

U.S. Environmental Protection Agency National Drinking Water Advisory Council (NDWAC) Virtual Public Meeting August 8, 2023

Meeting Summary

Opening

Ms. Elizabeth Corr, the Designated Federal Officer (DFO) for the NDWAC, opened the meeting. She introduced NDWAC Chair Lisa Daniels.

Chair's Welcome and Council's Introductions

Ms. Lisa Daniels, NDWAC Chair, welcomed participants to the meeting and reviewed the meeting agenda. She asked the NDWAC members and the Council's Centers for Disease Control liaisons to introduce themselves.¹ She then introduced Ms. Yu-Ting Guilaran, Deputy Director of EPA's Office of Ground Water and Drinking Water.

Office of Ground Water and Drinking Water's Welcome

Ms. Guilaran thanked Ms. Daniels and council members. She expressed appreciation for council members' robust participation during the April 2022 meeting where EPA consulted with the council on the proposed National Primary Drinking Water Regulation (NPDWR) for PFAS and looked forward to hearing more input during this meeting. Ms. Guilaran also expresses appreciation for the NDWAC's work in the past year regarding other rule making efforts by EPA, specifically Lead Copper Rule Revisions (LCRR), Lead and Copper Rule Improvements (LCRI), and Microbial and Disinfection Byproducts (MDBP) Rule Revisions.

Ms. Guilaran also acknowledged EPA's ongoing efforts related to addressing lead in drinking water, including plans to propose the LCRI in Fall 2023 and work that supports implementation of LCRR (e.g., EPA's June 2023 informational tool "Fact Sheet for Developing and Maintaining Lead Service Line Inventory" that serves as a companion to the August 2020 "Guidance for Developing and Maintaining a Service Line Inventory" and EPA's June 2023 document "Developing and Maintaining Service Line Inventory Small Entity Compliance Guide" focused on supporting small water systems in complying with LCRR initial inventory requirements).

¹ Please see Appendix A for the list of NDWAC members and liaisons. Note that NDWAC member Mac Underwood and Centers for Disease Control liaison Dr. Vincent Hill did not attend the meeting.

Ms. Guilaran also discussed some of EPA's work with states and other partners to implement Bipartisan Infrastructure Law (BIL funding provisions for the Drinking Water State Revolving Fund (DWSRF) including \$15 billion over 5 years for lead service line identification and replacement, which may also be funded using general supplemental DWSRF funding; and described funding allotments reflecting best available national and state level projections of service line counts based on information on lead service lines collected as part of EPA's 7th Drinking Water Infrastructure Needs Survey and Assessment (DWINSA). Ms. Guilaran described the Lead Service Line Replacement Accelerators initiative where EPA will partner with four states (Connecticut, Pennsylvania, New Jersey, and Wisconsin) and will work directly with 40 communities across these states to provide hands-on support to guide the communities through the lead service line replacement process.

Ms. Guilaran mentioned her appreciation of the NDWAC's effort with respect to the Council's MDBP Rule Revisions Working Group. In November 2021, EPA asked the NDWAC for advice and recommendations on potential revisions to certain MDBP rules. Ms. Guilaran provided an overview of the working group, formed in the Spring of 2022, which includes five NDWAC members and two members of EPA's National Environmental Justice Advisory Council (NEJAC), and mentioned that EPA hosted an in-person meeting of the working group in Washington, DC. in June 2023. She flagged an upcoming meeting between the NDWAC and the working group, following which the working group plans to send a report to NDWAC members. Ms. Guilaran noted that Ms. Daniels would provide a more complete update later in the meeting.

She transitioned to discuss the PFAS NPDWR, stating that the public comment period ended in May 2023 with the receipt of around 120,000 comments and noting that EPA's ambitious goal is to promulgate the PFAS NPDWR by the end of the calendar year. Ms. Guilaran noted that the BIL includes an investment of more than \$15.7 billion over 5 years in DWSRF funding that could be used in addressing PFAS and other emerging contaminants in drinking water systems.² The DWSRF Emerging Contaminants funds must be distributed to communities as entirely forgivable loans and grants, and the states are not required to provide a match for these funds. In addition to the DWSRF program, the BIL also established EPA's Emerging Contaminants in Small or Disadvantaged Communities (EC-SDC) grant program to provide funding to states and territories to assist public water systems in small or disadvantaged communities to address emerging contaminants including PFAS in drinking water.³ In February 2023, EPA announced the availability of \$2 billion for the EC-SDC grant program for the two years of FY2022-2023.

Ms. Guilaran again thanked the NDWAC members for all the work they have done for the Agency in these priority areas and their engagement during this busy season, including their engagement during this meeting.

Ms. Corr thanked Ms. Guilaran and opened the public comment portion of the meeting.

² The \$15.7 billion comprises \$11.7 billion in BIL DWSRF General Supplemental funds and \$4 billion in BIL DWSRF Emerging Contaminants funds.

³ The BIL provides for \$5 billion over 5 years for the EC-SDC grant program.

Public Comment to the NDWAC

NDWAC DFO Elizabeth Corr introduced commenters, who provided oral comment to the council as follows.⁴ Chris Moody provided verbal comments during the meeting and also submitted written comments. To view the written comments, please refer to Appendix D.

Karen Spencer provided verbal comments during the meeting and also submitted written comments. To view the written comments, please refer to Appendix D.

Linda Dell provided verbal comments during the meeting and also submitted written comments. To view the written comments, please refer to Appendix D.

Sean Lammerts provided verbal comments during the meeting and also submitted written comments. To view the written comments, please refer to Appendix D.

Rik Hull provided verbal comments only. Mr. Hull represented the National Association of Water Companies (NAWC) and wanted to discuss the MDBP Rule Revisions Working Group. As he understands, some of those discussions were around disadvantaged communities and how consolidation of systems may help them deal with DBPs. He hoped all forms of consolidation are being considered (public-public; public-private; private-private). Mr. Hill expressed that in order to address environmental justice concerns it is important that under-resourced communities have the ability to assess all options and added this may require additional funds to hire engineers and lawyers to fully assess options. He stated that NAWC would fully support the use of technical assistance grants or SRF grants for this purpose and that they would want to be part of any solutions and urge the council to take a holistic look at this issue.

Ms. Corr thanked the commentors for their contributions.

Consultation on Final National Primary Drinking Water Regulation (NPDWR) for per- and polyfluoroalkyl substances (PFAS)

Ms. Daniels introduced the EPA speakers for the consultation for the final PFAS rule,).

Dr. Ryan Albert, who supervises the Standards and Risk Reduction Branch (SRRB) in EPA's Office of Ground Water and Drinking Water, thanked everyone for their participation and stated that the purpose of this presentation was to provide the NDWAC with information on the proposed PFAS NPDWR, which was proposed in March 2023, and to consult with the NDWAC, as required under the SDWA, prior to proposing final regulation by the end of 2023. Dr. Albert's portion of the presentation included a discussion of background information on PFAS and SDWA, as well as certain requirements of the proposed PFAS NPDWR. Please see Appendix B for EPA's presentation.

PFAS Mixtures

As part of the presentation, Dr. Albert summarized input from NDWAC members on PFAS mixtures that was shared at the consultation for the proposed rule in April 2022. He then opened the meeting for questions and discussion.

Ms. Shellie Chard asked if EPA had considered waste streams that are generated from treatment processes and how that plays into affordability. She wondered if other technologies need to be

⁴ The NDWAC also received comments provided in written form only. Please refer to Appendix D.

investigated for disposal of this waste. Reverse osmosis may not be feasible, and while granular activated carbon (GAC) can be effective, it is unclear how to properly dispose of or regenerate it. She asked if all these points have been a part of EPA discussions.

Dr. Albert stated that these concerns have been considered. While PFAS are not considered hazardous waste under current regulations, several stakeholders noted that it could be treated as hazardous in the future, or that facilities will want to treat it as hazardous to limit liability. For the proposed rule, EPA conducted a sensitivity analysis looking at the national level increase in costs if PFAS were to be treated as hazardous. It was estimated that this treatment would cost another \$30-60 million per year nationally. EPA offered to provide results from this analysis following the meeting; results can also be found in Appendix N of the Proposed Rule Economic Analysis. EPA is considering public comment on this topic prior to rule finalization.

Mr. Jeffery Szabo had concerns about the hazard index and being able to clearly emphasize its' importance to the public. Providers often try to educate the people about maximum contaminant levels (MCLs). Mr. Szabo asked what the EPA plans to do to partner with these groups to explain what the hazard index means.

Dr. Albert agreed with the importance of this outreach and responded that while there are no final plans for outreach, EPA is taking suggestions for identifying needs EPA is aware that these are novel concepts for stakeholders, and it will be beneficial to have risk communication tools. Dr. Albert referenced a Hazard Index calculator as an example of a risk communication tool that can be produced, and this is something that will be considered if the regulation is finalized.

Mr. Jim Proctor was concerned about the use of the hazard index in this context, as the EPA has never used it in this context before. He believed that this dose additivity model⁵ has a tendency to overstate the volume of PFAS in water because it is difficult to apply to a family of chemicals instead of a single chemical. Mr. Proctor asked what EPA will do to consider the overestimate.

Dr. Albert, acknowledging that while he does not have a background in health science, provided that the general Hazard Index approach was presented to the Science Advisory Board (SAB) and received generally positive feedback. Dr. Albert noted that the approach does not necessarily account for too much PFAS but is a health protective approach that ensures EPA puts forward a Maximum Contaminant Level Goal (MCLG) that is conservative. Dr. Albert stated that there have been a considerable number of public comments on this approach, and they are being considered in the final rule.

Mr. Proctor had a follow-up question on the toxicity analysis. EPA typically recommends using human rather than animal data; however, these levels appear to have been based on animal data. Mr. Proctor wondered if this was consistent with Agency policy.

Dr. Albert stated that human epidemiological data were used in the development of the MCLG for PFOA and PFOS. For the health assessments, EPA considered SAB input prior to proposing the PFAS NPDR in March. Epidemiology data were also used for GenX and PFBS. However, for PFHxS and PFNA, the best available science at the time of proposal were the Agency for Toxic Substances and Disease Registry (ATSDR) assessments, which were based on animal data. Dr. Albert noted that this is consistent with EPA policy to use the best available science.

⁵ EPA presenters heard "dose additivity model" to mean "dose additivity assumption using a Hazard Index."

Ms. Quirk added that the hazard index has been discussed so much because it is new. She noted that the Hazard Index is using values for four compounds that do not always impact the same health effects. Ms. Quirk asked how EPA combines different health effects with different outcomes into the same calculation.

Dr. Albert stated his understanding that these PFAS impact multiple health endpoints so this is not an issue where the six PFAS only have one impact on one endpoint. The hazard index approach focuses on the most sensitive health endpoints for each of the individual PFAS within the Hazard Index. EPA believes that this is an appropriate way to regulate PFAS and mixtures with respect to health and to derive the MCLG, which is set at a level where there are no adverse health effects. Dr. Albert acknowledged the number of public comments made on the hazard index and that these comments are being considered prior to the final regulation.

Ms. Alexandra Campbell-Ferrari asked Dr. Albert to explain more about factors that impact the difference between MCLG and MCL. What is motivating the difference between those two numbers?

Dr. Albert responded that the MCLG is solely based on health information. It is a goal that is fully health protective with an adequate margin of safety. However, it is not always feasible to reach the MCLG. The MCL takes into consideration the availability of analytical methods, the practical quantitation limit (PQL), treatment, and cost. In the case of PFOA and PFOS where there is a difference between the MCL and MCLG, it would be technically challenging and extremely expensive to achieve a level of zero with available analytical methods and treatment. The MCL of 4.0 ppt is a feasible level that is as close to the MCLG of zero as possible. EPA is considering comments on where to set the MCL prior to finalizing regulation.

Ms. Jana Littlewood explained that she assists small water systems (e.g., condo associations and homeowners' associations serving between 12 - 300 homes) with coming into compliance with EPA regulations. A consensus among these systems is that EPA is not hearing their concerns. There has been a lot of public testimony related to four main concerns. The first is that the PFOS and PFOA MCLs are too low and it is not feasible to come into compliance in a cost-effective manner. Next, the hazard index is confusing. As someone who frequently explains concepts to customers such as the MCL and MCLG, the challenge of explaining the hazard index will be overwhelming. Third, PFAS mitigation will be more costly than EPA has estimated. There have been a lot of on-the-ground situations where states have required systems to come into compliance and the costs are overwhelming. Finally, the timeframe to come into compliance is not achievable. Ms. Littlewood was unsure if the three-year timeframe is set by the SDWA or if there was any flexibility. She wondered if EPA would consider all of these concerns prior to finalization of the rule or if it is too late to make changes. In addition, Ms. Littlewood asked if EPA would consider waiting for the data from the fifth Unregulated Contaminant Monitoring Rule (UCMR 5) to be released to see what those levels are. There is a lot of angst for small water systems, where the cost per customer to come into compliance with the regulation will be overwhelming.

Dr. Albert thanked Ms. Littlewood and added that EPA has received feedback on these topics through the public comments process. EPA is considering the points being raised and conducting additional technical analyses. Changes based on public comment considerations will be documented in the federal register and public comment document.

Ms. Yolanda Barney expressed her appreciation to EPA for sharing this information, saying that it is important to keep people informed. Ms. Barney asked that EPA figure out how to address PFAS

contamination in source water. She wondered if it could be handled through something like a chemical waiver.

Mr. Eagle Jones followed up on Ms. Littlewood's question by asking why EPA is not waiting for UCMR 5 data before making a final decision.

Dr. Albert answered that EPA is fortunate to currently have good quality data. Although UCMR 5 will be very useful, there are UCMR 3 data available for six PFAS, as well as a lot of state data for up to 29 different PFAS. To evaluate the current data, EPA developed a robust model to estimate national occurrence for the proposal. Dr. Albert reiterated that EPA is considering comments, including those related to PFAS occurrence, prior to finalize the rule. However, there is significant public interest at multiple levels to finalize the rule as soon as possible. If EPA were to wait for all UCMR 5 data to be available, there would be a multiple year delay. Based on public comments received, that delay would cause additional concern.

Dr. Arthur Chang added that the CDC and ATSDR have a lot of health information on their website. He noted that the CDC posts a lot of data on their site (both human/epidemiological and animal data), which can make it hard to distinguish and interpret. The hazard index is what PFAS scientists use to understand PFAS that do not have a lot of animal or human data, and that it is an acceptable tool. The CDC has a new tool on their website that predicts blood level concentrations based on levels of different PFAS. The tool can be used to find blood levels based on the proposed regulatory limits. The blood level data are collected as part of the National Health Nutrition Examination Survey (NHANES) and is the best human health data available.⁶

Dr. Albert thanked Dr. Chang. He noted that the current discussion is focused on the overview and background, health, and hazard index information. Ms. Greene will be providing further information about treatment and monitoring later in the presentation.

Mr. Szabo thanked Ms. Littlewood for her earlier comments. Next, he stated that using the hazard index as drafted without specifically knowing what EPA is going to do to help communicate the information to the people being served would cause mass confusion and would be detrimental on a daily basis. Without knowing what the commitment related to communications support is going to be and how to properly educate the people served, he thought it could be disastrous.

Dr. Albert thanked Mr. Szabo for his input in emphasizing the importance of effective communication. He explained that EPA has developed a two-page factsheet that explains the hazard index and acknowledged if the hazard index is finalized, risk communication will be exceptionally important.

Ms. Daniels asked about EPA's determination that the PFOA and PFOS MCLs of 4.0 ppt are feasible when the MCL is equal to the PQL. She suggested that the PQL could be the Detection Limit (DL) or the Reporting Level (RL). She offered that in her experience there may be confidence at those levels but considering the quality assurance/quality control (QA/QC) requirements takes us over the PQL level of 4 ppt in terms of the confidence in the result. She further noted that +/-thirty percent of 4 ppt would add another 1.2 ppt. Ms. Daniels expressed her concern about accuracy and whether the QA/QC data supports these low levels. She stated that she thinks that this would be the first time EPA is setting an MCL at the DL. She also wondered about implementation as there is usually time between when the

⁶ Dr. Arthur Chang provided the following link in the chat: <https://www.atsdr.cdc.gov/pfas/resources/index.html>.

water supply would detect the contaminant before generating an MCL violation. She asked Dr. Albert to describe the thought process for determining that the 4 ppt was a feasible MCL.

Dr. Albert thanked Ms. Daniels and responded that the PQL is based on UCMR 5 and is almost interchangeable with the RL. It is the level where we have a high degree of confidence but it is not the lowest level to which the contaminant can be detected. The DL is actually quite a bit lower than the RL or the PQL and is lab-specific. Based on previously observed data, it can be anywhere from 3 to 10 times lower than the RL. Utilities will have the ability to report the absence or presence for PFAS well below the PQL. In her portion of the presentation, Ms. Greene will talk about monitoring provisions and how compliance is calculated. Compliance is determined by a running annual average (RAA) calculation and not an instantaneous level. If there are three samples below the PQL and one that is slightly above it, that system would still be in compliance with the regulations. In response to the second question, Dr. Albert stated that this is the part of this rule that is not “novel.” EPA has a history of setting regulations at the PQL; it has been done with other NPDWRs in the past.

Ms. Daniels added a follow-up comment about the practicality of the data that has been observed, saying that labs struggle to get to the 4 ppt number and that she is concerned.

Dr. Albert thanked everyone for their questions and feedback, noting that NDWAC and public input is very important prior to rule finalization.

Ms. Daniels transitioned to Ms. Greene’s portion of the presentation.

Ms. Greene thanked Ms. Daniels for the introduction. She explained that she planned to cover topics related to monitoring, treatment technologies, and the economic analysis performed for the PFAS NPDWR.

Monitoring

Ms. Greene presented information related to the proposed NPDWR monitoring requirements. As part of the presentation, she summarized comments from NDWAC members on monitoring considerations shared at the proposed rule consultation in April 2022. Following her presentation, she opened the discussion for monitoring-related questions and comments.

Mr. Scott Borman asked a question with regard to consecutive systems and wholesale systems. If the parent system does not have any PFOA/PFOS issues, is there a potential for those consecutive systems to have waivers? This is not a DBP situation where they can increase in the distribution system. If PFAS are present, they will be in the source water. Mr. Borman asked if this situation would be a candidate where waivers could be issued, as these systems are 100% supplied by another system.

Ms. Greene responded that EPA has seen this concern as part of the public comments and it will be addressed in their responses to those comments. She acknowledged that there may be some rationale for having consecutive systems considered differently with respect to PFAS NPDWR requirements.

Mr. Borman stated that his concern relates to precision and bias of the testing, especially with Method 533. He expressed that because the MCL is low, he sees potential for a parent system to have no PFAS detected while the consecutive system has a violation due to the inconsistency with the methods. He stated that this is a concern that he has heard from many consecutive systems. They fear that they could inadvertently be hit with a violation because the numbers are so tight related to the methods and the RLs.

Ms. Daniels stated that the trigger levels are so low and are so much below the PQL, that she wonders about the feasibility of using the trigger level. She understands that those levels do not constitute a violation, but they trigger action. She asked if this is a legally defensible number if it is so far below the MCL. Ms. Daniels stated that with nitrate (an acute contaminant), a detection above 50% of the MCL would trigger increased monitoring. She wondered how EPA justified a lower trigger level of 1/3 the MCL for a chronic contaminant. Is this trigger level similar to nitrate or is it meant to be more of a “reliably and consistently” below criteria to allow a system to stay on reduced monitoring?

Ms. Greene responded by saying EPA received comments on quantitation and trigger levels. EPA is evaluating available data on what systems and states can achieve to help to determine how to proceed in the final rule. Trigger levels are meant to be for presence / absence to trigger increased monitoring and not an MCL violation which EPA believes is consistent with the standardized monitoring framework (SMF). EPA appreciates the public comment received, as well as the work being conducted in Pennsylvania to inform EPA as far as what levels can be achieved.

Mr. Jones stated that in Ms. Greene’s presentation he had seen an example of a 2.0 ppt result that would be considered as zero, and how that is related to the trigger level.

Ms. Greene explained that the reference is not related to the trigger level, but rather to the compliance calculation. As proposed, if a value for one of the regulated PFAS is below its specific PQL, which is different depending on the individual PFAS, then that value would be zero in the RAA compliance determination calculation. Since 4.0 ppt is the PQL for PFOA/PFOS, if a measured value is 2.0 ppt, then that value would use zero in the compliance calculation.

Mr. Steve Elmore added his concern on levels of PFOS and PFOA just below 4.0 ppt (e.g., in the range of 3.9) that would come up as zero in the RAA calculation. The system would meet the RAA of 4.0 ppt when in reality it may have two or three samples greater than 4.0 ppt but by averaging those samples with zero, the RAA would be 3.9 ppt which is not addressing the public health concern, particularly when the MCLG is zero. Mr. Elmore stated that this is a concern especially if labs can report reliably down to as low as 0.5 ppt as mentioned earlier. Throwing out results below 4.0 ppt, the public health impact is not adequately measured and the accuracy of the labs is not honored. Mr. Elmore also stated that lab capacity is also going to be a significant concern. In Wisconsin this year, all the public water systems have been testing for PFAS and there has been major capacity issues, even when drawing on labs from outside the state. It is difficult to get all samples run within a year.

Ms. Greene thanked Mr. Elmore for providing those perspectives.

Ms. Daniels shared Mr. Elmore’s concerns about lab capacity, noting that this is not only a concern for the drinking water world, pointing out that we will have 50,000 new systems monitoring drinking water which could identify more contamination sites and could lead to more surface water and ground water monitoring. She suggested that will mean much longer turnaround times and shared her observation that currently systems must wait three to four weeks to get sample results back, adding that there are only seven or eight states with their own MCLs. She also noted that long turn-around times cause implementation challenges related to treatment efficacy. Lastly, if EPA finalizes the rule with a low level of 4.0 ppt as an MCL, they will have to consider whether or not labs will have to routinely report all QA/QC data to ensure QA has been met. This is not something that states routinely review. Ms. Daniels wondered as we are moving towards a new Federal data system and electronic reporting, have we thought about what it would mean for the entire data package to be reported by the lab?

Mr. Jones asked whether EPA has considered the sampling challenges had by small water systems; collecting these samples is extremely sensitive and not following sampling techniques could result in inaccuracies. Mr. Jones stated that he has seen situations before where samples were collected by two neighboring agencies that came back with different results when similar sampling practices were not followed. If water systems are required to collect these samples, there must be funding for training and teaching operators the proper sampling techniques. Errors could result in misinformation. These are very unique samples that need to be collected.

Ms. Greene stated that EPA is considering all of the technical assistance, implementation guidance, and other materials to ensure successful implementation.

Public Notification

Ms. Greene presented information related to the proposed public notice requirements. As part of this information, she summarized comments from NDWAC members on public notification considerations provided during the April 2022 NDWAC consultation. Following her presentation, she opened the discussion for monitoring-related questions and comments.

Mr. Szabo asked for confirmation in his understanding that the public notification would not be for violating an MCL but would be required within 30 days.

Ms. Greene stated that if the rule is finalized as proposed, if a system monitored and their RAA was in violation of the MCL, then the water system would be required to notify the public that the violation has occurred.

Mr. Szabo asked how the Agency would handle that.

Ms. Greene responded that it would be the same procedures as under the Public Notification (PN) rule.

Mr. Szabo asked if the violation would also need to be reported in the annual Consumer Confidence Report (CCR).

Ms. Greene responded yes; violations would need to be published in community water systems' CCRs.

Mr. Szabo is concerned with the short time period for agencies to install treatment, making it tough for systems to meet the proposed deadline. Mr. Szabo also stated that there will be numerous notifications of the public which may confuse the already alarmed public.

Ms. Greene responded that, as proposed, EPA is following the PN rule and consumers would need to be notified of the violation.

Mr. Szabo stated that he is considering the real-world ramifications of the proposal. If a system is sampling and working with a local regulator, Mr. Szabo thinks that there will be a tremendous cost burden to put treatment in place. There will be mass confusion too. It will muddy the water regarding informing the public, as well.

Ms. Campbell-Ferrari stated that from a community perspective, the faster the notifications can go out, the better. It is better for public trust. These regulations are going to be new to everyone and recommended that an educational notification be included as part of the CCR to inform customers about what they should expect, what PFAS are, when they should / should not be concerned. It would be good to have preliminary information ready to go out to people as a lot of information is being

thrown at them from various sources, media, etc. Ms. Campbell-Ferrari stated that the faster the notification goes out, the greater the trust. She also encouraged a faster notification timeline than within 30 days.

Ms. Quirk asked about prior discussions related to making the language in the CCR more understandable to the general public. She asked if EPA would be flexible in their language to effectively communicate, or if standard language will be used that systems would be unable to amend.

Ms. Greene stated that she cannot answer that question at this time but language usage is under consideration. EPA is working with their colleagues who work on the CCR and the PN rule to ensure they are all moving forward similarly. EPA is also taking comments into consideration on the way they communicate and present PFAS information, as well as flexibilities that they may be able to offer primacy agencies.

Treatment Technologies

Ms. Greene presented information related to the PFAS drinking water treatment technologies and included a summary of NDWAC members' comments on treatment considerations provided during the April 2022 NDWAC consultation prior to rule proposal. Following her presentation, she opened the discussion for monitoring-related questions and comments.

Ms. Chard asked about treatment residuals in areas other than a water treatment plant. In Oklahoma, there are drinking water plants whose residuals are going to a Publicly Owned Treatment Works (POTW). If PFAS chemicals are in that waste, PFAS is just being transferred from one place to another. She also noted that point-of-use (POU) filters also generate waste, regardless of the user. If sent to a septic system, then PFAS is being moved to the ground water. If PFAS is going into the POTW, then we are potentially shifting it within the same utility/district. Then the same citizens who are paying to take the PFAS out of their drinking water are also paying to remove PFAS from their wastewater treatment plants.

Ms. Greene stated that EPA understands that PFAS has a whole life cycle and are taking this into account across many statutes. She stated that she is not a treatment engineer and referred Ms. Chard to the proposed rule support documents for more information. However, Ms. Greene did note that EPA is looking into available technologies that will be able to destroy and dispose of those materials in the most effective way to ensure water systems can comply with SDWA and that we are not just passing around the PFAS contamination. Ms. Greene also stated that the proposed rule did not name POU as a treatment technology. Homeowners may explore that option but that is not something that EPA actively included in the proposed rule.

Ms. Chard acknowledged that while she understands that the current proposed regulation relates to drinking water only, there are individuals who get their drinking water and wastewater services from the same entity so this issue needs to be dealt with holistically.

Dr. Albert reminded NDWAC members about the PFAS Strategic Roadmap, which works across multiple environmental statutes and offices within EPA. He stated that there has been a lot of discussion and coordination on these overlapping topics throughout the Agency.

Ms. Jennifer Peters expressed her curiosity about EPA's statement in their presentation about some systems switching drinking water sources rather than installing new treatment. This is something the

state of Michigan has investigated because they have some of the most stringent MCLs for PFAS. Ms. Peters stated that it was found that around 25% of public water systems in Michigan could switch to a new source rather than installing treatment which would eliminate the cost and burden of disposal. She asked if EPA had conducted a national estimate, acknowledging the difficulty in that estimate since many states are not as far along in the process as Michigan. Ms. Peters reiterated the importance of source water protection and expressed her disappointment that EPA has not been moving as quickly on the Clean Water Act (CWA) and guidelines on effluents. That rulemaking is moving much slower than on the drinking water side which is unfortunate because that would help reduce the impact to drinking water sources, as well as fish and wildlife. Additionally, Ms. Peters was curious how the cost estimates change when looking at different MCL levels. The slides presenting the different treatment techniques showed 99% reductions. She wondered how the cost burden would change if the MCL were to be increased from 4 ppt to 10 ppt. Finally, Ms. Peters emphasized the importance of helping small water systems find funding in the form of grants that they do not need to pay back in order to comply with these regulations because that is a huge concern.

Ms. Greene explained that EPA's economic analyses are extensive and information will be shared later in the presentation. She also referred Ms. Peters to the Federal Register Notice (FRN) for more details. Regarding systems that may be able to switch sources rather than install treatment, Ms. Greene could not provide the exact estimates but believed those estimates were presented in the Proposed Rule Economic Analysis. With respect to the cost associated with different MCLs, EPA looked at alternative MCLs as a part of the Health Risk Reduction Cost Analysis (HRRCA). These numbers are also within EPA's economic analysis and show the different costs and benefits at the different levels. She also noted with respect to small systems funding that there is \$5 billion in grant funding for dealing with PFAS and emerging contaminants in small and disadvantaged communities, stating that this is a significant investment that is available for these small systems.

Mr. Proctor stated that the economical treatment of PFAS requires significant innovation and creativity by the manufacturers of these technologies. Many manufacturers say they are concerned that if they enter this industry, they could potentially be accused of violating the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) down the road or become liable for their product. He asked if there has been any thought given regarding a "good Samaritan" consideration so that manufacturers are not disincentivized to innovate.

Ms. Greene stated that EPA is working with various offices under their environmental statutes (e.g., CERCLA) to determine how moving forward with those statutes will impact the drinking water regulation. Ms. Greene noted that she cannot comment on the status of those rules and when they will be finalized, but EPA is actively seeking input on how those rules should be implemented, developed, and finalized. She encouraged Mr. Proctor to engage with EPA and the rule making process for these concerns.

Mr. Eric Burneson added that EPA's Office of Enforcement and Compliance Assurance (OECA) have conducted listening sessions and there is current consideration for these issues.

Mr. Borman asked about EPA's evaluation of ongoing Operations and Maintenance (O&M) costs and whether the lifecycle of the material was considered. Mr. Borman noted that many systems that install this type of treatment technology will require a different level of certification for their operator. He noted that it is already challenging for these water systems to hire operators. The grants infrastructure money does not address these O&M costs down the road once the treatments are installed.

Ms. Greene stated that the O&M costs were considered in EPA's cost estimates.

Dr. Albert mentioned that EPA has information to share during the economic analysis portion of the presentation, and EPA economist Morgan Webster is available to address specific economics questions at that time.

Ms. Quirk reiterated that she was on a regulatory meeting for the Association of Metropolitan Water Agencies (AMWA) and there were many systems trying to install GAC treatment who had trouble installing the media. She said that only six firms make GAC in America and it is under high demand from both water and wastewater systems. Locally in Wisconsin, systems that are trying to get ahead of treatment are implementing anion exchange (AIX) and are going overseas for the product, which would disqualify them from using federal funds if the product is not made in the USA. Ms. Quirk wondered if this issue had been brought to EPA's attention.

Ms. Greene responded that EPA received many public comments surrounding the availability of treatment materials. It is something they are actively considering in reviewing the comments.

Ms. Quirk followed up by asking about "buying local."

Ms. Greene stated that EPA has seen many comments on that topic, as well.

Mr. Burneson stated that Build America, Buy America (BABA) provisions apply to the DWSRF and BIL funding. This is a legitimate concern that EPA is considering. This is a restriction on the use of those funds. However, he also noted that the proposed rule does not limit water systems to the use of media produced only in the United States if other types of funds are utilized.

Mr. Elmore shared information regarding the blending of sources, a practice that some systems in Wisconsin have done to address PFAS in one or more sources. These systems take a ground water source that has low or no PFAS and blends it with other wells that have higher concentrations of PFAS. He cautioned the use of this practice since the well that has no PFAS is usually in close proximity to the well with higher PFAS concentrations, and the PFAS plume will move and may cause PFAS detections where there was not before. He suggested that EPA should keep in mind that blending is not a final solution for addressing PFAS. If blending is used, the system should be required to pump equally from the two wells (50-50) to ensure that levels are not increasing where they were once lower or non-detection.

Ms. Daniels mentioned that Pennsylvania has experienced different treatment challenges to comply with their MCL. Pennsylvania has seen a need for pilot studies to ensure there is correct sizing of treatments, which is complicated when the NPDWR includes both short chain and long chain PFAS. Pilot studies need to be considered in order to understand when breakthrough will occur. Performance monitoring is also important (e.g., monitoring at the raw source, tracking breakthrough, etc.). This will also impact lab capacity and cost. It could take three to four weeks to get results back. Furthermore, similar to Mr. Elmore's comment about movement of contamination plumes, Pennsylvania has seen the contamination move to then impact other customers. You cannot just turn sources off. It may be a short-term measure while treatment is being put into place but it does not seem like a long-term solution.

Economic Analysis

Ms. Greene transitioned to the next section of her presentation where she shared information on the economic analysis, including the benefits and cost for the proposed rule, as well as available federal funds to help communities address emerging contaminants in their drinking water systems. In addition, Ms. Greene highlighted the key milestones and timeline of the PFAS NPDWR. Ms. Greene opened the meeting for discussion.

Dr. Albert asked to return to Mr. Borman's earlier question on operating costs and Ms. Peters' question related to costs at different MCL levels. He asked his colleague Ms. Webster to briefly address those concerns.

Ms. Morgan Webster first provided details related to Ms. Peters' comments and questions about the water system and whether they can pursue a non-treatment option to comply with the rule. EPA developed a compliance forecast that estimates 2% of water systems would elect to drill a new well to comply with the rule, and 6-7% of water systems would pursue an interconnection with another system to achieve compliance with the proposed rule. Ms. Webster was not familiar with the report used in the state of Michigan example mentioned earlier and stated that EPA will take it into consideration. EPA will also look at Ms. Daniel's point about plume migration. Lastly, EPA received comments on the compliance forecast and commenters recommended lowering the 2% and 6-7% estimates as they are too high, which EPA is taking into consideration. In regard to other MCLs, EPA considered regulatory alternatives in the economic analysis with alternative MCLs for PFOA/PFOS of 5 ppt and 10 ppt. EPA estimated the cost of the 10 ppt option at \$430-580 million per year. That cost change relates to the operational cost from treating from 4 ppt to 10 ppt and the number of water systems triggered into treatment under a 10 ppt standard. Ms. Webster also noted that the benefits are also reduced under the 10 ppt option. Further, she noted that the benefits outweighed the cost at the 4 ppt option. Finally, Ms. Webster provided an additional clarification – that EPA considers useful life of the treatment technologies in the cost analysis. She referred the meeting attendees to the Technologies and Costs proposed rule support document that provides detailed information on the useful life for each component.

Mr. Borman stated that he feels EPA's cost estimate is too low; the ongoing operation and maintenance (O&M) and associated costs are underestimated when comparing a 1 MGD plant to a 10 MGD plant to a 20 MGD plant. He said that there is also an ongoing problem of getting people to work; it is costing more money to entice operators to get into the industry. He also thought that EPA's cost estimates are too low for the initial installation as well as the ongoing O&M and suggested that it is also going to be difficult for the systems to get in compliance in only three years. For simultaneous compliance, using GAC is great for PFOS and PFOA, as well as other contaminants but it will need to be regenerated more frequently. Mr. Borman noted that the models are only as good as what you put into them. His real-world experience is telling him that EPA has underestimated the cost. Medium and large systems could probably absorb the costs but the small systems are going to have trouble keeping operators and staying in compliance.

Ms. Webster thanked Mr. Borman for sharing his concerns and mentioned that his concerns had been echoed in the public comments. EPA is actively considering how to refine and adjust the economic analysis for the final rulemaking. EPA recognizes the technical, managerial, and financial challenges faced by small water systems. EPA received comments suggesting specific co-occurrence and other adjustments that might be necessary in order to comply with the PFAS standards. Regarding

competition for absorptive sites, EPA considered the presence of total organic carbon (TOC) and the impact that would have on cost with change out times, etc.

Ms. Chard started by echoing Mr. Borman's point. Raising the MCL results in loss of protection of public health. However, that is assuming that everyone is complying the day that the MCL becomes effective and that is not realistic. Setting a standard does not protect public health; it is the ability to implement those standards that protects public health. It is important to consider what happens to small systems. Compliance assistance will be important. A lot of funding is available through BIL, the American Rescue Plan Act (ARPA) and DWSRF; however, we are seeing this money going to congressional earmarks that do not relate to emerging contaminants or small systems. This needs to be recognized as a real issue. On the cost of purchasing equipment, from a time that a project goes through the engineering phase to the bid, it is 25-30% higher than the original estimate. It takes 89 weeks to get certain equipment and water systems are running borrowed generators because the generator they ordered 18 months ago has not arrived. Generators and other equipment take a long time to ship and get installed. Regarding ongoing O&M costs, there have been grants and loans to build great plants to treat water, but there are not operators that are adequately trained to operate them. Plants are failing after 7-8 years that have not been fully paid for because they do not have operators that know how to run it. The big systems are figuring it out by hiring the small system operators. That does not help the small systems though.

Mr. Elmore commented on the cost model and urged EPA to ground truth the information they have as systems install treatment, collect information on real costs, and recalibrate their model with the new information. This tool will not only be helpful in embarking this MCL, but over years as EPA brings systems into compliance, they need to know those costs. This model could also be used by Congress and loan programs as they put forward new appropriations for water infrastructure improvements. Many shareholders would benefit from an accurate cost model; Mr. Elmore urged EPA to ground truth it.

Ms. Peters recognized that the more systems that must come into compliance, the more expensive it is going to be nationwide. She was curious about the difference in cost at the system-level (i.e., how the system would have to comply with a 10 ppt or 4 ppt MCL). Additionally, Ms. Peters asked if EPA is tracking each state's infrastructure money in a single place, noting that it would be helpful to see what states are taking advantage of the funding.

Ms. Webster responded that she does not have the system-level marginal cost of going from 4 ppt to 10 ppt; EPA only estimated that at the national-level.

In response to Ms. Peters' funding question, Dr. Albert stated that he did not have the specific resource to provide but would ask his colleagues to provide the requested information following the meeting.

Ms. Elin Betanzo mentioned that she conducted an analysis comparing the EPA and American Water Works Association (AWWA) cost models. There are concerns about the cost inputs, the increased costs to implement alternative MCLs, and the significantly higher cost estimates from the AWWA model. Ms. Betanzo stated that when looking at the two models, she identified a \$2.6 trillion difference based on the high levels of cost data in the AWWA model. Most of the difference comes from a different discount rate. In the AWWA estimate, if one entry point needed treatment then all entry points needed treatment. There were no non-treatment compliance options used in the AWWA cost estimate. She said that the huge cost difference comes from basic differences in cost inputs.

For the non-treatment options that play a significant role for cost, especially for small systems, Ms. Peters referred to Michigan, where 26% of systems have been able to comply using a non-treatment

option. She recognized that this is only one data point, but she encouraged the participants to look at that data. She said that Michigan has a higher percentage than EPA is considering.

Ms. Webster noted that she had not meant to imply that EPA had made a decision on the estimated number of systems that would select a non-treatment option.

Ms. Campbell-Ferrari mentioned that it is important for communities to feel like they are receiving the highest quality water as possible. There is more that EPA could do to support this process. There is no central database to track where the large amount of money is being distributed. The financial burden cannot fall solely on the ratepayer. EPA could help to prioritize money for systems. She also expressed concern on the point that if PFAS do not get into the source water, then the drinking water systems do not have to treat it. She recommended that EPA take the time to focus on source water protection.

Ms. Daniels thanked everyone for their contributions to EPA.

Dr. Albert thanked everyone for their time and for providing helpful input that will be considered as this regulation is being finalized, ideally by the end of this year.

Microbial & Disinfection Byproducts Rule Revisions Working Group Update

Ms. Daniels, co-chair for the MDBP Rule Revisions Working Group, presented an overview and an update on the MDBP Rule Revisions working group activities.⁷ Ms. Daniels opened the meeting up for NDWAC member discussion and requested that working group members speak first.

Mr. Borman expressed his pleasure with the process so far and thinks there has been lots of good discussion and ideas. It is his hope that their recommendations will be able to address all variables (e.g., different source water, etc.). The recommendations include enough flexibility while still protecting public health and supporting social justice requests from EPA. He feels there has been a lot of progress made and feels the working group members are moving towards an endpoint with work that will benefit everyone.

Ms. Quirk said that she has learned a lot over the last one and a half years with the working group. It is a diverse group with many different viewpoints. The technical experts and EPA staff have been helpful. Ms. Quick noted that she was on the premise plumbing subgroup. EPA has helped the working group to reach out to other federal organizations that can potentially help with certain issues. She agrees that there is good output coming forward.

Mr. Alex Rodriguez thanked Ms. Daniels for being the co-chair of the working group, and agreed that this has been a constructive and engaging experience. The in-person meeting brought them together to move in the same direction. He thanked EPA staff, technical advisors and everyone that participated, and is excited for future discussion and final recommendations.

Ms. Betanzo thanked Ms. Daniels and stated that this work touches on nearly all aspects of drinking water quality in some way. She appreciated the different perspectives, the ability for the group to troubleshoot problems, be all encompassing, and focus on public health protection. She is also looking forward to the final set of recommendations to be sent to NDWAC.

⁷ Please see Appendix C for Ms. Daniel's presentation.

Ms. Daniels thanked the group and echoed these perspectives. Initially the charge was overwhelming in scope, but it has gone well. She appreciates EPA staff and facilitators for getting the working group the resources they needed. They helped with problem characterization and gaining common understanding of the problem that needed to be solved, as well as the root causes of those problems. She thought everything really came together when they met in person, and she was also excited about the final set of recommendations. She then opened up comments and questions to non-working group members.

Ms. Littlewood thanked Ms. Daniels for her presentation and all of the working group members for their efforts. She recognized the level of attention to detail and knowledge necessary to complete this and it is a huge challenge. She read the charge from EPA and was overwhelmed with how they would come to a final consensus. Ms. Littlewood noted that the NDWAC may have more questions at this time if they had more information to review. She wondered if the working group could share if they felt like there is a desire to have all water systems chlorinating or disinfecting or if this will only pertain to water systems that are disinfecting. Ms. Littlewood stated that fewer than 5% of her water systems disinfect. There is a concern that all ground water systems would have to chlorinate.

Ms. Daniels welcomed EPA staff to respond if they had answers. She noted that the working group is focused on microbials and disinfectant byproducts in the context of the Surface Water Treatment Rule (SWTR). The Ground Water Rule (GWR) has not been brought up as part of that conversation. There have been discussions about some ancillary issues that may or may not be a part of that discussion.

Dr. Albert stated that in the third Six-Year Review (SYR3) findings, EPA considered eight rules for revision with a focus on microbial pathogens and DBPs in surface water. There have been a few topics that have touched on the GWR and the Revised Total Coliform Rule (RTCR) regarding monitoring but the primary focus has been on surface water.

Ms. Daniels agreed with Ms. Littlewood that people might not have questions but are open to comments or concerns.

Ms. Campbell-Ferrari asked if there are specific themes that have been more difficult to discuss and harder to reach a conclusion. Additionally, she asked if they should prepare for the next NDWAC meeting with a substantive presentation on the results, if there are things that will be helpful to think about until then with regards to preparing questions or opinions.

Ms. Daniels stated that the working group has not yet formed final recommendations. The preliminary work is still ongoing, so she cannot report on what points there will be consensus on. There has been valuable discussion in many different areas that will help inform these recommendations. She asked EPA to step in and answer what people should think about ahead of time for the NDWAC briefing in the fall. She suggested that NDWAC slides be sent out early to allow NDWAC members to prepare. She was aware that timing is tight and there is still much work to be completed before the end of the year.

Dr. Albert thanked Ms. Daniels and clarified that he is not trying to steer the deliberations of the working group or the recommendations they bring to the NDWAC. Keeping in mind that NDWAC members are very busy, he thought it would be helpful for them to take a look at the charge of the MDBP working group and early presentations from their meetings. Dr. Albert offered to prepare a list of resources for members who want to dive into specific issues. The meetings also have very detailed summaries available.

Ms. Daniels thanked Dr. Albert and mentioned upcoming meetings for NDWAC: one to talk about the preliminary scope in terms of the various recommendations, and one to present the report. Then there will be a meeting with ample time for deliberation. There will be multiple opportunities for getting questions, comments, and concerns back for the NDWAC.

Ms. Quirk added to a comment mentioned earlier about the extent of what is being accomplished to discuss where the efforts need to be concentrated. She mentioned Mr. Borman's concerns with the small systems' staffing and noted that there are also staffing issues at the state- and regional-level.

Ms. Daniels echoed Ms. Quirk's point of the scope of the issues and on allowing participants to provide a broad perspective on potential impacts that a rule making would have, both the positive side in terms of public health protection and those challenges such as unintended consequences.

Dr. Albert thanked Ms. Daniels, the workgroup members, and the rest of NDWAC. He hopes the NDWAC will be as impressed with the working group as he has been.

Closing Remarks

Ms. Daniels thanked Dr. Albert, as well as the rest of the NDWAC members. She thanked the EPA presenters for the opportunity to consult on the PFAS rulemaking effort and thanked the NDWAC members for their thoughtful comments. She also thanked her fellow MDBP working group members, as well as NDWAC members for questions and comments on that subject. Finally, she thanked the members of the public for joining the meeting and providing public comments earlier in the meeting.

Ms. Guilaran thanked Ms. Daniels for participating and leading the discussion. She also thanked Ms. Daniels for her MDBP workgroup leadership and acknowledged the challenge of finding the balance between the complexity of MDBPs and delivering public health protection. Ms. Guilaran thanked the EPA presenters and recognized Ms. Corr. Lastly, she thanked NDWAC members for devoting their time and energy, providing feedback, and for participating in the workgroup.

Ms. Corr thanked Ms. Guilaran and shared a comment from Ms. Yolanda Barney mentioned in the chat "I commend the group that worked on PFAS. Lots of good information and "things" to ponder upon in terms of operator certification, renovation(s)/ upgrade(s), and legal aspects. Great job!".

Ms. Corr adjourned the meeting.

APPENDIX A
NDWAC Roster: August 2023

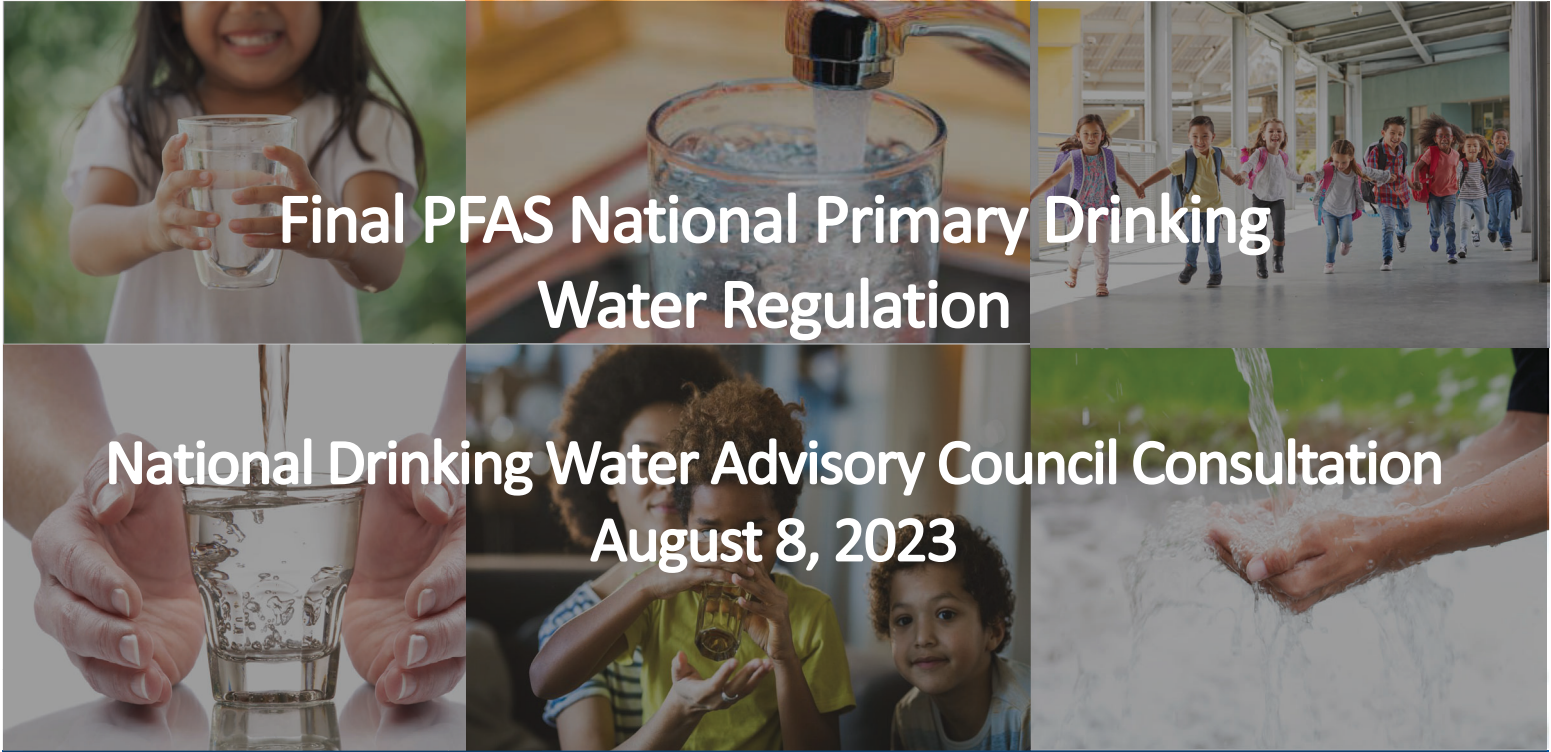
National Drinking Water Advisory Council Members	
Lisa D. Daniels, NDWAC Chair Director, Bureau of Safe Drinking Water Pennsylvania Department of Environmental Protection Harrisburg, PA	Yolanda Barney Environmental Program Manager Navajo Public Water System Supervision Program Navajo Nation Environmental Protection Agency Window Rock, AZ
Elin Warn Betanzo Founder and Principal Safe Water Engineering, LLC Detroit, MI	D. Scott Borman General Manager Benton/Washington Regional Public Water Authority Rogers, AR
Alexandra Campbell-Ferrari Co-Founder and Executive Director The Center for Water Security and Cooperation Washington, DC	Shellie R. Chard Director, Water Quality Division Oklahoma Department of Environmental Quality Oklahoma City, OK
Steven B. Elmore Program Director Bureau of Drinking Water and Groundwater Wisconsin Department of Natural Resources Madison, WI	Eagle Jones Director of Water Operations Pechanga Tribal Government Temecula, CA
Jana Littlewood National Rural Water Association Board of Directors -- Alaska Representative Wasilla, AK	Jennifer L. Peters National Water Programs Director Clean Water Action/Clean Water Fund Littleton, CO
James M. Proctor, II Senior Vice President and General Counsel McWane, Inc. Birmingham, AL	Nancy A. Quirk General Manager Green Bay Water Utility Green Bay, WI
Alex Rodriguez President and Chief Executive Officer DCG Public Affairs Diversity Consulting Group, LLC Santa Barbara, CA	Jeffrey W. Szabo Chief Executive Officer Suffolk County Water Authority Oakdale, NY
Macaroy "Mac" Underwood Principal Consultant Raftelis Financial Consultants, Inc. Vestavia, AL	
Centers for Disease Control Liaisons	
Dr. Arthur S. Chang Chief Medical Officer Division of Environmental Health Science and Practice National Center for Environmental Health Centers for Disease Control and Prevention Atlanta, GA	Dr. Vincent Hill Chief, Waterborne Disease Prevention Branch Division of Foodborne, Waterborne and Environmental Diseases National Center for Emerging and Zoonotic Infectious Diseases Centers for Disease Control and Prevention Atlanta, GA

APPENDIX B:

PRESENTATION: Final PFAS National Primary Drinking Water Regulation

NDWAC

August 8, 2023



Final PFAS National Primary Drinking Water Regulation

National Drinking Water Advisory Council Consultation August 8, 2023

Purpose

- To provide the National Drinking Water Advisory Council (NDWAC) with information on the proposed per-and polyfluoroalkyl substances (PFAS) National Primary Drinking Water Regulation (NPDWR)
- To consult with the NDWAC prior to promulgating the final PFAS NPDWR



Overview

- PFAS and Safe Drinking Water Act (SDWA) Background
- Proposed PFAS NPDWR Requirements
- Summary of NDWAC Member Perspectives on the Development of Key Areas of the Proposed Rule
- Questions and Discussion



PFAS Background

- PFAS are a category of manufactured chemicals that have been used in industry and consumer products since the 1940s.
- PFAS have characteristics that make them useful in a variety of products, including nonstick cookware, waterproof clothing, and firefighting foam, as well as in certain manufacturing processes.
- PFAS tend to break down extremely slowly in the environment and can build up in people, animals, and the environment over time.
- Even though some specific PFAS have been largely phased out due to health and environmental concerns, they may still be found in the environment and in drinking water.

PFAS Background

- We now know that over a long time PFAS may:
 - Lead to negative health effects on pregnant people and in developing babies
 - Weaken a body's ability to fight disease
 - An increased risk for some cancers, liver damage
 - Elevated cholesterol levels (which can increase the risk for heart attack or stroke)
- PFAS can enter drinking water in many ways, including discharges to rivers and lakes from manufacturing and processing facilities, as well as during industrial and commercial use. Areas can also be exposed due to proximity to industrial sites, airports, military installations, and other sites where PFAS have been produced or used.
- Drinking water is one of several ways people may be exposed to PFAS.
- Different PFAS are often found together and in combinations (or mixtures) in drinking water and the environment.
- EPA is acting to protect people's drinking water and reducing our exposure to PFAS, can lower our risk for these health effects.

Regulating PFAS in Drinking Water

- Under SDWA, EPA issued final regulatory determinations for PFOA and PFOS in March 2021. As a part of that action, EPA stated it would continue to evaluate additional PFAS to consider regulatory actions for other PFAS as supported by the best available science.
- In March 2023, EPA issued preliminary regulatory determinations to regulate four PFAS including PFHxS, PFNA, PFBS, HFPO-DA (commonly referred to as GenX Chemicals)
- Concurrent with these preliminary regulatory determinations, EPA proposed an NPDWR for these four PFAS as well as for PFOA and PFOS.
- This action is not final and does not require any actions until after EPA considers the public input and finalizes the regulation. Under SDWA, NPDWRs require compliance three years following rule promulgation.
- EPA anticipates that if fully implemented the rule will prevent tens of thousands of serious PFAS-attributable illnesses or deaths.



Overview of NPDWR Development Process

Evaluate Data Availability

What are the best available, peer-reviewed science and supporting studies?

Establish MCLG

What is the level at which no known or anticipated adverse effects on the health of persons occur and which allows for an adequate margin of safety?

Develop Rule Analyses

Develop a health risk reduction and cost analysis and determine what are the impacts of policy alternatives?

Set Standard as Close as Feasible to MCLG

What is the regulatory standard that may be achieved with the use of best available technologies taking cost into consideration?

Benefit-Cost Determination

Are the benefits justified by the costs?



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Stakeholder Input Informing the Proposed PFAS NPDWR

- To inform the proposed NPDWR, EPA gathered input from several stakeholder groups and public meetings including:
 - Local, state, and tribal governments and officials
 - Public drinking water systems,
 - Small system representatives to the Small Business Advocacy Review Panel
 - Science Advisory Board
 - National Drinking Water Advisory Council
 - Public meetings on environmental justice considerations



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NDWAC Consultation on the Proposed PFAS NPDWR

- In April 2022, EPA conducted a consultation with the NDWAC prior to rule proposal. EPA provided information related to informing considerations for key areas of the proposed rule including:
 - PFAS mixtures
 - Monitoring
 - Public notification
 - Treatment

EPA's Proposed Action for the PFAS NPDWR

- EPA proposed health-based, non-enforceable Maximum Contaminant Level Goals (MCLGs) for six PFAS.
 - PFOA and PFOS as individual contaminants, and
 - PFHxS, PFNA, GenX Chemicals, and PFBS as a PFAS mixture
 - MCLGs are the maximum level of a contaminant in drinking water where there are no known or anticipated negative health effects allowing for a margin of safety.
- EPA proposed an NPDWR to establish legally enforceable MCLs for these six PFAS in drinking water.

Proposed PFOA and PFOS MCLGs Considerations

- To establish the MCLGs for PFOA and PFOS, EPA assessed the peer reviewed science examining cancer and noncancer health effects associated with oral exposure.
- Consistent with SDWA statutory definition of an MCLG, EPA establishes MCLGs of zero for carcinogens classified as *Carcinogenic to Humans* or *Likely to be Carcinogenic to Humans* where there is insufficient information to determine that a carcinogen has a threshold dose below which no carcinogenic effects have been observed.
- Under the EPA Guidelines for Carcinogen Risk Assessment, EPA reviewed the weight of the evidence and determined that PFOA and PFOS are *Likely to Be Carcinogenic to Humans*.
 - For PFOA, this determination is based on the statistically significant evidence of kidney cancer in humans and Leydig cell tumors, pancreatic acinar cell tumors, and hepatocellular adenomas in rats.
 - For PFOS, this determination is based on the statistically significant evidence of potentially human relevant tumors, including hepatocellular tumors in male and female rats and pancreatic islet cell carcinomas in male rats.

EPA's Proposed Action for the PFAS NPDWR

Compound	Proposed MCLG	Proposed MCL (enforceable levels)
PFOA	0 ppt*	4.0 ppt*
PFOS	0 ppt*	4.0 ppt*
PFNA		
PFHxS	1.0 (unitless) Hazard Index	1.0 (unitless) Hazard Index
PFBS		
HFPO-DA (commonly referred to as GenX Chemicals)		

The Hazard Index is a tool used to evaluate potential health risks from exposure to chemical mixtures.

*ppt = parts per trillion (also expressed as ng/L)

Proposed MCLs Considerations

- EPA proposed MCLs as close as feasible to the MCLGs.
- For the feasibility determination, EPA considered factors including:
 - Availability of analytical methods: There are multiple methods available (EPA Methods 533 and 537.1) to reliably measure and quantify the six PFAS at or below their proposed MCLs.
 - Identification of treatment technologies: There are several treatment technologies available and currently in use to treat and remove the six PFAS to levels at or below their proposed MCLs.

What is a Hazard Index?

- The HI is a tool used to evaluate potential health risks from exposure to chemical mixtures, based on an assumption of dose additivity.
- EPA is proposing that water systems use this approach to limit any mixture containing one or more of PFHxS, PFNA, PFBS, and GenX Chemicals. The HI does not include PFOA and PFOS which are proposed for regulation as individual contaminants due to their likely carcinogenicity.
- To determine the HI, water systems would monitor and compare the amount of each of the four PFAS in drinking water to its associated HBWC, which is the level below which no health effects are expected for that PFAS. The proposed HBWCs are:

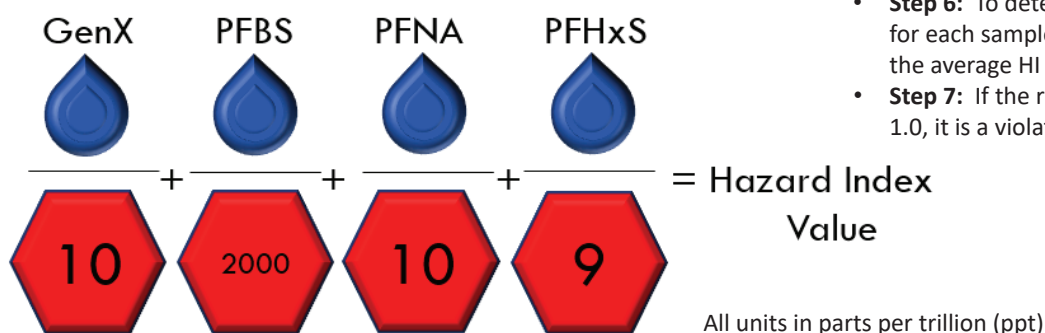
Compound	Proposed HBWC (ppt)
PFHxS	9.0
PFNA	10
PFBS	2000
HFPO-DA (commonly referred to as GenX Chemicals)	10

How do I calculate the Hazard Index?

The HI is used to understand health risks. For the PFAS NPDWR proposal, the HI considers the combined toxicity of PFNA, GenX Chemicals, PFHxS, and PFBS in drinking water.

What is a Hazard Index?

The HI is made up of a sum of fractions. Each fraction compares the level of each PFAS measured in the water to the level determined not to cause health effects (i.e., HBWC).



Steps:

- **Step 1:** Divide the measured concentration of **GenX** by the health-based value of **10 ppt***
- **Step 2:** Divide the measured concentration of **PFBS** by the health-based value of **2000 ppt**
- **Step 3:** Divide the measured concentration of **PFNA** by the health-based value of **10 ppt**
- **Step 4:** Divide the measured concentration of **PFHxS** by the health-based value of **9.0 ppt**
- **Step 5:** Add the ratios from steps 1, 2, 3, and 4 together
- **Step 6:** To determine HI compliance, repeat steps 1-5 for each sample collected in the past year and calculate the average HI for all the samples taken in the past year
- **Step 7:** If the running annual average HI greater than 1.0, it is a violation of the proposed HI MCL

Hazard Index MCL Calculation Examples

GenX Chemicals PFBS PFNA PFHxS HI

- **Example 1** – Exceedance of proposed Hazard Index MCL

$$\left(\frac{[5 \text{ ppt}]}{[10 \text{ ppt}]}\right) + \left(\frac{[200 \text{ ppt}]}{[2000 \text{ ppt}]}\right) + \left(\frac{[5 \text{ ppt}]}{[10 \text{ ppt}]}\right) + \left(\frac{[9 \text{ ppt}]}{[9.0 \text{ ppt}]}\right) = 2.1$$

- **Example 2** – Exceedance of proposed Hazard Index MCL

$$\left(\frac{[0 \text{ ppt}]}{[10 \text{ ppt}]}\right) + \left(\frac{[200 \text{ ppt}]}{[2000 \text{ ppt}]}\right) + \left(\frac{[2 \text{ ppt}]}{[10 \text{ ppt}]}\right) + \left(\frac{[7 \text{ ppt}]}{[9.0 \text{ ppt}]}\right) = 1.1$$

- **Example 3** – Exceedance of proposed Hazard Index MCL

$$\left(\frac{[12 \text{ ppt}]}{[10 \text{ ppt}]}\right) + \left(\frac{[0 \text{ ppt}]}{[2000 \text{ ppt}]}\right) + \left(\frac{[0 \text{ ppt}]}{[10 \text{ ppt}]}\right) + \left(\frac{[0 \text{ ppt}]}{[9.0 \text{ ppt}]}\right) = 1.2$$

- **Example 4** – Meets proposed Hazard Index MCL

$$\left(\frac{[0 \text{ ppt}]}{[10 \text{ ppt}]}\right) + \left(\frac{[100 \text{ ppt}]}{[2000 \text{ ppt}]}\right) + \left(\frac{[4 \text{ ppt}]}{[10 \text{ ppt}]}\right) + \left(\frac{[3 \text{ ppt}]}{[9.0 \text{ ppt}]}\right) = 0.8$$

NDWAC Member Perspectives in the Pre-Proposal Consultation: PFAS Mixtures

How should EPA consider or address potential mixtures of PFAS in the proposed drinking water standard?

- EPA should consider mixtures of PFAS, and to the extent possible, addressing multiple contaminants at once is important.
- EPA could evaluate PFAS in groups similar to what is done for total trihalomethanes and haloacetic acids regulations.
- There could be challenges in regulating groups of PFAS where not enough similarities in health effects.

Questions and Discussion

EPA's Proposed Action for the PFAS NPDWR

- The proposed rule would require public water systems to:
 - Monitor for these PFAS;
 - Notify the public of the levels of these PFAS; and
 - Reduce the levels of these PFAS in drinking water if they exceed the proposed standards.

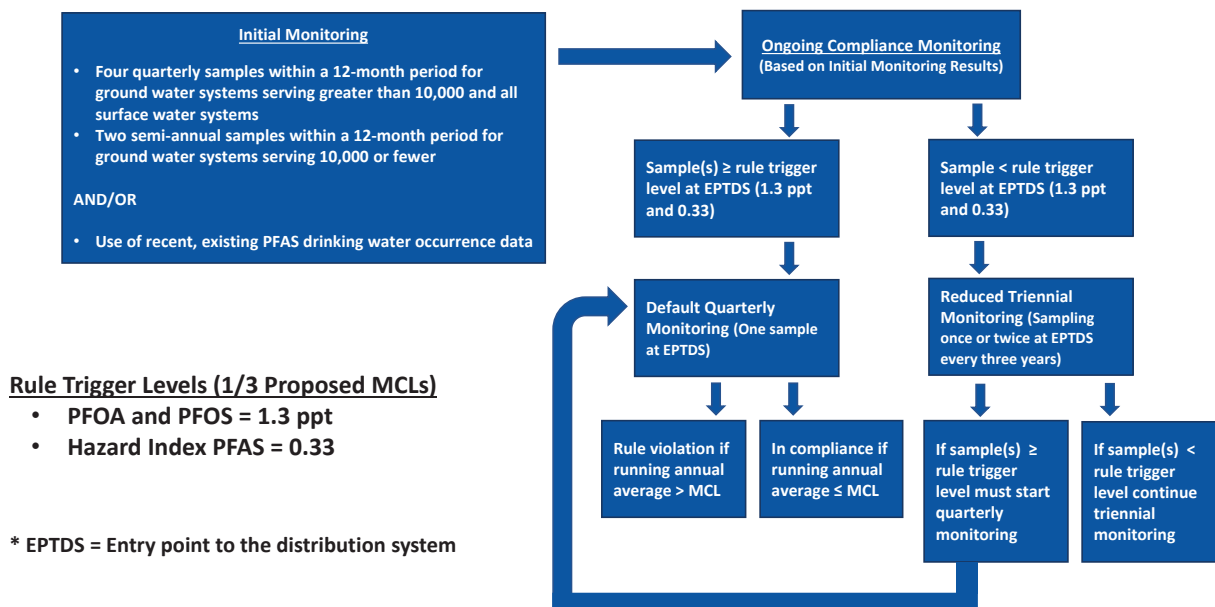
Proposed NPDWR Monitoring Requirements

- EPA's proposed requirements are based on EPA's Standardized Monitoring Framework for both initial and ongoing compliance monitoring of regulated PFAS to ensure that drinking water is not above MCLs.
- Initial monitoring must be completed in the three years between the rule promulgation date (anticipated end of 2023) and the rule effective date (anticipated end of 2026). Proposed initial monitoring requirements to establish baseline PFAS levels include any combination of:
 - Two or four samples collected at public water systems over one year, dependent on system population size and system type
 - Use of recent, previously acquired PFAS drinking water data from the fifth Unregulated Contaminant Monitoring Rule (UCMR 5), state-level drinking water occurrence monitoring, or other appropriate data collection program
- Initial monitoring results will determine the ongoing compliance monitoring requirements. Proposed ongoing compliance monitoring requirements include:
 - Quarterly monitoring as the normal frequency for all sampling locations
 - Reduced monitoring flexibility to once or twice every three years for sampling locations where the result is below 1/3 of the MCLs (i.e., rule trigger level)
- A system is in violation if monitoring results exceed one of the MCLs. Compliance calculations are based on running annual average, where if the measured value is below the PFAS practical quantitation level (PQL), zero is used for the calculation (e.g., a value of 2.0 for PFOA would default to zero).

Proposed NPDWR Monitoring Requirements

- EPA used PQLs for the six PFAS proposed for regulation in determining the proposed MCLs. PQLs are the lowest concentration of a contaminant that can be reliably achieved within specified limits of precision and accuracy during routine laboratory operating conditions.
- The proposed rule trigger levels are set at levels that are useful in determining whether the contaminant is present in a sample rather than to determine its specific concentration.
- EPA requested comment on establishing the proposed rule trigger levels at 1/3 of the proposed MCLs and on alternative trigger levels such as 1/2 of the proposed MCLs.

Proposed NPDWR Monitoring Requirements



NDWAC Member Perspectives in the Pre-Proposal Consultation: Monitoring

How should available monitoring data be considered for initial monitoring requirements and should the Standardized Monitoring Framework for SOC's be incorporated?

- EPA should utilize a Standardized Monitoring Framework for monitoring.
- It is unlikely that monitoring waivers could be issued given how ubiquitous PFAS are in the environment. If waivers are allowed, it should be based on sampling results, though for other systems it could be based on their source water if it known to show little variation.
- Recent data collected under UCMR 5 or other monitoring efforts using EPA approved analytical methods could be used.
- EPA could offer a phased schedule for monitoring to address any laboratory capacity issues.

Questions and Discussion

Proposed NPDWR Public Notification Requirements

- EPA proposed that public water systems be required to issue public notification to customers if the levels of regulated PFAS exceed the proposed PFAS NPDWR.
- Under the Public Notification Rule, EPA proposed the PFAS NPDWR as a “Tier 2” notification.
 - This would require notice as soon as possible, but within 30 days of the violation.
- EPA is proposing that community water systems be required to include PFAS information in the Consumer Confidence Report distribution to customers including:
 - The level of the regulated PFAS that is measured in their drinking water.
 - The potential health effects of the regulated PFAS detected in violation of the PFAS NPDWR.

NDWAC Member Perspectives in the Pre-Proposal Consultation: Public Notification

How quickly should water systems be required to notify the public following a violation of the PFAS standard and information that should be included in Consumer Confidence Reports regarding PFAS in drinking water?

- Members provided various comments on the public notification tier with some suggesting a Tier 2 or Tier 3 public notification is most appropriate, while other comments suggested a public notification tier between Tier 1 and Tier 2 and that EPA should identify a level above the MCL which would require an immediate notice.
- CCRs should include violations, however they should not include all detections. Other comment provided that all detections should be provided to the public as soon as possible, particularly for certain sensitive populations.
- Clear health effects language is critical, as well as explanation of how people can reduce their exposure beyond drinking water risks.

Questions and Discussion

PFAS Drinking Water Treatment Technologies

- Water systems with regulated PFAS above their proposed MCLs will be required to install treatment or take other action to reduce regulated PFAS levels in their drinking water and meet MCLs.
- As proposed, the rule would allow water systems the flexibility to determine the best actions and approaches to their specific situation.
- EPA evaluated technologies and has studies that demonstrate effective removal of all regulated PFAS. EPA has identified the following as best available technologies (BATs):
 - Granular activated carbon(GAC)
 - Anion Exchange (AIX)
 - Nanofiltration (NF) and Reverse Osmosis (RO)
- Some water systems may be able to reduce PFAS levels without installing treatment by using an alternative source of water that does not have PFAS contamination.

PFAS Drinking Water Treatment Technologies

- EPA conducted an extensive review of available PFAS removal treatment literature in EPA's Drinking Water Treatability Database and detailed in EPA's proposed rule support documents. The available data includes hundreds of studies conducted in the laboratory, in the field at pilot scale, and in full-scale application.
- Based on the best available science, EPA found that all of the best available technologies (GAC, AIX, RO, and NF) can exceed treatment removal efficiencies > 99% and can achieve concentrations below analytical detection limits.
- These technologies can also co-remove PFAS. For example, PFHxS is removed approximately as well as PFOA.

PFAS Treatment Residuals and Disposal

- EPA evaluated actions that public water systems must take to dispose of treatment residuals that contain PFAS.
- EPA has developed interim guidance for the destruction and disposal of PFAS and PFAS-containing materials from some products, including spent drinking water treatment media.
- EPA is aware that actions resulting from other environmental statutes (e.g., Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA)) may impact future drinking water treatment and disposal options.
 - As part of the proposed PFAS NPDWR, EPA has considered the costs of various disposal options for drinking water treatment residuals that contain PFAS.
- EPA is prioritizing research on PFAS disposal options in different environmental media and best management practices.

NDWAC Member Perspectives in the Pre-Proposal Consultation: Treatment

Are there other treatment technologies EPA should consider or other non-treatment options? How should EPA consider the disposal of PFAS treatment residuals?

- When considering point of use treatment, EPA should evaluate who owns and operates them and where the responsibility lies.
- Treatment residual disposal should be considered in EPA's evaluation of drinking water treatment.
- More research is needed for the ultimate destruction and disposal of PFAS.
- EPA should consider requiring systems to identify new sources of water before applying treatment to address PFAS.
- EPA should allow treatment technologies that have not currently been identified under the rule to be implemented later and meet rule requirements.
- The burden to remove PFAS should not solely fall on public water systems and manufacturers and producers of PFAS should be responsible for limiting the formation and discharges of PFAS.

Questions and Discussion

Economic Analysis for the Proposed Rule

- Benefits are assessed as avoided cases of illness and deaths associated with exposure to the six PFAS in the NPDWR. EPA's benefits analysis considered the strength of evidence for each effect and the availability of data to quantify the associated morbidity and mortality impacts.
- Costs are assessed as the expenses incurred by public water systems to monitor for the six PFAS included in the NPDWR, install and operate treatment technologies, inform consumers, and perform record-keeping and reporting responsibilities. State (or primacy agency) costs are assessed as expenses incurred to administer and implement the rule.
- EPA used the best available science and peer reviewed models to complete the economic analysis for the proposed rule. **The Administrator determined that the benefits of this proposed regulation justify the costs.**

National Benefits Summary

- EPA quantified some of the reduced adverse health effects expected from the proposed rule including kidney cancers, heart attacks, strokes, and developmental (birth weight) effects. EPA relied on the assessment of adverse health effects of PFOA and PFOS in the MCLG documents to inform the benefits analysis.
- EPA anticipates significant additional benefits beyond those that EPA has quantified associated with the following adverse health effects:
 - Immune
 - Developmental
 - Cardiovascular
 - Hepatic
 - Carcinogenic
 - Endocrine
 - Metabolic
 - Reproductive
 - Musculoskeletal

Annualized Quantified Rule Benefits (i.e., per year)	3% Discount Rate	7% Discount Rate
	\$1.23 billion	\$908 million

National Costs Summary

- EPA expects roughly 66,000 water systems to be subject to the rule, with approximately 3,400-6,300 systems anticipated to exceed one or more MCL.
- EPA has estimated the costs of the proposed rule to public water systems associated with administration, monitoring, and treatment and costs to primacy agencies associated with rule implementation and administration.
- Public water system treatment cost estimates include capital, and yearly operation and maintenance costs over the period of analysis and are derived using peer-reviewed work breakdown structure models.

Annualized Quantified Rule Costs (i.e., per year)	3% Discount Rate	7% Discount Rate
	\$772 million	\$1.20 billion

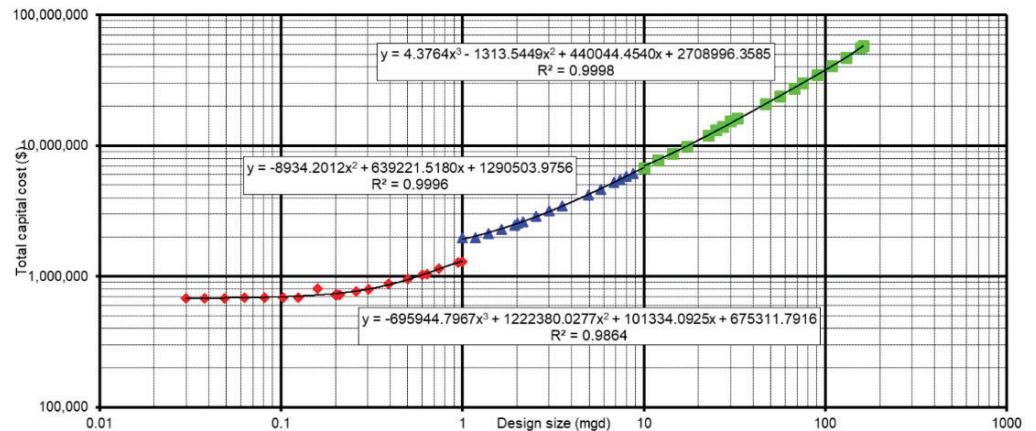
- EPA also prepared a supplemental cost analysis that estimates the annual costs would increase by \$30-\$61 million per year if water systems are required to dispose of PFAS treatment as hazardous waste.

Water System Treatment Costs

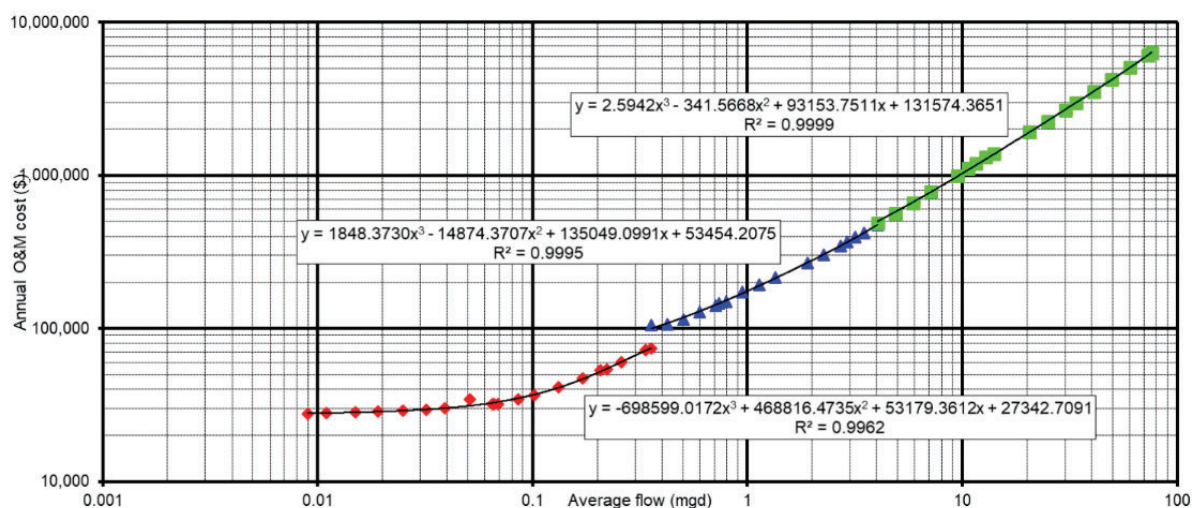
- EPA estimated annualized costs per year for water systems that treat or change water source.
 - Costs of system capital, operation, and maintenance are annualized.
- Quantified costs are estimated over a human lifetime (82 years) to be comparable to quantified benefits estimates.
- Costs factor in repairs and replacement of capital infrastructure at the end of its lifespan (variable, based on materials used; for example, useful life range of approximately 20-35 years for GAC capital).
- Costs differ based on treatment technology used.
- For more information, see USEPA (2023) Economic Analysis of the Proposed National Primary Drinking Water Regulation for Per- and Polyfluoroalkyl Substances. EPA-822-P-23-001.

Capital Cost Estimates

- EPA developed dozens of Work Breakdown Structure cost equations for treatment at surface and ground water systems across the range of bed life (5,000 to 150,000 BVs) and residuals management scenarios (hazardous and non-hazardous), including high, mid, and low-cost levels.
- The mid-level capital cost curve (right) estimates costs of removal of PFAS from surface water using GAC.
- These curves are used to inform the SafeWater model, which estimates national level treatment costs.



Operation and Maintenance Cost Estimates



Mid-level Cost Results for Removal of PFAS from Surface Water Using Gravity GAC (\$2020)

Bipartisan Infrastructure Law Funding for PFAS

- The Bipartisan Infrastructure Law provides \$9 billion to invest in drinking water systems specifically impacted by PFAS and other emerging contaminants.
 - \$4 billion through the Drinking Water State Revolving Fund (DWSRF)
 - \$5 billion through EPA's Emerging Contaminants in Small or Disadvantaged Communities Grant Program
- States and communities can also leverage an additional nearly \$12 billion in BIL DWSRF funds dedicated to making drinking water safer.

Proposed Rule Public Comment Period

- Following the proposed rule publishing, the public were invited to review the proposal and supporting information and provide their written input to EPA through the public docket.
 - The public docket can be accessed at: www.regulations.gov under Docket ID: EPA-HQ-OW-2022-0114.
- During the public comment period, EPA also held a public hearing to listen to the public's views about the proposal.
- EPA received approximately 122,000 public comments submitted to the docket and during the public hearing.
- EPA is currently evaluating and considering all the public comments to inform the final PFAS NPDWR.

PFAS NPDWR Key Milestones and Path Forward

Final Regulatory Determinations for PFOA and PFOS: March 2021

Preliminary Regulatory Determinations for PFHxS, PFNA, PFBS, GenX Chemicals, and their mixtures: March 2023

Proposed PFAS NPDWR for PFOA, PFOS, PFHxS, PFNA, PFBS, and GenX Chemicals: March 2023

Public Comment Period on Proposed PFAS NPDWR: March 29 – May 30, 2023

Public Hearing on Proposed PFAS NPDWR: May 4, 2023

Final PFAS NPDWR Promulgated: Anticipated December 2023

PFAS NPDWR Effective Date: Anticipated December 2026 (three years following final rule promulgation)



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Questions and Discussion



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EPA's PFAS NPDWR website:

<https://www.epa.gov/sdwa/and-polyfluoroalkyl-substances-pfas>

APPENDIX C

PRESENTATION: Microbial & Disinfection Byproducts Rule Revisions
Working Group Update

NDWAC
August 8, 2023

Potential MDBP Rule Revisions Update on Working Group Activities to Inform NDWAC Advice and Recommendations

Lisa Daniels

Co-Chair, Microbial and Disinfection Byproducts Rule Revisions

Working Group

August 8, 2023

Presentation Overview

- **Brief Recap:** Microbial and Disinfection Byproducts (MDBP) Rule Revisions Working Group (through November 2022)
- **Working Group Meetings:** December 2022 through June 2023
- **Next Steps:** Working Group and NDWAC

MDBP Rule Revisions Working Group Purpose

- **EPA is seeking advice and consensus recommendations from the NDWAC** that would improve public health protection provided by the MDBP regulations, better assure the regulations equitably protect consumers' health, particularly disadvantaged communities, and be implementable.
- **EPA charged the NDWAC** to provide the agency with advice and consensus recommendations on key issues related to potential revisions to MDBP rules.
- **To support the work of the NDWAC**, EPA requested that the Council form a working group (the MDBP Rule Revisions Working Group).
- **The MDBP Rule Revisions Working Group** is asked to consider issues related to potential rule revisions.
- **The Working Group is moving forward** to conclude its work this Fall.

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Working Group Membership

Lisa D. Daniels* – WG Co-chair, Pennsylvania Dept of Environmental Protection
Andy Kricun** – WG Co-chair, Moonshot Missions
Elin W. Betanzo* , Safe Water Engineering LLC
D. Scott Borman* , Benton/Washington Regional Public Water Authority
John Choate , Tri-County Regional Water Distribution District
Kay Coffey , Oklahoma Department of Environmental Quality
Jeffrey K. Griffiths , Tufts University School of Medicine
Michael Hotaling , Newport News Waterworks Department
Jolyn Leslie , Washington State Department of Health
Rosemary Menard , City of Santa Cruz
William F. Moody , Mississippi State Department of Health
Erik D. Olson , Natural Resources Defense Council
Benjamin J. Pauli** , Kettering University
Nancy A. Quirk* , Green Bay Water Utility
Lisa J. Ragain , Metropolitan Washington Council of Governments
Alex Rodriguez* , Diversity Consulting Group
Lynn W. Thorp , Clean Water Action/Clean Water Fund
Gary Williams , Florida Rural Water Association

*Member of U.S. EPA's National Drinking Water Advisory Council.

**Member of U.S. EPA's National Environmental Justice Advisory Council.

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Prior Public Engagement and Working Group's Formation and Initial Meetings

- **Prior to Working Group formation:** EPA held an initial virtual public meeting in October 2020 followed by six virtual meetings from May to November 2021 to solicit broad input and information on MDBPs in drinking water.
- **The Working Group was formed** in Spring 2022 and met virtually eight times from May 2022 through April 2023 and one time in person in June 2023.
- **The first four meetings focused on** characterizing problems related to opportunistic pathogens, disinfectant residuals, disinfection byproducts, finished water storage, implementation and compliance issues, environmental justice and other relevant topics.
- **EPA provided the Working Group** with information gathered through analyses and input from EPA and external technical experts and relevant published information, as well as with information gathered through the public docket and the prior public engagement.

5

MDBP Rule Revisions Working Group Meeting #5: December 13, 2022

- **Purpose** was to continue problem characterization discussions on opportunistic pathogens, disinfectant residuals, disinfectant byproducts, as well as risk-balancing/interdependencies, and begin focused discussion on problems relevant to disadvantaged communities.
- **Key meeting highlights** included an overview of a National Academies report on *Legionella*, a characterization of *Legionella* outbreak data, an evaluation of DBP precursor occurrence and control, and environmental justice in the MDBP context.

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MDBP Rule Revisions Working Group Meeting #6: January 24, 2023

- **Purpose** was to continue problem characterization discussions on environmental justice, begin focused discussion on problems relevant to implementation and compliance challenges, and continue discussions about preliminary findings for problem characterization.
- **Key meeting highlights** included implementation and compliance challenges from small and large systems, a synthesis of data and analysis on opportunistic pathogens and DBPs, premise plumbing, distribution systems, data and analysis, and preliminary findings for problem characterization.

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MDBP Rule Revisions Working Group Meeting #7: March 9, 2023

- **Purpose** was to continue discussions about preliminary understandings for problem characterization and begin discussion of interventions.
- **Key meeting highlights** included continued discussions of preliminary findings from Meeting 6, synthesis of source water conditions, storage tanks, consecutive systems, environmental justice, and implementation challenges, and introducing and planning for Working Group interventions discussions.

8

MDBP Rule Revisions Working Group Meeting #8: April 19, 2023

- **Purpose** was to create / characterize an initial list of intervention ideas and related implementation mechanisms; to set up the ability to formulate intervention/ implementation mechanism options for Working Group consideration at the June in-person meeting; and to understand where future information would be helpful to support discussions at the June in-person meeting.
- **Key meeting highlights** included breakout discussions on source water and treatment, distribution systems, premise plumbing and the SDWA enabling environment. Plenary discussions were held following the breakout discussions.

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MDBP Rule Revisions Working Group Meeting #9: June 27-29, 2023

- **Two and a half day in person Working Group meeting** with observer option for in person or virtual attendance.
- **Purpose:**
 - Review and discuss MDBP Working Group-generated interventions and implementation actions to form the basis for Working Group recommendations to the NDWAC.
 - Cross-check status of MDBP Working Group deliberations with the EPA charge to the NDWAC and the NDWAC request for recommendations from the MDBP Working Group.
 - Identify emergent recommendations that hold potential to receive Working Group member consensus support.

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MDBP Rule Revisions Working Group

Meeting #9: June 27-29, 2023

- **Key meeting highlights** included Facilitator Review of Interventions and Implementation Actions related to:
 - Premise Plumbing – Improve Building Water Quality
 - Distribution System – Disinfectant Residual, Overall Distribution Water Quality, and Opportunistic Pathogen Benefits
 - Treatment and Distribution System – Regulated and Unregulated DBPs
 - Distribution System – Finished Water Storage Tanks
 - Consecutive Systems (e.g., DBP Compliance)
 - Treatment – Improve Chloramination Practice
 - Source Water – Source Control and Source Water Assessment
 - Enabling Environment – Technical, Managerial, and Financial Capacity
 - Enabling Environment – State Capacity

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Moving Forward: Working Group Next Steps

- **Planning for Working Group meetings:**
 - Meeting #10 – September 14, 2023
 - Meeting #11 – October 5, 2023
 - Meeting #12 – October 31, 2023
- **Development of a report** to inform the NDWAC's deliberations in responding to EPA's request for advice and recommendations from the Council.

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Moving Forward: NDWAC Next Steps

- **Mid-October 2023 NDWAC Public Meeting:** The Working Group will brief the NDWAC on general direction and anticipated scope of the pending report to the Council.
- **Mid-November 2023:** Delivery of the Working Group's report to the NDWAC.
- **Late November 2023 NDWAC Public Meeting:** The NDWAC will meet to deliberate and develop advice and recommendations from the Council to EPA.
- **Early December 2023:** Delivery to EPA of NDWAC advice and recommendations on potential revisions to MDBP rules.

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Key Takeaways to Date

- **All Working Group members** have provided valuable input and contributed to meaningful discussions relevant to the Working Group purpose.
- **There is recognition of the depth and complexity** of the topics and of the technical information provided to the Working Group for supporting discussions and deliberations.
- **Information provided by the technical analysts** is helpful for supporting the Working Group.

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Open for NDWAC Discussion

- **Additional thoughts from NDWAC members serving on the Working Group:** Please share your observations and perceptions about Working Group proceedings.
- **Discussion among all NDWAC members:** Floor is open to all members of the NDWAC to share your thoughts, raise questions, and engage in conversation.

APPENDIX D

Public Comments

NDWAC

August 8, 2023



THE UNITED STATES
CONFERENCE OF MAYORS



May 30, 2023

The Honorable Radhika Fox
Assistant Administrator
Office of Water
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue NW
Washington, DC 20460

**RE: Per- and Polyfluoroalkyl Substances National Primary Drinking Water Regulation,
Docket ID No. EPA-HQ-OW-2022-0114**

Dear Assistant Administrator Fox,

On behalf of the nation's mayors, cities and counties, we appreciate the opportunity to submit comments on the U.S. Environmental Protection Agency's (EPA) Proposed Per- and Polyfluoroalkyl Substances (PFAS) National Primary Drinking Water Regulation (NPDWR). Local leaders are dedicated to the health, well-being and safety of their residents and communities and are therefore highly interested and concerned regarding the public health impact that PFAS substances may have on our drinking water supplies and systems.

We appreciated the opportunity to submit comments to EPA in response to the Agency's Federalism Consultation briefing last year. However, we are disappointed to see that our recommendations are not reflected in this proposed regulation. Given this, we urge EPA to move forward cautiously, consult with local governments, gather and utilize additional and updated scientific data, and reexamine the cost-benefit analysis to best inform this regulation. Specifically, should EPA move forward, we ask that the Agency:

- Provide maximum flexibility for local governments, including longer compliance timeframes;
- Reconsider regulatory alternative Option 1 c, which would establish a higher MCL at 10 parts per trillion (ppt) for PFOS and PFOA; and
- Provide additional direct funding for local governments to comply with the proposed regulation to avoid creating an unfunded mandate that will disproportionately impact low-income residents and communities.

These measures are important to ensuring that local governments, water utilities and ratepayers are not unduly burdened in trying to fix a problem they did not create.

Our organizations represent the nation's 3,069 counties, 19,000 cities and the mayors of the 1,400 largest cities throughout the United States. For the past several years, all levels of government, including the counties and cities we represent, have become increasingly concerned about drinking water contamination from PFAS. Created by private industry for use in a variety of sectors and applications around the globe, these chemicals have made their way into drinking water systems across the country and are heavily concentrated in communities near military installations or industrial sites.

The presence of these human-made chemicals has spurred action by state and local governments across the country. We continue to urge the federal government to take holistic and comprehensive action to address PFAS contamination through pollution prevention, cleanup, research and development, scientific and public health analysis, and prevention of further exposure.

As passive receivers of materials containing PFAS, local water systems neither caused nor contributed to the pollution. We urge EPA to adhere to the polluter pays model and provide sufficient direct funding to comply with this regulation. Additionally, in developing this regulation, EPA should provide local governments with maximum flexibility and a longer compliance timeframe to avoid overburdening communities and ratepayers.

As coregulators in implementing federal statutes, including the Safe Drinking Water Act, and as partners in protecting public health, it is important that federal, state and local governments work together to craft reasonable and practicable rules and regulations to address PFAS contamination. In order to achieve these goals, it is essential that EPA provide local governments with a clear understanding of the rules' and regulations' requirements and a full and complete cost-benefit analysis.

In general, our organizations support provisions in the 1996 Amendments to the Safe Drinking Water Act that requires drinking water standards to be based on sound science, public health protection and the occurrence of contaminants in drinking water supplies at levels of public health concern in order to reduce risk while also balancing costs. Consequently, we believe the NPDWR for PFAS, and any regulatory or legislative initiative addressing PFAS in drinking water, should balance public health and environmental priorities with technological and economic feasibility. Any federal mandate on local governments should include additional federal financial resources, as well as offer local water systems flexibility in implementation and compliance options. Further, our organizations support programs for public education regarding safe drinking water and innovative solutions that approach this problem beyond traditional command and control.

Local governments fund the majority of water infrastructure investments

Local governments fund over 98 percent of all capital, operations and maintenance investment in drinking water and wastewater infrastructure in the United States, primarily through user fees and bonds. The most recent U.S. Census data shows that local governments spent over \$142 billion on water and wastewater in 2020 alone, and, between 1993 and 2019, spent over \$2.38 trillion, not adjusted for inflation. Even with this significant investment by and commitment from local governments, many communities struggle to upgrade their drinking water and wastewater systems.

During this same time period, the federal government only appropriated approximately \$2 billion annually for both the Clean Water and Drinking Water State Revolving Loan Fund (SRF) programs. The SRF programs provide grants to states which, in turn, provide local governments with loans that must be repaid.

While we are pleased that the bipartisan Infrastructure Investment and Jobs Act (also known as the Bipartisan Infrastructure Law or BIL) provided record-high levels of funding for our nation's water infrastructure, including \$10 billion over five years for grants to address PFAS and other emerging contaminants in drinking water and wastewater, this funding is insufficient for local governments to meet the requirements of this proposed regulation and/or other rules that the Agency is considering.

At a minimum, it must be acknowledged that the timelines for the availability of funding under BIL, which is through FY 2026, and the likely compliance dates for a new NPDWR for PFAS do not align. Therefore, it is uncertain if local governments will be able to use BIL funding specifically for compliance with this forthcoming regulation or for future rulemakings pertaining to PFAS.

Taking a holistic approach toward drinking water regulations

Considering EPA is simultaneously undergoing other rulemaking processes that pertain to local drinking water and wastewater infrastructure management, among others, it is important that these rules and regulations are not developed in silos within the Agency. We urge the Agency to take a holistic and integrated approach and consider the cumulative impacts that the rules and regulations will have on local governments in terms of costs, compliance, and implementation timelines. Additionally, EPA should examine other related consequences of proposed rules, such as the impact on efforts to reduce greenhouse gas emissions.

Specifically, we are concerned that the Agency's rulemakings around NPDWR for PFAS, Lead and Copper Rule Improvements, and regulating PFAS under CERCLA and RCRA will individually and combined, create additional unfunded mandates on local governments that will be economically significant and, in many communities, unaffordable. If EPA moves forward with these proposed rules and regulations, new funding sources must be created to assist local governments with compliance and implementation. Even with the increased funding from BIL for the SRF programs, as well as for reducing lead in drinking water and addressing PFAS drinking water contamination, local governments will still face a significant water infrastructure needs gap that would exacerbate affordability and equity concerns for the many fixed- and low-income households that already spend a disproportionate amount of their income on water bills.

Overarching Concerns and Recommendations

As EPA continues to develop this regulation, we offer several of our priority concerns and recommendations relating to local governments' ability to effectively and cost-efficiently implement the proposed changes while also protecting public health.

1. Financial Impacts

The Agency's proposed regulation will have a significant impact on public water systems, and the financial burden will ultimately be passed on to local governments and community ratepayers. In proposing this new regulation, the Agency has conducted a cost-benefit analysis and concluded annual costs of \$770 million and benefits of approximately \$1.2 billion. However, the American Water Works Association (AWWA) has estimated annual costs between \$2.5-\$3.2 billion.¹

While we appreciate the recent significant investments in our nation's water infrastructure from BIL, it unfortunately is not enough to protect the health of our residents in the manner the Agency is attempting. We have serious concerns that not only will local governments be unable to afford the required costs to comply with this regulation, but also that the Agency has underestimated the cascading impacts this regulation will have on local communities, primarily in the form of higher water bills.

In general, local governments will be responsible, directly or indirectly, for a wide array of associated compliance costs, including for testing, monitoring and installing treatment. The

¹ WITAF 56 Technical Memorandum Update, "PFAS National Cost Model Report," May 26, 2023

proposed Maximum Contaminant Level (MCL) of 4 ppt will require 3,400-6,000 systems across the country to take action, significantly more than are currently doing so based on state-level standards or that would need to do so if the MCL was set at a higher level such as 10 ppt. Many of the roughly 66,000 water systems that will be subject to the regulation are small systems for which compliance will be even more challenging and the financial impacts more severe. Moreover, as the Agency moves forward with rulemakings designating PFOS and PFOA or other PFAS as hazardous substances under CERCLA, these costs will only increase as local governments will also be responsible for the appropriate removal and transport of hazardous chemicals.

As providers of public water, providing safe, clean and affordable drinking water to our communities is of utmost importance to local governments. It is worth emphasizing that local governments have limited financial resources to comply with a host of new and existing water-related mandates, including but not limited to testing for lead, removing lead service lines, upgrading cybersecurity and replacing aging infrastructure. Consequently, local utilities may be forced to fund the compliance costs associated with this new regulation by cutting back on infrastructure replacement and maintenance, reducing operational resiliency and reducing other expenditures that would otherwise benefit public health and access to clean and safe drinking water.

As local governments are forced to bear the brunt of the financial burden, an increase in water rates in communities across the nation is a near certainty. Indeed, this rising consumer cost for utilities to comply with the proposed MCL will be felt most harshly by the low-income households and small business community. The U.S. Census reports that local governments spent \$80 billion in 2020 on water supply utilities. A \$2.5-\$3.2 billion new unfunded federal mandate will require a 3.125-4% increase in national spending that will be passed on to consumers through rate increases and long-term debt, particularly where advanced treatment is required for compliance. Given this regulation will currently impact approximately 3,400-6,000 systems, the burden on those communities will be dramatically higher. Many communities and residents are already experiencing significant and widespread financial burdens, and this proposed regulation will add to that burden.

Because of this, we urge the Agency to reconsider the financial impact this proposed regulation will have on individual consumers, particularly on environmental justice and disadvantaged communities. These communities are often disproportionately impacted by both increased costs for their water bills and risk exposure to emerging contaminants. In examining the financial impacts this regulation could impose upon individual households, costs will vary depending on several factors, such as the size of the public water system. In scenarios where new treatment facilities would need to be installed and operated, individual households may see increases in the amounts of hundreds of dollars to their water bills. For systems serving smaller communities, this number extends into the thousands, according to an AWWA study.

Further, many American households currently face a significant and widespread financial burden when it comes to water bills. This burden falls disproportionately on fixed- and low-income households who must dedicate a significant portion of their income to water. Given the Administration's focus on environmental justice, water rate affordability must be a part of the consideration in this proposed regulation.

In light of the Administration's Justice40 initiative, EPA should additionally recognize that the financial burden on low-income and environmental justice communities associated with meeting environmental requirements is an important aspect of environmental justice. The financial

burden that increased rates will have on disadvantaged communities should be a consideration in this and other rules and regulations.

This burden can be alleviated by providing additional flexibility for local governments, a longer compliance timeframe, and additional direct funding for local governments. Should EPA move forward, the Agency should establish a higher MCL, such as 10 ppt under Option 1 c in the proposed regulation. This will capture the systems where PFAS is more prevalent, without overburdening many more communities.

2. Practicality of Implementation

As mentioned above, local governments play a critical role in the effective implementation of federal regulations. Recognizing this, as the Agency moves forward with this regulation, we have serious concerns with several practical aspects that will impact local governments' capacity to effectively and economically achieve compliance, as outlined below. Many of these practical concerns can be minimized by establishing a higher MCL for PFOS and PFOA, which would prioritize systems with higher concentrations of the chemicals, and by granting a longer compliance timeframe.

a. MCLs for PFOS and PFOA set at detection levels

The Agency has proposed drinking water standards for PFOS and PFOA at 4 ppt, which is the lowest detection level at which the contaminant can be reliably measured. When comparing these MCL levels with other datasets in the international community, such as those from the World Health Organization, Australia, Japan and Canada, EPA's proposed standards are significantly lower. Additionally, several states have recently set their own drinking water standards for PFOS and PFOA. As proposed, EPA's standards are lower than any current international or state standard.

When taking into account that states as well as the international community have access to the same available science EPA used to develop its proposed regulation, there are questions about this wide discrepancy that requires further examination. Additionally, EPA's standards are set so strictly that thousands of systems will be in violation from the outset, necessitating immediate decision making and costly actions.

This discrepancy also makes it difficult for local leaders to effectively communicate the risk associated with PFAS to the public and will likely lead to confusion among residents as they seek to determine if their water is safe to drink. EPA should work with local governments and water utilities to collaboratively develop a risk communication toolkit that explains the relative public health risks and clarifies why additional measures are necessary despite higher current state and international standards. Importantly, this language and toolkit should be voluntary for local governments to use, rather than mandatory.

b. Inadequate Laboratory Capacity

Laboratories across the nation will need to be drastically scaled up in order to manage the surging demand from public water systems as they implement and comply with the proposed regulation. Compliance monitoring is only a small fraction of the number of samples that will need to be collected and analyzed as public water systems that have test samples above the limit will also need to monitor for PFAS while installing and operating treatment techniques. Therefore, laboratory capacity must be able to not only handle initial compliance monitoring, but also monitoring from public water systems as they continue to operate and treat PFAS.

With such an increase in demand, we have serious concerns regarding laboratory capacity

across the nation and the ability for public water systems to receive and process sample results in a timely and cost-efficient manner. Currently, public water systems are reporting wait times of up to three months to receive PFAS sample results. With the Agency's current proposal giving public water systems only three years to comply, water systems will be forced to move as expeditiously as possible, resulting in increased monitoring and longer sample response times than what is already being reported. As laboratories across the nation become overwhelmed, EPA should consider the importance of having reliable, accessible and affordable laboratory capacity as it relates to local governments' capability to work towards compliance.

c. Supply Chain, Disposal and Workforce Challenges

The technologies used to remove PFOS and PFOA from drinking water include Granulated Activated Carbon (GAC) or reverse osmosis membranes. Supply chain disruptions are already apparent at the local level, with over a year wait for replacement carbon, and in many cases the cost of needed supplies has also increased. These factors will be further exacerbated if more water utilities add GAC to their systems as a treatment technique. Establishing a higher initial MCL would allow those public water systems with the highest PFOA or PFOS levels, and therefore also the highest public health risks, to be prioritized first. A rapid increase in demand resulting from detection-level MCL levels would only exacerbate the amount of time it takes to obtain and install necessary treatment.

Moreover, given that both technologies only remove but do not destroy PFOS and PFOA, local governments will have to find a way of disposing of the spent carbon and membranes. Considering that EPA is also moving forward to declare both chemicals as hazardous substances under CERCLA, local governments will be forced to use either hazardous waste landfills or hazardous waste incinerators. Unfortunately, there are very few of either of these facilities and will necessitate expensive long-hauling of material. Given the increase in energy usage associated with these treatments and the additional costs associated with appropriate disposal, EPA should focus more attention on an effective means of destroying these chemicals.

Additionally, many local governments and water systems are experiencing workforce shortages that will impact the timeline and cost to comply with this regulation. These challenges not only include trained water utility personnel, but also limited staff capacity at testing labs, in the transportation sector needed for shipping and handling equipment and lab tests, and at the state agency level for review and approval of monitoring and compliance data.

