THE STATE OF NEW HAMPSHIRE

MERRIMACK, SS

SUPERIOR COURT
DOCKET NO.
217-2019-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

COMPLAINT FOR DECLARATORY RELIEF AND FOR TEMPORARY, PRELIMINARY AND PERMANENT INJUNCTIVE RELIEF

The Plymouth Village Water & Sewer District, Resource Management, Inc., Charles G. Hanson, and 3M Company (collectively "Plaintiffs") file this Complaint for Declaratory Relief, and Temporary, Preliminary, and Permanent Injunctive Relief against the New Hampshire Department of Environmental Services ("NHDES" or the "Agency") to declare invalid and to enjoin enforcement of recently adopted rules pertaining to permitted levels of PFOA, PFOS, PFNA, and PFHxS scheduled to become effective on September 30, 2019.[1] In support of this Complaint, Plaintiffs state as follows:

[1] NHDES initially advised the public the new PFAS Rules were to become effective October 1, 2019. June 28, 2019 NHDES Press Release. "If approved by the JLCAR, the new rules are scheduled to become effective on October 1, 2019."

INTRODUCTION

1. This Complaint seeks a declaration that: (a) NHDES's adoption and impending enforcement of the following N.H. Code Admin Rules, NHDES PFOA, PFOS, PFNA, and PFHxS Rules, Exhibit 1, which encompasses specified provisions of Env-Dw, Env-Wq and Env-Or (together the "Final Rules") violate the State's constitutional and statutory prohibitions against legislative and agency impositions of unfunded mandates on political subdivisions under the New Hampshire Constitution, Part I, Art. 28-a, and RSA 541-A:25, and ignore NHDES Rule Env-Dw 102.01, which exempted cities and towns from other water quality rules on such grounds; (b) NHDES violated the Administrative Procedure Act ("APA"), RSA Ch. 541-A by failing to provide a renewed notice and comment period when it dramatically lowered the maximum contaminant levels ("MCLs") and Ambient Groundwater Quality Standards ("AGQS") in its final rule without prior notice and opportunity for public comment, (c) NHDES failed to fulfill its obligations under the New Hampshire Safe Drinking Water Act, RSA 485:3, I (b) to undertake an analysis of "the costs and benefits to the affected parties that will result from establishing the standard" for the PFOA, PFOS, PFNA, and PFHxS Rules; and (d) NHDES deprived citizens of property without consent or legislative authorization, in violation of the New Hampshire Constitution, Part 1, Art. 12 and Art. 15, and the due process clause of the Fourteenth Amendment to the United States Constitution.

2. Plaintiffs further seek to enjoin NHDES from implementing and enforcing the PFOA, PFOS, PFNA, and PFHxS Rules until NHDES promulgates new rules in compliance with all regulatory, statutory, and constitutional requirements.

PARTIES

3. Plaintiff Plymouth Village Water & Sewer District ("Plymouth Water District") is a village district and a political subdivision of the Town of Plymouth, New Hampshire, with a

principal place of business at 27 Old N Main St., Plymouth, NH 03264. Plaintiff Plymouth Water District will be subject to the new rules, and required to test for, and if necessary, remediate, PFOA, PFOS, PFNA, and PFHxS, if the rules are not enjoined.

4. Plaintiff Resource Management, Inc. ("RMI") is a New Hampshire corporation with a principal place of business at 1171 NH Rte. 175, Holderness, NH 03245. Plaintiff RMI provides, among other services, biosolids management for governments and private businesses. Plaintiff RMI will be subject to the new rules, and may be required to test for, and if necessary, remediate, PFOA, PFOS, PFNA and PFHxS, if the rules are not enjoined.

5. Plaintiff Charles G. Hanson ("Hanson") is a taxpayer and an individual who owns and operates a farm on 121 Dane Road, Center Harbor, New Hampshire 03226. Plaintiff Hanson will be subject to the new rules, and may be required to test for, and if necessary, remediate, PFOA, PFOS, PFNA and PFHxS, if the rules are not enjoined. Plaintiff Hanson also is an employee and principal of RMI.

6. Plaintiff 3M Company ("3M") is a Delaware corporation with a principal place of business at 3M Center, St. Paul, Minnesota 55133, and operates a facility having a community water system located at 11 Paper Trail, Tilton, New Hampshire 03276 which is subject to the Final Rules. On May 31, 2019, just weeks prior to the issuance of the final PFOA, PFOS, PFNA, and PFHxS Rules, the State of New Hampshire commenced two actions against 3M[2] alleging damages to, *inter alia*, surface water and ground water from PFAS. The damages the

[2] *State of New Hampshire v. 3M Company, et al.*, Hillsborough County Superior Court, North Division, 216-2019-CV-0445; *State of New Hampshire v. 3M Company, et al*., United States District Court, District of New Hampshire, 19-cv-0800 which was then transferred to Multi-District Litigation case pending in the United States District Court for the District of South Carolina, *In re: Aqueous Film-Forming Foam Products Liability Litigation*, transferred to that district as part of Multi-District Litigation involving Aqueous Film-Forming Products, MDL No. 2873.

State seeks from 3M include the costs of all PFAS testing and remediation incurred by all municipalities in New Hampshire.

7. Defendant Robert R. Scott is the Commissioner of the New Hampshire Department of Environmental Services, an agency of the State of New of Hampshire with an address of 29 Hazen Drive, P.O. Box 95 Concord, NH 03302.

JURISDICTION AND VENUE

8. This is an action to contest the validity of administrative rules for noncompliance with provisions of RSA Ch. 541-A, the New Hampshire Constitution and RSA Ch. 485. Pursuant to NH RSA 541-A:23, jurisdiction and venue lie in the Merrimack County Superior Court. This Court also has jurisdiction pursuant to RSA 541-A:24 and RSA 491:22. *Asmussen v. Comm'r, New Hampshire Dep't of Safety*, 145 N.H. 578, 585–86 (2000).

FACTS PERTINENT TO THE CLAIMS

9. Per and polyfluoralkyl substances ("PFAS") refers to a huge group of chemicals with widely varied properties. Among other uses, PFAS substances have been used for their water and stain repellency, resistance to high temperatures, and to reduce surface tensions. At issue in this suit are the four PFAS compounds for which NHDES has promulgated regulations: PFOA, PFOS, PFNA, and PFHxS.

10. On July 10, 2018, the Governor signed SB 309 which became 2018 Chapter Law 368. This law directed NHDES to commence rulemaking by January 1, 2019 to establish drinking water standards that would apply to all public water supplies called maximum contaminant levels (MCLs), and ambient groundwater quality standards (AGQS) for PFOA, PFOS, PFNA, and PFHxS. RSA 485:16-e, RSA 485-C:6, V, VI.

11. The legislature set no deadline for completion of the rulemaking process mandated by 2018 Chapter Law 368.

12. On January 4, 2019, NHDES proposed numeric MCLs and AGQS for PFOA, PFOS, PFNA, and PFHxS ("Proposed Rules"), and set a schedule for public hearing and comment.

13. On June 28, 2019, NHDES's final rulemaking ("Final Rules") set substantially lower MCLs and AGQS:

	PFOA	**PFOS**	**PFHxS**	**PFNA**
Proposed Rules– January, 2019	38 ppt	70 ppt	85 ppt	23 ppt
Final Rules– June, 2019	12 ppt	15 ppt	18 ppt	11 ppt

14. The Final Rules NHDES issued in June, 2019 used a significantly different toxicity study for PFOS, and used significantly different critical end points and exposure modeling approaches from those proposed in January.

15. NHDES never offered the public an opportunity to comment on these critical scientific changes or their cost implications.

16. When adopting new rules establishing MCLs and/or AGQS, NHDES is required not only to analyze the science, but also to consider the costs and benefits to all affected parties that will result from establishing the standard.

17. NHDES did not give the public the opportunity to comment upon the Technical Background Report for the Proposed Maximum Contaminant Levels and Ambient Groundwater Quality Standards for PFOA, PFOS, PFNA, and PFHxS that it developed, and used to support its June 2019 numeric MCL and AGQS standards. https://www4.des.state.nh.us/nh-pfas-investigation/?p=1063

18. NHDES never gave the public the opportunity to comment on the costs and benefits of the Final Rules for the four substances issued in June 2018. NHDES Summary of Comments on Initial Proposals with NHDES Responses June 28 2019, Exhibit 2, at 86.

19. NHDES did not base the Final Rules on an adequate assessment of the costs and benefits to all affected parties of adopting the lower MCLs and AGQSs for PFOA, PFOS, PFNA, and PFHxS. For example, based on changes between the January and June proposals, the annual Operation and Maintenance ("O&M") costs for public water systems are estimated to increase by up to 60 times, and initial treatment costs for public water systems are estimated to increase by approximately 30 times the original proposal. NHDES did not calculate the benefits to be gained by such increased costs.

20. Addressing the new Final Rules, Robert R. Scott, Commissioner of NHDES explained, "[t]he work . . . is expected to require substantive upgrades for facilities that exceed the new MCLs . . . at the time of filing the rule for approval, [it] was estimated to cost [public water systems] at least $190 million over the next two years."[3]

21. The magnitude of costs Plaintiffs and other public and private entities will incur were not addressed in the Commissioner's comments.

22. NHDES failed to evaluate whether the significantly lower limits for MCLs and AGQS in the PFOA, PFOS, PFNA, and PFHxS Rules achieved equivalent or more benefits than the costs required. NHDES failed to evaluate the value of any benefits achieved beyond the originally proposed limits.

[3] NHDES Commissioner's Column, Final PFAS Drinking Water Standards Established, September-October 2019, Exhibit 3.

23. The Final Rules and their application interfere with or impair, or threaten to interfere with or impair, the legal rights or privileges of the Plaintiffs.

24. 2018 Chapter Law 368 expressly required compliance with the New Hampshire APA, RSA Ch. 541-A.

25. The New Hampshire Safe Drinking Water Act provides:

> The commissioner shall adopt under RSA 541-A . . . drinking water rules . . . 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: …

RSA 485:3, I (emphasis added).

COMMENCEMENT OF RULEMAKING

THE FIRST PRESS RELEASE

26. On December 31, 2018 NHDES commenced the PFOA, PFOS, PFNA, and PFHxS rulemaking process. On January 2, 2019, the agency posted a press release on its website[4] (the "First Press Release") advising the public that it had initiated rulemaking to establish the PFOA, PFOS, PFNA, and PFHxS MCLs and AGQS as well as the locations of upcoming public hearings to be held beginning in March of 2019. NHDES First Press Release, January 2, 2019, Exhibit 4.

27. The First Press Release included a statement that, "using the most recent and best science available," it was proposing drinking water standards "that are protective of the most

[4] The link to the relevant portion of the website is: https://www4.des.state.nh.us/nh-pfas-investigation/?p=918

sensitive populations over a lifetime." The proposed standards published in the First Press Release were:

PFOA	38 ppt (parts per trillion)
PFOS	70 ppt
PFOA & PFOS (combined)	70 ppt
PFHxS	85 ppt
PFNA	23 ppt

28. The proposed Rules were intended to replace action taken by the agency in 2016 when it used emergency rulemaking powers to incorporate the Environmental Protection Agency's ("EPA") health advisory for two PFAS substances (PFOA and PFOS) as an AGQS at 70 ppt combined. May 31, 2016 NHDES Press Release, *NHDES Establishes Ambient Groundwater Quality Standard for {PFOA and PFOS}*.

THE SUMMARY REPORT

29. Two days after the First Press Release, on January 4, 2019, DES published a "Summary Report" on its website addressing Maximum Contaminants Levels and Ambient Water Quality Standards for . . . [PFOS, PFOA, PFNA, and PFHxS]. NHDES Summary Report, January 4, 2019, Exhibit 5.

30. The Summary Report is 86 pages long, including eleven (11) appendices.

31. The Summary Report purports to analyze, *inter alia*, costs and benefits of the MCL and AGQS numeric standards set out in the First Press Release. Table 1 of the Summary Report provides an overview of the proposed derived standards and the factors selected to derive

the proposed MCLs and AGQSs:

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)

NHDES Summary Report, January 4, 2019, Exhibit 5, at 7.

32. The Summary Report contains sections setting out, *inter alia*, "Costs to Affected Parties" and "Benefits to Affected Parties." NHDES Summary Report, January 4, 2019, Exhibit 5, at 11, 16.

33. Costs were calculated based upon the then NHDES proposed standards of 38 ppt, 70 ppt, 85 ppt and 23 ppt.

34. NHDES did not publish or otherwise make available to the public an amendment or revision to the Summary Report, taking account of new or revised numeric standards during the mandated comment period.

35. In the Summary Report, NHDES acknowledged that the analyses were keyed to the then-existing draft MCLs and AGQS (38 ppt, 70 ppt, 85 ppt and 23 ppt). NHDES also acknowledged that, even at the original proposed MCLs and AGQSs, it did not have the resources or expertise necessary to undertake a comprehensive analysis of costs or benefits, and that the analysis it made available for comments did not contemplate any different standards that might be adopted, rendering the Summary Report inapplicable to the final numeric values in the Final Rules:

> 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**.

NHDES Summary Report, January 4, 2019, Exhibit 5, at 11.

THE SECOND PRESS RELEASE

36. On February 21, 2019, seven (7) weeks after its initial notice, and only (7) seven business days before the required public hearings were to commence, NHDES issued a second press release[5] (the "Second Press Release"), notifying the public for the first time that "new scientific information was evaluated by NHDES that may change the proposed drinking water standards [pertaining to PFOA, PFOS, PFNA and PFHxS]," and that "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." (emphasis added). NHDES Second Press Release, February 21, 2019, Exhibit 6.

37. The Second Press Release had the effect of "moving the goalpost" for the purposes of public hearing and comment.

38. The Second Press Release did not identify the "new scientific information" other than by vague reference to "a new assessment tool developed by the Minnesota Department of Health." NHDES did not post this assessment tool, nor provide a link to it in the Second Press Release.

39. The Second Press Release did not advise the public what the potentially "significantly" lowered new standards were or would be.

40. The Second Press Release did not provide the public with a definition or indication of what was meant by use of the word "significantly."

41. NHDES fundamentally changed the scientific underpinnings of its analysis. Although NHDES purported to invite comment on this self-described significant change in rulemaking, in reality its invitation was a meaningless gesture. NHDES did not provide any

[5] New Information May Change NHDES Proposed PFAS Drinking Water Standards, NHDES PFAS Investigation website February 21, 2019, https://www4.des.state.nh.us/nh-pfas-investigation/?p=945.

information about what change it was considering, or how it would affect the level at which NHDES intended to regulate. The changes to the methodology NHDES used were so unclear that it was not possible to provide meaningful public input as required by RSA 541-A.

42. In the Second Press Release, NHDES acknowledged that it would "need to complete a review of the **technical and cost implications** of these health-based calculations, and any public comment received, prior to issuance of the Final Proposal." (emphasis added).

PUBLIC NOTICE AND HEARING

43. NHDES is required to provide at least 20 days' notice of the public hearing on proposed rules. RSA 541-A:6, I.

44. NHDES did not alter the schedule for Public Hearings subsequent to the February 21, 2019 Second Press Release. The required public hearings on the Proposed Rules commenced just seven (7) business days after the Second Press Release, dates unchanged from the First Press Release on January 2, 2019, and in violation of RSA 541-A:6, I.

45. NHDES never made its revised standards available to the public during the public hearing and comment period.

FINAL PROPOSED MCLs AND AGQSs

46. In its June 28, 2019 Final Proposed Rulemaking NHDES established the following MCLs and AGQSs:[6]

[6] *See* https://www4.des.state.nh.us/nh-pfas-investigation.

PFAS Final Proposed MCL and AGQS

PFOA	12 ppt
PFOS	15 ppt
PFHxS	18 ppt
PFNA	11 ppt

47. The first time NHDES provided notice of the revised numeric MCLs and AGQS was the same day it issued its Final Proposed Rulemaking. Plaintiffs and the interested and affected public had no notice of the numeric MCLs and AGQS such that they could have provided public comment.

48. To date, NHDES still has not provided an opportunity for public comment on the final MCL and AGQS standards in the Final Rules.

49. The proposed final MCLs and AGQS ranged from two (2) times to nearly five (5) times lower than the numeric standards initially disclosed for comment by NHDES more than six months earlier.

50. NHDES' purported cost benefit analysis did not identify, let alone compare, the benefits to be achieved for the costs to be incurred by the affected parties by so substantially reducing the MCL and AGQS standards from the January levels to the June levels.

51. No critique or analysis of the "new assessment tool developed by the Minnesota Department of Health" was made available by NHDES for public comment.

