses.

U.S. Environmental Protection Agency (EPA). 2006. SAB Review of EPA's Draft Risk Assessment of the Potential Human Health Effects Associated with PFOA and Its Salts. SAB06006.

U.S. Environmental Protection Agency (EPA). 2011. Exposure Factors Handbook: 2011 Edition. EPA/600/R-090/052F. Office of Research and Development, National Center for Environmental Assessment, Washington, D.C. 1436 pp. Accessed online at: https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=236252.

U.S. Environmental Protection Agency (EPA). 2016a. Health Effects Support Document for Perfluorooctanoic acid (PFOA). Document # EPA 822-R-16-003. May 2016. Retrieved from https://www.epa.gov/sites/production/files/2016-05/documents/pfoa_hesd_final_508.pdf.

U.S. Environmental Protection Agency (EPA). 2016b. Health Effects Support Document for Perfluorooctane Sulfonate (PFOS). Document # EPA 822-R-16-002. May 2016. Retrieved from https://www.epa.gov/sites/production/files/2016-05/documents/pfos_hesd_final_508.pdf.

U.S. EPA. 2018. National Primary Drinking Water Regulations. Retrieved from https://www.epa.gov/ground-water-and-drinking-water/national-primary-drinking-water-regulations#one.

U.S. Environmental Protection Agency (EPA). 2019. Exposure Factors Handbook: Chapter 3 Update. Office of Research and Development, National Center for Environmental Assessment, Washington, D.C. 1436 pp. Accessed online at: https://www.epa.gov/sites/production/files/2019-02/documents/efh_-_chapter_3_update.pdf.

Wang, J, Yan, S, Zhang, W, Zhang, H, Dai, J. 2015. Integrated proteomic and miRNA transcriptional analysis reveals the hepatotoxicity mechanism of PFNA exposure in mice. J Proteome Res. 14:330-41.

White SS, Calafat AM, Kuklenyik Z, *et al.* 2007. Gestational PFOA exposure of mice is associated with altered mammary gland development in dams and female offspring. Toxicol Sci 96(1):133-144.

White SS, Stanko JP, Kato K, *et al.* 2011. Gestational and chronic low-dose PFOA exposures and mammary gland growth and differentiation in three generations of CD-1 mice. Environ Health Perspect 119(8):1070-1076.

Winquist A, Lally C, Shin HM, Steenland K. 2013. Design, methods, and population for a study of PFOA health effects among highly exposed mid-Ohio valley community residents and workers. Environ Health Perspect. 121(8):893-9. doi: 10.1289/ehp.1206450.

Zhang Y, Beesoon S, Zhu L, *et al.* 2013. Biomonitoring of perfluoroalkyl acids in human urine and estimates of biological half-life. Environ Sci Technol 47(18):10619-10627. 10.1021/es401905e.

Attachment 1: "New Hampshire Department of Environmental Services Technical Background for the June 2019 Proposed Maximum Contaminant Levels (MCLs) for Perfluorooctane sulfonic Acid (PFOS), Perfluorooctanoic Acid (PFOA), Perfluorononanoic Acid (PFNA), and Perfluorohexane sulfonic Acid (PFHxS)" and findings of a peer review of NHDES's derivations conducted by Stephen M. Roberts, Ph.D.

Attachment 2: NHDES updated cost and benefit considerations

Attachment 3: NHDOJ letter

ATTACHMENT 1

New Hampshire Department of Environmental Services

Technical Background Report for the June 2019 Proposed Maximum Contaminant Levels (MCLs) and Ambient Groundwater Quality Standards (AGQSs) for Perfluorooctane sulfonic Acid (PFOS), Perfluorooctanoic Acid (PFOA), Perfluorononanoic Acid (PFNA), and Perfluorohexane sulfonic Acid (PFHxS)

And

Letter from Dr. Stephen M. Roberts, Ph.D. dated 6/25/2019 – Findings of Peer Review Conducted on Technical Background Report

June 28, 2019

New Hampshire Department of Environmental Services

Technical Background Report for the June 2019 Proposed Maximum Contaminant Levels (MCLs) and Ambient Groundwater Quality Standards (AGQSs) for Perfluorooctane sulfonic Acid (PFOS), Perfluorooctanoic Acid (PFOA), Perfluorononanoic Acid (PFNA), and Perfluorohexane sulfonic Acid (PFHxS)

June 28, 2019

Table of Contents

Abbreviations

AFFF - aqueous film forming foam

AGQS - Ambient Groundwater Quality Standard

APFO – ammonium perfluorooctanoate

ATSDR – Agency for Toxic Substances and Disease Registry

BMD – benchmark dose

BMDL – benchmark dose lower-bound confidence limit

C8 – an alternative name for perfluorooctanoic acid

CAR – constitutive androstane receptor

CAS# - Chemical Abstracts Service Registry Number

CDC – Centers for Disease Control and Prevention

CSF – cancer slope factor

d - day

DAF – dosimetric adjustment factor

IR – ingestion rate

IRIS - Integrated Risk Information System

kg - kilogram

L - liter

LHA – lifetime health advisory

Ln – natural logarithm

LOAEL – lowest observed adverse effect level

MCL – maximum contaminant level

mg - milligram

MDH – Minnesota Department of Health

MRL – minimal risk level

ng - nanogram

NHDES – New Hampshire Department of Environmental Services

NH DHHS – New Hampshire Department of Health & Human Services

NIS - National Immunization Survey

NJDWQI – New Jersey Drinking Water Quality Institute

NOAEL – no observed adverse effect level

NTP – National Toxicology Program

PFAS – perfluoroalkyl substances

PFHxS – perfluorohexane sulfonic acid

PFNA – perfluorononanoic acid

PFOA – perfluorooctanoic acid

PFOS – perfluorooctane sulfonic acid

POD – point of departure

PPAR - peroxisome proliferator-activated receptor

ppb –parts-per-billion

ppt – parts-per-trillion

RME – reasonable maximum exposure

RSC – relative source contribution

$t_{1/2}$ – half-life

UF – uncertainty factor

USEPA – U.S. Environmental Protection Agency

V_d – volume of distribution

WHO – World Health Organization

α – alpha, used to denote specific subtypes of biological molecules (i.e., proteins)

β – beta, used to denote specific subtypes of biological molecules (i.e., proteins)

γ - gamma, used to denote specific subtypes of biological molecules (i.e., proteins)

Acknowledgements

New Hampshire Department of Environmental Services would like to thank the numerous New Hampshire stakeholders and residents who provided valuable technical commentary on the initially proposed MCLs for PFOA, PFOS, PFNA and PFHxS. This includes New Hampshire's residents, academic institutions, community advocacy groups, representatives for the business community and municipalities. The science followed in deriving the currently proposed maximum contaminant levels was enacted in part as a result of their contributions. Additionally, NHDES is grateful for insights and information shared by professionals from other state agencies, interstate collaborative working groups and professional societies.

Section I. Executive Summary

The objective of the health-based risk assessment was identifying drinking water concentrations of perfluorooctanoic acid (PFOA), perfluorooctane sulfonic acid (PFOS), perfluorononanoic acid (PFNA) and perfluorohexane sulfonic acid (PFHxS) that provide adequate protection of human health at all life stages, including but not limited to pre-natal development. This document provides the technical basis for the proposed maximum contaminant levels (MCLs,) which by law become Ambient Groundwater Quality Standards (AGQSs), following evaluation of technical comments submitted up to April 12th, 2019, public comment deadline, as well as peer-reviewed scientific literature published since January 1st, 2019, and external review by Dr. Stephen Roberts at the University of Florida. As a result of this process, NHDES is proposing the following maximum contaminant levels (MCLs):

- **12 ng/L for Perfluorooctanoic acid, or perfluorooctanoate (PFOA)**
- **15 ng/L for Perfluorooctane sulfonic acid, or perfluorooctane sulfonate (PFOS)**
- **11 ng/L for Perfluorononanoic acid, or perfluorononanoate (PFNA)**
- **18 ng/L for Perfluorohexane sulfonic acid, or perfluorohexane sulfonate (PFHxS)**

These health-based values are intended as health-protective limits against the chronic health effects for a through-life exposure. The primary associated health outcomes are hepatotoxicity and changes in lipid metabolism (PFOA and PFNA), suppressed immune response to vaccines (PFOS) and impaired female fertility (PFHxS). Secondary associated health effects that are expected to be less sensitive are changes in thyroid and sex hormone levels, early-life growth delays, changes in cholesterol levels and biomarkers of liver function, neurobehavioral effects, and a possible risk for certain cancers (i.e., testicular and kidney cancer).

These proposed MCLs are lower than those proposed in January 2019 (NHDES 2019) as a result of new studies and models that indicate the standards need to be lower to be adequately protective of health at all life stages. Specifically, a peer reviewed toxicokinetic model was published by the Minnesota Department of Health (Goeden et al., 2019) that predicts blood serum levels across a lifetime. Using similar studies as those from the initial proposal and those suggested in technical comments submitted by April 12th, 2019, this model indicates lower standards are necessary to avoid unacceptable elevations in the serum levels of breastfed infants and children who were breastfed as infants.

The technical basis for the proposed MCLs is detailed in Sections III and IV, and the modeling results and conclusions are presented in Section V. Briefly, this risk assessment utilized upper value, “conservative” estimates regarding: daily water consumption rates throughout life, breastmilk consumption rates through infancy, the duration of exclusive breastfeeding (12 months), relative source contribution, absorption efficiency and consideration of breastmilk transfer. Central tendency, or less conservative, assumptions included: use of uncertainty factors, human half-life estimates, placental and breastmilk transfer efficiencies of PFAS, and the recommendation of individual MCLs instead of assuming toxicological equivalency among the four PFAS evaluated.

The health effects of PFAS is an evolving area of research and it is expected that future research will improve our understanding of the quantitative risks associated with PFAS. This may result in higher or lower recommendations for these and other PFAS in the future. NHDES is committed to reviewing new scientific information on PFAS to improve the understanding of this large group of chemicals and making future recommendations for evidence-based health protective drinking water standards.

Section II. Introduction

Perfluorooctanesulfonic acid (PFOS), perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), and perfluorohexanesulfonic acid (PFHxS) are individual compounds in a large class of chemicals known as perfluorinated compounds (PFCs) and more broadly as per- and polyfluoroalkyl substances (PFAS). They have been widely used since the 1940s in commercial, industrial, and household products and applications, including production of water, grease, and stain-resistant materials, fire suppression foams, non-stick cookware, wax removers, etc. (ATSDR 2018b).

All four compounds have been detected in New Hampshire's groundwater and surface water. Their widespread use, persistence and mobility in the environment and bioaccumulative properties has resulted in the detection of PFAS in blood serum in humans and animals worldwide. This has led to considerable research into their toxicity and health effects. The health effects associated with PFAS exposure are currently being researched extensively by toxicologists and epidemiologists worldwide, resulting in numerous publications being released on a continuous basis.

According to the Agency for Toxic Substances and Disease Registry (ATSDR)(ATSDR 2018b) the following health impacts may be associated with PFAS (specific compounds as noted by ATSDR):

- Hepatotoxicity - changes in certain liver enzymes in serum (PFOA, PFOS, PFHxS)
- Increases in total and LDL cholesterol levels (PFOA, PFOS, PFNA)
- Small decreases in birth weight (PFOA, PFOS)
- Endocrine system effects (PFOA, PFOS)
- Reproductive toxicity - decreased fertility (PFOA, PFOS)
- Immunotoxicity - decreased vaccine response (PFOA, PFOS, PFHxS)
- Suggestive evidence of carcinogenicity, specifically testicular and kidney cancer (PFOA, PFOS)
- Suggestive evidence of association with pregnancy-induced hypertension and/or pre-eclampsia (PFOA, PFOS)

For additional information on the toxicity and health effects of these compounds, please visit the ATSDR webpage at: https://www.atsdr.cdc.gov/pfas/health-effects.html

In addition to the ATSDR draft toxicological profile on perfluoroalkyls, several other state (NJDWQI 2017, 2018ab; MDH 2018, 2019ab; MI PFAS Science Advisory Panel 2018), federal (EPA 2016ab; NTP 2016) and international agencies (IARC 2016; Health Canada 2016ab; EFSA 2018) have reviewed the toxicological data related to PFAS and identified similar associated health impacts.

This document presents the health-based risk assessment that derived the proposed MCLs and Ambient Groundwater Quality Standards (AGQS) for these four compounds. In January 2019, NHDES released its initially proposed MCLs along with a supporting document that explained the rationale used and scientific literature reviewed to arrive at its recommendation (NHDES, 2019). The current report is not an exhaustive review of all existing studies that reference PFOA, PFOS, PFNA, PFHxS or other PFAS; rather, it is an update to the previous assessment after evaluation of newer studies and technical comments since the initial MCL proposal in January 2019 (NHDES, 2019).

Section III. Reference Dose Derivation

The U.S. EPA (2002) defines a reference dose (RfD) as:

> "An estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime."

For PFAS, a RfD can be expressed in units of nanograms of specified PFAS (ng), per kilogram of a person's body weight (kg), per day (ng/kg-d). This allows for estimation of chemical-specific daily doses that are readily scaled to persons of differing sizes. A RfD is not the same as the minimal risk levels (MRLs) developed and used by ATSDR in that 1) MRLs are not developed with the same considerations as RfDs, and 2) MRLs are not used to define action or clean up levels for chemical contaminants (EPA 2002; ATSDR 2018a). NHDES derived RfDs for PFOA, PFOS, PFNA and PFHxS (Table 1). *Additionally, it is important to note that a RfD is a population-level value and its associated blood concentration is not considered a clinically-relevant value for individuals.*

Table 1. Summary of RfDs and MCLs.

Compound	Reference dose (RfD)	Exposure Assumptions	Maximum Contaminant Level (MCL)
Perfluorooctanoic acid (PFOA)	6.1 ng/kg-d	See Section IV	12 ng/L
Perfluorooctanesulfonic acid (PFOS)	3.0 ng/kg-d	See Section IV	15 ng/L
Perfluorononanoic acid (PFNA)	4.3 ng/kg-d	See Section IV	11 ng/L
Perfluorohexanesulfonic acid (PFHxS)	4.0 ng/kg-d	See Section IV	18 ng/L

Derivation of a RfD requires selection of three components (Equation 2): a point of departure (POD), uncertainty factors (UF) and, where appropriate, a dosimetric adjustment factor (DAF). The POD is based on a sensitive and human-relevant critical health effect from either animal or human studies. For PFAS, this is typically a blood concentration of a certain compound at which there is no observable adverse effect in animals (e.g. rodents). As rodents are not humans, the UF is applied to be protective by reducing the animal POD to a lower and acceptable human target serum level. The DAF then converts, by estimation, the blood concentration (ng/mL) to a body weight-adjusted (kg) amount of the chemical (ng) external to the body that would need to be ingested on a daily basis to reach the human target serum level.

$$\text{Reference dose (ng/kg/d)} = \frac{\text{Point of departure (ng/mL)}}{\text{Total uncertainty factors (unitless)}} \times \text{Dosimetric adjustment factor (mL/kg/d)}$$

As the EPA RfDs for PFOA and PFOS were deemed insufficiently protective, and there are no values for PFNA or PFHxS in the EPA Integrated Risk Information System (IRIS) database, NHDES evaluated the RfDs proposed by other agencies and derived its own values. The remainder of Section III describes how RfDs for PFOA, PFOS, PFNA and PFHxS were derived following evaluation of relevant studies and technical comments submitted to NHDES by April 12th, 2019, as well as scientific uncertainties specific to the RfDs.

Perfluorooctanoic acid or perfluorooctanoate (PFOA), CAS# 335-67-1

Principal study & consideration of health effects

For the derivation of a RfD and MCL for PFOA, NHDES recommends the critical health effect of increased relative liver weight (Loveless et al., 2006; NJDWQI 2017) as an indicator for the onset of hepatotoxicity. This is the same critical health effect previously selected in the initial MCL proposal (NHDES 2019), and based on review of the literature and technical comments received, NHDES remains confident in this recommendation.

Since the initial MCL proposal by NHDES at the start of January 2019, additional studies have been published related to associations between PFOA and human health impacts along with studies demonstrating toxicity in rodent models. Relative to the critical effect proposed by NHDES, there are three new studies that merit acknowledgment with regard to relative liver toxicity. This includes two studies from highly-exposed populations (Bassler et al., 2019; Nian et al., 2019) and evaluation of background exposure levels from the 2011-2014 NHANES dataset (Jain and Ducatman 2019). Bassler and colleagues (2019) reported associations between non-clinical biomarkers of hepatocyte apoptosis (cell death) as well as altered inflammatory disease of the liver with exposure to PFOA and other PFAS within a subset of subjects from the C8 Cohort (mean PFOA serum level 94.6 ng/mL). In the C8 Health Study of China (n = 1,605 participants, median PFOA serum level of 6.19 ng/mL), liver enzyme markers such as ALT and AST showed significant increases with natural log (ln)-unit changes of PFOA, other PFAS and their isomers (Nian et al., 2019). Analysis of the 2011-2014 NHANES data (n=2,883 subjects) detected consistent associations between PFAS, including PFOA, and increased ALT and GGT in obese individuals. It is noted that the cross-sectional design of certain studies and the lack of adjustments for false discovery following multiple comparisons underscore typical challenges of relying on epidemiological studies to demonstrate causal relationships, or their utility for determining the POD in RfD development. Qualitatively, these studies reinforce NHDES consideration of altered liver function and hypertrophy in rodents as a critical health effect for the basis of its PFOA RfD.

Studies published prior to 2019 were considered as a part of the initial PFAS MCL proposal put forward by NHDES (2019). This included evaluation of peer-reviewed evidence for:

- associated immunotoxicity as summarized by the National Toxicology Program (NTP 2016), ATSDR (2018b), DeWitt et al., (2012), Kirk et al., (2018) and Chang et al., (2016),
- developmental toxicity in animal models (Butenhoff et al., 2004; Lau et al., 2006; White et al., 2007; Wolf et al., 2007; Hu et al., 2010; Onishchenko et al., 2011; White et al., 2011; Albrecht et al., 2013; Cheng et al., 2013; Koustas et al., 2014; Quist et al., 2015ab; Koskela et al., 2016), associated fetal and neonatal growth impacts in humans (reviewed by Verner et al., 2015; Negri et al., 2017; Rappazzo et al., 2017; Liew et al., 2018 and ATSDR 2018b) and consideration of developmental outcomes evaluated in the U.S. EPA LHA for PFOA of 70 ng/L (EPA 2016a),
- associated human-health outcomes based on the C8 studies (Frisbee et al., 2009, 2010; Steenland et al., 2009, 2010ab, 2013; Stein et al. 2009, 2013; Lopez-Espinosa et al., 2011, 2012ab; Gallo et al., 2012; Savitz et al., 2012ab; Steenland and Woskie 2012; Barry et al., 2013; Darrow et al., 2013; Fletcher et al., 2013; Vieira et al., 2013; Watkins et al., 2013; Winquist et al., 2013; Darrow et al., 2016),

- and delayed mammary gland development in mice (White et al., 2007, 2009, 2011; Macon et al., 2011; Tucker et al., 2015).

In its initial proposal, NHDES agreed with the assessment made by the New Jersey Drinking Water Quality Institute (NJDWQI) relative to adverse effects on the liver and NHDES maintains this position. In their 2017 document, NJDWQI summarized evidence from studies in non-human primates, various strains of rodents, including PPARα knock-out mice, as well as the existing epidemiologic studies. This lead the NJDWQI to the conclusion that there was "consistency among non-occupational studies, as well as evidence of specificity, exposure-response, strength, and biological plausibility for PFOA and ALT. These findings provide evidence supporting a causal relationship between PFOA and ALT" (NJDWQI 2017). They also acknowledge the limited epidemiologic evidence, as of 2017, to definitively prove a causal relationship with PFOA and liver disease, and the available studies did not find an association. (NJDWQI 2017). While NHDES does not agree with the application of a full database uncertainty factor (NJDWQI 2018), the arguments made for consideration of hepatic effects for human health risk assessment were deemed appropriate given the existing information on PFOA.

The ATSDR 2018 draft toxicity profile for perfluoroalkyls recognized the likely associations between PFOA and hepatotoxicity (e.g., increased serum enzyme concentrations and effects on serum bilirubin) after consideration of similar epidemiological studies and the NJDWQI 2017 report (NJDWQI 2017; ATSDR 2018b). After additional review of this same document (ATSDR 2018b), NHDES agrees there is concern for the associations between exposure to PFOA and the following human health outcomes: increases in serum lipids (i.e., total and LDL cholesterol), disruption of thyroid hormone function and transport, decreased vaccine response, decreased fertility and reduced birth weight. The scientific evidence is less clear regarding other suggested human health associations and merit further investigation to establish whether these effects are truly linked to PFOA exposure. As this relates to the RfD derived by NHDES, it was determined that the animal study selected by ATSDR was not appropriate for RfD derivation following NHDES understanding of EPA methodology (EPA 2002) and was therefore not selected for use in the initial or final MCL proposal.

Regarding carcinogenicity, NHDES derived a PFOA MCL based on non-cancer endpoints. The U.S. EPA and International Agency for Research on Cancer (IARC) determined that the current evidence indicates that PFOA is a suggestive (EPA 2016) or possible (IARC 2016) carcinogen in humans. This is specific to suggestive evidence for increased risks of kidney and testicular cancer seen in rodents and mixed associations from human studies (Barry et al., 2013). Two other agencies, the USEPA (2016a) and NJDWQI (2017), have derived cancer values for PFOA using the same principal rodent study for PFOA carcinogenicity (Butenhoff et al. 2012). The U.S. EPA (2016a) and NJDWQI (2017) arrived at possible MCL values of 500 ng/L and 14 ng/L, respectively, for a one-in-a-million risk for testicular cancer. More recently, the California Office of Environmental Health Hazard Assessment (2019) has recommended a similar value of 14 ng/L for PFOA citing concern for liver damage and cancer. This discrepancy in cancer-based MCL estimates highlights the need for better information to inform cancer risk assessment for PFOA, and is expected to be an evolving area of research in years to come. Regardless of whichever is the more accurate assessment, the proposed MCL for PFOA is lower than the more conservative of these two estimates.

Determination of a point of departure

As previously proposed by NHDES (2019), the principal study and point of departure (POD) was the same study (Loveless et al., 2006) recommended and benchmark dose modeled by the NJDWQI (2017). The critical health effect was increased relative liver weight in male mice following a 14-d oral exposure to APFO (Loveless et al., 2006). There is consistent evidence for liver toxicity across wild-type and PPARα knock-out mice (Butenhoff et al., 2004; Loveless et al., 2008; Son et al., 2008; Cui et al., 2009; Elcombe et al., 2010; Yahia et al., 2010; Tan et al., 2013; Wang et al., 2015; Rebholz et al., 2016; Li et al., 2017), as well as persistent effect on liver size and structure following gestational exposure to similar dosing regimens (Quist et al., 2015). Rat studies have suggested that this effect is an adaptive response that will dissipate following cessation of the exposure to PFOA (Butenhoff et al., 2004; Hall et al., 2012). Beyond rodent models, cynomolgus monkeys display hepatic hypertrophy, increased serum triglycerides and decreased serum T_4 following chronic exposure (26 weeks) to APFO (Butenhoff et al., 2002). As it relates to the present human health risk assessment for an MCL, these effects are not entirely adaptive as animal studies suggest persistent changes in the liver following exposure during early life stages (Quist et al., 2015a). NHDES also maintains its previous position that whether the response is adaptive is not relevant to drinking water exposures as the general population should not require recovery periods from public water. Furthermore, unlike rodents that display relatively short half-lives for PFOA and other PFAS, once humans are exposed to increased levels of PFOA they will maintain elevated serum levels on a time scale of months to years. This means that brief external exposures become chronic internal doses, especially if the external dose is relatively high. The effects on liver function are considered a chronic health outcome based on the existing body of literature.

This POD is based on the benchmark dose modeling work conducted by the NJDWQI (2017) in their technical documents for their proposed RfD and MCL of 2.0 ng/kg-d and 14 ng/L, respectively, that identified a POD for PFOA of 4,351 ng/mL based on increased liver weight. NHDES did not arrive at the same RfD due to differences in the application of uncertainty factors. Differences in the final MCL are due to NH's use of the transgenerational exposure model for breastfeeding (Goeden et al., 2019).

Application of uncertainty factors

A total uncertainty factor of 100 was applied to the POD for PFOA based on:

Intraspecies variability (10) × Interspecies variability (3) × Database limitations (3) = 100

For the non-risk assessor, the units of 3 and 10 are for partial (half) and full log units. So, a full log unit of 10 equals 10^1, but a half log unit of $10^{½}$ or $10^{0.5}$ is equal to 3.162. As a convention of risk assessment using EPA methodology (EPA 2002), the value of 3.162 is presented as 3. Thus, 10 × 3 × 3 is rounded to 100 from 99.982.

The full factor of 10 for intraspecies variability was deemed appropriate to protect for the poorly characterized differences in toxico-dynamics (× 3) and -kinetics (× 3) within the human population. As NHDES applied a DAF to convert the rodent serum concentration to an oral human dose, only a partial uncertainty factor (× 3) was applied for interspecies variability. As the NJDWQI (2017) derived a benchmark dose, there was no need for any additional uncertainty factors to account for lowest

observed adverse effect level (LOAEL) to no observed adverse effect level (NOAEL) conversion. As the critical effect of hepatic hypertrophy is considered the onset of the adverse effect in a sensitive model species, no additional uncertainty factor was applied to account for acute-to-chronic duration of exposure.

Although NHDES agrees with the NJDWQI selection of a critical health effect and derivation of the POD for PFOA (NJDWQI 2017), NHDES concluded there is insufficient evidence supporting the application of the more conservative full database uncertainty factor (× 10). In technical comments submitted on the initially proposed MCLs, this decision was the subject of multiple critiques. On one hand, some have argued the use of a partial uncertainty factor was under-protective as the NJDWQI applied a full factor (× 10) due to concerns for observations of delayed mammary gland development in mice exposed to PFOA during perinatal development (NJDWQI 2017, and references therein). NHDES notes that the USEPA LHA (2016a) and CDC's ATSDR draft report (2018b) did not apply any database uncertainty factor with respect to the mammary gland development studies in rodents given the lack of clarity towards human health relevance (Table 3). Similar to New Hampshire, two other state agencies, Minnesota (MDH 2018) and New York (presentation, October, 2018), derived RfDs for PFOA affording only a partial uncertainty factor for this and other adverse health impacts observed in rodent and epidemiological studies. It should be noted that both of these other agencies did not use the same POD as NJDWQI or NHDES, where Minnesota utilized a higher POD and New York utilized a lower POD compared to the benchmark dose (BMD) value from Loveless et al., (2006). Thus, NHDES believes that the application of a partial database uncertainty factor (× 3) is appropriately protective without being overly conservative given the critical health effect selected and the existing toxicological and epidemiological database.

Estimation of a human equivalent oral dose

The POD represents an internal animal serum level associated with the adverse health outcome of concern. Dividing the POD by the total uncertainty factor yields a protective target serum level equivalent for the human population. *This is not a clinical or diagnostic value, nor should it be interpreted as such*.

$$\text{Target serum level for PFOA} = \frac{4{,}351 \text{ ng/mL}}{100} = 43.5 \text{ ng/mL}$$

To estimate how this internal blood level corresponds to an external oral dose of the specified compound, a dosimetric adjustment factor is applied by multiplication to identify a dose in ng of specified PFAS, per kg of individual body weight, per day (ng/kg-d). This step accounts for the highly-bioaccumulative nature and unique half-life estimates of each compound, and is consistent with prior risk assessment methods for derivation of RfDs for PFAS (USEPA 2016ab; NJDWQI 2017, 2018a; ATSDR 2018b; MDH 2018, 2019ab). The human equivalent oral dose is estimated by the following equations:

$$\text{Reference dose (RfD)} = \frac{\text{Point of departure (POD)}}{\text{Total uncertainty factors (UF)}} \times \text{Dosimetric adjustment factor (DAF)}$$

Where the DAF is equal to,

$$DAF = V_d \times \left(\frac{Ln(2)}{t_{1/2}}\right)$$

$$DAF = 170\ mL/kg \times \left(\frac{Ln(2)}{840\ days}\right) = 1.40\times10^{-1}\ mL/kg\text{-}d$$

Consistent with the initial PFOA MCL proposal (NHDES 2019), the volume of distribution (V_d) for PFOA was 170 mL/kg (Thompson et al., 2010; EPA, 2016a). For its revised and final proposal, NHDES selected the serum half-life of 2.3 years for PFOA (Bartell et al., 2010). NHDES acknowledges that the half-life of 2.3 years is slightly less conservative than the initially proposed value for RfD derivation of 2.7 years (Li et al. 2018; NHDES 2019). This change was due, in part, to the consideration of this half-life being more appropriate given the significantly higher exposure specific to PFOA described in Bartell et al. (2010) and the larger sample size than that in Li et al. (2018).

Thus, using this chemical-specific DAF and the aforementioned point of departure and uncertainty factors, NHDES derived an oral reference dose for PFOA of 6.1 ng/kg-d.

$$\text{Reference dose (RfD)} = \frac{4{,}351\ ng/mL}{100} \times 1.40\times10^{-1}\ mL/kg\text{-}d = 6.1\ ng/kg\text{-}d$$

Perfluorooctane sulfonic acid or perfluorooctane sulfonate (PFOS), CAS# 1763-23-1

Principal study & consideration of health effects

For the derivation of a RfD for PFOS, NHDES recommends the critical health effect of suppressed immunoglobulin M (IgM) production in male mice as proposed by the Minnesota Department of Health (Dong et al., 2011; MDH, 2019a). While NHDES previously proposed a RfD based on developmental toxicity, the review of existing and emerging evidence and technical comments suggest that the use of this immunotoxic endpoint represents a more appropriately cautious approach for the risk assessment of PFOS.

Since the initial MCL proposal by NHDES at the start of January 2019, additional studies have been published related to associations between PFOS and human health impacts along with studies demonstrating toxicity in rodent models. In the same studies that found associations between PFOA and serological markers of liver function (Nian et al., 2019; Jain and Ducatman, 2019; Bassler et al., 2019), PFOS was also associated with liver dysfunction and markers of hepatic inflammatory responses. Relative to the critical health effect selected by NHDES, one additional study on immunosuppression in humans was published since January 2019. In a prospective study of 3-month old infants from China (n = 201 participants), cord blood levels of branched isomers of PFOS were associated with reduced concentrations of antibodies towards enterovirus 71 (a causative viral agent of hand-foot-and-mouth disease; Zeng et al., 2019). Aside from hepatic and immune effects, additional studies have suggested associations between prenatal PFOS levels and early onset of puberty in girls from the Danish Birth Cohort (Ernst et al., 2019) and an estrogen-mediated relationship between cord blood levels of PFOS and birth weight (Wang et al., 2019). As with many epidemiological studies on PFAS, many of these recent studies possessed various combinations of limitations including a lack of analysis for other environmental contaminants, limited sample size and lack of analysis for the influence of breastfeeding. However, they collectively demonstrate that there is a growing body of evidence for adverse health impacts associated with PFOS.

Studies published prior to 2019 were considered as a part of the initial PFAS MCL proposal put forward by NHDES (2019). This included evaluation of peer-reviewed evidence for:

- immunotoxicity as summarized by the National Toxicology Program (NTP 2016), ATSDR (2018b) DeWitt et al., (2012) and Chang et al., (2016),
- developmental toxicity in animal models (Lau et al., 2003; Thibodeaux et al., 2003; Luebker et al., 2005ab; Yahia et al., 2008; Butenhoff et al., 2009; Onishchenko et al., 2011; Rogers et al., 2014; Wan et al., 2014), fetal and neonatal growth impacts in humans (reviewed by Verner et al., 2015; Negri et al., 2017; Rappazzo et al., 2017; Liew et al., 2018 and ATSDR 2018b) and consideration of delayed development in the U.S. EPA LHA for PFOS of 70 ng/L (EPA 2016b),
- neurobehavioral and thyroid hormone-associated effects (as reviewed by ATSDR 2018b).

NHDES acknowledges that the current understanding of the immunotoxic effects of PFOS, other PFAS and their interactions is an evolving area of research. As described by DeWitt et al. (2019), the interpretation of immunosuppression is important to consider when evaluating the relevance of associated outcomes from human studies, as well as measured responses from rodents. The current body of literature is not mature enough to clearly evaluate clinical relevance to humans, or lack thereof

(Chang et al., 2016); however, the NTP (2016) concluded that PFOS is "presumed to be an immune hazard to humans" based on animal and human data available at that time. Mouse studies indicate that PFOS impairs the T cell-dependent antibody response at low doses following sub-chronic exposure durations (Dong et al., 2009, 2011; reviewed by DeWitt et al., 2012, 2019), and was selected as the basis for a PFOS RfD by several agencies including NJDWQI (NJDWQI 2018; further detailed by Pachkowski et al. 2019), NYDOH (2018) and proposed by MDH (2019a). Although the ATSDR MRL for PFOS was based on developmental delays (Luebker et al., 2005ab), they applied an additional uncertainty factor of 10 due to the evidence for immunotoxicity (ATSDR, 2018b). Collectively, this indicates that the lower dose range at which the immunotoxic effects occur in rodents is recognized as an appropriately protective range for selection of a POD. There is a critical need for replication and use of larger study populations for understanding the immunomodulatory associations reported for PFOS and other PFAS.

NHDES derived a PFOS MCL based on non-cancer endpoints due to a lack of adequate carcinogenicity studies. IARC has not classified the carcinogenicity of PFOS at this time. The U.S. EPA determined that PFOS was a suggestive carcinogen (EPA, 2016b). This is specific to suggestive evidence for increased incidence of liver and thyroid adenomas in rats following chronic exposure. The recommendation of using non-cancer endpoints over cancer endpoints is not unique to NHDES, as other agencies have concluded that non-cancer health endpoints are adequately protective (MDH 2018; Michigan PFAS Science Advisory Panel 2018). Should additional information become available that is adequate for derivation of a cancer slope factor (CSF) for PFOS, NHDES will consider this in the framework of the MCL process.

Determination of point of departure

Following review of the technical documents deriving RfDs for PFOS based on immunosuppression in mice (NJDWQI, 2018; ATSDR 2018b; Pachkowski et al., 2019; MDH, 2019), NHDES agreed with the RfD derivation recently proposed by the Minnesota Department of Health (MDH 2019). This POD is based on serum concentrations of PFOS at the no observable adverse effect level (NOAEL) for suppressed IgM production in male mice following 60-d oral exposure (Dong et al. 2011). As summarized by MDH (2019), the critical effect reported in Dong et al. (2011) was suppressed IgM production with a NOAEL of 2,620 ng/mL (oral dose, 0.0167 mg/kg-d) and a LOAEL of 10,750 ng/mL (oral dose, 0.083 mg/kg-d). A prior study by Dong et al. (2009) reported a NOAEL of 674 ng/mL (oral dose, 0.008 mg/kg-d) for reduced plaque forming cell response to sheep red blood cells, and a similar oral LOAEL as Dong et al. (2011). However, the early work by Dong et al. (2009) did not include the intermediate dose of 0.0167 mg/kg-d that was identified as a NOAEL in their later work (Dong et al. 2011). This is further complicated as the specific effect was not replicated in both studies where plaque forming cell response was only measured in Dong et al. (2009) and IgM concentrations in the later Dong et al. (2011). As both of these metrics describe different aspects of the same immune process they do support the consideration of immunosuppression at these low doses as a POD. There remains the issue of discordance in dosing. While benchmark dose modeling of these endpoints using the original data might prove valuable to demonstrating these different metrics support a similar POD, the original data was not available for modeling and the reported data has been described as unamenable to benchmark dose modeling (NJDWQI 2018). As a result, NHDES agreed with the use of the NOAEL (2,620 ng/mL) for IgM suppression (Dong et al., 2011) instead of the lower NOAEL of 674 ng/mL (Dong et al., 2009) as a POD.

Application of uncertainty factors

A total uncertainty factor of 100 was applied to the POD for PFOS based on:

Intraspecies variability (10) × Interspecies variability (3) × Database limitations (3) = 100

For the non-risk assessor, the units of 3 and 10 are for partial (half) and full log units. So, a full log unit of 10 equals 10^1, but a half log unit of $10^{½}$ or $10^{0.5}$ is equal to 3.162. As a convention of risk assessment using EPA methodology (EPA 2002), the value of 3.162 is presented as 3. Thus, 10 × 3 × 3 is rounded to 100 from 99.982.

The full factor of 10 for intraspecies variability was deemed appropriate to protect for the poorly characterized differences in toxico-dynamics (× 3) and -kinetics (× 3) within the human population. As NHDES applied a DAF to convert the rodent serum concentration to an oral human dose, only a partial uncertainty factor (× 3) was applied for interspecies variability. The POD was based on the NOAEL described in Dong et al. (2011); thus, there was no need for additional uncertainty factors to account for LOAEL to NOAEL conversion. Dong et al. (2011) conducted a 60-day exposure so no additional uncertainty factor was applied for acute-to-chronic duration of exposure. As described by MDH (2019), an additional partial (× 3) database uncertainty factor was applied due to concerns for reports of thyroid disruption (decreased T_4) in neonatal animals and the implications of these observations in terms of neurodevelopment that has not yet been adequately studied. NHDES agreed with this consideration given the suggestive evidence for the human relevance of altered T_4 levels (reviewed by Ballesteros et al., 2017 and ATSDR, 2018b) and their potential implications for impaired neurodevelopment in humans (Grandjean and Landrigan, 2014).

Estimation of a human equivalent oral dose

The POD represents an internal animal serum level associated with the adverse health outcome of concern. Dividing the POD by the total uncertainty factor yields a protective target serum level equivalent for the human population. *This is not a clinical or diagnostic value, nor should it be interpreted as such*.

$$\text{Target serum level for PFOS} = \frac{2{,}360\ \text{ng/mL}}{100} = 23.6\ \text{ng/mL}$$

To estimate how this internal blood level corresponds to an external oral dose of the specified compound, a dosimetric adjustment factor is applied by multiplication to identify a dose in ng of specific PFAS per kg of individual body weight per day (ng/kg-d). This step accounts for the highly-bioaccumulative nature and unique half-life estimates of each compound, and is consistent with prior risk assessment methods for derivation of RfDs for PFAS (EPA, 2016ab; NJDWQI, 2017, 2018a; ATSDR, 2018b; MDH, 2018, 2019ab). The human equivalent oral dose is estimated by the following equations:

$$\text{Reference dose (RfD)} = \frac{\text{Point of departure (POD)}}{\text{Total uncertainty factors (UF)}} \times \text{Dosimetric adjustment factor (DAF)}$$

Where the DAF is equal to,

$$DAF = V_d \times \left(\frac{Ln(2)}{t_{1/2}}\right)$$

$$DAF = 230\ mL/kg \times \left(\frac{Ln(2)}{1{,}241\ days}\right) = 1.28\times10^{-1}\ mL/kg\text{-}d$$

Consistent with the initial PFOS MCL proposal (NHDES 2019), the V_d for PFOS was 230 mL/kg (Thompson et al., 2010). In its revised and final proposal, NHDES maintains its use of a 3.4-year half-life estimate based on the average across men and women, described in Li et al. (2018; NHDES 2019). NHDES considered the longer half-life values reported for retired fluorochemical workers (Olsen et al. 2007), and deemed these to be inappropriately conservative given the use of the Minnesota transgenerational model for exposure assessment which emphasizes early-life and breastfeeding exposures.

Thus, using this chemical-specific DAF and the aforementioned point of departure and uncertainty factors, NHDES derived an oral reference dose for PFOS of 3.0 ng/kg-d.

$$\text{Reference dose (RfD)} = \frac{2{,}360\ ng/mL}{100} \times 1.28\times10^{-1}\ mL/kg\text{-}d = 3.0\ ng/kg\text{-}d$$

Perfluorononanoic acid or perfluorononanoate (PFNA), CAS# 375-95-1

Principal study & consideration of health effects

For the derivation of a RfD and MCL for PFNA, NHDES recommends the critical health effect of increased relative liver weight in pregnant mice (Das et al., 2015; NJDWQI, 2018) as an indicator for the onset of hepatotoxicity. This is the same critical health effect previously selected in the initial MCL proposal (NHDES, 2019), and based on additional review of the literature NHDES remains confident in this decision.

Since the initial MCL proposal by NHDES at the start of January 2019, additional studies have been published related to associations between PFNA and associated human health impacts along with studies demonstrating toxicity in rodent models. In the same studies that found associations between PFOA and serological markers of liver function (Nian et al., 2019; Jain and Ducatman, 2019; Bassler et al., 2019), PFNA was also associated with liver dysfunction and markers of hepatic inflammatory responses. As discussed later, this co-association between multiple PFAS and the same health outcomes is acknowledged as a present challenge of epidemiological research. The same study of the Danish Birth Cohort that associated PFOS with an early onset of puberty in girls found that prenatal serum levels of PFNA were associated with delayed onset of puberty in boys (Ernst et al., 2019). Ernst and colleagues (2019) noted that these associations merit caution in their interpretation and require replication due to their novelty. Unlike PFOA and PFOS, PFNA has been the subject of relatively less research and its lower background serum concentrations compared to PFOA and PFOS present a challenge to identifying its effects in human populations.

Studies published prior to 2019 were considered as a part of the initial PFAS MCL proposal put forward by NHDES (2019). At the time, two major documents reviewed the toxicity of PFNA in humans and rodents (NJDWQI, 2018; ATSDR, 2018b). As noted in both documents, relatively little research has been conducted on PFNA despite its historical use and presence in a variety of environmental media. The NJDWQI concluded there was limited evidence associating PFNA with changes in serum ALT as a biomarker of hepatotoxicity (NJDWQI, 2018), whereas the ATSDR determined these inconsistencies in epidemiological data did not merit inclusion of hepatotoxicity as an associated health outcome for PFNA (ATSDR, 2018b). In its initial proposal, NHDES agreed with the assessment made by the NJDWQI relative to adverse effects on the liver and NHDES maintains this position. Given the limited amount of epidemiological data currently available for PFNA and its similarity in chemical structure to PFOA and biological activities in animal models, NHDES determined that the associated hepatotoxic effects were more relevant and sensitive for human health risk assessment than the developmental and endocrine effects reported in animal studies. While NHDES does not agree with the application of the database uncertainty factor or animal-to-human dose extrapolation, the arguments made for consideration of hepatotoxicity by NJDWQI (2018) were deemed appropriate given the existing information.

To date, the carcinogenicity of PFNA has not been reported in a rodent model. The human carcinogenicity of PFNA has not been classified by the U.S. EPA, IARC or CDC (ATSDR). Therefore, NHDES did not conduct a cancer-based risk assessment for PFNA. Should additional information become available that is adequate for consideration of a cancer slope factor (CSF) for PFNA, NHDES recommends consideration as to whether its development and application of such values would be more protective than the proposed MCL.

Determination of a point of departure

As previously proposed by NHDES (2019), the principal study and point of departure (POD) was the same study (Das et al., 2015) recommended and benchmark dose modeled by the NJDWQI (2018). The critical health effect was increased relative liver weight in pregnant mice following a 17-d (duration of gestation) oral exposure to PFNA (Das et al., 2015). The internal LOAEL for these mice was 12,400 ng/mL which corresponded to an oral dose of 1.0 mg/kg-d (Das et al., 2015). While no significant mortality was observed at this dose, higher oral doses (>5.0 mg/kg-d) were associated with neonatal mortality in mice. Wolf et al. (2010) demonstrated the profound effects of PFNA on mouse pups were due to PPARα activation which raises uncertainty about the qualitative and quantitative relevance of this outcome to human health. Additional studies demonstrate that rodent models display hepatotoxic responses towards PFNA (Wolf et al., 2010; Wang et al., 2015), with evidence of PPARα-independent mechanisms (Rosen et al., 2017).

This POD is based on the benchmark dose modeling work conducted by the NJDWQI (2018) in their technical documents for their proposed MCL of 13 ng/L. It should be noted that NJDWQI did not derive a RfD as a part of the MCL development, as a ratio method was used instead of a DAF with water ingestion rate to convert the target serum level to a corresponding water concentration. NHDES did not arrive at the same MCL because NHDES opted to derive a RfD consistent with the other PFAS evaluated, as well as use of the transgenerational exposure model for breastfeeding (Goeden et al., 2019; MIDHHS, 2019).

Application of uncertainty factors

A total uncertainty factor of 100 was applied to the POD for PFNA based on:

Intraspecies variability (10) × Interspecies variability (3) × Database limitations (3) = 100

For the non-risk assessor, the units of 3 and 10 are for partial (half) and full log units. So, a full log unit of 10 equals 10^{1}, but a half log unit of $10^{½}$ or $10^{0.5}$ is equal to 3.162. As a convention of risk assessment using EPA methodology (EPA 2002), the value of 3.162 is presented as 3. Thus, 10 × 3 × 3 is rounded to 100 from 99.982.

The full factor of 10 for intraspecies variability was deemed appropriate to protect for the poorly characterized differences in toxico-dynamics (× 3) and -kinetics (× 3) within the human population. As NHDES applied a DAF to convert the rodent serum concentration to an oral human dose, only a partial uncertainty factor (× 3) was applied for interspecies variability. As the NJDWQI (2018) derived a benchmark dose, there was no need for any additional uncertainty factors to account for LOAEL to NOAEL conversion. As with PFOA, the critical effect of hepatic hypertrophy is considered the onset of the adverse effect in a sensitive model species. Consistent with PFOA, no additional uncertainty factor was applied to account for acute-to-chronic duration of exposure. The NJDWQI applied a full LOAEL to NOAEL uncertainty factor (× 10) to account for differences between the 17-d exposure in Das et al. (2015) and longer exposures resulting in reported adverse effects (summarized in NJDWQI, 2018). As increased liver weight in mice is already considered to be a highly-sensitive critical effect in response to PFAS, NHDES determined this was overly conservative given similar uncertainty factor considerations for the similar perfluorinated carboxylic acid, PFOA.

In its original proposal, NHDES applied a full database uncertainty factor (× 10) to account for the limited existing literature on PFNA (× 3), as well as the absence of a serum-derived human half-life estimate (× 3; NHDES 2019). As a part of its revision to the proposed RfDs and subsequent MCLs, NHDES utilized the more conservative half-life of PFNA derived for men and older women. Given the application of this more conservative half-life estimate, NHDES removed the associated partial uncertainty factor for PFNA. NHDES retained the partial uncertainty factor of × 3 to account for a lack of multigenerational rodent studies using PFNA, as well as concern for potential immunotoxic impacts seen with other PFAS (NTP 2016; DeWitt et al., 2012, 2019).

Estimation of a human equivalent oral dose

The POD represents an internal animal serum level associated with the adverse health outcome of concern. Dividing the POD by the total uncertainty factor yields a protective target serum level equivalent for the human population. *This is not a clinical or diagnostic value, nor should it be interpreted as such*.

$$\text{Target serum level for PFNA} = \frac{4{,}900\ \text{ng/mL}}{100} = 49.0\ \text{ng/mL}$$

To estimate how this internal blood level corresponds to an external oral dose of the specified compound, a dosimetric adjustment factor is applied by multiplication to identify a dose in ng of specific PFAS per kg of individual body weight per day (ng/kg-d). This step accounts for the highly-bioaccumulative nature and unique half-life estimates of each compound, and is consistent with prior risk assessment methods for derivation of RfDs for PFAS (USEPA 2016ab; NJDWQI 2017, 2018a; ATSDR 2018b; MDH 2019ab). The human equivalent oral dose is estimated by the following equations:

$$\text{Reference dose (RfD)} = \frac{\text{Point of departure (POD)}}{\text{Total uncertainty factors (UF)}} \times \text{Dosimetric adjustment factor (DAF)}$$

Where the DAF is equal to,

$$\text{DAF} = V_d \times \left(\frac{\text{Ln}(2)}{t_{1/2}}\right)$$

$$\text{DAF} = 200\ \text{mL/kg} \times \left(\frac{\text{Ln}(2)}{1{,}570\ \text{days}}\right) = 8.83 \times 10^{-2}\ \text{mL/kg-d}$$

Consistent with the initial PFNA MCL proposal (NHDES 2019), the V_d for PFNA was 200 mL/kg based on similar assumptions made by ATSDR (ATSDR 2018b). In this revised proposal, NHDES adjusted the half-life value from 2.5 to 4.3 years based on urinary half-lives estimated for men and older women, groups that tend to eliminate PFAS slower than younger and reproductive age women (Zhang et al., 2013; NHDES, 2019). As previously discussed in its initial proposal (NHDES, 2019), NHDES would prefer to have more reliable serum half-life estimates for PFNA instead of the urinary-derived estimates reported by Zhang and colleagues (2013). However, since the submission of the initial proposal no additional studies have been published that report a serum-based estimate for the half-life of PFNA in humans. Should additional peer-reviewed studies emerge that provide more rigorous estimates of these values, NHDES recommends consideration as to whether such data would represent and merit a significant change for the PFNA RfD.

Thus, using this chemical-specific DAF and the aforementioned point of departure and uncertainty factors, NHDES derived an oral reference dose for PFNA of 4.3 ng/kg-d.

$$\text{Reference dose (RfD)} = \frac{4{,}900 \text{ ng/mL}}{100} \times 8.83\times10^{-2} \text{ mL/kg-d} = 4.3 \text{ ng/kg-d}$$

Perfluorohexane sulfonic acid or perfluorohexane sulfonate (PFHxS), CAS# 355-46-4

Principal study & consideration of health effects

For the derivation of a RfD and MCL for PFHxS, NHDES recommends the critical health effect of impaired female reproduction as determined by reduced litter size initially reported in Chang et al. (2018). This RfD derivation is currently under peer-review with a scientific journal (Ali et al. *in review*). This is the same critical health effect previously proposed in the initial MCL proposal (NHDES 2019), albeit the present value is adjusted for benchmark dose modeling and selection of endpoint specific factors for dosimetric adjustment. NHDES developed the revised RfD in collaboration with external collaborators, Dr.'s Leah Stuchal and Stephen Roberts at the University of Florida, and awaits external peer-review on the soundness of its derivation. Should peer-review recommend revision and adjustment of the proposed RfD, NHDES will review the current MCL to determine if adjustments are required to be adequately protective of human health.

Since its initial proposal (NHDES, 2019), there has been a limited amount of new information generated relative to PFHxS. The Minnesota Department of Health proposed a RfD for PFHxS of 9.7 ng/kg-d based on reduced free T_4 in exposed rats using unpublished data from the NTP. At the time of writing this recommendation, the ATSDR has not released a revision to their 2018 draft MRL of 20 ng/kg-d based upon thyroid follicular cell damage in rats (ATSDR, 2018b). PFHxS showed similar associations with serological markers of liver function and inflammation as reported for PFOA, PFOS and PFNA (Nian et al., 2019; Jain and Ducatman, 2019; Bassler et al., 2019). Despite its legacy of widespread environmental occurrence associated primarily with AFFF use and growing regulatory interests, relatively little new toxicological information has emerged for PFHxS as of June 2019.

Studies published prior to 2019 were considered as a part of the initial PFAS MCL proposal put forward by NHDES (2019). This included re-evaluation of peer-reviewed evidence considered by ATSDR (2018b) including:

- thyroid toxicity including altered thyroid histology and reduced T_4 levels in rodent models (Butenhoff et al., 2008; Chang et al., 2018; Ramhøj et al., 2018), as well as epidemiology studies for altered T_4 levels (Ballesteros et al., 2017),
- immunomodulation in humans (Grandjean et al., 2012; Dong et al., 2013; Humblet et al., 2014; Okada et al., 2014; Buser and Scinicariello 2016; Stein et al., 2016; Zhu et al., 2016)
- reproductive and developmental toxicity in rodents (Butenhoff et al., 2008; Viberg et al., 2013; Chang et al., 2018; Ramhøj et al., 2018)
- hepatotoxicity or changes in lipid metabolism in rodents (Butenhoff et al., 2008; Bijland et al., 2011; Rosen et al., 2017; Chang et al., 2018; Ramhøj et al., 2018) and humans (Nelson et al., 2010; Starling et al., 2014; Mattsson et al. 2015).
- and human carcinogenicity (Hardell et al., 2010; Bonefel et al., 2014; Hurley et al., 2018).

To date, the carcinogenicity of PFHxS has not been reported in a rodent model. The human carcinogenicity of PFHxS has not been classified by the U.S. EPA, IARC or CDC (ATSDR). Therefore, NHDES did not conduct a cancer-based risk assessment for PFHxS. Should additional information become available that is adequate for consideration of a CSF for PFHxS, NHDES recommends consideration as to whether its development and application would be more protective than the proposed MCL.

Determination of a point of departure

As described in its initial MCL proposal (NHDES 2019), the principal study and point of departure (POD) was the same study (Chang et al., 2018) that has been adjusted primarily by use of benchmark dose modeling (Ali et al., *in review*). The critical health effect was reduced litter size in mice following a 14-d, prior to pregnancy, oral exposure to PFHxS (Chang et al., 2018). As mentioned above, the details and methodology for derivation of the POD for PFHxS are currently under review in Ali et al (*in review*). Benchmark dose (BMD) modeling was performed using Benchmark Dose Software (BMDS) (Version 3.1; USEPA, 2019). The critical effect endpoint was a change in the mean live litter size for adult CD-1 female mice, and due to the unavailability of litter-specific data was modeled based on PFHxS serum concentrations on study day 14 (reported in Chang et al., 2018). This resulted in a benchmark dose of 41,200 ng/mL and a 95% lower confidence limit on the benchmark dose (BMDL) of 13,900 ng/mL. NHDES determined that this is an appropriately cautious endpoint given the limited number of animal studies (reviewed in NHDES, 2019), considerably longer half-lives of PFHxS in humans when compared to other PFAS (Olsen et al., 2007; Zhang et al., 2013; Worley et al., 2017; Li et al., 2018), environmental occurrence and exposures (Daly et al., 2018), as well as suggestive associations of reproductive impacts in humans (Vélez et al., 2015; Zhou et al., 2017; Zhang et al., 2018).

Application of uncertainty factors

A total uncertainty factor of 300 was applied to the POD for PFHxS based on:

Intraspecies variability (10) × Interspecies variability (3) × Duration of exposure (3)
× Database limitations (3) = 300

For the non-risk assessor, the units of 3 and 10 are for partial (half) and full log units. So, a full log unit of 10 equals 10^1, but a half log unit of $10^{½}$ or $10^{0.5}$ is equal to 3.162. As a convention of risk assessment using EPA methodology (EPA 2002), the value of 3.162 is presented as 3. Thus, 10 × 3 × 3 × 3 is rounded to 300 from 316.14.

The full factor of 10 for intraspecies variability was deemed appropriate to protect for the poorly characterized differences in toxico-dynamics (× 3) and -kinetics (× 3) within the human population. As NHDES applied a DAF to convert the rodent serum concentration to an oral human dose, only a partial uncertainty factor (× 3) was applied for interspecies variability. As benchmark dose modeling was used to derive a POD, detailed in Ali et al. (*in review*), there was no need for any additional uncertainty factors to account for LOAEL to NOAEL conversion. After careful evaluation of technical comments and re-assessment of the literature and principal study, an additional but partial uncertainty factor (× 3) was applied to account for acute-to-chronic duration of exposure of female mice. In Chang et al. (2018), female mice received a less than chronic exposure (14 days) to PFHxS prior to the start of pregnancy. Because of the relatively limited number of studies on PFHxS and evidence for adverse impacts following longer exposure to similar compounds (i.e., PFOS), this was determined to be appropriate without being overly conservative (e.g., a full factor of × 10).

In its original proposal, NHDES applied a full database uncertainty factor (× 10) to account for the limited existing literature on PFHxS (× 3), as well as associations with thyroid hormone and transport interference (× 3; NHDES 2019). As a part of its revision to the proposed RfD and subsequent MCL,

NHDES determined the existing single-generation studies provide some basis for evaluating the reproductive and developmental toxicity of PFHxS. However, NHDES retained a partial uncertainty factor (× 3) to account for a lack of multigenerational rodent studies, as well as concern for potential immunotoxic impacts seen with other PFAS that have yet to be assessed (NTP 2016; DeWitt et al., 2019). The protracted human half-life of PFHxS relative to other PFAS underscores the need for additional research into biological impacts following chronic exposures.

Estimation of a human equivalent oral dose

The POD represents an internal animal serum level associated with the adverse health outcome of concern. Dividing the POD by the total uncertainty factor yields a protective target serum level equivalent for the human population. *This is not a clinical or diagnostic value, nor should it be interpreted as such*.

$$\text{Target serum level for PFHxS} = \frac{13{,}900\ \text{ng/mL}}{300} = 46.3\ \text{ng/mL}$$

To estimate how this internal blood level corresponds to an external oral dose of the specified compound, a dosimetric adjustment factor is applied by multiplication to identify a dose in ng of specific PFAS per kg of individual body weight per day (ng/kg-d). This step accounts for the highly-bioaccumulative nature and unique half-life estimates of each compound, and is consistent with prior risk assessment methods for derivation of RfDs for PFAS (USEPA 2016ab; NJDWQI 2017, 2018a; ATSDR 2018b; MDH 2019ab). The human equivalent oral dose is estimated by the following equations:

$$\text{Reference dose (RfD)} = \frac{\text{Point of departure (POD)}}{\text{Total uncertainty factors (UF)}} \times \text{Dosimetric adjustment factor (DAF)}$$

Where the DAF is equal to,

$$\text{DAF} = V_d \times \left(\frac{\text{Ln}(2)}{t_{1/2}}\right)$$

$$\text{DAF} = 213\ \text{mL/kg} \times \left(\frac{\text{Ln}(2)}{1{,}716\ \text{days}}\right) = 8.61\times10^{-2}\ \text{mL/kg-d}$$

In its revised MCL proposal for PFHxS, NHDES has changed both the V_d and half-life estimate for PFHxS to reflect the female-specific health impact utilized as the basis of the RfD. The V_d for PFHxS was reduced from 287 to 213 mL/kg which reflects a female-specific V_d value for PFHxS (Sundström et al., 2012). Sundström et al. (2012) reports the volume of distribution for cynomolgus monkeys, not humans, and no human V_d is currently available for PFHxS. Similar to ATSDR (ATSDR 2018b) and other agencies (MDH 2019b; MIDHHS 2019), NHDES used the non-human primate value as an estimate for the human volume of distribution. Similarly, NHDES adjusted the half-life value from 5.3 to the female-specific estimate of 4.7 years (average) based on a study of a community exposed to PFHxS through contaminated drinking water (Li et al. 2018; discussed in NHDES 2019). It is noted that use of this average half-life estimate for women is less conservative than longer average half-life estimates of 8.5 years (Olsen et al., 2007) or 7.4 years (Li et al., 2018) that rely on serum levels in men, or longer estimates of 7.7-35 years for women depending on age (Zhang et al., 2013). However, given the conservative nature and sex-specific effect selected for the POD of PFHxS, the use of a 4.7-year half-life in women was deemed appropriate without being overly-conservative.

Thus, using this chemical-specific DAF and the aforementioned point of departure and uncertainty factors, NHDES derived an oral reference dose for PFHxS of 4.0 ng/kg-d.

$$\text{Reference dose (RfD)} = \frac{13{,}900 \text{ ng/mL}}{300} \times 8.61\times10^{-2} \text{ mL/kg-d} = 4.0 \text{ ng/kg-d}$$

Summary of Recommended RfDs for PFOA, PFOS, PFNA and PFHxS

Recommended RfDs

NHDES recommends the following chronic oral RfDs for PFOA, PFOS, PFNA and PFHxS:

- PFOA, 6.1 ng/kg-d
- PFOS, 3.0 ng/kg-d
- PFNA, 4.3 ng/kg-d
- PFHxS, 4.0 ng/kg-d

These RfDs are for protection from the primary health effects of liver toxicity (PFOA and PFNA), immune suppression of antibody responses (PFOS) and reduced female fertility (PFHxS) based on evidence from animal studies. In addition to these primary health outcomes, these RfDs are expected to be reasonably protective for associated and secondary (less sensitive) health outcomes that occur at similar or higher serum concentrations in rodents. Secondary health effects for these and other PFAS include disruption of thyroid and sex hormone levels and their signaling, teratogenic effects, early-life growth delays, changes in cholesterol levels, neurobehavioral effects, renal toxicity and fertility in rodent models. NHDES believes its selection of PODs, uncertainty factors and DAFs for each RfD provides adequate protection of human health from appreciable risk of these primary and secondary health effects during a lifetime.

Table 2 presents the NHDES recommended RfDs or MRLs, along with their applied uncertainty factors those selected by other agencies that have evaluated these same PFAS. The application of uncertainty factors follows EPA guidance (EPA 2002), and is dependent on the principal study selected and consideration of other available studies. However, it is not uncommon for different risk assessors and toxicologists to arrive at different applications of uncertainty factors when considering where reasonable and health-protective conservatism is being applied in the risk assessment process.

Discussion of scientific uncertainties

While the human health effects of PFAS is a rapidly growing area of scientific research, the exact nature of their associated health effects in humans remains uncertain (ATSDR, 2018b; Michigan Panel, 2018). The cross-sectional nature of most epidemiological studies precludes proof of causality between measured PFAS serum concentrations and the reported associated health outcomes. This is especially problematic as the extraordinarily long half-lives of PFAS (years) make it difficult to disentangle the associated health effects in these studies from co-exposure to other environmental contaminants with relatively shorter half-lives (days to weeks). Additionally, there is a general lack of true control groups for comparison as various combinations of PFAS are detectable in the blood of virtually all populations from around the world. There is concern for the implications of reverse causation with certain health outcomes associated to PFAS. As an evolving area of scientific research, NHDES anticipates new findings will improve the understanding of PFAS-related health effects in humans.

Due to the limitations of epidemiological studies, RfDs were derived using animal data. There are inherent uncertainties associated with RfDs derived from animal studies (EPA 2002), specifically related

to considerations of human health relevance (e.g., biological plausibility) and translation of animal findings to human equivalent values (i.e., uncertainty factors and DAFs).

As a part of its initial proposal (NHDES, 2019), NHDES considered the contentious issue of peroxisome proliferator-activated receptor subtype α (PPARα) activation in rodents and its relevance to human health. The activation of PPARα is a contributing pathway for several of the reported toxic responses in rodent models evidenced by genetic knockout studies and gene expression profiling studies (reviewed by ATSDR 2018b and NHDES 2019). This is especially true for hepatotoxicity and changes in lipid metabolism in rodents following exposure to PFAS due to upregulation of rodent specific pathways leading to oxidative stress (Perkins et al., 2004; Loveless et al., 2006; Rosen et al., 2007, 2008, 2017; Das et al., 2017; reviewed by ATSDR, 2018b). *In vitro* testing demonstrates that PFAS show a stronger binding affinity for rodent PPARα when compared to human PPARα (Wolf et al., 2008). These and other studies reviewed by NHDES (2019) suggest qualitative and quantitative differences in toxicity between species for PPARα-dependent effects.

Such qualitative and quantitative differences raise concern for selection of critical health effects such as liver toxicity based on rodent studies (reviewed by Klaunig et al., 2012), and have been a major criticism of the half-lives derived by NHDES and other agencies for RfDs for PFOA, PFOS, PFNA and PFHxS. Based on existing toxicological information, NHDES contends that selected critical effects from animal studies are appropriate for the protection of human health. While the physiological roles of PPARs (i.e., PPARα, β and γ) in humans are less defined than those of the other nuclear receptors like the estrogen or androgen receptor, there is evidence that they are involved in lipid metabolism (Issemann and Green, 1990; Lee et al., 1995) and function of muscle, adipose and immune cells throughout the body (Tyagi et al., 2011). Independent of PPARα activation, there is evidence for other mechanisms for rodent toxicity (e.g. mitochondrial dysfunction) that are potentially relevant to humans and other organisms (Hagenaars et al., 2013; Cui et al., 2015; reviewed by Li et al., 2017; Li et al., 2018; NHDES, 2019). Furthermore, evidence from non-human primates further suggest that effects on the liver, cholesterol levels, thyroid hormones and the immune system are relevant to humans and not isolated to rodent studies (Griffith and Long 1980; Thomford 2001; Butenhoff et al., 2002; Seacat et al., 2002). Taken collectively, this supports the NHDES risk assessment and derivation of RfDs using the selected critical health effects.

With respect to uncertainty factors, NHDES received multiple comments regarding its application of uncertainty factors in the initially proposed MCLs (NHDES, 2019). Table 2 presents the uncertainty factors used by other state or federal agencies for the derivation of RfDs for PFOA, PFOS, PFNA or PFHxS, and demonstrates that NHDES's selections are within the norms of the professional practice. As previously explained for each compound, NHDES considered available information from human and animal studies to arrive at the total uncertainty factors applied for each RfD. Difference in principal study selection and consideration of available data results in differences in the selection and application of total uncertainty factors (EPA 2002). Given the selection of principal studies and considerations of exposure assumptions described in Section IV, NHDES remains confident that its application of uncertainty factors is appropriate without being overly conservative.

Table 2. Interagency Differences in Uncertainty Factors. Summary of uncertainty factor allocations, RfDs and MRLs by government risk assessment groups.

Specific Uncertainty Factors	ATSDR[a] (MRLs)	US EPA[b,c] (RfD)	TX CEQ[d] (RfD)	MN DOH[e-g] (RfD)	NJ DWQI[h-j] (RfD)	NH DES (RfD)	NY DOH[k] (RfD)
PFOA							
Principal Study	Koskela et al. 2016	Lau et al. 2006	Macon et al. 2011	Lau et al. 2006	Loveless et al. 2006	Loveless et al. 2006	Macon et al. 2011
Human Variability	10	10	10	10	10	10	10
Interspecies Differences	3	3	1	3	3	3	3
Duration of Exposure	1	1	1	1	1	1	1
LOAEL to NOAEL	10	10	30	1	1	1	1
Database Insufficiency	1	1	1	3	10	3	3
Total Uncertainty Factor	300	300	300	100	300	100	100
RfD (ng/kg-d)	3.0	20.0	12.0	18.0	2.0	6.1	1.5
PFOS							
Principal Study	Luebker et al. 2005	Luebker et al. 2005	Zeng et al. 2011	Dong et al. 2011	Dong et al. 2009	Dong et al. 2011	Dong et al. 2009
Human Variability	10	10	10	10	10	10	10
Interspecies Differences	3	3	1	3	3	3	3
Duration of Exposure	1	1	1	1	1	1	1
LOAEL to NOAEL	1	1	10	1	1	1	1
Database Insufficiency	10	10	1	3	1	3	1
Total Uncertainty Factor	300	300	100	100	30	100	30
RfD (ng/kg-d)	2.0	20.0	23.0	3.0	1.8	3.0	1.8
PFNA							
Principal Study	Das et al. 2015	n.a.	Fang et al. 2010	n.a.	Das et al. 2015	Das et al. 2015	n.a.
Human Variability	10	-	10	-	10	10	-
Interspecies Differences	3	-	1	-	3	3	-
Duration of Exposure	1	-	10	-	10	1	-
LOAEL to NOAEL	1	-	1	-	1	1	-
Database Insufficiency	10	-	10	-	3	3	-
Total Uncertainty Factor	300	-	1,000	-	1,000	100	-
RfD (ng/kg-d)	3.0		12.0		0.73	4.3	
PFHxS							
Principal Study	Butenhoff et al. 2009	n.a.	Hoberman & York 2003	Unpublished NTP data	n.a.	Chang et al. 2018	n.a.
Human Variability	10	-	10	10	-	10	-
Interspecies Differences	3	-	1	3	-	3	-
Duration of Exposure	1	-	1	1	-	3	-
LOAEL to NOAEL	1	-	3	1	-	1	-
Database Insufficiency	10	-	10	10	-	3	-
Total Uncertainty Factor	300	-	300	300	-	300	-
RfD (ng/kg-d)	20.0		3.8	9.7		4.0	

n.a. indicates the specific compound was not assessed or reported on by the specific agency.

[a] ATSDR, 2018b. Draft Toxicological Profile for Perfluoroalkyls

[b] U.S. EPA, 2016a. Health Effects Support Document for Perfluorooctanic Acid (PFOA)

[c] U.S. EPA, 2016b. Health Effects Support Document for Perfluorooctane Sulfonate (PFOS)

[d] TX Commission on Environmental Quality (TXCEQ), 2016. Perfluoro Compounds (PFCs): available at: https://www.tceq.texas.gov/assets/public/implementation/tox/evaluations/pfcs.pdf

[e] Minnesota Department of Health (MDH), 2018. Toxicological Summary for: Perfluorooctanoate.

[f] Minnesota Department of Health (MDH), 2019a. Toxicological Summary for: Perfluorooctane sulfonate.

[g] Minnesota Department of Health (MDH), 2019b. Toxicological Summary for: Perfluorohexane sulfonate.