Due to the challenges outlined above, attempting to implement this regulation in such a constrained time frame would only exacerbate current issues relating to skyrocketing supply prices, labor shortages, laboratory capacity, and surging market demands. Although EPA's proposal includes providing extensions on a case-by-case basis granted either from states or by EPA, this is not a guarantee. We strongly urge the Agency to clearly and definitively extend the current proposed three-year timeline for all public water systems.

3. Additional Scientific Data and Cost-Benefit Analysis

The Agency anticipates releasing updated data in its upcoming Fifth Unregulated Contaminant Monitoring Rule (UCMR 5), expected within the next nine months. According to EPA's website, "the monitoring provides EPA and other interested parties with nationally representative data on the occurrence of contaminants in drinking water, the number of people potentially being exposed, and an estimate of the levels of that exposure. These data can support future regulatory determinations and other actions to protect public health." Notably, this current

dataset includes monitoring for 29 different PFAS chemicals, including for all six PFAS chemicals being considered under this proposal.

The information obtained from this monitoring dataset would be of great value to support the Agency as it develops its regulatory framework for proposing new drinking water standards for PFAS. This updated data will provide a deeper insight into the monitoring and testing capacities of public water systems and laboratories across the nation, as well as better inform the cost-benefit analysis. Setting the MCL levels at the detect level when more data has yet to be released denies the Agency the ability to adjust these levels in the future, making permanent strict compliance actions for which the costs may not be justified. Therefore, we urge the Agency to either propose a higher MCL standard such as 10 ppt for PFOS and PFOA, or consider waiting to finalize any MCL levels until at least this new dataset becomes available for EPA to utilize.

Conclusion

In conclusion, we urge EPA to adhere to the polluter pay model and to move forward in a way that will not shift the cost burden of this regulation onto local governments, water utilities or ratepayers. In developing this regulation, EPA should adequately examine emerging data in an effort to further scientific consensus regarding PFAS. Further, should EPA move forward on this regulation, we urge the Agency to establish a higher MCL for PFOA and PFOS and to provide local governments with maximum flexibility, longer compliance timeframes, and additional direct funding.

On behalf of the nation's mayors, cities and counties, thank you for considering the local government perspective on this important issue. If you have any questions, please contact us: Judy Sheahan (USCM) at 202-861-6775 or jsheahan@usmayors.org; Carolyn Berndt (NLC) at 202-626-3101 or Berndt@nlc.org; or Sarah Gimont (NACo) at 202-942-4254 or sgimont@naco.org.

Sincerely,



Tom Cochran
CEO and Executive Director
The U.S. Conference of Mayors



Clarence E. Anthony
CEO and Executive Director
National League of Cities



Matthew D. Chase
CEO and Executive Director
National Association of Counties

NDWAC -

Item 1: Please schedule me for a 3 minute oral comment on August 8th and advise of approximately what time that will occur.

Item 2: Please distribute the following to your membership for review in advance of that meeting.

Councilors -

It is vitally important that as you consider the PFAS situation that you understand that:

1. The F in this class of chemicals stands for fluorine and as the most reactive element on the periodic table which results in all types of fluorides in the environment, the F is the most dangerous
2. PFAS do break down, albeit slowly, increasing fluoride pollution
3. Plans to filter out PFAS may result in more fluoridation chemicals being intentionally added to water supplies where 99% of the material goes directly to wastewater. These chemicals originate as the waste product of industry in the US, China and Mexico.
4. Techno-cons plans for dealing with fluoride include accelerating the decomposition of PFAS which dumps more fluoride into the environment.

The above is vitally important because although everyone is vulnerable to fluoride poisoning, we have robust and high quality scientific evidence that susceptible sub-populations most at risk of adverse effects even from low concentrations considered 'optimal' (0.7 ppm) by biased stakeholders include:

1. **Pregnant women & their fetuses and bottle-fed babies & young children** for whom 'optimal' exposure to fluorides results in a range of adverse effects including higher rates of learning disabilities, lower IQ, thyroid dysfunction and dental fluorosis. A 2021 Benchmark Dose Analysis found fluoride exposure of 0.2 mg/L (ppm equivalence) was harmful.
2. **Thyroid patients and those with low iodine** for whom fluoride exposure is at 0.5 mg/L has been documented as harmful
3. **Kidney patients** for whom any exposure is dangerous
4. **Diabetics & kidney patients** who consume much more water than is typical and therefor exceed so-called "safe" doses
5. **Consumers with inflammatory or autoimmune conditions** from allergies to UC for whom fluoride is a accelerant
6. **Elderly** for whom a lifetime of exposure results in arthritic conditions and brittle bones.

The attached annotated bibliography includes approximately 150 studies published since 2015 substantiating the claims above. I also suggest you review the following:

- **WTF about NTP with Dr. Christine Till, Ph.D.**
(2023): <https://www.youtube.com/watch?v=NOc4XYto8eo>
- **Dr. David Brownstein, M.D. on Iodine, Thyroid, Cholesterol & Fluoride**
(2023): <https://www.youtube.com/watch?v=9vUWLZG6dYY>

- **Letter to MWRA signed by Kilmer McCoy, M.D. et al.**
(2023): https://fluoridealert.org/wp-content/uploads/MWRAletter_2023.03.pdf

You may also be interested in perusing the links in my signature and the recent revelations on scientific fraud regarding fluoride science on the part of vested stakeholders.

- **HHS & CDC suppression of science (Capitol & Main/Mother Jones,**
2023): <https://capitalandmain.com/health-officials-delayed-report-linking-fluoride-to-brain-harm>
- **Scientific Fraud on part of government players (The Defender,**
2023): <https://childrenshealthdefense.org/defender/researchers-hid-data-fluoride-lower-iq/>

Ultimately, the EPA should set the MCLG for fluoride at zero and an MCL of no higher than 0.3 ppm while banning fluoridation practice based on the evidence of harm and the immorality of using water supplies to medicate the public whose individual dose is dependent on individual factors including consumption. The EPA should also take into account the SDWA bans national players from using water supplies to treat the public and that state and municipal players should be no less restrictive than national players, and yet fluoridation is encouraged & protected by national players using shells in state and local government.

- The first four minutes of this 2023 presentation (25 min) addresses legality & EPA complicity: https://www.youtube.com/watch?v=C7_K0ZA0-Tc

I also suggest the 2014 interview of Dr. Moolenburgh is worth your time:

- “As a summary of our research, we are now convinced that fluoridation of the water supplies causes a low grade intoxication of the whole population, with only the approximately 5% most sensitive persons showing acute symptoms. The whole population being subjected to low grade poisoning means that their immune systems are constantly overtaxed.... this can hasten health calamities.” - *Dr. Hans Moolenburgh, MD (1993 affidavit and 2014 interview)*

Regards,

Karen Favazza Spencer

Fluoridation Policy: *An Annotated Bibliography of Published Science*

A sampling of the scientific studies and reports relevant to water fluoridation published since the HHS 2015 recommendation to lower the fluoridation target to 0.7 ppm is listed below.

I suggest these items provide compelling evidence that 0.7 ppm is neither optimal nor safe and that any claims to the contrary are ill-founded. Moreover, protests that more study is required before banning fluoridation is a tacit endorsement of human experimentation without individual consent which is medical assault - *Karen F. Spencer*

2023

INFLAMMATION: Children living in communities with water sources containing less than 1 ppm fluoride concentration are still vulnerable to inflammatory, immunological, and other non-skeletal bodily harm. Dental fluorosis (DF) is present even at these low exposures and seems to be sex-mediated. DF a visible sign of fluoride poisoning while young.

<https://www.sciencedirect.com/science/article/abs/pii/S0269749122022102>

- Tang H, Wang M, Li G, Wang M, Luo C, Zhou G, Zhao Q, Dong L, Liu H, Cui Y, Liu L, Zhang S, Wang A. Association between dental fluorosis prevalence and inflammation levels in school-aged children with low-to-moderate fluoride exposure. *Environ Pollut.* 2023 Jan.

HYPOTHYROIDISM & PREGNANCY: Using multiple measures and gold-standard approaches to examine maternal fluoride exposure and thyroid function in a prospective pregnancy cohort receiving optimally fluoridated water, authors found that a 0.5 mg/L increase in drinking water fluoride concentration was associated with a 1.65 increased odds of primary hypothyroidism in pregnant women and that children born to women with primary hypothyroidism had lower FSIQ scores, particularly boys. The authors found no evidence of confounding by other environmental toxicants in this Canadian cohort (MIREC). Maternal urinary fluoride concentration was not associated with hypothyroidism, which may be due to fluctuations in exposure to fluoride from diet. Ultimately, the water concentration proved the more stable factor, and in fact water fluoridation is the primary source of exposure and consequently representative of body burden.

<https://www.sciencedirect.com/science/article/pii/S0048969722082523>

- Hall M, Lanphear B, Chevrier J, Hornung R, Green R, Goodman C, Ayotte P, Martinez-Mier EA, Zoeller RT, Till C. Fluoride exposure and hypothyroidism in a Canadian pregnancy cohort. *Science of The Total Environment.* 2023.

DIABETES: Low levels of Vitamin D are linked to osteoporosis, bone fractures, hypertension, cardiovascular diseases, chronic kidney disease (CKD), and diabetes. This study found fluoride-induced insulin resistance and vitamin D deficiency lead to diabetes, and confirms fluoride exposure through drinking tap water with F-concentrations considered “safe” by US authorities is a risk factor for developing diabetes. The study demonstrate that vitamin D deficiency and fasting blood glucose levels (BGLs) are significantly associated with water fluoride levels. The authors recommend using safe water (bottled or purified) and vitamin D supplementation.

<https://pubmed.ncbi.nlm.nih.gov/36705502/>

- Kumar P, Gupta R, Gupta A. Vitamin D deficiency in patients with diabetes and its correlation with water fluoride levels. *J Water Health.* 2023 Jan;21(1):125-137.

ECZEMA: Early-life exposure to fluoride is associated with atopic eczema, consistent with other observations of increased allergies and adverse impact of toxic metals on immunity.

<https://pubmed.ncbi.nlm.nih.gov/37422976/>

- Kampouri M, et al. Associations of gestational and early-life exposure to toxic metals and fluoride with a diagnosis of food allergy or atopic eczema at 1 year of age. *Environ Int.* 2023 Jun 29;178:108071.

Fluoridation Policy: *An Annotated Bibliography of Published Science*

CENTRAL NERVOUS SYSTEM: This study review recognizes that fluoride is an environmental pollutant which crosses the blood-brain-barrier as well as having an adverse effect on hormones and organs. Although the authors didn't find any studies that considered fluoride's association with the growing incidence of pediatric brain tumors, the indirect evidence of fluoride's effects on the central nervous system (CNS) is such that they urge research with a focus on the impact of fluoride on gliomas and other brain tumors. <https://www.mdpi.com/1422-0067/24/2/1558/htm>

- Żwieretko, W.; Maruszewska, A.; Skórka-Majewicz, M.; Gutowska, I. Fluoride in the Central Nervous System and Its Potential Influence on the Development and Invasiveness of Brain Tumours—A Research Hypothesis. *Int. J. Mol. Sci.* 2023, 24, 1558.

BAD BONES: Animal study finds low dose equivalencies of human fluoride exposure inhibited the GPX activity and increased the MDA level to activate oxidative stress as well as impairing cancellous bone and causing redox imbalance. <https://pubmed.ncbi.nlm.nih.gov/36640257/>

- Li H, Chen X, Zhang Z, Zhang J, Xu H. Microstructural Analysis of Cancellous Bone in Fluorosis Rats. *Biol Trace Elem Res.* 2023 Jan 14.

SYSTEMATIC REVIEW & META-ANALYSIS: Although there is no argument that fluoride exposure can result in dental and skeletal fluorosis, a structural defect, the impact on brains and bodies at current exposure levels is a source of controversy. The National Toxicology Program (NTP) reviewed the evidence of developmental neurotoxicity at concentrations considered optimal or safe by U.S. authorities. The NTP found that most studies suggested an adverse effect of fluoride exposure on children's IQ, starting at low levels of exposure.

<https://pubmed.ncbi.nlm.nih.gov/36639015/>

- Veneri F, Vinceti M, Generali L, Giannone ME, Mazzoleni E, Birnbaum LS, Consolo U, Filippini T. Fluoride exposure and cognitive neurodevelopment: Systematic review and dose-response meta-analysis. *Environ Res.* 2023 Mar 15;221:115239.

ARSENIC, FLUORIDE & KIDNEYS: Alone and in combination, arsenic and fluoride change the microbiome in such a way that it results in nephrotoxic impact” “The metabiome data showed that single and combined As and F exposure remarkably altered the serum metabolites...”

<https://pubmed.ncbi.nlm.nih.gov/36692655/>

- Tian X, Yan X, Chen X, Liu P, Sun Z, Niu R. Identifying Serum Metabolites and Gut Bacterial Species Associated with Nephrotoxicity Caused by Arsenic and Fluoride Exposure. *Biol Trace Elem Res.* 2023 Jan 24.

ORAL MICROBIOME: Two hundred students without cavities, oral health issues or other confounders were chosen from a college population with a dental fluorosis prevalence of 47%. Half were controls. Salivary microbiota was carefully analyzed: “Analyses of β diversity revealed significant differences between control individuals and dental fluorosis patients, while there was no significant difference in the saliva microbial diversity between patients with mild and moderate to severe dental fluorosis.” Gene expression, periodontitis and systemic lung or gut diseases are potential effects of the altered microbiome, regardless of severity of fluorosis.

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9946311/>

- Liu S, et al. (2023) Saliva microbiome alterations in dental fluorosis population. *J Oral Microbiol.* 2023 Feb 20;15(1):2180927.

EXECUTIVE FUNCTION: Prenatal exposure to ‘optimally’ fluoridated water in 616 maternal-child pairs in Alberta Pregnancy Outcomes and Nutrition (APrON) study between 2009 and 2012 found prenatal fluoride exposure increased behavioral and cognitive deficits associated with executive function, including lack of inhibitory control.

<https://pubmed.ncbi.nlm.nih.gov/37236475/>

- Dewey D, et al. APrON Study Team. Fluoride exposure during pregnancy from a community water supply is associated with executive function in preschool children: A prospective ecological cohort study. *Sci Total Environ.* 2023 May 25;891:164322.

Fluoridation Policy: *An Annotated Bibliography of Published Science*

TAIWAN, TEA & ALLERGIES: Non-fluoridated Taiwan study of 562 school children found a urinary fluoride range of 0.03 to 2.08 mg/L, with a geometric mean of 0.32 mg/L. Tea consumption was a factor in fluoride exposure and dental fluorosis which affected 23.67% of participants. The study did not find significant cognitive deficits. However, those with dental fluorosis had significantly more allergic conditions. <https://www.mdpi.com/2227-9067/10/6/987>

- Lin YY, Hsu WY, Yen CE, Hu SW. Association of Dental Fluorosis and Urinary Fluoride with Intelligence among Schoolchildren. *Children (Basel)*. 2023 May 31;10(6):987.

TOPICAL: A randomized clinical trial of two topical fluoride treatments and a placebo found, “there were no statistically significant differences in preventing of approximal caries development between the semiannual application of 5%NaF, 38%SDF, or placebo.” This finding further calls into question the narrative promoted by dentists who profit from fluoride mythology. <https://pubmed.ncbi.nlm.nih.gov/37268914/>

- Sirivichayakul P, Jirarattanasopha V, Phonghanyudh A, Tunlayadechanont P, Khumsub P, Duangthip D. The effectiveness of topical fluoride agents on preventing development of approximal caries in primary teeth: a randomized clinical trial. *BMC Oral Health*. 2023 Jun 2;23(1):349.

META-ANALYSIS: This meta-analysis “suggests that early exposure to fluoride may have neurotoxic effects on neurodevelopment affecting behavioral, cognitive and psychosomatic symptoms related to ADHD diagnosis.” Authors also mention the biological plausibility of such an effect as fluoride easily crosses the blood–brain barrier in prenatal and early life and concentrates in the brain areas responsible for memory and learning abilities as well as affects the metabolism and physiology of neuronal and glial cells through oxidative stress and can suppress thyroid function. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10143272/>

- Fiore G, Veneri F, Di Lorenzo R, Generali L, Vinceti M, Filippini T. Fluoride Exposure and ADHD: A Systematic Review of Epidemiological Studies. *Medicina (Kaunas)*. 2023 Apr 19;59(4):797.

PROLONGED EXPOSURE & BRAIN DAMAGE: Authors studied adolescent and adult male mice and found that even low F-exposure results in protein changes in the hippocampal region of the brain which can affect behavior, but that those changes are substantially different from the changes in that region at high exposure which are associated with cognitive changes. Also noted that higher exposures resulted in changes in neuron density. Authors suggest that prolonged long term adult exposure could be associated with degenerative neurological conditions like Alzheimers disease and that more research is required at different ages and durations of F-exposure at low levels. <https://pubmed.ncbi.nlm.nih.gov/37422569/>

- Bittencourt LO, Dionizio A, Ferreira MKM, Aragão WAB, de Carvalho Cartágenes S, Puty B, do Socorro Ferraz Maia C, Zohoori FV, Buzalaf MAR, Lima RR. Prolonged exposure to high fluoride levels during adolescence to adulthood elicits molecular, morphological, and functional impairments in the hippocampus. *Sci Rep*. 2023 Jul 8;13(1):11083.

Fluoridation Policy: *An Annotated Bibliography of Published Science*

2022

CATFISH REPORT: Despite the optimistic assumption of the pro-fluoridation National Institute for Health who funded this comprehensive review of fluoridation schemes in Cumbria, England, researchers have found no significant improvement for the 'poor children' and indeed little if any dental benefit, with only 0.3 fewer cavities in baby teeth and 0.2 fewer cavities in permanent teeth among the children surveyed in fluoridated communities as compared to children in unfluoridated communities. This obviously translates into no real economic benefit.

<https://www.journalslibrary.nihr.ac.uk/phr/SHMX1584/#/full-report>

- Goodwin M, Emsley R, Kelly MP, Sutton M, Tickle M, Walsh T, et al. Evaluation of water fluoridation scheme in Cumbria: the CATFISH prospective longitudinal cohort study. *Public Health Res* 2022;10(11).

CWF, DIET, PREGNANCY & TOXICITY: "There may be a place for the use of topical fluoride, including silver diamine fluoride, to prevent and arrest dental caries but the use of community water fluoridation for the prevention of dental caries can no longer be considered to be safe."

<https://www.fluorideresearch.online/epub/files/196.pdf>

- Research report: The Dark Side of Fluoride. Amir Hossein Mahvi, Bruce Spittle. *Fluoride* 55(4):388-390 October-December 2022

RIGHT QUESTION: Given the robust and consistent evidence regarding the developmental neurotoxic impact of fluoridation policy, the question that needs to be evaluated by communities should be: is this intervention worth the risk of lowering the IQ of at least certain individuals when we have a viable substitute, i.e. fluoridated toothpastes?

<https://www.karger.com/Article/Abstract/520789>

- Vieira AR The Overlooked Individual: Susceptibility to Dental Caries, Erosive Tooth Wear and Amelogenesis. *Monogr Oral Sci.* Basel, Karger, 2022, vol 30, pp 140–148.

PAROTID GLANDS: Animal study finds fluoride exposure results in oxidative stress and changes in oxidative biochemistry of the largest salivary gland which stimulates compensatory mechanisms and increases risk to the complex cell cytoskeleton.

<https://www.ncbi.nlm.nih.gov/labs/pmc/articles/PMC8794182/>

- Miranda GHN, et al. Effects of long-term fluoride exposure are associated with oxidative biochemistry impairment and global proteomic modulation, but not genotoxicity, in parotid glands of mice. *PLoS One.* 2022 Jan 27;17(1):e0261252.

NUTRITION & POLITICS: Private interests played a significant role at the start of 1940s public health endorsements of "fluoride-supplemented high-carbohydrate nutritional guidelines." This political effort biased scientific processes and required the reversals of three key scientific points in an absence of supporting evidence: 1) potential harms of fluoride exposure, 2) role of nutritional deficiencies in dental caries, and 3) that low-carbohydrate diets are recommended for dental caries prevention. This corrupted bias continues today: "The USDA and the WHO not only ignore the evidence that high-carbohydrate diets may lead to diseases other than dental caries, but also fail to prioritize high-quality evidence over low-quality evidence when writing their nutritional guidelines" per experts in evidence-based grading systems.

<https://www.mdpi.com/2072-6643/14/20/4263/html>

- Hujoel, P.P. Private Interests and the Start of Fluoride-Supplemented High-Carbohydrate Nutritional Guidelines. *Nutrients* 2022, 14, 4263.

Fluoridation Policy: *An Annotated Bibliography of Published Science*

LOWER PERFORMANCE IQ: Examined children's IQ at three separate time points (age 4, 5, and 6–12 years) to determine longitudinal and domain specific effects of prenatal fluoride exposure on IQ in mother-child dyads from the Early Life Exposures in Mexico to Environmental Toxicants (ELEMENT) cohort. Found prenatal exposure to fluoride, which is primarily from fluoridated salt programs, is associated with sustained impacts on IQ. Non-verbal abilities may be more susceptible to impairment from prenatal fluoride exposure as compared to verbal abilities.

<https://www.sciencedirect.com/science/article/abs/pii/S0013935122003206?via%3Dihub>

- Goodman C, et al. Domain-specific effects of prenatal fluoride exposure on child IQ at 4, 5, and 6–12 years in the ELEMENT cohort. *Environmental Research*. 2022

IODINE & IQ: MIREC study of child-mother dyads finds “the association between prenatal fluoride exposure and full-scale intelligence previously identified in this cohort was exacerbated by low maternal iodine in pregnancy among boys. These results, which were found among mother-child pairs living in fluoridated and non-fluoridated communities in Canada, underscore the importance of sufficient iodine intake in pregnancy to minimize the neurotoxicity of fluoride in boys.” <https://www.mdpi.com/2072-6643/14/14/2920/htm>

- Goodman CV, et al. Iodine Status Modifies the Association between Fluoride Exposure in Pregnancy and Preschool Boys' Intelligence. *Nutrients* 2022, 14, 2920.

HISTORICAL CONTEXT: Senior former public health officer and research scientist who has been skeptical about fluoridation since the 1950s with a particular interest in kidneys reflects on science, law, and conversations with notable pro and anti personalities over the decades.

<https://www.mdpi.com/2072-6643/14/17/3507/htm>

- Taves, Donald R. (2022) Fluoride: From Nutrient to Suspected Neurotoxin. *Nutrients* 14, no. 17: 3507.

GENOTOXIC: According to public health authorities, fluoride has a narrow range between the concentration which is beneficial and that which has adverse effects. The primary exposure to the fluoride-ion is through drinking water supplemented with fluorosilicic acid (FA). FA in ‘safe’ doses causes DNA damage in human osteoblast cells, reduces the telomere length and induces oxidative stress. Although combinations of fluoride with other toxins could have a synergistic effect, this study found that FA alone affects the genomic integrity of human bone cells.

<https://pubmed.ncbi.nlm.nih.gov/35483789/>

- Garcia ALH, Matzenbacher CA, Soares S, Rohr P, da Silva J. Fluorosilicic acid and cotinine, separately and in combination, induce genotoxicity and telomeric reduction in human osteoblast cell line MG63. *Mutat Res Genet Toxicol Environ Mutagen*. 2022 Apr-May;876-877:503474.

PROBIOTICS: Adding probiotics to school milk is more effective and less costly than fluoride varnish in preventing cavities in children. <https://pubmed.ncbi.nlm.nih.gov/35567374/>

- Rodriguez GA, Cabello RA, Borroni CP, Palacio RA. Cost-effectiveness of probiotics and fluoride varnish in caries prevention in preschool children. *J Public Health Dent*. 2022 May 14.

MALE FERTILITY: Fluoride, alone or in combination with arsenite both of which are common elements in drinking water, significantly suppressed the expression of steroidogenic enzymes and of the genes encoding these enzymes. “In conclusion, this study showed that exposure to As and F at environmentally relevant concentrations dispersed by water decreased testosterone production in Leydig cells, an important cell of the male reproductive system. The deleterious effects of even the lowest concentrations of As and F elements that can reach humans from the environment on the Leydig cell, and therefore on male infertility, emphasize necessity new safe limits for these elements.” <https://pubmed.ncbi.nlm.nih.gov/36182826/>

- Orta Yilmaz B, Aydin Y. Disruption of Leydig cell steroidogenic function by sodium arsenite and/or sodium fluoride. *Theriogenology*. 2022 Sep 22;193:146-156.

Fluoridation Policy: *An Annotated Bibliography of Published Science*

BODY & BRAIN: “Fluoride in higher concentrations or continuous exposure to lower doses are both found to induce mental imbalance in animals apart from the genotoxic, immunotoxic, and cytotoxic effects commonly observed. The behavioral profile of fluoride-treated animals has corroborated the clinical symptoms seen in fluoride-poisoned humans.”

<https://pubmed.ncbi.nlm.nih.gov/35488996/>

- Ottappilakkil H, Babu S, Balasubramanian S, Manoharan S, Perumal E. Fluoride Induced Neurobehavioral Impairments in Experimental Animals: a Brief Review. Biol Trace Elem Res. 2022 Apr 30.

OVARIAN & TESTICULAR: Animal study from in utero through puberty showing adverse impact on reproductive function. “Approximately 80–90% of fluoride absorbed by infants and children accumulates in the body. It can enter into the umbilical cord blood of the child from the mother through the placenta. In addition, significantly high fluoride content in breast milk is indicative of fluoride exposure to infants. Young children show less resistance to the toxic effects of fluoride than adults because of under-developed defense mechanisms and highly permeable blood-brain barrier.” <https://link.springer.com/article/10.1007/s12011-022-03220-8>

- Li, W., Sun, Z., Li, M. et al. Exposure to Fluoride From in Utero to Puberty Alters Gonadal Structure and Steroid Hormone Expression in Offspring Rats. Biol Trace Elem Res (2022).

BIRTH ANTHROPOMETRY: Using ELEMENT cohort, authors determined maternal exposure to fluoride affects length and weight of newborns with different susceptibility windows. Advises women avoid fluoride during pregnancy. <https://pubmed.ncbi.nlm.nih.gov/35660617/>

- Ortíz-García SG, Torres-Sánchez LE, Muñoz-Rocha TV, Mercado-García A, Peterson KE, Hu H, Osorio-Yáñez C, Téllez-Rojo MM. Maternal urinary fluoride during pregnancy and birth weight and length: Results from ELEMENT cohort study. Sci Total Environ. 2022 Jun 2.

KIDNEY KILLER: Using U.S. NHANES data, finds water fluoridation results in significantly higher plasma fluoride levels in healthy teens with lower renal function, suggesting a vicious feedback loop for those with CKD.

<https://www.sciencedirect.com/science/article/abs/pii/S0013935122009306>

- John Danziger, Laura E.Dodge, Howard Hu. Role of renal function in the association of drinking water fluoride and plasma fluoride among adolescents in the United States: NHANES, 2013–2016. Environmental Research. 7 June 2022.

BRAIN CONNECTION: Dental fluorosis (DF) is a structural tooth defect associated with a structural brain defects, i.e. “abnormal brain structure and brain function” which impacts concentration, learning, and memory, as well as increasing mental symptoms such as anxiety, tension, and depression. <https://pubmed.ncbi.nlm.nih.gov/36049331/>

- Ren C, Li HH, Zhang CY, Song XC. Effects of chronic fluorosis on the brain. Ecotoxicol Environ Saf. 2022 Aug 29;244:114021.

SKELETAL FLUOROSIS: Summary of the adverse effects of ≤ 0.7 mg/L exposure on skeletal and non-skeletal systems finds “low fluoride can indeed cause damage to human health. As a consequence, it is necessary to formulate the more appropriate water fluoride standard by taking into account the effects of low fluoride on various bodily systems. In addition, more and more evidence suggest that there exist individual differences in the effect of low fluoride on the body... most likely due to genetic polymorphisms. Therefore, it is necessary to consider both environmental exposure and the actual genetic situation of the individuals with respect to fluoride exposure.”

<https://pubmed.ncbi.nlm.nih.gov/35661326/>

- Zhou J, Sun D, Wei W. Necessity to Pay Attention to the Effects of Low Fluoride on Human Health: an Overview of Skeletal and Non-skeletal Damages in Epidemiologic Investigations and Laboratory Studies. Biol Trace Elem Res. 2022 Jun 6.

Fluoridation Policy: *An Annotated Bibliography of Published Science*

FLUORIDE-INDUCED DEVELOPMENTAL NEUROTOXICITY: Animal study using in vivo and in vitro data finds several mechanisms where fluoride exposure induced learning and memory deficits in male offspring mice affecting structure, chemical function, and size.

<https://www.sciencedirect.com/science/article/pii/S014765132200522X>

- Li W, Lu L, Zhu D, Liu J, Shi Y, Zeng H, Yu X, Guo J, Wei B, Cai Y, Sun M. Gestational exposure to fluoride impairs cognition in C57 BL/6 J male offspring mice via the p-Creb1-BDNF-TrkB signaling pathway. *Ecotoxicol Environ Saf*. 2022 Jul 1.

FLUORIDE-INDUCED LIVER INJURY: Animal study validates fluoride disrupted the glycolipid metabolism in the liver and resulted in the differential expression of 35 miRNAs and 480 mRNAs. <https://pubmed.ncbi.nlm.nih.gov/36222057/>

- Zhao Y, Yu Y, Ommati MM, Xu J, Wang J, Zhang J, Sun Z, Niu R, Wang J. Multiomics Analysis Revealed the Molecular Mechanism of miRNAs in Fluoride-Induced Hepatic Glucose and Lipid Metabolism Disorders. *J Agric Food Chem*. 2022 Oct 12.

NOT BENEFICIAL: Dental fluorosis (DF) is a qualitative defect in enamel from fluoride exposure early in life. DF is associated with other systemic conditions i.e. cognitive deficits, bone problems, thyroid disorders, etc. Significant incidences of DF are found in areas with 0.25 ppm fluoride in water concentration. Remineralization without fluoride exposure is advisable.

<https://www.mdpi.com/1660-4601/19/12/7153/htm>

- Struzycka I, et al. Assessing Fluorosis Incidence in Areas with Low Fluoride Content in the Drinking Water, Fluorotic Enamel Architecture, and Composition Alterations. *Int J Environ Res Public Health*. 2022 Jun 10;19(12):7153.

GENETIC SUSCEPTIBILITY: “In conclusion, there are alleles and genotypes of different single nucleotide polymorphisms involved in increasing or decreasing the risk of developing dental fluorosis.” <https://pubmed.ncbi.nlm.nih.gov/36354656/>

- González-Casamada C, Nevarez-Rascón M, Nevarez-Rascón A, et al. Single Nucleotide Polymorphisms and Dental Fluorosis: A Systematic Review. *Dent J (Basel)*. 2022 Nov 6;10(11):211.

FEMALE GUTS: Animal study details the process whereby fluoride exposure damages intestinal cells in the absence of estrogen which is consistent with a pattern of increased gastrointestinal illness among post-menopausal women in fluoridated communities.

<https://pubmed.ncbi.nlm.nih.gov/36252517/>

- Jin Y, Gao XY, Zhao J, Tian WS, Zhang YL, Tian EJ, Zhou BH, Wang HW. Estrogen deficiency aggravates fluoride-induced small intestinal mucosa damage and junctional complexes proteins expression disorder in rats. *Ecotoxicol Environ Saf*. 2022 Oct 14;246:114181.

INFLAMMATION: Using US NHANES data on children and animal studies, authors determined that fluoride exposure of typical children can and does result in inflammatory responses affecting immune function and disrupting white blood cell (WBC) function resulting in tissue damage. [ncbi.nlm.nih.gov/pmc/articles/PMC9608888/](https://pubmed.ncbi.nlm.nih.gov/36252517/)

- Den Besten P, Wells CR, Abduweli Uyghurturk D. Fluoride exposure and blood cell markers of inflammation in children and adolescents in the United States: NHANES, 2013-2016. *Environ Health*. 2022 Oct 27;21(1):102.

DOSE IN FOOD: Finds that fluoride exposure from foods prepared with fluoridated water have heavy fluoride concentrations not previously suspected that may exceed exposure from drinking water, dependent on dietary habits. Proposes reduction in fluoride exposure especially critical for low weight populations, such as infants, children and the frail.

<https://www.sciencedirect.com/science/article/pii/S0304389422019562>

- Sawangjang B, Takizawa S. Re-evaluating fluoride intake from food and drinking water: Effect of boiling and fluoride adsorption on food. *J Hazard Mater*. 2022 Oct 12.

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BLOOD PRESSURE: “This study suggested that fluoride exposure may affect childhood blood pressure.” <https://pubmed.ncbi.nlm.nih.gov/35168424/>

- Guo M, Afrim FK, Li Z, Li N, Fu X, Ding L, Feng Z, Yang S, Huang H, Yu F, Zhou G, Ba Y. Association between fluoride exposure and blood pressure in children and adolescents aged 6 to 19 years in the United States: NHANES, 2013-2016. *Int J Environ Health Res.* 2022 Feb 15:1-11.

LOW BIRTH WEIGHT & ENVIRONMENTAL JUSTICE: “Our findings suggest a significant association between excess water fluoride exposure (>0.7 ppm) and LBW weight in Hispanic women, independent of established LBW risk factors.” The EPA MCL/MCLG is 4 ppm.

<https://pubmed.ncbi.nlm.nih.gov/35897326/>

- Arun AK, Rustveld L, Sunny A. Association between Water Fluoride Levels and Low Birth Weight: National Health and Nutrition Examination Survey (NHANES) 2013-2016. *Int J Environ Res Public Health.* 2022 Jul 23;19(15):8956

EPA FAILURE & TSCA: Does not mention fluoride, but focuses on organizational patterns that result in greater risk and actual harm from chemical exposure to susceptible subpopulations.

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9454241/>

- Rayasam SDG, et al. Toxic Substances Control Act (TSCA) Implementation: How the Amended Law Has Failed to Protect Vulnerable Populations from Toxic Chemicals in the United States. *Environ Sci Technol.* 2022 Sep 6;56(17):11969-11982.

2021

BENCHMARK DOSE ANALYSIS: Using fluoride studies from MIREC and ELEMENT projects as input, the results of which are consistent with other studies, authors identify 0.2 mg/L as having an adverse impact on neurodevelopment. “The prospective studies offer strong evidence of prenatal neurotoxicity, and the benchmark results should inspire a revision of water-fluoride recommendations aimed at protecting pregnant women and young children.”

<https://pubmed.ncbi.nlm.nih.gov/34101876/>

- Grandjean P, Hu H, Till C, Green R, Bashash M, Flora D, Tellez-Rojo MM, Song P, Lanphear B, Budtz-Jørgensen E. A Benchmark Dose Analysis for Maternal Pregnancy Urine-Fluoride and IQ in Children. *Risk Analysis.* 8 June 2021.

LIFETIME EXPOSURE: Fluoridation is the primary source of fluoride exposure for 1,629 Canadians between 3 and 79 that finds substantially higher lifetime fluoride exposure in fluoridated communities using CHMS data, increasing with age. Vulnerable subpopulations to adverse effects of fluoride noted as the young, those who are iodine deficient, and post-menopausal women. <https://www.mdpi.com/1660-4601/18/12/6203/htm>

- Julia K. Riddell, Ashley J. Malin, Hugh McCague, David B. Flora, and Christine Till. Urinary Fluoride Levels among Canadians with and without Community Water Fluoridation. *Int. J. Environ. Res. Public Health* 2021, 18(12), 6203.

DF & COGNITION: Indian study finds significant relationship between impaired cognition and dental fluorosis in children. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8680901/>

- Prabhakar A, Abdulkhayarkutty K, Cheruvallil SV, Sudhakaran P. Effect of Endemic Fluorosis on Cognitive Function of School Children in Alappuzha District, Kerala: A Cross Sectional Study. *Ann Indian Acad Neurol.* 2021 Sep-Oct;24(5):715-720.

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KIDNEYS: This study of 1,070 adults found every 1 mg/L increment in the urinary fluoride concentrations was associated with significant increases of 22.8% in the risk of kidney function injury after adjusting for potential confounding factors. Authors conclude that long-term fluoride exposure is associated with compromised kidney function in adults, and that urinary NAG is a sensitive and robust marker of kidney dysfunction caused by fluoride exposure.

<https://pubmed.ncbi.nlm.nih.gov/34478979/>

- Wu L, Fan C, Zhang Z, Zhang X, et al. Association between fluoride exposure and kidney function in adults: A cross-sectional study based on endemic fluorosis area in China. *Ecotoxicol Environ Saf.* 2021 Aug 31;225:112735.

BEHAVIORAL CHANGES: Children in Cincinnati Childhood Allergy and Air Pollution Study (CCAAPS) assessed at age 12. Boys in particular did not experience significant anxiety or depression, yet had somatic behaviors based on their childhood urinary fluoride (CUF) concentrations, “seven times more likely to exhibit ‘at-risk’ internalizing symptomology.”

<https://pubmed.ncbi.nlm.nih.gov/34755609/>

- Adkins EA, Yolton K, Strawn JR, Lippert F, Ryan PH, Brunst KJ. Fluoride exposure during early adolescence and its association with internalizing symptoms. *Environ Res.* 2021 Oct 29;112296.

DENTAL FLUOROSIS: “The prevalence of dental fluorosis was 70% in the U.S. children and adolescents in survey of NHANES 2015–2016.” <https://pubmed.ncbi.nlm.nih.gov/34166938/>

- Dong H, Yang X, Zhang S, Wang X, Guo C, Zhang X, Ma J, Niu P, Chen T. Associations of low level of fluoride exposure with dental fluorosis among U.S. children and adolescents, NHANES 2015-2016. *Ecotoxicol Environ Saf.* 2021 Sep 15;221:112439.

CRITICAL WINDOWS: Using urine samples and test scores from 596 mother-child Canadian pairs in the MIREC prospective cohort, researchers found evidence that developmental neurological damage was based on timing of fluoride exposure and gender, “Associations between fluoride exposure and PIQ (performance IQ) differed based on timing of exposure. The prenatal window may be critical for boys, whereas infancy may be a critical window for girls.”

<https://pubmed.ncbi.nlm.nih.gov/34051202/>

- Farmus L, Till C, Green R, Hornung R, Martinez-Mier EA, Ayotte P, Muckle G, Lanphear B, Flora D. Critical Windows of Fluoride Neurotoxicity in Canadian Children. *Environ Res.* 2021 May 26;111315.

GENES: Several genes make individuals more vulnerable to the neurotoxic impact with gender differences, also affecting mitochondria and suggesting vulnerability to dementia. Chinese study of 952 school children between 7 and 13 using water, urinary, hair and nail fluoride identified multiple neurodevelopmental metabolic pathways that result in adverse effects from low fluoride exposures. <https://www.sciencedirect.com/science/article/pii/S0160412021003068>

- Yu X, Xia L, Zhang S, et al. Fluoride exposure and children's intelligence: Gene-environment interaction based on SNP-set, gene and pathway analysis, using a case-control design based on a cross-sectional study. *Environ Int.* 2021 Jun 4;155:106681.

GENETIC VULNERABILITY: Dopamine relative genes affect the susceptibility of individuals to fluoride toxicity even in safe water concentrations which result in lowered IQ so that “low-moderate fluoride exposure is inversely related to children's IQ.”

<https://pubmed.ncbi.nlm.nih.gov/33360592/>

- Zhao L, Yu C, Lv J, et al. Fluoride exposure, dopamine relative gene polymorphism and intelligence: A cross-sectional study in China. *Ecotoxicology and Environmental Safety.* 2021 Feb;209:111826.

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BRITTLE BONES: “In this cohort of postmenopausal women, the risk of fractures was increased in association with two separate indicators of fluoride exposure. Our findings are consistent with RCTs and suggest that high consumption of drinking water with a fluoride concentration of ~1 mg/L may increase both BMD (bone mineral density) and skeletal fragility in older women.” <https://pubmed.ncbi.nlm.nih.gov/33822648/>

- Helte E, Donat Vargas C, Kippler M, Wolk A, Michaëlsson K, Åkesson A. Fluoride in Drinking Water, Diet, and Urine in Relation to Bone Mineral Density and Fracture Incidence in Postmenopausal Women. *Environ Health Perspect.* 2021 Apr;129(4):47005.

OSTEOARTHRITIS: Identifies fluoride as an environmental chemical that has adverse effects on articular cartilage and osteoarthritis (OA) risk. “In full sample analysis, a 1 mg/L increase in UF (urinary fluoride) level was associated with a 27% higher risk of OA.”

<https://link.springer.com/article/10.1007/s12011-021-02937-2>

- Sowvanou, A., Meng, X., Zhong, N. et al. Association Between Osteoarthritis and Water Fluoride Among Tongyu Residents, China, 2019: a Case–Control of Population-Based Study. *Biol Trace Elem Res* (2021).

NO BENEFIT FOR PRESCHOOLERS: Polish study finds ‘optimal’ fluoride concentrations in water provide no dental benefit. Dental caries experience depended on oral hygiene and diet.

<https://www.sciencedirect.com/science/article/abs/pii/S0946672X2100016X>

- Opydo-Szymaczek J, et al. Fluoride exposure and factors affecting dental caries in preschool children living in two areas with different natural levels of fluorides. *Journal of Trace Elements in Medicine and Biology.* Volume 65. 2021.

REPRODUCTIVE HEALTH: “Median (IQR) water and plasma fluoride levels were 0.48 (0.53) mg/L and 0.34 (0.30) μ mol/L respectively. An IQR increase in water fluoride was associated with a 3.3 month earlier first menstrual period ($B = -0.28$, 95%CI: -0.54 , -0.02 , $p = 0.05$). Additionally, we observed a significant interaction between plasma fluoride and race/ethnicity in association with age of menarche ($p = 0.01$). For non-Hispanic black females, each IQR increase in plasma fluoride was associated with a 5-month earlier age of menarche ($B = -0.42$, 95%CI: -0.61 , -0.23 , $p < 0.001$).” <https://ehp.niehs.nih.gov/doi/abs/10.1289/isee.2020.virtual.O-OS-619>

- Fluoride exposure and reproductive health among adolescent females in the United States: NHANES 2013-2016. A. J. Malin, S. A. Busgang, J. C. Garcia, P. Curtin, and A. P. Sanders. ISEE National Meeting Presentation. *Environmental Health Perspectives.* 23-26 August 2021.

ALTERNATIVE: This systematic review and meta-analysis concludes that biomimetic hydroxyapatite-containing, fluoride-free oral care products are effective in reducing dental decay, especially in children without the risk of dental fluorosis and neurotoxicity inherent in topical use of fluoridated products. <https://files.cdha.ca/profession/journal/2752.pdf>

- Hardy Limeback, BSc, PhD, DDS; Joachim Enax, Dr; Frederic Meyer, Dr. Biomimetic hydroxyapatite and caries prevention: a systematic review and meta-analysis. | *Can J Dent Hyg* 2021;55(3): 148-159.

AMERICAN KIDNEYS: Using U.S. NHANES data from two recent cycles, finds ‘optimal’ amounts of fluoridated water results in high incidence of uric acid in adolescents suggesting higher risk of kidney disease and other illnesses. Identifies dose-response trend in plasma fluoride of teens.

<https://www.sciencedirect.com/science/article/pii/S0147651320315074>

- Yudan Wei, Jianmin Zhu, Sara Ann Wetzstein. Plasma and water fluoride levels and hyperuricemia among adolescents: A cross-sectional study of a nationally representative sample of the United States for 2013–2016. *Ecotoxicology and Environmental Safety.* Volume 208. 15 January 2021.

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TODDLERS: The Programming Research in Obesity, Growth, Environment and Social Stressors (PROGRESS) cohort included 948 mother-child pairs from Mexico City. Blinded testing of children between one and 24 months to examine associations between maternal fluoride intake from food and beverages during pregnancy and offspring neurodevelopment in this prospective and longitudinal study found, “higher exposure to fluoride from food and beverage consumption in pregnancy was associated with reduced cognitive outcome, but not with language and motor outcome in male offspring over the first two years of life.”

https://fluoridealert.org/wp-content/uploads/cantoral-2021.final_.pdf

- Alejandra Cantoral, Martha M. Tellez-Rojo, Ashley J. Malin, Lourdes Schnaas d, Erika Osorio-Valencia, Adriana Mercadob, E. Angeles Martínez-Mier, Robert O. Wright, Christine Till. Dietary fluoride intake during pregnancy and neurodevelopment in toddlers: A prospective study in the progress cohort. *Neurotoxicology* 87 (2021) 86–93.

CNS: Study of Chinese children consuming water with the low to moderate concentrations of fluoride considered safe by the US EPA determines that fluoride exposure was associated with the alteration of cholinergic system, dental fluorosis, IQ, and AChE, i.e the central nervous system is adversely impacted concurrent with fluoride exposure which also causes tooth defects and measurable deficits in cognition. <https://pubmed.ncbi.nlm.nih.gov/34808511/>

- Wang S, Zhao Q, Li G, Wang M, Liu H, Yu X, Chen J, Li P, Dong L, Zhou G, Cui Y, Wang M, Liu L, Wang A. The cholinergic system, intelligence, and dental fluorosis in school-aged children with low-to-moderate fluoride exposure. *Ecotoxicol Environ Saf.* 2021 Nov 20;228:112959.

NO SAFE DOSE: Study of Mexican children and their mothers using measurements of urinary fluoride and water concentrations associated dental fluorosis and lowered IQ with fluoride dose consistent with findings of larger studies in other countries. Authors declare WHO fluoride guidelines are unsafe and hypothesize that 0.045 F⁻ mg/day is a protective exposure

<https://www.mdpi.com/1660-4601/18/21/11490/htm>

- Fariás P, Estevez-García JA, Onofre-Pardo EN, Pérez-Humara ML, Rojas-Lima E, Álamo-Hernández U, Rocha-Amador DO. Fluoride Exposure through Different Drinking Water Sources in a Contaminated Basin in Guanajuato, Mexico: A Deterministic Human Health Risk Assessment. *International Journal of Environmental Research and Public Health.* 2021; 18(21):11490.

BABY BRAIN POISON: Exposure to fluoridated water (10 mg/L & 50 mg/L) beginning on the first day of pregnancy and continuing through the last day of breastfeeding shows chemical imbalances, cellular damage and changes in the hippocampus of Wistar rat offspring that would affect neurological development.

<https://pubmed.ncbi.nlm.nih.gov/33096359/>

- Ferreira MKM, Aragão WAB, Bittencourt LO, Puty B, Dionizio A, Souza MPC, Buzalaf MAR, de Oliveira EH, Crespo-Lopez ME, Lima RR. Fluoride exposure during pregnancy and lactation triggers oxidative stress and molecular changes in hippocampus of offspring rats. *Ecotoxicology and Environmental Safety.* 2021 Jan 15;208:111437.

BAD TEETH - BAD BRAIN: Chinese study confirm 1.6 ppm v. 0.1 ppm results in children with both damaged teeth and lower IQ. Authors validate that fluoride affects thyroid function, neurotransmitters and mitochondrial energy enzymes. There were no students with low IQ found in the area with low F level. There was high IQ among the 96.6% of the students who did not experience fluorosis.

<https://www.sciencedirect.com/science/article/pii/S021391121001965>

- Yani SI, Seweng A, Mallongi A, et al. The influence of fluoride in drinking water on the incidence of fluorosis and intelligence of elementary school students in Palu City. *Gac Sanit.* 2021;35 Suppl 2:S159-S163.

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GUTS & BRAINS: Memory function was reduced and gut microbiota structure was significantly altered in fluoride-exposed mice.

<https://www.sciencedirect.com/science/article/pii/S0147651321002190>

- Xin J, Wang H, Sun N, Bughio S, Zeng D, Li L, Wang Y, Khaliq A, Zeng Y, Pan K, Jing B, Ma H, Bai Y, Ni X. Probiotic alleviate fluoride-induced memory impairment by reconstructing gut microbiota in mice. *Ecotoxicol Environ Saf.* 2021 Jun 1;215:112108

INFLAMED GUTS: Exposure to fluoridated water at both doses (10 mg/L & 50 mg/L) inflame guts in rats and alters the gut microbiome as compared to control (0 mg/L).

<https://pubmed.ncbi.nlm.nih.gov/33508686/>

- Dionizio A, Uyghurturk DA, Melo CGS, Sabino-Arias IT, Araujo TT, Ventura TMS, Perles JVC, Zanon JN, Den Besten P, Buzalaf MAR. Intestinal changes associated with fluoride exposure in rats: Integrative morphological, proteomic and microbiome analyses. *Chemosphere.* 2021 Jan 11;273:129607.

PUBERTY: Black girls consuming optimally fluoridated water have earlier menarche.

<https://doi.org/10.1007/s12403-021-00448-y>

- Malin, A.J., Busgang, S.A., Garcia, J.C. et al. Fluoride Exposure and Age of Menarche: Potential Differences Among Adolescent Girls and Women in the United States. *Expo Health* (2021).

HARMFUL ADEQUATE INTAKE (AI): Study found "the levels of dietary F- intake were below the current AI, were greater towards the end of gestation and in women who were moderately and highly compliant with Mexican dietary recommendation" in ELEMENT cohort and recommended changing future dietary recommendations due to evidence of developmental neurotoxicity at even low dose exposure. <https://pubmed.ncbi.nlm.nih.gov/33602354/>

- Castiblanco-Rubio, G., Muñoz-Rocha, T., Cantoral, A., Téllez-Rojo, M., Ettinger, A., Mercado-García, A., Peterson, K.E., Hu, H., Martínez-Mier, E. (2021). Dietary Fluoride Intake Over the Course of Pregnancy in Mexican Women. *Public Health Nutrition*, 1-25.

CALCIUM & FLUORIDE IN PREGNANCY: Calcium intake during pregnancy lowers urinary fluoride (UF) concentrations by some unknown mechanism in ELEMENT cohort.

<https://pubmed.ncbi.nlm.nih.gov/34176079/>

- Castiblanco-Rubio GA, Muñoz-Rocha TV, Téllez-Rojo MM, Ettinger AS, Mercado-García A, Peterson KE, Hu H, Cantoral A, Martínez-Mier EA. Dietary Influences on Urinary Fluoride over the Course of Pregnancy and at One-Year Postpartum. *Biol Trace Elem Res.* 2021 Jun 26.

SAFETY: Evidence of dental fluorosis and other adverse effects to bodies and brains from supposed safe concentrations is alarming. "The safety of public health approach of drinking water fluoridation for global dental caries reduction are urgently needed further research."

<https://www.sciencedirect.com/science/article/pii/S0147651321005510?via%3Dihub>

- Dong H, Yang X, Zhang S, Wang X, Guo C, Zhang X, Ma J, Niu P, Chen T. Associations of low level of fluoride exposure with dental fluorosis among U.S. children and adolescents, NHANES 2015-2016. *Ecotoxicol Environ Saf.* 2021 Jun 22;221:112439.

SKELETAL FLUOROSIS: This Chinese study of the pathogenetic progression of skeletal fluorosis, details how local signaling pathways, hormones, promoter DNA hypermethylation, RNA expression etc. are affected by fluoride exposure leading to pain and disability.

<https://www.mdpi.com/1422-0067/22/21/11932/htm>

- Qiao L, Liu X, He Y, Zhang J, Huang H, Bian W, Chilufya MM, Zhao Y, Han J. Progress of Signaling Pathways, Stress Pathways and Epigenetics in the Pathogenesis of Skeletal Fluorosis. *International Journal of Molecular Sciences.* 2021; 22(21):11932.

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DEPRESSION: Animal study finds negative changes in brain structure and behavior with exposure to sodium fluoride (NAF). <https://pubmed.ncbi.nlm.nih.gov/34735150/>

- Zhou G, Hu Y, Wang A, Guo M, Du Y, Gong Y, Ding L, Feng Z, Hou X, Xu K, Yu F, Li Z, Ba Y. Fluoride Stimulates Anxiety- and Depression-like Behaviors Associated with SIK2-CRTC1 Signaling Dysfunction. *J Agric Food Chem*. 2021 Nov 4. PMID: 34735150.

OLD PEOPLE: Chinese study using individual measure of blood fluoride finds those consuming fluoride in water at or above 2 ppm as compared to those in areas with <0.8 ppm are more likely to suffer cognitive decline and dementia. The US EPA certifies that up to 4 ppm is safe.

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8656079/>

- Ren C, Zhang P, Yao XY, Li HH, Chen R, Zhang CY, Geng DQ. The cognitive impairment and risk factors of the older people living in high fluorosis areas: DKK1 need attention. *BMC Public Health*. 2021 Dec 9;21(1):2237.

DECEPTION: This historical analysis documents how the ADA suppressed the established science that vitamin D was necessary for healthy teeth and bones in order to promote falsely fluoride which was and is more profitable for their membership. “Public health may well depend on looking at professional societies no different than the way we look at the pharmaceutical industry—conflicted organizations with a power to shape conventional wisdom based on fragile evidence.” <https://www.mdpi.com/2072-6643/13/12/4361/htm#>

- Hujoel PE. How a Nutritional Deficiency Became Treated with Fluoride. *Nutrients*. 2021.

2020

AMERICAN FETAL EXPOSURE: Study on pregnant women in California and Montana find, “Fluoride concentrations in urine, serum, and amniotic fluid from women were positively correlated to public records of community water fluoridation” and that concentration is consistent with findings of Canadian studies that find these concentrations are associated with increased learning disabilities and lower IQ in offspring.

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7132865/>

- Abduweli Uyghurturk D, Goin DE, Martinez-Mier EA, Woodruff TJ, DenBesten PK. Maternal and fetal exposures to fluoride during mid-gestation among pregnant women in northern California. *Environ Health*. 2020 Apr 6;19(1):38.

THYROID & IQ: Concentrations of fluoride in drinking water considered optimal and safe in the US result in altered thyroid function and lowered IQ in Chinese children.

<https://www.sciencedirect.com/science/article/pii/S0160412019301370>

- Wang M, Liu L, Li H, et al. Thyroid function, intelligence, and low-moderate fluoride exposure among Chinese school-age children. *Environment International*. Volume 134, January 2020.

BLOOD: Canadian Health Measures Survey (CHMS) collects extensive biomonitoring data used to assess the exposure of Canadians to environmental chemicals finds higher fluoride in urine associated with significantly higher blood lead, urinary lead, etc. Also finds urinary selenium is significantly lower in fluoridated Canadian communities, “this is the first study where biomonitoring data from multiple cycles of CHMS were combined in order to generate robust estimates for subsets of the Canadian population. Such assessments can contribute to a regional-level prioritization of control measures to reduce the exposure of Canadians to chemicals in their environment.”

<https://www.ncbi.nlm.nih.gov/pubmed/31972364?dopt=Abstract>

- Valcke M, Karthikeyan S, Walker M, Gagné M, Copes R, St-Amand A. Regional variations in human chemical exposures in Canada: A case study using biomonitoring data from the Canadian Health Measures Survey for the provinces of Quebec and Ontario. *Int J Hyg Environ Health*. 2020 Jan 20;225:113451.

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OVERDOSED CANADIAN BABIES: MIREC study documents Canadian bottle-fed babies have lower IQ in optimally fluoridated communities while breast fed babies have extremely low F and significantly higher IQ. <https://www.sciencedirect.com/science/article/pii/S0160412019326145>

- Till C, Green R, Flora D, Hornung R, Martinez-Miller EA, Blazer M, Farmus L, Ayotte P, Muckle G, Lanphear B. Fluoride exposure from infant formula and child IQ in a Canadian birth cohort. *Environment International*. 2020.

BIASED NARRATIVES: Canadian researchers comment on “expert” attacks on the high quality studies that contradict the dental CWF narrative, i.e. political suppression of scientific facts. <https://www.nature.com/articles/s41390-020-0973-8>

- Till, C., Green, R. Controversy: The evolving science of fluoride: when new evidence doesn't conform with existing beliefs. *Pediatr Res* (2020).

BONE HEALTH: Low to moderate fluoride exposure weakens and damages bones in women. <https://www.sciencedirect.com/science/article/abs/pii/S0147651320308708>

- Minghui Gao et al, Association between low-to-moderate fluoride exposure and bone mineral density in Chinese adults: Non-negligible role of RUNX2 promoter methylation. *Ecotoxicology and Environmental Safety*. Volume 203, 15 October 2020.

BONES: Found an age-specific association between fluoride exposure and altered CALCA methylation in adult women, affecting bone health. <https://pubmed.ncbi.nlm.nih.gov/32283421/>

- Sun R, Zhou G, Liu L, Ren L, Xi Y, Zhu J, Huang H, Li Z, Li Y, Cheng X, Ba Y. Fluoride exposure and CALCA methylation is associated with the bone mineral density of Chinese women. *Chemosphere*. 2020 Aug;253:126616.

SEX HORMONES IN FLUORIDATED US: “The data indicated gender- and age-specific inverse associations of fluoride in plasma and water with sex steroid hormones of total testosterone, estradiol and SHBG in U.S. children and adolescents.”

<https://www.sciencedirect.com/science/article/pii/S0269749119357963>

- Bai, R., Huang, Y., Wang, F., & Guo, J. (2020). Associations of fluoride exposure with sex steroid hormones among U.S. children and adolescents, NHANES 2013–2016. *Environmental Pollution*, 114003

NERVOUS SYSTEM: The enteric nervous system (ENS) is called the second brain and governs the gastrointestinal track. Includes dopamine & serotonin function. Study finds “fluoride exposure during pregnancy and lactation might induce ENS developmental defects.”

<https://link.springer.com/article/10.1007/s12011-020-02249-x>

- Sarwar, S., Quadri, J.A., Kumar, M. et al. Apoptotic and Degenerative Changes in the Enteric Nervous System Following Exposure to Fluoride During Pre- and Post-natal Periods. *Biol Trace Elem Res* (2020).

ENDOCRINE SYSTEM REVIEW: The endocrine system includes the pineal gland, hypothalamus, pituitary gland, thyroid with parathyroid glands, thymus, pancreas (partial endocrine function), adrenal glands, as well as male and female gonads (testes and ovaries) which are adversely effected by exposure to fluoride.

<https://www.sciencedirect.com/science/article/abs/pii/S0045653520317604>

- Marta Skórka-Majewicz et al, Effect of fluoride on endocrine tissues and their secretory functions -- review. *Chemosphere*, Volume 260, December 2020, 127565.

PINEAL GLAND & MELATONIN: Fluoride calcifies the pineal gland and interferes with enzyme function, hormones and sleep patterns. <https://www.mdpi.com/2076-3417/10/8/2885>

- Dariusz Chlubek, Maciej Sikora. Fluoride and Pineal Glad. *Applied Sciences*. 22 April 2020.

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WHO IGNORES KIDNEYS: WHO guidelines of safety below 1.5 ppm fluoride concentration is wrong. “The available guidelines for drinking water are solely based on healthy populations with normal renal function. But, it is evident that once the kidney function is impaired, patients enter a vicious cycle as fluoride gradually accumulates in the body, further damaging the kidney tissue.”
<https://www.sciencedirect.com/science/article/abs/pii/S0045653520313795>

- Shanika Nanayakkara, et al. The Influence of fluoride on chronic kidney disease of uncertain aetiology (CKDu) in Sri Lanka. Chemosphere. Volume 257, October 2020, 127186

PEDIATRIC BONE DISEASE: Identifies fluoride concentrations in water above 1.2 ppm as “dangerously high” that can cause pediatric bone disease. Urine measurements of fluoride in those afflicted are below the fluoride concentrations in women living in optimally fluoridated communities per 2017 Canadian study by Green et al.

<https://pubmed.ncbi.nlm.nih.gov/32692054/>

- Nipith Charoenngam, Muhammet B Cevik, Michael F Holick. Diagnosis and management of pediatric metabolic bone diseases associated with skeletal fragility. Curr Opin Pediatr. 2020 Aug;32(4):560-573.

EPA ON ENVIRONMENTAL STRESS: EPA authors find that exposure to fluoride has the greatest adverse impact on cognitive ability in children, even more than lead.

<https://www.mdpi.com/1660-4601/17/15/5451/html>

- Frances M. Nilsen, Jazmin D.C. Ruiz and Nicole S. Tulve. A Meta-Analysis of Stressors from the Total Environment Associated with Children’s General Cognitive Ability. Int. J. Environ. Res. Public Health 2020, 17(15), 5451.

FLUORIDE SOURCE: Compared MIREC, ELEMENT & PROGRESS data. MIREC & ELEMENT differed from PROGRESS in that “daily food and beverage fluoride intake was not associated with CUF in PROGRESS” but study “found that CUF (child urinary fluoride) levels are comparable among children in Mexico City and fluoridated Canadian communities, despite distinct sources of exposure.” <https://pubmed.ncbi.nlm.nih.gov/33233802/>

- Green, R., Till, C., Cantoral Preciado, A. D. J., Lanphear, B., Angeles Martinez-Mier, E., Ayotte, P., Wright, R. O., Tellez-Rojo, M. M., & Malin, A. J. (2020). Associations between urinary, dietary, and water fluoride concentrations among children in Mexico and Canada. Toxics, 8(4), 1-11. [110].

SPERM MOTILITY: Animal study determines mechanisms how fluoride exposure lowers sperm quality and male reproductive function. <https://pubmed.ncbi.nlm.nih.gov/31901658/>

- Liang C, He Y, Liu Y, Gao Y, Han Y, Li X, Zhao Y, Wang J, Zhang J. Fluoride exposure alters the ultra-structure of sperm flagellum via reducing key protein expressions in testis. Chemosphere. 2020 May;246:125772.

DENTAL FLUOROSIS & CWF CESSATION: Dental literature review by dentists finds “a significant decrease in the prevalence of fluorosis post cessation or reduction in the concentration of fluoride added to the water supply.”

<https://pubmed.ncbi.nlm.nih.gov/32598322/>

- Nor Azlida Mohd Nor, Kuala Lumpur, Barbara L. Chadwick, Damian JJ. Farnell, Ivor G. Chestnutt. The impact of stopping or reducing the level of fluoride in public water supplies on dental fluorosis: a systematic review. Reviews on Environmental Health. 2020.

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2019

SLEEP & PINEAL GLAND: "Chronic low-level fluoride exposure may contribute to changes in sleep cycle regulation and sleep behaviors among older adolescents in the US."

<https://ehjournal.biomedcentral.com/articles/10.1186/s12940-019-0546-7>

- Malin, A.J., Bose, S., Busgang, S.A. et al. Fluoride exposure and sleep patterns among older adolescents in the United States: a cross-sectional study of NHANES 2015–2016. *Environ Health* 18, 106 (2019)

ADHD: Youth in optimally fluoridated Canadian communities are almost 3 times more likely to be diagnosed with ADHD and have significantly higher rates of other learning disabilities as compared to their counterparts in non-fluoridated communities on a dose-response trend line.

<https://www.sciencedirect.com/science/article/pii/S0160412019315971>

- Riddell JK, et al. Association of water fluoride and urinary fluoride concentrations with attention deficit hyperactivity disorder in Canadian youth. *Environment International*. Volume 133, Part B, December 2019.

ASD: Increased exposure to fluoride is associated with higher incidence of ASD in regions with fluoridated water or endemic fluorosis. Based on biological plausibility and incidence, authors hypothesize that increased fluoride exposure is an environmental risk factor for autism.

<https://www.mdpi.com/1660-4601/16/18/3431/html>

- Strunecka A, Strunecky O. Chronic Fluoride Exposure and the Risk of Autism Spectrum Disorder. *Int. J. Environ. Res. Public Health* 2019, 16(18), 3431.

PRENATAL: Three measurements in high quality NIH sponsored prospective cohort study (MIREC) found significantly lowered IQ in offspring of mostly white, well-educated Canadian women living in 'optimally' fluoridated communities.

<https://jamanetwork.com/journals/jamapediatrics/fullarticle/2748634>

- Green R, Lanphear B, Hornung R, et al. (2019) Association Between Maternal Fluoride Exposure During Pregnancy and IQ Scores in Offspring in Canada. *JAMA Pediatrics*. 2019.

KIDNEY & LIVER: Researchers at Mt. Sinai Medical School find American teens in optimally fluoridated American towns have markers for altered kidney & liver parameters that puts them at higher risk for kidney & liver disease as adults. Notes the primary source of fluoride is water.

<https://www.sciencedirect.com/science/article/pii/S0160412019309274>

- Malin AJ, Lesseur C, Busgang SA, Curtin P, Wright RO, Sanders AP. Fluoride exposure and kidney and liver function among adolescents in the United States: NHANES, 2013–2016. *Environment International*. August 8, 2019.

GUTS: Animal study on microbiome health and immunity documents fluoride causes serious damage to rectal structure and significantly inhibits proliferation of rectal epithelial cells.

<https://www.ncbi.nlm.nih.gov/pubmed/31885060/>

- Wang H., Miao C., Liu J. et al. Fluoride-induced rectal barrier damage and microflora disorder in mice. *Environ Sci Pollut Res* (2019).

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TEETH: An analysis of the dental fluorosis data in three U.S. NHANES reports noted that more than half of American teens have fluoride damaged teeth as the result of too much fluoride consumption during childhood. This results in costly cosmetic dentistry in young adulthood for millions as well as increased decay in the more severely affected.

(20% very mild + 15% mild + 28% moderate + 3% severe = 65% afflicted per 2011-12 data)

<http://fluoridealert.org/wp-content/uploads/neurath.2019-1.pdf>

- Neurath C, Limeback H, Osmunson B, et al. (2019) Dental Fluorosis Trends in US Oral Health Surveys: 1986 to 2012. JDR Clinical & Translational Research.

ALZHEIMER'S: Even low concentrations of fluoride in drinking water at or below concentrations deemed optimal or safe by the WHO result in a pattern of increased dementia.

<https://www.ncbi.nlm.nih.gov/pubmed/30868981>

- Russ TC, Killin LOJ, Hannah J, Batty GD. Aluminium and fluoride in drinking water in relation to later dementia risk. The British Journal of Psychology. March 2019.

DNA DAMAGE: Mitochondrial dysfunction associated with dental fluorosis observed in Chinese children with fluoride concentrations in water identified as optimal or safe per U.S. authorities. Gender differences to the fluoride induced oxidative stress also noted.

<https://www.sciencedirect.com/science/article/pii/S0160412018326291?via%3Dihub>

- Zhou G, Yang L, Luo C, et al. Low-to-moderate fluoride exposure, relative mitochondrial DNA levels, and dental fluorosis in Chinese children. Environment International. Volume 127, June 2019, Pages 70-77.

DEMENTIA: Describes mechanism by which the effectiveness of the two most popular drugs used to treat Alzheimer's & other neurodegenerative dementia disease is reduced or blocked by fluoride. <https://www.mdpi.com/1660-4601/16/1/10/html>

- Marta Goschorska, Izabela Gutowska, Irena Baranowska-Bosiacka, Katarzyna Piotrowska, Emilia Metryka, Krzysztof Safranow, Dariusz Chublek. Influence of Acetylcholinesterase Inhibitors Used in Alzheimer's Disease Treatment on the Activity of Antioxidant Enzymes and the Concentration of Glutathione in THP-1 Macrophages under Fluoride-Induced Oxidative Stress. Int. J. Environ. Res. Public Health, 2019, 16(1), 10.

ADULT BRAINS: First long term NaF animal study (10 weeks) using moderate levels of fluoride finds a number of histological changes including in parts of the brain associated with memory and learning. <https://www.sciencedirect.com/science/article/pii/S0045653518317508>

- Pei Jiang, Gongying Li, Xueyuan Zhou, Changshui Wang, Yi Qiao, Dehua Liao, Dongmei Shi. Chronic fluoride exposure induces neuronal apoptosis and impairs neurogenesis and synaptic plasticity: Role of GSK-3 β /b-catenin pathway. Chemosphere. Volume 214, January 2019, Pages 430-435.

DELAYED MALE PUBERTY: This 4th study from the NIH sponsored ELEMENT investigation of the prenatal impact of low-dose prenatal exposure found a significant pattern of delayed puberty for boys associated with maternal fluoride as measured in urine samples. Female data showed non-significant trend towards earlier menarche. More study needed to determine the impact on sexual development. <https://www.ncbi.nlm.nih.gov/pubmed/30922319>

- Liu Y, Téllez-Rojo M, Hu H, et al. Fluoride exposure and pubertal development in children living in Mexico City. Environ Health. 2019 Mar 29;18(1):26.

ANXIETY & DEPRESSION: Both rats and children experience changes in brain chemistry from extended exposure to fluoride which affects mood. Serotonin and the prefrontal cortex are impacted. Studies that only examine short-term exposure are inadequate to detect these changes which are more pronounced in females.

<https://www.sciencedirect.com/science/article/abs/pii/S0031938418309375>

- Lu F, Zhang Y, Trevedi A, et al. (2019) Fluoride related changes in behavioral outcomes may relate to increased serotonin. Physiology & Behavior.

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EYE DISEASE: Fluoride is a poison that has biological impact on consumers in any dose, contributing to the development of cataracts, glaucoma and macular degeneration.

<https://www.mdpi.com/1660-4601/16/5/856>

- Waugh DT. The Contribution of Fluoride to the Pathogenesis of Eye Diseases: Molecular Mechanisms and Implications for Public Health. *Int. J. Environ. Res. Public Health*. 2019, 16(5), 856.

BONES & GENES: This 30 day animal study at 8 mg/L fluoride documents DNA & RNA damage that inhibits gene expression which can be passed on through generations affecting bone development and contributing to weak bones, blood & bone cancers and skeletal fluorosis.

<https://www.sciencedirect.com/science/article/pii/S0147651318311734?via%3Dihub>

- Atule P. Daiwile, Prashant Tarale, Saravanadevi Sivanesan, et al. Role of fluoride induced epigenetic alterations in the development of skeletal fluorosis. *Ecotoxicology and Environmental Safety*. Volume 169, March 2019, Pages 410-417.

BRAIN INJURY: Fluoride interferes with calcium metabolism which impacts brain chemistry and poisons the hippocampus. “The imbalance of calcium metabolism caused by fluorosis may be a pathogenesis of brain injury induced by fluoride.”

<https://www.sciencedirect.com/science/article/pii/S0045653518324007>

- Qiuli Yu, Dandan Shao. Rui Zhang, Wei Ouyang, Zigui Zhang. Effects of drinking water fluorosis on L-type calcium channel of hippocampal neurons in mice. *Chemosphere*. Volume 220, April 2019, Pages 169-175. [Online Ahead of Print]

BRAIN DAMAGE: Prenatal & postnatal animal experiment using 10, 50 and 100 mg/L to simulate human experience documents mitochondrial damage and neuronal death as mechanism that result in learning and memory impairments.

<https://www.ncbi.nlm.nih.gov/pubmed/30659323>

- Zhao, Q., Niu, Q., Chen, J. et al. Roles of mitochondrial fission inhibition in developmental fluoride neurotoxicity: mechanisms of action in vitro and associations with cognition in rats and children. *Arch Toxicol* (2019).

IODINE: Identifies and discusses the biochemical and hormonal impact of fluoride and fluoridation policy on iodine metabolism with consideration of related neurodevelopmental and pathological disorders. <https://www.mdpi.com/1660-4601/16/6/1086>

- Waugh DT. Fluoride Exposure Induces Inhibition of Sodium/Iodide Symporter (NIS) Contributing to Impaired Iodine Absorption and Iodine Deficiency: Molecular Mechanisms of Inhibition and Implications for Public Health. *Int. J. Environ. Res. Public Health* 2019, 16, 1086.

BIOLOGY OF POISON: Deep dive into the biological impact of fluoride that affects metabolism, hormones, immune function, etc. “Moreover, the findings of this study further suggest that there are windows of susceptibility over the life course where chronic F exposure in pregnancy and early infancy may impair Na⁺, K⁺-ATPase activity with both short- and long-term implications for disease and inequalities in health.” <https://www.mdpi.com/1660-4601/16/8/1427>

- Waugh DT. Fluoride Exposure Induces Inhibition of Sodium-and Potassium-Activated Adenosine Triphosphatase (Na⁺, K⁺-ATPase) Enzyme Activity: Molecular Mechanisms and Implications for Public Health. *Int. J. Environ. Res. Public Health* 2019, 16(8), 1427

DOSE RESPONSE: Three month study on adult rats found “fluoride can impair the learning ability of rats, which may be related to the induction of autophagy in rat hippocampal neurons.”

<https://www.ncbi.nlm.nih.gov/pubmed/31111310>

- Zhang C, Huo S, Fan Y, Gao Y, Yang Y, Sun D. Autophagy May Be Involved in Fluoride-Induced Learning Impairment in Rats. *Biol Trace Elem Res*. 2019 May 20.

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GENETIC SUSCEPTIBILITY: Review of recent scientific literature on biological impact. Same exposure in same population affect individuals differently, suggesting genetic vulnerability.

<https://onlinelibrary.wiley.com/doi/full/10.1111/jcmm.14185>

- Wei, W, Pang, S, Sun, D. The pathogenesis of endemic fluorosis: Research progress in the last 5 years. *J Cell Mol Med.* 2019; 23: 2333– 2342.

MITOCHONDRIA: Prenatal and postnatal exposure to fluoride results in mitochondrial abnormalities, autophagy and apoptosis contributing to neuronal death.

<https://www.ncbi.nlm.nih.gov/pubmed/30659323>

- Zhao, Q., Niu, Q., Chen, J. et al. Roles of mitochondrial fission inhibition in developmental fluoride neurotoxicity: mechanisms of action in vitro and associations with cognition in rats and children. *Arch Toxicol* (2019).

NUTRITION: The f-ion is a poison but the bioavailability of CaF is different than NaF as calcium is the antidote to fluoride poisoning. In addition to being in water and dental products, 20% of pharma and 40% of agrichemicals have a fluoride base. Consequently, people are exposed to excessive amounts of fluoride which contributes to chronic disease.

<https://journals.matheo.si/index.php/ACSi/article/view/4932/2095>

- Stepec D, Ponikvar-Svet M. Fluoride in Human Health & Nutrition. *Acta Chim Slov.* 2019, 66.

SYNERGY, SUSCEPTIBILITY & TSCA: Accurately identifying highly exposed groups and the intrinsic and extrinsic factors that affect susceptibility require adequately assessing the aggregate exposure among vulnerable groups. The 2016 Lautenberg update to the 1976 Toxic Substance Control Act (TSCA) requires performing a challenging and scientifically disciplined risk assessment that mitigates risk, such as calculating the impact of combined fluoride exposure from fluoridated pesticides in food and fluoridated water on young children.

<https://www.ncbi.nlm.nih.gov/labs/pmc/articles/PMC6715167/>

- Koman PD, Singla V, Lam J, Woodruff TJ. Population susceptibility: A vital consideration in chemical risk evaluation under the Lautenberg Toxic Substances Control Act. *PLoS Biol.* 2019 Aug 29;17(8):e3000372.

2018

THYROID: 18% of people drinking 'optimally' fluoridated water in Canadian communities have a heightened risk of low thyroid function because fluoride interferes with iodine metabolism. Many of them will be sub-clinical and not know they are mildly hypothyroid, which nevertheless increases their risk for diabetes, high cholesterol, and other problems. Study excluded those already diagnosed with thyroid disease. (CHMS)

<https://www.sciencedirect.com/science/article/pii/S016041201830833X>

- Ashley J. Malin, Julia Riddell, Hugh McCague, Christine Till. Fluoride exposure and thyroid function among adults living in Canada: Effect modification by iodine status. *Environment International.* Volume 121, Part 1, December 2018, Pages 667-674.

THYROID: Even 0.5 ppm fluoride in water has an adverse impact on thyroid hormones. Water is currently fluoridated to 0.7 ppm, a reduction from up to 1.2 ppm in 2015.

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5805681/>

- Z. Kheradpisheh et al. (2018) Impact of Drinking Water Fluoride on Human Thyroid Hormones: A Case-Control Study. *Scientific Reports.* volume 8.

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OVERDOSED BABIES: Over one third of babies (37%) in fluoridated American communities consume amounts of fluoride in excess of the upper limits of fluoride considered safe per government regulations. Even 4% of babies in non-fluoridated communities are overdosed on fluoride due to consumption of products made with fluoridated water. At the very least, this puts these children at high risk for developing dental fluorosis. Dental fluorosis is associated with increased incidence of learning disabilities, broken bones and kidney disease.

<http://jocpd.org/doi/10.17796/1053-4625-43.1.7>

- Claudia X Harriehausen, Fehmida Z Dosani, Brett T Chiquet, Michelle S Barratt, and Ryan L Quock. Fluoride Intake of Infants from Formula. *Journal of Clinical Pediatric Dentistry*. 2018.

GOVERNMENT BIAS: A National Toxicology Program animal experiment studying the impact of fluoride consumption used the wrong rats, the wrong dose, and the wrong study design in order to manufacture a finding of no prenatal or postnatal effect.

<https://www.sciencedirect.com/science/article/pii/S0306987718308600>

- Karen Favazza Spencer, Hardy Limeback. Blood is Thicker Than Water: Flaws in a National Toxicology Program Study. *Medical Hypotheses*. Volume 121. December 2018. Pages 160-163.

ALZHEIMER'S DISEASE: Describes impact of fluoride-induced stress and inflammation in the development of Alzheimer's disease and demonstrates the mechanism for cell death in its worsening over time. <https://www.mdpi.com/1422-0067/19/12/3965>

- Goschorska M, et al. Potential Role of Fluoride in the Etiopathogenesis of Alzheimer's Disease. *Int. J. Mol. Sci.* 2018, 19 (12), 3965.

DENTAL FLUOROSIS: In 2001–2002, the prevalence of “very mild and above” dental fluorosis in adolescents aged 16 and 17 years was 29.7%. In 2011–2012, it was 61.3%. The authors of the study found: “There was a difference of 31.6% in dental fluorosis prevalence.” That difference was expressed in “percentage points” but the difference in “prevalence” of very mild and above dental fluorosis in 2011-2012 was twice as high as in 2001-2002. It went from 29.7% to 61.3%: 106% higher. The authors concluded: “The continued increase in fluorosis rates in the U.S. indicates that additional measures need to be implemented to reduce its prevalence.”

<https://pubmed.ncbi.nlm.nih.gov/29500282/>

- Wiener RC, Shen C, Findley P, Tan X, Sambamoorthi U. Dental Fluorosis over Time: A comparison of National Health and Nutrition Examination Survey data from 2001-2002 and 2011-2012. *J Dent Hyg.* 2018 Feb;92(1):23-29.

PREGNANT WOMEN: Pregnant Canadian women drinking 'optimally' fluoridated water had twice the fluoride exposure per individual testing as compared to pregnant women in non-fluoridated Canadian communities - and consistent with the range in the Mexican women in the ELEMENT cohort whose children had up to 6 points lowered IQ based on prenatal exposure to fluoride (from salt). The Canadian study excluded those with health conditions such as kidney disease as well as considered confounding factors such as tea consumption.

<https://ehp.niehs.nih.gov/doi/pdf/10.1289/EHP3546>

- Christine Till, Rivka Green, John G. Grundy, Richard Hornung, Raichel Neufeld, E. Angeles Martinez-Mier, Pierre Ayotte, Gina Muckle, and Bruce Lanphear. Community Water Fluoridation and Urinary Fluoride Concentrations in a National Sample of Pregnant Women in Canada. *Environmental Health Perspectives*. October 2018.

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LEARNING DISABILITIES: Over 200 children were individually tested. Study found attention deficit disorder apparently caused by their prenatal exposure to fluoride specific to dose. This is the 3rd report out of the NIH sponsored 12 year ELEMENT project that has confirmed low dose prenatal exposure to fluoride consistent with exposure in 'optimally' fluoridated communities causes subtle but permanent brain damage for many consumers. Excluded those with history of mental illness or conditions such as diabetes and renal disease.

<https://www.sciencedirect.com/science/article/pii/S0160412018311814>

- Morteza Bashash, Maelle Marchand, Howard Hu, Christine Till, Angeles Martinez-Mier, Brisa N. Sanchez, Niladri Basu, Karen Peterson, Rivka Green, Lourdes Schnaas, Adriana Mercado-García, Mauricio Hernández-Avila, Martha María Téllez-Rojo. Prenatal fluoride exposure and attention deficit hyperactivity disorder (ADHD) symptoms in children at 6–12 years of age in Mexico City. *Environment International*. Volume 121, Part 1, December 2018, Pages 658-666.

CANCER: Researchers who include an IARC scientist find esophageal cancer is 9.4 times more prevalent among those with dental fluorosis in the endemic fluorosis regions of Kenya. Provides biological plausibility that inflammatory fluoride affects microbiome and other biological mechanisms. Recommends more study. <https://www.ncbi.nlm.nih.gov/pubmed/30582155/>

- Menya D, Maina SK, Kibosia C, Kigen N, Oduor M, Some F, Chumba D3, Ayuo P, Middleton DR, Osano O, Abedi-Ardekani B, Schüz J, McCormack V. Dental fluorosis and oral health in the African Esophageal Cancer Corridor: Findings from the Kenya ESCCAPE case-control study and a pan-African perspective. *Int J Cancer*. 2018 Dec 23.

KIDNEYS: Fluoride is a common exposure that is selectively toxic to the kidneys.

<https://www.sciencedirect.com/science/article/pii/S0270929518301827>

- Lash LH. Environmental and Genetic Factors Influencing Kidney Toxicity. *Seminars in Nephrology*. Volume 39, Issue 2, March 2019, Pages 132-140.

IQ & DF: Between 0.5 and 3.9 mg/L, found every 0.1 mg/L increased dental fluorosis by 2.24% and every 0.5 mg/L decreases IQ by 2.67 points. Also found half as many kids with high IQ children with higher F- dose. <https://www.ncbi.nlm.nih.gov/pubmed/29870912>

- Yu X et al. Threshold effects of moderately excessive fluoride exposure on children's health: A potential association between dental fluorosis and loss of excellent intelligence. *Environ Int*. 2018 Jun 2;118:116-124.

WORSE THAN ARSENIC: "In conclusion, F exposure was related to the urinary excretion of early kidney injury biomarkers, supporting the hypothesis of the nephrotoxic role of F exposure."

<https://www.sciencedirect.com/science/article/pii/S0041008X18302382>

- Monica I. Jiménez-Córdova, Mariana Cardenas-Gonzalez, Guadalupe Aguilar-Madrid, Luz C. Sanchez-Peña, Ángel Barrera-Hernández, Iván A. Domínguez-Guerrero, Carmen González-Horta, Olivier C. Barbier, Luz M. Del Razo. Evaluation of kidney injury biomarkers in an adult Mexican population environmentally exposed to fluoride and low arsenic levels. *Toxicology and Applied Pharmacology*. May 2018.

Brain-derived neurotrophic factor (BDNF): Chinese animal study verifies mechanisms using in vivo and in vitro methodology for cognitive deficits that "suggest that the developmental neurotoxicity of fluoride is associated with the impairment of synaptogenesis, which is caused by ERK1/2-mediated BDNF-TrkB signaling disruption." BDNF is involved with learning, memory, aging and psychiatric disease <https://pubmed.ncbi.nlm.nih.gov/30130557/>

- Chen J, et al. ERK1/2-mediated disruption of BDNF-TrkB signaling causes synaptic impairment contributing to fluoride-induced developmental neurotoxicity. *Toxicology*. 2018 Dec 1;410:222-230.

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KIDNEY CASCADE: “Taken together, these findings indicate that there can be some alterations in liver enzyme activities at early stages of fluoride intoxication followed by renal damage.” <https://pubmed.ncbi.nlm.nih.gov/29769014/>

- Perera T. et al. Effect of fluoride on major organs with the different time of exposure in rats. *Environmental Health and Preventive Medicine* (2018) 23:17

2017

REVIEW: Concludes that fluoridation schemes whether from water, food or salt programs “pose risks of various diseases in the asthmatic-skeletal, neurological, endocrine and skin systems. Dental and skeletal fluorosis are signs of chronic and excessive ingestion of fluoride.”

<https://www.NCBI.nlm.nih.gov/pubmed/28453591>

- Verena Romero, Frances J. Norris, Juvenal A. Ríos, Isel Cortés, Andrea González, Leonardo Gaete, Andrei N. Tchernitchin. The impact of tap water fluoridation on human health. *Rev. méd. Chile* vol.145 no.2 Santiago Feb. 2017.

DOSE-RESPONSE: Validated that IQs of children are lowered on a dose-response trend line correlated with the amount of fluoride exposure as measured via urine tests of their mothers during pregnancy and individualized IQ tests of offspring. In the range consistent with doses in optimally fluoridated communities, there was up to a 6 point difference in IQ. This NIH sponsored 12 year longitudinal study conducted by researchers at world class American & Canadian universities excluded diabetics as well as those with kidney disease or pregnancy complications and allowed for many confounders.

<https://pubmed.ncbi.nlm.nih.gov/28937959/>

- Morteza Bashash, Deena Thomas, Howard Hu, et al. Prenatal Fluoride Exposure and Cognitive Outcomes in Children at 4 and 6–12 Years of Age in Mexico. *Environ Health Perspect.* Sept 2017. Vol 125, Issue 9.

GENES & BONES: “This study provides evidence that chronic oxidative and inflammatory stress may be associated with the fluoride-induced impediment in osteoblast differentiation and bone development.” <http://link.springer.com/article/10.1007/s12011-016-0756-6>

- Gandhi, D., Naoghare, P.K., Bafana, A. et al. Fluoride-Induced Oxidative and Inflammatory Stress in Osteosarcoma Cells: Does It Affect Bone Development Pathway? *Biol Trace Elem Res* (2017) 175: 103.

PRESCHOOL DIET: Diet of two year olds contain unsafe levels of fluoride.

<http://onlinelibrary.wiley.com/doi/10.1111/cdoe.12283/full>

- Martinez-Mier EA, Spencer KL, Sanders BJ, Jones JE, Soto-Rojas AE, Tomlin AM, Vinson LA, Weddell JA, and Eckert GJ. Fluoride in the diet of 2-years-old children. *Community Dent Oral Epidemiol.* 2017;00:1–7.

APOPTOSIS: “Enamel fluorosis is a developmental disturbance caused by intake of supraoptimal levels of fluoride during early childhood. The enamel defects consist of horizontal thin white lines, opacities (subsurface porosities), discolorations, and pits of various sizes. The molecular mechanism underlying enamel fluorosis is still unknown..... We can hypothesize that fluorosis is due to a combination of direct cytotoxic effects causing cell death, the delayed development of tight junctions, which are necessary to form a sealed barrier between apical and basolateral surfaces, and a direct inhibitory effect of fluoride on vectorial calcium and/or bicarbonate transport.” <https://www.NCBI.nlm.nih.gov/pmc/articles/PMC5770627/>

- Rácz, Róbert et al. “No Change in Bicarbonate Transport but Tight-Junction Formation Is Delayed by Fluoride in a Novel Ameloblast Model.” *Frontiers in Physiology.* 2017; 8: 940.

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FLUORIDE & DEMOGRAPHICS: “About 30% of the children were at the risk of dental fluorosis.” <https://pubmed.ncbi.nlm.nih.gov/28110134/>

- Jain RB. Concentrations of fluoride in water and plasma for US children and adolescents: Data from NHANES 2013-2014. *Environ Toxicol Pharmacol*. 2017 Mar;50:20-31.

DNA: Finds that “prolonged fluoride intake at chosen concentrations caused imbalance of the cellular oxidative state, affected DNA and disrupted cellular homeostasis... It is recommended that fluoride supplementation requires a fresh consideration in light of the current study.” <https://www.NCBI.nlm.nih.gov/pubmed/28089781>

- F.D. Campos-Pereira, L. Lopes-Aguiar, F.L. Renosto, et al. Genotoxic effect and rat hepatocyte death occurred after oxidative stress induction and antioxidant gene downregulation caused by long term fluoride exposure. *Chem Biol Interact*. 2017 Feb 25;264:25-33.

PRENATAL POISON: “F can pass through the cord blood and breast milk and may have deleterious impact on learning and memory of the mouse pups.”

<http://journals.sagepub.com/doi/abs/10.1177/0960327117693067>

- Y Zhang, X Xue, R Niu, J Wang. Maternal fluoride exposure during gestation and lactation decreased learning and memory ability, and glutamate receptor mRNA expressions of mouse pups. *Z Sun, Human & Experimental Toxicology*. February 13, 2017.

IMMUNITY: Prenatal and early postnatal exposure to fluoride impairs spleen function and development which damages spleen and lifelong immunity.

<https://www.NCBI.nlm.nih.gov/pubmed/28846973/>

- Yanqin Ma, Kankan Zhang, Fengjun Ren, Jundong Wang, Developmental fluoride exposure influenced rat's splenic development and cell cycle via disruption of the ERK signal pathway, In *Chemosphere*, Volume 187, 2017, Pages 173-180

NEUROINFLAMMATION: Toxic effects of fluoride on the central nervous system and immunity.

<https://link.springer.com/article/10.1007/s10753-017-0556-y>

- Chen R, Zhao LD, Liu H. et al. Fluoride Induces Neuroinflammation and Alters Wnt Signaling Pathway in BV2 Microglial Cells. *Inflammation*. 2017;40: 1123.

2016

CRITIQUE HHS RECOMMENDATION: Pro-fluoridation team of dental researchers determined that the Department of Health and Human Services reduction of the optimal fluoride concentration to a single 0.7 ppm target is lacking in sound science, i.e. that “policy need to be cognizant of the balancing of risk and protective exposures across the entire population and potentially all ages and to be based on recent data that are purposefully collected, critically analyzed and carefully interpreted... (the recommendation seems) premature in terms of its rationale and its use and interpretation of sometimes dated data.” These authors’ bias is to maintain 1 ppm; nevertheless, their rationale against the HHS document is appropriate. The HHS document is political, not scientific.

<https://www.NCBI.nlm.nih.gov/pubmed/26710669>

- Spencer AJ, Do LG. Caution needed in altering the 'optimum' fluoride concentration in drinking water. *Community Dent Oral Epidemiol*. 2016 Apr;44(2):101-8.

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OSTEOPOROSIS: “Consequently, although the World Health Organization continues to support F schemes for caries prevention despite a lack of scientific proof, the F schemes are not able to improve the crystal quality but rather contribute adversely to affect tooth development and increases the risk of developing postmenopausal osteoporosis.”

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<https://www.ncbi.nlm.nih.gov/pubmed/27740551>

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2015

COCHRANE CWF REVIEW: Estimates that 12% of the children living in fluoridated communities with 0.7 ppm fluoridation have aesthetically objectionable dental fluorosis with a total dental fluorosis effect of 40%. The effects were 47% & 15% for 1 ppm, only a minor impact on incidence of dental fluorosis and consistent with the findings of the 2000 York Review.

http://www.cochrane.org/CD010856/ORAL_water-fluoridation-to-prevent-tooth-decay

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- S Peckham, D Lowery, S Spencer. Are fluoride levels in drinking water associated with hypothyroidism prevalence in England? A large observational study of GP practice data and fluoride levels in drinking water. *J Epidemiol Community Health*. 24 February 2015.

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<http://www.ehjournal.net/content/pdf/s12940-015-0003-1.pdf>

- A Malin and C Till. Exposure to fluoridated water and attention deficit hyperactivity disorder prevalence. *Environmental Health* 2015, 14:17

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CWF INFLAMMATIONS: Found that “even in small concentrations fluoride changes the amounts and activity of COX-1 and COX-2 enzymes taking part in the initiating and development of inflammatory process.”

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NEUROTOXICANT: EPA scientists classify fluoride as a ‘gold standard’ developmental neurotoxicant with substantial evidence of harm.

<http://www.sciencedirect.com/science/article/pii/S0892036215300362>

- William R. Mundy, Stephanie Padilla, Joseph M. Breier, et al. Expanding the test set: Chemicals with potential to disrupt mammalian brain development. *Neurotoxicology and Teratology*. Volume 52, Part A, November–December 2015, Pages 25–35.

SUGAR: “Sugars as a cause of caries is underemphasized and not prominent in preventive strategies... researchers mistakenly consider caries to be a multifactorial disease; they also concentrate mainly on mitigating factors, particularly fluoride... “ Authors argue that modifying factors such as fluoride would not be needed if they tackled the one cause of cavities, sugar.

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PROPAGANDIZING: The proponents of fluoridation ignored concerning evidence and did not deliver on their promise of dental benefit then, and now. Neither did they do the expected due diligence re harms. <https://doi.org/10.2105/AJPH.2015.302660>

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GENETICS: Although everyone is vulnerable, some people are more susceptible to adverse neurological and dental effects due to genetic makeup. “In conclusion, fluoride exposure was adversely associated with children's intelligence, whereas the COMT polymorphism may increase the susceptibility to the deficits in IQ due to fluoride exposure. Moreover, the proteomic analysis can provide certain basis for identifying the early biological markers of fluorosis among children.” <https://pubmed.ncbi.nlm.nih.gov/25556215/>

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- Lee Ko & Kathleen M. Thiessen (2015) A critique of recent economic evaluations of community water fluoridation, *International Journal of Occupational and Environmental Health*, 21:2, 91-120

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Additional items of note:

2017 IAOMT Position Paper: <https://iaomt.org/iaomt-fluoride-position-paper-2/>

2018 Open Letter: <http://www.multibriefs.com/briefs/icim/nutrition.pdf>

2019 Children's Health Defense Statement: <https://childrenshealthdefense.org/news/u-s-water-fluoridation-a-forced-experiment-that-needs-to-end/>

2020 Expert Opinion: <https://www.ehn.org/fluoride-and-childrens-health-2648120286.html>

2023: NTP Report suppressed by HHS political machinations: <https://ntp.niehs.nih.gov/whatwestudy/assessments/noncancer/ongoing/fluoride/index.html>

"...fluoride is presumed to be a cognitive neurodevelopmental hazard to humans..."
- Draft Monograph from National Toxicology Program, "[Systematic Review of Fluoride Exposure and Neurodevelopmental and Cognitive Health Effects](#)" (Sept 6, 2019)

"The cessation of all compulsory water fluoridation schemes should be the goal of all public health agencies, ethical lawmakers, and informed citizens."
- Prof. Rita F. Barnett-Rose, J.D. in "[Compulsory Water Fluoridation](#)" (2014)

DEFINITIONS:

- **Endorsement:** An endorsement is an authoritative statement reflecting a point of view for the purpose of exerting influence. An endorsement is **not** an expert opinion.
 - **Authoritative statement:** An opinion that interprets a rule, law or policy for the purpose of guiding, influencing, or mandating action. Authoritative statements are not inherently trustworthy or reliable, but they are inherently manipulative. "Testimonial propaganda" utilizes authoritative statements in marketing and in politics. The slogan "question authority" was intended to encourage critical thinking in order to combat the blind acceptance of biased authoritative statements that endorse policy and/or sanctioned narratives. (*Logical Fallacies: Appeal to Authority*)
- **Expert Opinion:** An expert opinion is dependent on evidence and the due diligence of someone with substantial study in a field. The Daubert Standard is a legal process that validates the trustworthiness of experts offering opinion in a court of law.

EXAMPLES:

ENDORSEMENT: The April 2015 HHS statement recommending 0.7 ppm fluoride concentration in drinking water for 'safe & effective' prevention of tooth decay promoted the long standing fluoridation policy of the agency.

vs.

EXPERT OPINION: The June 2015 Cochrane report finds no reliable evidence of dental benefit to adults or low income children, but documents substantially higher rates of dental fluorosis, some of which will likely result in costly cosmetic dentistry. The 2019 National Toxicology Program systematic review offered an expert opinion based on the evidence that fluoride is a presumed hazard to human health specific to neurotoxic impact when exposure is pre- or post-natal.

Dear NDWAC Membership:

Please include an agenda item about water fluoridation and the neurodevelopmental toxicity of fluoride exposure among pregnant mothers according to the current science, which unfortunately and unethically is being suppressed by CDC, HHS and EPA leadership. President Biden's initiatives demand recognition and resolution of the environmental injustice of fluoridation policy to susceptible population groups.

Please advise EPA leadership to stop applying taxpayer dollars via the Department of Justice to defend in Federal District Court a policy and program that is dumbing down this great country's future brain trust.

Thank you.

John Mueller

To whom it may concern,

I would like to request to present an oral statement at the upcoming August 8, 2023 NDWAC meeting regarding PFAS.

I would also like to submit the following written statement for distribution to members of the Council:

Thank you for the opportunity to address the proposed National Primary Drinking Water Regulation for per- and polyfluoroalkyl substances (PFAS). My comments are focused on occurrence of PFAS and key questions to inform a meaningful regulation.

1. Occurrence data analysis was an overly complicated, statistical analysis that lacks transparency for the public. The lack of transparency extends beyond understanding the inner workings of the Bayesian Model to include a lack of a clear output of water systems that will be impacted. The public can't appreciate or understand who is impacted from estimated percentages of water system categories.
2. The EPA has real world results from actual water systems' UCMR 5 data sitting at its door, which will represent a substantial amount of data that will offer a high level of confidence on PFAS occurrence. (~3,500 systems across size categories/sources and MRLs in low single digits ppt concentrations). These data will let the public appreciate and understand who is impacted by PFAS and how much.
3. Specific notes about occurrence of PFAS compounds:
 - a. Available data for GenX is severely lacking and is not sufficient to characterize water occurrence regionally, let alone nationally.
 - b. Available data for PFBS demonstrates a complete lack of occurrence at levels of concern. UCMR 3 showed virtually no occurrence at levels above 4.5% of the HAL and subsequent state data confirms that lack of occurrence.
 - c. Available data for PFNA shows very low occurrence.
4. The EPA's proposal lacks sensitivity analyses to test the Bayesian model to understand how uncertainties may skew the overall EPA analyses.

Thank you for the opportunity to submit this written statement and present at the upcoming meeting.

Regards,

Chad

Chad Seidel, Ph.D., P.E.
President
Corona Environmental Consulting, LLC

Submission to the National Drinking Water Advisory Council: Comments on the National Primary Drinking Water Regulation for PFAS

August 1, 2023

Submitted by: Linda Dell, Harvey Clewell, Robinan Gentry (Ramboll US Consulting, Inc.)

In 2009, the United States Environmental Protection Agency (USEPA) established provisional health advisories (HAs) of 400 and 200 parts per trillion for PFOA and PFOS, respectively, in drinking water (USEPA 2009). In 2016, the USEPA revised and reduced the HAs to 70 ppt each for PFOA and PFOS, or for the combined concentration of PFOA and PFOS (USEPA, 2016a, 2016b). Last summer, the USEPA established an interim health advisory for PFOA of 0.004 ppt and an interim health advisory for PFOS of 0.02 ppt (USEPA 2022a, 2022b). That is a 17,500-fold reduction in the PFOA health advisory level from the previous one. The magnitude of the reduction implies that there are large and immediate risks to population health at concentrations of 70 ppt PFOA in drinking water.

The USEPA (2023a, 2023b) recognizes that current analytical methods cannot measure PFOA or PFOS in drinking water concentrations at extremely low concentrations that approach 0 ppt. Earlier this year, the USEPA proposed setting an enforceable standard, or a maximum contaminant level (MCL), at 4 parts per trillion each for PFOA and PFOS in drinking water (USEPA 2023a, 2023b). The USEPA also proposed using a hazard index (HI) of less than 1.0 (unitless) to regulate the combined concentrations of four additional PFAS chemicals in drinking water: PFBS, PFNA, PFHxS, and GenX (USEPA 2023c).

Our comments here focus on the USEPA's use of the available science related to PFAS exposure and selected health endpoints to quantify the health benefits of the proposed National Primary Drinking Water Regulation for PFAS. The USEPA quantified the health risk reduction associated with two critical endpoints that have been associated with PFOA or PFOS in epidemiological studies: decreases in birth weight and increases in total cholesterol. In addition, we also comment on issues with the proposed approach for regulating the four additional PFAS using a hazard index (HI) to evaluate whether the mixture of four PFAS poses a potential health risk.

The proposed MCLs for PFOA and PFOS are based on epidemiological data that have shown associations between PFOA and PFOS blood concentrations and differences in biomarkers of effect (specifically, increases in total cholesterol, decreases in antibody response, increases in certain liver enzymes, or small decreases in birth weight) (USEPA 2023a, 2023b). The USEPA's economic analysis suggests that the proposed MCLs will result in improved health benefits by reducing the number of adverse health effects associated with PFAS exposure (USEPA 2023d). The quantified expected benefits from implementing the proposed MCL standard include higher average birth weights, leading to fewer infant deaths and reduced medical expenditures due to fewer interventions that are associated with low birth weight infants. The quantified expected benefits also include fewer heart attacks, strokes or other cardiovascular-related morbidity and mortality due to lower average blood cholesterol levels in adults, and therefore fewer adults with high cholesterol in the general population. The USEPA estimated that full implementation of the standard will result in tens of thousands of avoided illnesses and deaths that would have been attributed to PFAS exposure (USEPA 2023d).

These quantified benefits of avoided mortality and illness are likely overstated. The available population health data suggest that further reductions of average PFOA and PFOS concentrations in blood serum are unlikely to result in measurable increases in average birth weight.

The Reference Doses for PFOA and PFOS, used in the derivation of the MCLs, are based on small decreases in birth weight (Wikström et al. 2021). Some studies have reported that the associations between PFOA and PFOS and decreases in birth weight are likely confounded by maternal hemodynamics, specifically plasma volume expansion and kidney filtration rate. Epidemiological studies reported larger decreases in birth weight when maternal PFAS was measured during the third trimester. When maternal PFAS was measured during the first trimester, there was no association between PFAS and decreased birthweight or the association was attenuated (Steenland et al. 2018, Dzierlenga et al. 2020). There is substantial uncertainty as to whether the associations between PFOA and PFOS and decreases in birth weight are causal.

Another of the critical studies that the USEPA (2023a) relied upon for deriving the Reference Dose for PFOA was an epidemiological study of a birth cohort born in 1997-2000 in the Faroe Islands (Grandjean et al. 2012). The study found that children at age 7 had reduced antibodies to diphtheria and tetanus boosters that were administered at age 5 and in relation to PFOA and PFOS concentrations measured at age 5 years. The biological significance of small decreases (which were imprecisely measured) in antibody response is unknown and a mode of action for this effect has not been postulated. Moreover, the population data from the Faroe Islands, does not support decreased birth weight in relation to PFAS levels reported in the birth cohort studies. Infants born in the Faroe Islands have larger mean birth weights (possibly the largest globally). When compared to other Nordic countries, this phenomenon has been measured since approximately the mid-1970s (Olsen and Joensen 1985). Average birth weight has continued to increase in more recent years in the Faroe Islands and the population has been described as having birth weight that is closer to optimal than other populations (Olsen and Joensen 1985; Olsen et al. 2023).