52. NHDES likewise did not explain how it was using the new model to revise its original proposed regulation.

53. Upon information and belief, the agency simply accepted the Minnesota assessment tool as valid, without independent inquiry.

54. During the comment period, NHDES did not identify technical, economic or other peer-reviewed science regarding potential benefits associated with the specific reduction in MCLs and AGQSs NHDES ultimately adopted.

JLCAR HEARING

55. RSA 541-A:13 provides that, before a rule becomes final, the General Court's Joint Legislative Committee on Administrative Rules ("JLCAR") reviews and then may comment on, object to, conditionally approve, or approve the rule.

56. Just fourteen business days after the "Proposed Final" MCLs and AGQS were published, JLCAR held a public hearing and approved the Final Rules. 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. JLCAR ignored specific requests by the public to defer the decision or release the revised proposal for public comment.

57. The Final Rules were initially noticed to become effective on October 1, 2019. NHDES changed that date, without notice, after the JLCAR approved the rules. The Final Rules are now set to be effective on September 30, 2019, requiring compliance sooner than previously announced.

NHDES RISK ASSESMENT

58. Had public comment on the Final NHDES decision been allowed, numerous detailed comments on the risks considered would have been provided, and NHDES would have been required to consider them. For example:

a) The risk analysis used to develop the MCLs and AGQS is based on non-cancer endpoints.[7]

[7] *See* NHDES July 9, 2019 Summary of the Technical Background Report for the Proposed Maximum Contaminant Levels and Ambient Groundwater Quality Standards for PFOA, PFOS, PFNA and PFHxS, Exhibit 7, at 6.

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 Perflouroalkyls, Draft for Public Comment, ATSDR 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.

NHDES ASSESMENT OF COSTS AND BENEFITS TO AFFECTED PARTIES

59. Had public comment on the Final NHDES decision 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, I (b).

b) NHDES' June 28, 2019 Update 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.[8]

60. 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.

61. NHDES did not conduct any of its own economic analysis at all regarding the cost and benefits of the proposed MCLs and AGQSs at the specific levels proposed.

62. The purported benefits of the Final Rules are grounded largely on a non-peer reviewed paper published by the Nordic Council of Ministers that NHDES acknowledged simply assumes the existence of unproven health effects.

63. The NHDES report largely fails to address any costs or benefits for private parties other than an estimate of $70 million for testing of private wells, which it notes need not be tested or treated unless the homeowner chooses to do so.

64. The NHDES ignored the enormous potential costs to private businesses and individuals - affected parties - throughout New Hampshire, including affected parties such as Plaintiffs.

65. The NHDES consideration of costs is an incoherent amalgam of objective data, and subjective, "qualitative" information.

[8] The detail required by EPA necessitates the involvement of economists given the importance and complexity of the process. For example, "[t]he height of the demand curve at a quantity Q d-1 gives the marginal WTP [willingness to pay] for the Qd-1th unit. The height of the demand curve at a quantity Qd gives the marginal WTP for the Qdth unit. Note that the marginal WTP is greater for the Q d-1th unit." EPA Guidelines for Preparing Economic Analyses.

66. NHDES admitted in response to public comments it did not perform a thorough and adequate cost-benefit analysis because it lacked needed data to provide a quantitative report. NHDES explained:

> 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 <u>needed data</u> 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 procedure. The office of the Attorney General found NHDES's interpretation of the requirement under RSA 485:3, I (b) to be reasonable and lawful…

NHDES Summary of Comments on Initial Proposals with NHDES Responses, June 28, 2019, <u>Exhibit 2</u> (emphasis added).

67. The New Hampshire Department of Justice has refused to produce its underlying analysis of the issue, citing privilege.

68. The costs and benefits of the June 2019 Final Rules are conclusory and result oriented:

> 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.

NHDES Summary of Comments on Initial Proposals with NHDES Responses, June 28, 2019, <u>Exhibit 2</u> (emphasis added).

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) and if, over the next four quarters, samplings exceed the MCLs, they will be required to develop an action plan for

achieving compliance with the standards, which is likely to include costly treatment to remove those chemicals to near zero. This new mandate is not in budgets, is an unknown number, and is precisely the sort of economic impact on public entities that the legislature has sought to prevent. All taxpayers that operate a public water system, wastewater treatment plant or landfill, or who are otherwise obligated by permit to test groundwater quality, will be subject to the rules and the related costs.

70. Numerous other entities, including New Hampshire's businesses and private individuals, face potentially huge costs to comply with the Final Rules with no clear delineation of the incremental benefit of using a final MCL versus the originally proposed standard.

COUNT ONE – DECLARATORY JUDGMENT, TEMPORARY, PRELIMINARY AND PERMANENT INJUNCTIVE RELIEF

71. Plaintiffs incorporate the allegations of paragraphs 1 through 70 of this Complaint.

72. Part I, Art. 28-a of the New Hampshire Constitution prohibits the State from mandating any new, modified or expanded programs or responsibilities in such a way as to necessitate local expenditures unless they are fully funded by the State or the local political subdivision approves by a vote of the local legislative body.

73. Part I, Art. 12 and Art. 15 of the New Hampshire Constitution prohibit the State from depriving citizens of property without consent, or constitutional, legislative authorization.

74. RSA 541-A: 25, the APA, incorporates the prohibitions of the New Hampshire Constitution Part I Art 28-a and is more specific and more expansive in its coverage.

75. The APA expressly prohibits the adoption of agency rules that 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 municipalities unless approved by a vote of the

local legislative body. RSA 541-A: 25 expressly provides the prohibition covers "programs . . . of a nature customarily performed by municipalities," whether or not required by statute, and expressly includes "solid waste, sewer and water" in the programs covered. The APA expressly enumerates "solid waste, sewer and water" among the programs for which new or expanded programs or responsibilities may not be expanded. RSA 541-A:25, II.

76. The Final Rules require local expenditures and constitute a new, expanded or modified program or responsibility.

77. The mandate to test and if necessary, remediate as set forth in the Final Rules is not funded at all by the State, much less fully funded.

78. N.H. Code Admin. Rules Env-Dw 102.01 exempts municipalities from certain requirements pursuant to Part 1, Article 28-a for water systems owned by political subdivisions.

79. Few if any municipalities have approved the increased expenditures imposed by the Final Rules by a local vote.

80. The General Court's adoption of 2018 Chapter Law 368 violates Article 28-a because it mandates NHDES adopt MCLs and AGQS requiring municipalities to incur enormous local expenditures to comply with the standards without funding.

81. NHDES's adoption, and impending implementation and enforcement, of the Final Rules violate RSA 541-A:25 and Admin R. Env-Dw 102.01 by requiring municipalities to incur local expenditures to comply with the standards without funding. The rule provides that water systems may not be burdened with extra expenditures arising from the adoption of new and stricter state water quality standards. NH Code Admin. Rule Env-Dw 102.01 stipulates a list of water quality rules that are not applicable to municipalities unless compliance is funded by the State.

82. The Final Rules violate Part 1, Art. 28-a of the New Hampshire Constitution because the requirements mandated by 2018 Chapter Law 368 constitute a new, expanded or modified program or responsibility necessitating enormous local expenditures that are not fully funded by the State.

83. The Final Rules violate RSA 541-A:25 because they mandate a new, expanded or modified program or responsibility to cities and towns by requiring them to expend funds testing for and remediating alleged PFOA, PFOS, PFNA, and PFHxS above the relevant MCLs and AGQSs without providing funding for those expenditures.

84. NHDES asserts in its Summary of Comments to Initial Proposals with NHDES Responses dated June 28, 2019 that its issuance of the Final Rules does not violate the Supreme Court's analysis of N.H. Const. Part I, Art. 28-a. *City of Concord v. State of New Hampshire,* 164 N.H. 130 (2012).

85. NHDES reads *City of Concord* too expansively, ignores previous Supreme Court precedent, and disregards the fact that RSA 541-A:25 was not construed at all. To interpret the case as NHDES does effectively repeals Art. 28-a and RSA 541-A:25.

86. The APA empowers the Court to fashion an appropriate remedy because the Final Rules violate RSA 541-A:25. RSA 541-A:23, III ("For other violations of this chapter, the court may fashion appropriate relief.").

87. Where a statute violates the NH Constitution and an express provision of law, an appropriate remedy is temporary, preliminary and permanent injunctive relief. Plaintiffs request that judgment be entered declaring the Final Rules invalid because they were promulgated in violation of the APA, RSA 541-A, the New Hampshire Safe Drinking Water Act, RSA 485, constitute an unfunded mandate in violation of Part I, Article 28-a of the New Hampshire

Constitution, and an impermissible deprivation of property without due process of law, consent or legislative authorization in violation of Part I, Article 12 and Article 15 of the New Hampshire Constitution and Section 1 to the Fourteenth Amendment to the United States Constitution.

88. Plaintiffs further request the Court temporarily, preliminarily and permanently enjoin the NHDES from enforcing the Final Rules.

COUNT TWO – VIOLATION OF STATUTORY AND CONSTITUIONAL DUE PROCESS

89. Plaintiffs incorporate the allegations of paragraphs 1 through 88 of this Complaint.

90. The APA sets forth procedures which must be followed to adopt administrative rules. RSA 541-A:5–14.

91. RSA 541-A:6 requires at least 20 days' notice of an agency's intent to hold a public hearing.

92. RSA 541-A:11 requires reasonable public notice, public hearings and public comment.

93. NHDES gave notice of intended rulemaking in early January, 2019 and established a public hearing schedule commencing on March 4, 2019, approximately 2 months after public notice.

94. NHDES announced for the first time that it would be considering "new scientific information" that would "significantly" impact the Proposed Rules on February 21, 2019, seven (7) business days before the first public hearing on March 4.

95. The public was given only seven (7) business days to try to locate, digest, analyze, and critique this new, complex scientific information before the first public hearing.

96. Despite knowing it intended to revise its proposal "significantly" downward based on undisclosed information being used by NHDES and an undisclosed way, NHDES failed to alter the Public Hearing schedule to provide the meaningful opportunity for public input and comments that the APA is meant to preserve.

97. NHDES violated Plaintiffs' due process rights inherent in RSA 541-A by announcing a "significant" and substantive change in the underlying scientific principles guiding analysis of the Proposed Rules, without providing any change in the public hearing schedule so as to allow for considered and meaningful scientific comment, without providing new numeric limits for public comment, and without conducting a proper cost-benefit analysis required by law.

98. In enacting the Final Rules, NHDES failed to follow the proper statutory procedure. NHDES enacted the Final Rules *ultra vires*.

99. For these reasons, the Final Rules were enacted in violation of Plaintiffs' due process rights under the APA, RSA 541-A; the New Hampshire Safe Drinking Water Act, RSA 485; the New Hampshire Constitution, Part 1, Arts. 12 , 15 and 28-a; and, the United States Constitution, U.S. CONST. Art. XIV.

100. Plaintiffs request the Court temporarily, preliminarily and permanently enjoin the NHDES from enforcing the Final Rules.

COUNT THREE – VIOLATION OF THE NEW HAMPSHIRE SAFE DRINKING WATER ACT, RSA 485

101. Plaintiffs incorporate the allegations of paragraphs 1 through 100 of this Complaint.

102. NHDES was directed by the legislature to undertake rulemaking to establish drinking water standards for PFOA, PFOS, PFNA and PFHxS that would apply to all public water supplies.

103. The statutory due process requirements for establishing new MCLs and AGQS are set forth in RSA 485. RSA 485:3, I requires that rulemaking conform to the requirements of the APA, RSA 541-A.

104. An economic analysis of costs and benefits of new MCLs and AGQS on all affected parties is required prior to the establishment of new standards.

> 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 . . . .

RSA 485:3, I (b) (emphasis added).

105. NHDES failed to comply with the scientific and economic requirements of the statute.

106. The scientific and economic analysis undertaken by NHDES to establish the Final Rules, as described in the Summary of the Technical Background Report for the Proposed Maximum Contaminant Levels and Ambient Groundwater Quality Standards for PFOA, PFOS, PFNA and PFHxS, is woefully deficient.

107. In violation of the statutory requirement of RSA 485:3 I (b), NHDES did not conduct an economically valid or appropriate consideration and evaluation of the costs and benefits of the new MCLs and AGQS.

108. The procedure by which the Final Rules were adopted violates the requirements of RSA 485:3 and RSA 541-A, and the Final Rules are therefore invalid.

109. RSA 541-A:23 and RSA 541-A:24 empower the court to enter an appropriate remedy if an agency violates the APA in the promulgation of rules.

110. Plaintiffs request that the Court temporarily, preliminarily and permanently enjoin NHDES from enforcing the Final Rules.

COUNT FOUR – VIOLATION OF CONSTITUTIONAL DUE PROCESS

111. Plaintiffs incorporate the allegations of paragraphs 1 through 110 of this Complaint.

112. The New Hampshire Constitution due process clause resides in Part I, Article 15. It states, in pertinent part:

> . . . No subject shall be arrested, imprisoned, despoiled, or deprived of his property, immunities, or privileges, put out of the protection of the law, exiled or deprived of his life, liberty, or estate, but by the judgment of his peers, or the law of the land; . . .

Part I, art. 15, N.H. Const. "[T]he law of the land" is synonymous with "due process of law." *Bragg v. Dir., New Hampshire Div. of Motor Vehicles*, 141 N.H. 677, 678 (1997) (quoting Petition of Bagley, 128 N.H. 275, 282 (1986)).

113. Part 1, Art. 12 of the New Hampshire Constitution prohibits the State from depriving citizens of property without consent, or constitutional, legislative authorization.

114. U.S. Const. amend. XIV, § 1 guarantees ". . . nor shall any State deprive any person of life, liberty, or property, without due process of law."

115. In New Hampshire, "[t]he fundamental requisite of due process is the right to be heard at a meaningful time and in a meaningful manner." *Petition of Bagley*, 128 N.H. 275, 282–83 (1986).

116. The rulemaking process undertaken by NHDES denied Plaintiffs the right to be heard by unreasonably shortening the public comment period, failing to adequately explain all of

the considerations underlying the Second Press Release, failing to comply with the cost benefit analysis required by RSA 485:3 and RSA 541-A:5, and publishing its proposed final MCLs and AGQSs without allowing any opportunity for comment prior to presenting its rules for approval to JLCAR, and then changing the effective date of the rules from the date originally published.

117. The failure by NHDES to follow the proper statutory procedure, and the failure to adequately allow comment prior to the adoption of the "significantly" changed, Final Rules, threatens to, and will, if the rules go into effect, or are enforced, materially deprive Plaintiffs of their liberty and property, and violates both the substantive and procedural due process protections of the New Hampshire and United States Constitutions.

118. If the *ultra vires* Final Rules go into effect, or are enforced, Plaintiffs' liberty and property interests protected by the substantive and procedural due process clauses of the New Hampshire and United States Constitutions will be irreparably harmed, and Plaintiffs will be deprived of their lawful assets and holdings through materially unfair, unconscionable, and substantially increased costs (1) to comply with the MCLs and AGQS established by the Final Rules (2) costs via potential exposure to damages in the two lawsuits against 3M Company by the State if assessed by the MCLs and AGQSs established by the Final Rules.

119. Plaintiffs' due process rights under Part I, Art. 12 and 15 of the New Hampshire Constitution and U.S. Const. amend. XIV, § 1 were thereby violated.

120. Plaintiffs request the Court temporarily, preliminarily and permanently enjoin the NHDES from implementing and enforcing the Final Rules.

WHEREFORE, Plaintiffs The Plymouth Village Water & Sewer District, Resource Management, Inc., Charles G. Hanson, and 3M Company respectfully request that this Court:

A. Enter an order declaring the Final Rules invalid as:

i. Promulgated in violation of the Administrative Procedure Act, RSA 541-A;

ii. Promulgated in violation of the New Hampshire Safe Drinking Water Act, RSA 485;

iii. An unfunded mandate in violation of Part 1, Article 28-a of the New Hampshire Constitution or RSA 541-A:25, or both;

iv. A deprivation of property without due process of law in violation of Part 1, Article 12;

v. A deprivation of property without due process of law in violation of Part 1, Article 15 of the New Hampshire Constitution; and,

vi. A deprivation of property without due process of law in violation of U.S. Const. amend. XIV, § 1;

B. Temporarily, preliminarily and permanently enjoin NHDES from implementing and enforcing the Final Rules until such time as new rules may be enacted in compliance with all regulatory, statutory, and constitutional requirements; and

C. Permanently enjoining NHDES from implementing and enforcing the Fianl Rules until such time as new rules may be enacted in compliance with all regulatory, statutory, and constitutional requirements.