[h] New Jersey Drinking Water Quality Institute (NJDWQI), 2017. Appendix A: Health-Based Maximum Contaminant Level Support Document: Perfluorooctanoic Acid (PFOA)

[i] New Jersey Drinking Water Quality Institute (NJDWQI), 2018a. Appendix A: Health-Based Maximum Contaminant Level Support Document: Perfluorooctane Sulfonate (PFOS)

[j] New Jersey Drinking Water Quality Institute (NJDWQI), 2018b. Appendix A: Health-Based Maximum Contaminant Level Support Document: Perfluorononanoic Acid (PFNA)

[k] New York Department of Health (NYDOH), 2018 and personal communications. Presentation available at: https://www.health.ny.gov/environmental/water/drinking/dwqc/

Section IV. Drinking Water Exposure Assumptions, Modeling and Resulting MCLs

Using the reference dose (RfD) derived in Section III, the MCL considers the estimated daily intake of water from a specific source and how much drinking water contributes to the total exposure from all other sources of a specified contaminant. Specific methodologies for deriving health protective water criteria are detailed by the EPA (USEPA 1989, 2004, 2017, 2018). Although NHDES chose a different approach, the conventional method for deriving drinking water values utilizes the following equation:

$$\text{Maximum contaminant level (ng/L)} = \frac{\text{Reference dose (ng/kg-d)}}{\text{Daily water ingestion rate (L/kg-d)}} \times \text{Relative source contribution (unitless)}$$

For a simple example, a drinking water value for PFOA using the currently recommended RfD, 95th percentile ingestion rate of lactating women and a relative source contribution of 0.5 (meaning 50%) is shown below. This approach was used in the initially proposed MCL, but is not being applied following consideration of breastfeeding (Goeden et al., 2019).

$$\text{Example for PFOA (not an actual MCL recommendation by NHDES)} = \frac{6.1 \text{ ng/kg-d}}{0.055 \text{ L/kg-d}} \times 0.5 = 55 \text{ ng/L}$$

The daily water ingestion rate is a body-weight adjusted factor specific to certain age groups, to gender, and to lactation or pregnancy status. In its initial proposal, NHDES selected the water ingestion rate of the 95th percentile of lactating women, an estimated value of 0.055 L/kg-d (EPA, 2011; NHDES, 2019). While lower estimates are more reflective of the central tendencies of the general population, especially non-lactating women, they were deemed inadequately protective for the larger population. The values are selected from the Exposure Factors Handbook (EPA 2011), which was recently updated specifically for these ingestion rates (see Chapter 3 of EPA, 2019). These updated values were used by NHDES.

Instead of applying a fixed daily water ingestion rate that is assumed to be protective across a lifespan, NHDES applied the toxicokinetic model described by Goeden et al. (2019) to consider how changes in water ingestion at a given MCL are predicted to influence internal blood levels of each PFAS. This is due to the prolonged and elevated internal doses (i.e., serum levels) predicted across infancy and childhood resulting from PFAS in breastmilk. NHDES acknowledges that this is a departure from typical methodology for deriving such a standard, but the unique properties of PFAS (i.e., long half-lives) merit its application to be truly protective across all life stages for the chronic health impacts associated with these chemicals.

The relative source contribution (RSC) is an estimate of how much of the typical daily exposure will be allowed to come from drinking water. EPA recommends an RSC floor of 20% of the RfD and a ceiling of 80% of the RfD. The intention of an RSC ceiling of 80% is to ensure that total exposure from all sources does not exceed 100% of the RfD with a margin of safety for potential unknown or underestimated exposures. PFAS are present in a wide variety of environmental media (Moriwaki et al., 2003; Trudel et al., 2008; Haug 2011; Haug et al., 2011; Winkens et al., 2017, 2018) and consumer products (Haug 2011; Carpet and Textile Treatment - Washburn et al., 2005; Winkens et al. 2017; Cosmetics - Kang et al., 2016; Fast Food Packaging – Schaider et al., 2017), with an ever-growing number of potential sources identified (Boronow et al., 2019; Kim et al., 2019; Nakayama et al., 2019). Thus, for the typical person, it is unlikely that drinking water is responsible for 100% of their exposure. However, an exact profile for the proportions of exposure from various sources remains poorly characterized. The latter part of this section details how this was evaluated by NHDES to arrive at a RSC of 50% for PFOA, PFOS, PFNA and PFHxS.

Application of Goeden et al. (2019) for exposure modeling

As a part of the evaluation of published research and technical comments on the initially proposed MCLs (NHDES, 2019), NHDES has adopted the use of the transgenerational toxicokinetic model (detailed in Goeden et al., 2019), for the determination of appropriately protective health-based MCLs. This is a toxicokinetic model that predicts the serum concentration of PFAS due to drinking water exposure and consumption of breastmilk or formula across a lifespan starting at birth (Goeden et al., 2019). It does not predict an effect (health outcome) due to exposure from drinking water, only the blood concentration for an individual in a reasonable maximum exposure (RME) scenario. The tolerable blood concentration in the RME scenario, or threshold, is determined by the chemical-specific RfD and RSC. This Excel-based model is available upon request from the MN Department of Health.

After review of the model and studies on the placental transfer (Fei et al., 2007; Midasch et al., 2007; Monroy et al., 2008; Fromme et al., 2010; Beesoon et al., 2011; Kim et al., 2011; Liu et al., 2011; Needham et al., 2011; Lee et al., 2013; Porpora et al., 2013; Zhang et al., 2013; Kato et al., 2014; Cariou et al., 2015; Manzano-Salgado et al., 2015; Fisher et al., 2016; Yang et al., 2016; Chen et al., 2017; Mamsen et al., 2019) and breastmilk transfer (Karrman et al., 2007; Haug et al., 2011; Kim et al., 2011; Liu et al., 2011; Cariou et al., 2015; Gyllenhammer et al., 2018) of PFOA, PFOS, PFNA and PFHxS, NHDES determined this novel and "fit-for-purpose" tool (Goeden et al., 2019) was necessary to evaluate exposure outcomes from the proposed MCLs. Specifically, the transfer of PFAS into breastmilk combined with the relatively high breastmilk and water ingestion rates of infants results in a prolonged elevation of serum levels throughout childhood. Under RME assumptions, the serum levels are predicted to be drastically higher than background serum levels seen in the general population, which is assumed to be free of widespread PFAS contamination in drinking water. Furthermore, this elevation throughout childhood into late adolescence limits the RSC allotment for exposure to other sources of PFAS in the environment that, to date, are not regulated.

The following subsections describe the inputs selected by NHDES for RME modeling using Goeden et al. (2019). A summary of model inputs, and associated references, used by NHDES for selection of the proposed MCLs are provided in Table 3.

Human half-life and V_d assumptions

Explanations of the selected half-lives for PFOA, PFOS, PFNA and PFHxS are described in the discussions of DAFs in Section III of this report. For PFOA, an average serum-based half-life was selected from Bartell et al. (2010), which was estimated from a sample population of 200 individuals from the Mid-Ohio valley who were exposed to PFOA from their drinking water supply due to contamination from a DuPont facility. NHDES selected the half-life estimates from Li et al. (2018) for PFOS and PFHxS. These serum-derived half-life estimates were determined to be more representative of the general population, and were obtained from a Swedish community (n = 106 participants) exposed to PFAS, namely PFOS and PFHxS, from drinking water contaminated by AFFF use at a nearby airbase (Li et al., 2018). Finally, the half-life estimate for PFNA was selected from Zhang et al. (2013) which reports urine-based values from a Chinese population (n = 86 participants).

Similar to the half-life values, the volume of distribution (V_d) estimates were identical to those selected by NHDES to derive RfDs for PFOA, PFOS, PFNA and PFHxS (Section III, and references therein).

Table 3. Exposure Model Parameters. Summary of parameters utilized in the transgenerational model (Goeden et al., 2019) by NHDES for derivation of proposed MCLs.

Model Parameter	Central or Upper Tendency of Parameter	PFOA	PFOS	PFHxS	PFNA
Half-Life, years (yrs)	Central	2.3 [a]	3.4 [b]	4.7 [b]	4.3 [c]
Placental Transfer Ratio	Central	0.72 [d]	0.40 [d]	0.70 [d]	0.69 [e]
Breastmilk Transfer Ratio	Central	0.050 [d]	0.017 [d]	0.014 [d]	0.032 [e]
Volume of Distribution (V_d), L/kg	Central	0.170 [f]	0.230 [f]	0.213 [g]	0.200 [e,h]
Relative Source Contribution (RSC), %	Central	50	50	50	50
		Same for All 4 PFAS Exposure Scenario Models			
Duration of Exclusive Breastfeeding, months	Upper	12			
Water Ingestion Rates, mL/kg-d [i] (EPA Exposure Factors Handbook, 2019 Update)					
Birth to <1 mon	Upper	224			
1 to <3 mons	Upper	267			
3 to <6 mons	Upper	158			
6 to <11 mons	Upper	133			
1 to <2 yrs	Upper	57			
2 to <3 yrs	Upper	67			
3 to <6 yrs	Upper	45			
6 to <11 yrs	Upper	41			
11 to <16 yrs	Upper	31			
16 to <18 yrs	Upper	31			
18 to <21 yrs	Upper	31			
21+ yrs	Upper	44			
Lactating Woman	Upper	47			
Breastmilk Ingestion Rates, mL/kg-d (EPA Exposure Factors Handbook, 2011)					
Birth to <1 mon	Upper	220			
1 to <3 mons	Upper	190			
3 to <6 mons	Upper	150			
6 to <12 mons	Upper	130			

[a] Bartell et al., 2010; [b] Li et al., 2018; [c] Zhang et al., 2013; [d] MDH, 2018, 2019ab
[e] MIDHHS, 2019; [f] Thompson et al., 2010; [g] Sundström et al., 2012; Ali et al., *in review*
[h] ATSDR, 2018b;
[i] Body weight and age-specific adjustments to the V_d were maintained the same as described in Goeden et al., 2019.

Placental & breastmilk transfer ratios

NHDES applied previously selected placental and breastmilk transfer ratios for PFOA (MDH 2018), PFOS (MDH 2019), PFNA (MIDHHS 2019) and PFHxS (MDH 2019). In line with the MDH and MIDHHS, NHDES opted to use central tendency values for each PFAS versus the upper or 95th percentile estimate for transfer in the RME scenarios (Table 3).

The exact quantitative nature of PFAS transfer across the placenta remains an active area of research. For example, Mamsen et al. (2019) demonstrated that the accumulation of PFAS in fetal tissues begins early in pregnancy and continues throughout gestation as specific PFAS are taken up by the forming organs with slightly different efficiencies. Several studies of cord blood compared to maternal serum levels of PFAS have been used to estimate placental transfer ratios and are used in the model to predict the "at birth" serum level (Fei et al., 2007; Midasch et al., 2007; Monroy et al., 2008; Fromme et al., 2010; Beesoon et al., 2011; Kim et al., 2011; Liu et al., 2011; Needham et al., 2011; Lee et al., 2013; Porpora et al., 2013; Kato et al., 2014; Cariou et al., 2015; Manzano-Salgado et al., 2015; Fisher et al., 2016; Yang et al., 2016; Chen et al., 2017; Mamsen et al., 2019). The average maternal-to-cord blood or placenta ratios ranged from 0.20 (Mamsen et al., 2019) to 1.24 (Midasch et al., 2007) for PFOA, 0.14 (Fisher et al., 2014) to 0.60 (Midasch et al., 2007) for PFOS, 0.24 (Mamsen et al., 2019) to 1.18 (Monroy et al., 2008) for PFNA, and 0.23 (Fisher et al., 2016) to 1.25 (Monroy et al., 2008) for PFHxS. A point of caution in interpreting placental transfer ratios in these studies is the trimester of pregnancy that data are collected. Changes in blood volume over the coruse of pregnancy are expected to affect the maternal blood concentration, thereby influences cord blood to maternal blood concentration ratios for various PFAS. Collectively, these studies provide valuable and reliable information for estimating the transfer from mother to newborn. This model does not predict fetal blood or tissue concentrations of PFAS as this compartmentalization is poorly understood, although recent work, such as Mamsen et al. (2019) may lead to the development of such models.

Compared to placental transfer efficiencies that are well-documented for PFAS, a small body of literature informs our understanding of the PFAS in breastmilk. As a part of its review of the technical documents described by MDH (2018, 2019ab) and MIDHHS (2019), NHDES reviewed the source papers for the breastmilk transfer ratios (Karrman et al., 2007; Haug et al., 2011; Kim et al., 2011; Liu et al., 2011; Cariou et al., 2015; Gyllenhammer et al., 2018). These studies demonstrate that the small average percentage (0.6-11% across various PFAS) transferred from a mother's serum, which is typically at concentrations of ng/mL or ppb, results in breastmilk at concentration ranges well above most existing drinking water advisories. Combined with relatively high ingestion rates of breastmilk relative to the infant's body weight, this results in a spike of infant blood concentrations that the model predicts will remain high through childhood.

Duration of breastfeeding

A major assumption for the breastfeeding component of this model is the duration of exclusive breastfeeding. Consistent with the RME scenarios selected by other states (MDH, 2018, 2019ab; MIDHHS, 2019), NHDES used a 12-month duration of *exclusive breastfeeding* for all four RME scenarios. Similar to the CDC, the World Health Organization (WHO) defines exclusive breastfeeding as:

> "Exclusive breastfeeding means that the infant receives only breast milk. No other liquids or solids are given – not even water – with the exception of oral rehydration solution, or drops/syrups of vitamins, minerals or medicines." – WHO eLENA (2019)

A central tendency assumption for the duration of exclusive breastfeeding would be 6 months, but NHDES selected a more conservative modeling parameter of 12 months of exclusive breastfeeding. A 12-month exclusive breastfeeding duration is a conservative assumption because the CDC recommends 6 months of exclusive breastfeeding and some continuation through infancy given the clear benefits to an infant's health and their long-term development. After 6 months of age, the recommendation is that other food items are introduced and breastfeeding continues for up to 2 years of age.

This assumption has been argued by some to be overly conservative relative to the RME scenarios as 1) CDC recommended exclusive breastfeeding for up to 6 months of age and 2) if an infant were exclusively breastfeeding at or after 12 months of age, it is unlikely they are not ingesting other fluids or foods. NHDES contends that this is a reasonable assumption given 1) the role that the duration of exclusive breastfeeding plays in the MN model and 2) the high rates of breastfeeding in New Hampshire and breastfeeding trends across the nation.

MDH notes that the duration of breastfeeding, along with breastmilk intake rates and water concentration, are the most sensitive parameters of the model (MDH 2017). The duration of exclusive breastfeeding and breastfeeding with complimentary foods varies, but the CDC recommends up to 2 years of breastfeeding with the addition of complimentary foods. The transgenerational model does not contain parameters for apportionment of exposure from breastmilk versus complimentary foods, or formula, across the first two years of life. Given this uncertainty for mixed exposures for breastfed infants, NHDES agreed that the assumption of a 12-month exclusive breastfeeding duration was appropriate for estimate for the purpose of the model.

Results from the National Immunization Survey (NIS) indicate that, in the general U.S. population of newborns, approximately 24.9% ± 1.2 (± half 95% CI) of infants are exclusively breastfed at 6 months of age. By 12 months, 35.9% ± 1.3 of infants consume breastmilk along with complimentary foods and liquids (CDC, 2018a). New Hampshire specific estimates from this same dataset are that 30.2% ± 5.8 of infants exclusively breastfeed at 6 months of age, while 45.6% ± 6.5 breastfeed at 12 months of age in addition to complimentary foods (CDC, 2018a). Based on the historical trends, the 2018 Breastfeeding Report Card (CDC, 2018b) indicates more women nationwide are breastfeeding or want to breastfeed their children, giving weight to the consideration of breastfeeding and selecting a conservative window of 12 months.

Breastmilk and drinking water ingestion rate assumptions

This transgenerational model evaluates the impact of changing water ingestion rates across a lifespan. These ingestion rates are expressed as liters of water per kilogram of an individual's body weight per day (L/kg-d). As a person grows, their physiological demand for water changes and this is reflected by age-specific ingestion rates, or life-process specific rates in the case of pregnant and lactating women. To put this in context of historical practice, the EPA typically assumed a drinking water ingestion rate of 2 L/d

for adults and 1 L/d for infants and children under 10 years of age (U.S. EPA, 2000). After adjusting for body weight, these typical rates would underestimate the water consumption of infants, children and lactating and pregnant women. Thus, consideration of these life-stage specific values is prudent for a persistent and highly-bioaccumulative class of drinking water contaminants.

To be protective of the general population including high-end water consumers, NHDES applied the 95th percentile water and breastmilk ingestion rates throughout life in the RME scenarios for PFOA, PFOS, PFHxS and PFNA. The use of the 95th percentile for water ingestion rates is consistent with the initial proposal, and this is simply an extension to other life stages. Recently updated values in 2019 Updated Chapter 3 of the Exposure Factors Handbook (EPA, 2019) were combined with estimated breastmilk ingestion rates from Chapter 15 of the 2011 Edition (EPA, 2011). As these changes were specific to water ingestion, not breastmilk, the difference between the 2011 and 2019 estimates for infants, a change of -9% to +3% for those <1 year of age, was determined to be a minor and tolerable change to the RME scenarios. The breastfed RME exposure was the driver of the MCL for all evaluated PFAS, and therefore protective of an individual in the formula-fed RME scenario.

Consideration of the Relative Source Contribution (RSC)

Exposure to PFAS is not solely due to drinking water, so in order for the MCL to be health protective NHDES needs to account for the contribution of other sources towards the reference dose (RfD). The proportion of exposure attributed to a specific source is accounted for through the relative source contribution (RSC). With respect to a MCL, the RSC is the percentage of total exposure typically accounted for by drinking water (EPA 2000). This value can be referred to as a proportion or percentage, and EPA recommends a ceiling of 80% and a floor of 20%. A smaller RSC for drinking water exposure results in a lower regulatory standard, but implies that sources other than water contribute more significantly to exposure.

Presently, there is no inventory of all relevant sources of PFAS exposure to determine what proportion each source shares in an RSC for the general population. Several studies have characterized specific media such as dust, food (Kowalczyk et al., 2013; reviewed by EFSA, 2018) and breastmilk (previously discussed) and estimated the percentages of total exposure attributable to these sources; but no single study has merged these findings to estimate the reasonable and realistic RSC for drinking water.

In the absence of such data, the EPA provides a decision tree for identifying an appropriate RSC (replicated in Figure 1; EPA 2000). Following this process, NHDES determined:

- (Box 6 to 8a) *Yes, there are significant known sources of these PFAS other than drinking water*. As a result of their dispersion into the environment and lack of adequate removal from waste streams, there are known sources of PFAS that contribute to environmental exposures. This includes release into surface water and implications for fish and shellfish consumption (Fair et al., 2019), and the impacts of PFAS contamination of soil (Filipovic et al., 2015; Scher et al., 2018), dust (Fu et al., 2015; Winkens et al., 2018) and agriculture-related exposures (Nascimento et al., 2018; reviewed by Ghisi et al., 2019).

- (Box 8a to 8c) *Yes, there is some information to make a characterization of exposure*. As mentioned above, there is some data on environmental sources to make rough characterizations. Additionally, there is blood data from the National Health and Nutrition Examination Survey (NHANES) to estimate the general exposure of the U.S. population to PFAS. The NHANES data for blood levels of PFAS is assumed to reflect general exposure to all sources in the U.S. population, and is presumed to not reflect the results of excessively high exposures, relative to the proposed MCLs, due to contaminated drinking water as seen in the communities of Southern New Hampshire Pease Tradeport and Southern New Hampshire.
- (Box 8c to 13) *NHDES performed apportionment with a 50% ceiling and 20% floor for each of the assessed PFAS*. This apportionment was achieved using the EPA subtraction method (EPA 2000).

The subtraction method (EPA 2000) estimates an apportionment of the RSC is based on assumed knowledge of the background exposure. For PFAS, the subtraction method has been mathematically applied as follows (NJDWQI 2018; MDH 2018, 2019ab):

$$\text{Relative Source Contribution} = \frac{\text{Target serum level } \left(\frac{\text{ng}}{\text{mL}}\right) - \text{Reference or background population level } \left(\frac{\text{ng}}{\text{mL}}\right)}{\text{Target serum level } \left(\frac{\text{ng}}{\text{mL}}\right)} \times 100\%$$

The difference between the target serum level and the RfD is that the former is an internal blood concentration while the latter is the external amount of the chemical that could come from multiple sources. For each of the compounds, the target serum levels were: PFOA – 43.5 ng/mL, PFOS – 23.6 ng/mL, PFNA – 49.0 ng/mL and PFHxS – 46.3 ng/mL. The reference population serum level is meant to reflect a background level of exposure from the general population, not one that is highly exposed due to a specific environmental source such as drinking water. Using the NHANES average serum values, subtracting this background level from the target serum level (the maximum allowable level) results in a proportion that is presumably permissible for drinking water alone. Other sources including food, dust, treated consumer products (e.g., carpeting, cookware, food packaging, etc.) are assumed to be included in the reference or background population blood concentrations.

Using this approach with the NHANES 2013-2014 data for children ranging in age from 3 to 19 years (as reported in Daly et al., 2018), NHDES arrived at RSCs of 50% for PFOA, PFOS, PFNA and PFHxS. Unlike its initial proposal, NHDES selected the NHANES dataset over the use of NH-specific estimates. The NH-specific blood data was focused on communities whose primary exposure was associated with drinking water, and would therefore overestimate non-drinking water exposure sources if used to establish an RSC as initially proposed in January (NHDES, 2019). Thus, the NHANES dataset was deemed more appropriate to account for other non-drinking water sources of exposure. For an understanding of how the NHANES data compares to that collected from one of the highly-exposed communities in New Hampshire and the limitations of interpreting these findings, readers are referred to Daly et al. (2018).

Instead of using the general population (i.e., all ages), NHDES estimated RSCs based on the serum concentrations from those younger than 19 years of age (Table 4). As emphasized in several comments made to NHDES on its initial proposal, the risk assessment needs to consider current information for children. Since the phase out of certain PFAS, but not all, the national average serum levels have declined suggesting some reduction of background exposure. Given the emphasis of the RME on infancy

and early childhood, NHDES determined it was appropriate to derive the RSC with specific consideration of this group. All of the values for PFOA, PFOS, PFNA and PFHxS were at or above 48.3%, therefore NHDES opted for an RSC of 50%.

NHDES acknowledges that the use of the general NHANES estimates that includes adults with historically high exposures results in similar or more restrictive RSC values; especially for PFOS. However, the RME scenarios for the proposed MCLs indicate that the predicted serum level for the 95th percentile of adult water consumers is approximately equal to or below the 20% RSC and therefore sufficiently protective after considering the context of the national dataset. Furthermore, the cap of 50% despite calculated higher RSCs for each of these accounts for the unknown and novel sources of PFAS exposure, as well as the higher serum levels of PFAS found in New Hampshire's highly-exposed communities.

Table 4. Relative Source Contribution Estimates. Various relative source contribution (RSC) values resulting from use of the EPA subtraction method (EPA 2002) in combination with available serum data for the geometric mean (GM) and 95th percentile from the NHANES 2013-2014 dataset, as reported in Daly et al. (2018).

Reference Population	Reference Serum level (ng/mL)	Target Serum Level (ng/mL)	Resulting RSC Allotment for Drinking Water (%)
PFOA			
3-5 year olds (GM)	2.00	43.5	95.4
6-11 year olds (GM)	1.89	43.5	95.7
12-19 year olds (GM)	1.66	43.5	96.2
3-5 year olds (95th percentile)	5.58	43.5	87.2
6-11 year olds (95th percentile)	3.84	43.5	91.2
12-19 year olds (95th percentile)	3.47	43.5	92.0
PFOS			
3-5 year olds (GM)	3.38	24.0	85.9
6-11 year olds (GM)	4.15	24.0	82.7
12-19 year olds (GM)	3.54	24.0	85.3
3-5 year olds (95th percentile)	8.82	24.0	63.3
6-11 year olds (95th percentile)	12.40	24.0	48.3
12-19 year olds (95th percentile)	9.30	24.0	61.3
PFNA			
3-5 year olds (GM)	0.76	49.0	98.4
6-11 year olds (GM)	0.81	49.0	98.3
12-19 year olds (GM)	0.60	49.0	98.8
3-5 year olds (95th percentile)	3.49	49.0	92.9
6-11 year olds (95th percentile)	3.19	49.0	93.5
12-19 year olds (95th percentile)	2.00	49.0	95.9
PFHxS			
3-5 year olds (GM)	0.72	46.3	98.4
6-11 year olds (GM)	0.91	46.3	98.0
12-19 year olds (GM)	1.27	46.3	97.3
3-5 year olds (95th percentile)	1.62	46.3	96.5
6-11 year olds (95th percentile)	4.14	46.3	91.1
12-19 year olds (95th percentile)	6.30	46.3	86.4

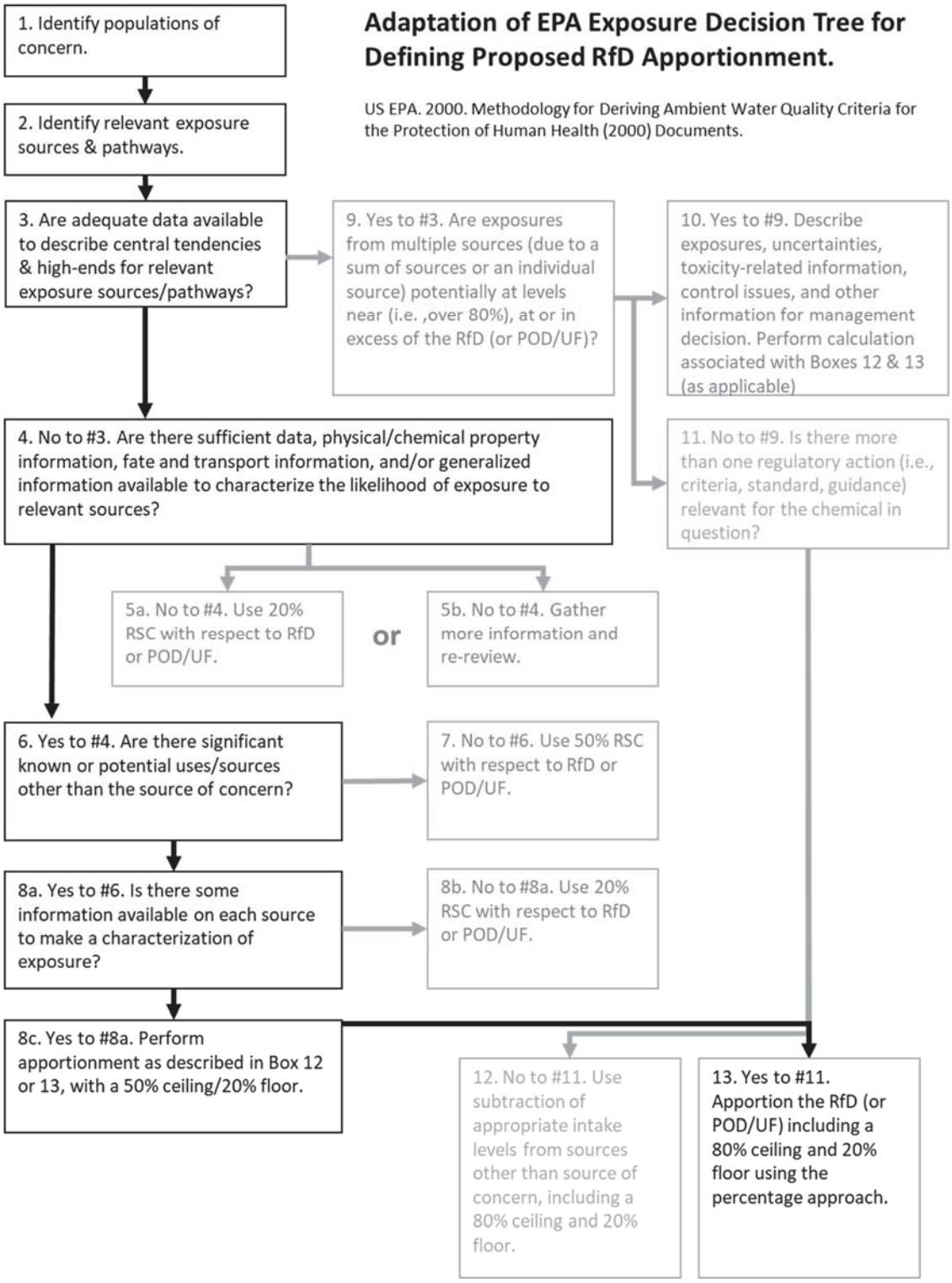


Figure 1. Adaptation of EPA decision tree (EPA, 2000) for determining the RSC. Black boxes, text and arrows outline the decision process used by NHDES to arrive at the subtraction method for PFAS with a 50% ceiling. The target serum level is a population assessment value, *not clinical*, from the derivation of the RfDs, detailed in Section III.

Section V. Discussion of the MCLs proposed by NHDES

Based on the previously described RfDs, exposure considerations and application of the transgenerational model (Figure 2), the proposed maximum contaminant levels (MCLs) are:

- **12 ng/L for Perfluorooctanoic acid, or perfluorooctanoate (PFOA)**
- **15 ng/L for Perfluorooctane sulfonic acid, or perfluorooctane sulfonate (PFOS)**
- **11 ng/L for Perfluorononanoic acid, or perfluorononanoate (PFNA)**
- **18 ng/L for Perfluorohexane sulfonic acid, or perfluorohexane sulfonate (PFHxS)**

These health-based values are intended as health-protective limits against the chronic health effects for a through-life exposure. The primary associated health outcomes are hepatotoxicity and changes in lipid metabolism (PFOA and PFNA), suppressed immune response to vaccines (PFOS) and impaired female fertility (PFHxS). Secondary associated health effects that are expected to be less sensitive are changes in thyroid and sex hormone levels, early-life growth delays, changes in cholesterol levels and biomarkers of liver function, neurobehavioral effects, and a possible risk for certain cancers (i.e., testicular and kidney).

Modeled Exposure Results

Figure 2 shows the model result for predicted serum concentrations at the proposed MCL for each PFAS. The exposure starts at birth with the assumption that the mother is at a steady-state serum level from consumption of water at the modeled drinking water concentration. The solid blue line represents the highest exposure in the RME model, showing the predicted serum level for a breastfed infant who consumes breastmilk and water at the 95th percentile ingestion rates throughout life and is born to and breastfeeds from a mother with a similar water consumption rate. The solid green line represents the predicted serum level for a formula-fed infant who consumes formula (reconstituted with water at the MCL) and water at the 95th percentile ingestion rates throughout life and is born to a mother with a similar water consumption rate. The dashed lines represent the predicted serum concentrations for individuals at the central tendency or average breastmilk, formula and water ingestion rates.

There is a clear spike in predicted serum levels of breastfed infants due to the aforementioned transfer efficiencies of PFAS into breastmilk. For infants, this is concerning due to the potential for hand-to-mouth behaviors in later infancy that have been shown to contribute to PFAS exposure in children of this age (Trudel et al., 2008). Because of these potential exposures and the suspected health impacts on early development, NHDES selected an MCL value that does not allow the predicted infant serum level to exceed the 50% RSC of the RfD or target serum level. It is true that the central tendency consumers fall well below this threshold. However, it has been shown that when considering variants on the RME scenarios the use of the 95th percentile ingestion rate is adequately protective for other factors (e.g., higher breastmilk transfer efficiencies or longer half-life estimates) (Goeden et al., 2019).

The long half-lives of these compounds result in significantly elevated serum levels peaking at the cessation of breastfeeding and continuing through the remainder of childhood. While the predicted steady-state concentrations for adults or formula-fed infants would allow less restrictive MCLs, breastfed children could potentially exceed the RfD due to other sources such as dust (Winkens et al., 2018) or foods and food packaging (D'eon et al., 2009; reviewed by EFSA, 2018). This point further emphasizes the appropriateness of the 50% cap on the RSC as selected by NHDES.

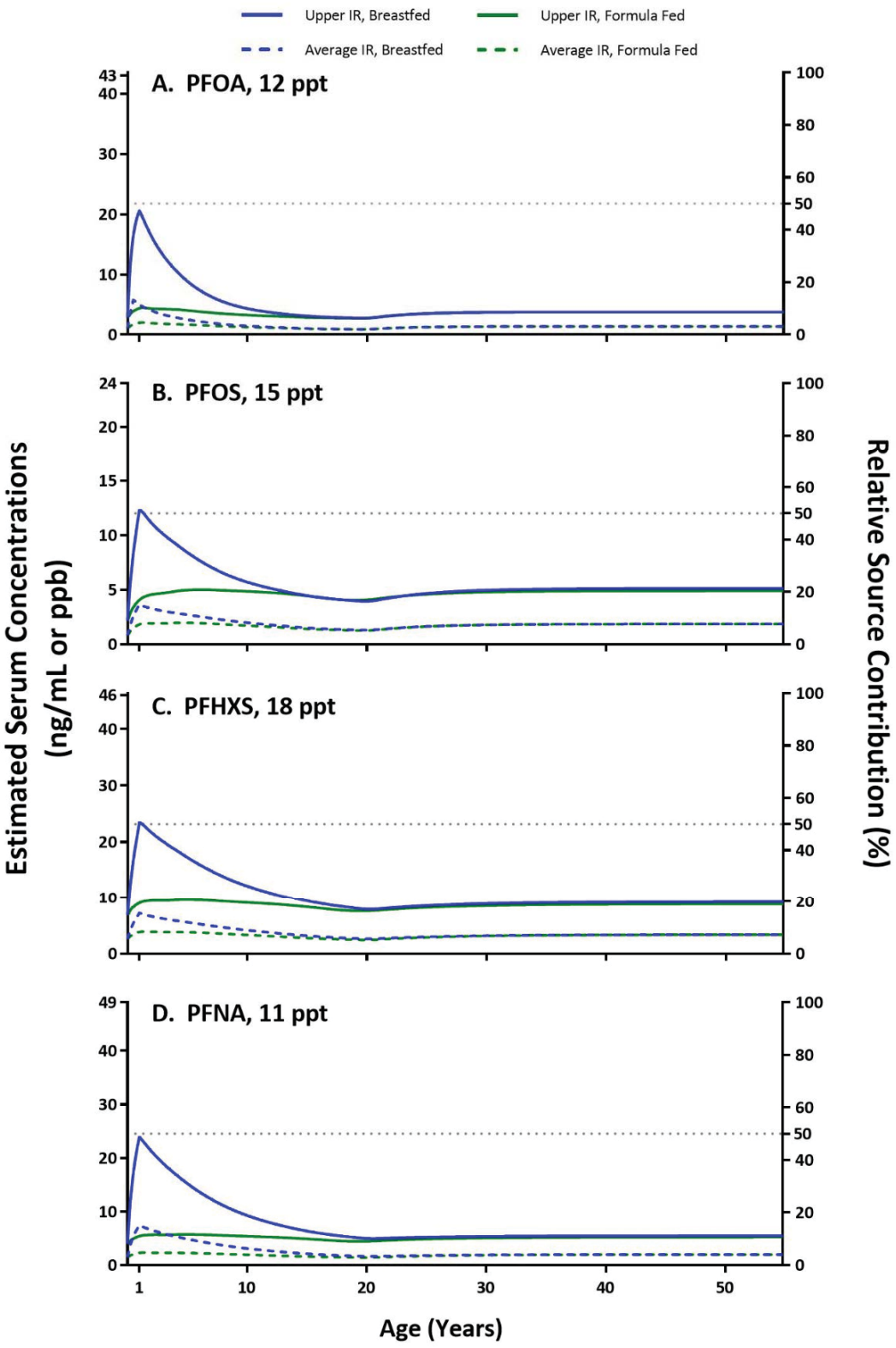


Figure 2. Predicted serum PFAS concentrations in response to upper (95th percentile) and average (mean) water ingestion rates (IR) at the proposed MCLs. Blue lines indicate results for breastfed infants with 12 months exclusive breastfeeding, and green lines indicate results for formula-fed infants. Solid lines represent upper IRs and dashed lines indicate average (mean) IRs. Estimates made using the model described in Goeden et al. (2019).

Using the proposed MCL values for each compound, serum concentrations attributable to drinking water can be estimated for an individual across various life stages (adapted from Figure 2). For newborns (at birth), the estimated drinking water contribution to serum concentrations for the 95th percentile consumer would be: 2.9 ng/mL for PFOA, 2.2 ng/mL for PFOS, 4.0 ng/mL for PFNA and 6.9 ng/mL for PFHxS. The model does not predict fetal tissue concentrations, so the predicted at-birth values represent the aforementioned placental transfer efficiencies. The predicted drinking water contribution to serum concentrations for the 95th percentile breastmilk consumer (at the end of 1 year of exclusive breastfeeding) would be: 20.6 ng/mL for PFOA, 12.4 ng/mL for PFOS, 25.1 ng/mL for PFNA and 23.5 ng/mL for PFHxS. Adults at steady state following constant water consumption at the 95th percentile are predicted to have drinking water contributions of PFAS equal to or less than: 3.8 ng/mL for PFOA, 5.1 ng/mL for PFOS, 5.7 ng/mL for PFNA and 9.2 ng/mL for PFHxS.

As a point of caution in interpretation, the previously described results assume no fluctuation from the 95th percentile drinking water consumption rate across an individual lifespan. That is to say, the 95th percentile consumer remains the 95th percentile consumer every day. These estimates include several conservative and protective assumptions, such as the use of the 95th percentile of drinking water ingestion rates (adjusted for body weight) throughout life, not the average water consumer or fluctuations between these tendencies. Additionally, the modeled outputs may not reflect individual variations in biology throughout life (Fàbrega et al., 2014; Worley et al., 2017) and are intended for population-level exposure assessment. However, as described by Goeden et al. (2019), this fit-for-purpose tool provides important insight into exposures during critical life stages of development. Further development and refinement of multi-compartment models will certainly prove useful for future risk assessments of these and other PFAS.

The proposed MCLs are predicted to result in a modest increase of serum concentrations due to drinking water levels; but, as argued by Post et al. (2017), such increases relative to background are preferred over the significantly larger serum levels that are predicted for the previously proposed MCLs (NHDES, 2019) or the EPA lifetime health advisories (EPA, 2016ab). Based on current evidence, this level of exposure is expected to be sufficiently health protective relative to current background levels reported in populations of concern, such as children and adolescents (Table 4).

Limitations and uncertainties

As with any risk assessment, this process was subject to uncertainty and limitations. Limitations included recommendation of individual versus group-based MCLs for PFAS, and consideration of background exposure using the RME scenarios described in Section IV. A major uncertainty was quantifying the exact risks of disease incidence for each compound, which is also a significant challenge for quantifying, or monetizing, the benefits of the proposed MCLs.

A limitation to the present assessment is that the transgenerational model's RME scenarios focus on the predicted impact of drinking water exposure, not other background sources of exposure. In general, there is a downward trend for the background levels of most measured PFAS based on the NHANES data. NHDES considered this with its use of the NHANES data to derive and apply a 50% RSC for each compound. Although PFOA and PFOS were recently phased out by most U.S. manufacturers, there remains potential for exposure to these and other PFAS from imported products or the degradation of

precursors into PFOA or PFOS in the environment. Nevertheless, the appropriate level of conservatism applied in the assumptions of drinking water ingestion rates and RSC provide reasonable protection.

At this time, NHDES is not recommending a class-based approach to regulation of these compounds. This is a limitation of the present risk assessment given the considerable number of PFAS detected in the environment and used in commerce. However, individual assessment of each compound found each one to have relatively unique toxico-dynamic and –kinetic properties based on consideration of existing animal toxicity and human data. Despite similarity in the range of the proposed MCLs for these 4 PFAS, it is likely that future individual assessments, using current EPA methodology, of shorter carbon chain PFAS will result in higher drinking water values for shorter carbon chain compounds as a result of shorter half-lives. Given these considerations, it was determined that a class based approach was not advisable at this time. Should other state agencies or the U.S. EPA identify science-based methods for group regulation that account for some of the unique properties of these compounds, NHDES will consider this approach.

Currently, there is uncertainty to quantifying the health risks associated with exposure to PFOA, PFOS, PFNA, PFHxS and other PFAS. A growing number of epidemiological and animal toxicity studies are adding to the body of evidence for the biological activity and health outcomes associated with these contaminants. However, the exact nature of PFAS-related health hazards remains elusive due to a variety of factors including, but not limited to: a limited understanding of the toxicological mechanism of action, their occurrence world-wide and lack of control (i.e., PFAS-free) populations to compare health outcomes against, lack of long-term studies despite decades of use, and co-exposure with other PFAS and other environmental contaminants. Additional research is critically needed to address this issue and better characterize and quantify the risks associated with PFAS.

Conclusions

The lower MCLs proposed in this report are primarily due to consideration of the elevated serum levels predicted for infants and young children under a reasonable maximum exposure scenario. At the initially proposed values, these spikes in infant blood levels of PFAS would result in unacceptable reductions in the margin of exposure from infancy through childhood due to the unique properties of PFAS. Their capacity to transfer through breastmilk combined with relatively long half-lives of each compound merits the use of novel methods (i.e., Goeden et al., 2019) to provide a more accurate assessment of exposure. This is not a recommendation against breastfeeding for women who are currently breastfeeding or plan to breastfeed as the benefits of breastfeeding are very well-defined relative to the potential risk associated with PFAS. NHDES recommends these MCLs to afford adequate long-term health protection of the population based on its assessment of these four PFAS.

The human health impacts of PFAS is a continuously evolving area of scientific research, and is expected to continue changing in the future. The assessments made by NHDES are based on currently available information but recognizes that science is a process, not an outcome. Future assessments of these and other PFAS compounds may result in higher or lower health protective values based on the best available science at the time. NHDES will continue to review emerging information as a part of its ongoing efforts to understand the impacts of PFAS contamination across New Hampshire.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2018a. Toxic Substances Portal: Minimal Risk Levels (MRLs) – For Professionals. Updated June 21, 2018. https://www.atsdr.cdc.gov/mrls/index.asp

Agency for Toxic Substances and Disease Registry (ATSDR). 2018b. Toxicological Profile for Perfluoroalkyls – Draft for Public Comment, June 2018. Accessed online at: https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf.

Albrecht PP, Torsell NE, Krishnan P, et al. 2013. A species difference in the peroxisome proliferator-activated receptor α-dependent response to the developmental effects of perfluorooctanoic acid. Toxicol Sci 131(2):568-582.

Ali JM, Roberts SM, Gordon DS, Stuchal LD. (in review) Derivation of a chronic reference dose for perfluorohexane sulfonate (PFHxS) for reproductive toxicity in mice.

Ballesteros V, Costa O, Iñiguez C, Fletcher T, Ballester F, Lopez-Espinosa MJ. 2017. Exposure to perfluoroalkyl substances and thyroid function in pregnant women and children: A systematic review of epidemiologic studies. Environ Int, 99:15-28. doi: 10.1016/j.envint.2016.10.015.

Bartell SM, Calafat AM, Lyu C, et al. 2010. Rate of decline in serum PFOA concentrations after granular activated carbon filtration at two public water systems in Ohio and West Virginia. Environ Health Perspect 118(2):222-228

Barry V, Winquist A, Steenland K. 2013. Perfluorooctanoic acid (PFOA) exposures and incident cancers among adults living near a chemical plant. Environ Health Perspect 121(11-12):1313-1318.

Bassler J, Ducatman A, Elliott M, Wen S, Wahlang B, Barnett J, Cave MC. 2019. Environmental perfluoroalkyl acid exposures are associated with liver disease characterized by apoptosis and altered serum adipocytokines. Environ Pollut. 247:1055-1063. doi: 10.1016/j.envpol.2019.01.064

Beesoon S, Webster GM, Shoeib M, Harner T, Benskin JP, Martin JW. 2011. Isomer profiles of perfluorochemicals in matched maternal, cord, and house dust samples: manufacturing sources and transplacental transfer. Environ Health Perspect. 119(11):1659-64. doi: 10.1289/ehp.1003265.

Bijland S, Rensen PC, Pieterman EJ, et al. 2011. Perfluoroalkyl sulfonates cause alkyl chain length-dependent hepatic steatosis and hypolipidemia mainly by impairing lipoprotein production in APOE*3-Leiden CETP mice. Toxicol Sci 123(1):290-303. 10.1093/toxsci/kfr142.

Boronow KE, et al. 2019. Serum concentrations of PFASs and exposure-related behaviors in African American and non-Hispanic white women. Journal of Exposure Science & Environmental Epidemiology, pp. 1-12.

Butenhoff J, Costa G, Elcombe C, et al. 2002. Toxicity of ammonium perfluorooctanoate in male Cynomolgus monkeys after oral dosing for 6 months. Toxicol Sci 69(1):244-257.

Butenhoff JL, Chang S, Ehresman DJ, et al. 2009a. Evaluation of potential reproductive and developmental toxicity of potassium perfluorohexanesulfonate in Sprague Dawley rats. Reprod Toxicol 27:331-341.

Butenhoff JL, Ehresman DJ, Chang SC, et al. 2009b. Gestational and lactational exposure to potassium perfluorooctanesulfonate (K+PFOS) in rats: Developmental neurotoxicity. Reprod Toxicol 27(3-4):319-330.

Butenhoff JL, et al. 2008. Evaluation of potential reproductive and developmental toxicity of potassium perfluorohexanesulfonate in Sprague Dawley rats. Reproductive Toxicology, 27, 331-341.

Butenhoff, J.L., G.L. Kennedy, Jr., S.-C. Chang, and G.W. Olsen. 2012. Chronic dietary toxicity and carcinogenicity study with ammonium perfluorooctanoate in Sprague-Dawley rats. Toxicology 298:1–13.

Butenhoff, J.L., G.L. Kennedy, S.R. Frame, J.C. O'Conner, and R.G. York. 2004. The reproductive toxicology of ammonium perfluorooctanoate (APFO) in the rat. Toxicology 196:95–116.

California Office of Environmental Health Hazard Assessment. 2019. PFOA and PFOS Notification Levels. https://www.waterboards.ca.gov/drinking_water/certlic/drinkingwater/PFOA_PFOS.html

Cariou R, Veyrand B, Yamada A, et al. 2015. Perfluoroalkyl acid (PFAA) levels and profiles in breast milk, maternal and cord serum of French women and their newborns. Environ Int 84:71-81.

Chang ET, et al. 2016. A critical review of perfluorooctanoate and perfluorooctanesulfonate exposure and immunological health conditions in humans. Crit Rev Toxicol., 46(4): 279-331.

Chang S, et al. 2018. Reproductive and developmental toxicity of potassium perfluorohexanesulfonate in CD-1 mice. Reproductive Toxicology 78: 150-168.

Chen F, Yin S, Kelly BC, Liu W. 2017. Isomer-Specific Transplacental Transfer of Perfluoroalkyl Acids: Results from a Survey of Paired Maternal, Cord Sera, and Placentas. Environ Sci Technol. 51(10):5756-5763. doi: 10.1021/acs.est.7b00268.

Cheng J, Fujimura M, Zhao W, et al. 2013. Neurobehavioral effects, c-Fos/Jun expression and tissue distribution in rat offspring prenatally co-exposed to MeHg and PFOA: PFOA impairs Hg retention. Chemosphere 91(6):758-764.

Cui L, Zhou QF, Liao CY, et al. 2009. Studies on the toxicological effects of PFOA and PFOS on rats using histological observation and chemical analysis. Arch Environ Contam Toxicol 56(2):338-349.

Cui Y, et al. 2015. Investigation of the Effects of Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS) on Apoptosis and Cell Cycle in a Zebrafish (Danio rerio) Liver Cell Line. Int J Environ Res Public Health. 12(12):15673-82.

Daly ER, Chan BP, Talbot EA, Nassif J, Bean C, Cavallo SJ, Metcalf E, Simone K, Woolf AD. 2018. Per- and polyfluoroalkyl substance (PFAS) exposure assessment in a community exposed to contaminated drinking water, New Hampshire, 2015. Int J Hyg Environ Health. 221(3):569-577. doi: 10.1016/j.ijheh.2018.02.007.

Das KP, Grey BE, Rosen MB, et al. 2015. Developmental toxicity of perfluorononanoic acid in mice. Reprod Toxicol 51:133-144. 10.1016/j.reprotox.2014.12.012.

Das KP, Wood CR, Lin MT, et al. 2017. Perfluoroalkyl acids-induced liver steatosis: Effects on genes controlling lipid homeostasis. Toxicology 378:37-52. 10.1016/j.tox.2016.12.007.

D'eon JC, Crozier PW, Furdui VI, Reiner EJ, Libelo EL, Mabury SA. 2009. Observation of a commercial fluorinated material, the polyfluoroalkyl phosphoric acid diesters, in human sera, wastewater treatment plant sludge, and paper fibers. Environ. Sci. Technol. 43: 4589-4594.

DeWitt JC, Blossom SJ, Schaider LA. 2019. Exposure to per-fluoroalkyl and polyfluoroalkyl substances leads to immunotoxicity: epidemiological and toxicological evidence. J Expo Sci Environ Epidemiol. 29(2):148-156. doi: 10.1038/s41370-018-0097-y

DeWitt JC, Peden-Adams MM, Keller JM, Germolec DR. 2012. Immunotoxicity of Perfluorinated Compounds: Recent Developments. Toxicologic Pathology, 40: 300-311.

Dong GH, Liu MM, Wang D, et al. 2011. Sub-chronic effect of perfluorooctanesulfonate (PFOS) on the balance of type 1 and type 2 cytokine in adult C57BL6 mice. Arch Toxicol 85(10):1235-1244.

Dong GH, Zhang YH, Zheng L, et al. 2009. Chronic effects of perfluorooctanesulfonate exposure on immunotoxicity in adult male C57BL/6 mice. Arch Toxicol 83(9):805-815.

Elcombe CR, Elcombe BM, Foster JR, et al. 2010. Hepatocellular hypertrophy and cell proliferation in Sprague-Dawley rats following dietary exposure to ammonium perfluorooctanoate occurs through increased activation of the xenosensor nuclear receptors PPARα and CAR/PXR. Arch Toxicol 84(10):787-798.

Ernst A, Brix N, Lauridsen LLB, Olsen J, Parner ET, Liew Z, Olsen LH, Ramlau-Hansen CH. 2019. Exposure to Perfluoroalkyl Substances during Fetal Life and Pubertal Development in Boys and Girls from the Danish National Birth Cohort. Environ Health Perspect. 127(1):17004. doi: 10.1289/EHP3567.

European Food Safety Authority (EFSA). 2018. Risk to human health related to the presence of perfluorooctane sulfonic acid and perfluorooctanoic acid in food. EFSA Journal, 16(12):5194

Fàbrega F, Kumar V, Schuhmacher M, Domingo JL, Nadal M. 2014. PBPK modeling for PFOS and PFOA: validation with human experimental data. Toxicol Lett. 230(2):244-51. doi: 10.1016/j.toxlet.2014.01.007.

Fair PA, Wolf B, White ND, Arnott SA, Kannan K, Karthikraj R, Vena JE. 2019. Perfluoroalkyl substances (PFASs) in edible fish species from Charleston Harbor and tributaries, South Carolina, United States: Exposure and risk assessment. Environ Res. 171:266-277. doi: 10.1016/j.envres.2019.01.021

Fang X, Fenga Y, Wang J, et al. 2010. Perfluorononanoic acid-induced apoptosis in rat spleen involves oxidative stress and the activation of caspase-independent death pathway. Toxicology 267: 54-59

Fei C, McLaughlin JK, Tarone RE, et al. 2007. Perfluorinated chemicals and fetal growth: A study within the Danish National Birth Cohort. Environ Health Perspect 115:1677-1682.

Filipovic M., Woldegiorgis A., Norström K., Bibi M., Lindberg M., Österås A.H. Historical usage of aqueous film forming foam: A case study of the widespread distribution of perfluoroalkyl acids from a military airport to groundwater, lakes, soils and fish. Chemosphere. 2015;129:39–45. doi: 10.1016/j.chemosphere.2014.09.005

Fisher M, Arbuckle TE, Liang CL, et al. 2016. Concentrations of persistent organic pollutants in maternal and cord blood from the maternal-infant research on environmental chemicals (MIREC) cohort study. Environ Health 15(1):59.

Fromme H, Mosch C, Morovitz M, et al. 2010. Pre- and postnatal exposure to perfluorinated compounds (PFCs). Environ Sci Technol 44(18):7123-7129.

Fu J, Gao Y, Wang T, Liang Y, Zhang A, Wang Y, Jiang G. 2015. Elevated levels of perfluoroalkyl acids in family members of occupationally exposed workers: the importance of dust transfer. Sci Rep. 20;5:9313. doi: 10.1038/srep09313.

Ghisi R, Vamerali T, Manzetti S. 2019. Accumulation of perfluorinated alkyl substances (PFAS) in agricultural plants: A review. Environ Res. 169:326-341. doi: 10.1016/j.envres.2018.10.023.

Gleason JA, Post GB, Fagliano JA. 2015. Associations of perfluorinated chemical serum concentrations and biomarkers of liver function and uric acid in the US population (NHANES), 2007-2010. Environ Res 136:8-14. 10.1016/j.envres.2014.10.004.

Goeden HM, Greene CW, Jacobus JA. 2019. A transgenerational toxicokinetic model and its use in derivation of Minnesota PFOA water guidance. J Expo Sci Environ Epidemiol. 29(2):183-195. doi: 10.1038/s41370-018-0110-5.

Grandjean P, et al. 2012. Serum Vaccine Antibody Concentrations in Children Exposed to Perfluorinated Compounds. JAMA, 307(4): 391-397.

Grandjean P, Landrigan PJ. (2014). Neurobehavioural effects of developmental toxicity. Lancet Neurol . 13, 330–338.

Gyllenhammar I, Benskin JP, Sandblom O, Berger U, Ahrens L, Lignell S, Wiberg K, Glynn A. 2018. Perfluoroalkyl Acids (PFAAs) in Serum from 2-4-Month-Old Infants: Influence of Maternal Serum Concentration, Gestational Age, Breast-Feeding, and Contaminated Drinking Water. Environmental Science and Technology. 2018 Jun 19;52(12):7101-7110. doi: 10.1021/acs.est.8b00770

Hagenaars A, et al. 2013. Mechanistic toxicity study of perfluorooctanoic acid in zebrafish suggests mitochondrial dysfunction to play a key role in PFOA toxicity. Chemosphere, 91(6): 844-56.

Hall AP, Elcombe CR, Foster JR, et al. 2012. Liver hypertrophy: A review of adaptive (adverse and non-adverse) changes- conclusions from the 3rd International ESTP Expert Workshop. Toxicol Pathol 40:971-994.

Haug LS, et al. 2011. Investigation on Per- and Polyfluorinated Compounds in Paired Samples of House Dust and Indoor Air from Norwegian Homes. Environmental Science & Technology, 45, 7991-7998.

Haug LS. 2011. Characterisation of human exposure pathways to perfluorinated compounds – comparing exposure estimates with biomarkers of exposure. Dissertation for the degree of Doctor of Philosophiae, University of Oslo.

Haug, L.S., Huber, S., Becher, G., Thomsen, C. 2011. Characterisation of human exposure pathways to perfluorinated compounds - comparing exposure estimates with biomarkers of exposure. Environ. Int. 37: 687-693.

Health Canada. 2016a. Perfluorooctanoic acid (PFOA) in drinking water. Available online at: https://www.canada.ca/content/dam/hc-sc/healthy-canadians/migration/health-system-systeme-sante/consultations/acide-perfluorooctanoic-acid/alt/perfluorooctanoic-eng.pdf

Health Canada. 2016b. Perflurooctane sulfonate (PFOS) in drinking water. Available online at: https://www.canada.ca/content/dam/hc-sc/healthy-canadians/migration/health-system-systeme-sante/consultations/perfluorooctane-sulfonate/alt/perfluorooctane-sulfonate-eng.pdf

Hu Q, Strynar MJ, DeWitt JC. 2010. Are developmentally exposed C57BL/6 mice insensitive to suppression of TDAR by PFOA? J Immunotoxicol 7(4):344-349.

International Agency for Research on Cancer (IARC) 2016: CAS No. 335-67-1, Agent = Perfluorooctanoic acid (PFOA) Group 2B, Volume 110, 2016 online, Available at: http://monographs.iarc.fr/ENG/Classification/latest_classif.php

Jain RB, Ducatman A. 2019. Selective Associations of Recent Low Concentrations of Perfluoroalkyl Substances with Liver Function Biomarkers: NHANES 2011 to 2014 Data on US Adults Aged ≥20 Years. J Occup Environ Med. 61(4):293-302.

Kang H, et al. 2016. Elevated levels of short carbon-chain PFCAs in breast milk among Korean women: Current status and potential challenges. Environmental Research, 148, 351-359.

Kärrman A, Ericson I, van Bavel B, et al. 2007. Exposure of perfluorinated chemicals through lactation: Levels of matched human milk and serum and a temporal trend, 1996-2004, in Sweden. Environ Health Perspect 115:226-230.

Kato K, Wong LY, Chen A, et al. 2014. Changes in serum concentrations of maternal poly- and perfluoroalkyl substances over the course of pregnancy and predictors of exposure in a multiethnic cohort of Cincinnati, Ohio pregnant women during 2003-2006. Environ Sci Technol 48(16):9600-9608.

Kim D-H, et al. 2019. Assessment of individual-based perfluoroalkyl substances exposure by multiple human exposure sources. Journal of Hazardous Materials, 365, 26-33.

Kim SK, Lee KT, Kang CS, et al. 2011. Distribution of perfluorochemicals between sera and milk from the same mothers and implications for prenatal and postnatal exposures. Environ Pollut 159(1):169-174.

Kirk M, Smurthwaite K, Bräunig J et al. (2018). The PFAS Health Study: Systematic Literature Review. Canberra: The Australian National University.

Klaunig JE, Hocevar BA, Kamendulis LM. 2012. Mode of action analysis of perfluorooctanoic acid (PFOA) tumorigenicity and human relevance. Reprod Toxicol 33(4):410-418.

Koskela A, Finnila MA, Korkalainen M, et al. 2016. Effects of developmental exposure to perfluorooctanoic acid (PFOA) on long bone morphology and bone cell differentiation. Toxicol Appl Pharmacol 301:14-21. 10.1016/j.taap.2016.04.002.

Koustas E, Lam J, Sutton P, et al. 2014. The Navigation Guide - evidence-based medicine meets environmental health: Systematic review of nonhuman evidence for PFOA effects on fetal growth. Environ Health Perspect 122(10):1015-1027.

Kowalczyk J., Ehlers S., Oberhausen A., Tischer M., Furst P., Schafft H., Lahrssen-Wiederholt M. Absorption, distribution, and milk secretion of the perfluoroalkyl acids PFBS, PFHxS, PFOS, and PFOA by dairy cows fed naturally contaminated feed. J. Agric. Food Chem. 2013;61:2903–2912. doi: 10.1021/jf304680j

Lau C, Anitole K, Hodes C, Lai D, Pfahles-Hutchens A, Seed J. 2007. Perfluoroalkyl acids: a review of monitoring and toxicological findings. Toxicol. Sci. 99: 366-394.

Lau C, Thibodeaux JR, Hanson RG, et al. 2003. Exposure to perfluorooctane sulfonate during pregnancy in rat and mouse. II: Postnatal evaluation. Toxicol Sci 74(2):382-392.

Lau C, Thibodeaux JR, Hanson RG, et al. 2006. Effects of perfluorooctanoic acid exposure during pregnancy in the mouse. Toxicol Sci 90(2):510-518.

Lee SS-T, Pineau T, Drago J, Lee EJ, Owens JW, Kroetz DL, Fernandez-Salguero PM, Westphal H, and Gonzalez FJ. 1995. Targeted disruption of the a isoform of the peroxisome proliferator-activated receptor gene in mice results in abolishment of the pleiotropic effects of peroxisome proliferators. Mol Cell Biol 15:3012–3022

Lee YJ, Kim M-K, Bae J, et al. 2013. Concentrations of perfluoroalkyl compounds in maternal and umbilical cord sera and birth outcomes in Korea. Chemosphere 90(5):1603-1609.

Li K, Gao P, Xiang P, Zhang X, Cui X, Ma LQ. 2017a. Molecular mechanisms of PFOA-induced toxicity in animals and humans: Implications for health risks. 99:43-54.

Li K, Sun J., Yang J, Roberts SM, Zhang X, Cui X, Wei S, Ma LQ. 2017b. Molecular Mechanisms of Perfluorooctanoate-Induced Hepatocyte Apoptosis in Mice Using Proteomic Techniques. Environmental Science & Technology, 51, 11380-11389.

Li Y, Fletcher T, Mucs D, et al. 2018. Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water. Occup Environ Med 75(1):46-51. 10.1136/oemed-2017-104651.

Liew Z, et al. 2018. Developmental Exposures to Perfluoroalkyl Substances (PFASs): An Update of Associated Health Outcomes. Current Environmental Health Reports 5:1-19.

Liu J, Li J, Liu Y, et al. 2011. Comparison on gestation and lactation exposure of perfluorinated compounds for newborns. Environ Int 37(7):1206-1212.

Loveless SE, Finlay C, Everds NE, et al. 2006. Comparative responses of rats and mice exposed to linear/branched, linear, or branched ammonium perfluorooctanoate (APFO). Toxicology 220:203-217.

Loveless SE, Hoban D, Sykes G, et al. 2008. Evaluation of the immune system in rats and mice administered linear ammonium perfluorooctanoate. Toxicol Sci 105(1):86-96.

Luebker DJ, Case MT, York RG, et al. 2005a. Two-generation reproduction and cross-foster studies of perfluorooctanesulfonate (PFOS) in rats. Toxicology 215(1-2):126-148.

Luebker DJ, York RG, Hansen KJ, et al. 2005b. Neonatal mortality from in utero exposure to perfluorooctanesulfonate (PFOS) in Sprague-Dawley rats: Dose-response, and biochemical and pharmacokinetic parameters. Toxicology 215(1-2):149-169.

Macon MB, Villanueva LR, Tatum-Gibbs K, et al. 2011. Prenatal perfluorooctanoic acid exposure in CD-1 mice: Low-dose developmental effects and internal dosimetry. Toxicol Sci 122(1):134-145.

Mamsen LS, Björvang RD, Mucs D, Vinnars MT, Papadogiannakis N, Lindh CH, Andersen CY, Damdimopoulou P. 2019. Concentrations of perfluoroalkyl substances (PFASs) in human embryonic and fetal organs from first, second, and third trimester pregnancies. Environ Int. 124:482-492. doi: 10.1016/j.envint.2019.01.010. Epub 2019 Jan 24.

Manzano-Salgado CB, Casas M, Lopez-Espinosa MJ, et al. 2015. Transfer of perfluoroalkyl substances from mother to fetus in a Spanish birth cohort. Environ Res 142:471-478. 10.1016/j.envres.2015.07.020

Michigan Department of Health and Human Services (MIDHHS). 2019. Public health drinking water screening levels for PFAS. Available online at: https://www.michigan.gov/documents/pfasresponse/MDHHS_Public_Health_Drinking_Water_Screening_Levels_for_PFAS_651683_7.pdf

Michigan PFAS Science Advisory Panel Report. 2018. Scientific Evidence and Recommendations for Managing PFAS Contamination in Michigan. December 7, 2018. Available online at: https://www.michigan.gov/documents/pfasresponse/Science_Advisory_Board_Report_641294_7.pdf

Midasch O, Drexler H, Hart N, et al. 2007. Transplacental exposure of neonates to perfluorooctanesulfonate and perfluorooctanoate: A pilot study. Int Arch Occup Environ Health 80:643-648.

Minnesota Department of Health. 2018 - Toxicological Summary for: Perfluorooctanoate: http://www.health.state.mn.us/divs/eh/risk/guidance/gw/pfoa.pdf

Minnesota Department of Health. 2019 - Toxicological Summary for: Perfluorooctane sulfonate: https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfos.pdf

Minnesota Department of Health. 2019 - Toxicological Summary for: Perfluorohexane sulfonate: https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfhxs.pdf

Monroy R, Morrison K, Teo K, et al. 2008. Serum levels of perfluoroalkyl compounds in human maternal and umbilical cord blood samples. Environ Res 108:56-62.

Moriwaki H, et al. 2003. Concentrations of perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) in vacuum cleaner dust collected in Japanese homes. J. Environ. Monit., 5, 753-757.

Nakayama SF, et al. 2019. Worldwide trends in tracing poly- and perfluoroalkyl substances (PFAS) in the environment. Trends in Analytical Chemistry. Article in press, available online 2/14/19.

Nascimento RA, Nunoo DBO, Bizkarguenaga E, Schultes L, Zabaleta I, Benskin JP, Spanó S, Leonel J. 2018. Sulfluramid use in Brazilian agriculture: A source of per- and polyfluoroalkyl substances (PFASs) to the environment. Environ Pollut. 242(Pt B):1436-1443. doi: 10.1016/j.envpol.2018.07.122.

Needham LL, Grandjean P, Heinzow B, et al. 2011. Partition of environmental chemicals between maternal and fetal blood and tissues. Environ Sci Technol 45(3):1121-1126.

Negri E, et al. 2017. Exposure to PFOA and PFOS and fetal growth: a critical merging of toxicological and epidemiological data. Critical Reviews in Toxicology 47: 482-508.

New Hampshire Department of Environmental Services (NHDES). 2019. Summary Report on the New Hampshire Department of Environmental Services Development of Maximum Contaminant Levels and Ambient Groundwater Quality Standards for Perfluorooctanesulfonic Acid (PFOS), Perfluorooctanoic Acid (PFOA), Perfluorononanoic Acid (PFNA), And Perfluorohexanesulfonic Acid (PFHxS). Available at: https://www.des.nh.gov/organization/commissioner/pip/publications/documents/r-wd-19-01.pdf

Nian M, Li QQ, Bloom M, Qian ZM, Syberg KM, Vaughn MG, Wang SQ, Wei Q, Zeeshan M, Gurram N, Chu C, Wang J, Tian YP, Hu LW, Liu KK, Yang BY, Liu RQ, Feng D, Zeng XW, Dong GH. 2019. Liver function biomarkers disorder is associated with exposure to perfluoroalkyl acids in adults: Isomers of C8 Health Project in China. Environ Res. 172:81-88. doi: 10.1016/j.envres.2019.02.013.

NJ DWQI 2017: NJ Drinking Water Quality Institute (DWQI). 2016. Health-Based Maximum Contaminant Level Support Document: Perfluorooctanoic Acid (PFOA). Available online at: https://www.state.nj.us/dep/watersupply/pdf/pfoa-appendixa.pdf

NJ DWQI 2018: NJ Drinking Water Quality Institute (DWQI). 2018. Health-Based Maximum Contaminant Level Support Document: Perfluorononanoic Acid (PFNA). Available online at: https://www.state.nj.us/dep/watersupply/pdf/pfna-health-effects.pdf

NJ DWQI 2018: NJ Drinking Water Quality Institute (DWQI). 2018. Health-Based Maximum Contaminant Level Support Document: Perfluorooctane Sulfonate (PFOS). Available online at: https://www.state.nj.us/dep/watersupply/pdf/pfos-recommendation-appendix-a.pdf

New York Department of Health (NYDOH), 2018 presentation and professional communications. Presentation available at: https://www.health.ny.gov/environmental/water/drinking/dwqc/

NTP 2016: National Toxicology Program. NTP Monograph: Immunotoxicity Associated with Exposure to Perfluorooctanoic Acid or Perfluorooctane Sulfonate. September 2016.

Olsen GW, Burris JM, Ehresman DJ, Froehlich JW, Seacat AM, Butenhoff JL, Zobel LR. 2007. Half-life of serum elimination of perfluorooctanesulfonate, perfluorohexanesulfonate, and perfluorooctanoate in retired fluorochemical production workers. Environ Health Perspect 115(9):1298–1305, PMID: 17805419, 10.1289/ehp.10009.

Onishchenko N, Fischer C, Wan Ibrahim WN, et al. 2011. Prenatal exposure to PFOS or PFOA altersmotor function in mice in a sex-related manner. Neurotox Res 19(3):452-461.

Perkins RG, Butenhoff JL, Kennedy GL, et al. 2004. 13-Week dietary toxicity study of ammonium perfluorooctanoate (APFO) in male rats. Drug Chem Toxicol 27(4):361-378.

Porpora MG, Lucchini R, Abballe A, et al. 2013. Placental transfer of persistent organic pollutants: A preliminary study on mother-newborn pairs. Int J Environ Res Public Health 10(2):699-711.

Post GB, Gleason JA, Cooper KR. 2017. Key scientific issues in developing drinking water guidelines for perfluoroalkyl acids: Contaminants of emerging concern. PLoS Biol. 15(12):e2002855. doi: 10.1371/journal.pbio.2002855.

Quist EM, Filgo AJ, Cummings CA, et al. 2015a. Hepatic mitochondrial alteration in CD-1 mice associated with prenatal exposures to low doses of perfluorooctanoic acid (PFOA). Toxicol Pathol 43(4):546-557. 10.1177/0192623314551841.

Quist EM, Filgo AJ, Cummings CA, et al. 2015b. Supplemental data: Hepatic mitochondrial alteration in CD-1 mice associated with prenatal exposures to low doses of perfluorooctanoic acid (PFOA). (Toxicol Pathol 43(4):546-557). Toxicol Pathol 43:546-557.

Ramhoj L, et al. 2018. Perfluorohexane Sulfonate (PFHxS) and a Mixture of Endocrine Disrupters Reduce Thyroxine Levels and Cause Antiandrogenic Effects in Rats. Toxicological Sciences, 163(2), 579-591.