Another critical effect on which the Reference Doses for PFOA and PFOS are based is increases in total cholesterol in the general population (Dong et al. 2019). The mode of action for increases in total cholesterol in relation to PFOA and PFOS blood concentrations seen in the general population is not understood. There is uncertainty as to whether the association between PFOA and PFOS and increased total cholesterol reported in epidemiological studies is a true causal association or whether it is confounded by physiological determinants (specifically, enterohepatic cycling of bile acids) that influence both PFAS concentrations in the blood and cholesterol levels (EFSA 2020, Andersen et al. 2021).

Similarly, a review of population health data indicates that consistent decreases in heart disease incidence and mortality rates have been reported since 1990 (and earlier). These data suggest improvements in cardiovascular disease risk factors and interventions have been largely successful. It seems unlikely that mean PFOA or PFOS serum concentrations at levels seen in the general public in recent years contribute to increased measurable risks of cardiovascular disease. Mean total cholesterol concentrations have fallen since the 1960s (Carroll et al. 2005). In 2017-2018, the age-adjusted prevalence of high total cholesterol (≥ 240 mg/dL) in US adults aged 20 and over was 10.5%, which had declined from 18.5% in 1999-2000

(Carroll and Fryar 2020). Therefore, it seems unlikely that the benefits of decreased CVD events are detectable with further declines in average blood serum concentrations of PFAS.

Overall, it seems likely that the quantified (and non-quantified) health risk reduction benefits, estimated assuming compliance with the proposed NPDWR, are overstated.

The USEPA has also proposed using a hazard index to regulate a mixture of four PFAS (PFNA, PFHxS, PFBS, GenX). Previously, an HI has commonly been used as a screening tool to make decisions about chemical remediation; it has never been used in a federal regulation as a standard to protect human health. In addition, the USEPA did not follow the recommendation of the USEPA Scientific Advisory Board (SAB) regarding the hazard index. The USEPA SAB (2022c) recommended that the USEPA use the hazard index approach for a common outcome. Instead, the HI for the four newly regulated PFAS are calculated using critical effects that are based on different endpoints and target tissues: delayed development in mouse offspring for PFNA; thyroid follicular epithelial hypertrophy/hyperplasia in parental male rats for PFHxS; decreased thyroxine in mouse offspring for PFBS; and liver toxicity in female rat dams for GenX (USEPA 2023c).

The USEPA defends the HI approach by making a broad claim that the diverse effects are all associated with a common mechanism involving disruption of cellular signaling. This is essentially an argument for a common mode of action. Yet, the USEPA (2023a, 2023b) has also determined that there are inadequate data supporting any mode of action, except for liver effects in rats which are dependent on peroxisome proliferator activated receptor alpha (PPAR α) activation in rodents administered PFOA or PFOS at doses that are a thousand fold higher (or more) than the exposure seen in the general population. Thus, this mode of action is not relevant to humans at blood concentrations seen in the general population. In a separate toxicity assessment of GenX chemicals, the USEPA (2021) also concluded that the liver effects of GenX (which are independent of PPAR α activation) are not consistent with a common mode of action for all PFAS.

The use of an HI based on different target organs or endpoints for estimation of a regulatory value has no support in existing agency guidelines or those of other national and international authoritative bodies. The agency should delay promulgating a HI-based assessment until they have developed the necessary Target Tissue Doses (TTDs). The TTDs can readily be derived using the existing ATSDR methodology (ATSDR 2018).

Finally, a commonly reported statistic is that certain PFAS (in particular, PFOA and PFOS) have been detected in the blood of more than 99% of the U.S. population at concentrations in the low parts per billion. Over a period of 20 years, PFOA serum concentrations in the general population fell from a median concentration of 5.2 ng/mL in 1999-2000 to 1.47 ng/mL in 2017-2018. Approximately 95% of the population now has PFOA blood concentrations below 3.77 ng/ml (CDC, 2023). During the same time, PFOS serum concentrations in the general population declined from a median concentration of 30.2 ng/mL to 4.3 ng/mL. As of 2018 (the year with the most recent data), 95% of the population have PFOS concentrations below 14.6 ng/ml, which is approximately one half of what the median PFOS concentration was in 1999. These concentrations have declined largely because PFAS manufacturers agreed to reduce emissions and/or phase out production of PFOA and PFOS and other long-chain PFAS,

and not as a result of decreasing acceptable levels of PFAS. It is unclear whether there are any incremental health benefits to be attained with the MCLs proposed under the NPDWR for PFAS; however, evidence of large and immediate risks to population health under the 2016 USEPA HAs of 70 ppt is lacking.

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August 1, 2023

Ms. Lisa D. Daniels
Chair
National Drinking Water Advisory Council
Environmental Protection Agency
1200 Pennsylvania Ave NW
Mail Code 4601
Washington, DC 20460

SUBMITTED ELECTRONICALLY

RE: AWWA Comments on Proposed PFAS Rulemaking for National Drinking Water Advisory
Council Consideration

Dear Ms. Daniels,

The American Water Works Association (AWWA) appreciates the public service provided by all of the National Drinking Water Advisory Council (NDWAC) members. The proposal that the NDWAC is reviewing is expected to have a significant impact on public health and the operation of water systems across thousands of communities in the United States. AWWA has been engaged on per- and polyfluoroalkyl substances (PFAS) issues for more than 2 decades since its addition to the Contaminant Candidate List, including supporting EPA's effort to develop national primary drinking water standards for the two most well-studied PFAS, perfluorooctanoic acid (PFOA), perfluorooctanesulfonic acid (PFOS). AWWA appreciates the opportunity to engage with the NDWAC about this proposal and issues that were identified in review of the Environmental Protection Agency's (EPA) proposal.

Following its publication of the proposed standards for PFOA, PFOS, and four other PFAS using a hazard index approach, AWWA provided an in-depth review and comment on the rulemaking for EPA's consideration. These comments included recommendations regarding the preliminary determinations for four PFAS (individually and as a group), the underlying analyses, monitoring requirements, and overarching concerns about the feasibility for the rule to be implemented as proposed. These detailed comments are attached for your convenience, but a few key issues are summarized below.

First, the rule's proposal is estimated to require from 4,500 to 7,500 water systems to install drinking water treatment for PFAS. Based on recent estimates, these communities will need to invest more than \$3.2 billion annually to meet the requirements of the rule. This estimate is based on work by Black & Veatch, a national engineering firm with extensive drinking water and PFAS treatment expertise nationally. To validate this work, AWWA collected case study data from more than a hundred utilities that have already developed cost estimates for PFAS treatment. The estimates by Black & Veatch and the case studies were closely aligned approximately 3 to 4 times higher than the EPA's. As proposed, EPA's

analysis significantly underestimates these costs for communities and the significant financial impacts to households.

Additionally, the EPA's proposed use of the hazard index for four PFAS using different health outcomes for each compound does not rely on sound science. This approach was not endorsed by the Science Advisory Board and is contrary to both EPA and ATSDR guidance materials which recommend using the hazard index when the health outcomes are the same. It is also not clear that EPA has the authority to issue a preliminary determination and a proposed drinking water standard simultaneously.

Finally, a significant concern about this rule is the feasibility for water systems to implement this rule, not only given the financial constraints but also given the compliance timeline. As thousands of systems work to implement the rule's requirements the demands for workforce, laboratory support, and construction materials will increase, straining an already challenging supply chain. At the moment, water systems are already facing long lead times for equipment, scheduling issues with construction crews, and a forthcoming 'grey wave' as many water operators and experts are preparing to retire. These challenges will also extend to the laboratory capacity for initial monitoring requirements, which will need to go from processing 20,000 samples annually under the Fifth Unregulated Contaminant Monitoring Rule (UCMR 5) to processing more than 220,000 water samples in the first year following the rule. Water utilities have already seen increased lead times for sample results, quality control challenges, and increased analytical costs for UCMR 5 and these impacts will be more severe with the proposed rule.

Another challenge for water utility implementation of the rule within the three, or up to five, year timeline is the need for appropriate assessment of PFAS treatment. Pilot testing will not only be needed to determine the most effective PFAS treatment technology but also to ensure that unintended consequences do not emerge causing simultaneous compliance issues with other rules, like the lead and copper rule.

AWWA encourages the NDWAC to consider these comments and the concerns highlighted here as they consider recommendations for the EPA's work towards finalizing the rule. AWWA supports EPA moving forward with drinking water standards when they are based on sound science and will ensure meaningful protection of public health. That begins, however, with analyses that transparently and accurately reflect the benefits, costs, and overall feasibility of the rulemaking.

If you have any questions or concerns regarding this correspondence or if AWWA can be of assistance in some other way, please contact me at (202) 326-6127 or cmoody@awwa.org.

Best Regards,

Chris Moody
Regulatory Technical Manager
American Water Works Association

CC: Elizabeth Corr

Attachment: AWWA Comments on the Proposed NPDWRs for PFAS



American Water Works Association

Dedicated to the World's Most Important Resource™

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May 30, 2023

Michael Regan
Administrator
Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Mail Code: 1309
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SUBMITTED ELECTRONICALLY

RE: Per- and polyfluoroalkyl substances (PFAS): Perfluorooctanoic acid (PFOA) and Perfluorooctanesulfonic acid (PFOS) National Primary Drinking Water Regulation Rulemaking, Docket No. [EPA-HQ-OW-2022-0114](#)

Dear Administrator Regan,

The American Water Works Association appreciates the U.S. Environmental Protection Agency's (EPA) efforts to propose national primary drinking water regulations for per- and polyfluoroalkyl substances (PFAS), including perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS). The proposal includes several major actions for PFAS in drinking water, including:

- Proposal for drinking water standards for PFOA and PFOS, individually,
- Preliminary determinations for perfluorohexanesulfonic acid (PFHxS), perfluorononanoic acid (PFNA), hexafluoropropylene oxide dimer acid (HFPO-DA), perfluorobutanesulfonic acid (PFBS), and the mixture of these four PFAS, and
- Proposal for drinking water standard for PFHxS, PFNA, HFPO-DA, and PFBS as a mixture using a hazard index.

AWWA supports the development of primary drinking water standards for PFOA and PFOS and supports the agency's interest in proposing regulatory determinations for additional PFAS. AWWA recommended the development of standards for PFOA and PFOS in comments to the EPA in 2021 and provided a shortlist of PFAS compounds for the agency's consideration for additional action as appropriate. In these comments, AWWA provided additional recommendations relating to the use of occurrence data, an approach to monitoring requirements, and available cost data for drinking water treatment facilities.

AWWA believes that EPA has put forward a rule framework that begins to address a number of stakeholder concerns. The proposal serves as a good starting point for finalizing a rule that will address PFAS compounds in drinking water. Attached are detailed comments on the proposed rule. In addition to providing feedback for improving the analyses to support the proposal, AWWA makes several key recommendations, which are further detailed in the comment letter.

These recommendations include:

1. The agency should consider withdrawing and re-proposing drinking water standards for PFOA and PFOS given the recurring issues with the underlying analyses. If the agency should finalize drinking water standards for PFOA and PFOS based on the current proposal, drinking water standards of 10 ppt, each, are most appropriate.
2. The agency's preliminary determinations for PFNA, HFPO-DA, and PFBS and the mixture of PFHxS, PFNA, HFPO-DA, and PFBS are not sufficiently supported by the available data. Moreover the available data currently suggests a negative determination for these PFAS is appropriate. The agency should re-issue these preliminary determinations following the availability of national monitoring data currently being collected, as part of the Fifth Unregulated Contaminant Monitoring Rule (UCMR 5).
3. The agency misuses the hazard index as a maximum contaminant level given it is not supported by federal guidance for assessing risk from mixtures and other issues.
4. The agency's proposed regulation concurrent with preliminary regulatory determinations is not within the scope of authorities granted by the Safe Drinking Water Act (SDWA), does not fulfill the obligations under the Administrative Procedures Act, and is inappropriate. Proposed regulation of additional PFAS should not occur until a determination to regulate is issued.

We hope that these comments will help EPA finalize the rule by effectively leveraging science and the authorities of the Safe Drinking Water Act. If you have any questions regarding this correspondence, please contact me or Chris Moody at 202.326.6127 or cmoody@awwa.org.

Best Regards,

ON BEHALF OF THE AMERICAN WATER WORKS ASSOCIATION



G. Tracy Mehan, III

Executive Director for Government Affairs

Attachment (1)

cc: Ryan Albert, EPA / OW
Radhika Fox, EPA / OW

Eric Burneson, EPA / OW
Jennifer McLain, EPA / OW

Who is AWWA

The American Water Works Association is an international, nonprofit, scientific and educational society dedicated to providing total water solutions assuring the effective management of water. Founded in 1881, the Association is the largest organization of water supply professionals in the world. Our membership includes more than 4,500 utilities that supply roughly 80 percent of the nation's drinking water and treat almost half of the nation's wastewater. Our 50,000-plus total membership represents the full spectrum of the water community: public water and wastewater systems, environmental advocates, scientists, academicians, and others who hold a genuine interest in water, our most important resource. AWWA unites the diverse water community to advance public health, safety, the economy, and the environment.

**AWWA Comments on the Proposed “PFAS National Primary Drinking Water
Regulation Rulemaking”**

[Docket ID No: EPA-HQ-OW-2022-0114](#)

*Prepared by the:
American Water Works Association*

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List of Acronyms

APA	Administrative Procedures Act
ATSDR	Agency for Toxic Substances and Disease Registry
AWWA	American Water Works Association
BIL	Bipartisan Infrastructure Law
CCT	Corrosion Control Treatment
CVD	Cardiovascular Disease
CWA	Clean Water Act
DBP	Disinfection Byproduct
DWSRF	Drinking Water State Revolving Fund
ELG	Effluent Limitation Guideline and Standard
EPA	Environmental Protection Agency
GAC	Granular Activated Carbon
HAL	Health Advisory Level
HBWC	Health Based Water Concentration
HFPO-DA	Hexafluoropropylene Oxide Dimer Acid
HRRCA	Health Risk Reduction and Cost Analysis
IRIS	Integrated Risk Information System
IX	Ion Exchange
LCRR	Lead and Copper Rule Revisions
MCL	Maximum Contaminant Level
MCLG	Maximum Contaminant Level Goal
NDAA 2020	National Defense Authorization Act for Fiscal year 2020
NHANES	National Health and Nutrition Examination Survey
NPDWR	National Primary Drinking Water Regulation
PFAS	Per- and Polyfluoroalkyl Substance
PFBS	Perfluorobutanesulfonic Acid
PFHxS	Perfluorohexanesulfonic Acid
PFNA	Perfluorononanoic Acid
PFOA	Perfluorooctanoic Acid
PFOS	Perfluorooctanesulfonic Acid
PQL	Practical Quantification Level
RAA	Running Annual Average
RCRA	Resource Conservation and Recovery Act
RO	Reverse Osmosis
SAB	Science Advisory Board
SDWA	Safe Drinking Water Act
SMF	Standard Monitoring Framework
TOC	Total Organic Carbon
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
UCMR 3	Third Unregulated Contaminant Monitoring Rule
UCMR 5	Fifth Unregulated Contaminant Monitoring Rule
WBS	Work Breakdown Structure
WIFIA	Water Infrastructure Finance and Innovation Act

AWWA Comments on the

Proposed “PFAS National Primary Drinking Water Regulation Rulemaking”

The American Water Works Association (AWWA) appreciates the opportunity to comment on the Environmental Protection Agency’s (EPA or agency) “Proposed Per- and polyfluoroalkyl substances (PFAS) National Primary Drinking Water Regulation Rulemaking” (the proposal). AWWA has prepared the following comments to assist EPA in moving forward with a final rule to protect public health that is scientifically grounded, legally defensible, and crafted in a manner that embraces the objectives of the Safe Drinking Water Act (SDWA).

1. Overarching Comments

AWWA appreciates EPA’s interest in addressing PFAS in drinking water to protect public health and maintain public trust in the nation’s drinking water supply. AWWA has been engaged on PFAS issues since the early 2000s and has leveraged technical expertise from our members, which include more than 50,000 professional members and 4,500 utilities, to support the agency’s broad efforts to address PFAS contamination. When the following guiding principles are followed, AWWA supports PFAS regulations:

1. Commitment to public health protection,
2. Fidelity to scientific process,
3. Setting regulatory requirements that are feasible to implement,
4. Ensuring affordability of safe drinking water, and
5. Effectively leveraging source water protection efforts.

In establishing drinking water regulations, embracing these guiding principles will ensure that communities and the public are effectively protected through a transparent rulemaking process and with a rule that prioritizes opportunities to reduce public health risks. The following comments and recommendations reflect these guiding principles.

Reinforcing the Polluter Pays Principle

EPA first published plans for a broad regulatory agenda to address PFAS as part of the EPA’s PFAS Action Plan in 2019 (EPA, 2019a). In 2021, the agency published the more detailed PFAS Strategic Roadmap for regulatory actions (EPA, 2021a). The EPA’s PFAS Action Plan and the PFAS Strategic Roadmap highlighted a variety of regulatory actions that the agency is pursuing, including setting effluent limitation guidelines and standards (ELGs) for industrial dischargers under the Clean Water Act (CWA). The implementation of these regulatory actions is critical in protecting the environment and the protection of drinking water sources. The actions, when completed, will reinforce the polluter pays principle for PFAS and help maintain the responsibility for the mitigation of PFAS contamination on the polluters instead of communities. While NPDWRs will require community investment to address PFAS, actions under the CWA, Resource Conservation and Recovery Act (RCRA), and the Toxic Substances Control Act (TSCA) will require manufacturers and users of PFAS to carry this burden.

To date, however, the agency’s actions on polluters have consistently lagged behind drinking water action. EPA originally identified PFAS as a potential priority for drinking water as part of the Contaminant Candidate List 3 in 2009 (EPA, 2009). In 2012 EPA advanced the Third Unregulated Contaminant Monitoring Rule (UCMR 3) that required water systems to monitor for six PFAS in finished drinking water

(EPA, 2012). With the proposal, EPA is proposing to set standards for PFAS in drinking water. At the same time, EPA has yet to advance regulations that require manufacturers and users to: (i) report about uses and releases of PFAS, (ii) control the release of PFAS to the environment, (iii) manage PFAS-containing wastes appropriately, and (iv) limit the use and manufacturing of PFAS (EPA, 2022a; EPA, 2022b; EPA, 2022c).

What is further concerning, is the lack of urgency in advancing these actions by the agency. The TSCA data reporting rule, which will require manufacturers and users to report on the production, use, and release of PFAS, was prompted by Congress as part of the National Defense Authorization Act for Fiscal Year 2020 (NDAA 2020) in December 2019 and proposed in June 2021 (Congress, 2019; EPA, 2022a). The rule has yet to be finalized, despite a statutory deadline of January 2023. Additionally, EPA initiated an effort under the CWA to consider ELGs for PFAS as part of the Preliminary Effluent Guidelines Program Plan 14 (EPA, 2019b). With this plan, EPA committed to performing a study of PFAS in industrial effluents for several industries. Program Plan 15 moved forward with a commitment to initiate two rulemakings for both manufacturers and metal finishers and to initiate additional studies on landfills and textile mills (EPA, 2021b). Neither of these rulemakings have been proposed. These actions, if advanced with the same sense of urgency as drinking water actions, would have provided invaluable information and protection for PFAS releases to the environment and the drinking water sources.

The responsibility of the Administrator is to ensure that regulatory actions are implemented in a cohesive manner for the effective protection of the environment and the public. It is imperative that the Administrator begin to advance these actions more meaningfully to minimize the role that communities play in addressing PFAS contamination that they were not responsible for causing. Advancing these actions in a more meaningful, cohesive manner has the potential to curb costly burdens on water system rate payers.

Ensuring Community Resources are Invested in High Priorities

In crafting NPDWRs, it is imperative that the abovementioned guiding principles be followed by the agency to ensure that community resources are invested in high priority risks. In crafting SDWA, Congress recognized the importance of addressing contaminants of greatest concern as part of the development of NPDWRs. EPA must consider the impact this final rule will have on communities as they are managing multiple priorities for community investments to protect public health. Important examples include the replacement of lead service lines, enhancing cybersecurity protections, continuing to improve risk reductions related to disinfection byproducts (DBPs), and the continuous efforts to replace and maintain aging infrastructure to avoid the risk of water main breaks and other threats to public health. The benefits and costs of new standards must be carefully, and accurately, weighed to ensure the investments needed to meet new regulatory requirements do not inappropriately lead to re-allocating available funds away from public health concerns of higher priority, causing unintended consequences.

A recent analysis by Black & Veatch estimated that the costs of the proposed standards could exceed \$2.5 to \$3.2 billion annually (Black & Veatch, 2023 – See Appendix B). The Administrator will need to determine if these costs are justified by the benefits, estimated to be \$0.8 to \$1.2 billion annually, and whether it is a meaningful opportunity to protect public health when this investment will divert water systems investments from other needs to assuring compliance with any final PFAS rule requirements.

Summary of Key Recommendations

AWWA reviewed all aspects of the proposal and the supporting documentation, including the agency's occurrence analysis, cost analysis, benefits analysis, and household affordability analysis. The proposal includes several major actions for PFAS in drinking water, including:

1. Proposal for drinking water standards for both PFOA and PFOS;
2. Preliminary determinations for perfluorohexanesulfonic acid (PFHxS), perfluorononanoic acid (PFNA), hexafluoropropylene oxide dimer acid (HFPO-DA), perfluorobutanesulfonic acid (PFBS), and the mixture of these four PFAS, and
3. Proposal for drinking water standard for PFHxS, PFNA, HFPO-DA, and PFBS as a mixture using a hazard index.

Based on the supporting documentation, and in consultation with drinking water technical experts, AWWA recommends that the agency consider withdrawing and re-proposing the drinking water standards for PFOA and PFOS given that the underlying analyses lack transparency, are not consistent with use of best available science, and are not clear. However, if EPA issues a final rule setting standards for PFOA and PFOS, the agency should set drinking water standards of 10 ppt PFOA and 10 ppt PFOS on the basis that these would be most defensible with the agency's current analysis.

The agency's preliminary determinations for PFNA, HFPO-DA, and PFBS are not sufficiently supported by the supporting documentation. The information included in the proposed rule docket does not suggest that there is a substantial likelihood of PFNA, HFPO-DA, and PFBS occurrence in drinking water with a frequency and at levels of public health concern. Instead, the available evidence indicates that a negative determination is appropriate. Similarly, the information on co-occurrence and overall occurrence of PFHxS, PFNA, HFPO-DA, and PFBS as a mixture also indicates that the agency only has supporting information for a negative determination. Preliminary determinations for these compounds and their mixture should be re-issued following completion of the Fifth Unregulated Contaminant Monitoring Rule (UCMR 5).

Additionally, the agency's proposed drinking water standard for the mixture of PFHxS, PFNA, HFPO-DA, and PFBS poses several issues that must be addressed prior to further action. As discussed above, the underlying data to support a regulatory determination is insufficient and likely suggests that regulation does not represent meaningful opportunity to protect public health. The agency's approach to using a general hazard index with multiple health outcomes lacks support from risk assessment guidance and professionals (ATSDR, 2018; ATSDR, 2022; EPA, 1986; EPA, 2000; SAB, 2022). Finally, the proposed regulation concurrent with preliminary determinations is not within the scope of the EPA's authority under SDWA. While EPA is authorized to issue a proposal concurrent with a determination to regulate, this is a distinctly different action from a preliminary determination.

Additional recommendations are detailed in this letter, including:

1. The proposal's underlying cost analysis is inaccurate and substantially underestimates costs to compliance costs and the financial impacts on consumers. The agency should work with AWWA and other stakeholders to develop a cost-estimating approach that is more reflective of best-engineering practices and should refine the cost estimate for the proposal. Any final rule should

re-evaluate the determinations that benefits justify the costs and the rule is affordable for small systems, considering the updated cost analysis to support any rule.

2. The standard implementation timeline is not appropriate for the current economic conditions. The process to monitor, plan, design, pilot, permit, and construct facilities will take much longer than the standard 3-year timeline given workforce and supply chain challenges. While states may be able to grant 2-year extensions, on a case-by-case basis the burden for state primacy agencies to process these requests will be significant. To avoid a final rule that is infeasible for water systems to comply with, the Administrator should exercise its authority under the SDWA to extend the effective date of compliance by two years for water systems installing capital improvements¹.
3. EPA's statistical approach to estimating occurrence of PFAS in drinking water is overly complicated and EPA would be ill-advised to move forward with a final rule without considering incoming data from more than 3,500 water systems currently collecting samples under the Fifth UCMR 5 (EPA, 2021c).
4. The results of the health risk reduction and cost analysis (HRRCA) are mixed and demonstrate the uncertainty of actual outcomes on communities. Under one analysis (3% discount rate), net benefits are expected for each rule option. Under the other analysis (7% discount rate), though, only one rule option (10 ppt PFOA and 10 ppt PFOS) has net benefits. EPA should recognize the significance of these conflicting results, in conjunction with the concerns regarding the cost analysis, prior to finalizing any rule.
5. The use of the Standard Monitoring Framework (SMF) for PFAS is appropriate where a running annual average (RAA) below one-half of the MCL is considered to be reliably below the MCL. However, the agency's proposed approach to require reporting results below the practical quantification level (PQL) to calculate the RAA for reduced monitoring is inappropriate and will cause equity issues with respect to access to high quality laboratories. This will lead systems with less financial capacity to have more stringent monitoring requirements. EPA should move forward with the SMF, where all results below the PQL are considered 0 ppt.
6. The affordability assessment relies on dated, inaccurate data and an approach that fails to capture affordability challenges for many communities. The affordability analysis should be updated to more accurately reflect household affordability and anticipated challenges for lower-income populations.

As further detailed below, unless the issues outline in this letter are addressed, AWWA is concerned that any final rule would be legally vulnerable for not complying with the SDWA and the Administrative Procedure Act (APA).

¹ Under 42 U.S.C. 300g-1(b)(10), the Administrator "may allow up to 2 additional years to comply with a maximum contaminant level if...additional time is necessary for capital improvements" (104th Congress, 1996).

3. Implementation Challenges

Public water systems subject to the proposal will need to comply within three years unless a two-year extension is provided by state primacy agencies or the Administrator. An estimated 67,000 water systems will need to perform initial monitoring at nearly 90,000 entry points used by water systems. Additionally, upwards of 4,300 water systems will need to take action to address PFAS levels above the maximum contaminant levels (MCLs) and continue to conduct quarterly sampling, according to the EPA's analysis. For water systems that need to install advanced treatment facilities for PFAS, a myriad of challenges will delay the implementation timeline for each system and will impact costs to implement these facilities.

Simply put, the current implementation timeline will cause the final rule to be infeasible, and therefore conflicts with the SDWA. When EPA establishes an MCL, the combination of technology, treatment techniques, or other means required to meet the level must not be more stringent than feasible.² The SDWA defines "feasible" to mean "feasible with the use of the best technology, treatment techniques and other means which . . . are available (taking cost into consideration)."³ The tight timeline for implementation here would render the "the combination of technology, treatment techniques, or other means" infeasible because of the additional costs and implementation considerations.

As required by the SDWA, AWWA encourages EPA to consider how these implementation challenges will impact not only the compliance cost of the rule, but the feasibility of the rule to be implemented on the standard timeline provided by the SDWA. These challenges are further detailed below.

Laboratory Capacity is Lagging Behind Demand

As noted above, more than 67,000 water systems will be driven to comply with the initial monitoring requirements to determine their PFAS levels. Given the timeline of the rule, as described in the proposed rule preamble and reflected in the proposed rule text, water systems that may not leverage previously collected PFAS sampling data, will need to perform initial monitoring during the 12-months immediately following the rule's promulgation. The product of this surge in water monitoring sampling will require laboratories to process more than 220,000 water samples being collected by these systems.

This is in addition to ongoing monitoring activities by the water sector that include compliance monitoring for systems subject to state drinking water standards, performance testing by systems with treatment facilities, and samples to support pilot testing by systems investigating and designing new treatment facilities. Examples of sampling programs for testing new treatment facilities are laid out in detail in AWWA's "Drinking Water Treatment Selection Guide for PFAS" (AWWA, 2020a). This is also in addition to sampling being performed outside of the water sector for environmental investigations and the implementation of recent actions for effluent discharges, which will largely rely on the same laboratories (EPA, 2022d; EPA, 2022e). By comparison, approximately 20,000 water samples will be processed annually as part of the UCMR 5 program. Initial monitoring requirements will increase the demand for laboratory capacity by a factor of more than 11.

Over the past few years, the demand for the analysis of samples has continued to grow and has outpaced the increase in laboratory capacity. Water systems are currently reporting sampling challenges like longer processing and turnaround times, higher analytical costs, and less reliable reporting data

² 42 U.S.C. 300g-1(b)(B)(5)(B)(ii).

³ 42 U.S.C. 300g-1(b)(B)(4)(D).

quality. The surge of sampling activity, especially with an emphasis on lower reporting levels, will further strain the existing laboratory capacity. EPA will therefore create unavoidable compliance risks for public water systems unless it extends the implementation timeline to the maximum extent possible.

Securing Financing is a Slow Process

A crucial step for installing capital improvement projects is to identify and secure a source of financing. In announcing this rule, EPA highlighted the funds through the Bipartisan Infrastructure Law (BIL) and the Drinking Water State Revolving Fund (DWSRF). Financing is also available through the Water Infrastructure Finance and Innovation Act (WIFIA) program. These programs provide an avenue for water systems to finance new treatment facilities, but these programs are known to be time consuming and sometimes take several years to acquire approval, which is in addition to the time to get to a project design that can be reviewed. These programs may also impose additional requirements for funding to be approved that may limit procurement options and costs. Whether a system utilizes the DWSRF, WIFIA, or through the market, the process may still be slow and will be independent of the typical financial planning process of developing a Capital Improvements Program. Planning through this program helps to assure that capital improvements are staged in a way that minimizes water rate impacts by staggering major investments within a community's water infrastructure.

Simultaneous Compliance will Slow Down Implementation

The installation of new water treatment facilities requires sufficient planning to ensure that bringing a plant into compliance with a new rule does not cause non-compliance with existing regulations. For most water systems in the U.S., the installation of PFAS treatment facilities will create challenges for simultaneous compliance with existing drinking water rules. Each of the best available technologies for PFAS will have impacts on the finished drinking water and may require post-treatment to avoid negative impacts. For example, the use of reverse osmosis (RO) and anion exchange (IX) treatment can increase the corrosivity of water impacting the potential for lead release into drinking water at homes. Granular activated carbon (GAC) has been known to contribute to distribution system nitrification. These impacts can be mitigated, but mitigation requires adequate evaluation.

One such example where simultaneous compliance concerns will delay the implementation of new drinking water treatment is the requirements Lead and Copper Rule Revisions (LCRR) (EPA, 2021d). When systems determine that mitigation is needed to comply with any new PFAS standards – either through new treatment or a change in the water supply source – they will need to comply with the LCRR requirements, which could include a lengthy process of analysis and subsequent studies to obtain approval from their primacy agency. The LCRR corrosion control studies and subsequent actions could take years to achieve. When complying with the LCRR leads to significant changes in corrosion control treatment (CCT) after the rule's administrative procedures are following, a system must have time to: (i) prepare the distribution system and customers for the transition, (ii) shift corrosion control practice at a pace that does not lead to water quality concerns, and (iii) simultaneously install the required PFAS treatment or water supply option. This will have a significant impact on the system's ability to install PFAS treatment within three to five years of any final PFAS rule and impact the cost of implementing new treatment for PFAS.

Most Systems will Need to Perform Pilot Testing

Finally, another important step in installing PFAS treatment facilities is pilot testing. While GAC, IX, and RO have been documented as being capable of removing PFAS effectively, they still require a sufficient level of pilot testing. Pilot testing typically takes at least six to nine months to complete, and costs vary but include the rental cost of equipment, engineering and other technical support, and appropriate monitoring and sample analysis costs. Bench scale testing can also be useful and is less costly for some systems.

There are a number of important goals associated with pilot and bench-scale testing are 3-fold: (i) demonstration of PFAS removal efficacy, (ii) characterizing pre- and post-treatment needs, and (iii) optimal treatment technology selection, (iv) confirmation of design and operational parameters, and (v) estimation of capital, operations and lifecycle costs (AWWA, 2020a). It is anticipated that most of the water systems that must install treatment to meet PFAS MCLs will need to perform pilot testing, especially given the permitting requirements to comply with the LCRR, as discussed above. While pilot testing may not seem appropriate for smaller systems, it is similarly vital for these systems to ensure that the expense of capital for a new long-term treatment facility is both cost-effective and appropriately designed to protect public health from secondary water quality changes. The potential cost to small systems if new treatment facilities fail to operate as intended can be severe, given the cost of identifying and implementing solutions cannot be distributed across a large number of households, particularly after water rates are already rising to take on debt of the initial PFAS rule compliance solution.

Implementation will Further Strain the Supply Chain

When considering the costs and feasibility of the timeline and proposed rule, EPA must also take into account current supply chain issues. Water systems have been faced with a strained supply chain, which were worsened following the start of the COVID-19 Pandemic. This strain has led to increased purchasing costs, longer lead times for equipment or materials, and limitations on the products that are available. Lead times for key equipment (e.g., vessels, carbon or resin media, electrical components, etc.) have already increased to beyond twelve months, depending on the equipment and the degree of specialization it requires. Vessels, GAC media, and IX resin are not widely available from more than a few suppliers. Lead times for replacement GAC media are currently six months or more and for new customers the lead time is in the range of twelve to eighteen months. The lead time for GAC media will increase as a surge of new systems begin ordering GAC and suppliers will need to acquire media internationally (e.g., China and India) as the domestic market becomes more strained. Manufacturing of IX resin is currently not domestic given the safety concerns regarding the chemicals used in its production as demand for IX resin increases the supply chain is anticipated to strain as well. These issues are also impacting major ancillary equipment like electrical panels, motor control centers, etc.

Workforce Limitations will be Worsened

The water sector is currently working to overcome workforce challenges, which EPA must also recognize when considering the feasibility of the timeline and proposed approach. EPA estimates that one-third of the sector's workforce is eligible to retire within the next 10 years and water systems are facing challenges in recruiting, training, and retaining employees (EPA, 2023a).

These challenges are expected to be more severe as more than 4,300 water systems are driven to advanced technologies that require more specialized technical skills. To install and operate these facilities, water systems will need to hire or contract engineers, manufacturers and suppliers, construction crews, and skilled operators. These service providers are already in high demand and in short supply. The resulting imbalance is impacting labor and material costs, lead times for materials, turnaround times for services (e.g., engineering, laboratory analysis, construction). The installation of new treatment facilities will surge following rule's promulgation, which will further worsen workforce challenges for water systems.

The demand for highly skilled water treatment operators will increase due to this rule. Systems currently not using filtration for water treatment may need to meet additional operator certification requirements. While each state independently sets certification requirements for water treatment plant operators, it is anticipated that systems requiring to install GAC, IX, or RO will need to staff operators with more advanced certification. Water systems in the states of Virginia⁴, California⁵, Colorado⁶, and Massachusetts⁷, for example, will all see impacts to operator certification requirements as a result of new treatment systems for PFAS. This change will have a significant impact on systems with a limited local labor pool or limited financial capacity to attract skilled operators that will be needed to safely operate these advanced treatment systems. As EPA recognizes, systems must have staffing with appropriate qualifications to operate 24 hours a day, 365 days a year. Many systems will struggle to find qualified operators for adequate staffing.

State Primacy Agency Capacity

Because the typical timeline for the planning, design, permitting, and construction of a new drinking water treatment facility for PFAS may take up to and exceed 5 years, the standard compliance window of three years under SDWA will not be feasible. This is especially important given that these implementation challenges will drive the timeline up as systems begin competing for the same limited supply of sector resources. Under SDWA, water systems may request a two-year extension for compliance with MCLs if it is determined that additional time is necessary for capital improvements.

It is anticipated that the vast majority of water systems that need to install treatment for PFAS will need to request this two-year extension, which is typically provided at the discretion of the state primacy agencies. State primacy agencies are currently working to review lead service line inventories, preparing to implement the corrosion control treatment requirements of the LCRR, administering the DWSRF and additional projects accessing funds from the Bipartisan Infrastructure Law, and working to ensure and improve water system compliance with existing rules. As the surge in water system requests for a two-year extension begins, these agencies will be strained as they work to review and process these requests.

However, the Administrator also has the authority under SDWA to provide this extension and can do so as a part of the rule as opposed to being done so on a case-by-case basis. In order to prevent issuing a

⁴ 18 Virginia Administrative Code 160-30-370 – Waterworks.

⁵ California Code Regs. Tit. 22, § 64413.1. - Classification of Water Treatment Facilities.

⁶ 5 Code of Colorado Regulations 1003-2 Regulation 100 - Water And Wastewater Facility Operators Certification Requirements: Sections 100.4 to 100.9.

⁷ 310 Massachusetts Register 22.11B.

final rule that is infeasible due to the implementation timeline or otherwise violates the APA as arbitrary and capricious, AWWA recommends that the Administrator leverage this authority to increase the likelihood that all water systems can comply with the timeline of the rule and take adequate, effective steps towards mitigating PFAS levels in drinking water.

4. Analysis of Occurrence Data

The SDWA requires that EPA rely upon the best available public health information, including the occurrence database.⁸ EPA must therefore ensure that the data on which it relies meets this standard. Because “best” is necessarily comparative, EPA must also provide sufficient explanation regarding the data selected and not selected as the basis of EPA’s decision so that the public can meaningfully comment on the data selected as well as evaluate the data that EPA did not decide to rely upon.

According to the proposal, EPA applied a statistical modeling approach to characterize occurrence data for PFAS using a combination of both national occurrence monitoring data from the UCMR 3 and more recently collected state data. While EPA typically relies on nationally representative occurrence data from the UCMR program to drive decisions for NPDWRs, the agency previously noted an interest in using data collected by state monitoring programs given the UCMR 3 database’s high reporting limits relative to the potential levels of health concern (EPA, 2021e). AWWA appreciates the agency’s interest in advancing this rulemaking by leveraging more recently collected data using improved methods. A detailed understanding of contaminant occurrence in drinking water across the country is a key factor for developing not only regulatory determinations, but also drinking water standards. In review of this approach, several opportunities to improve the analysis were identified.

Application of the Bayesian Statistical Model

The engine of the occurrence analysis for the proposal is the Bayesian hierarchical statistical model (the Bayesian Model), that uses PFAS occurrence data from more recent monitoring programs to provide improved understanding of the UCMR 3 data below the reporting limits. Similarly, the occurrence analysis is the engine of the entire rulemaking, informing the EPA’s understanding of the regulatory impacts of the rule. For this reason, it is imperative that the Bayesian Model be utilized appropriately and with statistical confidence.

While the Bayesian Model approach is sophisticated technically, it is an overly complex approach for characterizing national occurrence. Bayesian models can be useful in many applications but there are some key challenges that arise with the use of these models that make it non-optimal for regulatory applications. The key challenge is that the selection of priors and the posterior conditions is a very subjective decision that is subject to the discretion of the statistician that is crafting the model. As such, it is expected that the assumptions around these decisions are documented clearly and in detail. While EPA has provided a copy of the code used for the Bayesian Model, along with a recent publication about the model, there is a lack of a non-technical description of the agency’s intended approach. The lack of a clear explanation of intent regarding the model’s code leaves stakeholders unable to confirm that the code is accurately developed, and therefore unable to meaningfully comment on this aspect of the proposal.

⁸ 42 U.S.C. § 300g-1(b)(1)(B)(ii)(II).

Additionally, EPA only provided a 60-day public comment period for review and analysis of the entire proposal, including the occurrence analysis. AWWA and numerous additional stakeholders requested an extension of the comment period to support more in-depth of the occurrence analysis and the rule more broadly but the agency declined this request. The comment period is inadequate for reviewing this model and EPA neglects to provide informative sensitivity analyses and to clearly present model assumptions and outputs. In order to fulfill its obligations under the APA, AWWA therefore requests that EPA provide this information during a supplemental comment period prior to finalizing any rule.

Use of Non-UCMR 3 Data

EPA's occurrence analysis relies on data from both UCMR 3 and state monitoring programs. AWWA supports the consideration of the more recently collected data from state monitoring programs to improve understanding of occurrence, but there are several concerns about the agency's use of this data and the degree of quality control. These issues are discussed in more detail in the following paragraphs. As previously noted, the SDWA requires that EPA rely upon the best available public health information, including the occurrence database.⁹ EPA must also provide sufficient information and explanation regarding the data selected and not selected as the basis of EPA's decision so that the public can meaningfully comment on the data selected as well as evaluate the data that EPA did not decide to rely upon.

It is unclear how data was screened for inclusion as part of the analysis. Several state monitoring datasets are documented to have reporting thresholds far below what is considered reliable for a national occurrence analysis; for example, reporting thresholds below 1 ppt are indicated for several states including New Jersey, Massachusetts, and California. In other cases, states did not indicate the applicable reporting thresholds. While the proposal acknowledges these data quality issues, the agency nonetheless elected to utilize this data without quality control. As AWWA noted in 2020, EPA should supplement monitoring data from UCMR 3 with high quality occurrence data (AWWA, 2020b). It is recommended that EPA re-evaluate the non-UCMR 3 data that is being leveraged and ensure that monitoring results that are neither achievable using the robust methods approved by EPA nor representative of high-accuracy data should not be considered as part of this analysis.

Additionally, EPA has noted that the Bayesian Model's incorporation of state monitoring data excludes all non-UCMR 3 data that was collected by water systems that did not participate in UCMR 3. While EPA rationalizes this decision by highlighting that the UCMR 3 program was designed to collect data that is nationally representative, this approach fails to realize an opportunity to leverage the vast quantity of non-UCMR 3 data that is available. A project was conducted by Corona Environmental Consultants for AWWA that collected PFAS monitoring data from both UCMR 3 and state monitoring programs (Corona, 2021). This work successfully aggregated data from these programs from nearly 8,000 public water systems from across the country. Of these systems, 668 systems had participated in UCMR 3 and had more recent data available through state monitoring programs. Additionally, data was available for more than 3,100 water systems had participated in state monitoring programs but not UCMR 3. More effective inclusion of this data would expand the more recent data set for PFAS occurrence by a factor of nearly 5, which could improve our understanding of occurrence for smaller systems significantly.

⁹ 42 U.S.C. § 300g-1(b)(1)(B)(ii)(II).

It is reasonable that the EPA is interested in leveraging the non-UCMR 3 data in a way that is nationally representative, but there are two aspects of the proposed approach that require a more detailed review. First, it is not clear why EPA is willing to leverage an overly complicated Bayesian Model to assess occurrence but at the same time is unwilling to develop a statistical approach to incorporating the additional non-UCMR 3 data in a manner that is nationally representative. EPA has existing experience under the UCMR program selecting small water systems for participation in the UCMR program that will be nationally representative. It is not apparent that EPA considered or attempted to leverage the significantly sized dataset of non-UCMR 3 data more effectively, especially to improve the occurrence analysis for smaller systems. In order to fulfill its obligations under the SDWA, EPA must likewise use a statistical approach to the smaller water systems in order to make better use of this dataset.

Additionally, it is likely that the non-UCMR3 data that EPA used for the Bayesian Model is biased and not nationally representative. Consequently the data EPA used is likely not the best available public health information to support the rulemaking. Bias is present in the data collected after UCMR 3 is correlated to states with elevated concerns about statewide PFAS contamination following UCMR 3 detections and improved understanding of likely sources of PFAS in the state. While EPA notes that non-UCMR 3 data collected by systems that did not participate in UCMR 3 should be excluded because it may not be nationally representative, the agency did not determine whether the systems that were included are nationally representative. Unless EPA addresses its inconsistent treatment of available data before issuing any final rule, it risks violating both the SDWA and the APA.

Finally, the occurrence analysis was improperly used to project a probabilistic distribution of PFAS levels across all water systems in violation of the SDWA and APA. This approach may be appropriate for systems where there is not available data as that actual data is the best available data. However, in taking this approach EPA cannot ignore data for specific system PFAS levels. These systems, that have previously collected data, should be captured in the occurrence analysis based on their previously collected data. Similarly, EPA should reflect actual data when available in its EPA's occurrence analysis of PFAS and total organic carbon (TOC). Besides ensuring that the costs for these systems are accurately reflected, this will ensure that any unique relationships that may exist between TOC occurrence and PFAS occurrence will be captured.

The SDWA explicitly provides a mechanism for EPA to obtain nationally representative occurrence data for contaminants in drinking water by requiring EPA issue a new list of unregulated contaminants to be monitored in drinking water every five years.¹⁰ This list is known as the UCMR. The UCMR serves to better inform regulatory determinations, as contaminants are evaluated based on health effects and occurrence information, and EPA has historically relied on the UCMR process to collect occurrence data on contaminants to support a determination on whether to regulate contaminants. There are times when more recent or robust data may be available outside of UCMR collection,¹¹ and in such cases, EPA can appropriately rely on a combination of UCMR and non-UCMR data when available, so long as in doing so it provides a reasoned explanation for its approach and ensures that it is relying on the best available data for national occurrence. EPA has not done so here and must revise its data and provide greater transparency into its data in order to fulfill its obligations under the SDWA.

¹⁰ 42 U.S.C. § 300g-1(b)(1)(B).

¹¹ 87 Fed. Reg. 68060, 68062 (November 14, 2022).

Inclusion of Data from Fifth Unregulated Contaminant Monitoring Rule

This year, drinking water monitoring for PFAS under the Fifth Unregulated Contaminant Monitoring Rule (UCMR 5) began. UCMR 5 is expected to be the most comprehensive occurrence dataset for PFAS collected to-date. Indeed Congress took the additional step of explicitly instructed EPA to include these substances in UCMR 5 as part of the National Defense Authorization Act for Fiscal Year 2020 (Congress, 2019). In accordance with UCMR 5, more than 10,300 systems will monitor for 29 PFAS using EPA Methods 533 and 537.1 using single-digit minimum reporting limits. While a complete dataset will not be compiled until 2026, more than 3,500 water systems are actively collecting monitoring data this year. By the end of summer this year, EPA will have at least one sample result from these 3,500 water systems. Previous research has shown that preliminary data collected from UCMR monitoring provides accurate insights about occurrence when compared to the complete dataset (Eaton et al, 2018).

UCMR 5 data collected during the first half of 2023 would represent a significant increase in the available data that is nationally representative. Specifically, this dataset will provide information on nearly 70% of the number of water systems that are typically represented by a UCMR program. While this data would not be a full UCMR sampling program, it would significantly expand the universe of nationally representative data at method reporting levels deemed appropriate in the proposed rule. As the goal of the UCMR 5 program is to inform EPA on the occurrence of PFAS, EPA can ill afford to ignore these data, which would be of a much higher quality and value than its current Bayesian Model approach.

The UCMR program is designed to collect national occurrence data on contaminants not currently subject to NPDWRs, and EPA “require[ed] collection of data under UCMR 5 to inform EPA regulatory determinations and risk-management decisions” (EPA, 2021c). Given that Congress explicitly instructed EPA to include PFAS chemical in UCMR 5, Congress clearly intended for the national occurrence data resulting from UCMR 5 to inform EPA’s regulatory determinations about these substances. This strongly suggests that EPA should wait to take final regulatory action on these substances until all UCMR 5 data has been collected so that that its decisions can be fully informed with the best available information. This is particularly true given that the SDWA’s anti-backsliding provisions will require continued regulation of these substances once EPA has issued a final NPDWR: EPA must make the most informed decision possible at this stage to fulfill its statutory obligations and prevent unnecessary and unjustified regulations.¹²

But even if EPA does not wait until all UCMR-5 data has been collected, it must at the very least incorporate and prioritize data already provided to the agency under UCMR-5 in making regulatory decisions under this proposal. Given that much of the UCMR 5 data has already been collected, EPA cannot meet the SDWA’s directive to rely on the best available public health information without taking into account this most current and comprehensive set of data.

¹² See 42 U.S.C. § 300g-1(b)(9) (“Any revision of a national primary drinking water regulation shall be promulgated in accordance with this section, except that each revision shall maintain, or provide for greater, protection of the health of persons.”).

Distinguishing Between PFAS Detections and Levels of Health Risk Concern

The SDWA only allows EPA to regulate a substance when “the contaminant is known to occur or there is a substantial likelihood that the contaminant will occur in public water systems with a frequency *and at levels of public health concern*.”¹³ The agency’s discussion about the occurrence of PFAS in drinking water frequently discusses the likelihood of detections of PFAS. While this is useful, it would be more relevant, and therefore beneficial, for EPA to provide information about PFAS occurrence at levels closer to the relevant health risk levels, particularly the proposed MCLGs or MCLs. While occurrence at any level is useful to understand, it is misleading to the public and it is important that occurrence be contextualized with the relevant levels of health concern, given that that is the proper statutory focus under the SDWA. A detection of PFBS at 5 ppt, for example, represents a level that is less than 0.25% of the EPA’s lifetime health advisory level (HAL). In comparison, a detection of PFHxS at 5 ppt represents a level that is 55% of the EPA’s proposed health-based water concentration (HBWC). Put plainly, a detection of PFBS represents a much different level of risk than a detection of PFHxS. For this reason, EPA should ensure that the occurrence analysis provides context on PFAS occurrence that is more useful than detection.

Providing Transparency of Occurrence Analysis Outputs

While it has been previous practice of EPA to depict the conclusions of its occurrence analysis by presenting the number of water systems (in addition to the population) impacted by the proposed rule and the rule options. EPA failed to follow that practice here and did so without providing a proper explanation for this change in its analytical approach, as required by the APA. Instead, EPA provided a breakdown of the population impacted along with a more limited set of information about the small systems that are impacted.

AWWA previously inquired about additional information on Bayesian Model in Fall 2022 and received a series of statistical outputs that did not assist AWWA in an understanding the PFAS occurrence at systems. Following the rule’s publication to the Federal Register AWWA discussed the absence of this data in the Docket with EPA staff during a conference call; in discussion with staff, EPA clarified that the data, in their entirety, was not available as part of the supporting information provided for this rulemaking.¹⁴ AWWA made a written request for this data following this conference call and has not received the completed information (Moody, 2023).

It is important that relevant occurrence information underpinning a rulemaking analysis be made available for public comment and included in the record as part of any final PFAS rule, and for all future proposals for national primary drinking water regulations, so that the public can understand the overall impact of the rule on communities and to confirm that the benefits and costs attributed to the rule are accurate.

¹³ 42 U.S.C. § 300g-1 (b)(1)(A)(ii).

¹⁴ Conference call on March 28, 2023, between AWWA and EPA staff.

Summary of Recommendations for Improving Occurrence Analysis

In order to comply with the requirements of the SDWA and APA, AWWA makes the following recommendations to improve quality and transparency of the occurrence analysis:

1. The agency should provide clearer and more transparent information on the intended approach of the Bayesian Model, including information on the model outputs with respect to the number and type (e.g., system size, source) of water systems impacted by the various regulatory options.
2. The agency's consideration of non-UCMR 3 data needs additional quality control to exclude system data that does not meet the necessary data quality for national representation, such as reported results that are below nationally reliable reporting limits, as recognized by EPA.
3. Non-UCMR 3 data should be leveraged more effectively. Specifically:
 - a. The agency should leverage existing methodologies used to by the UCMR program to incorporate data from systems beyond the scope of UCMR 3 as part of the Bayesian Model in a way that maintains the national representation of the data and provides additional confidence.
 - b. If the agency determines that there are insufficient resources within the agency to do this, the non-UCMR 3 data that is excluded from the Bayesian Model should be used to evaluate the Model outputs.
4. The non-UCMR 3 data that is included in the Bayesian Model and considered to be nationally representative should be evaluated and EPA should substantiate the basis for its inclusion.
5. The agency should consider a 2-tier approach that relies on best-available, system specific data on PFAS levels and TOC levels and relies on a probabilistic distribution approach to the remaining systems without known PFAS and TOC levels.
6. EPA should not finalize the occurrence analysis without considering the availability of high-quality, nationally representative data from the UCMR 5 program to either improve the existing occurrence analysis or replace the analysis.

5. Preliminary Regulatory Determinations

The proposal includes preliminary regulatory determinations for four PFAS (and their mixture) concurrently with a proposed drinking water regulation of these compounds. AWWA supports the agency's interest in looking at PFAS beyond PFOA and PFOS for potential action and has previously recommended that the agency do so by applying adequate resources to fill data gaps (AWWA, 2020b).

Under the SDWA, EPA may only issue a NPDWR for a contaminant that is known to occur or there is a substantial likelihood that it will occur in public water systems at a level of public health concern.¹⁵ In the preamble to the proposal, the agency notes that there is not a "bright-line threshold for occurrence in drinking water that triggers whether a contaminant is of public health concern". AWWA agrees that SDWA does not define a "bright-line threshold" that would define that a contaminant is of public health

¹⁵ 42 U.S.C. § 300g-1 (b)(1)(A)(ii).

concern but given the statutory focus on the “adverse effect on the health of persons”¹⁶ it would be arbitrary and capricious and conflict with the SDWA if EPA did not use the level of adverse health effect to represent the level at which a contaminant starts to be considered a public health concern. AWWA also notes that EPA should consider its past practices for determining whether a contaminant reaches a level of public health concern and ensure that its approach in any final rule is consistent with past practice or that it provides a reasoned explanation for any deviation from past practice. AWWA offers recommendations for each of these options, with this and other aspects in consideration. AWWA further notes that the best available health information indicates that a negative determination is appropriate for PFNA, HFPO-DA, and PFBS at this time.

PFHxS

As part of the proposal, EPA has developed a HBWC using minimal risk levels that were developed by the Agency for Toxic Substances and Disease Registry (ATSDR) (ATSDR, 2021). AWWA supports the use of the proposed HBWC as a screening level for PFHxS in support of the regulatory determination as EPA’s Integrated Risk Information System (IRIS) program works towards completing its health assessment (EPA, 2023b).

Occurrence data for PFHxS is available not only through the UCMR 3 program but also as part of numerous state monitoring programs. Data is also currently being collected through the UCMR 5 program by 3,500 systems this year and more than 10,000 systems by the end of 2025 (EPA, 2021c). In review of the UCMR 3 data that is currently available, approximately 1.1% of water systems detected PFHxS at 30 ppt, more than 3 times higher than the proposed HBWC of 9.0 ppt. Additionally, data from California, Colorado, Pennsylvania, Vermont and Ohio show a similar trend of occurrence at levels above the proposed HBWCs (California Water Boards, 2023; CDPHE, 2023; Ohio EPA, 2023; PADEP, 2023; VTDEC, 2023). In review of the available occurrence data in comparison with the EPA’s proposed HBWC, AWWA agrees that there is evidence that PFHxS occurs in drinking water at potential levels of concern.

HFPO-DA

The UCMR 3 program did not include monitoring for HFPO-DA. Therefore, EPA is not able to determine the national occurrence of the chemical. While some states have conducted monitoring for HFPO-DA, these states provide only a limited understanding of national occurrence.

In review of the EPA’s analysis, HFPO-DA monitoring was conducted in only 16 states and the data does not provide sufficient evidence that there is national occurrence of HFPO-DA. In two states, North Carolina and Alabama, sampling data was available, but the extent of the program was unknown and so statewide occurrence levels can not be determined. In eight states, HFPO-DA was not detected in any systems and in another three states there was less than 0.2% of systems with detections of HFPO-DA, let at levels above the EPA’s lifetime health advisory level of 10 ppt (EPA, 2022f).

The only state with a significant number of detections of HFPO-DA at systems was Kentucky. A total of 81 systems were sampled across the state. Of these 81 systems, eleven detected HFPO-DA. An in-depth review of this data shows that all of these systems, except one, rely on the Ohio River as a water supply source. The last system relies on the Ohio River Alluvium. This data suggests that HFPO-DA contamination in Kentucky is not widespread but rather linked directly to recent releases directly to the

¹⁶ 42 U.S.C. § 300g–1 (b)(1)(A)(i).

Ohio River. Specifically, it is anticipated that the Ohio River, and these systems, have been impacted by discharges of HFPO-DA from the Washington Works PFAS manufacturing plant in Parkersburg, West Virginia.

The Washington Works plant has long been a center for discussions on PFAS contamination. However, the EPA recently took landmark action against this plant for violations of the Clean Water Act related to discharges of HFPO-DA to the Ohio River (EPA, 2023c). With this action, it is anticipated that HFPO-DA levels in the Ohio River will drop, which will lead to a reduction in contamination of affected systems. Similar action has been taken by the North Carolina Department of Environmental Quality (NCDEQ) against the Fayetteville Works facility along the Cape Fear River. In 2019 NCDEQ issued a consent order that required the facility to begin taking mitigative measures against the release and contamination of HFPO-DA in the area surrounding the facility. Following EPA's publication of the lifetime health advisory level in 2022, this consent order was updated and now will limit discharges to a maximum of 10 ppt HFPO-DA. This action will reduce HFPO-DA contamination in the Cape Fear River.

Data is available on the production, use, and release of HFPO-DA from the EPA's supporting documentation and shows that HFPO-DA was released by five facilities in five states. According to the most recent TRI program data for HFPO-DA, 72% of the total HFPO-DA and its ammonium salt released was from the Fayetteville Works facility in North Carolina; the Washington Works facility in West Virginia accounted for 5.7% to the total releases (EPA, 2023d). Given that both of these facilities are reducing releases, as discussed above, this will reduce the total release of PFAS by as much as 77.7% from these two facilities alone. Further reductions are anticipated following the promulgation of the ELGs for manufacturers and metal finishers under the CWA, which has been identified by EPA as a part of the Effluent Guidelines Program Plan 15 (EPA, 2021b). Similar reductions may be anticipated as EPA and states work towards addressing PFAS as part of the National Pollutant Discharge Elimination System (NPDES), as directed by the agency in April and December of 2022 (EPA, 2022d; EPA, 2022e).

Overall, UCMR data is not currently available for HFPO-DA and the available state data is not sufficient to determine the national occurrence of HFPO-DA in drinking water. Furthermore, the limited occurrence observed by state monitoring programs and the limited extent of production shown by information from the TRI program data is not suggestive of a substantial likely of HFPO-DA occurrence in drinking water with a frequency and at levels of public health concern. Instead, the available evidence indicates that a negative determination is appropriate for HFPO-DA. However, given that the EPA is currently collecting occurrence data for HFPO-DA in drinking water as part of the UCMR 5 program, EPA could consider re-issuing a preliminary determination for HFPO-DA following the completion of this program. This approach would ensure that the best available occurrence data is used.

PFNA

As with PFHxS, EPA also developed a HBWC for PFNA using data from ATSDR (ATSDR, 2021). AWWA supports the use of the proposed HBWC as a screening level for PFNA to guide this determination in the absence of a completed IRIS program health assessment (EPA, 2023b).

Occurrence data for PFNA is available not only through the UCMR 3 program but also as part of numerous state monitoring programs. Data is also currently being collected through the UCMR 5 program by 3,500 systems this year and more than 10,000 systems by the end of 2025 (EPA, 2021c). In review of the UCMR 3 data that is currently available, less than 0.3% of water systems detected PFNA at

20 ppt (twice the level of the proposed HBWC of 10 ppt). Data from state monitoring programs showed similarly extremely low occurrence of PFNA in drinking water. Vermont, for example, required sampling of 1,794 water systems across the state and PFNA was detected above 10 ppt in only 12 systems, or 0.7% of systems (VTDEC, 2023). California monitoring, for example, found that 95% of samples with detections were below 3.2 ppt (California Water Boards, 2023). Monitoring data from Pennsylvania Department of Environmental Protection showed a maximum PFNA concentration of 14 ppt in the state, with a median PFNA level of 5.6 ppt (PADEP, 2023). Data from showed a similar trend of low to minimal occurrence at the HBWC (CDPHE, 2023).

The available data on the production, use, and release of PFNA from the EPA's occurrence analysis indicates that there are not significant sources of PFNA in the United States. While this could be due to inefficiencies in the reporting requirements under the EPA's authorities of the Emergency Planning and Community Right To Know Act's Toxics Release Inventory (TRI) program, the agency must rely on the best available data (AWWA, 2023). EPA recently proposed rules that will require more improved data reporting on PFAS production, use, and release in the following years (EPA, 2022b).

In review of the available UCMR 3 data, state monitoring data, and manufacturing data for PFNA, the best available evidence does not suggest that there is a substantial likelihood of PFNA occurrence in drinking water with a frequency and at levels of public health concern. Based on the available evidence, and the SDWA statutory criteria, a negative determination is most appropriate for PFNA. However, given that the EPA is currently collecting occurrence data for PFNA in drinking water as part of the UCMR 5 program, EPA could consider re-issuing a preliminary determination for PFNA following the completion of this program. This approach would ensure that the best available data is utilized, not only occurrence data but also a forthcoming health assessment for PFNA under IRIS program (EPA, 2023b).

PFBS

The method reporting limits of the UCMR 3 program for PFBS provide sufficient clarity on occurrence at levels far below levels of health concern. For example, EPA's health advisory level is 2,000 ppt for PFBS and was only detected in less than 0.2% of systems above the reporting limit of 90 ppt under UCMR3 (EPA, 2022g) . Data from the state of Pennsylvania showed that the maximum PFBS level in drinking water was 13.0 ppt, with a median detected concentration of 4.2 ppt (PADEP, 2023). California monitoring data found that the maximum concentration across the state did not exceed 120 ppt and 95% of systems detected PFBS had levels below 15 ppt (California Water Boards, 2023). Monitoring data from several other states, including Ohio, Colorado, and Vermont show a similar trend (CDPHE, 2023; Ohio EPA, 2023; VTDEC, 2023).

Additionally, the available data on the production, use, and release of PFBS from the EPA's occurrence analysis indicates that there are not significant sources of PFBS in United States. While this could be due to inefficiencies in the reporting requirements of the TRI Program, the agency must rely on the available data. EPA recently proposed rules that will require more improved data reporting on PFAS production, use, and release in the following years.

In review of the available occurrence data, both from UCMR 3 and from state monitoring programs, it is apparent that PFBS does not occur in public water systems with a frequency, and at levels, of public health concern. The evidence from the production, use and release data that is available supports this conclusion. Therefore, at this time the best available public health information does not support a

determination to regulate PFBS under the SDWA. In contrast, there is strong evidence that a negative determination is appropriate for PFBS and AWWA recommends that a negative determination be issued. EPA can revise its determination as to PFBS in the future, if new data indicates that such a determination is warranted.

Alternatively, given that the EPA is currently collecting occurrence data for PFBS in drinking water as part of the UCMR 5 program, EPA could consider re-issuing a preliminary determination for PFBS following the completion of this program.

PFHxS, PFNA, HFPO-DA, and PFBS as a Mixture

As part of the proposal, EPA also sought public comment on the preliminary regulatory determination for PFHxS, PFNA, HFPO-DA, and PFBS as a mixture. Specifically, EPA highlights that Section 1401(6) of SDWA defines the term contaminant to mean “any physical, chemical or biological or radiological substance or matter in water” and therefore a mixture of two or more “contaminants” qualifies as a “contaminant” because the mixture itself is “any physical, chemical or biological or radiological substance or matter in water.”

AWWA appreciates the agency’s interest in addressing additional PFAS beyond PFOA and PFOS. However, the proposed approach to address these additional PFAS through a preliminary determination that these PFAS as a mixture meet the definition of being a single “contaminant” under SDWA is not appropriately supported. As recognized by the TSCA, any mixture is not considered a chemical substance, instead a mixture of contaminants formed either naturally or through a chemical formulation process may be considered a chemical substance (EPA, 2023e). EPA’s “Supplemental Guidance for Conducting Health Risk Assessment of Chemical Mixtures”, which EPA cites in crafting the risk assessment framework for the proposal, specifically indicates that opportunities to infer hazard for a mixture must be from a **“sufficiently similar mixture”** (EPA, 1986). The guidance goes on further to note that a mixture is sufficiently similar when the **“components and respective portions exist in approximately the same pattern”** (EPA, 1986).

The occurrence analysis provided by EPA does not demonstrate that PFHxS, PFNA, HFPO-DA, and PFBS can be grouped together as a mixture within a manner consistent with well-established agency guidance. First, there is a complete lack of national occurrence data for HFPO-DA and the data for PFBS shows a significant lack of occurrence in drinking water. Furthermore, data for PFNA shows a very low level of occurrence on its own, let alone with the other three PFAS. EPA’s groupwise occurrence provides neither a clear nor transparent characterization of occurrence, nor co-occurrence, of PFNA, PFHxS, PFBS, and HFPO-DA. The supporting documentation fails to illustrate a pattern of co-occurrence of these four compounds; in fact, most of the information on co-occurrence of these compounds is relative to PFOA and PFOS. Given that the determination is for PFHxS, PFNA, HFPO-DA, and PFBS the co-occurrence of these individual compounds with PFOA and PFOS does not demonstrate the other four compounds co-occur.

Furthermore, the information that is provided does not demonstrate occurrence of PFHxS, PFNA, HFPO-DA, and PFBS at or above levels of potential health risk. Instead, occurrence is consistently described as a function of detection. Unfortunately, this is also not useful in supporting a preliminary determination. Detections are not equivalent to potential risk, which is most easily demonstrated in comparing the meaning of a detection for PFBS and PFHxS. While a detection of PFHxS at 10 ppt is 100% of the

proposed HBWC while a detection of PFBS at 10 ppt is only 0.5% of the proposed HBWC. Detection of PFBS is likely to bias these results and represents a significantly different level of public health concern in comparison to a detection with other PFAS.

To further investigate co-occurrence for the PFAS, AWWA conducted an analysis of PFAS occurrence data that was collected from nearly 8,000 water systems by Corona Environmental Consulting (Corona, 2021). The results of this analysis are shown in the table. While some systems may detect more than one of these PFAS, the occurrence of these PFAS together at levels above the HBWC are much more limited, if at all.

Table 4-1: Co-Occurrence of PFBS, PFHxS, and PFNA in Drinking Water (N=7,989) (Corona, 2021)

	Number of Systems with Detections (% of Systems)	Number of Systems with PFAS Level(s) Exceeding HBWCs (% of Systems)
PFHxS & PFNA	605(7.5%)	32 (0.4%)
PFBS & PFHxS	765 (%)	0 (0%)
PFBS & PFNA	561 (%)	0 (0%)
PFBS, PFHxS & PFNA	551 (%)	0 (0%)

While there are scientific studies evaluating the hypothesis that exposure to multiple PFAS may lead to adverse health effects, the proposal and its supporting documentation do not substantiate a preliminary determination nor a determination to regulate the mixture of PFHxS, PFNA, HFPO-DA, and PFBS. There is a lack of information showing co-occurrence and, therefore, co-exposure to these compounds.

Equally importantly, EPA's proposed approach to using the general hazard index is significantly flawed and is not supported by federal agency guidance nor recommendations from the Science Advisory Board (SAB) (ATSDR, 2018; ATSDR, 2022; EPA, 1986; EPA, 2000; SAB, 2022), which collectively recommend that a common health outcome should be used as the basis for a hazard index in this context.

AWWA does not support the preliminary determination of PFHxS, PFNA, HFPO-DA, and PFBS as a mixture as the statutory factors under the SDWA to support a determination are not present. While EPA claims that mixtures of these PFAS may co-occur and represent a combined risk, the supporting information fails to create a sufficient record that this is the case. This is true both for evidence of occurrence and demonstration that these PFAS pose a combined risk. This lack of toxicological support for this approach is apparent in the agency's proposed approach to use a hazard index for these compounds through a methodology that is contrary to federal agency guidance. The preliminary determination for PFHxS, PFNA, HFPO-DA, and PFBS as a mixture is not sufficiently supported and the information for three of these compounds suggests a negative determination is most appropriate.

AWWA recommends that if EPA is interested in addressing additional PFAS through a regulatory determination for a mixture of PFAS, that EPA reconsider their approach to addressing PFAS as a mixture and delay re-issuing a preliminary determination until after the data collection activities for UCMR 5 are complete. Delaying this action would also ensure that EPA may consider health assessments for additional PFAS, which are currently in development (EPA, 2023b).

6. Proposed Maximum Contaminant Level Goals

The derivation of a science-based maximum contaminant level goal (MCLG) is crucial because it means that it is both protective of public health and can transparently be communicated to inform decision-making for the public. EPA did not use the best available science in proposing the MCLGs (and MCLs) for these substances as required by SDWA. EPA must also ensure that the underlying science is review by the EPA SAB. Because EPA has not demonstrated that PFNA, PFHxS, HFPO-DA, and PFBS warrant regulation under SDWA, it cannot finalize the proposed MCL and MCLG. AWWA offers the following specific comments on the proposed derivation of the MCLGs for PFOA, PFOS, and the mixture of PFHxS, PFNA, PFBS, and HFPO-DA.

PFOA

According to the proposal, EPA is proposing an MCLG of 0 ppt (zero) for PFOA based on a determination that PFOA is likely to be carcinogenic. AWWA has previously reviewed the EPA's determination that PFOA is carcinogenic and provided comments (AWWA, 2021a). Key aspects of those comments are shown below and can be found in more detail in Appendix A.

1. EPA cites Shearer et al (2021) as a key study showing that PFOA may be carcinogenic. This study may not be suitable as evidence to support this determination given that the study duration spanned less than 18 years. Given the half-life of PFOA, it is unlikely to accurately portray the exposure relevant to the development of kidney cancer.
2. In epidemiological studies of higher exposures there has been inconsistent evidence of increased cases of kidney cancer. For example, epidemiological studies of residents exposed to PFOA and other PFAS in contaminated drinking water have reported modest increases whereas occupational cohorts have shown increased and decreased risk of kidney disease, despite higher exposure and longer study durations.

If EPA moves forward with a conclusion that PFOA is carcinogenic, AWWA agrees that the appropriate MCLG for a carcinogen is 0 ppt (zero).

PFOS

As with PFOA, EPA is proposing to establish a MCLG of zero (0 ppt) for PFOS following a determination that PFOS is suggestive to be carcinogenic. As noted in comments in 2021, AWWA supports this determination (AWWA, 2021a). If EPA moves forward with a conclusion that PFOA is carcinogenic, AWWA agrees that the appropriate MCLG for a carcinogen is 0 ppt (zero).

Combined MCLG for PFHxS, PFNA, HFPO-DA, and PFBS

According to the proposal, EPA is proposing to establish a combined MCLG for four PFAS set at a hazard index of 1.0. In proposing this MCLG, EPA is making several key scientific determinations to support this decision:

1. PFNA, PFHxS, HFPO-DA, and PFBS are likely to co-occur in water in a way that is a “sufficiently similar mixture”,
2. Co-exposure to a mixture of PFNA, PFHxS, HFPO-DA, and PFBS can lead to an aggregate health effect as a result of dose additivity, and

3. The dose additivity of PFAS can be applied through the hazard index with dissimilar health effects, or outcomes.

AWWA contracted with Ramboll Consulting U.S. to assist in reviewing the EPA's approach and to offer detailed recommendations to improve this work. Ramboll has provided a detailed letter with recommendations, which is included as part of Appendix A.

AWWA supports the agency's interest in taking a public health protective stance on PFAS. It cannot do so based on the assumption of dose additivity without sufficient evidence. There are numerous concerns regarding the agency's determination that these compounds co-occur and that their co-exposure has a dose-additive effect on dissimilar outcomes. Based on the information that EPA has currently provided and relied upon, EPA has not met the statutory or scientific requirements to make a positive regulatory determination for these substances individually or as a mixture.

First, AWWA notes that EPA's novel use of the hazard index approach in the proposal is not clearly permissible under the SDWA. The SDWA is designed for an individual assessment of contaminants and an individualized assessment of appropriate MCLG and MCL, as the statute uses the singular "contaminant" when defining "maximum contaminant level."¹⁷ The proposal runs counter to this statutory focus by a Hazard Index approach rather than a specific concentration level for proposing an MCL and MCLG for PFNA, PFHxS, PFBS, and HFPO-DA.

The Hazard Index approach is also arguably inconsistent with SDWA because it is not a "level" but instead a calculated sum of component hazard quotients using a highly variable equation that can change over time.

Earlier in these comments, AWWA raised issues regarding the EPA's occurrence analysis and the proposal's lacking evidence for occurrence, let alone co-occurrence, of these four PFAS. Notably, EPA has not demonstrated that there is or "there is a substantial likelihood that the contaminant will occur in public water systems with a frequency and at levels of public health concern"¹⁸ as required under the SDWA, and at most has shown that they may potentially occur at levels of concern. Additionally, the **agency's approach is contrary to the agency's own guidance for assessing risks of mixtures, which states that it must be "sufficiently similar mixture" where "components and respective portions exist in approximately the same pattern" (EPA, 1986).** As indicated in this guidance, a key feature of a mixture is the mixtures composition and consistent co-occurrence of the components (PFNA, PFHxS, PFBS, and HFPO-DA). The EPA's occurrence analysis fails to sufficiently document co-occurrence of this mixture of PFAS and AWWA's analysis of data from nearly 8,000 water systems does not demonstrate a pattern of co-occurrence.

Additionally, the agency has failed to provide adequate information to support the proposed approach to apply the hazard index to these compounds using reference doses based on the compound-specific critical health outcome, not a similar health outcome. In review of the draft approach, the SAB provided EPA with support for the determination of "dose additivity based on a common outcome" while also noting that this was appropriate "instead of a common mode of action as a health protective default assumption" (SAB, 2022). Additionally, the support document for this MCLG states that "component-

¹⁷ 42 U.S.C. § 300f(3).

¹⁸ 42 U.S.C. § 300g-1(b).

based approaches for assessing risks of PFAS mixtures are focused on evaluation of similarity of toxicological endpoint/effect rather than similarity in MOA [mode of action]”. The proposed approach is inconsistent with this statement and is contrary to the SAB recommendations.

In addition, ATSDR and EPA guidance on risk assessment for mixtures recommends against the use of a common mode of action when using a hazard index. ATSDR’s Framework for Assessing Health Impacts of Multiple Chemicals and Other Stressors notes that the hazard index method is most appropriately “applied to components that cause the same effect by the same mechanism or mode of action” but “may be applied to components with different target organs as a screening measure” (ATSDR, 2018). ATSDR’s Public Health Assessment Guidance Manual indicates that when “the health guideline for each contaminant is based on different target organs, health assessors will need to calculate a target-organ-specific HQ [hazard quotient] for each contaminant” (ATSDR, 2022). Both ATSDR guidance and EPA’s own guidance for risk assessment of mixtures clearly indicates that the use of critical effects from multiple tissues in a hazard index is generally inappropriate, specifically noting that the index requires “similarity in target organ” (EPA, 1986). In fact, the guidance states that “because the hazard index is tied to a specific effect, the underlying data should be on that effect.” As part of this guidance, the agency recommends that target organ toxicity doses as opposed to a critical effect dose, which is what is proposed in this action.

Detailed comments and recommendations on the proposed hazard index approach for PFNA, PFHxS, HFPO-DA, and PFBS were prepared for AWWA by Ramboll US Consulting and can be found in Appendix A of these comments.

AWWA recommends that the EPA re-issue the preliminary determination for PFHxS, PFNA, HFPO-DA, and PFBS as a mixture and recommends that prior to re-issuing a preliminary determination for a mixture of PFAS work towards refining the proposed approach to align with guidance from federal agencies (including the agency itself) and recommendations from the SAB. Aligning the use of the hazard index with agency guidance and SAB recommendations is not only necessary for sound policy, but necessary to comply with both the SDWA and the APA. Further, EPA should follow its own guidance before finalizing any risk assessment for mixtures of PFAS, by EPA ensuring that appropriate peer review be conducted to confirm the agency is relying on the best available science.¹⁹ AWWA notes that EPA followed a more robust process in evaluating PFOA and PFOS and recommends that EPA apply at least the same level of rigor to its analysis of PFHxS, PFNA, HFPO-DA, and PFBS. Failing to do so, or failing to acknowledge this change, and providing a reasoned explanation for the change, would violate the APA.

EPA stated that SDWA provides the agency with the authority to concurrently propose a preliminary regulatory determination and to propose a drinking water regulation for these PFAS, but this is not the case. SDWA provides EPA with the authority to “publish such proposed regulation concurrent with the *determination to regulate*”. A determination to regulate is distinctly different from a preliminary determination. Specifically, a preliminary determination applies to the proposed action while a determination to regulate applies to a final action. While this language authorizes EPA to propose regulation as part of the same action as a determination to regulate, it does not authorize that the proposed regulation be concurrent with a preliminary determination.

¹⁹ U.S. EPA, Peer Review Handbook, 4th edition, 2015, available at: https://www.epa.gov/sites/default/files/2015-10/documents/epa_peer_review_handbook_4th_edition_october_2015.pdf.

EPA itself has distinguished between a preliminary and final regulatory determination, the latter of which it interprets to mean the “determination to regulate.”²⁰

Second, SDWA Section 1412(b)(1)(B)(ii) specifically uses different terms for a determination to regulate and a preliminary determination:

“Not later than 5 years after August 6, 1996, and every 5 years thereafter, the Administrator shall, after notice of the *preliminary determination* and *opportunity for public comment* . . . make *determinations* of whether or not to regulate such contaminants.”²¹

In addition, collapsing these steps into a single proposal undermines the SDWA’s mandate that EPA use the best available public health information to make regulatory determinations in accordance with the three statutory criteria. While EPA is collecting public comment on the preliminary determination (and is seeking more studies and health information from the public), how would it know that regulation is warranted when it lacks a complete record? Instead, the notice and comment provisions exist to allow EPA to collect the data it needs to decide whether to regulate and to ensure the statutory criteria for it to do so are present. EPA’s approach is also inconsistent with SDWA Section 1412(b)(4)(C), which states that “[a]t the time the Administrator proposes a national primary drinking water regulation under this paragraph, the Administrator shall publish a determination as to whether the benefits of the maximum contaminant level justify, or do not justify, the costs...”²² EPA cannot reach such a decision while it is collecting public health data from the preliminary determination phase because it cannot simultaneously determine whether the benefits of regulation justify the costs. And notably, Congress knows how to direct simultaneous regulatory actions under the SDWA when it intends to, but did not do so here.²³

Because the SDWA does not provide EPA with the authority to propose a preliminary determination to regulate at the same time as a proposed regulation (and as a result it has not been agency practice to do so previously), EPA must re-issue a proposed regulation for PFHxS, PFNA, HFPO-DA, and PFBS after accepting public comment on its preliminary determination to regulate those substances in order to comply with its obligations under the SDWA and APA.

²⁰ See 76 Fed. Reg. 7762, 7763 (Feb. 11, 2011) (“What is EPA’s final regulatory determination on perchlorate and what happens next?” “the Agency has made a determination to regulate perchlorate in drinking water [and] EPA is initiating the development of a proposed NPDWR for perchlorate.”).

²¹ 42 U.S.C. § 300g–1(b)(1)(B)(ii). See also *NRDC v. EPA*, No. 20-133, slip op. at 4 (D.C. Cir. May 9, 2023) (“After the comment period ends, EPA must make its final regulatory determination.”) (emphasis added); id. at 11 (“[T]he preliminary determination precedes the notice and comment period. Once that period ends, the agency makes its regulatory determination, and that determination is final.”).

²² 42 U.S.C. § 300g–1(a)(3).

²³ For example, Section 1412(a)(3)) states: “Whenever a national primary drinking water regulation is proposed under subsection (b) for any contaminant, the maximum contaminant level goal for such contaminant shall be proposed simultaneously. Whenever a national primary drinking water regulation is promulgated under subsection (b) for any contaminant, the maximum contaminant level goal for such contaminant shall be published immediately.”

7. Compliance Cost Analysis

According to the proposal, EPA anticipates that nearly 67,000 water systems will need to comply with the proposed rule. These water systems will need to review and understand how to implement the rule, conduct initial monitoring of the regulated PFAS at each entry point to the distribution system, and potentially work to install drinking water treatment systems or take another mitigation strategy. The proposal does not fully capture these costs. Because the SDWA requires EPA to take costs into consideration, including when setting the appropriate MCL, and to issue a determination as to whether the benefits of the maximum contaminant level justify, or do not justify, the costs, EPA can only comply with its statutory requirements by conducting an analysis that fully captures these costs and making them available for public comment.²⁴ The following sections provide recommendations to improve the cost analysis.

Monitoring Requirements for Systems Participating in UCMR 5

Under the proposed rule, community water systems and non-transient non-community water systems will need to conduct initial monitoring, unless the state primacy agency approves the use of previously collected monitoring data. AWWA appreciates the agency's interest in providing this flexibility for water systems that may have already collected PFAS monitoring data for UCMR 5 or state monitoring programs. As part of the compliance cost analysis for the proposal, EPA did not include the monitoring costs associated with certain systems that *may be eligible* to take advantage of this flexibility. While it is reasonable to assume that some systems may potentially avoid initial monitoring for PFAS, it is not appropriate to exclude these costs from the analysis. While all systems serving more than 3,300 are currently already required to monitor for PFAS in accordance with UCMR 5, many of these systems may need to conduct additional monitoring to comply with the rule and to meet the timeline for compliance set by EPA. Examples of these systems may include:

- Large groundwater systems (serving more than 10,000 persons): Large groundwater systems will be required to collect quarterly samples while UCMR 5 requires the collection of two samples for all groundwater systems. There are more than 1,650 systems in this category that would need to collect additional samples beyond UCMR 5 to take advantage of this flexibility. Additionally, systems that are actively collecting samples during 2023 are unlikely to have the opportunity to adjust monitoring plans.
- Water systems scheduled for UCMR 5 monitoring in 2025: The proposal provides a 3-year timeline for water systems to comply with both the initial monitoring requirements and compliance with the MCL. Initial monitoring for the rule will need to begin immediately following promulgation of the rule to ensure that there is adequate time to take necessary action if PFAS levels exceed one or more of the MCLs. Given that the EPA's target date for a final rule is December 2023, all water systems without pre-existing data sufficient to meet these requirements will need to be monitored during 2024. This precludes the use of samples from 2025 under UCMR 5 program, as these results would give these systems less than a year to comply with the three-window for compliance, if treatment was needed.

EPA's WBS Model

²⁴ See 42 U.S.C. §§ 300g-1 (b)(3)-(4).

According to the proposal, EPA considered three treatment options that may be used by as many as 4,300 water systems to reduce PFAS levels to below the MCLs. These treatment options included GAC, IX, and RO filtration facilities. Additionally, EPA considered other options to address PFAS levels in drinking water, such as interconnections, new wells, and point-of-use RO systems. To estimate the costs to install and operate these systems, EPA relied on their Work Breakdown Structure (WBS) for these strategies, which were developed more than two decades ago and were updated as part of the proposal.

As highlighted by the EPA's "Technologies and Costs for Removing Per- and Polyfluoroalkyl Substances (PFAS) from Drinking Water" document, there are full-scale facilities that are currently using these treatment technologies for PFAS removal. To support EPA's cost analysis, AWWA contracted with Black & Veatch to prepare a cost model (the BV Model) for PFAS treatment using GAC, IX, or RO using their national drinking water treatment expertise and with support from water systems and experts from across the sector (See full report in Appendix B). AWWA has also worked with water systems to compile information on the costs to install PFAS treatment systems.

The BV Model and the case studies were used to compare with the model outputs from the EPA's WBS Model for GAC and IX to better understand the accuracy of the EPA's WBS unit cost models. Figure 7-1 shows an example of a comparison of the available case study data to the BV and EPA cost models, specifically showing capital costs associated with installing GAC treatment facilities for PFAS treatment for systems up to 2.5 MGD.

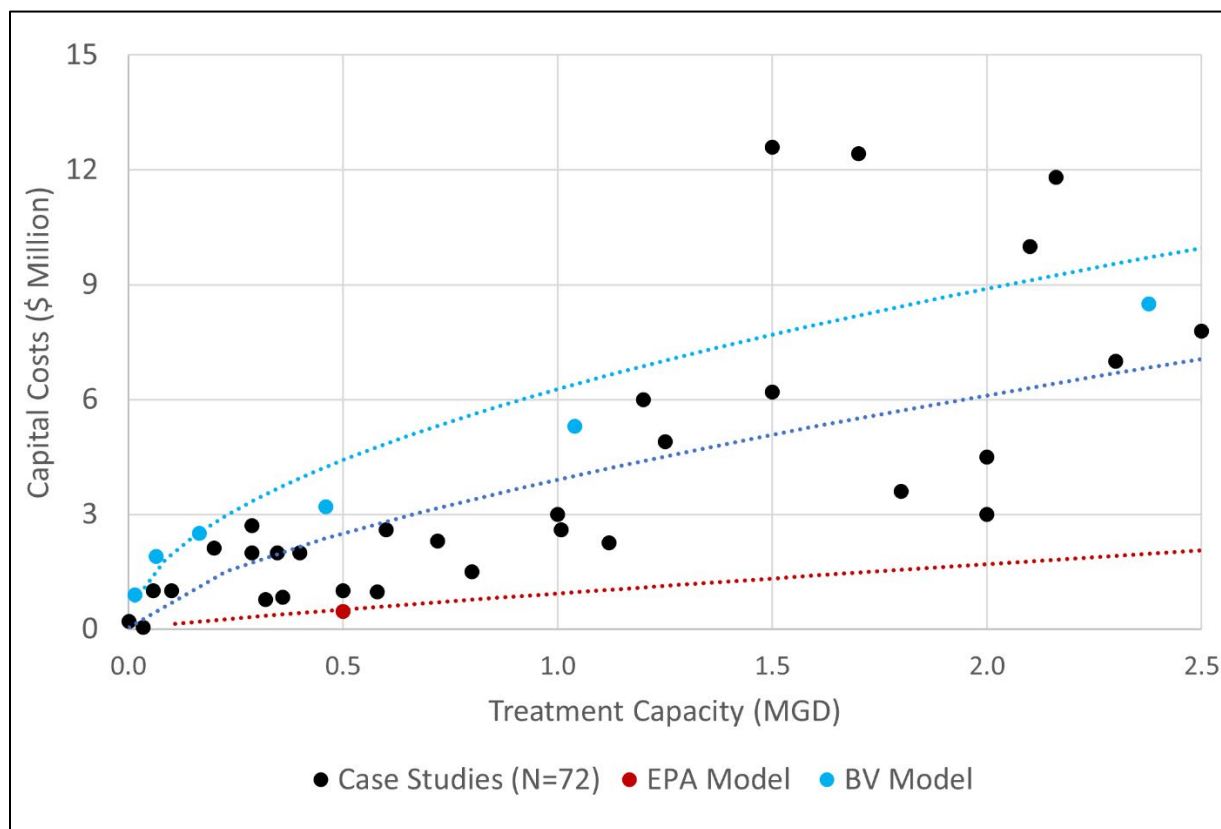


Figure 7-1: Capital Expenses for PFAS Treatment Facilities using GAC Compared to EPA and BV Models

This range was selected as it represents more than 75% of the systems that would be impacted by the proposed rule. Appendix C provides supplemental figures showing a similar comparison for additional ranges of treatment capacity, types of treatment, and operating costs. This data was developed through the use of available data from the Black & Veatch cost model, the example model outputs provided by EPA in the supporting documentation, and more than 100 case studies that were collected earlier this year. For a full list of treatment case studies, refer to Appendix D.

As shown in this figure, the EPA’s WBS model significantly underestimates the costs associated with PFAS treatment using a GAC treatment facility. Data from the case studies in this range shows that the typical PFAS treatment system costs 330% more than the estimated cost by the EPA’s WBS Model (based on 2021\$). The extent of the EPA’s WBS Model’s underestimation of cost is similarly demonstrated by the BV Model, which shows cost figures in 2022 dollars. These patterns are similarly observed when looking at larger treatment facilities, operating costs, and other treatment technologies. Refer to Appendix B and C for more information.

AWWA also compared the capital cost estimates for GAC under the proposed PFAS rule with the agency’s estimates for the Stage 2 Disinfectants and Disinfection Byproducts Rule (EPA, 2005). Under the Stage 2 rule, EPA also estimated costs for systems installing GAC with a 20-minute empty bed contact time. The results of this comparison, Figure 7-1, show an alarming issue: EPA’s cost estimate for PFAS removal in 2021 dollars is nearly the same as TOC removal in 2003 dollars. As a point of comparison, the Engineering News Record building cost index has increased from 6,654 in 2003 to 13,288 in 2023 representing a nearly double increase in construction costs alone, which do not include the additional cost increases water systems have been faced with (ENR, 2023).

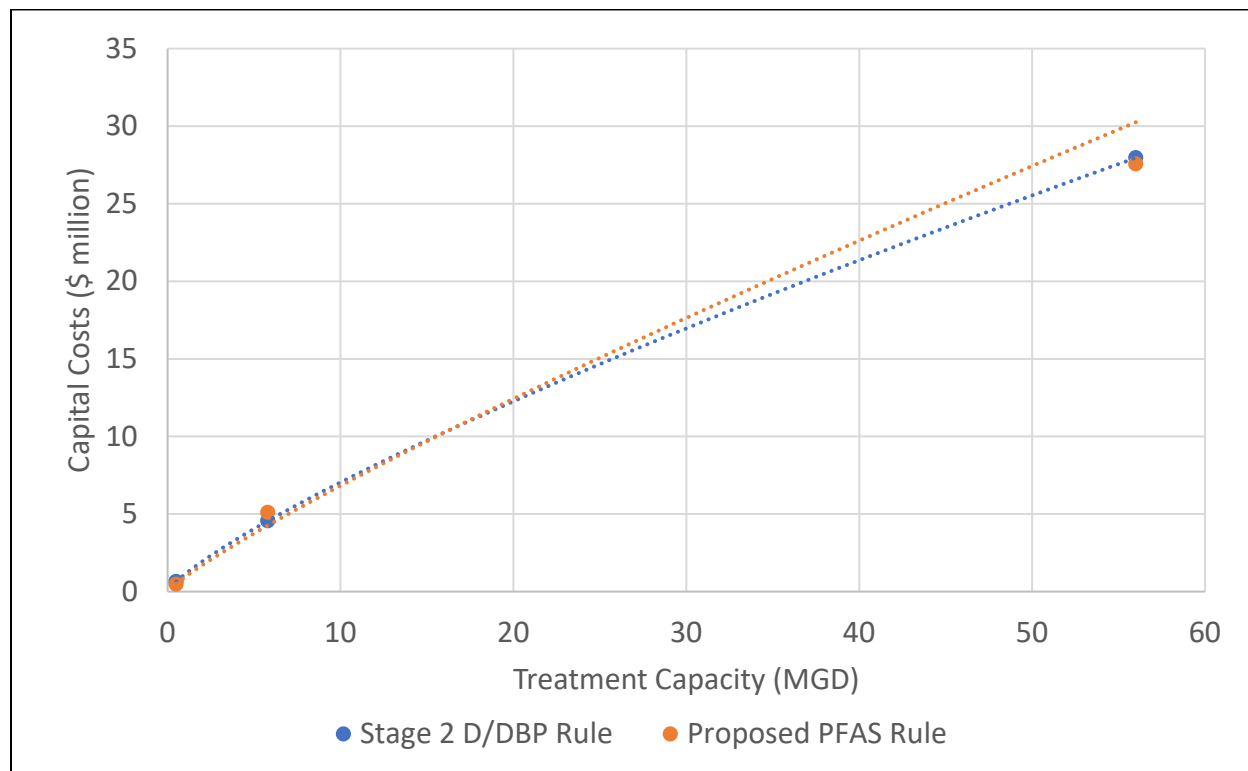


Figure 7-2: Comparison of GAC Capital Costs for GAC for Stage 2 D/DBP and Proposed PFAS Rule

AWWA requested an extension of the public comment period to accommodate a more detailed analysis of the EPA's updated WBS. Given that the agency did not extend the comment period, an exhaustive review of the WBS could not be provided within the 60-day period for review and comment. However, in review of the data from Figure 7-1, Figure 7-2, and the supplemental figures, there is no question that the WBS struggles to accurately capture costs of new treatment facilities. There are some aspects of the WBS that are anticipated to cause a significant underestimation of the true costs of installing PFAS treatment systems. These are further described in the following sections. Given the central role that costs play in EPA's determination, and in order to provide an opportunity for meaningful public comment on the costs associated with this proposal, EPA should work with AWWA and other drinking water treatment experts to revise the cost analysis and provide for an additional comment period on the updated analysis.

General Comments on EPA's WBS Model

Model Accuracy & Contingency

To estimate the costs associated with different treatment strategies, EPA's WBS model uses inputs to prepare a cost estimation for an individual water system. While the model is detailed with respect to potential costs that are considered, there are limitations of this approach, which may require the application of correction factors (similar to the toxicological uncertainty factors).

For each system, EPA estimates the service population using data available through the Safe Drinking Water Information System. The service population provides a reasonable estimate of the general water supply design requirements, which with some broad assumptions (e.g., daily per capita water use, peaking factor, etc) can inform an estimate of the overall water supply capacity needed for each entry point to the distribution system. Then, EPA relies on probabilistic distributions of PFAS and TOC to determine specific unit operation costs for each system in a Monte Carlo Simulation.

Ultimately, the WBS model relies on three key system characteristics to drive the cost estimate: (i) water treatment flow capacity, (ii) PFAS levels, and (iii) TOC levels. This information is used by EPA to estimate the implementation costs for each water system, which is intended to cover planning, design, testing, permitting, and construction of the system. While it is not uncommon for budgetary estimates to be prepared using a limited set of data, it is extremely rare for budgetary estimates of these systems to not recognize that the design is not exhaustive. Good engineering practice is to include an adequate contingency and to transparently describe the level of conservatism in the estimate based on uncertainty in the information available to prepare the cost estimate.

The American Association of Cost Engineering describes five classes of cost estimates that are distinguished by maturity level of project definition, end usage, methodology, and the expected accuracy range. A Class 1 estimate represents a level of project definition exceeding 50% where a detailed unit cost and detailed take-off have been used to estimate the costs and the cost could be as much as 15% higher. Alternatively, a Class 5 estimate represents a level of project definition of less than 2% where concepts are being screened and the use of parametric models were used and so the costs could be as much as 100% higher. In consideration of the data that are available for EPA to consider in estimating costs and given that site-specific conditions cannot be factored into the estimate, the EPA WBS Model is likely to be considered a Class 4 or Class 5 estimate where costs could be 50% or 100% higher.

EPA provides various example model outputs of their cost estimate for different systems, including the estimate of contingency. Table 7-1 provides an overview of the contingency for each of these estimates. As shown in the table, the vast majority of the model outputs show cost estimates for systems with 0% contingency included. This is for 100% of the systems with a treatment capacity below 5.809 MGD, which is pertinent to more than 75% of the water systems that EPA anticipates will be impacted by this rule. For the remaining example outputs, a very low level of contingency is included. This is inconsistent with recommended best practices for cost estimators and is expected to be a major contributor to the EPA WBS' failure to accurately capture costs for PFAS treatment facility implementation.

Table 7-1: Assumed Contingency in EPA WBS Model Example Outputs

Example Output	WBS Estimated System Cost (\$)	WBS Contingency (\$ / %)
0.500 to 5.809 MGD GAC Systems	\$470,000 - \$5,117,960	\$0 / 0%
56.271 MGD GAC System	\$27,557,434	\$1,085,355/ 3.9%
0.500 to 5.809 MGD IX Systems	\$367,110 - \$3,920,210	\$0 / 0%
56.271 MGD IX System	\$39,765,958	\$2,303,916 / 5.8%
0.500 – 5.809 MGD RO Systems	\$1,804,014 - \$7,613,306	\$0 / 0%
56.271 MGD RO System	\$36,366,653	\$0 / 0%
0.500 – 1.000 MGD Interconnections	\$463,316 - \$479,635	\$0 / 0%
3.536 MGD Interconnection	\$2,006,014	\$56,535/ 2.8%
0.500 – 1.000 MGD New Wells	\$266,255 - \$959,977	\$0 / 0%
3.536 MGD New Well	\$3,040,861	\$85,650 / 2.8%

Therefore, the minimal level of contingency, or lack thereof, in the WBS cost estimates wildly overestimates the WBS' ability to capture system-specific water quality, site conditions, community needs, and the overall cost factors for the new treatment facility. The EPA should adjust this approach and ensure that the appropriate levels of contingency are included to ensure that cost estimates are consistent with the level of project definition afforded by the available data in keeping with sound engineering practice.

Accurately Reflecting Current Economic Conditions

Another limitation of the EPA's WBS Model is that it reflects outdated 2021 construction costs. Additionally, the model relies on a variety of cost indices to scale the costs from a previous year to the relevant year of the analysis. It is reasonable to scale data from one year to another year using indices, but EPA fails to account for the fact that there is always a lag in the data for the most recent periods of time. The agency must recognize that the past two years, from 2021, have shown significant cost increases relevant to drinking water treatment systems.

These increases in costs have stemmed from the COVID-19 pandemic, high inflation, and increasing interest rates for borrowing. Construction costs, for example, have increased steadily by more than 15% to 30% in the past 2 years according to several different sources (Mortenson, 2023; Turner Constructon, 2023; USBR, 2023). By comparison, construction costs during 2020 only increased by less than 1.5%. A similar trend can be observed in analysis of inflation since 2021, which has averaged 5.81% annually. The federal funds rate has also increased from 0.08% to 5.25%, which will also drive up the costs for water

systems to secure financing for new projects. EPA's analysis must reflect these increases to properly account for the costs of implementation.

In addition to the increase in costs driven by economic conditions since 2021, it is also important to note that the proposal itself will further increase the costs; 67,000 systems conducting monitoring and upwards of 4,300 water systems installing treatment facility will increase demand for laboratories, engineering consultants, planners, contractors performing site investigation and construction work, and skilled treatment operators.

In order to comply with its statutory obligations, including under the SDWA²⁵, EPA should ensure that the cost analysis of any rule is accurately reflecting costs due to economic conditions and anticipated increases in demand that will drive the planning and construction costs of new facilities significantly higher than the current estimates.

Recognizing the Importance of Ancillary Systems

Another potential limitation of the EPA's WBS Model is that it only considers capital upgrades for PFAS removal from water. This is a significant analytical gap because many systems will likely need to make improvements to other areas of the treatment facility to support the PFAS treatment process. For example, some systems installing GAC treatment may determine that the concentration and form of manganese will cause problems in the vessel, requiring pre-treatment. A variety of other water quality characteristics may impact the need for pre-treatment and site-specific conditions may drive the need for significant upgrades to critical treatment support systems (e.g., pump stations, chemical feed systems, etc.). It is not uncommon for upgrades for PFAS treatment to require these types of improvements, none of which EPA's WBS Model takes into account.

Lifespan of Treatment Equipment

The proposal provides inconsistent information related to the total number of years that are used for the annualization of costs. In the Economic Analysis, EPA notes that both costs and benefits are annualized over 82 years. Alternatively, the example outputs for each treatment system in the Technologies and Costs document lists useful life for each piece of equipment that is included in the capital costs. In the same document a system-specific useful life is listed as part of the cost equations. The supporting documentation from the EPA does not provide a clear explanation of how costs are annualized. Additionally, the useful life varies for equipment from as low as 7 years to as long as 35 years; it is unclear from the supporting documentation the methodology EPA used to substantiate these assumptions. This approach is not consistent with previous practice; the agency's approach to annualizing costs under the Arsenic Rule was based on a 20-year useful life for equipment (EPA, 20XX). In order to fulfill its obligations under the APA, AWWA recommends that EPA provide this information during a supplemental comment period prior to finalizing any PFAS rule. Failing to do so or failing to acknowledge this change in the assumption for treatment facility lifespan and providing a reasoned explanation for the change, would violate the APA.

²⁵ 42 U.S.C. 300g-1(b)(B)(4)(D).

Interconnections

EPA anticipates that some water systems may take non-treatment actions to respond to PFAS levels above the MCLs, such as the installation of an interconnection. Overall, AWWA agrees that some systems could potentially decide to install an interconnection if it is a viable alternative. Regionalization may have benefits for consecutive systems and can help provide smaller systems with access to economies of scale. Alternatively, regionalization can have unintended consequences on the water quality for the consecutive system, such as elevated water age, nitrification, and DBPs. For this reason, systems considering interconnections will need to thoroughly investigate this option and determine if it is both cost effective and appropriate given the water quality impacts.

For the analysis, EPA estimates that upwards of 7% of small systems will install an interconnection to comply with the PFAS MCLs, it is unclear from the supporting information how this assumption was made, and EPA should provide additional information. The potential use of an interconnection to comply with the proposed rule has not previously been included as part of a drinking water rule's compliance analysis and the EPA's approach poses significant issues that exclude significant cost factors. AWWA is providing the following recommendations for necessary considerations for installing interconnections.

- *Compatibility of Secondary Disinfectants:* The use of disinfectants for maintaining a residual in the distribution system varies by system and not all systems use the same disinfectant chemical to maintain distribution system residuals (if they are required to). The selection of a disinfectant for maintaining a residual is not regionally uniform and varies on a number of other factors (e.g., water source, finished water quality, distribution system size, water supply capacity, etc.). Subsequently, there is a significant likelihood that a purchasing system may be using a different disinfectant than a supplying water system, which would require the installation of a facility that can convert free chlorine to chloramine, or vice versa. Some systems may need pH adjustment as well. EPA's cost analysis for interconnections does not consider this. If needed, disinfectant conversion facilities require substantially more upgrades than the EPA's WBS for interconnection considers. Costs for these facilities will need to include, at a minimum:
 - Land purchasing,
 - Building construction,
 - Chemical feed pumps, storage tanks, and spill containment,
 - Mixing and storage tanks, and
 - Water quality monitoring devices.
- *Simultaneous Compliance with LCRR:* LCRR became effective in 2021 and water system compliance with all provisions of the current LCRR will be required in October 2024. Water systems are required to fully evaluate and ensure adequate corrosion control when adding or changing sources of water. Specifically, LCRR expands on existing requirements to include this assessment when adding new sources as previously described. The challenges posed by LCRR will impact the number of systems for which purchased water from wholesale supplier is a viable near-term option. Furthermore, if both systems are using CCT, the compatibility of each CCT must be considered.