D. Grant such other and further relief as is just and necessary.

Respectfully submitted,

THE PLYMOUTH VILLAGE WATER & SEWER DISTRICT, RESOURCE MANAGEMENT, INC., AND CHARLES G. HANSON

By their Attorneys,

PASTORI|KRANS, PLLC

Dated: September 30, 2019 By: */s/ Terri L. Pastori*

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3M COMPANY

By their Attorneys,

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Dated: September 30, 2019 By: */s/ Mark C. Rouvalis*

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BEVERAGE & DIAMOND

Dated: September 30, 2019 By: */s/ Nessa Horewitch Coppinger*

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CERTIFICATE OF COMPLIANCE WITH RSA 541-A:23, IV AND RSA 541-A:24

Undersigned counsel certifies, in compliance with RSA 541-A:23, IV and RSA 541-A:24, that Plaintiffs have provided the State of New Hampshire Office of Legislative Services, Administrative Rules Division, Scott F. Eaton, Administrative Rules Director, with notice of its pleadings by emailing and mailing by U.S. mail a copy of its Complaint and any related submissions on the date of filing to its office at 25 Capitol Street, Rm 219, Concord, NH 03301-6312.

In addition, undersigned counsel has emailed a copy of this complaint to Senior Assistant Attorneys General K. Allen Brooks and Christopher G. Aslin of the New Hampshire Department of Justice.

/s/ Mark C. Rouvalis
Mark C. Rouvalis

EXHIBIT 1

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

EXHIBIT 2

The State of New Hampshire
DEPARTMENT OF ENVIRONMENTAL SERVICES

Robert R. Scott, Commissioner

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.

<u>Comment</u>: *NHDES should reconsider whether its assumption that the water intake rate of lactating women is appropriately protective across a lifetime.*

<u>Response</u>: 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.

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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

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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

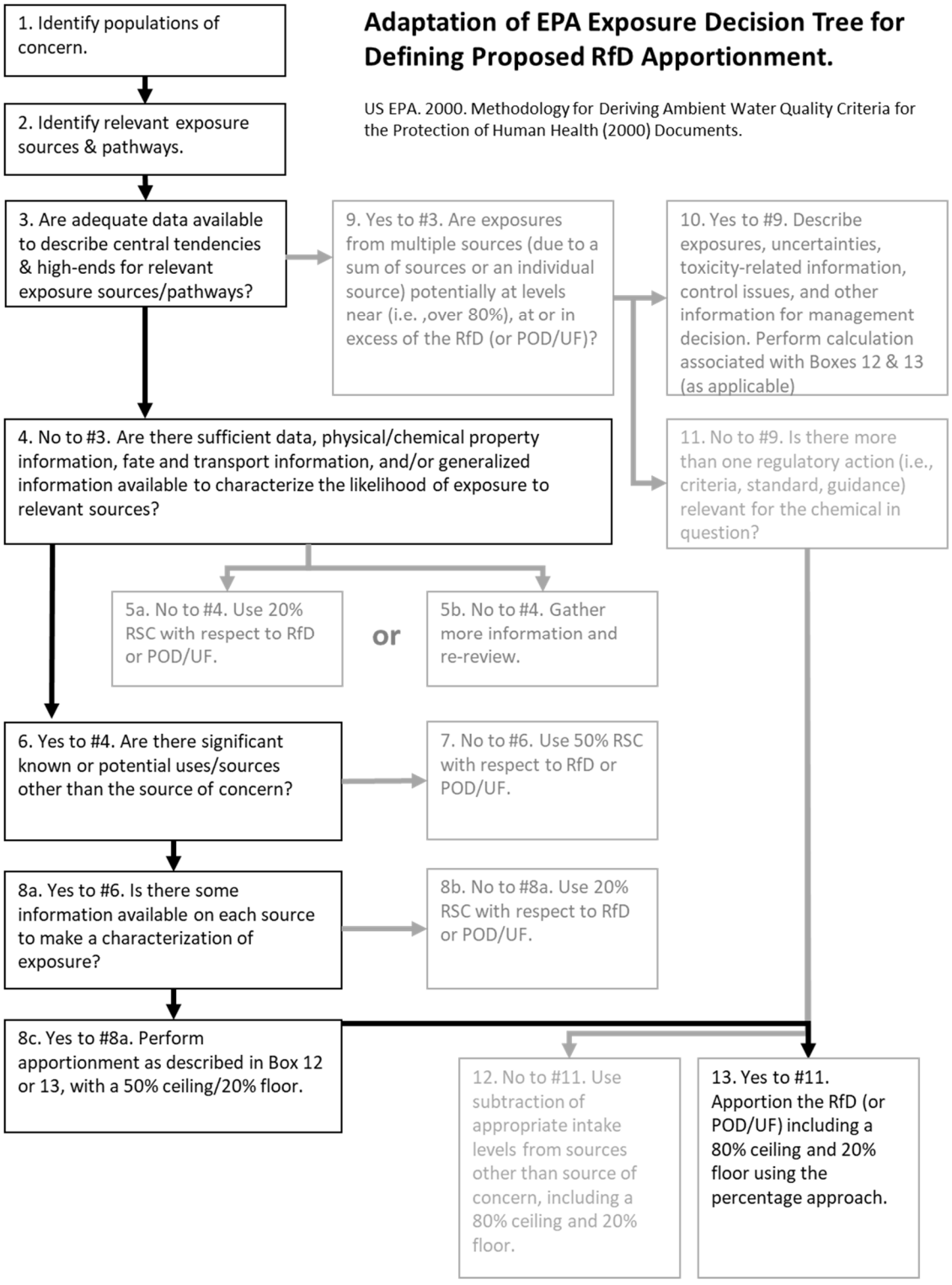

Figure 1. Adaptation of EPA decision tree (EPA, 2000) for determining the RSC. Black boxes, text and arrows outline the decision process used by NHDES to arrive at the subtraction method for PFAS with a 50% ceiling. The target serum level is a population assessment value, *not clinical*, from the derivation 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.

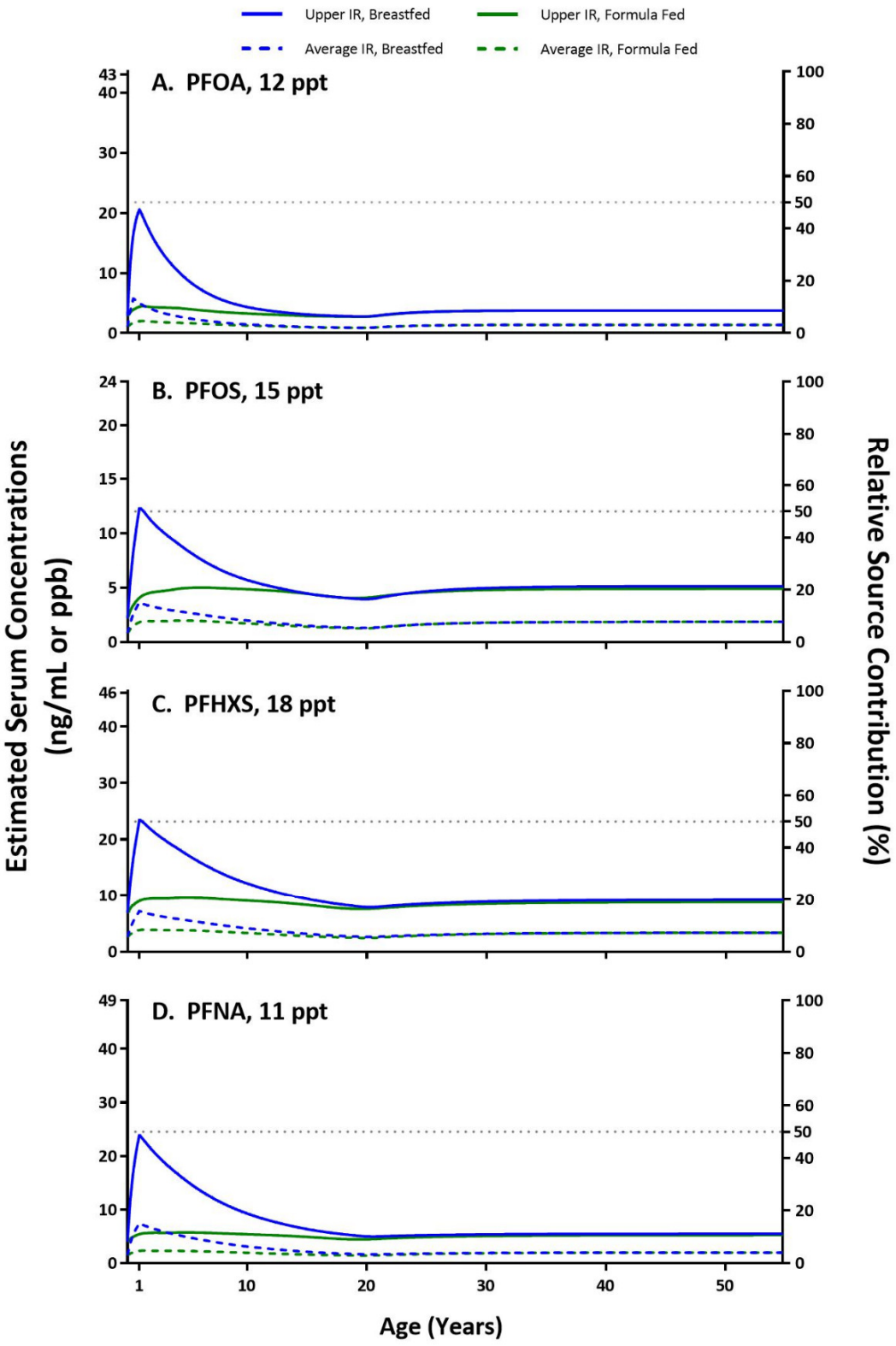

Figure 2. Predicted serum PFAS concentrations in response to upper (95th percentile) and average (mean) water ingestion rates (IR) at the proposed MCLs. Blue lines indicate results for breastfed infants with 12 months exclusive breastfeeding, and green lines indicate results for formula-fed infants. 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.

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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.

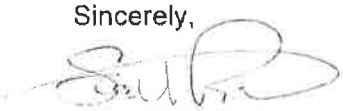
Another issue raised by public commenters is the overall level of conservatism inherent in the originally proposed MCL values, with comments offered in both directions — too conservative or not conservative enough. Concern that the initial MCLs were not

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.

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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***
		A Monitoring Network Enhancments			**A Annual Sampling and Reporting**		
101	53	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.

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

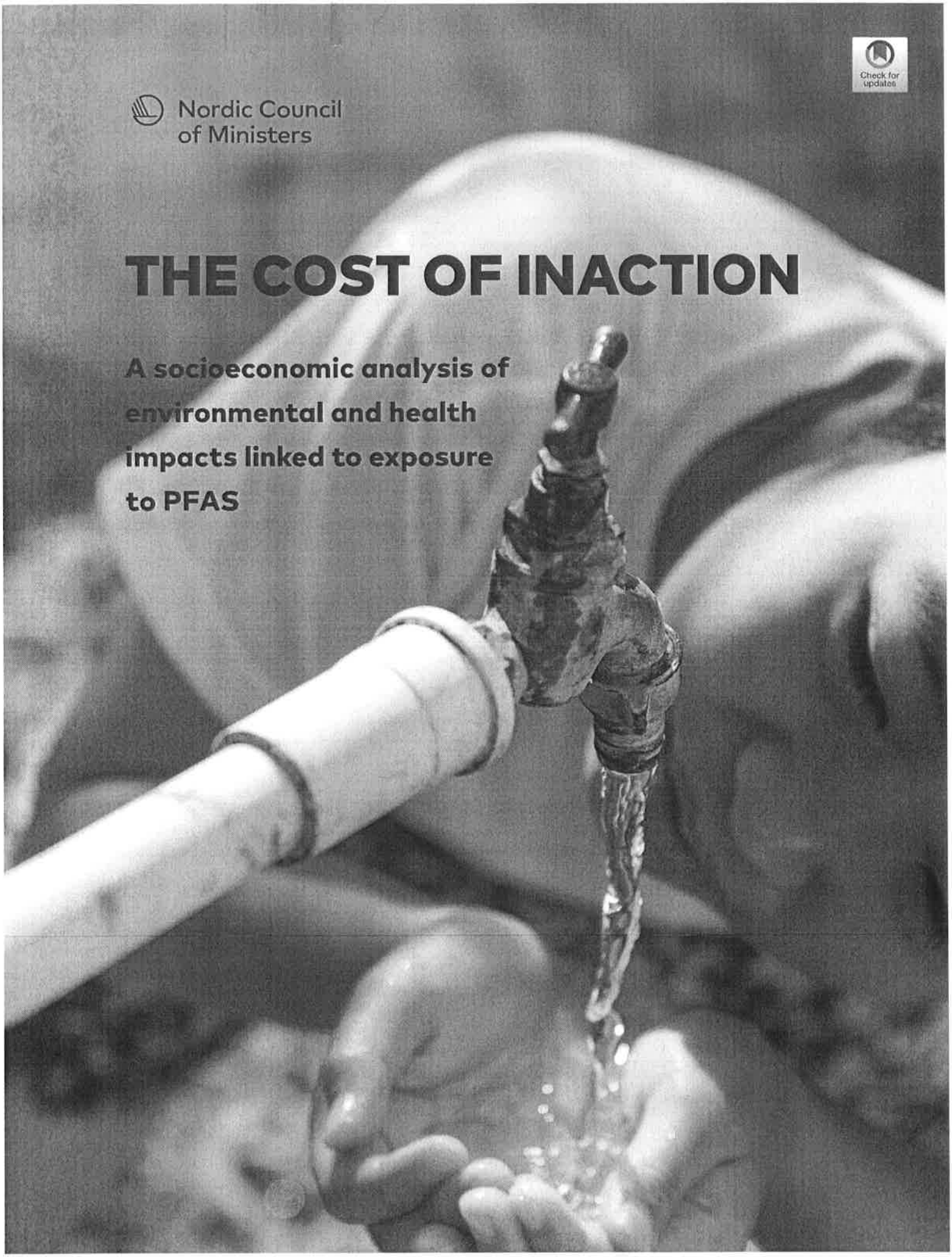
New PFAS Standard Evaluated:
PFOA: 12 ppt
PFOS: 15 ppt
PFNA: 11 ppt
PFHxS: 19 ppt

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
(603) 271-3679
christopher.aslin@doj.nh.gov

CGA/cga

EXHIBIT 3

ENVIRONMENTAL NEWS

NEW HAMPSHIRE DEPARTMENT OF Environmental Services

Newsletter of the New Hampshire Department of Environmental Services September-October 2019

COMMISSIONER'S COLUMN

Final PFAS drinking water standards established

The State of New Hampshire's dedication to being proactive and protective in its investigation of per- and polyfluoroalkyl substances (PFAS) in our environment has led to new, lower drinking water and groundwater standards for four PFAS, established in July and scheduled to take effect September 30, 2019. Now, we prepare to enter the next phase: implementation, which means facilities such as public water systems and groundwater discharge permittees, and contaminated sites will need to start testing for the compounds in their next round of sampling.

PFAS are part of a large class of chemicals that have been used for decades in commercial, industrial and household products and applications, including production of water resistant materials, fire suppression foams (a.k.a. aqueous film forming foam or AFFF), non-stick cookware, stain removers, etc. Because of their wide use, persistence in the environment and bio-accumulative properties, these compounds have been detected in blood serum levels in humans and other animals everywhere. The health effects linked to PFAS exposure have been identified through epidemiological studies and animal studies, and continue to be researched extensively by toxicologist and epidemiologists worldwide to provide greater specificity, especially to additional compounds beyond those which have been most studied to date. According to the Agency for Toxic Substances and Disease Registry,

Commissioner's Column, cont. page 2

Statewide private well sampling initiative

NHDES is sampling 500 randomly selected private wells that are evenly distributed statewide for over 250 chemicals and parameters, including volatile organic compounds, metals, radionuclides, per- and polyfluoroalkyl substances (PFAS) and pesticides. The sampling is funded through a grant from the New Hampshire Drinking Water and Groundwater Trust Fund. The program will offer important information to homeowners about the quality of their drinking water and, when necessary, steps that can be taken to improve water quality.