Rappazzo KM, et al. 2017. Exposure to Perfluorinated Alkyl Substances and Health Outcomes in Children: A Systematic Review of the Epidemiologic Literature. International Journal of Environmental Research and Public Health, 14, 691.

Rebholz SL, Jones T, Herrick RL, et al. 2016. Hypercholesterolemia with consumption of PFOA-laced Western diets is dependent on strain and sex of mice. Toxicology reports 3:46-54. 10.1016/j.toxrep.2015.11.004.

Rogers JM, Ellis-Hutchings RG, Grey BE, et al. 2014. Elevated blood pressure in offspring of rats exposed to diverse chemicals during pregnancy. Toxicol Sci 137(2):436-446. 10.1093/toxsci/kft248.

Rosen MB, Abbott BD, Wolf DC, et al. 2008a. Gene profiling in the livers of wild-type and PPARα-null mice exposed to perfluorooctanoic acid. Toxicol Pathol 36(4):592-607.

Rosen MB, Das KP, Rooney J, et al. 2017. PPARα-independent transcriptional targets of perfluoroalkyl acids revealed by transcript profiling. Toxicology Toxicology. 15;387:95-107. doi: 10.1016/j.tox.2017.05.013.

Rosen MB, Lee JS, Ren H, et al. 2008b. Toxicogenomic dissection of the perfluorooctanoic acid transcript profile in mouse liver: Evidence for the involvement of nuclear receptors PPARα and CAR. Toxicol Sci 103(1):46-56.

Rosen MB, Thibodeaux JR, Wood CR, et al. 2007. Gene expression profiling in the lung and liver of PFOA-exposed mouse fetuses. Toxicology 239:15-33.

Schaider LA, et al. 2017. Fluorinated Compounds in U.S. Fast Food Packaging. Environmental Science & Technology Letters, 4, 105-111.

Scher DP, Kelly JE, Huset CA, Barry KM, Hoffbeck RW, Yingling VL, Messing RB. 2018. Occurrence of perfluoroalkyl substances (PFAS) in garden produce at homes with a history of PFAS-contaminated drinking water. Chemosphere. 196:548-555. doi: 10.1016/j.chemosphere.2017.12.179.

Son H, Kim S, Shin HI, et al. 2008. Perfluorooctanoic acid-induced hepatic toxicity following 21-day oral exposure in mice. Arch Toxicol 82:239-246.

Stein CR, McGovern KJ, Pajak AM, et al. 2016. Perfluoroalkyl and polyfluoroalkyl substances and indicators of immune function in children aged 12-19 y: National Health and Nutrition Examination Survey. Pediatr Res 79(2):348-357.

Suh KS, et al. 2017. Perfluorooctanoic acid induces oxidative damage and mitochondrial dysfunction in pancreatic β-cells. Mol Med Rep. 15(6): 3871-3878.

Sundström M, Chang SC, Noker PE, et al. 2012. Comparative pharmacokinetics of perfluorohexanesulfonate (PFHxS) in rats, mice, and monkeys. Reprod Toxicol 33(4):441-451.

Tan X, Xie G, Sun X, et al. 2013. High fat diet feeding exaggerates perfluorooctanoic acid-induced liver injury in mice via modulating multiple metabolic pathways. PLoS ONE 8(4):e61409.

Texas Commission on Environmental Quality (TCEQ). 2016. Perfluorocompounds (PFCs). Available online at: https://www.tceq.texas.gov/assets/public/implementation/tox/evaluations/pfcs.pdf

Thibodeaux JR, Hanson RG, Rogers JM, et al. 2003. Exposure to perfluorooctane sulfonate during pregnancy in rat and mouse. I: Maternal and prenatal evaluations. Toxicol Sci 74(2):369-381.

Thomford PJ. 2001. 4-Week capsule toxicity study with ammonium perfluorooctanoate (APFO) in Cynomolgus monkeys. APME Ad-Hoc APFO toxicology working group.

Thompson J, Lorber M, Toms LM, et al. 2010. Use of simple pharmacokinetic modeling to characterize exposure of Australians to perfluorooctanoic acid and perfluorooctane sulfonic acid. Environ Int 36(4):390-397. 10.1016/j.envint.2010.02.008.

Trudel D, et al. 2008. Estimating Consumer Exposure to PFOS and PFOA. Risk Analysis, 28(2), 251-269. Erratum issued, 2008. Risk Analysis, 28(3), 807.

Trudel D, Horowitz L, Wormuth M, Scheringer M, Cousins IT, Hungerbuheler K. 2008. Estimating consumer exposure to PFOS and PFOA. Risk Anal. 28: 251-269.

Tucker DK, Macon MB, Strynar MJ, et al. 2015. The mammary gland is a sensitive pubertal target in CD-1 and C57Bl/6 mice following perinatal perfluorooctanoic acid (PFOA) exposure. Reprod Toxicol 54:26-36. 10.1016/j.reprotox.2014.12.002.

Tyagi S, Gupta P, Saini AS, Kaushal C, Sharma S (October 2011). "The peroxisome proliferator-activated receptor: A family of nuclear receptors role in various diseases". J Adv Pharm Technol Res. 2(4): 236–40.

USEPA (U.S. Environmental Protection Agency). 2000. Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000) Documents. Accessed online at: https://www.epa.gov/wqc/methodology-deriving-ambient-water-quality-criteria-protection-human-health-2000-documents

USEPA (U.S. Environmental Protection Agency). 2002. A Review of the Reference Dose and Reference Concentration Processes. EPA/630/P-02/0002F. Risk Assessment Forum, Washington, DC. Accessed online at: https://www.epa.gov/osa/review-reference-dose-and-reference-concentration-processes

USEPA (U.S. Environmental Protection Agency). 2011. Exposure Factors Handbook: 2011 Edition. EPA/600/R-090/052F. Office of Research and Development, National Center for Environmental Assessment, Washington, D.C. 1436 pp. Accessed online at: https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=236252.

USEPA (U.S. Environmental Protection Agency). Benchmark Dose Technical Guidance. Document # EPA/100/R-12/001. June 2012. Accessed online at: https://www.epa.gov/risk/benchmark-dose-technical-guidance

USEPA (U.S. Environmental Protection Agency). 2016a. Health Effects Support Document for Perfluorooctanoic acid (PFOA). Document # EPA 822-R-16-003. May 2016. Accessed online at: https://www.epa.gov/sites/production/files/2016-05/documents/pfoa_hesd_final_508.pdf

USEPA (U.S. Environmental Protection Agency). 2016b. Health Effects Support Document for Perfluorooctane Sulfonate (PFOS). Document # EPA 822-R-16-002. May 2016. Accessed online at: https://www.epa.gov/sites/production/files/2016-05/documents/pfos_hesd_final_508.pdf

USEPA (U.S. Environmental Protection Agency). 2019. Exposure Factors Handbook: Chapter 3 Update. EPA/600/R-090/052F. Office of Research and Development, National Center for Environmental Assessment, Washington, D.C. Accessed online at: https://www.epa.gov/sites/production/files/2019-02/documents/efh_-_chapter_3_update.pdf

Vanden Heuvel JP, Thompson JT, Frame SR, et al. 2006. Differential activation of nuclear receptors by perfluorinated fatty acid analogs and natural fatty acids: A comparison of human, mouse, and rat peroxisome proliferator-activated receptor-α, -ß, and -γ, liver x receptor-ß, and retinoid x receptor-α. Toxicol Sci 92(2):476-489.

Vélez MP, Arbuckle TE, Fraser WD. 2015. Maternal exposure to perfluorinated chemicals and reduced fecundity: The MIREC study. Hum Reprod 30(3):701-709. 10.1093/humrep/deu350.

Verner MA, Loccisano AE, Morken NH, et al. 2015. Associations of perfluoroalkyl substances (PFAS) with lower birth weight: An evaluation of potential confounding by glomerular filtration rate using a physiologically based pharmacokinetic model (PBPK). Environ Health Perspect 123(12):1317-1324.

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

Vieira VM, Hoffman K, Shin M, et al. 2013. Perfluorooctanoic acid exposure and cancer outcomes in a contaminated community: A geographic analysis. Environ Health Perspect 121(3):318-323.

Wan HT, Zhao YG, Leung PY, et al. 2014b. Perinatal exposure to perfluorooctane sulfonate affects glucose metabolism in adult offspring. PLoS ONE 9(1):e87137. 10.1371/journal.pone.0087137.

Wang H, Du H, Yang J, Jiang H, O K, Xu L, Liu S, Yi J, Qian X, Chen Y, Jiang Q, He G. 2019. S, PFOA, estrogen homeostasis, and birth size in Chinese infants. Chemosphere. 221:349-355. doi: 10.1016/j.chemosphere.2019.01.061.

Wang J, Yan S, Zhang W, et al. 2015. Integrated proteomic and miRNA transcriptional analysis reveals the hepatotoxicity mechanism of PFNA exposure in mice. J Proteome Res 14(1):330-341. 10.1021/pr500641b.

Washburn ST, et al. 2005. Exposure assessment and risk characterization for perfluorooctanoate in selected consumer articles. Environmental Science & Technology, 39(11), 3904-10.

White SS, Calafat AM, Kuklenyik Z, et al. 2007. Gestational PFOA exposure of mice is associated with altered mammary gland development in dams and female offspring. Toxicol Sci 96(1):133-144.

White SS, Kato K, Jia LT, et al. 2009. Effects of perfluorooctanoic acid on mouse mammary gland development and differentiation resulting from cross-foster and restricted gestational exposures. Reprod Toxicol 27(3-4):289-298.

White SS, Stanko JP, Kato K, et al. 2011. Gestational and chronic low-dose PFOA exposures and mammary gland growth and differentiation in three generations of CD-1 mice. Environ Health Perspect 119(8):1070-1076.

WHO. e-Library of Evidence for Nutrition Actions (eLENA). 2019. Exclusive breastfeeding for optimal growth, development and health of infants. Available online at: https://www.who.int/elena/titles/exclusive_breastfeeding/en/

Winkens K, et al. 2017. Early life exposure to per- and polyfluoroalkyl substances (PFASs): A critical review. Emerging Contaminants, 3, 55-68.

Winkens K, et al. 2018. Perfluoroalkyl acids and their precursors in floor dust of children's bedrooms – Implications for indoor exposure. Environment International, 119, 493-502.

Winkens K, Giovanoulis G, Koponen J, Vestergren R, Berger U, Karvonen AM, Pekkanen J, Kiviranta H, Cousins IT. 2018. Perfluoroalkyl acids and their precursors in floor dust of children's bedrooms - Implications for indoor exposure. Environ Int. 119:493-502. doi: 10.1016/j.envint.2018.06.009.

Wolf CJ, Fenton SE, Schmid JE, et al. 2007. Developmental toxicity of perfluorooctanoic acid in the CD-1 mouse after cross-foster and restricted gestational exposures. Toxicol Sci 95(2):462-473.

Wolf CJ, Schmid JE, Lau C, et al. 2012. Activation of mouse and human peroxisome proliferator-activated receptor-alpha (PPARa) by perfluoroalkyl acids (PFAAs): Further investigation of C4-C12 compounds. Reprod Toxicol 33:546-551.

Wolf CJ, Takacs ML, Schmid JE, et al. 2008. Activation of mouse and human peroxisome proliferator-activated receptor alpha by perfluoroalkyl acids of different functional groups and chain lengths. Toxicol Sci 106(1):162-171.

Wolf CJ, Zehr RD, Schmid JE, et al. 2010. Developmental effects of perfluorononanoic Acid in themouse are dependent on peroxisome proliferator-activated receptor-alpha. PPAR Res 2010 10.1155/2010/282896.

Worley RR, Yang X, Fisher J. 2017. Physiologically based pharmacokinetic modeling of human exposure to perfluorooctanoic acid suggests historical non drinking-water exposures are important for predicting current serum concentrations. Toxicol Appl Pharmacol. 330:9-21. doi: 10.1016/j.taap.2017.07.001.

Yahia D, El-Nasser MA, Abedel-Latif M, et al. 2010. Effects of perfluorooctanoic acid (PFOA) exposure to pregnant mice on reproduction. J Toxicol Sci 35(4):527-533.

Yahia D, Tsukuba C, Yoshida M, et al. 2008. Neonatal death of mice treated with perfluorooctane sulfonate. J Toxicol Sci 33(2):219-226.

Yang L, Wang Z, Shi Y, et al. 2016. Human placental transfer of perfluoroalkyl acid precursors: Levels and profiles in paired maternal and cord serum. Chemosphere 144:1631-1638. 10.1016/j.chemosphere.2015.10.063.

Zeng HC, Li YY, Zhang L, et al. 2011. Prenatal exposure to perfluorooctanesulfonate in rat resulted in long-lasting changes of expression of synapsins and synaptophysin. Synapse 65(3): 225-33.

Zeng XW, Bloom MS, Dharmage SC, Lodge CJ, Chen D, Li S, Guo Y, Roponen M, Jalava P, Hirvonen MR, Ma H, Hao YT, Chen W, Yang M, Chu C, Li QQ, Hu LW, Liu KK, Yang BY, Liu S, Fu C, Dong GH. 2019. Prenatal exposure to perfluoroalkyl substances is associated with lower hand, foot and mouth disease viruses antibody response in infancy: Findings from the Guangzhou Birth Cohort Study. Sci Total Environ. 663:60-67. doi: 10.1016/j.scitotenv.2019.01.325.

Zhang T, Sun H, Lin Y, Qin X, Zhang Y, Geng X, Kannan K. 2013. Distribution of poly- and perfluoroalkyl substances in matched samples from pregnant women and carbon chain length related maternal transfer. Environ Sci Technol. 47(14):7974-81. doi: 10.1021/es400937y.

Zhang Y, Beesoon S, Zhu L, et al. 2013. Biomonitoring of perfluoroalkyl acids in human urine and estimates of biological half-life. Environ Sci Technol 47(18):10619-10627. 10.1021/es401905e.

Zhu Y, Qin XD, Zeng XW, et al. 2016. Associations of serum perfluoroalkyl acid levels with T-helper cell-specific cytokines in children: By gender and asthma status. Sci Total Environ 559:166-173. 10.1016/j.scitotenv.2016.03.187.

June 25, 2019

Clark Freise
Assistant Commissioner
New Hampshire Department of Environmental Services
29 Hazen Drive
Concord, NH 03302

Dear Mr. Freise:

I have reviewed at your request the *New Hampshire Department of Environmental Services Technical Background for the June 2019 Proposed Maximum Contaminant Levels (MCLs) for Perfluorooctanoate (PFOA), Perfluorooctane sulfonate (PFOS), Perfluorononanoate (PFNA) and Perfluorohexane Sulfonate (PFHxS).* This document was prepared by Jonathan Ali, Ph.D., Mary Butow, M.S., and David Gordon, M.S., of the Permitting & Environmental Health Bureau and is dated June 7, 2019. This document updates drinking water standards for PFOA, PFOS, PFNA, and PFHxS originally proposed by the Department on December 31, 2018, taking into consideration recently published studies, as well as public comments on the original proposed Maximum Contaminant Levels (MCLs). Because the updated analysis is intended to be responsive to public comments, I have also read the public comments on the original proposed MCLs as part of my review.

All of the proposed MCLs are risk-based, meaning that the numerical value of the MCL is determined solely by what is determined to be a safe dose limit for the chemical in drinking water. Typically, risk-based criteria (i.e., concentration limits) for drinking water are derived using rather simplistic equations that combine some expression of the safe dose of the chemical with assumptions regarding drinking water consumption rate. The drinking water consumption rate is usually derived from an upper percentile value for a segment of the population [often, all adults]. Poly- and perfluoroalkyl substances (PFAS) are among the few environmental contaminants for which significant data are available regarding blood concentrations associated with adverse health effects, both in humans and animal models used in toxicity studies. This information, combined with information on the toxicokinetics of PFAS in humans and animals, allows safe levels of exposure to be based on blood concentrations and drinking water consumption that would produce those blood concentrations. Although this requires a more complex analysis than traditional methods for deriving MCLs, it provides a more rigorous and scientifically defensible basis for extrapolating dose-response relationships for toxicity observed in animals to humans.

The New Hampshire Department of Environmental Services (NHDES) and others have taken this approach for development of risk-based standards for PFAS in drinking water, but NHDES has taken it a step further. There is concern for PFAS exposure in infants, not only because some PFAS have been shown to produce adverse developmental effects in animals, but also because infants may have the highest blood concentrations of any life stage due to their small body weight and intake from

breastmilk or from formula made from PFAS contaminated water. This means that infants may be more susceptible to not only developmental effects from PFAS, but to other PFAS effects as well. To address explicitly potential risks from early life exposure to the four PFAS for which MCLs are proposed, NHDES has used a model recently developed by the Minnesota Department of Health (Goeden et al. 2019) that predicts blood concentrations of PFAS beginning at birth and extending into adulthood. The predicted blood concentrations of PFOA, PFOS, PFNA, and PFHxS using this model show clearly the importance of considering early life drinking water exposures, both direct and indirect, and allow demonstration that the proposed MCLs are protective at all life stages. This is a significant advance over the previous derivation of PFAS MCLs by the Department, and over most of the drinking water standards for PFAS developed elsewhere.

A critical aspect of the calculation of risk-based MCLs for PFAS is the derivation of safe dose limits, or reference doses. Development of these reference doses requires identification of a critical effect and study that provides dose-response information for that effect, determining a no-effect level from the data, selection of uncertainty factors to insure a health protective value in the face of limitations in the available data, and identifying a human equivalent dose based upon the toxicokinetics of the chemical in humans. The proposed MCLs in the June 2019 document include refinements in the reference doses for PFOA, PFOS, PFNA, and PFHxS presented in the January 2019 report based on consideration of new information, new analyses, and public comments. These include a change in critical effect (PFOS), total uncertainty factor (PFNA), modeling of toxicity data (PFHxS), and Dosimetric Adjustment Factor (PFOA, PFNA, PFHxS) to estimate a human equivalent oral dose. The rationale for each of the changes is clearly articulated in the report and all are well justified scientifically, in my opinion. I should note that a colleague, Dr. Leah Stuchal, and I collaborated with Dr. Ali of NHDES on the dose-response analysis for PFHxS presented in this report.

A number of public commenters took issue with one or more of the uncertainty factors selected for the derivation of initial reference doses for PFOA, PFOS, PFNA, and PFHxS in the January 2019 document. The selection of uncertainty factors for these and other chemicals is undoubtedly important as they have a direct impact on the risk-based drinking water standards that are derived. I have served as a peer reviewer for the U.S. EPA for many years on topics including proposed reference doses for several chemicals, primary through service on the Chartered Science Advisory Board and the Chemical Assessment Advisory Committee. Selection of uncertainty factors involves a good deal of scientific judgment, and despite guidance from the U.S. EPA on how uncertainty factor values should be selected in a given situation, it is often difficult to get complete agreement among objective scientists. So the number, and sometimes contradictory nature, of suggestions among public commenters regarding choices of uncertainty factors is not surprising. As with other aspects of reference dose development, I found the rationale for selection of uncertainty factors presented in the current document to be clear and consistent with U.S. EPA guidance. The comparison in Table 2 of uncertainty factors selected by NHDES with those chosen by other agencies that have developed reference doses for these chemicals shows that they are in line with judgments made by other regulatory scientists.

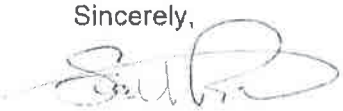
Another issue raised by public commenters is the overall level of conservatism inherent in the originally proposed MCL values, with comments offered in both directions — too conservative or not conservative enough. Concern that the initial MCLs were not

sufficiently conservative in that they were not clearly protective of infants has been addressed by NHDES through use of modeling that includes breastfed and formula-fed infants. For other, more general aspects of MCL derivation, NHDES is reasonably transparent in its attempts to strike the right balance of conservatism — conservative enough to provide confidence that the proposed MCLs are health protective without excessive conservatism that undermines the credibility of the results. Conservative choices are identified as such, and are used in combination with central tendency values for other inputs in an effort to create upper end, but not unrealistic estimates of risk. In my opinion, the level of conservatism achieved is entirely consistent with current risk assessment practice by state and federal environmental agencies.

As noted in the report, study of the potential health impacts of PFAS exposure is a rapidly changing field, and new information is becoming available almost continuously. Nevertheless, environmental regulatory agencies must often capture existing science as best they can and move forward with environmental criteria. Overall, I found the derivation of the MCLs proposed in the Technical Background document to be clearly described and scientifically sound, taking advantage of the most recent data and technical approaches.

The opinions expressed in this review are solely my own and do not necessarily reflect those of my employer, the University of Florida.

Sincerely,



Stephen M. Roberts, Ph.D.

Reference cited:

Goeden; HM, Greene CW, Jacobus JA. A transgenerational toxicokinetic model and its use in derivation of Minnesota PFOA water guidance. J. Expos. Sci. Environ. Epi. 29:183-195, 2019.

ATTACHMENT 2

New Hampshire Department of Environmental Services

Update on Cost and Benefit Consideration

June 28, 2019

NEW HAMPSHIRE DEPARTMENT OF ENVIRONMENTAL SERVICES

UPDATE ON CONSIDERATION OF THE COSTS AND BENEFITS RELATED TO FINAL PROPOSED MAXIMUM CONTAMINANT LEVELS AND AMBIENT GROUNWATER QUALITY STANDARDS FOR PERFLUOROOCTANESULFONIC ACID (PFOS), PERFLUOROOCTANOIC ACID (PFOA), PERFLUORONONANOIC ACID (PFNA), AND PERFLUOROHEXANESULFONIC ACID (PFHXS)

6/28/2019

Chapter Law RSA 345 requires the New Hampshire Department of Environmental Services to consider what is known about cost and benefit to affected parties when proposing maximum contaminant levels (MCLs) and ambient groundwater quality standards (AGQSs). This consideration was documented in the "Summary Report on the New Hampshire Department of Environmental Services Development of Maximum Contaminant Levels and Ambient Groundwater Quality Standards for Perfluorooctanesulfonic Acid (PFOS), Perfluorooctanoic Acid (PFOA), Perfluorononanoic Acid (PFNA), and Perfluorohexanesulfonic Acid (PFHxS)", dated January 4, 2019 (January 2019 report), for the initial proposed rules and is updated here for the final proposal. As was the case for the initial proposal, the emerging nature of PFAS contamination limits the availability of certain information that would be needed for a complete quantification of all the costs and benefits that will result from adopting these rules. Examples of these limitations include not having extensive sampling data for all potential contamination sources and public water systems statewide and having an incomplete understanding of all the health impacts associated with exposure to these four PFAS. Since the initial proposal, NHDES has continued to gather information and further research what is known about costs and benefits to consider in determining the standards to be included in the final proposal. Consideration of the updated information was performed and due to the clear, although difficult to quantify, health benefits in limiting exposure, the department chose to not alter the health based standards, despite recognizing the significant implementation costs.

Additional information on costs and benefits considered is provided below:

BENEFITS:

In the case of benefits, a number of new studies continue to suggest significant health impacts related to these four compounds, confirming that PFAS may:

- Increase cholesterol levels
- Increase liver enzyme levels
- Affect growth, learning, and behavior
- Interfere with the body's natural hormones, including thyroid hormone levels and sex hormone levels that could affect reproductive development and a woman's fertility
- Affect the immune system (e.g., decrease how well the body responds to vaccines)
- Increase the risk of certain types of cancers

These same health risks are identified by the Agency for Toxic Substances and Disease Registry (ATSDR) an agency within the Centers for Disease Control (CDC). https://www.atsdr.cdc.gov/pfas/health-effects.html

Additionally, the recent publication "A transgenerational toxicokinetic model and its use in derivation of Minnesota PFOA water guidance" provides a peer reviewed method to estimate blood serum levels that result from exposure to PFOA (later papers and one currently under peer review documented similar capabilities for PFOS, PFNA and PFHxS) in infants and children. As the statute specifically required that proposed standards provide "*an adequate margin of safety to protect human health at all life stages, including but not limited to pre-natal development*", this insight into how developmental-stage blood serum levels respond to different amounts of each of the four PFAS in drinking water strongly suggests that the proposed lower MCLs/AGQSs are necessary to keep infant and children blood serum levels below the levels that indicate enhanced risk of the various health endpoints identified by the ATSDR above.

As was described in the January 2019 report, NHDES was not able to monetize the avoided health impact costs. However, some of these impacts are clearly associated with the developmental stage of life and therefore can have significant through-life costs such as direct health care treatment costs, the associated losses of economic production and income of those impacted, and the associated impacts to families and caregivers. NHDES came to this conclusion after reviewing the most recent published research and speaking with experts, including a group of professors and researchers at the University of New Hampshire (UNH) with whom NHDES recently contracted to quantify the benefits of reducing the arsenic MCL. After filing the initial proposal, NHDES continued to reach out to experts and search for valid methods for quantifying benefit. Two recent studies were identified that have attempted to quantify benefits. The utility of both these studies is discussed below. The lack of science identifying direct causality between health impacts and these compounds continues to limit quantification of benefit, as was discussed in the January 2019 report related to utilizing contingent valuation studies. It should be noted that this is not unique to PFAS regulation in other states, other compounds have been regulated once the linkage to negative health impacts was documented, but before direct causality and dose/rate relationships were clearly known. This precautionary process is followed in drinking water regulation to limit the harm identified while the exact benefit is quantified through longer term studies. NHDES, based on the most recent studies, is confident that there is a clear and significant benefit to reducing exposure to these compounds through drinking water while additional studies will help to more accurately quantify the specific health care costs avoided from the known, and to be discovered, specific health impacts caused by these four PFAS compounds.

A new study produced by the Nordic Council of Ministers "The Cost of Inaction, A socioeconomic analysis of environmental and health impacts linked to exposure to PFAS" has attempted to quantify costs associated with low, medium and high risks of exposure to PFAS. This report assumes that PFAS as a group directly causes certain associated health impacts and then assumes a percentage of reported health events, for instance for kidney cancer, is caused by exposure to PFAS above certain levels. While not directly of utility to quantifying the health benefit associated with the proposed standards for these four compounds in New Hampshire, it does provide further estimation of the avoided costs that could be associated with reduced exposure to PFAS. A summary of the report is attached.

Similarly, a recent study used a previous study, that showed a clear link between low to moderate exposure to PFOA and reduced birth weights, to estimate health impact costs. This study, "Perfluorooctanoic acid and low birth weight: Estimates of US attributable burden and economic costs from 2003 through 2014", showed that while blood serum levels in the general US population are going down, there are still impacts to birth weights and attempted to quantify the through-life cost impacts of

those reduced birth weights. This is based on the National Health and Nutrition Examination Survey (NHANES) database where the general population is measured on a number of factors, including PFAS blood serum levels. It is important to note that a number of New Hampshire communities have measured blood serum levels significantly above those found in the NHANES data, which implies there is significant benefit in reducing exposures to better align with the national averages, as this study indicates there are still health impacts (reduced birth weight) that could be reduced by limiting exposure prior to and during pregnancy. While this study cannot be directly related to NH's population to quantify a benefit due to health cost mitigation, it did calculate (for the entire United States population) that the health impacts due to reduced birth weight were $347 million in 2013-2014. It is a consideration that the national averages for PFOA blood serum levels during this time period were half what has been measured recently in some impacted NH communities. The cost implications estimated in the study when the US population had similar blood serum levels to NH's impacted communities was approximately $2.7B. While this does not quantify the benefits of reduced PFAS exposure, it does imply that the benefits are significant.

Finally, the treatment that will be used at most public water systems that exceed an MCL(s) is granular activated carbon. This treatment may provide an ancillary benefit of removing many other substances such as any new emerging chemicals and other unregulated, not well studied PFAS.

COSTS

Where data was available to derive estimates of implementation costs, the information including all assumptions was provided in the January 4, 2019, report. These estimates have been updated based on the newly proposed standards (i.e. costs to public water systems, groundwater discharge permittees and landfill and hazardous waste site ground water management permittees). Public comments were broadly received commenting on the methods used by NHDES and providing recent quotes for treatment systems in design or implementation. Some of these updated costs validated the methods used by NHDES and none of the comments identified any systemic flaws in the approach used. Therefore, NHDES has chosen to continue to use the original assumptions which provide uniformity across source types and allow direct comparison of the costs resulting from the lowered standards. The following table provides the summary of the initial cost estimates and the new estimated costs.

PFAS Source Type	Initial Proposal Estimate	Final Proposal Estimate
Public Water Systems*	Initial Treatment Costs: $1,851,354 - $5,171,022 Initial Sampling: $1,102,500 - $2,836,000 Annual O&M Costs: $114,912 - $223,439 Annual Sampling Costs $73,055 - $184,825	Initial Treatment Costs: $65,046,987 - $142,822,884 Initial Sampling: $1,102,500 - $2,836,000 Annual O&M Costs: $6,914,552 - $13,444,963 Annual Sampling Costs $174,257 - $444,409
Active Hazardous Waste Sites*	Initial Corrective Action Costs:	Initial Corrective Action Costs:

	$1,350,000 _ $2,310,000 Annual Operating Costs: $570,000 - $1,020,000	$2,315,000 - $4,440,000 Annual Operating Costs: $980,000 - $1,795,000
Municipal Landfills*	Initial Corrective Action Costs: $380,000 – 755,000 Annual Operating Costs: $260,000 - $390,000	Initial Corrective Action Costs: $935,000 - $1,755,000 Annual Operating Costs: $465,000 - $770,000
Wastewater Discharges to Groundwater*	Initial Corrective Action Costs: $1,100,000 Annual Operating Costs: $200,000 - $400,000	Initial Corrective Action Costs: $5,000,000 Annual Operating Costs: $ 849,000 - $1,600,000

* Assumptions for public water systems are contained in the January 9, 2019, report and include treatment of sources that exceed the MCL verses taking the well off line, blending or inter-connecting. For all other costs categories, see attached tables that provide assumptions and calculations used to create these estimates.

Adopting MCLs and AGQSs does not require private well owners to test for or treat their water supplies. However, given the publicity concerning these contaminants and the low standards for them in public drinking water, it is likely that many homeowners may voluntarily choose to test and install treatment in their homes. Based on sampling in areas without likely sources of PFAS contamination, NHDES estimates that as much as 9% of the estimated 250,000 private wells will exceed the proposed standards which could result in an estimated initial cost of treatment of $70,895,522 and annual maintenance cost of $21,268,657. This is likely an overestimation since some homeowners will choose not to test, and some who test will choose not to treat.

In general, the qualitative explanation for sites that may be potential sources of contamination for which we have no or very limited data remains the same as what was presented in the January report. An exception to this is municipal fire stations. Based on an ongoing initiative to test 34 fire stations that may have used AFFF foams and are located in close proximity to wells, only 2 have levels above the proposed standards to date. This suggests there may be limited occurrence of PFAS at levels above the proposed standards near fire stations and accordingly costs associated with this potential source type may be overestimated in the January 9, 2019 report.

Table 1. Estimated Cost To Hazardous Waste and Landfill Sites for Proposed PFAS MCLs

Est. No. Hazardous Waste Sites	Est. No. of Landfill Sites	Additional Capital Costs	Hazardous Waste Sites	Landfill Sites	Additional Annual Costs	Hazardous Waste Sites	Landfill Sites
Projected # of existing Sites w/ PFAS Exceedances		*GMP Expansion of Existing Sites*	*Est. Cost*	*Est. Cost*	*GMP Expansion of Existing Sites*	*Est. Cost*	*Est. Cost*
		A **Monitoring Network Enhancments**			A **Annual Sampling and Reporting**		
252	84	Monitoring Well Install (assume 3 wells) + Initial Sampling Round	$ 12,000	$ 12,000	Annual Sampling/Lab fee (1 round, 3 wells)	$ 3,000	$ 3,000
		Receptor Survey	$ 1,000	$ 1,000	Annual GMP Reporting	$ 2,400	$ 2,400
		Est. Subtotal Capital Cost	$ 13,000	$ 13,000	Est. Subtotal Annual Cost	$ 5,400	$ 5,400
		Numbers below rounded to the nearest $5,000			Numbers below rounded to the nearest $5,000		
		Est. Total Capital Costs for GMP Expansion *(assumes 35% of all sites require expansion)*	**$ 1,145,000**	**$ 385,000**	***Est. Total Annual Monitoring/Reporting Costs*** *(assumes 35% of all sites require expansion)*	**$ 475,000**	**$ 160,000**
		Est. Total Capital Cost for GMP Expansion *(assumes 75% of all sites require expansion)*	**$ 2,455,000**	**$ 820,000**	***Est. Total Annual Monitoring/Reporting Costs*** *(assumes 60% of all sites require expansion)*	**$ 1,020,000**	**$ 340,000**
		B **Water Supply Well Treatment**			B **Water Supply Well Treatment**		
		POE Install -assume 3 per site	$ 3,000	$ 3,000	Annual O&M of POE (assume 3 per site)	$ 1,000	$ 1,000
		Est. Subtotal Cost	$ 9,000	$ 9,000	Est. Subtotal Annual O&M Cost	$ 3,000	$ 3,000
		Numbers below rounded to the nearest $5,000			Numbers below rounded to the nearest $5,000		
		Est. Total for Expansion of Sites 15% of all sites will have 3 new POEs	**$ 340,000**	**$ 115,000**	***Est. Total for Expansion of Sites 15% of all sites will have 3 new POEs***	**$ 115,000**	**$ 40,000**
		Est. Total for Expansion of Sites - 25% of all sites will have 3 new POEs	**$ 565,000**	**$ 190,000**	***Est. Total for Expansion of Sites 25% of all sites will have 3 new POEs***	**$ 190,000**	**$ 65,000**
					C **NHDES Staff Time (Assume Annual Salary/benefits for 2 FTE staff will be required at $120/yr)**	$ 120,000	$ 120,000
		I. Est. Capital Cost range for GMZ Expansion: Low	**$ 1,485,000**	**$ 500,000**	**I. Est. Annual Cost range for GMZ Expansion: Low**	**$ 710,000**	**$ 320,000**
		High	**$ 3,020,000**	**$ 1,010,000**	**High**	**$ 1,330,000**	**$ 525,000**
Projected # of Sites w/ PFAS Exceedances as new Contaminant of Concern		*Sites that may be required to address PFAS as a new Contaminant of Concern*	*Est. Cost*	*Est. Cost*	*Sites that may be required to address PFAS as a new Contaminant of Concern*	*Est. Cost*	*Est. Cost*
101	53	A **Monitoring Network Enhancments**			A **Annual Sampling and Reporting**		
		Monitoring Well Install (assume 5 wells) + Initial Sampling Round	$ 18,000	$ 18,000	Annual Sampling/Lab fee (1 round, 5 wells)	$ 3,500	$ 3,500
		Receptor Survey	$ 1,500	$ 1,500	Annual GMP Reporting	$ 2,900	$ 2,900
		Est. Subtotal Cost	$ 19,500	$ 19,500	Est. Subtotal Cost	$ 6,400	$ 6,400
		Numbers below rounded to the nearest $5,000			Numbers below rounded to the nearest $5,000		
		Est. Total for New Sites - 35%	**$ 695,000**	**$ 365,000**	***Est. Total Annual Monitoring Costs for New Sites - 35% of all sites***	**$ 225,000**	**$ 120,000**
		Est. Total for New Sites - 60%	**$ 1,190,000**	**$ 625,000**	***Est. Total Annual Monitoring Costs for New Sites - 60% of all sites***	**$ 390,000**	**$ 205,000**
		B **Water Supply Well Treatment**			B **Water Supply Well Treatment**		
		POE Install - assume 3 per site	$ 3,000	$ 3,000	Annual O&M of POE (assume 3 per site)	$ 1,000	$ 1,000
		Est. Subtotal Cost	$ 9,000	$ 9,000	Est. Subtotal Cost	$ 3,000	$ 3,000
		Numbers below rounded to the nearest $5,000			Numbers below rounded to the nearest $5,000		
		Est. Total for New Sites 15% of all sites will have 3 new POEs	**$ 135,000**	**$ 70,000**	***Est. Total for New Sites 15% of all sites will have 3 new POEs***	**$ 45,000**	**$ 25,000**
		Est. Total for New Sites 25% of all sites will have 3 new POEs	**$ 230,000**	**$ 120,000**	***Est. Total for New Sites 25% of all sites will have 3 new POEs***	**$ 75,000**	**$ 40,000**
		II. Est. Cost range for Sites w/ PFAS as New COC: Low	**$ 830,000**	**$ 435,000**	**I. Est. Annual Cost range for Sites w/ PFAS as New COC: Low**	**$ 270,000**	**$ 145,000**
		High	**$ 1,420,000**	**$ 745,000**	**High**	**$ 465,000**	**$ 245,000**
		Est. Total Capital Cost Impacts for Proposed MCLs: Low	**$ 2,315,000**	**$ 935,000**	**Est. Total Annual Operating Budget Impacts for Proposed MCLs: Low**	**$ 980,000**	**$ 465,000**
		High	**$ 4,440,000**	**$ 1,755,000**	**High**	**$ 1,795,000**	**$ 770,000**

Hazardous Waste Sites	Landfills	
$2.32M to $4.44M	$935K to $1.80M	Additional capital cost to expand existing GMZs, establish new sites and treat impacted drinking water supply wells
$935K to $1.76M	$465K to $770K	Additional annual operating costs (monitoring and reporting), and NHDES permit administration costs

For the Following Standards (ng/L):
PFOA = 12
PFOS = 15
PFNA = 11
PFHxS = 18

Updated: 5/7/2019

Table 1. Estimated Cost To Hazardous Waste and Landfill Sites for Proposed PFAS MCLs

Hazardous Waste Site Projections are based on:	
515	Hazardous Waste Sites
137	Number of sites PFAS Sampling has been completed
27%	Percent of Sites Sampled
Analysis of Existing Data and Current Standard of 70 PPT PFOA + PFOS	
Of the 137 sites sampled:	
49%	had exceedances of the current standard
9%	had water supply wells with exceedances of current standards
Estimate of # of Hazardous Waste Sites with Existing PFAS Compliance Issues	
Assumption: *Apply similar trend of existing data outlined above.*	
252	sites may have exceedances of the current standard
25 to 50	estimated number of sites with drinking water impacts[1]
Analysis of Existing Data and Proposed Standards in Parts per Trillion	
PFOA	12
PFOS	15
PFNA	11
PFHxS	18
69%	of sites sampled w/ exceed. of proposed stds of one or more compounds
53 to 88	estimated number of sites with drinking water impacts[1]

Notes:
1. Based on the limited data to estimate this, NHDES used a range of 15-25% of the projected number of sites with exceedances.

Landfill Site Projections are based on:	
201	Landfill Sites
117	Number of sites PFAS Sampling has been completed
58%	Percent of Sites Sampled
Analysis of Existing Data and Current Standard of 70 PPT PFOA + PFOS	
Of the 117 sites sampled:	
42%	had exceedances of the current standard
1%	had water supply wells with exceedances of current standards
Estimate of # of Landfill Sites with Existing PFAS Compliance Issues	
Assumption: *Apply similar trend of existing data outlined above.*	
84	sites may have exceedances of the current standard
8 to 17	estimated number of sites with drinking water impacts[1]
Analysis of Existing Data and Proposed Standards in Parts per Trillion	
PFOA	12
PFOS	15
PFNA	11
PFHxS	18
68%	sites sampled w/ exceed. of proposed stds of one or more compounds
21 to 34	estimated number of sites with drinking water impacts[1]

Notes:
1. Based on the limited data to estimate this, NHDES used a range of 15-25% of the projected number of sites with exceedances.

300

Updated: 5/7/2019

Cost Estimates - Reduction in PFAS Standards - Groundwater Discharge Permit Sites

Isolated Sites : Non-Developed Areas, Able to Expand GDZ, No Private/Public Water Supply Receptors

Small GWDP Sites
Non POTW sites, usually privately owned

Additional Capital Costs

Item	Count	Unit Cost	Total
Mon Well	3	$ 12,000	$ 36,000
Priv Well Svy	1	$ 1,000	$ 1,000
		Total	*$ 37,000*
5X	*Add'l sites*		*$ 185,000*

Additional Annual Costs

Item	Count	Unit Cost	Total
Smpl Rnd	6	$ 1,000	$ 6,000
Rpting	1	$ 2,400	$ 2,400
		Total	*$ 8,400*
5X	*Add'l sites*		*$ 42,000*

Large GWDP Sites
POTW sites, usually publicly owned

Additional Capital Costs

Item	Count	Unit Cost	Total
Mon Well	6	$ 12,000	$ 72,000
Priv Well Svy	1	$ 1,000	$ 1,000
		Total	*$ 73,000*
18X	*Add'l sites*		*$ 1,314,000*

Additional Annual Costs

Item	Count	Unit Cost	Total
Smpl Rnd	12	$ 1,000	$ 12,000
Rpting	1	$ 2,400	$ 2,400
		Total	*$ 14,400*
18X	*Add'l sites*		*$ 259,200*

Non-Isolated Sites : Developed Areas, Not (Easily) Able to Expand GDZ, Private/Public Water Supply Receptors Present

Small GWDP Sites
Non POTW sites, usually privately owned

Additional Capital Costs

Item	Count	Unit Cost	Total
Mon Well	2	$ 12,000	$ 24,000
Priv Well Svy	1	$ 2,500	$ 2,500
POE-PFAS	3	$ 3,000	$ 9,000
		Total	*$ 35,500*
Fac Trtmnt	*Range: 10k to 100k*		
4X	*Add'l sites*		*$ 142,000*

Additional Annual Costs

Item	Count	Unit Cost	Total
Smpl Rnd	4	$ 1,000	$ 4,000
Rpting	1	$ 2,400	$ 2,400
O&M	3	$ 900	$ 2,700
		Total	*$ 9,100*
4X	*Add'l sites*		*$ 36,400*

Large GWDP Sites
POTW sites, usually publicly owned

Additional Capital Costs

Item	Count	Unit Cost	Total
Mon Well	4	$ 12,000	$ 48,000
Priv Well Svy	1	$ 5,000	$ 5,000
POE-PFAS	6	$ 3,000	$ 18,000
		Total	*$ 71,000*
Fac Trtmnt	*Flows too large*		
2X	*Add'l sites*		*$ 142,000*

Additional Annual Costs

Item	Count	Unit Cost	Total
Smpl Rnd	8	$ 1,000	$ 8,000
Rpting	1	$ 2,400	$ 2,400
O&M	6	$ 900	$ 5,400
		Total	*$ 15,800*
2X	*Add'l sites*		*$ 31,600*

Multiplier 2.3

Additional Capital Costs

Add'l at new PFAS stds	*$*	*4,100,900*

Additional Annual Costs

Add'l at new PFAS stds	*$*	*849,160*

5x sites

Fac Trtmnt Range : up to $2,100,000	

*Small Facilities only

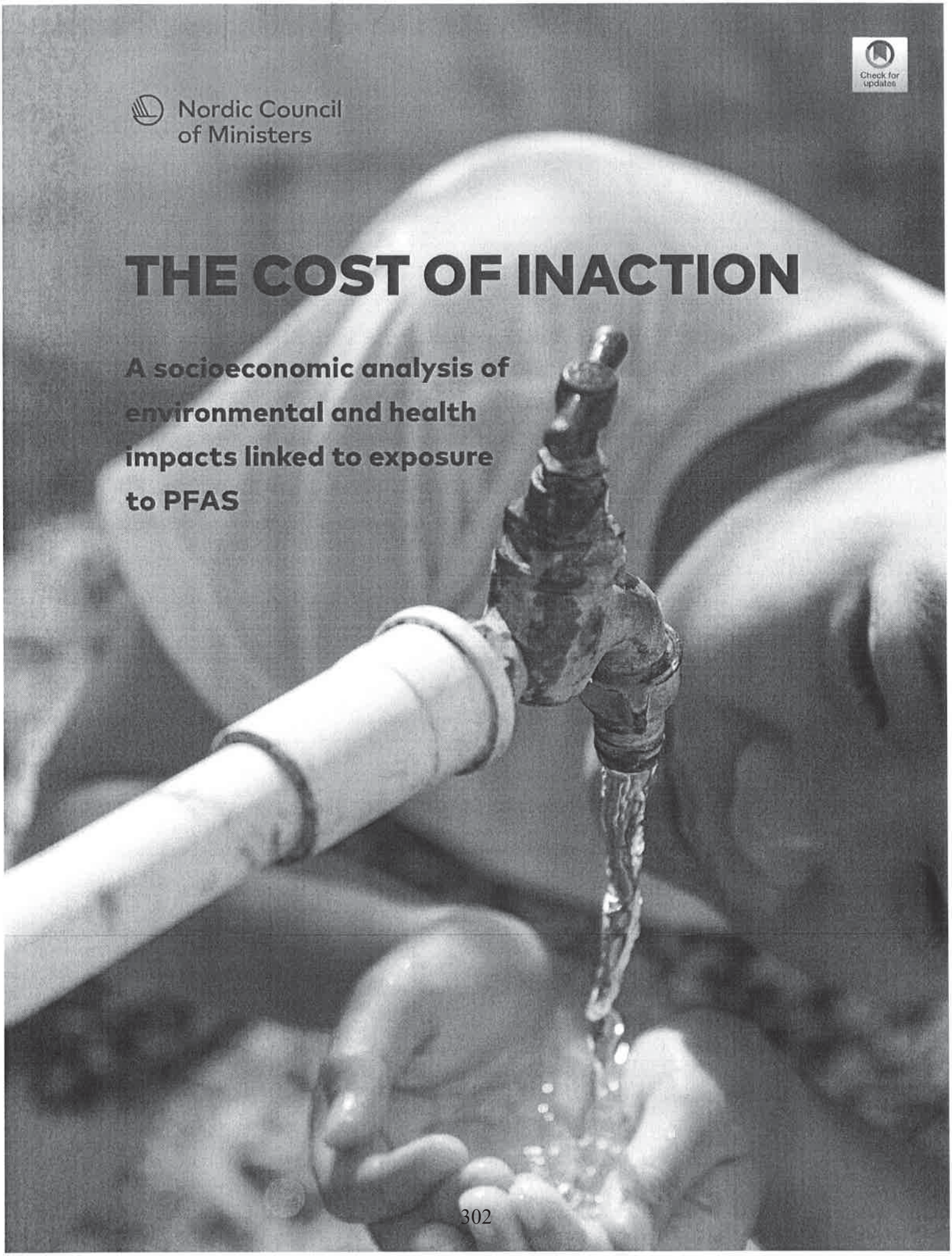
New PFAS Standard Evaluated:
PFOA: 12 ppt
PFOS: 15 ppt
PFNA: 11 ppt
PFHxS: 19 ppt

SUMMARY

For change to lower PFAS standards:
- A total of 27 GWDP sites with PFAS compliance issues - projected across full list of GWDP sites is 37.
-Adds ~ $4.1M to capital costs
-Adds ~ $900K to annual costs

Sites with Existing PFAS issues:
-Potential additional costs to sites with existing compliance issues that exceed the current PFAS standard : ~$800K

Cost impact to small (mostly privately owned) GWDP sites could be greater if WW pre-treatment is put in place: estimate ~ $2M to capital costs


Nordic Council
of Ministers
Check for updates
THE COST OF INACTION
A socioeconomic analysis of environmental and health impacts linked to exposure to PFAS

Summary

This study investigates the socioeconomic costs that may result from impacts on human health and the environment from the use of PFAS (per and polyfluoroalkyl substances). Better awareness of the costs and long-term problems associated with PFAS exposure will assist authorities, policy-makers and the general public to consider more effective and efficient risk management.

The production of PFAS, manufacture and use of PFAS-containing products, and end-of-life disposal of PFAS have resulted in widespread environmental contamination and human exposure. PFAS have been found in the environment all around the world and almost everyone living in a developed country has one or more PFAS in his/her body.

Because of the extreme persistence of PFAS in the environment, this contamination will remain on the planet for hundreds if not thousands of years. Human and environmental exposure will continue, and efforts to mitigate this exposure will lead to significant socioeconomic costs – costs largely shouldered by public authorities and ultimately taxpayers.

The focus of this study is on the costs of inaction with respect to regulation of PFAS in the countries comprising the European Economic Area (EEA). Costs of inaction are defined as the costs that society will have to pay in the future if action is not taken to limit emissions of PFAS today. The PFAS covered in this study are the C4-14 non-polymer fluorosurfactants.

The goal for the study has been two-fold:

1. to establish a framework for estimating costs for society related to negative impacts on health and the environment associated with PFAS exposure; and
2. to provide monetary values for those societal costs, documented by case studies.

Conclusions

The work of estimating the health and environment-related costs to society related to PFAS exposure has relied on the development of assumption-based scenarios. This reflects the limited data available in the academic literature, government documents and press reports. Whilst the uncertainties of the analysis need to be acknowledged, it is also important to recognise that, for several issues, there is little or no uncertainty:

1. PFAS are ubiquitous in the environment, and almost all people have PFAS in their bodies today. Monitoring in both Sweden and the USA concludes that around 3% of the population are currently exposed above proposed limit values, primarily through contamination of drinking water but also via other sources;
2. Many sources of PFAS exposure exist, linked to specialist applications (e.g. AFFFs for firefighting at airports and some industrial locations) and non-specialist uses (e.g. use in consumer goods such as pizza boxes, clothing and cosmetics);
3. Non-fluorinated alternatives for many of these uses are already on the market, and therefore certain uses of PFAS can be reduced;
4. The costs for remediating some cases of contamination run to many millions of EUR. Total costs at the European level are expected to be in the hundreds of millions of EUR as a minimum;
5. A large and growing number of health effects have been linked to PFAS exposure and evidence is mounting that effects occur even at background level exposures.

Current and proposed limit values for drinking water may be further reduced in recognition of growing information on, health and environmental risks. This would increase the costs of environmental remediation estimated here.

As explained throughout the study, the calculations rest on a number of assumptions, though these have been checked against e.g. data on costs incurred to ensure that they are linked to real-world experience. As more information becomes available, calculations will become more precise. Moreover, these findings are conservative. The figures are likely to get larger, in that the numbers of PFAS on the market and the volumes produced keep increasing. Further inaction will lead to more sources of contamination, more people exposed, and higher costs for remediation. The longer that PFAS contamination remains in the environment without remediation, the wider it will spread and the greater the quantity of soil or groundwater that will need to be decontaminated.

Methodology

Two methodologies have been developed, one for estimating health-related costs, the other for estimating costs of environmental remediation. Both methodologies are based on cases concerning exposure to PFAS. Data from the Nordic countries have been used when available, but the estimates also draw on cost data from other European countries, the USA and Australia, where relevant.

Impact pathways (the case studies)

Five case studies following the life-cycle of PFAS, from their production and use in product manufacturing, to the product's use and end-of-life disposal are used to illustrate how exposures to humans and the environment occur. Other instances of PFAS contamination provide additional data on direct costs incurred.

Case Study 1 considers exposures due to the production of PFAS in Europe. It reviews pollution linked to the Chemour factories in Dordrecht, Netherlands, the Miteni facility in the Veneto region of Italy, and the 3M plant near Antwerp, Belgium. The study estimates that up to 20 facilities actively produce fluorochemicals in Europe, that these facilities are significant sources of PFAS released to the environment, and that the exposure of workers at these plants is high.

The impacts from the manufacture and commercial use of PFAS-containing products are the focus of Case Study 2. Industrial activities with the potential to release PFAS to the environment include textile and leather manufacturing; metal plating, including chromium plating; paper and paper product manufacturing; paints and varnishes; cleaning products; plastics, resins and rubbers; and car wash establishments. The study assumes that a range of 3% to 10% of these facilities use PFAS. The study did not identify any fluorochemical production facilities in the Nordic countries. However, Eurostat statistics indicate that other industrial activities with the potential to release PFAS to the environment do take place in the region, such as metal plating and manufacture of paper products.

Case Studies 3 and 4 consider the use phase of PFAS-containing products. Case Study 3 examines exposure to PFAS-containing aqueous film-forming foams (AFFFs) used in firefighting drills and to extinguish petroleum-based fires. The AFFFs have contributed to groundwater contamination, especially around airports and military bases. Nearby communities have been affected by elevated levels of PFAS in their drinking water. Case Study 4 looks at PFAS-treated carpets, PFAS-treated food contact materials, and cosmetics as examples of how a product's use is likely to lead to direct human exposure through ingestion and dermal absorption. The use of products also result in releases to the environment when the product is washed off or laundered, entering sewers and treatment plants, and eventually waterways.

Case Study 5 looks at end-of-life impacts of PFAS-treated products. Municipal waste incineration may destroy PFAS in products if 1000 °C operating temperatures are reached. If landfilled, the PFAS will remain even after the product's core materials break down. The compounds will eventually migrate into liquids in the landfill, then into leachate collection systems or directly into the natural environment. They may then contaminate drinking water supplies, be taken up by edible plants and bioaccumulate in the food chain.

Health-related costs to society

To calculate health-related costs to society, the researchers looked for consensus regarding health endpoints affected by exposure to PFAS. Reviews of the scientific evidence have reached contradictory conclusions about the relevant health endpoints of human exposure to PFAS. However, some consensus has emerged concerning liver damage, increased serum cholesterol levels (related to hypertension), decreased immune response (higher risk of infection), increased risk of thyroid disease, decreased fertility, pregnancy-induced hypertension, pre-eclampsia, lower birth weight, and testicular and kidney cancer.

The methodology draws upon risk relationships developed in the course of specific epidemiological studies for populations exposed to PFAS at different levels. Workers exposed to PFAS in the workplace were used to exemplify a high level of exposure. Communities affected by PFAS, e.g. because of proximity to manufacturing sites or sites where fluorinated AFFFs were used, were assumed to have been exposed at a medium level; this level of exposure was assumed to have been experienced by 3% of the European population. The general population was considered to have experienced exposure at low (background) levels.

Table 1 provides an overview of the estimated annual costs for just a few health endpoints where risk ratios were available for affected populations. For example, the annual health-related costs for the elevated risk of kidney cancer due to occupational exposure to PFAS was estimated to be on the order of EUR 12.7 to EUR 41.4 million in the EEA countries. The estimated costs were substantially higher for elevated and background levels of exposure due to the greater number of persons affected. The total annual health-related costs, for the three different levels of exposure, was found to be at least EUR 2.8 to EUR 4.6 billion in the Nordic countries and EUR 52 to EUR 84 billion in the EEA countries.[1] Despite the high level of uncertainty and the assumptions underlying the calculations, the findings suggest that the health-related costs of exposure to PFAS are substantial.

[1] The health-related costs due to occupational exposure to PFAS in the Nordic countries was not estimated due to an absence of information about the number and location of chemical production plants or manufacturing sites.

Table 1: Estimates of annual health impact-related costs (of exposure to PFAS)

Exposure level	"Exposed" population and source	Health endpoint	Nordic countries		All EEA countries	
			Population at risk	Annual costs	Population at risk	Annual costs
Occupational (high)	Workers at chemical production plants or manufacturing sites	Kidney cancer	n.a.	n.a.	84,000–273,000	EUR 12.7–41.4 million
Elevated (medium)	Communities near chemical plants, etc. with PFAS in drinking water	All-cause mortality	621,000	EUR 2.1– 2.4 billion	12.5 million	EUR 41–49 billion
		Low birth weight	8,843 births	136 births of low weight	156,344 births	3,354 births of low weight
		Infection	45,000 children	84,000 additional days of fever	785,000 children	1,500,000 additional days of fever
Background (low)	Adults in general population (exposed via consumer products, background levels)	Hypertension	10.3 million	EUR 0.7– 2.2 billion	207.8 million	EUR 10.7–35 billion
Totals			*Nordic countries*	*EUR 2.8–4.6 billion*	*All EEA countries*	*EUR 52–84 billion*

Some overlap occurs in the figures above, because workers and affected communities are also exposed to background levels of PFAS. At the same time, these costs are likely to be underestimates due to the lack of epidemiological-based risk relationships for calculating other health endpoints and related costs.

Non-health (environment-related) costs to society

The second methodology compiled information on direct costs incurred by communities taking measures to reduce PFAS exposure through remediation of drinking water. Based on these direct costs, ranges of costs per persons affected or per case were developed. These unit costs then became the foundation for aggregating the costs of remediation when environmental contamination, e.g., PFAS concentrations in drinking water, reach certain levels. It should be noted that the ranges are broad, even when normalized against population.

The approach to derive ranges for the mean is dependent on the amount of data available. For the costs of water treatment, for example, several estimates were available, and in such cases it is unlikely that the true mean will be at either extreme of the range from the studies. Therefore, it is reasonable to truncate the observed range, for example by removing estimates that are sufficiently removed from other data as to be considered outliers. For some costs, however, very few estimates are available, each of which may be equally valid for representation of the average: in such a case the observed range in values is adopted as the range of plausible mean values.

Where no range is available from the studied literature, a range has been estimated. For example, the range of +/-90% is used for establishing a health assessment regime (here considered as a non-health cost as it deals with management of the problem, rather

than impacts on the health of society). In this example, the range is extremely broad for two reasons, first because of the lack of data available and second because of the potential for variation in the implementation of a health assessment programme.

As with the health-based estimates, the study assumes that 3% of the European population is exposed to drinking water with PFAS concentrations over regulatory action levels, such that the water treatment works serving them will require upgrading and maintenance over the next 20 years. The assumption of 20 years reflects potential for remediation to resolve problems perhaps through decontamination or the use of alternative supplies, or the potential for remedial action to persist for many years. Recognising the uncertainties that exist in the analysis and the available data, costs of remediation have been quantified using a scenario-based approach. For each scenario a number of parameters are specified, relating for example to the size of the affected population and the duration of maintenance works.

Table 2 shows the range of costs for the various categories of actions related to environmental remediation.

Table 2: Summary of estimates of mean cost data for non-health expenditures, 20 years

Action taken when PFAS found	Unit	Best estimate	Range from studies	Adopted range
Monitoring – checks for contamination due to industrial or AFFF use	Cost per water sample tested	EUR 340	EUR 278–402	EUR 278–402
	Cost/case of contamination	EUR 50,000	EUR 5,200–5.8 million	EUR 25,000–500,000
Health assessment (including biomonitoring)	Cost/person	EUR 50	No range	EUR 5–95 (+/-90%)
	Total biomonitoring and health assessment per case where considered appropriate	EUR 3.4 million	EUR 2.5 million–4.3 million	EUR 1 million–5 million
Provision of temporary uncontaminated supply	Cost/person	No relevant data		
Provision of a new pipeline	Cost/person	EUR 800	EUR 37–5,000	EUR 100–1,500
Upgrading water treatment works (capital)	Cost/person	EUR 300	EUR 8–2,200	EUR 18–600
Upgrading water treatment works (maintenance)	Cost/person	EUR 19	EUR 8–30	EUR 8–30
Excavation and treatment of soils – contamination from industrial or AFFF use	Cost/kg PFAS	EUR 280,000	EUR 100,000–4.3 million	EUR 100,000–1 million
	Cost/case	EUR 5 million	EUR 100,000–3 billion	EUR 300,000–50 million

In Table 3 the range of costs for the various categories of actions related to environmental remediation for the five Nordic countries are shown. The overall range of costs is EUR 46 million – 11 billion.

Table 3: Detailed breakdown of ranges for non-health costs to the Nordic countries, assuming that 1 to 5% (best estimate 3%) of the population is exposed above a statutory limit and that water treatment is required over a 20 year period

	N people affected (3%)	Screening and monitoring	Health assessment	Upgrade treatment works and maintenance	Soil remediation	Total
Denmark	170,000	EUR 70,000–8.3 million	EUR 280,000–27 million	EUR 7.4 million–274 million	EUR 0–798 million	EUR 8 million–1.1 billion
Finland	160,000	EUR 250,000–22 million	EUR 270,000–26 million	EUR 7.2 million–265 million	EUR 2.2 million–2.1 billion	EUR 10 million–2.4 billion
Iceland	10,000	EUR 10,000–900,000	EUR 20,000–1.6 million	EUR 400,000–1.6 million	EUR 100,000–86 million	EUR 1 million–105 million
Norway	160,000	EUR 170,000–20 million	EUR 260,000–25 million	EUR 6.8 million–250 million	EUR 1.6 million–1.9 billion	EUR 9 million–2.2 billion
Sweden	290,000	EUR 480,000–47 million	EUR 490,000–46 million	EUR 13 million–472 million	EUR 4.3 million–4.5 billion	EUR 18 million–5.1 billion
Nordic total	*790,000*					*EUR 46 million–11 billion*

The cost estimates provided in the table are likely to be more robust at the aggregate, European level than at the national level.

Table 4 provides aggregated costs covering environmental screening, monitoring (where contamination is found), water treatment, soil remediation and health assessment for the five Nordic countries and for the other EEA countries and Switzerland.

Table 4: Aggregated costs covering environmental screening, monitoring where contamination is found, water treatment, soil remediation and health assessment

	Best estimate	Low	High
Denmark	EUR 145 million	EUR 8 million	EUR 1.1 billion
Finland	EUR 214 million	EUR 10 million	EUR 2.4 billion
Iceland	EUR 12 million	EUR 1 million	EUR 105 million
Norway	EUR 194 million	EUR 9 million	EUR 2.2 billion
Sweden	EUR 423 million	EUR 18 million	EUR 5.1 billion
Other EEA+CH	EUR 15,9 billion	EUR 776 million	EUR 159.9 billion
Total	EUR 16.9 billion	EUR 821 million	EUR 170.8 billion

Parallel calculations for all 31 EEA Member Countries and Switzerland arrive at a range of costs for environmental remediation totalling EUR 821 million to EUR 170 billion. The

lower and upper bounds should be considered illustrative because of the limited information available. However, based on the literature review, there is a firm basis for concluding that the lower bound estimates would be exceeded. A best estimate in the order of EUR 10–20 billion is certainly plausible. The potential for higher costs is also possible: An estimate of the costs for one case identified in the course of the research, concerning the town of Rastatt in Baden-Wurttemberg in Germany is in the range of EUR 1 to 3 billion, with the estimated extent of the problem being seen to increase over time. The source of contamination in this case is understood to be contaminated waste paper materials that were spread on agricultural land, demonstrating that serious problems are not always linked to airfields and PFAS manufacture.

A number of other costs related to PFAS contamination are outside the scope of the quantification carried out in this report. These include loss of property value, reputational damage to a polluting company, ecological damage and the costs incurred by public authorities in responding to affected communities – including public outreach, surveys of contamination and remedial measures.

ATTACHMENT 3

Letter from NH Department of Justice dated 6/26/2019 Regarding NHDES Interpretation of RSA 485:3, I(b)

June 28, 2019

ATTORNEY GENERAL
DEPARTMENT OF JUSTICE

33 CAPITOL STREET
CONCORD, NEW HAMPSHIRE 03301-6397

GORDON J. MACDONALD
ATTORNEY GENERAL

JANE E. YOUNG
DEPUTY ATTORNEY GENERAL

June 26, 2019

Clark Freise
Assistant Commissioner
New Hampshire Department of Environmental Services
29 Hazen Drive
P.O. Box 95
Concord, New Hampshire 03302-0095

Re: NHDES Interpretation of RSA 485:3, I(b)

Dear Assistant Commissioner Freise

In response to the Department of Environmental Service's request for a legal opinion regarding the Department's interpretation of the costs and benefits clause included in RSA 485:3, I(b), as amended by Laws 2018, ch. 368, the Office of Attorney General provided a privileged and confidential letter containing legal advice to the Department. Without waiving the attorney-client privilege, this letter serves as confirmation that the Office of the Attorney General finds the Department's interpretation of RSA 485:3, I(b) to be reasonable and lawful.

Sincerely,

Christopher G. Aslin
Senior Assistant Attorney General
Environmental Protection Bureau
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R-WD-19-01

SUMMARY REPORT ON THE NEW HAMPSHIRE DEPARTMENT OF ENVIRONMENTAL SERVICES DEVELOPMENT OF MAXIMUM CONTAMINANT LEVELS AND AMBIENT GROUNDWATER QUALITY STANDARDS FOR PERFLUOROOCTANESULFONIC ACID (PFOS), PERFLUOROOCTANOIC ACID (PFOA), PERFLUORONONANOIC ACID (PFNA), AND PERFLUOROHEXANESULFONIC ACID (PFHxS)

Prepared by
New Hampshire Department of Environmental Services

Robert R. Scott, Commissioner
Clark B. Freise, Assistant Commissioner

January 4, 2019

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TABLE OF CONTENTS

LIST OF COMMON ACRONYMS

AGQS – Ambient Groundwater Quality Standard

ATSDR – Agency for Toxic Substances and Disease Registry

CDC – Centers for Disease Control

COC – Contaminant of Concern

DWEL – Drinking Water Equivalency Level

EPA – United States Environmental Protection Agency

HED – Human Equivalent Dose

MCL – Maximum Contaminant Level

MRL – Minimum Risk Level

NHDES – New Hampshire Department of Environmental Services

NHDHHS – New Hampshire Department of Health and Human Services

NOAEL – No Observed Adverse Effect Level

PFAS – Per- and polyfluoroalkyl substances

PFHxS – Perfluorohexanesulfonic Acid

PFOA – Perfluorooctanoic Acid

PFOS – Perfluorooctanesulfonic Acid

PFNA – Perfluorononanoic Acid

PoD – Point of Departure

PWS – Public Water System

RfD – Reference Dose

RSC – Relative Source Contribution Factor

SDWA – Safe Drinking Water Act

UFs – Uncertainty Factors

1. Background

Perfluorooctanesulfonic acid (PFOS), perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), and perfluorohexanesulfonic acid (PFHxS) are part of a large class of chemicals known as perfluorinated compounds (PFCs) and more broadly as per- and polyfluoroalkyl substances (PFAS). They have been widely used since the 1940s in commercial, industrial, and household products and applications, including production of water resistant materials, fire suppression foams, non-stick cookware, stain removers, etc. All four compounds have been detected in New Hampshire's groundwater and surface water. Because of their widespread use, persistence and mobility in the environment and bioaccumulative properties, these compounds have been detected in blood serum in humans and animals worldwide and have been studied for their toxicity and health effects. The health effects associated with PFAS exposure are currently being researched extensively by toxicologists and epidemiologists worldwide, resulting in numerous publications being released on a continuous basis. The New Hampshire Departments of Environmental Services (NHDES) and Health and Human Services (NHDHHS) continue to review and evaluate the toxicity and health effects of these compounds as research becomes available. According to the Centers for Disease Control's (CDC) Agency for Toxic Substances and Disease Registry (ATSDR), some, but not all, studies in humans have shown health effects possibly associated with PFAS exposure including:

- Altered growth, learning and behavior of infants and older children.
- Lowering a woman's chance of getting pregnant.
- Interference with the body's natural hormones.
- Increased cholesterol levels.
- Modulation of the immune system.
- Increased risk of certain cancers.

For additional information on the toxicity and health effects of these compounds, please visit the ATSDR webpage at: https://www.atsdr.cdc.gov/pfas/health-effects.html.

New Hampshire Chapter Laws 345 of 2018 (i.e., SB309, see Appendix 1) authorize NHDES to consult with NHDHHS and to initiate rulemaking to adopt maximum contaminant levels (MCLs) for PFOA, PFOS, PFHxS and PFNA by January 1, 2019. The legislation requires that NHDES consider, 1) the extent the contaminant is found in New Hampshire; 2) the ability to detect the compound; 3) the ability to treat the contaminant; 4) benefits associated with adopting an MCL; and 5) the costs associated with adopting an MCL. MCLs are water quality standards that apply to public water systems (PWS). Most MCLs , including those proposed in this report, are set for long-term, chronic exposure to a contaminant and only apply to non-transient public water systems (water systems serving 25 or more of the same population of people, six months of the year). Public water systems (PWS) sample all of their water sources for compounds with MCL standards, and submit the results to NHDES to demonstrate compliance with water quality standards.

Existing state law requires NHDES to adopt rules establishing Ambient Groundwater Quality Standards (AGQS) that are the same as any MCLs established by NHDES. Existing state law also requires that AGQS be the same or more stringent than any federal MCL or health advisory established under the federal Safe Drinking Water Act (SDWA). AGQS are the standards used to require site investigations and remedial action at and around contamination sites. AGQS are also used to identify where the provision of alternative drinking water is required when contaminated sites impact offsite private and/or public water supply wells. An AGQS also dictates the conditions under which wastewater and wastewater residuals may be discharged to groundwater. Although NHDES adopted an AGQS for PFOA and PFOS of 70 nanograms per Liter (ng/L) [or

parts per trillion (ppt)[1]] for these two compounds combined in May of 2016, the laws enacted in 2018 require NHDES to re-assess these standards and to also adopt AGQS for PFHxS and PFNA.

This report provides information on how New Hampshire's proposed MCLs and AGQSs for PFOA, PFOS, PFNA and PFHxS were developed to ensure they are protective of human health at all life stages. The report also provides information on the criteria that the law requires NHDES to consider when establishing MCLs including: occurrence in drinking water, the ability to detect the contaminant, the ability to treat to achieve compliance with the MCLs, and the costs and benefits to parties affected by establishing the standards.