- *Simultaneous Compliance with Microbial and Disinfection Byproduct Rules:* As noted previously, regionalization can provide benefits but can also have negative impacts on water quality particularly because of increased water age. For systems that install an interconnection to a consecutive system, a thorough investigation will be needed to determine if water age will be an issue and whether DBPs may need to be addressed at the to prevent MCL violations. This could potentially require systems installing interconnections to install GAC filters and/or transition to a different disinfectant residual. Similarly, systems may determine that inadequate disinfectant residual is present in the water to support the longer water age and so a boosting station is required.
- *Pressure Differences:* In Table 5-15 of the Economic Analysis, the EPA notes that booster pumps and/or pressure reducing valves are included as direct capital costs by the WBS cost model. The agency later notes, however, that to generate cost equations for interconnections the agency has assumed a minimal pressure difference between each water system so that neither booster pumps nor pressure reducing valves are needed. AWWA understands that it may be impossible for the agency to surmise the average pressure difference between two water systems, however it is nonetheless unrealistic to assume that booster pumps are unlikely to be necessary. Pressure loss associated with friction could be significant, especially for an interconnection that may span 10,000 feet or more. For an interconnection of this distance, the pressure loss associated with water flow through an appropriately sized pipe (to maintain water velocity from 5 to 7 feet per second), would be approximately 50 psi. The inclusion of purchasing booster pumps, at minimum, should be included as part of this analysis.
- *Unit Cost of Purchased Water:* According to the proposal, an assumed average cost of purchased water is \$3.00 per thousand gallons (2021\$) based on wholesale rates that were available online. Currently there are 3,258 water systems in SDWIS categorized as wholesaler systems. These systems range in service population size from 25 to up to 2.5 million persons, which represents a significant range in their economy of scale. The WBS for non-treatment options and the documentation that is provided does not clearly illustrate what data were considered to estimate the national average cost of purchased water and whether those data are nationally representative. It is possible that if the available data may be only from cities with a water supply that is relatively inexpensive to treat and supply to purchasers. Transparency on this data is necessary to ensure that this unit cost is accurate and reflective of the national perspective. EPA should therefore provide the underlying analysis and an explanation for the model provided in order to allow for a meaningful opportunity for public comment.

To further illustrate this, a report by the Department of Energy from 2017 assessed water rates nationally and estimated that in 2016 the average water rate was \$3.38 per thousand gallons (DOE, 2017). More recent data from Circle of Blue similarly analyzed water rates nationally and estimated that average national water rate in 2019 to be \$6.22 per thousand gallons (Circle of Blue, 2019). These national data points highlight a stark difference between the EPA's data and highlight that water rates have increased substantially as water supplies have become more severely impacted by drought and water quality challenges. Additionally, it is important to note that both reports do not reflect water system cost increases related to LCRR, the economic effects of the COVID-19 pandemic (e.g., increased price of chemicals, materials, and labor).

These cost increases have been previously described in this letter. EPA should clearly communicate the sources of the wholesale water rate data so that additional supporting data can be provided to improve EPA's analysis.

- *Available Capacity Without Improvements:* Finally, the EPA's approach makes a blanket assumption that these water systems will be able to identify a supplier water system that has existing available capacity to provide finished drinking water with PFAS levels below the MCL without needing to install any treatment. It is highly likely that a supplier will need to install additional drinking water treatment systems to accommodate the purchasing water system's water supply capacity. This is an especially important consideration for regions of the U.S. where drought is creating water supply challenges already as well as areas where source waters are becoming increasingly challenging with respect to accessibility and ease of treatment. On top of this challenge, there is a significant possibility that the PFAS contamination impacting a purchasing water system is also impacting the supplying water system, especially given the low levels of concern identified by the proposal. In this case, the system providing water supply would need to install PFAS treatment capacity for their current water supply capacity in addition to the purchasing water system's demands.

Finally, in review of the EPA's example outputs for interconnections, the projected costs for a 1 MGD interconnection with mid-cost components were estimated to be less than the projected costs for a 0.5 MGD interconnection with low-cost components. It's unclear whether the model requires correction. Nonetheless, EPA is encouraged to review the model and subsequent cost analysis to ensure that this and other potential errors are addressed prior to using this analysis to support any final rule.

Development of New Wells

EPA also estimates that some water systems will develop new wells instead of installing treatment. The development of new wells also relies on assumed conditions that may make the development of new wells to be more cost effective than treatment. One key assumption is that the PFAS contamination impacting the water system's current groundwater source is not impacting another local source where a new well can be constructed. This is a flawed assumption and likely overestimates the number of water systems for which this is a viable option.

This option also appears to be underestimating the costs for performing this task. In review of the example cost model outputs for a new well of 0.5 MGD, several aspects of the cost estimate are significantly low. A recent budgetary estimate for a water system in Pennsylvania for a new well with a capacity of 0.144 MGD is approximately \$1.5 million, which does not include planning and design services totaling another \$532,000 (Horsham, 2023a).

The referenced budgetary estimate was compared with EPA's model output for the development of a new 0.5 MGD well. Several aspects of the project are substantially low compared with the referenced estimate. Construction management, for example, is estimated to be less than \$16,000 by EPA's WBS whereas the construction management services for this recently developed well will exceed \$175,000. The overall cost of this well will exceed EPA's estimate by a factor of 5 for a well that has less than a third of the capacity. Another water system in Washington submitted comments to the EPA similarly illustrating that these costs are underestimated by a factor of 4 (LWD, 2023).

In order to provide an accurate assessment of costs, EPA must therefore re-evaluate the WBS for new wells and address errors in its estimates prior to using its new well costing analysis to support any final rule.

Social Costs of Carbon Dioxide

To comply with this rule, most water systems with PFAS exceeding the MCL(s) will need to install drinking water treatment facilities that rely on either GAC, IX, or RO. In many cases, it is likely that this will create a new hydraulic profile for the water system which requires additional facility pumping and consequently electricity demand. The use of GAC and IX also requires disposal of spent material involving transport of that material via train or truck to an appropriate facility. These activities can be reasonably anticipated to have a significant impact on the carbon footprint of water systems nationally.

EPA is currently heavily involved in addressing challenges with climate change and in advancing sectors of the U.S. economy towards reducing greenhouse gas emissions. Section 5 of President Biden's Executive Order 13990, notes that it is "essential that agencies capture the full costs of greenhouse gas emissions as accurately as possible, including by taking global damages into account" and that doing so "facilitates sound decision-making" (Biden, 2021).

The proposal lacks an analysis of the social costs of carbon. AWWA recommends that the agency consider the social costs of carbon as part of any PFAS rule's cost analysis to be comprehensive as well as to understand how this rule may have unintended consequences like increased social costs relating to carbon dioxide emissions.

In considering the social costs of carbon, the agency is encouraged to review a recent report by Policy Navigation Group (PNG, 2023). The current estimate for the social cost of carbon dioxide emissions varies, but a low estimate range is \$130 to \$190 per ton based on a recent EPA report (EPA, 2022h). In the Policy Navigation Report, they estimate the social costs of carbon using data from the EPA's current economic analysis and using available EPA guidance for estimating such costs. Policy Navigation Group estimates carbon emissions related to additional pumping, lighting, and ventilation associated with the PFAS proposed rule and concludes that the potential national social costs of the carbon emissions are \$5 million annually. The \$5 million would be in addition to the social costs associated with replacement of GAC and IX media as breakthrough occurs. Given that more than 4,300 water systems will rely on GAC and IX treatment for PFAS and will begin generating tens of thousands, if not hundreds of thousands of tons of spent GAC and IX resin it is important that the associated social costs are considered.

EPA estimated the total GAC and IX waste generation annually to perform a sensitivity analysis for managing these materials as hazardous wastes, but the estimated amount of waste generated is not reported. EPA estimated that water systems will need to install treatment for more than 64.8 million people to comply with the proposed rule; this will amount to more than 3 trillion gallons that will need to be treated annually. Calgon Carbon estimated one water treatment plant's GAC usage rate for PFAS treatment was as high as 0.07 pounds GAC per 1000 gallons (Calgon, 2023). Based on these figures, the annual demand for GAC could exceed 100,000 tons, which potentially has a carbon dioxide footprint of 850,000 tons (He, 2012). This could have a social cost of more than \$160 million annually.

With such a significant potential impact on society, EPA should conduct the same analysis to determine the social costs of carbon associated with each of the treatment technologies and the rule options. This

analysis should also be included as a matter of maintaining consistency across the agency's rulemaking processes. In two recent rulemaking, EPA estimated the social costs of the rule in recognition that changes to the operation of complex treatment systems can provide both benefits and unintended consequences (EPA, 2023f; EPA 2023g).

Shifting Landscape of Residual Management Practices

During the stakeholder engagement in advance of publishing the proposal, EPA was encouraged to consider the impacts of new regulatory actions that would impact disposal of GAC, IX, and RO waste streams with PFAS. In September 2022, EPA proposed to designate PFOA and PFOS as hazardous substances under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA); a broader action was proposed for additional PFAS in April 2023 (EPA, 2022i; EPA, 2023h). Separately, EPA is also preparing to list PFOA, PFOS, PFBS, and HFPO-DA as hazardous constituents (EPA, 2022c).

EPA considers these actions as part of the economic analysis, particularly through a sensitivity analysis. As noted above, the quantity of GAC and IX that EPA estimates will be disposed of annually is not reported in the supporting information. As noted above, the demand for GAC may exceed 100,000 tons annually. EPA has recently estimated that the costs to incinerate hazardous waste ranges from \$400 to \$1,700 per ton depending on the type of waste (EPA, 2020). At a minimum, the annual costs to incinerate GAC and IX as hazardous waste will exceed \$40 million and are likely to be beyond \$100 million. Just the costs of incineration significantly exceeds the \$30 million estimate, which EPA describes as including hazard waste disposal and associated costs (e.g., transportation and handling).

While EPA notes that hazard waste disposal costs are excluded from the estimate of annual costs, these costs should not be ignored. EPA's current commitment is to finalize the PFOA and PFOS hazardous substance designations under CERCLA this summer. Furthermore, as these actions are advanced, waste management practices are shifting and leading to increased disposal prices for water systems and, in some cases, facilities are refusing to accept water treatment residuals (AWWA, 2022).

AWWA recommends that the EPA (i) more closely look at the costs associated with hazardous waste disposal, including available guidance from Office of Land and Emergency Management (OLEM) and (ii) include the cost of hazard waste disposal of spent media. The current EPA regulatory agenda will drive impacts the cost of treatment options for removing PFAS from drinking water and will be legally binding by the time water systems must comply with any PFAS rule.

Importance of an Accurate Cost Analysis

The EPA is encouraged to review the cost analysis, and the EPA's WBS Model, to ensure that the monitoring and treatment costs are accurate. As noted above, EPA cannot fulfill its obligations under the SDWA unless the cost assessment is accurate. In addition, this cost analysis is critical given that it underpins the HRRCA as well as the household affordability analysis. An inaccurate estimation of costs will mischaracterize the proposal's regulatory impact, merits, and the affordability for households in smaller communities. These analyses are critical for the agency, and water systems, to understand public health priorities. Water systems are currently working to address various public health priorities, including lead service lines, cybersecurity, microbials and DBPs, water supply challenges (such as drought, increasingly impaired water sources), and aging infrastructure.

Black & Veatch estimates that the cost of rule will range from more than \$2.5 to \$3.2 billion annually (Black & Veatch, 2023 – See Appendix B). As proposed, this rule will be one of the most costly rules under SDWA ever proposed and will be a burden carried by less than 10% of water systems. It is imperative that the overall analysis of the proposal's impacts be accurate so that water systems and communities are assured that the investments in infrastructure being made represent the investments with the greatest societal benefit.

8. Health Risk Reduction Analysis

As required by SDWA, EPA has prepared a health risk reduction analysis for each of the proposal's regulatory options. AWWA contracted with Ramboll Consulting U.S. to assist in reviewing the EPA's approach and to offer detailed recommendations to improve this important work. Ramboll has provided a detailed letter with recommendations, which is included in these comments in Appendix A.

AWWA requests that EPA update its health risk reduction analysis in light of Ramboll's report and the recommendations it contains so as to ensure that the agency is relying on the best available public health information in reaching any regulatory decisions. MCLs are appropriately set at a level where the benefits justify the costs, and without a reliable assessment of both the costs and benefits of the proposal, EPA cannot do so. Based on the information provided in the proposal, the benefits do not justify the costs at the proposed MCL levels and particularly do not justify the costs under EPA's proposed compliance timeline.

Estimating Reductions in Cardiovascular Disease Risks

According to the proposal, increased PFOA and PFOS serum concentrations may lead to an increased risk of cardiovascular disease (CVD), including myocardial infarctions and strokes. To support this analysis EPA relies on an assumption that there is a causal relationship between PFAS exposure and CVD. In particular, this analysis uses exposure-response functions from a meta-analysis that was conducted using data from several epidemiological studies on the general population. In review of this approach, several critical issues have been raised:

- While EPA relies on epidemiological studies of participants from the National Health and Nutrition Examination Surveys (NHANES) to support its meta-analysis of PFAS exposure and CVD risk. These studies did not observe increased risks of CVD in the participants of these studies, including those participants with highest exposures yet EPA characterizes the results of these studies as 'inconsistent'.
- The analysis of CVD risk projects changes to total cholesterol and blood pressure based on PFOA and PFOS exposure but excludes changes to high density lipid cholesterol, which has also been observed. A sensitivity analysis of a hypothetical exposure reduction of 1 ppt PFOA and PFOS, which included the effects of changes to HDL-C, found that annualized CVD risk reduction benefits were decreased by 23 to 25%. By comparison, when changes to blood pressure were excluded the reduction in annualized CVD risk reduction benefits only decreased by 1.8 to 2.3%. This suggests that the benefits analysis, which excludes the impact of PFOA and PFOS on HDL-C, is significantly overestimated.
- Throughout the supporting documentation, the EPA makes contradictory statements about the strength of associations of PFOA and PFOS on blood pressure and HDL-C. Furthermore, the

inclusion of blood pressure impacts from PFOA and PFOS exposure but not the inclusion of changes to HDL-C is unclear, especially given that these impacts were observed equivocally.

- The biological mechanism for the association of PFOA and PFOS with cholesterol is not yet identified in humans. In fact, recent work demonstrates that a lifestyle intervention on cholesterol led to a decrease in both the cholesterol and serum PFOA and PFOS concentrations.
- Finally, given the recent downward trend in decreasing total and low-density lipid cholesterol since the 1970s coupled with the decreasing PFOA and PFOS serum levels suggests that there is a substantial likelihood that the proposed MCLs for PFOA and PFOS are unlikely to result in benefits as great as is reported as part of this proposal, given the outsized impact from other risk factors.

Additional details on these and other comments for the EPA's analysis of cardiovascular disease risk reduction are available in Appendix A. Based on the materials made available, it does not appear that EPA's conclusion is fully supported by the evidence or record before the agency.

Estimating the Reduced Impact of Low Birth Weights

In review of the EPA's approach to estimating the benefits of reducing the incidence of low birth weights resulting from prenatal PFOA and PFOS exposure, it appears that EPA is conflating risk of low birth weight with differences in mean birth weight. In review of this approach, several critical issues have been raised:

- The use of low birth weight as the critical effect is inconsistent with other regulatory agencies that found small decreases in birth weight but not increased risk of low birth weight in relation to PFOA and PFOS (ATSDR, 2021; EFSA, 2020; EFSA, 2018).
- In deriving the Reference Dose for both PFOA and PFOS, EPA has noted that the derivations for PFOA and PFOS were based on low birth weight (defined as birth weight less than 2,500 grams). However, the exposure-response coefficients used for these efforts were based on decreases in birth weight. In particular, the studies used for these derivations evaluated differences in average birth weight but not risk of low birth weight. EPA's conflation of low birth weight with decreases in birth weight is prevalent in the economic analysis as well. While these endpoints are correlated, they are not equivalent and the should not be evaluated as if they are the same.
- The benefits analysis relies on exposure-response functions based on coefficients for decreases in birth weight from the main analysis of two different meta-analyses for PFOA and PFOS. The critical study that serves as the basis for the PFOA meta-analysis (Steenland et al, 2018) concluded that there was no effect on birth weight when the results of the C8 Science Study were included and noted that the results were consistent with confounding and/or reverse causality. EPA's conclusion is at odds with Steenland et al's own conclusion for their work.
- EPA's characterization of the supporting studies for this health effect is inconsistent with the data that is provided by the studies. In the toxicity assessment for PFOA, EPA concludes that the majority of the studies considered showed supportive evidence of an increased risk of low birth weight with increasing PFOA exposures. However, closer inspection of the studies showed conflicting results. For example, several studies that stratified results by sex provided mixed

results on gender-specific impacts. Similarly mixed results were noted for studies that stratified results by country of birth. Furthermore, exposure-response results were not consistent with results from studies where exposures were measured in the general population; in particular, these studies generally reported no associations or very limited evidence of associations of PFOA and PFOS with low birth weight.

- While the SAB requested that USEPA reevaluate and consider studies published before the 2016 Health Effects Support Documents for PFOA and PFOS, at least seven studies that were available prior to 2017 were not included (Darrow et al. 2013; Nolan et al. 2009; Savitz et al. 2012a, 2012b; Stein et al. 2009; Chen et al. 2012; Wu et al. 2012). None of these studies found an increased risk of low birth weight in relation to PFOA or PFOS. EPA must include studies that do not support its preferred outcome in its analysis and explain why, in light of these studies, it has still reached its determination.
- The EPA also failed to acknowledge the significance of the results from the sensitivity analysis assessing bias associated with timing of the maternal sampling during late pregnancy versus sampling during early pregnancy. This sensitivity analysis highlighted that there was no effect on birth weight when maternal blood was sampled early in pregnancy whereas late pregnancy sampling showed a larger effect on birth weight than the meta-analysis. This suggests that the evidence for a conclusion that PFAS exposure is associated with decreased birth weight is inconsistent after considering potential confounding.

Additional details on these and other comments for the EPA's analysis of decreased birth weights are available in Appendix A. Based on the materials made available, it does not appear that EPA's conclusion is supported by the evidence or record before the agency.

9. Monitoring Requirements

The agency is providing a compliance timeline of three years for water systems subject to the rule to perform initial monitoring requirements for PFAS at each entry point to the distribution system. The initial monitoring requirements may be waived for some systems that are either participating in UCMR 5 or have participated in eligible state monitoring programs since January 2019. Initial monitoring will determine if systems are eligible for a reduced monitoring frequency under the proposed framework and if the system will need to install treatment (or take non-treatment action) to reduce PFAS to levels below the MCL. According to the proposal, compliance with the proposed rule will be determined using the RAA at each entry point to the distribution system. As part of the calculation of the RAA, EPA is also proposing that systems use 0 ppt for results that are below the practical quantification limit (PQL) of 4.0 ppt. At the same time, EPA is proposing that to calculate the RAA for determining a system's eligibility for reduced monitoring, that only values below the detection limit be considered as 0 ppt and all reported results above the detection limit be used. The following sections provide a detailed review of these requirements and their proposed alternatives.

Initial Monitoring: Use of Existing Data and Timeline

AWWA appreciates the agency's interest in reducing public water system monitoring burdens, especially where existing monitoring data exists. The agency's proposed approach to accept data collected since January 1, 2019 is appropriate. Reduced regulatory monitoring demands will be a welcome relief for

water systems already working to understand PFAS levels in their water, but it is uncertain what magnitude of impact this will have on water systems.

It is difficult to estimate the magnitude of the reduced monitoring impact because data will be (i) dependent on fully fulfilling the initial monitoring requirements and the quality control that they require and (ii) subject to state primacy agency approval. For example, many states may have data for PFOA, PFOS, PFHxS, and PFNA but not PFBS and HFPO-DA. In other cases, water systems may not have received results for PFAS below the proposed PQL. Another challenge for systems to use previously collected data, such as data from UCMR 5, is that the results may not have been reported at the necessary level to be used for EPA's monitoring and will likely not be feasible for all water systems to acquire data below the PQL.

Additionally, the timeline for the initial monitoring period of three years is appropriate. It is important to note, however, that this timeline is in concert with the compliance deadline for meeting the MCLs. As such, water systems will not be able to wait until the third year of the compliance window to perform initial monitoring requirements because this would not leave time to comply with the MCLs, if elevated levels of PFAS are found.

Use of Standard Monitoring Framework

The proposed approach to use the SMF as a basis for triggering reduced monitoring is appropriate. In review of the proposal, however, AWWA has significant concerns surrounding the proposed approach to require water systems to consider low quality, unreliable analytical results below the PQL. Specifically, the EPA is proposing that 1.3 ppt PFOA and PFOS and one-third of the hazard index be used as a trigger level for reporting for the purposes of determining reduced monitoring eligibility under the SMF. The EPA also requested input on the use of 2.0 and one-half the hazard index.

AWWA recommends against the use of any data below the PQL to drive regulatory requirements. Data should likewise not be used to determine nor treatment, or more frequent monitoring. For the reasons detailed below, use of this below-PWL data would be arbitrary and capricious.

The PQLs for PFOA and PFOS are set at 4.0 ppt each in the proposal, which is consistent with the currently active UCMR 5 monitoring program's minimum reporting limits. In finalizing the UCMR 5 monitoring program in 2021, EPA recognized that while EPA Methods 533 and 537.1 can both be used by laboratories to achieve lower reporting limits but concluded that the available lab capacity could not support establishing lower reporting limits to collect national occurrence data. Data below this level is less accurate and is not achievable by all water systems.

As a matter of policy, EPA should not set a precedent for the use of analytical results that are not reliably achievable for all water systems as this would create an equity issue. Moreover, the current minimum reporting levels for EPA Methods 533 and 537.1 are appropriate based on ongoing experience with PFAS analytical results.

As PFAS occurrence data are required to be collected and used to drive decision-making, there will be higher demand for laboratories that are able to analyze samples with a greater degree of reliability at single digit, part per trillion levels to minimize the risks of inaccurately higher sample results from interferences and other technical challenges. Increased demand for better laboratories will contribute to higher lead times, per sample costs, and more frequent recognition of sample analysis errors. The

consequences of these pressures on the analytical services market, will most negatively impact smaller systems with less financial capacity to access more experienced and better performing laboratories. To the degree small systems have limited access to high quality laboratory service, it creates inequitable access to reliable sample analysis.

Beyond the technical challenges of this aspect of the proposed monitoring requirements, there are challenges with the risk communication of associated results it would produce. As proposed, EPA would require that PFAS monitoring levels be described in two different ways. Risk communication will be especially challenging for water systems with observed values below the PQL, as they will have a reporting PFAS values based on the RAA for MCL compliance and a separate value for reduced monitoring eligibility. It is unclear if EPA has considered how these data would be reported and communicated to the public in a meaningful manner.

Analytical results below the PQL should not be used, rather the rule requirements should use 0 ppt for all analytical results below the PQL. Consistent with previous AWWA comments, AWWA recommends that one-half the MCL be used to determine if PFAS levels at an entry point are reliably below the MCL.

10. Public Notifications

Public notifications serve an important role in protecting public trust in drinking water and their usefulness for communities relies on a solid foundation for risk communication. The proposal requires that water systems with PFAS levels exceeding the MCLs must provide a Tier 2 notification to the public and Tier 3 notifications when a monitoring and/or testing procedure violation has occurred. Water systems will also be required to include information about detections of regulated PFAS in the Consumer Confidence Report.

AWWA supports the proposed approach for public notifications for PFOA and PFOS. While AWWA is recommending in this letter that the proposed regulation for PFHxS, PFNA, HFPO-DA, and PFBS be revised based on the discussed technical and legal issues, the agency is reminded that the risk communication for PFAS must be carefully and thoughtfully structured. The role of public communications is to provide useful information to the public about their drinking water. As EPA considers any rule for PFAS through a hazard index, it is important that regulations be structured in a manner that facilitates useful risk communication. EPA's proposed use of the general hazard index combining risks across multiple health outcomes prevents water systems from having an effective risk communication strategy. If EPA moves forward with any rule using the hazard index, risk communication should be considered more carefully.

11. Household Affordability and Small System Compliance Technologies

As noted in the first section, it is critical that drinking water be affordable and that smaller systems more susceptible to affordability challenges have access to compliance technologies. This is true for all consumers, and particularly for those in environmental justice communities. The proposal highlights several variations of the household affordability analysis beyond the EPA's previously utilized approach, including an approach that has been developed and recommended by AWWA and other water sector associations (AWWA, 2021b). AWWA appreciates that the agency is interested in utilizing recommendations previously made by stakeholders regarding alternative metrics for this analysis.

These comments have already highlighted significant concerns about EPA’s underlying approach to the cost analysis and the anticipated inaccuracies of the EPA’s WBS Model. EPA should refine that approach and re-evaluate the affordability analysis for any rule.

As part of its analysis, Black & Veatch assessed household impacts of the various rule options (Black & Veatch – See Appendix B). Table 11-1 provides an overview of those results for Options 1a (4 ppt PFOA and 4 ppt PFOS) and 1c (10 ppt PFOA and 10 ppt PFOS) in comparison with EPA’s estimated expenditure margins for the affordability analysis. As shown in Table 10-1, the household costs for each of these options significantly exceed the expenditure margin for systems serving less than 1,100 persons.

Table 11-1: Comparison of EPA Affordability Margin and Treatment Cost Estimates

Population Range	Household Costs Estimated by BV Model	EPA Estimated Expenditure Margins
25 to 100	\$3,570	\$877
101 to 500	\$1,675 - \$1,750	\$877
501 to 1,100	\$1,360 - \$1,390	\$753
1,101 to 3,300	\$575 - \$640	\$753
3,301-10,000	\$305 - \$327	\$855

It is also important to note that these household costs are only reflective of treatment costs, monitoring costs are not included here, which for smaller systems will have a greater household impact. A treatment technology that is not considered as part of this analysis is the use of point-of-use reverse osmosis (POU RO) systems. While POU RO may become available in the future following NSF/ANSI certification standard that is based on achieving levels at or below the proposed MCLs, it is currently not a compliance option. AWWA agrees with the agency’s decision to not include POU devices in its analysis of rule compliance affordability for small systems. Certification is currently not available, and demonstration of effectiveness is a critical aspect of including compliance technologies in this analysis. NSF/ANSI certification will be necessary if any rule considers POU RO as a small system compliance technology.

EPA should re-consider the proposal as the small system household costs for centralized water treatment exceed EPA’s estimated expenditure margins for these systems, a more affordable POU treatment option is not available, and EPA has not identified a small system variance technology.

Accounting for Financial Assistance

In its affordability analysis, EPA also cites that an additional analysis was conducted accounting for funds that are nationally available, such as the DWSRF program and funds from BIL. As EPA notes in the proposal, \$800 million is available annually for systems addressing emerging contaminants like PFAS. EPA also announced the availability of \$1 billion annually through the Emerging Contaminants in Small or Disadvantaged Communities grant program. Both programs are appropriated through Fiscal Year 2026. These programs make \$1.8 billion available for fiscal years 2024, 2025, and 2026 (a total of \$5.4 billion).

The EPA estimates that the total capital cost needs for small systems will range from 1.1 to 2.5 billion; however, as previously noted the cost analysis used by the EPA is significantly flawed and

underestimates financial impacts on communities. The occurrence analysis also presents several issues in characterizing the total small system impacts. For a more accurate comparison to available funding, and therefore potential offsetting of household costs, data from Black & Veatch was considered. Black & Veatch estimates that the total capital cost for small systems for a 4 ppt PFOA and 4 ppt PFOS rule will exceed \$21.6 billion based on the occurrence data collected by Corona Environmental (Black & Veatch, 2023; Corona, 2021). This is a stark difference from EPA's estimate of \$1.1 to \$2.5 billion total capital cost for small systems.

Even if the occurrence analysis from the EPA is used these estimates are substantially low. Policy Navigation Group estimated the number of systems that would potentially exceed the MCLs using data from the EPA (PNG, 2023). Using these figures and the estimated capital costs for each system size from Black & Veatch, the total capital cost exceeds \$10 billion. This is approximately more than the EPA's estimate by a factor of four.

The availability of \$5.4 billion for these systems will help alleviate the costs for individual households. That impact, however, is limited to systems that receive financial assistance. Unless EPA plans to work with states to develop a method for distributing these funds equally to all impacted water systems, the financial assistance will not be evenly distributed across small systems. This approach will inaccurately depict the financial impacts to households in communities where financial assistance is not provided. Therefore, AWWA recommends that the EPA not consider this financial assistance in assessing household affordability for small systems.

12. Executive Order 12898 – Achieving Environmental Justice

AWWA and our members have first-hand knowledge and experience of how the increased costs associated with new regulations such as the ones proposed here directly impact the customers of our water system members. Many water systems are small, public, or quasi-public entities. Increased compliance costs are necessarily passed on to customers in the form of high rates for their drinking water. As a result, unjustified compliance costs have a disproportionate impact on economically disadvantaged customers as they are least able to afford these rate increases. This disproportionate impact is particularly acute with respect to water infrastructure for several reasons. First, water systems serve local customers, they do not have the ability to spread costs out across a national customer-base. Second, because household water use is a necessity and most households cannot meaningfully scale back on their needs for drinking water when prices increase. Consequently, they are unable to take steps to reduce their water bills when water systems are forced to increase rates.

Congress amended the SDWA in 1996, recognizing that the Act's prior requirements, and associated economic burdens on water systems and the States were making the SDWA unworkable.²⁶ As a member of Congress explained at the time, "[c]ustomers will pay for safe drinking water . . . [b]ut are not willing to pay for complying with drinking water rules that provide only marginal increases in health protection

²⁶ S. Rep. No. 104-169 at 2, 11, 17 (1995); H.R. Rep. No. 104-632 at 9 (1996); *see also* S. Rep. No. 104-169 at 12–13 (noting that the prior version of the Act was the "quintessential example of an arbitrary Federal law imposing burdens on consumers and the taxpayers of other governments with no rational relationship to the public benefits that might be realized.").

at significant costs, particularly when there is so much uncertainty concerning both the occurrence and real threat to public health of many contaminants.”²⁷

EPA’s current analysis also fails to consider how these increased compliance costs will impact environmental justice communities, as required by Executive Order 12898. As you know, Executive Order 12898 directs each Federal agency to “make achieving environmental justice part of its mission by identifying and addressing, as appropriate, disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority populations and low-income populations.” EPA should revise its environmental justice analysis to reflect the burdens that the compliance costs associated with the new proposed requirements would place on environmental justice communities and further consider whether these additional burdens are appropriate in light of these impacts.

AWWA further notes that prematurely issuing national primary drinking water regulations for contaminants when the occurrence data indicates that it only occurs in drinking water at levels of public health concern in localized areas would cause communities, rather than those responsible for the pollution, to foot the bill for the problem. To avoid this inequitable result EPA should focus on using its other authorities to address any necessary clean ups, rather than pass the costs on in the form of higher rates due to increased SDWA compliance costs.

13. Alternative Regulatory Options for Drinking Water Standards

The proposal provides a detailed overview of the four regulatory alternatives that were considered by the Administrator to support this rulemaking. These options are laid out both in the preamble of the proposal and the economic analysis. The economic analysis frames both the costs and benefits of the proposed option and its alternatives. The proposed option is 4 ppt PFOA, 4 ppt PFOS, and a hazard index for PFHxS, PFNA, HFPO-DA, and PFBS of 1.0. Option 1a, 1b, and 1c propose MCLs for PFOA and PFOS each at 4 ppt, 5 ppt, and 10 ppt, respectively. Figures 13-1 and 13-2 depict the results of EPA’s analysis based on both discount rates of 3% and 7%. Presentation of both discount rates to inform decision-making is consistent with Office of Management and Budget guidance for regulatory analyses.

A striking observation from both figures is the difference in annualized costs for the Proposed Option and Options 1a and 1b compared with Option 1c. Under both discount rates, the cost to implement the rule doubles from Option 1c to 1b. This is reflective of the significant number of systems that would be required to install PFAS treatment facilities to mitigate PFAS to comply with rule Option 1b compared to Option 1c. This difference is expected to impact systems that have PFAS levels ranging from 4 to 8 ppt, as EPA assumes that only systems within 80% of the MCL will make costly infrastructure investments.

These figures demonstrate that, regardless of the discount rate, there are minimal incremental benefits with the addition of the hazard index MCL for PFHxS, PFNA, PFBS, and HFPO-DA. This is substantiated by the available occurrence data, which demonstrates that very few systems would be required to install treatment for the PFHxS, PFNA, PFBS, and HFPO-DA MCL but not the PFOA and PFOS MCL.

²⁷ H.R. Rep. No. 104-632 at 9 (quoting Ronald Dungan, President of the National Association of Water Companies).

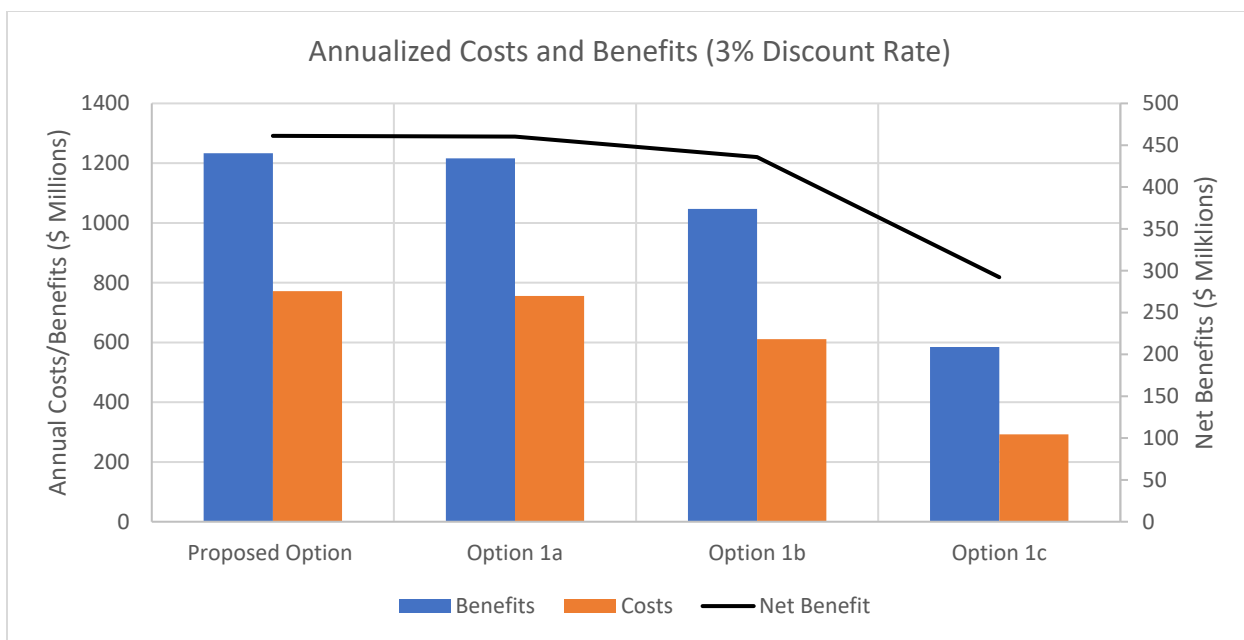


Figure 13-1 : Annualized Costs and Benefits (3% Discount Rate)

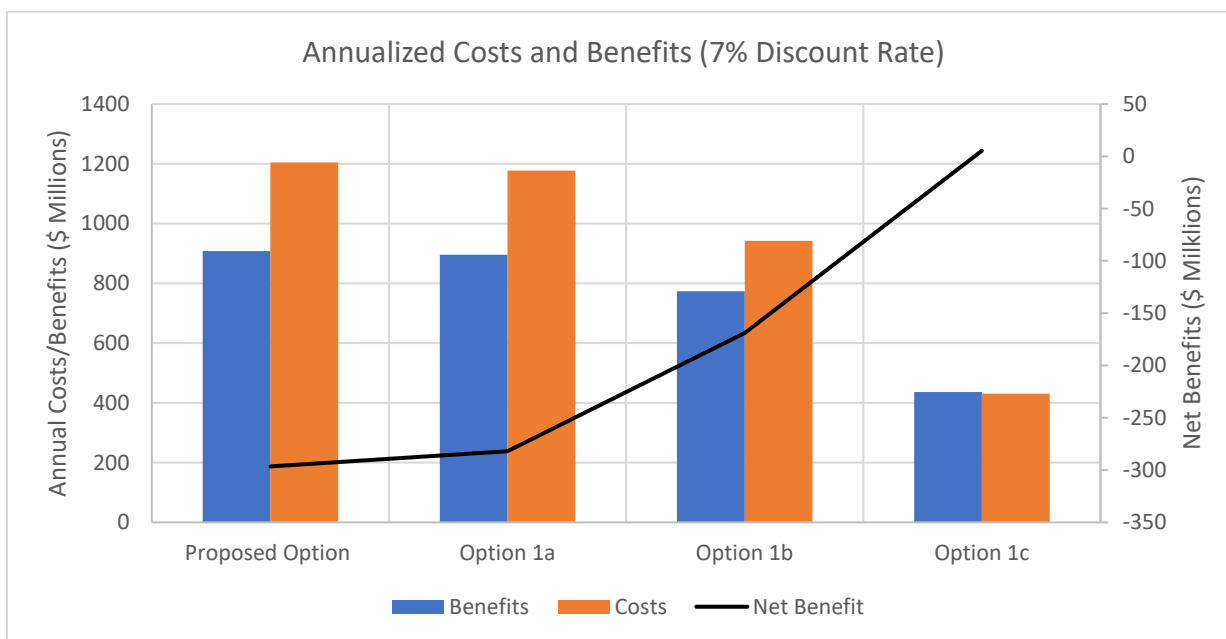


Figure 13-2 : Annualized Costs and Benefits (7% Discount Rate)

To evaluate each regulatory option, AWWA considered the various concerns with the underlying analyses to support the rule that are discussed throughout this letter and the guiding principles for PFAS regulation that were noted earlier, and has determined that any rule will require a substantial level of work to improve the analyses that support the decision by the EPA. Furthermore, AWWA recommends that – if any rule is finalized based on the proposal – EPA finalize the MCLs for PFOA and PFOS at 10 ppt each. AWWA makes this recommendation for several reasons, laid out in more detail below.

The EPA's proposed approach (4 ppt PFOA, 4 ppt PFOS, and hazard index of 1.0 for PFHxS, PFNA, HFPO-DA, and PFBS) will not be feasible, as they will create significant challenges for water systems to implement, is not clearly EPA's legal authority under SDWA, and rely on a series of critically flawed analyses that mischaracterize the impacts of the proposed rule. As discussed earlier, the large number of systems that will need to install drinking water treatment will create challenges in implementing the MCLs on the timeline provided by the EPA and, ultimately, systems will not be able to meet these timelines while also applying best engineering practices to plan, design, and construct these facilities. Additionally, the EPA's statement that it has legal authority to concurrently propose a drinking water regulation with a preliminary determination is a misinterpretation of SDWA. Finally, the hazard index approach is insufficiently substantiated by occurrence data and toxicological science.

As with the EPA selected rule option, setting the nation's first drinking water MCLs for PFOA and PFOS at levels as low as 4 or 5 ppt will create a significant combination of implementation challenges. The effect of such a rule option selection will be to delay all water systems in installing treatment facilities and increase the burden on households to pay for doing so, rendering such levels infeasible. Instead, EPA should place an emphasis on higher-priority water systems by targeting those systems higher levels of PFAS first. Focusing on systems with higher levels of PFAS will ensure that communities with the greatest risk of exposure to PFOA and PFOS are able to control exposure via drinking water more cost-effectively and promptly. Setting the nation's first drinking water standard for PFOA and PFOS at 10 ppt does not preclude EPA from further reducing these standards as technology advances and available occurrence and toxicological data improve.

Ultimately, each of the proposed options is likely to represent a net cost to society (and drinking water consumers), requiring investments that will outweigh the benefits. These impacts will be most dramatically felt by smaller systems serving less than 10,000 people and the affordability analysis suggests that the costs to implement these treatment facilities will range from hundreds to thousands of dollars annually for individual households, significantly exceeding affordable margins for household expenditures for drinking water (i.e. drive the cost of water services beyond EPA's measure of affordable drinking water).

Consequently, EPA should significantly improve upon the analyses to strengthen any rule and to accurately capture the impacts on water systems and public health. The Administrator should re-evaluate this rule with the improved analyses to determine if the benefits justify the costs and that the rule is feasible for small systems to implement. If any rule is finalized without the additional analysis and public review AWWA recommends that EPA utilize Option 1c and set MCLs of 10 ppt PFOA and 10 ppt PFOS. Option 1c affords the greatest opportunity for health benefit for impacted communities while reducing affordability concerns associated with the rule. If EPA determines that regulation of additional PFAS is merited, the agency should propose a rule following a final determination to regulate, consistent with the authority provided by SDWA.

14. Summary of Key Recommendations

Advancing public health is a shared goal between drinking water systems and EPA and AWWA has evaluated the rule to support EPA's actions moving forward meaningfully and legally, using sound science. AWWA appreciates the agency's interest in preparing a thoughtful, thoroughly crafted proposal to establish NPWDRs for PFOA, PFOS, and additional PFAS.

As previously discussed, the regulation of PFAS in drinking water as proposed will create numerous implementation challenges. Although EPA is interested in an expeditious rulemaking to reduce PFAS exposure, it is nonetheless important that EPA finalize a rule that is based on sound science, recognizes the importance of drinking water affordability, and be feasible to implement.

While EPA has a stated interest in advancing immediate protection of communities from PFAS exposure, water systems still need to perform the necessary work to implement any rule requirements – regardless of the timeline. Three years is insufficient time for water systems to comply with EPA's proposed rule option. The Administrator should provide a 2-year extension as part of the final rule per authority provided by SDWA, instead of relying on already overextended state primacy agencies.

Several of the EPA's analyses underlying the rulemaking need improvement to be credible. The occurrence analysis lacks transparency on the levels of PFAS in communities nationally and criteria for data inclusion/exclusion is not clear. It is also an overly complicated approach to assessing national occurrence data for PFAS, which are currently being collected as part of the UCMR 5 program. The cost analysis for drinking water treatment is demonstrably underestimating the impacts of the rule based on case study data and a model by Black & Veatch, which was crafted leveraging long-standing national PFAS treatment design expertise. Finally, the benefits analysis includes several assumptions that are not clearly and consistently discussed by the agency. For example, the analysis of CVD risk reduction accounts for impacts to total cholesterol from PFAS exposure but excludes the impacts to HDL-C, which decreases risks of CVD. These analyses, which underpin the rulemaking's HRRCA and affordability analysis, need significant improvements.

As part of this proposal, EPA proposes preliminary determinations for PFHxS, PFNA, HFPO-DA, and PFBS concurrently with a proposed drinking water standard for these compounds. The preliminary determinations for PFNA, HFPO-DA, and PFBS are not supported by the available occurrence data and the determination for the mixture of these PFAS is similarly lacking in co-occurrence data and is inconsistent with EPA guidance. Furthermore, the proposed regulation of these compounds concurrently with the preliminary determination is beyond EPA's authority under SDWA. EPA should (i) not finalize the preliminary determinations, (ii) evaluate and, if appropriate, re-issue the preliminary determinations following the availability of UCMR 5 data, and (iii) withdraw and re-issue a scientifically sound and adequately supported proposal to regulate the PFAS among these four which EPA can provide a sound basis for a positive determination to regulate.

With respect to the proposed drinking water standards for PFOA and PFOS, the docket does not support finalizing a rule, particularly a rule where attributable benefits outweigh quantifiable costs. The affordability of EPA's rule options is especially questionable for households served by small systems – systems which SDWA requires EPA give particular attention to in crafting a drinking water standard. If EPA moves forward with a final rule, setting MCLs for PFOA and PFOS at 10 ppt is the most appropriate

option among the options EPA has analyzed. This option will prioritize water systems with the highest PFAS levels moving forward immediately.

EPA's proposed approach to require monitoring under the Standard Monitoring Framework and the use of the one-half of the MCL as a trigger level is appropriate. However, the use of reporting results below the PQL is inappropriate as it requires water systems to rely on unreliable data to determine monitoring requirements as part of the regulatory requirements.

In summary, EPA is strongly encouraged to consider the impacts of this rule carefully and to ensure that, if finalized, the regulations are feasible, based on the best available public health information, based on accurate cost assessments, and legally defensible. While the agency has a strong interest in expeditious action, it is important to move actions forward meaningfully and in a way that avoids negative consequences that are avoidable. AWWA's recommendations are intended to assist EPA ensure that high-risk water systems are prioritized while also providing EPA with additional time to get better data and make additional sound, defensible risk management decisions. AWWA's recommendations also reflect EPA placing the onus of PFAS risk reduction on polluters rather than communities through the source water protection actions framed in the agency's Strategic Roadmap for PFAS.

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Appendix A

Detailed Technical Comments on PFAS Toxicological Assessments

Intended for
American Water Works Association (AWWA)

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Report

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Comments on the Proposed National Primary Drinking Water Regulation for Per- and Polyfluoroalkyl Substances (PFAS)

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1. Introduction

This document provides comments on the science considered and technical methods and approaches applied in the development of the US Environmental Protection Agency's (USEPA) proposed National Primary Drinking Water Regulation (NPDWR) for six per- and polyfluoroalkyl substances (PFAS) including perfluorooctanoic acid (PFOA), perfluorooctane sulfonic acid (PFOS), perfluorononanoic acid (PFNA), hexafluoropropylene oxide dimer acid (HFPO-DA and its ammonium salt, commonly known as GenX Chemicals), perfluorohexane sulfonic acid (PFHxS), and perfluorobutane sulfonic acid (PFBS). Our review for these comments includes the documentation of these methods and approaches in the following public comment drafts:

- Toxicity Assessment and Proposed Maximum Contaminant Level Goal for Perfluorooctanoic Acid (PFOA) in Drinking Water (USEPA 2023a) and Appendices (USEPA 2023b);
- Toxicity Assessment and Proposed Maximum Contaminant Level Goal for Perfluorooctane Sulfonic Acid (PFOS) in Drinking Water (USEPA 2023c) and Appendices (USEPA 2023d);
- Economic Analysis for the Proposed Per- and Polyfluoroalkyl Substances National Primary Drinking Water Regulation (USEPA 2023e) and Appendices (USEPA 2023f);
- Maximum Contaminant Level Goal (MCLG) Summary Document for a Mixture of Four Per- and Polyfluoroalkyl Substances (PFAS): HFPO-DA and its Ammonium Salt (also known as GenX Chemicals), PFBS, PFNA, and PFHxS (USEPA 2023g); and
- Framework for Estimating Noncancer Health Risks Associated with Mixtures of Per- and Polyfluoroalkyl Substances (PFAS) (USEPA 2023h).

These public comment drafts also include the USEPA's attempts to respond to the Scientific Advisory Board's detailed comments and review (August 2022) of the following four draft documents:

- Proposed Approaches to the Derivation of a Draft Maximum Contaminant Level Goal for Perfluorooctanoic Acid (PFOA) (CASRN 335-67-1) in Drinking Water (December 2021) (USEPA 2021a);
- Proposed Approaches to the Derivation of a Draft Maximum Contaminant Level Goal for Perfluorooctane Sulfonic Acid (PFOS) (CASRN 1763-23-1) in Drinking Water (December 2021) (USEPA 2021b);
- Draft Framework for Estimating Noncancer Health Risks Associated with Mixtures of Per- and Polyfluoroalkyl Substances (PFAS) (December 2021) (USEPA 2021c); and
- Analysis of Cardiovascular Disease Risk Reduction as a Result of Reduced PFOA and PFOS Exposure in Drinking Water (December 2021) (USEPA 2021d).

The USEPA made substantial revisions and added new material, which is now included in the public comment drafts, to address the extensive comments made by the Science Advisory Board (SAB). In addition, the USEPA responded to the SAB comments in the following report:

- USEPA Response to Final Science Advisory Board Recommendations (August 2022) on Four Draft Support Documents for the USEPA's Proposed PFAS National Primary Drinking Water Regulation (USEPA 2023i).

The revisions to the 2023 public comment drafts include the following, none of which have been peer-reviewed by the SAB, and therefore have to be peer-reviewed during this 60 day public comment period (which will end on May 30, 2023):

- A review of mechanistic data, which was lacking in the December 2021 draft documents, and synthesis of mechanistic data with the animal and human data. The SAB specifically stated "(US)EPA should include an evaluation of mechanistic/mode of action data for

those effects considered as the potential basis for the reference doses (RfDs) and cancer slope factors (CSFs)." (p. 2 of introductory letter, USEPA 2022a);

- Newly derived candidate RfDs for total cholesterol are now included in the proposed MCLG documents to align with the cardiovascular disease (CVD) benefits analysis. The SAB specifically stated *"(US)EPA should ensure that recommendations for the draft MCLG documents relating to evidence identification and synthesis are applied to the CVD endpoint."* (p. 4 of introductory letter, USEPA 2022a);
- USEPA quantified benefits of changes in high-density lipoprotein cholesterol (HDL) in relation to PFOA and PFOS;
- USEPA quantified benefits of changes in elevated blood pressure in relation to PFOS (although this had not requested by the SAB);
- Addition of quantified benefits of birth weight associated with reductions in PFOA/PFOS (SAB requested that USEPA consider risk reduction for additional endpoints);
- Newly derived candidate RfDs for decreases in birth weight (which is sometimes described as low birth weight) in relation to PFOA and PFOS, which were needed to align with quantified birth weight benefits. However, low birth weight has a specific definition (i.e. birth weight below 2500 grams), and this is not the endpoint for which the RfD is derived. The tiered risk assessment approach described in the December 2021 Draft *Framework for Estimating Noncancer Health Risks Associated with Mixtures of Per- and Polyfluoroalkyl Substances (PFAS)* was replaced with a data-driven/menu-based framework for the selection of component-based approaches for PFAS mixture assessment, and the interpretation of any approach as being "screening" or preliminary was minimized. The SAB specifically stated, *"Methods analogous to those classified by USEPA as 'Screening Level' or 'Tier 1' in the framework are potentially being used by states in a decision-making capacity. Issuance of this framework without recognition of that fact may create confusion for public water supplies and risk communication challenges for the public."* (p. 93-94, USEPA SAB 2022a).

The above changes to the underlying documents and assessments are not exhaustive; however, the comments below are largely focused on a review of these proposed changes.

The proposed approach for the regulation of these compounds includes the development of a hazard index (HI) that is used to determine if the combined levels of 4 PFAS pose a potential health risk. The use of this approach represents the first time a HI approach has been applied for a federal regulation; it has traditionally been applied as a screening tool to make initial decisions regarding chemical remediation. It is also important to note that the approach used by the agency in this case is inconsistent with existing regulatory guidelines; therefore, one of the main focuses of these comments are around the application of a HI outside of a screening approach and the challenges in finding support for this approach in the available science for PFAS.

As the proposed PFAS NPDWR is a significant regulatory action that was submitted to the Office of Management and Budget (OMB) for review, an economic analysis is required under Executive Order 12866. The remaining comments focus on the USEPA's use of the available science related to PFAS exposure and selected endpoints, specifically low birth weight and cardiovascular disease (CVD) to attempt to demonstrate quantifiable and nonquantifiable health risk reduction benefits are likely to occur as the result of compliance with the proposed NPDWR.

2. Comments on the Hazard Index Approach

USEPA has proposed the use of a General HI approach for regulation of drinking water concentrations of four PFAS: PFNA, PFHxS, PFBS, and GenX (USEPA 2023g, p.8-18). However,

rather than following the recommendation of the SAB (USEPA 2022a) to conduct the assessment on the basis of common outcome, the Hazard Index for these 4 compounds are calculated using critical effects (RfDs or MRLs) that are based on different endpoints and target tissues:

- PFNA: Delayed development in mouse offspring.
- PFHxS: Thyroid follicular epithelial hypertrophy/hyperplasia in parental male rats.
- PFBS: Decreased thyroxine in mouse offspring.
- GenX: Liver toxicity in female rat dams.

USEPA attempts to defend this approach using arguments that are inaccurate and contradictory. Initially, USEPA tries to make a broad claim that the diverse effects of PFAS are all associated with a common mechanism involving disruption of cellular signaling:

"PFAS, including HFPO-DA, PFBS, PFNA, and PFHxS, disrupt signaling of multiple biological pathways resulting in common adverse effects on several biological systems and functions, including thyroid hormone levels, lipid synthesis and metabolism, development, and immune and liver function (ATSDR 2021; EFSA 2018, 2020; USEPA 2022c)." (USEPA 2023g, p.2)

However, the USEPA suggestion that PFAS causes common disruption of biological pathway signaling that results in common adverse effects is essentially an argument for a common mode of action. This argument is difficult to support, given the agency's determination that information to support a common mode of action for PFAS is inadequate:

"Because PFAS are an emerging chemical class of note for toxicological evaluations and human health risk assessment, mode of action (MOA) data may be limited or not available for many PFAS." (USEPA 2023g, p.3).

Moreover, in contradiction to the USEPA suggestion of a common mode of action across all PFAS, the agency concluded in the USEPA Toxicity Assessment for GenX (USEPA 2021a), one of the 4 chemicals included in the HI, that the liver effects of GenX are not consistent with there being a common mode of action for all PFAS:

"Although there is evidence for a PPAR α MOA in the liver, particularly in the high-dose groups in the available studies, data indicate that liver toxicity extends beyond a single PPAR α -based MOA." (USEPA 2021a, p.84).

USEPA then makes a case for an assumption of dose additivity based on common outcome rather than common mode of action (USEPA 2023g, p.3), citing the USEPA (2000) mixtures guidance. Inexplicably, the USEPA applied a General HI approach across different outcomes for each chemical, apparently based on their assertion of a common mode of action (above), and despite the fact that the multi-outcome approach clearly ignores the recommendation of the SAB (USEPA 2022a). It is also inconsistent with existing USEPA guidelines.

USEPA Mixtures Guidance (1986) does not support the use of dissimilar effects in a Hazard Index:

"Since the assumption of dose addition is most properly applied to compounds that induce the same effect by similar modes of action, a separate hazard index should be generated for each end point of concern. Dose addition for dissimilar effects does not have strong scientific support, and, if done, should be justified on a case-by-case basis in terms of biological plausibility." (USEPA 1986, p.9)

The USEPA Risk Assessment Forum (2000) Mixtures Guidance clearly indicates that the use of critical effects from multiple tissues in a Hazard Index is generally inappropriate. The guidance describes the HI method only in terms of similarity in target organ:

"The Hazard Index method has weaker assumptions and data requirements, is more generally applicable, and has more uncertainty in the resulting assessment. Instead

of requiring knowledge of similar mode of action, the Hazard Index method requires only similarity in target organ.” (USEPA 2000, p.71)

“One of the key desirable features is the constraint to use only data on the effect of concern. Because the Hazard Index is tied to a specific effect, the underlying data should be on that effect. Substituting data on the critical effect introduces an unknown degree of conservatism, so that the Hazard Index is inflated by an unknown amount.” (USEPA 2000, p.85)

“The use of an acceptable level in the relative toxicity scaling factor (e.g. 1/RfD) may be overly health protective in that the RfD (or RfC) is based on the critical effect, defined as the toxic effect occurring at the lowest dose. When the Hazard Index is calculated for some different, less sensitive effect, the RfD will be too low, so the factor (1/RfD) will overestimate the relative toxicity and the Hazard Index will be too large. One alternative that avoids this critical effect conservatism is to use a toxicity-based exposure level that is specific to the target organ of interest and is derived similarly to an RfD (or RfC). For oral exposures, this value is called the target organ toxicity dose or TTD (Mumtaz et al., 1997).” (USEPA 2000, p.82)

Indeed, the use of a Hazard Index based on the combination of endpoints from multiple target tissues, apart from screening purposes, is not supported by any national risk assessment agency. It is inconsistent with ATSDR guidance.

The ATSDR (2022) Public Health Assessment Guidance Manual indicates that when “the health guideline for each contaminant is based on different target organs, health assessors will need to calculate a target-organ-specific HQ for each contaminant. These target-organ HQs can now be added together to give a Tier 3 HI based on the same target organ.” (ATSDR 2022, p.8)

The ATSDR Framework for Assessing Health Impacts of Multiple Chemicals and Other Stressors (2018) indicates that the use of different target organ toxicities is reserved for screening:

“Because it is based on the assumption of dose additivity, the hazard index method is most appropriately applied to components that cause the same effect by the same mechanism or mode of action. In practice, it may be applied to components with different target organs as a screening measure.” (ATSDR 2018, p.43)

USEPA (2023g) does not provide any justification for failing to apply the Target-organ Toxicity Dose (TTD) methodology that ATSDR has developed specifically to address situations where there is an overlap in effects across a mixture of chemicals, but where the critical effects are different: “A Target-organ Toxicity Dose (TTD) for each end point of concern is calculated using appropriate MRL (or RfD) methodology, and then used in estimating the end-point specific HQs and hazard indices.” (ATSDR 2018, p.45)

Use of the ATSDR methodology is particularly important in the case of this NPDWR, since the General HI used by the agency is based on the critical (lowest) effect for each chemical, regardless of target tissue. As pointed out by ATSDR, a HI based on TTDs will certainly be higher than one based on the General HI, indicating that the currently proposed General HI approach is overly conservative.

The draft mixtures framework (USEPA 2021b) referred to the HI based on multiple target tissues as a “Screening-Level HI” to differentiate it from a Target Organ Specific Hazard Index (TOSHI), which they indicated would be more consistent with the USEPA (2000) mixtures guidelines. The SAB specifically supported the USEPA Framework’s use of the TOSHI rather than the Screening Level HI:

"The SAB supports dose additivity based on a common outcome, instead of a common mode of action as a health protective default assumption and does not propose another default approach." (USEPA 2022a, p.90)

However, the SAB indicated that the agency should avoid referring to the multiple target tissue HI approach as a "Screening-Level HI", to avoid the appearance of disparaging the work of states that have been using it in their regulations:

"Methods analogous to those classified by USEPA as 'Screening Level' or 'Tier 1' in the framework are potentially being used by states in a decision-making capacity. Issuance of this framework without recognition of that fact may create confusion for public water supplies and risk communication challenges for the public." (USEPA 2022a, p.3-94)

In response to this SAB concern, the revised Framework (USEPA 2023h) refers to the Screening-Level HI as a "General HI". USEPA then applied the General HI approach rather than the TOSHI approach in deriving the MCLG for mixtures of 4 PFAS (USEPA 2023g). However, as pointed out above, the USEPA SAB (2022a) had clearly indicated that the USEPA should base their assessment on common outcome, which would require the use of the TOSHI approach.

2.1 Conclusions and Recommendations

- The proposed use of a HI based on different target organs or endpoints for estimation of a regulatory value has no support in existing agency guidelines or those of other national and international authoritative bodies.
- The agency should delay promulgating a HI-based assessment until they have developed the necessary Target Tissue Doses (TTDs) to support the use of the TOSHI approach.
- The TTDs can readily be derived using the existing ATSDR methodology (ATSDR 2018).

3. Comments on the Birth Weight Risk Reduction Analysis

In the 2021 documents, the USEPA had not quantified benefits of birth weight risk reductions associated with reductions in exposure to PFOA and PFOS in drinking water. The SAB had recommended that the USEPA consider risk reduction analyses for other endpoints, provided a sufficient rationale existed. The quantified health benefits associated with birth weight impacts now include the following, which have not been peer-reviewed by the SAB:

- Increase in birth weight (in millions of grams); and
- Number of birth-weight related deaths avoided.

Under the Proposed Option (MCL of 4 ppt for PFOA and 4.0 ppt for PFOS and an HI of 1.0 for PFNA, HFPO-DA (GenX chemicals), PFHxS, and PFBS), the USEPA calculated an expected increase in birth weight of 209,300,000 grams and 1,232.7 birth-weight related deaths avoided when the Proposed Option was compared to baseline drinking water concentrations.

Previously, after integrating the evidence, the USEPA had been unclear regarding its strength of evidence conclusion that PFOA and PFOS are associated with low birth weight or decreases in birth weight. Nevertheless, the USEPA (2021a, 2021b) had derived candidate RfDs for decreases in birth weight based on epidemiological studies before selecting a critical effect with the lowest point of departure human equivalent dose (which was for vaccine response). The SAB (USEPA 2022a) requested that the "USEPA consider reevaluating its strength of evidence conclusions for some human endpoints, including (but not necessarily limited to) decreased immune response, increased liver enzymes, increased serum lipids (for PFOA) and decreased fetal growth to determine if they are better described as having "likely" or "strong" evidence rather than "suggestive" or "moderate" evidence of an association with exposure to PFOA/PFOS. **Such a**

reevaluation should consider studies included in the 2016 HESD and more recent studies published after the end date of the literature search for the current draft." (emphasis added, p. 23, USEPA 2022a).

Most recently, the USEPA (2023a, 2023c) judged the evidence of an association between PFOA or PFOS and fetal growth restriction as "likely" based on "moderate" evidence in humans (Note: The evidence integration included a review of mechanistic data which had not been reviewed previously by the SAB. There were also new figures that had not been reviewed previously, including the forest plots for low birth weight and small for gestational age, which had been omitted from the USEPA 2021a, 2021b documents). This conclusion of a likely association between PFOA or PFOS and fetal growth restriction based on moderate evidence in humans also allowed for the justification of quantified health benefits from birth weight impacts associated with the Proposed Option (as well as other regulatory options).

3.1 Candidate Reference Dose for Low Birth Weight

For PFOA, the USEPA derived a Reference Dose (RfD) for low birth weight (LBW, defined as birth weight < 2500 grams) using a hybrid approach for defining the benchmark response (BMR), where the adverse health effect (LBW) was estimated using the dose that increases the percent of responses falling below the clinical definition of LBW (< 2500 g). In 2018, 8.27% of live births fell below 2500 g (CDC 2023a as reported in USEPA 2023b and 2023d). As a result, the USEPA selected a BMR of 5% and the background response of 8.27% to calculate a dose that results in 12.86% of the responses falling within a clinical definition of low birth weight (< 2500 g). For the dose-response association, the USEPA (2023a) chose the coefficient for the effect of PFOA on decreased birth weight from Wikstrom et al. (2020) (β -68.0 g per ln-ng/mL, 95% CI -112.0 to -24.0). When re-expressed to ng/mL (which was used to estimate the benchmark dose (BMD) and the lower bound on the BMD (BMDL)), β was -41.0 per ng/mL, 95% CI -67.5 to -14.5 g per ng/mL.

The use of LBW as the critical effect is inconsistent with other regulatory agencies that found small decreases in birth weight in relation to PFOA and PFOS but not increased risk of low birth weight. These include the following examples:

- ATSDR (2021) reported "*Small (<20-g or 0.7-ounce decrease in birth weight per 1 ng/mL increase in either PFOA or PFOS blood level) decreases in birth weight (PFOA, PFOS).*"
- ATSDR (2021) also reported "*most studies found no association between maternal serum PFOA levels and the risk of low birth weight infants (typically defined as <2,500 g) (Chen et al. 2012a; Darrow et al. 2013; Fei et al. 2007, 2008a; Manzano-Salgado et al. 2017a; Savitz et al. 2012b; Stein et al. 2009) or found a decreased risk of low birth weight infants (Nolan et al. 2009; Savitz et al. 2012a). Similarly, most studies found no increases in the risk for small for gestational age (Chen et al. 2012a; Fei et al. 2007, 2008a; Hamm et al. 2010; Lauritzen et al. 2017; Manzano-Salgado et al. 2017a; Savitz et al. 2012b; Wang et al. 2016; Whitworth et al. 2012a).*" (p. 465)
- EFSA (2020) reported that PFOA or PFOS exposure was associated with "*reduced birth weight.*" Furthermore, EFSA (2018, 2020) concluded that the decrease in birth weight is small after adjusting for confounders and "*the potential longer term consequences of this decrease are unclear.*"
- EFSA (2020) also stated "*As already explained in the previous Opinion on PFOA and PFOS (EFSA CONTAM Panel, 2018), the association with reduced birth weight might at least partly be explained by changes in the physiology during pregnancy, although a recent study seemed to strengthen the causality of the effect (Meng et al., 2018; see also Section 3.3.4.1.1). The remaining decrease in birth weight after adjusting for confounders was not large and the potential longer term consequences of this decrease*

are unclear. Thus far, there is little evidence for an increase in the proportion of children with low birth weight (< 2,500 g).” (p. 138)

In the hazard assessment, the USEPA (2023a, 2023c) evaluated and integrated evidence for fetal growth restriction by combining epidemiological studies on the risk of LBW (birth weight < 2500 grams) with studies on the risk of small for gestational age (SGA, primarily defined in epidemiological studies as birth weight below the 10th percentile for the gestational age). Although these endpoints can be correlated, they are *not* equivalent endpoints and they should not be evaluated as if they are same. The forest plots (figures 3-54, 3-55, 3-56, and 3-57, USEPA 2023a) are new and were not included in the 2021 draft documents. The USEPA (2023a) concluded that the evidence supports increased risk of LBW and SGA in relation to PFOA:

“Overall, nine of the eleven informative studies reporting main effects for either SGA or LBW or both showed some increased risks with increasing PFOA exposures. The magnitude of the associations was typically from 1.2 to 2.8 with limited evidence of exposure-response relationships among the studies with categorical data. Although the number of studies was fairly small, few discernible patterns across study characteristics or confidence ratings were evident across the SGA or LBW findings. For example, four of the nine studies showing increased odds of either SGA or LBW were based on early sampling biomarkers. Collectively, the majority of SGA and LBW studies were supportive of an increased risk with increasing PFOA exposures.” (p. 3-212, USEPA 2023a).

The tables and figures in the PFOA toxicity assessment (USEPA 2023a) reported that eight studies were informative for a total of nine results (Chu et al. 2020, Wang et al. 2016, Wikström et al. 2020, Lauritzen et al. 2018, Manzano-Salgado et al. 2017, Govarts et al. 2016, Hjermitsev et al. 2020, Meng et al. 2018). Closer inspection showed that the results were not consistent within or between studies. For example:

- Three studies stratified results according to sex. One study found increased risk of LBW in girls, but not boys (Wikström et al. 2020), one study found increased risk of SGA in girls and decreased risk of SGA in boys (Wang et al. 2016), and one study found increased risk of LBW in boys, and decreased risks in girls (Manzano-Salgado et al. 2017). Separately, Manzano-Salgado et al. (2017) also reported increased risk of SGA in boys and decreased risks in girls.
- Lauritzen et al. (2018) stratified by country of birth and reported decreased risk of SGA in Norway (median PFOA concentration, 1.62 ng/mL and median PFOS concentration, 9.74 ng/mL) and increased risk of SGA in Sweden (median PFOA concentration, 2.33 ng/mL and median PFOS concentration 16.4 ng/mL). (The range of PFOA and PFOS was similar among study participants from both countries, suggesting other explanations are likely for the differences in risks).
- Exposure-response results were not consistent within studies where exposures were measured in the general population (at low concentrations of PFOA and PFOS):
 - Meng et al. (2018) reported no association between risk of LBW per doubling of PFOA exposure (OR 1.0, 95% CI 0.7–1.5) and slightly increased odds ratios when PFOA exposure was categorized into quartiles and Q2 (OR 1.5, 95% CI 0.8–3.1), Q3 (OR 1.2, 95% CI 0.5–2.5), and Q4 (OR 1.5, 95% CI 0.7–3.3) were compared to Q1.
 - Chu et al. (2020) reported a slightly increased OR for LBW of 1.16 per ng/mL increase of PFOA (median concentration 1.54 ng/mL, interquartile range 0.957 to 2.635 ng/mL), but no increased risks when exposure was categorized into quartiles (OR 1.0 for 4th quartile (≥2.64 ng/ml) compared to 1st quartile (≤0.096 ng/ml)).

- Three studies did not show exposure-response relationships or trends when exposure was categorized into quartiles of exposure (Meng et al. 2018; Wikström et al. 2020; Chu et al. 2020). Wikström et al. (2020) showed an increased risk only for PFOA > 2.30 ng/mL (4th quartile) compared to < 1.1 ng/mL (1st quartile). Chu et al. (2020) did not find any increased risk of LBW when exposure was categorized by quartiles of exposure.
- When stratified by maternal sampling in early pregnancy (1st trimester) and maternal sampling in later pregnancy (2nd 3rd trimesters, cord blood, after delivery), there were 4 studies of sampling in early pregnancy (Hjermitslev et al. 2020; Wikström et al. 2020; Meng et al. 2018; Manzano-Salgado et al. 2017) and 4 studies of sampling in late pregnancy (Chu et al. 2020; Govarts et al. 2016; Wang et al. 2016; Lauritzen et al. 2018). Although Wikström et al. (2020) reported an increased risk of LBW in girls when the highest exposure was compared to the lowest exposure, other studies that sampled early in pregnancy reported decreased risks of LBW (Hjermitslev et al. 2020; Manzano-Salgado et al. 2017).

Importantly, and despite the request by the SAB that the USEPA re-evaluate studies published before and included in the 2016 Health Effects Support Documents (HESD) for PFOA (USEPA 2016a) and PFOS (USEPA 2016b), the USEPA (2023a, 2023c) did not include studies that evaluated risk of LBW or risk of SGA and exposure to PFOA or PFOS that were published prior to 2017 in the overall integration of evidence. Consequently, the USEPA did not consider at least 7 studies that evaluated LBW and did not find an increased risk of low birth weight in relation to PFOA or PFOS (Darrow et al. 2013; Nolan et al. 2009; Savitz et al. 2012a, 2012b; Stein et al. 2009; Chen et al. 2012; Wu et al. 2012). [Note: These studies were summarized in Table D.1.2 in the Appendix to the PFOA Toxicity Assessment (USEPA 2023b) and the study quality evaluation was presented in Figure 3-45 (USEPA 2023a), but these studies were not included in the forest plots (Figures 3-54 and 3-55, USEPA 2023a) or discussed in the integration of evidence]. In contrast, and in response to the request by the SAB (USEPA 2022a), the USEPA (2023a, 2023c) had integrated evidence regarding immunotoxicity studies and cholesterol studies that were older and included in the 2016 HESD documents (USEPA 2016a, 2016b).

The USEPA concluded in the PFOS toxicity assessment (USEPA 2023c):

"Collectively, the majority (7 of 10) of SGA and LBW studies were supportive of an increased risk with increasing PFOS exposures. The increased odds ranged from 1.19 to 4.14 although evidence of exposure-response relationships was lacking. There was no evidence of differences by study confidence as five of these seven were either high (n=4) or medium (n=1) confidence. There was also no evidence of sample timing differences as the majority of studies with associations were reported in studies based on early sampling periods." (p. 3-209, USEPA 2023c).

The USEPA (2023c) did not provide references within the above sentence; however, review of tables and figures reported the following studies were high confidence (Chu et al. 2020, Manzano-Salgado et al. 2017, Lauritzen et al. 2018, Wikström et al. 2020) or medium confidence (Govarts et al. 2016; Hjermitslev et al. 2020; Meng et al. 2018) despite the following issues:

- Exposure-response relationships were generally not seen.
- Four studies (Manzano Salgado et al. 2017; Wikström et al. 2020; Meng et al. 2018; Hjermitslev et al. 2020) were based on sampling during early pregnancy while three studies (Lauritzen et al. 2018, Govarts et al. 2016, Chu et al. 2020) were based on sampling late in pregnancy.

The USEPA (2023e) conflates decreases in birth weight with low birth weight in the economic analysis. The USEPA provided a rationale for estimating medical costs associated with changes in infant birth weight and the value of avoiding infant mortality at various birth weights by citing to health effects in relation to low birth weight specifically:

"LBW is linked to a number of health effects that may be a source of economic burden to society in the form of medical costs, infant mortality, parental and caregiver costs, labor market productivity loss, and education costs (Chaikind et al., 1991; J. R. Behrman et al., 2004; R. E. Behrman et al., 2007; Joyce et al., 2012; Kowlessar et al., 2013; Colaizy et al., 2016; Nicoletti et al., 2018; Klein et al., 2018). Recent literature also linked LBW to educational attainment and required remediation to improve student outcomes, childhood disability, and future earnings (Jelenkovic et al., 2018; Temple et al., 2010; Elder et al., 2020; Hines et al., 2020 Chatterji et al., 2014; Dobson et al., 2018)." (USEPA 2023e, p. 6-360)

"Low birth weight (LBW) is an important health outcome affected by PFOA/PFOS exposure because it is a significant factor in survival rates and medical care costs among infants (ATSDR, 2021)." (USEPA 2023e, p. 6-13)

"Epidemiology studies on PFOA supported an increased risk of LBW in infants with PFOA exposures (USEPA, 2023a). Similarly, epidemiology studies on PFOS showed an increased risk of LBW infants with PFOS exposures. Overall, most epidemiology studies evaluating the association between maternal serum PFOA/PFOS and birth weight reported negative relationships (i.e. increased exposure is associated with decreased birth weight) (Darrow et al., 2013; Verner et al., 2015; Govarts et al., 2016; Negri et al., 2017; Starling et al., 2017; Sagiv et al., 2018; Chu et al., 2020; Dzierlenga et al., 2020; Wikström et al., 2020; Yao et al., 2021). FN30: Recent evidence indicates that relationships between maternal serum PFOA/PFOS and birth weight may be impacted by changes in pregnancy hemodynamics (Sagiv et al., 2018; Steenland et al., 2018)." (USEPA 2023e, 6.13)

When considering the evidence for risk of low birth weight in relation to PFOA and PFOS, the USEPA combines studies of the risk of LBW with studies of the risk of SGA (USEPA 2023a, 2023c). There is little evidence that the risk of LBW or the risk of SGA is increased (see remarks above).

3.2 Exposure-response functions for PFOA and PFOS and decreases in birth weight used in the Economic Analysis

The USEPA (2023e) Economic Analysis relies on the exposure-response coefficients (slope factors) for decreases in birth weight from the main analyses of a meta-analysis of birth weight effects in relation to PFOA (Steenland et al. 2018) which reported a mean birth weight decrease of 10.5 g per ng/ml (95% CI -16.7, -4.4) and a separate meta-analysis of birth weight effects in relation to PFOS (Dzierlenga et al., 2020) which reported a mean birth weight decrease of 3.0 g per ng/ml (95% CI -4.9, -1.1). [NOTE: An average decrease of 10 grams is equivalent to a decrease of approximately 0.35 ounces]. The biological or clinical significance of such small changes in birth weight is uncertain. The exposure-response function (β -10.5 g birth weight per ng/mL serum) for PFOA used in the economic analysis is also considerably smaller than the coefficient (β -41.0 g per ng/mL, 95% CI -67.5, -14.5 for PFOA) from the study selected for the critical effect and the calculation of the BMD and BMDL (Wikström et al. 2020). For PFOS, the exposure-response function for the economic analysis (β -3.0 g per ng/mL) is slightly smaller than the coefficient (β -8.4 g per ng/mL, 95% CI -16.0, -0.5) used for deriving the candidate RfD for low birth weight based on Wikström et al. 2020).

Both of these meta-analyses (Steenland et al. 2018; Dzierlenga et al. 2020) conducted specific sensitivity analyses to evaluate bias associated with maternal sampling during late pregnancy

compared to maternal sampling during early pregnancy. Both meta-analyses reported that essentially no effect on birth weight was seen when maternal blood is sampled early in pregnancy, while a relatively larger effect on birth weight was seen when maternal blood is sampled late in pregnancy. In general, this suggests that any effect of PFOA or PFOS on birth weight is confounded by the time of sampling (Steenland et al. 2018, 2020; Dzierlenga et al. 2020). In brief, an increased glomerular filtration rate and maternal plasma volume expansion during pregnancy leads to an increased elimination of PFOA and PFOS. Plasma volume expansion and glomerular filtration rate are also related to birth weight. When PFAS in serum is sampled late in pregnancy, the magnitude of the glomerular filtration rate and the plasma volume expansion can distort the association between PFAS and birth weight. Therefore, using the main effect from the meta-analysis (which is essentially an average of birth weight effects reported from early in pregnancy and late in pregnancy) will overestimate the health benefits associated with birth weight risk reductions under the assumption that pregnancy hemodynamics confound the association.

Steenland et al. (2018) found that there was no effect on birth weight after including the C8 Science study in the meta-analysis:

*"Our meta-analysis including nine new studies, with an almost equal number of births as prior studies, shows a modest inverse association between maternal or cord PFOA and birthweight, with large heterogeneity across studies. **The two studies with exposure above background levels showed no association, and similarly, restriction to studies with blood sampling conducted early in pregnancy or shortly before conception showed little or no association. These findings are consistent with confounding and/or reverse causality being responsible for the inverse association seen in studies with low background exposure levels and blood sampling conducted later in pregnancy, when confounding and/or reverse causality are likely to be more important.**"*

Overall, there is little evidence that PFOA or PFOS at serum concentrations reported in the general population affect developmental outcomes. The USEPA (2023e) confirmed that there was generally a lack of evidence for exposure-response associations between PFOA and PFOS and other development outcomes:

"Additionally, the magnitude of birth weight changes may be correlated with other developmental outcomes such as preterm birth, gestational duration, fetal loss, birth defects, and developmental delays. As described in Section 6.2, these developmental outcomes have limited epidemiology and toxicology evidence showing associations with PFOA/PFOS exposure and due to this uncertainty, these outcomes were not further assessed." (p. 6-36).

3.3 Other factors that have affected mean birth weight

The USEPA (2023e) economic analysis calculates that the expected value of birth weight increases, assuming the MCLs are set to 4.0 ppt for PFOA and PFOS plus a hazard index of 1.0 for PFNA, PFBS, PFHxS, and HFPO-DA, is an average increase of 50 grams (1.8 ounces) in mean birth weight. According to US Natality data (CDC 2023a), the mean birth weight in 2018 was 3261.64g and 8.3% of births were low birth weight (<2500 g). If average birth weight were to increase by 50 grams, the mean birth weight would be 3,316.84 g.

During 2003-2018, median PFOS in blood serum decreased substantially by 12 ng/mL, from 14.6 ng/mL in 2003-2004 to 2.6 ng/mL in 2017-2018 (USEPA 2022b) in the population of women aged 16-49 years old (women of childbearing age). During the same years, median PFOA in blood serum also decreased but by a smaller absolute change of 2.1 ng/mL, from 3 ng/mL in 2003-2004 to 0.9 ng/mL in 2017-2018) (USEPA 2022b).

Although the average birth weight in 2003 was 3291.03 grams, which was 30 grams higher than average birth weight in 2018, there were fewer births of low birth weight babies (CDC 2023a). In 2003, 7.9% of births were of low birth weight while in 2018, 8.3% of births were of low birth weight. Together, these data suggest further reductions of PFOA and PFOS concentrations in blood serum are unlikely to result in measurable increases in average birth weight (CDC 2023a).

Tilstra and Masters (2020) reported that average birth weight decreased in the United States since at least 1990. In 1990, average birth weight was 3314.5 grams (approximately 50 grams more than in 2018). However, Tilstra and Masters (2020) provided an analysis that argued that the shift to lower average birth weight is due to changes in obstetric practices (more c-sections and scheduled births). As a result, there are fewer and fewer vaginal births at 40-42 weeks, when babies are heavier; most of these births have shifted to 37-39 weeks because of changes in obstetric practices. This shift has affected the average birth weight.

Data from other areas where PFOS and PFOA are found in the blood serum at similar concentrations to the US also provided evidence that PFOS and PFOA in blood serum at general population levels do not result in decreased birth weight. For example, birth weight in the Faroe Islands (where decreases in antibody response to diphtheria vaccination in relation to increases in PFOA and PFOS form the basis for the RfD for immunotoxicity effects, specifically decreases in vaccine response at age 7 in relation to PFOA or PFOS at age 5) has increased over the past 50 years. For the years 1969-1981, Olsen and Joensen (1985) reported that the average birth weight of liveborn infants delivered in the Faroe Islands was the highest average weight (3,610 g) reported by 33 countries. More recently, Olsen et al. (2023) found that that the mean birthweight in the Faroe Islands was higher than other Nordic countries and had increased during 2010–2019 in the Faroe Islands.

3.4 Conclusions and Recommendations

- When integrating the evidence of birth weight effects and arriving at an evidence stream judgment for humans, the USEPA should consider older studies of LBW in relation to PFOA and PFOS that predated the 2016 HESD (USEPA 2016a, 2016b). Reconsidering these studies likely decreases confidence in the judgment of evidence.
- The USEPA should provide further rationale for using Wikström et al. (2020) as the critical study for the association of decreased birth weight given that it is only one study that sampled PFAS in serum during early pregnancy that showed an association between decreased birth weight and increases in PFAS; other studies that sampled during early pregnancy did not show an association or they showed an attenuated association, which potentially leads to a conclusion that the evidence for an association between PFAS and decreased birth weight is inconsistent after considering potential confounding.
- The USEPA should provide a quantified sensitivity analysis and further discussion of the effects of confounding or reverse causation by pregnancy hemodynamics on the health benefits analysis.
- Available data suggest that further reductions of PFOA and PFOS concentrations in blood serum are unlikely to result in measurable increases in average birth weight. Data from other areas where PFOS and PFOA concentrations are elevated suggest that increases in average birth weight and decreases in infant mortality are likely not expected. For example, PFOS and PFOA in blood serum have been measured in maternal serum in birth cohort studies in the Faroe Islands with mean concentrations similar to that reported in the general population in the US (Grandjean et al. 2012). Studies have reported that the mean birthweight in the Faroe Islands was higher than other Nordic countries and had continued to increase during 2010–2019 in the Faroe Islands (Olsen and Joensen 1985; Olsen et al. 2023). Although there are no published studies of birth weight in relation to PFAS serum concentrations in the Faroe Islands, it is unlikely that small decreases in birth

weight in relation to PFAS – should the association exist in this population – have adverse health consequences.

4. Comments on the Cardiovascular Disease Risk Reduction Analysis

Previously, the USEPA had proposed using the atherosclerotic cardiovascular disease (ASCVD) model to estimate reductions in CVD risks associated with reductions in exposure to PFOA and PFOS in drinking water. The SAB was generally supportive of the overall approach for estimating reductions in CVD risk; however, the SAB noted that the approach did not mesh with the USEPA's conclusion that there was insufficient evidence of increased CVD risk to inform a candidate RfD. In response to SAB feedback, the USEPA (2023a, 2023c) developed RfDs for total cholesterol (TC) as a precursor to CVD and to further justify its use of the ASCVD model – which uses TC as one of several variables to estimate 10-year risk of CVD events – for quantifying CVD benefits. The quantified health benefits now include the following:

- Number of non-fatal myocardial infarction (MI) cases avoided;
- Number of non-fatal ischemic stroke (IS) cases avoided; and
- Number of CVD deaths avoided.

Under the Proposed Option (MCL of 4 ppt for PFOA and 4 ppt for PFOS and an HI of 1.0 for PFNA, HFPO-DA (GenX chemicals), PFHxS, and PFBS), the USEPA calculated that the following morbidity and mortality are avoided when the Proposed Option is compared to baseline drinking water concentrations: 6,081.0 non-fatal MI cases avoided; 8,870.8 non-fatal IS cases avoided; and 3,584.6 CVD deaths avoided (USEPA 2023e).

In order to calculate the avoided CVD related mortality and morbidity, the USEPA used exposure-response functions of serum PFOA and PFOS on TC, and serum PFOS (but not serum PFOA) on systolic blood pressure (BP) to estimate annual changes in TC and BP biomarkers.

4.1 Exposure-response functions for PFOA and PFOS and TC

The USEPA (2023f) conducted a meta-analysis of epidemiological studies of the general population of the associations between certain PFAS and total cholesterol to estimate the exposure-response function. Using these exposure-response functions presumes that further reductions in average PFOA and PFOS concentrations in serum in the general population will result in decreases in serum cholesterol, and that decreases in serum cholesterol will lead to decreases in CVD. In other words, this presumes that serum cholesterol is an intermediate variable on the causal pathway between PFAS exposure and CVDs.

The PFOA-TC exposure-response function developed by USEPA (2023f) is the summary estimate from a meta-analysis of four studies of the general population (Nelson et al. 2010; He et al. 2018; Dong et al. 2019; Fan et al. 2020). [NOTE: The slope factor is 1.57 mg/dL per ng/mL serum PFOA. The number of studies is either four (see p. F-11, Table F-2, and p. F-12, Figure 2, USEPA 2023f) or six (see p. F-10, USEPA 2023f and elsewhere in the documents).] Similarly, the PFOS-TC exposure-response function developed by USEPA (2023f) is the summary estimate from a meta-analysis of five studies of the general population (Chateau-Degat et al. 2010, Nelson et al. 2010, He et al. 2018, Dong et al. 2019, Fan et al. 2020), including the same four studies used for the PFOA-TC exposure-response function. Four of these studies were based on cross-sectional analyses serum PFAS and TC from National Health and Nutrition Examination Surveys (NHANES) and included overlapping years. For example:

- Nelson et al. (2010) included NHANES participants from 2003 to 2004; individuals taking cholesterol lowering medication were excluded.

- He et al. (2018) included NHANES participants from 2003-2004 to 2011-2012; individuals taking cholesterol lowering medication were not excluded.
- Dong et al. (2019) included NHANES participants from 2003-2004 to 2013-2014; individuals taking cholesterol lowering medications were excluded.
- Fan et al. (2020) included NHANES participants from 2011-2012 to 2013-2014; individuals taking cholesterol lowering medication were not excluded.

Overall, the USEPA (2023f) identified and included 14 studies in the meta-analysis of exposure-response relationships between PFOA or PFOS and TC. When all 11 studies of PFOA and TC are included in the meta-analysis, the slope factor is 0.003 mg/dL TC per ng/mL in serum PFOA. However, the exposure-response relationship used for the benefits analysis was based only on the summary estimate of the four (PFOA) or five (PFOS) studies that reported linear slope-relationships (beta coefficient for a change in TC or HDL-C in mg/dL to increases in serum PFOA or PFOS in ng/mL). These are the studies of the general population and the coefficient (1.57 mg/dL TC per ng/mL serum PFOA) used in the benefits analysis is 500-fold higher than when all 11 studies are included. This means that the estimated benefits will be much greater using the coefficient from the meta-analysis of the four (overlapping) general population studies than from using the coefficient from the 11 studies of PFOA-TC.

In fact, there appears to be a non-linear association between PFOA and TC, which is not accounted for when a linear slope factor is used over a relatively narrow range of PFOA in serum seen in the general population. In an evaluation of serum lipids in participants in the C8 Health Science study, Steenland et al. (2009) reported that the exposure-response function was steeper at concentrations of total cholesterol (TC) below approximately 208 mg/dL. Predicted TC leveled off at around 50 ng/mL of PFOA. By relying on the exposure-response function from four studies (PFOA) or five studies (PFOS) of the general population (with average PFOA and PFOS serum concentrations below 25 ng/mL), the calculated health benefits are greater than would be expected than if the exposure-response function was based on the distribution of serum PFOA and PFOS seen in occupational populations and studies of communities with drinking water contaminated by PFOA or PFOS. These populations have blood serum concentrations that are 2 to 4 orders of magnitude greater than those in the general population.

4.2 Issues related to use of the ASCVD model

The USEPA assumes that CVD (myocardial infarctions and strokes) can be reduced indirectly by decreasing average serum PFOA and PFOS concentrations further, which would lead to decreased total cholesterol; however, the USEPA has not shown evidence that PFAS exposure (particularly PFOA or PFOS) directly increases the risk of CVD. The USEPA (2023a, 2023c) did not acknowledge that epidemiological studies of PFAS exposures have not observed increased risks of CVD even in studies of populations exposed to the highest concentrations; instead, the USEPA suggested that the results are inconsistent.

The benefits analysis does not directly use PFOA or PFOS concentrations as inputs to the pooled cohort ASCVD model that evaluates the 10-year probability of CVD outcomes (Goff et al. 2014). Instead, the benefits analysis focuses on the exposure-response function between PFOA and PFOS and the precursor endpoint (e.g. total cholesterol) from a meta-analysis of results from epidemiological studies to calculate inputs to the ASCVD model.

Even under the assumption that PFOA or PFOS in serum leads to high total cholesterol, and a shift in the distribution of average cholesterol leads to an increased proportion of individuals with high cholesterol (which potentially affects a large population), there is a lack of evidence that such a shift has occurred based on PFOA and PFOS concentrations in the general population over the past 20 years. As such, there is substantial uncertainty in the population health benefit of

reduced TC by further reducing PFOA and PFOS concentrations from the baseline assumptions to 4 ppt each. The USEPA partially acknowledged this uncertainty when it stated:

"The analysis assumes that the CVD risk impact of changes in TC/BP from reductions in serum PFOA/PFOS is the same as the CVD risk impact of changes in these biomarkers due to other reasons such as behavioral changes or medication." (P. 6-117,6-118, USEPA 2023e).

The ASCVD model uses the following inputs to estimate a 10-year probability of a first hard ASCVD event in adults, 40 to 79 years of age (who are free from ASCVD): age, sex, TC, HDL-C, systolic BP, use of antihypertensive therapy, diabetes, and current smoking (Goff et al. 2014). However, the USEPA (2023e) used changes in TC (in relation to PFOA and PFOS) and changes in blood pressure (BP) (in relation to PFOS, discussed further below), but not changes in high density lipoprotein cholesterol (HDL-C), as inputs to ASCVD model.

Risks of CVD are lower in individuals or populations with higher levels of HDL-C. If PFOA or PFOS are also associated with higher HDL-C, it is plausible that risks of CVD would not be impacted by higher TC (Steenland et al. 2020). In a meta-analysis, the USEPA (2023f) found that, on average, HDL-C increased with PFOA and PFOS, although the summary results of the meta-analyses were not statistically significant. Across the documents, the USEPA is not consistent in their conclusions regarding PFOA or PFOS and HDL-C. Separately, the USEPA made the following conflicting statements regarding the strength of evidence for HDL-C and PFOA:

"Positive associations between PFOA and HDL were also observed in most studies in the general population." (p. 3-155, USEPA 2023a)

"HDL was not associated with PFOA." (p. 3-173, USEPA 2023a).

"The available evidence does not support a consistent association between PFOS and reduced HDL." (p. 3-164, USEPA 2023c).

In contrast, the USEPA presented similarly inconsistent language for effects of PFOS on BP, but included the effect of PFOS on BP in the calculation of the ASCVD model, while excluding the effect of HDL-C on PFOA or PFOS (see next section, Exposure-response function for PFOS and increases in blood pressure for additional information).

The SAB (USEPA SAB 2022a) requested that the USEPA address whether the inclusion of HDL-C would influence the results of the benefits analysis. In response, the USEPA (2023f) conducted a sensitivity analysis of a hypothetical exposure reduction of 1 ppt PFOA and 1 ppt PFOS and found that inclusion of the HDL-C effects (from the meta-analysis) decreases the annualized CVD benefits by 23-25%. Meanwhile, exclusion of the BP effects decreases annualized CVD benefits by approximately 1.8% to 2.2%. In other words, the annualized CVD benefits may be substantially overstated by excluding HDL-C from the model. In any event, the uncertainty associated with the estimated benefits from the proposed MCLs is large. It is not clear that the proposed MCLs will further drive down average PFOA or PFOS in blood serum and therefore if the estimated benefits will materialize.

4.3 Exposure-response function for PFOS and increases in blood pressure

The USEPA (2023e) also justified including changes in BP associated with PFOS as an input into the ASCVD model, and stated that the USEPA (2023c) had concluded "there was overall consistent evidence of an association between PFOS and BP in studies conducted in general adult populations." (p. 6-15, USEPA 2023e).

Subsequently, the USEPA (2023e) used the exposure-response function between PFOS and increases in blood pressure from a study of NHANES participants, 2003-2012 (Liao et al. 2020). This addition has not been peer-reviewed by the SAB and there is actually inconsistent language regarding the strength of evidence conclusions in the USEPA PFOS Toxicity Assessment (2023c),

which also indicated that the evidence of an association between PFOS and BP is uncertain. Examples of the inconsistency follow here:

"High and medium confidence studies reported positive associations with blood pressure and increased risk of hypertension." (P. 3-176, USEPA 2023c)

"While there is some evidence that PFOS exposure might also have the potential to affect blood pressure and other cardiovascular responses in humans given relevant exposure circumstances, the human evidence underlying this possibility is uncertain and without support from animal or mechanistic studies." (P. 3-176, USEPA 2023c).

"Results from studies of varying confidence reported mixed results for changes in blood pressure, including DBP and SBP, and risk of hypertension for all study populations. Studies in children (10) reported mostly non-significant associations with blood pressure and/or hypertension, though two studies in adolescents reported significantly increased (1/10) and decreased (1/10) DBP in males. In adults (13), one study reported a significantly increased risk of hypertension (1/13), but associations from other studies did not reach significance (3/13). When stratified by sex, there were mixed results. One study reported a higher risk of hypertension for males (1/13), while another reported higher risk for females (1/13). One study reported an inverse association for DBP (1/13), while others reported positive associations for DBP (6/13), but only three studies reached significance. SBP was significantly increased for all adults (4/13), in females only (2/13), and in males only (1/13). No studies examined blood pressure or hypertension in occupational populations." [USEPA 2023c, p. 3-177]

Overall, the rationale for including changes in BP in relation to PFOS is not clear; the evidence regarding BP effects from PFOS is equivocal, similar to that of changes in HDL-C. Furthermore, to include changes in BP but not include changes in HDL-C in relation to PFOS is inconsistent, especially considering that the sensitivity analysis that included HDL-C effects in the ASCVD model showed a reduction of as much as 25% in the annualized CVD benefits if the USEPA meta-analysis slope factors are used. In contrast, exclusion of BP effects decreases annualized CVD benefits by 1.8%-2.2% if USEPA meta-analysis slope factors are used.

The estimated health benefits do not consider the potential impact of clinical management of CVD risks. That is, clinicians use the ASCVD risk model to evaluate 10-year risk of hard CVD events and inform decisions about risk management, with one of the common methods for modifying CVD risk being the use of cholesterol-lowering medications. At least two scenarios involving the use of cholesterol-lowering medications can result in overestimated CVD risk reductions in relation to PFAS based on the observed association of increased cholesterol with increased PFOA or PFOS:

1. Clinicians recommend that individuals with high cholesterol be administered cholesterol-lowering medication, with statins typically recommended first. As described in the next section on biological mechanisms, PFAS serum concentrations in individuals who use statins have not been reported to differ from serum concentrations in individuals who do not use of statins. Assuming that statins decrease circulating cholesterol levels but do not effect PFAS serum concentrations (that is, PFAS serum concentrations remain relatively unchanged), the 10-year risk of a hard CVD event will decrease due to medication use but it is unrelated to a decrease in PFAS serum concentrations. The CVD benefits calculated from reductions in PFAS levels using the ASCVD model are overstated.
2. On the other hand, the clinician may prescribe a bile acid sequestrant to lower cholesterol. As described in the next section on biological mechanisms, there is some evidence that use of bile acid sequestrants decreases PFOA serum concentrations as well as circulating cholesterol levels. In this scenario, the CVD health benefits (calculated using the ASCVD model) resulting from medication use are misattributed to decreases in PFOA (or PFOS) serum levels (whether PFAS exposure decreases or not) because the association between

PFOA and serum cholesterol is confounded by underlying physiological processes (for example, enterohepatic cycling, which could explain the reported association between bile acids and PFOA and distort the magnitude of the association between PFOA and high cholesterol).

These are theoretical examples for illustrative purposes; however, in the absence of a better understanding of the biological mechanisms that underpin the association between increases in PFOA or PFOS in blood serum in the general population and increases in total cholesterol, the quantified benefits analysis may be less than estimated.

Although the USEPA has added discussion of biological mechanisms that inform the strength of evidence conclusion for increased CVD impacts associated with PFOA and PFOS, the discussion largely focuses on biological mechanisms that inform the decreases with cholesterol seen with PFOA and PFOS at much higher serum concentrations than those reported in the general population on which the RfD is based.

Biological mechanisms for the association between PFOS or PFOA (and other PFAS) and cholesterol has not yet been identified in humans. Some information regarding potential mechanisms, however, may be gleaned from epidemiological analyses of associations between PFAS concentrations and cholesterol among those who take medications to lower cholesterol. For example:

- *Statins* (HMG-CoA reductase inhibitors) inhibit the synthesis of cholesterol in the liver and increase the removal of low-density lipoprotein cholesterol (LDL-C) that is in the blood. Andersen et al. (2021) analyzed National Health and Nutrition Examination Survey (NHANES) data from 2003 to 2016 (while accounting for NHANES sampling parameters) and reported a 2.9% increase in PFOS concentrations ($p=0.001$) among participants who reported using statins. Statin use was not associated with increased or decreased PFOA concentrations (Andersen et al. 2021). Similarly, Ma and Ducatman (2022) found that statin use was associated with statistically significantly increased PFOS concentrations and borderline significantly increased PFOA concentrations (when compared to non-users) in the C8 Health Study.
- *Bile acid sequestrants* (cholestyramine) remove bile acids that are made when LDL cholesterol breaks down. Cholestyramine lowers cholesterol by increasing bile acid secretion. Cholestyramine increased fecal elimination of PFOS and PFOA and decreased blood serum concentrations in an individual who self-administered cholestyramine (Genius et al. 2010). In the cross-sectional analysis of NHANES data from 2003-2016, Andersen et al. (2021) reported that use of cholestyramine was associated with a 1.3% reduction in PFOA and a 15.1% reduction in PFOS serum concentrations. Similarly, when compared to non-users, use of cholestyramine was associated with statistically significant decreases in serum PFAS (and the effect was strongest for PFOS) in the C8 Health Science study (Ducatman et al. 2021).
- *Probenecid* lowers cholesterol by inhibiting organic ion transporters (OAT). Probenecid helps the kidneys remove uric acid from the blood and is also used in the treatment of gout. Ducatman et al. (2021) compared Probenecid users to non-users and found that use of Probenecid was associated with a small increase in serum PFAS which was not statistically significant for PFOA or PFOS in the C8 Health Study (Ducatman et al. 2021)
- *Ezetimibe* inhibits the absorption of cholesterol in the small intestine primarily by inhibiting Niemann-Pick C1-like 1 (NPC1L1) protein. Ma and Ducatman (2022) reported that when compared to non-users, ezetimibe was not associated with blood concentrations of PFAS. Ezetimibe use was not associated with PFOA or PFOS serum concentrations in the NHANES analysis, either (Andersen et al. 2021).

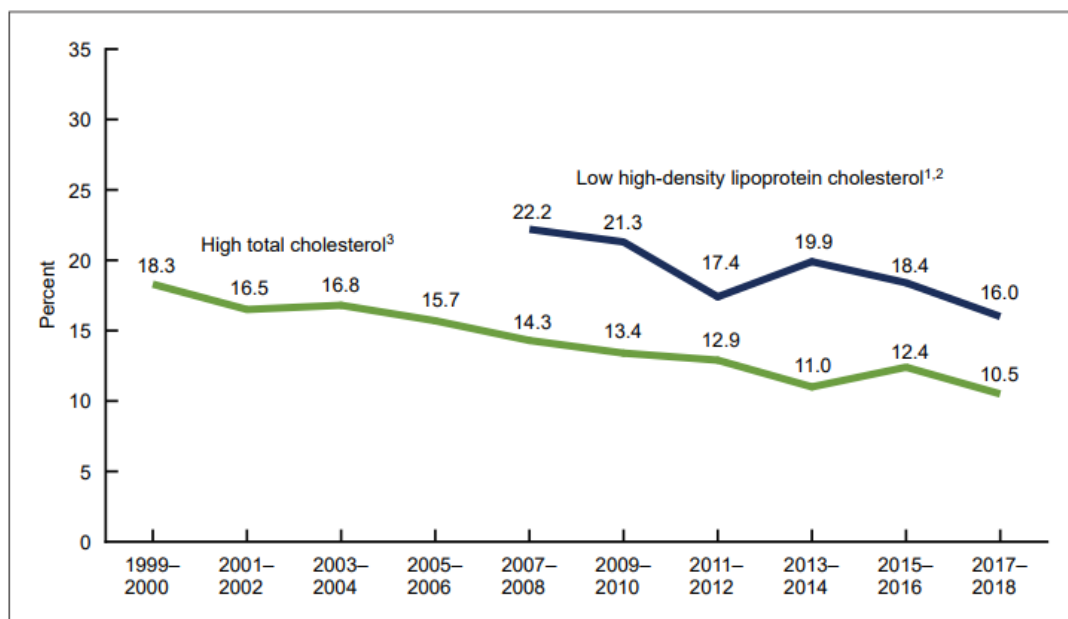
Separately, the effects of lifestyle interventions on cholesterol and PFAS blood concentrations are mixed: Morgan et al. (2023) found that lifestyle interventions over 6 months significantly reduced cholesterol; blood concentrations of PFOS and PFOA (as well as other PFAS) decreased significantly as well. After lifestyle interventions, only PFOS and total cholesterol were positively correlated and PFOS was only distributed in albumin lipoprotein fractions. Before the interventions, PFOS was found in both the albumin and non-albumin lipoprotein fractions.

4.4 Effects of other factors that mediate cholesterol levels likely dominate cardiovascular disease risks

Indirect evidence exists that suggests that the MCLs of 4.0 ppt each for PFOA and PFOS are unlikely to result in benefits as great as that reported by the USEPA (2023e) because other risk factors have a considerably larger impact on cholesterol levels. Collectively, this evidence suggests that a population shift in average cholesterol levels by further decreases in serum concentrations of PFOS and PFOA would not be detected. Over the past 20 years, PFOA and PFOS in serum have decreased in the general population. In adults 20 years and older, PFOA in serum decreased from a median of 5.20 ng/mL in 1999-2000 to 1.47 ng/mL in 2017-2018 (CDC 2023b). In adults 20 years and older, PFOS in blood serum decreased from a median of 30.3 ng/mL in 1999-2000 to 4.7 ng/mL in 2017-2018 (CDC 2023b). An examination of cholesterol levels over the past 60 years indicates substantial reductions have occurred, even during the time period when serum concentrations in PFOS and PFOA were likely increasing (before 1999-2000 when use of PFOA and PFOS in industrial applications and consumer products was greatest):

- Since 1960, TC levels have declined across all adult age groups, with the steepest declines seen in the older age groups. For example, average TC decreased from approximately 250 mg/dL in 1960 to approximately 215 mg/dL in adults 60-74 in 1999-2002 (Carroll et al. 2005). Mean TC in adults aged 20 and older declined to 188 mg/dL in 2017-2018 from 203 mg/dL in 1999-2000, a decrease of 15 mg/dL.
- In adults ages 20 and older, the prevalence of high cholesterol (total cholesterol level of at least 240 mg/dL) was 20% during 1988-1994 (Carroll et al. 2005). Since then, the prevalence of high cholesterol in adults has continued to decline from 18% during 1999-2000 to less than 11% in 2017-2018 (Carroll and Fryar 2020, Figure 4 below).
- The prevalence of high LDL-C decreased from 59% in the late 1970s to 27% in 2007-2010 (Kuklina et al. 2013). This trend was attributed to an increased percentage of adults eating diets low in saturated fats over time (from 25% during the late 1970s to 42% during 1988-1994). The percentage of adults eating diets low in saturated fats remained unchanged from 1988-1994 to 2007-2010 (Kuklina et al. 2013).
- From the late 1980s to 2007-2010, the percentage of adults using cholesterol-lowering medication increased from 5% to 23% (Kuklina et al. 2013).