This information will be used by state officials and scientists to evaluate the occurrence, concentration and sources of certain emerging contaminants in drinking water, including perchlorate, 1,4-dioxane, PFAS and pesticides, and their breakdown products. Additionally, this sampling program will deliver the first statewide assessment of bacteria, nitrate, lead, fluoride, manganese, arsenic, radionuclides and salt in water obtained from private wells, and it will build upon previous statewide assessments that have been conducted on other contaminants such as arsenic and radon. The data collected will provide a holistic snapshot of the quality of water in private wells and identify trends and patterns of the water quality relative to location of the well, nearby land uses, geology, well type and other factors that can impact water quality. Based on this information, strategies will be developed and implemented to mitigate and

Sampling, cont. page 3

Commissioner's Column *continued from page 1*

some known health effects may include interference with the body's natural hormones, increased cholesterol levels, effects to the immune system and increased risk of certain types of cancer.

Using the most recent and best science available, the department established drinking water standards, called maximum contaminant levels (MCLs), and ambient groundwater quality standards (AGQS) that are protective for the most sensitive populations over a lifetime of exposure, and on July 18, the New Hampshire Joint Legislative Committee on Administrative Rules (JLCAR) approved them. The new standards for the four PFAS, include:

- 12 parts per trillion (ppt) for perfluorooctanoic acid (PFOA)
- 15 ppt for perfluorooctanesulfonic acid (PFOS)
- 11 ppt for perfluorononanoic acid (PFNA)
- 18 ppt for perfluorohexanesulfonic acid (PFHxS)

When I last wrote in this space about our proposed MCLs, in the January/February issue, I announced that the department had submitted its initial proposed levels to the state Legislature. Shortly after that, new studies and models became available that indicated that the initial proposed MCLs should be further lowered to reduce exposure to be protective of health over a lifetime. Specifically, a peer-reviewed exposure model was developed and published by the Minnesota Department of Health. This new model influenced the department's decision to reconsider the proposed rules. Using this tool, NHDES continued developing the MCLs, and on June 28, proposed the levels that are now being implemented. A complete description of the development of the proposed MCLs is available on the NH PFAS Investigation website.

The final MCLs apply to non-transient public water systems (water systems serving the same 25 people more than six months per year). An AGQS is the standard used to require remedial action and the provision of alternative drinking water at a contaminated site. It also dictates the conditions under which treated and untreated wastewater may be discharged to groundwater. Current law requires AGQS be the same value as any MCL established by NHDES.

To establish these standards, the department had to consider the extent to which the contaminants are found in New Hampshire, the ability to detect them in public water systems, and the ability to remove the contaminants from drinking water while considering the costs and benefits to affected parties that will result from establishing the standard. The non-transient public water systems will be required to test for these four PFAS compounds in their next quarter of sampling. If sampling results averaged over four quarters of sampling exceed the MCLs, a public water system will need to develop an action plan for achieving compliance with the standards. The work of reducing these compounds in drinking water across the state is expected to require substantive upgrades for facilities that exceed the new MCLs, such as adding filtration systems, and, at the time of filing the rule for approval, was estimated to cost at least $190 million over the next two years.

It's important to note that the new drinking water standards do not apply to private well owners. We recommend that anyone with a private well should periodically have their drinking water tested for a number of different contaminants that can affect water quality and health, including common contaminants like arsenic, lead and radon. The NHDES list of recommended tests for private well water is available on the Private Well Testing Program webpage. If you decide to test for PFAS and find levels above the MCLs, you should consider installing a treatment system. NHDES has posted in-home water filtration information on the NH PFAS Investigation website.

Alongside the development of MCLs, New Hampshire took further steps to protect our residents from PFAS contamination and mitigate the effects of these chemicals. On May 29, the State filed two lawsuits against the original makers of PFAS chemicals, 3M and DuPont, and eight companies that manufacture AFFF (including 3M and DuPont), for the contamination of drinking water. This historic lawsuit represents the statewide effort to protect our citizens and environment from these harmful chemicals.

NHDES will also continue to investigate potentially impacted areas, and, as directed by the New Hampshire Legislature, develop a plan to establish surface water quality standards for the state. NHDES will continue to work with the Department of Health and Human Services to review the latest science and work to educate and inform citizens, healthcare providers, municipalities and other stakeholders about PFAS. For more information about the department's development of the MCLs and the overall PFAS investigation, visit the NH PFAS Investigation website. ■

ENVIRONMENTAL NEWS

Environmental News is published six times a year by the New Hampshire Department of Environmental Services.

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Sampling *continued from page 1*

prevent exposures to unsafe levels of contaminants in drinking water obtained from private wells. NHDES will analyze the sampling results, working with stakeholders and the public, and publish the results over the next 12 months.

Furthermore, the program will collect samples and analyze the water from households that were randomly selected to participate in the 2019 New Hampshire Tracking and Assessment of Chemical Exposures Study. This biomonitoring study includes testing blood and urine from people for many of the same chemicals being analyzed in water. This collaboration will provide crucial information about the relationship between chemicals measured in drinking water and in the bodies of study participants, giving additional insight to the ongoing effort to improve drinking water quality in New Hampshire and beyond. ■

Resilient tidal crossings

The NHDES Coastal Program recently released new mapping products and data that characterize tidal crossings for community and ecosystem resilience. This information is intended to be used by community officials and road managers to enact strategic repair or replacement of tidal crossing infrastructure, and to identify high-priority restoration and conservation opportunities at tidal crossing sites.

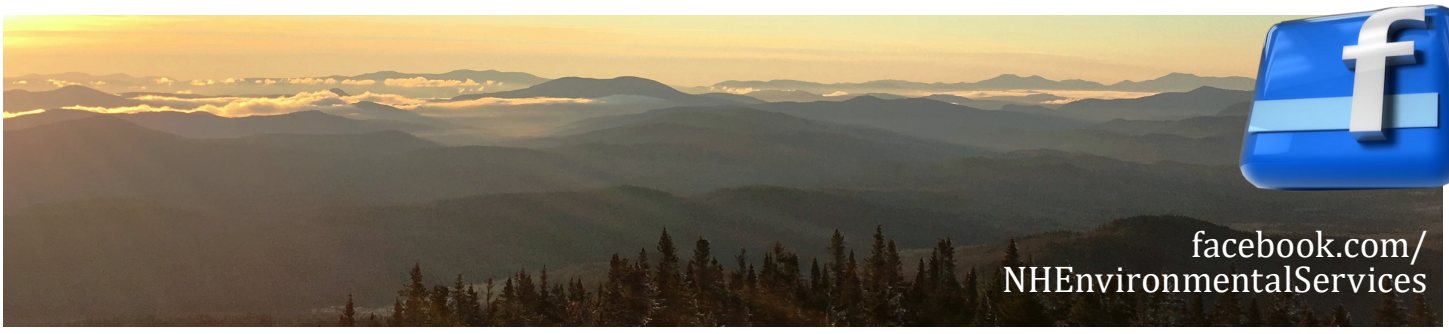
A tidal stream crossing (tidal crossing) is a bridge or culvert that conveys two-directional tidal flow. Tidal crossings are a unique and challenging class of transportation assets that have different engineering, regulatory and risk management considerations than their freshwater counterparts. For instance, properly designed tidal crossings need to convey enough tidal flow to periodically cover the salt marsh and at the same time be of sufficient size to accommodate freshwater flows from upstream sources. Additionally, tidal crossing infrastructure is especially vulnerable to coastal storm surge, flooding and sea-level rise.

The NHDES Coastal Program was awarded a $187,500 grant from the National Oceanic and Atmospheric Administration for the Resilient Tidal Crossings Project in 2017. The grant enabled the NHDES Coastal Program to work with a team of partners, including The Nature Conservancy and the University of New Hampshire, to implement the New Hampshire Tidal Crossing Assessment Protocol at all 118 tidal crossings in New Hampshire during the summer of 2018. This collaborative project brought together robust technical expertise and innovative remote sensing mapping products to implement an ambitious field and data analysis project. The project utilized the Statewide Asset Database Exchange System (SADES), New Hampshire's primary inventory of transportation assets, which enabled efficient data collection and establishes a digital inventory of tidal crossing infrastructure for reliable long-term asset management.

Tidal crossing assessment data were then used to score sites based on salt marsh migration potential, tidal restriction overall, vegetation evaluation, structure condition, inundation risk and tidal aquatic organism passage. The results of the Resilient Tidal Crossing Project show that among 118 tidal crossings, 23 were identified as highest replacement priority and 32 sites were identified as high replacement priority.

Although the data collection and assessment aspects of the project have concluded, there is still work to be done to leverage, implement and advance the project's findings. Initiatives include data sharing and maintenance, creation of crossing design standards, continuous research and advancing high priority tidal crossings through design and replacement. Implementation is already underway on the Lubberland Creek culvert replacement project in Newmarket to increase its climate resiliency. In addition, NHDES Wetlands Rules were recently updated to include a new category (Tier IV) within the stream crossing rules; creating for the first time in New Hampshire a regulatory standard for tidal crossing replacement projects.

More results, mapping products and other Resilient Tidal Crossings Project materials are available on the Resilient Tidal Crossings webpage. ■



Gulf of Maine Visionary Awards

Two New Hampshire award winners, including a NHDES employee, were among the individuals and organizations honored by the Gulf of Maine Council at an international ceremony held in Nova Scotia for making a significant difference in protecting the health and sustainability of the Gulf of Maine watershed.

The Gulf of Maine Visionary Awards are given to two individuals or organizations within each state and province to recognize their innovation, creativity and commitment to marine protection.

Kevin Lucey, NHDES Coastal Program restoration coordinator, was recognized for his exemplary work and leadership on the coastal watershed region's most significant restoration efforts including the Resilient Tidal Crossings Project as well as multiple dam removal projects. Kevin recently led an on-the-ground effort to assess every tidal crossing in New Hampshire, which resulted in new mapping products and data that characterize tidal crossings for community and ecosystem resilience. This information can be used by community officials and road managers to enact strategic repair/replacement of tidal crossing infrastructure and to identify high priority restoration and conservation opportunities at tidal crossings sites.

Abigail Lyon, community technical assistance program manager at the Piscataqua Region Estuaries Partnership, was recognized for her outstanding commitment to improving the environmental state of affairs in the Gulf of Maine through her current position as well as many work and volunteer experiences sharing her love and enthusiasm for the natural world and how to care for it.

The Council, a U.S.-Canadian partnership dedicated to protecting environmental quality in the Gulf of Maine, annually recognizes extraordinary work in its five jurisdictions, which include the states of New Hampshire, Maine and Massachusetts as well as the Canadian provinces of New Brunswick and Nova Scotia.

Additional information about the Gulf of Maine Council 2019 awards with detailed recipient bios is available on the Gulf of Maine website. ■

Walpole company honored for environmental stewardship

Chamberlain Machine Inc. of Walpole was recently awarded the national 2019 Small Business Environmental Stewardship Award for its work with the NHDES Small Business Technical Assistance Program (SBTAP) and Pollution Prevention Program (NHPPP) to significantly reduce or eliminate waste streams, and improve energy conservation and recycling.

The award, presented by the National Small Business Environmental Assistance Program, recognizes outstanding environmental leadership among small businesses and small business assistance providers.

"Chamberlain Machine is pleased to be recognized for our sustainability initiatives. Reducing or eliminating waste streams, energy conservation and recycling are key to our business success and demonstrate our commitment to environmental responsibility. SBTAP and NHPPP continue to be a valuable resource in pursuing our environmental goals," said Scott Boynton, President of Chamberlain Machine.

With the assistance of SBTAP and NHPPP, Chamberlain Machine thoroughly reviewed its waste streams, revamped an aqueous waste disposal process and installed a wastewater centrifuge. These updates and improvements proved to be successful: process-specific discharge was reduced by 93%. Moreover, the company installed a solar array that supplies 25% of its electricity needs and converted all lighting to LED, further reducing the company's electricity demand.

The awards program is sponsored by the National Steering Committee of Small Business Environmental Assistance Programs and Small Business Ombudsmen, in partnership with the EPA Asbestos and Small Business Office. ■

Geologic mapping program

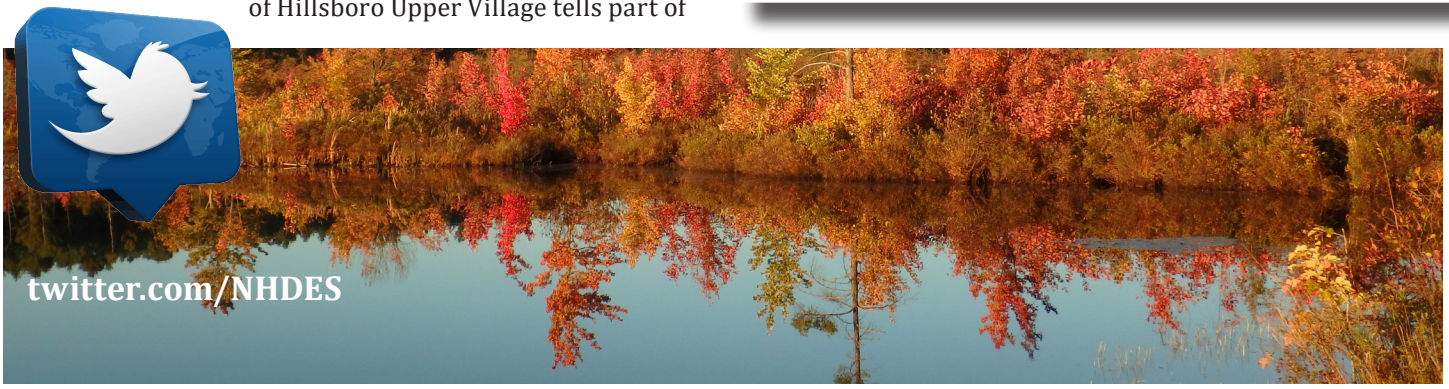
The New Hampshire Geological Survey (NHGS) at NHDES has been working since its inception in 1839 to map the bedrock geology and surficial deposits of the entire state. Two NHGS geologists, Greg Barker and Joshua Keeley, routinely go out in the field and map as part of this program.

Similar to the rest of the country, New Hampshire is divided into quadrangles, or areas defined by 7.5-minute grids, which are typically 49 to 70 square miles. In July, Greg and Joshua went out to the Hillsboro Upper Village quadrangle, which includes parts of the towns of Hillsborough, Henniker, Bradford and Warner.

At the Hillsboro Upper Village quadrangle, Greg and Joshua focused on surficial mapping, which is one of the program's main efforts. Surficial maps characterize the different earth materials of varying thicknesses that lie above the bedrock. In New Hampshire, these materials were at one time eroded and then transported by continental glaciers, the latest of which is known as the Laurentide Ice Sheet. The mapped sediments were generally deposited either directly by the ice (glacial till) or by streams of melt-water (stratified drift) as the glaciers melted, thinned and retreated. The sediment size of these materials varies greatly, and can tell us a lot about the geological history of the quadrangle. The size of the particles clearly reflects whether the water was flowing when deposition occurred; boulders, gravels and coarser sands indicate high-energy environments of deposition whereas fine sands, silts and clay indicate accumulation at the bottom of bodies of standing water. This particular map of Hillsboro Upper Village tells part of the story of Glacial Lake Contoocook.

The only tools Greg and Joshua need for the day are a shovel, a compass and a geographic information system (GIS) handheld device. Based on previous maps, they already had an idea of where they wanted to look for the glacial tills and stratified drift. Additionally, the mappers use a new type of high resolution topographic data called LiDAR. Because of the detailed topography, these new data allow greater understanding and confidence of how sediments were likely deposited. Navigating through many unpaved and unmaintained roads, they examined the sediment size by digging holes a few inches to two feet deep and visually inspecting the sediments for finer materials, such as sand, silt and clay, throughout the quadrangle. They logged the results and data on the GIS device, which will be used to create maps and for other analyses. New mapping will be available to the public after September 15.

Geology may not get the attention it deserves; however, geologic maps are highly informative and important. For example, they may be useful in construction and engineering projects, and city planning. Dams, roads, buildings and bridges require geologic analyses, as well as other smaller projects like water wells and septic systems. Surficial maps play an important role in understanding the way the land has been influenced by glacial melting over hundreds of thousands of years. ■



NHDES Snapshot: Fresh Water Beach Sampling

NHDES staff can't fulfill the agency's mission only from our desks. To protect environmental quality and public health in New Hampshire, we are out in the field every day: testing water quality in our ponds and lakes, sampling private well water, monitoring air emissions, assessing storm damage, responding to oil and chemical spills, training water works and solid waste operators, and so much more. "NHDES Snapshot" is an occasional series that takes a quick look inside the day of one of those employees.