It is important to note that New Hampshire, like most other states, has always relied on the U.S. Environmental Protection Agency (EPA) to set MCLs. EPA and the few other states that set drinking water standards employ a variety of experts who derive protective health-based standards (e.g., toxicologists and health risk assessors), economists trained in cost and benefit analysis, and chemists and engineers who can determine lab and treatment capabilities. SB309 included funding for a toxicologist and health risk assessor, who both began work at NHDES on October 12, 2018. NHDES was also able to engage the services of an outside expert to provide some additional assistance in the review of toxicological information. NHDES did not have resources to fully evaluate costs and benefits, as would have been done on the federal level, but has attempted to provide an analysis of each based upon available information.

The majority of the work NHDES has performed to date has been focused on deriving the individual standards for PFOA, PFOS, PFNA and PFHxS. During the rulemaking process, NHDES expects to continue researching health studies on these chemicals as well as risk management approaches that are scientifically valid and could address any compounding effects between chemicals when the chemicals are found in combination in a drinking water source. Further exploration on quantifying benefit to affected parties will also occur. This continued effort will be done in tandem with considering public comments received on the initial rule proposal.

2. Proposed MCLs and AGQSs

Establishing MCLs is done in accordance with guidance developed by EPA and other health agencies and programs. Details of how health protective drinking water standards are usually developed are presented in Appendix 2. The sequence of steps is summarized below:

- The most sensitive adverse effect that is thought to be relevant to humans is chosen. The lowest dose that has no significant toxic effect is the usual initial starting point (a no observed adverse effect level or NOAEL).

- The NOAEL or the *lowest* observed adverse effect level (LOAEL), if there is no NOAEL, is converted to a human equivalent dose (HED) through physiological models or other dose adjustment methods. The HED becomes the point of departure (PoD) for deriving the ultimate drinking water standard.

- The PoD is reduced by uncertainty factors (UFs) of either 10- or 3-fold to take into account incomplete knowledge regarding critical factors such as when there is incomplete knowledge of human variability and sensitivity; in cases where short-term studies are used to protect against

[1] Both the MCL and the AGQS are specified in nanograms per Liter (ng/L), a unit of concentration that is equivalent to parts per trillion (ppt) in water. In this document, concentrations are stated in ppt except in quoted references and tables that use ng/L.

effects from long-term exposure, and when the usual required studies to set a standard (e.g., reproductive effects studies, developmental studies or cancer studies) are missing.

- The toxicity value developed, which EPA refers to as a Reference Dose (RfD) and ATSDR refers to as a Minimal Risk Level (MRL), is converted to an equivalent dose in drinking water by selecting a sensitive human receptor and using their body weight and drinking water ingestion rate to calculate a drinking water equivalency level (DWEL). The DWEL is 100% of a dose not expected to cause any toxic effects to even the most sensitive subpopulation.

- For most chemicals, exposure from sources other than drinking water, such as from air, food and soil, is also possible. Therefore, the DWEL must be reduced by estimated doses coming from all other potential sources using a relative source contribution factor (RSC), so that the total exposure dose does not exceed 100% of the RfD, MRL or DWEL.

It is important to understand that drinking water standards for the same chemical often differ depending on the entity setting them. This is not unexpected, since standard setting guidance is not simply a mathematical formula and anticipates the need for professional judgment, which is involved in several stages of the standard setting process. Information about the relevancy of effects on animals to humans is often incomplete and contradictory, which will influence the toxic effect that is chosen. The selection of appropriate UFs is another area where judgment is critical. Whether a full UF of 10 or a partial one of 3 is used for an UF, it will change the resulting standard by just over 3-fold. The RSC chosen can also have a significant influence on the final standard. If one Risk Assessor determines that the data required to select an RSC are inadequate, EPA's guidance recommends using a default RSC of 20%. Another Assessor may determine the data on background exposure are adequate and choose an RSC of 60% based on them. The choice between those two RSCs will also change the standard selected by 3-fold. In a world of complete knowledge about a chemical's effects, relevance to humans and background exposure, health-protective drinking water standards calculated by different practitioners should be identical. However, in the real world, the lack of knowledge about a chemical and the appropriate degree of protectiveness to apply in the face of uncertainty results in differing choices, which can change the value selected for a standard.

In order to ensure that NHDES was aware of all the current, relevant health studies and information available in deriving the proposed MCLs/AGQSs, the agency solicited input from stakeholders through a series of public meetings held for this purpose. A list of the documents/references received following these meetings is available on the NHDES website at: https://www4.des.state.nh.us/nh-pfas-investigation/wp-content/uploads/2018/11/Draft_PFAS-Reference-List-as-of-11-07-18_For-Posting-to-Website.pdf. Comments received are available on the NHDES website at: https://www.des.nh.gov/organization/commissioner/max-contaminant-levels.htm. Studies selected and utilized in the derivation of the standards are listed in Appendix 8.

The following Table (Table 1) provides an overview of the proposed derived standards and the factors selected to derive the proposed MCL/AGQS. Appendices 4-7 include a description for each of the chemicals and how the standard was derived. These derivations were reviewed by Dr. Stephen M. Roberts, Ph.D., who also assisted NHDES with the review of ATSDR's Draft Toxicological Profile released in June 2018. In addition to the individual standards for PFOA and PFOS, the proposed rulemaking keeps the combined 70 ppt for PFOA and PFOS as an AGQS and also proposes that it be adopted as an MCL. This is consistent with existing law, which requires that an AGQS shall be no less stringent than an EPA health advisory.

Table 1: Summary of MCL Derivation Factors				
	PFOA*	PFOS*	PFHxS	PFNA
Health Effect Endpoint	Altered Liver Size/Function	Delayed Development	Impaired Reproduction	Altered Liver Size/Function
Animal Serum Dose (ng/mL)	4,351[a]	6,260[b]	27,200[c]	4,900[d]
Total Uncertainty Factor HUF x AUF x MF[e]	100 10 x 3 x 3	100 10 x 3 x 3	300 10 x 3 x 10	300 10 x 3 x 10
Target Human Serum Dose (ng/mL)	43.5	62.6	90.7	16.3
Human Half-life (years)	2.7[f]	3.4[f]	5.3[f]	2.5[g]
Dosimetric Adjustment Factor (L/kg/d)	$1.20E^{-04}$	$1.28E^{-04}$	$1.03E^{-04}$	$1.52E^{-04}$
Reference Dose (ng/kg/d)	5.2	8.0	9.3	2.5
Relative Source Contribution[h]	40%	50%	50%	50%
Water Ingestion Rate[i]	0.055 L/kg d	0.055 L/kg d	0.055 L/kg d	0.055 L/kg d
MCL/AGQS ppt (ng/L)	**38**	**70[j]**	**85**	**23**

[a] Loveless et al., 2006, NJ DWQI 2017, increased relative liver weight in mice;
[b] Luebker et al., 2005a, EPA 2016b, reduced pup weight and developmental delays in rats;
[c] Chang et al., 2018, reduced litter size in mice;
[d] Das et al., 2015, NJ DWQI 2018, increased relative liver weight in mice;
[e] HUF (Human-to-Human Uncertainty) x AUF (Animal-to-Human Uncertainty) x MF (Modifying Factor)
[f] Li et al., 2017, serum-derived half-life estimates from men and women exposed to PFAS via drinking water;
[g] Zhang et al., 2013, ATSDR 2018, urine-derived half-life from community exposure to PFNA;
[h] The RSC was derived using NH-specific blood data from high-exposed populations of Pease and Southern NH. This was calculated using the subtraction method described in the EPA 2000 Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health. Details about this approach are summarized in Appendices 4-7;
[i] EPA 2011 Exposure Factors Handbook, lactating women 95th percentile;
[j] PFOS rounded down to 70 ppt from 73 ppt, per the current EPA Health Advisory for PFOS.

*The derivation of the 70 ppt standard for PFOA and PFOS combined is based on the U.S. Environmental Protection Agency's November 2016 Health Advisory (https://www.epa.gov/ground-water-and-drinking-water/drinking-water-health-advisories-pfoa-and-pfos)

Each MCL/AGQS was calculated through a risk assessment process that: 1) assessed sensitive and human-relevant health effects of each PFAS in rodent models, 2) evaluated non-cancer endpoints due to uncertainty about cancer endpoints observed in rodent models, and 3) estimated health-protective doses for exposure to individual compounds across sensitive life stages. Under State law, development of MCLs necessitates evaluation of, and possible modification based on, the availability and accuracy of detection and treatment technology, as well as the costs associated with compliance. While these factors were considered, NHDES has determined that, for these compounds at this time, adjustments to the standards based on detection/treatment technology or projected compliance costs are not warranted, as both technology challenges and compliance costs can be addressed by means other than standards that do not adequately protect health. Therefore, NHDES has proposed the health-protective levels calculated using the science-based process as both the drinking water standard and the ambient groundwater standard for New Hampshire.

Animal studies, namely rodents, served as the basis for the derived dose of each MCL/AGQS. Human epidemiology studies were evaluated to identify relevant health effects observed in rodent models, but did not serve as the basis for dose calculation. The use of animal studies for risk assessment is consistent with the approach of other states (e.g., Minnesota, New Jersey, and New York) and federal agencies (EPA and ATSDR). Due to differences in methodology, exposure history and data reporting, the existing human epidemiological studies alone were determined to be insufficient for deriving the dose for MCL/AGQS in a manner that would be consistent with other drinking water standards. Although a novel method for epidemiology-based risk assessment has been applied by a single European agency (European Food Safety Authority 2018), this approach is self-acknowledged to either overestimate or underestimate reference doses and has not been adopted by other U.S. regulatory bodies.

The critical health effects selected from the toxicology literature were non-cancer endpoints, including liver enlargement (PFOA and PFNA), delayed development (PFOS) and impaired reproduction (PFHxS). Recognizing that epidemiological studies have identified associations between certain PFAS and cancer, NHDES also considered the feasibility of deriving a MCL/AGQS for a cancer endpoint using its standard risk assessment approach. Of the four PFAS assessed by NHDES, only PFOA had a study for consideration of a cancer-based endpoint. However, this study (Butenhoff et al., 2012) had technical limitations that hinder extrapolation of serum doses, as well as uncertainty regarding the biological relevance to humans. Thus, it was determined that there was insufficient information to conduct an accurate risk assessment for a cancer endpoint given the existing scientific literature. This has similarly been studied by both EPA and ATSDR, and both determined that if a cancer endpoint would have been chosen, the resulting standard would have been at a higher (less protective) level and therefore, the endpoint chosen is fully protective for all health effects.

Due to the current lack of information on the toxicity of PFAS mixtures, NHDES conducted its risk assessment for each compound on an individual basis. There is emerging evidence that suggests various PFAS may affect similar organ systems, but these effects occur at differing doses depending on experimental design and their relative potency has not been quantified. To address this concern for mixture effects, other states have exercised a risk management strategy, instead of risk assessment, by applying a combined standard for the sum total of multiple PFAS. While perceived as protective, this risk management strategy lacks a scientific basis as the combined toxicity of multiple PFAS is poorly understood. As there is uncertainty about the specific health effects of PFAS and the growing number of different PFAS identified in the environment, the scientific and practical merits of any risk management approach should be carefully

evaluated as an alternative to standard risk assessment. NHDES continues to study developments in scientifically based approaches to regulating combinations of PFAS.

Consistent with the previous points, Michigan recently released a report summarizing the challenges for deriving health-based standards for PFAS under the current risk assessment paradigm. This report was prepared by an independent panel of scientists from government and academic institutions with technical expertise on PFAS health effects, exposure and remediation. Given the current limitations of animal studies and human epidemiology, the expert panel recommended developing regulatory approaches that consider both of these lines of scientific evidence. Yet they did not provide technical guidance on how that might be achieved. The panel also stated that the non-cancer endpoint of PFAS seem to be more sensitive than cancer endpoints and may be more important for setting regulatory limits. Furthermore, the panel emphasized caution in using combined regulatory approaches due to the lack of quantitative evidence for assuming similar potency of different PFAS. Additional discussion of these technical issues and their relation to the derivation of the proposed MCL/AGQS are detailed in Appendices 3-7.

Finally, it is important to note the toxicity values for the MCL/AGQS were derived from the lowest doses in animal studies that were determined to be relevant to human health. This included selection of health effects associated with developmental delays from *in utero* exposure (i.e. PFOS), or other effects that occur at lower doses than those that induce developmental defects in animals (i.e., liver toxicity for PFOA and PFNA, and impaired reproduction for PFHxS). To afford additional protection for chronic exposure, daily water intake was assumed to be that of the 95th percentile for lactating women, which is the highest water in-take rate for adults (i.e., for a 175 lb. person, this would equal about 4.4 liters of water consumed each day. By using this rate of water intake to calculate the MCLs, the levels are expected to be safe for pregnant mothers and their fetuses, lactating mothers and their infants, and all children, adolescents, and adults). This high intake rate was assumed "through life" as a protective measure.

3. Occurrence, Ability to Reliably Quantify and Ability to Treat

The statute concerning how the State develops MCLs was amended in 2018 to clarify that New Hampshire's process should align with the process followed by EPA and most of the few other states that set MCLs. This section addresses three of the criteria that the law now requires be considered in the development of an MCL. It is important to note that no additional resources were provided to NHDES to produce information on these considerations or for cost and benefit estimates. Accordingly, NHDES used available data and work done under other investigations/projects or by others to address these aspects of determining a MCL.

3.1 Occurrence in Drinking Water

In New Hampshire, two contaminated sites, one involving contamination of Portsmouth, New Hampshire's municipal water system wells at the Pease Tradeport and another involving contamination of wells used as a source of water for Merrimack Village District in Merrimack, New Hampshire, raised awareness of these compounds and led NHDES and others to perform state-wide sampling at public water systems and other suspected sites. Based on these data, PFOA, PFOS, PFHxS, and PFNA occur in drinking water, groundwater and surface water in New Hampshire in proximity to releases of these contaminants to the environment. The following table describes the results of analysis for these chemicals at 402 of the 1,880 sources of drinking water that supply non-transient public water systems in New Hampshire.

Table 2: PFAS Concentrations Detected in Sources of Drinking Water for Non-Transient Public Water Systems (data provided by NHDES Sampling or PWS voluntary sampling conducted March 2016 to December 2018)

	Number of PFAS Sources			
Concentration (ppt)	**PFHxS**	**PFNA**	**PFOS**	**PFOA**
Not Detected	357	390	336	253
Detected at less than 10 ppt	35	6	47	125
10-20 ppt	2	3	14	13
20-40 ppt	7	3	2	8
40-60 ppt	1	0	2	0
Greater than 60 ppt	0	0	1	3

3.2 Ability to Reliably Quantify in Drinking Water

The following excerpt from the Association of State Drinking Water Administrator's PFAS Lab Testing Primer (https://www.asdwa.org/wp-content/uploads/2018/10/ASDWA-PFAS-Lab-Testing-Primer-10-10-18-Final.pdf) describes the current status of the ability to reliably quantify PFAS, including the four subject compounds, in drinking water:

"Laboratory analytical methods with reporting limits (RL) of at least 2-4 nanograms per liter (ng/L) parts-per-trillion (ppt) should be utilized. Many commercial labs are achieving reporting limits of less than 1 ng/L ppt. Additional health studies are rapidly evolving and some states have determined that PFAS health advisory concentrations in drinking water should be based on the additive effect of PFAS compounds. Obtaining water quality results with low RL will improve the utility of the data in the event health guidance or standards are changed or that the state you are in develops health guidance or standards based on the additive effects of PFAS.

It is important to understand the difference between a reporting limit (RL) and a detection limit (DL). An RL or reporting detection limit is the limit of detection in which the concentration of a contaminant can be reliably quantified. In contrast, the DL or method DL is lower than the RL and is below the point of calibration such that results reported below the RL are unreliable and as such, must be qualified as estimated values by carrying a "J" or "E" (NELAP) qualifier/flag."

Typical PFAS Reporting Limits	
Method 537	Range from 2.9 to 14 ng/L
Isotope Dilution	Varies by lab and compound but can be: • Below 1 ng/L for some compounds and • Up to 3 ng/L for others

3.3 Ability to Treat Drinking Water

Based on published literature, PFOA, PFOS, PFNA and PFHxS can be removed from drinking water with varying success using a number of treatment options. The most common treatment for PFAS removal, both in the literature and in practice, including at wells in New Hampshire, is granulated activated carbon (GAC). Data from a variety of sites, including at full-scale and fully operational municipal wells, clearly demonstrate that compliance with the proposed MCLs can be achieved using GAC or other approaches such as combining GAC with resin.

4. Costs to Affected Parties

NHDES used available water quality data to estimate potential costs to affected parties of compliance with the MCLs/AGQSs. For certain types of waste and groundwater discharge sites, this involved determining the frequency of exceeding the proposed standards for the sites sampled and applying that to the universe of sites. For other types of sites for which there are limited data, a qualitative description of anticipated costs is provided. As noted previously, with existing resources and expertise, NHDES was unable to analyze costs in keeping with EPA and Office of Management and Budget guidance, which entails determining costs associated with a number of different potential standards and capturing marginal costs.

For affected parties such as public water systems, landfill and hazardous waste site owners, and groundwater discharge permittees, NHDES had sufficient sampling data to estimate a cost range associated with setting these standards. In the case of affected public water systems that have already made significant investment in meeting the current AGQS, these costs were not included as new costs resulting from setting the standards. In the case of waste and discharge sites, where only initial sampling has occurred, the costs of compliance with the existing and new standards are included. The assumptions and analysis used to derive costs is included as an appendix to this report.

4.1 Estimated Costs to Public Water Systems to Comply with New MCLs

The MCLs for PFOA, PFOS, PFNA and PFHxS will apply to PWSs that serve residential populations (community PWSs) and those that serve the same 25 or more people each day for at least 6 months of the year (non-transient, non-community PWSs), such as schools and places of work with their own wells. There are currently 1,880 sources of water for PWSs that would be subject to the adoption of these MCLs. The costs incurred by these PWSs include the cost of routine sampling, the frequency of which will depend on compliance with the MCLs. For public water systems that exceed any of the MCLs based on a running annual average, the costs will also include treatment such as GAC, and operation and maintenance costs associated with the installed treatment. The methodology and assumptions made for estimating each of these costs is contained in Appendix 9. To summarize, NHDES estimated the following:

The initial cost of sampling for PWSs is estimated to be $1,102,500 - $2,836,000. Based on the anticipated percentage of detections, the costs of sampling for non-transient PWSs in year 2 thru 9 after the MCLs are established are estimated to be $73,055 - $184,825.

To date, sampling has occurred at 402 of the 1,880 sources of non-transient public drinking water in New Hampshire (see Table 2 in the Occurrence in Drinking Water subsection). Comparing these analytical results to the proposed standards allows estimation of the number of public water systems that will require treatment. The cost of treatment at PWSs associated with these standards is estimated to range from $1,800,000 - $5,200,000.

NHDES utilized operation and maintenance estimates from PWSs that have developed cost estimates for maintaining PFAS treatment systems under construction to comply with the current PFOA and PFOS 70 ppt combined AGQS to estimate operation and maintenance costs associated with the new MCLs. Operation and maintenance costs are estimated to range from $114,912 - $223,439 per year.

New Hampshire does not require drinking water not supplying public water systems to comply with MCLs. However, it is anticipate that homeowners and others with private wells will incur costs to ensure

their drinking water meets health based standards. NHDES estimates that 3,125 of the 250,000 private wells in New Hampshire will have drinking water that exceeds the MCLs. The cost of point-of-entry treatment for those wells is estimated to be $9,375,000, with an annual maintenance cost of $2,812,500.

4.2 Estimated Costs to Comply with New and Existing AGQS

4.2.1 Municipal Solid Waste Facilities (Groundwater Management/Release Detection Permits)
The vast majority of the unlined/lined solid waste disposal facilities or synthetic lined waste water treatment lagoons in New Hampshire are municipally owned, and as such, the municipality is responsible for maintaining the water quality systems and monitoring water quality associated with a permit. There are roughly 200 of these facilities that currently have groundwater release detection or groundwater management permits that have been issued by NHDES, in accordance with its administrative rules. These permits prescribe programs for periodic groundwater quality monitoring and reporting, provide for groundwater remediation either through active measures or natural attenuation, specify performance standards for remedies, and describe procedures for performing site investigations and implementing remedial action plans.

NHDES has required sampling for PFAS at all of these sites. To date, 58% have sampled and approximately 42% of those have exceedances of the current AGQS for PFOA and/or PFOS. Based on the proposed MCLs, 44% are estimated to have exceedances. NHDES has assumed that 25% to 50% of these sites will require either an expansion of the existing groundwater management zone where PFAS is already an established contaminant of concern (COC) or will require investigation where PFAS will become a new COC. The capital costs are estimated to be in the range of $380,000 - $755,000, and the annual operating costs could range from $260,000 - $390,000 per year. This includes assumptions concerning the cost to install additional monitoring wells, comply with permit sampling and reporting requirements, sample private wells and provide treatment to some percentage of the private wells tested, and administration of the permits. The worksheet that includes the assumptions and unit costs is provided in Appendix 10.

4.2.2 Hazardous Waste Remediation Sites (Groundwater Management Permits)
Hazardous waste remediation sites include all sites where a hazardous substance or waste has been released, and often have a long-term remediation and management component prescribed and regulated through an NHDES-issued groundwater management permit or remedial action plan. There are roughly 515 waste sites, including State-listed hazardous waste, CERCLA, and brownfields sites, that have an open status and are currently regulated by NHDES.

NHDES has required waste sites that meet certain criteria to complete an initial screening for the presence of PFAS. To date, 27% have sampled and approximately 49% of those have exceedances of the current AGQS for PFOA and/or PFOS. Based on the proposed MCLs, 53% are estimated to have an exceedance. NHDES has assumed that 25% to 50% of these sites will require either an expansion of the existing groundwater management zone where PFAS is already an established COC or will require investigation where PFAS will become a new COC. Assuming these percentages of non-compliance for the universe of waste sites, with the exceptions noted below, the capital costs are estimated to be in the range of $1,150,000 - $2,310,000 and the annual operating costs could range from $570,000 - $1,020,000 per year. Not included in the estimate above are costs associated with a few unprecedented, large-scale site investigations and associated response actions currently ongoing in southern New Hampshire to mitigate PFAS-contaminated drinking water. Response

actions at these sites have included providing treatment or alternative water sources to affected properties. Based on site-specific data collected to date, it is estimated that the proposed MCLs will result in an expanded area requiring investigation and additional properties requiring sampling and treatment. The additional capital costs unique to these southern New Hampshire sites are estimated to be in the range of $1.52M - $2.53M and the additional annual operating costs could range from $220,000 - $365,000 per year.

The cost estimates for waste sites include assumptions concerning the cost to install additional monitoring wells, comply with permit sampling and reporting requirements, sample private wells and provide treatment to some percentage of the private wells tested, and administration of the permits. The worksheet that includes the assumptions and unit costs is provided in Appendix 10.

4.2.3 Oil Remediation Sites (Groundwater Management Permits)
Oil remediation sites include all sites where long-term remediation and management of petroleum contamination occurs primarily through a NHDES-issued groundwater management permit or remedial action plan. There are approximately 1,500 active petroleum sites, including, but not limited to, leaking underground/above ground storage tank sites, and spill sites that have an open status and are currently regulated by NHDES.

NHDES has recently undertaken an initiative requesting a small initial subset of these petroleum sites to voluntarily complete an initial screening for the presence of PFAS. To date, only an estimated 1% of all petroleum sites have sampled for PFAS. The data indicate that some percentage of sites will have exceedances of the proposed MCLs. However, based on the limited nature of information and the types of releases/release mechanisms associated with petroleum sites, the capital and annual costs associated with the proposed MCLs is indeterminate at this time.

4.2.4 Wastewater Disposal to Groundwater (Groundwater Discharge Permits)
A number of municipalities and some private entities dispose of wastewater to the ground through such practices as discharges to lagoons, rapid infiltration basins, spray irrigation systems and very large leach fields. There are 96 of these facilities that currently have a groundwater discharge permit, which allows the discharge in accordance with rules that protect against impact to other properties and wells. NHDES has required sampling for these and other PFAS at all of these sites. To date, 44% have sampled and, of those, 29% have exceeded one or more of the proposed MCLs. Assuming this same percentage of non-compliance for the universe of sites, the capital costs are estimated to be approximately $1,100,000 and the annual operating costs are estimated in the range of $200,000 - $400,000. This includes assumptions concerning the cost to install additional monitoring wells at these sites, sample private wells and provide treatment to some percentage of the private wells tested. Given the variety of groundwater discharge sites and that wastewater discharge volumes at many permitted facilities are on the order of hundreds of thousands of gallons per day, available treatment technologies would not suitably treat these flows in a manner that is cost effective. The worksheet that includes the assumptions and unit costs is provided in Appendix 11.

4.2.5 Biosolids and Sludge Processing and Application Sites and Septage Land Spreading.
Biosolids are produced by municipally owned wastewater treatment facilities when they receive a sludge quality certification from NHDES approving the material for beneficial use as a fertilizer in New Hampshire. Some industrial sludge, such as short paper fiber or water treatment residuals, may also be approved for land application for their organic content or ability to bind phosphorous,

respectively. Before biosolids or sludge can be applied to the land for agricultural purposes, they must receive a Sludge Quality Certification that ensures that over 159 potential contaminants are at acceptable levels, following strict screening guidelines that protect groundwater and human contact. Until a leaching standard (the amount that can be in the biosolid or sludge without its land application resulting in an exceedance of AGQS) is set for these four PFAS, it is impossible to quantify the costs resulting from establishing these standards. In some cases, biosolids and sludge that are now being applied for beneficial purposes (i.e., fertilizer or organic material) may no longer be able to be used and communities and industry may see a rise in their biosolid and sludge disposal costs. A similar cost increase could occur at the five domestic septage (i.e., material pumped from residential septic tanks) land spreading sites if PFOA, PFOS, PHNA, PFHxS are found to leach into groundwater at unacceptable levels (i.e., causes an exceedance of AGQSs set for the four PFAS).

At the present time, New Hampshire has only one biosolids processing site that must sample and comply with the four PFAS AGQSs that are established as a result of setting the MCLs. This facility is currently sampling for PFAS, specifically to comply with the existing combined standard for PFOA and PFOS of 70ppt. The new AGQSs may require the installation of new sampling wells and modification of the facility to protect groundwater by controlling and treating runoff, etc. These costs are unknown at this time. This facility primarily serves municipalities and any increase in costs is likely to be reflected in increased tipping fees paid for by the New Hampshire municipalities who utilize this facility.

4.2.6 Fire Station/Fire Foam Sites

A known source of PFAS in the environment is the use of certain formulations of firefighting foams, referred to as Class B foam or aqueous film-forming foam (AFFF), which contains PFAS. Certain fire training areas and discrete locations across the state where AFFF has been applied historically are currently undergoing remedial investigation and/or cleanup of PFAS-contaminated groundwater. The recent discovery of contamination in drinking water wells at fire stations has prompted additional sampling in the vicinity of those fire departments, and has resulted in the detection of elevated PFAS concentrations in nearby private and public drinking water supply wells. Of the 16 fire departments that have sampled their private water supply wells and provided results to NHDES, five (or 31%) would exceed the proposed MCLs.

Based on review of available information, there are an estimated 293 fire stations in New Hampshire of which potentially just over 175 may be serviced by a private water supply well. Furthermore, information suggests that there are over 120 active public water supplies and potentially over 4,600 private wells within 1000 feet of a known fire station. Given the limited information, the capital and annual costs associated with the existing AGQS and the proposed MCLs is indeterminate at this time.

4.2.7 Air Deposition Sites

In addition to instructing NHDES to set MCLs, which in turn become AGQSs, for PFOA, PFOS, PFNA, PFHxS, SB 309 also require the agency to limit air emissions from facilities that cause or contribute to an exceedance of an AGQS and otherwise address the contamination caused. It is not possible to determine the number of facilities that have emissions that cause or contribute to contamination above the AGQS(s) or the costs associated with treatment, investigation and remediation.

NHDES has identified one current and one former industrial facility that have emissions that resulted in the exceedance of the current AGQS for PFOA and PFOS and is evaluating Best Available Control

Technology for PFAS emissions for the current facility. Estimated capital costs for the control devices under consideration range from $2,000,000 - $3,000,000 with annual operating costs of $200,000 - $400,000. In addition, the facility would be subject to air emission stack testing that could cost approximately $100,000 per test, depending on testing methodologies employed. Other potentially affected parties include:

1. Facilities with evaporators used to reduce the volume of liquid wastes if the liquid contains PFAS compounds.

2. Landfill gas (LFG) emissions at solid waste landfills, if it is determined that LFG contains PFAS. Further study as to the effectiveness of combustion of LFG in boilers, engines, turbines or flares as well as current treatment occurring at some LFG to energy facilities would be necessary to identify the impact from this potential source.

3. Other industrial facilities identified as using PFAS where emissions to air might be of concern. Specifically, this could be chrome plating operations that historically used PFAS mist suppressants.

4.2.8 Miscellaneous Sources

Highly fluorinated chemicals can be found in commercially available products and that are used in households, institutions and commercial and industrial facilities. Examples of items that *may* contain PFAS include but are not limited to:

1) Paints.
2) Sealants, including products used on grout, countertops and floor treatments.
3) House cleaners and stain removers.
4) Floor wax removers.
5) Stain-resistant textiles (or chemicals used to treat textiles in homes and businesses) including, but not limited to, carpets, shoes and clothing.
6) Furniture with stain-resistant fabric.
7) Water proof textiles.
8) Food cooking ware and utensils.
9) Ski and boat waxes.
10) Dental floss, cosmetics, sunscreen and other personal care products.
11) Construction materials, including caulk sealants and plumbing sealants.
12) Pesticides.
13) Treated paper.
14) Chemical coatings for metal roofing.
15) Solar panels.
16) Purchased garden soils.
17) Automotive supplies, including waxes, cleaners, windshield wipers and additives to fluids used in automobiles.
18) Camping and other outdoor gear.
19) Spray- and grease-based lubricants.
20) Inks.

The possible presence of PFAS in these items not only presents other exposure potential for PFAS to individuals in the home and at businesses, but also another potential source of contamination to

wastewater, groundwater, storm water and/or surface water. NHDES lacks sufficient data to estimate the potential costs to facility owners of addressing contaminated sites that result from the use of these products.

5. Benefits to Affected Parties

In general, it is difficult to quantify the monetized benefits for environmental and public health standards, and often the case is made that EPA's guidance on deriving benefits for MCLs underestimates benefit, particularly in the area of indirect costs such as reduced quality of life for both the sick individual and their family caregivers. *Contingent valuation,* which is a survey-based economic method for valuing non-market resources (e.g., asking people what they would pay to lower the risk of an adverse health outcome) is a widely accepted economic method to evaluate benefits in such cases as establishing a MCL when reduction in risk can be reasonably quantified. Contingent valuation is based on the economic principle that value equates to willingness to pay. Unfortunately, the type of information needed to use contingent valuation is not yet available for PFAS. While PFOA, PFOS, PFHxS and PFNA have clearly been associated with numerous adverse health outcomes in animals, the mechanism for, and risks related to, similar outcomes in humans are not well understood. Accordingly, NHDES currently has no quantified value of benefit, although there is likely significant benefit to reducing exposure to these compounds through drinking water given the findings of the few previous direct exposure studies and the emerging findings from current epidemiological studies. Qualitatively, given the potential for direct health care treatments costs, associated losses of economic production and income of those impacted, and associated impacts to families and caregivers, limiting exposure to PFOA, PFOS, PFNA and PFHxS at unsafe levels may result in numerous and significant avoided costs.

NHDES researched the subject of benefit quantification and spoke with experts, including a group of professors and researchers at the University of New Hampshire (UNH), with whom NHDES recently contracted to quantify the benefits of reducing the arsenic MCL. NHDES intends to further evaluate the possibility of quantifying benefit of these standards with the group at UNH to see whether studies exist or emerge that would allow the department to do so. In addition, through previous stakeholder engagements, a number of stakeholder groups have been engaging with other research institutions throughout the United States to find recent methods or studies that can help quantify the benefits.

APPENDICES

Appendix 1: Senate Bill 309-FN- Final Version

Below is an image of the final bill text of Senate Bill (SB) 309-FN- Final Version. Please visit the following webpage for an HTML or PDF version of the final bill text:
http://gencourt.state.nh.us/bill_status/Results.aspx?q=1&txtbillnumber=SB309&txtsessionyear=2018

CHAPTER 368
SB 309-FN - FINAL VERSION

03/08/2018 0973s
12Apr2018... 1310h
26Apr2018... 1580h

2018 SESSION

18-2838
08/10

SENATE BILL ***309-FN***

AN ACT regulating groundwater pollution caused by polluting emissions in the air and relative to standards for perfluorochemicals in drinking water, ambient groundwater, and surface water.

SPONSORS: Sen. Innis, Dist 24; Sen. Bradley, Dist 3; Sen. Avard, Dist 12; Sen. Fuller Clark, Dist 21; Sen. Gannon, Dist 23; Sen. Ward, Dist 8; Sen. Carson, Dist 14; Sen. Birdsell, Dist 19; Sen. Feltes, Dist 15; Rep. Messmer, Rock. 24; Rep. H. Marsh, Rock. 22; Rep. Emerick, Rock. 21; Rep. Bean, Rock. 21; Rep. Murray, Rock. 24

COMMITTEE: Energy and Natural Resources

AMENDED ANALYSIS

This bill:

I. Allows the department of environmental services to make rules regarding air pollution and the deposit of such pollutants on soils and water.

II. Regulates devices emitting or having the potential to emit air pollutants that may harm soil and water through the deposit of such pollutants.

III. Clarifies the basis for and requires periodic review of ambient groundwater quality standards.

IV. Directs the department to evaluate the ambient ground water quality standards for perfluorooctanoic acid (PFOA) and perfluoroctanesulfonic acid (PFOS) and set ambient groundwater quality standards for perfluorononanoic acid (PFNA) and perfluorohexanesulfonic acid (PFHxS).

V. Establishes the criteria for setting maximum contaminant limits for public drinking water and directs the department to set maximum contaminant limits for perfluorooctanoic acid (PFOA), perfluoroctanesulfonic acid (PFOS), perfluorononanoic acid (PFNA), and perfluorohexanesulfonic acid (PFHxS).

VI. Establishes a toxicologist position and a human health risk assessor position in the department of environmental services and makes an appropriation to fund the positions.

VII. Directs the department to develop a plan, including a schedule and cost estimates, for establishing surface water quality standards for perfluorooctanesulfonate (PFOS), perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), and perfluorohexanesulfonic acid (PFHxS) in class A and class B waters.

CHAPTER 368
SB 309-FN - FINAL VERSION

Explanation: **Matter added to current law appears in *bold italics.***
Matter removed from current law appears [~~in brackets and struckthrough.~~]
Matter which is either (a) all new or (b) repealed and reenacted appears in regular type.

CHAPTER 368
SB 309-FN - FINAL VERSION

03/08/2018 0973s
12Apr2018... 1310h
26Apr2018... 1580h

18-2838
08/10

STATE OF NEW HAMPSHIRE

In the Year of Our Lord Two Thousand Eighteen

AN ACT regulating groundwater pollution caused by polluting emissions in the air and relative to standards for perfluorochemicals in drinking water, ambient groundwater, and surface water.

Be it Enacted by the Senate and House of Representatives in General Court convened:

368:1 New Subparagraph; Rulemaking; Air Contaminant Impacts on Soil and Water. Amend RSA 125-C:4, I by inserting after subparagraph (s) the following new subparagraph:

(t) The determination of air contaminants subject to regulation, applicability thresholds, determination of best available control technology, and procedures to determine potential impacts of the deposit of such contaminants from the air on soils or water resources to implement RSA 125-C:10-e.

368:2 New Section; Requirements for Air Emissions of Perflourinated Compounds Impacting Soil and Water. Amend RSA 125-C by inserting after section 10-d the following new section:

125-C:10-e Requirements for Air Emissions of Perfluorinated Compounds Impacting Soil and Water.

I. For the purposes of this section:

(a) "Best available control technology" means "best available control technology" as defined in RSA 125-C:10-b, I(a).

(b) "Ambient groundwater quality standard" means "ambient groundwater quality standard" as defined in RSA 485-C:2, I.

(c) "Surface water quality standard" means "surface water quality standard" established in or pursuant to RSA 485-A.

(d) "Perfluorinated Compounds" or "PFCs" means the list of compounds identified in paragraph 1.1 of Environmental Protection Agency Document #: EPA/600/R-08/092 Method 537. "Determination of Selected Perfluorinated Alkyl Acids in Drinking Water by Solid Phase Extraction and Liquid Chromatography/Tandem Mass Spectrometry (LC/MS/MS)", Version 1.1 (September 2009).

(e) "Precursor" means any substance that has been shown by sound science to be transformed into a PFC under ambient conditions reasonably expected to occur in New Hampshire.

II. A device that emits to the air any PFCs or precursors that have caused or contributed to an exceedance of an ambient groundwater quality standard or surface water quality standard as a result of the deposition of any such PFCs or precursors from the air, shall be subject to the determination and application of best available control technology. Within 6 months of the department determining that the device is subject to such control technology, the owner of the device shall submit to the department an application for a permit. Within 12 months of permit issuance, the applicant shall complete construction and installation of controls consistent with the permit. Operation of the source may continue through the permitting, construction, and installation time period. A source which can demonstrate to the department that its device no longer contributes to an exceedance of an ambient groundwater quality standard or surface water quality standard shall be exempt from this section.

III. The construction, installation, or modification of any device that has the potential, based on an applicability threshold adopted by the department, to cause or contribute to an exceedance of an ambient groundwater quality standard or surface water quality standard as a result of the deposition of any PFCs or precursors from the air, shall be prohibited without first applying for and obtaining a permit from the department that establishes emission limitations for such device based on best available control technology.

IV. Part of the initial application for a permit under this section shall include an analysis of best available control technology for controlling emissions. Any permit issued shall contain inspection, testing, and reporting requirements, as applicable, to ensure the conditions of the permit are met.

V. Any determination of best available control technology under this section shall be subject to the following:

(a) In no event shall application of best available control technology result in:

(1) Emission of any air contaminant that would exceed the emissions allowed by any applicable standard under RSA 125-C or RSA 125-I or rules adopted pursuant to either chapter.

(2) Emission of any air contaminant subject to this section in an amount disproportionate to the emissions of such air contaminant from other similar air pollution control devices for that air contaminant at facilities using similar technology.

(3) Emission of any air contaminant subject to this section which causes or contributes to or has the potential to cause or contribute to an exceedance of an ambient groundwater quality standard or surface water quality standard, as a result of the deposition of the contaminant from the air.

(b) If the department determines that the facility has more than one device

that emits air contaminants subject to this section, the department shall determine best available control technology emission limitations for each such device.

VI. This section shall only pertain to PFCs for which at least one study has been conducted in accordance with generally accepted scientific principles that demonstrates that the PFC of concern is known to cause or may reasonably be anticipated to cause acute, chronic, mutagenic, reproductive, or developmental health effects in humans as a result of exposure to such PFC. The implementation of this section shall only rely upon standards that are based on federal maximum contaminant levels, health advisories, provisional health advisories, standards that are derived from federally published toxicological data, or more restrictive New Hampshire state standards.

368:3 New Subparagraph; Statement of Purpose. Amend RSA 485:1, II by inserting after paragraph (h) the following new subparagraph:

(i) Adopt primary drinking water standards by establishing maximum contaminant limits or treatment techniques.

368:4 Drinking Water Rules. Amend RSA 485:3, I(b) to read as follows:

(b) ***After consideration of the extent to which the contaminant is found in New Hampshire, the ability to detect the contaminant in public water systems, the ability to remove the contaminant from drinking water, and the costs and benefits to affected parties that will result from establishing the standard, a*** specification for each contaminant of either:

(1) A maximum contaminant level that is acceptable in water for human consumption[~~, if it is feasible to ascertain the level of such contaminant in water in public water systems~~]; or

(2) One or more treatment techniques or methods which lead to a reduction of the level of such contaminant sufficient to protect the public health, if it is not feasible to ascertain the level of such contaminant in water in the public water system; and

368:5 New Subdivision; Perfluorochemicals. Amend 485 by inserting after section 16-d the following new subdivision:

Perfluorochemicals

485:16-e Perfluorochemicals. By January 1, 2019, the commissioner shall, in consultation with the commissioner of the department of health and human services and other interested parties, initiate rulemaking in accordance with RSA 541-A to adopt a maximum contaminant limit for perfluorooctanoic acid (PFOA), perfluoroctanesulfonic acid (PFOS), perfluorononanoic acid (PFNA), and perfluorohexanesulfonic acid (PFHxS).

368:6 Ambient Groundwater Quality Standards. Amend RSA 485-C:6 to read as follows:

485-C:6 Ambient Groundwater Quality Standards.

I. The commissioner shall establish and adopt ambient groundwater quality standards for regulated contaminants which adversely affect human health or the environment. Ambient groundwater standards shall apply to all regulated contaminants which result from human operations or activities, but do not apply to naturally occurring contaminants. ***Where state maximum contaminant levels have been adopted under RSA 485:3, I(b), ambient groundwater quality standards shall be equivalent to such standards.*** Where federal maximum contaminant level or health advisories have been promulgated under the Federal Safe Drinking Water Act or rules relevant to such act, ambient groundwater quality standards shall be [~~equivalent to~~] ***no less stringent than*** such standards. ***The commissioner may adopt standards more stringent than federal maximum contaminant levels or health advisories if, accounting for an adequate margin of safety to protect human health at all life stages, including but not limited to pre-natal development, the commissioner determines federal standards are insufficient for protection of human health.*** Where such standards are ***established*** based upon ***health advisories that address*** cancer risks, the ambient groundwater quality standards shall be equivalent to that exposure which causes a lifetime exposure risk of one cancer in 1,000,000 exposed population. Where no federal ***or state*** maximum contaminant level or health advisory has been issued, the commissioner may adopt ambient groundwater quality standards on a basis which provides for an adequate margin of safety to protect human health and safety.

II. ***Health advisories that are adopted as ambient groundwater quality standards shall be reviewed by the department at least every 5 years to determine if new research warrants revising the current ambient groundwater quality standard. If the department finds a revision is necessary it shall conduct rulemaking to adopt the revised standard.***

III. Ambient groundwater quality standards shall be the water quality basis for issuance of groundwater discharge permits under RSA 485-A: 13.

[~~III.~~] ***IV.*** Except for discharges of domestic wastewater regulated under RSA 485-A:13 and RSA 485-A:29, no person shall violate ambient groundwater quality standards.

V. By January 1, 2019, the commissioner shall, in consultation with the commissioner of the department of health and human services and interested parties, initiate rulemaking to adopt ambient groundwater quality standards for perfluorononanoic acid (PFNA) and perfluorohexanesulfonic acid (PFHxS).

VI. By January 1, 2019, the commissioner shall, in consultation with the commissioner of the department of health and human services and interested parties, conduct a review to determine whether current research warrants revising the existing ambient groundwater quality standards for perfluorooctanoic acid (PFOA) and

perfluorooctanesulfonic acid (PFOS).

368:7 Department of Environmental Services; Positions Established; Appropriation. There is established within the department of environmental services one classified toxicologist position and one classified human health risk assessor for the purposes of developing appropriate standards to protect groundwater and drinking water quality under RSA 485-C. The sum necessary to pay the salary, benefits, and other costs related to the positions established in this section is hereby appropriated to the department of environmental services for the biennium ending June 30, 2019. This appropriation shall be in addition to any other appropriations made to the department in the biennium. The governor is authorized to draw a warrant for said sum out of any money in treasury not otherwise appropriated.

368:8 Department of Environmental Services; Surface Water Quality Standards. The commissioner of environmental services shall develop a plan, including a schedule and cost estimates, to establish surface water quality standards for perfluorooctanesulfonate (PFOS), perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), and perfluorohexanesulfonic acid (PFHxS) in class A and class B waters for all designated uses. The commissioner shall submit the plan upon its completion, but no later than January 1, 2020, to the house resources, recreation, and development committee and the senate energy and natural resources committee.

368:9 Effective Date.

I. Sections 1 and 2 of this act shall take effect 60 days after its passage.

II. The remainder of this act shall take effect upon its passage.

Approved: July 10, 2018
Effective Date:
I. Sections 1 and 2 shall take effect September 8, 2018.
II. Remainder shall take effect July 10, 2018.

Appendix 2: The Basic Steps Used by NHDES Environmental Health Program to Propose Health Based Drinking Water Standards for Perfluoroalkyl Substances

The Basic Steps Used by NHDES Environmental Health Program to Propose Health Based Drinking Water Standards for Perfluoroalkyl Substances

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Step 1:

Find a **no observed adverse effect level** (**NOAEL**) or **lowest observed adverse effect** (**LOAEL**) for the **critical health effect** in an animal study. Usually in units of milligrams of chemical/kilograms of animal body weight/ day (mg/kg/day).

NOAEL/LOAEL = To be protective against all other toxic effects, the critical effect (s) occurring at the lowest NOAEL is usually chosen. If even the lowest dose in the animal study has an effect, then the LOAEL must be used.

Critical health effect = adverse health effect in animal that is relevant to humans; generally occurs at very low exposures.

Step 2:

NOAEL/LOAEL dose (mg/kg/day) goes into a **pharmacokinetic model** = **point of departure (PoD in mg/kg/day)**

Pharmacokinetic model = model to convert an animal dose to a human exposure dose based on physiological parameters of each and knowledge of how chemicals act in the body (metabolism)

PoD = human dose (mg/kg/day) that is starting point for developing a toxicity value (100% of the safe chemical dose)

If no pharmacokinetic model exists, 2nd choice is a **dosimetric adjustment factor (DAF)** to go from NOAEL/LOAEL to PoD.

DAF = ratio of human half-life of chemical in the blood to the animal half-life of chemical in the blood.

Step 3:

PoD (human dose in mg/kg/day)/total uncertainty factors (UFs) = Reference Dose (RfD) or Minimal Risk Level (MRL in (mg/kg/day).

RfDs and MRLs are the same. Just different terminology used by EPA and ATSDR.

UFs = adjustment factors used when knowledge about a chemical's toxicity or effect on animal and human's is incomplete. UFs are usually either 10 or 3. Examples of common UFs: going from an animal study to a human exposure; accounting for human variability and sensitivity; if the lowest dose in an animal study still has an effect (no NOAEL); if a short-term study is used to develop a drinking water standard to protect against effects from long-term exposure, if the usual required studies such as developmental or cancer studies to understand how a chemical affects different life stages are missing (called a database deficiency UF).

RfD/MRL = the total safe non-cancer dose of a chemical to a human (mg/kg/day)

Step 4:

RfD/MRL (mg/kg/day) X Receptor (exposure factors) = drinking water equivalency level (DWEL in micrograms per liter (µg/L)

Receptor (exposure factors) = the sensitive exposed person used in the calculations (infant, young child, adult, pregnant or lactating woman) and their applicable bodyweight in kilograms and water ingestion rate in Liters/day.

DWEL (µg/L) = 100% of the safe dose expressed as the concentration in water for the receptor chosen.

Step 5:

DWEL (µg/L) /relative source contribution factor (RSC) = proposed drinking water standard (µg/L)

RSC = accounts for exposure to the chemical from sources other than drinking water. Examples are exposure from air, food, soil, non-ingestion drinking water exposure, such as breathing in the chemical when bathing (if the chemical is volatile) and absorption through the skin when bathing.

EPA guidance states that the highest RSC should be 80% (ceiling) and the lowest RSC should be 20% (floor). If there are sufficient data to calculate an RSC, one should be calculated. If data are insufficient, EPA recommends using the floor of 20% as a default value.

If data exist to calculate an RSC, EPA guidance recommends using **average** exposure values, not high-end.

For PFAS and some other chemicals, data on background exposure to humans has been collected and analyzed. CDC conducts the National Health and Nutrition Examination Survey (NHANES) to determine the nutritional and health status of the U.S. population. From blood samples of randomly selected volunteers, NHANES analyzes for several chemicals. In general, blood is not collected from the very young (less than 6 years of age). PFOA, PFOS, PFHxS, and PFNA are among the chemicals analyzed in blood serum by NHANES.

NHANES data are one of the best sources of background chemical exposure data for calculating an RSC. This is especially true for PFAS because of the long half-lives in human blood for many PFAS. Examples – PFOA half-life = 2.3 to 3.8 years; PFOS half-life = 5.4 years; PFHxS half-life = 8.5 years; PFNA half-life = 2.5 years).

NHANES has PFAS blood data results analyzed from 1999 through 2013-14. Because use of PFOA and PFOS has been phased out over time in the U.S., the concentrations found in the U.S. population by NHANES have been declining for years. See the Table below for the first and most recent PFAS sample results:

Concentrations in blood serum in micrograms per liter (µg/L = parts per billion (ppb))

Collection year	PFOA		PFOS	
	Geometric Mean Concentration	95th Percentile Concentration	Geometric Mean Concentration	95th Percentile Concentration
1999-2000	5.2	11.9	30.4	75.6
2013-2014	1.94	5.57	4.99	18.5

Geometric mean = 50% of the results are above and 50% are below this value. 95th percentile = 95% of the results are below and 5% are above this value.

Appendix 3: Technical Considerations for Health-Based Risk Assessment & References

Appendix 3: Technical Considerations for Health-Based Risk Assessment & References

The following is a summary of certain technical factors considered by NHDES in the derivation of the MCL/AGQS for PFOA, PFOS, PFHxS and PFNA. It should be noted that NHDES conducted a focused review of the existing information based on recent reports from state and federal agencies, public comments from technical workshops and recently published studies. Appendices 3-7 are not an exhaustive summary of all studies evaluated by NHDES; rather, they are a summary of critical information needed to understand the process by which the proposed MCL/AGQS values were derived. As the study of PFAS is an evolving area of science, NHDES is monitoring for emerging studies that would change the current understanding of PFAS-related health effects. NHDES will reevaluate the proposed standards if studies are published that demonstrate new and strong evidence for re-evaluating the toxicity values used to derive the currently proposed values.

In deriving the standards, there were two major technical considerations that influenced the NHDES evaluation of studies and selection of health effects. The first is discussion of issues related to the mechanism(s) of action associated with effects in animals and in vitro human models. The second was the determination to utilize non-cancer endpoints given the limited amount of information available for carcinogenicity of these specific PFAS.

Mechanism of Action

A mechanism of action is the biochemical process that allows a chemical to cause a physiological response. Mechanisms of action vary between chemicals and could include: interactions with receptors, interference of enzymes, mimicking of hormones or the formation of chemical bonds with biomolecules like cellular proteins or DNA. For toxicologists, knowledge about a chemical's mechanism of action is crucial for evaluating toxicity and relevance toward human health. Some mechanisms of action are unique to certain species or groups of animals and may have limited relevance to human health. If the mechanism of action is unknown, it is difficult to demonstrate a causal relationship between a chemical and a human health effect, even if there are associations.

Currently, there is no consensus in the scientific literature for the mechanism of action by which PFAS elicit their effects. There are two categories that the suspected mechanisms and their underlying studies can be classified into. The first mechanism is the activation of nuclear receptors, such as the peroxisome proliferator-activated receptor subtype alpha (PPARα). Activation of PPARα leads to peroxisome proliferation and oxidative stress in rodents, and altered lipid metabolism in humans. The second proposed mechanism is the induction of cellular stress and mitochondrial dysfunction independent of PPARα. The current literature presents evidence for both pathways, with more publications that focus on PPARα activation. Recent studies have sought to evaluate the role of PPARα-independent pathways in PFAS-related effects. It should be noted that the following summary does not seek to define a known mechanism of action for PFAS, as this is beyond the scope of the NHDES risk assessment. Rather, it is an overview of the issues surrounding the mechanism of action, which are critical to selecting appropriate health effects for risk assessment.

PPAR and Nuclear Receptor Mediated Effects

Peroxisome proliferator-activated receptor (PPAR) activation is the presumed mechanism of action for several forms of PFAS-induced toxicity in rodents. There are multiple isoforms of PPAR including subtypes alpha (α), beta (β) and gamma (γ), where PPARα is one of the most commonly studied isoforms in mammals. As nuclear receptors, PPARs are capable of initiating gene expression, thereby producing proteins that regulate lipid and energy metabolism (Issemann and Green, 1990; Lee et al., 1995). This includes elevating enzyme levels responsible for enzymatic-oxidation, ketogenesis, and lipoprotein metabolism (reviewed by Sertznig et al., 2007). Rodent studies demonstrate that PFAS exposure is associated with increased transcription of PPARα-regulated genes, palmitoyl CoA oxidase activity and perturbed lipid homeostasis and peroxisome proliferation (Perkins et al., 2004; Loveless et al., 2006; Rosen et al., 2007, 2008, 2017; Das et al., 2017; reviewed by ATSDR, 2018). An adverse side effect of this metabolic pathway is the generation of reactive oxygen species (ROS) that damage cellular structures and organelles, culminating in pathological effects observed in animal studies. PPARα activation in humans does not result in the same peroxisome proliferation effects, but does induce changes in lipid metabolism and gene transcription.

The role of PPARα in PFAS toxicity continues to be a major criticism against the use of rodent studies for human risk assessment (Klaunig et al., 2012). This criticism is based on quantitative and qualitative differences between rodent and human PPARα biology. When compared to humans, rodents overexpress PPARα by an approximate factor of 10 in certain tissues, namely the liver (Palmer et al., 1998; Corton et al., 2014). This overexpression of PPARα in rodents creates more molecular targets, thereby enhancing their sensitivity to PFOA and other PPARα agonists. Along with quantitative differences in the abundance of PPARα, structural differences between human and rodent PPARα enhance the sensitivity of rodents to certain PPARα agonists (Klaunig et al., 2003; Gonzalez and Shah, 2008; Tyagi et al., 2011). In light of these differences, *responses that are exclusively mediated by PPARα in rodents may overestimate toxicity for humans*.

The low expression of PPARα and other PPARs is not to be mistaken for lack of a functional role in human physiology. Human PPARs are involved in lipid and energy metabolism and are primarily expressed in liver, muscle, adipose tissues and certain cell types in the immune system (Tyagi et al., 2011). Hypolipidemic drugs such as fibrates act on human PPARs to manage clinically-high cholesterol levels (Brunton et al., 2011; Ferri et al., 2017). Some in vitro evidence shows that PFAS can activate human PPAR, albeit with less efficiency than rodent PPARs (Wolf et al., 2008). Additional studies are required to understand what role, if any, that PPARs play in human responses to PFAS.

Evidence from gene knock-out studies in mice (i.e., PPARα-null) and primates indicates that there are potentially PPARα-independent mechanisms of PFAS toxicity that involve other nuclear receptors (reviewed by Li et al., 2017a). The constitutive androstane receptor (CAR), estrogen receptor subtype-α (ERα), farnesoid X receptor (FXR), retinoid X receptor (RXR) and pregnane-X receptor (PXR) contribute to PFAS toxicity in wild-type and knock-out mice (Vanden Heuvel et al., 2006; Bjork et al., 2011; Rosen et al., 2017); albeit to a lesser degree in human cell models (Behr et al., 2018). Activation of these nuclear receptors can be influenced by activation of PPARα as ligand-bound nuclear receptors can form heterodimers (e.g. PPARα and RXR) with each other to initiate changes in gene expression (Evans and Mangelsdorf, 2014; Cave et al., 2016). Given the uncertainty about nuclear receptor and co-activator protein interactions, further research is needed before the role of other nuclear receptors in PFAS toxicity can be clearly demonstrated or refuted.

Non-Nuclear Receptor Mediated Effects

Aside from nuclear receptors, there is growing evidence that PFAS induce cellular dysfunction via PPARα-independent mechanisms. The alternative mechanisms with limited evidence include disruption of the: *i*) nuclear factor kappa(κ) B (NFκB) pathway, *ii*) intercellular gap-junction communication, *iii*) lipid membrane stability, and *iv*) mitochondrial signaling pathways (EPA 2016ab; Li et al., 2017a; ATSDR, 2018). Of these, recent evidence from rodent exposures and human cell lines points to disrupted mitochondrial signaling as a plausible PPARα-independent mechanism of PFAS toxicity.

Mitochondria are primarily responsible for maintaining chemical energy levels within cells through the production of ATP. Disruption of the mitochondrial membrane or proteins facilitating ATP production results in imbalanced energy metabolism and the formation of ROS. In response to this stress, cells will undergo programed cell death (apoptosis). In human HepG2 (hepatoma) cells, PFOA induces apoptosis that is preceded by ROS formation, loss of mitochondrial membrane potential and activation of the apoptosis regulating protein known as caspase-9 (Shabalina et al., 1999; Panaretakis et al., 2001; Yao and Zhong, 2005). Eriksen et al. (2010) reported a pronounced effect of PFOA and PFOS on ROS generation in HepG2 cells, but only PFNA was associated with DNA damage. In non-cancerous cell lines, Li et al. (2017b) documented dose-dependent apoptosis in HL-7702 (human liver) cells treated with PFOA (2,500-7,500 ppt). At these same doses they also observed increased production of caspase-9 and the formation of 8-hydroxydeoxyguanosine (8-OHdG), a marker of ROS damage to DNA. While the exact mechanism for mitochondrial dysfunction in human cells remains unidentified, there is evidence that both abnormal (i.e., cancerous) and normal *in vitro* cell lines are responsive to PFAS.

Beyond human cell lines, the mitochondrial effects of PFOA have been documented across a variety of *in vivo* models in the presence and absence of PPARα activation. Similar to human liver cells, PFOA-treated mice showed a dose-responsive increase in hepatic production of caspase-9 and 8-OHdG (Li et al., 2017b). Proteomic analysis of these mice found that ROS formation was independent of PPARα and likely due to suppression of proteins involved with ATP formation in the electron transport chain (ETC). Of note, these effects were observed following a 28-day *in vivo* exposure with average PFOA serum concentrations of 970 ng/mL. This pathway was associated with hepatic hypertrophy and signs of apoptosis.

In vitro animal studies have further substantiated PFAS-associated mitochondrial dysfunction. Suh et al. (2017) reported impaired mitochondrial metabolism combined with ROS formation in a rat pancreatic β-cell line exposed to PFOA. Mitochondria isolated from the livers of male rats and treated with various PFAS showed reduced membrane potential that was attributed to destabilization of lipid structures and subsequently enhanced ion exchange; however, this was at concentrations above extreme occupational exposures for individual PFAS (Starkov and Wallace, 2002). Compared to other PFAS, PFOS showed the most potent inhibitory effect on mitochondrial respiration in an isolated system (Wallace, 2013). In isolated rat mitochondria, Mashayekhi et al (2015) found that PFOA increased ROS generation, interfered with ETC complexes I, II and III activity and contributed to collapse of mitochondrial membrane potential. Additionally, there is some evidence for mitochondrial effects across broader classes of vertebrates including fish (Hagenaars et al., 2013; Cui et al., 2015). The ubiquity and conservative evolution of mitochondria makes this pathway potentially more relevant to human health than PPARα, but further research is needed before this can be confirmed, or excluded, as a mechanism of action for PFAS.

Conclusions

Current evidence suggests that the effects of PFAS in animal models may be due to various mechanisms of action, where activation of PPAR is critical for advanced toxicity observed in rodents. The latter PPAR-independent pathways have only recently received as much research attention as PPARα and *require further investigation*. As stated by EPA's own Health Advisory for PFOA (2016a) and PFOS (2016b), there is no known unifying mechanism of action for the wide-array of effects associated with PFOA, PFOS and other PFAS. Yet, there is some evidence that these compounds affect biological targets in animals and humans and thus does not preclude the necessity for assessment of the myriad of health effects observed through animal studies and human epidemiology.

If all PFAS shared an identical molecular mechanism of action, a class-based MCL/AGQS would be a scientifically reasonable method for risk management. Such approaches have been applied to other chemical classes where there is a known and common mechanism of action (e.g., polychlorinated biphenyls). However, based on current literature, the only demonstrated common target for PFAS appears to be the activation of PPARα. If this is true for all PFAS, then rodent-derived toxicity values for a class of "PPARα activators" are 3-10x more protective, given the overexpression and sensitivity of PPARα in rodents relative to humans. However, this would mean that the Animal-to-Human Uncertainty Factor (discussed in the Derivation Appendices) of 3 that is used to derive the human doses may overestimate human sensitivity. As there is currently evidence for compound-specific effects through other nuclear receptors and PPAR-independent pathways, NHDES assessed the health impacts of each PFAS individually.

It should be noted that in conducting this assessment NHDES observed a potential bias in the current understanding of the mechanism(s) of action for PFAS. In older animal studies, there is a tendency to focus on PPARα-related enzyme activity without measuring other biochemical processes that would substantiate, or rule-out, other mechanisms of action. This is, in part, due to an under-utilization of methods for identifying mechanisms of action. This is not unreasonable, as current approaches for identifying pathways were once very cost prohibitive. High-throughput approaches that are readily applied in today's research laboratories were not well standardized until quite recently. Now, the rapidly changing technologies in molecular biology, and the fairly recent application of these tools for toxicological studies, are allowing a better understanding of subtle biological processes. *Although not currently available, NHDES expects that future studies will provide important information about the mechanism(s) of action that will be critical to identifying relevant human health risks associated with PFOA, PFOS, PFHxS, PFNA and other PFAS*.

Non-Cancer Versus Cancer Endpoints

NHDES risk assessment of PFOA, PFOS, PFHxS and PFNA used non-cancer health effects for derivation of toxicity values and subsequent MCL/AGQS values. This is due to a current lack of adequate information to derive reliable cancer-based toxicity values from animal studies. Human epidemiological studies show some associations between these PFAS and certain cancers, but these associations are inconsistent with limited data on serum concentrations required to confidently develop health-based guidance values. Of the four PFAS, the most information is available for PFOA and PFOS and is discussed below. To the best of NHDES' knowledge, there are currently no peer-reviewed rodent studies that evaluate the carcinogenicity of either PFNA or PFHxS. This precludes risk assessment for cancer-based endpoints for PFNA and PFHxS at this current time.

PFOA is classified as possibly carcinogenic to humans (IARC, 2016) based on evidence from the C8 Study population (Barry et al., 2013) and a limited number of toxicology studies that identified kidney and testicular tumors in rats (Butenhoff et al., 2012; Biegel et al., 2001). In the 2016 Drinking Water Health Advisory document, EPA found suggestive evidence of carcinogenic potential in humans (EPA, 2016a). In humans, Barry et al. (2013) found an increased risk of testicular cancer with estimated exposure to PFOA in a highly exposed population, but others have reported no association with testicular cancer (Vieira et al., 2013). Steenland and Woskie (2012) reported an increase in kidney cancer associated with modeled exposure to PFOA, whereas others have found no association (Leonard, 2006; Leonard et al., 2008; Barry et al., 2013; Raleigh et al., 2014). Inconsistencies in the epidemiological evidence are likely due to the limited information regarding PFOA exposure, which is modeled in some studies to address a lack of exposure history. Additional sources of variation likely include differences between populations in lifestyle and background exposure to other environmental agents. However, these studies are associative and cannot demonstrate causation for increased or decreased risks making these studies ill-suited for deriving toxicity values. Therefore, risk assessment for PFOA currently would rely on evidence from more controlled animal studies to determine a cancer-based toxicity value for MCL/AGQS derivation.

While the animal studies provide limited support for PFOA-induced testicular tumors, the study that includes a dose-response relationship suitable for risk assessment did not measure serum concentrations (Butenhoff et al., 2012). Due to the profound differences in the half-lives of PFAS between rodents and humans, this omission introduces a large measure of uncertainty, since orally-administered doses of PFOA do not result in the same serum levels across species. Different approaches for estimating the serum concentrations from this study result in vastly different toxicity value and subsequent health advisory numbers (EPA, 2016a; NJ DWQI, 2017). Furthermore, there is no known mechanism of action for the carcinogenic potential of PFOA, and some potential pathways have questionable relevance to human health. Thus, NHDES found the existing database to be inadequate for assessing carcinogenic potential of PFOA and utilized non-cancer endpoints.

Currently, there is little evidence linking PFOS to a specific human cancer with inconsistent associations reported from epidemiological studies. For example, PFOS was associated with breast cancer in a study of Inuit women in Greenland (Bonefeld-Jørgensen et al., 2011), yet a later study of a larger Danish cohort did not substantiate the association (Bonefeld-Jørgensen et al., 2014). A single animal study that evaluated carcinogenicity in rats observed an increased incidence of hepatocellular adenomas at the highest dose, as well as a small number of thyroid tumors that did not display a dose-response relationship. As PFOS is shown to be a PPAR-activator, the hepatic tumors are unlikely to be relevant to human health assessment (Klaunig et al., 2003; Corton et al., 2014), and are not supported by epidemiological evidence (Eriksen et al., 2009). Given this and the EPA conclusion that there was insufficient evidence to pursue a cancer endpoint for PFOS (2016b), NHDES did not select cancer as an endpoint for risk assessment of PFOS.

In its 2018 draft, ATSDR identified on-going studies sponsored by the National Institute of Environmental Health Sciences (NIEHS) that aim to identify the carcinogenic potential of PFOA. To date, NHDES is unaware of other research teams that are investigating the carcinogenicity of other PFAS. Related to this, an independent panel of scientists commissioned by the state of Michigan noted that:

> "Although cancer often receives more attention than other potential adverse health effects that may result from a toxicant exposure, based in part on the presumption that it is the most sensitive outcome, this is not always the case. Indeed, for PFOA and PFOS, developmental and immune

> effects seem to be among the most sensitive in both animal and human studies and may be more important for setting advisory and regulatory limits on exposure. Developmental, immune, and liver effects were often drivers for determining the recent advisory levels of PFOA and PFOS from EPA, ATSDR, and state agencies." - Michigan PFAS Science Advisory Panel (2018)

If additional studies are published that demonstrate human-relevant mechanisms for carcinogenicity, combined with sufficient data for reliable and accurate extrapolation, NHDES recommends re-assessment of the proposed toxicity values.

Appendix 4: PFOA Derivation

Appendix 4: PFOA Derivation

Toxicity Endpoint: **Altered Liver Weight and Function**

Of the four PFAS for which MCL/AGQS values were derived, PFOA has the largest body of scientific literature for evaluation. Despite a large number of epidemiological studies showing a variety of associated health effects, these studies did not provide sufficient information for derivation of reference doses based on the recommended guidelines used by NHDES. However, NHDES did evaluate the human health literature to identify health endpoints with the greatest weight of evidence to narrow its search to animal studies with similar effects.

In humans, prolonged exposure to PFOA has been associated with alterations in markers of hepatic function and lipid metabolism. In the 2018 draft report, ATSDR found current epidemiological studies provide adequate evidence for alterations in serum levels of hepatic enzymes, as well as elevations in serum lipids (i.e., total and LDL cholesterol). A recent analysis of the current epidemiological literature by a team at the Australian National University found inadequate evidence for altered liver function in response to PFAS, but identified sufficient evidence for association between PFOA and PFOS exposure with hypercholesterolemia (Kirk et al., 2018). Most recently, an independent panel of academic and government scientists agreed with ATSDR's assessment of associations between PFAS exposure and liver enzyme levels (Michigan PFAS Science Advisory Panel, 2018), although additional research is needed to determine if such changes in these clinical markers translate into liver disease following chronic exposure.

As a critical health effect, altered liver weight and function are potentially adaptive, meaning they are expected to recede in the absence of the stimulating chemical. Hall et al. (2012) contend that such adaptive effects should not serve as the basis for risk assessment as the effect is dependent on continuous exposure. Kirk et al. (2018) suggest that any adverse effect related to changes in cholesterol metabolism and downstream effects may not be of public health relevance due to treatability. However, the NHDES risk assessment process assumed that the MCL/AGQS should allow for prolonged water consumption without the need for recovery from an adaptive response in the liver or associated effects on lipid metabolism. Furthermore, the relatively long half-lives of PFOA, and other PFAS, in humans prolong exposure on a scale of months to years making such depurations suspect. Thus, the NHDES risk assessment of PFOA evaluated and selected increased relative liver weight in rodents as a sensitive precursor effect for altered liver function and changes in lipid metabolism.

Several research teams have evaluated the hepatotoxicity of PFOA in non-human primates, rodents and other non-mammalian model organisms. Hepatotoxicity is of particular interest as PFOA concentrations are frequently higher in the liver than circulating serum levels. Furthermore, considerable resources have been dedicated to investigating the hepatic effects of PFOA across *in vitro*, *in vivo* and epidemiological studies. This is due to concern for prolonged liver damage and its implications for chronic diseases, such as non-alcoholic fatty liver disease. However, indicators of hepatotoxicity in animal models may be overly sensitive when compared to human biology due to PPARα activation, making outcomes like liver cancer in rodents less relevant to human health (Hall et al., 2012). Given the suggestive evidence for liver impacts in humans, NHDES evaluated the consistency of adverse hepatic outcomes across animal studies and their relevance to human health as determined by PPARα-independent effects.

One the most consistently documented responses to PFOA across rodent models is hepatic hypertrophy. As reviewed by Hall et al. (2012), hepatic hypertrophy has various connotations including increases in the i)

organ weight, ii) average size of hepatocytes, and iii) expression levels or activity of hepatic enzymes (also referred to as functional hypertrophy). The occurrence of any one of these forms of hepatic hypertrophy alone may not indicate liver toxicity. This is due to rodent-specific sensitivity in the activation of cellular responses that are mediated by the PPARα pathway. Thus, the presence of multiple forms of hepatic hypertrophy in animals and evidence for a non-PPARα mechanism of action would suggest hepatotoxicity that is relevant to humans. Regarding PFOA, there is evidence for multiple forms of hepatic hypertrophy in animal models, summarized below. As mentioned in Appendix 3, the mechanism of action was evaluated and it was determined that liver hypertrophy could be associated with non-PPARα mechanisms.