Figure 4. Trends in age-adjusted prevalence of high total cholesterol and low high-density lipoprotein cholesterol among adults aged 20 and over: United States, 1999–2000 through 2017–2018



¹Percentages prior to 2007–2008 are not presented due to changes in laboratories and methods.

²Significant decreasing linear trend from 2007–2008 to 2017–2018.

³Significant decreasing linear trend from 1999–2000 to 2017–2018.

NOTES: High total cholesterol is 240 mg/dL or more. Low high-density lipoprotein cholesterol is less than 40 mg/dL. All estimates were age adjusted by the direct method to the projected 2000 U.S. Census population using the age groups 20–39, 40–59, and 60 and over. Access data table for Figure 4 at: <https://www.cdc.gov/nchs/data/databriefs/db363-tables-508.pdf#4>.

SOURCE: NCHS, National Health and Nutrition Examination Survey, 1999–2018.

Source of Figure 4: Carroll and Fryar 2020.

With respect to heart disease specifically, the heart disease death rate for men aged 45–64 years in the US declined from 235.7 per 100,000 in 1999 to 183.5 per 100,000 in 2011. It increased to 192.9 per 100,000 in 2018. For women, the heart disease death rate declined from 96.8 per 100,000 in 1999 to 74.9 per 100,000 in 2011, and increased to 80.3 per 100,000 in 2016 before leveling off (Curtin, 2020). Incidence of CVD has also declined globally over the period from 1990 to 2017 (Amini et al. 2021).

Overall, evidence of consistent decreases in heart disease incidence and mortality rates since 1990 (and earlier) suggests improvements in CVD risk factors and interventions related to diet, physical activity, and cholesterol medications are largely successful and largely drove decreases in CVD incidence and mortality until more recent years. It seems implausible that mean PFOA or PFOS serum concentrations at levels seen in the general public in recent years contribute to increased measurable risks of CVD, given that mean TC concentrations have fallen since the 1960s while PFAS blood concentrations were more likely to be increasing until the late 1990s or 2002. It is unlikely that the benefits of decreased CVD events are detectable with further declines in average blood PFAS serum concentrations.

4.5 Conclusions and Recommendations

- The USEPA should be consistent with its recent decision to include systolic blood pressure (SBP) in the ASCVD analysis related to PFOS (which is inconsistently related to SBP) and include HDL-C in the benefits analysis based on the ASCVD model. The ASCVD model uses HDL-C, and collectively, there is some evidence that PFOA and PFOS are positively correlated with HDL-C concentrations.
- The USEPA should include a more expansive discussion of biological mechanisms for the correlation of PFOA and PFOS concentrations with TC. The mechanisms which explain

decreased cholesterol with higher PFOA or PFOS serum concentrations in animals are not likely to explain the small modest increases in cholesterol in relation to small increases in PFAS concentrations in the general population.

- The USEPA should use sensitivity analyses to further explore the potential for confounding by underlying biological processes.
- The USEPA should consider whether the quantified benefits (which are substantial) make sense within the broader context of trends over time for cholesterol levels and heart disease incidence and mortality. Cholesterol levels and heart disease incidence and mortality were decreasing even before PFOA and PFOS concentrations in the blood of the general population began decreasing (since early 2000's).

5. Overall Conclusions/Comments

- The use of a HI based on different target organs or endpoints for estimation of a regulatory value has no support in existing agency guidelines or those of other national and international authoritative bodies. The agency should delay promulgating a HI-based assessment until they have developed the necessary Target Tissue Doses (TTDs), which can readily be derived using the existing ATSDR methodology (ATSDR 2018).
- Available data suggest that further reductions of average PFOA and PFOS concentrations in blood serum are unlikely to result in measurable increases in average birth weight. Data from other areas where PFOS and PFOA concentrations are elevated suggest that increases in average birth weight and decreases in infant mortality are not expected with lower PFOS and PFOA in blood serum (Olsen and Joensen 1985; Olsen et al. 2023) found that that the mean birthweight in the Faroe Islands was higher than other Nordic countries and had increased during 2010–2019 in the Faroe Islands.
- Evidence of consistent decreases in heart disease incidence and mortality rates since 1990 (and earlier) suggests improvements in CVD risk factors and interventions are largely successful. It is unlikely that mean PFOA or PFOS serum concentrations at levels seen in the general public in recent years contribute to increased measurable risks of cardiovascular disease, given that mean TC concentrations have fallen since the 1960s. It is unlikely that the benefits of decreased CVD events are detectable with further declines in average blood serum concentrations.
- Importantly, the quantified health benefits are likely to be overstated. Although epidemiological studies have reported consistent differences in biomarkers of effect (increases in total cholesterol, decreases in antibody response, increases in certain liver enzymes or small decreases in birth weight), there are only inconsistently reported increased risks of adverse health events (e.g. frequency or duration of infections) and there is generally no evidence of increased risks of low birth weight (birth weight < 2500 g) or increased risk of cardiovascular disease, the two adverse health endpoints on which the health benefits are quantified.
- Separately, it is easy to misinterpret and overstate the health benefits potentially associated with decreasing PFOA and PFOS in drinking water by “double-counting” the benefits. As evidenced by the Faroe Islands population, there is likely to be a smaller benefit than estimated (e.g. some Faroe Islands birth cohorts showed decreases in anti-diphtheria and anti-tetanus responses in relation to PFOA and PFOS in birth cohorts that have some of the largest mean birth weights globally). In other words, any quantifiable health benefit for the population would apply to some combination of endpoints (some small benefits associated with increased birth weight, some small benefits associated with decreased cholesterol) but not the cumulative endpoints (benefits associated with decreased birth weight plus benefits associated with decreased cardiovascular diseases).

Because the actual nature of the mechanisms for the observed associations between PFOA and PFOS and the health effects are unknown, there remains substantial uncertainty in the range of the quantified benefits associated with the Proposed Option as well as the alternative regulatory options.

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December 30, 2021

Dr. Weihsueh A. Chiu, Ph.D.
Chair
Science Advisory Board PFAS Review Panel
Environmental Protection Agency
1300 Pennsylvania Ave NW
Washington, DC 20460

SUBMITTED ELECTRONICALLY

RE: AWWA Comments for Science Advisory Board PFAS Review Panel Consideration

Dear Dr. Chiu,

The American Water Works Association (AWWA) appreciates the public service provided by the Science Advisory Board (SAB) PFAS Review Panel members. The documents the Panel is reviewing will lay the foundation for the national primary drinking water regulation (NPDWR) for at least two per- and polyfluoroalkyl substances (PFAS) in drinking water and perhaps other PFAS through EPA's current effort and future rulemakings. AWWA looks forward to the SAB Panel's feedback. As the Panel is aware, the Safe Drinking Water Act (SDWA) and Executive Orders set a clear bar for transparent analysis and use of sound science in setting NPDWRs. AWWA appreciates the Panel's assistance to EPA in this process. AWWA offers the following comments for the Panel's consideration in its deliberation and for the Agency as it addresses the Panel's review.

Sufficient Resources are Needed to Ensure Scientific Integrity

Under the SDWA, the EPA has a responsibility to use the best-available science in accordance with sound and objective scientific practices. Doing so is imperative in ensuring that drinking water contaminants are addressed in a meaningful way that protects the public and can be implemented feasibly. As EPA has already presented and discussed during the Panel's Dec. 16 meeting, there are five significant draft documents for your consideration relating to the draft approaches for developing a perfluorooctanoic acid (PFOA) MCLG and a perfluorooctanesulfonic acid (PFOS) MCLG, evaluating cardiovascular health effects of PFAS, and evaluating non-cancer health risks associated with PFAS mixtures. This represents a substantial body of work for the Panel to review and on which to provide feedback.

The SAB PFAS Review Panel's report of recommendations is anticipated to be completed in May 2022. If EPA adheres to the schedule set in its PFAS Strategic Roadmap, this leaves less than six months for the Agency to review these recommendations, implement appropriate changes, incorporate resulting differences into its cost analysis, craft a regulatory proposal, and complete the associated procedural

requirements for proposing rulemakings. Normally one-third of that six-month period would be taken up by inter-Agency review. This schedule is well within EPA's statutory deadline for proposal of the rule. AWWA urges the EPA Administrator to afford the SDWA program staff sufficient resources to act on your review so that the proposal is based on the best available science and that the analyses are sound. This is a critical rulemaking for the program, and the Administrator must be sure that EPA employs a defensible premise for benefits anticipated to be accrued through this rulemaking.

Charge Questions and Ensuring Recommendations Reflect Purpose of Documents

On Dec. 16, EPA presented the Panel with its intended use of the documents being reviewed. The Agency made clear why it included five economists in a Panel charged with reviewing scientific assessments of health data. Unfortunately, the final charge questions distributed to the Panel do not address the key questions EPA must answer or make best use of the expertise of the economists on the Panel.

To inform its deliberations, AWWA recommends that the Panel request a briefing from the National Center for Economic Analysis on the construction of benefits analyses to support SDWA regulatory standard setting. Such a briefing would provide the Panel members a common understanding of the task before EPA using the materials the Panel is reviewing. With that basis, the panelists with expertise in national benefit analysis would be able to speak to the strengths and weaknesses of the Agency approach for purposes of the benefits analysis. For example, the Agency is positing in one of its analyses that a fraction of cardiovascular disease in the United States is attributable to PFOA and PFOS. The U.S. Centers for Disease Prevention and Control currently estimates that the mortality rate for diseases of the heart in the United States is 200.8 per 100,000. There are recognized risk factors, some of which have marked socio-economic correlations. It is important that EPA (1) neither grossly over- or under-estimate benefits from risk reduction in its rulemakings and (2) understand and communicate how assumptions and uncertainties in its analysis impact use of the risk reduction model. The plausibility of the Agency's analytical approach being adequate to underpin an economic analysis for an SDWA primary standard warrants discussion.

In-Depth Review of the Documents Provided for Panel Review

AWWA contracted with Ramboll U.S. Consulting, Inc. (Ramboll) to prepare a review of the documents before the Panel and compile comments relative to the charge questions posed to the Panel. The scientists at Ramboll included experts in both cancer and non-cancer health risk assessments, physiologically based pharmacokinetic modeling, and epidemiological research. A summary of the review by Ramboll scientists is attached and is organized to align with the Agency's charge to the Panel. Some key points are:

1. EPA did not apply the Agency's current systematic review process to all the studies it utilized; studies central to its quantitative analysis were likely not held to the expectations applied in the current systematic review process.
2. The evidence for a causal relationship between PFOA or PFOS exposure and cardiovascular disease is weak. It is plausible that PFOA and PFOS exposure is associated with higher cholesterol levels but without an increased risk of cardiovascular disease. Because epidemiological evidence of increased risks of cardiovascular disease in relation

to PFOA and PFOS exposure is weak, it is currently speculative to assume that the small increases in total cholesterol or low-density lipoprotein cholesterol (LDL-C, the “bad” cholesterol) are causally related to increased incidence of cardiovascular disease.

- EPA utilizes studies based on an apparent association between PFOA and PFOS exposure and total cholesterol/ LDL-C when the weight-of-evidence is limited and there are scientific reasons to suspect the association is not meaningful for EPA’s analysis.
 - Furthermore, if PFOA and PFOS are associated with small increases in high density lipoprotein cholesterol (HDL-C), it is biologically plausible that the risks of cardiovascular disease remain unchanged. HDL-C is considered to be the “good” cholesterol and higher levels of HDL-C are associated with a decreased risk of cardiovascular disease. The EPA should consider this as part of the analysis.
 - The analysis presented in the cardiovascular disease risk reduction document concludes that PFOA and PFOS levels lead to an increase in cholesterol. This is not a certainty, and it is possible that the correlation between PFOA and PFOS levels and cholesterol levels is more likely related to the transport mechanisms of cholesterol and PFOA and PFOS within the body.
3. Shearer et al. (2021), one of the key studies in EPA’s analyses, should not be used as a basis for either cancer characterization or dose-response assessment. Despite analyses that adjusted for estimated glomerular filtration, there is the possibility of additional confounding by effects of the underlying cancer induction processes on other aspects of kidney function, such as the renal transporters that are required for control of PFOA excretion, that could also lead to higher PFOA blood concentrations. In addition, the maximum latency in the study was 18 years since the blood collection, which is generally inadequate for kidney cancer, which has a long disease latency. Separately, there is an apparent anomaly between the number of cases and controls in the referent category of exposure (<4 ng/mL) which may lead to under- or over-estimated odds ratios in the higher quartiles of exposure.
 4. EPA’s analyses for PFOA and PFOS rely upon a half-life from a study of retired workers exposed occupationally (Olsen et al., 2007) and thought to have been exposed intermittently since retirement. Recent work by the Alliance for Risk Assessment concluded that the most appropriate studies support a much lower half-life. EPA acknowledges the study’s shortcomings but does not provide reasoning for nonetheless using the higher half-lives.
 5. EPA’s analyses rely on human epidemiological studies with published findings of reduced vaccination efficacy based on cohorts in the Faroe Islands. There are several concerns associated with these studies relating to the level of clinical protection, inconsistencies regarding study subjects included in various studies, and confounding resulting from contaminants (e.g., polychlorinated biphenyl, methyl mercury) anticipated to be high in

the Faroese diet. Should the EPA decide to use these studies, the data should be obtained from the study investigators and independently evaluated prior to finalizing the Agency's analysis.

6. Several key aspects of these analyses are not presented for the SAB PFAS Review Panel and the public to review and verify, including:
 - Neither EPA's animal nor human pharmacokinetic model files were made available for public verification. Without these model files, the model cannot be verified. Lack of access to files also limits the ability to evaluate the pregnancy and lactation model. All model files (including R scripts) should be made available for review by the SAB PFAS Review Panel, as well as the public, to provide scientific transparency.
 - The source for the milk ingestion for animal pups was not fully documented, nor was it peer reviewed. EPA should fully describe the basis for milk ingestion in the PFOA and PFOS documents to adequately support the Panel's review.
7. The draft framework for assessing non-cancer health effects of PFAS mixtures is a significant improvement upon previous approaches applied by other regulatory agencies. However, the document relies on the conclusion that dose additivity occurs for chemicals with a similar toxic endpoint. This begs the question of "How similar?". While chemicals may share a common toxicity endpoint, if the health effects vary, then it becomes necessary to define sub-classes of chemicals for which dose-additivity is appropriate.

If you have any questions regarding this correspondence or if we can be of assistance in some other way, please contact Chris Moody (202.326.6127, cmoody@awwa.org).

Best regards,

FOR THE AMERICAN WATER WORKS ASSOCIATION



G. Tracy Mehan III
Executive Director – Government Affairs
American Water Works Association

cc: Suhair Shallal Al-Mudallal, EPA/SAB
Al McGartland, EPA/OP/NCEE
Erik Helm, EPA/OW
Betsy Behl, EPA/OW/OST
Jennifer McLain, EPA/OW/OGWDW
Eric Burneson, EPA/OW/OGWDW/SRMD

Attachment (1)

Who is AWWA

The American Water Works Association (AWWA) is an international, nonprofit, scientific and educational society dedicated to providing total water solutions assuring the effective management of water. Founded in 1881, the Association is the largest organization of water supply professionals in the world. Our membership includes more than 4,500 utilities that supply roughly 80 percent of the nation's drinking water and treat almost half of the nation's wastewater. Our 50,000-plus total membership represents the full spectrum of the water community: public water and wastewater systems, environmental advocates, scientists, academicians, and others who hold a genuine interest in water, our most important resource. AWWA unites the diverse water community to advance public health, safety, the economy, and the environment.

ATTACHMENT 1
Technical Expert Review of
Agency Review Documents Relating to PFAS Health Risks in Drinking Water

**Technical Expert Review of
Agency Review Documents Relating to PFAS Health Risks in Drinking Water**

Prepared by
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December 23, 2021

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PROPOSED APPROACHES TO THE DERIVATION OF A DRAFT MAXIMUM CONTAMINANT LEVEL GOALS FOR PFOA AND PFOS IN DRINKING WATER

General Comment: *As noted in the background sections for both documents, because PFOA and PFOS are listed on the Fourth Drinking Water Contaminant Candidate List (USEPA 2021), EPA made a determination to regulate PFOA and PFOS with a National Primary Drinking Water Regulation. While there are separate documents to discuss the approach for the development of the Maximum Contaminant Level Goal (MCLG) for each compound, there is a lot of information focused on the combination of these compounds or additional PFAS as a class of compounds. For example, in the Occurrence Summary (Section 1.4), examination of the occurrence relies upon the data from the third Unregulated Contaminant Monitoring Rule (from 2013-2015; Section 1.4) and is focused on the occurrence of PFOS and PFOA in aggregate, summing concentrations. This concept, as well as others throughout the documents, should be focused on each compound separately when considering data in the estimation of the individual MCLGs for each compound.*

Study Identification and Inclusion

1. EPA used systematic review methods consistent with the current ORD systematic review practice to ensure transparency and completeness of literature identification, sorting, and study quality evaluation. Is the process clearly described? Please identify additional peer-reviewed studies that the panel is aware of that could inform toxicity value derivation.

Comment: *As noted in both the PFOA and PFOS documents, EPA has built upon the data included and analyses conducted as part of the 2016 Health Effects Support Documents (HESD) for the Health Advisories for each compound. In the identification of relevant studies, EPA conducted broad literature searches focused on the chemical name/synonyms with no limitations on lines of evidence (Appendix A of the documents). Therefore, any relevant study published since the 2016 HESDs should have been identified.*

EPA has also noted in both the PFOA and PFOS documents that all studies relied upon for quantitative analysis were not put through the same systematic review process. Many of the epidemiological and animal studies are qualitatively incorporated into this assessment based on the HESD. Specifically, EPA notes that only the animal studies supporting the candidate Reference Doses (RfDs) derived in the 2016 HESDs were incorporated into the systematic review methods outlined in the current SAB External Peer Review Draft MCLG documents. EPA notes that all other studies referenced from the 2016 HESD adhered to the specific criteria for inclusion in the 2016 HESDs, but study confidence between the studies included in the 2016 HESD and this assessment cannot be compared. Therefore, only the animal studies supporting the candidate RfDs derived in the 2016 HESD were considered quantitatively in this assessment.

It is important that all of the studies relied upon quantitatively be put through the same evaluation process to determine study confidence and quality. Because processes have changed since the 2016 HESDs, the requirements for a high-quality study may have changed. EPA (2021a, b) indicates that the current systematic review processes have been applied to the animal studies being used quantitatively and this would also be important for the epidemiological studies. The epidemiological studies were the focus and played an integral role in the assessment, with the animal studies

providing support. In reviewing all of the studies considering quantitatively, it is also important for EPA to confirm that the majority of the epidemiological studies relied upon for quantitative analysis in the current SAB External Peer Review Draft documents are more recent than the 2016 HESDs and therefore, should have been put through the same systematic review processes. Because there are a limited number of epidemiological studies that are pre-2016, it would not be a large effort to put these studies through the same critical review to ensure that all confidence ratings are comparable.

Noncancer Hazard Identification

1. Please comment on the health effect/outcome categories identified from the review of the available literature. Do you agree with the strong vs. suggestive evidence designations for the various health outcome categories? Do any other health systems or endpoints need to be considered for POD derivation?

Comment: *The most striking aspect of the EPA review of the health effect/outcome categories identified from the PFOA and PFOS literature is that, while the evidence is characterized as suggestive for many endpoints, there is only one health outcome where the EPA characterizes the evidence as strong: an apparent association of PFOS exposure and Total Cholesterol/LDL-C (but not directly with CVD). Even in this case, the PFOA document does not characterize the association as strong. These equivocal characterizations reflect the fact that, despite the large number of studies that have been carried out on PFOA, the evidence from animal studies is almost exclusively from studies conducted with dosing in the range where effects may be associated with activation of PPAR-mediated disruption of lipid metabolism, which is not relevant to the much lower exposures experienced by human populations. On the other hand, the evidence from epidemiological studies is highly susceptible to confounding by pharmacokinetic interactions between the health outcome being studied and the transport/excretion of PFAS (Andersen et al. 2021a,b, open access).*

Reduced birthweight provides an example of the potential for unrecognized confounding in epidemiological studies with PFAS. As cited by EPA, numerous studies of human populations have reported small decreases in birth weight in relation to increasing PFOA and PFOS blood concentrations. However, the C8 Science Panel evaluated the epidemiological evidence in 2011 and concluded that there was not a probable link between exposure to PFOA and low birth weight (C8 Science Panel 2011; Stein et al. 2009; Savitz et al. 2012; 2011b). Since then, additional epidemiological studies have reported small reductions in infant birth weight (less than 20 grams per ng/ml increase of PFOA or PFOS) (ATSDR 2021). Steenland et al. (2020) recently reviewed literature published since 2011. They attributed the association between PFOA or PFOS and decreases in birth weight as possibly due to reverse causality or confounding. Studies with insufficient exposure contrast (i.e., low exposures with little variability), such as those in the general population (in the absence of drinking water contamination) are particularly prone to distorted effects due to reverse causality and confounding. Stronger associations with birth weight are seen in studies when PFOA is measured later in pregnancy (Steenland et al. 2018; Apelberg et al. 2007; Chu et al. 2020). When PFOA is measured earlier in pregnancy, the associations with birth weight are largely null (Darrow et al. 2013; Manzano-Salgado et al. 2017; Steenland et al. 2018). In addition, most epidemiological studies that specifically evaluated the risk of low birth weight (that is, birth weight <2500 grams) have reported null associations with increased concentrations of PFOA or PFOS (Savitz et al. 2012a; 2012b; Darrow et al. 2013; Stein et al. 2009; Chen et al. 2012; Manzano-Salgado et al. 2017).

These findings are consistent with the hypothesis that the apparent association between PFOA and birth weight is confounded by the magnitude of plasma volume expansion during pregnancy and glomerular filtration rate (Steenland et al. 2018; Verner et al. 2015). Evaluations of potential pharmacokinetic bias have demonstrated that associations between prenatal exposure to PFAS and lower birth weight in epidemiological studies may actually be driven by changes in glomerular filtration, which increases by about 50% during the first half of pregnancy followed by a slight decline in the second half (Andersen et al. 2021a). Studies have shown that women with less of an increase in GFR tended to have smaller babies (Gibson, 1973; Morken et al., 2014). Verner et al. (2015) used a PBPK model to run simulations of a study population and to generate pairs of predictions for PFAS level and birth weight. Results obtained from simulated PFAS levels and birth weights were compared with published epidemiological studies to evaluate how much of this association might be attributable to the influence of GFR. The analysis used a detailed PBPK model of PFOA and PFOS during pregnancy (Loccisano et al., 2013) that was modified to describe the association of GFR with birth weight. The model was then used to simulate study populations exposed to PFOA or PFOS and to predict the resulting distributions of concentrations in maternal and cord plasma. Results from Monte Carlo PBPK model simulations (of longitudinal data) indicated that even controlling just for the effect of GFR changes accounted for the majority of the association of PFOA and PFOS with reduced birth weight.

2. Elevation of liver serum biomarkers in humans is frequently used as an indication of liver injury, although it has not been shown to be as specific as functional tests, such as histology findings and liver disease (Boone, 2005, HERO ID: 782862). However, greater than 2-fold increases in alanine aminotransferase (ALT) activity, the most sensitive test of hepatocellular injury in humans, above the upper limit of normal are considered indicative of hepatocellular injury. EPA concluded that the available data in adults show a consistent positive association between PFOA and/or PFOS exposure and increased serum ALT levels in the epidemiological literature. However, this response was not selected for dose response modeling because 1) the magnitude of the effect was not large compared to control levels; and 2) concerns about the clinical relevance of the findings and non-specificity of the biomarkers relationship to adverse liver injury and disease.

Comment: EPA concerns regarding the clinical relevance of the small increases in ALT reported in the epidemiological literature and the non-specificity of ALT as a biomarker of liver injury/disease are justified. They would not be appropriate as a basis for setting a quantitative exposure guideline.

3. Does the SAB panel agree with EPA's rationale for not considering the ALT endpoint reported in the epidemiological studies for the derivation of a POD for the liver health effects? Please provide your justification and if you suggest that EPA consider this endpoint for POD derivation, please provide your recommendations for a modeling approach.

Comment: The decision not to select ALT for dose-response modeling is appropriate due to the questionable clinical relevance of the small increases in ALT reported in the epidemiological literature and the non-specificity of ALT as a biomarker of liver injury/disease particularly. The relevance of these observations of small changes in ALT is of particularly questionable in the case of PFAS, due to the likelihood that the associations may be secondary to a reverse relationship between altered liver function and the control of PFAS transport/excretion, where individuals with impaired liver function may have reduced transport/clearance of PFAS, resulting in relatively higher blood

concentrations compared to healthy individuals (similar to the case of impaired kidney function, described in the comment on cancer classification below).

- A. Are you aware of additional studies that support the ALT levels as markers of adverse liver effects? Please provide citations.

Comment: *No.*

- B. Are there other adverse liver endpoints identified in the epidemiological literature that need to be considered?

Comment: *No.*

Cancer

1. Cancer classification for PFOA/PFOS

- A. PFOA: Based on new cancer studies identified since the 2016 PFOA Health Advisory (HA), EPA concludes that the available cancer data for PFOA indicate a ‘likely carcinogen’ categorization which is a change from ‘suggestive’ in the 2016 HA. Does the panel agree with the ‘likely’ designation based on the new evidence? If yes, is the rationale clearly described? If no, please provide an explanation for arriving at a different conclusion.

Comment: *The EPA’s proposed change in the categorization of PFOA from “suggestive evidence” to “likely carcinogen” is not justified. The EPA’s determination appears to be based on an epidemiology study reporting an association between PFOA concentrations and incidence of renal cell carcinoma (Shearer et al. 2021). PFOA (median 5.5 ng/mL sampled during 1993-2001) was measured in blood serum at least 2 to 18 years before diagnosis of kidney cancer; given the half-life of <2 years (see comments under Toxicokinetics section), a single PFOA measurement is unlikely to accurately portray the exposure relevant to the development of disease. In epidemiological studies of higher exposures, there is inconsistent evidence of increased kidney cancer risk. Epidemiological studies of residents exposed to PFOA and other PFOS in contaminated drinking water have reported modest increases in kidney cancer (Li et al. 2022; Vieira et al. 2013). Studies of occupational cohorts have been inconsistent, with one cohort showing decreased risk of kidney cancer (Raleigh et al. 2014) and another cohort showing increased risk of kidney cancer (Steenland and Woskie, 2012); however, the number of kidney cancer cases or deaths in the occupational cohorts have been relatively few, and the investigators have cited low statistical power to draw conclusions regarding the reported associations. Nevertheless, if there were a strong causal association between PFOA or PFOS exposure and kidney cancer, it would be expected that much higher estimates of relative risk (a magnitude of 3-fold or more) would be seen in occupational cohorts who were exposed to PFOA at much higher concentrations than the general population and who were followed for more than 30 years on average (Raleigh et al. 2014; Steenland and Woskie 2012). Separately, kidney cancer is frequently associated with impaired kidney function. Lower renal function (calculated as estimated glomerular filtration rate (eGFR)) is likely to result in decreased PFOA excretion and a consequent increased concentration in serum. Cross-sectional analyses of adults exposed at background levels (Shankar et al. 2011) and of children exposed at high levels (Watkins et al. 2013) found a positive*

association between lower kidney function (i.e., lower eGFR) and higher measured serum PFOA. Dhingra et al. (2016), performed an analysis of cross-sectional studies reporting associations between PFOA and renal function, and concluded that reverse causation led to the observed associations. Shearer et al. (2021) reported that a higher percentage of cases (9%) than controls (5.6%) had diminished kidney function; however, the overall difference in kidney function between cases and controls was not statistically significant when kidney function was stratified by normal ($\text{eGFR} \geq 90 \text{ mL/min/1.73 m}^2$), mild loss ($\text{eGFR} 60\text{--}89 \text{ mL/min/1.73 m}^2$) or diminished kidney function ($\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$). In sensitivity analyses, Shearer et al. (2021) stratified by kidney function and separately restricted analyses to study subjects with high kidney function. In both analyses, the odds ratios for kidney cancer were statistically significantly increased for PFOA exposure; however, it is not clear that eGFR based on a single sample collected 2 to 18 years before the diagnosis of kidney cancer falls within a relevant time window for kidney cancer induction and latency associated with impaired kidney function. Steenland and Vieira (2021) reviewed these studies (including the Shearer et al. 2021) and concluded that the evidence from epidemiologic studies of PFAS in relation to cancer “remains limited.”

- B. PFOS: Based on a small number of new cancer studies identified since the 2016 PFOS HA, EPA concludes that the available cancer data for PFOS indicate a ‘suggestive’ categorization which is unchanged from the categorization identified in the 2016 HA. Does the panel agree that the new studies do not change the designation? If yes, is the rationale clearly described? If no, please provide an explanation for arriving at a different conclusion.

Comment: The decision to continue the ‘suggestive’ categorization is appropriate. Although Shearer et al. (2021) also reported statistically significantly increased odds ratios for kidney cancer when PFOS was measured as a continuous variable, the odds ratios did not increase with increasing exposure when PFOS exposure was categorized. After adjusting for PFOA and PFHxS, there was a non-significantly decreased odds ratio for kidney cancer (OR 0.92, 95% CI 0.60–1.42).

2. Cancer Slope Quantification: EPA used the Shearer et al., 2021 epidemiological study to quantify a cancer slope factor using peak exposure for PFOA. Has EPA adequately justified the use of this study and peak exposure for the quantification of a cancer slope factor for PFOA? If no, please describe alternate approaches that SAB recommends.

Comment: The EPA relies extensively (and exclusively, for epidemiological studies generated since the 2016 HESD) on the Shearer et al. (2021) study. However, there are earlier studies of occupational groups exposed to much higher exposures that were discounted largely because of small numbers of cases and questions about whether the studies had adequate power to detect an excess cancer risk if one existed (Steenland and Woskie et al. 2012; Raleigh et al. 2014). See response to Cancer Classification.

- (i) Use of Shearer et al. (2021) study: For purposes of deriving the cancer slope factor, the EPA estimated the dose-response between PFOA and kidney cancer using a weighted linear regression of the quartile-specific odds ratios where the weights were inverse variance of each OR. Although Shearer et al. (2021) reported a statistically significant increased risk of renal cell carcinoma when exposure was modeled as a continuous variable. In a separate categorical analysis, an exposure-response relationship was not seen, that is, the odds ratios did not increase with increasing exposure. In the analysis that adjusted (OR 1.71, 95% CI 1.23–2.37) after

adjusting for body mass index, smoking status, history of hypertension, estimated glomerular filtration rate, previous freeze-thaw cycle, and calendar year of blood draw, the OR was statistically significant for the highest quartile of PFOA exposure only.), a separate categorical analysis did not show an exposure-response relationship. The odds ratios did not increase with increasing exposure, although the OR was increased for PFOA in blood serum concentrations >7.2 to 27.2 ng/mL when compared to concentrations <4.0 ng/mL. After further adjustment for exposure to other PFAS, the OR for the 4th quartile was attenuated, and was not statistically significant. (see Table below). The p-for-trend was 0.13 (not significant). The Shearer et al. (2021) study should not be used to derive a POD for calculating a cancer slope factor.

Shearer et al. (2021)

Cases / Controls	ng PFOA/mL blood	OR	95% CI
47 / 81	<4.0	1.00	Reference
83 / 79	≥4.0 – 5.5	1.41	0.69–2.90
69 / 83	>5.5 – 7.3	1.12	0.52–2.42
125 / 81	>7.3 – 27.2	2.19	0.86–5.61
	Continuous	1.68	1.07 – 2.63

Study participants were 55-74 years at time of blood draw. Serum samples were collected at a single point in time (before diagnosis of kidney cancer). Kidney cancers were diagnosed on average 8.8 years after the blood draw (range, 2-18 years). In comparison, Steenland and Woskie (2012) studied mortality among 5,791 workers exposed to PFOA during 1952 to 2004. These workers had mean duration of employment of 19 years, a mean duration of follow up of 30 years, and an estimated mean annual serum concentration of 350 ng/mL. When kidney cancer mortality was compared to other workers in the same region, there was no exposure-response relationship when PFOA was categorized according to quartiles of cumulative PFOA exposure. Although an excess of kidney cancer deaths was seen for workers exposed to ≥1,819 ng/ml-years (the 4th quartile) when exposure was lagged by 20 years to account for latency, the SMR was decreased for the second quartile and there were no kidney cancer deaths that occurred in the third quartile of exposures (Steenland and Woskie, 2012). Although some have argued that the results from the worker studies are limited because workers are healthier than adults in the general population, the healthy worker effect is not a significant source of bias when evaluating cancers with long latencies (Checkoway et al, 2014).

Under a hypothetical assumption of 30 years of exposure to 15 ng/mL (which represents a mid-range estimate from the highest quartile of exposure (>7.3 to 27.2 ng/mL) in Shearer et al. (2021), the cumulative exposure would be 450 ng/mL-years for cases and controls in the highest quartile. These cases from the Shearer et al. (2021) population would fall within the lowest occupational exposure category from the Steenland and Woskie (2012) workers (1st quartile, 0 to <515 ng/mL-years, when exposure was lagged 20 years to allow for an appropriate induction and latency for kidney cancer). Among workers exposed to PFOA, three kidney cancer deaths were reported (compared to 2.2 expected, SMR 1.34, 95% CI 0.28 – 3.91) (Steenland and Woskie et al. 2012). Raleigh et al. (2014) relied on a job-exposure matrix to estimate individual concentrations of APFO in micrograms per cubic meter of air, rather than drawing blood for analysis. Although these estimates of inhalation exposure reported by Raleigh et al. (2014) cannot be directly converted into serum PFOA concentrations, other studies of workers that overlapped with the Raleigh et al. (2014) cohort (Olsen et al. 2003; Olsen and Zobel, 2007) reported very high serum concentrations of PFOA in the blood of workers (>1,000 ng/mL) and at concentrations higher than those reported by Steenland and Woskie (2012). In contrast to

Steenland and Woskie (2012), Raleigh et al. (2014) did not find an increased risk of kidney cancer in the highest category of exposure (4th quartile, hazard ratio (HR) 0.73, 95% CI 0.21–0.48) when compared to the non-exposed population based on four incident kidney cancers. There was also no increased risk of kidney cancer when the 3rd and 4th quartiles were combined (8 kidney cancers, HR 0.85, 95% CI 0.36–2.06).

Shearer et al. (2021) chose quartile cut-points based on the serum PFOA exposure in the controls. There was, however, a substantial difference in the number of kidney cancer cases (n=47) and controls (n=81) for the lowest exposures (1st quartile, <4.0 ng/mL). This concentration is similar to the geometric mean concentration for PFOA (4.8 ng/mL for participants age 60 years and older) in a nationally representative sample (NHANES) collected during 1999–2000. It seems unlikely that there would be a discrepancy in kidney cancer incidence in a population exposed at background levels and there is no logical explanation for a deficit in kidney cancers at these low concentrations. The clear discrepancy between the number of cases and controls in the referent category may potentially create a spurious association between PFOA and kidney cancer when higher quartiles of exposure are compared to the referent group (1st quartile of exposure).

The Shearer et al. (2021) study does not consider cancer induction and latency, which is a study limitation, especially considering population-level exposures. Presumably, high exposures in occupational groups would result in a shorter latency than population level exposures; however, inconsistent associations between PFOA and kidney cancer have been reported in workers exposed to much higher concentrations and followed for 30 years or more (Raleigh et al. 2014; Steenland and Woskie, 2012). Solid-tumor cancers are unlikely to have cancer induction and latency periods that are shorter than 20 years at higher exposures. For example, Smith et al. (2018) reported latency periods for kidney cancer were at least 20 years following exposure to arsenic in drinking water and remained elevated after 40 years of follow up. Because the time to diagnosis of kidney cancer ranged from 2 to 18 years since blood in Shearer et al. (2021), an inadequate latency for cancer development raises the possibility of reverse causality or confounding, i.e., impairment of kidney function associated with renal cell cancer induction results in increased concentrations of PFOA. Dhingra et al. (2017) reported that decreased renal function (lower glomerular filtration rate) led to higher PFOA concentrations. Decreased renal function is expected as kidney cancer develops, although it may not be noticed before clinical manifestation of kidney cancer. Although Shearer et al. (2021) adjusted their results for estimated glomerular filtration rate (eGFR), the estimate was based on a single blood serum sample, which is inadequate for an evaluation of glomerular filtration rate. Moreover, glomerular filtration is only one of the many kidney functions that control the excretion of PFOA; multiple renal transporters, including the basolateral and apical organic acid transporters and the urate transporter, are also involved in the regulation of PFOA excretion, and hence blood levels.

- (i) Use of peak exposure: Peak exposure was not discussed as an exposure metric in Shearer et al. (2021), and a search of the EPA PFOA MCLG document did not identify any statement to indicate that peak exposure was used in the EPA analysis. While the California Environmental Protection Agency's Office of Environmental Health Hazard Assessment (OEHHA) used peak concentration in their derivation of a Public Health Goal (PHG) for PFOA, and it seems likely that the EPA followed a similar approach, there is no citation to the OEHHA PHG document in the EPA MCLG document. The*

uncertainty regarding the approach taken by EPA represents a serious lack of transparency in the document.

The use of peak exposure is not appropriate for a chemical like PFOA, which has a half-life on the order of a year or more. With such a long human half-life, it can take as much as a decade of continued exposure at a given intake to produce a corresponding change in target tissue concentration, so the peak internal exposure would not be well represented by the peak intake. There is also no rationale for using peak exposure as the dose metric for renal cell carcinoma from human exposures to PFOA on the basis of potential nonlinear pharmacokinetics or pharmacodynamics of PFOA over the range of human exposures. Therefore, the time-weighted average exposure should be used as the dose metric for the analysis, or cumulative exposure (that is, the product of intensity and duration) as used by Steenland and Woskie (2012).

Toxicokinetic Models

1. Human model –

- A. For endpoints observed in adults, EPA used a steady-state approach to calculate the HED, which assumes a relatively constant exposure and clearance during adulthood. Please comment on this method of HED calculation. Are there alternative approaches that EPA should consider? If so, please describe the rationale for recommending this approach(es).

Comment: *The use of steady-state to calculate HED is an acceptable approach for cross-species extrapolation of the key model derived points of departure in this document. For longer half-lived PFAS compounds like PFOA and PFOS, it is unlikely that event-based approaches would yield different results. This may not be the case for PFAS with shorter half-lives.*

- B. Two key parameters are the half-life and volume of distribution, which were used to calculate clearance. Half-life and volume of distribution were assumed to be constant across sex and age groups because of a lack of strong quantitative data to parametrize changes across sex and age. Please comment on the strengths and weakness of the use of this assumption and the choice of these parameters by the EPA. Please describe the rationale for alternative recommended approaches. For endpoints observed in human neonates or children, EPA used a one-compartment TK model to simulate dosimetry during pregnancy and a two-compartment TK model (one-compartment models for the mother and the child) to simulate dosimetry during lactation, to calculate the HED for each POD. Please comment on the strengths and weaknesses of this choice of model structure for the task of predicting dosimetry in the human fetus and child compared to dosimetry in mice and rats in the similar lifestyles. Please provide the rationale for any alternative recommended approaches.

Comment: *The use of Thompson et al. (2010) for the Volume of Distribution (VD) for PFOA and PFOS is appropriate for human and the animal. However, the half-lives for humans used by EPA for the clearance calculation was 3.8 years for PFOA and 5.4 years for PFOS, derived from studies of retired workers (Olsen et al. 2007). Given the extensive discussion in the MCLG document of additional studies reporting the half-life of PFOA in environmentally exposed populations, it is unclear why the half-life from an occupational study that included*

only a small number of retired workers was chosen over other reported values for larger populations exposed at environmental levels, including the more accepted half-life value of 2.3 years reported by Bartell et al. (2010) study. The Zhang et al. (2013) study, which included the collection of urinary data to support an estimate of renal clearance, and which was reviewed in the EPA's half-life discussion, also appears to support the results of the Bartell that the 3.8 year half-life is incorrect. These studies provide a strong argument against using the half-life reported in Olsen et al. (2007), which is thought to include subjects that intermittently had occupational level exposures after retiring.

A recent review of studies with human PFOA half-life information by an international collaboration sponsored by the Alliance for Risk Assessment (Campbell et al., 2022) concluded that the results from the most appropriate studies support a PFOA half-life in the range of 0.5 to 1.7 years, indicating that the half-life of 3.8 years (Olsen et al. 2007) is much too high. Verner et al. (2016) did not justify the use of 3.8 years nor did they evaluate the impact of the shorter estimated half-lives in their analysis and noted that the "much greater ratio of estimated intakes of PFOA may be partially due to the half-life we used (3.8 years); others have suggested a lower value (e.g., 2.3 years)." The Agency discusses this in their half-life review but does not provide reasoning as to why they nevertheless used the 3.8-year half-life from the model in Verner et al. (2016). A broader evaluation of the half-lives reported for PFOA would indicate that, when accounting for continued exposure, the half-life is likely to be below 2 years. The Olsen et al. (2007) study also serves as the basis for the PFOS half-life used in the Verner et al. (2013) model by the EPA; it is likely that the correct half-life for PFOS is also much shorter than the value used by EPA.

- C. The key chemical-specific parameters that describe the transfer of the chemical from the mother to the child during gestation and lactation are the maternal to fetal serum ratio and the ratio of maternal serum to milk PFOA/S concentration. These ratios were assumed to be constant during gestation and lactation, respectively. Another important parameter is the rate of milk ingestion, which is chemical-independent and varies throughout lactation. Please comment on the strengths and weaknesses of the choice of parameters for fetal to maternal partitioning and partitioning into breastmilk, as well as the choice for lactation rate. Please also comment on the choice to assume that fetal to maternal partitioning and partitioning to breastmilk did not vary in time. Please describe whether there are other methods you would recommend to account for these changes over time and across development.

Comment: *The use of the serum to milk and serum to fetus ratios is a default approach that, while increasing uncertainty in the fetal/lactational modeling, is not unreasonable. The application of the model to the available time-course data in the rat during lactation indicates that the approach was valid for PFOA and PFOS.*

2. Animal Model –

- A. After a review of the available toxicokinetic models for PFOA/S predictions in laboratory animals, EPA selected the Wambaugh et al. (2013) model because it was parametrized using all species of interest, demonstrated good agreement with training and test datasets, and used a single, biologically motivated, model structure across all species. Does the panel agree with selecting this model? If not, please describe the rationale for alternative recommended approaches for the calculation of the internal dose metrics in adult animals.

Comment: *The choice of a simple PK model that accounts for saturable resorption is reasonable, and the approach taken (e.g., choice of dose metrics) in deriving the animal dose metrics, as well as the PODs in humans, are reasonable. Based on the model fits presented in the PFOA and PFOS documents, the models provide acceptable fits to the data overall. However, neither the animal nor the human PK model files were available to allow verification of the EPA's implementation of the published models that they actually used to derive the dose metrics. The EPA routinely verifies the code of submitted models, and the same possibility should be provided to the public for agency model code. All model files (including R scripts) should be available for review by the SAB, as well as the public, to provide full disclosure.*

- B. The animal model parameters were obtained through a Bayesian inference parameterization which produced wide credible intervals for some parameter values, but relatively tight credible intervals for the predicted serum concentration. Does the panel agree with using the median values of the estimated animal parameter distributions for prediction of serum concentration and internal dose metrics?

Comment: *Median values from Bayesian inference using a PK model are reasonable to the extent that the PK model adequately captures the kinetic data used in the calibration and the implementation of the models by EPA are error-free. (see previous).*

- C. Based on visual inspection of model predictions to the calibration datasets, EPA utilized sex-independent parameters for PFOS. The male-specific parameters were used for all rat-specific PFOS predictions including predictions in pregnant and nursing dams and the female-specific parameters were used for all mouse-specific PFOS predictions because the parameter values obtained from fitting the female-specific rat data and male-specific mouse data were not consistent with the overall TK parameters for PFOS and produced poor fits to the training and test datasets. Does the panel agree with this approach and justification for this assumption for PFOS? If not, please describe other approaches that could be considered?

Comment: *While it is reasonable to suggest that the kinetics of PFOS in male and female rat are similar, the impact of that assumption on the simulations of the Chang et al (2012) study presented in Wambaugh et al. (2013), and of the Kim study shown in supplemental E (Figure E-7 left panel) should be presented. Wambaugh et al. (2013) did not show the model fit to the female rat and the agency has not included simulations using the Wambaugh et al. (2013) model in Appendix E. Wambaugh et al. (2013) did not discuss their use of gender-specific parameters for PFOS in the rat, which had not been required in the previous modeling efforts. It is not possible to fully evaluate whether there is an error in the approach taken with the PFOS animal modeling, because there is a less than full documentation of the EPA model.*

- D. EPA assumed a one compartment model for the developing infant based on the lack of infant-specific toxicokinetic data from rats and mice. This model utilizes averages of half-life and volume of distribution from the literature coupled with physiologically relevant lactational parameters for pup nursing. Does the panel agree with the decision to use this model structure for infant animals? If not, please provide data on infant-specific changes during the animal lactational-period that could be used to account for toxicokinetic differences between the adult and infant rats and mice.

Comment: *The model structure for the nursing pup is reasonable. The potential issues noted for the adult animal; however, could lead to issues with the prediction of fetal dose metrics. Given the adult model cannot be fully evaluated due to limited documentation, the pregnancy and lactation model can also not be fully evaluated to determine whether or not the results are correct.*

- E. Several parameters dictate the transfer of chemical from the mother to her pup. Does the panel agree with the selection of these parameters for the animal model? If not, please provide your justification and alternative parameters.

Comment: *The source for the milk ingestion for animal pups was not fully documented and has not been peer reviewed – cited as in prep (Kapraun et al. 2021). The PFOA and PFOS MCLG documents should provide the information on milk ingestion by mouse and rat pups, describe whether the values are consistent or different from those used in previous lactational modeling in mouse and rat, and justify the choice of intakes. There was no documentation provided on the lactational transfer in humans and very little information is provided regarding the Verner model. While Verner et al. (2016) includes a model file in his manuscript, this does not preclude the agency from providing their model files for the SAB review, as well as for the public, since the parameters actually used in the model scripts determine the output, not the parameters in the publication.*

- F. For neonatal animals, EPA assumed no sex differences in clearance in neonatal animals based on the lack of identification of sex-dependent differences in PFOA/S toxicokinetics from the available data. Does the panel agree with this assumption? If not, please provide your justification and available data on sex differences in neonatal rats.

Comment: *Sex differences in PFOA clearance have only been observed for adult rat where the female rat exhibited a much shorter half-life than the male rat. The evidence supports an assumption that PFOA and PFOS clearance in neonatal animals is similar to adults, and that it is similar to the adult female rat for PFOA.*

Epidemiological Study RfD Derivation

1. EPA evaluated potential confounding as part of their study quality evaluation of the epidemiological studies and selected only ‘medium’ and ‘high’ quality studies for POD derivation. Have the epidemiological studies that were selected for dose-response modeling sufficiently addressed confounding? If not, are there key additional analyses that could be performed to further address the potential confounding of PFAS exposures in these studies?

Comment: *The EPA appropriately evaluated confounding as a risk of bias domain when evaluating the quality of an individual study. Based on a visual review of the heat maps, it appears that a study can be deemed deficient with respect to confounding and still be judged to be a “medium” quality study. It is not clear if each of the risk of bias domains were considered of equal weight when judging the study confidence level (high, medium, low, or uninformative) or if there were certain domains that were given greater weight when assessing study confidence. A written protocol would provide guidance for the evaluation of study quality.*

In the overall synthesis of evidence from epidemiological studies, the EPA appeared to focus primarily on co-exposures to other PFAS. Potential confounding from exposures to other environmental contaminants is only discussed by citing statements in the study publications that exposures to a particular compound (e.g., PCBs) were not highly correlated with PFAS exposure. However, an evaluation of the correlation between the two exposures is not a substitute for including the co-exposures in the analysis as an additional covariate. Previous studies for other compounds by some of the same investigators have failed to include important covariates out of concern that there might no longer be a significant association for the main effect. Given the importance of these studies for the risk assessment, the EPA should not finalize this document without obtaining the data from the critical studies and performing their own analysis, so that it would be available for public scrutiny.

Another potential mechanism of confounding is referred to as pharmacokinetic (PK) bias. PK bias due to confounding arises when a confounding factor affects both the biomarker (e.g., PFAS blood concentration) and the health outcome (e.g., decreased birthweight). PK bias can also result from reverse causation, that is, when the health outcome alters biomarker levels. Importantly, PK bias analysis, whether for reverse causality or effects of confounding factors, can readily be conducted with pharmacokinetic models to examine the influence of confounding factors or health outcomes on pharmacokinetic processes and the resulting epidemiological associations (Andersen et al. 2021a). EPA has the capability to review published studies of PK bias and to perform PK bias analysis themselves, as evidenced by their previous applications of PBPK and BBDR modeling.

2. Studies of developmental immune health outcomes (Grandjean et al., 2012 [HERO ID: 1248827]; Grandjean et al. 2017 [HERO ID: 3858518]; Grandjean et al., 2017 [HERO ID: 4239492]; and Budtz-Jorgensen and Grandjean, 2018 [HERO ID: 5083631]) after PFOA/S exposure identified associations with very low doses of either PFOA or PFOS with developmental immune effects. The RfD for this outcome was selected as the critical effect because it was the lowest among the candidate RfDs for PFOA or PFOS and can result in severe illness. Does the panel agree with the selection of the critical study and critical effect for the derivation of chronic RfDs for PFOA and PFOS?
 - A. If so, please explain your justification.
 - B. If not, please provide your rationale and detail an alternative critical study and/or critical effect you would select to support the derivation of chronic RfDs.
 - C. Are any additional analyses or rationales needed to increase the confidence in the chronic RfDs for PFOA and PFOS?

Comment: *The EPA stated that human epidemiological studies consistently reported decreases in antibody response following vaccination and recommended antibody response to vaccination in children as an outcome for POD derivation. For the POD derivation, the EPA relied an analysis of response to tetanus and diphtheria vaccination in two birth cohorts of children in the Faroe Islands (Budtz-Jorgensen and Grandjean, 2018). Should the EPA decide to use the Faroe Island studies as the basis of the RfD, the data should be obtained from the study investigators and independently evaluated. The analysis and interpretation of the results from the series of studies deserves further review due to inconsistencies within and between the studies regarding participants and methods. Additional concerns are provided below.*

Level of clinical protection: Grandjean et al. (2012) reported 2-fold and 4-fold increased ORs for falling below a clinically significant protective level for tetanus and diphtheria antibodies at age 5 years and age 7 years, respectively. The clinically protective level used by the study investigators was 0.1 IU/mL. Grandjean et al. (2012) reported: “[s]erum concentrations of antibodies against the tetanus toxoid were measured in coded samples by the Statens Serum Institut using enzyme-linked immunosorbent assay (Hendriksen et al. 1988).” Hendriksen et al. (1988) describes the toxin binding inhibition test (ToBI), an assay which is a modified ELISA, for which clinical protection is achieved at 0.01 IU/mL (WHO, 2018; WHO, 2017).

The WHO (2017) reports:

“There is no definitive immunological correlate of protection for tetanus. The minimum amount of circulating antibody that, in most cases, ensures immunity to tetanus is assay-specific. Using in vivo neutralization tests or modified enzyme-linked immunosorbent assays (ELISA), concentrations exceeding 0.01 IU/ml are usually considered protective, whereas antibody concentrations of at least 0.1–0.2 IU/ml are defined as protective when using standard ELISA techniques. [p. 61]”

The WHO (2018) states:

“A toxin binding inhibition (ToBI) assay has been reported and demonstrated to show good correlation with the neutralization assay (correlation coefficient = 0.95) (Hendriksen et al., 1988). The assay determines the level of inhibition of binding of TT to a polyclonal antitoxin by tetanus antibodies in the test sera. The ToBI assay has been subsequently demonstrated to be able to measure tetanus antibody levels below 0.01 IU/ml, making the test attractive for assessing tetanus immunity (van Gageldonk et al., 2008). [p.7]”

For vaccination against tetanus and diphtheria, the children in the Faroe Islands cohorts followed a vaccination schedule of 3 shots in the primary series (at ages 3 months, 5 months, and 1 year) and one booster at age 5. The current recommendation by the WHO for primary vaccination and booster doses in children recommends a primary series of 3 doses of TTCV (similar to the Faroe Islands schedule for primary series) and a boosting regime of 3 doses of TTCV for a total of 6 doses in order to achieve long-term immunity (WHO, 2017):

“The 3 TTCV booster doses should be given at: 12–23 months of age; 4–7 years of age; and 9–15 years of age. Ideally, there should be at least 4 years between booster doses.”

The duration of protection and requirements for booster is further illustrated in the WHO Immunological Basis for Immunization Series, Module 3: Tetanus (WHO, 2018):

“To illustrate the kinetics of immunity among children ≥ 1 year, adolescents and adults following primary and booster vaccination with TTCV, Figure 2 provides a schematic diagram of the typical response. A single dose of TT in the absence of priming induces little, if any, protection. Two to four weeks after the second dose, the mean level of tetanus antitoxin typically exceeds the minimum putatively protective level of 0.01 IU/mL. One year after the second dose, the mean antibody levels are expected to decline and may fall to the protective threshold level. After each subsequent dose of vaccine, immunity is boosted, then persists above the protective threshold for a time, and then wanes over time. Putatively protective levels of immunity are induced by a primary series of three TTCV doses and immunity typically persists for at least 5 years. After the third dose, each additional booster dose given after at least a one-year interval increases tetanus antitoxin levels and further prolongs the duration of immunity. Immunity may persist for approximately 10 years after the fourth dose of TTCV and for at least 20 years after the fifth dose. [WHO, 2018, pp. 14-15].”

Figure 2: Schematic diagram of the antibody response to tetanus toxoid (TT) among children ≥ 1 year, adolescents and adults

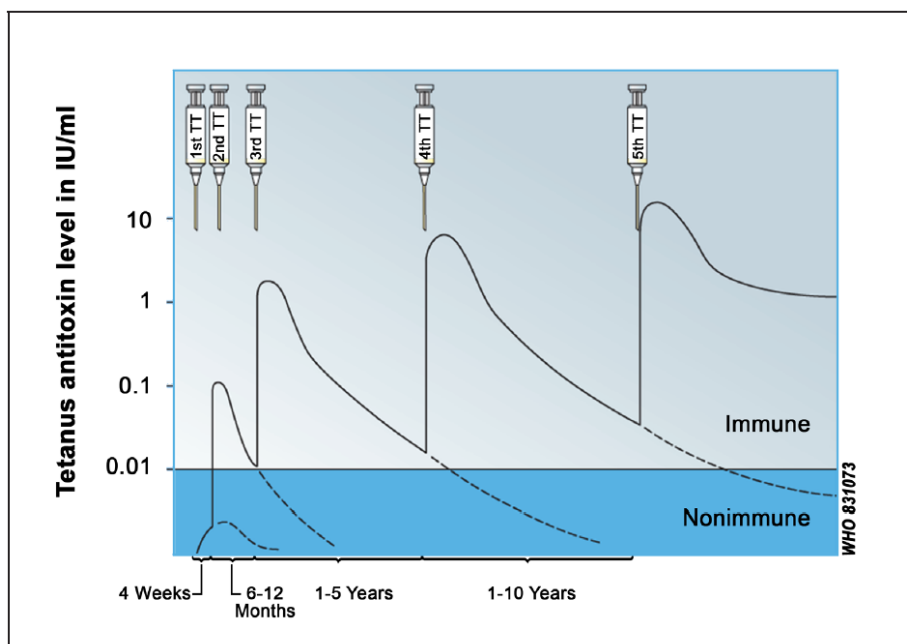


Figure reproduced from: Plotkin S, Orenstein W, Offit P, Edwards KM. Plotkin's vaccines, seventh edition. New York (NY): Elsevier; 2017: Chapter 58, Tetanus toxoid, p. 1071, reproduced by permission.

"Five properly spaced doses [given to children ≥ 1 year, adolescents and adults provides] protection lasting at least 20 years and probably substantially longer for most recipients. (Threshold of protection of 0.01 IU/mL applies to values measured by in vivo neutralization, modified ELISA [or bead-based immunofluorescence]; for standard ELISA, protection is [usually defined between 0.10–20 IU/mL, based on the assay])." (Modified source: Borrow R, Balmer P, Roper MH. The immunological basis for immunization. Module 3: Tetanus (update 2006). Geneva: World Health Organization; 2007.)

Although the WHO (2012) Guidelines for Immunotoxicity Risk Assessment recommend measures of vaccine response as a measure of immune effects, the guidelines refer to EHC 180 (UNEP, 1996):

"A description of biomarkers in epidemiological studies is provided in EHC 180: Principles and methods for assessing direct immunotoxicity with exposure to chemicals (IPCS, 1996). The risk assessor should refer to the assay descriptions in EHC 180 for immunotoxicity endpoints contained in the data set for the chemical in question to provide specific context, cautions and information that may assist in the interpretation of immunosuppression data for risk assessment. In addition, it is recommended that the risk assessor consult an expert in immunotoxicology or clinical immunology to help interpret the biological plausibility of the study results. [p.51]"

EHC 180 Principles and methods for assessing direct immunotoxicity with exposure to chemicals (UNEP, 1996) described assays recommended by WHO (UNEP, 1996) and the National Research Council (NRC) of the National Academy of Sciences (NRC, 1992) for preliminary assessment of individuals exposed to immunotoxicants, including secondary antibody responses to proteins (e.g., diphtheria, tetanus, poliomyelitis) and polysaccharides (e.g., pneumococcal, meningococcal). Specifically, the WHO (1996) describes the tests for antibody response to immunization:

“In order to test for T cell-dependent antibody responses, commercially available diphtheria-tetanus vaccine can be given in recommended doses. Blood is taken two weeks after each injection and tetanus and diphtheria antibodies are determined. In patients who have been immunized with diphtheria-tetanus or diphtheria-pertussis-tetanus vaccine, one booster injection is given before determination of antibodies. In testing for T cell-independent antibody responses, commercially available pneumococcal vaccine can be given in recommended doses. Three doses of killed poliomyelitis vaccine (10 ml intramuscularly, at intervals of two weeks) can also be used as the immunogen. Blood is taken two weeks after the last injection, and antibody is usually determined by virus neutralization. [WHO, 1996, pp. 238-239]”

Therefore, it seems that the most relevant findings from the studies in the Faroe Islands are the associations between 5 year post-booster responses compared to PFAS serum measured at age 5 years pre-booster, four weeks earlier. None of the studies of the Faroe Islands reported whether any person had a post-booster concentration that fell below 0.1 IU/mL four weeks after receiving a booster.

Although there is no information on the number of individuals who received a booster but failed to mount an appropriate response within four weeks, there is evidence that the booster worked overall. For tetanus, the post-booster median concentration (35 IU/mL, interquartile range (IQR) 16-96) in 456 children was 159-fold higher than the pre-booster median concentration based on 532 children (0.22 IU/mL, IQR 0.10 – 0.51) (Grandjean et al. 2012, Table 1). There is approximately the same variability in post-booster and pre-booster concentrations based on the IQR (6-fold difference from 75th to 25th percentiles post booster and 5-fold difference from 75th to 25th percentiles in antibody concentrations pre-booster). For diphtheria, the post-booster median concentration (13 IU/mL, IQR 6.4–26) was 108-fold higher than the pre-booster median concentration (0.12 IU/mL, IQR 0.05 – 0.40). There was a 4-fold difference between the 75th to 25th percentiles after boosting, and an 8-fold difference between the 75th and 25th percentiles before boosting.

Inconsistencies regarding study subjects included in various analyses: Although the Faroe Islands are four separate studies, they each address one or two birth cohorts (1997-2000 and 2007-2009) from a single hospital in the Faroe Islands. There are inconsistencies reported in relation to the number of participants at different ages in these cohorts. For the 1997-2000 birth cohort, Grandjean et al. (2012) and subsequent studies reported “[a] total of 587 children (89% of the cohort) participated in 1 or more of the examinations, which took place at age 5 years pre-booster, approximately 4 weeks after the booster, and at age 7 years.” Separately, Grandjean et al. (2012) reported “[a] lower antibody response was observed in 2 groups of 173 and 168 children, who had been inoculated with combination booster vaccines containing pertussis, polio, or both, as compared with the 151 who received diphtheria and tetanus toxoids only.” The total of 492 children receiving a booster dose does not match with the 532 children attending the age-5 pre-booster examination or the 456 children attending the age-5 post-booster exam (Grandjean et al. 2012, table 1) or the 537 children included in the 5-year pre-booster analysis or the 440 children included in the 5-year post-booster analysis (Grandjean et al. 2012, table 2).

It is also not clear that the children examined at the 5-year post-booster examination were restricted to the same children that participated in the 5-year pre-booster exam. In fact, it seems more likely that there were children who participated in the 5-year post-booster exam that were not part of the study population that had attended the 5-year pre-booster exam (and therefore, had not provided a PFAS serum sample 4 weeks earlier). Grandjean et al. (2012) reported that “[f]or the 5-year post-

booster data, we adjusted for the time since vaccination, using a restricted cubic spline (Heilmann 2006).” There would be no need to make this adjustment for time since last vaccination if all of the 5-year post-booster results were restricted to the same individuals who attended the 5-year pre-booster exam 4 weeks earlier. Potentially, some of the children at the 5 year post-booster exam were sampled for PFAS serum and antibody concentration immediately after they had their booster shot. If the 5-year post-booster serum samples described mixed populations (individuals who were last vaccinated four weeks earlier as well as individuals who were last vaccinated at one year), the analyses of 5-year post-booster data would have significant variability in levels of circulating antibodies. Regardless of PFAS serum concentrations, some proportion of individuals included in the post-booster analysis would have had relatively high levels of circulating antibodies (that is, individuals who were boosted 4 weeks earlier) while others would have had low levels of circulating antibodies (those who had last received a vaccination at age 1) due to the well-known effect of waning antibodies 1 to 3 years after the primary series (WHO, 2017; 2018). The analysis of post-booster results should be restricted to the individuals who provided serum four weeks earlier so that the boosting effect is not diluted.

Confounding: Many factors affect humoral immunity and vaccine response. Possible confounding due to co-exposures to PCBs and other persistent organic compounds, as well as methyl mercury (MeHg), existing in studies of the Faroese Island populations. The Faroese diet includes a high proportion of whale meat, which contains high levels of PFAS (Weihe et al. 2008), but it also contains high levels of PCBs, polybrominated flame retardants and MeHg. (Andvik et al. 2021). The PFAS exposure from eating whale meat is significant: “On a relative scale, a high intake of two pilot whale dinners per month is associated with increases in the 14-year serum concentrations of PFOS, PFNA, and PFDeA by almost 25%, 50%, and 100%, when compared to concentrations in subjects eating little or no whale at all (Table 3). Fish dinners had a much weaker effect, although each weekly fish dinner augmented the PFHxS concentration by about 10%.” (Weihe et al. 2008)

Budtz-Jorgensen and Grandjean (2018) cited Grandjean et al. (2012) when they reported that confounding by methyl mercury and polychlorinated biphenyls was unlikely because of their weak correlations with serum concentrations of PFAS. The evaluation of methyl mercury as a potential confounder should be independently confirmed by EPA. Although Grandjean et al. (2012) reported that PCBs were weakly correlated with PFAS, methyl mercury was not discussed in these analyses and was presumably not part of the evaluation. The IPCS (1996) reported that MeHg decreased humoral immunity in mice in a study conducted by Blackley et al. (1980).

Conclusion: It seems more likely than not that the results of this study are merely consistent with heterogeneity in vaccine response. Furthermore, reverse causality and/or confounding are likely to be an issue in studies with little variation in exposure and low exposure contrasts. Exposure varied little in the Faroe Island studies; the exposure contrasts were low based on measurements of PFAS in serum. Although the study investigators did not provide minimum or maximum concentrations, they reported interquartile ranges. In children measured for PFAS in serum at age 5, the IQRs were: PFOA, 3.33 to 4.96 ng/mL (median 4.06); PFOS, 13.5 to 21.1 ng/mL (median 16.7); PFHxS 0.45 to 0.88 ng/mL (median 0.63) ; PFNA 0.76 to 1.24 ng/mL (median 1.00); and PFDA, 0.21 to 0.38 ng/mL (median 0.28). Grandjean et al. (2012) and subsequent evaluations of the Faroe Islands cohort used log base 2 transformations of these exposure data (with low exposure contrasts) and found that the strongest effect was at the lowest concentrations, which seems biologically implausible.

3. The health outcomes identified in the critical studies were decreased antibody response, specifically in serum anti-tetanus and anti-diphtheria, in children after vaccination (Grandjean et al., 2012 [HERO ID: 1248827]; Grandjean et al. 2017 [HERO ID: 3858518]; Grandjean et al., 2017 [HERO ID: 4239492]; and Budtz-Jorgensen and Grandjean, 2018 [HERO ID: 5083631]). This health outcome represents an increased susceptibility to a disease that can cause very severe symptoms, including lethality. Furthermore, children who are immunocompromised may mount a lower antibody response and in turn, be more susceptible to contracting the disease, if exposed than healthy children. Because this health outcome has the potential for severe illness and was assessed in children (i.e., EPA guidelines [US EPA, 1991] support a 5% BMR for developmental effects), a benchmark response (BMR) of 5% was selected for benchmark dose modeling. While some clinical findings are available, the clinical relevance of a 5% decrease in antibody response is not clear. Given the need to protect sensitive subpopulations (e.g., children, individuals with pre-existing conditions) and the available clinical data (i.e., antibody response clinical level), does the SAB support the 5% BMR selection for modeling to identify the POD? If not, please recommend the BMR level and a scientific rationale for an alternative selection.

Comment: As stated in the comments on the previous question, the level of circulating antibody that correlates with clinical protection is assay-specific. Based on the ToBI assay used in the Faroe Islands, the relevant clinical level of protection for the population in that study would be 0.01 IU/mL, not the value of 0.1 IU/mL used by the EPA. Furthermore, none of the studies of the Faroe Islands population provided information on whether the prevalence of failure to respond to secondary immunization was beyond that expected from natural variation in vaccine response. In addition, epidemiological studies of PFOA and/or PFOS and common infectious diseases and their symptoms (including otitis media, common colds, gastroenteritis, respiratory tract infections, fevers) have reported inconsistent associations (Granum et al. 2013, Impinen et al. 2018, Dalsager et al. 2016; Looker et al. 2014; Okada et al. 2012), bringing into question the validity of the EPA's assumptions regarding the clinical relevance of the antibody titer endpoint.

4. EPA has evaluated and applied where appropriate uncertainty factors to account for intraspecies variability (UFH), interspecies differences (UFA), database limitations (UFD), duration (UFS), and LOAEL-to-NOAEL extrapolation (UFL) for PFOA and PFOS.

- A. Has uncertainty been adequately accounted for in the derivation of the RfDs? Please describe and provide suggestions, if needed.

Comment: Yes. Since the RfDs are derived from data in human populations, an uncertainty factor of 10 to consider human interindividual variability is more than adequate to properly account for uncertainty in the derivation of the RfDs.

- B. Does the provided scientific rationale support the application of the selected uncertainty factors? Please explain.

Comment: Yes. Since the RfDs are derived from data in human populations, no uncertainty factors are required apart from one for human interindividual variability.

Relative Source Contribution

1. EPA applies a Relative Source Contribution (RSC) when calculating the MCLG to provide a margin of safety that an individual's total exposure from a contaminant does not exceed the RfD. The RSC is the portion of an exposure for an individual in the general U.S. population estimated to equal the RfD that is attributed to drinking water; the remainder of the exposure equal to the RfD is allocated to other potential sources. Based on the physical properties, detected levels, and available exposure information, there are significant potential sources other than drinking water ingestion for PFOA and PFOS; however, information is not available to quantitatively characterize exposure from these different sources. EPA followed Agency guidance on how to derive an RSC (U.S. EPA, 2000; available online at: <https://www.epa.gov/sites/default/files/2018-10/documents/methodology-wqc-protection-hh-2000.pdf>) and recommends an RSC of 20 percent (0.20) for PFOA and PFOS. This RSC is the same as what was used in the 2016 HAs for PFOA and PFOS.

- A. Are you aware of additional relevant exposure data that EPA should consider in developing the RSCs for PFOA and PFOS? If so, please provide citations.
- B. Please provide comment on whether the recommended RSC of 20 percent (0.20) for PFOA and PFOS is adequately supported and clearly described.

Comment: *The basis for the recommended RSCs of 50% for infants/children and 20% for adults is adequately supported.*

EPA'S DRAFT FRAMEWORK FOR ESTIMATING NONCANCER HEALTH RISKS ASSOCIATED WITH MIXTURES OF PER- AND POLYFLUOROALKYL SUBSTANCES

Overall charge: EPA is seeking SAB comment on whether the framework and illustrative examples provided in the document are scientifically supported, clearly described, and informative for assessing potential health risk(s) associated with exposure to mixtures of PFAS.

General Comment: *The EPA Draft Framework is a very impressive document that proposes a scientifically sound approach and applies widely accepted practices for mixture risk assessment. It also provides informative examples that help to clarify the proposed approach. It represents a major improvement over some approaches that have been used by regulatory agencies, such as applying the PFOA RfD to total PFAS concentrations. It is important to emphasize, however, that these approaches are best suited for site- or source-specific assessments, where the composition of the mixture at the site or source has been determined. While the approaches described in the framework are sound risk assessment tools, it should be made clear that they are not as well suited for use by states or other entities in the promulgation of drinking water standards, where the composition of the mixtures of PFAS compounds in drinking water may not be consistent across locations and sources.*

Charge questions

1. The component-based mixtures approaches presented in the framework are based on dose addition. Traditionally, an assumption of dose addition for a mixture is based on components sharing a common mode of action (MOA) for a given health effect. However, EPA's supplementary guidance (EPA, 2000) states: "The common mode-of-action (MOA) assumption can be met using a surrogate of toxicological similarity, but for specific conditions (endpoint, route, duration)." This suggests that although the common MOA metric for application of dose addition is optimal, there is flexibility in the level of biological organization at which "similarity" can be determined among mixture components. As an emerging chemical class, MOA data is limited or not available for many PFAS. For purposes of a component-based evaluation of mixtures additivity for PFAS, EPA assumes similarity at the level of toxicity endpoint/health effect rather than MOA.
 - A. Please comment on the appropriateness of this approach for a component-based mixture evaluation of PFAS under an assumption of dose additivity.

Comment: *Grouping chemicals for dose-additivity on the basis of similarity in toxicity/health effects begs the question of "how similar". For example, dose additivity is not necessarily appropriate for a chemical that causes centrilobular hypertrophy and a chemical that causes single cell hepatocellular necrosis. In the specific case of PFAS, if the effects in a tissue differ somewhat, it is critical that the assumption of dose-additivity be restricted to compounds that also have sufficient structural similarity to support the likelihood that the key elements of the Mode of Action are similar (that is, similar interactions of the compounds with the key proteins controlling the pharmacokinetics and pharmacodynamics of PFAS). As in the case of pyrethroids and PCBs, it may be necessary to define sub-categories of PFAS for which dose-additivity can be applied. Two examples of the potential considerations are short vs. long chain length, and linear fluoroalkyl acids vs. branched-chain fluoroether acids.*

- B. If common toxicity endpoint/health effect is not considered an optimal similarity domain for those PFAS with limited or no available MOA-type data, please provide specific alternative methodologies for integrating such chemicals into a component-based mixture evaluation(s).

Comment: *Common toxicity endpoint/health effect is an acceptable similarity domain for those PFAS with limited or no available MOA-type data. However, the concerns raised in the comment on the previous question need to be adequately addressed as part of the evaluation of similarity.*

2. Section 4.3 (Hazard Index; HI) of the framework document demonstrates the application of a component-based mixture approach, based on dose addition, using available oral reference doses from completed EPA human health assessments, and hypothetical exposure information. The example calculations presented are primarily focused on four PFAS with finalized EPA Human Health Assessments: perfluorooctanoic acid (PFOA), perfluorooctane sulfonic acid (PFOS), perfluorobutane sulfonic acid (PFBS), and hexafluoropropylene oxide (HFPO) dimer acid and HFPO dimer acid ammonium salt (referred to as “GenX chemicals”).

- A. Please provide specific feedback on whether the HI approach is a reasonable methodology for indicating potential risk associated with mixtures of PFAS. If not, please provide an alternative.

Comment: *The proposed approach is reasonable and is consistent with EPA practice.*

- B. Please provide specific feedback on whether the proposed HI methodologies in the framework are scientifically supported for PFAS mixture risk assessment.

Comment: *The proposed HI methodologies in the framework are adequately supported for use in preliminary site-specific risk assessments for a specific PFAS mixture composition.*

3. Section 4.4 (Relative Potency Factor; RPF) of the framework document demonstrates the application of a component-based mixture approach, based on dose addition, using available dose-response information (i.e., points-of-departure) from completed EPA human health assessments, and hypothetical exposure information. The example RPFs and corresponding Index Chemical Equivalent Concentration (ICEC) calculations presented are primarily focused on four PFAS with finalized EPA Human Health Assessments: PFOA, PFOS, PFBS, and HFPO dimer acid and GenX chemicals.

- A. Please provide specific feedback on whether the RPF approach is a reasonable methodology for estimating risk associated with mixtures of PFAS. If not, please provide an alternative.

Comment: *The proposed RPF approach is a reasonable methodology for estimating risk associated with specific mixtures of PFAS and is consistent with EPA practice.*

- B. Please provide specific feedback on whether the proposed RPF methodology in the framework is scientifically supported for PFAS mixture risk assessment.

Comment: *The proposed RPF methodology is scientifically supported as an approach for a specific PFAS mixture during a site- or source-specific assessment, but it should be made clear that it is not well suited for the development of general standards that are intended to be applied across sites or sources with different mixing-ratios of component chemicals.*

4. Section 4.5 (Mixture BMD) of the framework document demonstrates the application of a component-based mixture approach using established EPA dose-response modeling (i.e., benchmark dose; BMD) of hypothetical PFAS dose-response data, and hypothetical exposure information.

- A. Please provide specific feedback on whether the Mixture BMD approach is a reasonable methodology for estimating what is in essence a mixture-based point-of-departure. If not, please provide an alternative.

Comment: *The Mixture BMD approach is a reasonable methodology for estimating what is in essence a point-of-departure for a specific mixture composition, but the resulting POD should not be applied across mixtures with different compositions.*

- B. Please provide specific feedback on whether the proposed Mixture BMD methodology in the framework is scientifically supported for PFAS mixture risk assessment.

Comment: *The proposed Mixture BMD methodology in the framework is scientifically supported for a PFAS mixture with a specific mixing-ratio of component chemicals, and can be applied during a site- or source-specific assessment. It is important that the EPA make it clear that it should not be applied in the development of a single standard that is intended to be applied across sites or sources with different mixing-ratios of component chemicals.*

ANALYSIS OF CARDIOVASCULAR DISEASE RISK REDUCTION AS A RESULT OF REDUCED PFOA AND PFOS EXPOSURE IN DRINKING WATER

Overall charge: EPA is seeking SAB comment on the extent to which the approach to estimating reductions in CVD risk associated with reductions in exposure to PFOA and PFOS in drinking water is scientifically supported and clearly described.

Charge Questions

1. Section 4.2 presents EPA's meta-analysis for the total cholesterol dose-response function.
 - A. Please provide specific feedback on the extent to which the study selection criteria, the identified studies, and the methodological approach of the meta-analysis are complete and capture up to date scientific literature.

Comment: Although the EPA describes the study selection criteria, there is no apparent integration of study quality criteria in the meta-analysis. Several of the identified studies were judged by reviewers to be of low (or deficient) quality. Separately, there was no information on the risk of bias analysis for five studies (of 14 total studies considered in the meta-analysis) conducted before 2018. Meta-estimates should be derived for studies which were considered higher quality and/or studies that adjusted for certain confounding factors (lipid-lowering medication). In addition, the EPA meta-analysis did not address the associations between PFOA or PFOS and LDL-C, which were discussed in the draft documents. LDL-C is well established as a causal risk factor for CVD; it is recommended that individuals with high LDL-C (and not high TC) take LDL-C lowering medication to manage CVD risk (Grundy et al. 2019). Presumably, EPA excluded LDL-C from the meta-analysis because the ASCVD risk calculator does not include LDL-C as a predictor. The majority of cholesterol in human lipid profiles is LDL-C so the exclusion of LDL-C does not appear to be a fatal flaw. However, the ASCVD risk model is intended solely for patients with LDL-C <190 mg/dL, without ASCVD, and not on LDL-C lowering therapy. In contrast, studies in the general population did not consistently adjust for lipid-lowering medication and did not exclude individuals with LDL-C $\geq$ 190 mg/dL.

An international scientific panel (Andersen et al. 2021b) concluded that correlated net absorption or excretion of bile salts and PFAS in the gut enterocytes could give rise to the apparent associations of cholesterol and PFAS in blood observed in epidemiological studies. It has been demonstrated that several bile acid transporters expressed in enterocytes and hepatocytes can also transport PFAS, suggesting that PFAS could be entrained within the enterohepatic recirculation of bile acids. Co-modulation of the kinetics of bile acids and PFAS at these specific transporters by cholesterol has been shown in the rat. Correlated uptake/biliary excretion of PFAS and bile salts could serve as a confounding link between cholesterol homeostasis and PFAS kinetics, leading to an apparent association between Total Cholesterol (TC) and PFAS concentrations in serum. Importantly, if PFAS and cholesterol kinetics were both correlated with a common confounding process (e.g., bile acid recirculation), the fractional change in TC (compared to the average) in a given study would be expected to be the same as the fractional change in PFAS serum (compared to the average) in a same study. Moreover, this expected relationship would hold whether the exposures were at low or high PFAS

concentrations. In fact, this relationship is consistently seen in studies of PFAS exposures, whether the study subjects are exposed to low environmental concentrations or are occupationally exposed to high concentrations. In other words, a similar fractional change in PFAS concentration (rather than the concentration itself) is associated with a fractional change in TC across many orders of magnitude of exposure concentrations. However, cross-sectional epidemiological studies have evaluated the concentration of PFAS in relation to TC concentrations (using cross-sectional study designs). When evaluating the body of evidence, the apparent effect on cholesterol is stronger at lower concentrations than higher concentrations; however, it is biologically implausible that the potency of PFAS on cholesterol homeostasis decreases as PFAS concentration increases. Therefore, it is more likely that the association is not causal, and that bile acid recirculation distorts the association between PFOA or PFOS and TC.

A recent review of epidemiological studies and other scientific evidence published since 2012 (Steenland et al. 2020) discussed confounding by enterohepatic cycling of PFAS and bile acids as a possible explanation for the positive association between PFOA and total cholesterol. Genius et al. (2014) discussed supporting evidence that cholestyramine, a bile acid sequestrant, also reduced PFOS, PFOA, and PFHxS serum concentrations in humans and rats. Epidemiological studies have not shown increased risks of cardiovascular disease in relation to PFOA or PFOS, even among workers with the highest exposures (Steenland et al. 2015; Steenland and Woskie, 2021; Alexander et al, 2014) or community members exposed to PFOA in contaminated drinking water (Winquist and Steenland, 2014).

- B. To inform the CVD risk reduction analysis for those ages 40-89 using the ASCVD risk model, EPA used a meta-analysis approach for the total cholesterol dose-response function. Please provide specific feedback on the extent to which this approach is reasonable for this application, or whether using a single dose-response study (e.g. Dong et al., 2019) selected in the analysis of cholesterol impacts in the *Proposed Approaches for Deriving Maximum Contaminant Level Goals for PFOA and PFOS in Drinking Water* would add additional strengths for the CVD risk reduction application.

Comment: Small increases in HDL cholesterol have also been reported in relation to PFOA and/or PFOS concentrations in blood serum in cross-sectional studies. In addition to a small, non-statistically significant increase in total cholesterol represented in the pooled estimate seen in Figure A-4 of the Serum Cholesterol Dose-Response Function Appendix, the meta-analysis also showed a small, non-statistically significant increase in HDL cholesterol (Figure A-4 in relation to PFOA and Figure A-8 in relation to PFOS). Presumably, a pooled estimate for the total cholesterol dose-response function reduces the random error associated with relying on a dose-response estimate from a single study. Nevertheless, the pooled estimates showed significant heterogeneity in the underlying studies (I^2 values >70% for both the PFOA (Table A-2) and the PFOS meta-analyses (Table A-3)). This also suggests a single dose-response study should not be relied upon. In any case, the meta-analysis did not address the issue of systematic error and it included studies judged by reviewers to be low quality and/or deficient.

1. Section 5.1 presents EPA's life table approach methodology.

- A. Please comment on the extent to which this analysis is scientifically supported and clearly described. To the extent improvements are suggested, please provide specific changes that are implementable in a U.S. national-level benefits analysis with readily available data.

Comment: *The life table approach is clearly described and is a standard life table approach. Because there is substantial uncertainty introduced in using the ASCVD model to predict CVD events in general (see Comment below, Question 2), and substantial uncertainty regarding the risk of CVD in relation to PFOA and PFOS exposure, a simpler approach to the quantifiable impacts of a potential reduction in cholesterol in relation to reductions in PFOA and PFOS exposure is preferred. Instead of attempting to quantify the impacts of avoided CVD events in relation to reduced total cholesterol, the EPA could consider quantifying avoided health care costs associated with treating high LDL-C (for example, avoided prescriptions for cholesterol-lowering medication) or measuring a reduction in the population eligible for treatment with cholesterol-lowering medication. There are uncertainties with this suggested approach as well; however, it avoids the potential amplification of uncertainty associated with the EPA approach.*

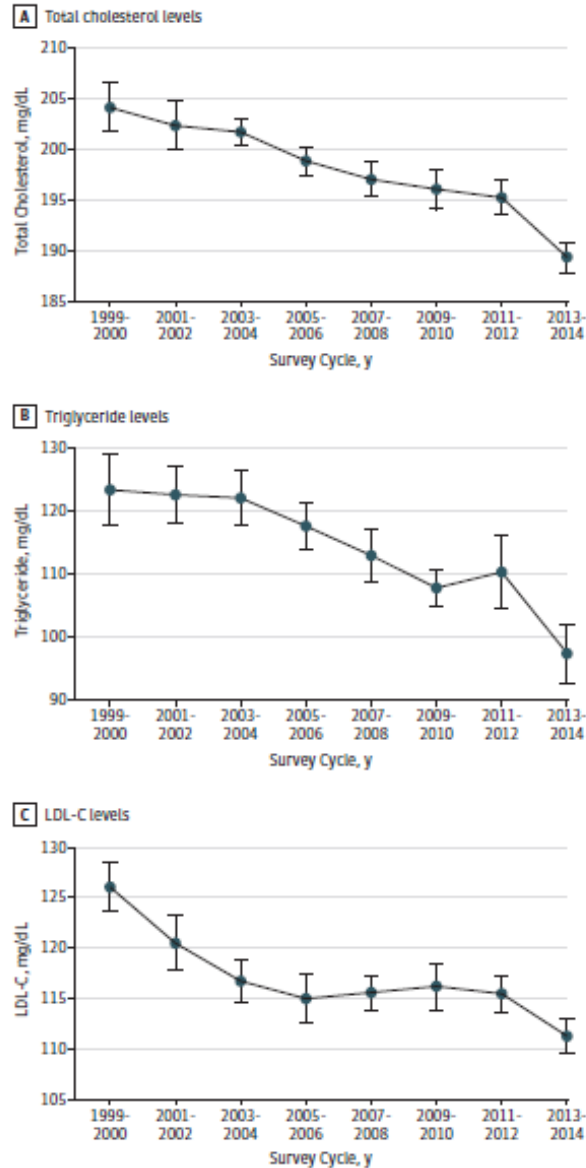
2. Section 5.2 presents EPA's application of the atherosclerotic cardiovascular disease (ASCVD) risk model used to estimate the probability of hard CVD events corresponding to total cholesterol changes.

- A. Please comment on the scientific validity of the ASCVD model application for estimating the probability of first time CVD events in various sub-populations and the extent to which it is clearly described.

Comment: *The ASCVD risk model was developed for use in clinical practice to provide a quantitative risk score for an individual based on an estimate of the 10-year probability of an initial CVD event based on several inputs. The calculator uses age, total and HDL-cholesterol levels, systolic blood pressure, antihypertensive therapy status, history of diabetes and current smoking and assumes that the individual has LDL-C <190 mg/dL, is free of CVD, and is not taking lipid-lowering medication. The pooled cohort equations that form the basis for the risk estimates are based on older population studies that enrolled volunteers (for example, the Framingham Cohort study, and the Atherosclerosis Risk in Communities (ARIC) study including other cohorts). Although the data may be useful for informing a clinician-patient discussion of managing potential CVD risk for a particular patient, it is not valid for estimating current population-level risks of CVD. Several validation studies have reported that the ASCVD model has overestimated the rate of CVD (DeFilippis et al. 2015; Cook and Ridker, 2014; Rana et al. 2016). The ASCVD model does not account for changes in CVD risk predictors (including cholesterol) over time at a population level. For example, Rosinger et al. (2017) reported that mean cholesterol levels (both TC and LDL-C) and mean triglyceride levels decreased from 1999 to 2014, based on estimates using eight NHANES cycles (see figure below). The decreases over time were similar when stratified by lipid-lowering medication use and are possibly related to the reduction and elimination of artificial trans fats in foods.*

Rosinger et al. 2017:

Figure. Age-Adjusted Total Cholesterol, Triglyceride, and Low-Density Lipoprotein Cholesterol (LDL-C) Trends for US Adults Aged 20 Years and Older, 1999 to 2014



A, Predicted total cholesterol levels and 95% confidence intervals in a sample size of 39 049. B, Predicted log-transformed triglyceride levels and 95% confidence intervals; log-transformed values were exponentiated after the regression, sample size of 17 406. C, Predicted LDL-C levels and 95% confidence intervals in a sample size of 17 096. Figure generated using marginal standardization from age-adjusted linear regression models. Data source: Centers for Disease Control and Prevention/National Center for Health Statistics, the National Health and Nutrition Examination Survey.

SI conversion factors: To convert LDL-C to micromoles per liter, multiply by 0.0259; to convert total cholesterol to micromoles per liter, multiply by 0.0259; to convert triglycerides to micromoles per liter, multiply by 0.0113.

- B. Please comment on whether EPA's approach and assumption, of a uniform first CVD event hazard distribution over the 10-year period, is sufficiently robust given current data sources and literature. If additional distributional sources of information are suggested, please provide specific citations/sources for EPA's consideration.

Comment: *Avoided CVD events should not be the basis for the regulatory impact assessment. It is unclear whether the EPA approach and assumption of a uniform first CVD event over a 10-year period is sufficiently robust. Moreover, the link between PFAS exposure and CVD is too tenuous to support a meaningful cost-benefit comparison.*

- C. Please comment on the scientific validity of using the ASCVD risk model for estimating reduced CVD risk stemming from changes in total cholesterol in response to reducing exposure to PFOA and PFOS in drinking water.

Comment: *The evidence for a causal relationship between PFOA or PFOS exposure and cardiovascular disease is weak, and it is plausible that PFOA and PFOS exposure is associated with higher total cholesterol levels but without an increased risk of cardiovascular disease (Steenland et al. 2020). It is highly uncertain that reduced PFOA or PFOS exposure will lead to lower cholesterol concentrations that can then be quantified as avoided CVD events. As stated previously, the ASCVD model has not accounted for decreases in total cholesterol over the past 20 years or more and is intended for individuals with LDL-C < 190 mg/dL. A more direct and relevant regulatory cost analysis that requires fewer assumptions (and less uncertainty) is preferred. A regulatory analysis of quantified health risk reduction that focuses on cholesterol reduction (and specifically LDL-C reduction) as the quantified endpoint rather than CVD events avoided would reduce uncertainties, such as use of ASCVD to calculate avoided CVD cases when the epidemiological literature does not show increased risks of CVD in relation to PFOA or PFOS exposure. For example, the EPA could consider avoided use of cholesterol-lowering medication or the reduction in number of adults eligible for treatment for high cholesterol. However, this approach would still not address the possibility that confounding by enterohepatic cycling of PFAS and bile acids is actually responsible for the positive association between PFOA and total cholesterol (Steenland et al. 2020).*

5. Section 7 and Appendix A describe the limitations and uncertainties of the CVD risk reduction analysis. Has EPA clearly described the individual contributions of the sources of uncertainty?

Comment: *If PFOA and PFOS are associated with small increases in TC and small increases in HDL-C, it is biologically plausible that the risks of cardiovascular disease remain unchanged (Steenland et al 2020). The uncertainties described in the ASCVD model included whether PFOA and PFOS potentially impact other risk factors in the ASCVD model (diabetes and systolic blood pressure, for example); however, the ASCVD model does not include all identified or important risk factors (e.g., elevated LDL-C and elevated C-reactive protein (CRP) levels, for example). Although the association between PFOA or PFOS and CRP levels is not well studied, Genser et al. (2015) reported that CRP levels decreased with increasing serum PFOA concentration in an analysis of adults older than 18 years who resided in water districts contaminated with PFOA (the C8 Health Study).*

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Appendix B

WITAF 056 Technical Memorandum Update: PFAS National Cost Model Report

WITAF 56 TECHNICAL MEMORANDUM UPDATE

PFAS National Cost Model Report

B&V PROJECT NO. 409850

PREPARED FOR



**American Water Works
Association**

American Water Works Association

26 MAY 2023



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Abbreviations

AACE	Association for the Advancement of Cost Engineering
AWWA	American Water Works Association
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFPUA	Cape Fear Public Utility Authority
CIP	Clean-In-Place
CWS	Community Water System
DBP	Disinfection Byproducts
EBCT	Empty Bed Contact Time
EPA	U.S. Environmental Protection Agency
EPTDS	Entry Point to the Distribution System
FRP	Fiberglass Reinforced Plastic
ft	Feet
GAC	Granular Activated Carbon
g/cc	Grams per Cubic Centimeter
gpm	Gallons per Minute
gpm/sf	Gallons per Minute per Square Feet
HRT	Hydraulic Retention Time
IX	Ion Exchange
kWh	Kilowatt-hour
lb/gal	Pounds per Gallon
LHHCWD	La Habra Heights County Water District
MCL	Maximum Contaminant Level
mgd	Million Gallons per Day
mg/L	Milligrams per Liter
NF	Nanofiltration
NTNCWS	Non-Transient Non-Community Water System
NPDWR	National Primary Drinking Water Regulation
PFAS	Per- And Polyfluoroalkyl Substances
PFBS	Perfluorobutane Sulfonic Acid
PFHpA	Perfluoroheptanoic Acid
PFHxS	Perfluorohexane Sulfonate
PFNA	Perfluorononanoic Acid
PFOA	Perfluorooctanoic Acid
PFOS	Perfluorooctane Sulfonic Acid
ppt	Parts Per Trillion
PWS	Public Water System
RO	Reverse Osmosis
SDWIS	Safe Drinking Water Information System
sf	Square Feet
SLR	Surface Loading Rate
T&O	Taste and Odor

TDH	Total Dynamic Head
TDS	Total Dissolved Solids
TOC	Total Organic Carbon
UCMR	Unregulated Contaminant Monitoring Rule
WITAF	Water Industry Technical Action Fund

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Partner Utilities:

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- City of Ann Arbor
- Greater Cincinnati Water Works
- Plainfield Charter Township
- City of North Miami Beach
- Miami-Dade County Water and Sewer Department
- Tucson Water

Steering Committee Members:

- Amy Stoffer – Northern Kentucky Water District
- Cynthia Lane – Platte Canyon
- Carel Vandermeiden – CFPUA
- Robert Cheng – Coachella Valley Water District
- Zaid Chowdhury – Garver USA
- Chuck Hertz – Retired

2.0 Introduction

Known as “forever chemicals” because they do not easily biodegrade, per- and polyfluoroalkyl substances (PFAS) are drawing increased scrutiny from health agencies, water utilities, and the public for their presence in drinking water and their effects on human and environmental health. They have quickly become contaminants of great concern in drinking water.

Six PFAS compounds were monitored in finished drinking water as part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) between 2013 and 2015 to quantify their prevalence across the United States. The UCMR program provides the U.S. Environmental Protection Agency (EPA) with nationally representative occurrence data to inform drinking water regulations. Using the results from UCMR 3, in February 2021, the EPA published a final determination to regulate perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS) and signaled an interest in considering the regulation of additional PFAS. On March 14, 2023 the EPA announced the first proposed National Primary Drinking Water Regulation (NPDWR) for six PFAS compounds, including PFOA, PFOS, perfluorononanoic acid (PFNA), hexafluoropropylene oxide dimer acid (HFPO-DA, commonly known as GenX Chemicals), perfluorohexane sulfonic acid (PFHxS), and perfluorobutane sulfonic acid (PFBS). The deadline for public comment on this proposed regulation is May 30th, 2023 and the EPA has publicly committed to promulgate the PFAS NPDWR by the end of 2023.

U.S. federal laws and executive orders stipulate that the U.S. EPA estimate the cost of compliance for this new primary drinking water regulation. Black & Veatch was selected by the American Water Works Association (AWWA) to develop a national cost estimate for water systems to remove PFAS from drinking water to better understand the financial impacts to communities and the costs to comply with a national primary drinking water regulation, a policy that could impact each of the more than 66,500 public water systems.

The project was funded by the Water Industry Technical Action Fund (WITAF), which is managed by the AWWA’s Water Utility Council to support projects, studies, analyses, reports, and presentations in support of the organization’s legislative and regulatory agenda. The national cost estimate and its cost models, developed under WITAF 056, are intended to support to AWWA’s engagement with the U.S. EPA and Congress on the differences in financial impacts of treating drinking water to various PFAS regulatory limits. WITAF funded a separate project (WITAF 057) to generate a national PFAS occurrence database using data from state monitoring and UCMR3. This national database was used as an input for the WITAF 057 project.

The national cost modeling tool programmatically evaluates each public water system (PWS) with occurrence data from WITAF 057 to generate a dataset of the most probable capital and operating costs. Those costs are then scaled up nationally to account for the PWSs without data captured in WITAF 057 to quantify the national cost of compliance of a proposed regulation, bringing flexibility for data-driven responses to EPA cost assessments. This project brought together occurrence data, cost data, and best practice design methodology to help ensure the U.S. EPA’s proposed national primary drinking water regulations for PFAS accurately reflect cost estimates for drinking water treatment.

3.0 PFAS Treatment Technologies

Treatment strategies for PFAS in drinking water include proven, commercially available technologies as well as emerging technologies. Many of these developing technologies have been demonstrated on the bench scale but have not yet been proven at the full scale or are not yet commercially available. Commercially available technologies that have been demonstrated at full scale in the field to reduce concentrations of PFAS in drinking water are limited to the following:

- Granular activated carbon (GAC).
- Ion exchange (IX).
- Nanofiltration (NF) and reverse osmosis (RO).

Treatment considerations for the application of each of these technologies are described in the following subsections.

3.1 Granular Activated Carbon

GAC media is a well-known adsorbent for organics and has been widely applied in water treatment. GAC is produced from carbon-based materials such as coal, coconut shells, peat, or wood that has been “activated” to produce a highly porous media with adsorptive properties. The pores contain sites on which organic compounds become attached and are adsorbed onto the activated carbon matrix.

GAC treatment applications include removal of organics, such as color, disinfection byproducts (DBP) and their precursors, taste and odor (T&O) causing compounds, industrial chemicals, and emerging contaminants such as PFAS, endocrine disrupting compounds, and pharmaceuticals and personal care products. Each of these contaminants compete for adsorption sites on GAC media with targeted PFAS if present. In some cases, co-adsorption can be viewed as a benefit for using GAC as the co-contaminants are simultaneously removed. Cost analyses and removal performance models must balance competitive adsorption of co-contaminants and its associated detrimental performance impact on PFAS removal.

GAC has a finite capacity for adsorbing compounds. High concentrations of organics or high flow rates will lead to more frequent media replacement. In general, short-chained PFAS are less readily adsorbed and less strongly bound than long chain compounds. The overall efficacy of GAC removal of PFAS highly dependent on the water matrix, the water treatment goals, and the design of the system. One of the most important design parameters is the empty bed contact time (EBCT), or the time during which the water is in contact with the media bed (also the duration at which adsorption can occur), assuming the water flows through the entire bed at a constant velocity. A desired EBCT will result in breakthrough when the adsorptive capacity of the media has been exhausted. The media must be either replaced or reactivated at that time.

3.1.1 Implementation and Operational Considerations

GAC applied for PFAS removal is most effective when used solely as an adsorbent. Conventional granular media filters containing GAC are typically designed for short EBCTs and must be frequently backwashed for removal of particulate material that is retained in the media. Such backwashing disrupts the adsorption front. Short EBCTs and backwashing lead to fast breakthrough of contaminants and underutilization of GAC media. If a water treatment facility contains conventional filters, contactors for GAC adsorption are typically located downstream.

Process selection (including GAC media selection) is typically confirmed through demonstration testing (bench-, pilot- or full-scale studies) to account for the unique characteristics of the source water.

GAC adsorption treatment systems installed for PFAS removal typically provide a 10 to 20 minute EBCT and a surface loading rate of 4 to 10 gallon per minute (gpm) per square foot of media (gpm/sf). PFAS adsorbers are applied in two main configurations: pressure vessels or gravity basins.

- Pressure vessel configurations are more common in small systems (less than approximately 10 million gallons per day [mgd]). Pre-engineered pressure-vessel type GAC treatment systems are widely available. Vessels are typically carbon steel or fiberglass reinforced plastic (FRP). Pressure vessels may be installed in single (parallel) or dual stage (series/lead-lag) arrangements.
 - The single stage arrangement allows for columns to be operated in various stages of breakthrough or exhaustion, resulting in an overall effluent below the treatment target. This arrangement can result in better media utilization, produce a more consistent product water quality, and lessen impact of potential overruns on individual vessels. Single stage systems typically include N+1 redundancy.
 - The dual stage arrangement allows for simultaneous production during media replacement, and sampling between vessels ensures that lag vessel effluent always meets treatment targets. The lead vessel can be in service until the media is completely exhausted, leading to higher utilization of the adsorbent media. The dual stage arrangement includes built-in redundancy as either the lead or lag vessel can be removed from service without reducing the treatment flow rate. Thus, no dedicated redundant vessels are typically provided.
- To avoid an excessive number of pressure vessels, gravity basin configurations are typically applied by large systems with design flows greater than approximately 10 mgd. Gravity basins are typically single stage and operated at various stages of breakthrough, similar to a single stage pressure vessel arrangement. The basins themselves are typically constructed of concrete with an N+1 redundancy because of the single stage arrangement.

Exhausted GAC filter media will be saturated with PFAS. Bulk GAC can be reactivated by the media supplier through thermal treatment at high temperatures (up to 1800° F) to remove and destroy adsorbed contaminants (Rebecca DiStefano, 2022). This reactivation process restores the media's adsorptive capacity, allowing the media to be returned for reuse. GAC is sometimes regenerated by heating the media to temperatures typically less than 400° F to remove a portion of the adsorbed contaminants. However, this process will not remove all the compounds and will not destroy the PFAS compounds; therefore, it is not appropriate for GAC utilized for PFAS removal. Media suppliers may not accept the low volumes of GAC required by small systems for reactivation, forcing them to dispose of spent GAC and replace it with new (virgin) material.

Disposal alternatives for exhausted GAC that will not be reactivated for municipal reuse include disposal by reactivation for industrial reuse, incineration, and landfilling. The cost of each disposal method depends on proximity to disposal sites, hazardous waste classification, and volume of material. Disposal costs can be a significant operational cost for GAC treatment systems.

The EPA proposed to designate PFOS and PFOA as hazardous substances under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) in August of 2022. This designation is expected to limit the disposal sites willing to accept spent GAC media. Additionally, the practice of reactivating GAC media contaminated with PFAS is expected to be more limited in drinking water applications.

3.1.2 Assumptions for Cost Estimation

The cost model includes capital costs, annual operating and maintenance costs, life-cycle costs, and annualized costs. The assumptions that drove the results of those cost estimates are summarized in this section.

The costs for GAC contactors depend on the contactor type, size, number, and ancillary processes such as backwash pumps/recovery basins and contactor influent pumps/wet wells. The primary process design assumptions for each of these factors are summarized in Table 3-1.

Table 3-1 GAC Design Process Assumptions

Contactor Type	Parameter		Assumption/Input
Pressure Vessel	Treatment Plant Capacity		1-12 mgd
	Surface Loading Rate ^(Note 1)		4-10 gpm/sf (most likely 6 gpm/sf)
	Empty Bed Contact Time ^(Note 1)		10-20 min (most likely 18 min)
	Vessel Diameter		6-12 ft
	Arrangement		Dual Stage
	Redundancy		None
	Influent Pump Station	TDH (total dynamic head)	45 ft
Gravity Basin		Design HRT (hydraulic retention time)	15 min
	Treatment Plant Capacity		> 12 mgd
	Surface Loading Rate ^(Note 1)		4-10 gpm/sf (most likely 4 gpm/sf)
	Empty Bed Contact Time ^(Note 1)		10-20 min (most likely 18 min)
	Filter Dimensions		8-20 ft cell width, 2:1 length to width ratio
	Arrangement		Single Stage
	Redundancy		N+1
	Influent Pump Station TDH, design HRT		30 ft, 15 min

Contactor Type	Parameter		Assumption/Input
Common	Backwash ^(Note 2)	Loading Rate	13 gpm/sf
		Duration	30 min
		Frequency	30 days
		Pump Design TDH	60 ft
	Influent Pump Station ^(Note 3)	Pump Efficiency	70%
		Motor Efficiency	85%
	Backwash Water Recovery Basin ^(Note 4)	Water Depth	20 ft
		Backwash Cycles Held	1.0
	GAC Media	Apparent Density	0.5 gram per cubic centimeter (g/cc)
	Contactor Area Factor (for pipe gallery and appurtenances)		2.0

Notes:

1. For adsorptive media, the major specified process design inputs are the surface loading rate (SLR) and the EBCT. For each of these factors, a minimum, maximum, and most likely number was assumed using feedback from existing treatment systems. The minimum, maximum, and most likely numbers for the published model outputs are summarized herein. National variability in SLR and EBCT is included in the model using a Monte Carlo simulation. The details of how this statistical method was employed within the cost modeling tool is described in Section 5.3.
2. Backwash pumps are required for periodic backwashing of the media.
3. An influent pump station is presumed to be required to accommodate the additional headloss necessary to an existing process train.
4. Backwash recovery basin omitted from systems for size category 1 and 2.