Andrea hops out of the car, gathers three sample bottles and writes the time on each one. While walking toward this freshwater beach, she eyes the three sections of the lake that she will be taking samples from, which are established stations reported to the EPA. To collect a sample, she wades into the water to her knees and fills the bottle with water. She also determines the water temperature and makes an inspection, noting the number of people swimming, number of animals or birds, and overall conditions of the beach and water. Then she returns to the sand, collects her things and heads back to the car.

This summer she's working for the Beach Inspection Program, resampling fresh and marine waterbodies to update current beach advisories. The work takes her all across New Hampshire; she'll visit a different area of the state each day of the week. She and her co-workers, Tammi and Della, monitor about 100 public beaches between Memorial Day and Labor Day. These beaches are sampled throughout the month and, in the event of an advisory, they are resampled within two days.

They are testing public swimming beaches for fecal indicator bacteria, which is used to judge the water quality and possible presence of pathogens. They also collect samples when there is either a definite or suspected cyanobacteria bloom in order to test for species of toxic cyanobacteria. Toxic cyanobacteria can affect anyone who comes into contact with it, but it is particularly harmful to highly vulnerable groups such as children, dogs and people with compromised immune systems.

After Andrea returns to the car, she ensures the samples are labeled correctly and places them in the cooler. She'll repeat this process for the next few beaches she has to resample, returning to the office within six hours from the first sample taken.

When she returns to the NHDES Concord office, she drops off the day's fecal bacteria samples at the Department of Health and Human Services lab, where staff there will test and get the results back to her within 24 hours. The cyanobacteria samples are looked at immediately, identified and enumerated by Amanda McQuaid, NHDES Beach Program Coordinator.

If the bacteria amount for those they study exceeds the limits set by the State, NHDES issues an advisory, warning the public of the potential swimming hazards. Andrea will then resample the beach to find out if the levels have decreased enough for the advisory to be lifted.

After looking at the previous day's results to see what beaches require an advisory, she'll plan to revisit beaches with elevated fecal bacteria and prepare for the next day. Andrea also travels to locations with reported blooms and will resample where cyanobacteria samples are needed.

When talking to Andrea about the importance of her work at NHDES, she said it's all about protecting public health and making sure people stay safe: "Water quality sampling is important to the community because it is a preventative science. We sample and test for fecal bacteria to prevent the public from getting sick while enjoying the beauty of New Hampshire's beaches."

To stay up-to-date on beach advisories, subscribe to the Beach Program's weekly enewsletter, follow on Twitter (@NHDES_Beaches) or view the interactive map. ■

Summer food drive

In June, NHDES hosted a two-week long internal food drive where NHDES employees donated more than $300 and 2,760 food and hygiene items. The donations were delivered to the Friends of Forgotten Children, a local organization that provides assistance to children dependent on free and reduced-cost lunches. ■

The hazy days of summer

Have you ever taken a summer trip to the New Hampshire White Mountains and wished you could see Mt. Washington or the many other mountains more clearly through the haze? One of the most basic forms of air pollution, haze, degrades visibility in many scenic areas. Haze is caused when sunlight encounters tiny pollution particles in the air, which reduce the clarity and color of what we see, particularly during humid conditions, which is why it is often worst during humid summer days. Of course, Mt. Washington is often covered in clouds, which is just part of the natural mystique of New Hampshire and has nothing to do with pollutants.

During hazy days, visibility reduction is normally caused by sulfate, nitrate and organically based particles. Air pollution emissions of sulfur dioxide (SO_2), nitrogen oxides (NOx) and organic gases react in the atmosphere to create ammonium sulfate, ammonium nitrate and organically based particles. Higher concentrations of these particles results in lower visibility. These particles also affect our health, thus improving our visibility also improves our health. There are also some days where visibility is reduced by smoke particles caused by forest fires, sometimes thousands of miles away. Events such as these are due to accidents or natural events, such as lightning strikes, and are not part of normal human controlled air pollution emissions.

The federal regional haze rule seeks to make steady visibility improvement toward natural visibility conditions by the year 2064. Every five to 10 years, the state needs to provide status updates and an updated state implementation plan for making emission reductions designed to help visibility at federally designated Class I areas. In New Hampshire, the Great Gulf wilderness and the Presidential Range – Dry River Wilderness, located on the north and south flanks of Mt. Washington, are federally designated Class I areas. Lye Brook Wilderness Area in Vermont and Acadia National Park and Moosehorn Wilderness Area, both in Maine, are other Class I areas within reach of New Hampshire air pollution emissions.

NHDES is working with our neighboring states to develop plans to ensure we still have the views of our precious landscapes. We are already seeing signs of improvement. The regional haze rule has been in place for 17 years. In that time visibility during the worst days has improved around Mt. Washington; you can now see about three times farther on these bad visibility days. Similar improvement has been noted at the nearby Class I areas, so we are clearly on the right track. With each passing year, your trips to the White Mountains will be greeted with better odds of a good view of Mt. Washington and the surrounding mountains. ■

Robert Fitzpatrick won the #ThisIsNH July photo contest with this photo of "A Beautiful Morning on Swanzey Lake." The theme of the photo contest was "Lakes Appreciation Month." Photos submitted to the This Is New Hampshire story map were entered into the contest, and our Facebook and Twitter followers were invited to vote for their favorite. The prize was having the winning photo featured as the NHDES Facebook and Twitter cover photo, and the cover of the story map website. ■

Charge Forward EV Relay

NHDES and Granite State Clean Cities Coalition are among the partners supporting a first-of-its-kind Electric Vehicle (EV) event in New Hampshire. On Monday, September 16, the Charge Forward EV Relay will showcase the diversity of EV models and the accessibility of charging stations to major destinations across the Granite State.

The relay will feature the latest EVs, driven by popular New Hampshire personalities, along each leg of the relay, demonstrating the power and practicality of the next generation in transportation. Each leg of the route will showcase a different EV model and highlight a unique New Hampshire landmark or destination. Public is welcome at all stops and EV drivers are welcome to "caravan" along the way. Visit the Drive Electric NH – Charge Forward EV Relay website for details. ■

NEW HAMPSHIRE DEPARTMENT OF
Environmental Services

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GSCCC reports fuel and emissions reductions

Have you ever considering fueling your vehicle with something other than gasoline or diesel? Do you modify your driving habits to improve your fuel economy? Are you a stickler about turning off your engine to prevent unnecessary idling?

Clean Cities, a program supported by the United States Department of Energy, works with public and private fleets, and businesses to advance affordable, domestic transportation fuels and technologies. In New Hampshire, the Granite State Clean Cities Coalition (GSCCC) is made up of 140 stakeholders working to reduce petroleum use by adopting and advancing alternative fuels (such as biodiesel, natural gas and propane), advanced technology vehicles (such as electric and hybrid), and other fuel-saving strategies (such as idle-reduction). The GSCCC Coordinator works with stakeholders to support these efforts, promote their achievements and provide education and outreach around the state.

Each year, Clean Cities Coalitions across the nation perform stakeholder outreach for the Clean Cities Annual Report. This year, GSCCC stakeholders provided data on their use of alternative fuels, advanced technology vehicles and other fuel-saving strategies during 2018. The data reflect the impact of stakeholder efforts in reducing petroleum consumption and vehicle emissions.

In 2018, GSCCC Stakeholders reduced petroleum use by over 1.4 million gallons and Greenhouse Gas Emissions by over 8,500 tons! This goes to show that even small steps can make big impressions.

If you are interested in more information about alternative fuels and advanced technology vehicles , visit the GSCCC website. The latest news includes the launch of Destination Electric, a program that promotes businesses and destinations in the Northeast that are near electric vehicle charging stations. ■

National Drive Electric Week

Join us at the Drive Electric kick-off event taking place in downtown Concord on Saturday, September 14 from 8:30 AM-1 PM. We'll be at City Plaza in front of the State House, next to the Concord Farmer's Market. Find out more details about our kick-off and other of free events scheduled around New Hampshire, on the Drive Electric Week website. ■

EXHIBIT 4

NH PFAS Investigation

Release: NHDES Proposes New PFAS Drinking Water Standards

Posted on January 2, 2019 by Jim Martin

News from the New Hampshire Department of Environmental Services

FOR IMMEDIATE RELEASE

DATE: January 2, 2019

CONTACT: Jim Martin, (603) 271-3710

NHDES Proposes New PFAS Drinking Water Standards

Initiates Rulemaking for PFOA, PFOS, PFHxS and PFNA

Concord, NH – On December 31, 2018, the New Hampshire Department of Environmental Services (NHDES) initiated rulemaking to establish Maximum Contaminant Levels (MCLs) and Ambient Groundwater Quality Standards (AGQS) for four per- and polyfluoroalkyl substances (PFAS) – perfluorooctanoic acid (PFOA), perfluorooctanesulfonic acid (PFOS), perfluorononanoic acid (PFNA) and perfluorohexanesulfonic acid (PFHxS) to ensure greater protection of public health related to the consumption of drinking water. Specifically, NHDES filed a request for a fiscal impact statement for the new MCLs with the New Hampshire Legislative Budget

ADDITIONAL RESOURCES

Water Line Extension Projects
Investigation Documents
Be Well Informed Guide
Pease Tradeport Investigation Archive

RECENT POSTS

Summary of the Technical Background Report for the Proposed Maximum Contaminant Levels and Ambient Groundwater Quality Standards for PFOA, PFOS, PFNA and PFHxS.
NHDES Submits Final Rulemaking Proposal for PFOA, PFOS, PFHxS and PFNA
Slides from East Kingston PFAS Update Meeting, June 20, 2019
EPA Office of Research and Development

Assistant, meeting the January 1 deadline established in New Hampshire Chapter Laws 345 and 368 of 2018 (i.e. SB 309).

These MCLs are drinking water quality standards that non-transient public water systems (water systems serving the same 25 people at least 60 days a year) must comply with. An AGQS is the standard used to require remedial action and the provision of alternative drinking water at a contaminated site. It also dictates the conditions under which treated and untreated wastewater may be discharged to groundwater. Current law requires AGQSs be the same value as any MCL established by NHDES and also that they be as stringent as health advisories set by the U.S. Environmental Protection Agency (EPA). In 2016, NHDES adopted EPA's health advisory for PFOA and PFOS as an AGQS (70 parts per trillion (ppt) combined).

To establish MCLs for PFOA, PFOS, PFHxS and PFNA, which by law then become AGQSs, NHDES had to also address 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, and then develop a MCL for each compound that is protective of the most sensitive population at all life stages. The development of these standards was greatly enhanced by affected parties responding to NHDES' request for studies and information to be considered in deriving the MCLs (https://www.des.nh.gov/organization/commissioner/max-contaminant-levels.htm).

Using the most recent and best science available, NHDES is proposing the following drinking water standards that are protective of the most sensitive populations over a lifetime:

PFAS	Proposed MCL and AGQS
PFOA	38 ppt
PFOS	70 ppt
PFOA & PFOS (combined)	70 ppt
PFHxS	85 ppt
PFNA	23 ppt

Within the next few days, NHDES will release a summary report on the development of the drinking water standards (MCLs) including an explanation of the health risk assessment for each compound and

Report #6 (stack test emissions)
Public Information Meeting – East Kingston, June 20
EPA Office of Research and Development Report #5 (raw materials)
State of New Hampshire Announces Historic Lawsuit, Actions to Protect Clean Drinking Water in New Hampshire

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NHDES Public Information Officer

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information on cost, benefit, occurrence, and ability to detect and treat these chemicals. The report will be posted on the NHDES PFAS webpage – https://www4.des.state.nh.us/nh-pfas-investigation/

The majority of the work NHDES has performed to date has been focused on deriving the individual standards for PFOA, PFOS, PFNA and PFHxS that protect the most sensitive population through their lives. During the rulemaking process, NHDES expects to continue researching health studies on these chemicals as well as risk management approaches that are scientifically valid that could address any compounding effects between chemicals. 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. NHDES recognizes and thanks the many stakeholder groups who have participated to date, and hopes they continue to be engaged throughout the public comment process.

Public hearings on the proposed MCLs will occur in southern NH, at Pease Tradeport, and at the NHDES offices in Concord in early March, which will provide the public more than a month to review the proposal and companion report. Depending on the comments received, it is anticipated that the final proposals will be filed by summer. The effective date of the new rules has yet to be determined.

# # #

Posted in Uncategorized

← NHDES Report on PFAS MCL/AGQS Development

Results of Saint-Gobain, October 2018, on-site groundwater samples →

EXHIBIT 5

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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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

Contact with questions or comments:
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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

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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 $10^{0.5} \times 10^{0.5} = 10$.

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

Appendix 6: PFNA Derivation

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

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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.

$DAF = V_d$ x (Ln(2) ÷ $t_{½}$)

*$DAF = 0.287$ L/kg x (Ln(2) ÷ (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.

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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.

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

	Item	Count	Unit Cost	Total
Additional Capital Costs	Mon Well	3	$ 12,000	$ 36,000
	Priv Well Svy	1	$ 1,000	$ 1,000
			Total	*$ 37,000*
1X	***Add'l sites***			***$ 37,000***

	Item	Count	Unit Cost	Total
Additional Annual Costs	Smpl Rnd	6	$ 1,000	$ 6,000
	Rpting	1	$ 2,400	$ 2,400
			Total	*$ 8,400*
1X	***Add'l sites***			***$ 8,400***

Large GWDP Sites
POTW sites, usually publicly owned

	Item	Count	Unit Cost	Total
Additional Capital Costs	Mon Well	6	$ 12,000	$ 72,000
	Priv Well Svy	1	$ 1,000	$ 1,000
			Total	*$ 73,000*
3X	***Add'l sites***			***$ 219,000***

	Item	Count	Unit Cost	Total
Additional Annual Costs	Smpl Rnd	12	$ 1,000	$ 12,000
	Rpting	1	$ 2,400	$ 2,400
			Total	*$ 14,400*
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

	Item	Count	Unit Cost	Total
Additional Capital Costs	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***		
2X	***Add'l sites***			***$ 71,000***

	Item	Count	Unit Cost	Total
Additional Annual Costs	Smpl Rnd	4	$ 1,000	$ 4,000
	Rpting	1	$ 2,400	$ 2,400
	O&M	3	$ 900	$ 2,700
			Total	*$ 9,100*
2X	***Add'l sites***			***$ 18,200***

Large GWDP Sites
POTW sites, usually publicly owned

	Item	Count	Unit Cost	Total
Additional Capital Costs	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***		
1X	***Add'l sites***			***$ 71,000***

	Item	Count	Unit Cost	Total
Additional Annual Costs	Smpl Rnd	8	$ 1,000	$ 8,000
	Rpting	1	$ 2,400	$ 2,400
	O&M	6	$ 900	$ 5,400
			Total	*$ 15,800*
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

EXHIBIT 6

NH PFAS Investigation

New Information May Change NHDES Proposed PFAS Drinking Water Standards

Posted on February 21, 2019 by Jim Martin

New Information May Change NHDES Proposed PFAS Drinking Water Standards

On December 31, 2018, the New Hampshire Department of Environmental Services (NHDES) initiated rulemaking to establish Maximum Contaminant Levels (MCLs) and Ambient Groundwater Quality Standards (AGQS) for four per- and polyfluoroalkyl substances (PFAS) – perfluorooctanoic acid (PFOA), perfluorooctanesulfonic acid (PFOS), perfluorononanoic acid (PFNA) and perfluorohexanesulfonic acid (PFHxS).

After the initial proposal, new scientific information was evaluated by NHDES that may change the proposed drinking water standards. Specifically, a new assessment tool developed by the Minnesota Department of Health allows for a quantitative estimate of infant and child exposure to PFAS through breastmilk and/or formula. This peer-reviewed model was published at the beginning of January after NHDES filed its Initial Proposal. 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. NHDES is continuing to review the suitability of this assessment tool for PFHxS and PFNA based on this and other studies released in 2019. NHDES will need to complete a review of the technical and cost implications

ADDITIONAL RESOURCES

Water Line Extension Projects
Investigation Documents
Be Well Informed Guide
Pease Tradeport Investigation
Archive

RECENT POSTS

Summary of the Technical Background Report for the Proposed Maximum Contaminant Levels and Ambient Groundwater Quality Standards for PFOA, PFOS, PFNA and PFHxS.
NHDES Submits Final Rulemaking Proposal for PFOA, PFOS, PFHxS and PFNA
Slides from East Kingston PFAS Update Meeting, June 20, 2019
EPA Office of Research and Development

of these health-based calculations, and any public comment received, prior to issuance of the Final Proposal.

NHDES feels that it is important to release this information prior to the upcoming PFAS public hearings, so that there is plenty of time for people and organizations to examine this model and its use while developing their comments.