Several studies have demonstrated that exposure to PFOA through food or water induces increased liver weights in mice and rats (reviewed by EPA 2016 and ATSDR, 2018, and references therein). This is associated with changes in hepatocellular structure that include hepatocellular hypertrophy, cytoplasmic vacuolization, necrosis, signs of apoptosis and persistent changes in liver structure following prenatal exposure (Griffith and Long, 1980; Butenhoff et al., 2004a; Loveless et al., 2008; Son et al., 2008; Cui et al., 2009; Elcombe et al., 2010; Yahia et al., 2010; Wang et al., 2013; Quist et al., 2015; Li et al., 2017b). Changes in clinical chemistry markers, including alanine aminotransferase (ALT) and aspartate aminotransferase (AST), can be observed after exposure to drinking water laced with PFOA (21-d; Son et al., 2008). Others reported no changes in ALT and AST despite the occurrence of liver necrosis in rodents (Kennedy et al., 1985), suggesting that AST and ALT may not be accurate indicators for chronic disease in rodents (Hall et al., 2012). Additionally, hepatic hypertrophy from PFOA is associated with reductions in circulating cholesterol levels in rodents (Haughom and Spydevold, 1992; Loveless et al., 2006, 2008; Elcombe et al., 2010; Quist et al., 2015ab). While hypocholesterolemia is the opposite effect of that generally seen in epidemiological studies, hypercholesterolemia has been observed in PFOA-exposed rodents that are also fed a high-fat or Westernized diet (Tan et al., 2013; Rebholz et al., 2016).

As discussed in Appendix 3, recent studies indicate that there are PPARα-independent pathways associated with altered liver size and function making the hepatic effects in rodents relevant to human health risk assessment.

In primates, Butenhoff et al. (2004b) used male cynomolgus monkeys to assess liver toxicity from ammonium perfluorooctanoate (APFO) at 3, 10 and 30 mg/kg/d over the course of 26 weeks. They observed increased absolute liver weights, although relative liver weight (liver weight relative to body weight) was only significantly elevated at the highest dose, along with serum triglycerides and thyroid hormones. Consistent with other primate studies using cynomolgus monkeys (Thomford, 2001) and Rhesus monkeys (Griffith Long, 1980), no histological changes were observed in the liver. A lack of change in hepatic palmitoyl CoA oxidase activity at all but the highest dose led the authors to conclude that peroxisome proliferation did not play a role in the observed toxicity. The authors also noted that:

> “increase in liver weights seen following the administration of APFO to cynomolgus monkeys was, at least in part, due to hepatocellular hypertrophy (as demonstrated by decreased hepatic DNA content) which in turn may be explained by mitochondrial proliferation (as demonstrated by increased succinate dehydrogenase activity).” - Butenhoff et al. (2004b)

The strength of these observations is limited by inherent challenges with primate research including a limited sample size combined with high inter-individual variability in wild-caught animals (as referenced by the need to determine age by dentition). Additional issues in this study add greater scrutiny, such as

changes in the high-dose treatment mid-way through the experiment and attrition of animals from what were assumed to be non-treatment-related causes (Butenhoff et al., 2004b).

Consideration of Other PFOA-Related Effects from Animal Studies

As outlined by EPA (2016), National Toxicology Program (NTP 2016) and the draft assessment by ATSDR (2018), PFOA has also been shown to affect the functions of the immune, thyroid and reproductive systems, along with effects on early growth and development. The sensitivity of early life stages requires additional consideration regarding developmental effects associated with PFOA. As discussed below, EPA based its 2016 Health Advisory for PFOA on developmental delays in mice following an in utero exposure to PFOA (Lau et al., 2006; EPA, 2016). Another developmental endpoint of concern is delayed mammary gland development, which has been a contentious endpoint in recent health-based risk assessments of PFOA. Most regulatory bodies have deferred from its use as a critical health endpoint given uncertainty about its functional significance and relevance to human health. Given concerns for developmental outcomes, NHDES decided it was important to detail its decision not to use these health endpoints as the basis for PFOA's reference dose.

Early-life exposure to PFOA elicits responses from a variety of physiological systems and age-dependent-processes. Rodent responses to in utero, perinatal, lactational or peripubertal exposures include: pre- and post-birth loss of pups, reduced neuro-motor activity, delays in developmental hallmarks, reduced bone ossification and impaired growth (Butenhoff et al., 2004a; Lau et al., 2006; White et al., 2007; Wolf et al., 2007; Hu et al., 2010; Onishchenko et al., 2011; White et al., 2011; Albrecht et al., 2013; Cheng et al., 2013; Quist et al., 2015ab; Koskela et al., 2016). The variety of developmental endpoints reflects experiments using both standardized and non-traditional toxicological endpoints. The use of different rodent strains, routes of administration and exposure periods makes it difficult to discern common effects. However, a meta-analysis of seven fetal growth studies estimated a negative relationship between PFOA and rodent pup weight, where body mass is reduced by 0.23 g per 1 mg/kg/d increase in PFOA (Koustas et al., 2014). Together, there is evidence that PFOA is detrimental to growth and development in rodent models.

EPA and ATSDR considered certain developmental impacts of PFOA to be sufficient critical effects for their derivation of final and draft reference doses, respectively. The developing fetus is often more sensitive to chemical insults meaning that standards based upon developmental exposures in mice or rats, spanning gestation and subsequent window of lactation, are considered protective for sensitive subpopulations (EPA, 2016a). In both cases, EPA and ATSDR selected studies that reported alterations in bone development, along with additional developmental effects unrelated to the skeletal system. However, there were stark differences between these studies in their suitability for human health risk assessment.

Lau et al. (2006) evaluated the pre- and post-natal effects of in utero PFOA exposure in CD-1 mice. Developmental effects were observed in pups across all doses (1-40 mg/kg/d), where the lowest dose was associated with reduced bone ossification, precocious male puberty, and increased weight gain in later life. Higher doses (10-20 mg/kg/d) were associated with increased incidence of full fetal reabsorption, microcardia, delayed eye-opening, as well as reductions in fetal survival, birth weight. At 40 mg/kg/d there was a complete loss of pregnancy in all treated mice. Lau et al. (2006) concluded that reduced ossification of the forelimb phalanges (long-bones of the paw) was the most sensitive endpoint in prenatally-exposed pups. A weakness of this study was the lack of information regarding PPARα activity, or other biochemical

measures, that might have pointed to a mechanism of action for developmental toxicity. A good experimental design, adequate sample sizes and thorough characterization of fetal growth and survival were strengths of the study, making it a credible basis for risk assessment.

Another developmental study, presented across two publications (Onischenko et al., 2011, Koskela et al., 2016), reported behavioral and skeletal changes in C57BL/6 mice. This study used a single dose level of PFOA (0.3 mg/kg/d) based on the lowest effect doses estimated by Lau et al. (2006), and exposed the mice throughout gestation (Onischenko et al., 2011). It is not explicitly stated when, but, somewhere between 5-8 weeks of age the mice were evaluated for locomotor activity and changes in circadian rhythms, then again at 3-4 months for coordination and muscle strength. Onischenko et al. (2011) found that PFOA exposure was associated with a decrease in the number of inactive periods in group social settings. However, there was no effect on other endpoints including novelty exploration, anxiety and coordination. In a subsequent analysis of the bones from these same mice, Koskela et al. (2016) reported changes in bone morphology in the PFOA-exposed mice when compared to controls. These effects were subtle, and the authors even acknowledged that these morphological changes might be due to increased body-weight of PFOA-treated mice. They augmented their study with a dose-response experiment using *in vitro* osteoblast cells that showed some PFOA-induced changes in metabolism, altered nuclei features and relative gene expression (Figures 5 and 6 of Koskela et al., 2016). The observations for morphological features, organ weights and birth defects were poorly characterized in this study, only reporting a significant increase in the absolute liver weight of PFOA-exposed pups (Onischenko et al., 2011) and significant body weight gains in treated adults (Koskela et al., 2016). At best, this study demonstrated that the lowest effect dose estimated by Lau et al. (2006) for neonatal survival can be considered a LOAEL for behavioral, skeletal and liver weight effects of PFOA. The combined lack of a dose-response relationship, questionable statistical power and inadequate study design precluded these combined works from further consideration by NHDES.

It is noteworthy that the study by Onischenko et al. (2011) and Koskela et al. (2016) selected their PFOA singular dose based on the low doses for effects estimated by Lau et al. (2006). Of the biological effects observed in pups and their dams, the most sensitive response was the increased maternal liver weight and not the developmental delays observed in pups (Lau et al., 2006). Given PFOA's effects on hepatic function, oxidative stress and cholesterol metabolism, it is not unreasonable to question if these responses in the dam contributed to the developmental effects observed in pups. Thus, increased liver weight of the dam was the most sensitive response from a gestational exposure, not the developmental delays observed in pups.

Other animal studies provide limited insight into the developmental toxicity and teratogenicity of PFOA. Most studies have focused on morphological endpoints with little to no anchoring in biochemical or histological changes observed in exposed pups. This lack of molecular details with these observations raises challenges for interpreting their relevance for human health. The exception to this has been work by the National Toxicology Program that has evaluated the effects of PFOA on mammary gland development in mice.

Nine studies have evaluated altered mammary gland development in female mice following exposure to PFOA either during gestation, nursing/lactation or puberty (White et al., 2007; Yang et al., 2009; White et al., 2009; Zhao et al., 2010; Macon et al., 2011; White et al., 2011; Zhao et al., 2012; Albrecht et al., 2013; Tucker et al., 2015). All but one (Albrecht et al., 2013) have reported altered timing of mammary gland development in response to PFOA. This suggests a consistent biological effect in an animal model that is commonly used to study mammary gland development.

Mammary gland development starts in the fetus, followed by a second window of maturation during puberty in response to hormonal changes, and undergoes a third period of maturation in preparation for lactation (Rudel et al., 2011; Osborne et al., 2015). In animal models, this has been evaluated through subjective scoring of whole-mount tissues, as well as quantitative measures of gland-specific tissue structures such as tubules, terminal end buds and duct ends. Altered developmental timing of the mammary gland is a proposed susceptibility factor for an increased risk of mammary gland-related diseases, such as breast cancer (Rudel et al., 2011; Tiede and Kang, 2011; Macon and Fenton, 2013; Osborne et al., 2015). It should be noted that these references are not studies that demonstrate PFOA-associated delays in mammary gland development are a risk factor for breast cancer; rather, they are primarily reviews and perspectives of why this should be investigated. Aside from cancer outcomes, there is concern for detrimental impacts of altered mammary gland development on lactation and ability to adequately support nursing offspring.

In utero exposure to PFOA delays mammary gland development in female mice. White et al. (2007) evaluated fetal windows of susceptibility toward PFOA-induced delay in mammary gland development. They found that exposure to PFOA delayed mammary gland development in both pups and dams. In a follow-up study, White et al. (2009) demonstrated that intrauterine and/or lactational exposure to PFOA (5 mg/kg/d) delayed mammary gland development in CD-1 mice, emphasizing the sensitivity of the mammary gland during pre- and post-natal development. In a third publication, White et al. (2011) showed that gestational and chronic life exposure to PFOA (1, 5 mg/kg/d; some animals supplemented with 5 ppb-laced drinking water) leads to delayed mammary gland development in daughters and granddaughters of exposed CD-1 mice. From a functional standpoint, this had no significant effect on lactational support of their offspring despite the observed changes in gland structure (White et al., 2011) and milk-related gene expression (White et al., 2007). A related study characterized the internal dosimetry of PFOA treated CD-1 mice, showing that PFOA crosses the placenta and leads to delayed mammary gland development at relatively low serum concentrations (Macon et al., 2011).

Strain- and age-specific differences in mice affect whether there is a delay, acceleration or no effect on mammary gland development. Tucker et al. (2015) evaluated strain differences between CD-1 and C57BL/6 mice for susceptibility towards delayed mammary gland development after gestational exposure to PFOA (0.01-1 mg/kg/d). They found that both strains were susceptible to delayed mammary gland development but at different doses. Yang et al. (2009) compared strains of mice (Balb/c and C57BL/6 mice) for differences in PFOA's effect on peri-pubertal development of the mammary ducts, uterus and estrus cycling. Balb/c mice experienced delayed mammary duct development, and liver hypertrophy, whereas C57BL/6 mice experienced accelerated mammary gland development at 5 mg/kg/d and delayed development at higher doses. This effect has been speculated to be the result of differences between *in utero* and peri-pubertal exposure (Yang, 2009; Tucker et al., 2015).

This effect is possibly due to PPAR activation in mice. PPAR-associated binding proteins have been implicated in mammary duct development in mice models, as their inactivation results in delayed mammary gland development. Peroxisome proliferator-activated receptor-binding protein (PBP) is a transcription factor that supports the activation of PPARs, as well as other nuclear receptors (Zhu et al., 1997). Jia et al. (2005) showed that PBP is involved in normal mammary gland development in mice, and that its inactivation results in impaired gland function and responsiveness to hormone signals, as well as delayed development. This same research group reported that another PPAR coactivator protein was involved in delayed mammary gland development and impaired milk production in mice (Qi et al., 2004). Yang et al. (2006)

demonstrated that PPARα activation leads to delays in mammary gland development following treatment with a PPARα activator, or constitutive activation of PPARα in transgenic mice. Curiously, this same study found no delays in gland development of PPARα-null mice indicating that PPARα-activation is not necessary for normal mammary gland development. More recently, Albrecht et al. (2013) reported no effect of PFOA on mammary gland development in mice with normal PPAR function, humanized PPAR function or a loss of PPAR function (knock-out mice). This would suggest that the rodent-specific sensitivity of the PPAR pathway might be responsible for this critical effect. To date, the role of these proteins and PPAR-signaling on PFOA-induced delays in mammary gland development has not been clearly studied, nor is it clear if PPAR-activation during mammary gland development is of direct relevance to human health.

Aside from potential detriments to lactation, there is a concern for increased cancer risks due to abnormal mammary gland development. Rudel et al. (2011) argued that enhanced cancer susceptibility can be induced by delays in mammary gland development that lead to a higher number of terminal end buds, such as those seen within rats exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (Brown et al., 1998; Fenton et al., 2002). There is also evidence for concern from accelerated mammary gland development (reviewed by Tiede and Kang, 2011; Macon and Fenton, 2013; Osborne et al., 2015; and references therein). The problem with applying this is that PFOA is associated with a reduced number of terminal end buds, but the TCDD model is associated with an increased number of terminal end buds. This does not appear to align with mechanisms proposed in other reviews (Tiede and Kang, 2011; Macon and Fenton, 2013; Osborne et al., 2015). To date, we are unaware of any study that links the observed structural delays seen in mice after PFOA exposure with enhanced susceptibility toward carcinogenesis. If future evidence arises that addresses the shortcomings of this health endpoint and identifies clear linkage to human relevance, this endpoint should be re-assessed as a potential critical health effect of PFOA.

Other state agencies, including Texas and New Jersey, have considered delayed mammary gland development as a critical health effect towards setting regulatory limits. However, the two agencies reached starkly different numbers with this same biological endpoint. The New Jersey Drinking Water Quality Institute (NJ DWQI) calculated a reference dose that would have resulted in an MCL of < 1.0 ppt, although NJ DWQI ultimately selected increased relative liver weight and arrived at an MCL of 14 ppt. The NJ DWQI Subcommittee found the delay in mammary duct development concerning in their health-based risk assessment, but determined the limited existing information only supported justification of using a modifying factor of 10 out of precaution for this and other developmental impacts. The Texas Commission on Environmental Quality (CEQ) derived a protective concentration level (PCL) of 290 ppt based on delayed mammary gland development, although their estimations rely on the orally-administered dose instead of serum concentrations. EPA (2016) concluded there was insufficient evidence demonstrating that delays in mammary gland development resulted in a permanent adverse effect, thus excluded this critical effect for calculation of the current health advisory level of 70 ppt.

Animal Serum Dose: **4,351 ng/mL**

The reference study used to derive the animal serum dose was Loveless et al. (2006) that reported the responses of rodents (rats and mice) toward i) linear PFOA, ii) branched PFOA and iii) a mixture of linear and branched isoforms. PFOA was administered in the form of ammonium perfluorooctanoate (APFO) via oral gavage with APFO-treated water. All three forms of PFOA displayed hepatotoxic responses in male mice and

rats. Given the occurrence of different PFOA isoforms in the environment, it was decided that this study was well-suited for characterizing response to a relevant mixture of PFOA isoforms.

Loveless et al. (2006) reported serum concentrations for PFOA for both the LOAEL and NOAEL. When feasible, it is recommended to utilize benchmark dose (BMD) modeling to address technical uncertainties related to the use of NOAELs for determining a point of departure from animal studies (EPA 2002). Given the time required for *de novo* development and appropriate validation of BMD models, we deferred to the BMD model described by the NJ DWQI for the same study by Loveless et al. (2006) (methodology is summarized in NJ DWQI, 2017). Briefly, BMD analysis estimated the serum dose for a 10% increase in relative liver weight from a branched and linear mixture of PFOA. The average serum concentration for the lower 95% confidence limit (the BMDL) from the two best fit models was determined to be 4,351 ng/mL (NJ DWQI, 2017).

Uncertainty Factors (UF): Total UF of 100

A full UF of 10 was applied to account for differences in sensitivity and toxicokinetics (e.g., half-lives and elimination rates) across the human population. Given the uncertainty surrounding the exact mechanism(s) of action for PFOA, a partial UF of 3 was applied for rodent-to-human differences in toxicodynamics to account for unknown differences in sensitivity between humans and rodents for PPARα-independent effects. In practice, an additional UF can be applied to account for suspected differences in toxicokinetics between rodents and humans (i.e., half-life); however, the use of a dosimetric adjustment factor can replace this UF of 3. A UF of 3 was applied due to evidence for associated effects on other physiological systems including immune function observed in animal and human epidemiological studies.

UF 10 (Human-to-Human) x UF 3 (Animal-to-Human) x UF 3 (Other Toxicities) = Total UF 100

Note that an UF of 3 is a simplification of a half-log unit ($10^{0.5}$ = 3.16), where $10^{0.5}$ x $10^{0.5}$ = 10.

Dividing the Animal Serum Dose by the Total Uncertain give the Target Serum Level in humans.

Target Serum Level = Animal Serum Dose ÷ Total uncertainty Factor

43.5 ng/mL = 4,351 ng/mL ÷ 100

Dosimetric Adjustment: $1.20E^{-04}$ L/kg/d, assuming 2.7-year half-life

The dosimetric adjustment factor (DAF) estimates an externally administered (ingested) dose corresponding to the internal serum dose of concern (i.e., the Human Equivalent Dose). This is a necessary step since the half-lives of PFAS in rodents are profoundly shorter than the half-lives in humans. The NHDES approach is similar to the EPA method used for deriving the reference dose for PFOA (EPA, 2016). This approach requires a volume of distribution (V_d; 0.17 L/kg, Thompson et al. 2010) and the chemical's half-life ($t_{½}$) in humans.

$DAF = V_d$ x (Ln(2) ÷ $t_{½}$)

*$DAF = 0.17$ L/kg x (Ln(2) ÷ (2.7 y * 365 d/y)) = $1.1954E^{04}$ L/kg/d*

The half-life for PFOA was assumed to be 2.7 years, based on a recent study by Li et al. (2018). This study evaluated the half-lives of PFOA, PFOS and PFHxS in a population that was exposed to these compounds via drinking water. The strengths of this study included its sample size, relevance to drinking water exposure, inclusion of a broad age range (15-50) and balanced representation of both sexes. Amongst the 106 participants of the study, the average (± SD) serum concentration of PFOA was 21.1 ± 14.7 ng/mL. No difference was detected between the average half-life of PFOA in men and women from this study (Li et al., 2018).

Reference Dose (RfD): 5.2 ng/kg/d

The RfD is calculated as:

RfD = (Animal Serum Dose / Total UF) x DAF

RfD = (4,351 ng/mL ÷ 100) x 1.20E^{-04} L/kg/d = 5.2 ng/kg/d

This RfD is less than EPA's current RfD for PFOA (20 ng/kg/d) and greater than ASTDR's draft MRL for PFOA (3.0 ng/kg/d). This difference from both agencies in not unexpected as the NHDES assessment utilized a different study, a lower total uncertainty factor (100 versus 300 for both EPA and ATSDR) and a longer half-life for PFOA estimated from a non-occupational exposure.

It should be noted that in the RfD calculation there is no term that adjusts for the proportion of PFOA actually absorbed following ingestion. This is because NHDES assumed that 100% of the PFOA ingested from environmental sources is absorbed within the gastrointestinal tract. Although ingestion is the primary route of exposure to PFAS, the mechanisms and efficiency of uptake is poorly understood. This is a health-protective assumption as the actual uptake efficiency is currently unknown in humans (summarized by ATSDR, 2018), but may be less than 100% as indicated by animal studies following exposure through food or water.

Exposure Assumptions: Relative Source Contribution of 40%,
Water consumption rate for lactating women

The relative source contribution (RSC) for drinking water is typically set between 20-80%. When possible, the RSC is calculated using quantitative information for exposure from other sources such as air, food and soil. However, sufficient information is currently unavailable for accurate estimation of daily exposure to PFOA from non-drinking water sources such as food and inhalation. Thus, the cumulative background exposure to PFOA is estimated from serum concentrations in the general population.

In this assessment, the RSC was derived using the subtraction method in conjunction with the EPA decision tree for RSC determination (EPA, 2000). The subtraction method derives a RSC from the background level of exposure and the target serum level, where:

RSC = (Target Serum Level – Background exposure level) ÷ Target Serum Level

When population-specific data for background exposure are not available, it is recommended to utilize the average from datasets such as NHANES. The 2013-2014 NHANES report shows an average PFOA serum

concentration of 1.9 ng/mL for all ages, with a high end estimate (95th percentile) of 5.6 ng/mL for those age 12 years or older (NH HEALTH WISDOM, accessed December, 2018; ATSDR 2018). Utilizing either the average or the 95th percentile for exposure from the 2013-2014 NHANES data would result in an RSC >80%. However, more recent and population-specific data for serum PFOA concentrations are available for New Hampshire. Across adults and children (n=219) in Southern New Hampshire, the average and 95th percentile for PFOA serum concentrations were 4.4 ng/mL and 26.6 ng/mL, respectively (NH HEALTH WISDOM, accessed December, 2018). Based on the 95th percentile for New Hampshire-specific data, the chemical-specific RSC for PFOA was determined to be 40%.

RSC = (43.5 ng/mL – 26.6 ng/mL) ÷ 43.5 ng/mL = 0.38, rounded to 0.40 or 40%

NHDES calculated the exposure using the water ingestion rate of a lactating woman (0.055 L/kg d). This was based on the 95th percentile consumers estimate for combined direct and indirect community water ingestion for lactating women (EPA, 2011). The water ingestion rate of lactating women is greater than that of non-lactating women, pregnant women or men, and is therefore more protective as it over-estimates an individual's chronic exposure via drinking water.

MCL for PFOA: 38 ppt (ng/L)

The RfD is converted to an equivalent dose in drinking water by selecting a sensitive human receptor and using their drinking water ingestion rate to calculate a drinking water equivalency level (DWEL). The DWEL is 100% of a dose not expected to cause any toxic effects.

DWEL = RfD ÷ Water Ingestion Rate

DWEL = 5.2 ng/kg/d ÷ 0.055 L/kg d = 94.5 ng/L

Taken together with the RSC to account for background sources of exposure, the MCL is derived as follows:

MCL = (DWEL x RSC)

MCL = (94.5 ng/L x 0.40) = 38 ng/L

NHDES is currently reviewing emerging information for the impact the proposed MCL will have on serum concentrations relative to background sources of PFOA.

Appendix 5: PFOS Derivation

Appendix 5: PFOS Derivation

Toxicity Endpoint: **Developmental Delays**

After PFOA, PFOS is one of the most studied PFAS in the toxicological literature. Epidemiology studies associate PFOS with similar effects as PFOA, with some emphasis on developmental delays and immunotoxicity (as reviewed by NTP 2016; Rappazzo et al. 2017; ATSDR 2018; Liew et al. 2018), although it is noted that these latter effects in humans have been disputed (Chang et al. 2016; Negri et al. 2017). Based on more controlled rodent studies, PFOS has been shown to affect the liver, thyroid function, immune system and early development. Developmental delays were determined to be a sensitive and consistent critical effect for reference dose derivation, and concern for immunotoxic effects warranted a UF of 3, discussed below.

As with most PFAS outcomes, the epidemiological studies do not present a clear understanding for the relationship of PFOS and fetal growth and early life development. Most PFAS have been shown to readily cross the placenta, resulting in exposure levels reflecting the mother's blood concentration of PFAS. Of the studies identified by ATSDR (2018), three identified a significant negative association between maternal PFAS levels and low birth weight in infants (Washino et al. 2009; Chen et al. 2012; Maisonet et al. 2012). The 2018 ATSDR draft MRL found that other epidemiology studies have not detected significant effects on birth weight and early growth in infants, but meta-analyses across studies indicate a negative association between PFOS and other PFAS with growth and development (Koustas et al. 2014; Verner et al. 2015). Interpreting these associations in humans is difficult, in part, due to physiological changes in pregnant women that affect how the body clears chemicals like PFOS. To address this, Verner et al. (2015) conducted a meta-analysis of birth weight studies and adjusted for the kidney physiology (glomerular filtration rate) of pregnant women. Physiologically-adjusted analysis revealed that a 1 ng/mL increase in PFOS was associated with a 2.72 g reduction in birth weight. Although some individual studies currently present mixed observations for an effect of PFOS on growth, additional lines of evidence from animal studies support the observation of delayed growth and development following gestational exposure to PFOS.

Several toxicological studies have reported delayed development across different strains of mice and rats following pre- and post-natal exposure to PFOS (Yahia et al. 2008; Butenhoff et al. 2009; Rogers et al. 2014; Wan et al. 2014). In the study ATSDR used for evaluating PFOS, Onishchenko et al. (2011) observed decreased locomotor activity and coordination in adult mice with early-life exposure to PFOS. However, the limitations of this study are similar to those discussed for PFOA in Appendix 4. A comparative study between rats and mice found delayed growth in rat pups following gestational exposure, and the induction of several birth defects in both rodents and mice at higher doses (10-20 mg/kg/d; Thibodeaux et al. 2003; Lau et al. 2003). As concluded by the EPA (2016b), these and other studies support the selection of delayed development as a critical health effect for PFOS.

The reference study selected for deriving the MCL/AGQS was Luebker et al (2005ab), consistent with the EPA (2016) and ATSDR draft MRL for PFOS (2018). This two-generational study evaluated the long-term and reproductive impacts of PFOS on rats and their progeny (Luebker et al. 2005a). Female rats were treated prior to and throughout pregnancy and lactation, and pups birthed to these dams were continuously exposed throughout life. Some of these treated pups were switched with control pups to evaluate the specific role of exposure via gestation and lactation on early growth and development. Pups born to PFOS exposed dams displayed impaired growth, developmental delays and reduced survival. The LOAEL for the

developmental delays was 0.1 mg/kg/d based on transient delays in growth and delayed onset of eye opening. Maternal exposure was a major driver of the observed effects, as determined by cross-fostering of exposed and control animals and evaluation of serum concentration of PFOS in dams and pups (Luebker et al. 2005b). The transient effect on growth is argued to be of questionable significance. From a risk assessment perspective, given the protracted human half-life of PFOS when compared to rats, there is valid concern for what effect modest delays may have on developmental trajectories following in utero exposure.

Experiments using transgenic knock-out mice (PPARα) found the developmental effects of PFOS in rodents are likely PPARα-independent (Abbott et al. 2009). The study exposed mice during the late-stages of gestation and noted decreased survival in both types of mice. Similar to Luebker et al. (2005a), there was a delay in the time to eye opening in both wild-type and the PPARα knock-out mice. There was no transient delay in growth, which may be due to the differences in the start of maternal exposure (Abbott et al. 2009). Such evidence that developmental delays are a PPAR-independent effect further supports the selection of this critical endpoint.

Aside from developmental delays, PFOS is an immunotoxicant in rodent models. Evidence for this was reviewed and summarized by the National Toxicology Program in an assessment of PFOS and PFOA (NTP 2016). NTP found moderate evidence that PFOS was immunotoxic in humans, but had high confidence it was immunotoxic in rodents (NTP 2016). The difference in conclusion is not unexpected, as epidemiological studies in humans and toxicological studies in rodents provide different lines of evidence. The strength of the animal models for studying immunotoxicity is the amount of control the experimenter has for factors that may affect the high-sensitive responses of the immune system. For studies of PFOS and PFOA, the disadvantage of animal models has been the considerable species- and strain-specific differences in immunological responses. For a more thorough review on the effects of PFOS and other PFAS in animal models and their relation to human health outcomes, see DeWitt et al. (2012).

Epidemiology studies have identified varying associations for PFOS with immunomodulation (reviewed NTP 2016; ATSDR 2018), although these associations have been disputed for a variety of criteria (Chang et al. 2016). These effects include hyper-sensitivity, autoimmunity and immunosuppression. Of particular concern for public health is the association between PFOS, and other PFAS, with reduced vaccine response. The primary evidence for suppressed vaccine responses associated with PFOS has come from studies of a highly-exposed population in the Faroe Islands and evidence from the Norwegian birth cohort study (Grandjean et al. 2012; Granum et al. 2013; Kielsen et al. 2015; Looker et al. 2014). In the Faroese, PFOS has been specifically associated with decreases in diphtheria antibodies in children by the age of seven (Grandjean et al. 2012; Mogensen et al. 2015). In surveys of the U.S. population (NHANES), Stein et al. (2016) reported reduction in rubella and mumps antibodies associated with each doubling of serum PFOS concentrations. Re-analysis of similar data from the U.S. population using methods that account for biological differences between men and women found that PFOA was associated with reduced vaccine titers in adults, but there was no association between PFOS and vaccine titers in youths or adults (Pilkerton et al. 2018).

Currently, there is no known mechanism for the associated immunological effects observed in humans. This is a major challenge for scientifically demonstrating causality between PFOS, and other PFAS, with the associated immunomodulatory effects. The growing number of studies is highly suggestive that PFAS act as an immunomodulatory; however, the current evidence is not conclusive.

Despite there being a limited number of studies, there is evidence that PFOS is immunosuppressive in rodents. At low doses, B6C3F1 mice showed a suppressed response to sheep's red blood cells (sRBCs) (1.66

µg/kg/d for 28 days; Peden-Adams et al. 2008) and lower resistance to viral infection by influenza (25 µg/kg/d for 21 days; Guruge et al. 2009). Dong et al. (2009; 2011) evaluated immunosuppression in a different strain of mice following a 60-day exposure to PFOS. The NOAELs for suppressed antibody response from these two studies were 8.3 µg/kg/d (Dong et al. 2009) and 16.7 µg/kg/d (Dong et al. 2011), but these were determined using different assays with different low doses. While there is some evidence for suppressed antibody production, there are technical inconsistencies that limit its use for reference dose derivation and therefore justified an UF of 3.

In light of this evidence, an additional UF of 3 was applied to PFOS to address the potential for immunotoxicity observed in rodents at the NOAEL serum concentrations reported in Dong et al. (2011).

Animal Serum Dose: **6,260 ng/mL**

The animal serum dose used for deriving the MCL for PFOS was the same as that estimated by EPA (2016b) and Minnesota Department of Health (2017), which is based on the NOAEL for reduced pup body weight in the two-generation study in rats (Luebker et al. 2005a). In the 2016 Health Advisory for PFOS, EPA (2016b) summarizes the consistency of this serum dose with NOAEL and LOAEL values from other developmental delays associated with PFOS exposure. NHDES noted that the estimated serum concentration is based on an EPA model that utilized the data reported in Luebker et al. (2005ab).

Uncertainty Factors (UF): **Total UF of 100**

A full UF of 10 was applied to account for differences in sensitivity and kinetics across the human population. Given the uncertainty surrounding the exact mechanism(s) of action for PFOS, a partial UF of 3 was applied for rodent-to-human differences in toxicodynamics to account for unknown differences in sensitivity between humans and rodents toward PPARα-independent effects. In practice, an additional UF can be applied to account for suspected differences in toxicokinetics between rodents and humans (i.e., half-life); however, the use of a dosimetric adjustment factor can replace this UF of 3. An UF of 3 was applied due to concern for PFOS' effects on other physiological processes including the immune system (NTP 2016; and lipid metabolism (ATSDR 2018).; Perkins et al. 2018).

UF 10 (Human-to-Human) x UF 3 (Animal-to-Human) x UF 3 (Other Toxicities) = Total UF 100

Note that an UF of 3 is a simplification of a half-log unit ($10^{0.5}$ = 3.16), thus $10^{0.5}$ x $10^{0.5}$ = 10.

Dividing the Animal Serum Dose by the Total Uncertain gives the Target Serum Level in humans.

Target Serum Level = Animal Serum Dose ÷ Total uncertainty Factor

62.6 ng/mL = 6,260 ng/mL ÷ 100

Dosimetric Adjustment: **1.28E-04 L/kg/d, assuming 3.4-year half-life**

The dosimetric adjustment factor (DAF) estimates an externally administered (ingested) dose corresponding to the internal serum dose of concern (i.e., the Human Equivalent Dose). This is necessary since the half-lives

of PFAS in rodents are profoundly shorter than their half-lives in humans. The NHDES approach is similar to the EPA method used for deriving the reference dose for PFOS (EPA 2016). This approach requires a volume of distribution (V_d; 0.23 L/kg, Thompson et al. 2010) and the chemical's half-life ($t_{½}$) in humans.

$$DAF = V_d \times (Ln(2) \div t_{½})$$

$$DAF = 0.17\ L/kg \times (Ln(2) \div (3.4\ y * 365\ d/y)) = 1.2844E^{-04}\ L/kg/d$$

The half-life for PFOS was assumed to be 3.4 years based on the same study selected for the half-life of PFOA (Li et al. 2018). The strengths of this study included its sample size, relevance to drinking water exposure, inclusion of a broad age range (15-50) and balanced representation of both sexes. The average (±SD) serum concentration of PFOS was 387 ± 259 ng/mL amongst 106 participants. Unlike PFOA, there were sex-specific differences in the half-life of PFOS where the half-life in men was 4.6 years (95% CI 3.7-6.1 years) and for women was 3.1 years (95% CI 2.7-3.7 years). The average across both sexes was 3.4 years. NHDES used the reported average across both sexes as a more protective half-life for a lactating women.

Reference Dose (RfD): 8.0 ng/kg/d

The RfD is calculated as:

$$RfD = (Animal\ Serum\ Dose / Total\ UF) \times DAF$$

$$RfD = (6{,}260\ ng/mL \div 100) \times 1.28E^{-04}\ L/kg/d = 8.0\ ng/kg/d$$

This RfD is lower than EPA's current RfD for PFOS (20 ng/kg/d) and greater than the ATSDR's draft MRL for intermediate PFOS (2.0 ng/kg/d). The NHDES assessment utilized the same study as both agencies for the basis of the PFOS RfD development; however, there were differences in the application of Total Uncertainty Factors (EPA applied 30 and ATSDR applied 300) and a shorter half-life for PFOS based on a non-occupational exposure.

It should be noted that in the RfD calculation there is no term that adjusts for the proportion of PFOS actually absorbed following ingestion. This is because NHDES assumed that 100% of the PFOS ingested from environmental sources is absorbed within the gastrointestinal tract. Although ingestion is the primary route of exposure to PFAS, the mechanisms and efficiency of uptake is poorly understood. This is a health-protective assumption as the actual uptake efficiency is currently unknown in humans (summarized by ATSDR 2018), and may be less than 100% as indicated by animal studies following exposure through food or water.

Exposure Assumptions: **Relative Source Contribution of 50%,**
Water consumption rate of a lactating woman

Similar to PFOA, the chemical-specific RSC for PFOS was derived using the subtraction method in conjunction with the EPA decision tree for RSC determination (EPA 2000). The subtraction method derives a RSC from the background level of exposure and the target serum level, where:

RSC = (Target Serum Level – Background exposure level) ÷ Target Serum Level

When population specific data for background exposure is not available, it is recommended to utilize the average from datasets such as NHANES. The 2013-2014 NHANES report shows an average PFOS serum concentration of 5.0 ng/mL for all ages, with a high end estimate for the NHANES data shows a 95th percentile of 18.5 ng/mL for those age 12 years or older (NH HEALTH WISDOM, accessed December 2018; ATSDR 2018). Utilizing either the average or the 95th percentile for exposure from the 2013-2014 NHANES data would result in an RSC >80%. However, more recent and population specific data for serum PFOS concentrations is available for New Hampshire, specifically the Pease community. Across those in the 2016 Pease group (n=242), the average and 95th percentile for PFOS serum concentrations were 10.2 ng/mL and 31.7 ng/mL, respectively (NH HEALTH WISDOM accessed December 2018). Based on the 95th percentile for New Hampshire-specific data, the chemical-specific RSC for PFOS was determined to be 50%.

RSC = (62.2 ng/mL – 31.7 ng/mL) ÷ 62.2 ng/mL = 0.49, rounded to 0.50 or 50%

NHDES calculated the exposure using the water ingestion rate of a lactating woman (0.055 L/kg d). This was based on the 95th percentile consumers estimate for combined direct and indirect community water ingestion for lactating women (EPA 2011). The water ingestion rate of lactating women is greater than that of non-lactating women or men, and is therefore more protective as it over-estimates an individual's chronic exposure via drinking water.

MCL for PFOS: **70 ppt (ng/L)**

The RfD is converted to an equivalent dose in drinking water by selecting a sensitive human receptor and using their drinking water ingestion rate to calculate a drinking water equivalency level (DWEL). The DWEL is 100% of a dose not expected to cause any toxic effects.

DWEL = RfD ÷ Water Ingestion Rate

DWEL = 8.0 ng/kg/d ÷ 0.055 L/kg d = 145.5 ng/L

Taken together with the RSC to account for background sources of exposure, the MCL is derived as follows:

MCL = (DWEL x RSC)

MCL = (145.5 ng/L x 0.50) = 73 ng/L, rounded down to 70 ng/L

This was rounded down to 70 ppt to comply with the existing EPA Health Advisory for PFOS.

NHDES is currently reviewing emerging information for the impact the proposed MCL will have on serum concentrations relative to background sources of PFOS.

Appendix 6: PFNA Derivation

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Toxicity Endpoint: **Altered Liver Weight and Function**

Significantly less peer-reviewed literature is available for PFNA than PFOA and PFOS, with only slightly more studies than PFHxS. Relative to human epidemiological studies, PFNA has been studied in the context of exposure to multiple PFAS and is loosely associated with altered liver enzyme activity and potential effects on the immune system (as reviewed by ATSDR). However, PFNA-specific effects on human health are unknown as there remains insufficient information to draw conclusions about the human health effects from the observed associations (summarized by ATSDR 2018 and NJ DWQI 2018). Based on more controlled rodent studies, PFNA seems to have similar biological properties as PFOA as seen through effects on the liver, immune system and early development; although the degree to which these two are similar is poorly quantified. Limited data on PFNA results in greater uncertainty regarding PFNA-specific health effects and its relative potency when compared with similar PFAS.

Relatively fewer epidemiological studies have characterized the associations of PFNA with health outcomes. As with most PFAS, the existing literature is focused on changes with clinical measures of enzymes, hormones and blood chemistry with far fewer evaluating specific disease diagnoses. Many of the findings are conflicting, emphasizing the need for additional research to understand the effects, if any, PFNA has on human health (reviewed by ATSDR 2018). An example for how little is known about PFNA is the fact that there is no reported serum half-life for this compound. In developing the 2018 draft MRL for PFNA, ATSDR (2018) relied on estimated half-lives based on urine measurements (Zhang et al. 2013) which are less accurate than serum-derived half-lives. No associations have been found between PFNA and cancer.

Similar to PFOA, the most consistent effect observed in animal studies has been increased relative liver weight and altered lipid metabolism (Wolf et al. 2012; Das et al. 2015, 2017; Wang et al. 2015; Rosen et al. 2017). Wolf et al. (2012) showed that PFNA is a stronger activator of PPARα than PFOA using *in vitro* assays. As discussed in Appendix 3, a PPARα-dependent mechanism of toxicity may not be relevant to human health. Gene expression profiles show that PFNA does activate PPARα, but can also act on the liver via other nuclear receptors including PPARγ and the estrogen receptor (Rosen et al. 2017). In addition to liver toxicity, PFNA has been associated with immunotoxic effects in rodents following acute exposures (Fang et al. 2009), but these studies provide limited information for understanding chronic exposures or PFNA-related effects during early development.

The reference study used to derive the MCL/AGQS was Das et al. (2015) which characterized the toxicity of PFNA in pregnant CD-1 mice and their pups. This study was a follow-up to another toxicity study of PFNA that showed some of the adverse developmental impacts of PFNA were dependent on PPARα activation (Wolf et al. 2010). Similar to gestational exposure to PFOA (Lau et al. 2006), relative liver weights of pregnant and non-pregnant mice displayed dose-dependent increases with PFNA treatment. Fetal effects included increased fetal liver weight, reduced pup weight and delays in developmental milestones (Das et al. 2015). In PPARα-null mice (genetic knockouts), the developmental effects of PFNA are absent, but the effects on maternal liver weight are retained at slightly higher doses (Wolf et al. 2010). As noted by Das et al. (2015), benchmark dose analysis found that increased relative liver weight was more sensitive than many of the developmental outcomes.

The similarity in hepatic effects observed with PFOA and evidence for potential relevance to human health based on the available, but limited, human evidence was the basis for selecting increased relative liver

weight as a precursor for altered liver function. The developmental toxicity in rodents appears to be highly dependent on PPARα, which may translate into limited relevance for human health. If the observed developmental outcomes seen in rodents are relevant to human health, liver toxicity is the more sensitive and therefore protective health endpoint. Given the lack of a robust database on the effects of PFNA, additional studies that quantify the serum half-life in humans and the basis for developmental impacts seen in animals would merit re-evaluation of this critical health effect and its derived RfD.

Animal Serum Dose: **4,900 ng/mL**

Das et al. (2015) reported serum concentrations for PFNA at both the LOAEL and NOAEL. When feasible, it is recommended to utilize benchmark dose (BMD) modeling to address technical uncertainties related to the use of NOAELs for determining a point of departure from animal studies (EPA 2002). Given the time required for *de novo* development and appropriate validation of BMD models, NHDES deferred to the BMD model previously derived by NJ DWQI for the same study by Das et al. (2015) (detailed methodology is summarized in NJ DWQI 2018). Briefly, BMD analysis estimated the serum concentration for a 10% increase in relative liver weight from exposure to PFNA. The serum concentration for the lower 95% confidence limit (the BMDL) from the best fit model was found to be 4,900 ng/mL (NJ DWQI 2018).

Uncertainty Factors (UF): **Total UF of 300**

A full UF of 10 was applied to account for differences in sensitivity and toxicokinetics (e.g., half-lives and elimination rates) across the human population. Given the uncertainty surrounding the exact mechanism(s) of action for PFNA, a partial UF of 3 was applied for rodent-to-human differences in toxicodynamics to account for unknown differences in sensitivity between humans and rodents toward PPARα-independent effects. In practice, an additional UF can be applied to account for suspected differences in toxicokinetics between rodents and humans (i.e, half-life); however, the use of a dosimetric adjustment factor can replace this UF of 3. A UF of 10 was applied due to the limited number of studies on PFNA, specifically the lack of information for a serum half-life in humans, as well as uncertainty for associated effects on other physiological processes including the immune system (summarized by ATSDR 2018).

UF 10 (Human-to-Human) x UF 3 (Animal-to-Human) x MF 10 (Limited Database and Other Toxicities) = Total UF 300

Note that an UF of 3 is a simplification of a half-log unit ($10^{0.5}$ = 3.16), thus $10^{0.5}$ x $10^{0.5}$ = 10. In the case of 300, this is rounded down from 316.

Dividing the Animal Serum Dose by the Total Uncertainty Factor gives the Target Serum Level in humans.

Target Serum Level = Animal Serum Dose ÷ Total Uncertainty Factor

16.3 ng/mL = 4,900 ng/mL ÷ 300

Dosimetric Adjustment: **$1.52E^{-04}$ L/kg/d, assuming 2.5-year half-life**

The dosimetric adjustment factor (DAF) estimates an externally administered (ingested) dose corresponding to the internal serum dose of concern (i.e., the Human Equivalent Dose). This is necessary since the half-lives of PFAS in rodents are profoundly shorter than their half-lives in humans. The NHDES approach is similar to the EPA method used for deriving the reference dose for PFOA and PFOS (EPA 2016ab). This approach requires a volume of distribution (V_d; 0.20 L/kg, ATSDR 2018) and the chemical's half-life ($t_{½}$) in humans.

$$DAF = V_d \times (Ln(2) \div t_{½})$$

$$DAF = 0.20\ L/kg \times (Ln(2) \div (2.5\ y * 365\ d/y)) = 1.5189E^{-04}\ L/kg/d$$

The half-life for PFNA was assumed to be 2.5 years. Unlike PFOA, PFOS and PFHxS, Li et al. (2018) did not quantify serum PFNA or its half-life in the community exposed via drinking water. A single study has estimated half-lives of PFNA in a Chinese population by measuring urinary concentrations of PFNA (Zhang et al. 2013). It should be noted that serum derived half-lives are preferable to those derived from urine concentrations of PFAS. Consistent with ATSDR (2018), we applied an assumed half-life of 2.5 years for women under the age of 50. The uncertainty for a potentially longer half-life is addressed by the previously discussed MF of 3.

Reference Dose (RfD): **2.5 ng/kg/d**

The RfD is calculated as:

$$RfD = (Animal\ Serum\ Dose / Total\ UF) \times DAF$$

$$RfD = (4{,}900\ ng/mL \div 300) \times 1.52E^{-04}\ L/kg/d = 2.5\ ng/kg/d$$

This RfD is slightly lower than the ATSDR's draft MRL for intermediate exposure to PFNA (3.0 ng/kg/d). The US EPA has not developed an RfD for PFNA. The NHDES assessment utilized the same study as the basis for RfD development; however, there was a difference in selection of critical effects and application of uncertainty/modifying factors.

It should be noted that in the RfD calculation there is no term that adjusts for the proportion of PFNA actually absorbed following ingestion. This is because NHDES assumed that 100% of the PFNA ingested from environmental sources is absorbed within the gastrointestinal tract. Although ingestion is the primary route of exposure to PFAS, the mechanisms and efficiency of uptake is poorly understood. This is a health-protective assumption as the actual uptake efficiency is currently unknown in humans (summarized by ATSDR 2018), and may be less than 100% as indicated by animal studies following exposure through food or water.

Exposure Assumptions: **Relative Source Contribution of 50%,**
Water consumption rate of a lactating woman

Similar to PFOA and PFOS, the chemical-specific RSC for PFNA was derived using the subtraction method in conjunction with the EPA decision tree for RSC determination (EPA 2000). The subtraction method derives a RSC from the background level of exposure and the target serum level, where:

RSC = (Target Serum Level – Background exposure level) ÷ Target Serum Level

When population specific data for background exposure is not available, it is recommended to utilize the average from datasets such as NHANES. The 2013-2014 NHANES report shows an average PFNA serum concentration of 0.68 ng/mL for all ages, with a high end estimate (95th percentile) of 2.00 ng/mL for those age 12 years or older (ATSDR 2018). Utilizing either the average or the 95th percentile for exposure from the 2013-2014 NHANES data would result in an RSC >80%. Additionally, more recent and population specific data for serum PFNA concentrations is available for New Hampshire. Across adults and children (n=219) in Southern New Hampshire the average and 95th percentile for PFNA serum concentrations were 0.66 ng/mL and 1.70 ng/mL, respectively (provided by NHDHHS Environmental Public Health Tracking program). Based on the 95th percentile for New Hampshire-specific data, the chemical-specific RSC for PFNA was determined to be 90%.

RSC = (16.3 ng/mL – 1.70 ng/mL) ÷ 16.3 ng/mL = 0.90, or 90%

However, uncertainty about uncharacterized sources of PFNA in the environment resulted in the decision to limit the RSC to 50% (EPA 2000).

NHDES calculated the exposure using the water ingestion rate of a lactating woman (0.055 L/kg d). This was based on the 95th percentile consumers estimate for combined direct and indirect community water ingestion for lactating women (EPA 2011). The water ingestion rate of lactating women is greater than that of non-lactating women or men, and is therefore more protective as it over-estimates an individual's chronic exposure via drinking water.

MCL for PFNA: 23 ppt (ng/L)

The RfD is converted to an equivalent dose in drinking water by selecting a sensitive human receptor and using their drinking water ingestion rate to calculate a drinking water equivalency level (DWEL). The DWEL is 100% of a dose not expected to cause any toxic effects.

DWEL = RfD ÷ Water Ingestion Rate

DWEL = 2.5 ng/kg/d ÷ 0.055 L/kg d = 45.5 ng/L

Taken together with the RSC to account for background sources of exposure, the MCL is derived as follows:

MCL = (DWEL x RSC)

MCL = (45.5 ng/L x 0.50) = 23 ng/L

NHDES is currently reviewing emerging information for the impact the proposed MCL will have on serum concentrations relative to background sources of PFNA.

Appendix 7: PFHxS Derivation

Appendix 7: PFHxS Derivation

Toxicity Endpoint: **Impaired Reproduction (Reduced Litter Size)**

Significantly less peer-reviewed literature is available for PFHxS than PFOA and PFOS. NHDES identified six animal studies on PFHxS (Butenhoff et al. 2008; Bijland et al. 2011; Viberg et al. 2013; Das et al. 2017; Chang et al. 2018; Ramhøj et al. 2018), where only four evaluated more than one dose level of PFHxS. Relative to human epidemiological studies, PFHxS has been evaluated in the context of exposure to multiple PFAS. This makes it challenging to discern PFHxS-specific effects on human health from those of other PFAS typically detected at higher concentrations in human serum. A result of this paucity of data is greater uncertainty regarding specific health effects and relative potency of PFHxS when compared with similar PFAS.

Based on the small number of animal studies, there appears to be limited evidence that PFHxS affects the thyroid gland and liver, with subtle effects on growth and development. Butenhoff et al. (2008) reported thyroid hypertrophy and altered clinical chemistry in male rats following exposure to PFHxS. This same study served as the basis of the 2018 ATSDR draft MRL for PFHxS (20 ng/kg/d), although it was noted that the thyroid effects may be related to enzyme activity that, at present, is not clearly relevant to human health. Ramhøj et al. (2018) reported altered thyroid hormone levels in rats and their pups following gestational exposure to PFHxS, where the effects were potentiated by the presence of other endocrine disrupting compounds. As reviewed and summarized by ATSDR (2018), very few associations have been found between PFHxS and clinical markers of thyroid function in humans, with no associations to clinical thyroid disease. Most of these associations were found in women, not men, which is the opposite of what is seen in rodent models. Similar to other PFAS, PFHxS can elicit hepatic hypertrophy and altered lipid metabolism at higher doses (Butenhoff et al. 2008; Bijland et al. 2011; Das et al. 2017) and are also associated with mixed responses of clinical markers of hepatic function in humans (reviewed by ATSDR 2018).

The most recent study, and basis for the NHDES derivation of a reference dose for PFHxS, was conducted on mice to evaluate reproductive and developmental impacts associated with PFHxS (Chang et al. 2018). In this study, male and female mice were treated with PFHxS by oral gavage and evaluated for a battery of clinical and reproductive outcomes. Male mice were exposed for 42 days, whereas females were exposed for 14-days prior to pregnancy and through gestation and lactation. PFHxS exposure was found to affect liver weight and cholesterol in males, with no alterations in other clinical markers including thyroid function (Chang et al. 2018). Of key interest was a reduction in litter size of female mice starting at the administered dose of 1.0 mg/kg/d, with a NOAEL of 0.3 mg/kg/d. In male mice, there was no relationship between PFHxS exposure and sperm quality, suggesting the reduction in litter size was the result of a female-specific effect. Unlike PFOS in rats (Luebker et al. 2005a), there was no sign of in utero loss of fetal pups, as determined by the pup-born-to-implant ratio, suggesting an effect prior to implantation.

It is acknowledged that the authors of Chang et al. (2018) regard the observed reduction in litter size as toxicologically insignificant. This is based on the contention that this effect is inconsistent with two other studies showing no reduction in the litter size of rats that were exposed to PFHxS (Butenhoff et al. 2008; Ramhøj et al. 2018). However, these comparisons are complicated by the issues of exposure dose and timing. It is true that Butenhoff et al. (2008) did not see reduced litter size from female rats that were administered higher doses of PFHxS than those used in Chang et al. (2018). However, the highest internal dose observed in female rats prior to breeding (42,000 ng/mL; Butenhoff et al. 2008) was approximately half

of the lowest internal dose observed in female mice with reduced litters (89,000 ng/mL; Chang et al. 2018). Thus, the dose that elicited reduced litter size in mice was not achieved in rats. This difference is likely due to the shorter half-life of PFHxS in rats compared to mice. Ramhøj et al. (2018) also reported that higher administered doses than those used by Chang et al. (2018) did not reduce litter size at birth. This does not address the issue of exposure timing as Ramhøj et al. (2018) initiated PFHxS treatment *after* female rats were confirmed to be pregnant, unlike Chang et al. (2018) that had initiated treatment prior to pregnancy. Taken together, the evidence from Butenhoff et al. (2008) and Ramhøj et al. (2018) does not support the contention that the reduction in litter size observed by Chang et al. (2018) is an inconsistent effect.

To date, there are two studies that have evaluated associations between PFHxS and reproductive outcomes in women. Vélez et al. (2015) evaluated a cohort of 1,743 women from the Maternal-Infant Research on Environmental Chemicals (MIREC) Study, all of which were recruited before 14 weeks of gestation from ten Canadian cities between 2008 and 2011. They found significant associations for PFHxS with reduced fecundability and increased infertility (Vélez et al. 2015). This observation is contrasted with the lack of association with fertility reported in a comparably sized population with lower median PFHxS levels (Bach et al. 2015). It should be noted that these studies do not prove or disprove a relationship between PFHxS and human fertility due to several factors addressed by the authors, including limitations of experimental design, statistical analyses and evaluation of male reproductive effects. However, the limited number of human epidemiology studies, and limitations of data therein, preclude them as the basis of RfD determination. Thus, the Chang et al. (2018) was deemed sufficient for identifying the RfD required for MCL/AGQS derivation. Additional epidemiological studies are needed to determine if there is a causal relationship between PFHxS and human reproduction.

Given the lack of a robust database on the effects of PFHxS, additional studies that further assess reproductive impacts, changes in thyroid function and other health outcomes would merit re-evaluation of this critical health effect and its derived RfD.

Animal Serum Dose: **27,200 ng/mL**

The animal study selected for PFHxS was a mouse study conducted by Chang et al. (2018). In the study, male and female mice were administered PFHxS by oral gavage at doses of 0, 0.3, 1.0 and 3.0 mg/kg/d. Female mice showed a statistically significant reduction in litter size with a LOAEL of 1.0 mg/kg/d, and a NOAEL of 0.3 mg/kg/d. Additionally, the study reported an increase in the anogenital distance in male pups born to females across all doses. As noted by the authors of the study, the biological implications of an increased anogenital distance are unclear as this would suggest masculinization by androgens, and this effect was not observed in female pups. Given some evidence for associated impacts on fertility and limited database on the effects of PFHxS in animals, reduced litter size was selected as the critical health effect. Instead of benchmark dose modeling to determine a dose from a specified threshold, the serum concentration at the NOAEL before pregnancy was selected as the animal serum dose (0.3mg/kg/d, 14-d exposure, 27.2µg/mL). Due to current feasibility, and as recommended by the EPA guidance (2002; 2012), the NOAEL was used in place of BMD modeling.

Uncertainty Factors (UF): **Total UF of 300**

A full UF of 10 was applied to account for differences in sensitivity and kinetics across the human population. Given the uncertainty surrounding the exact mechanism(s) of action for PFHxS, a partial UF of 3 was applied for rodent-to-human differences in toxicodynamics to account for unknown differences in sensitivity between humans and rodents toward PPARα-independent effects. In practice, an additional UF can be applied to account for suspected differences in toxicokinetics between rodents and humans (i.e, half-life); however, the use of a dosimetric adjustment factor can replace this UF of 3. An UF of 10 was applied due to the limited number of studies on PFHxS, both animal and epidemiological, as well as uncertainty for associated effects on other physiological processes including the thyroid system (ATSDR 2018).

UF 10 (Human-to-Human) x UF 3 (Animal-to-Human) x MF 10 (Limited Database and Other Toxicities) = Total UF 300

Note that an UF of 3 is a simplification of a half-log unit ($10^{0.5}$ = 3.16), thus $10^{0.5}$ x $10^{0.5}$ = 10. In the case of 300, this is rounded down from 316.

Dividing the Animal Serum Dose by the Total Uncertain gives the Target Serum Level in humans.

Target Serum Level = Animal Serum Dose ÷ Total Uncertainty Factor

90.7 ng/mL = 27,200 ng/mL ÷ 300

Dosimetric Adjustment: $1.03E^{-04}$ L/kg/d, assuming 5.3-year half-life

The dosimetric adjustment factor (DAF) estimates an externally administered (ingested) dose that corresponds to the internal serum dose of concern (i.e., the Human Equivalent Dose). This is necessary since the half-lives of PFAS in rodents are profoundly shorter than their half-lives in humans. The NHDES approach is similar to the EPA method used for deriving the reference dose for PFOA and PFOS (EPA 2016ab). This approach utilizes a volume of distribution (V_d, 0.287 L/kg; ATSDR 2018; Sundström et al. 2012) and the chemical's half-life ($t_{½}$) in humans.

$DAF = V_d \times (Ln(2) \div t_{½})$

$DAF = 0.287\ L/kg \times (Ln(2) \div (5.3\ y * 365\ d/y)) = 1.03^{-04}\ L/kg/d$

The half-life for PFHxS was assumed to be 5.3 years based on the same study selected for the half-lives of PFOA and PFOS (Li et al. 2018). The strengths of this study included its sample size, relevance to drinking water exposure, inclusion of a broad age range (15-50) and balanced representation of both sexes. The average (±SD) serum concentration of PFHxS was 353 ± 260 ng/mL amongst 106 participants. Unlike PFOA, there were sex-specific differences in the half-life of PFHxS where the half-life in men was 7.4 years (95% CI 6.0-9.7 years) and 4.7 years for women (95% CI 3.9-5.9 years). The average across both sexes was 5.3 years.

Reference Dose (RfD): **9.3 ng/kg/d**

The RfD is calculated as:

RfD = (Animal Serum Dose / Total UF) x DAF

RfD = (27,200 ng/mL ÷ 300) x 1.03E^{-04} L/kg/d = 9.3 ng/kg/d

This RfD is lower than the ATSDR's draft MRL for intermediate exposure to PFHxS (20 ng/kg/d). EPA has not developed an RfD for PFHxS. The NHDES assessment utilized an entirely different study and critical health effects than those selected by ATSDR.

It should be noted that in the RfD calculation there is no term that adjusts for the proportion of PFHxS actually absorbed following ingestion. This is because NHDES assumed that 100% of the PFHxS ingested from environmental sources is absorbed within the gastrointestinal tract. Although ingestion is the primary route of exposure to PFAS, the mechanisms and efficiency of uptake are poorly understood. This is a health-protective assumption as the actual uptake efficiency is currently unknown in humans (summarized by ATSDR 2018), and may be less than 100% as indicated by animal studies following exposure through food or water.

Exposure Assumptions: **Relative Source Contribution of 50%,**
Water consumption rate of a lactating woman

Similar to PFOA, PFOS and PFNA, the chemical-specific RSC for PFHxS was derived using the subtraction method in conjunction with the EPA decision tree for RSC determination (EPA 2000). The subtraction method derives a RSC from the background level of exposure and the target serum level, where:

RSC = (Target Serum Level – Background exposure level) ÷ Target Serum Level

When population specific data for background exposure is not available, it is recommended to utilize the average from datasets such as NHANES. The 2013-2014 NHANES report shows an average PFHxS serum concentration of 1.4 ng/mL for ages 12 and older, with a high end estimate (95th percentile) of 5.6 ng/mL for those age 12 years or older (NH HEALTH WISDOM, accessed December 2018; ATSDR 2018). Utilizing either the average or the 95th percentile for exposure from the 2013-2014 NHANES data would result in an RSC >80%. However, more recent and population specific data for serum PFHxS concentrations is available for New Hampshire. Across those 12 and older in the 2016 Pease group (n=242), the average and 95th percentile for PFHxS serum concentrations were 4.5 ng/mL and 26.0 ng/mL, respectively (NH HEALTH WISDOM accessed December 2018). Based on the 95th percentile for New Hampshire-specific data, the chemical-specific RSC for PFHxS was determined to be 70%.

RSC = (90.7 ng/mL – 26.0 ng/mL) ÷ 90.7 ng/mL = 0.71, rounded to 0.70 or 70%

However, uncertainty about uncharacterized sources of PFHxS in the environment resulted in the decision to limit the RSC to 50% (EPA 2000).

NHDES calculated the exposure using the water ingestion rate of a lactating woman (0.055 L/kg d). This was based on the 95th percentile consumers estimate for combined direct and indirect community water ingestion for lactating women (EPA 2011). The water ingestion rate of lactating women is greater than that

of non-lactating women or men, and is therefore more protective as it over-estimates an individual's chronic exposure via drinking water. Additionally, the critical health effect of impaired reproduction was specific to females as no effects were observed in male sperm (Chang et al. 2018).

MCL for PFHxS: 85 ppt (ng/L)

The RfD is converted to an equivalent dose in drinking water by selecting a sensitive human receptor and using their drinking water ingestion rate to calculate a drinking water equivalency level (DWEL). The DWEL is 100% of a dose not expected to cause any toxic effects.

DWEL = RfD ÷ Water Ingestion Rate

DWEL = 9.3 ng/kg/d ÷ 0.055 L/kg d = 169.1 ng/L

Taken together with the RSC to account for background sources of exposure, the MCL is derived as follows:

MCL = (DWEL x RSC)

MCL = (169.1 ng/L x 0.50) = 85 ng/L

NHDES is currently reviewing emerging information for the impact the proposed MCL will have on serum concentrations relative to background sources of PFHxS.

Appendix 8: References

Appendix 8: References

This list includes references for the main summary report and Appendices 3-7. This list is for documents specifically cited within these appendices and does not contain all of the research articles, reviews, technical document and various reports reviewed by NHDES.

Abbott BD, Wolf CJ, Das KP, et al. 2009. Developmental toxicity of perfluorooctane sulfonate (PFOS) is not dependent on expression of peroxisome proliferator activated receptor-alpha (PPARα) in the mouse. Reprod Toxicol 27(3-4):258-265.

Agency for Toxic Substances and Disease Registry (ATSDR). 2018. Toxicological Profile for Perfluoroalkyls – Draft for Public Comment, June 2018. Accessed online at: https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf.

Albrecht PP, Torsell NE, Krishnan P, et al. 2013. A species difference in the peroxisome proliferator-activated receptor α-dependent response to the developmental effects of perfluorooctanoic acid. Toxicol Sci 131(2):568-582.

Bach CC, Bech BH, Nohr EA, et al. 2015. Serum perfluoroalkyl acids and time to pregnancy in nulliparous women. Environ Res 142:535-541. 10.1016/j.envres.2015.08.007.

Barry V, Winquist A, Steenland K. 2013. Perfluorooctanoic acid (PFOA) exposures and incident cancers among adults living near a chemical plant. Environ Health Perspect 121(11-12):1313-1318.

Behr AC, et al. 2018. Perfluoroalkylated substances (PFAS) affect neither estrogen and androgen receptor activity nor steroidogenesis in human cells in vitro. Toxicology Letters, 291: 51-60.

Biegel LB, Hurtt ME, Frame SR, et al. 2001. Mechanisms of extrahepatic tumor induction by peroxisome proliferators in male CD rats. Toxicol Sci 60(1):44-55.

Bijland S, Rensen PC, Pieterman EJ, et al. 2011. Perfluoroalkyl sulfonates cause alkyl chain length-dependent hepatic steatosis and hypolipidemia mainly by impairing lipoprotein production in APOE*3-Leiden CETP mice. Toxicol Sci 123(1):290-303. 10.1093/toxsci/kfr142.

Bjork JA, Butenhoff JL, Wallace KB. 2011. Multiplicity of nuclear receptor activation by PFOA and PFOS in primary human and rodent hepatocytes. Toxicology 288(1-3):8-17. 10.1016/j.tox.2011.06.012.

Bonefeld-Jorgensen EC, et al. 2011. Perfluorinated compounds are related to breast cancer risk in Greenlandic Inuit: A case control study. *Environmental Health*, **10**:88.

Bonefeld-Jorgensen EC, et al. 2014. Breast cancer risk after exposure to perfluorinated compounds in Danish women: a case-control study nested in the Danish National Birth Cohort. *Cancer Causes Control*, **25**:1439-1448.

Brown NM, Manzolillo PA, Zhang JX, Wang J, Lamartiniere CA. Prenatal TCDD and predisposition to mammary cancer in the rat. Carcinogenesis. 1998;19(9):1623–1629.

Brunton, L. L.; Chabner, Bruce; Knollmann, Björn C., eds. (2011). Goodman & Gilman's The Pharmacological Basis of Therapeutics (12th ed.). New York: McGraw-Hill. ISBN 978-0-07-162442-8. 2084

Butenhoff, J.L., G.L. Kennedy, S.R. Frame, J.C. O'Conner, and R.G. York. 2004a. The reproductive toxicology of ammonium perfluorooctanoate (APFO) in the rat. Toxicology 196:95–116.

Butenhoff, J.L., G.L. Kennedy Jr, P.M. Hinderliter, P.H. Lieder, R. Jung, J.K. Hansen, G.S. Gorman, P.E. Noker, and P.J. Thomford. 2004b. Pharmacokinetics of perfluorooctanoate in cynomolgus monkeys. Toxicological Sciences 82(2):394–406.

Butenhoff JL, et al. 2008. Evaluation of potential reproductive and developmental toxicity of potassium perfluorohexanesulfonate in Sprague Dawley rats. Reproductive Toxicology, 27, 331-341.

Butenhoff JL, Chang S, Ehresman DJ, et al. 2009a. Evaluation of potential reproductive and developmental toxicity of potassium perfluorohexanesulfonate in Sprague Dawley rats. Reprod Toxicol 27:331-341.

Butenhoff JL, Ehresman DJ, Chang SC, et al. 2009b. Gestational and lactational exposure to potassium perfluorooctanesulfonate (K+PFOS) in rats: Developmental neurotoxicity. Reprod Toxicol 27(3-4):319-330.

Butenhoff, J.L., G.L. Kennedy, Jr., S.-C. Chang, and G.W. Olsen. 2012a. Chronic dietary toxicity and carcinogenicity study with ammonium perfluorooctanoate in Sprague-Dawley rats. Toxicology 298:1–13.

Butenhoff JL, Chang SC, Olsen GW, et al. 2012b. Chronic dietary toxicity and carcinogenicity study with potassium perfluorooctanesulfonate in Sprague Dawley rats. Toxicology 293(1-3):1-15.

Cave et a. 2016. Nuclear receptors and nonalcoholic fatty liver disease. Biochimica et Biophysica Acta (BBA) - Gene Regulatory Mechanisms, 1859:9, 1083-1099. https://doi.org/10.1016/j.bbagrm.2016.03.002

Chang ET, et al. 2016. A critical review of perfluorooctanoate and perfluorooctanesulfonate exposure and immunological health conditions in humans. Crit Rev Toxicol., 46(4): 279-331.

Chang S, et al. 2018. Reproductive and developmental toxicity of potassium perfluorohexanesulfonate in CD-1 mice. Reproductive Toxicology 78: 150-168.

Chen MH, Ha EH, Wen TW, et al. 2012. Perfluorinated compounds in umbilical cord blood and adverse birth outcomes. PLoS ONE 7(8):e42474.

Cheng J, Fujimura M, Zhao W, et al. 2013. Neurobehavioral effects, c-Fos/Jun expression and tissue distribution in rat offspring prenatally co-exposed to MeHg and PFOA: PFOA impairs Hg retention. Chemosphere 91(6):758-764.

Corton JC, Cunningham ML, Hummer BT, et al. 2014. Mode of action framework analysis for receptor-mediated toxicity: The peroxisome proliferator-activated receptor alpha (PPARα) as a case study. Crit Rev Toxicol 4444(1):1-49. 10.3109/10408444.2013.835784.

Cui L, Zhou QF, Liao CY, et al. 2009. Studies on the toxicological effects of PFOA and PFOS on rats using histological observation and chemical analysis. Arch Environ Contam Toxicol 56(2):338-349.

Cui Y, et al. Investigation of the Effects of Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS) on Apoptosis and Cell Cycle in a Zebrafish (Danio rerio) Liver Cell Line. Int J Environ Res Public Health. 2015 Dec 9;12(12):15673-82.