3.2 Ion Exchange

IX is an adsorptive water treatment process that involves the selective exchange of ions in solution with ions bound to a resin matrix. IX has a long history in water treatment, and resins are manufactured for a variety of contaminants, including PFAS. Several manufacturers provide specific IX resins designed to be selective for PFAS as the market has expanded for their use. Some resins originally intended for removal of other contaminants (such as perchlorate) have shown a high degree of selectivity and capacity for PFAS as well.

IX resins, like GAC, have a limited capacity for adsorption. The adsorptive capacity of IX resins is affected by contaminant concentrations and flow rates in the same manner as GAC. However, the IX resins surveyed have proven to be highly selective toward PFAS removal, exhibiting minimal removal of other contaminants. This may result in a greater adsorptive capacity for PFAS compared to GAC, without, however, the co-contaminant removal benefits of other technologies. In general, short-chained PFAS are less readily adsorbed and less strongly bound than long chain compounds. The overall efficacy of IX for PFAS removal is highly individual to the water matrix, the water treatment goals, and the design of the system.

An IX treatment process does not result in a fixed percentage removal of a contaminant over time, as there is a variable degree of contaminant removal and gradual or sharp contaminant breakthrough. Although it is selective to certain contaminant groups, the resin can experience interference from other compounds in the water matrix. The most preferred compound will tend to exhibit long runs and sharp breakthroughs; less preferred compounds will have earlier, more gradual breakthroughs.

Exhaustion of the media is determined (in a fashion similar to that for GAC) through the measure of the contaminant in the effluent (breakthrough). When the adsorptive capacity has been exhausted, the resins require replacement or regeneration. Because of the proposed CERCLA hazardous substance designations for PFOA and PFOS as discussed in Subsection 3.1.1, single use (fixed-bed) systems are currently being considered for IX, requiring disposal of spent media and replacement with new resin when exhausted. PFAS destruction technologies are currently in research and development that may be able to destroy PFAS in the brine stream, although that technology is not yet matured enough for full-scale implementation.

Fixed-bed IX has been demonstrated at full scale in the field as a proven PFAS removal technology. Fixed-bed ion exchangers applied for PFAS removal consist of carbon steel or FRP pressure vessels and typically 1.5 to 3 minutes of EBCT (as compared to 10 to 20 minutes for GAC). IX can be favorable because of the smaller footprint required.

3.2.1 Implementation and Operational Considerations

The efficacy of an IX treatment system will likely be improved by a pretreatment step to remove interferences such as suspended solids, particulate natural organic matter, and colloidal compounds. Commercially available filters can be selected depending on the pretreatment needs to improve the treatment capacity of the IX system. This prefiltration step can prevent deposition of fine particles on the resin, reduce pressure drop across a column, and increase run time.

Process selection (including resin selection) is typically confirmed through demonstration testing (bench-, pilot- or full-scale studies) to account for the unique characteristics of the source water.

Ion exchange treatment systems are conventionally installed in pressure filters in lieu of gravity basins. As with GAC, the pressure vessels can be implemented in single or dual stage arrangements. Considerations for the single or dual stage arrangements are summarized in Subsection 3.1.1.

Exhausted IX resin will be saturated with PFAS. Disposal alternatives for exhausted IX resins include incineration and landfilling. The cost of each disposal method depends on proximity to disposal sites, hazardous waste classification, and volume of material. Disposal costs can be a significant operational cost for IX treatment systems.

3.2.2 Assumptions for Cost Estimation

The costs for IX Contactors depend on the contactor type, size, number, and ancillary processes such as backwash pumps/recovery basins and contactor influent pumps/wetwells. The primary process design assumptions for each of these factors are summarized in Table 3-2.

Table 3-2 IX Design Process Assumptions

Parameter		Assumption/Input
Surface Loading Rate ^(Note 1)		5-12 gpm/sf (most likely 8 gpm/sf)
Empty Bed Contact Time ^(Note 1)		1.5-3.0 min (most likely 2.0 min)
Vessel Diameter		4-12 ft
Contact Mode		Lead-Lag
Redundancy		None
Influent Pump Station ^(Note 2)	Pump Efficiency	70%
	Motor Efficiency	85%
	TDH	60 ft
	Design HRT	15 min
Backwash ^(Note 3)	Loading Rate	5 gpm/sf
	Duration	30 min
	Frequency	30 days
	Pump Design TDH	60 ft
Backwash Water Recovery Basin ^(Note 4)	Water Depth	20 ft
	Backwash Cycles Held	1.0
IX Resin	Apparent Density	1.05 g/cc
Contactor Area Factor (for pipe gallery and appurtenances)		2.0

Notes:

1. For adsorptive media, the major specified process design inputs are the SLR and the EBCT. For each of these factors, a minimum, maximum, and most likely number was assumed using feedback from existing systems. The minimum, maximum, and most likely numbers used for the published model outputs are summarized herein. National variability in SLR and EBCT is included in the model using a Monte Carlo simulation. The details of how this statistical method was employed within the cost modeling tool is described in Section 5.3.
2. An influent pump station is presumed to be required to accommodate the additional headloss necessary to an existing process train.
3. Backwash pumps are required for periodic backwashing of the media.
4. Backwash recovery basin omitted from systems for size category 1 and 2.

3.3 Reverse Osmosis and Nanofiltration

RO and NF are membrane-based water treatment processes in which a semi-permeable barrier removes dissolved contaminants from water. This capability is attractive when considering the need to remove total dissolved solids (TDS), specific ions such as calcium, magnesium, sodium, chloride, sulfate, and hardness; DBP precursors; and T&O causing compounds as well as high levels of PFAS. RO/NF processes are commonly applied in water treatment plants and have applications ranging from desalination of brackish water, softening, and the removal of nitrate, agricultural chemicals (e.g., atrazine), color, total organic carbon (TOC), DBP precursors, and PFAS. Both RO and NF processes are capable of a high rejection of PFAS. While RO/NF systems are more expensive than GAC or IX systems, they are most viable when the GAC/IX replacement frequency requirements are cost-prohibitive because of high concentrations of influent PFAS.

The key differences between RO and NF are salt passage and feed pressure. RO membranes reject a higher percentage of dissolved ions in the feed water and require a greater feed pressure than NF membranes. NF membranes preferentially remove larger divalent ions or molecules compared to monovalent ions. Thus, NF systems generally exhibit lower energy use and lower operating cost than RO systems. The lower feed pressure required for NF generally translates to a slightly favorable capital cost in relation to RO systems treating the same flow rate. However, the benefits of higher salt rejection and flexibility of systems designed for RO to utilize either NF or RO membranes typically results in utilities favoring RO over marginally lower cost NF systems.

For a typical RO/NF system, membrane elements are mounted into pressure vessels that are arranged in stages, banks, or arrays. The number of stages required depends on specified recovery. Two stages are typically used for recovery less than 80 percent, and three stages are required for higher recovery. RO/NF is a cross flow filtration method, in which only a portion of the feedwater becomes permeate (finished water). The remainder leaves the system as concentrate (brine) that carries away the concentrated material before precipitation or scaling forms on the membrane surface or in the device. Antiscalant is used to control the precipitation of sparingly soluble salts such as calcium carbonate, calcium sulphate, barium sulfate, calcium fluoride, silicon dioxide, etc.

3.3.1 Implementation and Operational Considerations

The recovery of the RO/NF treatment systems depends on the concentrations of the sparingly soluble salts and typically ranges from 75 to 85 percent. Pretreatment requirements include pH depression, antiscalant chemical products to reduce scaling, and cartridge filters to protect the RO/NF membranes from particulates.

The combination of pH depression in the feedwater and the removal of alkalinity through the process results in a low pH (acidic) finished water. Gases pass through NF/RO membranes, resulting in the potential need for removal of hydrogen sulfide and carbon dioxide from the treated water. Post-treatment generally consists of gas stripping through a decarbonation tower and chemical conditioning by addition of a base such as lime or sodium hydroxide (caustic) to raise pH, alkalinity, and hardness to render the water less corrosive. Sometimes a corrosion inhibitor is also added to prevent distribution system corrosion.

A major challenge to implementing centralized NF/RO treatment for PFAS removal is in dealing with the concentrated waste stream generated by the treatment process. Contaminants are rejected into a waste brine stream that is typically around 15 percent by volume of the feedwater (for low salinity feed waters) and 4 to 7 times more concentrated than the raw water fed to the membranes. As a result,

additional raw water is required to achieve the desired finished water capacity, and the waste stream requires disposal. Traditional alternatives for disposal include sending the stream to a downstream water reclamation facility, discharging to surface water, or injection into underground deep wells. However, because of the CERCLA regulations for PFOA and PFOS as discussed in Subsection 3.1.1 and pending effluent limit goals for PFAS, concentrate treatment may be required before disposal using these methods.

3.3.2 Assumptions for Cost Estimation

The costs for RO systems depend on the number of trains, permeate flow, and ancillary processes such as the RO feed tank, low-pressure feed pump, high-pressure feed pump, chemical pretreatment, chemical post-treatment, flush pump/tank, clean-in-place (CIP) system, decarbonation system, building requirements, and brine disposal. The primary process design assumptions for each of these factors are summarized in Table 3-3.

Table 3-3 RO Design Process Assumptions

Sub-System	Parameter	Assumption/Input
RO System Design	Recovery ^(Note 1)	70-85%
	PFAS Rejection	95%
	RO Element Membrane Area	400 ft ²
	Design Flux	15 gallons per foot-squared per day (gfd)
	Redundancy	N+1
	Concentrate Recycle	0%
	Number of Elements per Pressure Vessel	6
	First Stage Pressure Vessel Ratio	4
	Second Stage Pressure Vessel Ratio	2
	Third Stage Pressure Vessel Ratio	1
RO Feed Tank	Hydraulic Detention Time	30 min
RO Low Pressure Feed Pump Sizing	Pump Design TDH	30 ft
RO High Pressure Feed Pump Sizing	Pump Design TDH	350 ft
Chemical Pretreatment ^(Note 2) Antiscalant Chemical	Density	10.01 pounds per gallon (lb/gal)
	Design Dose	3 mg/L
	Storage	30 days

Sub-System	Parameter	Assumption/Input
Chemical Pretreatment ^(Note 2) Sulfuric Acid (98%)	Density	15.26 lb/gal
	Design Dose	30 milligrams per liter (mg/L)
	Storage	30 days
Chemical Post-Treatment ^(Note 2) Caustic (50%) or Liquid Lime	Density	12.78 lb/gal
	Design Dose	45 mg/L
	Storage	30 days
RO Flush Pump Sizing	Flow Rate per Pressure Vessel	30 gpm/1st stage pressure vessel
	Pump Design TDH	140 ft
	Flush Frequency	12 hrs/yr/train
RO Flush Tank Sizing	Volume per Pressure Vessel	7 cubic feet (ft ³)
	Number of Flushes in Tank	2
	Safety Factor	50%
CIP System Sizing	Flush Flow	50 gpm/1st stage pressure vessel
	Time/skid	4 minutes
	CIP Pump TDH	140 ft
	CIP Interval	90 days
	Time/CIP	6 hrs
	CIP Temperature Increase	65 °F
	Heater Losses	10%
Forced Draft Degasifier (decarbonation)	Loading Rate	30 gallons per minute per square foot (gal/min/ft ²)
Building Calculations	RO Equipment Area Factor	2.0
	Unit Area	880 ft ² /mgd

Sub-System	Parameter	Assumption/Input
Brine Disposal		Deep Well Injection
Deep Well Injection	Flow per well	1 mgd

Notes:

1. For RO, the critical design input is percent recovery. A minimum and maximum recovery, but no most likely number, is specified. The minimum and maximum recovery used for the published model outputs are summarized herein. National variability in recovery is included in the model using a Monte Carlo simulation. The details of how this statistical method was employed within the cost modeling tool is described in Section 5.3.
2. Chemical systems include pumps, bulk storage, piping, and containment. No day tanks were included in the estimate.

4.0 Estimating National Occurrence

To estimate the costs of removing PFAS from drinking water nationally, national occurrence must be characterized. In parallel to this project, AWWA funded WITAF 057 to compile an occurrence database for PFAS in drinking water. In addition to data available for UCMR 3, WITAF 057 facilitated the collection of PFAS monitoring data from state databases and integrated these sources into a single data set. PWSs in this database included only active Community Water Systems (CWSs) and active Non-Transient Non-Community Water Systems (NTNCWSs). The inactive and transient non community water systems were eliminated from the dataset. Consecutive systems receiving all water from treated water wholesaler systems were not excluded from the database or from representation in the national cost estimation.

The WITAF 057 dataset consisted of 7,842 PWSs within these categories as compared to the 49,193 PWSs in the Safe Drinking Water Information System (SDWIS). To account for this incomplete occurrence data, the percent of systems impacted by a potential PFAS regulation within each system size category was multiplied by the active number of CWSs or NTNCWSs in EPA's SDWIS system at each size category to estimate the anticipated number of total water systems impacted in each size category. This methodology therefore assumed that existing occurrence data is representative of national occurrence. This assumption is considered conservative given a significant fraction of existing occurrence data came from UCMR 3, where the reporting limits of 20 parts per trillion (ppt) and 40 ppt for PFOA and PFOS, respectively, likely bias existing occurrence data to underrepresent true national occurrence that would be measured using the current reporting limits.

Monitoring data for PFAS compounds in the WITAF 057 database included more than 30 individual compounds but for this work was limited to the six PFAS covered by UCMR 3: PFOS, perfluoroheptanoic acid (PFHpA), PFHxS, PFNA, and PFBS. As compiled, the WITAF 057 database includes all monitoring results under UCMR 3 and various state monitoring programs, which may include multiple sample results for specific PFAS at a given PWS. Reported data were reviewed to ensure correct translation of reporting units; fields were included for PWS identification number, state, number of people served, source type, and system type. These data were analyzed to determine the maximum and average sample results for each PFAS at each PWS in the database.

5.0 Individual Treatment Facility Cost Methodology

The next step in estimating the national costs to remove PFAS from drinking water is to use the occurrence database to estimate the costs associated with treatment for individual PWSs. The following subsections summarize how capital, operating, and life-cycle costs are calculated for each system and for each technology.

The spreadsheet tool developed to perform this task accepts inputs for individual or combined target effluent levels for the six PFAS compounds represented in the database. After both occurrence data and potential regulatory levels are input, Visual Basic scripts within Excel may be initiated by a user to run a Monte Carlo analysis and generate a 10th percentile, 90th percentile, and most probable costs for the capital, operations and maintenance (O&M), and life-cycle costs for a typical entry point to the distribution system (EPTDS) for each PWS in the database. For each system, the tool selects the treatment technology with the lowest life-cycle cost.

This methodology assumes installation of a treatment system at each EPTDS associated with PWSIDs where the maximum PFAS concentration is greater than the potential regulatory level for the corresponding PFAS. The details of individual system and EPTDS cost methodology are described in the following subsections. A list of output fields generated by the cost modeling tool for each PWS with occurrence data is shown in Table 5-1.

Table 5-1 Model Outputs for Individual PWS with Occurrence Data

Model Outputs for Each PWS with Occurrence Data
Design Flow (mgd)
Average Flow (mgd)
Capital Expenditure for GAC Vessels
Annual Operations and Maintenance Costs for GAC Vessels
Life-Cycle Costs for GAC Vessels
Capital Expenditure for GAC Basins
Annual Operations and Maintenance Costs for GAC Basins
Life-Cycle Costs for GAC Basins
Capital Expenditure for Ion Exchange Vessels
Annual Operations and Maintenance Costs for Ion Exchange Vessels
Life-Cycle Costs for Ion Exchange Vessels
Capital Expenditure for Reverse Osmosis
Annual Operations and Maintenance Costs for Reverse Osmosis
Life-Cycle Costs for Reverse Osmosis
Capital Expenditure for Lowest Life-Cycle Cost Technology
Annual Operations and Maintenance Costs for Lowest Life-Cycle Cost Technology

Model Outputs for Each PWS with Occurrence Data
Life-Cycle Costs for Lowest Life-Cycle Cost Technology
10th Percentile Capital Expenditure for Lowest Life-Cycle Cost Technology
10th Percentile Operations and Maintenance Cost for Lowest Life-Cycle Cost Technology
10th Percentile Life-Cycle Cost for Lowest Life-Cycle Cost Technology
90th Percentile Capital Expenditure for Lowest Life-Cycle Cost Technology
90th Percentile Operations and Maintenance Cost for Lowest Life-Cycle Cost Technology
90th Percentile Life-Cycle Cost for Lowest Life-Cycle Cost Technology
Capital Expenditure for Manganese Pretreatment
Annual Operations and Maintenance Costs for Manganese Pretreatment
Life-Cycle Cost for Manganese Pretreatment
Lowest Life-Cycle Cost Treatment Technology

5.1 Determining Design Parameters

5.1.1 Treatment Design Flow Determination

PWS data available in SDWIS do not include water usage data for each PWS and EPTDS. Instead, service population data from SDWIS was used and the average flow for each PWS was assumed based on a per capita per day usage of 150 gallons. While not reflective of each state's dynamics with respect to water usage, this was considered a reasonable number from a national perspective. Peaking factors for different size systems from the EPA's "Cost and Technology Document for Final Groundwater Rule" were used and are shown in Table 5-2. The trend of this dataset was best fit to a power equation to calculate peaking factor as a function of average daily flow as shown on Figure 5-1.

Table 5-2- EPA Peaking Factor for Various Average System Flows

Design Flow (MGD)	Average Flow (MGD)	Peaking Factor	Design Flow (MGD)	Average Flow (MGD)	Peaking Factor
0.007	0.0015	4.7	2	0.77	2.6
0.022	0.0054	4.1	3.5	1.4	2.5
0.037	0.0095	3.9	7	3	2.3
0.091	0.025	3.6	17	7.8	2.2
0.18	0.054	3.3	22	11	2
0.27	0.084	3.2	76	38	2
0.36	0.11	3.3	210	120	1.8
0.68	0.23	3	430	270	1.6
1	0.3	3.3	520	350	1.5

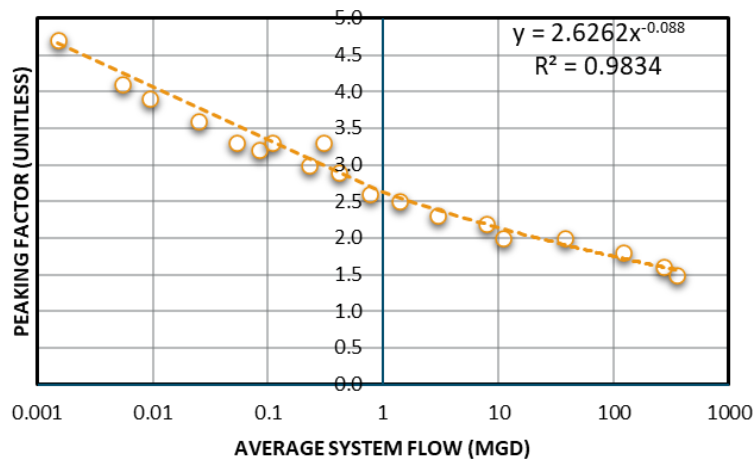


Figure 5-1 Peaking Factor as a Function of Average System Flow

The treatment design flow per EPTDS was determined by Equation 1:

$$\text{Design Flow per EPTDS} = \frac{(\text{number of customers per PWS})(150 \text{ gpcd})(\text{peaking factor})}{\text{EPTDS per PWS size category}} \quad (1)$$

Where:

$$\text{Peaking factor} = 2.6262(\text{PWS Average Flow})^{-0.088}$$

The estimated number of EPTDS per system size is based on an evaluation by EPA published with the proposed national primary drinking water regulation for PFAS from March 2023. The number of EPTDS per system broken out by groundwater and surface water systems within each system size bin is summarized in Table 5-3.

Table 5-3 Number of EPTDS as a Function of System Size

Size Category	Population Range	Entry Points/System	
		Groundwater	Surface Water
1	0-100	1	1
2	101-500	1	1
3	501-1,000	2	1
4	1,001-3,300	2	1
5	3,301-10,000	2	1

Size Category	Population Range	Entry Points/System	
		Groundwater	Surface Water
6	10,001-50,000	4	1
7	50,001-100,000	10	2
8	100,001-1,000,000	12	2
9	>1,000,001	39	4

5.1.2 Water Quality Considerations Incorporated

5.1.2.1 Influent and Effluent PFAS Levels

For each PWS in the occurrence database, any single PFAS monitoring result above either existing state or potential regulatory limit was assumed to incur a capital expenditure for treatment. Data down to the resolution of each individual source was not considered for this modeling effort; instead, the number of projected water treatment facilities per system was based on the EPTDS factors as summarized in the previous section. Maximum PFAS monitoring data were assumed to compel treatment for the PWS as a whole and, thus, all the projected water treatment facilities. The average PFAS monitoring data were used to estimate long-term costs of removal (annual O&M costs).

The target effluent PFAS levels for treatment was determined as an input percentage of a potential regulatory limit. For example, treatment could be triggered at 80, 90, or 100 percent of the potential regulatory level. For this work, a threshold of 80 percent was used in alignment with previous practice for estimating costs of potential regulations for drinking water, since water systems will target and operate below this threshold to ensure that the limit is not exceeded if the water quality suddenly increases.

5.1.2.2 Other Water Quality Considerations

Other water quality contaminants may impact PFAS treatment performance (and therefore costs), such as TOC and manganese. The longevity of GAC media, IX resin, and membrane operations are significantly affected by the quality of the source. Differences in source water quality parameters not specifically included (e.g., TOC, sulfate, pH, alkalinity, etc.) with pertinence to design or performance were reflected in cost by varying design parameters and treatment system performance according to probability functions using Monte Carlo analysis. This is primarily controlled through variation of the treatment performance factors (e.g., EBCT, surface area loading rate) to reflect less or more challenging water quality characteristics. The methodology for the Monte Carlo Simulation is covered in Section 5.2. Work is in progress to estimate costs associated with removing manganese and will be made available at a later date.

5.2 Monte Carlo Simulation for Design and Performance Variability

Water treatment system design is a practice that evolves non-uniformly across the country. Decisions in the design process are driven in some cases by rigorous engineering standards and in others by regional and geographic considerations, or owner and operator preferences. The result is a landscape of treatment systems across the United States that cannot be effectively modeled by clear and simple rules and frameworks. Additionally, water quality characteristics vary both regionally and locally, and these variations cannot be fully captured in the model with distinct data. These water quality characteristics

may improve or hinder performance as well as increase costs to ensure water quality downstream is not altered and complies with other regulations.

To compensate for this uncertainty, Monte Carlo methods were applied to simulate variation and to account for unknowns in major factors influencing design, operation, and, ultimately, cost for PFAS reduction systems. The @RISK Probabilistic Risk Analysis Software by Lumivero, which functions through an Excel add-in, was utilized for the Monte Carlo analysis.

Monte Carlo methods consist of randomizing inputs (e.g., loading rate, GAC media life, RO recovery) according to a defined distribution and number of iterations while calculating the impact to the outputs (e.g., number of vessels, media replacement frequency, cost). As the number of variables undergoing Monte Carlo analysis increases, computer processing power and the time to simulate one scenario both increase exponentially. Thus, Monte Carlo analysis was limited to only major factors considered to exert significant influence on design, performance, and cost of the individual systems. The major factors subjected to Monte Carlo are shown in Table 5-4.

Table 5-4 Major Factors for Monte Carlo Analysis

Parameter	Value
GAC - Pressure	
Surface Loading Rate	
Distribution Type	Triangular
Minimum Value	4 gpm/sf
Maximum Value	10 gpm/sf
Most Likely Value	6 gpm/sf
EBCT	
Distribution Type	Triangular
Minimum Value	10 min
Maximum Value	20 min
Most Likely Value	18 min
GAC - Basins	
Surface Loading Rate	
Distribution Type	Triangular
Minimum Value	4 gpm/sf
Maximum Value	10 gpm/sf
Most Likely Value	4 gpm/sf
EBCT	
Distribution Type	Triangular

Parameter	Value
Minimum Value	10 min
Maximum Value	20 min
Most Likely Value	18 min
GAC Bed Volumes to Breakthrough ^(Note 1)	
Distribution Type	Triangular
Minimum Value	75 percent of prediction
Maximum Value	175 percent of prediction
Most Likely Value	Prediction
IX - Vessels	
Surface Loading Rate	
Distribution Type	Triangular
Minimum Value	5 gpm/sf
Maximum Value	12 gpm/sf
Most Likely Value	8 gpm/sf
EBCT	
Distribution Type	Triangular
Minimum Value	1.5 min
Maximum Value	3 min
Most Likely Value	2 min
IX Bed Volumes to Breakthrough ^(Note 1)	
Distribution Type	Triangular
Minimum Value	75 percent of prediction
Maximum Value	175 percent of prediction
Most Likely Value	Prediction
Reverse Osmosis/Nanofiltration	
Surface Loading Rate	
Distribution Type	Uniform
Minimum Value	70 percent
Maximum Value	85 percent
Notes:	

Parameter	Value
1. GAC and IX Performance (i.e. determination of media life) is described in Section 5.4.1. Predicted value is determined using the generalized logistic function of the Clark model.	

With the exception of RO recovery, all Monte Carlo inputs were assigned a triangular distribution. A triangular distribution is a probability distribution where the probability decreases linearly on either side of the most likely value (highest probability) to the minimum and maximum, at which point the probability is zero. Triangular distributions were used where typical industry design values exist. RO recovery was modeled using a uniform distribution where each value between the minimum and maximum have an equivalent probability of occurrence.

The result of the Monte Carlo analysis is a distribution of possible costs for each technology (i.e., low [10th percentile], high [90th percentile], and most probable). For each modeled scenario, each of these costs was stored as a modeled output for each system represented in the occurrence database for use in determining the overall national cost of compliance with the modeled limit.

5.3 Capital Cost Calculation

Capital costs were calculated for each EPTDS of a PWS based on the design flow per EPTDS (refer to Equation 1). The design flow was used for capital costs estimates since equipment should be sized for peak treatment flow rates. Costs were independently calculated for IX, GAC vessels, GAC basins, and RO as described in the following subsections. Capital costs generated for individual systems represent a Class 5 Association for the Advancement of Cost Engineering (AACE) estimate, at approximately 1 to 2 percent maturity level of deliverable definition.

5.3.1 Major Hardware Components

5.3.1.1 GAC Gravity Basins

The major cost components incorporated into the capital cost estimate for this option are the concrete basins themselves, an influent pump station, media for the initial fill, and a building to house the system. The design assumptions for each element are summarized in Subsection 3.1.2.

The concrete basin includes costs for influent and effluent piping, isolation valves, and monitoring instruments. Using the design flow rate and the SLR, a required surface area for filtration is calculated and used to determine the appropriate number of basin cells and anticipated basin dimensions for costing.

Once number and size of basins are calculated, the design flow and specified EBCT is used to determine the volume of media needed. Cost of media was determined by converting volume to mass using an average GAC density of 0.5 g/cc and an average cost per pound of \$1.40. It should be noted that cost changes were not projected into the cost model resulting from increased demand for adsorbent media.

The pump station includes costs for influent pumps, backwash pumps, an influent wetwell, and a backwash recovery basin. The independent design inputs for the influent pumps are total dynamic head (TDH) and total number of pumps. The independent design parameters for backwash pump and backwash recovery basin calculations are backwash loading rate, backwash duration, backwash frequency, and backwash pump TDH. Costs for backwash pumping include a single duty pump and a single standby pump.

The sum of the square footage required for the contactor basins was multiplied by a sizing factor of two to account for the ancillary equipment and space for access and maintenance. Pump station square footage, including all pumps and the wet well, was estimated by benchmarking design flow against previous designs. Building area was assumed to be the sum of contactor facility area (including sizing factor), pump station area, and backwash recovery basin area (assumed to be indoors). The building cost was assumed to be \$200/sf.

Black & Veatch utilized empirically derived cost curves as a function of size from several decades of infrastructure project design and delivery to estimate cost for these major components. A curve for concrete basins provides cost as a function of square footage. A curve for steel tanks provides costs as a function of volume in gallons, and a curve for pumps provides cost as a function of horsepower.

Installation fees were included at 20 percent for all major equipment components, as summarized in Table 5-5. These cost factors are identical to those for GAC and IX pressure vessels.

Table 5-5 GAC and IX Equipment Installation Cost Factors

Component	Percent Multiplier of Unit Cost
Basins/Pressure Vessels	20%
Influent Pumps	20%
Backwash Pumps	20%
Influent Wetwell	20%
Backwash Recovery Basin	20%

5.3.1.2 GAC, IX and Manganese Pretreatment Pressure Vessels

Capital equipment costs were calculated using the total contactor footprint, contactor building footprint, and media volume required. Capital costs were calculated for the ancillary pump stations using the building footprint, number and size of influent pumps, backwash pumps, influent wetwell, and backwash recovery basin. The model incorporated a building cost of \$200/ft². The installation fees for the various components are the same as those summarized in Table 5-6.

Calculated capital cost for manganese pretreatment for each system was considered a stand-alone output and was not included in the capital, operational, or life-cycle cost outputs for PFAS treatment.

5.3.1.3 Reverse Osmosis

Capital costs were calculated for the RO system and building, low- and high-pressure feed pumps and their associated building, storage tanks, cartridge filters, chemical treatment system, decarbonation system, and brine disposal. The model incorporated a building cost of \$200/ft². The installation fees for the various components are summarized in Table 5-6.

Table 5-6 RO Equipment Installation Cost Factors

System	Component	Percent Multiplier of Unit Cost
Storage Tanks	RO Feed Tank	15%
	CIP Tank	15%
	CIP Neutralization Tank	15%
	Flush Tank	15%
Pump Stations	RO Low Pressure Feed Pumps	25%
	RO High Pressure Feed Pumps	25%
	CIP Pumps	20%
	Flush Pumps	20%
Cartridge Filter	RO Feed Cartridge Filter	20%
	CIP Cartridge Filter	20%
Chemical Feed Systems	Antiscalant	20%
	Sulfuric Acid	20%
	Caustic/Liquid Lime	20%
Decarbonation System	All related equipment	20%

5.3.1.4 Additional Capital Costs

In addition to equipment costs, the capital costs for GAC, IX, RO, and manganese pretreatment included additional project costs (site work, yard piping, electrical, and instrumentation and controls), contractor markup costs, and non-construction costs. The multipliers used for each of these factors are summarized in Table 5-7.

Table 5-7 Additional Capital Cost Assumptions

Additional Capital Costs	Description	Percent Multiplier of Total Equipment Costs
Additional Project Costs	Site Work	8.0%
	Yard Piping	9.0%
	Electrical	10.0%
	Instrumentation & Controls	2.5%

Additional Capital Costs	Description	Percent Multiplier of Total Equipment Costs
Contractor Markup Costs	Overhead	7.0%
	Profit	10.0%
	Mobilization/Bonds/Insurance	3.0%
	Contingency	4.0%
Non-Construction Costs	Permitting	1.0%
	Engineering	8.0%
	Legal/Administration	0.5%
	Construction Services	7.0%
	Commissioning/Startup	3.0%
	Contingency	30.0%

5.4 Operating Cost Calculation

The operational costs for GAC, IX, and RO were calculated using the average flow rate for each EPTDS, as represented by the average flow per water system divided by the number of EPTDS. Whereas capital costs were driven by maximum PFAS levels, the operating costs incurred were driven by the average influent PFAS concentrations to reflect long-term operating conditions. The tool allows entry of a treatment goal expressed as a percent of the potential regulatory limit, and the resulting target concentration serves as the effluent concentration trigger for replacement of media. This target may be expressed either as a concentration of a single PFAS compound or as a combination of compounds.

Operating costs that were considered for this work included replacement costs (using the calculated bed volumes to breakthrough or media replacement frequency), power consumption in the pumps and buildings, maintenance costs, waste disposal, and labor costs. Analytical monitoring costs were not included in the life-cycle cost calculations. Table 5-8 provides an overview of the O&M cost assumptions.

Table 5-8 O&M Cost Assumptions

O&M Category	Description	Value
Media Replacement	GAC Virgin Media ^(Note 1)	\$1.40/lb
	GAC Reactivated Media	\$1.20/lb
	IX Resin	\$240/ft ³ (\$3.70/lb @ apparent density of 1.05 g/cc)
Membrane Replacement	Membrane Cost	\$600/element

O&M Category	Description	Value
Power	Unit Cost	\$0.10/kilowatt-hour (kWh)
	Unit Building Power Usage	19.5 kWh/ft ² /yr
	Building Utilization Factor	365 days/year
Maintenance	Installed Equipment	1.5% Percent Multiplier of Capital Costs
	Structures and Facilities	1.0% Percent Multiplier of Capital Costs
Waste Disposal	Incineration ^(Note 2)	\$720/ton
	GAC Density	0.5 g/cc
	IX Density	1.05 g/cc
	Mn Adsorptive Media Density	1.8 g/cc
Chemical Consumption Costs	Antiscalant	\$15.00/gal
	Sulfuric Acid	\$2.50/gal
	Caustic	\$4.50/gal
Labor	Operator Rate	\$30/hr
	Admin Rate	\$25/hr
	Number of Valves	3 per vessel/basin, 2 per pump (additional requirements for RO system include 2 per cartridge filter, 3 per decarbonation system, and 2 per tank)
	Number of Instruments	2 per vessel/basin, 1 per pump (additional requirements for RO system include 2 per cartridge filter, 2 per decarbonation system, and 1 per tank)
	Record Keeping and Sampling	5 minutes per day per instrument
	Pump Operation (adjustments)	5 minutes per day per pump
	Valve Adjustments	5 minutes per week per valve
	GAC Contactor Maintenance	1 hour per week per vessel/basin
	IX Replacement	16 hours per bed volume
	Cartridge Filters	12 hours per year per cartridge filter
	RO Membrane Process Labor	120 hours per week

Notes:

1. Life-cycle cost factors were chosen to match the EPA's standard practice for estimating life-cycle cost
2. Spent GAC media and IX resin was assumed to be incinerated because of the unknown viability of GAC media reactivation under CERCLA. Replacement costs were therefore assumed to be virgin media.

5.4.1 Estimation of Media Life and Disposal

The generalized logistic function of the Clark model (Clark, 1987), represented in Equation 2, was the basis for calculations for estimation of media life for both GAC and IX. While more rigorous techniques exist for modeling adsorption, Clark's model was utilized for its relative simplicity and accuracy.

$$C = \frac{C_o^{n-1}}{1 + B e^{-r't}} \quad (2)$$

Where:

C_o is the influent contaminant concentration, C is the concentration of a given contaminant at time t , n is the inverse of the slope of the Freundlich isotherm, and r' and B are constants. Rearranging the equation above to:

$$\ln \left[\left(\frac{C_o}{C} \right)^{n-1} - 1 \right] = -r't + \ln B$$

r' and B can be solved for from the slope and intercept of the plot of $\ln[(C_o/C)^{1/n}-1]$ versus time. If a constant flow is assumed, the number of bed volumes becomes directly proportional to time, allowing these relationships to be expressed as a function of bed volumes treated rather than time. B , n , and r' values utilized for GAC and IX are expressed in Table 5-9. The values utilized for GAC were derived from data collected during a Black & Veatch GAC pilot study for CFPWA. The values utilized for IX were derived partially from data collected during a Black & Veatch IX pilot study for CFPWA and partially from data collected during an IX pilot study for La Habra Height County Water District (LHHCWD).

Table 5-9 Values Variables in Modeled Bed Life

Media	Constant	PFOA	PFOS	PFHxS	PFNA	PFHpA	PFBS
IX ^(Note 1)	n	1.25	1.25	1.25	1.25	1.25	1.25
	B	4.8	1.8	1.4	6.0	3.6	3.2
	$-r'$	-2.55E-05	-3.33E-06	-3.40E-06	-1.70E-05	-2.62E-05	-6.23E-06
GAC	n	1.49	1.54	3.23	1.79	1.67	1.56
	B	141.7	15.8	666.0	49.1	49.1	11.3
	$-r'$	-6.21E-04	-2.07E-04	-3.77E-04	-3.46E-04	-4.81E-04	-3.28E-04

Notes:

- Parameters for PFOA and PFHpA were derived from the CFPWA data set. Parameters for PFBS, PFHxS, and PFOS were derived from the LHHCWD data set. Parameters for PFNA were estimated by extrapolating data for PFOA and PFHpA because insufficient pilot data were available to support a curve fit determination.

For each system with occurrence data, *C* was calculated for each PFAS compound at a specified bed volume increment. Increments of 250 bed volumes up to a maximum of 40,000 were calculated for GAC. Increments of 5,000 bed volumes up to a maximum of 800,000 were calculated for IX. The number of bed volumes at which *C* exceeded the specified target replacement concentration was determined, and the number of bed volumes for the first contaminant to breach its target concentration was used to calculate media replacement frequency. The number of bed volumes treated before the first contaminant exceeded the target concentration was subjected to Monte Carlo variability as described in Section 5.2.

5.5 Life-Cycle Costs

The model determines 20-year life-cycle costs, which combines the capital costs and annual operating and maintenance costs. Life-cycle costs provide a means of comparing the costs of alternative technologies over the life cycle of the equipment. The life-cycle costs in the body of this report were calculated assuming a 20-year lifespan and a discount rate of 3 percent. A comparison of annualized NPDWR costs by system size at 3 and 7 percent is included in Table A-5 of Appendix A . While typical practice to determine life-cycle costs may incorporate other factors, such as the inflation and loan interest, the discount rate was used to match the approach that is standard practice for the EPA in promulgating national primary drinking water regulations.

6.0 National Cost Assessment Methodology

The conceptual framework for assessing the national costs is as follows:

- Assess capital, annual O&M, and life-cycle costs for each EPTDS in every water system for which potential regulatory limits for PFAS may require treatment.
- Average the costs by system size category and system type (ground or surface water).
- Multiply those average costs by the total anticipated number of systems of each type impacted in each system size category based on the percentage of systems in the database impacted by a proposed regulatory limit for PFAS.

The following subsections summarize the process and details associated with the national cost estimation methodology.

6.1 Estimating National Costs Using Model Outputs

Due to the difference in number of EPTDS for groundwater and surface water systems, the national cost calculations were completed separately for groundwater and surface water systems. The following methodology was utilized for each source water classification:

1. Using the treatment facility costs for systems from the occurrence database, the costs were binned by system size, and average EPTDS costs per system size bin were calculated.
2. Using the occurrence database, the number of impacted systems per size category was calculated, and the corresponding percent of the systems in the database was determined.
3. To estimate the number of impacted systems nationally, the percentage of impacted systems in the occurrence database was multiplied by the total number of systems in SDWIS for each size category.
4. The estimated number of impacted systems per size category multiplied by the average cost per EPTDS and the assumed number of entry points yields the total cost per size category. The sum of all costs per size category yields the estimated national cost of removing PFAS to a potential regulatory limit.

A summary output is included in Table 6-1, which displays the costs associated with achieving a maximum contaminant level (MCL) in drinking water of 4 ppt for PFOA and 4 ppt for PFOS for groundwater systems only.

Table 6-1 Example Summary Capital Cost Table for an MCL of 4 ppt for PFOA and PFOS (Groundwater Systems Only)

Size Category	PWSs In Database (Note 1)	Impacted PWSs in Database	% Impacted in Database	Active PWSs in SDWIS (Note 1)	Estimated Nationally Impacted PWSs	EPTDS per PWS	Average Capital Cost per EPTDS	National Capital Cost
1	1262	264	21%	10,654	2,229	1	\$900,000	\$2,006,100,000
2	1031	191	19%	13,037	2,415	1	\$1,900,000	\$4,588,500,000
3	285	37	13%	4,132	536	2	\$2,500,000	\$2,680,000,000
4	301	40	13%	5,503	731	2	\$3,200,000	\$4,678,400,000
5	373	65	17%	2,784	485	2	\$5,300,000	\$5,141,000,000
6	1221	106	9%	1,385	120	4	\$8,500,000	\$4,080,000,000
7	171	19	11%	162	18	10	\$12,500,000	\$2,250,000,000
8	78	11	14%	74	10	12	\$17,700,000	\$2,124,000,000
9	2	2	100%	2	2	39	\$40,600,000	\$3,166,800,000
All Systems	4724	735	16%	37,733	6,546	N/A	N/A	\$30,714,800,000

Notes:

1. The number of PWSs was updated per information included in EPA's March 2023 proposed regulation package and accounts for only community water systems (CWSs), which are PWSs that serve more than 25 people year round.

6.2 Accounting for State Level Regulatory Costs

The model includes consideration of state regulatory actions that may have driven PWSs to remove PFAS already. Consideration of state regulatory actions is necessary to characterize the compliance costs of a potential NPDWR for PFAS. All state regulations incorporated into modeled cost output are shown in Table 6-2

Table 6-2 State Maximum Contaminant Levels Modeled for State Regulatory Cost Estimate

States	Type	PFOA	PFOS	PFHxS	PFNA	PFHpA	PFBS
Connecticut	Individual		10				
Delaware	Individual	14	21				
Delaware	Combined	17	17				
Massachusetts	Combined	20	20	20	20	20	
Michigan	Individual	8	16	51	6		
New Hampshire	Individual	12	15	18	11		
New Jersey	Individual	14	13		13		
New York	Individual	10	10				
Ohio	Combined	70	70				
Ohio	Individual			140	21		140,000
Vermont	Individual	20	20	20	20	20	
Wisconsin	Combined	70	70				

To differentiate federal regulatory costs from costs incurred because of existing state regulations, the cost tool includes an input sheet for all existing state MCLs as either individual limits or group totals. The Visual Basic Script references both the state MCLs and the projected federal MCLs. In the absence of a federal regulation (or if the state MCL is more stringent than the federal MCL), the cost tool generates costs for treatment to comply with the state MCLs on the input sheet. An example of this is shown in Table 6-3, which displays treatment costs incurred as a result of state regulations shown in Table 6-2.

Table 6-3 Summary of Estimated Costs Associated with State PFAS MCLs

PWS Size Category	Population Range	% Impacted	Estimated Number of Impacted PWSs	Average CAPEX/PWS	Average O&M/PWS	Annualized PWS Cost	Annualized Total Cost	Present Value of Lifecycle Cost ¹
1	25 to 100	6%	718	\$800,000	\$20,000	\$74,000	\$53,132,000	\$790,500,000
2	101 to 500	5%	763	\$1,700,000	\$30,000	\$145,000	\$110,635,000	\$1,646,000,000
3	501 to 1,100	4%	232	\$4,400,000	\$68,000	\$364,000	\$84,448,000	\$1,256,400,000
4	1,001 to 3,300	1%	111	\$5,300,000	\$100,000	\$457,000	\$50,727,000	\$754,700,000
5	3,301 to 10,000	5%	240	\$8,600,000	\$200,000	\$779,000	\$186,960,000	\$2,781,500,000
6	10,001 to 50,000	3%	99	\$20,400,000	\$300,000	\$1,672,000	\$165,528,000	\$2,462,600,000
7	50,001 to 100,000	1%	6	\$46,200,000	\$800,000	\$3,906,000	\$23,436,000	\$348,700,000
8	100,001 to 1,000,000	3%	11	\$50,200,000	\$1,100,000	\$4,475,000	\$49,225,000	\$732,300,000
9	>1,000,000	4%	1	\$1,095,900,000	\$23,010,000	\$96,672,000	\$96,672,000	\$1,438,200,000
All Systems		4%	2181	N/A	N/A	N/A	\$820,763,000	\$12,210,900,000

Notes:

1. Present value of lifecycle costs estimated based on 3% discount rate and 20-year lifespan of equipment.
2. National costs for two potential MCLs are summarized in Section 7.1. The differentials between state costs in this table the total national costs represent the cost associated with any modeled NPDWR.

7.0 Summary of Results

A summary of the cost model results for various potential federal MCL alternatives on the national and household level is presented in this section.

7.1 National Cost Estimates

The national cost modeling tool was used to evaluate both the national financial burdens on communities from PFAS drinking water contamination (the National Burden) and the costs for water systems to comply with a potential NPDWR for PFAS (NPDWR Compliance Costs).

The National Burden is reflective of the total, cumulative impact to water systems and communities across the United States from PFAS contamination of drinking water. It is calculated by estimating the drinking water PFAS treatment costs associated with the number of systems with PFAS occurrence data above the target limit. The National Burden assumes the same target limit for water systems across all states and includes systems in states with existing drinking water regulations for PFAS. The NPDWR Compliance Costs are determined by estimating the national financial burden and excluding costs for systems already triggered into treatment by existing drinking water regulations at the state level. The difference between the National Burden and the NPDWR Compliance Costs is therefore calculated using the data presented in Table 6-3.

The National Burden and NPDWR Compliance Costs were estimated for two different scenarios. The first scenario is based on a target PFOA and PFOS level of 4 ppt each. The second scenario is based on target PFOA and PFOS levels of 10 ppt each.

An overview of the present value of the life-cycle cost for the National Burden and NPDWR compliance cost for each of these scenarios is displayed on Figure 7-1.

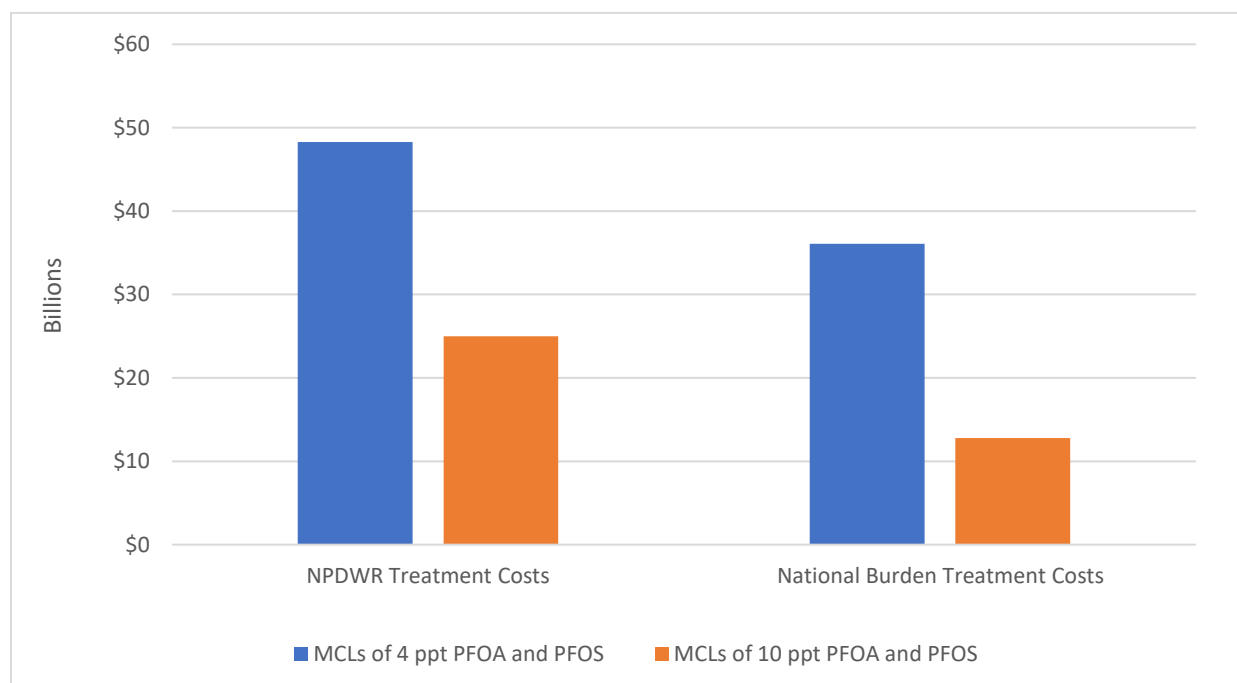


Figure 7-1 Summary of Present Value of Life-Cycle Costs for National Burdens and NPDWR Compliance Costs for Each Scenario based on a 3% discount rate

Annualized costs were also calculated using Formula 3. An overview of the National Burden and NPDWR Compliance Annualized Cost for each of these scenarios is presented on Figure 7-2.

$$\text{Annualized Costs} = \frac{(\text{Capital Costs})(\text{Discount Rate})}{1 - (1 + \text{Discount Rate})^{-n}} + \text{Annual Operating Costs} \quad (3)$$

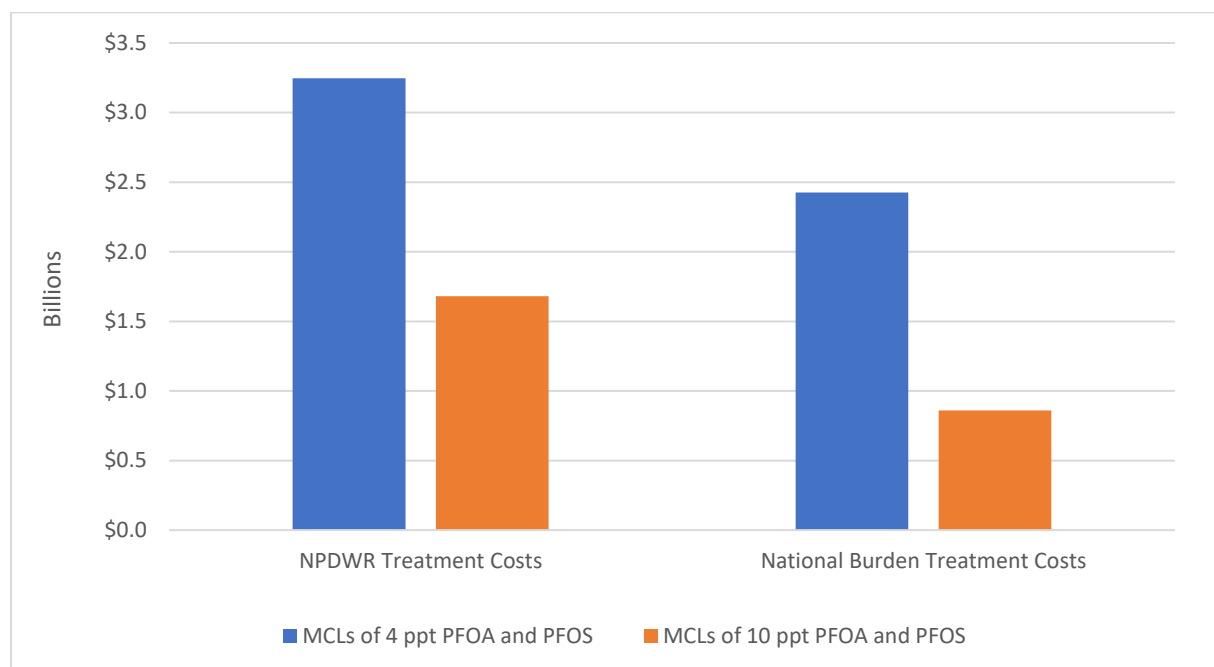


Figure 7-2 Summary of Annualized Costs for National Burdens and NPDWR Compliance for Each Scenario based on a 3% discount rate

A more detailed breakdown of these costs by system size is presented in Appendix A.

7.2 Household Financial Impacts

As part of this analysis, the annual financial impacts to individual households from costs associated with the installation and operation of drinking water treatment facilities for PFAS were determined. The financial impacts to individual households will vary by specific PFAS levels, system size, and other factors. Additionally, the impacts to individual households arising from a potential NPDWR will differ depending on whether there is an existing state regulation for PFAS in drinking water. Table 7-1 shows the individual household impacts as a function of system size for each of the three scenarios discussed in Section 7.1. These household level cost impacts are based on the annualized costs for each system size and an average of 2.6 persons per household and incorporate estimated average service populations for each size category based on SDWIS data. The range of household level costs in the table is reflective of communities where new treatment facilities will need to be installed and operated.

Table 7-1 Annual Costs to Household for Removing PFAS from Drinking Water

PWS Size Category	Population Range	Average Service Population	Approximate Range of Costs per Household
1	25 to 100	59	\$3570 - \$3570
2	101-500	245	\$1675 - \$1750
3	501-1,100	736	\$1360 - \$1390
4	1,001-3,300	1,939	\$575 - \$640
5	3,301-10,000	5,696	\$305 - \$325
6	10,001-50,000	20,613	\$200 - \$225
7	50,001-100,000	67,417	\$155 - \$175
8	100,001-1,000,000	204, 194	\$65 - \$70
9	>1,000,000	1,700,000	\$115 - \$120

Appendix A. Modeled Cost MCL and Discount Rate Comparison Tables

Table A-1 National Cost Burden by System Size for 4 ppt PFOA, PFOS (Groundwater Systems)

Size Category	Population Range	Estimated Number of PWSs Impacted	Estimated Number of EPTDS Impacted	Average CAPEX per EPTDS	Average OPEX per EPTDS	Total CAPEX	Total OPEX
1	25 to 100	2,229	2,229	\$900,000	\$20,000	\$2,006,100,000	\$44,580,000
2	101 to 500	2,415	2,415	\$1,900,000	\$30,000	\$4,588,500,000	\$72,450,000
3	501 to 1,100	536	1,072	\$2,500,000	\$39,000	\$2,680,000,000	\$41,808,000
4	1,001 to 3,300	731	1,462	\$3,200,000	\$41,000	\$4,678,400,000	\$59,942,000
5	3,301 to 10,000	485	970	\$5,300,000	\$82,000	\$5,141,000,000	\$79,540,000
6	10,001 to 50,000	120	480	\$8,500,000	\$156,000	\$4,080,000,000	\$74,880,000
7	50,001 to 100,000	18	180	\$12,500,000	\$215,000	\$2,250,000,000	\$38,700,000
8	100,001 to 1,000,000	10	120	\$17,700,000	\$388,000	\$2,124,000,000	\$46,560,000
9	>1,000,000	2	78	\$40,600,000	\$845,000	\$3,166,800,000	\$65,910,000
All Systems		6,546	9,006	N/A	N/A	\$30,714,800,000	\$524,370,000

Table A-2 National Burden Costs per System Size for 4 ppt PFOA, PFOS (Surface Water Systems)

Size Category	Population Range	Estimated Number of PWSs Impacted	Estimated Number of EDTDS Impacted	Average CAPEX per EPTDS	Average OPEX per EPTDS	Total CAPEX	Total OPEX
1	25 to 100	41	41	\$900,000	\$20,000	\$36,900,000	\$820,000
2	101 to 500	125	125	\$1,900,000	\$30,000	\$237,500,000	\$3,750,000
3	501 to 1,100	63	63	\$2,600,000	\$43,000	\$163,800,000	\$2,709,000
4	1,001 to 3,300	137	137	\$3,400,000	\$51,000	\$465,800,000	\$6,987,000
5	3,301 to 10,000	294	294	\$5,500,000	\$82,000	\$1,617,000,000	\$24,108,000
6	10,001 to 50,000	134	134	\$9,200,000	\$171,000	\$1,232,800,000	\$22,914,000
7	50,001 to 100,000	46	92	\$13,000,000	\$243,000	\$1,196,000,000	\$22,356,000
8	100,001 to 1,000,000	61	122	\$18,600,000	\$469,000	\$2,269,200,000	\$57,218,000
9	>1,000,000	2	8	\$32,000,000	\$1,270,000	\$256,000,000	\$10,160,000
All Systems		903	1,016	N/A	N/A	\$7,475,000,000	\$151,022,000

Table A-3 National Burden Costs by System Size for 10 ppt PFOA, PFOS (Groundwater Systems)

Size Category	Population Range	Estimated Number of PWSs Impacted	Estimated Number of EPTDS Impacted	Average CAPEX per EPTDS	Average OPEX per EPTDS	Total CAPEX	Total OPEX
1	25 to 100	861	861	\$900,000	\$20,000	\$774,900,000	\$17,220,000
2	101 to 500	835	835	\$2,000,000	\$30,000	\$1,670,000,000	\$25,050,000
3	501 to 1,100	290	580	\$2,700,000	\$33,000	\$1,566,000,000	\$9,570,000
4	1,001 to 3,300	201	402	\$3,100,000	\$37,000	\$1,246,200,000	\$7,437,000
5	3,301 to 10,000	246	492	\$5,500,000	\$71,000	\$2,706,000,000	\$17,466,000
6	10,001 to 50,000	84	336	\$8,900,000	\$119,000	\$2,990,400,000	\$9,996,000
7	50,001 to 100,000	12	120	\$12,700,000	\$170,000	\$1,524,000,000	\$2,040,000
8	100,001 to 1,000,000	10	120	\$18,300,000	\$277,000	\$2,196,000,000	\$2,770,000
9	>1,000,000	2	78	\$42,100,000	\$790,000	\$3,283,800,000	\$1,580,000
All Systems		2,541	3,824	N/A	N/A	\$17,957,300,000	\$93,129,000

Table A-4 National Burden Costs by System Size for 10 ppt PFOA, PFOS (Surface Water Systems)

Size Category	Population Range	Estimated Number of PWSs Impacted	Estimated Number of EPTDS Impacted	Average CAPEX per EPTDS	Average OPEX per EPTDS	Total CAPEX	Total OPEX
1	25 to 100	21	21	\$900,000	\$20,000	\$18,900,000	\$420,000
2	101 to 500	83	83	\$2,000,000	\$30,000	\$166,000,000	\$2,490,000
3	501 to 1,100	21	21	\$2,800,000	\$40,000	\$58,800,000	\$840,000
4	1,001 to 3,300	39	39	\$3,700,000	\$55,000	\$144,300,000	\$2,145,000
5	3,301 to 10,000	155	155	\$5,600,000	\$70,000	\$868,000,000	\$10,850,000
6	10,001 to 50,000	93	93	\$9,400,000	\$138,000	\$874,200,000	\$12,834,000
7	50,001 to 100,000	29	58	\$13,400,000	\$199,000	\$777,200,000	\$5,771,000
8	100,001 to 1,000,000	45	90	\$19,800,000	\$382,000	\$1,782,000,000	\$17,190,000
9	>1,000,000	1	4	\$37,200,000	\$1,100,000	\$148,800,000	\$1,100,000
All Systems		487	564	N/A	N/A	\$4,838,200,000	\$53,640,000

Table A-5 Annualized NPDWR Costs by System Size per Discount Rate

PWS Size Category	Population Range	4 ppt PFOA/PFOS		10 ppt PFOA/PFOS	
		3%	7%	3%	7%
1	25 to 100	\$130,738,000	\$169,422,000	\$18,310,000	\$23,682,000
2	101-500	\$290,685,000	\$387,667,000	\$40,835,000	\$55,309,000
3	501-1,100	\$151,558,000	\$200,989,000	\$35,287,000	\$51,609,000
4	1,001-3,300	\$365,882,000	\$489,646,000	\$55,914,000	\$77,610,000
5	3,301-10,000	\$391,743,000	\$518,888,000	\$101,870,000	\$142,992,000
6	10,001-50,000	\$291,366,000	\$381,145,000	\$119,121,000	\$169,335,000
7	50,001-100,000	\$269,744,000	\$355,852,000	\$139,532,000	\$194,533,000
8	100,001-1,000,000	\$350,471,000	\$454,900,000	\$238,755,000	\$331,892,000
9	>1,000,000	\$209,464,000	\$272,704,000	\$136,734,000	\$200,238,000
All Systems		\$2,451,651,000	\$3,231,213,000	\$886,358,000	\$1,247,200,000

Appendix C

Supplemental Figures Comparing Case Study Data with EPA and BV Cost Models

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- Figure A 2: Comparison of GAC Operating Costs for Small Systems (<2.5 MGD)
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- Figure A 11: Comparison of RO Capital Costs for Medium Systems (<10 MGD)
- Figure A 12: Comparison of RO Operating Costs for Medium Systems (<10 MGD)

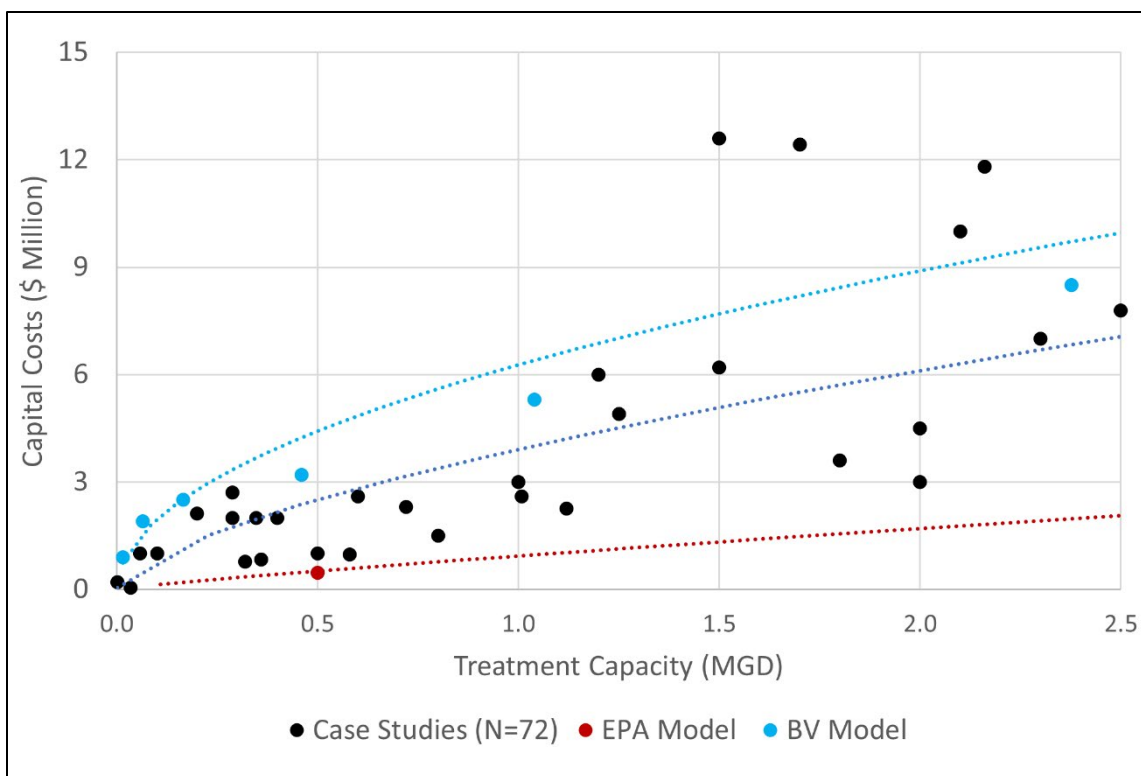


Figure A 13: Comparison of GAC Capital Costs for Smaller Systems (<2.5 MGD)

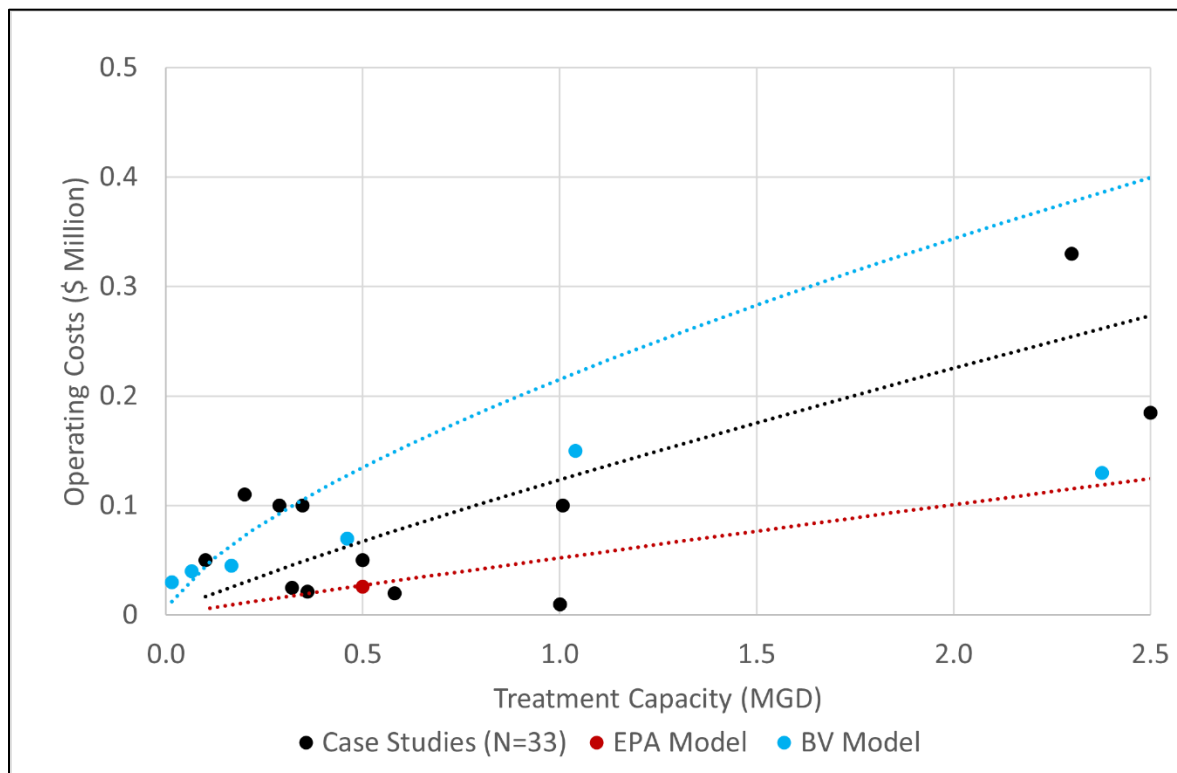


Figure A 14: Comparison of GAC Operating Costs for Small Systems (<2.5 MGD)

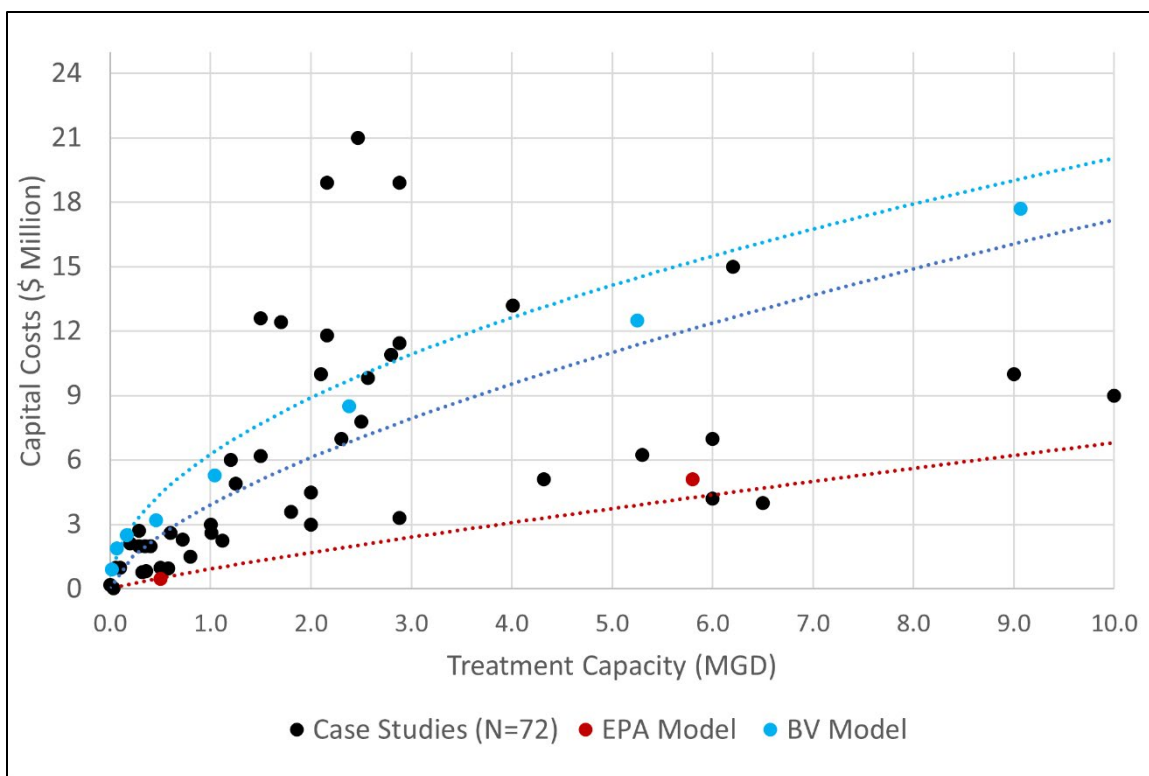


Figure A 15: Comparison of GAC Capital Costs for Medium Systems (<25 MGD)

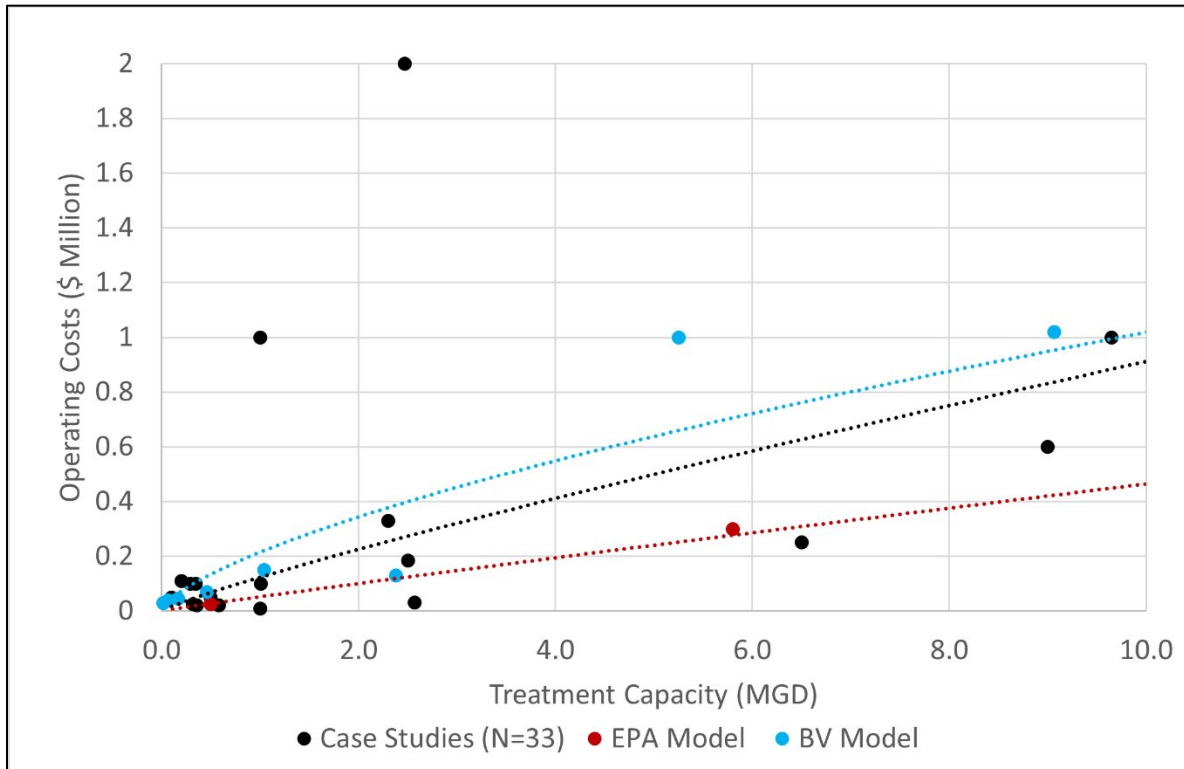


Figure A 16: Comparison of GAC Operating Costs for Medium Systems (<10 MGD)

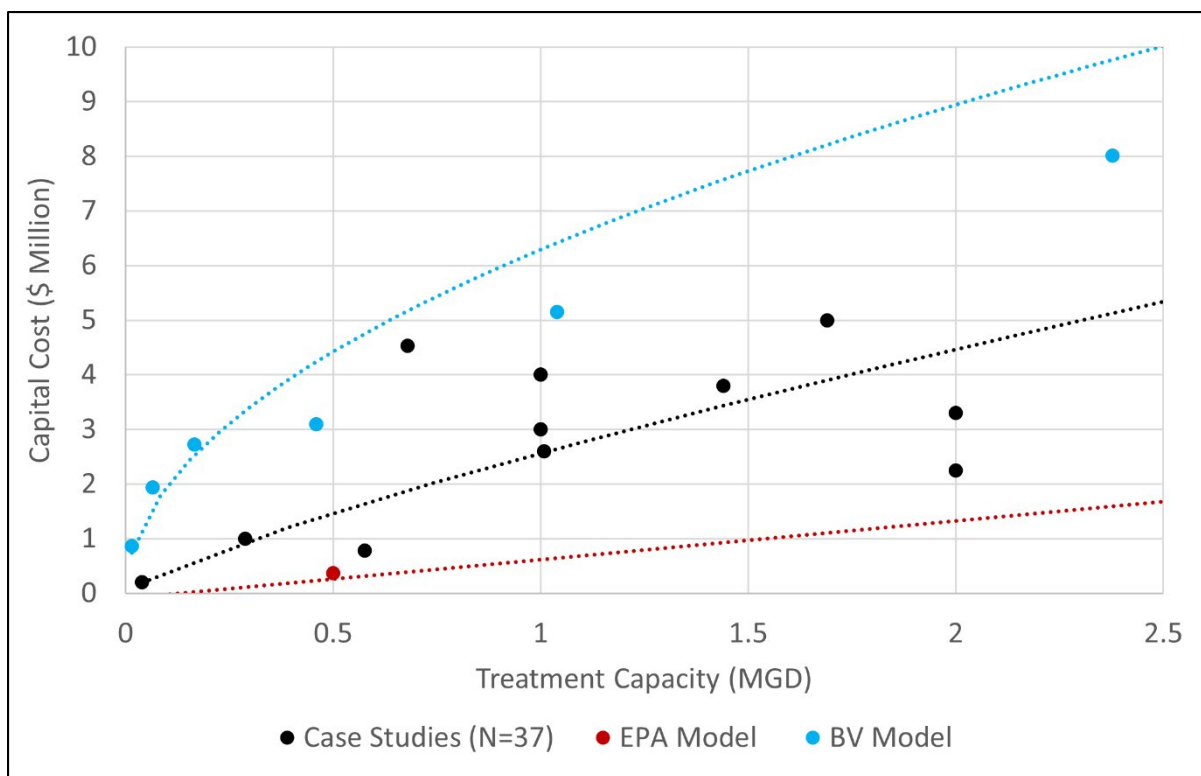


Figure A 17: Comparison of IX Capital Costs for Small Systems (<2.5 MGD)

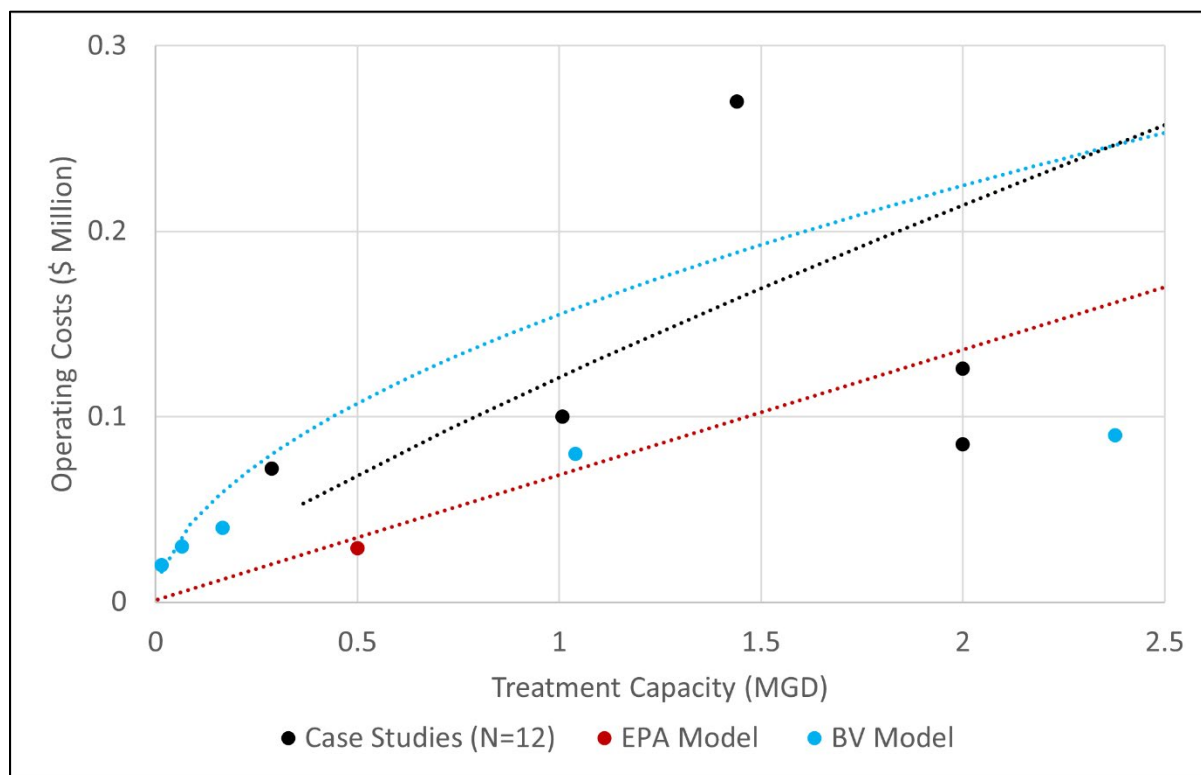


Figure A 18: Comparison of IX Operating Costs for Small Systems (<2.5 MGD)

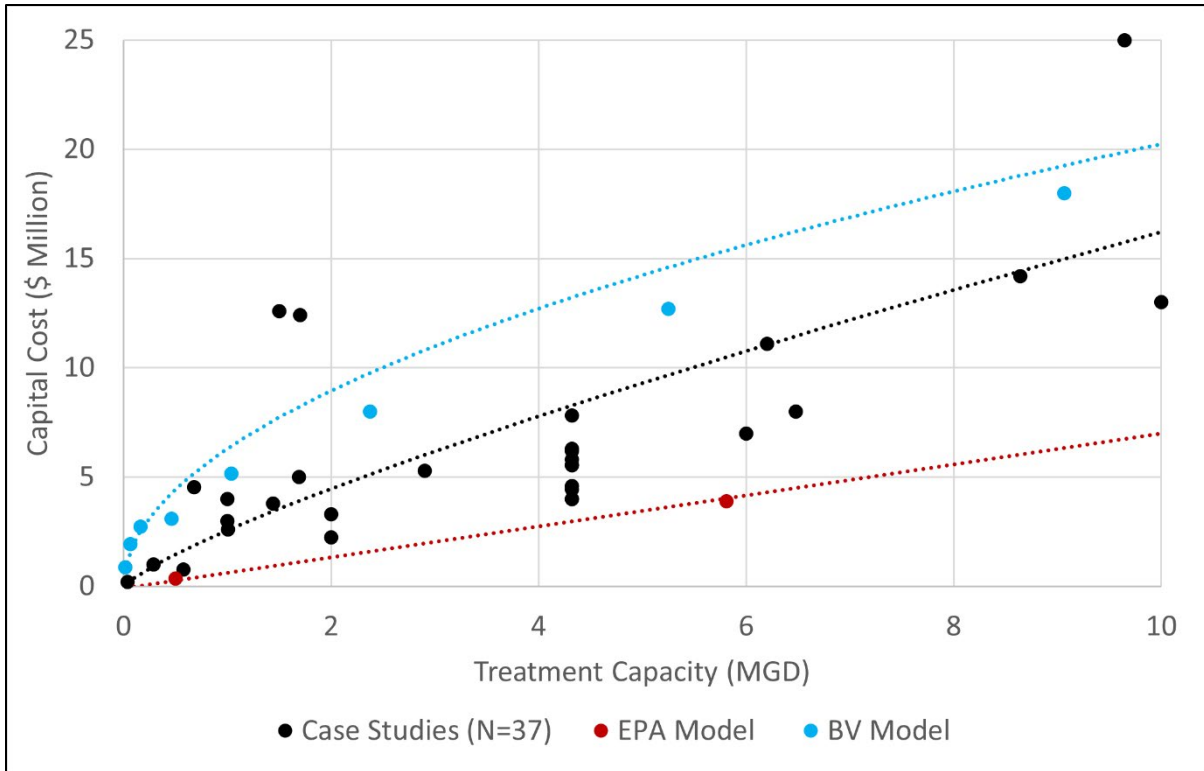


Figure A 19: Comparison of IX Capital Costs for Medium Systems (<10 MGD)

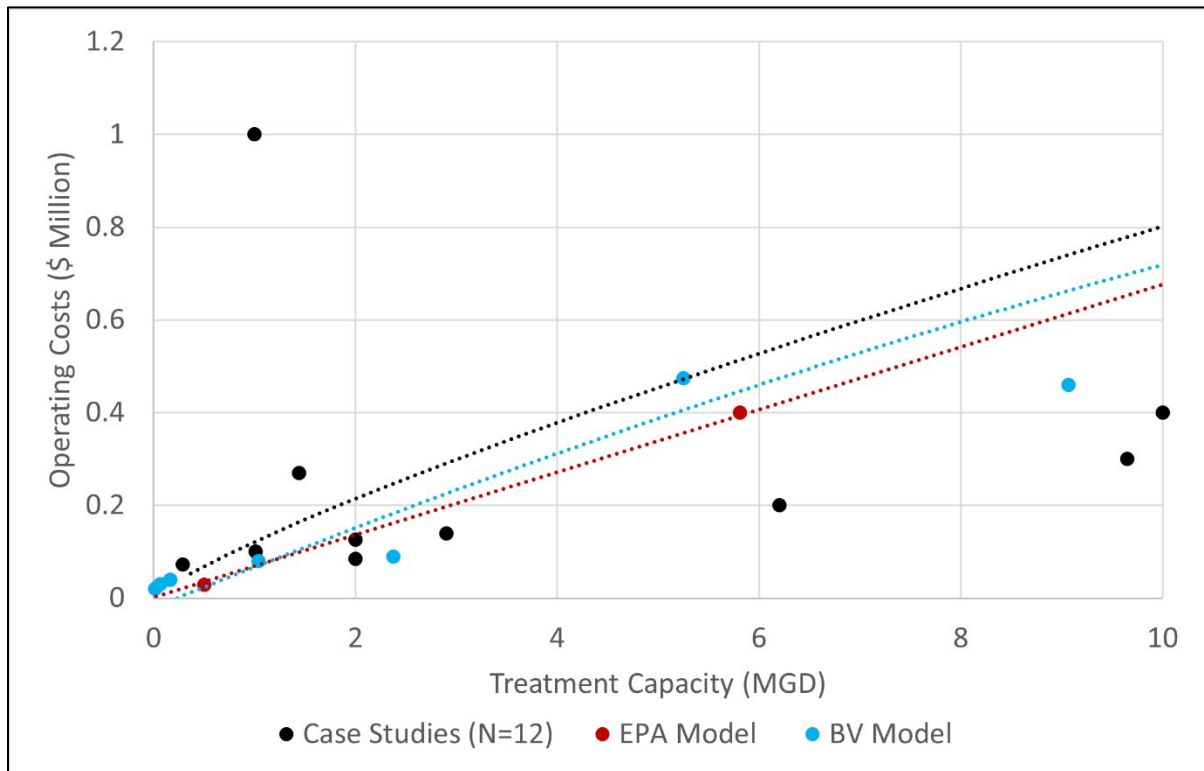


Figure A 20: Comparison of IX Operating Costs for Medium Systems (<10 MGD)

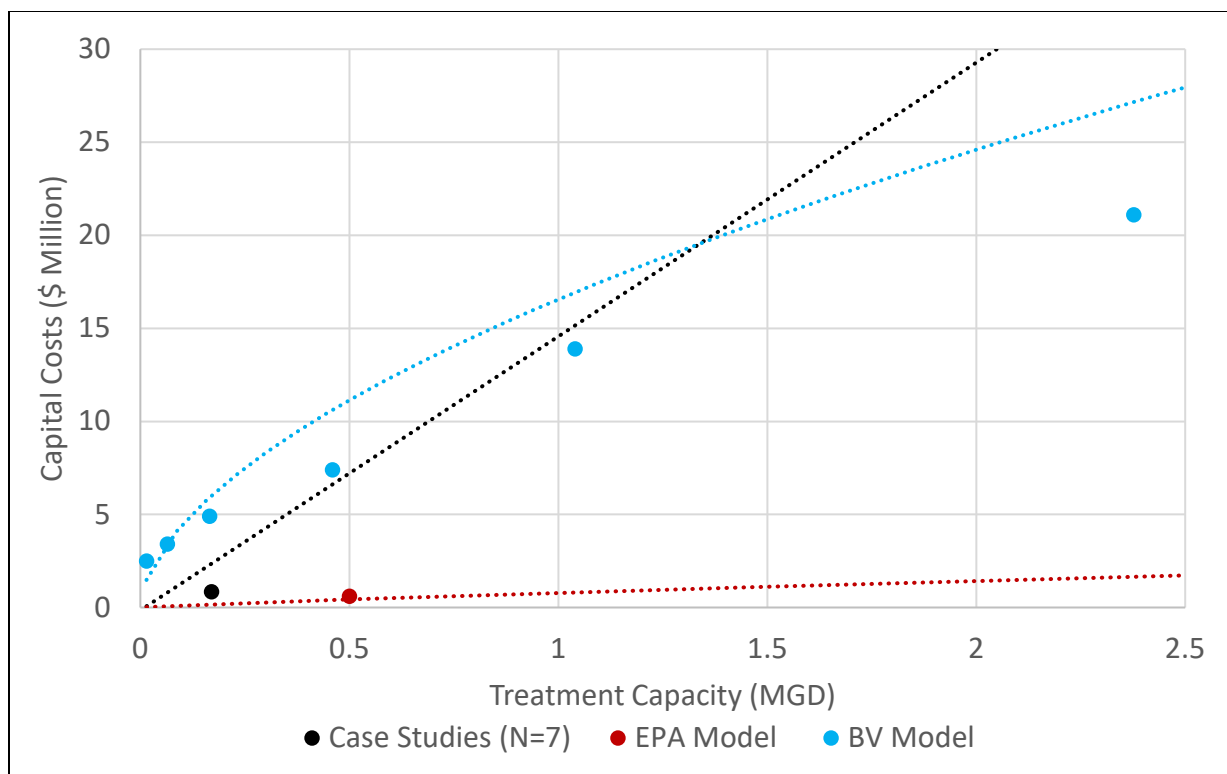


Figure A 21: Comparison of RO Capital Costs for Small Systems (<2.5 MGD)

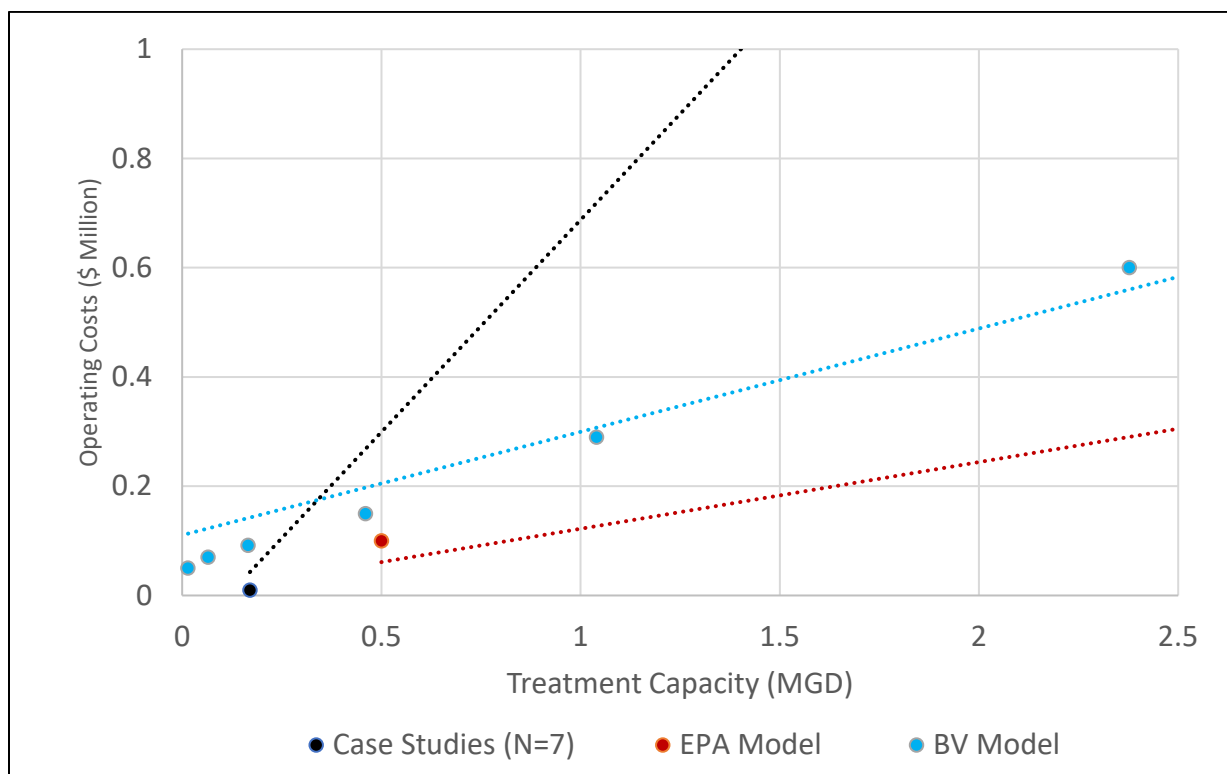


Figure A 22: Comparison of RO Operating Costs for Small Systems (< 2.5 MGD)

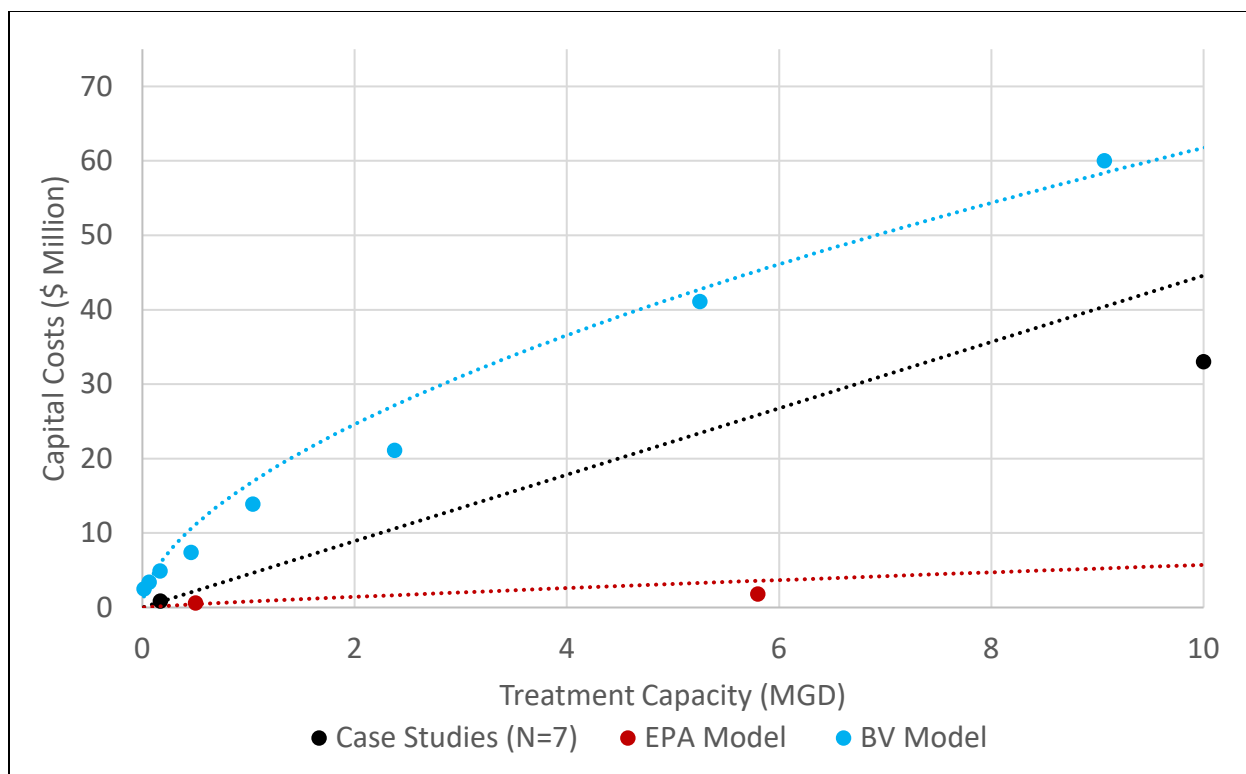


Figure A 23: Comparison of RO Capital Costs for Medium Systems (<10 MGD)

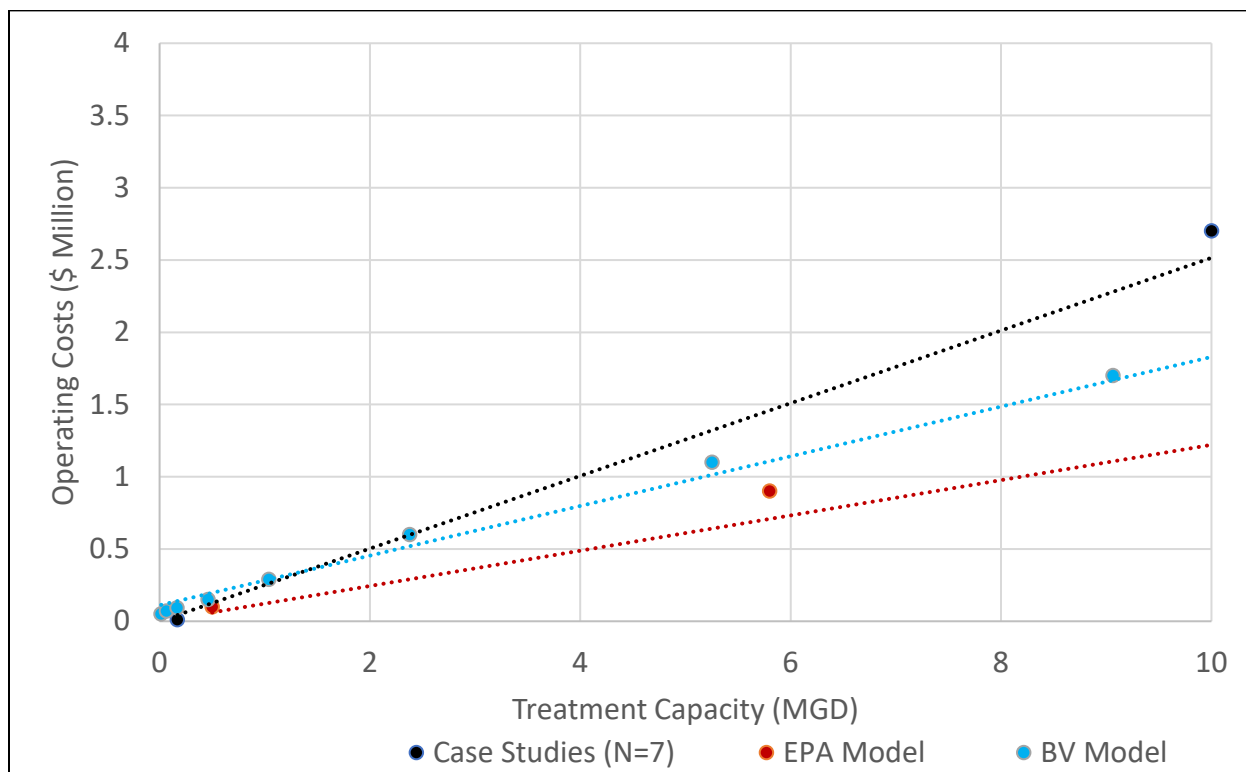


Figure A 24: Comparison of RO Operating Costs for Medium Systems (<10 MGD)

Appendix D

Summary of PFAS Treatment Cost Case Studies

Water System	Treatment Technology	Facility Capacity (MGD)	Facility Capital Cost (\$)	Facility Operating Cost (\$)	Source
Pease International Tradeport Drinking Water System	GAC	0.4	2	Not Available.	Kleinfelder, 2023; AWWA, 2022b ¹
Merrimack Village District	GAC	2.8	10.9	Not Available.	Kleinfelder, 2023; AWWA, 2023b ¹
Merrimack Village District	GAC	1.8	3.6	Not Available.	Kleinfelder, 2023; AWWA, 2023b ¹
Devens Public Water Supply	GAC	1.25	4.9	Not Available.	Kleinfelder, 2023; AWWA, 2023b ¹
Devens Public Water Supply	GAC	1.12	2.25	Not Available.	Kleinfelder, 2023; AWWA, 2023b ¹
Town of Canton	GAC	2.1	10	Not Available.	Kleinfelder, 2023; AWWA, 2023b ¹
Mashpee Water District	GAC	0.6	2.6	Not Available.	Kleinfelder, 2023; AWWA, 2023b ¹
Mashpee Water District	GAC	0.72	2.3	Not Available.	Kleinfelder, 2023; AWWA, 2023b ¹
Wellesley Water Division	GAC	1.5	6.2	Not Available.	Kleinfelder, 2023; AWWA, 2023b ¹
Anonymous	GAC	0.8	1.5	Not Available.	Kleinfelder, 2023; AWWA, 2023b ¹
Anonymous	GAC	1.2	6	Not Available.	Kleinfelder, 2023; AWWA, 2023b ¹
Anonymous	GAC	2.16	11.8	Not Available.	Kleinfelder, 2023; AWWA, 2023b ¹
Anonymous	GAC	4.03	38	Not Available.	Kleinfelder, 2023; AWWA, 2023b ¹
Anonymous	GAC	0.001	0.2	Not Available.	Kleinfelder, 2023; AWWA, 2023b ¹
Anonymous	GAC	0.03456	0.047	Not Available.	Kleinfelder, 2023; AWWA, 2023b ¹
City of Ames	GAC	15	36.84	1.483	Strand, 2023.
South Central Connecticut Regional Water Authority	GAC	2.5	7.784	0.185	Barger, 2023
South Central Connecticut Regional Water Authority	GAC	2.568	9.816	0.032	Barger, 2023
Pownal Water District	GAC	0.2	2.112	0.110366667	Bennington, 2022
Horsham Water & Sewer District	GAC	0.1	1	0.05	Horsham, 2023b
Horsham Water & Sewer District	GAC	0.5	1	0.05	Horsham, 2023b
Orange County Water District	GAC	62.4	105	Not Available.	Dadakis, 2023
Essential Utilities	GAC	0.32	0.78	0.024811	Essential, 2023
Essential Utilities	GAC	0.58	0.97	0.020054	Essential, 2023
Essential Utilities	GAC	0.36	0.84	0.021642	Essential, 2023
Cape Fear Public Utility Authority	GAC	44	46		AWWA, 2021c
Water System in DE	GAC	30	50.6	2.3	Walczyk, 2023
Water System in ID	GAC	25	105.6	4.8	Walczyk, 2023

Water System	Treatment Technology	Facility Capacity (MGD)	Facility Capital Cost (\$)	Facility Operating Cost (\$)	Source
Water System in NY	GAC	50	250	11.2	Walczyk, 2023
Hazen & Sawyer Project: Alabama (10 MGD)	GAC	10	9	0.65	Rosenfeldt, 2021
Hazen & Sawyer Project: Alabama (6 MGD)	GAC	6	4.2		Rosenfeldt, 2021
Hazen & Sawyer Project: New Mexico (2 MGD)	GAC	2	4.5	0.088	Rosenfeldt, 2021
Hazen & Sawyer Project: New Mexico (200 gpm)	GAC	0.288	2.7	0.076	Rosenfeldt, 2021
Hazen & Sawyer Project: New York (40 gpm)	GAC	0.0576	1	0.025	Rosenfeldt, 2021
Hazen & Sawyer Project: California (6.2 MGD)	GAC	6.2	15	0.1	Rosenfeldt, 2021
Hazen & Sawyer Project: MAssachusetts (2 MGD)	GAC	2	3	0.045	Rosenfeldt, 2021
Town of Webster, MA	GAC	2.16	18.9	Not Available.	Tighe & Bond, 2022
Town of Webster, MA	GAC	2.88	18.9	Not Available.	Tighe & Bond, 2022
Town of Webster, MA	GAC	2.88	11.45	Not Available.	Tighe & Bond, 2022
Town of Webster, MA	GAC	4.3	35.8	Not Available.	Tighe & Bond, 2022
Lakewood, Colorado	GAC	2.88	3.3	Not Available.	LWD, 2023
Lakewood, Colorado	GAC	4.32	5.1	Not Available.	LWD, 2023
Anonymous	GAC	9.648	26	1	AWWA, 2023b ¹
Anonymous	GAC	44	26.7	2	AWWA, 2023b ¹
Fayetteville Public Works Commission	GAC	39.5	60	7	AWWA, 2023b ¹
Cobb County-Marietta Water Authority	GAC	36	20	1	AWWA, 2023b ¹
Anonymous	GAC	120	240	30	AWWA, 2023b ¹
Anonymous	GAC	9	10	0.6	AWWA, 2023b ¹
Townsend Water	GAC	2.47	21	2	AWWA, 2023b ¹
Town of West Springfield DPW Water Division	GAC	6.5	4	0.25	AWWA, 2023b ¹
Anonymous	GAC	60	0.5	3	AWWA, 2023b ¹
City of Thornton	GAC	50	100	4	AWWA, 2023b ¹
City of Hamilton IL	GAC	1		0.009777	AWWA, 2023b ¹
Anonymous	GAC	5.3	6.251	Not Available.	AWWA, 2023b ¹
Town of Sharon, MA	GAC	2.3	7	0.33	AWWA, 2023b ¹
Anonymous	GAC	80	80	12	AWWA, 2023b ¹

Water System	Treatment Technology	Facility Capacity (MGD)	Facility Capital Cost (\$)	Facility Operating Cost (\$)	Source
Anonymous	GAC	20	13.8	0.5	AWWA, 2023b ¹
Fayetteville Public Works Commission	GAC	18	20	3	AWWA, 2023b ¹
Norwell Water Department	GAC	0.347	2	0.1	AWWA, 2023b ¹
Anonymous	GAC	60	20	3	AWWA, 2023b ¹
City of Thornton	GAC	20	65	2.6	AWWA, 2023b ¹
Anonymous	GAC	4.0104	13.19	Not Available.	AWWA, 2023b ¹
Anonymous	GAC	70	70	8	AWWA, 2023b ¹
Orange County Utilities	GAC	21.28	35.88	2.166	AWWA, 2023b ¹
Norwell Water Department	GAC	0.288	2	0.1	AWWA, 2023b ¹
Orange County Utilities	GAC	34.56	58.24	1.987	AWWA, 2023b ¹
Merrimack Village District	GAC/IX	1.7	12.42	Not Available.	Kleinfelder, 2023. See note.
Town of Canton	GAC/IX	2.88	25.2	Not Available.	Kleinfelder, 2023. See note.
Frisco, CO Public Works	GAC/IX	1	3	1	AWWA, 2023b ¹
Anonymous	GAC/IX	1.5	12.6		AWWA, 2023b ¹
Norwell Water Department	GAC/IX	1.008	2.6	0.1	AWWA, 2023b ¹
Anonymous	GAC/IX	6	7	Not Available.	AWWA, 2023b ¹
Anonymous	IX	0.576	0.78	Not Available.	Kleinfelder, 2023. See note.
Anonymous	IX	1	4	Not Available.	Kleinfelder, 2023. See note.
City of Ames	IX	15	32.94	2.713	Strand, 2023.
Orange County Water District	IX	4.32	5.805	Not Available.	Dadakis, 2023
Orange County Water District	IX	23.76	24.457	Not Available.	Dadakis, 2023
Orange County Water District	IX	4.32	5.55	Not Available.	Dadakis, 2023
Orange County Water District	IX	4.32	6.194	Not Available.	Dadakis, 2023
Orange County Water District	IX	8.64	25.762	Not Available.	Dadakis, 2023
Orange County Water District	IX	10.8	10.02	Not Available.	Dadakis, 2023
Orange County Water District	IX	25.92	27.7	Not Available.	Dadakis, 2023
Orange County Water District	IX	4.32	6.3	Not Available.	Dadakis, 2023
Orange County Water District	IX	4.32	7.807	Not Available.	Dadakis, 2023

Water System	Treatment Technology	Facility Capacity (MGD)	Facility Capital Cost (\$)	Facility Operating Cost (\$)	Source
Orange County Water District	IX	8.64	14.2	Not Available.	Dadakis, 2023
Orange County Water District	IX	4.32	4	Not Available.	Dadakis, 2023
Orange County Water District	IX	4.32	4	Not Available.	Dadakis, 2023
Orange County Water District	IX	6.48	8	Not Available.	Dadakis, 2023
Orange County Water District	IX	4.32	4.6	Not Available.	Dadakis, 2023
Orange County Water District	IX	4.32	5.55	Not Available.	Dadakis, 2023
Orange County Water District	IX	4.32	4.431	Not Available.	Dadakis, 2023
Essential Utilities	IX	0.68	4.53	Not Available.	Essential, 2023
Essential Utilities	IX	0.04	0.2	Not Available.	Essential, 2023
Water System in NJ	IX	188	550	25	Walczyk, 2023
Hazen & Sawyer Project: Alabama (10 MGD)	IX	10	13	0.4	Rosenfeldt, 2021
Hazen & Sawyer Project: New Mexico (2 MGD)	IX	2	3.3	0.126	Rosenfeldt, 2021
Hazen & Sawyer Project: New Mexico (200 gpm)	IX	0.288	1	0.072	Rosenfeldt, 2021
Hazen & Sawyer Project: California (6.2 MGD)	IX	6.2	11.1	0.2	Rosenfeldt, 2021
Hazen & Sawyer Project: Massachusetts (2 MGD)	IX	2	2.25	0.085	Rosenfeldt, 2021
Anonymous	IX	2.9	5.3	0.14	AWWA, 2023b ¹
Anonymous	IX	9.648	25	0.3	AWWA, 2023b ¹
Town of Sharon, MA	IX	1.44	3.8	0.27	AWWA, 2023b ¹
Town of Sharon, MA	IX	1.69	5	Not Available.	AWWA, 2023b ¹
City of Ames	RO	15	63.02	4.065	Strand, 2023.
Brunswick County Public Utilities	RO	36	99	2.9	CDM Smith, 2018.
Hazen & Sawyer Project: Alabama (10 MGD)	RO	10	33	2.7	Rosenfeldt, 2021
Anonymous	RO	0.17	0.85	0.01	AWWA, 2023b ¹
Anonymous	RO	150	706	42	AWWA, 2023b ¹
Anonymous	RO	60	372	20	AWWA, 2023b ¹
Anonymous	RO	250	1.077	60	AWWA, 2023b ¹

¹ For detailed survey data, contact Chris Moody, Regulatory Technical Manager at American Water Works Association.