The Rule Making Notice/Initial Proposal packages for the amendments to Env-Dw 700-800, Env-Or 603.03, and Env-Wq 402 are available on-line at:

https://www.des.nh.gov/organization/commissioner/legal/rulemaking/index.htm#pdrinking

https://www.des.nh.gov/organization/commissioner/legal/rulemaking/index.htm#poil

https://www.des.nh.gov/organization/commissioner/legal/rulemaking/index.htm#pwaterq

Public Hearing Dates:

Monday, March 4, 2019, 5:30 PM; All Purpose Room, James Mastricola Upper Elementary School, Merrimack, NH

Tuesday, March 5, 2019, 1:00 PM; Auditorium, DES Offices, 29 Hazen Drive, Concord NH

Tuesday, March 12, 2019, 5:30 PM; NHDES Pease Field Office, Room A, 222 International Drive, Suite 175, Portsmouth, NH

You may submit written comments even if you do not attend a public hearing.

LAST DAY TO FILE WRITTEN COMMENTS**: Friday, April 12, 2019 (4:00 PM)**

For Drinking Water Standards (MCLs) (Env-Dw 700-800) – Submit comments to: Chip Mackey (Harrison.Mackey@des.nh.gov), DWGB Drinking Water Quality Manager

For Ambient Groundwater Quality Standards (AGQS) (Env-Or 603.03) – Submit comments to: Lea Anne Atwell (LeaAnne.Atwell@des.nh.gov), Haz. Waste Remediation Bureau, Emerging Contaminants Coordinator

For Discharges to Groundwater of Wastewater Containing Certain Perfluorochemicals (Env-Wq 402) – Submit comments to: Stephen Roy

Report #6 (stack test emissions)
Public Information Meeting – East Kingston, June 20
EPA Office of Research and Development Report #5 (raw materials)
State of New Hampshire Announces Historic Lawsuit, Actions to Protect Clean Drinking Water in New Hampshire

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CATEGORIES

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(Stephen.Roy@des.nh.gov), DWGB Technical Section Manager, Groundwater Permitting

(Primary reference: Goeden et al. 2019. A transgenerational toxicokinetic model and its use in derivation of Minnesota PFOA water guidance. *Journal of Exposure Science & Environmental Epidemiology*. vol 29, pages 183–195. https://www.nature.com/articles/s41370-018-0110-5)

# # #

Posted in Uncategorized

← Information for Upcoming PFAS Rulemaking Hearings

EPA to Announce First-Ever Comprehensive Nation Wide PFAS Action Plan →

EXHIBIT 7

NEW HAMPSHIRE DEPARTMENT OF Environmental Services

Summary of the Technical Background Report for the Proposed Maximum Contaminant Levels and Ambient Groundwater Quality Standards for PFOA, PFOS, PFNA and PFHxS.

Stakeholder Meeting
07/09/2019

NEW HAMPSHIRE DEPARTMENT OF Environmental Services

Presentation Overview

1. Health-Based Risk Assessment Process

2. Chemical-Specific Reference Doses for:
   PFOA PFOS
   PFNA PFHxS

3. Exposure Assumptions
   Use of the “Minnesota” Model
   Relative Source Contribution

4. Modeled Exposures & Proposed MCLs

5. Questions

image: medium.com

Acknowledgements

The New Hampshire Department of Environmental Services (NHDES) acknowledges the following groups for technical comments submitted by New Hampshire's:

- **residents and community stakeholders,**
- **academic institutions,**
- **community advocacy groups,**
- **representatives for the business community,**
- **and municipalities.**

Additionally, NHDES acknowledges the productive and professional discussions and information sharing by the following entities:

- **Connecticut Department of Public Health (CTDPH)**
- **Environmental Council of the States (ECOS) PFAS Caucus**
- **Federal-State Toxicology & Risk Analysis Committee (FSTRAC)**
- **Interstate Technology & Regulatory Council (ITRC) PFAS Working Group**
- **Massachusetts Department of Environmental Protection (MADEP)**
- **Michigan Department of Health & Human Services (MIDHHS)**
- **Minnesota Department of Health (MDH)**
- **New England Interstate Water Pollution Control Commission (NEIWPCC)**
- **New Jersey Department of Environmental Protection (NJDEP)**
- **Northeast Waste Management Officials' Association (NEWMOA)**

Health-Based Risk Assessment Process

1. **Identify the chemicals of concern**:

Perfluorooctanoic acid (PFOA)	Perfluorooctane sulfonic acid (PFOS)
Perfluorononanoic acid (PFNA)	Perfluorohexane sulfonic acid (PFHxS)

2. Identify sensitive and human-relevant health effects due to exposure to the chemical, and **derive a reference dose (RfD)** for the effects.
   - Is the chemical a carcinogen?
   - Are non-cancer health effects more protective than cancer endpoints?
   - Do epidemiological studies provide clear evidence?
   - Are there appropriate animal models for quantifying toxicity?

3. **Characterize an exposure scenario** using protective assumptions to determine an environmental concentration (*i.e.*, drinking water level) that will not exceed the RfD.

Health-Based Risk Assessment Process

Per the CDC's **Agency for Toxic Substances and Disease Registry** (ATSDR) draft toxicity profile on PFAS (ATSDR, 2018), suspected health outcomes include:

Rodent Experiments

- Immune system modulation
- Altered lipid metabolism
- Liver stress and inflammation
- Altered liver enzyme levels
- Thyroid disruption
- Reduced birth weight
- Fetal skeletal defects
- Fetal loss (death)
- Neurobehavioral defects
- Delayed mammary gland development
- Liver, testicular & kidney cancer

?

Human Epidemiology

Possible effects based on associations:

- Immune system modulation
- Altered lipid metabolism
- Altered liver enzyme levels
- Altered thyroid hormone levels
- Altered behavior in infants, children & adolescents
- Infertility in women
- Reduced birth weight
- Potentially testicular & kidney cancer

NEW HAMPSHIRE DEPARTMENT OF Environmental Services

Health-Based Risk Assessment Process

Proposed MCLs based on non-cancer endpoints

Specific PFAS	NHDES Revised MCLs	Animal Health Outcome
PFOA	12 ng/L	Liver toxicity & altered lipid metabolism
PFOS	15 ng/L	Suppressed immune response to vaccines
PFHxS	18 ng/L	Reduced female fertility
PFNA	11 ng/L	Liver toxicity & altered lipid metabolism

Chemical-Specific Reference Doses

A **reference dose (RfD)** is:
"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." – EPA 2002

RfDs are not synonymous to ATSDR minimal risk levels (MRLs).

Measured internal dose from animal study

Estimated an external (oral) dose

Used for the calculation of a drinking water or other environmental standard

$$\frac{\textbf{Animal Serum (ng/mL)}}{\textbf{Total Uncertainty}} \times \textbf{Dosimetric Adjustment Factor (mL/kg–d)} = \textbf{Reference Dose (ng/kg–d)}$$

Accounts for animal-to-human differences and quality of studies

Chemical-Specific Reference Doses

Animal studies selected for RfDs in the Initial (January) MCL proposal.

Specific PFAS	Animal Study Health Effect	Notes & Corresponding Animal Serum Concentration
Perfluorooctanoic acid (PFOA)	Increased relative liver weight	Male mouse study Duration: 14 days 4,351 ng/mL $BMDL_{10}$; Loveless et al. 2006, NJDWQI 2017
Perfluorooctane sulfonic acid (PFOS)	Delayed pup growth & development	Reproductive & transgenerational rat study Duration: 2 generations 6,260 ng/mL Modeled; Luebker 2005ab, EPA 2016
Perfluorohexane sulfonic acid (PFHxS)	Reduced litter size	Reproductive & developmental CD-1 mouse study Duration: 14 days prior to & through gestation 27,200 ng/mL NOAEL; Chang et al. 2018
Perfluorononanoic acid (PFNA)	Increased relative liver weight	Reproductive & developmental CD-1 mouse study Duration: through gestation, 17 days 4,900 ng/mL $BMDL_{10}$; Das et al. 2015, NJDWQI 2018

Perfluorooctanoic acid (PFOA) RfD Derivation

Animal Starting Point (Internal Dose and Effect)

Animal Serum Level
(Benchmark Model, NJDWQI calculation)

Increased relative liver weight, or the onset of hepatotoxicity — 4,351 ng/mL

Uncertainty Factors

Human-to-Human Variation	10
Rodent versus Human Sensitivity (assumes humans are more sensitive than mice)	$10^{0.5}$
Database Uncertainty (suspected growth & immune effects)	$\times 10^{0.5}$
Total Uncertainty Factor	100

Internal Target Serum Level →

4,351 ng/mL
÷ 100
43.5 ng/mL

Estimation of Human External Dose

Dosimetric Adjustment Factor (DAF)

Converts the internal blood dose (above) to an external (oral) dose of the chemical.

$$DAF = Vd \times \left(\frac{Ln2}{Halflife\ (days)}\right)$$

$$DAF = 0.17\ L/kg \times \left(\frac{Ln2}{\mathbf{840\ days}}\right) = 1.40 \times 10^{-4}\ L/kg\text{-}d$$

Assumed a **2.3 year half-life**

43.5 ng/mL
1.40×10^{-4} L/kg-d
× 1,000 mL/L
6.1 ng/kg-d

PFOA RfD, 6.1 ng/kg-d

Perfluorooctane sulfonic acid (PFOS) RfD Derivation

Animal Starting Point (Internal Dose and Effect)

Animal Serum Level
(No Observed Adverse Effect Level, Agreed with MDH 2019 Assessment)

Decreased immunoglobulin production, Or reduced vaccine response — 2,360 ng/mL

Uncertainty Factors

Human-to-Human Variation	10
Rodent versus Human Sensitivity (assumes humans are more sensitive than mice)	$10^{0.5}$
Database Uncertainty (suspected growth & fetal thyroid effects)	$\times 10^{0.5}$
Total Uncertainty Factor	100

Internal Target Serum Level →

2,360 ng/mL
÷ 100
23.6 ng/mL

Estimation of Human External Dose

Dosimetric Adjustment Factor (DAF)

Converts the internal blood dose (above) to an external (oral) dose of the chemical.

$$DAF = Vd \times \left(\frac{Ln2}{Halflife\ (days)}\right)$$

$$DAF = 0.23\ L/kg \times \left(\frac{Ln2}{1{,}241\ days}\right) = 1.28 \times 10^{-4}\ L/kg\text{-}d$$

Assumed a 3.4 year half-life

23.6 ng/mL
1.28×10^{-4} L/kg-d
× 1,000 mL/L
3.0 ng/kg-d

PFOS RfD, 3.0 ng/kg-d

Perfluorononanoic acid (PFNA) RfD Derivation

Animal Starting Point (Internal Dose and Effect)

Animal Serum Level
(Benchmark Model, NJDWQI calculation)

Increased relative liver weight, or the onset of hepatotoxicity — 4,900 ng/mL

Uncertainty Factors

Human-to-Human Variation	10
Rodent versus Human Sensitivity (assumes humans are more sensitive than mice)	$10^{0.5}$
Database Uncertainty (lack of multigenerational studies)	$\times 10^{0.5}$
Total Uncertainty Factor	100

4,900 ng/mL
÷ 100
49.0 ng/mL

Internal Target Serum Level →

Estimation of Human External Dose

Dosimetric Adjustment Factor (DAF)

Converts the internal blood dose (above) to an external (oral) dose of the chemical.

$$DAF = Vd \times \left(\frac{Ln2}{Halflife\ (days)}\right)$$

$$DAF = 0.20\ L/kg \times \left(\frac{Ln2}{\mathbf{1{,}570\ days}}\right) = 8.83 \times 10^{-5}\ L/kg\text{-}d$$

Assumed a **4.3 year half-life**

49.0 ng/mL
8.83×10^{-5} L/kg-d
× 1,000 mL/L
4.3 ng/kg-d

PFNA RfD, 4.3 ng/kg-d

Perfluorohexane sulfonic acid (PFHxS) RfD Derivation

Animal Starting Point (Internal Dose and Effect)

Animal Serum Level
(Benchmark Model, *under peer-review*)

Reduced litter size in female mice, 13,900 ng/mL

Uncertainty Factors

Human-to-Human Variation	10
Rodent versus Human Sensitivity (assumes humans are more sensitive than mice)	$10^{0.5}$
Duration of Exposure (14-day effect)	$10^{0.5}$
Database Uncertainty (lack of studies, fetal thyroid effects)	$\times 10^{0.5}$
Total Uncertainty Factor	300

Internal Target Serum Level →

13,900 ng/mL
÷ 300
46.3 ng/mL

Estimation of Human External Dose

Dosimetric Adjustment Factor (DAF)

Converts the internal blood dose (above) to an external (oral) dose of the chemical.

$$DAF = Vd \times \left(\frac{Ln2}{Halflife\ (days)}\right)$$

$$DAF = 0.213\ L/kg \times \left(\frac{Ln2}{\mathbf{1{,}716\ days}}\right) = 8.61 \times 10^{-5}\ L/kg\text{-}d$$

Assumed a **4.7 year half-life**

46.3 ng/mL
8.61×10^{-5} L/kg-d
× 1,000 mL/L
4.0 ng/kg-d

PFHxS RfD, 4.0 ng/kg-d

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Comparison of Reference Doses

RfDs for the four evaluated PFAS in comparison to values from other agencies.
All values below are presented in **ng/kg-d**

Specific PFAS	NHDES (01/2019) (RfD)	NHDES (06/2019) (RfD)	US EPA 2016 (RfD)	ATSDR 2018 (MRL)	EFSA 2019 (RfD)
PFOA	5.2	**6.1**	20	3.0	0.8
PFOS	8.0	**3.0**	20	2.0	1.8
PFHxS	9.3	**4.0**	-	20	-
PFNA	2.5	**4.3**	-	3.0	-

USEPA. 2016. Drinking Water Advisory for Perfluoroctanoic acid (PFOA).
USEPA. 2016. Drinking Water Advisory for Perfluorooctane sulfonic acid (PFOS).
ASTDR. 2018. Toxicological Profile for Perfluoroalkyls Draft for Public Comment. https://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=1117&tid=237
EFSA.

Exposure Assumptions

Exposure characterization considers how much PFAS is permissible given:

1. Protective assumptions about drinking water ingestion rates
2. Estimation of other non-drinking water sources of exposure.

The U.S. EPA (2016) assumed the drinking water ingestion rate of the 90th percentile of lactating women, and that 20% of exposure is permissible through drinking water (PFOA & PFOS at 70 ng/L).