Das KP, Grey BE, Rosen MB, et al. 2015. Developmental toxicity of perfluorononanoic acid in mice. Reprod Toxicol 51:133-144. 10.1016/j.reprotox.2014.12.012.

Das KP, Wood CR, Lin MT, et al. 2017. Perfluoroalkyl acids-induced liver steatosis: Effects on genes controlling lipid homeostasis. Toxicology 378:37-52. 10.1016/j.tox.2016.12.007.

DeWitt JC, et al. 2012. Immunotoxicity of Perfluorinated Compounds: Recent Developments. Toxicologic Pathology, 40: 300-311.

Elcombe CR, Elcombe BM, Foster JR, et al. 2010. Hepatocellular hypertrophy and cell proliferation in Sprague-Dawley rats following dietary exposure to ammonium perfluorooctanoate occurs through increased activation of the xenosensor nuclear receptors PPARα and CAR/PXR. Arch Toxicol 84(10):787-798.

Eriksen KT, Sorensen M, McLaughlin JK, et al. 2009. Perfluorooctanoate and perfluorooctanesulfonate plasma levels and risk of cancer in the general Danish population. J Natl Cancer Inst 101(8):605-609.

Eriksen K.T., Raaschou-Nielsen O., Sørensen M., Roursgaard M., Loft S., Møller P. Genotoxic potential of the perfluorinated chemicals PFOA, PFOS, PFBS, PFNA and PFHxA in human HepG2 cells. Mutat. Res. 2010;700:39–43.

Evans RM, Mangelsdorf DJ. Nuclear receptors, RXR, and the big bang. Cell. 2014;157:255–266. doi: 10.1016/j.cell.2014.03.012.

Fang X, Feng Y, Shi Z, et al. 2009. Alterations of cytokines and MAPK signaling pathways are related to the immunotoxic effect of perfluorononanoic acid. Toxicol Sci 108(2):367-376. 10.1093/toxsci/kfp019.

Felter SP, Foreman JE, Boobis A, Corton JC, Doi AM, Flowers L, Goodman J, Haber LT, Jacobs A, Klaunig JE, Lynch AM, Moggs J, Pandiri A. Human relevance of rodent liver tumors: Key insights from a Toxicology Forum workshop on nongenotoxic modes of action. Regul Toxicol Pharmacol. 2018 Feb;92:1-7. doi: 10.1016/j.yrtph.2017.11.003.

Fenton SE, et al. 2002. Persistent abnormalities in the rat mammary gland following gestational and lactational exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Toxicological Sciences, 67(1): 63-74.

Ferri N, Corsini A, Sirtori C, Ruscica M. PPAR-α agonists are still on the rise: an update on clinical and experimental findings. Expert Opin Investig Drugs. 2017 May;26(5):593-602. doi: 10.1080/13543784.2017.1312339

Frank J. Gonzalez, Yatrik M. Shah. 2008. PPARα: Mechanism of species differences and hepatocarcinogenesis of peroxisome proliferators. Toxicology, Volume 246, Issue 1: 2-8, https://doi.org/10.1016/j.tox.2007.09.030.

Gleason JA, Post GB, Fagliano JA. 2015. Associations of perfluorinated chemical serum concentrations and biomarkers of liver function and uric acid in the US population (NHANES), 2007-2010. Environ Res 136:8-14. 10.1016/j.envres.2014.10.004.

Grandjean P, et al. 2012. Serum Vaccine Antibody Concentrations in Children Exposed to Perfluorinated Compounds. *JAMA*, **307(4)**: 391-397.

Granum B, et al. 2013. Pre-natal exposure to perfluoroalkyl substances may be associated with altered vaccine antibody levels and immune-related health outcomes in early childhood, *Journal of Immunotoxicology*, **10:4**: 373-379.

Griffith FD, Long JE. 1980. Animal toxicity studies with ammonium perfluorooctanoate. Am Ind Hyg Assoc J 41(8):576-583.

Hagenaars A., Vergauwen L., De Coen W., Knapen D. Structure-activity relationship assessment of four perfluorinated chemicals using a prolonged zebrafish early life stage test. Chemosphere. 2011;82:764–772. doi: 10.1016/j.chemosphere.2010.10.076

Hagenaars A, et al. 2013. Mechanistic toxicity study of perfluorooctanoic acid in zebrafish suggests mitochondrial dysfunction to play a key role in PFOA toxicity. Chemosphere, 91(6): 844-56.

Hall AP, Elcombe CR, Foster JR, et al. 2012. Liver hypertrophy: A review of adaptive (adverse and non-adverse) changes- conclusions from the 3rd International ESTP Expert Workshop. Toxicol Pathol 40:971-994.

Haughom B, Spydevold O. 1992. The mechanism underlying the hypolipemic effect of perfluorooctanoic acid (PFOA), perfluorooctane sulphonic acid (PFOSA) and clofibric acid. Biochim Biophys Acta 1128(1):65-72.

Hu Q, Strynar MJ, DeWitt JC. 2010. Are developmentally exposed C57BL/6 mice insensitive to suppression of TDAR by PFOA? J Immunotoxicol 7(4):344-349.

IARC 2016: CAS No. 335-67-1, Agent = Perfluorooctanoic acid (PFOA) Group 2B, Volume 110, 2016 online, Available at: http://monographs.iarc.fr/ENG/Classification/latest_classif.php

Issemann I, Green S. 1990. Activation of a member of a steroid hormone receptor superfamily by peroxisome proliferators. Nature 347:645-650.

Jia, Y., Qi, C., Zhang, Z., Zhu, Y. T., Rao, S. M., and Zhu, Y. J. (2005). Peroxisome proliferators-activated receptor-binding protein null mutation results in defective mammary gland development. J. Biol. Chem. 280, 10766–10773.

Kennedy GL. 1985. Dermal toxicity of ammonium perfluorooctanoate. Toxicol Appl Pharmacol 81(2):348-355.

Kielsen K, Shamin Z, Ryder LP, et al. 2015. Antibody response to booster vaccination with tetanus and diphtheria in adults exposed to perfluorinated alkylates. *J Immunotoxicol* **13(2):**270-273.

Kirk M, Smurthwaite K, Bräunig J et al. (2018). The PFAS Health Study: Systematic Literature Review. Canberra: The Australian National University.

Klaunig JE, Babich MA, Baetcke KP, et al. 2003. PPARα agonist-induced rodent tumors: Modes of action and human relevance. Crit Rev Toxicol 33(6):655-780.

Klaunig JE, Hocevar BA, Kamendulis LM. 2012. Mode of action analysis of perfluorooctanoic acid (PFOA) tumorigenicity and human relevance. Reprod Toxicol 33(4):410-418.

Koskela A, Finnila MA, Korkalainen M, et al. 2016. Effects of developmental exposure to perfluorooctanoic acid (PFOA) on long bone morphology and bone cell differentiation. Toxicol Appl Pharmacol 301:14-21. 10.1016/j.taap.2016.04.002.

Koustas E, Lam J, Sutton P, et al. 2014. The Navigation Guide - evidence-based medicine meets environmental health: Systematic review of nonhuman evidence for PFOA effects on fetal growth. Environ Health Perspect 122(10):1015-1027.

Lau C, Thibodeaux JR, Hanson RG, et al. 2003. Exposure to perfluorooctane sulfonate during pregnancy in rat and mouse. II: Postnatal evaluation. Toxicol Sci 74(2):382-392.

Lau C, Thibodeaux JR, Hanson RG, et al. 2006. Effects of perfluorooctanoic acid exposure during pregnancy in the mouse. Toxicol Sci 90(2):510-518.

Lee SS-T, Pineau T, Drago J, Lee EJ, Owens JW, Kroetz DL, Fernandez-Salguero PM, Westphal H, and Gonzalez FJ (1995) Targeted disruption of the a isoform of the peroxisome proliferator-activated receptor gene in mice results in abolishment of the pleiotropic effects of peroxisome proliferators. Mol Cell Biol 15:3012–3022

Leonard RC. 2006. Ammonium perfluorooctanoate: Phase II. Retrospective cohort mortality analyses related to a serum biomarker of exposure in a polymer production plant. Wilmington, DE: E.I. du pont de Nemours and Company.

Leonard RC, Kreckmann KH, Sakr CJ, et al. 2008. Retrospective cohort mortality study of workers in a polymer production plant including a reference population of regional workers. Ann Epidemiol 18:15-22.

Li K, Gao P, Xiang P, Zhang X, Cui X, Ma LQ. 2017a. Molecular mechanisms of PFOA-induced toxicity in animals and humans: Implications for health risks. 99:43-54.

Li K, Sun J., Yang J, Roberts SM, Zhang X, Cui X, Wei S, Ma LQ. 2017b. Molecular Mechanisms of Perfluorooctanoate-Induced Hepatocyte Apoptosis in Mice Using Proteomic Techniques. Environmental Science & Technology, 51, 11380-11389.

Li Y, Fletcher T, Mucs D, et al. 2018. Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water. Occup Environ Med 75(1):46-51. 10.1136/oemed-2017-104651.

Liew Z, et al. 2018. Developmental Exposures to Perfluoroalkyl Substances (PFASs): An Update of Associated Health Outcomes. Current Environmental Health Reports 5:1-19.

Lin CY, Lin LY, Chiang CK, et al. 2010. Investigation of the associations between low-dose serum perfluorinated chemicals and liver enzymes in US adults. Am J Gastroenterol 105(6):1354-1363.

Looker C, et al. 2014. Influenza Vaccine Response in Adults Exposed to Perfluorooctanoate and Perfluorooctanesulfonate. *Toxicological Sciences*, **138(1):**76-88.

Loveless SE, Finlay C, Everds NE, et al. 2006. Comparative responses of rats and mice exposed to linear/branched, linear, or branched ammonium perfluorooctanoate (APFO). Toxicology 220:203-217.

Loveless SE, Hoban D, Sykes G, et al. 2008. Evaluation of the immune system in rats and mice administered linear ammonium perfluorooctanoate. Toxicol Sci 105(1):86-96.

Luebker DJ, Case MT, York RG, et al. 2005a. Two-generation reproduction and cross-foster studies of perfluorooctanesulfonate (PFOS) in rats. Toxicology 215(1-2):126-148.

Luebker DJ, York RG, Hansen KJ, et al. 2005b. Neonatal mortality from in utero exposure to perfluorooctanesulfonate (PFOS) in Sprague-Dawley rats: Dose-response, and biochemical and pharmacokinetic parameters. Toxicology 215(1-2):149-169.

Macon MB, Villanueva LR, Tatum-Gibbs K, et al. 2011. Prenatal perfluorooctanoic acid exposure in CD-1 mice: Low-dose developmental effects and internal dosimetry. Toxicol Sci 122(1):134-145.

Macon MB and Fenton SE. 2013. Endocrine Disruptors and the Breast: Early Life Effects and Later Life Disease. J Mammary Gland Biol Neoplasia. 18(1): 43-61.

Maisonet M, Terrell ML, McGeehin MA, et al. 2012. Maternal concentrations of polyfluoroalkyl compounds during pregnancy and fetal and postnatal growth in British girls. Environ Health Perspect 120(10):1432-1437.

Mashayekhi V., Tehrani K.H.M.E., Hashemzaei M., Tabrizian K., Shahraki J., Hosseini M. Mechanistic approach for the toxic effects of perfluorooctanoic acid on isolated rat liver and brain mitochondria. Hum. Exp. Toxicol. 2015;34:985–996. doi: 10.1177/0960327114565492.

Michigan PFAS Science Advisory Panel Report. 2018. Scientific Evidence and Recommendations for Managing PFAS Contamination in Michigan. December 7, 2018. Available online at: https://www.michigan.gov/documents/pfasresponse/Science_Advisory_Board_Report_641294_7.pdf.

Minnesota Department of Health. 2017 - Toxicological Summary for: Perfluorooctanoate: http://www.health.state.mn.us/divs/eh/risk/guidance/gw/pfoa.pdf

Mogensen UB, Grandjean P, Heilmann C, et al. 2015. Structural equation modeling of immunotoxicity associated with exposure to perfluorinated alkylates. *Environ Health* **14**:47.

Negri E, et al. 2017. Exposure to PFOA and PFOS and fetal growth: a critical merging of toxicological and epidemiological data. Critical Reviews in Toxicology 47: 482-508.

NH HEALTH WISDOM: Perfluorochemical (PFC) Blood Testing and Community Exposure. https://wisdom.dhhs.nh.gov/wisdom/#main

NJ DWQI 2017: NJ Drinking Water Quality Institute (DWQI). 2016. Health-Based Maximum Contaminant Level Support Document: Perfluorooctanoic Acid (PFOA). February 15, 2017. Available online at: https://www.state.nj.us/dep/watersupply/pdf/pfoa-appendixa.pdf.

NJ DWQI 2018: NJ Drinking Water Quality Institute (DWQI). 2018. Health-Based Maximum Contaminant Level Support Document: Perfluorooctane Sulfonate (PFOS). June 5, 2018. Available online at: https://www.state.nj.us/dep/watersupply/pdf/pfos-recommendation-appendix-a.pdf.

NJ DWQI 2018: NJ Drinking Water Quality Institute (DWQI). 2018. Health-Based Maximum Contaminant Level Support . Document: Perfluorononanoic Acid (PFNA)

NTP 2016: National Toxicology Program. NTP Monograph: Immunotoxicity Associated with Exposure to Perfluorooctanoic Acid or Perfluorooctane Sulfonate. September 2016.

Onishchenko N, Fischer C, Wan Ibrahim WN, et al. 2011. Prenatal exposure to PFOS or PFOA altersmotor function in mice in a sex-related manner. Neurotox Res 19(3):452-461.

Osborne G, et al. 2015. Evaluating chemical effects on mammary gland development: A critical need in disease prevention. Reproductive Toxicology, 54, 148-155.

Palmer CN, Hsu MH, Griffin KJ, et al. (1998). Peroxisome proliferator activated receptor-alpha expression in human liver. Mol Pharmacol, 53, 14–22

Panaretakis, T., Shabalina, I.G., Grandér, D., Shoshan, M.C., DePierre, J.W., 2001. Reactive oxygen species and mitochondria mediate the induction of apoptosis in human hepatoma hepg2 cells by the rodent peroxisome proliferator and hepatocarcinogen, perfluorooctanoic acid. Toxicol. Appl. Pharmacol. 173, 56–64.

Perkins RG, Butenhoff JL, Kennedy GL, et al. 2004. 13-Week dietary toxicity study of ammonium perfluorooctanoate (APFO) in male rats. Drug Chem Toxicol 27(4):361-378.

Pilkerton CS, et al. 2018. Rubella immunity and serum perfluoroalkyl substances: Sex and analytic strategy. PLOS One, 13(9):e0203330.

Qi, C., Kashireddy, P., Zhu, Y. T., Rao, S. M., and Zhu, Y. J. (2004). Null mutation of peroxisome proliferators-activated receptor-interacting protein in mammary glands causes defective mammopoiesis. J. Biol. Chem. 279, 33696–33701.

Quist EM, Filgo AJ, Cummings CA, et al. 2015a. Hepatic mitochondrial alteration in CD-1 mice associated with prenatal exposures to low doses of perfluorooctanoic acid (PFOA). Toxicol Pathol 43(4):546-557. 10.1177/0192623314551841.

Quist EM, Filgo AJ, Cummings CA, et al. 2015b. Supplemental data: Hepatic mitochondrial alteration in CD-1 mice associated with prenatal exposures to low doses of perfluorooctanoic acid (PFOA). (Toxicol Pathol 43(4):546-557). Toxicol Pathol 43:546-557.

Raleigh KK, Alexander BH, Olsen GW, et al. 2014. Mortality and cancer incidence in ammonium perfluorooctanoate production workers. Occup Environ Med 71(7):500-506. 10.1136/oemed-2014-102109.

Ramhoj L, et al. 2018. Perfluorohexane Sulfonate (PFHxS) and a Mixture of Endocrine Disrupters Reduce Thyroxine Levels and Cause Antiandrogenic Effects in Rats. Toxicological Sciences, 163(2), 579-591.

Rappazzo KM, et al. 2017. Exposure to Perfluorinated Alkyl Substances and Health Outcomes in Children: A Systematic Review of the Epidemiologic Literature. International Journal of Environmental Research and Public Health, 14, 691.

Rebholz SL, Jones T, Herrick RL, et al. 2016. Hypercholesterolemia with consumption of PFOA-laced Western diets is dependent on strain and sex of mice. Toxicology reports 3:46-54. 10.1016/j.toxrep.2015.11.004.

Rogers JM, Ellis-Hutchings RG, Grey BE, et al. 2014. Elevated blood pressure in offspring of rats exposed to diverse chemicals during pregnancy. Toxicol Sci 137(2):436-446. 10.1093/toxsci/kft248.

Rosen MB, Thibodeaux JR, Wood CR, et al. 2007. Gene expression profiling in the lung and liver of PFOA-exposed mouse fetuses. Toxicology 239:15-33.

Rosen MB, Abbott BD, Wolf DC, et al. 2008a. Gene profiling in the livers of wild-type and PPARα-null mice exposed to perfluorooctanoic acid. Toxicol Pathol 36(4):592-607.

Rosen MB, Lee JS, Ren H, et al. 2008b. Toxicogenomic dissection of the perfluorooctanoic acid transcript profile in mouse liver: Evidence for the involvement of nuclear receptors PPARα and CAR. Toxicol Sci 103(1):46-56.

Rosen MB, Das KP, Rooney J, et al. 2017. PPARα-independent transcriptional targets of perfluoroalkyl acids revealed by transcript profiling. Toxicology [In press].

Rudel RA, et al. 2011. Environmental exposures and mammary gland development: state of the science, public health implications, and research recommendations. Environmental Health Perspectives, 119(8):1053-61.

Sertznig P., Seifert M., Tilgen W., Reichrath J. Present concepts and future outlook: Function of peroxisome proliferator-activated receptors (PPARs) for pathogenesis, progression, and therapy of cancer. J. Cell. Physiol. 2007;212:1–12.

Shabalina, I.G., Panaretakis, T., Bergstrand, A., DePierre, J.W., 1999. Effects of the rodent peroxisome proliferator and hepatocarcinogen, perfluorooctanoic acid, on apoptosis in human hepatoma hepg2 cells. Carcinogenesis 20, 2237–2246.

Son H, Kim S, Shin HI, et al. 2008. Perfluorooctanoic acid-induced hepatic toxicity following 21-day oral exposure in mice. Arch Toxicol 82:239-246.

Starkov AA, Wallace KB. 2002. Structural determinants of fluorochemical-induced mitochondrial dysfunction. Toxicol Sci 66(2):244-252.

Stein CR, McGovern KJ, Pajak AM, et al. 2016. Perfluoroalkyl and polyfluoroalkyl substances and indicators of immune function in children aged 12-19 y: National Health and Nutrition Examination Survey. *Pediatr Res* **79(2):**348-357.

Suh KS, et al. 2017. Perfluorooctanoic acid induces oxidative damage and mitochondrial dysfunction in pancreatic β-cells. Mol Med Rep. 15(6): 3871-3878.

Sundström M, Chang SC, Noker PE, et al. 2012. Comparative pharmacokinetics of perfluorohexanesulfonate (PFHxS) in rats, mice, and monkeys. Reprod Toxicol 33(4):441-451.

Tan X, Xie G, Sun X, et al. 2013. High fat diet feeding exaggerates perfluorooctanoic acid-induced liver injury in mice via modulating multiple metabolic pathways. PLoS ONE 8(4):e61409.

Thibodeaux JR, Hanson RG, Rogers JM, et al. 2003. Exposure to perfluorooctane sulfonate during pregnancy in rat and mouse. I: Maternal and prenatal evaluations. Toxicol Sci 74(2):369-381.

Thomford PJ. 2001. 4-Week capsule toxicity study with ammonium perfluorooctanoate (APFO) in Cynomolgus monkeys. APME Ad-Hoc APFO toxicology working group.

Thompson J, Lorber M, Toms LM, et al. 2010. Use of simple pharmacokinetic modeling to characterize exposure of Australians to perfluorooctanoic acid and perfluorooctane sulfonic acid. Environ Int 36(4):390-397. 10.1016/j.envint.2010.02.008.

Tiede B and Kang Y. 2011. From milk to malignancy the role of mammary stem cells in development, pregnancy and breast cancer. Cell Research, 21:245-257.

Tucker DK, Macon MB, Strynar MJ, et al. 2015. The mammary gland is a sensitive pubertal target in CD-1 and C57Bl/6 mice following perinatal perfluorooctanoic acid (PFOA) exposure. Reprod Toxicol 54:26-36. 10.1016/j.reprotox.2014.12.002.

Tyagi S, Gupta P, Saini AS, Kaushal C, Sharma S (October 2011). "The peroxisome proliferator-activated receptor: A family of nuclear receptors role in various diseases". J Adv Pharm Technol Res. 2(4): 236–40.

USEPA (U.S. Environmental Protection Agency). 2000. Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000) Documents. Accessed October 2018. Accessed online at: https://www.epa.gov/wqc/methodology-deriving-ambient-water-quality-criteria-protection-human-health-2000-documents

USEPA (U.S. Environmental Protection Agency). 2002a. A Review of the Reference Dose and Reference Concentration Processes. EPA/630/P-02/0002F. Risk Assessment Forum, Washington, DC. Accessed October 2018. Accessed online at: https://www.epa.gov/osa/review-reference-dose-and-reference-concentration-processes

USEPA (U.S. Environmental Protection Agency). 2011. Exposure Factors Handbook: 2011 Edition. EPA/600/R-090/052F. Office of Research and Development, National Center for Environmental Assessment, Washington, D.C. 1436 pp. Accessed October 2018. Accessed online at: https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=236252.

USEPA (U.S. Environmental Protection Agency). Benchmark Dose Technical Guidance. Document # EPA/100/R-12/001. June 2012. Accessed October 2018. Accessed online at: https://www.epa.gov/risk/benchmark-dose-technical-guidance

USEPA (U.S. Environmental Protection Agency). Health Effects Support Document for Perfluorooctanoic acid (PFOA). Document # EPA 822-R-16-003. May 2016. Accessed online at: https://www.epa.gov/sites/production/files/2016-05/documents/pfoa_hesd_final_508.pdf

USEPA (U.S. Environmental Protection Agency). Health Effects Support Document for Perfluorooctane Sulfonate (PFOS). Document # EPA 822-R-16-002. May 2016. Accessed online at: https://www.epa.gov/sites/production/files/2016-05/documents/pfos_hesd_final_508.pdf

Vanden Heuvel JP, Thompson JT, Frame SR, et al. 2006. Differential activation of nuclear receptors by perfluorinated fatty acid analogs and natural fatty acids: A comparison of human, mouse, and rat peroxisome proliferator-activated receptor-α, -ß, and -γ, liver x receptor-ß, and retinoid x receptor-α. Toxicol Sci 92(2):476-489.

Vélez MP, Arbuckle TE, Fraser WD. 2015. Maternal exposure to perfluorinated chemicals and reduced fecundity: The MIREC study. Hum Reprod 30(3):701-709. 10.1093/humrep/deu350.

Verner MA, Loccisano AE, Morken NH, et al. 2015. Associations of perfluoroalkyl substances (PFAS) with lower birth weight: An evaluation of potential confounding by glomerular filtration rate using a physiologically based pharmacokinetic model (PBPK). Environ Health Perspect 123(12):1317-1324.

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

Vieira VM, Hoffman K, Shin M, et al. 2013. Perfluorooctanoic acid exposure and cancer outcomes in a contaminated community: A geographic analysis. Environ Health Perspect 121(3):318-323.

Wallace K, Kissling G, Melnick R, et al. 2013. Structure-activity relationships for perfluoroalkane-induced in vitro interference with rat liver mitochondrial respiration. Toxicol Lett 222(3):257-264.

Wan HT, Zhao YG, Leung PY, et al. 2014b. Perinatal exposure to perfluorooctane sulfonate affects glucose metabolism in adult offspring. PLoS ONE 9(1):e87137. 10.1371/journal.pone.0087137.

Wang J, Yan S, Zhang W, et al. 2015. Integrated proteomic and miRNA transcriptional analysis reveals the hepatotoxicity mechanism of PFNA exposure in mice. J Proteome Res 14(1):330-341. 10.1021/pr500641b.

Washino N, Saijo Y, Sasaki S, et al. 2009. Correlations between prenatal exposure to perfluorinated chemicals and reduced fetal growth. Environ Health Perspect 117:660-667.

White SS, Calafat AM, Kuklenyik Z, et al. 2007. Gestational PFOA exposure of mice is associated with altered mammary gland development in dams and female offspring. Toxicol Sci 96(1):133-144.

White SS, Kato K, Jia LT, et al. 2009. Effects of perfluorooctanoic acid on mouse mammary gland development and differentiation resulting from cross-foster and restricted gestational exposures. Reprod Toxicol 27(3-4):289-298.

White SS, Stanko JP, Kato K, et al. 2011. Gestational and chronic low-dose PFOA exposures and mammary gland growth and differentiation in three generations of CD-1 mice. Environ Health Perspect 119(8):1070-1076.

Wolf CJ, Fenton SE, Schmid JE, et al. 2007. Developmental toxicity of perfluorooctanoic acid in the CD-1 mouse after cross-foster and restricted gestational exposures. Toxicol Sci 95(2):462-473.

Wolf CJ, Takacs ML, Schmid JE, et al. 2008. Activation of mouse and human peroxisome proliferator-activated receptor alpha by perfluoroalkyl acids of different functional groups and chain lengths. Toxicol Sci 106(1):162-171.

Wolf CJ, Zehr RD, Schmid JE, et al. 2010. Developmental effects of perfluorononanoic Acid in themouse are dependent on peroxisome proliferator-activated receptor-alpha. PPAR Res 2010 10.1155/2010/282896.

Wolf CJ, Schmid JE, Lau C, et al. 2012. Activation of mouse and human peroxisome proliferator-activated receptor-alpha (PPARa) by perfluoroalkyl acids (PFAAs): Further investigation of C4-C12 compounds. Reprod Toxicol 33:546-551.

Woskie SR, Gore R, Steenland K. 2012. Retrospective exposure assessment of perfluorooctanoic acid serum concentrations at a fluoropolymer manufacturing plant. Ann Occup Hyg 56(9):1025-1037. 10.1093/annhyg/mes023.

Yahia D, Tsukuba C, Yoshida M, et al. 2008. Neonatal death of mice treated with perfluorooctane sulfonate. J Toxicol Sci 33(2):219-226.

Yahia D, El-Nasser MA, Abedel-Latif M, et al. 2010. Effects of perfluorooctanoic acid (PFOA) exposure to pregnant mice on reproduction. J Toxicol Sci 35(4):527-533.

Yang C, Tan YS, Harkema JR, Haslam SZ. Differential effects of peripubertal exposure to perfluorooctanoic acid on mammary gland development in C57Bl/6 and Balb/c mouse strains. Reprod Toxicol. 2009;27:299–306.Yao X, Zhong L. Genotoxic risk and oxidative DNA damage in HepG2 cells exposed to perfluorooctanoic acid. Mutat Res. 2005 Nov 10;587(1-2):38-44.

Zhang Y, Beesoon S, Zhu L, et al. 2013. Biomonitoring of perfluoroalkyl acids in human urine and estimates of biological half-life. Environ Sci Technol 47(18):10619-10627. 10.1021/es401905e.

Zhao Y, et al. 2010. Perfluorooctanoic Acid Effects on Steroid Hormone and Growth Factor Levels Mediate Stimulation of Peripubertal Mammary Gland Development in C57BI/6 Mice. Toxicological Sciences, 115(1), 214-224.

Zhao Y, et al. 2012. Perfluorooctanoic acid effects on ovaries mediate its inhibition of peripubertal mammary gland development in Balb/c and C57BI/6 mice. Reproductive Toxicology, 33(4): 563-576.

Zhu Y., Qi C., Jain S., Rao M.S., Reddy J.K. Isolation and characterization of PBP, a protein that interacts with peroxisome proliferator-activated receptor. J. Biol. Chem. 1997;272:25500–25506. doi: 10.1074/jbc.272.41.25500

Appendix 9: Analysis of Increased Costs for PWS to comply with Proposed MCLs for PFOA, PFOS, PFNA, PFHxS

Appendix 9: Cost of Compliance with Proposed MCL for PFOA, PFOS, PFNA & PFOA/PFOS Combined for PWS and Private Wells

1.0 PFAS Treatment Costs

Costs to operate and maintain treatment systems to remove PFAS has been prepared assuming treatment is required when

a. PFOS & PFOA combined exceeds 70 parts-per-trillion (ppt);
b. PFOA exceeds 38 ppt;
c. PFNA exceeds 23 ppt; or
d. PFHXS exceeds 85 ppt.

1.1 Occurrence Information

The PFAS sampling results for non-transient PWS were reviewed. Four hundred and two sources of water associated with non-transient PWS were sampled. Two sources of water (0.5% of the sources sampled) equaled or exceeded 70 ppt for PFOA and PFOS combined. Three sources of water (0.75% of the sources sampled) equaled or exceeded 38 ppt for PFOA. Three sources of water exceeded 23 ppt for PFNA, however two of these three sources of water already exceeded the standard for PFOA and PFOA and PFOS combined. None of the results exceeded 85 ppt for PFHxS. Non-transient PWS sources around the Saint Gobain site and the Haven well at Pease Tradeport are not included in the occurrence analysis above, as there are likely not any sources of public drinking water near the type of large scale contamination sources that impacted these wells.

1.2 Costs for Water Treatment for Water Sources Associated with Non-transient PWS With Sampling Results

All sources for PWS that exceed 38 ppt for PFOA and/or 70 ppt for PFOA and PFOS combined already exceed the existing 70 ppt AGQS for PFOA and PFOS combined and costs are already being incurred by these water systems to comply the current AGQS. Therefore, the proposed values of 38 ppt for PFOA and 70 ppt for PFOS and PFOA combined do not require the expenditure of additional funds.

1.3 Costs for Water Treatment for Water Sources Associated with Non-transient Public Water Systems Without Sampling Results

In order to estimate the volume of water that may require treatment for non-transient public water systems that were not sampled, the daily flow volumes for these systems were estimated based on the volume of flow associated with the wellhead protection area for each unsampled source. Generally, this flow volume is the maximum volume that would be used from a particular source.

The cost per gallon to treat water for PFAS can vary broadly. Issues such as the potential for the need to construct a new building, volume of flow, initial PFAS concentrations or pretreatment requirements for constituents such as iron, manganese and radon can cause costs to vary by up to 300% from source to source. The costs per unit of flow used in the estimate were based on the costs associated with treatment at sites in New Hampshire and New York. These are summarized below. The lowest cost per gallon ($2.91 for MVD 4 & 5) and the highest cost per gallons ($8.10 for Pease) were used to develop high and low end estimates.

PFAS Treatment Costs Associated with PWS in New Hampshire and New York:

	Gallons Per Day	Cost	Cost Per Gallon
MVD 4/5	1,152,000	$3,350,000	$2.90
MVD 7/8	1,800,000	$8,000,000	$4.44
Pease	1,728,000	$14,000,000	$8.10
Hooksick Falls	500,000	$3,000,000	$6
Marlow School	1,125	$4,000	$3.56

The treatment costs for sources of water associated with non-transient PWS were estimated. The production volumes associated with the wellhead protection area for the unsampled sources were summed and multiplied by the 0.5% to estimate treated costs associated with sources of water that may exceed 70 ppt PFOA and PFOS combined. Similarly, the production volumes were summed and multiplied by 0.75% to estimate the treatment costs for sources that may exceed 38 ppt for PFOA.

The spreadsheet used to complete the calculations is attached.

The total cost estimates are below:

	Low Estimate	High Estimate
PWS with Sampling Results	$0	$0
Unsampled Public Water Systems	$1,851,354	$5,171,022
Total Cost	$1,851,354	$5,171,022

1.4 Operation and Maintenance Costs for PFAS Water Treatment Systems for Public Water Systems

The operation and maintenance (O&M) cost estimates for PWS were developed using estimated O&M costs associated with the treatment system being constructed for Merrimack Village District's wells 4/5 and the estimated O&M costs associated with the treatment system being constructed at Pease. The estimated annual O&M cost based on the average daily volume that is anticipated to require treatment is $0.18 per gallon to $0.35 per gallon

The annual O&M costs are estimated to be *$114,912 - $223,439* per year.

The cost estimates do not include O&M costs for non-transient public water systems that currently exceed the current AGQS of 70 ppt for PFOA and PFOS combined.

1.5 Chemical Monitoring Costs

Upon the adoption of the proposed MCLs, all non-transient public water systems will be required to sample all sources of their water for four consecutive quarters. After the first year of initial sampling, the average concentration of PFOA, PFOS, PFNA and PFHxS will be calculated for each water source to determine compliance with the MCLs. After the first year of sampling, the frequency of future sampling will be dictated by the results of the first year of sampling. The tables below estimate the cost associated with testing all sources of water on a quarterly basis for the first year and estimated ongoing sampling costs after the first year of sampling.

Assuming Sample Analysis cost of $175 - $450 per sample
1st Year Laboratory Costs - Quarterly Compliance Sampling

Owner	# PWS	# Sites	Initial Cost
State	6	13	$9100 - $23,400
Federal	3	4	$2800 - $7200
Local	274	472	$330,400 - $849,600
Others	907	1086	$760,200 - $1,955,800
TOTAL	1190	1575	$1,102,500 - $2,836,000

Projected Percentage of PWS Sample Sites at Various Contaminant Levels

% of MCL	PFHXS	PFNA	PFOA	PFOS	PFOA + PFOS
ND	86.4%	92.8%	50.3%	79.7%	
<20%	11.4%	3.0%	37.1%	16.4%	38.3%
20 to 75%	2.2%	2.5%	10.7%	3.2%	8.9%
>75% to MCL	0%	1.0%	1.2%	0.5%	1.2%
>=100%	0%	0.7%	0.7%	0.2%	0.5%

Projected Annual Compliance Monitoring Laboratory Costs (years 2 - 9)

Contaminant Range	% of Sites	# of Sites	Sampling Frequency	Cost/Site/Year	Total Sampling Cost/Year
>MCL or Treatment	2%	32	Quarterly	$700 - $1800	$22,400 - $57,600
>75% to MCL	3%	47	Annually	$175 - $450	$8225 - $21,150
20 to 75%	15%	236	Every 3 Years	$60 - $150	$14,160 - $35,400
<20%	19.5%	307	Every 6 Years	$30 - $75	$9210 - $23,025
ND**	60.5%	953	Every 9 Years	$20 - $50	$19,060 - $47,650
				Average Annual Cost	$73,055 - $184,825

**Most sites that have any detection will exceed the threshold value for more than one contaminant. Preliminary study shows 243 out of 402 sites tested as having no detections (60.5%).

1.6 Other Potential Costs that Could Impact Public Water Systems

In southern New Hampshire, several square miles of soil have been contaminated with PFAS due to air emissions. Water utilities completing construction projects in these areas may incur increased costs associated with managing potentially contaminated soils and construction dewatering in these areas.

2.0 Cost Estimates for Private Wells

It is estimated that there are 250,000 private wells in New Hampshire. If it is assumed 0.75% of the private wells in the state will require treatment for PFOA exceeding 38 ppt and 0.5% of the private wells will require treatment for PFOA and PFOS exceeding 70 ppt, the treatment costs will be approximately *$9,375,000 for 3125 private wells*. This assumes there are 250,000 private wells and it will cost $3000 per well to install treatment. [(0.75% x 250,000 wells + 0.5% x 250,000 wells) x $3000/well]

It is estimated that it will cost $900 per year per well to sample and test and maintain treatment systems for PFOS and PFOA. *The total cost annual cost to test and maintain treatment systems for 3125 private wells is estimated to be $2,812,500*.

Appendix 10: Analysis of Increased Costs for Municipal and Private Landfills and Hazardous Waste Sites to comply with Proposed MCLs for PFOA, PFOS, PFNA, PFHxS

Appendix 10: Table 1- Estimated Cost to Hazardous Waste and Landfills Sites for Proposed PFAS MCLs

Est. No. Hazardous Waste Sites	Est. No. of Landfill Sites		Additional Capital Costs	Hazardous Waste Sites	Landfill Sites		Additional Annual Costs	Hazardous Waste Sites	Landfill Sites
Projected # of existing Sites w/ PFAS			*GMP Expansion of Existing Sites*	*Est. Cost*	*Est. Cost*		*GMP Expansion of Existing Sites*	*Est. Cost*	*Est. Cost*
		A	**Monitoring Network Enhancments**			A	**Annual Sampling and Reporting**		
252	**84**		Monitoring Well Install (assume 3 wells) + Initial Sampling Round	$ 12,000	$ 12,000		Annual Sampling/Lab fee (1 round, 3 wells)	$ 3,000	$ 3,000
			Receptor Survey	$ 1,000	$ 1,000		Annual GMP Reporting	$ 2,400	$ 2,400
			Est. Subtotal Capital Cost	$ 13,000	$ 13,000		Est. Subtotal Annual Cost	$ 5,400	$ 5,400
			Numbers below rounded to the nearest $5,000				Numbers below rounded to the nearest $5,000		
	25%		***Est. Total Capital Costs for GMP Expansion*** *(assumes 25% of all sites require expansion)*	**$ 820,000**	**$ 275,000**		***Est. Total Annual Monitoring/Reporting Costs*** *(assumes 25% of all sites require expansion)*	**$ 340,000**	**$ 115,000**
	50%		***Est. Total Capital Cost for GMP Expansion*** *(assumes 50% of all sites require expansion)*	**$ 1,635,000**	**$ 545,000**		***Est. Total Annual Monitoring/Reporting Costs*** *(assumes 50% of all sites require expansion)*	**$ 680,000**	**$ 225,000**
		B	**Water Supply Well Treatment**			B	**Water Supply Well Treatment**		
			POE Install -assume 3 per site	$ 3,000	$ 3,000		Annual O&M of POE (assume 3 per site)	$ 1,000	$ 600
			Est. Subtotal Cost	$ 9,000	$ 9,000		Est. Subtotal Annual O&M Cost	$ 3,000	$ 1,800
			Numbers below rounded to the nearest $5,000				Numbers below rounded to the nearest $5,000		
	10%		***Est. Total for Expansion of Sites 10% of all sites will have 3 new POEs***	**$ 225,000**	**$ 75,000**		***Est. Total for Expansion of Sites 10% of all sites will have 3 new POEs***	**$ 75,000**	**$ 15,000**
	20%		***Est. Total for Expansion of Sites - 20% of all sites will have 3 new POEs***	**$ 455,000**	**$ 150,000**		***Est. Total for Expansion of Sites 20% of all sites will have 3 new POEs***	**$ 150,000**	**$ 30,000**
						C	**NHDES Staff Time (Assume Annual Salary/benefits for 2 FTE staff will be required at $120,000/yr)**	$ 120,000	$ 120,000
			I. Est. Capital Cost range for GMZ Expansion: Low	**$ 1,045,000**	**$ 350,000**		**I. Est. Annual Cost range for GMZ Expansion: Low**	**$ 535,000**	**$ 250,000**
			High	**$ 2,090,000**	**$ 695,000**		**High**	**$ 950,000**	**$ 375,000**
Projected # of Sites w/ PFAS Exceedances			*Sites that may be required to address PFAS as a new Contaminant of Concern*	*Est. Cost*	*Est. Cost*		*Sites that may be required to address PFAS as a new Contaminant of Concern*	*Est. Cost*	*Est. Cost*
19	**5**	A	**Monitoring Network Enhancments**			A	**Annual Sampling and Reporting**		
			Monitoring Well Install (assume 5 wells) + Initial Sampling Round	$ 18,000	$ 18,000		Annual Sampling/Lab fee (1 round, 5 wells)	$ 3,500	$ 3,500
			Receptor Survey	$ 1,500	$ 1,500		Annual GMP Reporting	$ 2,900	$ 2,900
			Est. Subtotal Cost	$ 19,500	$ 19,500		Est. Subtotal Cost	$ 6,400	$ 6,400
			Numbers below rounded to the nearest $5,000				Numbers below rounded to the nearest $5,000		
	25%		**Est. Total for New Sites - 25%**	**$ 90,000**	**$ 25,000**		***Est. Total Annual Monitoring Costs for New Sites - 25% of all sites***	**$ 30,000**	**$ 10,000**
	50%		**Est. Total for New Sites - 50%**	**$ 185,000**	**$ 50,000**		***Est. Total Annual Monitoring Costs for New Sites - 50% of all sites***	**$ 60,000**	**$ 15,000**
		B	**Water Supply Well Treatment**			B	**Water Supply Well Treatment**		
			POE Install - assume 3 per site	$ 3,000	$ 3,000		Annual O&M of POE (assume 3 per site)	$ 1,000	$ 600
			Est. Subtotal Cost	$ 9,000	$ 9,000		Est. Subtotal Cost	$ 3,000	$ 1,800
			Numbers below rounded to the nearest $5,000				Numbers below rounded to the nearest $5,000		
	10%		***Est. Total for New Sites 10% of all sites will have 3 new POEs***	**$ 15,000**	**$ 5,000**		***Est. Total for New Sites 10% of all sites will have 3 new POEs***	**$ 5,000**	**$ - **
	20%		***Est. Total for New Sites 20% of all sites will have 3 new POEs***	**$ 35,000**	**$ 10,000**		***Est. Total for New Sites 20% of all sites will have 3 new POEs***	**$ 10,000**	**$ - **
			II. Est. Cost range for Sites w/ PFAS as New COC: Low	**$ 105,000**	**$ 30,000**		**I. Est. Annual Cost range for or Sites w/ PFAS as New COC: Low**	**$ 35,000**	**$ 10,000**
			High	**$ 220,000**	**$ 60,000**		**High**	**$ 70,000**	**$ 15,000**
			Est. Total Capital Cost Impacts for Proposed MCLs: Low	**$ 1,150,000**	**$ 380,000**		**Est. Total Annual Operating Budget Impacts for Proposed MCLs: Low**	**$ 570,000**	**$ 260,000**
			High	**$ 2,310,000**	**$ 755,000**		**High**	**$ 1,020,000**	**$ 390,000**

Hazardous Waste Sites — Landfills

$1.15M to $2.31M — $380K to $755K — Additional capital cost to expand existing GMZs, establish new sites and treat impacted drinking water supply wells.

$570 to $1.0M — $260K to $390K — Additional annual operating costs (monitoring and reporting), and NHDES permit administration costs

For the Following Standards (PPT):
PFOA = 38
PFOS = 70
PFNA = 23
PFHxS = 85
PFOA+PFOS = 70

Appendix 10: Table 2 Estimated Cost to Hazardous Waste and Landfill Sites for Proposed MCLs

Hazardous Waste Site Projections are based on:	
515	Hazardous Waste Sites
137	Number of sites PFAS Sampling has been completed
27%	Percent of Sites Sampled
Analysis of Existing Data and Current Standard of 70 PPT PFOA + PFOS	
Of the 137	sites sampled:
49%	had exceedances of the current standard
9%	had water supply wells with exceedances of current standards
Estimate of # of Hazardous Waste Sites with Existing PFAS Compliance Issues	
Assumption: *Apply similar trend of existing data outlined above.*	
252	sites may have exceedances of the current standard
25 to 50	estimated number of sites with drinking water impacts[1]
Analysis of Existing Data and Proposed Standards in Parts per Trillion	
PFOA	38
PFOS	70
PFNA	23
PFHxS	85
PFOA+PFOS	70
53%	of sites sampled w/ exceed. of proposed stds of one or more compounds
27 to 54	estimated number of sites with drinking water impacts[1]

Notes:
1. Based on the limited data to estimate this, NHDES used a range of 10-20% of the projected number of sites with exceedances.

Landfill Site Projections are based on:	
201	Landfill Sites
117	Number of sites PFAS Sampling has been completed
58%	Percent of Sites Sampled
Analysis of Existing Data and Current Standard of 70 PPT PFOA + PFOS	
Of the 117	sites sampled:
42%	had exceedances of the current standard
1%	had water supply wells with exceedances of current standards
Estimate of # of Landfill Sites with Existing PFAS Compliance Issues	
Assumption: *Apply similar trend of existing data outlined above.*	
84	sites may have exceedances of the current standard
8 to 17	estimated number of sites with drinking water impacts[1]
Analysis of Existing Data and Proposed Standards in Parts per Trillion	
PFOA	38
PFOS	70
PFNA	23
PFHxS	85
PFOA+PFOS	70
44%	sites sampled w/ exceed. of proposed stds of one or more compounds
9 to 18	estimated number of sites with drinking water impacts[1]

Notes:
1. Based on the limited data to estimate this, NHDES used a range of 10-20% of the projected number of sites with exceedances.

394

Appendix 10: Table 3-Estimated Cost to Select Southern New Hampshire Hazardous Waste Sites for Proposed MCLs

Additional Capital Costs			Additional Annual Costs		Hazardous Waste Sites
	Additional Private Well Testing [2,3]	***Est. Cost***		***Additional Private Well Testing***	***Est. Cost***
A	**Additional Private Well Testing**		**A**	**Additional Annual Private Well Sampling and Reporting**	
	Initial Sampling Round (assume 500 wells)	$ 500,000		Annual Sampling/Lab fee (2 rounds, 50 wells)	$ 100,000
	Receptor Survey	$ 10,000		Annual GMP Reporting	$ 10,000
	Est. Subtotal Capital Cost	$ 510,000		Est. Subtotal Annual Cost	$ 110,000
	Provision of Alternate Water [5]	***Est. Cost***		***Provision of Alternate Water***	***Est. Cost***
B	**Water Supply Well Treatment** [5]		**B**	**Water Supply Well Treatment**	
	POE installations (assume 180)	$ 3,000		Annual O&M of POE (assume 150)	$ 1,000
	Est. Subtotal Cost	$ 540,000		Est. Subtotal Annual O&M Cost	$ 180,000
C	**Waterline Connections** [6]		**C**	**Waterline Connections**	
	In areas with existing waterlines (assume 65)	$ 15,000		N/A	
	Est. Subtotal Cost	$ 975,000		Est. Subtotal Annual O&M Cost	$ -
	Total Costs (A,B, and C)	$2,025,000		Total Costs (A,B, and C)	$ 290,000
	Est. Total Capital Cost Impacts for Proposed MCLs: Low (75% of Total Costs)	**$1,520,000**		**Est. Total Annual Cost Impacts for Proposed MCLs: Low (75% of Total Costs)**	**$ 220,000**
	High (125% of Total Costs)	**$2,530,000**		**High (125% of Total Costs)**	**$ 365,000**

Notes and Assumptions:

Costs presented in the table above are for two large sites in southern New Hampshire, where groundwater in portions of the communities of Amherst, Bedford, Hollis, Litchfield, Londonderry, Manchester, and Merrimack has been impacted by PFAS.

1. The number of additional potentially impacted properties is unknown. An extrapolation of the sample results from private drinking water wells was completed to provide a general screening-level approximation of the number of additional properties that could potentially be impacted. Note the dataset used in the extrapolation contains data from both overburden and bedrock wells and wells of various depths, and most of the well were only sampled on one occasion. Additional sampling will be required to evaluate actual concentrations in groundwater. In areas where information about water sources for individual properties was not available, it was assumed that properties within a proximity of a waterline were connected to public water; all other properties were assumed to be served by private wells. This information needs to be confirmed.

2. Based on the extrapolation, approximately 500 properties are located in areas where groundwater could be impacted by PFOA at concentrations greater than half of the proposed AGQS. The actual number will likely vary based on further evaluation of sample results.

3. Potential additional site investigation costs are not able to be determined, as plans for off-site investigations have not yet been developed.

4. A determination of sources of alternate water will be made following an evaluation of additional sampling data and feasibility. For this cost estimate, it was assumed that approximately half of the properties sampled would need alternate water.

5. For purposes of this cost estimate, it was assumed that point-of-entry treatment systems (POEs) would be provided in areas where waterlines are currently not present.

6. For purposes of this cost estimate, it was assumed that connections to public water would be provided only in areas where waterlines are already present. These costs assume that no new water main extensions would needed.

Capital costs would be significantly higher if water main extensions would be required to service those properties in Section B that are assumed to be covered by POEs. Costs for additional waterline extensions are not able to be determined at this time and would vary significantly based on the number of properties served, length of water main needed, service connection lengths, water source, and contractor pricing, but could potentially be in the ballpark of $10-45 MM.

Appendix 11: Analysis of Increased Costs for Groundwater Discharge Permittees to comply with Proposed MCLs for PFOA, PFOS, PFNA, PFHxS

Cost Estimates - Reduction in PFAS Standards - Groundwater Discharge Permit (GWDP) Sites

Isolated Sites : Non-Developed Areas, Able to Expand Groundwater Discharge Zones (GDZ), No Private/Public Water Supply Receptors

Small GWDP Sites
Non POTW sites, usually privately owned

Additional Capital Costs					Additional Annual Costs			
	Item	Count	Unit Cost	Total	Item	Count	Unit Cost	Total
	Mon Well	3	$ 12,000	$ 36,000	Smpl Rnd	6	$ 1,000	$ 6,000
	Priv Well Svy	1	$ 1,000	$ 1,000	Rpting	1	$ 2,400	$ 2,400
			Total	*$ 37,000*			*Total*	*$ 8,400*
	1X	***Add'l sites***		***$ 37,000***	1X	***Add'l sites***		***$ 8,400***

Large GWDP Sites
POTW sites, usually publicly owned

Additional Capital Costs					Additional Annual Costs			
	Item	Count	Unit Cost	Total	Item	Count	Unit Cost	Total
	Mon Well	6	$ 12,000	$ 72,000	Smpl Rnd	12	$ 1,000	$ 12,000
	Priv Well Svy	1	$ 1,000	$ 1,000	Rpting	1	$ 2,400	$ 2,400
			Total	*$ 73,000*			*Total*	*$ 14,400*
	3X	***Add'l sites***		***$ 219,000***	3X	***Add'l sites***		***$ 43,200***

Non-Isolated Sites : Developed Areas, Not (Easily) Able to Expand GDZ, Private/Public Water Supply Receptors Present

Small GWDP Sites
Non POTW sites, usually privately owned

Additional Capital Costs					Additional Annual Costs			
	Item	Count	Unit Cost	Total	Item	Count	Unit Cost	Total
	Mon Well	2	$ 12,000	$ 24,000	Smpl Rnd	4	$ 1,000	$ 4,000
	Priv Well Svy	1	$ 2,500	$ 2,500	Rpting	1	$ 2,400	$ 2,400
	POE-PFAS	3	$ 3,000	$ 9,000	O&M	3	$ 900	$ 2,700
			Total	*$ 35,500*			*Total*	*$ 9,100*
	Fac Trtmnt	***Range: 10k to 100k***						
	2X	***Add'l sites***		***$ 71,000***	2X	***Add'l sites***		***$ 18,200***

Large GWDP Sites
POTW sites, usually publicly owned

Additional Capital Costs					Additional Annual Costs			
	Item	Count	Unit Cost	Total	Item	Count	Unit Cost	Total
	Mon Well	4	$ 12,000	$ 48,000	Smpl Rnd	8	$ 1,000	$ 8,000
	Priv Well Svy	1	$ 5,000	$ 5,000	Rpting	1	$ 2,400	$ 2,400
	POE-PFAS	6	$ 3,000	$ 18,000	O&M	6	$ 900	$ 5,400
			Total	*$ 71,000*			*Total*	*$ 15,800*
	Fac Trtmnt	***Flows too large***						
	1X	***Add'l sites***		***$ 71,000***	1X	***Add'l sites***		***$ 15,800***

	Additional Capital Costs	Additional Annual Costs
Multiplier 2.3	**Add'l at new PFAS st $ 915,400**	**Add'l at new PFAS st $196,880**
4x sites	***Fac Trtmnt Range : $20,000 to $200,000***	*Small Facilities only

New PFAS Standard Evaluated:
PFOA: 38 ppt
PFOS: 70 ppt
PFOA + PFOS: 70 ppt
PFNA: 23 ppt
PFHxS: 85 ppt

SUMMARY

-For change to lower PFAS standards:
- Adds ~12 GWDP sites to the list of sites with PFAS compliance issues.
-Adds ~ $900K to capital costs
-Adds ~ $200K to annual costs

Sites with Existing PFAS issues:
-Potential additional costs to sites with existing compliance issues that exceed the current PFAS standard : ~$200K

Cost impact to small (mostly privately owned) GWDP sites could be greater if WW pre-treatment is put in place: estimate ~ $20K to $200K capital costs

Assumption Summary for development of Cost Impacts to Groundwater Discharge Permit (GWDP) sites due to the lowering of the PFAS standards

Breakdown of all Sites in GWDP program: 96 GWDP sites - Four Categories

Geographically Isolated Sites: -Located in non-developed area -Commonly able to easily expand GDZ -No public or private water wells nearby (no receptors)	**Small sites:** -Flows less than 50K per day -Usually privately owned -Contaminant specific treatment may be feasible
	Large sites: -Flows greater than 50K per day -Usually publically owned POTW -Contaminant specific treatment usually NOT feasible
Non-isolated Sites: -Located in developed area -Not easily able to expand GDZ -Public and/or private water wells nearby (receptors)	**Small sites:** -Flows less than 50K per day -Usually privately owned -Contaminant specific treatment may be feasible
	Large sites: -Flows greater than 50K per day -Usually publically owned POTW -Contaminant specific treatment usually NOT feasible

Breakdown of GWDP sites with PFAS in groundwater at or above current AGQS based on sampling:

- 1-Isolated Small sites
- 2-Isolated Large sites
- 0-Non Isolated Small sites
- 1-Non Isolated Large sites

Assumptions related to number of GWDP sites affected by lowering of PFAS standards:

- For new PFAS standard, the number of current sites that would exceed standards at those sites that have sampled would increase from 4 sites to 7 sites.
- Forty two (42) of 96 sites have sampled, therefore number of exceeding sites were scaled up by a factor or 2.3 (96/42) projecting exceedances at approximately 16 groundwater discharge permit sites across the entire population of permit holders.

Response actions at sites that exceed the new standard that impact cost:

- Isolated sites:
  - Conduct Receptor Survey
  - Expand GDZ where feasible
  - Add monitoring wells (3 per small site, 6 per large site)
  - Conduct additional annual sampling
- Non-Isolated sites
  - Conduct Receptor Survey
  - Expand GDZ where feasible
  - Add monitoring wells (less than isolated sites)
  - Conduct additional annual sampling
  - Install POE treatment systems (up to 3 units per small site, up to 6 units per large site)

Private Well Mitigation Considerations: POE only, no public water system extensions or connections
WW Treatment Considerations: Modifications to WW treatment systems are only feasible at Small Sites

LBAO
18-2838
12/19/17

SB 309-FN- FISCAL NOTE
AS INTRODUCED

AN ACT relative to standards for perfluorochemicals in drinking water, ambient groundwater, and surface water.

FISCAL IMPACT: **[X] State** **[X] County** **[X] Local** **[ ] None**

	Estimated Increase / (Decrease)			
STATE:	**FY 2019**	**FY 2020**	**FY 2021**	**FY 2022**
Appropriation	$0	$0	$0	$0
Revenue	$0	$0	$0	$0
Expenditures	Indeterminable Increase	Indeterminable Increase	Indeterminable Increase	Indeterminable Increase
Funding Source:	[X] General	[ ] Education	[ ] Highway	[ ] Other

COUNTY:

Revenue	$0	$0	$0	$0
Expenditures	Indeterminable Increase	Indeterminable Increase	Indeterminable Increase	Indeterminable Increase

LOCAL:

Revenue	$0	$0	$0	$0
Expenditures	Indeterminable Increase	Indeterminable Increase	Indeterminable Increase	Indeterminable Increase

METHODOLOGY:

This bill requires the commissioner of the Department of Environmental Services to adopt a state drinking water standard relative to perfluorochemicals (PFCs); establish ambient groundwater quality standards relative to PFCs; and establish surface water quality standards relative to PFCs.

Regarding section 1 of the bill, the Department of Environmental Services indicates there are approximately 4,200 active sources of water for public water systems that will need to be sampled routinely if a Maximum Contaminant Level (MCL) is adopted. The frequency of sampling would be increased or decreased based on previous monitoring results from a given source. Laboratory costs for perfluorinated compounds ranges from $180 to $400 per sample. Therefore a single round of sampling for locals, counties and other entities that own or operate public water systems would range from $756,000 to $1,680,000 or higher depending on how low the standard is set. The impact on expenditures cannot be determined because the frequency of sampling and the number of water sources exceeding the MCL cannot be determined in

advance. The cost to the Department associated with administering compliance are indeterminable for the same reasons.

Regarding section 2, the Department would need to independently review available research and analyze whether that research warrants revision of ambient groundwater quality standards (AGQS) on an annual basis for perfluorooctanesulfate (PFOS) and perfluorooctanic acid (PFOA). The Department assumes an additional Toxicologist IV, labor grade 29, step 4 position would be necessary, starting on July 1, 2018 with the following estimated costs:

	FY 2019	FY 2020	FY 2021	FY 2022
Salary	$66,905	$69,791	$69,791	$72,911
Benefits	$31,484	$33,289	$34,614	$36,573
Total Salary & Benefits:	$98,389	$103,080	$104,405	$109,484
Other Expenses:				
Current Expenses	$5,200	$5,200	$5,200	$5,200
Equipment	$3,926	$500	$500	$500
Office Space	$3,693	$3,806	$3,880	$3,960
Travel	$4,500	$2,750	$2,750	$2,750
DoIT Charges, Training and Telecommunications	$9,112	$2,064	$2,064	$2,064
Total Other Expenses:	$26,431	$14,320	$14,394	$14,474
Total:	**$124,820**	**$117,400**	**$118,799**	**$123,958**

In addition, a potential reduction in the current AGQS for PFOA and PFOS may result in additional indeterminable costs to local and county government entities that hold groundwater discharge or management permits such as those associated with landfills.

Regarding section 3, in order to establish surface water quality standards for PFCs, the Department would need funds to hire a contractor, experienced in PFCs and the EPA's methodologies for developing aquatic life and human health surface water quality criteria. The contractor would review existing literature, including criteria and assumptions used in other states, and develop a report with defensible aquatic life and human health surface water criteria and supporting documentation consistent with EPA methodologies. To accomplish this within 120 days, as required, the Department assumes the cost would exceed $100,000. For surface water quality standards, the cost to the municipalities could be significant, but indeterminable. Many municipalities operate waste water treatment facilities that discharge treated water to local groundwater or surface waters. Should those surface waters fail to meet surface water quality standards, treatment technologies or industrial pretreatment programs, may need to be developed. For larger facilities, the expense of that treatment could be millions of dollars. In addition, some municipalities have firefighting or fire training facilities which have the potential to cause surface water impairments, the remediation cost for these facilities could be high. For counties, the cost is likely to be lower unless contamination is found to originate from a county facility.

The total costs to the Department and other entities are indeterminable, however at least one general funded position and funds for a contractor would be necessary as outlined above. In addition, potential costs to local and county governments for treatment, mitigation, and remediation in order to comply with a new MCL, ambient groundwater standard, and surface water quality standard could be significant, but are also indeterminable.

AGENCIES CONTACTED:

Department of Environmental Services

Effective September 30, 2019, Env-Dw 701.03 reads as follows [new paragraph (d), existing paragraphs (d) and (e) renumbered as (e) and (f)]:

Env-Dw 701.03 Units of Measure for Maximum Contaminant Levels (MCLs) and Maximum Contaminant Level Goals (MCLGs). The units of measure for MCLs and MCLGs shall be as follows:

(a) Picocuries per liter, abbreviated as pCi/L;

(b) Milligrams per liter, abbreviated as mg/L;

(c) Micrograms per liter, abbreviated as µg/L;

(d) Nanograms per liter, abbreviated as ng/L;

(e) Millirem per year, abbreviated as mrem/year; and

(f) Fibers per liter, abbreviated as fibers/L.

Effective September 30, 2019, Env-Dw 705.06 reads as follows:

Env-Dw 705.06 MCLs and MCLGs for Per- and Polyfluoroalkyl Substances (PFAS) Contaminants.

(a) The MCLs and MCLGs for the per- and polyfluoroalkyl substances contaminants specified in (b), below, shall apply to community water systems and non-transient non-community water systems.

(b) The MCLs and MCLGs for PFAS contaminants shall be as specified in Table 705-7, below:

Table 705-7: PFAS Contaminant MCLs and MCLGs

PFAS Contaminant	MCL (mg/L)	MCLG (mg/L)
Perfluorohexane sulfonic acid (PFHxS)	0.000018	0
Perfluorononanoic acid (PFNA)	0.000011	0
Perfluorooctane sulfonic acid (PFOS)	0.000015	0
Perfluorooctanoic acid (PFOA)	0.000012	0

(c) Monitoring and compliance for PFAS contaminants shall be as specified in Env-Dw 707, Env-Dw 708, and Env-Dw 712.

Effective September 30, 2019, Env-Dw 707.06 reads as follows [changes to (d) and (e) only]:

Env-Dw 707.06 Sample Analysis Methods; Sample Collection Protocol; Approval of Alternative Methods.

(a) Acceptable laboratory methods, detection limits, and sample collection protocols shall be those specified in 40 CFR 141, 142, or 143, as applicable.

(b) The O/O of a PWS having its own laboratory or the O/O of a laboratory used by one or more PWS who wishes to use a method other than one specified in (a), above, shall obtain written permission from the department as specified in (c) through (e), below, prior to using any alternative method.

(c) The O/O shall submit a request to use an alternative method in writing to the program manager of the NH environmental laboratory accreditation program (NH ELAP) at the address specified in Env-C 303.01(a).

(d) The request shall include all relevant information, including:

(1) The reason(s) for requesting approval of the alternate method; and

(2) Analytical data demonstrating the precision and accuracy of the alternative method as it relates to the determination of compliance with the applicable standard.

(e) An alternative method shall be approved only if the NH ELAP program manager with the concurrence of the administrator of the U.S. EPA determines that the method is equivalent to or better than the prescribed test in both precision and accuracy as it relates to the determination of compliance with the applicable standard.

Effective September 30, 2019, Env-Dw 712.23 through Env-Dw 712.30 read as follows [all new]:

Env-Dw 712.23 Initial Monitoring for Per- and Polyfluoroalkyl Substances (PFAS) Contaminants.

(a) Beginning with the first quarter following the 2019 effective date of this section, the O/O of an existing community water system or existing non-transient, non-community water system shall collect 4 consecutive quarterly samples for the per- and polyfluoroalkyl substances contaminants listed in Env-Dw 705.06 at each sampling point identified in the sampling schedule established pursuant to Env-Dw 708.01.

(b) Beginning with the first quarter following the initiation of operations of a new community water system or new non-transient, non-community water system, the O/O shall collect 4 consecutive quarterly samples for the PFAS contaminants listed in Env-Dw 705.06 at each sampling point identified in the sampling schedule established pursuant to Env-Dw 708.01.

(c) If the results of the samples from the first 2 quarters are below the detection limits specified in Env-Dw 712.28(c), the O/O may submit a written request to the department for the monitoring frequency to be reduced.

(d) A written request submitted pursuant to (c), above, shall include the following:

(1) The name of the PWS;

(2) The PWS identifier for the PWS; and

(3) A summary of the historical PFAS data from the system and nearby systems, when available.

(e) If the department determines that the results are all below the detection limits listed in Table 712-2, the final 2 quarters of the initial monitoring shall be waived and the monitoring frequency shall be as specified in Env-Dw 712.24.

Env-Dw 712.24 Monitoring Frequency for PFAS Contaminants.

(a) Subsequent to the initial monitoring required by Env-Dw 712.23 and subject to Env-Dw 712.26, the O/O shall monitor for all PFAS contaminants based on the PFAS contaminant with the most frequent monitoring period calculated from the average of the results of the initial monitoring required by Env-Dw 712.23, as specified in Table 712-1, below, and as demonstrated in Appendix D for specific PFAS contaminants:

Table 712-1: Monitoring Frequency Based on PFAS Contaminant Concentrations

Average Monitoring Result (ng/L)	Frequency
Greater than 50% of MCL to 100% of MCL	Annually
50% of MCL or less	Once every 3 years

(b) If the average monitoring result exceeds 100% of the MCL, the O/O shall monitor as specified in Env-Dw 712.27.

(c) The O/O shall monitor for PFAS contaminants during the quarter in which the highest analytical result was observed.

(d) Subsequent sample results shall be used to establish future PFAS contaminant sampling schedules using the shortest PFAS monitoring period specified in Table 712-1.

(e) Based on a review of the submitted results, the department shall:

(1) Modify the system's schedule in accordance with Table 712-1 or (b), above, as applicable; and

(2) Notify the O/O in writing of the new monitoring requirements.

Env-Dw 712.25 Monitoring Location for PFAS Contaminants.

(a) The O/O of a PWS supplied by a groundwater source shall collect at least one sample to be analyzed for PFAS contaminants at every entry point to the distribution system. Each entry point shall be representative of each well after treatment, as specified in the sampling schedule established pursuant to Env-Dw 708.01.

(b) The O/O of a PWS supplied by a surface water source or a combination of surface water and groundwater shall collect at least one sample to be analyzed for PFAS contaminants at points in the distribution system that are representative of each source or at each entry point to the distribution system after treatment, as specified in the sampling schedule established pursuant to Env-Dw 708.01.

(c) If the O/O believes that conditions make another sampling point more representative of a source, treatment plant, or distribution system for purposes of sampling for PFAS contaminants, the O/O shall request a change in sampling location for such contaminants pursuant to Env-Dw 708.04.

(d) If a PWS obtains water from more than one source and the sources are combined prior to entering the distribution system, the O/O shall collect the samples to be analyzed for PFAS contaminants at an entry point to the distribution system during periods of normal operating conditions, when water from all sources is being used.

Env-Dw 712.26 Confirmation Sampling for PFAS Contaminants.

(a) Subject to (c), below, if a PFAS contaminant is detected in a representative sample at a level greater than 50% of the MCL, the O/O shall:

(1) Collect a confirmation sample under the same contributing conditions within 14 days of being notified of the result; and

(2) Have the sample analyzed for the contaminant(s) detected.

(b) If a confirmation sample is required pursuant to (a) above, the results of the initial and confirmation samples shall be averaged to determine compliance with the MCL specified in Env-Dw 705.06.

(c) If results from the sampling point or the contributing sources have historically demonstrated the presence of that PFAS contaminant at a level greater than 50% of the MCL, then:

(1) A confirmation sample shall not be required; and

(2) The monitoring frequency for the approved sampling point shall be determined pursuant to Env-Dw 712.24 or Env-Dw 712.27, as applicable.

Env-Dw 712.27 Increased Monitoring for PFAS Contaminants. The O/O shall collect and analyze quarterly PFAS samples at all sampling points if:

(a) The running annual average for any PFAS contaminant at the sampling point is above the applicable MCL; or

(b) The PWS is operating any type of treatment to reduce the amount of a PFAS contaminant.

Env-Dw 712.28 Laboratory Methods, Sampling Protocols, and Detection Limits for PFAS Contaminants.

(a) Analysis for PFAS contaminants shall be conducted only by laboratories that are accredited by the department for such analyses pursuant to Env-C 300.

(b) Samples to be analyzed for PFAS contaminants shall be collected in accordance with the protocol specified in the sample analysis method approved per Env-Dw 707.06.

(c) Detection limits for PFAS contaminants shall not exceed those set forth in Table 712-2, below:

Table 712-2: Detection Limits for PFAS Contaminants

PFAS Contaminant	Detection Limit
Perfluorohexane sulfonic acid (PFHxS)	2 ng/L
Perfluorononanoic acid (PFNA)	2 ng/L
Perfluorooctane sulfonic acid (PFOS)	2 ng/L
Perfluorooctanoic acid (PFOA)	2 ng/L

Env-Dw 712.29 Compliance Determination for PFAS Contaminants; Limiting Public Notice.

(a) Compliance with Env-Dw 705.06 shall be determined using the analytical results obtained at each sampling point that is an entry point to the distribution system, as specified in the sampling schedule established pursuant to Env-Dw 708.01.

(b) For any PWS that conducts monitoring for PFAS contaminants at a frequency greater than annually, the department shall determine compliance by calculating a running annual average of all samples collected at each sampling point. If the annual average of any sampling point is greater than the MCL, then the department shall identify the PWS as out of compliance.

(c) If monitoring for PFAS contaminants is conducted annually or less frequently, then the department shall identify the PWS as being out of compliance if the level of a PFAS contaminant at any sampling point is greater than the MCL.

(d) If a PWS has a distribution system with portions that are hydraulically separate from other parts of the distribution system, the O/O may request approval from the department pursuant to Env-Dw 801 to limit the notice to only that portion that is out of compliance.

Env-Dw 712.30 Recordkeeping and Reporting for PFAS Contaminants. An O/O shall:

(a) Maintain records of PFAS contaminant analyses for not less than 10 years and as specified in Env-Dw 718; and

(b) Report monitoring results for PFAS contaminants as specified in Env-Dw 719.