KRISTEN ATHA
Director

August 1, 2023

FILED VIA E-MAIL NDWAC@epa.gov
Elizabeth Corr
NDWAC Designated Federal Officer
Office of Ground Water and Drinking Water
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue NW
Washington, DC 20460

**RE: NDWAC Meeting on Proposed Rule, PFAS National Primary Drinking Water Regulation
FRL-11126-01-OW; 88 FR 44128 (July 11, 2023)**

Dear Ms. Corr:

The City of Columbus, Department of Public Utilities (“CDPU”) appreciates the opportunity to provide testimony for consideration by the National Drinking Water Advisory Council (Council) on EPA’s proposed National Primary Drinking Water Regulation for PFOA, PFOS, and four other PFAS. CDPU operates drinking water and wastewater treatment plants serving over 220 square miles in the greater Columbus area. Our interest in PFAS stretches from the source water that feeds our drinking water plants to the effluent released from our wastewater plants, and everywhere in between. We are committed to improving water quality and protecting the environment, and we share EPA’s concerns about the risks that PFAS chemicals pose to the public.

In May, CDPU submitted comments to EPA regarding the proposed National Primary Drinking Water Regulation for PFAS (attached). As a vested stakeholder with a duty to the public, CDPU supports EPA’s efforts to set its initial federal drinking water standard for PFAS at a protective, feasible maximum contaminant level (MCL). In the letter, CDPU urged EPA to consider the active loading of PFAS into water occurring through the manufacture and use of PFAS and PFAS containing products. CDPU also urged EPA to consider the accurate costs and feasibility, consumer confidence, and regulatory alternatives before it mandates its first MCL at the practical quantification level (PQL). Several states have established feasible state standards for PFOA and PFOS at twice the currently proposed federal standard. Those plans would result in the best use of limited resources because they address the greatest health risks by prioritizing systems with the highest concentrations of PFAS. The May comment letter is attached as it details CDPU’s rationale and support.

CDPU respectfully requests that the Council review the May comment letter and consider the following points as it provides EPA with advice and recommendations on this important issue:



Customer Service | Telephone (614) 645-8276 | Fax (614) 645-0222 | TDD (614) 645-7188

Power Division | 3500 Indianola Avenue | Columbus, OH 43214 | Telephone (614) 645-8371 | Fax (614) 645-7830
Sewerage and Drainage | 1250 Fairwood Avenue | Columbus OH 43206 | Telephone (614) 645-7175 | Fax (614) 645-3801
Water Administration | 910 Dublin Road Columbus OH | 43215 | Telephone (614) 645-7020 | Fax (614) 645-3993
Director’s Office | 910 Dublin Road | Columbus OH 43215 | Telephone (614) 645-6141 | Fax (614) 645-8019
columbus.gov

1. EPA should consider a direct campaign to eliminate additional PFAS load in our water and soil by instituting a ban on the manufacture and use of PFAS, except for in extraordinary circumstances with stringent controls.
 - CDPU does not use, produce, or profit from PFAS compounds.
 - CDPU provides critical services to our community and, due to that function, CDPU is a passive receiver of source water that might contain PFAS from industrial, commercial, and domestic sources.
 - On the whole, the resource intensive efforts that will be required of water utilities might reduce the current amount of PFOA and PFOS in a cup of water from low parts per trillion to an amount in parts per quadrillion, but it will not reduce the amount of PFAS that the public is exposed to everyday through other common household and environmental sources.
2. MCLs set at the PQL are not feasible.
 - Under section 1412(b)(4)(B) of SDWA, EPA must take costs into consideration when setting an enforceable MCL as close to the maximum contaminant level goal (MCLG) as feasible. The standard is feasibility, which contemplates expense, rather than mere capability.
 - EPA's estimate of nationwide proposed costs to treat to 4 ppt for PFOA and PFOS falls between \$776M and \$1.2B.
 - Water utilities are reporting treatment cost estimates that appear to be much higher than EPA's estimate.
 - Additionally, this range is much too low after factoring in inflation, supply and demand, and increased health risks from the greenhouse gas emissions that will result from these treatment processes.
3. CDPU's estimate for the cost of implementing granulated activated carbon treatment at its water plants is close to half of EPA's total nationwide estimate.
 - Estimated capital costs for just CDPU's four plants falls between \$338 million to \$363 million, but could realistically approach \$544 million.
 - Additionally, the current estimate for operation and maintenance costs is between \$29.4 million to \$43.9 million per year.
 - To put that in perspective, CPDU—one public water system out of 3,500—may need to spend **over half a billion dollars by 2030** to treat the very low levels of PFAS in our drinking water. The result would be a high cost for small benefit.
4. EPA's proposed timeline to meet an MCL of 4 ppt is unreasonable.
 - Meeting an MCL of 4 ppt will require the commitment of an extremely large amount of public money to accomplish these significant construction projects.
 - On top of the typical lengthy project timeline, if 3,500 water utilities enter the market for the same few treatment techniques, there will be massive supply/demand issues.
 - Three years is not enough time to get us to the finish line, especially when compliance and the corresponding consumer confidence are at risk. Utilities are struggling with maintaining affordable rates as they face an onslaught of mandated capital and regulatory projects.
 - *One option* to solve the timing issues would be to extend the compliance timeline.

- *Another option* is a phased approach to the MCL (starting with a higher initial MCL) that would address these problems by reducing the competition for limited resources for installing treatment and allowing the science to catch up to policy. It would tackle the bulk of, and most significant, human health risks by targeting systems with the highest PFAS concentrations first (see #6 below).
5. EPA should consider reliability and consumer confidence before setting its initial MCL at the PQL.
- Consumer confidence is key in any water regulation. Clean, safe drinking water is ultimately only as drinkable as the public understands it to be.
 - An overly-aggressive MCL for PFAS will shake consumer confidence.
 - Here, where there is scientific uncertainty and the proposed MCL is pushing the available treatment and sampling techniques to their physical extremes, EPA should set a MCL that meets the requirements of the SDWA that also can be supported as rational to the public. As more data becomes available from UCMR 5 and other studies, there will be opportunities to issue a more stringent MCL.
 - Issuing the initial MCL at the PQL has high consequences because once EPA sets the MCL, water utilities will have to start expending the billions of dollars to comply to meet it.
 - When the MCL is at the PQL and measured in parts per trillion, the difference between installing hundreds of millions of dollars of treatment, or not having to treat, is one drop in 20 Olympic size swimming pools. Quantitative reliability is a necessity. The margins for error are tight right now when the science is still emerging and the risks of overcommitting public money is high.
6. EPA could reconsider the option of addressing the water systems with the most harmful, highest concentrations *first* by setting the MCL at 10 ppt.
- If this case, the result would be targeted funding and treatment to the areas that need it the most, which will also protect our most vulnerable citizens from soaring utility bills that may threaten their food and housing stability.
 - It will lessen the impact of such a large number of water utilities vying for limited treatment equipment, labor, and resources.
 - It will give time for regulation or legislation to prevent further introduction of PFAS into our environment through consumer products.
 - It will allow labs to develop the capability to test on a larger scale.
 - It will allow public water utilities to have flexibility in their budgets to address the current, multiple regulatory challenges of PFAS, lead and copper, and aging infrastructure.
 - And it will preserve consumer confidence in the safety of people's utmost necessity: drinking water.

August 1, 2023

CDPU Testimony for NDWAC Consideration on Proposed PFAS National Primary Drinking Water Regulation

Page 4 of 4

CDPU looks to the Council and EPA to help ensure that access to the most basic human necessity—safe, clean drinking water—continues to be affordable and equitable. As EPA continues its determination on a final National Drinking Water Regulation related to these six PFAS, CPDU asks that Council urge EPA to re-examine alternatives—regulations, timelines, and limits—that would provide a reliable, comprehensive benefit to the public by removing the greatest PFAS hazards, judicially using public funds, and ensuring the eventual end of the PFAS cycle.

The City of Columbus, Department of Public Utilities appreciates your consideration of these comments. Should you have any questions, please contact Kristen Atha at klatha@columbus.gov or call (614) 645-7541. Thank you again for your attention to and consideration of these comments.

Sincerely,



Kristen L. Atha

Director

Columbus Department of Public Utilities

klatha@columbus.gov

Ecc: Alana Shockey, Deputy Director
Janean Weber, Assistant Director, Regulatory Compliance
John Newsome, Columbus CDPU, DOW Administrator
Matthew Steele, Columbus CDPU, DOW Assistant Administrator
Robert Priestas, Columbus CDPU, DOSD Administrator
Stacia Eckenwiler, Columbus CDPU, DOSD Assistant Administrator



August 17, 2023

Lisa D. Daniels, Chair
Elizabeth Corr, Designated Federal Officer
National Drinking Water Advisory Council
Environmental Protection Agency
Office of Ground Water and Drinking Water
Mail Code 4601M
1200 Pennsylvania Ave, N.W.
Washington, D.C. 20460

Dear Ms. Daniels and Ms. Corr:

Members of the Environmental Protection Network (EPN) listened to EPA's August 8, 2023, consultation with the National Drinking Water Advisory Council (NDWAC) on the proposed PFAS national drinking water rule. EPN is an organization of nearly 600 former EPA career and political appointee scientists and regulators who provide pro bono technical assistance to communities with environmental problems and analyze EPA policies, regulations, and guidance.

Our members are concerned that NDWAC received very little information on the significance of PFAS exposures, namely the health effects of PFAS, and the scientific justification for use of the hazard index. The discussion at the August 8 meeting principally focused on the costs of implementing the proposed maximum contaminant levels (MCLs) and difficulties communicating the hazard index to the public. This letter summarizes the extensive health effects information on the six PFAS included in EPA's rule and justifies the use of a hazard index for four of those PFAS.

In addition to EPA's 2022 review of the PFAS health effects literature, systematic reviews were also conducted by the European Food Safety Authority in 2020, the Agency for Toxic Substances and Disease Registry in 2021, and the National Academies of Sciences, Engineering and Medicine (NASEM) in 2022. All these reviews found extensive human and animal data on the health effects of PFOA and PFOS. The animal health data were highly consistent with the human health effects data. Both human and animal studies link exposure to PFOA, PFOS, and other PFAS with multiple health effects, including immune system effects; increased cholesterol; liver, kidney, and thyroid problems; reproductive and developmental harm; and multiple types of cancer. These reviews document multiple health effects within the general population's range of exposure to PFOA and PFOS with blood serum or plasma levels in nanograms per milliliter (ng/mL) or parts per billion (ppb), not just in the range of highly exposed workers.

NDWAC is aware of the 2022 NASEM findings,¹ which focused on seven PFAS, four of which are included in EPA's proposed drinking water rule and are the most commonly detected PFAS in the blood of the U.S. population (PFOA, PFOS, PFHxS, and PFNA). NASEM reports that they found sufficient evidence of an association between exposure to these four PFAS and three others (MeFOSAA, PFDA, and PFUnDA) and the following diseases and health outcomes: decreased antibody response in adults and children; dyslipidemia in adults and children; decreased infant and fetal growth; and increased risk of kidney cancer in adults. NASEM also found limited or suggested evidence between exposure to these PFAS and the following health effects: increased risk of breast cancer in adults; liver enzyme alteration in adults and children; increased risk of testicular cancer in adults; thyroid disease and dysfunction in adults; and increased risk of ulcerative colitis in adults. In short, NASEM documented evidence of an association of those general population exposures across multiple body systems: immune system, reproductive and development system, metabolism, and renal carcinogenicity. NASEM found suggestive effects for five other adverse effects: breast and testicular cancers, liver damage in both children and adults, thyroid effects and damage to the gastrointestinal system. Again, note these effects are evident in the range of general population exposures which are associated with blood levels in the ng/mL or ppb range.

Consequently, NASEM recommends that clinicians measure PFAS in blood serum or plasma for patients with occupational or locational exposure to sources of PFAS contamination. For patients with blood serum or plasma concentrations between 2 ng/mL and 20 ng/mL (2 and 20 ppb, respectively), NASEM recommends clinicians encourage PFAS exposure reduction and screen for dyslipidemia, hypertensive disorder during pregnancy, and breast cancer. For patients with blood serum or plasma concentrations at or above 20 ng/mL (20 ppb), NASEM recommends clinicians encourage PFAS exposure reduction and screen for dyslipidemia; conduct thyroid function testing for those over 18 years old; assess for signs of kidney cancer for those over 45 years old; and assess for signs of testicular cancer and ulcerative colitis for those over 15 years old. NASEM recommends that this clinical guidance be updated every five years in order to keep current with the evolving science on PFAS.

Because this is the first time drinking water standards have been based on a hazard index, water systems do not have experience with communicating such a standard. For years, the Superfund program has successfully communicated to communities living near contaminated sites the risks of multiple pollutants using a hazard index approach. EPA's drinking water program is fortunate to have such in-house expertise and can build on the Superfund risk communication materials to develop information for public water systems. While those communication materials are being developed, NDWAC should understand that the hazard index accounts for the combined toxic effects of four frequently occurring PFAS in drinking water on thyroid hormone levels, lipid synthesis and metabolism, fetal and infant development, and immune and liver function.

Participants in the August 8 NDWAC meeting expressed concern that the health-based water concentrations (HBWCs) for the four PFAS used in the hazard index were not based on the same target organ. Because studies on all four PFAS have documented toxic effects on the thyroid, liver, and fetal/infant development, EPA could have selected any one of these common effects as the basis of the HBWCs. In order to be more health protective, however, EPA based the HBWCs on the most sensitive health effect for each of the PFAS as follows: thyroid effects for PFHxS; developmental effects for PFNA; liver effects for GenX; and thyroid effects for PFBS. The hazard index then adds the toxicity of all four PFAS to account for their effects on

¹ National Academies of Sciences, Engineering, and Medicine. 2022. Guidance on PFAS Exposure, Testing, and Clinical Follow-Up. Washington, DC: The National Academies Press. <https://doi.org/10.17226/26156>

multiple biological systems and functions. Given the weight of the evidence of damage throughout the human body, this approach is clearly more appropriate than a single target-organ hazard index.

In summary, EPN is concerned that NDWAC was not well-served by the August 8, 2023, consultation and should be better informed of the serious health risks posed by these six PFAS chemicals in advising EPA on how best to regulate these toxic chemicals in the nation's public water systems. The August 8, 2023, consultation failed to communicate the extent and severity of those risks.

Sincerely,

A handwritten signature in cursive script that reads "Michelle Roos".

Michelle Roos
Executive Director
Environmental Protection Network

cc: Bruno Pigott, Principal Deputy Assistant Administrator, EPA Office of Water
Jennifer McClain, Director, Office of Groundwater and Drinking Water, EPA Office of Water

KRISTEN ATHA
Director

May 30, 2023

FILED VIA FEDERAL eRULEMAKING PORTAL
Assistant Administrator Radhika Fox
Office of Water
Office of Ground Water and Drinking Water
Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, DC 20460

**RE: Proposed Rule, PFAS National Primary Drinking Water Regulation
EPA-HQ-OW-2022-0114; 88 FR 18638 (March 29, 2023)**

Dear Assistant Administrator Fox:

The City of Columbus, Department of Public Utilities (“CDPU”) appreciates the opportunity to comment on EPA’s proposed National Primary Drinking Water Regulation for PFOA, PFOS, and four other PFAS. CDPU operates drinking water and wastewater treatment plants serving over 220 square miles in the greater Columbus area. Our interest in PFAS stretches from the source water that feeds our drinking water plants to the effluent released from our wastewater plants, and everywhere in between. We are committed to improving water quality and protecting the environment, and we share EPA’s concerns about the risks that PFAS chemicals pose to the public.

CDPU provides critical services to our community. The three drinking water plants operated by CDPU delivered over 50 billion gallons of clean, safe drinking water last year, averaging 145 million gallons daily, to over 1.3 million residents of the Greater Columbus area. The City of Columbus has many residents living in environmental justice communities including Linden, Franklinton, Hilltop, and Near South. CDPU uses a complex multi-barrier approach utilizing state of the art equipment and the latest treatment technologies to ensure all requirements of the Safe Drinking Water Act (SDWA) are continuously met. Ratepayers keep these plants running.

CDPU drinking water facilities do not use, produce, or profit from PFAS compounds. We passively receive source water into our facilities that may contain PFAS from industrial, commercial, and domestic sources. We treat and distribute water 24 hours a day, 7 days a week, 365 days each year. And we comply with our permits and protect public health and the environment by providing safe drinking water.



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EPA requested comment on implementation challenges and considerations for setting the maximum contamination level (MCL) at the practical quantification level (PQL) for PFOA and PFOS, including on the costs and benefits related to this approach. EPA also asked for comment related to its evaluation of feasibility under the SDWA for the proposed PFOA and PFOS and hazard index MCLs. CDPU concurs with the comments made by the National Association of Clean Water Agencies (NACWA) and makes the additional comments below.

As a vested stakeholder with a duty to the public, CDPU supports EPA in its efforts to set its initial federal drinking water standard for PFAS at a protective, feasible (MCL). But CDPU urges EPA to consider the active loading of PFAS into water occurring through the manufacture and use of PFAS and PFAS containing products. CDPU also urges EPA to consider the accurate costs and feasibility, consumer confidence, and regulatory alternatives before it mandates its first MCL at the PQL. Several states have established feasible state standards for PFOA and PFOS at twice the currently proposed federal standard. Those plans would result in the best use of limited resources because they address the greatest health risks by prioritizing systems with the highest concentrations of PFAS.

I. EPA should consider source control as it implements its PFAS Strategic Roadmap to avoid cost-shifting PFAS remediation to water utilities and ratepayers.

Working its way through its PFAS Strategic Roadmap, EPA is using CERCLA to address sites that are contaminated with PFAS, and the SDWA to address water that is contaminated with PFAS. One option that it did not consider is a direct campaign to eliminate additional PFAS load in our water and soil: to ban the manufacture and use of PFAS, except for in extraordinary circumstances with stringent controls. While most manufacturers voluntarily phased out PFOA and PFOS in the early 2000's for most uses, there is nothing being done to stop the active loading of new PFAS into our environment—onto our soil and into our water—from PFAS laden products that continue to be rolled out to consumers. At this very minute, manufacturers that produce and use PFAS are profiting from the pollution that ratepayers will be paying to clean out of their water for years to come. Water utilities can dedicate billions of dollars to treat water for decades but will never be able to tackle the source. And the source, those companies profiting from a product that is a known health concern, can continue to lawfully pump those pollutants into our world.

An MCL set without proper recognition of implementation, social, and opportunity costs shifts the financial burden of minimizing risk of human exposure to PFAS from private businesses to the public. The proposed drinking water regulation (as currently written) would shift the financial and technical responsibility from the manufacturers that create and use PFAS in their processes to downstream utilities. This burden will be borne by ratepayers.

The MCL should be set with the understanding that PFAS exists in the background of the environment and everyday life. The cost-benefit to achieve these regulatory requirements in the water industry needs to be weighed against the cost-benefit of removing it from food packaging, personal care products, clothing, and other direct and indirect exposure routes. The only way to address and stop the growing concentration of PFAS contaminants in the hydrological cycle is to stop introducing additional PFAS loads. EPA should re-assess the navigation of its PFAS Roadmap.

II. EPA should consider costs and timing before settling its initial MCL at the PQL.

EPA is mandated to consider costs when setting the MCL. The standard is feasibility, which contemplates expense, rather than mere capability. The regulation as proposed is not feasible, nor is it capable of being met under EPA's proposed timeline.

A. MCLs at 4 ppt PFOA and PFOS are not feasible.

Under section 1412(b)(4)(B) of SDWA, EPA must conduct an economic analysis during the development of drinking water contaminant regulations. EPA must take costs into consideration when setting an enforceable MCL as close to the maximum contaminant level goal (MCLG) *as feasible*. The SDWA analysis on feasibility is only acceptable if it uses accurate assumptions for its inputs. EPA's cost estimate falls short.

The actual costs that water utilities will face for treatment are significantly higher than EPA's estimate. EPA's estimate of nationwide proposed costs to treat to 4 ppt PFOA and PFOS falls between \$776M and \$1.2B. This range is much too low. First, there are a few factors that will drive the true cost of meeting 4 ppt much higher. The first is inflation. Last summer, construction costs skyrocketed 25 to 30% higher than 2021 costs.¹ EPA used 2021 dollars in its estimates so construction costs will already be 25 to 30% higher than the estimates used in the analysis. Second, supply and demand will force additional upward pressure on prices. Prices will increase significantly based on scarcity in the supply chain and labor force as 3,500 utilities nationwide hustle to comply.² This demand will continue to drive up prices on equipment, lab services, media, and reactivation. Third, EPA's cost estimate fails to account for the increased costs associated with increased health risks from the GHG emissions that will result from these treatment processes. Reactivation, disposal, incineration, and trucking of granulated activated carbon media will all result in additional greenhouse gas emissions. This treatment process (and its resulting emissions) does not currently exist at most plants so there will be an increase in overall emissions industry-wide. EPA should balance the benefits of pushing the MCL down to the PQL with the costs—financial, human health, and environmental—that will come from the added greenhouse gas emissions created through treatment.³

As part of its cost analysis, EPA also failed to recognize the factor of the opportunity costs of this regulatory action. As drinking water rates soar in response to the MCL and the required implementation of expensive treatment technologies, there is also an opportunity cost that will be realized by households and businesses. Consumers will have to make tough choices as they have less money. Businesses will raise prices to reflect their increased costs. Products and services will then cost consumers more and they will have even less money for discretionary spending. This cycle is exacerbated in Justice40 neighborhoods where there is already a lack of extra money and increases in the cost of basic needs results in going without basic necessities.

¹ *Inflation Taking the Bite out of New Infrastructure Projects*, US News and World Report article. June 19, 2022. [Inflation Taking Bite Out of New Infrastructure Projects \(usnews.com\)](https://www.usnews.com/story/news/economy/2022/06/19/inflation-taking-the-bite-out-of-new-infrastructure-projects)

² In the proposed rule, EPA estimates that 3,400-6,300 water systems will be impacted by the proposed regulation of 4 ppt for PFOA and PFOS with the 1.0 hazard index for the four other PFAS.

³ Beyond the factors that are not yet reflected in EPA's cost estimate, NACWA's members participated in a survey that proved that EPA's estimates were significantly lower than real world data from utilities that have already implemented treatment. EPA's estimate for capital costs was 2.9 times lower on average than the reported value for 60 utilities.

EPA's acute underestimation is borne out in CDPU's numbers. In February 2023, CDPU received an estimate for the cost of implementing granulated activated carbon treatment at its water plants, which includes three existing plants and one plant currently in design.⁴ Estimated capital costs for CDPU's four plants falls between \$338 million to \$363 million, but could realistically approach \$544 million. Additionally, the current estimate for *annual* operation and maintenance costs is between \$29.4 million to \$43.9 million, depending on whether virgin or reactivated carbon is used. To put that in perspective, CPDU—one public water system out of 3,500—may need to spend over half a billion dollars by 2030 to treat the very low levels of PFAS in our drinking water. The result would be an extreme cost for miniscule benefit. EPA's cost-benefit analysis is distorted because its nationwide estimate does not come close to being accurate.

Even with a historically high level of federal funding reaching billions of dollars, the proposed rule's estimated costs will far exceed available funding. NACWA's benefit-cost report projected that water utilities and their ratepayers will pay six times the amount allocated, plus operation and maintenance costs.⁵ While the federal funding will provide much needed assistance, it will not offset the necessary expenditure enough to prevent adverse outcomes for water utilities and ratepayers.

Utilities are struggling with maintaining affordable rates as they face an onslaught of mandated capital and regulatory projects.⁶ Public water utilities are stewards of public funds and must choose how to best protect human health with limited resources. To accommodate the costs of new treatment, public water utilities will either have to devote fewer resources to ongoing operations, infrastructure repairs, and responses to other health priorities, or they will have to raise rates. Ratepayers—especially in our environmental justice communities—will feel the burden of higher utility bills. Regulating without proper consideration of cost will affect affordability and could result in negative outcomes in disadvantaged communities.

Importantly, these resource intensive efforts by water utilities are only treating the symptoms rather than the disease. Treatment might reduce the current amount of PFOA and PFOS in a cup of water from low parts per trillion to an amount in parts per quadrillion, but it will not reduce the amount of PFAS that the public is exposed to everyday through other common household and environmental sources. This continued exposure will continue unabated so long as PFAS chemicals are permitted to be used in consumer goods manufactured in, or imported to, the United States.

B. Public water utilities will need more time to comply, especially if the MCL is 4 ppt.

Even if EPA believes that the 4 ppt MCL is generally feasible, EPA's proposed timeline to meet it is not. Meeting an MCL of 4 ppt will require the commitment of an extremely large amount of public money to accomplish these significant construction projects. Public water utilities across the nation,

⁴ Engineers at Hazen and Sawyer produced a memo detailing the Cost of Implementation of GAC for PFAS Treatment at Columbus, OH Water Plants, dated March 9, 2023. Hazen and Sawyer is a National A/E firm which has worked in surface water and groundwater plants, developed solutions for meeting PFAS targets, installed granular activated carbon and ion exchange facilities, and researched innovative treatment approaches.

⁵ *Federal Funding Analysis*, at page 67 of the Black and Veatch Study attached to NACWA's comment letter.

⁶ Impending regulatory challenges for water utilities are the Lead and Copper Rule Revision, Lead and Copper Rule Improvements, and CERCLA designation of PFAS. Additionally, water utilities are struggling with replacing aging infrastructure and meeting the challenges of climate change, including reducing greenhouse gas emissions while simultaneously preparing for its effects.

like CDPU, must navigate their budgeting, funding, and procurement processes before a shovel strikes the ground to add a treatment process. It will take time to do enough sampling to ensure that the expenditure of hundreds of millions of dollars is warranted, and then to design and plan the project. Then comes the long haul of actually constructing the treatment. Added to these considerations is the requirement that the public water utility must maintain 100% of their operations while building out the treatment. In short, public projects of this magnitude take time.

On top of the typical lengthy project timeline, if 3,500 water utilities enter the market for the same few treatment techniques, there will be massive supply/demand issues. There are already limited resources for granular activated carbon equipment, media, and regeneration services. The same can be said for lab services for sampling. Even though EPA received dozens of applications for certification, on a recent call NACWA members from around the country only identified two labs that the entirety of utilities present were using for PFAS analysis. Compounding this difficulty are the general supply chain issues and labor shortages that linger across the United States. Build America, Buy America and other federal requirements add more hurdles. Three years is not enough time to get us to the finish line, especially when compliance and the corresponding consumer confidence are hanging in the balance.

Allowing more time for compliance will also enable EPA to tackle the data gap issues that are currently plaguing water utilities and PFAS science. UCMR 5 data will be instrumental in the nation's understanding of its PFAS problem. This crucial information will come too late with the current compliance timeline to allow EPA and water utilities to respond accordingly.

One option to solve the timing issues would be to extend the compliance timeline. Another option is a phased approach to the MCL (starting with a higher initial MCL) that would address these problems by reducing the competition for limited resources for installing treatment and allowing the science to catch up to policy. It would tackle the bulk of, and most significant, human health risks by targeting systems with the highest PFAS concentrations first.

III. EPA should consider reliability and consumer confidence before setting its initial MCL at the PQL.

Issuing the initial MCL at the PQL has high consequences because once EPA sets the MCL, water utilities will have to start expending the billions of dollars to comply to meet it. Here, where there is scientific uncertainty and the proposed MCL is pushing the available treatment and sampling techniques to their physical extremes, EPA should set a MCL that meets the requirements of the SDWA that also can be supported as rational to the public. As more data becomes available from UCMR 5 and other studies, there will be opportunities to issue a more stringent MCL.

A. The MCL and the PQL are too close.

EPA requested comment on setting the MCL so close to the PQL and its effect on water utilities. In that discussion, EPA recognized that “[t]he agency must have a high degree of confidence in the quantified result as it may compel utilities to make potentially costly compliance decisions in order to comply with the MCL.” It also recognized that water utilities operate conservatively to maintain water quality and “typically aim to achieve lower than the MCL to avoid a violation.” In reality, this results in proactively adding treatment if there is any possibility of violating the MCL (as will play out at CDPU).

But when the MCL is so low—measured in parts per trillion—and the difference between installing hundreds of millions of dollars of treatment, or not having to treat, is one drop in 20 Olympic size swimming pools, it is absolutely necessary to provide for quantitative reliability. The margins are too tight right now when the science is still emerging and the risks of overcommitting public money is high. The reality is that a water utility could graze the MCL level if the sampler applied deodorant that morning, wore a jacket while grabbing the sample, or used a typical sampling container.⁷ CDPU supports EPA in its decision that sample results below the PQL are to be recorded as zero. Setting the first ever PFAS MCL this low is not feasible (as discussed above) or reasonable when so much benefit can be realized with setting a higher MCL first to address the systems with significant PFAS contamination.

B. An overly-aggressive MCL for PFAS will shake consumer confidence.

Consumer confidence is key in any water regulation. Clean, safe drinking water is ultimately only as drinkable as the public understands it to be. In the case of PFAS, the science is clear on the risks of consuming high levels of the most damaging formulas of PFAS. Where science is still developing is in relation to the impacts and risks of health implications of low levels of less studied versions. Still further undeveloped is the science on how exposures vary between drinking water and the hundreds of other ways humans are exposed every day. What we know is that drinking water only accounts for 20% of human exposure to PFAS,⁸ but the risk that the public will be dissuaded from drinking safe tap water is increasingly high. This is especially true if the MCL is issued at the level proposed by EPA alongside the current compliance timeline. Widespread violations of the MCL may be triggered by EPA's proposed approach, and even if levels are close to the PQL and much lower than other sources of PFAS exposure, consumers will lose confidence in the safety of publicly supplied drinking water.⁹

Additionally, EPA's use of a hazard index adds another layer of confusion. If EPA includes a hazard index in the final version of the regulation, water utilities will be relying on EPA to develop a communication strategy to protect consumer confidence. Feasible standards with reasonable timelines will safeguard consumer confidence and protect our most vulnerable residents from additional burden.

IV. EPA should reconsider the proposed alternatives before setting its initial MCL at the PQL.

As it conducted its required analysis when crafting the proposed rule, EPA deliberated various options to reduce PFAS from drinking water. One option that was considered but not selected, was to address the water systems with the most harmful, highest concentrations first with a MCL at 10 ppt. As discussed above, the feasibility analysis for that decision was flawed.

⁷ https://www.epa.gov/sites/default/files/2020-01/documents/pfas_methods-sampling_tech_brief_7jan2020-update.pdf

⁸ Relative Source Contribution (RSC) is an estimate that drinking water contributes 20% of total exposure to PFOA and PFOS. Other sources contributing 80% of exposure to PFOA and PFOS include but are not limited to air, foods, incidental soil/dust ingestion, and consumer products (U.S. EPA, 2016b) (U.S. EPA, 2016a).

⁹ Consumer confidence concerns affect low-income and underserved environmental justice area residents even more if they spend their limited resources on purchasing bottled water that is less-regulated.

The crux of EPA's decision to set the MCL at 4 ppt instead of 10 ppt is that EPA found the former to be feasible. But that determination was erroneous because EPA did not accurately account for the cost of treatment and other social costs to environmental justice communities and from increased GHG emissions. Once EPA adjusts its calculation, if it determines that an MCL of 4 ppt is not feasible, then it will have an opportunity to revisit the regulatory alternative of 10 ppt and perform the analysis again with more realistic costs and benefits to determine if a higher MCL is appropriate and justifiable under the SDWA statutory criteria.

EPA has already acknowledged that a higher MCL would decrease the number of water utilities that will need to treat their water at this time. But it will also result in targeted funding and treatment in the areas across the country that need it the most, which will protect our most vulnerable citizens from soaring utility bills that may threaten their food and housing stability. It will lessen the impact of such a large number of water utilities vying for limited treatment equipment, labor, and resources. It will give time for regulation or legislation to prevent further introduction of PFAS into our environment through consumer products. It will allow labs to develop the capability to test on a larger scale. It will allow public water utilities to have flexibility in their budgets to address the current, multiple regulatory challenges of PFAS, lead and copper, and aging infrastructure. And it will preserve consumer confidence in the safety of people's utmost necessity: drinking water.

V. CONCLUSION

CDPU looks to EPA to help ensure that access to the most basic human necessity—safe, clean drinking water—continues to be affordable and equitable. As EPA continues its determination on a final National Drinking Water Regulation related to these six PFAS, CDPU asks that EPA recognize that public water utility resources are limited as we try to rise to the challenge of meeting each increasingly stringent drinking water standard simultaneously. Additionally, CDPU asks that EPA's feasibility analysis include accurate costs for treatment, a quantification for the burden of opportunity costs, and the financial, human health, and environmental costs of increased greenhouse gas emissions, which will result from an MCL of 4 ppt. Finally, CDPU asks that EPA re-examine alternatives—regulations, timelines, and limits—that would provide a reliable, comprehensive benefit to the public by removing the greatest PFAS hazards, judicially using public funds, and ensuring the eventual end of the PFAS cycle.

The City of Columbus, Department of Public Utilities appreciates your consideration of these comments. Should you have any questions, please contact Kristin Atha at klatha@columbus.gov or call (614) 645-7541. Thank you again for your attention to and consideration of these comments.

Sincerely,



Kristin L. Atha
Director
Columbus Department of Public Utilities
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May 30, 2023

CDPU Comments to the Proposed PFAS National Primary Drinking Water Regulation

Page 8 of 8

Ecc: Alana Shockey, Deputy Director
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Date: August 8, 2023

Comments for EPA NDWAC

Sean Lammerts, PE – Black & Veatch

Thank you for allowing me the opportunity to speak with you all today. My name is Sean Lammerts and I am with Black & Veatch. Black & Veatch is a global engineering firm, headquartered in the United States, that has extensive experience with the design and construction of drinking water treatment systems. In this capacity we are routinely relied upon to generate accurate cost estimates for water treatment projects. To this end, we recently developed a cost model for PFAS treatment to estimate the capital, operating, and maintenance costs necessary for these advanced treatment systems.

When EPA proposed the national primary drinking water regulations earlier this year for PFOA and PFOS, the EPA cost model was used to project the national cost impacts for communities that rely on public water systems. Using our model, we estimate that the national costs could exceed \$3.2 billion annually. By comparison, the EPA's proposal estimated that the costs would be \$1.2 billion annually using a similar set of occurrence and entry point assumptions.

To better understand these differences, the cost model from EPA was compared to our cost model on a treatment flow basis. Additionally, both models were compared to a large set of case studies that was collected by the American Water Works Association earlier this year. The results of this analysis showed that both the actual costs associated with the case studies and the Black & Veatch cost model projections were upwards of 3 to 4 times higher than the EPA model projections. For your convenience, I have provided our report.

Further, our model shows that the impacts will be non-linear across the system size ranges with smaller systems faced with higher per-user cost of compliance. The cost estimates by Black & Veatch show that household impacts could range from \$60 per household annually in very large communities up to thousands of dollars annually for households in smaller communities.

Given the divergence in actual costs versus the EPAs cost estimates, the parity concerns across system sizes and the importance of this regulation, it appears that a review of the cost and funding assumptions would be prudent before finalizing this regulation.

WITAF 56 TECHNICAL MEMORANDUM

PFAS National Cost Model Report

B&V PROJECT NO. 409850

PREPARED FOR



**American Water Works
Association**

American Water Works Association

7 MARCH 2023



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Abbreviations

AACE	Association for the Advancement of Cost Engineering
AWWA	American Water Works Association
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFPUA	Cape Fear Public Utility Authority
CIP	Clean-In-Place
CWS	Community Water System
DBP	Disinfection Byproducts
EBCT	Empty Bed Contact Time
EPA	U.S. Environmental Protection Agency
EPTDS	Entry Point to the Distribution System
FRP	Fiberglass Reinforced Plastic
ft	Feet
GAC	Granular Activated Carbon
g/cc	Grams per Cubic Centimeter
gpm	Gallons per Minute
gpm/sf	Gallons per Minute per Square Feet
HRT	Hydraulic Retention Time
IX	Ion Exchange
kWh	Kilowatt-hour
lb/gal	Pounds per Gallon
LHHCWD	La Habra Heights County Water District
MCL	Maximum Contaminant Level
mgd	Million Gallons per Day
mg/L	Milligrams per Liter
NF	Nanofiltration
NTNCWS	Non-Transient Non-Community Water System
NPDWR	National Primary Drinking Water Regulation
PFAS	Per- And Polyfluoroalkyl Substances
PFBS	Perfluorobutane Sulfonic Acid
PFHpA	Perfluoroheptanoic Acid
PFHxS	Perfluorohexane Sulfonate
PFNA	Perfluorononanoic Acid
PFOA	Perfluorooctanoic Acid
PFOS	Perfluorooctane Sulfonic Acid
ppt	Parts Per Trillion
PWS	Public Water System
RO	Reverse Osmosis
SDWIS	Safe Drinking Water Information System
sf	Square Feet
SLR	Surface Loading Rate
T&O	Taste and Odor

TDH	Total Dynamic Head
TDS	Total Dissolved Solids
TOC	Total Organic Carbon
UCMR	Unregulated Contaminant Monitoring Rule
WITAF	Water Industry Technical Action Fund

1.0 Acknowledgement

This study was a collaborative effort; many individuals and utilities spent time compiling data, answering questions, and making contacts. We wish to thank the following utilities for sharing data and Steering Committee Members for their time and insight:

Partner Utilities:

- Cape Fear Public Utility Authority (CFPUA)
- City of Ann Arbor
- Greater Cincinnati Water Works
- Plainfield Charter Township
- City of North Miami Beach
- Miami-Dade County Water and Sewer Department
- Tucson Water

Steering Committee Members:

- Amy Stoffer – Northern Kentucky Water District
- Cynthia Lane – Platte Canyon
- Carel Vandermeiden – CFPUA
- Robert Cheng – Coachella Valley Water District
- Zaid Chowdhury – Garver USA
- Chuck Hertz – Retired

2.0 Introduction

Known as “forever chemicals” because they do not easily biodegrade, per- and polyfluoroalkyl substances (PFAS) are drawing increased scrutiny from health agencies, water utilities, and the public for their presence in drinking water and their effects on human and environmental health. They have quickly become contaminants of great concern in drinking water.

Six PFAS compounds were monitored in finished drinking water as part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) between 2013 and 2015 to quantify their prevalence across the United States. The UCMR program provides the U.S. Environmental Protection Agency (EPA) with nationally representative occurrence data to inform drinking water regulations. Using the results from UCMR 3, in February 2021, the EPA published a final determination to regulate perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS) and signaled an interest in considering the regulation of additional PFAS. The EPA is expected to promulgate the first national primary drinking water regulation for PFAS in the United States in September of 2024 as a result of this regulatory determination.

U.S. federal laws and executive orders stipulate that the U.S. EPA estimate the cost of compliance for this new primary drinking water regulation. Black & Veatch was selected by the American Water Works Association (AWWA) to develop a national cost estimate for water systems to remove PFAS from drinking water to better understand the financial impacts to communities and the costs to comply with a national primary drinking water regulation (NPDWR), a policy that could impact each of the more than 66,500 public water systems.

The project was funded by the Water Industry Technical Action Fund (WITAF), which is managed by the AWWA’s Water Utility Council to support projects, studies, analyses, reports, and presentations in support of the organization’s legislative and regulatory agenda. The national cost estimate and its cost models, developed under WITAF 056, are intended to support to AWWA’s engagement with the U.S. EPA and Congress on the differences in financial impacts of treating drinking water to various PFAS regulatory limits. WITAF funded a separate project (WITAF 057) to generate a national PFAS occurrence database using data from state monitoring and UCMR3. This national database was used as an input for the WITAF 057 project.

The national cost modeling tool programmatically evaluates each public water system (PWS) with occurrence data from WITAF 057 to generate a dataset of the most probable capital and operating costs. Those costs are then scaled up nationally to account for the PWSs without data captured in WITAF 057 to quantify the national cost of compliance of a proposed regulation, bringing flexibility for data-driven responses to EPA cost assessments. This project brought together occurrence data, cost data, and best practice design methodology to help ensure the U.S. EPA’s proposed national primary drinking water regulations for PFAS accurately reflect cost estimates for drinking water treatment.

3.0 PFAS Treatment Technologies

Treatment strategies for PFAS in drinking water include proven, commercially available technologies as well as emerging technologies. Many of these developing technologies have been demonstrated on the bench scale but have not yet been proven at the full scale or are not yet commercially available. Commercially available technologies that have been demonstrated at full scale in the field to reduce concentrations of PFAS in drinking water are limited to the following:

- Granular activated carbon (GAC).
- Ion exchange (IX).
- Nanofiltration (NF) and reverse osmosis (RO).

Treatment considerations for the application of each of these technologies are described in the following subsections.

3.1 Granular Activated Carbon

GAC media is a well-known adsorbent for organics and has been widely applied in water treatment. GAC is produced from carbon-based materials such as coal, coconut shells, peat, or wood that has been “activated” to produce a highly porous media with adsorptive properties. The pores contain sites on which organic compounds become attached and are adsorbed onto the activated carbon matrix.

GAC treatment applications include removal of organics, such as color, disinfection byproducts (DBP) and their precursors, taste and odor (T&O) causing compounds, industrial chemicals, and emerging contaminants such as PFAS, endocrine disrupting compounds, and pharmaceuticals and personal care products. Each of these contaminants compete for adsorption sites on GAC media with targeted PFAS if present. In some cases, co-adsorption can be viewed as a benefit for using GAC as the co-contaminants are simultaneously removed. Cost analyses and removal performance models must balance competitive adsorption of co-contaminants and its associated detrimental performance impact on PFAS removal.

GAC has a finite capacity for adsorbing compounds. High concentrations of organics or high flow rates will lead to more frequent media replacement. In general, short-chained PFAS are less readily adsorbed and less strongly bound than long chain compounds. The overall efficacy of GAC removal of PFAS highly dependent on the water matrix, the water treatment goals, and the design of the system. One of the most important design parameters is the empty bed contact time (EBCT), or the time during which the water is in contact with the media bed (also the duration at which adsorption can occur), assuming the water flows through the entire bed at a constant velocity. A desired EBCT will result in breakthrough when the adsorptive capacity of the media has been exhausted. The media must be either replaced or reactivated at that time.

3.1.1 Implementation and Operational Considerations

GAC applied for PFAS removal is most effective when used solely as an adsorbent. Conventional granular media filters containing GAC are typically designed for short EBCTs and must be frequently backwashed for removal of particulate material that is retained in the media. Such backwashing disrupts the adsorption front. Short EBCTs and backwashing lead to fast breakthrough of contaminants and underutilization of GAC media. If a water treatment facility contains conventional filters, contactors for GAC adsorption are typically located downstream.

Process selection (including GAC media selection) is typically confirmed through demonstration testing (bench-, pilot- or full-scale studies) to account for the unique characteristics of the source water.

GAC adsorption treatment systems installed for PFAS removal typically provide a 10 to 20 minute EBCT and a surface loading rate of 4 to 10 gallon per minute (gpm) per square foot of media (gpm/sf). PFAS adsorbers are applied in two main configurations: pressure vessels or gravity basins.

- Pressure vessel configurations are more common in small systems (less than approximately 10 million gallons per day [mgd]). Pre-engineered pressure-vessel type GAC treatment systems are widely available. Vessels are typically carbon steel or fiberglass reinforced plastic (FRP). Pressure vessels may be installed in single (parallel) or dual stage (series/lead-lag) arrangements.
 - The single stage arrangement allows for columns to be operated in various stages of breakthrough or exhaustion, resulting in an overall effluent below the treatment target. This arrangement can result in better media utilization, produce a more consistent product water quality, and lessen impact of potential overruns on individual vessels. Single stage systems typically include N+1 redundancy.
 - The dual stage arrangement allows for simultaneous production during media replacement, and sampling between vessels ensures that lag vessel effluent always meets treatment targets. The lead vessel can be in service until the media is completely exhausted, leading to higher utilization of the adsorbent media. The dual stage arrangement includes built-in redundancy as either the lead or lag vessel can be removed from service without reducing the treatment flow rate. Thus, no dedicated redundant vessels are typically provided.
- To avoid an excessive number of pressure vessels, gravity basin configurations are typically applied by large systems with design flows greater than approximately 10 mgd. Gravity basins are typically single stage and operated at various stages of breakthrough, similar to a single stage pressure vessel arrangement. The basins themselves are typically constructed of concrete with an N+1 redundancy because of the single stage arrangement.

Exhausted GAC filter media will be saturated with PFAS. Bulk GAC can be reactivated by the media supplier through thermal treatment at high temperatures (up to 1800° F) to remove and destroy adsorbed contaminants (Rebecca DiStefano, 2022). This reactivation process restores the media's adsorptive capacity, allowing the media to be returned for reuse. GAC is sometimes regenerated by heating the media to temperatures typically less than 400° F to remove a portion of the adsorbed contaminants. However, this process will not remove all the compounds and will not destroy the PFAS compounds; therefore, it is not appropriate for GAC utilized for PFAS removal. Media suppliers may not accept the low volumes of GAC required by small systems for reactivation, forcing them to dispose of spent GAC and replace it with new (virgin) material.

Disposal alternatives for exhausted GAC that will not be reactivated for municipal reuse include disposal by reactivation for industrial reuse, incineration, and landfilling. The cost of each disposal method depends on proximity to disposal sites, hazardous waste classification, and volume of material. Disposal costs can be a significant operational cost for GAC treatment systems.

The EPA proposed to designate PFOS and PFOA as hazardous substances under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) in August of 2022. This designation is expected to limit the disposal sites willing to accept spent GAC media. Additionally, the practice of reactivating GAC media contaminated with PFAS is expected to be more limited in drinking water applications.

3.1.2 Assumptions for Cost Estimation

The cost model includes capital costs, annual operating and maintenance costs, life-cycle costs, and annualized costs. The assumptions that drove the results of those cost estimates are summarized in this section.

The costs for GAC contactors depend on the contactor type, size, number, and ancillary processes such as backwash pumps/recovery basins and contactor influent pumps/wet wells. The primary process design assumptions for each of these factors are summarized in Table 3-1.

Table 3-1 GAC Design Process Assumptions

Contactor Type	Parameter		Assumption/Input
Pressure Vessel	Treatment Plant Capacity		1-12 mgd
	Surface Loading Rate ^(Note 1)		4-10 gpm/sf (most likely 6 gpm/sf)
	Empty Bed Contact Time ^(Note 1)		10-20 min (most likely 18 min)
	Vessel Diameter		6-12 ft
	Arrangement		Dual Stage
	Redundancy		None
	Influent Pump Station	TDH (total dynamic head)	45 ft
Gravity Basin		Design HRT (hydraulic retention time)	15 min
	Treatment Plant Capacity		> 12 mgd
	Surface Loading Rate ^(Note 1)		4-10 gpm/sf (most likely 4 gpm/sf)
	Empty Bed Contact Time ^(Note 1)		10-20 min (most likely 18 min)
	Filter Dimensions		8-20 ft cell width, 2:1 length to width ratio
	Arrangement		Single Stage
	Redundancy		N+1
	Influent Pump Station TDH, design HRT		30 ft, 15 min

Contactor Type	Parameter	Assumption/Input
Common	Backwash ^(Note 2)	Loading Rate
		13 gpm/sf
		Duration
		30 min
	Influent Pump Station ^(Note 3)	Frequency
		30 days
		Pump Design TDH
		60 ft
	Backwash Water Recovery Basin ^(Note 4)	Pump Efficiency
		70%
	GAC Media	Motor Efficiency
		85%
	Water Depth	20 ft
		Backwash Cycles Held
	Apparent Density	1.0
		0.5 gram per cubic centimeter (g/cc)
	Contactor Area Factor (for pipe gallery and appurtenances)	
	2.0	

Notes:

1. For adsorptive media, the major specified process design inputs are the surface loading rate (SLR) and the EBCT. For each of these factors, a minimum, maximum, and most likely number was assumed using feedback from existing treatment systems. The minimum, maximum, and most likely numbers for the published model outputs are summarized herein. National variability in SLR and EBCT is included in the model using a Monte Carlo simulation. The details of how this statistical method was employed within the cost modeling tool is described in Section 5.3.
2. Backwash pumps are required for periodic backwashing of the media.
3. An influent pump station is presumed to be required to accommodate the additional headloss necessary to an existing process train.
4. Backwash recovery basin omitted from systems for size category 1 and 2.

3.2 Ion Exchange

IX is an adsorptive water treatment process that involves the selective exchange of ions in solution with ions bound to a resin matrix. IX has a long history in water treatment, and resins are manufactured for a variety of contaminants, including PFAS. Several manufacturers provide specific IX resins designed to be selective for PFAS as the market has expanded for their use. Some resins originally intended for removal of other contaminants (such as perchlorate) have shown a high degree of selectivity and capacity for PFAS as well.

IX resins, like GAC, have a limited capacity for adsorption. The adsorptive capacity of IX resins is affected by contaminant concentrations and flow rates in the same manner as GAC. However, the IX resins surveyed have proven to be highly selective toward PFAS removal, exhibiting minimal removal of other contaminants. This may result in a greater adsorptive capacity for PFAS compared to GAC, without, however, the co-contaminant removal benefits of other technologies. In general, short-chained PFAS are less readily adsorbed and less strongly bound than long chain compounds. The overall efficacy of IX for PFAS removal is highly individual to the water matrix, the water treatment goals, and the design of the system.

An IX treatment process does not result in a fixed percentage removal of a contaminant over time, as there is a variable degree of contaminant removal and gradual or sharp contaminant breakthrough. Although it is selective to certain contaminant groups, the resin can experience interference from other compounds in the water matrix. The most preferred compound will tend to exhibit long runs and sharp breakthroughs; less preferred compounds will have earlier, more gradual breakthroughs.

Exhaustion of the media is determined (in a fashion similar to that for GAC) through the measure of the contaminant in the effluent (breakthrough). When the adsorptive capacity has been exhausted, the resins require replacement or regeneration. Because of the proposed CERCLA hazardous substance designations for PFOA and PFOS as discussed in Subsection 3.1.1, single use (fixed-bed) systems are currently being considered for IX, requiring disposal of spent media and replacement with new resin when exhausted. PFAS destruction technologies are currently in research and development that may be able to destroy PFAS in the brine stream, although that technology is not yet matured enough for full-scale implementation.

Fixed-bed IX has been demonstrated at full scale in the field as a proven PFAS removal technology. Fixed-bed ion exchangers applied for PFAS removal consist of carbon steel or FRP pressure vessels and typically 1.5 to 3 minutes of EBCT (as compared to 10 to 20 minutes for GAC). IX can be favorable because of the smaller footprint required.

3.2.1 Implementation and Operational Considerations

The efficacy of an IX treatment system will likely be improved by a pretreatment step to remove interferences such as suspended solids, particulate natural organic matter, and colloidal compounds. Commercially available filters can be selected depending on the pretreatment needs to improve the treatment capacity of the IX system. This prefiltration step can prevent deposition of fine particles on the resin, reduce pressure drop across a column, and increase run time.

Process selection (including resin selection) is typically confirmed through demonstration testing (bench-, pilot- or full-scale studies) to account for the unique characteristics of the source water.

Ion exchange treatment systems are conventionally installed in pressure filters in lieu of gravity basins. As with GAC, the pressure vessels can be implemented in single or dual stage arrangements. Considerations for the single or dual stage arrangements are summarized in Subsection 3.1.1.

Exhausted IX resin will be saturated with PFAS. Disposal alternatives for exhausted IX resins include incineration and landfilling. The cost of each disposal method depends on proximity to disposal sites, hazardous waste classification, and volume of material. Disposal costs can be a significant operational cost for IX treatment systems.

3.2.2 Assumptions for Cost Estimation

The costs for IX Contactors depend on the contactor type, size, number, and ancillary processes such as backwash pumps/recovery basins and contactor influent pumps/wetwells. The primary process design assumptions for each of these factors are summarized in Table 3-2.

Table 3-2 IX Design Process Assumptions

Parameter		Assumption/Input
Surface Loading Rate ^(Note 1)		5-12 gpm/sf (most likely 8 gpm/sf)
Empty Bed Contact Time ^(Note 1)		1.5-3.0 min (most likely 2.0 min)
Vessel Diameter		4-12 ft
Contact Mode		Lead-Lag
Redundancy		None
Influent Pump Station ^(Note 2)	Pump Efficiency	70%
	Motor Efficiency	85%
	TDH	60 ft
	Design HRT	15 min
Backwash ^(Note 3)	Loading Rate	5 gpm/sf
	Duration	30 min
	Frequency	30 days
	Pump Design TDH	60 ft
Backwash Water Recovery Basin ^(Note 4)	Water Depth	20 ft
	Backwash Cycles Held	1.0
IX Resin	Apparent Density	1.05 g/cc
Contactor Area Factor (for pipe gallery and appurtenances)		2.0

Notes:

1. For adsorptive media, the major specified process design inputs are the SLR and the EBCT. For each of these factors, a minimum, maximum, and most likely number was assumed using feedback from existing systems. The minimum, maximum, and most likely numbers used for the published model outputs are summarized herein. National variability in SLR and EBCT is included in the model using a Monte Carlo simulation. The details of how this statistical method was employed within the cost modeling tool is described in Section 5.3.
2. An influent pump station is presumed to be required to accommodate the additional headloss necessary to an existing process train.
3. Backwash pumps are required for periodic backwashing of the media.
4. Backwash recovery basin omitted from systems for size category 1 and 2.

3.3 Reverse Osmosis and Nanofiltration

RO and NF are membrane-based water treatment processes in which a semi-permeable barrier removes dissolved contaminants from water. This capability is attractive when considering the need to remove total dissolved solids (TDS), specific ions such as calcium, magnesium, sodium, chloride, sulfate, and hardness; DBP precursors; and T&O causing compounds as well as high levels of PFAS. RO/NF processes are commonly applied in water treatment plants and have applications ranging from desalination of brackish water, softening, and the removal of nitrate, agricultural chemicals (e.g., atrazine), color, total organic carbon (TOC), DBP precursors, and PFAS. Both RO and NF processes are capable of a high rejection of PFAS. While RO/NF systems are more expensive than GAC or IX systems, they are most viable when the GAC/IX replacement frequency requirements are cost-prohibitive because of high concentrations of influent PFAS.

The key differences between RO and NF are salt passage and feed pressure. RO membranes reject a higher percentage of dissolved ions in the feed water and require a greater feed pressure than NF membranes. NF membranes preferentially remove larger divalent ions or molecules compared to monovalent ions. Thus, NF systems generally exhibit lower energy use and lower operating cost than RO systems. The lower feed pressure required for NF generally translates to a slightly favorable capital cost in relation to RO systems treating the same flow rate. However, the benefits of higher salt rejection and flexibility of systems designed for RO to utilize either NF or RO membranes typically results in utilities favoring RO over marginally lower cost NF systems.

For a typical RO/NF system, membrane elements are mounted into pressure vessels that are arranged in stages, banks, or arrays. The number of stages required depends on specified recovery. Two stages are typically used for recovery less than 80 percent, and three stages are required for higher recovery. RO/NF is a cross flow filtration method, in which only a portion of the feedwater becomes permeate (finished water). The remainder leaves the system as concentrate (brine) that carries away the concentrated material before precipitation or scaling forms on the membrane surface or in the device. Antiscalant is used to control the precipitation of sparingly soluble salts such as calcium carbonate, calcium sulphate, barium sulfate, calcium fluoride, silicon dioxide, etc.

3.3.1 Implementation and Operational Considerations

The recovery of the RO/NF treatment systems depends on the concentrations of the sparingly soluble salts and typically ranges from 75 to 85 percent. Pretreatment requirements include pH depression, antiscalant chemical products to reduce scaling, and cartridge filters to protect the RO/NF membranes from particulates.

The combination of pH depression in the feedwater and the removal of alkalinity through the process results in a low pH (acidic) finished water. Gases pass through NF/RO membranes, resulting in the potential need for removal of hydrogen sulfide and carbon dioxide from the treated water. Post-treatment generally consists of gas stripping through a decarbonation tower and chemical conditioning by addition of a base such as lime or sodium hydroxide (caustic) to raise pH, alkalinity, and hardness to render the water less corrosive. Sometimes a corrosion inhibitor is also added to prevent distribution system corrosion.

A major challenge to implementing centralized NF/RO treatment for PFAS removal is in dealing with the concentrated waste stream generated by the treatment process. Contaminants are rejected into a waste brine stream that is typically around 15 percent by volume of the feedwater (for low salinity feed waters) and 4 to 7 times more concentrated than the raw water fed to the membranes. As a result,

additional raw water is required to achieve the desired finished water capacity, and the waste stream requires disposal. Traditional alternatives for disposal include sending the stream to a downstream water reclamation facility, discharging to surface water, or injection into underground deep wells. However, because of the CERCLA regulations for PFOA and PFOS as discussed in Subsection 3.1.1 and pending effluent limit goals for PFAS, concentrate treatment may be required before disposal using these methods.

3.3.2 Assumptions for Cost Estimation

The costs for RO systems depend on the number of trains, permeate flow, and ancillary processes such as the RO feed tank, low-pressure feed pump, high-pressure feed pump, chemical pretreatment, chemical post-treatment, flush pump/tank, clean-in-place (CIP) system, decarbonation system, building requirements, and brine disposal. The primary process design assumptions for each of these factors are summarized in Table 3-3.

Table 3-3 RO Design Process Assumptions

Sub-System	Parameter	Assumption/Input
RO System Design	Recovery ^(Note 1)	70-85%
	PFAS Rejection	95%
	RO Element Membrane Area	400 ft ²
	Design Flux	15 gallons per foot-squared per day (gfd)
	Redundancy	N+1
	Concentrate Recycle	0%
	Number of Elements per Pressure Vessel	6
	First Stage Pressure Vessel Ratio	4
	Second Stage Pressure Vessel Ratio	2
	Third Stage Pressure Vessel Ratio	1
RO Feed Tank	Hydraulic Detention Time	30 min
RO Low Pressure Feed Pump Sizing	Pump Design TDH	30 ft
RO High Pressure Feed Pump Sizing	Pump Design TDH	350 ft
Chemical Pretreatment ^(Note 2) Antiscalant Chemical	Density	10.01 pounds per gallon (lb/gal)
	Design Dose	3 mg/L
	Storage	30 days

Sub-System	Parameter	Assumption/Input
Chemical Pretreatment ^(Note 2) Sulfuric Acid (98%)	Density	15.26 lb/gal
	Design Dose	30 milligrams per liter (mg/L)
	Storage	30 days
Chemical Post-Treatment ^(Note 2) Caustic (50%) or Liquid Lime	Density	12.78 lb/gal
	Design Dose	45 mg/L
	Storage	30 days
RO Flush Pump Sizing	Flow Rate per Pressure Vessel	30 gpm/1st stage pressure vessel
	Pump Design TDH	140 ft
	Flush Frequency	12 hrs/yr/train
RO Flush Tank Sizing	Volume per Pressure Vessel	7 cubic feet (ft ³)
	Number of Flushes in Tank	2
	Safety Factor	50%
CIP System Sizing	Flush Flow	50 gpm/1st stage pressure vessel
	Time/skid	4 minutes
	CIP Pump TDH	140 ft
	CIP Interval	90 days
	Time/CIP	6 hrs
	CIP Temperature Increase	65 °F
	Heater Losses	10%
Forced Draft Degasifier (decarbonation)	Loading Rate	30 gallons per minute per square foot (gal/min/ft ²)
Building Calculations	RO Equipment Area Factor	2.0
	Unit Area	880 ft ² /mgd

Sub-System	Parameter	Assumption/Input
Brine Disposal		Deep Well Injection
Deep Well Injection	Flow per well	1 mgd

Notes:

1. For RO, the critical design input is percent recovery. A minimum and maximum recovery, but no most likely number, is specified. The minimum and maximum recovery used for the published model outputs are summarized herein. National variability in recovery is included in the model using a Monte Carlo simulation. The details of how this statistical method was employed within the cost modeling tool is described in Section 5.3.
2. Chemical systems include pumps, bulk storage, piping, and containment. No day tanks were included in the estimate.

4.0 Estimating National Occurrence

To estimate the costs of removing PFAS from drinking water nationally, national occurrence must be characterized. In parallel to this project, AWWA funded WITAF 057 to compile an occurrence database for PFAS in drinking water. In addition to data available for UCMR 3, WITAF 057 facilitated the collection of PFAS monitoring data from state databases and integrated these sources into a single data set. PWSs in this database included only active Community Water Systems (CWSs) and active Non-Transient Non-Community Water Systems (NTNCWSs). The inactive and transient non community water systems were eliminated from the dataset. Consecutive systems receiving all water from treated water wholesaler systems were not excluded from the database or from representation in the national cost estimation.

The WITAF 057 dataset consisted of 7,842 PWSs within these categories as compared to the 49,424 PWSs in the Safe Drinking Water Information System (SDWIS). To account for this incomplete occurrence data, the percent of systems impacted by a potential PFAS regulation within each system size category was multiplied by the active number of CWSs or NTNCWSs in EPA's SDWIS system at each size category to estimate the anticipated number of total water systems impacted in each size category. This methodology therefore assumed that existing occurrence data is representative of national occurrence. This assumption is considered conservative given a significant fraction of existing occurrence data came from UCMR 3, where the reporting limits of 20 parts per trillion (ppt) and 40 ppt for PFOA and PFOS, respectively, likely bias existing occurrence data to underrepresent true national occurrence that would be measured using the current reporting limits.

Monitoring data for PFAS compounds in the WITAF 057 database included more than 30 individual compounds but for this work was limited to the six PFAS covered by UCMR 3: PFOS, perfluoroheptanoic acid (PFHpA), perfluorohexane sulfonate (PFHxS), perfluorononanoic acid (PFNA), and perfluorobutane sulfonic acid (PFBS). As compiled, the WITAF 057 database includes all monitoring results under UCMR 3 and various state monitoring programs, which at times includes multiple sample results-specific PFAS at PWS. Reported data were reviewed to ensure correct translation of reporting units; fields were included for PWS identification number, state, number of people served, source type, and system type. These data were analyzed to determine the maximum and average sample results for each PFAS at each PWS in the database.

5.0 Individual Treatment Facility Cost Methodology

The next step in estimating the national costs to remove PFAS from drinking water is to use the occurrence database to estimate the costs associated with treatment for individual PWSs. The following subsections summarize how capital, operating, and life-cycle costs are calculated for each system and for each technology.

The spreadsheet tool developed to perform this task accepts inputs for individual or combined target effluent levels for the six PFAS compounds represented in the database. After both occurrence data and potential regulatory levels are input, Visual Basic scripts within Excel may be initiated by a user to run a Monte Carlo analysis and generate a 10th percentile, 90th percentile, and most probable costs for the capital, operations and maintenance (O&M), and life-cycle costs for a typical entry point to the distribution system (EPTDS) for each PWS in the database. For each system, the tool selects the treatment technology with the lowest life-cycle cost.

This methodology assumes installation of a treatment system at each EPTDS associated with PWSIDs where the maximum PFAS concentration is greater than the potential regulatory level for the corresponding PFAS. The details of individual system and EPTDS cost methodology are described in the following subsections. A list of output fields generated by the cost modeling tool for each PWS with occurrence data is shown in Table 5-1.

Table 5-1 Model Outputs for Individual PWS with Occurrence Data

Model Outputs for Each PWS with Occurrence Data
Design Flow (mgd)
Average Flow (mgd)
Capital Expenditure for GAC Vessels
Annual Operations and Maintenance Costs for GAC Vessels
Life-Cycle Costs for GAC Vessels
Capital Expenditure for GAC Basins
Annual Operations and Maintenance Costs for GAC Basins
Life-Cycle Costs for GAC Basins
Capital Expenditure for Ion Exchange Vessels
Annual Operations and Maintenance Costs for Ion Exchange Vessels
Life-Cycle Costs for Ion Exchange Vessels
Capital Expenditure for Reverse Osmosis
Annual Operations and Maintenance Costs for Reverse Osmosis
Life-Cycle Costs for Reverse Osmosis
Capital Expenditure for Lowest Life-Cycle Cost Technology
Annual Operations and Maintenance Costs for Lowest Life-Cycle Cost Technology

Model Outputs for Each PWS with Occurrence Data
Life-Cycle Costs for Lowest Life-Cycle Cost Technology
10th Percentile Capital Expenditure for Lowest Life-Cycle Cost Technology
10th Percentile Operations and Maintenance Cost for Lowest Life-Cycle Cost Technology
10th Percentile Life-Cycle Cost for Lowest Life-Cycle Cost Technology
90th Percentile Capital Expenditure for Lowest Life-Cycle Cost Technology
90th Percentile Operations and Maintenance Cost for Lowest Life-Cycle Cost Technology
90th Percentile Life-Cycle Cost for Lowest Life-Cycle Cost Technology
Capital Expenditure for Manganese Pretreatment
Annual Operations and Maintenance Costs for Manganese Pretreatment
Life-Cycle Cost for Manganese Pretreatment
Lowest Life-Cycle Cost Treatment Technology

5.1 Determining Design Parameters

5.1.1 Treatment Design Flow Determination

PWS data available in SDWIS do not include water usage data for each PWS and EPTDS. Instead, service population data from SDWIS was used and the average flow for each PWS was assumed based on a per capita per day usage of 150 gallons. While not reflective of each state's dynamics with respect to water usage, this was considered a reasonable number from a national perspective. Peaking factors for different size systems from the EPA's "Cost and Technology Document for Final Groundwater Rule" were used and are shown in Table 5-2. The trend of this dataset was best fit to a power equation to calculate peaking factor as a function of average daily flow as shown on Figure 5-1.

Table 5-2- EPA Peaking Factor for Various Average System Flows

Design Flow (MGD)	Average Flow (MGD)	Peaking Factor	Design Flow (MGD)	Average Flow (MGD)	Peaking Factor
0.007	0.0015	4.7	2	0.77	2.6
0.022	0.0054	4.1	3.5	1.4	2.5
0.037	0.0095	3.9	7	3	2.3
0.091	0.025	3.6	17	7.8	2.2
0.18	0.054	3.3	22	11	2
0.27	0.084	3.2	76	38	2
0.36	0.11	3.3	210	120	1.8
0.68	0.23	3	430	270	1.6
1	0.3	3.3	520	350	1.5

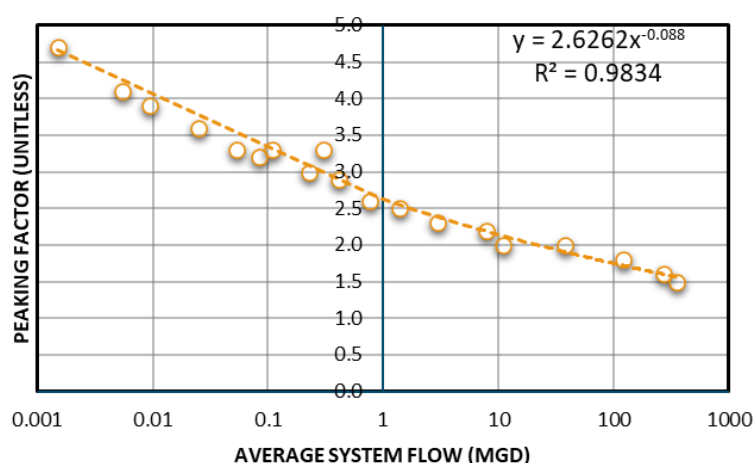


Figure 5-1 Peaking Factor as a Function of Average System Flow

The treatment design flow per EPTDS was determined by Equation 1:

$$\text{Design Flow per EPTDS} = \frac{(\text{number of customers per PWS})(150 \text{ gpcd})(\text{peaking factor})}{\text{EPTDSs per PWS size category}} \quad (1)$$

Where:

$$\text{Peaking factor} = 2.6262(\text{PWS Average Flow})^{-0.088}$$

The estimated number of EPTDS per system size bin is taken from the AWWA Letter to Congressional Budget Office Re: S.1507 - PFAS Release Disclosure Act, dated August 8, 2019, which incorporated updates to information originally collected by EPA's Community Water System Survey. The estimated number of EPTDS by system size bin is summarized in Table 5-3.

Table 5-3 Number of EPTDS as a Function of System Size

Size Category	Population Range	Entry Points/System
1	0-100	2.4
2	101-500	2.0
3	501-1,000	2.1
4	1,001-3,300	1.9
5	3,301-10,000	2.2

Size Category	Population Range	Entry Points/System
6	10,001-50,000	3.1
7	50,001-100,000	4.1
8	100,001-1,000,000	6.6
9	>1,000,001	14.5

5.1.2 Water Quality Considerations Incorporated

5.1.2.1 Influent and Effluent PFAS Levels

For each PWS in the occurrence database, any single PFAS monitoring result above either existing state or potential regulatory limit was assumed to incur a capital expenditure for treatment. Data down to the resolution of each individual source was not considered for this modeling effort; instead, the number of projected water treatment facilities per system was based on the EPTDS factors as summarized in the previous section. Maximum PFAS monitoring data were assumed to compel treatment for the PWS as a whole and, thus, all the projected water treatment facilities. The average PFAS monitoring data were used to estimate long-term costs of removal (annual O&M costs).

The target effluent PFAS levels for treatment was determined as an input percentage of a potential regulatory limit. For example, treatment could be triggered at 80, 90, or 100 percent of the potential regulatory level. For this work, a threshold of 80 percent was used in alignment with previous practice for estimating costs of potential regulations for drinking water, since water systems will target and operate below this threshold to ensure that the limit is not exceeded if the water quality suddenly increases.

5.1.2.2 Other Water Quality Considerations

Other water quality contaminants may impact PFAS treatment performance (and therefore costs), such as TOC and manganese. The longevity of GAC media, IX resin, and membrane operations are significantly affected by the quality of the source. Differences in source water quality parameters not specifically included (e.g., TOC, sulfate, pH, alkalinity, etc.) with pertinence to design or performance were reflected in cost by varying design parameters and treatment system performance according to probability functions using Monte Carlo analysis. This is primarily controlled through variation of the treatment performance factors (e.g., EBCT, surface area loading rate) to reflect less or more challenging water quality characteristics. The methodology for the Monte Carlo Simulation is covered in Section 5.2. Work is in progress to estimate costs associated with removing manganese and will be made available at a later date.

5.2 Monte Carlo Simulation for Design and Performance Variability

Water treatment system design is a practice that evolves non-uniformly across the country. Decisions in the design process are driven in some cases by rigorous engineering standards and in others by regional and geographic considerations, or owner and operator preferences. The result is a landscape of treatment systems across the United States that cannot be effectively modeled by clear and simple rules and frameworks. Additionally, water quality characteristics vary both regionally and locally, and these variations cannot be fully captured in the model with distinct data. These water quality characteristics may improve or hinder performance as well as increase costs to ensure water quality downstream is not altered and complies with other regulations.

To compensate for this uncertainty, Monte Carlo methods were applied to simulate variation and to account for unknowns in major factors influencing design, operation, and, ultimately, cost for PFAS reduction systems. The @RISK Probabilistic Risk Analysis Software by Lumivero, which functions through an Excel add-in, was utilized for the Monte Carlo analysis.

Monte Carlo methods consist of randomizing inputs (e.g., loading rate, GAC media life, RO recovery) according to a defined distribution and number of iterations while calculating the impact to the outputs (e.g., number of vessels, media replacement frequency, cost). As the number of variables undergoing Monte Carlo analysis increases, computer processing power and the time to simulate one scenario both increase exponentially. Thus, Monte Carlo analysis was limited to only major factors considered to exert significant influence on design, performance, and cost of the individual systems. The major factors subjected to Monte Carlo are shown in Table 5-4.

Table 5-4 Major Factors for Monte Carlo Analysis

Parameter	Value
GAC - Pressure	
Surface Loading Rate	
Distribution Type	Triangular
Minimum Value	4 gpm/sf
Maximum Value	10 gpm/sf
Most Likely Value	6 gpm/sf
EBCT	
Distribution Type	Triangular
Minimum Value	10 min
Maximum Value	20 min
Most Likely Value	18 min
GAC - Basins	
Surface Loading Rate	
Distribution Type	Triangular
Minimum Value	4 gpm/sf
Maximum Value	10 gpm/sf
Most Likely Value	4 gpm/sf
EBCT	
Distribution Type	Triangular
Minimum Value	10 min

Parameter	Value
Maximum Value	20 min
Most Likely Value	18 min
GAC Bed Volumes to Breakthrough ^(Note 1)	
Distribution Type	Triangular
Minimum Value	75 percent of prediction
Maximum Value	175 percent of prediction
Most Likely Value	Prediction
IX - Vessels	
Surface Loading Rate	
Distribution Type	Triangular
Minimum Value	5 gpm/sf
Maximum Value	12 gpm/sf
Most Likely Value	8 gpm/sf
EBCT	
Distribution Type	Triangular
Minimum Value	1.5 min
Maximum Value	3 min
Most Likely Value	2 min
IX Bed Volumes to Breakthrough ^(Note 1)	
Distribution Type	Triangular
Minimum Value	75 percent of prediction
Maximum Value	175 percent of prediction
Most Likely Value	Prediction
Reverse Osmosis/Nanofiltration	
Surface Loading Rate	
Distribution Type	Uniform
Minimum Value	70 percent
Maximum Value	85 percent

Notes:

1. GAC and IX Performance (i.e. determination of media life) is described in Section 5.4.1. Predicted value is determined using the generalized logistic function of the Clark model.

With the exception of RO recovery, all Monte Carlo inputs were assigned a triangular distribution. A triangular distribution is a probability distribution where the probability decreases linearly on either side of the most likely value (highest probability) to the minimum and maximum, at which point the probability is zero. Triangular distributions were used where typical industry design values exist. RO recovery was modeled using a uniform distribution where each value between the minimum and maximum have an equivalent probability of occurrence.

The result of the Monte Carlo analysis is a distribution of possible costs for each technology (i.e., low [10th percentile], high [90th percentile], and most probable). For each modeled scenario, each of these costs was stored as a modeled output for each system represented in the occurrence database for use in determining the overall national cost of compliance with the modeled limit.

5.3 Capital Cost Calculation

Capital costs were calculated for each EPTDS of a PWS based on the design flow per EPTDS (refer to Equation 1). The design flow was used for capital costs estimates since equipment should be sized for peak treatment flow rates. Costs were independently calculated for IX, GAC vessels, GAC basins, and RO as described in the following subsections. Capital costs generated for individual systems represent a Class 5 Association for the Advancement of Cost Engineering (AACE) estimate, at approximately 1 to 2 percent maturity level of deliverable definition.

5.3.1 Major Hardware Components

5.3.1.1 GAC Gravity Basins

The major cost components incorporated into the capital cost estimate for this option are the concrete basins themselves, an influent pump station, media for the initial fill, and a building to house the system. The design assumptions for each element are summarized in Subsection 3.1.2.

The concrete basin includes costs for influent and effluent piping, isolation valves, and monitoring instruments. Using the design flow rate and the SLR, a required surface area for filtration is calculated and used to determine the appropriate number of basin cells and anticipated basin dimensions for costing.

Once number and size of basins are calculated, the design flow and specified EBCT is used to determine the volume of media needed. Cost of media was determined by converting volume to mass using an average GAC density of 0.5 g/cc and an average cost per pound of \$1.40. It should be noted that cost changes were not projected into the cost model resulting from increased demand for adsorbent media.

The pump station includes costs for influent pumps, backwash pumps, an influent wetwell, and a backwash recovery basin. The independent design inputs for the influent pumps are total dynamic head (TDH) and total number of pumps. The independent design parameters for backwash pump and backwash recovery basin calculations are backwash loading rate, backwash duration, backwash frequency, and backwash pump TDH. Costs for backwash pumping include a single duty pump and a single standby pump.

The sum of the square footage required for the contactor basins was multiplied by a sizing factor of two to account for the ancillary equipment and space for access and maintenance. Pump station square footage, including all pumps and the wet well, was estimated by benchmarking design flow against previous designs. Building area was assumed to be the sum of contactor facility area (including sizing

factor), pump station area, and backwash recovery basin area (assumed to be indoors). The building cost was assumed to be \$200/sf.

Black & Veatch utilized empirically derived cost curves as a function of size from several decades of infrastructure project design and delivery to estimate cost for these major components. A curve for concrete basins provides cost as a function of square footage. A curve for steel tanks provides costs as a function of volume in gallons, and a curve for pumps provides cost as a function of horsepower.

Installation fees were included at 20 percent for all major equipment components, as summarized in Table 5-5. These cost factors are identical to those for GAC and IX pressure vessels.

Table 5-5 GAC and IX Equipment Installation Cost Factors

Component	Percent Multiplier of Unit Cost
Basins/Pressure Vessels	20%
Influent Pumps	20%
Backwash Pumps	20%
Influent Wetwell	20%
Backwash Recovery Basin	20%

5.3.1.2 GAC, IX and Manganese Pretreatment Pressure Vessels

Capital equipment costs were calculated using the total contactor footprint, contactor building footprint, and media volume required. Capital costs were calculated for the ancillary pump stations using the building footprint, number and size of influent pumps, backwash pumps, influent wetwell, and backwash recovery basin. The model incorporated a building cost of \$200/ft². The installation fees for the various components are the same as those summarized in Table 5-6.

Calculated capital cost for manganese pretreatment for each system was considered a stand-alone output and was not included in the capital, operational, or life-cycle cost outputs for PFAS treatment.

5.3.1.3 Reverse Osmosis

Capital costs were calculated for the RO system and building, low- and high-pressure feed pumps and their associated building, storage tanks, cartridge filters, chemical treatment system, decarbonation system, and brine disposal. The model incorporated a building cost of \$200/ft². The installation fees for the various components are summarized in Table 5-6.

Table 5-6 RO Equipment Installation Cost Factors

System	Component	Percent Multiplier of Unit Cost
Storage Tanks	RO Feed Tank	15%
	CIP Tank	15%
	CIP Neutralization Tank	15%
	Flush Tank	15%
Pump Stations	RO Low Pressure Feed Pumps	25%
	RO High Pressure Feed Pumps	25%
	CIP Pumps	20%
	Flush Pumps	20%
Cartridge Filter	RO Feed Cartridge Filter	20%
	CIP Cartridge Filter	20%
Chemical Feed Systems	Antiscalant	20%
	Sulfuric Acid	20%
	Caustic/Liquid Lime	20%
Decarbonation System	All related equipment	20%

5.3.1.4 Additional Capital Costs

In addition to equipment costs, the capital costs for GAC, IX, RO, and manganese pretreatment included additional project costs (site work, yard piping, electrical, and instrumentation and controls), contractor markup costs, and non-construction costs. The multipliers used for each of these factors are summarized in Table 5-7.

Table 5-7 Additional Capital Cost Assumptions

Additional Capital Costs	Description	Percent Multiplier of Total Equipment Costs
Additional Project Costs	Site Work	8.0%
	Yard Piping	9.0%
	Electrical	10.0%
	Instrumentation & Controls	2.5%

Additional Capital Costs	Description	Percent Multiplier of Total Equipment Costs
Contractor Markup Costs	Overhead	7.0%
	Profit	10.0%
	Mobilization/Bonds/Insurance	3.0%
	Contingency	4.0%
Non-Construction Costs	Permitting	1.0%
	Engineering	8.0%
	Legal/Administration	0.5%
	Construction Services	7.0%
	Commissioning/Startup	3.0%
	Contingency	30.0%

5.4 Operating Cost Calculation

The operational costs for GAC, IX, and RO were calculated using the average flow rate for each EPTDS, as represented by the average flow per water system divided by the number of EPTDS. Whereas capital costs were driven by maximum PFAS levels, the operating costs incurred were driven by the average influent PFAS concentrations to reflect long-term operating conditions. The tool allows entry of a treatment goal expressed as a percent of the potential regulatory limit, and the resulting target concentration serves as the effluent concentration trigger for replacement of media. This target may be expressed either as a concentration of a single PFAS compound or as a combination of compounds.

Operating costs that were considered for this work included replacement costs (using the calculated bed volumes to breakthrough or media replacement frequency), power consumption in the pumps and buildings, maintenance costs, waste disposal, and labor costs. Analytical monitoring costs were not included in the life-cycle cost calculations. Table 5-8 provides an overview of the O&M cost assumptions.

Table 5-8 O&M Cost Assumptions

O&M Category	Description	Value
Media Replacement	GAC Virgin Media ^(Note 1)	\$1.40/lb
	GAC Reactivated Media	\$1.20/lb
	IX Resin	\$240/ft ³ (\$3.70/lb @ apparent density of 1.05 g/cc)
Membrane Replacement	Membrane Cost	\$600/element

O&M Category	Description	Value
Power	Unit Cost	\$0.10/kilowatt-hour (kWh)
	Unit Building Power Usage	19.5 kWh/ft ² /yr
	Building Utilization Factor	365 days/year
Maintenance	Installed Equipment	1.5% Percent Multiplier of Capital Costs
	Structures and Facilities	1.0% Percent Multiplier of Capital Costs
Waste Disposal	Incineration ^(Note 2)	\$720/ton
	GAC Density	0.5 g/cc
	IX Density	1.05 g/cc
	Mn Adsorptive Media Density	1.8 g/cc
Chemical Consumption Costs	Antiscalant	\$15.00/gal
	Sulfuric Acid	\$2.50/gal
	Caustic	\$4.50/gal
Labor	Operator Rate	\$30/hr
	Admin Rate	\$25/hr
	Number of Valves	3 per vessel/basin, 2 per pump (additional requirements for RO system include 2 per cartridge filter, 3 per decarbonation system, and 2 per tank)
	Number of Instruments	2 per vessel/basin, 1 per pump (additional requirements for RO system include 2 per cartridge filter, 2 per decarbonation system, and 1 per tank)
	Record Keeping and Sampling	5 minutes per day per instrument
	Pump Operation (adjustments)	5 minutes per day per pump
	Valve Adjustments	5 minutes per week per valve
	GAC Contactor Maintenance	1 hour per week per vessel/basin
	IX Replacement	16 hours per bed volume
	Cartridge Filters	12 hours per year per cartridge filter
	RO Membrane Process Labor	120 hours per week

Notes:

1. Life-cycle cost factors were chosen to match the EPA's standard practice for estimating life-cycle cost
2. Spent GAC media and IX resin was assumed to be incinerated because of the unknown viability of GAC media reactivation under CERCLA. Replacement costs were therefore assumed to be virgin media.

5.4.1 Estimation of Media Life and Disposal

The generalized logistic function of the Clark model (Clark, 1987), represented in Equation 2, was the basis for calculations for estimation of media life for both GAC and IX. While more rigorous techniques exist for modeling adsorption, Clark's model was utilized for its relative simplicity and accuracy.

$$C = \frac{C_o^{n-1}}{1 + B e^{-r't}} \quad (2)$$

Where:

C_o is the influent contaminant concentration, C is the concentration of a given contaminant at time t , n is the inverse of the slope of the Freundlich isotherm, and r' and B are constants. Rearranging the equation above to:

$$\ln \left[\left(\frac{C_o}{C} \right)^{n-1} - 1 \right] = -r't + \ln B$$

r' and B can be solved for from the slope and intercept of the plot of $\ln[(C_o/C)^{1/n}-1]$ versus time. If a constant flow is assumed, the number of bed volumes becomes directly proportional to time, allowing these relationships to be expressed as a function of bed volumes treated rather than time. B , n , and r' values utilized for GAC and IX are expressed in Table 5-9. The values utilized for GAC were derived from data collected during a Black & Veatch GAC pilot study for CFPUA. The values utilized for IX were derived partially from data collected during a Black & Veatch IX pilot study for CFPUA and partially from data collected during an IX pilot study for La Habra Height County Water District (LHHWCWD).

Table 5-9 Values Variables in Modeled Bed Life

Media	Constant	PFOA	PFOS	PFHxS	PFNA	PFHpA	PFBS
IX ^(Note 1)	n	1.25	1.25	1.25	1.25	1.25	1.25
	B	4.8	1.8	1.4	6.0	3.6	3.2
	$-r'$	-2.55E-05	-3.33E-06	-3.40E-06	-1.70E-05	-2.62E-05	-6.23E-06
GAC	n	1.49	1.54	3.23	1.79	1.67	1.56
	B	141.7	15.8	666.0	49.1	49.1	11.3
	$-r'$	-6.21E-04	-2.07E-04	-3.77E-04	-3.46E-04	-4.81E-04	-3.28E-04

Notes:

- Parameters for PFOA and PFHpA were derived from the CFPUA data set. Parameters for PFBS, PFHxS, and PFOS were derived from the LHHWCWD data set. Parameters for PFNA were estimated by extrapolating data for PFOA and PFHpA because insufficient pilot data were available to support a curve fit determination.

For each system with occurrence data, C was calculated for each PFAS compound at a specified bed volume increment. Increments of 250 bed volumes up to a maximum of 40,000 were calculated for GAC. Increments of 5,000 bed volumes up to a maximum of 800,000 were calculated for IX. The number of bed volumes at which C exceeded the specified target replacement concentration was determined, and the number of bed volumes for the first contaminant to breach its target concentration was used to calculate media replacement frequency. The number of bed volumes treated before the first contaminant exceeded the target concentration was subjected to Monte Carlo variability as described in Section 5.2.

5.5 Life-Cycle Costs

The model determines 20-year life-cycle costs, which combines the capital costs and annual operating and maintenance costs. Life-cycle costs provide a means of comparing the costs of alternative technologies over the life cycle of the equipment. The life-cycle costs were calculated assuming a 20-year lifespan and a discount rate of 7 percent. While typical practice to determine life-cycle costs may incorporate other factors, such as the inflation and loan interest, the discount rate was used to match the approach that is standard practice for the EPA in promulgating national primary drinking water regulations.

6.0 National Cost Assessment Methodology

The conceptual framework for assessing the national costs is as follows:

- Assess capital, annual O&M, and life-cycle costs for EPTDSs in every water system for which potential regulatory limits for PFAS may require treatment.
- Average the costs by system size category.
- Multiply those average costs by the total anticipated number of systems impacted in each system size category based on the percentage of systems in the database impacted by a proposed regulatory limit for PFAS.

The following subsections summarize the process and details associated with the national cost estimation methodology.

6.1 Estimating National Costs Using Model Outputs

Using the treatment facility costs for systems from the occurrence database, the costs were binned by system size, and average EPTDS costs per system size bin were calculated. Using the occurrence database, the number of impacted systems per size category was calculated, and the corresponding percent of the systems in the database was determined. To estimate the number of impacted systems nationally, the percentage of impacted systems was multiplied by the total number of systems in SDWIS for each size category.

The estimated number of impacted systems per size category multiplied by the average cost per EPTDS and the assumed number of entry points yields the total cost per size category. The sum of all costs per size category yields the estimated national cost of removing PFAS to a potential regulatory limit. A summary output is included in Table 6-1 which displays the costs associated with a potential regulatory maximum contaminant level (MCL) of 4 ppt for PFOA and 4 ppt for PFOS.

Table 6-1 Example Summary Cost Table for Potential Regulatory MCL of 4 ppt PFOA and PFOS

Size Category	PWSs In Database (Note 1)	Impacted PWSs in Database	% Impacted in Database	Active PWSs in SDWIS (Note 1)	Nationally Impacted PWSs	EPTDSs per PWS	Nationally Impacted EPTDSs	Average Capital Cost per EPTDS	National Cost
1	1298	242	19%	11,622	2,167	2.4	5201	\$ 800,000	\$ 4,160,640,000
2	1080	177	16%	15,064	2,469	2	4938	\$ 1,700,000	\$ 8,394,600,000
3	341	39	11%	5,324	609	2.1	1279	\$ 2,200,000	\$ 2,813,580,000
4	427	46	11%	7,964	858	1.9	1630	\$ 2,900,000	\$ 4,727,580,000
5	660	103	16%	5,002	781	2.2	1718	\$ 4,800,000	\$ 8,247,360,000
6	3007	224	7%	3,419	255	3.1	791	\$ 7,900,000	\$ 6,244,950,000
7	579	64	11%	582	64	4.1	262	\$ 11,100,000	\$ 2,912,640,000
8	425	72	17%	422	71	6.6	469	\$ 16,800,000	\$ 7,872,480,000
9	25	4	16%	25	4	14.5	58	\$ 35,000,000	\$ 2,030,000,000
Total	7,842	971	12%	49,424	7,278	N/A	N/A	N/A	\$47,403,830,000

Note 1: The current analysis accounts for only those costs associated with community water systems (CWSs), which are PWSs that serve more than 25 people for more than 6 months of the year.

6.2 Accounting for State Level Regulatory Costs

The model includes consideration of state regulatory actions that may have driven PWSs to remove PFAS already. Consideration of state regulatory actions is necessary to characterize the compliance costs of a potential NPDWR for PFAS. All state regulations incorporated into modeled cost output are shown in Table 6-2

Table 6-2 State Maximum Contaminant Levels Modeled for State Regulatory Cost Estimate

States	Type	PFOA	PFOS	PFHxS	PFNA	PFHpA	PFBS
Connecticut	Individual		10				
Delaware	Individual	14	21				
Delaware	Combined	17	17				
Massachusetts	Combined	20	20	20	20	20	
Michigan	Individual	8	16	51	6		
New Hampshire	Individual	12	15	18	11		
New Jersey	Individual	14	13		13		
New York	Individual	10	10				
Ohio	Combined	70	70				
Ohio	Individual			140	21		140,000
Vermont	Individual	20	20	20	20	20	
Wisconsin	Combined	70	70				

To differentiate federal regulatory costs from costs incurred because of existing state regulations, the cost tool includes an input sheet for all existing state MCLs as either individual limits or group totals. The Visual Basic Script references both the state MCLs and the projected federal MCLs. In the absence of a federal regulation (or if the state MCL is more stringent than the federal MCL), the cost tool generates costs for treatment to comply with existing state MCLs. An example of this is shown in Table 6-3, which displays treatment costs incurred as a result of state regulations.

Table 6-3 Summary of Estimated Costs Associated with State PFAS MCLs

PWS Size Category	Population Range	% Impacted	Average CAPEX/PWS	Average O&M/PWS	Annualized PWS Cost	Estimated Number of Impacted PWSs	Annualized Total Cost	Present Value of Lifecycle Cost
1	<100	7%	\$1,920,000	\$48,000	\$229,000	761	\$174,269,000	\$1,846,200,000
2	101-500	5%	\$3,400,000	\$60,000	\$381,000	809	\$308,229,000	\$3,265,400,000
3	501-1,100	5%	\$4,620,000	\$63,000	\$499,000	250	\$124,750,000	\$1,321,600,000
4	1,001-3,300	1%	\$5,510,000	\$76,000	\$596,000	112	\$66,752,000	\$707,200,000
5	3,301-10,000	5%	\$10,560,000	\$132,000	\$1,129,000	243	\$274,347,000	\$2,906,400,000
6	10,001-50,000	3%	\$24,180,000	\$310,000	\$2,592,000	99	\$256,608,000	\$2,718,500,000
7	50,001-100,000	1%	\$43,050,000	\$594,500	\$4,658,000	6	\$27,948,000	\$296,100,000
8	100,001-1,000,000	3%	\$98,340,000	\$1,848,000	\$11,131,000	11	\$122,441,000	\$1,297,100,000
9	>1,000,000	4%	\$407,450,000	\$8,555,000	\$47,015,000	1	\$47,015,000	\$498,100,000
All Systems		4%				2292	\$1,402,359,000	\$14,856,600,000

Note: National costs for various potential MCLs are summarized in Section 7.1. The differentials between state costs in this table and various total national costs represent the cost associated with any modeled NPDWR.

7.0 Summary of Results

A summary of the cost model results for various potential federal MCL alternatives on the national and household level is presented in this section.

7.1 National Cost Estimates

The national cost modeling tool was used to evaluate both the national financial burdens on communities from PFAS drinking water contamination (the National Burden) and the costs for water systems to comply with a potential NPDWR for PFAS (NPDWR Compliance Costs).

The National Burden is reflective of the total, cumulative impact to water systems and communities across the United States from PFAS contamination of drinking water. It is calculated by estimating the drinking water PFAS treatment costs associated with the number of systems with PFAS occurrence data above the target limit. The National Burden assumes the same target limit for water systems across all states and includes systems in states with existing drinking water regulations for PFAS. The NPDWR Compliance Costs are determined by estimating the national financial burden and excluding costs for systems already triggered into treatment by existing drinking water regulations at the state level. The difference between the National Burden and the NPDWR Compliance Costs is therefore calculated using the data presented in Table 6-3.

The National Burden and NPDWR Compliance Costs were estimated for three different scenarios. The first scenario is based on a target PFOA and PFOS level of 4 ppt each. The second scenario is based on target concentrations for PFOA, PFOS, PFHxS, PFHpA, and PFNA (collectively referred to as “long-chain PFAS”) of 4 ppt each. The third scenario is based on target concentrations for the same long-chain PFAS compounds of 10 ppt each.

An overview of the present value of the life-cycle cost for the National Burden and NPDWR compliance cost for each of these scenarios is displayed on Figure 7-1.

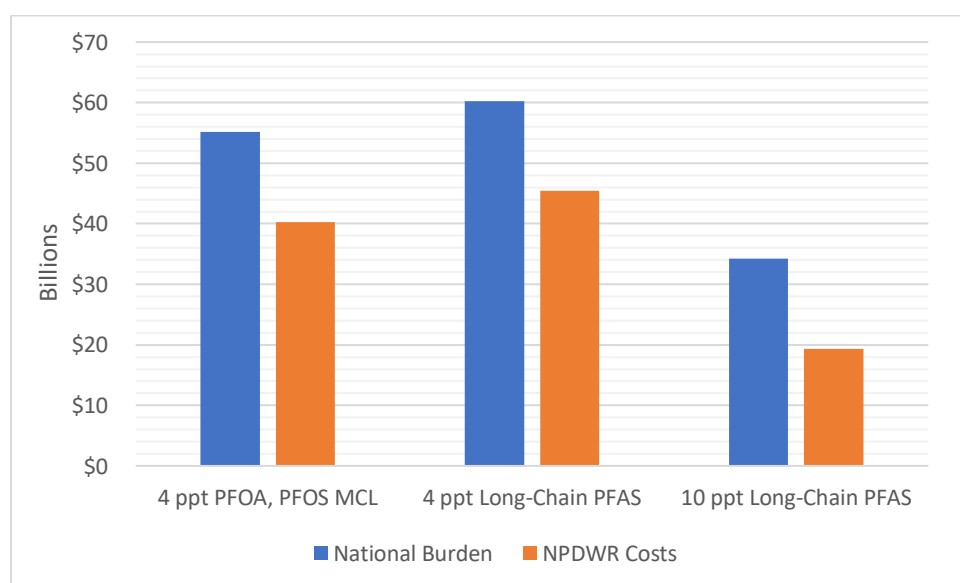


Figure 7-1 Summary of Present Value of Life-Cycle Costs for National Burdens and NPDWR Compliance Costs for Each Scenario

Annualized costs were also calculated using Formula 3. An overview of the National Burden and NPDWR Compliance Annualized Cost for each of these scenarios is presented on Figure 7-2.

$$\text{Annualized Costs} = \frac{(\text{Capital Costs})(\text{Discount Rate})}{1 - (1 + \text{Discount Rate})^{-n}} + \text{Annual Operating Costs} \quad (3)$$

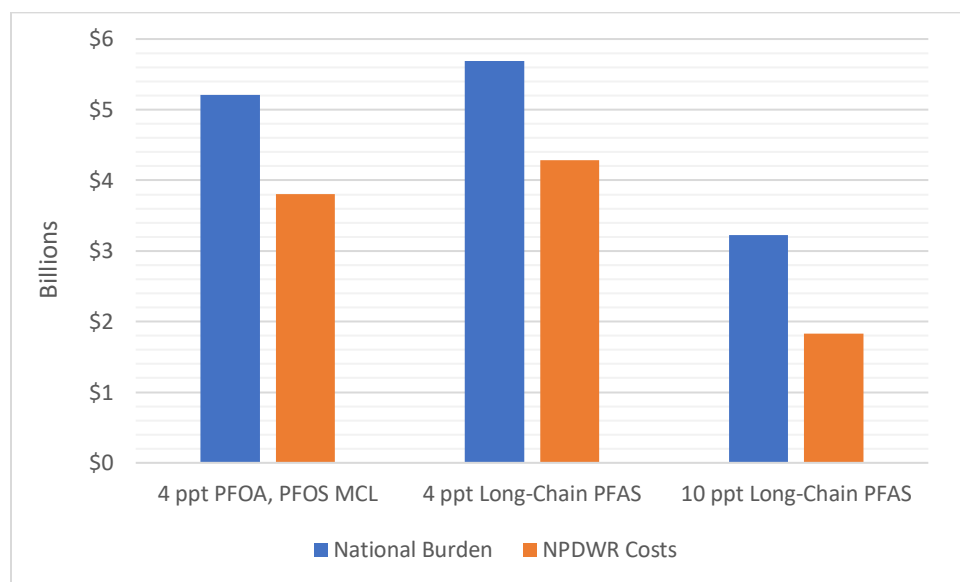


Figure 7-2 Summary of Annualized Costs for National Burdens and NPDWR Compliance for Each Scenario

A more detailed breakdown of these costs by system size is presented in Appendix A.

7.2 Household Financial Impacts

As part of this analysis, the annual financial impacts to individual households from costs associated with the installation and operation of drinking water treatment facilities for PFAS were determined. The financial impacts to individual households will vary by specific PFAS levels, system size, and other factors. Additionally, the impacts to individual households arising from a potential NPDWR will differ depending on whether there is an existing state regulation for PFAS in drinking water. Table 7-1 shows the individual household impacts as a function of system size for each of the three scenarios discussed in Section 7.1. These household level cost impacts are based on the annualized costs for each system size and an average of 2.6 persons per household and incorporate estimated average service populations for each size category based on SDWIS data. The range of household level costs in the table is reflective of communities where new treatment facilities will need to be installed and operated.