These assumptions vary by state agencies, sometimes resulting in different drinking water values despite similar RfDs.

image: medium.com

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Exposure Assumptions: Initial Proposal (January 4th, 2019)

$$\frac{\text{RfD (ng/kg–day)} \times \text{Relative Source Contribution (\%)}}{\text{Water Ingestion Rate (L/kg–day)}} = \text{Maximum Contaminant Level (ng/L)}$$

Specific PFAS	Reference Dose (ng/kg-day)	Water Ingestion Rate (L/kg-day)	Relative Source Contribution	Proposed MCL (ng/L)
PFOA	These values changed in response to technical comments	These values changed in the EPA Exposure Factor Handbook (Feb 2019)	These values changed in response to technical comments	38
PFOS				70
PFHxS				85
PFNA				23

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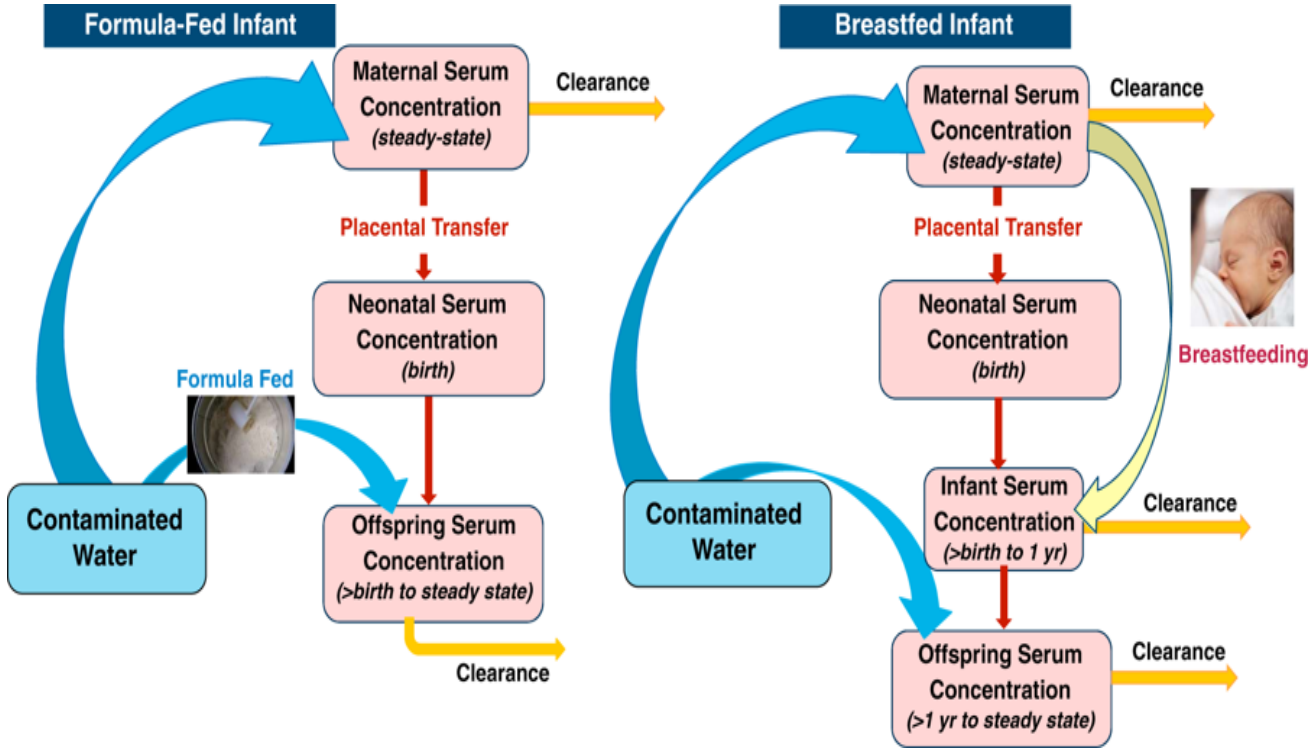
Exposure Assumptions: Example using June 2019 proposal

$$\frac{\text{RfD (ng/kg–day)} \times \text{Relative Source Contribution (\%)}}{\text{Water Ingestion Rate (L/kg–day)}} = \text{Maximum Contaminant Level (ng/L)}$$

Specific PFAS	Reference Dose (ng/kg-day)	Water Ingestion Rate (L/kg-day)	Relative Source Contribution	*Example* Drinking Water Value (ng/L)
PFOA	6.1	These values do not account for the transfer of PFAS across the placenta and into breastmilk.	50%	These values would result in unacceptable serum levels in breastfed infants.
PFOS	3.0		50%	
PFHxS	4.0		50%	
PFNA	4.3		50%	

Exposure Assumptions: Minnesota Model

What is the Transgenerational (or Minnesota) Model?



The conceptual diagram for the toxicokinetic model.
Image from: Goeden et al. (2019), *Journal of Exposure Science & Environmental Epidemiology* vol. 29, 183–195.

Excel-based model is available upon request from Minnesota Department of Health.

Human Half-life Assumptions

- NHDES applied **average** (**central tendency**) half-life estimates for PFOA (2.3 years), PFOS (3.4 years), PFNA (4.3 years) and PFHxS (4.7 years).
- NHDES did not apply the 95th percentile, or other high-end values derived from occupational exposures.

Placental & breastmilk transfer efficiencies

- NHDES applied **average** (**central tendency**) transfer efficiencies, similar to MDH and MIDHHS.

Duration of *exclusive* breastfeeding

- NHDES applied a **conservative** **12-month exclusive breastfeeding duration** for the modeled exposure scenarios.

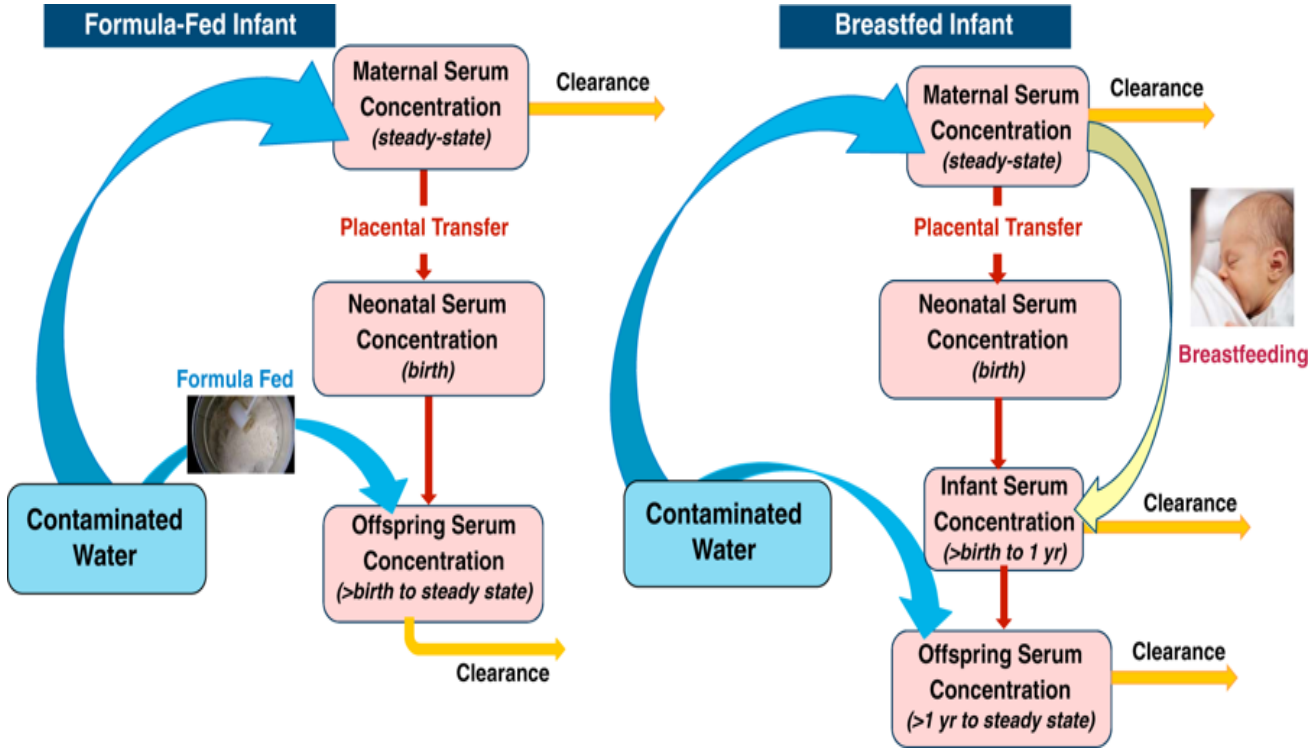
Breastmilk & water ingestion rates

- NHDES applied the **95th percentile** (**conservative**) ingestion rates for water and breastmilk across life.

Values are summarized in Table 3 of the June Report.

Exposure Assumptions: Minnesota Model

What is the Transgenerational (or Minnesota) Model?



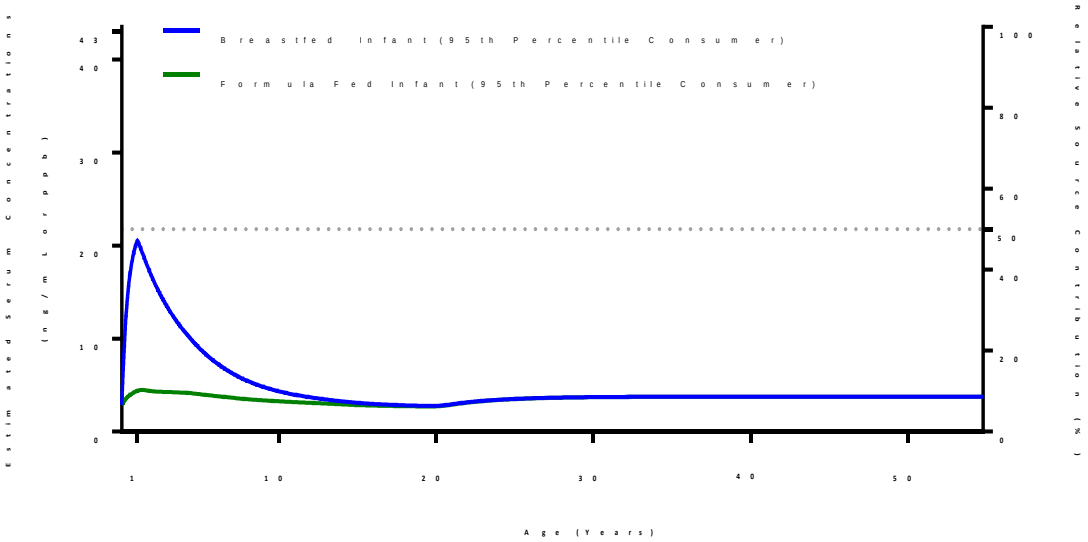
The conceptual diagram for the toxicokinetic model.
Image from: Goeden et al. (2019), *Journal of Exposure Science & Environmental Epidemiology* vol. 29, 183–195.

Excel-based model is available upon request from Minnesota Department of Health.

The model allows for the comparison of:

- **predicted blood levels (left y-axis) to**
- **the % of allowable maximum dose (right y-axis).**

Example model output for a PFOA MCL of 12 ng/L using NHDES's risk assessment assumptions.

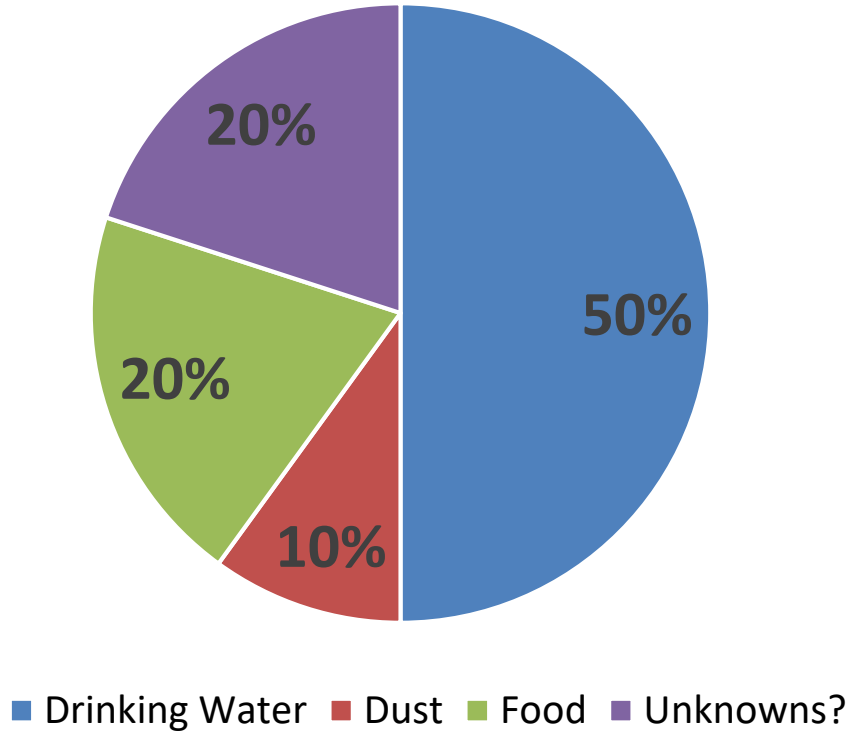


Exposure Assumptions: Relative Source Contribution

This is **how we "budget" the daily dose (RfD)** for water versus non-drinking water sources of exposure.

- **20%** - Low and the default EPA recommendation when "we don't know". Results in the most restrictive MCL.
- **50%** - Consistent with values derived from NHANES to estimate background
- **80%** -Results in a higher MCL value and assumes that other sources are not contributing to exposure (20% or less).

Relative Source Contribution
(example below for visualization purposes)



Exposure Assumptions: Relative Source Contribution

20%

U.S. EPA (2016)
- **20% RSC for PFOA & PFOS** for the lifetime health advisory of 70 ng/L, based on RfDs of 20 ng/kg-d.

Vermont - VTDOH (2016-2017)
- **20% RSC across all** for health-based screening values (HBSVs).

New Jersey - NJDWQI (2017-2018)
- **20% RSC for PFOA & PFOS** because of insufficient serum data (proposed MCL).
- **50% RSC for PFNA** because of sufficient serum data from NHANES and a NJ community (MCL).

New York - NYDWQC (2018)
- **≤60% RSC for PFOA & PFOS** recommendation based on serum data (proposed MCL).

Minnesota - MDH (2017-2019)
- **50% RSC for PFOA, PFOS & PFHxS** in their model for (HBSVs).

50-60%

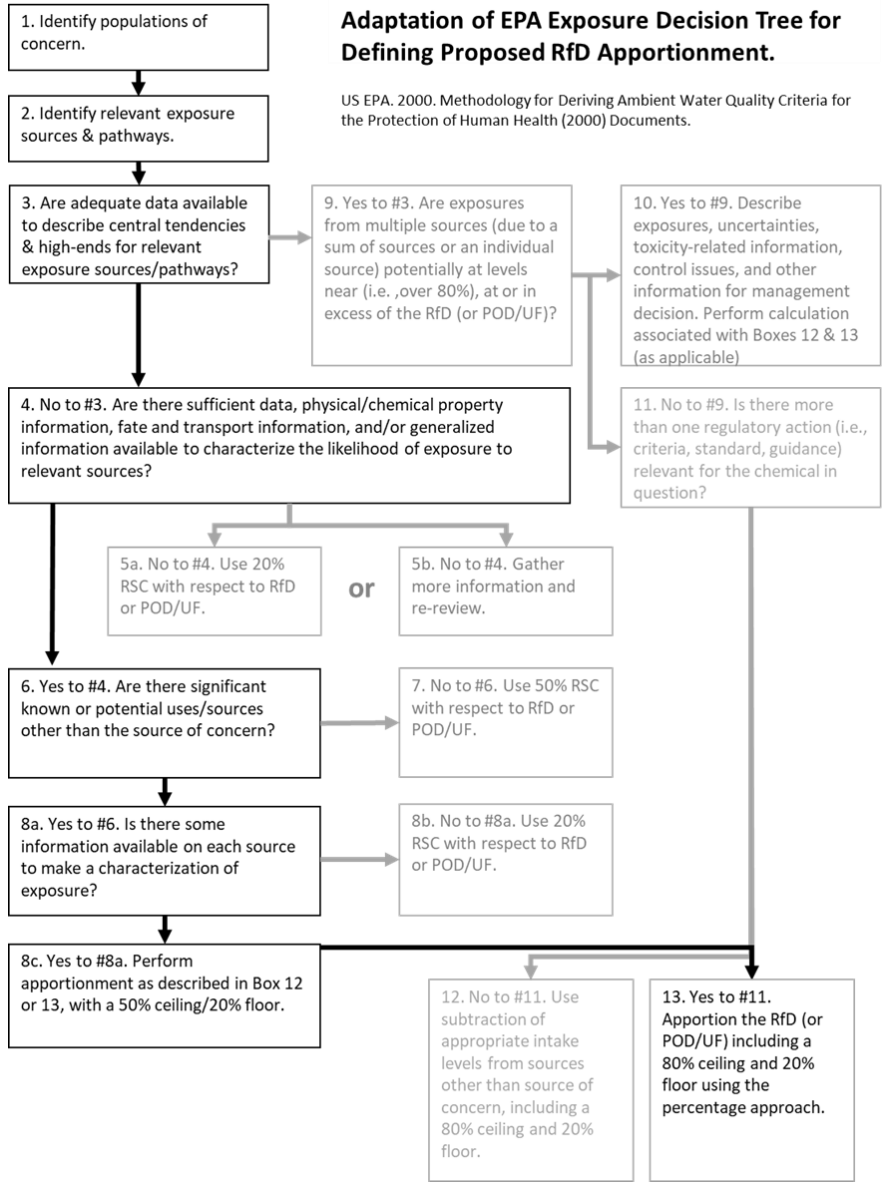
Michigan - MIDHHS (2019)
- **50% RSC for PFOA, PFOS, PFNA & PFHxS** in MDH's transgenerational model (HBSVs).

How did the NHDES MCLs arrive at a 50% RSC?

Exposure Assumptions: Relative Source Contribution

NHDES referred to the EPA Decision Tree for determining the relative source contribution.

Arrived at a **50% ceiling** combined with apportionment (**subtraction method**) to derive chemical specific RSCs.



US EPA. 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

Exposure Assumptions: Relative Source Contribution

In the initial proposal, NHDES estimated "background" using existing blood data. However, this value should reflect the typical non-drinking water exposures.