Effective September 30, 2019, Env-Dw 808.01 reads as follows:

PART Env-Dw 808 HEALTH EFFECTS LANGUAGE FOR SYNTHETIC ORGANIC CHEMICAL (SOC) CONTAMINANTS AND PER- AND POLYFLUOROALKYL SUBSTANCES (PFAS) CONTAMINANTS

Env-Dw 808.01 Required Health Effects Language for Regulated Synthetic Organics Chemical (SOC) Contaminants and Per- and Polyfluoroalkyl Substances (PFAS) Contaminants. The O/O shall use the statements specified in this part, as applicable, as the statement required by Env-Dw 801.03(a)(3) to describe the adverse health effects for the synthetic organic chemical (SOC) contaminants specified in Env-Dw 705.02 and the per- and polyfluoroalkyl substances (PFAS) contaminants specified in Env-Dw 705.06.

Effective September 30, 2019, new sections Env-Dw 808.27 through Env-Dw 808.30 read as follows [former sections Env-Dw 808.27 through Env-Dw 808.34 renumbered as Env-Dw 808.31 through Env-Dw 808.38]:

Env-Dw 808.27 Perfluorohexane Sulfonic Acid (PFHxS). For perfluorohexane sulfonic acid (PFHxS) violations, the statement shall read as follows:

> "Some people who drink water containing perfluorohexane sulfonic acid (PFHxS) in excess of the MCL over many years could experience problems with their liver, endocrine system, or immune system, or may experience increased cholesterol levels. It may also lower a women's chance of getting pregnant."

Env-Dw 808.28 Perfluorononanoic Acid (PFNA). For perfluorononanoic acid (PFNA) violations, the statement shall read as follows:

> "Some people who drink water containing perfluorononanoic acid (PFNA) in excess of the MCL over many years could experience problems with their liver, endocrine system, or immune system, or may experience increased cholesterol levels."

Env-Dw 808.29 Perfluorooctane Sulfonic Acid (PFOS). For perfluorooctane sulfonic acid (PFOS), violations, the statement shall read as follows:

> "Some people who drink water containing perfluorooctane sulfonic acid (PFOS) in excess of the MCL over many years could experience problems with their liver, endocrine system, or immune system, may experience increased cholesterol levels, and may have an increased risk of getting certain types of cancer. It may also lower a women's chance of getting pregnant."

Env-Dw 808.30 Perfluorooctanoic Acid (PFOA). For perfluorooctanoic acid (PFOA) violations, the statement shall read as follows:

> "Some people who drink water containing perfluorooctanoic acid (PFOA) in excess of the MCL over many years could experience problems with their liver, endocrine system, or immune system, may experience increased cholesterol levels, and may have an increased risk of getting certain types of cancer. It may also lower a women's chance of getting pregnant."

Effective September 30, 2019, Env-Dw 811.02 reads as follows [only (d) revised]:

Env-Dw 811.02 Definitions. For purposes of this part, the following definitions shall apply unless otherwise specified:

(a) "Action level (AL)" means the concentration of a contaminant which, if exceeded, triggers treatment or other requirements which a water system must follow;

(b) "Consumer confidence report (CCR)" means an annual report supplied by a CWS O/O to customers which contains information on the quality of their drinking water;

(c) "Customers" means billing units or service connections to which water is delivered by a CWS;

(d) "Detected" means the presence of any primary or secondary drinking water contaminant including:

(1) Microbiological contaminants;

(2) Radiological contaminants;

(3) IOC contaminants;

(4) VOC contaminants;

(5) SOC contaminants;

(6) PFAS contaminants; and

(7) Disinfection by-products;

(e) "Regulated contaminant" means a contaminant that is subject to a maximum contaminant level (MCL), action level (AL), maximum residual disinfectant level (MRDL), or treatment technique (TT); and

(f) "Unregulated contaminant" means a contaminant specified in 40 CFR 141.40.

Effective September 30, 2019, Env-Dw 811.07(c) reads as follows [no change to (a), (b), or (d)]:

Env-Dw 811.07 Health Effects Language.

(c) Subject to (d), below, the CWS O/O shall use the following language to satisfy the requirements of (b), above:

"The sources of drinking water (both tap water and bottled water) include rivers, lakes, streams, ponds, reservoirs, springs, and wells. As water travels over the surface of the land or through the ground, it dissolves naturally-occurring minerals and, in some cases, radioactive material, and can pick up substances resulting from the presence of animals or from human activity.

Contaminants that may be present in source water include:

Microbial contaminants, such as viruses and bacteria, which may come from sewage treatment plants, septic systems, agricultural livestock operations, and wildlife.

Inorganic contaminants, such as salts and metals, which can be naturally occurring or result from urban stormwater runoff, industrial or domestic wastewater discharges, oil and gas production, mining or farming.

Pesticides and herbicides, which may come from a variety of sources such as agriculture, urban stormwater runoff, and residential uses.

Organic chemical contaminants, including per- and polyfluoroalkyl substances, synthetic organic chemicals, and volatile organic chemicals, which are byproducts of industrial processes, wastewater treatment, residuals from firefighting foams, and petroleum production, and can also come from gas stations, urban stormwater runoff, and septic systems.

Radioactive contaminants, which can be naturally- occurring or be the result of oil and gas production and mining activities.

In order to ensure that tap water is safe to drink, EPA and the State of New Hampshire prescribe regulations that limit the amount of certain contaminants in water provided by public water systems. The United States Food and Drug Administration (FDA) regulations establish limits for contaminants in bottled water, which must provide the same protection for public health."

Effective September 30, 2019, Env-Dw 811.22(b) intro and Table 811-1 as to per- and polyfluoroalkyl substances contaminants are cited and read as follows:

Env-Dw 811.22 Contaminant Source Information.

(b) If the O/O lacks specific information on the likely source of the detected contaminant(s), the owner shall use the contaminant source information specified below in Table 811-1, as applicable:

Table 811-1: Contaminant Origin

Contaminant	Common Source in Drinking Water
Per- and Polyfluoroalkyl Substances (PFAS) Contaminants	
Perfluorohexane sulfonic acid (PFHxS)	Discharge from industrial processes, wastewater treatment, residuals from firefighting foam, runoff/leachate from landfills and septic systems
Perfluorononanoic acid (PFNA)	Discharge from industrial processes, wastewater treatment, residuals from firefighting foam, runoff/leachate from landfills and septic systems
Perfluorooctane sulfonic acid (PFOS)	Discharge from industrial processes, wastewater treatment, residuals from firefighting foam, runoff/leachate from landfills and septic systems
Perfluorooctanoic acid (PFOA)	Discharge from industrial processes, wastewater treatment, residuals from firefighting foam, runoff/leachate from landfills and septic systems

Effective September 30, 2019, Env-Dw 811.25(a) intro and Table 811-2 as to per- and polyfluoroalkyl substances contaminants are cited and read as follows:

Env-Dw 811.25 Converting MCL Water Quality Compliance Values.

(a) The MCL, MRDL, MCLG, and MRDLG for a contaminant shall be expressed in identical units as a number equal to or greater than 1.0, as specified in table 811-2, below, subject to the notes in (b), below:

Table 811-2: Converting MCL Water Quality Compliance Values

Contaminant	Traditional MCL in compliance units (mg/L)	To convert to a whole number, Multiply by ...	MCL in CCR units	MCLG in Whole Numbers
Per- and Polyfluoroalkyl Substances (PFAS) Contaminants				
Perfluorohexane sulfonic acid (PFHxS)	0.000018	1,000,000	18 ppt	0
Perfluorononanoic acid (PFNA)	0.000011	1,000,000	11 ppt	0
Perfluorooctane sulfonic acid (PFOS)	0.000015	1,000,000	15 ppt	0
Perfluorooctanoic acid (PFOA)	0.000012	1,000,000	12 ppt	0

APPENDIX A - STATUTES/REGULATIONS IMPLEMENTED

Rule Section(s)	State Statute(s) Implemented	Federal Regulation(s) Implemented
Env-Dw 701.03(d)-(f)	RSA 485:3, I; RSA 485:16-e	
Env-Dw 705.06	RSA 485:3, I; RSA 485:16-e	
Env-Dw 707.06(d)-(e)	RSA 485:3, I; RSA 485:16-e	
Env-Dw 708.01(e)	RSA 485:3, I; RSA 485:16-e	
Env-Dw 712.23 - 712.30	RSA 485:3, I; RSA 485:16-e	
Env-Dw 808.01; Env-Dw 808.27-808.30	RSA 485:43; RSA 485:16-e	
Env-Dw 811.02(d); Env-Dw 811.07(c); Env-Dw 811.22(b), Table 811-1; Env-Dw 811.25(a), Table 811-2	RSA 485:43; RSA 485:16-e	

APPENDIX B - FEDERAL DEFINITIONS

[No new definitions]

APPENDIX C: DEFINITION OF PESTICIDE

[Not applicable to this rulemaking]

APPENDIX D: MONITORING FREQUENCY FOR PFAS CONTAMINANTS BASED ON SPECIFIED MCL

Perfluorohexane sulfonic acid (PFHxS); MCL = 18 ng/L

Average Monitoring Result (ng/L)	Frequency
> 9 to 18	Annually
≤ 9	Every 3 years

Perfluorononanoic acid (PFNA); MCL = 11 ng/L

Average Monitoring Result (ng/L)	Frequency
> 5.5 to 11	Annually
≤ 5.5	Every 3 years

Perfluorooctane sulfonic acid (PFOS); MCL = 15 ng/L

Average Monitoring Result (ng/L)	Frequency
> 7.5 to 15	Annually
≤ 7.5	Every 3 years

Perfluorooctanoic acid (PFOA); MCL = 12 ng/L

Average Monitoring Result (ng/L)	Frequency
> 6 to 12	Annually
≤ 6	Every 3 years

Effective September 30, 2019, Env-Wq 402.05 reads as follows [only (c) revised]:

Env-Wq 402.05 Exemptions to Groundwater Quality Criteria. Groundwater shall be exempt from the groundwater quality criteria of Env-Wq 402.04(a) and (b) if:

(a) The groundwater is within a groundwater discharge zone that has been permitted in accordance with Env-Wq 402.23;

(b) The groundwater is within a groundwater management zone that has been permitted in accordance with Env-Or 607; or

(c) The only source of the groundwater contamination is:

(1) Salt and other de-icing chemicals applied for winter road maintenance, provided an active source of drinking water is not made unsuitable for use as drinking water without treatment; or

(2) Residual 1,4-dioxane, perfluorohexane sulfonic acid (PFHxS), perfluorononanoic acid (PFNA), perfluorooctane sulfonic acid (PFOS), or perfluorooctanoic acid (PFOA), or any combination thereof, from any facility that discharges treated wastewater to groundwater, provided:

a. The requirements of Env-Wq 402.251 are met; and

b. An active source of drinking water is not made unsuitable for use as drinking water without treatment.

Effective September 30, 2019, Env-Wq 402.24 reads as follows:

Env-Wq 402.24 Groundwater Discharge Permit Compliance Criteria.

(a) Domestic wastewater shall receive primary treatment by settling of solids in subsurface disposal systems and at least secondary treatment as defined in 40 CFR 133 for other disposal methods, before discharge to the ground or to groundwater.

(b) Municipal wastewater, alone or in combination with domestic wastewater, shall receive treatment in compliance with RSA 485-A:13, I(a) before being discharged to the ground or to groundwater.

(c) Non-domestic wastewater, alone or in combination with domestic wastewater, shall be treated by BAT before being discharged to the ground or to groundwater.

(d) Except as provided in Env-Wq 402.251 for 1,4-dioxane, perfluorooctanoic acid, perfluorooctane sulfonic acid, perfluorononanoic acid, and perfluorohexane sulfonic acid, no discharge shall cause the groundwater quality criteria set forth in Env-Wq 402.04 to be violated at any point beyond the boundary of a groundwater discharge zone.

(e) No discharge shall cause or contribute to a violation of surface water quality standards set forth in RSA 485-A or Env-Wq 1700.

(f) Subject to Env-Wq 402.251, the concentration in treated wastewater to be discharged to groundwater of the contaminants listed in Table 402-2, below, shall not exceed the specified concentration:

Table 402-2: Maximum Concentration of Certain Contaminants in Treated Wastewater Discharged to Groundwater

Contaminant	Maximum Concentration
1,4-dioxane	2 µg/L
Perfluorohexane sulfonic acid (PFHxS)	Twice the AGQS established in Env-Or 603.03
Perfluorononanoic acid (PFNA)	Twice the AGQS established in Env-Or 603.03
Perfluorooctane sulfonic acid (PFOS)	Twice the AGQS established in Env-Or 603.03
Perfluorooctanoic acid (PFOA)	Twice the AGQS established in Env-Or 603.03

Effective September 30, 2019, Env-Wq 402.25(a) reads as follows [changes to (a)(4) & (a)(5) only]:

Env-Wq 402.25 Response to Exceedances.

(a) If any regulated contaminant is detected by the permittee's monitoring at a concentration that exceeds the applicable AGQS, the permittee shall:

(1) Within 10 days of receiving the test results that show the exceedance, notify the department of the exceedance;

(2) Within 21 days of receiving the test results that show the exceedance, test water for the regulated contaminant that exceeds the AGQS from each private or public drinking water supply well within 1,000 feet of the location where the exceedance occurred;

(3) Report the results of the testing required by (2), above, to the department within 45 days of collecting the samples;

(4) For exceedances of contaminants other than 1,4-dioxane, perfluorooctanoic acid, perfluorooctane sulfonic acid, perfluorononanoic acid, or perfluorohexane sulfonic acid, or any combination thereof, from a facility that discharges treated wastewater to groundwater, prepare, submit, and implement a written response plan in accordance with (b) through (g), below, to ensure that groundwater quality criteria are not violated at the boundary of the groundwater discharge zone; and

(5) For exceedances of 1,4-dioxane, perfluorooctanoic acid, perfluorooctane sulfonic acid, perfluorononanoic acid, or perfluorohexane sulfonic acid, or any combination thereof, from a facility that discharges treated wastewater to groundwater, proceed as specified in Env-Wq 402.251.

Effective September 30, 2019, Env-Wq 402.251 reads as follows:

Env-Wq 402.251 Treatment for Excess 1,4-Dioxane and Certain Per- and Polyfluoroalkyl Substances in Wastewater Discharged to Groundwater.

(a) If the level of any of the contaminants identified in Env-Dw 402.24(f), Table 402-2, in treated wastewater to be discharged to groundwater exceeds the maximum concentration established in Table 402-2 or if the level of any of the contaminants identified in Env-Dw 402.24(f), Table 402-2, in the groundwater at the perimeter of or outside the groundwater discharge zone exceeds the applicable ambient groundwater quality standard (AGQS) established in Env-Or 603, the facility discharging the wastewater shall:

(1) If the testing done pursuant to Env-Wq 402.25(a)(2) does not show the presence of any of the contaminants identified in Env-Dw 402.24(f), Table 402-2, in a private or public drinking water supply well at a concentration that exceeds the applicable AGQS, either:

a. Treat the wastewater effluent using best available technology (BAT); or

b. Implement an investigation and corrective action program (I&CA program) as described in (c) or (d), below, as applicable, to identify, assess, and address the potential source(s) of the contaminant(s); or

(2) If the testing done pursuant to Env-Wq 402.25(a)(2) shows the presence of any of the contaminants identified in Env-Dw 402.24(f), Table 402-2 in a private or public drinking water supply well at a concentration that exceeds the applicable AGQS and the department determines that it is more likely than not that the permitted wastewater discharge is the source of the contaminant(s), implement the response described in (1)a. or b., above, and (e), below.

(b) Within 90 days of initiating the implementation of the response, the facility shall submit to the department a report of the response implemented that describes all investigative actions taken, the nature and date of each corrective action taken, and the results as demonstrated by sampling of the treated wastewater.

(c) If the permittee is a public wastewater collection and treatment system, the I&CA program required by (a)(2), above, shall include the following:

(1) Assessment of each facility that discharges non-domestic wastewater to the wastewater system;

(2) Sampling within the wastewater system or at facilities connected to the wastewater system to evaluate potential sources of the contaminant(s); and

(3) Modification of operations at facilities discharging non-domestic wastewater as needed to reduce or eliminate sources that cause or contribute to elevated concentrations of the contaminant(s).

(d) If the permittee is not a public wastewater collection and treatment system, the I&CA program required by (a)(2), above, shall include the following:

(1) A review of the materials used in the facility to identify potential sources of the contaminant(s);

(2) Sampling of the materials used in the facility to evaluate potential sources of the contaminant(s); and

(3) Modification of facility operations, such as installing treatment systems for wastewater or replacing the materials that are causing or contributing to elevated concentrations of the contaminant(s) to the extent practicable.

(e) If required by (a)(2), above, the permittee shall:

(1) Expand the testing of public and private drinking water wells beyond 1,000 feet as necessary to determine the extent of the exceedance of the applicable AGQS in drinking water supplies; and

(2) Within 21 days of receiving the test results obtained pursuant to (1), above, submit a proposed response plan to the department that evaluates the relative costs and benefits of:

a. Installing treatment to remove the contaminant(s) from the water supplied from the well; or

b. Providing alternate water to those served by the drinking water supply by:

1. Supplying bottled water as an interim mitigation measure until a long-term water supply alternative is provided; and

2. Providing a long-term alternative water supply by:

(i) Installing, testing, and maintaining a point-of-entry water treatment system at each structure served; or

(ii) Connecting each structure served to a public water system.

(f) The response plan submitted pursuant to (e)(2), above, shall include:

(1) A recommendation for providing alternate water; and

(2) A schedule for implementing the response plan.

(g) The department shall:

(1) Approve the plan, including the schedule, if it determines that the plan is adequate to protect public health; and

(2) Notify the permittee of its determination in writing, provided that if the plan is not approved the department shall identify the reason(s) why.

(h) The permittee shall implement the response plan in accordance with the schedule approved by the department.

APPENDIX A: STATE STATUTES & FEDERAL REGULATIONS IMPLEMENTED

Rule Section(s)	State Statute(s) Implemented	Federal Regulations Implemented
Env-Wq 402.05 intro & (c)	RSA 485-C:6	40 CFR 144, 145, & 146
Env-Wq 402.24; 402.25(a) intro, (4) & (5); 402.251	RSA 485-A:13, I(a)	40 CFR 144, 145, & 146

Effective September 30, 2019, Env-Or 603.03(b) reads as follows:

(b) The following shall apply to Table 600-1, below:

(1) The standard for total trihalomethanes, namely bromoform, bromodichloromethane, dibromochloromethane and trichloromethane (chloroform), shall be 80 micrograms per liter (µg/L) if the groundwater is contaminated by chlorinated water supplies;

(2) Positives for total coliform shall be confirmed by the presence of other wastewater parameters, such as fecal coliform, Escherichia coli, fecal streptococcus, nitrates, and chlorides;

(3) Unless otherwise noted, concentrations shall be measured in micrograms per liter (µg/L), which is equivalent to parts per billion (ppb); and

(4) Gross alpha radionuclides, radium 226 and 228, strontium 90, and tritium shall be measured in picocuries per liter (pCi/L).

Effective September 30, 2019, Env-Or 603.03(c) intro & Table 600-1 relative to perfluorohexane sulfonic acid (PFHxS), perfluorononanoic acid (PFNA), perfluorooctane sulfonic acid (PFOS), and perfluorooctanoic acid (PFOA) read as follows:

(c) AGQS shall be as set forth in Table 600-1 below:

Table 600-1
AMBIENT GROUNDWATER QUALITY STANDARDS

Chemical Name	CAS No.	AGQS µg/L (ppb)
Perfluorohexane sulfonic acid (PFHxS), total of all isomers	335-46-4	0.018
Perfluorononanoic acid (PFNA), total of all isomers	375-95-1	0.011
Perfluorooctane sulfonic acid (PFOS), total of all isomers	1763-23-1	0.015
Perfluorooctanoic acid (PFOA), total of all isomers	335-67-1	0.012

APPENDIX A: STATE STATUTES IMPLEMENTED

Rule	State Statutes Implemented
Env-Or 603.03(b) and (c) intro & Table 600-1	RSA 485:16-e; RSA 485-C:4, III; RSA 485-C:6, V & VI

STATE OF NEW HAMPSHIRE

MERRIMACK, SS. SUPERIOR COURT

CASE NO. 217-2019-CV-00650

The Plymouth Village Water & Sewer District, et al

Plaintiffs,

v.

Robert R. Scott, as Commissioner of the Department of Environmental Services

Defendant.

OBJECTION TO MOTION FOR PRELIMINARY INJUNCTION

NOW COMES Robert R. Scott, as Commissioner of the State of New Hampshire, Dept. of Environmental Services ("NHDES") and objects to the plaintiffs' motion for a preliminary injunction. In support of this objection, NHDES avers as follows:

I. PRELIMINARY INJUNCTION STANDARD

The plaintiffs face a heavy burden in their attempt to obtain a preliminary injunction.

> The issuance of injunctions, either temporary or permanent, has long been considered an extraordinary remedy…. An injunction should not issue unless there is an immediate danger of irreparable harm to the party seeking injunctive relief, and there is no adequate remedy at law. Also, a party seeking an injunction must show that it would likely succeed on the merits.

N.H. Dep't of Envtl. Servs. v. Mottolo, 155 N.H. 57, 63 (2007) (internal citations omitted). Therefore, a preliminary injunction should be issued sparingly, only when (1) the movant shows that it will likely succeed on the merits and when there is an (2) immediate danger of (3) irreparable harm.

The N.H. Supreme Court has not adopted the presumption of irreparable harm for violations of constitutional rights espoused by the plaintiffs. This proposition is found in one

Merrimack County Superior Court decision from 2013 – *Deere and Co. v. New Hampshire*, 2013 WL 9889004 (2013). In any event, the Merrimack County Superior Court in *Deere*, and the cases cited by the superior court, clearly establish merely a presumption, not an automatic finding. The superior court quoted *Donohue v. Mangano*, 886 F. Supp. 2d 126 (E.D.N.Y. 2012) in which the district court stated that "the assertion of a constitutional injury is insufficient to *automatically* trigger a finding of irreparable harm" and allowed the injunction to issue in that case because "the constitutional deprivation [was] convincingly shown" further finding that the "violation carrie[d] noncompensable damages." *Mangano 886 F. Supp. 2d at 150* (emphasis added). The court in *Mangano* agreed that a preliminary injunction cannot issue if "Plaintiffs cannot assert a constitutional injury *at this time*," citing to other cases for the proposition that "[b]are allegations, without more, are insufficient for the issuance of a preliminary injunction." *Id*. at 150-151 (internal citations omitted) (emphasis added). In *Univ. of Hawaii Prof'l Assembly v. Cayetano*, 16 F. Supp. 2d 1242, 1247 (D. Haw. 1998), although the court recognized that the Ninth Circuit found that "[a]n alleged constitutional infringement will often alone constitute irreparable harm," it went on to determine whether the premise applied, stating that:

> the Court finds that irreparable harm is possible because, as discussed above, many of the 3157 members of UHPA may experience harm from a pay lag including incurring late fees for bills and credit cards and delays in mortgage payments. In some cases, a delay of even five days could effect a person's credit report. It is highly unlikely that any damages remedy would adequately compensate the injury of each and every member of UHPA

Univ. of Hawaii Prof'l Assembly v. Cayetano, 16 F. Supp. 2d 1242, 1247 (D. Haw. 1998), *aff'd sub nom. Univ. of Hawai'i Prof'l Assembly v. Cayetano*, 183 F.3d 1096 (9th Cir. 1999), and *order dissolved*, 125 F. Supp. 2d 1237 (D. Haw. 2000).

Other cases cited by the superior court made similar findings. *Goings v. Court Servs. & Offender Supervision Agency for D.C.*, 786 F. Supp. 2d 48, 78 (D.D.C. 2011) ("Given that the

conditions imposed on the plaintiff limit his ability to see and interact with his family, his freedom of movement, and association, the Court finds that the plaintiff has demonstrated irreparable harm"); *Kendall-Jackson Winery, Ltd. v. Branson*, 82 F. Supp. 2d 844, 878 (N.D. Ill. 2000) ("plaintiffs are being compelled to do business with distributors whom they wish to terminate and, because of provisions of the Act barring any state judicial interference with the proceedings before the Liquor Control Commission, cannot, in the foreseeable future, seek a remedy from the Illinois courts"). For its part, the U.S. Supreme Court limited its application to issues involving First Amendment freedoms. *Elrod v. Burns*, 427 U.S. 347, 373–74 (1976). In general, all of the movants in these cases had to show an actual injury, not simply a claim of general or hypothetical constitutional infirmity. It is worth noting that *Awad v. Ziriax*, 670 F.3d 1111 (10th Cir. 2012), cited by the superior court, applied the presumption to the "irreparable" prong of the test, but not to whether the plaintiff "face[d] a concrete and imminent injury." *Id.* at 1131.

In contrast, longstanding New Hampshire law indicates that invalidating an action of the legislature or executive on a preliminary basis cannot be taken lightly.

> It has always been the practice in this jurisdiction to follow the universally accepted doctrine that the constitutionality of an act passed by the coordinate branch of the government is to be presumed. It will not be declared to be invalid except upon unescapable grounds; and the operation under it of another department of the state government will not be interfered with until the matter has received full and deliberate consideration.
>
> …
>
> Unless irreparable loss will be caused, no restraining order should issue until the subject has been passed upon by the court of last resort.

Musgrove v. Parker, 84 N.H. 550 (1931).

The Court should note that the rules at issue in this case only require quarterly testing of each water source for a period of one year (four samples for each water source from October 1,

2019-September 30, 2020). Env-Dw 712.23. The initial quarterly test need not be completed until December 31, 2019. To the extent that a plaintiff tests above standards, as determined by calculating the average of the four quarterly samples collected over the first year, further actions may be required such as the installation of treatment. *Id.* DES estimates that testing costs up to $350 per test. Exhibit A. By way of example, the Plymouth Water District must conduct four tests on each of its two wells over the next year, which NHDES estimates may total up to $2800. Only two tests are required by December 31, 2019, totaling approximately $700. The frequency of testing after the first year is based on the results from the first year as specified in the rules, but could decrease to as little as testing each source once every three years. Also, by rule, if a system has non-detect in their initial two samples, the system may immediately change its sampling regime to sample only once every three years which, in this case, would limit costs to up to $1400 over those three years.

II. THE PLAINTIFFS HAVE NOT DEMONSTRATED A HIGH LIKELIHOOD OF SUCCESS ON THE MERITS

A. The Complaint and Motion for Preliminary Injunction Allege No Facts Upon Which the Court Could Establish Harm

The complaint includes allegations from four very differently situated plaintiffs consisting of:

1. The Plymouth Village Water and Sewer District ("Plymouth Water District")

The Plymouth Water District runs a public water system and stands alone among the plaintiffs as the only entity purporting to be a subdivision of a municipality.

2. The 3M Company ("3M")

The 3M property located at 11 Paper Trail in Tilton includes a public water system; specifically a non-transient non-community water system (i.e., a water system for employees).

Exhibit A. As a public water system, it is subject to quarterly testing requirements; however, the site at 11 Paper Trail is also subject to numerous other requirements due to its Groundwater Discharge Permit ("GDP"). Exhibit A. As a result, 3M has already tested for all of the compounds in this case. Exhibit A. The results came back non-detect for all compounds except PFOA which showed concentrations of 2 parts per trillion ("ppt") – well below the current limits promulgated by NHDES. Exhibit A.

3. Resource Management Inc., ("RMI")

RMI is a residuals processing facility. It does not have a public water system and, therefore, is not subject to the testing requirements of a public water system. Exhibit A. Instead, because the RMI facility processes wastewater "residuals" (meaning treated sludge and septage) for land application for beneficial agricultural use, it operates under a Sludge Facility Permit (#SL96002). RMI also holds a Sludge Site Permit (#SL96010S) for the on-site land application of residuals. Exhibit B. The required groundwater monitoring for the Sludge Facility is included in the Sludge Facility Permit Conditions. Exhibit B. Results obtained as part of this monitoring have shown that RMI already exceeded the old NHDES Ambient Groundwater Quality Standards (AGQS) for PFOA/PFOS and nitrate in its groundwater. Exhibit B. In accordance with Env-Wq 808.03, RMI created and submitted a corrective action plan (CAP) to address the AGQS exceedances of PFOA/PFOS and nitrate in groundwater at the sludge facility. The last revised version of the CAP was received by NHDES RMS on February 2, 2019. RMI has also submitted a waiver request received by NHDES RMS on February 1, 2019, in accordance with Env-Wq 811, requesting relief from Env-Wq 808.03(f)(1) which requires the permit holder to "cease operation immediately" if the concentration of any monitored constituent detected in any down-gradient monitoring well exceeds the AGQS. Exhibit B. Review of these documents by NHDES is pending. Also under pre-existing rules (Env-Wq 808.03(a)), whenever a contaminant

is detected above "background value," the permit holder must "[c]ommence monthly monitoring for each constituent for which background has been exceeded at each well where background has been exceeded." Env 808.03(a)(2). "Background" is defined as "the analytical detection limit for that constituent" – a stricter standard than the MCL at issue in this case. Env 808.03(b)(1). The RMI sludge facility has experienced groundwater concentrations for several metals, TKN, and nitrate, that exceed "background" levels, and continues to perform monthly monitoring and reporting for these constituents. Exhibit B.

To the extent treatment of impacted groundwater must occur at this facility/site, the two viable options for treatment most likely consist of either a groundwater treatment system or monitored natural attenuation ("MNA"). Exhibit C. Treatment systems are designed to treat to non-detect, therefore, the capital cost of such a system is the same whether one uses the old or new standard. Exhibit C. MNA would consist of monitoring contaminant concentrations in on-site groundwater samples without active treatment and is essentially the same under the current or old PFAS AGQS. Exhibit C. In short, RMI already has an obligation to monitor greater than what would be required under the new rules. It has not articulated what immediate and irreparable impact the new standards might have on its facility.

With respect to RMI's business operation generally, sludge taken from a facility for land application for beneficial agricultural use, must first be tested and obtain/maintain a Sludge Quality Certification (SQC) from NHDES (Env-Wq 804.04 and Env-Wq 809). This testing must be done for the initial certification and on an annual basis (Env-Wq 809). RMI already tests for 9 PFAS compounds, including all four at issue in this case. Exhibit B. Currently, NHDES does not have MCL for PFAS in sludge, therefore, a positive PFAS result in sludge will not affect sludge facility operations at this time unless it exceeds a contact limit with is and will likely

remain an order of magnitude higher than the current AGQS/MCL. NHDES does not require further testing of biosolids that RMI takes directly from a source (such as Plymouth) to an application site.

4. Charles G. Hanson

Mr. Hanson owns and operates Hilltop Farm located at 121 Dane Road, Center Harbor, New Hampshire. Mr. Hanson is also a Director and Secretary of RMI. Mr. Hanson stated in the complaint that "Plaintiff Hanson will be subject to new rules, and may be required to test for, and if necessary, remediate, PFOA, PFOS, PFNA, and PFHxS, if the rules are not enjoined." Complaint, pg. 3, para. 5. However, NHDES does not require groundwater monitoring for sludge site permits. RMI obtained a "Sludge Site Permit Renewal" (#SLS-01-004) for the land application of treated sludge at agronomic rates on "The Hanson Hilltop Farm" fields. Exhibit B. As the permit holder, RMI, would be subject to the terms of the sludge site permit. As stated above, RMI would only have to test biosolids at its sludge facility that it planned to land apply at permitted sites, in accordance with the facility's SQC requirements.

Despite fundamentally different characteristics, the complaint never attributes factual allegations to any particular plaintiff. Allegations either relate to the public generally or to the plaintiffs as a group. For instance, Count I discusses Part I, Art. 28-A, "unfunded mandates," and a concomitant section in the State Administrative Procedures Act, RSA 541-A ("APA"). If the arguments in this count have any validity, it is only with respect to the Plymouth Water District. Nevertheless, Count I either talks about municipalities generally or the "Plaintiffs" collectively. Complaint, pg. 19, para. 79 ("Few if any municipalities have approved the

increased expenditures….")[1]; *id*. at pg. 19, para. 80 ("…requiring municipalities to incur enormous local expenditures…"); *id*. at pg. 20, para. 83 ("…responsibility to cities and towns by requiring them to expend funds…"); *id*. at pg. 20, para. 87 (after discussing only requirements related to municipalities, the complaint states: "*Plaintiffs* request that judgment be entered declaring the Final Rules invalid ….") (emphasis added); *id*. at pg. 20, para. ("pg. 21, para. 88 ("*Plaintiffs* further request the Court temporarily, preliminarily and permanently enjoin the NHDES from enforcing the Final Rules") (emphasis added). Nothing indicates whether the Plymouth Water District, or the Town of Plymouth (which has chosen not to be a party) has budgeted for this expenditure or whether the Plymouth Water District or the Town of Plymouth is actually likely to experience "enormous local expenditures." Information available to NHDES indicates that it may not as it already performed testing on two of the four regulated compounds and the results came back at non-detect with a reporting limit (at the time) of 20-40 ppt and a detection limit of 1-8 ppt. Exhibit A.

The closest that any allegation comes to linking the Plymouth Water District to an actual fact is on page 6 of the *Memorandum of Law* which states:

> The burden thrust upon political subdivisions of the State such as Plaintiff Plymouth Village Water & Sewer District to ensure filter water resources [sic] to near-zero levels of PFOA, PFOS, PFNA, and PFHxS is a new and/or expanded undertaking, and necessitates a material change to the duties, responsibilities, and costs incurred by political subdivisions.

Nothing indicates that the current standards will require the Plymouth Water District to "filter…to near-zero levels" or that it would "necessitate[] a material change to the duties, responsibilities, and costs" incurred by the Plymouth Water District[2] – an entity whose job it is to

[1] During discovery, the State intends to inquire about the status of funding for testing by the Plymouth Water District.

[2] The official government record, consisting of a video, of a meeting of the Plymouth Water District in which this case was discussed shows no concern about immediate or irreparable harm

manage water resources. As discussed further below, RSA 485:4 already allows NHDES to require municipalities to remediate for anything it considers to be a health hazard. More information on the Plymouth Water District is provided below. In any event, three of the four plaintiffs have no standing to make this claim.

In sections dedicated to claims other than the one related to allegedly unfunded mandates, the plaintiffs memorandum lumps the plaintiffs together with all parties located in the State without any distinct claim of harm related to any specific plaintiff. *See* Memo.,[3] pg. 16 ("Plaintiffs and other interested and affected public [sic]."); *id*. at 17 ("The Final Rules threaten to … deprive Plaintiffs and all political subdivisions of the State…"); *id*. at 18 ("Plaintiffs, political subdivisions and businesses across the State will be deprived…"); *id*. ("Many political subdivisions lack the technology, personnel, infrastructure, training, and other resources to filter

to the Plymouth Water District, merely a desire by 3M's attorneys to include a town. At the meeting of September 24, 2019, at approximately 14 minutes and 41 seconds into the video, the Plymouth Water District states: "There is a coalition coming together to file an appeal of the PFAS regulations. I have talked to the lawyers, McLane Graf is the representative for the State of New Hampshire, they are targeting, they want to have it filed in time for an injunction, PFAS regs become effective October 1, so they [McLane] are scurrying, they [McLane] have the lawyer on standby for six oclock if there are questions, … but essentially we would be looking for a modest contribution overall … they [McLane] need us a lot at this point, they [McLane] need a municipal entity, there may be others that they're trying to bring on board but the reason they need it is because the constitution provides for – prohibits unfunded mandates, and municipalities are the only entities that are covered by that unfunded mandate provision, so that adds horses to the nature of the appeal, so my recommendation is that you authorize me to officially join the appeal and commit $1,000 dollars at the front end and with the stipulation that we will entertain but not guarantee further commitment…. " This video is available at the Plymouth Water District site at http://pvwsd.org/ by clicking on the "TV" icon to the right or directly at https://www.youtube.com/watch?v=4uka1UtjNqs&list=PLbPTWBdOlg4DQD80ONdBPCNmTQOM6xV3H&index=2&t=0s.

[3] "Memo." refers to the plaintiffs "Memorandum in Support of Temporary and Preliminary Injunction."

water to near-zero levels of these compounds.")[4]; *id*. ("Political subdivisions and other interested private parties that are involved in the supply and treatment of water resources across the State will incur all of these costs…."); *id*. at 24 (The rule will "impose an unlawful, costly burden on the political subdivisions and taxpayers of the State").

Similarly, the complaint makes no allegations specific to any plaintiff with respect to the public comment/rulemaking process. Instead, again, the complaint advocates for the public at-large. Complaint, pg. 5, para. 15 ("NHDES never offered the public an opportunity to comment…."); *id*. at pg. 5, para. 17 ("NHDES did not give the public an opportunity to comment…."); *id*. at pg. 17, para. 18 ("NHDES never gave the public an opportunity to comment….) *id*. at pg. 14, para. 56 ("Although many members of the public were present at the JLCAR hearing and some requested to speak in opposition to the rules, JLCAR refused to accept public comment."); *id*. ("JLCAR ignored specific requests by the public…."); *id*. at pg. 17, para. 69 ("For the first time, political subdivisions and municipalities will be required to test for PFOA, PFOS, PFNA, and PFHxS as part of any mandated groundwater sampling (e.g., water discharge, leachate discharge and groundwater management permit)"); *id*. at pg. 18, para. 69 ("All taxpayers that operate a public water system…"); *id*. at pg. 18, para. 70 ("Numerous other entities…face potentially huge costs….").

At other times, the complaint passively and ambiguously lumps all of the plaintiffs together in ways that make commonality among the plaintiffs more than dubious. For instance, pg. 14 of the complaint states that: "Had public comments…been allowed, numerous detailed

[4] The State believes it may well establish that the Plymouth Water District has the requisite capability and, in any event, is already subject to remediation for any contamination impacting public health pursuant to RSA 485:4. In addition, treatment systems used for PFAS provide clean water, i.e., they treat to "near-zero levels" anyway. Exhibit A. That is true whether the MCL is 70 ppt or 14 ppt. Therefore, this allegation, and those like it, appear to be mere puffing.

comments on the risks considered would have been provided." Complaint, pg. 14, para. 58. It then goes on to list four very technical, sophisticated, and specific comments; namely:

a. The risk analysis used to develop the MCLs and the AGQS is based on non-cancer endpoints.

b. The EPA does not classify PFOA, PFOS, PFNA, PFHxS as known human carcinogens.

c. "The available human studies have identified some potential targets of toxicity; however, cause and effect relationships have not been established for any of the effects, and the effects have not been consistently found in all studies." Toxicological Profile for Perfluoroalkyls, Draft for Public Comment, ASTDR 2018; p.p. 635-36.

d. There is no scientifically established risk of humans developing cancer at the low parts-per-trillion levels in the Proposed Rules, let alone the dramatically lower parts per trillion limits of the Final Rules.

A similar allegation is repeated in the next paragraph which states:

> Had public comment … been allowed, numerous detailed comments on the costs considered would have been provided. For example:
>
> a. NHDES failed to fully evaluate the costs and benefits to all affected parties that result from MCL and AGQS standards in the June 2019 Final Rules as required by RSA 485:3, 1(b).
>
> b. NHDES' June 28, 2019 Updated on Cost and Benefit Considerations report runs a mere four pages, plus attachments.
>
> c. EPA is developing MCLs for some of the same PFAS substances. Part of that process includes a detailed and rigorous consideration of costs and benefits. EPA's Guidelines for Preparing Economic Analyses, National Center for Environmental Economics Office of Policy U.S. Environmental Protection Agency, December 17, 2010 (updated May 2014), stretches to well over 300 pages and references methodologies for discounting future benefits and costs, analyzing benefits, analyzing costs, conduct of an economic impact analysis, and other factors, including an appendix devoted to Economic Theory.

Complaint, pg. 15, para. 59. The complaint does not allege which if any of the plaintiffs actually would have made such comments. Discovery is needed, but it appears likely that some of the

plaintiffs intended to do nothing of the sort and it is almost a certainty that not all four of the plaintiffs would have made these exact comments.

In fact, missing from the complaint is the fact that on April 12, 2019, 3M actually provided 50 pages of comments to NHDES along with multiple pages of attachments. As part of those comments, 3M noted: "On February 21, 2019, NHDES announced that there was 'new information that may change its proposed PFAS drinking water standards' and identified 'a new assessment tool developed by the Minnesota Department of Health'" described in an article by Goeden et al. Exhibit C.4 (pg. 6. of 3M comments). In response, 3M stated that it had "hired Dr. Anne Loccisano (Exponent), a nationally recognized expert on pharmacokinetic modeling, to review Goeden et al." and included both a summary of her findings and an attachment of her "review in its entirety." *Id*.

Many of 3M's comments mirror those found in the complaint that it now claims could not be made.[5] For instance, 3M asserted that it was "scientifically unjustified to use a single endpoint of slightly reduced litter size in a mouse reproductive and developmental study," i.e., a non-cancer endpoint. Exhibit C.4 (pg. 2. of 3M comments). 3M further stated, "there is an absence of data for PFAS that would support: (1) carcinogenicity in humans." Exhibit C.4 (pg. 6. of 3M comments). In language tracking the complaint almost verbatim, 3M told NHDES:

> In its 2018 Draft Toxicological Profile for Perfluoroalkyls, ATSDR recently acknowledged that for PFAS there is no cause and effect established between health effects and exposure to humans, when it stated: "The available human studies have identified some potential targets of toxicity; however, ***cause and effect relationships have not been established for any of the effects, and the effects have not been consistently found in all studies***." ATSDR 2018; pages 635-636.

[5] The similarity of 3M's comments to NHDES to those found in the complaint indicates that they likely originated solely from 3M and not any of the other plaintiffs. Discovery on this issue is needed.

Exhibit C.4 (pg. 5. of 3M comments) (emphasis original).

At the time of its comments, the NHDES methodology for determining costs and benefits was known even if the final numerical standards had not yet been released. 3M chose to say nothing about this methodology. It is also worth noting that the president of RMI attended and provided comments at the second of three NHDES public hearings. Exhibit C.

In contrast to the plaintiffs aspirational pleadings, the law requires each plaintiff to recite the injury to *it*. Indeed, a plaintiff must allege more than an infirmity with a rulemaking process, it must show that it "suffered harm." *Nevins v. New Hampshire Dep't of Res. & Econ. Dev.*, 147 N.H. 484, 488 (2002). In this case, with respect to the rulemaking process, the complaint lacks even one concrete factual recital indicating that the process precluded any particular plaintiff from submitting comments and certainly fails to allege that the NHDES decision on its final rule would have been different if it had allowed additional public input. *Id*. (wherein the plaintiffs claim failed partly because it did not "assert that if DRED had followed proper rulemaking procedures, thereby allowing for public input into the rulemaking process, DRED would not have entered into the lease with U.S. Cellular").

With respect to actual injury, other than the Plymouth Water District, no plaintiff articulates how, given their current status and other existing obligations, they could possibly be harmed. Specifically, as stated above, RMI has to test its site for other reasons, 3M already tested and came up under 2 ppt, and Mr. Hanson need not test at all. Certainly, nothing rises to the level of being immediate and irreparable. With respect to the Plymouth Water District, NHDES estimates that it will need to take quarterly samples for each of its two sources of water by September 30, 2020, for a total of eight samples. The frequency of future sampling will be determined by the results of sampling for the first year and could be reduced to sampling each

source of water only once every three years if the compounds are not detected in the water samples. NHDES estimates that a reasonable high estimate for each test is $350. Exhibit A. The Plymouth Water District already tested for PFOA, PFNA and PFHxS and PFOS in September 2016 and came back below both the laboratory detection and reporting limits (reporting limits at that time equaling about 20-40 pp and detection limits of 1-8 ppt). Exhibit A. The detection limits associated with the results of Plymouth Water District's testing results are below the MCLs and all of the results associated with the compounds MCLs have been established for were "ND" meaning "not detected." Plymouth reported to NHDES that the PFAS was not detected in its water. Exhibit A. If PFAS were detected above the current limits, the Plymouth Water District would simply need to conduct four more quarterly tests to see if levels persisted. Only after more than a year of high readings might a corrective action plan be requested. RSA 485:4 already gives NHDES the authority to require such measures. Hardly the basis for the issuance of an "extraordinary remedy."[6]

In total, other than a possible legal argument regarding the Plymouth Water District, not only does the complaint as written provide no basis for a preliminary injunction, it provides almost no grounds upon which relief could be granted at all. It strays even farther from providing a factual basis that could support a finding of immediate and irreparable harm. Most striking, neither the complaint nor the motion and associated memorandum include affidavits supporting any factual allegation related to injury, irreparability, or immediacy. SUPER. CT. RULE 11 states: "The court will not hear any motion grounded upon facts, unless such facts are verified by affidavit…." Had such factual allegations been made, and had such affidavits been

[6] Alleging that the Plymouth Water District could be penalized is non-sequitur, RSA 31:3-a frees it from any such threat.

provided, the State would have investigated the allegations and affirmations, contradicted those it found inaccurate or groundless, and provided a thorough response. As it stands, there is nothing to which the State can respond. Therefore, the motion, taken together with the complaint, provides no basis for preliminary relief.

B. NHDES Followed the Process Set Forth in the APA

Even if the plaintiffs had alleged actual injuries, they cannot demonstrate a high likelihood of success on the underlying merits. The facts demonstrate the following chronology. *See* Exhibit C:[7]

- March 2016 – NHDES creates its first webpage dedicated to PFAS
- September 1, 2017- NHDES establishes a PFAS "blog" meaning, essentially, a website with sequential updates
- October 16, 17, 18, 2018 - NHDES holds stakeholder Public Meetings
- December 31, 2018 - NHDES files a Request for Fiscal Impact Statement to the Legislative Budget Assistant ("LBA")
- January 1, 2019 – NHDES receives a fiscal impact statement from LBA
- January 24, 2019 – NHDES publishes a Rulemaking Notice
- February 21, 2019 – NHDES blog updated with post entitled "New Information May Change NHDES Proposed PFAS Drinking Water Standards"
- March 4, 2019 – NHDES conducts its First Public hearing
- March 5, 2019 – NHDES conducts its Second Public hearing
- March 12, 2019 –NHDES conducts a Third Public hearing
- April 12, 2019 – Written comments due
- June 28, 2019 – NHDES publishes its Final Proposed Rule (includes final standards)
- July 18, 2019 – JLCAR holds public meeting and approves the Final Proposed Rule
- July 24, 2019 – Adoption letter sent to Office of Legislative Services ("OLS")
- July 25, 2019 – Rules adopted
- September 30, 2019 – Rules effective

[7] The parties are working to submit a stipulated chronology to the Court. Nevertheless, NHDES has confirmed the accuracy of this outline. Exhibit C.

This chronology demonstrates that NHDES published a rulemaking notice on January 24, 2019. On February 21, 2019, NHDES updated its PFAS blog with a post entitled: "New Information May Change NHDES Proposed PFAS Drinking Water Standards." Exhibit C.1. In that post, NHDES references "a new assessment tool developed by the Minnesota Department of Health." It goes on to state:

> NHDES's assessment of the exposure model for the interaction of drinking water levels of PFAS and breastfeeding (Goeden et al, 2019) indicates that health-based drinking water or groundwater standards for PFOA and PFOS *would potentially be lowered significantly below the initial proposal figures of 38 parts per trillion (ppt) and 70 ppt, respectively*.

Exhibit C.1 (emphasis added). The February 21, 2019 blog post (which the plaintiffs call "Second Press Release") includes a link to the referenced, peer-reviewed work of Goeden, et al.: https://www.nature.com/articles/s41370-018-0110-5. Among other things, Goeden et al., analyzed and referenced the Minnesota assessment tool. All of this information was published 11 days before the first public hearing, 12 days before the second public hearing, 19 days before the third public hearing, and 49 days before comments were due. The APA does not require the publication of any of it.

After receiving and considering comments from 3M and others by the April 4, 2019 deadline, NHDES published a Final Proposed Rule on June 28, 2018 – 20 days before the JLCAR hearing on the rules and 5 days prior to its deadline to submit said rules (July 3, 2018). From these facts, the plaintiffs' claim violations of what they call "statutory due process" and constitutional due process, alleging that "*Plaintiffs* and other interested and affected public [sic] *had no notice* of the numeric MCLs and AGQS *such that they could have provided public comment*." Memo., pg. 16 (emphasis added).

1. The Plaintiffs Fail to Show a Violation of Constitutional Due Process

The plaintiffs claim, essentially, that either the timeline related to rulemaking was too short or that NHDES' final rule was significantly different from the original proposed rule and, therefore, the rulemaking process should have started over with new notice, new hearings, and the like. The plaintiffs' claims related to constitutional due process are misplaced. Rulemaking is not an adjudicative function; it is a quasi-legislative function.[8] In fact, the endpoint of rulemaking is distinctly legislative as the rules must pass through JLCAR and are eventually given to OLS for publication. The APA also give the General Court notice so that it may cure any alleged defect. RSA 541-A:13, VII.

When examining whether enactment of a zoning ordinance "without notice and hearing" violated due process, the Indiana Supreme Court found such acts "to be an exercise of the legislative power of State government, and as such [] exempt from the due process requirements of a trial-type hearing." *Krimendahl v. Common Council of City of Noblesville*, 256 Ind. 191, 197–98, 267 N.E.2d 547, 551 (1971). It went on to find that such acts "[are] not subject to the requirements of the state and federal due process provisions requiring a trial-type hearing with prior notice and the application of standards." *Id*.; *see also Rassi v. Trunkline Gas Co.*, 262 Ind. 1, 8, 240 N.E.2d 49, 53 (1968) (finding determination of public need in eminent domain proceeding to be a legislative determination such that judicial review "would violate the doctrine of separation of powers," and that such decisions are, in any event, not subject to procedural "due process").

[8] Part of the NHDES process could be described as "discretionary" rather than "legislative," but it is in no sense "adjudicatory."

The U.S. Supreme Court similarly held that legislative decisions do not trigger due process considerations. In *N. Laramie Land Co. v. Hoffman*, 268 U.S. 276 (1925), the plaintiffs in an eminent domain proceeding alleged "a denial of an opportunity to plaintiff in error to be heard." *Id*. at 278. The Court held that: "the necessity and expediency of the taking of property for public use 'are legislative questions, no matter who may be charged with their decision, and a hearing thereon is not essential to due process in the sense of the Fourteenth Amendment.'" *Id*. at 284; *see also Rindge Co. v. Los Angeles Cty.*, 262 U.S. 700, 709 (1923) ("The necessity for appropriating private property for public use is not a judicial question. This power resides in the Legislature, and may either be exercised by the Legislature or delegated by it to public officers").

Nevertheless, nothing indicates that the process in this case violated some fundamental notion of due process. NHDES promulgated its rule package in January. It described the likely change in the numerical limits and identified the studies upon which they were based 19 days before the last public hearing and 42 days before the deadline to submit comments. By way of comparison, the N.H. Court System also engages in rulemaking. SUP. CT. RULE 50. SUPREME COURT RULE 50 anticipates that the public will only be given 30 days notice prior to adoptance of a rule and does not require a public hearing at all unless the Supreme Court justices so require. The rule also anticipates that the final rule may differ from the proposed rule. No one believes that this timeframe violates due process.

The Legislature itself reviews many hundreds of pieces of legislation each year. The Legislature often schedules public hearings with far less advanced notice than the 47 days given by NHDES since the first notification or the 19 days given after informing the public of new studies. Amendments may occur on the House of Senate floor or in committee of conference with little to no chance for public comment. No party can raise procedural due process concerns

with these non-adjudicative functions. The same is true here. Instead, the only real question in this case is whether the process met the requirements of the APA.

2. The Plaintiffs' Allegations Fail to Support any Violation of What They Call "Statutory Due Process"

The plaintiffs allege that the rulemaking process allegedly violated their "statutory due process" rights. New Hampshire has not recognized any separate cause of action for alleged violations of "statutory due process." Federal courts that have used the phrase "statutory due process" appear to use it merely to refer to alleged violations of statutory procedures.

> Bankruptcy proceedings involve both statutory and constitutional due process rights. Statutory due process rights arise from the Bankruptcy Code and the Federal Rules of Bankruptcy Procedure. … violation of a "right granted by a procedural rule," without more, does not rise to the level of a constitutional violation.

Fed. Nat'l Mortg. Ass'n v. Meeko, No. 3:15-CV-01200-AA, 2016 WL 1108941, at *4 (D. Or. Mar. 17, 2016). Such allegations do not invoke constitutional due process rights. The sole issue is whether the agency conformed to the statute, in this case, the APA.

The APA requires an initial notice of rulemaking. A public hearing on the proposed rule must occur at least 20 days after this initial notice and the agency must give notice of the public comment period. RSA 541-A:6 ("The agency shall give at least 20 days' notice of its intent to hold a public hearing and shall also give notice of the cut-off date for the submission of written testimony"). NHDES satisfied all of these provisions, publishing its intent on January 24, 2019, and holding not one, but three public hearings on the proposed rules on March 4, 5, and 12.

Part of the information received during this process consisted of a study by Goeden et al. based partly on information from the Minnesota Department of Health. NHDES notified the public of this information on its "blog" on February 21, 2019. Exhibit C.1. NHDES also stated that the "standards for PFOA and PFOS would potentially be lowered significantly below the

initial proposal figures of 38 parts per trillion (ppt) and 70 ppt, respectively." Exhibit C.1. It was under no obligation to provide any of this information. *See* APA generally; *see also* RSA 541-A:3 (describing what must occur to initiate rulemaking and having no requirement to provide background information).

NHDES accepted comments on proposed rules until April 3, 2019. As stated above, 3M submitted over 50 pages of detailed comments including ones in its complaint. Exhibit C.4.[9] After NHDES had reviewed all of the comments and information available to it, it determined that the final rule should include lower standards. The APA does not preclude this. To the contrary, the APA anticipates that the final rule may be influenced by the information received during the public process and may differ from the proposed rule. In fact, the APA does not allow the agency to alter its proposed rule *before* the public hearing. RSA 541-A:10 states:

> I. At the same time the notice required by RSA 541-A:6, I is filed, the agency shall file the text of the proposed rule with the director of legislative services. The text of the proposed rules as filed by the agency pursuant to RSA 541-A:3, III *shall not be changed prior to the hearing* held pursuant to RSA 541-A:11, I(a).

RSA 541-A:10 (emphasis added). Further, the text of the rule cannot be finalized by the agency until after the public hearing and after the end of the comment period:

> II. *The agency shall not establish the text of the final proposal until after the conclusion of the public comment period* established pursuant to RSA 541-A:11, I(b). If the agency elects to solicit comment pursuant to RSA 541-A:11, I(c), the agency shall prepare a draft final proposal that is annotated to show how the rules as initially proposed are proposed to be changed. In response to comment received, the agency may revise the draft prior to filing the final proposal in accordance with RSA 541-A:12.

RSA 541-A:10 (emphasis added). The APA does allow the agency to hold a second public hearing with a revised proposal but this decision is purely discretionary.

[9] Exhibit C.3 also includes the NHDES response to many of the comments received by the public.

> (c) An agency *may* hold a public hearing or otherwise solicit public comment on a draft final proposed rule prior to filing the final proposed rule pursuant to RSA 541-A:3, V.

RSA 541-A:11; *see also Appeal of Rowan*, 142 N.H. 67, 71 (1997) (reiterating the "general rule that in statutes the word 'may' is permissive only, and the word 'shall' is mandatory"). An agency need only specify how the final rule changed from the proposed rule. RSA 541-A:12, II(d) states:

> The final proposal shall include . . . [a] copy of the fixed text of the final proposed rule annotated clearly to show how the final proposed rule differs from the rule as initially proposed, if the text has changed.

RSA 541-A:12. Therefore, NHDES adhered to the terms of the APA.[10]

The plaintiffs also find fault with the effective date of the rule. The effective date is not part of the rule and NHDES was under no obligation to announce what it predicted to be the effective date. The APA allows the agency to choose an effective date simply by sending a letter to OLS after adoption: "Adopted rules shall become effective under RSA 541-A:16, III on the day after filing by the agency, or at a later date, *provided that the agency so specifies in a letter to the director of legislative services*…." RSA 541-A:14, IV (emphasis added). The APA further allows the agency to change the effective date, also simply by filing a letter: "If the agency has specified a later effective date, the agency may modify the date by providing a statement to the director of legislative services which shall indicate the new effective date and all reasons for modifying the date." *Id*.

The plaintiffs also level claims against JLCAR. JLCAR is, of course, a legislative

[10] The plaintiffs also take issue with the fact that NHDES addressed Part I, Art. 28-a in its rulemaking notice but not RSA 541-A:25; however, the APA only requires an agency to provide "a statement that the proposed rule does not violate the New Hampshire constitution, part I, article 28-a." RSA 541-A:3, I. There is no requirement to explain its relationship to all other state laws.

committee. RSA 541-A:2. Claims of due process do not apply to JLCAR for the reasons discussed above. The APA also does not require JLCAR to hold a public hearing. RSA 541-A:2, III states: "The committee *may* hold public hearings on a proposed or previously adopted rule on its own initiative." RSA 541-A:2, III (emphasis added). If JLCAR decides to hold a public hearing, it need only give notice "7 days in advance." *Id*. For its part, NHDES simply had to ensure that it transmitted its final proposal to JLCAR "no later than 14 days before a regularly scheduled committee meeting…." RSA 541-A:12. NHDES transmitted its rule to JLCAR on June 28, 2019, several days before it was due. There is no provision requiring JLCAR to accept public comment. Therefore, JLCAR fulfilled the APA requirements in this case.

C. The NHDES Rulemaking Is Not An "Unfunded Mandate"

The plaintiffs make two arguments regarding what for purposes of this objection can be be called "unfunded mandates": (1) a constitutional argument under N.H. Constitution, Part I, Art. 28-a; and (2) a statutory argument under RSA 541:A-25. Although the "plaintiffs" request an injunction based on these arguments, only the Plymouth Water District has standing to raise them. The State will address each of these arguments in turn.

1. The NHDES Rulemaking Does Not Violate Part I, Art. 28-a

Article 28–a provides:

> The state shall not mandate or assign any new, expanded or modified programs or responsibilities to any political subdivision in such a way as to necessitate additional local expenditures by the political subdivision unless such programs or responsibilities are fully funded by the state or unless such programs or responsibilities are approved for funding by a vote of the local legislative body of the political subdivision.

N.H. CONST. pt. I, art. 28–a. The rule, required by SB 309, does not place any new responsibility on municipalities. Municipalities are not required to own or operate water

systems. By way of example, Exhibit A lists 45 municipalities that do not have a municipal water system, a village district, a school, or a standalone municipal building system that would have to test for PFAS. Exhibit A.

"Invoking the constitutional prohibition requires *both a mandate of responsibility* to the political subdivision and a requirement of additional local political subdivision expenditures by virtue of the mandate." *Opinion of the Justices (Materials in Solid Waste Stream)*, 135 N.H. 543, 545 (1992). Municipalities clearly have no responsibility to own or operate a water system.

Municipalities that own and operate water systems have historically done so in a manner consistent with laws and rules related to public health and safety. In a previous case involving town roads, the N.H. Supreme Court noted that "because [t]owns have historically been responsible for the local roads within their boundaries, [w]e … concluded that the reclassification was not an unconstitutional unfunded mandate." *City of Concord v. State,* 164 N.H. 130, 137 (2012), *as modified on reconsideration* (Sept. 28, 2012) (internal quotations and citations omitted) interpreting *Town of Nelson v. N.H. Dept. of Transportation*, 146 N.H. 75 (2001). The same is true here.

The Plymouth Water District has also provided no information as to what Plymouth or any subdivision of the town "funded prior to the adoption of Article 28–a." *City of Concord v. State*, 164 N.H. at 139. In fact, the plaintiffs, including the Plymouth Water District, concede that monitoring or treating for PFAS is already the practice and will remain the practice. Specifically, to support their argument that "NHDES will suffer no, or virtually on harm if an injunction issues," the plaintiffs state that "An injunction will not bar regulation of PFOA, PFOS, PFNA and PFHxS. They will simply be regulated at the current levels, which were the levels NHDES initially proposed, pending the proper adoption of new rules." Memo., pg. 24.

In addition, the State's Safe Drinking Water Act originated in 1977 (originally RSA 148-B, re-codified in 1989). "Accordingly," testing water quality was "previously mandated by other statutes." *City of Concord v. State*, 164 N.H. at 136. Indeed, "where a local subdivision has historically had responsibility for the subject matter of the mandate, some change in the scope of that responsibility does not result in a violation of Article 28–a."[11] *Id*. at 140. To the extent the Plymouth Water District argues that it may have to incur slightly higher costs to test for two new PFAS compounds, "an increase in expenditures alone is not dispositive of whether a program or responsibility has been expanded or modified." *Id*.

The Plymouth Water District provides nothing but speculation as to what costs municipalities in general may experience without any analysis of what the district itself will incur. Again, the Plymouth Water District already performed testing for two PFAS compounds (PFOA and PFOS) which came back "non-detect" at the then-available detection limit of 1-8 ppt. A claim regarding cost, therefore, would be "merely speculative." *City of Concord v. State*, 164 N.H. at 130. The N.H. Supreme Court in *New Hampshire Ass'n of Ctys. v. State*, 158 N.H. 284, 291 (2009) held that such speculative injury did not satisfy the requirement that "there must be a clear and substantial conflict with the constitution to declare a legislative act unconstitutional." *Id*. at 291.

Even the dissent in *City of Concord* would not find a violation of Part, I, Art. 28-a in this case. In her dissent, Justice Conboy relied on State law mandating participation in the State retirement system, pointing out that "local governments … do not have the option to withdraw." *City of Concord v. State*, 164 N.H. at 147. In this case, a municipality can simply choose not to

[11] The Plaintiffs point to a NHDES rule enacted prior to the N.H. Supreme Court cases on unfunded mandates. The rule, and exemption table affixed thereto, does not include many contaminants including the previous PFAS standard of 70 ppt.

own and operate what in many places is already a private enterprise.

NHDES successfully made the arguments above regarding the scope of Part I, Art. 28-a during prior rulemaking which, as the plaintiffs point out, prompted a change to the law – *not the constitution*. The plaintiffs note that during the passage of RSA 541-A:25, lawmakers noted that they believed the new statute embodied the "spirit" of the amendment. Memo. pg. 8. The "spirit" with which a lawmaker may have wanted the amendment invested does not change what the amendment actually says. In any event, the cited opinion stated that the new law captured the "spirit" that some aspired to in passing the amendment, not the actual requirements of Part, I, Art. 28-a. This Court must "not redraft the constitution in an attempt to make it conform to an intention not fairly expressed in it." *City of Concord*, 164 N.H. at 141 (internal quotation omitted). The constitution says what it says which is that the current standards at issue in this case do not qualify as an unfunded mandate. Again, the only real issue lies with the interpretation of the statute.

2. The NHDES Rulemaking Does Not Violate the "Unfunded Mandate" Provision in the APA

As the plaintiffs state, the Legislature amended the APA in 1994. Among other provisions, the Legislature created RSA 541-A:25. The first part of RSA 541-A:25 tracks the constitutional provision. The last sentence in paragraph I, as well as paragraphs II and III go beyond the constitutional language. RSA 541-A:25 states:

> I. A state agency to which rulemaking authority has been granted, including those agencies, the rulemaking authority of which was granted prior to May 6, 1992, shall not mandate or assign any new, expanded, or modified programs or responsibilities to any political subdivision in such a way as to necessitate further expenditures by the political subdivision unless such programs or responsibilities are approved for funding by a vote of the local legislative body of the political subdivision. *Such programs include those functions of a nature customarily undertaken by municipalities whether or not performance of such functions is required by statute.*

> II. *Such programs also include, but are not limited to,* functions such as police, fire and rescue, roads and bridges, solid waste, sewer and *water,* and construction and maintenance of buildings and other municipal facilities or other facilities or functions undertaken by a political subdivision.
>
> III. *Included in the scope and nature of such programs are those municipal functions which might be undertaken by a municipality or by a private entity and those functions which a municipality may legally choose not to undertake.*

RSA 541-A:25 (emphasis added to show additional requirements). These changes statutorily addressed whether certain optional programs, like those enumerated, would be included in an analysis of whether or not a State law or rule created an unfunded mandate. It left unchanged, however, other analyses related to such mandates. For instance, the court must still analyze whether the activity was "previously mandated by other statutes" (*City of Concord v. State*, 164 N.H. at 136); must acknowledge that "some change in the scope of that responsibility does not result in a violation" (*id*. at 140); must acknowledge that "an increase in expenditures alone is not dispositive of whether a program or responsibility has been expanded or modified" (*id*. at 136); and that recourse is inappropriate if the injury would be "merely speculative" (*id*. at 130). All of these factors weigh heavily, at a minimum, in favor of denying the plaintiffs' request for a preliminary injunction. Instead, the Plymouth Water District should be required to make specific allegations, be required to provide affidavits regarding these allegations as well as issues such as actual cost and past practice, and the State should be allowed to obtain discovery on these topics.

In addition, although the Legislature enacted RSA 541-A:25, it also passed SB 309. SB 309 specifically requires NHDES to make the standards at issue in this case. It contains no exemption for municipalities. When the Legislature requires NHDES to make rules, it does just that.

Although NHDES may struggle at times to reconcile its Legislative directives with RSA

541-A:25, the import of SB 309 was clear. PFAS constituted a health threat that must be addressed at all public water systems. *See* SB 309 Fiscal Note (discussing methods for determining effects on "human health"). The Legislature intended it to apply to municipalities and municipalities knew that it would. Specifically, the Fiscal Note for SB 309 states: "a potential reduction in the current AGQS for PFOA and PFOS may result in additional indeterminable costs to local and county government…." Exhibit D (from SB 309 Senate file). Summarized testimony of Senator Dan Innis indicates that the Legislature familiarized itself with this Fiscal Note. Exhibit D (from SB 309 Senate file). Written testimony from the N.H. Municipal Association, as summarized in Senate reports, states: "A fiscal note prepared by DES states the additional costs to municipalities, while indeterminable, could be 'significant' and cost 'millions of dollars.'" Exhibit D (from SB 309 Senate file). The association specifically raised concerns regarding unfunded mandates. The bill passed regardless.

NHDES itself ensured that the Legislature knew that this bill would require local expenditures. A letter from NHDES Commissioner Robert Scott to Senator Kevin Avard, Chair of the Senate Energy and Natural Resources Commission, dated January 23, 2018, states: "costs to government entities and rate payers will result from establishing the standard." Exhibit D (from SB 309 Senate file). A similar letter from Commissioner Scott to Representative Chris Christensen, Chair, House Resources, Recreation, and Development Committee states: "The cost to municipalities and other stakeholders could be large, in the event that treatment technologies, industrial pretreatment programs, or remediation efforts may be required." Exhibit D (from SB 309 Senate file).

In addition, although the Plymouth Water District complains that the cost it may incur in coming into conformance with standards creates a conflict with RSA 541-A:25, other laws

already specifically subject municipal water systems to such requirements. Specifically, RSA 483:4 entitled "Power to Require Improvements" related to public health states:

> I. *The department is empowered to investigate the sanitary conditions and methods pertaining to the source, treatment, and distribution of all public water supplies for domestic use, and to require the application of any treatment or improvement in conditions and methods as it may deem necessary to insure fitness and safety and adequate protection of the public health. If the department determines that improvements are necessary,* ***the municipality****, corporation, or person shall be so notified in writing and the requirements so ordered shall be effected pursuant to RSA 38:25 within a reasonable time to be fixed by the department. Appeals of actions of the department may be made as provided in RSA 485:59.* The department may set intermediate goals and time frames to assist municipalities, corporations, or persons to abide by an order of the department under this paragraph.
>
> II. Upon complaint of not less than 10 customers of an existing public water system or not less than 10 residents not currently served by a public water supply, the department shall make an investigation of conditions regarding water quality or quantity problems described in the complaint. *If, as a result of any such investigation, the department concludes that a significant public health or safety problem exists due to water supply quality or quantity, it shall perform a preliminary analysis of alternatives which address the problem.* The department may request additional information from the complainants and nearby public water supply system owners, such as data on water supply quality and quantity, well characteristics, and water distribution system characteristics, as is necessary to perform its investigation and analysis. If the department determines that an extension of water service from an existing public water supply system to the area of impaired water quality or quantity is the most feasible and cost-effective alternative, that the extension is consistent with municipal master planning, local water system policies and rules, RSA 9-B, and RSA 162-C:2, V, and that the existing public water system has adequate water supply and system capacity to serve the problem area, ***the municipality***, corporation, or person who owns the public water system shall be ordered to allow connection to its water distribution system from the identified area, regardless of existing municipal or public water system service area boundaries. The connection so ordered shall be effected pursuant to RSA 38:25 within a reasonable time to be fixed by the department and may contain limitations on water system connections unrelated to the original petition in order to limit unintended land use impacts. Appeals of actions of the department may be made as provided in RSA 485:59. The department may set intermediate goals and time frames to assist municipalities, corporations, or persons to abide by an order of the department under this paragraph. The provisions of this paragraph or of any order issued under this paragraph shall not delegate any costs associated with a connection to the person receiving the order from the department.

> III. *The department may investigate the sanitary conditions and methods pertaining to pumper stations, piping, storage, and treatment facilities of privately owned redistribution systems which present a threat to public health and safety. If the department determines that action, such as disinfection, is necessary, the* ***municipality****, corporation, or person shall be so notified in writing and the action so ordered shall be effected within a reasonable time to be fixed by the department. Replacement of existing infrastructure shall only be required in response to a specific public health threat.* Appeals of actions of the department may be made under RSA 485:59. The department may set intermediate goals and time frames to assist municipalities, corporations, or persons to abide by an order of the department under this paragraph.

RSA 483:4 (emphasis added). Therefore, a rule requiring a municipality to undertake corrective actions is not a new mandate, it is a longstanding statutory requirement.

Therefore, the rule pertains to an activity that municipalities generally do anyway, have been required to do for a long time, and to requirements related to corrective actions that always applied. There is no basis, on these facts, to issue a preliminary injunction.

III. NHDES PROPERLY CONSIDERED COSTS AND BENEFITS

The plaintiffs argue that NHDES did not thoroughly review the costs and benefits associated with the required rulemaking. The requirement to do so stems from SB 309 which "[a]mended RSA 485:3, I(b) with respect to the required rulemaking to say:

> (b) After *consideration* of the extent to which the contaminant is found in New Hampshire, the ability to detect the contaminant in public water systems, the ability to remove the contaminant from drinking water, and the costs and benefits to affected parties that will result from establishing the standard, a specification for each contaminant of either:

(emphasis added). However, the plaintiffs own documents demonstrate significant "consideration" of costs and benefits. Memo., pg. 19. NHDES is in the process of producing copious information to the plaintiffs on this same topic. Some costs could not be numerically quantified but were considered nonetheless. *Id*. at 20 ("it was not able to monetize the avoided health impact costs"). The Court should note that neither the APA nor SB 309 require NHDES

to publish its cost and benefit consideration or justify it to the public.

The plaintiffs also state: "NHDES did not conduct an economic analysis of costs and benefits of the type or detail required of the federal government when it must do a cost-benefit analysis in setting an MCL." Complaint, pg. 10. But neither the APA nor SB 309 require NHDES to commence the arduous and expensive analysis required of the U.S. EPA under federal rules. The law merely says that rulemaking shall commence "after consideration" of "costs and benefits."

In *Appeal of Nationwide Ins. Co.*, 120 N.H. 90 (1980), the N.H. Supreme Court analyzed what it meant for the Commissioner of the N.H. Dept. of Insurance to give "'due consideration' to the factors enumerated in RSA 412:15 and RSA 414:3…." *Id*. at 93. The Court determined that the Commissioner had discretion to determine how to give various factors "due consideration." It stated:

> RSA 413:3(b) (Supp.1977) does not prescribe the weight to be accorded to the various factors considered by the commissioner in ratemaking, and it is within his discretion to determine both the method to be used in deriving rates and the weight to be given to each factor. Nationwide has not overcome the presumption that the commissioner's decision is prima facie lawful and reasonable.

Id. at 94 (internal citations omitted). In this case, NHDES simply had to give costs and benefits "consideration." It did so. The attached affidavit from Sarah Pillsbury, Administrator of the NHDES Drinking Water and Groundwater Bureau, and the two publicly-available cost/benefit reports attached thereto, provide information about this process. Exhibit C; C.2; and C.3. Everything indicates that NHDES considered costs and benefits to the best of its ability. Nothing more is required.

IV. 3M CANNOT USE THIS CASE AS A TOOL TO OBTAIN RESULTS IN A DIFFERENT CASE

3M attempts to use its possible liability in another case to demonstrate standing in this

case. Memo., pg. 18. However, the case filed by the State against 3M will proceed in accordance with Court rules, 3M will have every opportunity to argue whether and to what extent damages are justified, cross-examine witnesses, and generally enjoy all of the process afforded to all litigants. It is not appropriate to ask this Court to use this action related to rulemaking as a way for 3M to litigate its damages in that case.

V. THE PLAINTIFFS HAVE NOT DEMONSTRATED WHAT ALLEGED "LIBERTY INTEREST" MAY BE IMPACTED BY PFAS REGULATIONS

The plaintiffs allege that the current PFAS regulations will deprive them of a "liberty interest" in relation to their due process[12] claims addressed above. Memo., pg. 17. However, "[n]ot every [] liberty interest lends itself to judicial enforcement or vindication." *Baker v. Cunningham*, 128 N.H. 374, 378–80 (1986). Of the recognized "categories of such interests," the plaintiffs' complaint and motion have alleged violations of none. *Id*. In this respect, they "are not adequate pleadings, because courts should not be forced to engage in inference, or guesswork, to identify a specific liberty interest that might be thought to have been infringed by an alleged failure to afford procedural due process." *Id*.

[12] The State reserves its right to question "whether a municipality may assert a due process claim against a state agency under Part I, Article 15 of our State Constitution." *In re Town of Bethlehem*, 154 N.H. 314, 328 (2006).

WHEREFORE, for the reasons stated above, NHDES requests that this Honorable Court:

A. Deny the plaintiffs request for a preliminary injunction; and,

B. Grant such other relief as it deems just and equitable.

Respectfully submitted,

THE STATE OF NEW HAMPSHIRE

By its attorney,

GORDON J. MACDONALD
ATTORNEY GENERAL

Dated: October 10, 2019

/s/ K. Allen Brooks
K. Allen Brooks, Bar No. 16424
Senior Assistant Attorney General
Environmental Protection Bureau
NH Department of Justice
33 Capitol Street
Concord, New Hampshire 03301-6397
(603) 271-3679

CERTIFICATE OF SERVICE

October 10, 2019

I hereby certify that on October 10, 2019 the foregoing was filed electronically and sent via electronic filing service, to the Plaintiff, through counsel.

/s/ K. Allen Brooks
K. Allen Brooks

EXHIBIT 1

THE STATE OF NEW HAMPSHIRE

MERRIMACK, SS SUPERIOR COURT

DOCKET NO. 2019-CV-00650

THE PLYMOUTH VILLAGE WATER & SEWER DISTRICT, RESOURCE MANAGEMENT, INC., CHARLES G. HANSON, and 3M COMPANY

Plaintiffs

v.

ROBERT R. SCOTT, AS COMMISSIONER OF THE NEW HAMPSHIRE DEPARTMENT OF ENVIRONMENTAL SERVICES

Defendant

AFFIDAVIT OF JASON C RANDALL IN SUPPORT OF MOTION FOR TEMPORARY AND PRELIMINARY INJUNCTIVE RELIEF

I, Jason C. Randall hereby depose and state:

1. I am District Superintendent of the Plymouth Village Water & Sewer District ("PVWSD" or "the District"). I make this Affidavit on personal knowledge.
2. After graduating with B.S. in Natural Science from Lyndon State College, I began my career as a Laboratory Field Services Technician in Concord, NH and later as the Laboratory Supervisor for the New Hampshire Department of Environmental Services Winnipesaukee River Basin Program. In 2014, I became Superintendent of the PVWSD where I am responsible for operation, maintenance, and administrative needs for the water and sewer systems, serving a population of approximately 7,000. I am a certified Grade 4 New Hampshire Wastewater Operator, as well as a Grade 2 New Hampshire Water Works Treatment and Distribution Operator. I was the recipient of the 2016 New England Water Environment Association: Operator Award

for New Hampshire, the 2017 Granite State Rural Water Association: Water System of the Year, the 2017 New Hampshire Department of Environmental Services: Source Water Sustainability Award, and the 2018 New England Water Environment Association: Asset Management Excellence Award. I also serve as a Director for the Granite State Rural Water Association, a member of the New Hampshire Water Pollution Control Association, a member New England Water Environment Association, and a member of the New Hampshire Drinking Water Coalition. I am also affiliated through PVWSD with North East Biosolids & Residuals Association, NH Municipal Association, NH Water Works Association, New England Water Works Association, and NH Public Works Association.

3. PVWSD and its staff are members of numerous trade and municipal organizations including, North East Biosolids & Residuals Association, New Hampshire Municipal Association and Granite State Rural Water Association.

4. The process by which the New Hampshire Department of Environmental Services ("NHDES") adopted new rules governing four PFAS compounds: PFOA, PFOS, PFNA and PFHxS was rushed and failed to adequately identify and consider the costs to be imposed on municipal water and sewer utilities relative to real health benefits for the utility customer. This results in potential massive costs to those municipal water and sewer utilities impacted by PFAS rules, without funding from the State, and takes away from other very real health and infrastructure related issues.

5. PVWSD is a village district and a political subdivision of the Town of Plymouth, New Hampshire, with a principal place of business at 227 Old N Main Street, Plymouth, NH 03264.

6. PVWSD is not part of the Town government, but instead is a separate municipality that serves water and sewer infrastructure including two gravel packed water wells, a water distribution system, two water storage tanks, nine sewer pump stations, gravity and force main sewer collection system, and a wastewater treatment plant.

7. PVWSD has approximately 1,100 water and sewer connections, and serves a population of approximately 7,000, including a State University, a hospital, public schools, and a private high school ("District Users"). Its 2019 water and sewer budget is $2.7 million, with additional planned capital investments of $14.5 million over the next three years.

8. Out of 234 towns in the state, the median household income in Plymouth is ranked 228th, second lowest in Grafton County. The ability to dramatically increase water and sewer rates in the District to cover the costs of an unfunded mandate is not an option and would place other necessary and inevitable capital improvements on hold, some of which NHDES indicated should be a "high priority" for the District Users. Yet, it is my understanding that these new rules could well require such actions.

9. Based on my experience and understanding, these new rules place major financial responsibility and liability of drinking water and groundwater contamination by PFOA, PFOS, PFNA and PFHxS on the customers of our small water and sewer district and will have a significant financial impact on every other municipal water and sewer utility in New Hampshire.

10. To my knowledge based on personal research PFAS compounds are commonly found in every household in New Hampshire and throughout the United States, including in products as diverse as: non-stick cookware, furniture, clothes, dental floss, cosmetics,

lubricants, paint, carpets, pizza boxes, popcorn bags, and PFAS compounds are used for a variety of applications.

PVWSD's Water Treatment and Distribution

11. PVWSD has two gravel packed wells on Foster Street in Plymouth. Average daily flow pumped from these wells is approximately 400,000 gallons per day in 2018. Water pumped from these wells is stored in two water tanks and then delivered by gravity or pumping to water distribution and service lines, and ultimately to the Users' homes, businesses, and institutions throughout the District.

12. PVWSD is required to deliver water to District Users that is safe to drink under the U.S. Environmental Protection Agency's Safe Drinking Water Act and NHDES rules. To do so, the District provides treatment of the groundwater prior to entry into the water distribution system by addition of chlorination for disinfection, caustic soda to raise pH and control pipe corrosion, as well as provides manganese and iron sequestration and additional pipe corrosion control by treating with ortho-polyphosphate. The chemicals for this treatment process currently cost approximately $35,000 per year. No other treatment is currently required to ensure water quality meets the requirements of NHDES and EPA, and District Users.

13. PVSWD is subject to the new NHDES rules as a public water system, and will be required to test for PFOA, PFOS, PFNA, and PFHxS at or below the new, dramatically reduced MCL levels in its well water in order to be in compliance. The rules require PVSWD to spend money for testing on a schedule established in the rules for 2019 and beyond. Testing for 2019 is not in the budget.

14. Should PFOA, PFOS, PFNA and PFHxS be detected at a level over the new MCL for the average of four consecutive quarterly samples and tests beginning in the fourth quarter of 2019, PVWSD will be required to take the contaminated water source out of service or treat its water, which includes the purchase, installation and operation a new treatment system. If a treatment system is required it will come with a significant capital, operation and maintenance investment, as well as take upwards of greater than a year to complete and place online. Also, as a practical matter, the treatment method required may exceed the current certification of the District's operators which will require additional training and certification or hiring of additional staff to be in compliance.

15. On May 31, 2016, NHDES adopted the US Environmental Protection Agency's 2016 Health Advisory limits of 70 parts per trillion for combined PFOA and PFOS.

16. At NHDES's request in 2016, PVWSD volunteered to test its two sources of drinking water and sampled on September 20, 2016. PVWSD received results for both wells of non-detect for six PFAS compounds using EPA method 537. Although the results were non-detect, the reporting detection limit (the lowest level established by the lab for the detection of a compound) provided by the laboratory was 20 parts per trillion for PFOA and 40 parts per trillion for PFOS, which was below the 70 parts per trillion adopted Health Advisory limits in place at that time. The reporting detection limits of 20 ppt and 40 ppt for the September 20, 2016 sample are above NHDES's PFOA and PFOS MCL's for those respective compounds in the new rules. To my knowledge the testing laboratory did not report results above the method detection limits (the ability of instrumentation to detect a compound) of 1-19 ppt for PFOA and

4-39 ppt for PFOS and flag them as qualifiers. This currently places a level of uncertainty if there may be detectable levels of PFOA, PFOS, PFNA and PFHxS in the District's water sources today, at or above the new, dramatically reduced MCL's. This also questions the reliability of the current technology and test methods available, and a laboratory's ability to detect, and confidently report to levels at or near the detection limits in parts per trillion.

17. PVWSD is regulated by NHDES and the U.S. Environmental Protection Agency. We pride ourselves as being in the environmental protection business right along with NHDES, and we are not opposed to appropriate regulation based on sound science and a reasoned consideration of costs and benefits, but believe this regulation was rushed and developed in advance of the scientific and public process. The public and political concern has led to stringent MCL's for PFOA, PFOS, PFNA and PFHxS in NH that has and will have significant unintended financial and operational impacts on PVWSD and other New Hampshire water and sewer utilities. Given the ubiquity of PFOA, PFOS, PFNA and PFHxS, and the comparative background levels which may be found in wastewater and biosolids, setting drinking water and groundwater standards near analytical detection limits may not provide a discernible benefit to public health. The District would like to work with NHDES to determine the most cost-effective steps needed to reduce human exposure and implement them within the broad context of protecting public health. This requires differentiating the high concentration sites from background concentrations and taking actions to mitigate concentrations at high use sites. It also demands a realistic assessment of how much

any regulatory action will be able to control PFOA, PFOS, PFNA and PFHxS already in the background environment.

18. Without the funding to support implementation, the new NHDES program constitutes an unfunded mandate that the District cannot absorb. The full costs for complying with this new regulation are still unknown and not something that has been planned for in our current and future budgets. Based upon our research and input from consulting engineers and scientists we anticipate these capital, and operational and maintenance costs will be significant in the range of hundreds of thousands of dollars and will place a severe burden on the customers in our District as well as for other municipalities, utilities, and communities throughout the state.

19. Once treatment is installed, annual Operation and Management costs will be incurred to maintain compliance.

20. Numerous water utilities in New Hampshire will face similar expenses.

21. No state funding has been guaranteed to pay for all of the costs of this new program.

22. Because of the rushed rulemaking process used by NHDES, there was no time to evaluate the MCL's and AGQS released on June 28, 2019. I am not aware of any effort by NHDES to reach out to small utilities like PVWSD, nor any opportunity to comment on the rules before they were approved by JLCAR on July 18, 2019.

23. It is my understanding that NHDES believes some water utilities will be able to stop use of a well that tests higher than the new rules allow for PFOA, PFOS, PFNA, and/or PFHxS. Shutting off a contaminated well is not an option for PVWSD, which has only 2 wells. The District's two water sources/wells on at the Foster Street site are only about 170 feet apart and share the same cone of influence in respect to

groundwater recharge based on observed drawdown during operation, wellhead protection area, and vulnerabilities due to their close proximity to Interstate 93. It would be highly likely that both wells would either be deemed contaminated under the new standards or not, not one or the other. Shutting off both wells is also not an option for PVWSD who will then be forced to implement emergency water distribution efforts to approximately 7,000 people.

24. It is understood that the first year under the PFAS rules will involve sampling only; 4th quarter 2019 through 3rd quarter 2020. More significantly, after the first year, substantial expenses associated with capital upgrades for treatment could be required, and that too is unfunded, leaving perhaps millions of dollars of capital expense to be borne solely by PVWSD and its customers.

25. This funding problem will be the same for numerous municipal water utilities throughout New Hampshire.

PVWSD Sewer Collection, Septage, and Wastewater Treatment Facility

26. Based on the Final Report of the Commission to Study Methods and Costs of Sewage, Sludge, and Septage Disposal (HB 699, Chapter 253, Laws of 2007) published November 1, 2008, it is my understanding that PVWSD is one of 85 municipal and 27 private WWTFs in New Hampshire, all of which by one process or another separate sludge and/or biosolids from the liquid portion of sewage and septage. Sewage is collected and conveyed through approximately 25 miles of gravity sewer and 5 miles of sewer force mains, some of which is pumped uphill by 9 pump stations located throughout the District.

27. Sewage is defined in accordance with RSA 485-A:2, X as "the water carried waste products from buildings, public or private, together with such groundwater infiltration and surface water as may be present."

28. PVWSD and every other municipal water and sewer utility throughout New Hampshire provide essential public services with a common mission to protect public health and the environment. PVWSD and other water and sewer utilities are not "producers" of PFOA, PFOS, PFNA and PFHxS. Rather, they are "receivers" of these chemicals used by customers, and merely convey the traces of those PFAS compounds that we encounter in our daily lives.

29. PVWSD also owns and operates a wastewater treatment facility ("WWTF") that recently completed upgrades in order to provide every town in New Hampshire with a receiving facility for septage, municipal sludge, and holding tank waste. In 2018, the District received septage, municipal sludge, and holding tank waste from 73 of the 234 towns (30%) in New Hampshire. This is a service the District provides to New Hampshire residents and businesses that are on private "decentralized" septic systems and as an alternative for municipal systems that are not able to manage wastewater sludge (i.e. emergencies, equipment malfunctions, lagoon sludge dewatering, etc.…).

30. PVWSD's WWTF processes approximately 1,000 wet tons of Class B Biosolids every year. Biosolids are the nutrient rich organic byproducts resulting from wastewater treatment. Those Biosolids have been treated and tested and meet strict EPA and NHDES Sludge Quality Certification standards for use as fertilizers and soil amendments at NHDES permitted sites. Biosolids provide plant nutrients and organic matter to soils when recycled and land applied on farm fields. The District has

promoted and practiced the beneficial use of Class A compost and Class B Biosolids for over 30 years within compliance with the regulations of EPA and NHDES Sludge Quality Certification.

31. Based on reading the Final Report of the Commission to Study Methods and Costs of Sewage, Sludge, and Septage Disposal (HB 699, Chapter 253, Laws of 2007) published November 1, 2008, it is my understanding that in 2007 it is estimated that 97,600 wet tons of sludge/biosolids were generated by such municipal WWTFs in the state and approximately 40% were of Class A or Class B quality that have the potential to be applied on farm fields for beneficial use. The remaining sludge was disposed of by landfill, incineration, and out of state.

32. The District has concern under the new, dramatically reduced PFAS MCL's that its Biosolids, which contain PFAS compounds based on recent District and NHDES testing, may no longer be viable for recycling at land application sites in New Hampshire because NHDES has taken the position that they may have the potential to impact nearby groundwater and drinking water, especially at the dramatically reduced levels in the new rules.

33. Biosolids are organic solids resulting from the treatment of wastewater.

a. The solids or "sludge" collected from the primary and secondary treatments, and septage are mixed together and undergo further treatment for vector attraction and pathogen reduction.

b. These combined solids typically are "dewatered" to make a more manageable, semi-solid material averaging a 30% solid cake (70% water).

c. The solids are also "stabilized" by some form of digestion (aerobic or anaerobic), and/or composting, or some other treatment (District utilizes the lime method that increases pH to accomplish vector attraction and pathogen reduction). There are rigorous federal guidelines which identify approved treatment processes.

d. Only after they have been through these treatment and testing procedures can "sludges" be called "biosolids" and used as fertilizers and soil amendments.

34. Currently, PVWSD recycles its Class B biosolids for beneficial use as fertilizer at a cost to the District of $70.72 per wet ton. Under the new rules it is possible that biosolids may no longer be considered viable for use as fertilizer by NHDES. In that case, we will be required to further treat the wastewater sludge/solids and then pay the current cost of $155.00 per ton to dispose of it in Canada or $200.00 per ton for disposal at a landfill in Bethlehem, New Hampshire. I have been notified that those two landfilling disposal options may not be available based on current market demand. If the availability of disposal in a landfill is the District's only option, costs alone will likely be $155,000.00 to $200,000.00 in the first year (compared to $70,720 currently), then every year thereafter subject to market prices. There may be additional costs for treatment as different disposal locations have specific requirements for odors reduction and elimination. I understand that in the near future NHDES plans to explore disposal options that may destroy PFAS compounds in wastewater solids, sludge, and biosolids utilizing technology and science still being developed and researched, such as high temperature incineration but those possible solutions are years away.

35. I understand that the law requires NHDES to begin a rulemaking process for surface water discharges for PFAS compounds by January 2020 which will impact the District's WWTF discharge of wastewater effluent to the Pemigewasset River. It would be in the best interest of the citizens and businesses of New Hampshire to participate in the rulemaking process for surface water standards and biosolids leaching standards as my understanding is that they may be identified as potential responsible parties (PRP's) for contamination of water's of the State including groundwater, drinking water, surface water, and wastewater solids due to their daily use and conveyance of PFAS containing products and materials.

36. To my knowledge NHDES plans to include municipal stakeholders in the rulemaking process by conducting workshops to determine options and feasibility of wastewater effluent and solids treatment and disposal/recycling to determine costs and impacts associated by the rulemaking for all sewer utilities in the State. The process by which NHDES proposes the surface water quality and biosolids leaching standards should fully comply with the law which would provide treatment cost solutions and an implementation plan for all sewer utilities impacted. There is currently no funding proposed or budgeted in the District's 2020 budget and beyond for these unanticipated costs of wastewater effluent and solids treatment and disposal/recycling.

EXHIBIT 1

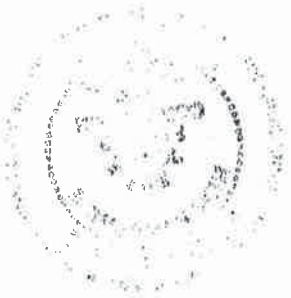

EXHIBIT 1

Dated: October 16, 2019 By: _____

Before me at Plymouth New Hampshire, on October 16, 2019, personally appeared Jason Randall, who has sworn and signed before me that the above is true and accurate to the best of his knowledge.

KIM A. HAINES NOTARY PUBLIC STATE OF NEW HAMPSHIRE MY COMMISSION EXPIRES JUNE 4 2024

_____
Notary Public / Justice of the Peace

My Commission Expires

_____

STATUTORY PROVISIONS AND RULES

Statutes

RSA 485:1-a Definitions. (Excerpts)

I. "Community water system" means a public water system which serves at least 15 service connections used by year-round residents or regularly serves at least 25 year-round residents.

XI. "Non-transient non-community water system" means a system which is not a community water system and which serves the same 25 people, or more, over 6 months per year.

RSA 485:3 Drinking Water Rules.

I. The commissioner shall adopt under RSA 541-A, following public hearing, drinking water rules and primary drinking water standards which are necessary to protect the public health and which shall apply to all public water systems. Such rules shall include:

(a) identification of contaminants which may have an adverse effect on the health of persons;

(b) After consideration of the extent to which the contaminant is found in New Hampshire, the ability to detect the contaminant in public water systems, the ability to remove the contaminant from drinking water, and the costs and benefits to affected parties that will result from establishing the standard, a specification for each contaminant of either:

(1) A maximum contaminant level that is acceptable in water for human consumption; or
(2) One or more treatment techniques or methods which lead to a reduction of the level of such contaminant sufficient to protect the public health, if it is not feasible to ascertain the level of such contaminant in water in the public water system; and

(c) criteria and procedures to assure compliance with the levels or methods determined under subparagraph (b), including quality control monitoring and testing procedures and standards to ensure compliance with such levels or methods; criteria and standards to ensure proper operation and maintenance of the system; requirements as to the minimum quality of water which may be delivered to the consumer; and requirements with respect to siting new facilities. Such rules shall be no less stringent than the most recent national Primary Drinking Water Regulations in effect, as issued or promulgated by the United States Environmental Protection Agency.

II. The commissioner may adopt secondary drinking water rules, which are necessary to protect the public welfare. Such rules may apply to any contaminant in drinking water which may adversely affect the color, odor, taste or appearance of the water and consequently may

cause a substantial number of persons to discontinue using a public water system, or which may otherwise adversely affect the public welfare. Such rules may vary according to geographic, economic, technical or other relevant circumstances. Such rules shall reasonably assure the protection of the public welfare and the supply of aesthetically adequate drinking water.

III. The commissioner shall adopt under RSA 541-A all rules necessary to implement the requirements of the following sections of this chapter:

(a) RSA 485:42.

(b) RSA 485:43.

IV. The commissioner may adopt rules specifying criteria and procedures for requiring public water systems to conduct monitoring programs for contaminants which are not identified in the national primary drinking water regulations, but which have been identified by the administrator of the United States Environmental Protection Agency as "unregulated contaminants." Such rules shall require monitoring of drinking water supplied by the system and shall vary the frequency and schedule of monitoring requirements for systems. An unregulated contaminant is one for which no maximum contaminant level or treatment technique has been established under paragraph I or II. In developing such rules, the commissioner shall consider materials submitted by the department of health and human services, pursuant to RSA 125-H:3. Rules adopted under this paragraph shall list unregulated contaminants for which public water systems may be required to monitor. Any list established pursuant to this paragraph shall be consistent with, but not limited by, the list of unregulated contaminants identified in regulations promulgated by the administrator of the United States Environmental Protection Agency.

V. The commissioner may adopt rules specifying the criteria under which filtration, including coagulation and sedimentation, as appropriate, is required as a treatment technique for public water systems supplied by surface water sources. In developing such rules the commissioner shall consider the quality of source waters, protection afforded by watershed management, treatment practices such as disinfection and length of water storage and other factors relevant to protection of health. The commissioner may require any public water supply system to assist in determining the necessity of filtration in that system. The commissioner shall provide an opportunity for notice and public hearing prior to implementation of any filtration requirement. Following such hearing, the commissioner shall prescribe, by rule adopted pursuant to RSA 541-A, a compliance schedule for such filtration requirement.

VI. The commissioner may adopt rules requiring disinfection as a treatment technique for all public water systems.

VII. The commissioner may adopt rules specifying the criteria and procedures to be used to identify and notify persons who may be affected by lead contamination of their drinking water when such contamination results from either the lead content in the construction materials of the

public water system or the corrosivity of the water supply, or both. The commissioner may also adopt rules prohibiting the use of lead pipes, solder and flux in the installation or repair of any public water system or any plumbing in a residential or nonresidential facility providing water for human consumption. Such rules shall not prohibit the use of leaded joints necessary for the repair of cast iron pipes.

VIII. The commissioner may adopt rules relative to defining the best available technology, treatment techniques, or other means which are feasible for the purpose of meeting the federal maximum contaminant level. In defining the best available technology, treatment technique or other means, the commissioner may consider the number of persons served by the system, other physical conditions related to engineering feasibility and cost of compliance, and information contained in health risk assessments provided by the department of health and human services pursuant to RSA 125-H:3, II and IV. Such rules shall specify all applicable criteria relative to the commissioner's determination.

IX. The commissioner may adopt rules to implement a wellhead protection program pursuant to RSA 485:48.

X. The commissioner may adopt rules to implement the Underground Injection Control Program of the federal Safe Drinking Water Act, 42 U.S.C. section 300f et seq., as well as rules pertaining to permits for the regulation and remediation of contamination from previous discharges or disposal of waste to the groundwater. The commissioner's rules shall include criteria and procedures to ensure that past and present underground injection will not endanger drinking water sources, and shall provide for consideration of varying geologic, hydrologic, or other conditions in different areas within the state.

XI. The commissioner shall adopt rules, pursuant to RSA 541-A, specifying the water quality standards and other criteria and procedures for obtaining a permit to use a source of water for the manufacture of bottled water.

XII. The commissioner may adopt rules to ensure long-term viability of public drinking water systems as required by section 119 of the federal Safe Drinking Water Act Amendments of 1996, 42 U.S.C. section 300g-9 to qualify for full eligibility for federal and state revolving fund capital grants.

XIII. The commissioner shall adopt rules, pursuant to RSA 541-A, relative to new groundwater withdrawals of 57,600 gallons or more in any 24-hour period by public water systems. Such rules shall include:

(a) Criteria and procedures for requiring public water systems to identify and address impacts of withdrawals on surface waters, subsurface waters, water-related natural resources, and public, private, residential, and farm wells within the anticipated zone of

contribution to the withdrawal.

(b) Requirements relative to conservation management plans which demonstrate the need for the proposed withdrawals, to be submitted by the public water system seeking approval for a withdrawal.

(c) Procedures by which the department may deny permission for withdrawals or order the applicant to provide a response policy, as provided by department rules, for provision of alternative water supply at no initial capital cost to persons whose wells are adversely affected by the proposed withdrawal or order reduced withdrawals if hydrogeologic data indicate that water-related resources are being adversely affected by the withdrawals.

XIV. The commissioner may adopt rules to:

(a) Regulate the heat exchange fluids utilized in closed loop geothermal systems. The commissioner's rules shall include criteria and procedures to ensure that these substances when released to the environment will not endanger drinking water sources.

(b) Prohibit the construction of open loop geothermal systems where such process will contaminate freshwater aquifers with brackish or saline groundwater.

RSA 485:41 <u>Duties of Department</u>.

The department shall:

I. Monitor the operation and maintenance of new and existing public water systems and privately owned redistribution systems.

II. Adopt rules governing the maintenance and operation of public water systems to ensure compliance with drinking water standards and to protect the public health.

III. Adopt rules governing the installation of pipes, fixtures and other apparatus which are used to connect the water system or privately owned redistribution system to a building. Such rules shall be considered minimum standards. The department shall adopt the International Plumbing Code as published by the International Code Council by reference, provided the department specifies which sections of the code are in force in New Hampshire and makes specific any discretionary provisions in the code subject to approval by the state building code review board. The department shall periodically review the rules adopted under this paragraph to assure that they are no less stringent than the requirements of the current code.

IV. Adopt rules establishing recordkeeping, reporting and testing requirements for public water systems.

V. Enter, and authorize its employees and agents to enter, the premises of all public water systems and privately owned redistribution systems for the purpose of carrying out inspection and for the purpose of taking water samples, to determine compliance with the provisions of this

chapter or rules adopted under it, and to inspect any and all records and facilities of such public water supply or privately owned redistribution system in order to determine compliance with this chapter and rules adopted under it.

VI. Undertake long-range planning and studies, within available state and federal funding, relating to the purity of drinking water in the state.

VII. Make available to the public the analytical results of all monitoring and testing undertaken pursuant to this chapter.

VIII. Adopt a fee system in recognition of services provided by the water supply engineering bureau including the issuance of an operational permit for public water systems subject to this chapter. The commissioner shall adopt rules establishing the application process for the issuance of operational permits pursuant to RSA 541-A. The fee category for community systems per year shall be $300, but in no case shall the fee exceed $10 per household or household equivalent. The fee category for non-transient and non-community systems shall be $150 per year. All fees shall be paid to the department for deposit in the operational permits account. Moneys in the operational permits account shall be used to pay the salaries, benefits and expenses of the staff in the department's drinking water supply program. Any revenues generated in excess of the costs of funding the drinking water supply program's expenses, shall lapse to the general fund at the close of each fiscal year to be used to offset the future general fund appropriation for the water supply program.

IX. Adopt rules applying to privately owned redistribution systems requiring:

(a) Periodic monitoring of coliform bacteria and public notification, and remedial action in case of violation of bacterial water quality standards, consistent with the rules which apply to public water systems for such bacterial water quality standards.

(b) Retention of a primary water operator who maintains an operating certificate at a minimum grade 1-A level.

(c) Inspection and maintenance of exterior pumping stations, distribution networks, and exterior storage tanks.

(d) Design standards for new and replacement facilities consistent with the rules which apply to public water systems as limited by the provisions of RSA 485 concerning privately owned redistribution systems, and provided that such rules require that any plans review required by RSA 485 shall be completed within 30 days of the submission of such plans.

RSA 485:58 <u>Enforcement and Penalties</u>.

I. If the department determines that a primary standard has violated, or that, in its judgment, a condition exists in a public water system which will cause a violation of a primary standard and may result in a serious risk to public health, it may issue an order requiring:

(a) The prohibition of transportation, sale, distribution or supplying of water;

(b) The repair, installation or operation of purification equipment or methods;

(c) The notification of all potential users of the system, including travelers, of the nature, extent and possible health effects of the imminent hazard, and precautions to be taken by users; or

(d) The testing, sampling or other analytical operations required to determine the nature, extent, duration or termination of the imminent hazard.

The superior court shall place any action filed by the department to enforce an order under this section at the top of its calendar of cases and shall provide an expeditious hearing on such order.

II. Any reckless violation of any provision of this chapter, any rule adopted under this chapter, any term or condition of an approval, exemption, variance or order issued under this chapter, or any misstatement of a material fact required to be disclosed under this chapter shall constitute a misdemeanor for a natural person and a felony for any other person.

III. Unless otherwise provided, any purposeful or knowing violation of any provision of this chapter, any rule adopted under this chapter, any term or condition of an approval, exemption, variance, or order issued under this chapter, or any misstatement of a material fact required to be disclosed under this chapter shall constitute a class B felony.

IV. Any person who violates any provision of this chapter or any rule adopted or any term or condition of an approval, exemption, variance or order issued under this chapter shall be liable to the state, upon suit brought by the attorney general, for a civil forfeiture in an amount not to exceed $25,000 for each day of such violation. (Amended 2019)

V. The commissioner of environmental services, after notice and hearing pursuant to RSA 541-A, may impose an administrative fine not to exceed $2,000 for each offense upon any person who violates any provision of this chapter including any rule adopted under the provisions of this chapter. Rehearings and appeals from a decision of the commissioner under this paragraph shall be in accordance with RSA 541. Any administrative fine imposed under this section shall not preclude the imposition of further penalties under this chapter. The proceeds of administrative fines levied pursuant to this paragraph shall be deposited by the department in the general fund. The commissioner shall adopt rules, under RSA 541-A, relative to:

(a) A schedule of administrative fines which may be imposed under this paragraph for violations of this chapter as provided above.

(b) Procedures for notice and hearing prior to the imposition of an administrative fine.

(Amended 2019)

VI. Any act or failure to act in violation of RSA 485:8, II; 31; 42; 43; 46; or 48; or any rule adopted under RSA 485:2; 3; 4; 40; 41; 44; or 47 may be enjoined.

VII. Notwithstanding RSA 651:2, any person may, in addition to any sentence of imprisonment, probation or conditional discharge, be fined not more than $25,000 if found guilty of any violation of paragraph II or III of this section. The court may also order the person to pay the costs of remediation. Each day of violation shall constitute a separate offense. (Amended 2019)

Rules

N.H. Admin R. Env-Dw 103.11.

"Community water system" means "community water system" as defined in RSA 485:1- a, I, as reprinted in Appendix B.

N.H. Admin R. Env-Dw 103.38.

"Non-transient non-community water system (NTNC)" means "non-transient noncommunity water system" as defined in RSA 485:1-a, XI, as reprinted in Appendix B.

N.H. Admin R. Env-Dw 705.06 MCLs and MCLGs for Per- and Polyfluoroalkyl Substances (PFAS) Contaminants.

(a) The MCLs and MCLGs for the per- and polyfluoroalkyl substances contaminants specified in (b), below, shall apply to community water systems and non-transient non-community water systems.

(b) The MCLs and MCLGs for PFAS contaminants shall be as specified in Table 705-7, below:

Table 705-7: PFAS Contaminant MCLs and MCLGs

PFAS Contaminant	MCL (mg/L)	MCLG (mg/L)
Perfluorohexane sulfonic acid (PFHxS)	0.000018	0
Perfluorononanoic acid (PFNA)	0.000011	0
Perfluorooctane sulfonic acid (PFOS)	0.000015	0
Perfluorooctanoic acid (PFOA)	0.000012	0

(c) Monitoring and compliance for PFAS contaminants shall be as specified in Env-Dw 707, Env-Dw 708, and Env-Dw 712.

N.H. Admin R. Env-Dw 708.09 Public Notice of Non-Compliance.

(a) Subject to (b), below, the O/O shall provide public notice as required by Env-Dw 800 if any violation occurs of any applicable MCL, MRDL, monitoring requirement, treatment technique established in RSA 485 or Env-Dw 700, or reporting or recordkeeping requirement specified in Env-Dw 709, Env-Dw 720.14(b), or Env-Dw 720.16(b).

(b) If a system has a distribution system with portions that are hydraulically separate from other parts of the distribution system, the O/O may request approval from the department pursuant to Env-Dw 801 to limit the public notice to users of only that part of the system in which the applicable standard is exceeded.

N.H. Admin R. Env-Dw 712.23 Initial Monitoring for Per- and Polyfluoroalkyl Substances (PFAS) Contaminants.

(a) Beginning with the first quarter following the 2019 effective date of this section, the O/O of an existing community water system or existing non-transient, non-community water system shall collect 4 consecutive quarterly samples for the per- and polyfluoroalkyl substances contaminants listed in Env-Dw 705.06 at each sampling point identified in the sampling schedule established pursuant to Env-Dw 708.01.

(b) Beginning with the first quarter following the initiation of operations of a new community water system or new non-transient, non-community water system, the O/O shall collect 4 consecutive quarterly samples for the PFAS contaminants listed in Env-Dw 705.06 at each sampling point identified in the sampling schedule established pursuant to Env-Dw 708.01.

(c) If the results of the samples from the first 2 quarters are below the detection limits specified in EnvDw 712.28(c), the O/O may submit a written request to the department for the monitoring frequency to be reduced.

(d) A written request submitted pursuant to (c), above, shall include the following:

(1) The name of the PWS;

(2) The PWS identifier for the PWS; and

(3) A summary of the historical PFAS data from the system and nearby systems, when available.

(e) If the department determines that the results are all below the detection limits listed in Table 712-2, the final 2 quarters of the initial monitoring shall be waived and the monitoring frequency shall be as specified in Env-Dw 712.24.

N.H. Admin R. Env-Dw 712.24 Monitoring Frequency for PFAS Contaminants.

(a) Subsequent to the initial monitoring required by Env-Dw 712.23 and subject to Env-Dw 712.26, the O/O shall monitor for all PFAS contaminants based on the PFAS contaminant with the most frequent monitoring period calculated from the average of the results of the initial monitoring required by Env-Dw 712.23, as specified in Table 712-1, below, and as demonstrated in Appendix D for specific PFAS contaminants:

Table 712-1: Monitoring Frequency Based on PFAS Contaminant Concentrations

Average Monitoring Result (ng/L)	Frequency
Greater than 50% of MCL to 100% of MCL	Annually
50% of MCL or less	Once every 3 years

(b) If the average monitoring result exceeds 100% of the MCL, the O/O shall monitor as specified in Env-Dw 712.27.

(c) The O/O shall monitor for PFAS contaminants during the quarter in which the highest analytical result was observed.

(d) Subsequent sample results shall be used to establish future PFAS contaminant sampling schedules using the shortest PFAS monitoring period specified in Table 712-1.

(e) Based on a review of the submitted results, the department shall:

(1) Modify the system's schedule in accordance with Table 712-1 or (b), above, as applicable; and

(2) Notify the O/O in writing of the new monitoring requirements.

N.H. Admin R. Env-Dw 712.25 Monitoring Location for PFAS Contaminants.

(a) The O/O of a PWS supplied by a groundwater source shall collect at least one sample to be analyzed for PFAS contaminants at every entry point to the distribution system. Each entry point shall be representative of each well after treatment, as specified in the sampling schedule established pursuant to Env-Dw 708.01.

(b) The O/O of a PWS supplied by a surface water source or a combination of surface water and groundwater shall collect at least one sample to be analyzed for PFAS contaminants at points in the distribution system that are representative of each source or at each entry point to the distribution system after treatment, as specified in the sampling schedule established pursuant to Env-Dw 708.01.

(c) If the O/O believes that conditions make another sampling point more representative of a source, treatment plant, or distribution system for purposes of sampling for PFAS contaminants, the O/O shall request a change in sampling location for such contaminants pursuant to Env-Dw 708.04.

(d) If a PWS obtains water from more than one source and the sources are combined prior to entering the distribution system, the O/O shall collect the samples to be analyzed for PFAS contaminants at an entry point to the distribution system during periods of normal operating conditions, when water from all sources is being used.

N.H. Admin R. Env-Dw 712.26 Confirmation Sampling for PFAS Contaminants.

(a) Subject to (c), below, if a PFAS contaminant is detected in a representative sample at a level greater than 50% of the MCL, the O/O shall:

(1) Collect a confirmation sample under the same contributing conditions within 14 days of being notified of the result; and

(2) Have the sample analyzed for the contaminant(s) detected.

(b) If a confirmation sample is required pursuant to (a) above, the results of the initial and confirmation samples shall be averaged to determine compliance with the MCL specified in Env-Dw 705.06.

(c) If results from the sampling point or the contributing sources have historically demonstrated the presence of that PFAS contaminant at a level greater than 50% of the MCL, then:

(1) A confirmation sample shall not be required; and

(2) The monitoring frequency for the approved sampling point shall be determined pursuant to EnvDw 712.24 or Env-Dw 712.27, as applicable.

N.H. Admin R. Env-Dw 712.27 Increased Monitoring for PFAS Contaminants.

The O/O shall collect and analyze quarterly PFAS samples at all sampling points if:

(a) The running annual average for any PFAS contaminant at the sampling point is above the applicable MCL; or

(b) The PWS is operating any type of treatment to reduce the amount of a PFAS contaminant.

N.H. Admin R. Env-Dw 712.29 Compliance Determination for PFAS Contaminants; Limiting Public Notice.

(a) Compliance with Env-Dw 705.06 shall be determined using the analytical results obtained at each sampling point that is an entry point to the distribution system, as specified in the sampling schedule established pursuant to Env-Dw 708.01.

(b) For any PWS that conducts monitoring for PFAS contaminants at a frequency greater than annually, the department shall determine compliance by calculating a running annual average of all samples collected at each sampling point. If the annual average of any sampling point is greater than the MCL, then the department shall identify the PWS as out of compliance.

(c) If monitoring for PFAS contaminants is conducted annually or less frequently, then the department shall identify the PWS as being out of compliance if the level of a PFAS contaminant at any sampling point is greater than the MCL. NEW HAMPSHIRE CODE OF ADMINISTRATIVE RULES 43 Env-Dw 709-713

(d) If a PWS has a distribution system with portions that are hydraulically separate from other parts of the distribution system, the O/O may request approval from the department pursuant to Env-Dw 801 to limit the notice to only that portion that is out of compliance.

N.H. Admin R. Env-Dw 719.02 Required Reporting and Public Notice.

(a) An O/O shall submit all monitoring data and other required information as specified in this part.

(b) An O/O shall provide public notice as specified in Env-Dw 800.

N.H. Admin R. Env-Dw 719.03 Third-Party Laboratory Obligations.

(a) Before an O/O submits samples for analysis to meet monitoring requirements to a third-party laboratory, the O/O and third-party laboratory shall enter into a binding written agreement that requires the laboratory to submit all information required by, and as specified in, Env-Dw 719.05.

(b) Each time an O/O submits any sample to a third-party laboratory for the purpose of meeting a monitoring requirement, the O/O shall inform the laboratory that the analyses being requested are to meet department monitoring requirements.

N.H. Admin R. Env-Dw 719.04 Registering for Electronic Reporting System.

(a) In order to access the electronic reporting system, the O/O or third-party laboratory shall register as a data provider under the department's OneStop portal at https://www4.des.state.nh.us/OnestopDataProviders/DESLogin.aspx, by:

(1) Providing the information required in (b), below;

(2) Selecting the "DWGB Certified Lab Upload" or "DWGB Login Samples" as applicable; and

(3) Accepting the agreement specified in (c), below.

(b) The O/O or third-party laboratory shall provide the following information:

(1) The type of requestor, specifically consultant, contractor, individual, federal government, laboratory, local government, non-profit business, other business, PWS contact, state government;

(2) The first and last name of the individual who is applying for access;

(3) The applicant's email address;

(4) A user name and password;

(5) The name, mailing address, daytime telephone including area code and extension, if any, email address, and fax number including area code of the business or other organization with which the requestor is affiliated;

(6) If the requestor has another telephone number, the telephone number including area code and extension, if any; and

(7) If the requestor has another email address, the secondary email address.

(c) The agreement between the data provider and the department shall be as follows:

"I understand that I am responsible for the accuracy of the information submitted in this request for a PIN and Password that will allow me to access, via the Internet, certain information held by the Department of Environmental Services.

I further understand that the information I am submitting and/or requesting access to is security-sensitive, and I agree to (1) protect the PIN and Password provided to me by the Department of Environmental Services to prevent any unauthorized use thereof; (2) protect against further dissemination and/or unauthorized use of any and all information obtained using the PIN and Password to the extent reasonable in light of the legitimate use for which the information is needed, as described above.

I affirm that the information I am submitting with this request is true, complete, and not misleading. I understand that the submittal of false, incomplete, or misleading information could result in denial or revocation of data provider access and/or could subject me to the criminal penalties established for falsification in official matters, currently in RSA 641.

I understand that by clicking on I AGREE, I am solely responsible for the PIN and Password I am about to receive and for any and all information collected using the PIN and Password, and that NO LIABILITY IS INCURRED BY THE STATE by reason of providing the requested access.

I understand that if I do not agree to be the sole responsible party, I should click CLOSE to discontinue the PIN/Password request process."

N.H. Admin R. Env-Dw 719.05 <u>Reporting Method Prior to CMDP Activation</u>.

(a) Until such time as notification is made that the CMDP is available for use, the following information shall be submitted as specified in (b), below, for each sample analyzed to meet monitoring requirements:

(1) The PWS identifier of the system;

(2) The name of the PWS and the city or town in which the system is located;

(3) Sample collection information, including:

a. The sample location identifier and name; and

b. The date and time the sample was collected;

(4) Whether the data being submitted is original or a revision;

(5) The name of contaminant(s) or contaminant code(s) for which the sample was collected and tested;

(6) Whether the sample is a routine, repeat, make-up, confirmation, or replacement sample;

(7) The monitoring period for which the sample was collected, by month or quarter, as applicable, and year;

(8) The first and last name and daytime telephone number including area code of the individual who collected the sample, or the person responsible for any sample(s) collected by individual homeowners;

(9) The date and time the sample was received by the laboratory;

(10) The temperature of the sample(s) upon receipt, in degrees Celsius;

(11) The accreditation identifier of the laboratory that performed the analysis;

(12) The laboratory sample identifier and batch identifier, if different;

(13) The results of the analysis, including units and an explanation of whether the result is an actual or calculated value, and any qualifiers associated with the result(s);

(14) The date and time the sample was:

a. Prepared for analysis, if applicable; and

b. Analyzed;

(15) The analytical method used, including reportable and method detection limits, if applicable, with applicable units; and

(16) If the sample was initially received by a laboratory other than the one performing the analysis, the name, accreditation identifier, and daytime telephone number including area code of the laboratory that initially received the sample.

(b) The required information shall be submitted using the electronic reporting system for drinking water monitoring data.

N.H. Admin R. Env-Dw 719.06 Reporting Content and Method When CMDP Is Available.

When the CMDP becomes available, then:

(a) The department shall notify each O/O of the date the system will be available for use;

(b) The information regarding samples analyzed to meet monitoring requirements shall be the information required by the CMDP; and

(c) The information required by (b), above, shall be submitted to the department using the CMDP.

N.H. Admin R. Env-Dw 719.07 Timing of Reporting.

(a) Except where a shorter period is specified, such as where there is an exceedance of an acute contaminant MCL as specified in Env-Dw 801, the O/O or third-party laboratory, as applicable, shall report to the department within 2 business days of the analysis being completed the results of any test measurement or analysis required by:

(1) Env-Dw 723 relative to non-central treatment; or

(2) Env-Dw 709 through Env-Dw 715 and Env-Dw 717 relative to water quality monitoring.

(b) The O/O or third-party laboratory, as applicable, shall notify the department by telephone as soon as possible, but within 24 hours, of:

(1) The presence of fecal coliforms or E. coli in any sample; and

(2) The exceedance of any nitrate or nitrite MCL as specified in Env-Dw 704.02, Table 704-1.

N.H. Admin R. Env-Dw 801.03 Elements of a Public Notice.

Unless otherwise specified in Env-Dw 802 or Env-Dw 803, public notice shall:

(a) For each violation or situation for which notice is being given:

(1) Describe the violation or situation, including identifying each contaminant of concern and the corresponding contaminant level;

(2) Identify the compliance period, including year, when the violation or situation occurred;

(3) Describe any potential adverse health effects from the violation or situation using the applicable health effects language for that contaminant as specified in Env-Dw 804 through EnvDw 810; and

(4) Identify the population(s) at risk, including each subpopulation that is particularly vulnerable if exposed to the contaminant in the drinking water;

(b) Advise whether alternative water supplies should be used;

(c) Identify what actions a consumer should take, including when to seek medical help, if known and applicable;

(d) Describe actions the O/O is taking to correct the violation(s) or situation(s);

(e) Identify when the O/O is expected to return to compliance or otherwise resolve the situation(s);

(f) Provide the name, business address, and telephone number of the PWS owner, certified operator, or designee as a source of additional information concerning the notice;

(g) Include the following statement: “Please share this information with all the other people who drink this water, especially those who may not have received this notice directly (for example, people in apartments, nursing homes, schools and businesses). You can do this by posting this notice in a public place or distributing copies by hand or by mail.”; and

(h) Include the following statement, including the information necessary to fill in the blanks, if the public notice is issued for a monitoring and reporting or monitoring and testing procedure violation as set forth in Env-Dw 707 through Env-Dw 713: “We are required to monitor your drinking water for specific contaminants on a regular basis. Results of regular monitoring are an indicator of whether or not your drinking water meets health standards. During [compliance period], we ‘did not monitor or test’ or ‘did not complete all monitoring or testing’ for [contaminants(s)], and therefore cannot be sure of the quality of your drinking water during that time.”

N.H. Admin R. Env-Dw 801.04 Acute Public Notice and Required Consultations.

(a) For purposes of this section, the following definitions shall apply:

(1) "Consult with the department" means the O/O has engaged in 2-way communications directly with an individual in the department's drinking water program, whether such communications are in person, by telephone, by fax, or by e-mail;

(2) "Initiate consultations" means the O/O has made reasonable efforts to communicate with the department, whether or not the efforts were initially successful; and

(3) "Made reasonable efforts" means the O/O conveyed information to the department by telephone, fax, or e-mail regarding the occurrence of the violation or situation, which included current contact information for the O/O or designee.

(b) As soon as practical but no later than 24 hours after learning of a turbidity MCL violation as listed in Env-Dw 801.05(g) or a treatment technique violation as listed in Env-Dw 801.05(h), the O/O shall consult with the department as specified in Env-Dw 801.06 relative to consultation for acute public notice violations or situations.

(c) As soon as practical, but no later than 24 hours after learning of a violation or situation listed in Env-Dw 801.05(a)-(f) or (i)-(k), the O/O shall:

(1) Issue acute public notice as specified in Env-Dw 801.07; and

(2) Initiate consultations with the department as specified in Env-Dw 801.06 to determine whether additional public notice is required.

N.H. Admin R. Env-Dw 801.05 Violations and Other Situations for Which Acute Public Notice is Required.

The O/O shall provide acute public notice, in accordance with Env-Dw 801.01 through Env-Dw 801.04 and Env-Dw 801.07, of any of the following violations or situations:

(a) Any violation of the MCL for E. coli specified in Env-Dw 702;

(b) Any detection of E. coli as specified in Env-Dw 709.19, subject to Env-Dw 709.20;

(c) Any detection of E. coli, enterococci, or coliphage in the groundwater source at a system that is subject to Env-Dw 717;

(d) Any violation of the MCL for nitrate, nitrite, or total nitrate and nitrite as specified in Env-Dw 704.02 and determined in accordance with Env-Dw 707, Env-Dw 708, and Env-Dw 711;

(e) Any failure to collect a confirmation sample of nitrate, nitrite, or total nitrate and nitrite within 24 hours of the water system's receipt of the first sample results showing an exceedance of the nitrate or nitrite MCL, if required pursuant to Env-Dw 708 or Env-Dw 711;

(f) Any violation of the chlorine dioxide MRDL as specified in Env-Dw 705.04(c), where:

(1) The required samples were not collected in the distribution system; or

(2) One or more samples collected in the distribution system the day following an exceedance of the MRDL at the entrance of the distribution system exceed the MRDL;

(g) Any violation of the turbidity MCL specified in 40 CFR 141.13(b) if:

(1) The O/O fails to consult with the department within 24 hours of learning of the violation as required by Env-Dw 801.04(b); or

(2) After the required consultation, the department determines, based on the circumstances causing or contributing to the violation, that public notice is required to protect public health and safety;

(h) Any violation of a treatment technique requirement specified in the federal requirements relating to filtration and disinfection that are incorporated by Env-Dw 716, resulting from a single exceedance of the maximum allowable turbidity limit, if:

(1) The O/O fails to consult with the department within 24 hours of learning of the violation as required by Env-Dw 801.04(b); or

(2) After the required consultation, the department determines, based on the circumstances causing or contributing to the violation, that public notice is required to protect public health and safety;

(i) Any occurrence of a waterborne disease outbreak as defined in 40 CFR 141.2;

(j) Any occurrence of a waterborne emergency, including, but not limited to:

(1) A failure or significant interruption of key water treatment processes or distribution;

(2) A natural disaster that disrupts the water supply or distribution system; or

(3) A chemical spill or the unexpected introduction of possible pathogens or substances into the source water that significantly increases the potential for drinking water contamination; and

(k) Any other violation or situation that has significant potential to cause serious adverse effects on human health as a result of short-term exposure, that is:

(1) Identified in Env-Dw 700; or

(2) Determined by the department after consultation with the O/O to warrant public notice in order to protect public health and safety.

N.H. Admin R. Env-Dw 801.06 <u>Consultation for Acute Public Notice Violations or Situations</u>.

(a) The consultation between the department and the O/O required by Env-Dw 801.04(c)(2) shall be to determine:

(1) The degree of risk to public health from the violation or situation;

(2) Whether additional public notice will be required to be given by the O/O; and

(3) If additional public notice is required, the parameters for such notice, including timing, form, manner and frequency of distribution, and content.

(b) The determinations in (a), above, shall be based on:

(1) The nature of the specific violation or situation, including whether it exists throughout the entire PWS or is confined to only a portion of the PWS;

(2) When the violation or situation first occurred and how long it has been on-going;

(3) The severity of the violation or situation; and

(4) The potential health risk posed by the violation or situation

N.H. Admin R. Env-Dw 801.07 <u>Acute Public Notice; Methods of Delivery</u>.

(a) The O/O shall notify persons served by the PWS of any of the violations or situations listed in Env-Dw 801.05 within 24 hours of learning of the violation or situation.

(b) The O/O shall deliver the notice in a manner that is calculated to reach all persons served, by using at least one of the following forms of delivery:

(1) Broadcast media, such as radio and television, by furnishing a copy of the public notice for broadcast to radio or television stations, or both, that broadcast in the area served by the PWS;

(2) Written notice to all persons served by the PWS using one or more of the following methods:

a. If the area is served by a daily newspaper of general circulation, by publication in 3 consecutive issues of that newspaper;

b. By door-to-door hand delivery; or

c. For non-transient PWS, by posting the public notice in conspicuous locations throughout the area served by the PWS for as long as the violation persists or 7 days, whichever is longer; or

(3) Subject to (c) below, reverse 911 telephone service to all persons served by the PWS, provided:

a. Current phone numbers are known for all service connections; and

b. A receipt mechanism confirms that notice was received within 24 hours of transmittal.

(c) When reverse 911 is used but all current phone numbers are not known, one of the delivery methods specified in (b)(1) or (2), above, shall be used for each person for whom the number is not known.

(d) To supplement the delivery of notice by one or more methods listed in (b), above, the O/O may also distribute the public notice to persons served by the system using any of the methods specified below:

(1) Delivery of multiple copies for distribution by customers who provide the water to others, such as apartment building owners, schools, or large private employers;

(2) Posting on the internet; or

(3) Delivery of one or more copies to community organizations.

(e) Within 10 days of providing notice to each consumer, the O/O shall submit to the department the certification specified in Env-Dw 801.18.

(f) Public notices for the violations described in (a), above, shall be repeated every 3 months for as long as the violation or situation persists unless the O/O requests, and the department approves, a different frequency pursuant to Env-Dw 801.10.

N.H. Admin R. Env-Dw 801.08 <u>Standard Public Notice for Community Water Systems (CWS)</u>.

(a) The O/O of a community water system (CWS) shall provide notice, as required by Env-Dw 801.01, in accordance with (b), below, within 30 days of learning of any of the following violations or situations:

(1) Any violation of an applicable MCL, MRDL, treatment technique, monitoring, or testing procedure requirements specified in Env-Dw 700 that is not an acute violation specified in EnvDw 801.05;

(2) Any violation of the reporting or recordkeeping requirements specified in Env-Dw 709, EnvDw 720.14(b), or Env-Dw 720.16(b);

(3) Any violation of the turbidity MCL requirements established in 40 CFR 141.13(b) where the department determines, after consultation in accordance with Env-Dw 801.06, that the violation does not impose an acute health risk that warrants notification to the public within 24 hours;

(4) Any violation of a federal treatment technique relating to filtration and disinfection as incorporated by Env-Dw 716, as noted in Env-Dw 801.05(h), where the department determines, after consultation in accordance with Env-Dw

801.06, that the violation does not impose an acute health risk that warrants notification to the public within 24 hours;

(5) Failure to take corrective action or failure to maintain at least 4-log treatment of viruses before or at the first customer as specified in Env-Dw 717; or

(6) Failure to comply with ambient groundwater quality standards (AGQS) as required by EnvDw 707.02(b).

(b) The CWS O/O shall notify each customer receiving a bill and the O/O of any other service connection through which water is delivered to the public of any of the violations listed in (a), above, in such a manner that is calculated to reach all persons served by the CWS, by using at least one of the following forms of delivery:

(1) Mail delivery; or

(2) Door-to-door hand delivery.

(c) If the CWS O/O determines that not all persons served by the CWS were reached using the method chosen by the O/O from those described in (b), above, the O/O shall issue notice to persons served by the CWS using one of the following methods:

(1) Publication in a local newspaper or newsletter distributed to all persons served by the CWS;

(2) Delivery of multiple copies for distribution by customers that provide the water to others, such as apartments building owners, schools, or large private employers;

(3) Posting in public places served by the CWS;

(4) Posting on the internet or email broadcast to all persons served by the CWS; or

(5) Delivery of one or more copies to community organizations.

(d) A CWS O/O who chooses to use the method in (c)(3), above, shall post the public notice for as long as the violation persists or 7 days, whichever is longer.

(e) Within 10 days of providing notice to its customers, the CWS O/O shall submit to the department the certification specified in Env-Dw 801.18.

(f) Public notices for the violations described in (a), above, shall be repeated every 3 months for as long as the violation persists unless the CWS O/O requests, and the department approves, a different frequency pursuant to Env-Dw 801.10.

N.H. Admin R. Env-Dw 801.09 <u>Request for Extension of Standard Public Notice for CWS</u>.

(a) If a CWS O/O is unable to provide public notice within 30 days as required by Env-Dw 801.08(a), the O/O shall submit a request for an extension in writing to the department.

(b) A request for an extension shall:

(1) Be filed prior to the expiration of the 30-day notice period;

(2) Identify the CWS by name and PWS identifier;

(3) Explain why the O/O is unable to provide the required notice within the required time;

(4) Identify the length of the extension being requested; and

(5) Describe what alternative(s), if any, will be used by the O/O prior to providing the required notice to ensure that public health will be protected.

(c) The department shall respond to the extension request in writing. If the request is denied, the department shall specify the reason(s) for the denial.

(d) The department shall approve the extension request if it finds that the alternative(s) proposed will:

(1) Adequately protect human health and the environment; and

(2) Meet all applicable federal requirements.

(e) An extension granted by the department shall extend the time for providing the initial notice not more than 3 months from the date the water system learns of the violation.

(f) In no event shall an extension request be approved for any ongoing violations.

N.H. Admin R. Env-Dw 801.10 <u>Alternate Frequency for Repeat Standard Public Notice for CWS</u>.

(a) If a CWS O/O who is required to provide repeat notice under Env-Dw 801.08(f) wishes to provide repeat notice less frequently than once every 3 months, the O/O shall submit a request for a modification of the repeat notice frequency in writing to the department.

(b) A request for an alternate repeat notice frequency shall:

(1) Be filed prior to the expiration of the notice period required by Env-Dw 801.08(a) or extension thereof granted pursuant to Env-Dw 801.09;

(2) Identify the CWS by name and PWS identifier;

(3) Explain why the O/O is unable or unwilling to provide the required repeat notice every 3 months;

(4) Identify the frequency being requested for the repeat notices; and

(5) Explain how public health will be protected even though the frequency of repeat notices would be reduced.

(c) The department shall respond to the request for a modification of the repeat notice frequency in writing. If the request is denied, the department shall specify the reason(s) for the denial.

(d) The department shall approve the request if it finds that the proposed frequency will:

(1) Adequately protect human health and the environment; and

(2) Meet all applicable federal requirements.

(e) In no event shall repeat notice be given less frequently than once per year.

(f) In no event shall the department approve a less frequent repeat notice for an MCL or treatment technique violation under Env-Dw 709.23 or for violations of federal treatment techniques incorporated by Env-Dw 716, as noted in Env-Dw 801.05(h).

N.H. Admin R. Env-Dw 801.11 <u>Standard Public Notice for Non-Community Water Systems (NCWS)</u>.

(a) The O/O of a non-community water system (NCWS) shall provide notice, as required by Env-Dw 801.01, in accordance with (b), below, within 30 days of learning of any of the following violations or situations:

(1) A violation of an applicable MCL, MRDL, treatment technique, monitoring, or testing procedure requirements specified in Env-Dw 700 that is not an acute violation specified in EnvDw 801.05;

(2) Any violation of the reporting and recordkeeping requirements specified in Env-Dw 709, Env-Dw 720.14(b) or Env-Dw 720.16(b);

(3) Any violation of the turbidity MCL requirements established in 40 CFR 141.13(b) where the department determines, after consultation in accordance with Env-Dw 801.06, that the violation does not impose an acute health risk that warrants notification to the public within 24 hours;

(4) Any violation of a federal treatment technique relating to filtration and disinfection as incorporated by Env-Dw 716, as noted in Env-Dw 801.05(h), where the department determines, after consultation in accordance with Env-Dw 801.06, that the violation does not impose an acute health risk that warrants notification to the public within 24 hours;

(5) Failure to take corrective action or failure to maintain at least 4-log treatment of viruses before or at the first customer as specified in Env-Dw 717;

(6) Failure to comply with ambient groundwater quality standards (AGQS) as required by EnvDw 707.02(b); or

(7) Failure to complete seasonal start-up procedures and certification as required by Env-Dw 506.

(b) The NCWS O/O shall notify each customer receiving a bill and the owner of any other service connection through which water is delivered to the public of any of the violations listed in (a), above, in such a manner that is calculated to reach all persons served by the NCWS, by using at least one of the following forms of delivery:

(1) Mail delivery;

(2) Door-to-door hand delivery; or

(3) Posting the notice in conspicuous locations throughout the distribution system frequented by persons served by the system.

(c) If the NCWS O/O determines that persons served by the NCWS cannot be reached using the delivery methods specified in (a), above, the O/O shall issue notice to persons served by the NCWS using one of the following methods:

(1) Publication in a local newspaper or newsletter distributed to persons served by the NCWS;

(2) Delivery of multiple copies for distribution by customers that provide the water to others, such as apartment building owners, schools, or large private employers;

(3) Posting on the internet or email broadcast to all persons served by the system; or

(4) Delivery of one or more copies to community organizations.

(d) Within 10 days of providing notice to its customers, the NCWS O/O shall submit to the department the certification specified in Env-Dw 801.18.

(e) If the NCWS O/O elects to provide public notice by posting the notice, posting shall continue for as long as the violation persists or 7 days, whichever is longer.

(f) Subject to (g), below, the NCWS O/O shall repeat the public notice for the standard violations described in (a), above, every 3 months for as long as the violation persists unless the NCWS owner requests, and the department approves, a different frequency pursuant to Env-Dw 801.13.

(g) If a monitoring violation occurs at a seasonal system that will not be in operation for at least 3 months following the issuance of the public notice, the NCWS O/O shall also post the notice upon the reopening of the system the following season. If such additional posting is required, the notice shall remain posted for 7 days or as long as the violation persists, whichever is longer.

N.H. Admin R. Env-Dw 801.17 <u>Department Action When O/O Fails to Notify</u>.

(a) The department shall issue public notice on behalf of the O/O if the department determines that the O/O failed to issue public notice as required by this part and:

(1) The failure to issue public notice may pose a risk to human health as identified in Env-Dw 700; or

(2) The O/O fails to recognize or acknowledge any other violation of Env-Dw 700.

(b) Even if the department provides notice pursuant to (a), above, the O/O shall remain subject to enforcement under RSA 485:58 for failing to provide public notice and for any other violations of this part.

(c) The department shall issue a public notice violation if the O/O fails to comply with the timing, content, delivery, or certification requirements of this part.

N.H. Admin R. Env-Dw 808.27 Perfluorohexane Sulfonic Acid (PFHxS).

For perfluorohexane sulfonic acid (PFHxS) violations, the statement shall read as follows:

"Some people who drink water containing perfluorohexane sulfonic acid (PFHxS) in excess of the MCL over many years could experience problems with their liver, endocrine system, or immune system, or may experience increased cholesterol levels. It may also lower a woman's chance of getting pregnant."

N.H. Admin R. Env-Dw 808.28 Perfluorononanoic Acid (PFNA).

For perfluorononanoic acid (PFNA) violations, the statement shall read as follows:

"Some people who drink water containing perfluorononanoic acid (PFNA) in excess of the MCL over many years could experience problems with their liver, endocrine system, or immune system, or may experience increased cholesterol levels."

N.H. Admin R. Env-Dw 808.29 Perfluorooctane Sulfonic Acid (PFOS).

For perfluorooctane sulfonic acid (PFOS) violations, the statement shall read as follows:

"Some people who drink water containing perfluorooctane sulfonic acid (PFOS) in excess of the MCL over many years could experience problems with their liver, endocrine system, or immune system, may experience increased cholesterol levels, and may have an increased risk of getting certain types of cancer. It may also lower a woman's chance of getting pregnant."

N.H. Admin R. Env-Dw 808.30 Perfluorooctanoic Acid (PFOA).

For perfluorooctanoic acid (PFOA) violations, the statement shall read as follows:

"Some people who drink water containing perfluorooctanoic acid (PFOA) in excess of the MCL over many years could experience problems with their liver, endocrine system, or immune system, may experience increased cholesterol levels, and may have an increased risk of getting certain types of cancer. It may also lower a woman's chance of getting pregnant."

N.H. Admin R. Env-Dw 811.02 Definitions.

For purposes of this part, the following definitions shall apply unless otherwise specified:

(a) "Action level (AL)" means the concentration of a contaminant which, if exceeded, triggers treatment or other requirements which a water system must follow;

(b) "Consumer confidence report (CCR)" means an annual report supplied by a CWS O/O to customers which contains information on the quality of their drinking water;

(c) "Customers" means billing units or service connections to which water is delivered by a CWS;

(d) "Detected" means the presence of any primary or secondary drinking water contaminant including:

(1) Microbiological contaminants;

(2) Radiological contaminants;

(3) IOC contaminants;

(4) VOC contaminants;

(5) SOC contaminants;

(6) PFAS contaminants; and

(6) Disinfection by-products;

(e) "Regulated contaminant" means a contaminant that is subject to a maximum contaminant level (MCL), action level (AL), maximum residual disinfectant level (MRDL), or treatment technique (TT); and

(f) "Unregulated contaminant" means a contaminant specified in 40 CFR 141.40.

N.H. Admin R. Env-Dw 811.03 Timing and Certification of Distribution.

(a) No later than July 1 of each year, each CWS O/O shall provide a copy of the CCR to each customer and the department as specified in Env-Dw 811.24.

(b) By July 10 of each year, the CWS O/O shall certify to the department that:

(1) The CCR has been distributed to customers; and

(2) The information in the CCR is correct and consistent with the compliance monitoring data previously submitted to the department.

(c) The O/O of a new CWS shall file the first CCR by July 1 of the year after its first full calendar year in operation and annually thereafter.

(d) A CWS O/O who sells water to another CWS shall provide the buyer with applicable information required in this part to the receiving system:

(1) No later than April 1 of each year; or

(2) On a date mutually agreed upon by the seller and the purchaser, and specifically included in a contract between the parties.

N.H. Admin R. Env-Dw 811.09 Detected Contaminants.

(a) The CWS O/O shall include data in the manner specified in Env-Dw 811.11 if any of the following are detected in the water provided by the CWS:

(1) Regulated contaminant(s) as defined in Env-Dw 811.02(e);

(2) Unregulated contaminant(s) as regulated under 40 CFR 141.40;

(3) Sodium, if monitoring is required per Env-Dw 713.08; or

(4) Disinfection by-products or microbial contaminants other than cryptosporidium for which monitoring is required by Env-Dw 712.17 and that are detected in the finished water.

(b) The CCR shall identify the probable source of the contaminant. If the CWS O/O lacks specific information on the likely source(s) of contamination, the O/O shall use the language specified in Env-Dw 811.22.