Used the EPA subtraction method:

$$\frac{\text{Target serum level (ng/mL)} - \text{Population background (ng/mL)}}{\text{Target serum level (ng/mL)}} = \text{RSC}$$

Using the NHANES (average) for PFOA:

$$\frac{43.5\ \text{ng/L} - 1.8\ \text{ng/L}}{43.5\ \text{ng/L}} = 0.96 \text{ or } 96\%$$

Using Adults from Southern NH (95th percentile) for PFOA:

$$\frac{43.5\ \text{ng/L} - 26.6\ \text{ng/L}}{43.5\ \text{ng/L}} = 0.39 \text{ or } 39\%$$

The use of the *NH-specific data likely overestimates* the background (non-drinking water) exposure.

But, the current lack of regulations on PFAS means an 80% RSC, especially for adults, is inadequately protective.

US EPA. 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

Exposure Assumptions: Relative Source Contribution

Estimation of RSC by Subtraction Method Using NH-specific data

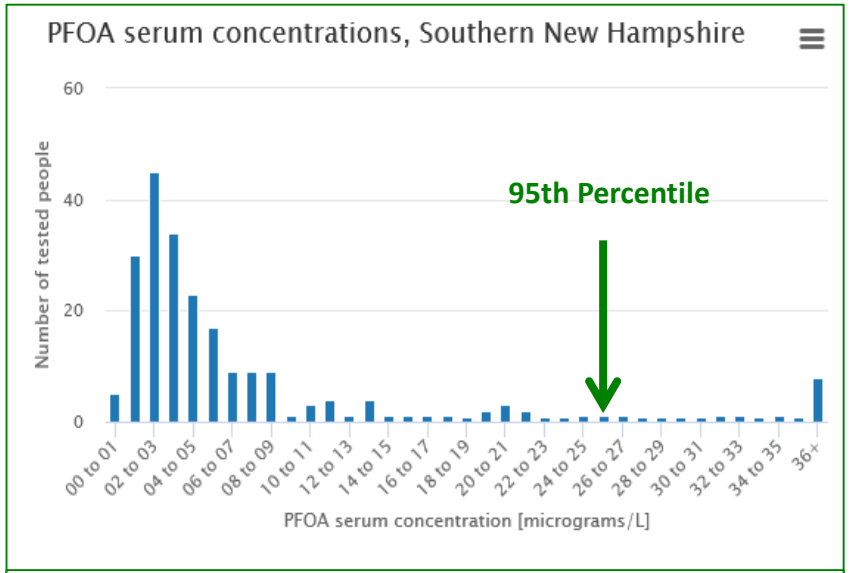
Subtraction method applied to all 4 PFAS using blood data collected by NH Dept. Health & Human Services from highest exposed populations.

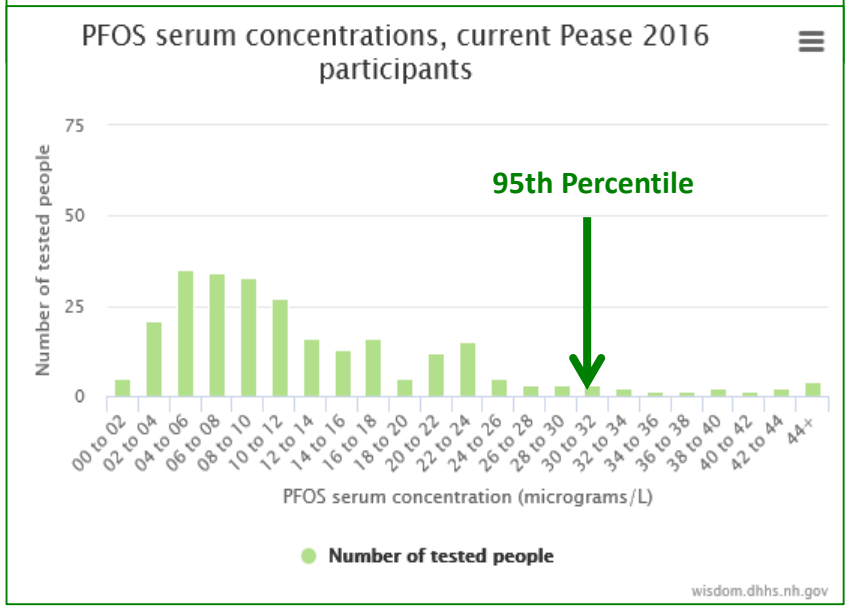
Used NH-specific PFAS blood concentrations:

	Geometric mean	95th Percentile
PFOA*	4.40 ng/mL	**26.6 ng/mL**
PFOS**	10.2 ng/mL	**31.7 ng/mL**
PFHxS**	4.50 ng/mL	**26.0 ng/mL**
PFNA	0.66 ng/mL	**1.70 ng/mL**

* **PFOA** concentrations from exposed population in Merrimack (217 participants) & Southern NH (219 participants).

** **PFOS** & **PFHxS** concentrations from exposed population in Pease, NH (256 participants).





NH Wisdom Portal, Per- and Polyfluoroalkyl Substances (PFAS) Blood Testing and Community Exposure: https://wisdom.dhhs.nh.gov/wisdom/#TopicGroup_8203D9F1281247419C5C417B8E591CE7

Exposure Assumptions: Relative Source Contribution

Estimation of RSC Using NHANES data

RSC estimates using the NHANES 2013-2014 dataset (summarized by Daly et al. 2018):

- **geometric mean (GM) and**
- **95th percentile.**

NHANES data more likely to reflect background exposure levels from non-drinking water sources.

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**

Modeled Exposures & Proposed MCLs

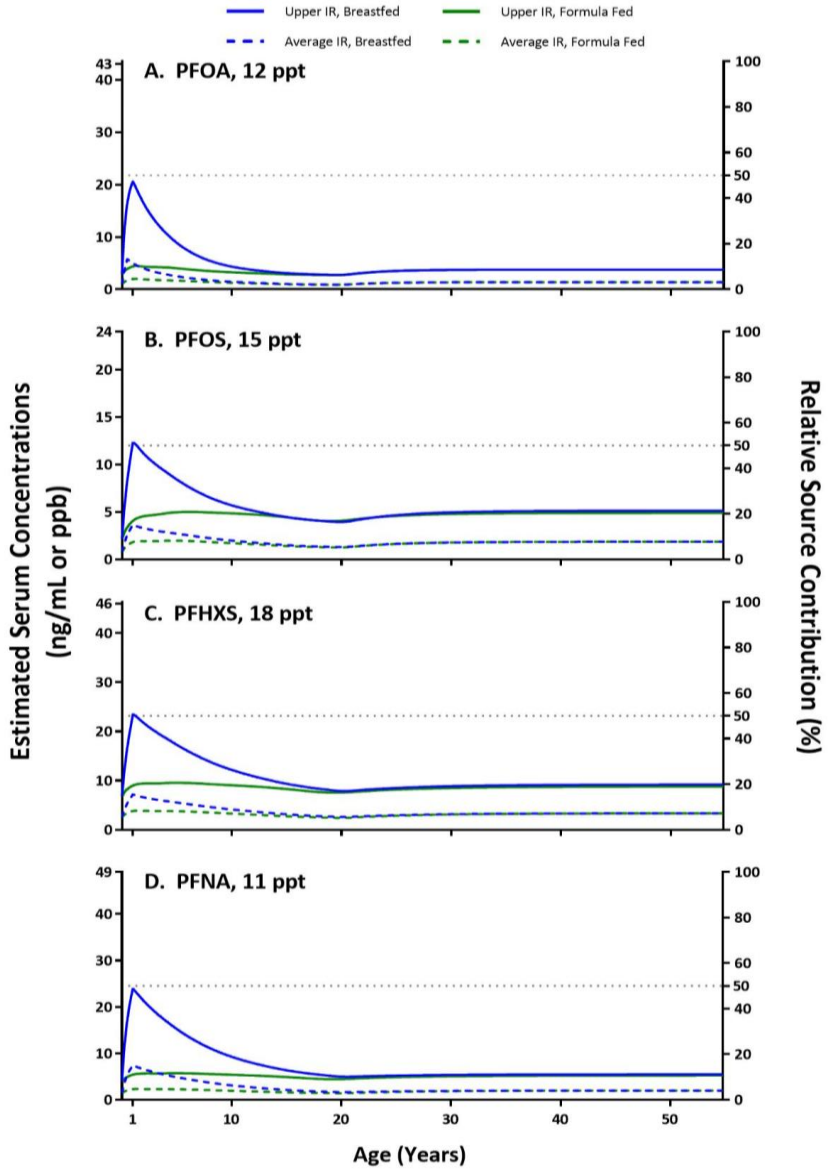
Given these **reference doses** and **exposure assumptions**, the proposed MCLs/AGQS are:

PFOA	**12 ng/L**
PFOS	**15 ng/L**
PFHxS	**18 ng/L**
PFNA	**11 ng/L**

Because of the unique properties of PFAS, accounting for breastmilk transfer is necessary.

The 50% RSC (upper limit) protects children from additional exposures to from other non-drinking water sources of PFAS.

Thus, these proposed MCLs are protective across all life stages for associated chronic health outcomes.



Modeled Exposures & Proposed MCLs

Where was NHDES conservative in its health-based risk assessment?

Central Tendency Assumptions	Conservative (High-End) Assumptions
1. Application of Uncertainty Factors (see page 23 of the June Technical Report)	1. Accounting for breastmilk & placental transfer in a drinking water standard (MDH model)
2. Human half-life estimates (average values)	2. 95th percentile water consumptions rates, *throughout life*
3. Placental & breastmilk transfer estimates (average values)	3. Assumed 12-month exclusive breastfeeding period
4. Individual MCLs specific to each compound instead of a class-based MCL.	4. Assuming 100% absorption in GI tract
5. Relative Source Contribution cap of 50%*	5. Relative Source Contribution cap of 50%*

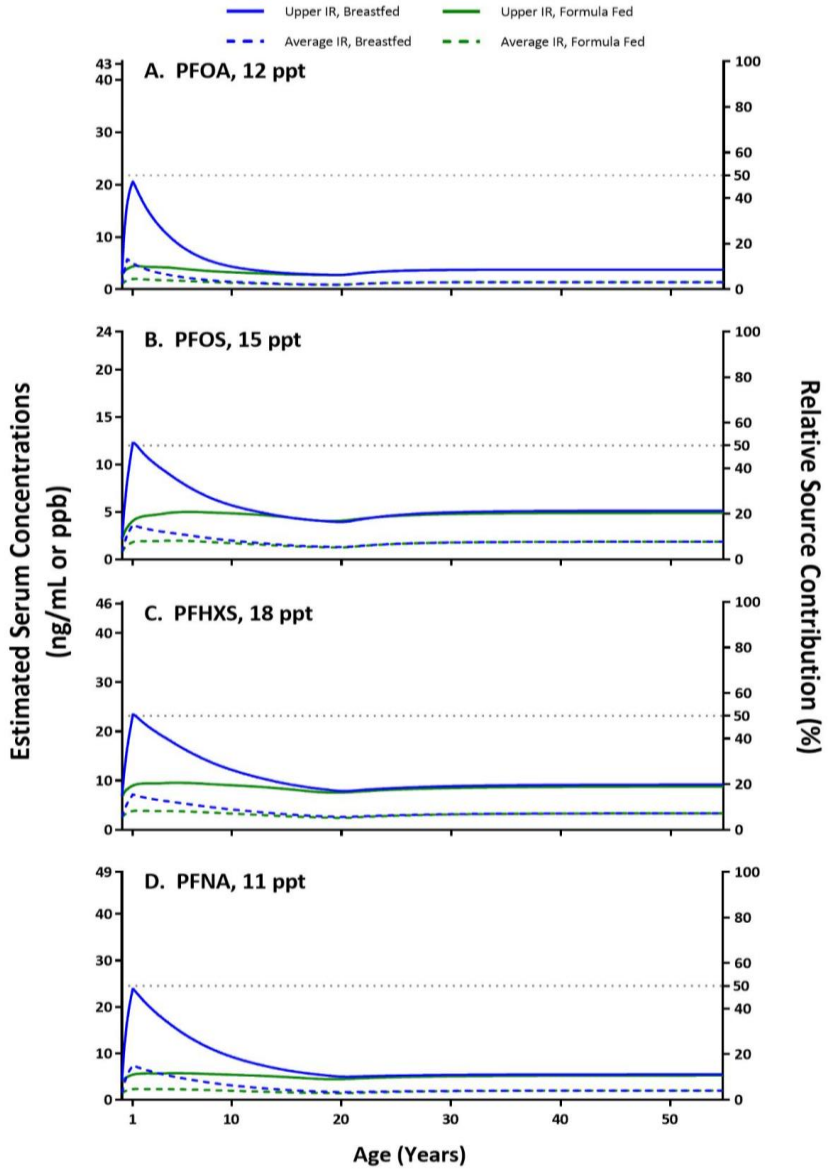
Modeled Exposures & Proposed MCLs

Given these **reference doses** and **exposure assumptions**, the proposed MCLs/AGQS are:

PFOA	**12 ng/L**
PFOS	**15 ng/L**
PFHxS	**18 ng/L**
PFNA	**11 ng/L**

NHDES is *currently* not recommending a class- or subclass-based approach to regulating PFAS.

NHDES is committed to continuing to review the scientific literature for advances in risk assessment for these and other PFAS.



Questions

References and Supporting Documents can be found in the Reference List of the June 2019 Technical Report:
https://www.des.nh.gov/organization/commissioner/legal/rulemaking/documents/pfas-scr-attch-1-w-ltr.pdf

Technical Questions about this presentation can be submitted to the **NHDES Permitting & Environmental Health Bureau**:

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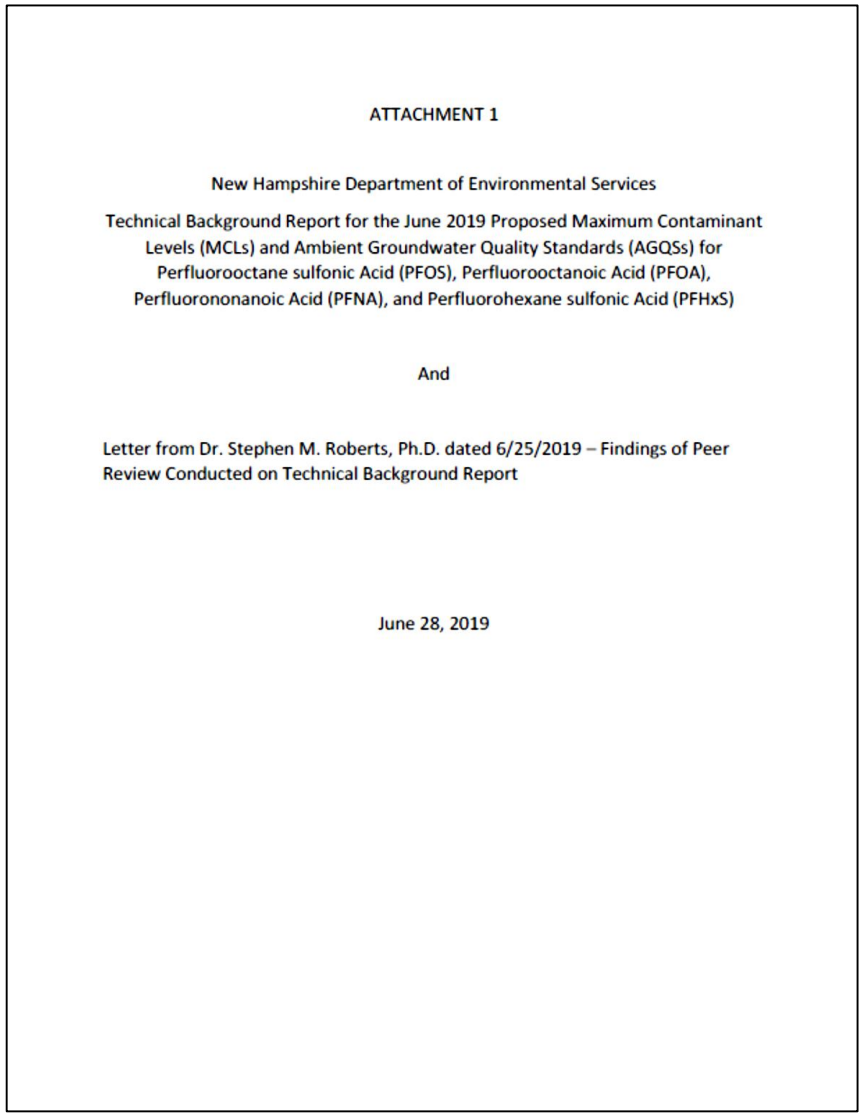

ATTACHMENT 1

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