Table 7-1 Annual Costs to Household for Removing PFAS from Drinking Water

PWS Size Category	Population Range	Average Service Population	Approximate Range of Costs per Household
1	<100	59	\$10,090 - \$11,150
2	101-500	245	\$4,045 - \$4,245
3	501-1,100	736	\$1,765 - \$1,910
4	1,001-3,300	1,939	\$765 - \$800
5	3,301-10,000	5,696	\$525 - \$545
6	10,001-50,000	20,613	\$335 - \$340
7	50,001-100,000	67,417	\$185 - \$195
8	100,001-1,000,000	204, 194	\$145 - \$160
9	>1,000,000	1,700,000	\$80 - \$105

Appendix A. Modeled Cost Comparison Tables

Table A-1 National Cost Burden by System Size for 4 ppt PFOA, PFOS

PWS Size Category	Population Range	4 ppt PFOA, PFOS MCL			Estimated Number of Impacted PWSs	Annualized National Cost
		Average CAPEX/PWS	Average O&M/PWS	Annualized PWS Cost		
1	<100	\$1,920,000	\$72,000	\$253,000	2167	\$548,251,000
2	101-500	\$3,400,000	\$60,000	\$381,000	2469	\$940,689,000
3	501-1,100	\$4,620,000	\$63,000	\$499,000	609	\$303,891,000
4	1,001-3,300	\$5,510,000	\$57,000	\$577,000	858	\$495,066,000
5	3,301-10,000	\$10,560,000	\$176,000	\$1,173,000	781	\$916,113,000
6	10,001-50,000	\$24,490,000	\$372,000	\$2,684,000	255	\$684,420,000
7	50,001-100,000	\$45,510,000	\$512,500	\$4,808,000	64	\$307,712,000
8	100,001-1,000,000	\$110,880,000	\$891,000	\$11,357,000	71	\$806,347,000
9	>1,000,000	\$507,500,000	\$3,045,000	\$50,949,000	4	\$203,796,000
	All Systems				7278	\$5,206,285,000

Table A-2 National Burden Costs per System Size for 4 ppt Long-Chain PFAS

PWS Size Category	Population Range	4 ppt Long-Chain PFAS			Estimated Number of Impacted PWSs	Annualized National Cost
		Average CAPEX/PWS	Average O&M/PWS	Annualized PWS Cost		
1	<100	\$1,920,000	\$48,000	\$229,000	2265	\$518,685,000
2	101-500	\$3,400,000	\$60,000	\$381,000	2553	\$972,693,000
3	501-1,100	\$4,830,000	\$84,000	\$540,000	640	\$345,600,000
4	1,001-3,300	\$5,510,000	\$76,000	\$596,000	933	\$556,068,000
5	3,301-10,000	\$11,000,000	\$154,000	\$1,192,000	811	\$966,712,000
6	10,001-50,000	\$24,800,000	\$372,000	\$2,713,000	282	\$765,066,000
7	50,001-100,000	\$45,100,000	\$779,000	\$5,036,000	68	\$342,448,000
8	100,001-1,000,000	\$110,880,000	\$2,277,000	\$12,743,000	75	\$955,725,000
9	>1,000,000	\$508,950,000	\$18,487,500	\$66,529,000	4	\$266,116,000
	All Systems				7631	\$5,689,113,000

Table A-3 National Burden Costs by System Size for 10 ppt Long-Chain PFAS

PWS Size Category	Population Range	10 ppt Long-Chain PFAS			Estimated Number of Impacted PWSs	Annualized National Cost
		Average CAPEX/PWS	Average O&M/PWS	Annualized PWS Cost		
1	<100	\$1,920,000	\$48,000	\$229,000	1039	\$237,931,000
2	101-500	\$3,600,000	\$60,000	\$400,000	1032	\$412,800,000
3	501-1,100	\$4,620,000	\$84,000	\$520,000	328	\$170,560,000
4	1,001-3,300	\$5,320,000	\$66,500	\$569,000	261	\$148,509,000
5	3,301-10,000	\$10,560,000	\$154,000	\$1,151,000	455	\$523,705,000
6	10,001-50,000	\$24,490,000	\$341,000	\$2,653,000	205	\$543,865,000
7	50,001-100,000	\$44,690,000	\$656,000	\$4,874,000	47	\$229,078,000
8	100,001-1,000,000	\$112,200,000	\$1,848,000	\$12,439,000	61	\$758,779,000
9	>1,000,000	\$545,200,000	\$15,950,000	\$67,413,000	3	\$202,239,000
	All Systems				3431	\$3,227,466,000

Dear NDWAC Members,

Attached please find comments (and Exhibits) filed by 36 organizations regarding the proposed National Primary Drinking Water Regulations setting Maximum Contaminant Levels (MCLs) for 6 PFAS.

While the attached comments address many of the issues raised by the EPA proposal in detail, I wanted to respond to comments made to NDWAC by some water utility representatives at the NDWAC meeting today. Specifically, the NDWAC should be aware of the substantially inflated costs of the proposed EPA MCLs presented to NDWAC by those representatives. The attached comments, and Exhibit C specifically, include a detailed analysis and comparison of EPA's and AWWA's (Black & Veatch's) cost estimates completed by Elin Betanzo of Safe Water Engineering (SWE). The SWE analysis concludes (on page 6, emphasis added):

The evidence provided in this memo demonstrates that the EPA cost estimate is robust. While there are several items that could not be directly compared to the AWWA cost model, there is no evidence that EPA is consistently underestimating occurrence or costs. According to calculations shown in Appendix G, if AWWA's O&M costs are accurate (\$30,000-125,000 per entry point), this could mean that EPA's O&M cost estimates (\$27,000-2,515,000) are larger than necessary and may be lower in practice. If Michigan's rate of non-treatment options is relevant nationwide, the total EPA cost would fall even further.

The EPA cost estimate of \$658.5 million appears to be the more realistic result based on the calculations and findings presented here.

While professional judgement must be used in applying cost modeling assumptions, it appears that several of the assumptions in the AWWA cost model are too conservative. These assumptions consistently result in higher capital and O&M costs for treatment, especially for systems serving fewer than 10,000. As shown in Table 2, the analyses presented here demonstrate **the AWWA estimate includes at least \$2.6 billion in excess costs**. Many of these overestimates have cascading effects that could not be modeled with available data. The cumulative impact of these corrections is likely even larger than estimated here. **Subtracting the excess costs from AWWA's total estimate would result in a maximum annual cost of \$1.2 billion for treatment installation and O&M.**

Overall, the comments of the 36 organizations conclude:

The Proposed Rule is an important step forward. EPA correctly found that there is no safe exposure level for many PFAS, including PFOA and PFOS, and it proposed maximum contaminant levels ("MCLs") for PFOA and PFOS that are readily achievable using existing treatment technologies. EPA also recognized the serious health risks associated with exposures to mixtures of GenX, PFNA, PFBS, and PFHxS—PFAS that are frequently found in the same drinking water supplies—and it proposed an MCL that is designed to protect people who are exposed to those contaminants individually or in combination. EPA's proposed Maximum Contaminant Level Goals ("MCLGs") and MCLs are supported by an extensive factual record and are required by the SDWA. EPA must resist efforts to weaken those levels and diminish the rule's protections.

At the same time, EPA must revise aspects of its Proposed Rule that would limit the rule's reach and undermine its effectiveness. First, EPA should update its Health Based Water Concentrations ("HBWCs")—the toxicity values that EPA uses to calculate the drinking water limits—for PFBS, GenX, PFNA, and PFHxS to address the dangers those chemicals pose to infants, children, and other higher-risk populations. Second, while EPA conducted an extensive economic analysis and found that the Proposed Rule's benefits outweigh its costs, that analysis understates the benefits of reduced PFAS exposures and should be expanded to better account for health benefits that EPA has acknowledged but has not yet quantified or monetized. Third, when determining compliance with the new drinking water standards, EPA should consider all monitoring results with detectable PFAS rather than treating samples with lower but still harmful levels of PFAS as though they were PFAS-free. Fourth, EPA should maintain the minimum requirement of quarterly PFAS monitoring for all water systems with prior PFAS detections and should not permit such systems to evade further detections and necessary treatment by monitoring just once or twice every three years. Finally, EPA should mandate public notification of MCL violations within 24 hours, as is required for all violations that may cause serious, short-term health effects.

With more than half of the nation drinking PFAS-contaminated water, the Proposed Rule is urgently needed and should be finalized expeditiously, with the revisions outlined below. But as EPA Administrator Michael Regan acknowledged, "[w]hile this proposal is a step forward, there's no doubt there's more work left to do." In a separate rulemaking, EPA should establish drinking water standards for the PFAS that are not covered by EPA's current proposal, including class-based standards that address the harms from additional PFAS mixtures. We urge EPA to use the full extent of its SDWA authority to ensure that no one suffers the harms associated with PFAS-contaminated drinking water.

Thank you for your attention to this important matter.

ERIK D. OLSON

Senior Strategic Director for Health & Food

HEALTHY PEOPLE & THRIVING COMMUNITIES PROGRAM
NATURAL RESOURCES
DEFENSE COUNCIL

May 30, 2023

Via Regulations.gov

Assistant Administrator Radhika Fox
Office of Water
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, DC 20460

Re: PFAS National Primary Drinking Water Regulation Rulemaking, Docket No. EPA–HQ–OW–2022–0114

Dear Assistant Administrator Fox:

The undersigned 36 organizations submit these comments on EPA’s proposed National Primary Drinking Water Regulation for six per- and polyfluoroalkyl substances (“PFAS”) (the “Proposed Rule”).¹ Our organizations include communities with PFAS-contaminated drinking water, scientists who study the harms associated with PFAS, and longtime advocates for health-protective PFAS drinking water standards.

We strongly support EPA’s issuance of PFAS drinking water standards, which are a critical and long overdue step to address a public health crisis that threatens the health and lives of hundreds of millions of people in the United States. For decades, communities across the country have been drinking tap water contaminated with PFAS, a large class of long-lasting and dangerous chemicals. People in those communities have lost parents, children, and other loved ones to cancer, liver and heart disease, and other diseases associated with PFAS. The longer that EPA waits to establish federal drinking water standards, the more people will be exposed, in violation of the Safe Drinking Water Act (“SDWA”) mandate to reduce the harmful effects from drinking water contaminants as much as feasible.

The Proposed Rule is an important step forward. EPA correctly found that there is no safe exposure level for many PFAS, including PFOA and PFOS, and it proposed maximum contaminant levels (“MCLs”) for PFOA and PFOS that are readily achievable using existing treatment technologies. EPA also recognized the serious health risks associated with exposures to mixtures of GenX, PFNA, PFBS, and PFHxS—PFAS that are frequently found in the same drinking water supplies—and it proposed an MCL that is designed to protect people who are exposed to those contaminants individually or in combination. EPA’s proposed Maximum Contaminant Level Goals (“MCLGs”) and MCLs are supported by an extensive factual record

¹ Preliminary Regulatory Determination and Proposed Rule, PFAS National Primary Drinking Water Regulation Rulemaking, 88 Fed. Reg. 18,638 (Mar. 29, 2023). The six PFAS covered by the Proposed Rule are perfluorooctanoic acid (“PFOA”), perfluorooctane sulfonic acid (“PFOS”), perfluorohexane sulfonic acid (“PFHxS”), hexafluoropropylene oxide dimer acid and its ammonium salt (“GenX”), perfluorononanoic acid (“PFNA”), and perfluorobutane sulfonic acid (“PFBS”) (collectively, the “Six PFAS”).

and are required by the SDWA. EPA must resist efforts to weaken those levels and diminish the rule's protections.

At the same time, EPA must revise aspects of its Proposed Rule that would limit the rule's reach and undermine its effectiveness. First, EPA should update its Health Based Water Concentrations ("HBWCs")—the toxicity values that EPA uses to calculate the drinking water limits—for PFBS, GenX, PFNA, and PFHxS to address the dangers those chemicals pose to infants, children, and other higher-risk populations. Second, while EPA conducted an extensive economic analysis and found that the Proposed Rule's benefits outweigh its costs, that analysis understates the benefits of reduced PFAS exposures and should be expanded to better account for health benefits that EPA has acknowledged but has not yet quantified or monetized. Third, when determining compliance with the new drinking water standards, EPA should consider all monitoring results with detectable PFAS rather than treating samples with lower but still harmful levels of PFAS as though they were PFAS-free. Fourth, EPA should maintain the minimum requirement of quarterly PFAS monitoring for all water systems with prior PFAS detections and should not permit such systems to evade further detections and necessary treatment by monitoring just once or twice every three years. Finally, EPA should mandate public notification of MCL violations within 24 hours, as is required for all violations that may cause serious, short-term health effects.

With more than half of the nation drinking PFAS-contaminated water,² the Proposed Rule is urgently needed and should be finalized expeditiously, with the revisions outlined below. But as EPA Administrator Michael Regan acknowledged, "[w]hile this proposal is a step forward, there's no doubt there's more work left to do."³ In a separate rulemaking, EPA should establish drinking water standards for the PFAS that are not covered by EPA's current proposal, including class-based standards that address the harms from additional PFAS mixtures. We urge EPA to use the full extent of its SDWA authority to ensure that no one suffers the harms associated with PFAS-contaminated drinking water.

I. EPA Must Expeditiously Finalize Health-Protective PFAS Drinking Water Standards

A. EPA Should Move Quickly to Finalize PFAS MCLs

As described by Dr. Patrick Breyse, the former Director of the Center for Environmental Health in the U.S. Centers for Disease Control and Prevention, PFAS present "one of the most

² David Q. Andrews and Olga V. Naidenko, *Population-Wide Exposure to Per- and Polyfluoroalkyl Substances from Drinking Water in the United States*, Env't. Sci. & Tech. Letters 931 (2020), <https://doi.org/10.1021/acs.estlett.0c00713>.

³ Michael Regan, Admin., EPA, Remarks for the PFAS Drinking Water Standard Event, As Prepared for Delivery (Mar. 14, 2023), <https://www.epa.gov/speeches/administrator-michael-regan-remarks-pfas-drinking-water-standard-event-prepared-delivery>.

seminal public health challenges for the next decades.”⁴ When Dr. Breyse made that statement in October 2017, he estimated that “up to 10 million Americans” could be drinking water with unsafe levels of PFAS.⁵ Today, that estimate is approximately 200 million.⁶ Impacted communities experience increased risks of cancer and other severe effects, with some exposed to PFAS levels that are tens of thousands of times higher than the levels that EPA has already determined present serious health risks.⁷

Because of their chemical structure, PFAS are highly persistent, “indicat[ing] the potential for long-lasting environmental and human exposure ... that is difficult to control and reverse.”⁸ Many PFAS also bioaccumulate in animals and people, with low-level exposures building up in people’s bodies and causing serious harm. More than 98% of people tested in the United States have PFAS in their blood.⁹ Communities of color often experience the greatest PFAS exposures and risks; a recent study found that the “watersheds serving higher proportions of Hispanic/Latino and non-Hispanic Black populations had significantly greater odds of containing PFAS sources.”¹⁰

⁴ Pat Rizzuto et al., *CDC Sounds Alarm on Chemical Contamination in Drinking Water*, Bloomberg Env’t (Oct. 17, 2017), <https://news.bloomberglaw.com/environment-and-energy/cdc-sounds-alarm-on-chemical-contamination-in-drinking-water>.

⁵ *Id.*

⁶ See Andrews & Naidenko (2020); Annie Sneed, *Forever Chemicals Are Widespread in U.S. Drinking Water*, Sci. Am. (Jan 22, 2021), <https://www.scientificamerican.com/article/forever-chemicals-are-widespread-in-u-s-drinking-water/>.

⁷ See, e.g., EPA, *Hoosick Falls, New York, Drinking Water and Groundwater Contamination, Frequently Asked Questions* (Jan. 12, 2016), https://www.epa.gov/sites/default/files/2016-01/documents/hoosickfalls_faqs.pdf (describing PFOA detections of 600 parts-per-trillion (ppt) in drinking water in Hoosick Falls, New York—150,000 times higher than EPA’s health advisory level of .004 ppt); WRAL News, *Report: Extremely High Levels of GenX-like Chemicals in Wilmington Drinking Water for Years* (Oct. 9, 2019), <https://www.wral.com/story/report-extremely-high-levels-of-genx-like-chemicals-in-wilmington-drinking-water-for-years/18688129/> (describing GenX detections of 130,000 ppt in the Cape Fear River near Wilmington, NC, drinking water intake—13,000 times higher than EPA’s health advisory level of 10 ppt).

⁸ See, e.g., Ian T. Cousins et al., *Why is High Persistence Alone a Major Cause of Concern?*, 21 Env’t Sci. Processes & Impacts 781 (2019), <https://doi.org/10.1039/C8EM00515J>.

⁹ Antonia M. Calafat et al., *Polyfluoroalkyl Chemicals in the U.S. Population: Data from the National Health and Nutrition Examination Survey (NHANES) 2003–2004 and Comparisons with NHANES 1999–2000*, 115 Env’t Health Persp. 1596 (2007), <http://doi.org/10.1289/ehp.10598>.

¹⁰ Jahred M. Liddie et al., *Sociodemographic Factors Are Associated with the Abundance of PFAS Sources and Detection in U.S. Community Water Systems*, Env’t Sci. Tech. (2023), <https://pubs.acs.org/doi/pdf/10.1021/acs.est.2c07255>.

The health risks associated with PFAS are well established and have been widely recognized by international scientific organizations,¹¹ federal and state regulatory agencies,¹² and other leading scientific bodies.¹³ Individually, the PFAS covered by EPA’s Proposed Rule are associated with cancer (PFOA, PFOS, and GenX), developmental harm (PFOA, PFOS, PFHxS, GenX, PFNA, and PFBS), reproductive harm (PFOA, PFOS, GenX, and PFNA), immune system toxicity (PFOA, PFOS, GenX, and PFNA), liver toxicity (PFOA, PFOS, PFHxS, GenX, and PFNA), thyroid toxicity (PFOA, PFOS, PFHxS, and PFBS) and kidney toxicity (PFOA, PFOS, GenX, and PFBS), among other adverse effects.¹⁴ Because of these common health effects, people who are exposed to multiple PFAS—whether through their combined presence in drinking water or from other sources—face even greater risks of harm. Studies have shown that exposure to PFAS mixtures alter critical biological processes in the developing fetus, infants, and children that are separately associated with an increased risk of developmental disorders, cardiovascular disease, and many types of cancer.¹⁵ Recent human birth cohort studies also reported associations between multiple PFAS exposures during pregnancy and adverse health outcomes, including an increased risk of gestational diabetes and altered glucose levels during pregnancy, altered levels of thyroid hormones in pregnant people and newborns, and liver injury

¹¹ See United Nations Env’t Programme, UNEP/POPS/POPRC.2/17/Add.5, *Report of the Persistent Organic Pollutants Review Committee on the Work of its Second Meeting* add. 25–26 (Nov. 2006) (Risk Profile on Perfluorooctane Sulfonate), <http://chm.pops.int/Portals/0/download.aspx?d=UNEP-POPS-POPRC.2-17-Add.5.English.PDF>; United Nations Env’t Programme, UNEP/POPS/POPRC.12/11/Add.2, *Report of the Persistent Organic Pollutants Review Committee on the Work of Its Twelfth Meeting* add. 24–26 (Oct. 2016) (Risk Profile on Pentadecafluorooctanoic Acid (PFOA, Perfluorooctanoic Acid), its Salts and PFOA-related Compounds), <http://chm.pops.int/Portals/0/download.aspx?d=UNEP-POPS-POPRC.12-11-Add.2.English.PDF>.

¹² Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Perfluoroalkyls*, at 5–21, 26–29 (May 2021) (“ATSDR 2021”), <https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf>; Cal. Env’t Protection Agency, *Public Health Goals: Perfluorooctanoic Acid and Perfluorooctane Sulfonic Acid in Drinking Water (First Public Review Draft)*, Off. of Env’t Health Hazard Assessment, at 62–166 (July 2021), <https://oehha.ca.gov/sites/default/files/media/downloads/crn/pfoapfosphgdraft061021.pdf>.

¹³ Nat’l Acad. of Sci., Eng’g, & Med., *Guidance on PFAS Exposure, Testing, and Clinical Follow-Up*, at 6–8 (2022) (“NAS 2022”), <https://nap.nationalacademies.org/catalog/26156/guidance-on-pfas-exposure-testing-and-clinical-follow-up>; see also Arlene Blum et al., *The Madrid Statement on Poly- and Perfluoroalkyl Substances (PFASs)*, 123 *Env’t Health Persp.* A107 (2015), <https://ehp.niehs.nih.gov/doi/epdf/10.1289/ehp.1509934> (statement of more than 250 scientists expressing “concern[] about the production and release into the environment of an increasing number of [PFAS]”).

¹⁴ See Proposed Rule, 88 *Fed. Reg.* at 18,645–47, 18,656–63, 18,704, 18,718.

¹⁵ Jesse A. Goodrich et al., *Metabolic Signatures of Youth Exposure to Mixtures of Per- and Polyfluoroalkyl Substances: A Multi-Cohort Study*, 131 *Env’t Health Persp.* Art. No. 27005 (2023), <https://ehp.niehs.nih.gov/doi/epdf/10.1289/EHP11372>.

in children.¹⁶ An accompanying analysis by Drs. Anna Reade and Katherine Pelch of the Natural Resources Defense Council discusses additional health effects that are linked to PFAS exposure, but which were not well described in EPA’s toxicity assessments for PFOA and PFOS, such as disruption of mammary gland development and reduced duration of lactation.¹⁷

EPA has known of the harms associated with PFAS since at least 1998, and it has known of the presence of PFAS in drinking water since at least 2001.¹⁸ More than two decades later, however, EPA has yet to establish any federal limits on PFAS levels in drinking water. The Proposed Rule, which would regulate six widespread and highly toxic PFAS, is necessary and long overdue. EPA should act swiftly to issue final drinking water standards for PFAS, with the changes recommended below.

B. EPA Should Pursue Additional, Class-Based PFAS Drinking Water Standards

While EPA’s proposal marks an important step towards addressing the PFAS crisis, further action is needed to protect communities who are exposed to additional PFAS in their drinking water. A recent peer-reviewed, published study of tap water collected from 16 states, including more than 20 samples from public water supplies, detected 26 different PFAS, only six

¹⁶ Guoqi Yu et al., *Environmental Exposure to Perfluoroalkyl Substances in Early Pregnancy, Maternal Glucose Homeostasis and the Risk of Gestational Diabetes: A Prospective Cohort Study*, 156 *Env’t Int’l Art. No. 106621* (2021), <https://pubmed.ncbi.nlm.nih.gov/33984575/>; i: [10.1016/j.envint.2021.106621](https://doi.org/10.1016/j.envint.2021.106621); Blanca Sarzo et al., *Maternal Perfluoroalkyl Substances, Thyroid Hormones, and DIO Genes: A Spanish Cross-sectional Study*, 55 *Env’t Sci. Tech.* 11144 (2021), <https://pubs.acs.org/doi/10.1021/acs.est.1c01452>; Arash Derakhshan et al., *Association of Per- and Polyfluoroalkyl Substances with Thyroid Homeostasis During Pregnancy in the SELMA Study*, 167 *Env’t Int’l Art. No. 107420* (2022), <https://www.sciencedirect.com/science/article/pii/S0160412022003476?via%3Dihub>; Richard Christian Jensen et al., *Higher Free Thyroxine Associated with PFAS Exposure in First Trimester. The Odense Child Cohort*, 212 *Env’t Rsch. Art. No. 113492* (2022), <https://pubmed.ncbi.nlm.nih.gov/35597289/>; Jianqiu Guo et al., *Umbilical Cord Serum Perfluoroalkyl Substance Mixtures in Relation to Thyroid Function of Newborns: Findings From Sheyang Mini Birth Cohort Study*, 273 *Chemosphere Art. No. 129664* (2021), <https://pubmed.ncbi.nlm.nih.gov/33493812/>; Qian Yao et al., *Prenatal Exposure To Per- and Polyfluoroalkyl Substances, Fetal Thyroid Hormones, and Infant Neurodevelopment*, 206 *Env’t Rsch. Art. No. 112561* (2022), <https://www.sciencedirect.com/science/article/abs/pii/S0013935121018624?via%3Dihub>; Nikos Stratakis et al., *Prenatal Exposure to Perfluoroalkyl Substances Associated With Increased Susceptibility to Liver Injury in Children*, 72 *Hepatology* 1758, 1758–70 (2020), <https://onlinelibrary.wiley.com/doi/full/10.1002/hep.31483>.

¹⁷ Ltr. from Drs. Anna Reade and Katherine Pelch, Natural Resources Defense Council, re PFAS National Primary Drinking Water Regulation Rulemaking (May 30, 2023) (“Reade and Pelch 2023”) (attached as **Exhibit A**).

¹⁸ Scott Faber, *For 20-plus Years, EPA Has Failed to Regulate ‘Forever Chemicals’*, *Env’t Working Grp.* (Jan. 9, 2020), <https://www.ewg.org/research/20-plus-years-epa-has-failed-regulate-forever-chemicals>.

of which are covered by EPA's proposed rule.¹⁹ Another study of U.S. surface waters found 35 PFAS across 29 states and the District of Columbia, with one or more PFAS detected in 83% of the water bodies sampled.²⁰ Many of the PFAS detected but not covered by EPA's proposal, such as perfluorobutanoic acid ("PFBA") and perfluoroheptanoic acid ("PFHpA"), are associated with an increased risk of thyroid toxicity, liver damage, and developmental impairment, exacerbating the harms from the PFAS that are subject to EPA's proposed standards.²¹ Notably, PFBA, perfluorohexanoic acid ("PFHxA"), and perfluorodecanoic acid ("PFDA"), all have finalized or draft toxicity assessments performed by EPA's Integrated Risk Information System ("IRIS").²²

Like GenX, PFBS, PFNA, and PFHxS (collectively, the "Hazard Index PFAS" or "HI PFAS"), these other detected PFAS also meet the three statutory criteria for regulation under the SDWA.²³ EPA has found that PFBA, PFHxA, and PFDA "may have an adverse effect on the health of persons,"²⁴ and that they are associated with many of the same health effects observed following exposure to other PFAS, as summarized in Table 1 below. There are sufficient occurrence data from state monitoring efforts to support the need to protect against exposure to these PFAS in drinking water.²⁵ Regulation of these PFAS in drinking water, individually and as

¹⁹ Katherine E. Pelch et al., *70 Analyte PFAS Test Method Highlights Need for Expanded Testing of PFAS in Drinking Water*, 876 Sci. of the Total Env't Art. No.162978 (2023), <https://www.sciencedirect.com/science/article/pii/S0048969723015966>.

²⁰ Waterkeeper All., *Invisible, Unbreakable, Unnatural: PFAS Contamination of U.S. Surface Waters*, at 13 (Oct. 2022), <https://waterkeeper.org/wp-content/uploads/2022/10/Waterkeeper-Alliance-PFAS-Report-FINAL-10.14.22.pdf>.

²¹ EPA, *IRIS Toxicological Review of Perfluorobutanoic Acid (PFBA, CASRN 375- 22-4) and Related Salts*, at 4-1–4-2 (Dec. 2022), https://www.epa.gov/system/files/documents/2022-12/10945-%20PFBA%20ToxReview%20Final%20December%202022-HERO_partial-508%20%28updated%20page%20100%29.pdf; Health and Env't All., *The Curious Case of PFHpA and Why This and All Forever Chemicals Should be Banned Under REACH* (Dec. 13 2022), <https://www.env-health.org/the-curious-case-of-pfhpa-and-why-this-and-all-forever-chemicals-should-be-banned-under-reach/>.

²² EPA, *IRIS Toxicological Review of Perfluorobutanoic Acid*; EPA, *IRIS Toxicological Review of Perfluorohexanoic Acid [PFHxA, CASRN 307-24-4] and Related Salts* (Apr. 2023), https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0704tr.pdf; EPA, *IRIS Toxicological Review of Perfluorodecanoic Acid [PFDA, CASRN 335-76-2] and Related Salts (Public Comment and External Review Draft)* (Apr. 2023), https://ordspub.epa.gov/ords/eims/eimscomm.getfile?p_download_id=546623.

²³ See 42 U.S.C. § 300g-1(b)(1)(A) (requiring EPA to develop MCLGs and national primary drinking water regulations for contaminants that EPA determines "may have an adverse effect on the health of persons," are known or substantially likely to occur in public water systems "with a frequency and at levels of public health concern," and present "a meaningful opportunity for health risk reduction for persons served by public water systems").

²⁴ *Id.* § 300g-1(b)(1)(A)(i).

²⁵ See *id.* § 300g-1(b)(1)(A)(ii).

a mixture with other PFAS, “presents a meaningful opportunity for health risk reduction for persons served by public water systems.”²⁶

TABLE 1 Adverse Effects	PFBA ²⁷ osRfD ²⁸ (mg/kg-d)	PFHxA ²⁹ osRfD (mg/kg-d)	PFDA ³⁰ osRfD (mg/kg-d)
Hepatic	1 x 10⁻³ Increased hepatocellular hypertrophy in adult rats	4 x 10 ⁻⁴ Increased hepatocellular hypertrophy in adult rats	7 x 10 ⁻⁷ Increased relative liver weight in female rats <i>*subchronic RfD only</i>
Thyroid	1 x 10⁻³ Decreased total T4 in adult rats	1 x 10 ⁻³ Decreased free T4 in adult male rats <i>*subchronic RfD only</i>	
Developmental	6 x 10 ⁻³ Developmental delays in mice	5 x 10⁻⁴ Decreased F₁ body weight at PND 0 in rats	3 x 10⁻¹⁰ Decreased birth weight in male and female children
Hematopoietic	-	5 x 10 ⁻³ Decreased red blood cells in adult rats	
Immune			4 x 10⁻¹⁰ Decreased serum antibody concentrations for tetanus and diphtheria in children at age 7 years
Male reproductive			5 x 10 ⁻⁶

²⁶ *Id.* § 300g-1(b)(1)(A)(iii).

²⁷ EPA, *IRIS Toxicological Review of Perfluorobutanoic Acid*, at xiii (Table ES-1. Evidence integration judgements and derived toxicity values for PFBA).

²⁸ Organ/system-specific oral reference dose.

²⁹ EPA, *IRIS Toxicological Review of Perfluorohexanoic Acid [PFHxA, CASRN 307-24-4] and Related Salts*, at xv (Apr. 2023).

³⁰ EPA, *IRIS Toxicological Review of Perfluorodecanoic Acid (PFDA) and Related Salts (Public Comment and External Review Draft*, at xvii-xviii (Apr. 2023).

			Decreased absolute whole epididymis weight in rats <i>*subchronic RfD only</i>
Female reproductive			3 x 10 ⁻⁶ Increased number of days spent in diestrus in rats <i>*subchronic RfD only</i>

Health effects in **bold** were selected by EPA as the chronic or lifetime RfD.

Moreover, with more than 1,000 PFAS already in commerce and dozens of new PFAS awaiting EPA approval, EPA cannot fully protect public health or the environment by regulating individual PFAS (or even small sub-groups of PFAS) one at a time. Separate from its Proposed Rule, EPA should pursue a broader, class-based PFAS drinking water standard. Leading scientists have called for class-based standards³¹ and the European Union has established a drinking water standard, which will take effect in January 2026, for “the totality of per- and polyfluoroalkyl substances.”³² Similarly, the Canadian government recently found that “a precautionary, class-based approach to addressing PFAS is needed to protect the environment and people from anticipated adverse effects.”³³ “Addressing PFAS as a class of chemicals would also reduce the chance of regrettable substitution,” or the replacement of PFAS that regulated under the SDWA with equally toxic but less studied PFAS that are not subject to SDWA controls.³⁴

The SDWA does not require detailed information about every member of the class to protect the public from their cumulative health effects. EPA previously established a class-based drinking water limit for polychlorinated biphenyls (“PCBs”) despite acknowledging that “the

³¹ Carol F. Kwiatkowski et al., *Scientific Basis for Managing PFAS as a Chemical Class*, 7 Env’t Sci. & Tech. Letters 532, 532–43 (2022), <https://pubs.acs.org/doi/10.1021/acs.estlett.0c00255>.

³² Directive 2020/2184 of the European Parliament and of the Council of the European Parliament and of the Council of 16 December 2020 on the Quality of Water Intended for Human Consumption, 2020 O.J. (L 435), <https://eur-lex.europa.eu/legal-content/EN/TXT/HTML/?uri=CELEX:32020L2184>; *see also* Health Canada, *Draft Objective for Per- and Polyfluoroalkyl Substances in Canadian Drinking Water: Rationale*, <https://www.canada.ca/en/health-canada/programs/consultation-draft-objective-per-polyfluoroalkyl-substances-canadian-drinking-water/rationale.html> (last updated Feb. 10, 2023) (proposing 30 ppt drinking water limit for “total PFAS in drinking water,” using detection methods capable of measuring at least 18 PFAS).

³³ Environment and Climate Change Canada and Health Canada, *Draft State of Per- and Polyfluoroalkyl Substances (PFAS) Report* at 113-114 (May 2023), <https://www.canada.ca/content/dam/ecccc/documents/pdf/pded/pfas/draft-state-pfas-report.pdf>.

³⁴ *Id.* at 116.

toxicity of [the 209 possible PCB isomers] has not been fully characterized.³⁵ Here, too, the presence of multiple PFAS in the same drinking water supplies, as well as those chemicals' shared persistence and potential for common health effects, supports a class-based MCL. In addition to finalizing the Proposed Rule to protect communities with PFOA, PFOS, and the HI PFAS in their drinking water, EPA should also pursue a separate rulemaking process, beginning with a class-based PFAS regulatory determination, to establish drinking water standards that cover all mixtures of PFAS in drinking water.

II. The Proposed MCLs are Required by the SDWA and Supported by an Extensive Factual and Scientific Record

A. Statutory and Regulatory Background

"The Safe Drinking Water Act . . . was enacted to ensure that public water supply systems meet minimum national standards for the protection of public health."³⁶ To prevent drinking water contamination, the SDWA requires EPA to establish National Primary Drinking Water Regulations ("NPDWRs") that specify the "maximum levels for contaminants that may have an adverse effect on the health of consumers."³⁷

EPA's obligation to issue NPDWRs is triggered by the Administrator's determination that: (1) a contaminant "may have an adverse effect on the health of persons," (2) "the contaminant is known to occur or there is a substantial likelihood that the contaminant will occur in public water systems with a frequency and at levels of public health concern," and (3) "regulation of such contaminant presents a meaningful opportunity for health risk reduction for persons served by public water systems."³⁸ EPA's regulatory determinations, and other science-based decisions under the SDWA, "shall use . . . the best available, peer-reviewed science and supporting studies."³⁹ Here, EPA made regulatory determinations for PFOA and PFOS in March 2021, and it made a preliminary regulatory determination for the HI PFAS in the Proposed Rule.⁴⁰

An NPDWR must contain either a "maximum contaminant level" or a "treatment technique."⁴¹ A maximum contaminant level, or "MCL," is "the maximum permissible level of a contaminant in water which is delivered to any user of a public water system."⁴² An MCL must be set at a level that is "as close . . . as is feasible" to the "level at which no known or anticipated

³⁵ National Primary Drinking Water Regulations—Synthetic Organic Chemicals and Inorganic Chemicals; Monitoring for Unregulated Contaminants; National Primary Drinking Water Regulations Implementation; National Secondary Drinking Water Regulations, 56 Fed. Reg. 3,526, 3,546 (Jan. 30, 1991).

³⁶ *Nat'l Wildlife Fed'n v. EPA*, 980 F.2d 765, 768 (D.C. Cir. 1992).

³⁷ *Id.* (citing 42 U.S.C. § 300g-1).

³⁸ 42 U.S.C. § 300g-1(b)(1)(A).

³⁹ *Id.* § 300g-1(b)(3)(A)(i).

⁴⁰ Proposed Rule, 88 Fed. Reg. at 18,638; *see also* 42 U.S.C. § 300g-1(b)(1)(E) (authorizing EPA to publish a proposed NPDWR "concurrent with the determination to regulate").

⁴¹ 42 U.S.C. § 300f(1)(C).

⁴² *Id.* § 300f(3).

adverse effects on the health of persons occur and which allows an adequate margin of safety,” also known as the Maximum Control Level Goal or “MCLG.”⁴³ The analysis of feasibility “tak[es] costs into consideration,”⁴⁴ but it does not prioritize those considerations over public health protection or require EPA to find that the economic benefits of an MCL outweigh the costs.⁴⁵

EPA has broad authority under the SDWA to set MCLs for groups of related contaminants. The SDWA broadly defines “contaminant” as “any physical, chemical, biological, or radiological substance or matter in water,” encompassing both individual chemicals and chemical mixtures that are found in the same water supplies.⁴⁶ EPA set, and the D.C. Circuit Court of Appeals upheld, a single MCL for combined levels of radium-226 and radium-228 based on those substances’ co-occurrence in drinking water and common carcinogenic effects.⁴⁷ Similarly, EPA’s NPDWR for disinfectants and disinfection byproducts set combined MCLs for four different trihalomethanes (“THMs”) and five different haloacetic acids (“HAA5”).⁴⁸ In setting its original drinking water standards for THMs, EPA rejected calls to establish chemical-specific MCLs for individual THMs, such as chloroform, explaining that “as a family of compounds, the THMs are similar in chemical composition and nature” and are commonly found together in drinking water.⁴⁹ As explained above, EPA’s NPDWR for PCBs established a single MCL for “complex mixtures” of up to 209 possible PCB isomers.⁵⁰ In each of those rules, the use of a class-based MCL protects communities that are exposed to mixtures of related contaminants and furthers the SDWA’s purpose of “prevent[ing] the harmful contamination of public water systems.”⁵¹

⁴³ *Id.* § 300g–1(a)(4). The SDWA authorizes, but does not require, EPA to set an MCL above the most health-protective, feasible level if EPA determines that the “benefits of [the] maximum contaminant level ... would not justify the costs of complying with the level.” *Id.* § 300g–1(b)(6)(A). EPA did not make such a finding in its proposed rule, and, as described in greater detail below, EPA’s analysis of the rule’s costs and benefits precludes such a finding. *See pp.* 18–20 *infra*.

⁴⁴ 42 U.S.C. § 300g-1(a)(4)(D).

⁴⁵ *See* S. Rep. No. 104-169, at 33 (Nov. 7, 1995) (“The Administrator is not precluded from ... set[ting] a maximum contaminant level as close to the maximum contaminant level goal as feasible, even if the Administrator determines that the benefits of the MCL at this level do not justify the costs.”); *see also* 42 U.S.C. § 300g-1(b)(3)(C)(i) (requiring EPA to consider “nonquantifiable health risk reduction benefits” when establishing MCLs); *City of Portland, Oregon v. EPA*, 507 F.3d 706, 712 (D.C. Cir. 2007).

⁴⁶ 42 U.S.C. § 300f(6) (emphasis added).

⁴⁷ *See* Final Rule, National Primary Drinking Water Regulations; Radionuclides, 65 Fed. Reg. 76,708, 76,718, 76,720 (Dec. 7, 2000); *City of Waukesha v. E.P.A.*, 320 F.3d 228 (D.C. Cir. 2003).

⁴⁸ Final Rule, National Primary Drinking Water Regulations: Disinfectants and Disinfection Byproducts, 63 Fed. Reg. 69390 (Dec. 16, 1998).

⁴⁹ Final Rule, National Interim Primary Drinking Water Regulations; Control of Trihalomethanes in Drinking Water, 44 Fed. Reg. 68,624, 68,627 (Nov. 29, 1979).

⁵⁰ 56 Fed. Reg. at 3,546.

⁵¹ *Int’l Fabricare Inst. v. EPA*, 972 F.2d 384, 387 (D.C. Cir. 1992).

B. EPA's MCLs for PFOA and PFOS are Consistent with the SDWA and Supported by the Record

i. EPA Correctly Determined That There is No Safe Exposure Level for PFOA or PFOS

EPA's proposed MCLGs for PFOA and PFOS are consistent with the SDWA's mandate to identify the "level at which no known or anticipated adverse effects on the health of persons occur and which allows an adequate margin of safety."⁵² EPA appropriately proposed MCLGs of 0 ppt based on its longstanding policy of "establish[ing] MCLGs of zero for carcinogens ... where there is insufficient information to determine ... a threshold dose below which no carcinogenic effects have been observed."⁵³ There is substantial evidence that both PFOA and PFOS are carcinogenic, with no known safe level of exposure. "The carcinogenicity of PFOA has been observed in both human epidemiological and animal toxicity studies."⁵⁴ EPA documented the evidence of PFOA's carcinogenicity in its 2016 PFOA Health Effects Support Document, and subsequent studies have only strengthened and reinforced that finding.⁵⁵ A 2022 report from the National Academies of Sciences, Engineering and Medicine ("NAS") found "that there is sufficient evidence for an association between PFAS," including PFOA, "and kidney cancer,"⁵⁶ and the Agency for Toxic Substances and Disease Registry ("ATSDR") has reported "increases in the risk of testicular and kidney cancer associated with PFOA."⁵⁷ Similarly, human and animal studies of PFOS "reported elevated risk of bladder, prostate, kidney, and breast cancers after chronic PFOS exposure."⁵⁸ California's Office of Environmental Health Hazard Assessment ("OEHHA") has listed both PFOA and PFOS as chemicals "known . . . to cause cancer," based on its independent review of the scientific literature.⁵⁹ Additionally, OEHHA has published draft public health goals for PFOA and PFOS in drinking water based on their

⁵² 42 U.S.C. § 300g-1(b)(4)(A).

⁵³ Proposed Rule, 88 Fed. Reg. at 18,659.

⁵⁴ *Id.* at 18,656.

⁵⁵ *Id.*

⁵⁶ NAS 2022 at 74.

⁵⁷ ATSDR 2021 at 523.

⁵⁸ Proposed Rule, 88 Fed. Reg. at 18,660.

⁵⁹ OEHHA, *Notice to Interested Parties, Chemical Listed Effective February 25, 2022, As Known to the State of California to Cause Cancer: Perfluorooctanoic Acid* (2022) ("OEHHA 2022"), <https://oehha.ca.gov/media/downloads/cnr/listingnoticepfoa022522.pdf>; OEHHA, *Notice to Interested Parties, Chemicals Listed Effective December 24, 2021, As Known to the State of California to Cause Cancer: Perfluorooctane Sulfonic Acid (PFOS) and Its Salts and Transformation and Degradation Precursors* (2021), <https://oehha.ca.gov/media/downloads/cnr/noticepfossandsaltstransdegradprecursor122421.pdf>.

carcinogenicity.⁶⁰ EPA thus appropriately concluded that both PFOA and PFOS are “likely to be carcinogenic to humans.”⁶¹

EPA’s established practice, which has been endorsed repeatedly by EPA’s Science Advisory Board, the NAS, and other leading authorities, assumes that cancer risks follow a linear dose response curve, with no safe exposure threshold, in the absence of “scientific evidence demonstrating a threshold level of exposure below which there is no appreciable cancer risk.”⁶² Here, there is no evidence of a safe level for PFOA and PFOS, and accordingly no basis to depart from EPA’s standard approach. Based on substantial evidence of their carcinogenicity and the absence of a safe exposure threshold, EPA appropriately determined that there is no safe level for either PFOA or PFOS.

EPA’s proposed MCLGs are further supported by PFOA’s and PFOS’s severe non-cancer effects, which occur at levels far below those that can be detected in drinking water. As recognized EPA’s toxicity assessments for both PFOA and PFOS, those contaminants cause immune system harm (including reduced vaccine effectiveness in children), developmental toxicity, and heart and kidney damage at levels ranging as low as 3×10^{-8} mg/kg/day (PFOA) and 1×10^{-7} mg/kg/day (PFOS).⁶³ Those levels equate to drinking water toxicity in the parts-per-quadrillion range, well below the detection limit for either chemical.⁶⁴ While cancer risks alone are sufficient to support the proposed zero ppt MCLGs for PFOA and PFOS, their serious noncancer risks reinforce the need for EPA to reduce exposure to both chemicals as much as feasible.

ii. EPA’s Proposed PFOA and PFOS MCLs are Feasible Using Readily Available Detection and Treatment Technologies

EPA’s proposed PFOA and PFOS MCLs are “as close . . . as is feasible” to those chemicals’ MCLGs, as required by the SDWA.⁶⁵ The SDWA defines feasibility as “feasible with the use of the best technology” that has been tested under “field conditions” and is “available.”⁶⁶ When it amended the SDWA in 1986, Congress specified that “granular activated carbon is feasible for the control of synthetic organic chemicals,” like PFAS.⁶⁷ Congress added that “other

⁶⁰ OEHHA, *Public Health Goals, Perfluorooctanoic Acid and Perfluorooctane Sulfonic Acid in Drinking Water (First Public Review Draft)*, at 10 (2021) (“OEHHA 2021”), <https://oehha.ca.gov/sites/default/files/media/downloads/crn/pfoapfosphgdraft061021.pdf>.

⁶¹ Proposed Rule, 88 Fed. Reg. at 18,659–60.

⁶² *Id.* at 18,652–53; *see also* EPA, *Guidelines for Carcinogen Risk Assessment*, at 3-21–3-22 (2005).

⁶³ Proposed Rule, 88 Fed. Reg. at 18,659, 18,662–63.

⁶⁴ *See id.* at 18,669 (“The level at which no known or anticipated [non-cancer] effects on the health of persons would occur is well below current analytical quantitation level for PFOA and PFOS.”).

⁶⁵ 42 U.S.C. § 300g-1(b)(4)(B).

⁶⁶ 42 U.S.C. § 300g-1(b)(4)(D).

⁶⁷ Pub. L. No. 99-339, 100 Stat. 642, 644-645 (June 19, 1986) (currently codified at 42 U.S.C. § 300g-1(b)(4)(D)).

means found to be the best available for the control of synthetic organic chemicals must be at least as effective in controlling synthetic organic chemicals as granular activated carbon.”⁶⁸

Here, EPA correctly found that granular activated carbon (“GAC”), anion exchange, and high pressure membranes such as those used in reverse osmosis systems “can achieve [PFAS] concentrations less than 4 [ppt]” and may “exceed >99 percent [PFAS removal].”⁶⁹ Those technologies are not only readily available, but they have been deployed and proven effective in communities across the country. The Cape Fear Public Utility Authority reported no PFAS detections in water treated by granular activated carbon at a Wilmington, NC drinking water treatment plant, despite high levels of PFAS in the water before treatment.⁷⁰ In nearby Brunswick County, another utility used reverse osmosis to reduce PFOA and PFOS to non-detectable levels.⁷¹ The use of granular activated carbon treatment in New Jersey yielded similar results:

Seven different GAC treatment plants operating for years . . . removed PFOA, PFOS, and other PFAS chemicals to nondetectable levels. Three of those GAC plants were treating groundwater with concentrations of PFOA or PFOS of >500 ppt to nondetectable levels. Since 2019, 12 New Jersey plants, seven that use GAC and five that use IEX, have been achieving nondetectable levels of PFOA and PFOS in >99.9% of treated water with detection limits ranging from 0.53 to 5 ppt.⁷²

Moreover, as described below, EPA’s benefit-cost analyses found that these systems are cost-effective, with health benefits exceeding treatment costs in many circumstances.

Despite acknowledging that existing technologies can reduce PFOA and PFOS levels below 4 ppt, EPA proposed a 4 ppt MCL based on those chemicals’ practical quantitation levels (“PQL”), or the lowest level that can be detected “by capable analysts at 75 percent or more of

See id.

⁶⁸ *Id.*

⁶⁹ Proposed Rule, 88 Fed. Reg. at 18,684–86.

⁷⁰ See WECT News, *CFPUA: No PFAS Found in Water Treated by GAC Filters* (Oct. 11, 2022), <https://www.wect.com/2022/10/11/cfpua-no-pfas-found-water-treated-by-gac-filters/>.

⁷¹ Brunswick Cnty. Gov. Complex, *Brunswick County Commissioners Receive Final Report Showing PFAS Not Detected in LPRO Treated Water* (Apr. 17, 2018), <https://www.brunswickcountync.gov/brunswick-county-commissioners-receive-final-report-showing-pfas-not-detected-in-lpro-treated-water/>; CDM Smith, *Advanced Treatment Options for the Northwest Water Treatment Plant: Brunswick County*, App’x A (Apr. 2018), <https://www.brunswickcountync.gov/wp-content/uploads/2018/04/CDM-Smith-Brunswick-Final-Report-April-2018.pdf>.

⁷² Elizabeth Southerland & Linda S. Birnbaum, *What Limits Will the World Health Organization Recommend for PFOA and PFOS in Drinking Water*, 57 Env’t Sci. & Tech. 7103, 7103–7105 (2023), <https://doi.org/10.1021/acs.est.3c02260>; N.J. Drinking Water Quality Inst., Treatment Subcomm., *Recommendation on Perfluorinated Compound Treatment Options for Drinking Water*, at 6–8 (June 2015), <https://www.nj.gov/dep/watersupply/pdf/pfna-pfc-treatment.pdf>.

the laboratories using a specified analytical method[.]”⁷³ Because no laboratories can currently measure PFOA or PFOS levels down to the 0 ppt MCLG and not all labs can reliably measure those chemicals below the PQL, “EPA often bases the MCL on the PQL.”⁷⁴ EPA’s latest six-year review of National Primary Drinking Water Treatment Standards identified at least 14 contaminants for which EPA set MCLs based on the PQL, including benzo[a]pyrene, PCBs, and 2,3,7,8-Tetrachlorodibenzo-p-Dioxin.⁷⁵ Similarly, setting the PFOA and PFOS MCLs at the PQL is consistent with the SDWA and with EPA’s past practice.

As testing technologies advance, however, EPA must review and reduce those MCLs. The SDWA provides that “not less often than every 6 years,” EPA must “review and revise, as appropriate, each national primary drinking water regulation promulgated under this subchapter.”⁷⁶ As EPA acknowledges, laboratory testing capacity “can improve over time,” and “the Six-Year Review process is an opportunity to evaluate whether new information . . . shows that PQLs for carcinogens can be reduced, which introduces the possibility of reducing the MCLs[.]”⁷⁷ Many labs already have the ability to measure PFOA and PFOS well below the MCLs, and some emerging technologies can detect PFOA, PFOS, GenX, and PFBS in the parts-per-quadrillion range.⁷⁸ EPA should reassess laboratories’ PFOA and PFOS detection capacity during each six-year review period and decrease the MCL whenever new information supports a lower PQL.

C. EPA’s Proposed MCL for GenX, PFBS, PFNA and PFHxS is Consistent with the SDWA and Supported by the Record

i. A Hazard Index Is a Well Established and Appropriate Method of Addressing the Risks Posed by Mixtures of Multiple PFAS

For the HI PFAS, EPA proposed an MCLG and MCL using an approach that addresses the harms caused by each of those contaminants individually, as well as by their combined presence in drinking water supplies. This approach is well grounded in the SDWA, and it is needed to protect communities that have multiple PFAS in their drinking water supplies.

⁷³ Proposed Rule, 88 Fed. Reg. at 18,666–67.

⁷⁴ EPA, *EPA 810-R-16-002, Development of Estimated Quantitation Levels for the Third Six-Year Review of National Primary Drinking Water Regulations (Chemical Phase Rules)*, Off. of Water, at 1-3 (Oct. 2016), <https://www.epa.gov/sites/default/files/2016-12/documents/810r16002.pdf>.

⁷⁵ *Id.* at 1-3 – 1-4.

⁷⁶ 42 U.S.C. § 300g–1(b)(9).

⁷⁷ EPA, *EPA 810-R-16-002, Development of Estimated Quantitation Levels for the Third Six-Year Review of National Primary Drinking Water Regulations (Chemical Phase Rules)*, at 1-2.

⁷⁸ Phenomenex, *Achieving Low Parts-per-Quadrillion Detection Limits for PFAS Analysis in Drinking Water (TN-1316)*, at 1, <https://www.phenomenex.com/documents/2022/09/29/17/52/achieving-low-partsperquadrillion-detection-limits-for-pfas-analysis-in-drinking-water-tn1316>.

A hazard index is “commonly used” to measure and regulate risks from mixtures, or combinations, of contaminants that cause similar health effects.⁷⁹ Under this approach, EPA calculates each contaminant’s “hazard quotient,” or individual risk, by dividing the level of human exposure (i.e., the concentration of the contaminant in drinking water) by the level at which the contaminant presents risk to human health (referring to be EPA as a Health-Based Water Concentration or “HBWC”).⁸⁰ A hazard quotient below 1 indicates an individual chemical is present below the level that is known to cause risk. To calculate the risk from the mixture, EPA adds the chemicals’ hazard quotients to calculate a hazard index, with a hazard index above 1 generally indicating elevated risk to human health.⁸¹ However, the use of a hazard index of 1 as an adequate health threshold relies on EPA’s ability to address all of the harms associated with the chemicals at issue in their underlying toxicity assessments. Given the uncertainties inherent in the estimation of risk and the multitude of factors that are not included in EPA calculations—including the effects of non-chemical stressors and co-exposures to other PFAS that are not covered by the Proposed Rule – EPA should consider the use of a hazard index below 1 to provide the “adequate margin of safety” required by the SDWA.⁸²

Hazard indices have long been used by EPA offices and endorsed by leading scientific authorities, including EPA’s Science Advisory Board. EPA calculates hazard indices under the Comprehensive Environmental Response, Compensation, and Liability Act to measure the cumulative effects of multiple contaminants at a Superfund site and to develop health-protective clean-up goals.⁸³ They are also used under the Clean Air Act to calculate chronic risks from multiple chemicals released by a given source category.⁸⁴ Hazard indices address the potential for “low levels of multiple [chemicals] that individually would not likely result in adverse health effects . . . to result in adverse health effects” when combined in a mixture. This approach to calculating risk – also known as dose additivity – “has found widespread acceptance as an

⁷⁹ Proposed Rule, 88 Fed. Reg. at 18,639.

⁸⁰ *Id.*

⁸¹ *Id.*

⁸² 42 U.S.C. § 300g–1(b)(4)(A); *see also* Devon Payne-Sturges et al., *Cumulative Risk Evaluation of Phthalates Under TSCA*, 57 Env’t Sci. & Tech. 6403, 6409 (2023), <https://pubs.acs.org/doi/full/10.1021/acs.est.2c08364> (challenging the “traditional use of HI ≤ 1 as being ‘safe’ or acceptable for mixtures/multiple chemical exposures” and proposing “the use of a HI of 0.1–0.2 as a benchmark”).

⁸³ *See* EPA, EPA/540/1-89/002, *Risk Assessment Guidance for Superfund: Volume I Human Health Evaluation Manual (Part A)*, Off. of Emergency and Remedial Response, at 8-11–8-13, <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=2000KLZ9.txt> (“At most Superfund sites, one must assess potential health effects of more than one chemical ... Estimating risk or hazard potential by considering one chemical at a time might significantly underestimate the risks associated with simultaneous exposures to several substances ... To assess the overall potential for noncarcinogenic effects posed by more than one chemical, a hazard index (HI) approach has been developed based on EPA’s (1986b) *Guidelines for Health Risk Assessment of Chemical Mixtures*.”)

⁸⁴ *See* National Emission Standards for Hazardous Air Pollutants: Cyanide Chemicals Manufacturing Residual Risk and Technology Review, 86 Fed. Reg. 3906, 3910-11 (Jan. 15, 2021).

assessment concept for combined exposures to multiple chemicals . . . and is extensively used by regulatory authorities as a protective default approach.”⁸⁵

Here, a hazard index is necessary and appropriate to address the harms associated with the HI PFAS. As EPA found, “PFHxS, [GenX], PFNA, and PFBS . . . result[] in common adverse effects on several biological systems including thyroid hormone levels, lipid synthesis and metabolism, as well as on development, and immune and liver function.”⁸⁶ Exposure to mixtures of those chemicals poses greater risks than exposure to each chemical in isolation, such that setting individual chemical MCLs would not fully protect people who have combinations of the HI PFAS in their drinking water. EPA also found, based on nationwide monitoring data, that “there is a substantial likelihood PFHxS, [GenX], PFNA, and PFBS will occur and co-occur with a frequency of public health concern.”⁸⁷ “When three or four HI PFAS were monitored, over 40 percent of systems reported detections of two to three of the HI PFAS.”⁸⁸ An MCL that ignores those co-exposures could leave millions of people at risk.⁸⁹

This is the precise scenario that has justified prior, mixture-based MCLs. In its disinfection byproducts rule, EPA set a combined MCL for five THMs that are detected together in drinking water and cause similar health effects.⁹⁰ Because of their combined effects, regulating each component of that mixture in isolation would “permit a substantial number of communities . . . to avoid any improvement of treatment practice and, by implication, water quality.”⁹¹ As described above, EPA also regulated all PCBs under a single MCL because individual isomer limits would understate their combined risks.⁹² Here, too, EPA cannot protect communities with multiple HI PFAS in their drinking water unless its MCL accounts for the harms associated with those chemicals’ mixtures.

- ii. EPA Should Maintain its Hazard Index Approach to Setting the MCLGs and MCLs for the HI PFAS While Updating its Hazard Index Calculations to Reflect the Best Available Science

With appropriate inputs, a hazard index of 1 reflects “the level at which no known or anticipated adverse effects on the health of persons occur” from exposure to the HI PFAS, as required for an MCLG.⁹³ Because the HI PFAS cause a range of harmful effects at different exposure levels, simply setting a maximum concentration for their combined presence in

⁸⁵ Org. for Econ. Coop. and Dev., *ENV/JM/MONO(2018)37, Considerations for Assessing the Risks of Combined Exposure to Multiple Chemicals: Series on Testing and Assessment No. 296*, at 19 (Dec. 6, 2018), <https://www.oecd.org/chemicalsafety/risk-assessment/considerations-for-assessing-the-risks-of-combined-exposure-to-multiple-chemicals.pdf>.

⁸⁶ Proposed Rule, 88 Fed. Reg. at 18,647.

⁸⁷ *Id.*

⁸⁸ *Id.* at 18,676.

⁸⁹ *See id.* at 18,678

⁹⁰ 44 Fed. Reg. at 68,624, 68,626-28.

⁹¹ *Id.* at 68,628.

⁹² 56 Fed. Reg. at 3,546.

⁹³ 42 U.S.C. § 300g-1(b)(4).

drinking water, as EPA has done for prior contaminant mixtures, would not address their “known or anticipated” adverse effects.⁹⁴ By dividing each contaminant’s exposure level by the lowest level at which the contaminant is known to pose harm, EPA can calculate hazard quotients that are tailored to each contaminant and that protect against effects that occur at higher exposure levels. And by adding those quotients to calculate the hazard index, EPA can protect against adverse effects from mixtures of the HI PFAS and ensure that people who are exposed to multiple HI PFAS are not placed at risk.⁹⁵ However, this approach requires EPA to set HBWCs—the denominators in its hazard quotient equations⁹⁶—at levels that protect against all of a contaminants’ adverse health effects, including effects to “subgroups . . . such as infants, children, pregnant women, the elderly, individuals with a history of serious illness, or other subpopulations[] that are identifiable as being at greater risk of adverse health effects due to exposure to contaminants in drinking water than the general population.”⁹⁷ As explained below, EPA’s proposed HBWCs do not address the HI PFAS’ increased risks to infants and must be revised in a manner consistent with the best available science. We urge EPA to use the reduced HBWCs recommended below when calculating the Hazard Index for the purposes of setting the HI PFAS MCLG.

A hazard index of 1 is also “feasible with the use of the best [available] technology,” and is a proper MCL. Here, as well, EPA should revise the denominators for its hazard index calculations. Because the HBWCs proposed below are significantly lower than the practical quantitation level for the HI PFAS, laboratories may not be able to detect MCL exceedances that are based on those levels. Therefore, when calculating the hazard index for the purpose of setting and implementing the MCL, we recommend that EPA use the PQL for each HI PFAS as the denominator, similar to EPA’s approach for PFOA, PFOS, and other chemicals that pose health risks below their respective PQLs. This would ensure that the HI PFAS MCL is feasible, because (1) laboratories can already detect each HI PFAS down to the PQL and (2) water systems can reduce levels of the HI PFAS below their respective PQLs by using the same treatment technologies that EPA has proposed to address PFOA and PFOS, including granular activated carbon and reverse osmosis.⁹⁸ Similar to PFOA and PFOS, many communities are already using those technologies to treat water contaminated with GenX and other HI PFAS.

The SDWA does not dictate the form of an MCL or MCLG; it merely requires the MCLG to be set at a health-protective “level” and the MCL to be set as close as feasible to that level. EPA’s Proposed Rule, with the changes recommended herein, satisfies those statutory requirements. In the past, EPA has set MCLs based on the percentage of water samples that detected a class of contaminants (total coliforms), as opposed to a density-based limit, because “the presence-absence concept is simpler and mathematically more precise than the current

⁹⁴ *Id.*

⁹⁵ As described below, EPA should update its toxicity values (or Health Based Water Concentrations) for the HI PFAS to reflect the latest available science on those chemicals’ hazards. Those revisions would not change the MCL or MCLG; they would merely ensure that the calculations that EPA used to calculate the hazard index are fully protective of human health.

⁹⁶ Proposed Rule, 88 Fed. Reg. at 18,665.

⁹⁷ 42 U.S.C. § 300g–1(b)(1)(C).

⁹⁸ Proposed Rule, 88 Fed. Reg. at 18,665–66.

density standard for total coliforms[.]”⁹⁹ Similarly, a hazard index provides a “more precise” estimate of the HI PFAS’ effects than a concentration-based limit, consistent with the SDWA’s mandate to minimize those contaminants’ adverse effects to the extent feasible.

III. EPA Should Take Steps to Strengthen the Economic Analysis Supporting the Proposed Rule

As required by the SDWA, EPA’s Proposed Rule is supported by a draft Economic Analysis (“Draft EA”) that assesses the proposal’s “[q]uantifiable and nonquantifiable health risk reduction benefits,” its “[q]uantifiable and nonquantifiable costs,” the incremental costs and benefits of the alternative MCLs considered, the effects of the Six PFAS on the general population and greater-risk subpopulations, any increased health risks associated with compliance with the Proposed Rule, and other relevant factors such as uncertainties in the analysis.¹⁰⁰ As described in the accompanying expert review of EPA’s Draft EA by Dennis Guignet, Ph.D., many features of the Draft EA are exceptionally thorough and transparent,¹⁰¹ and the Draft EA provides ample justification for EPA’s conclusion that the Proposed Rule’s quantified and unquantified benefits justify its costs.¹⁰² Further, as discussed in the accompanying expert review by Elin Betanzo, EPA’s treatment cost estimates are robust and, if anything, may overstate actual costs.¹⁰³ Nevertheless, there are important steps EPA can and should take to strengthen the EA to better support the proposed drinking water standards.

At the outset, we encourage EPA to maintain, and consider expanding upon, several key methodological strengths of the Draft EA.¹⁰⁴ The Draft EA is predicated on a detailed, data-driven Monte Carlo simulation model that supports comprehensive sensitivity analyses, which evaluate the impact of specific variables on estimates of the Proposed Rule’s net benefits. As described by Dr. Guignet, this is the most thorough approach to account for multiple sources of uncertainty in the economic analysis simultaneously.¹⁰⁵ The Draft EA accounts appropriately for existing state-level drinking water standards when estimating the costs and benefits attributable

⁹⁹ Drinking Water; National Primary Drinking Water Regulations; Total Coliforms (Including Fecal Coliforms and E. coli), 54 Fed. Reg. 27,544, 27,548 (June 29, 1989).

¹⁰⁰ 42 U.S.C. § 300g-1(b)(3)(C)(i); see EPA, *Economic Analysis for the Proposed Per- and Polyfluoroalkyl Substances National Primary Drinking Water Regulation* (EPA Doc. No. EPA-822-P-23-001) (Draft for Public Comment) (Mar. 2023).

¹⁰¹ Memorandum from Dennis Guignet, Ph.D., to Earthjustice, Re. Review of the Economic Analysis for the Proposed PFAS NPDWR (May 26, 2023) (“Guignet 2023”) (Attached as **Exhibit B**).

¹⁰² See Proposed Rule, 88 Fed. Reg. at 18,689, 18,727–29. At the same time, we stress that EPA has the authority to set the MCL “as close to the [MCLG] as feasible, even if [EPA] determines that the benefits of the MCL at this level do not justify the costs.” S. Rep. No. 104-169 at 33.

¹⁰³ Elin Betanzo, Safe Water Engineering, *Analysis of the USEPA Proposed PFAS National Primary Drinking Water Regulation Treatment Costs and Comparison to the AWWA National PFAS Cost Model Report* (May 30, 2023) (“Betanzo 2023”) (Attached as **Exhibit C**).

¹⁰⁴ See Guignet 2023 at 2–7.

¹⁰⁵ *Id.* at 2–3.

to the Proposed Rule.¹⁰⁶ In addition, the EA relies appropriately on unquantified health benefits (though, as discussed below, the record supports quantification of additional health benefits).¹⁰⁷ EPA’s reliance on unquantified health benefits is consistent with the agency’s standard practice,¹⁰⁸ and is expressly required by the SDWA, which mandates that EPA’s health risk reduction and cost analysis account for all “[q]uantifiable and nonquantifiable health risk reduction benefits for which there is a factual basis in the rulemaking record to conclude that such benefits are likely to occur” due to compliance with the MCL.¹⁰⁹ Congress “require[d] [EPA] to determine whether the benefits of a [drinking water] standard ‘justify’ (rather than ‘exceed’ or ‘outweigh’) the costs to reflect the nonquantifiable nature of some of the benefits and costs that may be considered. [EPA] is not required to demonstrate that the dollar value of the benefits are greater (or lesser) than the dollar value of the costs,” and “[a]ll costs and benefits, both quantifiable and nonquantifiable, must be considered when making determinations under this authority.”¹¹⁰

EPA’s analysis of treatment costs associated with the Proposed Rule is also well supported and more accurately forecasts costs than the competing analysis submitted by the American Water Works Association (“AWWA”).¹¹¹ For example, in contrast to AWWA, EPA properly screened the PFAS occurrence data incorporated into its treatment cost assessment to avoid bias in the data set from non-public water system PFAS samples and samples collected by water systems investigating known PFAS contamination.¹¹² EPA also appropriately calculated treatment costs based on the number of water system entry points with modeled MCL violations, whereas AWWA assumed without justification that *every* entry point within a water system will require treatment if *any* entry point within the system violates the MCL.¹¹³ Critically, EPA also incorporated detailed estimates of the compliance strategies that water systems are likely to select—including non-treatment options—and associated costs.¹¹⁴ EPA’s cost estimates also

¹⁰⁶ *Id.* at 5.

¹⁰⁷ *Id.* at 6–7.

¹⁰⁸ *Id.*

¹⁰⁹ 42 U.S.C. § 300g-1(b)(3)(C)(i)(I)–(II).

¹¹⁰ S. Rep. No. 104-169 at 33. Moreover, Congress recognized the inherent difficulty and subjectivity in fully quantifying the economic benefits of rules, so the SDWA authorizes EPA to establish an MCL at a feasible level even if the agency cannot formally determine that MCL is justified by the economic costs. *Id.* We note that, for example, there are often equity considerations, as there are with PFAS, whereby certain populations, often low-income communities and communities of color, bear disproportionate burdens from exposure to environmental contaminants. See Jahred M. Liddie et al., *Sociodemographic Factors Are Associated with the Abundance of PFAS Sources and Detection in U.S. Community Water Systems*, Env’t Sci. Tech. (2023), <https://pubs.acs.org/doi/pdf/10.1021/acs.est.2c07255>. Such equity considerations often are not reflected in economic analyses but are valid considerations under the SDWA.

¹¹¹ Betanzo 2023; see Am. Water Works Ass’n, *PFAS National Cost Model Report* (Black & Veatch 2023).

¹¹² Betanzo 2023 at 4, 6.

¹¹³ *Id.* at 4–5.

¹¹⁴ Guignet 2023 at 4; Betanzo 2023 at 2–3, 9–11.

rely appropriately on inventoried flow rates, whereas AWWA utilizes a standardized 150 gpm/person flow rate that is biased high and fails to account for regional water-use differences.¹¹⁵ Overall, EPA’s cost estimate is “robust” and “there is no evidence that EPA is consistently underestimating occurrence or costs,” while AWWA’s estimate includes excess treatment costs of at least \$2.6 billion.¹¹⁶

In addition to maintaining or expanding upon these robust features of the Draft EA, there are several ways that EPA can and should strengthen the EA. These are outlined in detail in Dr. Guignet’s analysis, and we highlight several key recommendations here:

First, EPA should utilize the best available scientific and economic information to quantify and/or monetize additional benefits of the rule in the final EA. In the Proposed Rule, EPA correctly determined that:

PFAS exposure is associated with a wide range of adverse health effects including reproductive effects such as decreased fertility; increased high blood pressure in pregnant women; developmental effects or delays in children, including low birth weight, accelerated puberty, bone variations, or behavioral changes; increased risk of some cancers, including prostate, kidney, and testicular cancers; reduced ability of the body’s immune system to fight infections, including reduced vaccine response; interference with the body’s natural hormones; and increased cholesterol levels and/or risk of obesity.¹¹⁷

Yet EPA quantified only “three PFOA- and PFOS-related health endpoints in [the economic] analysis,” while recognizing that the rule is “expected to produce substantial benefits that have not been quantified.”¹¹⁸

We urge EPA to expand upon its quantification and, where supported, monetization of the Proposed Rule’s benefits. As explained by Dr. Guignet, EPA’s economic analysis guidance dictates that the agency

should try to get as far as possible in first *identifying* all key benefit and cost categories. The next step (when possible) is to then *quantify* the projected change in each benefit and cost outcome that is expected to result from the policy option, relative to the baseline. Quantifying in this case means to measure the change in terms of some quantitative metric, such as the number of lives saved, number of cases prevented, etc. The final step is to monetize the quantified change, meaning that a dollar value is assigned.¹¹⁹

Here, consistent with recently proposed revisions to OMB’s Circular A4, EPA should assess and disclose the expected magnitude of benefits that EPA recognized but did not quantify.¹²⁰ This

¹¹⁵ Betanzo 2023 at 5, 12.

¹¹⁶ *Id.* at 22.

¹¹⁷ Proposed Rule, 88 Fed. Reg. at 18,725.

¹¹⁸ *Id.*

¹¹⁹ Guignet 2023 at 9.

¹²⁰ *Id.* at 10.

would enable some quantification of additional benefits, even if fully monetizing a benefit category is not possible. In addition, where quantification is not possible, EPA should utilize available monetary cost-of-illness or willingness to pay estimates to illustrate the potential magnitude of benefits discussed qualitatively.¹²¹

In addition, as summarized in the accompanying analysis by Drs. Anna Reade and Katherine Pelch, the best available science supports EPA's consideration, and potential quantification and monetization, of additional regulatory benefits.¹²² When measuring the Proposed Rule's benefits, EPA ignored or improperly dismissed evidence of the connection between PFAS and liver disease, impaired mammary gland development and reduced lactation duration, and immune system suppression and increased susceptibility to infectious disease. EPA thus failed to measure all of the benefits attributable to the Proposed Rule's reduction in PFAS exposures. At a minimum, EPA should assess these benefits qualitatively and it should utilize the analysis provided by Drs. Reade and Pelch to attempt to quantify and, where possible, monetize these benefits as well.¹²³

EPA should also reconsider its omission of reduced testicular cancer incidence from its assessment of the Proposed Rule's benefits. To justify that omission, EPA asserts in the Draft EA that "testicular cancer is rarely fatal which implies low expected economic value of reducing this risk because Value of Statistical Life is the driver of the economic benefits evaluated in the EA."¹²⁴ But that assertion is not well supported. While the Draft EA relies on the Value of Statistical Life metric "[t]o estimate the economic value of avoided premature deaths" associated with the rule, it utilizes the cost of illness (COI) valuation approach "[t]o estimate the economic value of avoided morbidity (i.e., non-fatal heart attacks and ischemic strokes, birth weight decrements, and cancers)," with the COI values "reflect[ing] medical care expenditures and opportunity costs associated with managing/treating the condition."¹²⁵ EPA has not explained why COI-based valuation of avoided non-fatal testicular cancer cases is not justified. EPA also has not provided a reasoned basis to dismiss as "imply[ing] low" the expected economic value of reducing testicular cancer risks associated with PFOA exposure because EPA has not attempted to estimate the economic value of reducing this risk.¹²⁶ Moreover, as explained by Dr. Guignet, the COI values EPA did employ to evaluate the benefits of reducing other non-fatal health effects incorrectly omit the opportunity cost of time, which, as elaborated below, "likely results in a significant underestimate of the benefits."¹²⁷ For this reason too, EPA's speculation that the economic value of avoided testicular cancer cases would be "low" is unsupported. In the final EA, EPA should estimate the economic value of reduced testicular cancer cases associated

¹²¹ *Id.*

¹²² *See generally* Reade and Pelch 2023.

¹²³ *Id.*; Guignet 2023 at 10.

¹²⁴ Draft EA at 6-21– 6-22.

¹²⁵ *Id.* at 2-4.

¹²⁶ *Id.* at 6-21.

¹²⁷ Guignet 2023 at 12.

with the Proposed Rule.¹²⁸ In doing so, EPA should utilize COI estimates that properly account for the opportunity cost of time in addition to avoided medical expenses or use willingness-to-pay estimates where supported by the literature.¹²⁹

Second, while it is appropriate for EPA to consider co-benefits when estimating the health benefits of the Proposed Rule,¹³⁰ EPA's analysis of co-benefits is incomplete. The Draft EA considers only reduced bladder cancer risks from co-removal of disinfection byproducts associated with PFAS drinking water technology,¹³¹ ignoring the benefits that will arise from co-removal of additional synthetic organic contaminants, including additional PFAS that are not directly regulated by the Proposed Rule.

Third, EPA should apply no (or at most a very low) discount rate to account for the intergenerational harms associated with PFAS and the nature of the rule's economic impacts.¹³² In the event a discount rate for future benefits is applied, we agree with EPA's conclusion in the Draft EA that a lower, consumption-based discount rate is "more appropriate for this rulemaking" than a higher, capital-based discount rate¹³³ given the 80-year timeframe for analysis, the impacts on future generations, and the extent of uncertainties in the magnitude of future health benefits.¹³⁴ Thus, if a discount rate is used, EPA should ensure that both the final EA and the final rule reflect and explain EPA's determination that a lower, consumption-based discount rate is more appropriate.¹³⁵ EPA's draft preamble does not include or explain this determination and instead presents benefit estimates based on a 3% consumption-based discount rate and a 7% capital-based discount rate as equally relevant to assessing the net benefits of the rule.¹³⁶ Further, if EPA continues to apply a discount rate in the final EA, it should consider

¹²⁸ Peer-reviewed literature is available to support the monetization of avoided testicular cancer cases. *See, e.g.,* Michael Aberger et al., *Testicular Self-Examination and Testicular Cancer: A Cost Utility Analysis*, 3 *Cancer Med.* 1629 (2014), <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4298389/>.

¹²⁹ Guignet 2023 at 12.

¹³⁰ *Id.* at 5.

¹³¹ Proposed Rule, 88 Fed. Reg. at 18,721.

¹³² *See, e.g.,* Frank Ackerman and Lisa Heinzerling, *Pricing the Priceless: Cost-Benefit Analysis of Environmental Protection*, 150 U. Pa. L. Rev. 1553, 1571 (2002), https://scholarship.law.upenn.edu/penn_law_review/vol150/iss5/6 (questioning the use of discounting to address long-term, intergenerational harms, including those associated with "persistent toxins"); *see also* Institute for Policy Integrity, *Comments on National Primary Drinking Water Regulations: Lead and Copper Rule Revisions* (Feb. 12, 2020), https://policyintegrity.org/documents/EPA_Lead_Copper_Rule_Comments_2020.02.11.pdf (arguing for a "3% or lower discount rate" for benefits of EPA drinking water rule).

¹³³ Draft EA at 2-3.

¹³⁴ Guignet 2023 at 11; Peter Howard & Jason A. Schwartz, *Valuing the Future: Legal and Economic Considerations for Updating Discount Rates*, 39 *Yale J. Regul.* 595, 599, 603 (2022) (explaining why a consumption-based discount rate is appropriate for estimating benefits of rules designed to affect public health over a long time horizon).

¹³⁵ Draft EA at 2-3.

¹³⁶ *E.g.,* Proposed Rule, 88 Fed. Reg. at 18,724, table 66.

utilizing a reduced consumption-based discount rate—below 2%—as the 3% rate used in the Draft EA does not reflect the best available economic data and literature.¹³⁷

Fourth, as noted above, EPA should account for opportunity costs, in addition to avoided medical expenditures, in its final cost-of-illness estimates for non-fatal health effects associated with PFAS exposure.¹³⁸ As Dr. Guignet explains, “assuming the opportunity cost of time associated with these adverse health outcomes is zero (as the EPA currently does) is not correct” and likely yields a substantial underestimate of the COI-based benefits for reducing non-fatal adverse health effects.¹³⁹ While recognizing limitations in the relevant literature, Dr. Guignet has identified multiple approaches for EPA to account for these benefits in the final EA.

Fifth, EPA should develop an estimate of the benefits of managing spent filtration materials as hazardous waste, which would reduce environmental releases of PFAS and associated human exposures. In assessing the Proposed Rule’s costs in the Draft EA, EPA correctly excludes the incremental costs to water systems from potential future requirements to manage spent filtration materials as hazardous waste, electing instead to calculate such costs as part of an illustrative sensitivity analysis because these costs are not attributable to the rule under consideration.¹⁴⁰ While EPA’s inclusion of this sensitivity analysis enhances the transparency value of the EA, it improperly considers only the costs of potential hazardous waste management requirements, without accounting for the benefits.¹⁴¹ If EPA maintains this illustrative analysis as part of the final EA, it also must include a benefits estimate to ensure that its analysis is comprehensive and balanced.¹⁴²

Finally, EPA should evaluate a regulatory option that is more stringent than the proposed MCLs. Contrary to OMB Circular A4 and EPA’s economic analysis guidelines, all the alternatives analyzed in the Draft EA are less stringent than the proposed option. EPA should evaluate a more stringent regulatory option in the final EA or, at a minimum, explain why such an analysis is not appropriate.¹⁴³

IV. EPA Should Strengthen its Proposed Rule to Protect the Public and Promote Compliance

A. EPA Should Revise its Health Based Water Concentrations (“HBWCs”) to Fully Address the HI PFAS’ Harms to Susceptible Populations

¹³⁷ Howard & Schwartz (2022) at 595–96, 599, 610–11, 617–19; *see also* White House Off. of Mgmt. and Budget, *Circular A-4 (Public Review Draft)*, at 76 (Apr. 6, 2023) (proposing consumption-based discount rate of 1.7%), www.whitehouse.gov/wp-content/uploads/2023/04/DraftCircularA-4.pdf.

¹³⁸ Guignet 2023 at 11–12.

¹³⁹ *Id.*

¹⁴⁰ *Id.* at 4–5.

¹⁴¹ *Id.* at 5.

¹⁴² *Id.*

¹⁴³ *Id.* at 10.

EPA’s proposed MCLG and MCL for the HI PFAS incorporate HBWCs to indicate the levels at which PFBS, GenX, PFNA, and PFHxS pose no known adverse health effects.¹⁴⁴ When establishing MCLs, the SDWA requires EPA to consider contaminants’ effects not only on the general population but also on “groups . . . such as infants, children, pregnant women, the elderly, individuals with a history of serious illness, or other subpopulations that are identified as likely to be at greater risk of adverse health effects due to exposure to contaminants in drinking water[.]”¹⁴⁵ However, EPA’s proposed HBWCs fail to address risks to infants and other populations that experience the greatest risks from the HI PFAS, leaving those populations exposed to serious harm. In its final rule, we urge EPA to revisit and strengthen its HBWCs for each of the HI PFAS.

Developing infants and children are most at risk of the long-term effects of PFAS exposure. There are two reasons for this. First, the fetal and early childhood life stages are when the body’s systems are being established and developed. Small changes that disrupt or permanently alter the course of development can increase the risk of later-life disease. Second, formula fed infants and lactating people consume more drinking water per unit of body weight.¹⁴⁶ Infants, for example, may be exposed to PFAS via contaminated breastmilk and/or infant formula prepared with PFAS contaminated water. It is important that these factors are adequately accounted for in the MCL or health-based value calculation process, since developing children are both the most sensitive population as well as the population with the highest estimated exposure.

Unless there is substantial data showing that an endpoint studied in adults is not relevant in infants and children, it is EPA’s responsibility to set standards that are protective of all populations. Furthermore, the assumption for PFAS should be that there is a need to protect infants and children. Given the similarity among PFAS, EPA does not require developmental studies each particular PFAS to conclude that infants and children are susceptible to harm from exposure. In this case, drinking water intake assumptions for infants and children should be used. As stated by EPA in the Drinking Water Health Advisory for PFBS, “[w]hen multiple potentially sensitive populations or life stages are identified based on the critical effect or other health effects data (from animal or human studies), EPA selects the population or life stage with the greatest [drinking water intake rate adjusted for body weight] DWI-BW” because it is the most health protective.”¹⁴⁷ Despite this strong statement and the potential for developmental effects

¹⁴⁴ As described above, EPA divides the measured concentration of each HI PFAS by its HBWC to calculate a hazard quotient, which is then added to the other HI PFAS’ hazard quotients to calculate the hazard index.

¹⁴⁵ 42 U.S.C. § 300g–1(b)(3)(C)(i)(V) (requiring EPA to assess health impacts on greater-risk populations when establishing MCLs).

¹⁴⁶ EPA, *EPA/600/R-18/259F, Update for Chapter 3 of the Exposure Factors Handbook: Ingestion of Water and Other Select Liquids*, Off. of Rsch. and Dev., at 3-14, 3-23 (Feb. 2019), https://www.epa.gov/sites/default/files/2019-02/documents/efh_-_chapter_3_update.pdf.

¹⁴⁷ EPA, *EPA/822/R-22/006, Drinking Water Health Advisory: Perfluorobutane Sulfonic Acid (CASRN 375-73-5) and Related Compound Potassium Perfluorobutane Sulfonate (CASRN 29420-49-3)*, Off. of Water, at 18–19 (June 2022), <https://www.epa.gov/system/files/documents/2022-06/drinking-water-pfbs-2022.pdf>.

from PFAS exposure in the Proposed Rule, EPA consistently failed to use the more protective DWI-BW for infants or children, even when the critical effect is developmental, as is the case for PFNA. EPA chose a DWI-BW for lactating women for PFNA and GenX, a DWI-BW for “women of childbearing age” for PFBS and a DWI-BW for the adult general population for PFHxS.¹⁴⁸ The application of DWI-BW for these PFAS is not the most health protective and puts infants and children at risk.

Health protective approaches are used in other steps of risk assessment when evidence is lacking, for example, when deriving a chronic reference dose in the absence of a chronic study. Risk assessors often determine the risk of acute, subchronic, and chronic exposures to a chemical. For PFAS in drinking water, the most protective and realistic exposure scenario is typically a chronic exposure. However, since chronic exposure studies are not always available, EPA derives reference doses (“RfDs”) for chronic exposure from subchronic studies by applying an uncertainty factor instead of improperly assuming a RfD based on a subchronic exposure is protective of chronic exposure.

Furthermore, NAS has recommended the use of an additional uncertainty factor of 10 to ensure protection of fetuses, infants and children groups which are often are not sufficiently protected from toxic chemicals such as pesticides by the traditional intraspecies uncertainty factor.¹⁴⁹ Congress adopted this requirement in the Food Quality Protection Act (“FQPA”) for pesticides in or on foods.¹⁵⁰ The uneven application of this additional uncertainty factor to protect these vulnerable populations across EPA is concerning. Considering the many health effects linked to PFAS that affect these vulnerable populations and the substantial data gaps on exposure and toxicity of these compounds in complex mixtures, EPA must do a better job of protecting sensitive and vulnerable populations in its assessments and actions on all toxic chemicals, regardless of their regulatory context.

i. EPA Should Revise its HBWC for PFHxS to 2 ng/L

When calculating its HBWC for PFHxS, EPA relied on a RfD derived by ATSDR for thyroid follicular cell damage in adult male rats from a study by Butenhoff et al.¹⁵¹ However, in March 2022, OEHHHA published a risk assessment analysis for PFHxS as part of its Notification Level Recommendation for PFHxS in Drinking Water.¹⁵² In its analysis, OEHHHA evaluated the

¹⁴⁸ EPA, *EPA-822-P-23-004, Maximum Contaminant Level Goal (MCLG) Summary Document for a Mixture of Four Per- and Polyfluoroalkyl Substances (PFAS): HFPO-DA and its Ammonium Salt (also known as GenX Chemicals), PFBS, PFNA, and PFHxS*, Off. of Water, at 9 (Mar. 2023), <https://www.regulations.gov/document/EPA-HQ-OW-2022-0114-0906>.

¹⁴⁹ Nat’l Acad. of Sci., *Pesticides in the Diets of Infants and Children*. National Research Council 361 (1993), <https://www.ncbi.nlm.nih.gov/books/NBK236275/>.

¹⁵⁰ 21 U.S.C. § 346a(b)(2)(C)(ii)(II).

¹⁵¹ See ATSDR 2021 at 21, A54–A57 (citing John L. Butenhoff et al., *Evaluation of Potential Reproductive and Developmental Toxicity of Potassium Perfluorohexanesulfonate in Sprague Dawley Rats*, 27 *Reprod. Toxicology* 331, 331–334 (June 2009)).

¹⁵² OEHHHA, *Notification Level Recommendation: Perfluorohexane Sulfonic Acid in Drinking Water* (Mar. 2022), <https://oehha.ca.gov/media/pfhxsnl031722.pdf>.

same studies as ATSDR and a newer toxicological study by the National Toxicology Program (“NTP”).¹⁵³ Ultimately, OEHHA derived Public Health-Protective Concentrations (equivalent to EPA’s health-based water concentrations) for three sensitive endpoints because they occur in different populations.¹⁵⁴ In contrast, ATSDR only derived a single RfD. The analysis of multiple sensitive endpoints in deriving a final drinking water value is critical to ensure protection from all adverse health effects in all populations.

First, choosing the lowest human equivalent dose (“HED”) to derive a RfD does not guarantee that the RfD will protect against all health effects. A less sensitive HED could reasonably result in a lower RfD due to differences in study design and overall application of uncertainty. The IRIS PFAS assessments follow best practices in calculating organ-specific RfDs for multiple identified health effects.¹⁵⁵ OEHHA also followed these best practices and derived RfDs for decreased thyroxine (T4) (which is associated with thyroid toxicity) in adult male rats, decreased litter size in female mice, and increased relative liver weight in female rats.¹⁵⁶ Whereas the lowest HED was for decreased litter size, the lowest RfD was identified as decreased total T4 due to the application of different uncertainty factors to the two outcomes.¹⁵⁷

Secondly, choosing the lowest RfD to derive an HBWC does not guarantee that all health effects will be protected against. The influence of population specific drinking water exposure assumptions is also important to consider. In the case of OEHHA’s analysis, the final health-protective concentration in drinking water was lowest for decreased T4 (2 ng/L), when protecting against possible health effects in infants.¹⁵⁸

In its analysis, OEHHA states, “[f]or PFHxS, there are no developmental studies of thyroid hormone levels in animals, and no mouse studies reporting T4 or T3 levels. Despite this uncertainty, the point of departure (“POD”) for decreased T4 in male rats is a suitable candidate for PFHxS HPC derivation due to the severity of possible developmental consequences of decreased T4 in humans.”¹⁵⁹ Therefore, because infants are a sensitive group for decreased total T4, OEHHA applied a 0- to 6-month infant DWI-BW of 0.237 L/kg/day to derive a health-based water concentration.

We strongly support OEHHA’s health-protective approach to the lack of developmental data on thyroid hormone disruption for PFHxS. There is no reason to assume that this health effect is limited to an adult male population. Rather, when data are available, decreased T4

¹⁵³ *Id.* at 15 (citing, *inter alia*, NTP, *NTP Technical Report on the Toxicity Studies of Perfluoroalkyl Sulfonates (Perfluorobutane Sulfonic Acid, Perfluorohexane Sulfonate Potassium Salt, and Perfluorooctane Sulfonic Acid)* (August 2019), <https://cebs.niehs.nih.gov/cebs/publication/TOX-96>)).

¹⁵⁴ *Id.* at 30.

¹⁵⁵ See, e.g., EPA, *Toxicological Review of Perfluorohexanoic Acid and Related Salts*, at 5–28.

¹⁵⁶ OEHHA, *Notification Level Recommendation: Perfluorohexane Sulfonic Acid in Drinking Water*, at 26.

¹⁵⁷ *Id.* at 28.

¹⁵⁸ *Id.* at 28–29.

¹⁵⁹ *Id.* at 26.

during development has been identified as a sensitive endpoint for other PFAS. Importantly, OEHHA and IRIS have argued that even though decreased thyroid hormone levels appear less severe than classical hypothyroidism and are not associated with increased levels of thyroid-stimulating hormone (TSH), decreased T4 is correlated with neurodevelopmental and cognitive deficits in children, highlighting the importance of protecting the developing fetus, infants, and children against PFAS exposure.¹⁶⁰

The analysis by OEHHA indicates that a HBWC of 2 ng/L should be set to protect the most vulnerable and sensitive populations. Because that level is lower than the PQL of 3 ng/L, when calculating the hazard index for the purpose of establishing and monitoring compliance with the PFHxS MCL EPA should rely on the PQL, as opposed to EPA's currently proposed and under-protective HBWC.¹⁶¹

ii. EPA Should Revise its HBWC for PFBS to 240 ng/L

EPA's highest, and least protective, HBWC is for PFBS, a chemical that is often "considered [as] a replacement for PFOS."¹⁶² EPA's proposed HBWC of 2000 ppt PFBS is significantly higher than toxicity values and drinking water standards adopted by California (500 ppt), Michigan (420 ppt), Washington (345 ppt), and Minnesota (100 ppt).¹⁶³

In deriving a RfD for PFBS, EPA, California, Michigan and Washington used the same health effect (impaired thyroid development) and the same approach for calculating a human equivalent dose, resulting in similar RfDs.¹⁶⁴ However, EPA's PFBS Lifetime Health Advisory for drinking water, which serves as the foundation for EPA's proposed HBWC, deviated from state risk assessments most notably in the choice of DWI-BW (rate of water intake). In order to protect infants from harmful PFBS exposures, California, Michigan, and Washington selected a

¹⁶⁰ See *id.* at 20.

¹⁶¹ Proposed Rule, 88 Fed. Reg. at 18,680. Although we strongly recommend EPA adopt the analysis conducted by OEHHA, we acknowledge and support EPA's choice to apply an additional UF of 10 to adjust for subchronic-to-chronic duration (i.e., UFS), per agency guidance. *Id.* at 18,645–46.

¹⁶² EPA, *Drinking Water Health Advisories for PFAS Fact Sheet for Communities*, at 2 <https://www.epa.gov/system/files/documents/2022-06/drinking-water-ha-pfas-factsheet-communities.pdf>.

¹⁶³ Cal. Water Boards, *Notification Level Issuance*, State Water Res. Control Bd. (Mar. 5, 2021), https://www.waterboards.ca.gov/drinking_water/certlic/drinkingwater/pfas.html; Mich. PFAS Action Response Team, *Maximum Contaminant Levels (MCLs)*, <https://www.michigan.gov/pfasresponse/drinking-water/mcl> (last accessed May. 25, 2023); Wash. State Dep't of Health, *Recommended State Action Levels for Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water: Approach, Methods, and Supporting Information* (Nov. 1, 2021), <https://doh.wa.gov/sites/default/files/2022-02/331-673.pdf>; Minn. Dep't of Health, *Per- and Polyfluoroalkyl Substances (PFAS) and Health*, at 3–4 (Sept. 6, 2022), <https://www.health.state.mn.us/communities/environment/hazardous/docs/pfashealth.pdf>.

¹⁶⁴ We support the use of a more chemical-specific dose adjustment factor in EPA's final toxicity assessment versus the allometric scaling performed in EPA's draft document.

higher value for drinking water intake associated with infant drinking water consumption when deriving their drinking water limits for PFBS.¹⁶⁵ EPA, on the other hand, relied on a lower water intake rate associated with “women of childbearing age”¹⁶⁶ and thus failed to address PFBS’ increased risks to infants, a “potentially susceptible life stage[] for the types of effects observed in animal testing with PFBS.”¹⁶⁷ Thus, EPA’s HBWC for PFBS falls short of protecting one of the most “sensitive population(s) or life stage(s) (i.e., those that may be more susceptible or sensitive to a chemical exposure).”¹⁶⁸

The thyroid harm identified by EPA resulted from decreased serum levels of T4 from PFBS exposure during a developmental life stage, effects that begin prenatally and continue into infancy. Decreased levels of T4 indicate dysfunction or underdevelopment of the thyroid gland. While a decrease in T4 affects the pregnant mice, those effects can also carry over to their offspring and “persist[] until the pubertal and adult periods.”¹⁶⁹ The authors of the study that EPA relied upon concluded that PFBS “may impair thyroid development in offspring, leading to permanent hypothyroxinemia”¹⁷⁰ Infants with hypothyroxinemia experience impaired growth and development because the thyroid orchestrates processes that are critical to their growth, including brain development. Thus, many infants with hypothyroxinemia experience intellectual disabilities and growth failures that require treatment through puberty and, in some cases, into adulthood.¹⁷¹ While women of childbearing age are sensitive to developmental toxicity and persistent changes in thyroid hormone levels associated with PFBS, maternal thyroid hormones play a critical role in fetal and infant growth and neurodevelopment. Thyroid development and stores of T4 are especially important in infants whose T4 stores are lower and not as capable of

¹⁶⁵ OEHHHA, *Notification Level Recommendation: Perfluorobutane Sulfonic Acid in Drinking Water*, at 29–30 (Jan. 2021),

<https://oehha.ca.gov/media/downloads/water/chemicals/nl/pfbsnl121820.pdf>; Wash. Dep’t of Health, *Recommended State Action Levels for Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water: Approach, Methods, and Supporting Information*, Off. of Pub. Health Sci., at 80–81 (Nov. 1, 2021), <https://doh.wa.gov/sites/default/files/2022-02/331-673.pdf>.

¹⁶⁶ EPA, *EPA-822-P-23-004, Maximum Contaminant Level Goal (MCLG) Summary Document for a Mixture of Four Per- and Polyfluoroalkyl Substances (PFAS): HFPO-DA and its Ammonium Salt (also known as GenX Chemicals), PFBS, PFNA, and PFHxS*, Off. of Water, at 12 (Mar. 2023), <https://www.regulations.gov/document/EPA-HQ-OW-2022-0114-0906>.

¹⁶⁷ Wash. Dep’t of Health, *Recommended State Action Levels for PFAS*, at 79.

¹⁶⁸ EPA, *MCLG Summary Document for a Mixture of Four PFAS*, at 5.

¹⁶⁹ Xuejiao Feng et al., *Exposure of Pregnant Mice to Perfluorobutanesulfonate Causes Hypothyroxinemia and Developmental Abnormalities in Female Offspring*, 155 *Toxicological Sciences*, 409, 417 (2017), <https://doi.org/10.1093/toxsci/kfw219>.

¹⁷⁰ *Id.* at 414.

¹⁷¹ See generally Noora Moog et al., *Influence of Maternal Thyroid Hormones During Gestation on Fetal Brain Development*, 342 *Neuroscience* 68, 68–100 (2017), <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4819012/>; Stanford Medicine, *Congenital Hypothyroidism in Children*, Children’s Health, <https://www.stanfordchildrens.org/en/topic/default?id=hypothyroidism-in-children-90-P01963>.

offsetting declines.¹⁷² Drinking water intake is a pertinent exposure factor that is “intended to protect sensitive populations and life stages within the general population from adverse effects.”¹⁷³ EPA’s failure to consider the increased drinking water intake of infants understates the exposure to PFBS and its health effects on a sensitive population. EPA’s HBWC may provide protections for adults and fetuses but it ignores the risks that PFBS poses for infants and does not address “adverse effects can result from short or intermittent exposure during a critical period of development.”¹⁷⁴

Using the DWI-BW listed on Table 3 of the Drinking Water Health Advisory for PFBS¹⁷⁵ for formula fed infants (0.249 L/kg/day), the HBWC for PFBS should be no more than 240 ng/L.

$$\begin{aligned}\text{HBWC} &= (\text{RfD}/\text{DWI-BW}) * \text{RSC} \\ &= ((0.0003 \text{ mg/kg-bw/day}) / (0.249 \text{ L/kg-bw/day})) * 0.2 \\ &= 0.00024 \text{ mg/L} = 240 \text{ ng/L}\end{aligned}$$

iii. EPA Should Revise its HBWC for PFNA to 2 ng/L

In deriving a health-based water value for PFNA from ATSDR’s RfD, EPA chose the drinking water intake estimate for lactating women (0.0469 L/kg-bw/day).¹⁷⁶

However, the critical effects selected by ATSDR (decreased body weight and developmental delays including delayed eye opening, preputial separation and vaginal opening) occur during development.¹⁷⁷ Furthermore, a transgenerational toxicokinetic model for PFNA has been developed and used by some states which demonstrates a significantly higher level of exposure for breastfed infants.¹⁷⁸ We recommend that EPA either evaluate and use this transgenerational toxicokinetic model or apply a drinking water intake rate for infants. Using the

¹⁷² Francesca Coperchini et al., *Thyroid Disrupting Effects of Old and New Generation PFAS*, 11 *Frontiers in Endocrinology* Art. No. 612320 (2021), <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7851056/>; Hui Min et al., *Maternal Hypothyroxinemia-Induced Neurodevelopmental Impairments in the Progeny*, 53 *Molecular Neurobiology* 1613, 1613–1624 (2016), <https://pubmed.ncbi.nlm.nih.gov/25666160/>; Miller, M.D., et al., *Thyroid-Disrupting Chemicals: Interpreting Upstream Biomarkers of Adverse Outcomes*, 117 *Env’t Health Persp.* 1033, 1033–41 (2009), <https://pubmed.ncbi.nlm.nih.gov/19654909/>.

¹⁷³ EPA, *Drinking Water Health Advisory: Perfluorobutane Sulfonic Acid Potassium Perfluorobutane Sulfonate*, at 18.

¹⁷⁴ *Id.* at 17 (citing EPA, EPA/600/FR-91/001, *Guidelines for Developmental Toxicity Risk Assessment*, (Dec. 5, 1991), https://www.epa.gov/sites/default/files/2014-11/documents/dev_tox.pdf).

¹⁷⁵ EPA, *Drinking Water Health Advisory: Perfluorobutane Sulfonic Acid and Perfluorobutane Sulfonate*, at 19.

¹⁷⁶ EPA, *MCLG Summary Document for a Mixture of Four PFAS*, at 15.

¹⁷⁷ *Id.* at 13.

¹⁷⁸ Wash. Dep’t of Health, *Recommended State Action Levels for PFAS*, at 21–24.

drinking water intake rate for formula fed infants (0.249 L/kg-bw/day) (see above), the health-based value goal for PFNA should be no more than 2 ng/L.

$$\begin{aligned}\text{HBWC} &= (\text{RfD/DWI-BW}) * \text{RSC} \\ &= ((0.000003 \text{ mg/kg-bw/day}) / (0.249 \text{ L/kg-bw/day})) * 0.2 \\ &= 0.0000024 \text{ mg/L} = 2 \text{ ng/L}\end{aligned}$$

The HBWC for PFNA should be no higher than 2 ng/L to protect the most vulnerable and sensitive populations. Because that level is lower than the PQL of 4 ng/L, when calculating the hazard index for the purpose of establishing and monitoring compliance with the PFNA MCL, EPA should rely on the PQL, as opposed to EPA's currently proposed and under-protective HBWC.¹⁷⁹

iv. EPA Should Revise its HBWC for GenX to 2 ng/L

EPA finalized the Human Health Toxicity Assessment for GenX in October 2021.¹⁸⁰ In the development of the Lifetime Health Advisory for GenX, EPA “identified three potentially sensitive life stages for GenX chemical exposure—women of childbearing age (13 to < 50 years), pregnant women, and lactating women.”¹⁸¹ In setting its HBWC, EPA ultimately chose the drinking water intake estimate for lactating women, stating that this would be protective of the other two populations as well (i.e., pregnant women and women of childbearing age).¹⁸² However, there is no analysis to suggest that infants and young children would be sufficiently protected from liver or other developmental effects due to exposure during this critical stage. Furthermore, the NOAEL for developmental effects linked to GenX exposure is within the same range as the NOAEL for liver effects (i.e., within one order of magnitude).¹⁸³ We therefore

¹⁷⁹ Proposed Rule, 88 Fed. Reg. at 18,680.

¹⁸⁰ See EPA, *Human Health Toxicity Values for Hexafluoropropylene Oxide (HFPO) Dimer Acid and Its Ammonium Salt (CASRN 13252-13-6 and CASRN 62037-80-3), Also Known as ‘GenX Chemicals’* at 86-88 (Oct. 2021), https://www.epa.gov/system/files/documents/2021-10/genx-chemicals-toxicity-assessment_tech-edited_oct-21-508.pdf. We support the changes to the draft document that were made in response to the public comment process. Specifically, we support the NTP Pathology Working Group findings on liver lesions, which were based on more contemporary pathology guidelines than were used in prior analyses. We further support the application of a full uncertainty factor to account for the use of a study with less chronic exposure and a full uncertainty factor for database deficiencies.

¹⁸¹ EPA, *Drinking Water Health Advisory: Hexafluoropropylene Oxide (HFPO) Dimer Acid (CASRN 13252-13-6) and HFPO Dimer Acid Ammonium Salt (CASRN 62037-80-3), Also Known as ‘GenX Chemicals’*, Off. of Water, at 21 (June 2022), <https://www.epa.gov/system/files/documents/2022-06/drinking-water-genx-2022.pdf>.

¹⁸² EPA, *MCLG Summary Document for a Mixture of Four PFAS*, at 9.

¹⁸³ EPA, *Human Health Toxicity Values for Hexafluoropropylene Oxide (HFPO) Dimer Acid and Its Ammonium Salt (CASRN 13252-13-6 and CASRN 62037-80-3) Also Known as ‘GenX Chemicals’*, (Oct. 2021) https://www.epa.gov/system/files/documents/2021-10/genx-chemicals-toxicity-assessment_tech-edited_oct-21-508.pdf

recommend that EPA apply a DWI-BW for infants. Using the DWI-BW listed on Table 3 of the Drinking Water Health Advisory for GenX for formula-fed infants (0.249 L/kg/day), the health-based value goal for GenX should be no more than 2 ng/L.

$$\begin{aligned}\text{HBWC} &= (\text{RfD}/\text{DWI-BW}) * \text{RSC} \\ &= ((0.000003 \text{ mg/kg-bw/day}) / (0.249 \text{ L/kg-bw/day})) * 0.2 \\ &= 0.0000024 \text{ mg/L} = 2 \text{ ng/L}\end{aligned}$$

The HBWC for GenX should be no higher than 2 ng/L to protect the most vulnerable and sensitive populations. Because that level is lower than the PQL of 5 ng/L, when calculating the hazard index for the purpose of establishing and monitoring compliance with the GenX MCL EPA should rely on the PQL, as opposed to EPA's currently proposed and under-protective HBWC.¹⁸⁴

* * *

We reiterate our support for EPA's use of a hazard index, or some other method that accounts for adverse effects associated with mixtures of the HI PFAS, when setting its MCLG and MCL. In order to comply with the SDWA and protect communities who are exposed to those PFAS mixtures, however, the HBWCs that EPA uses to calculate the hazard index must reflect the "best available science" on those chemicals' risks to infants and other susceptible subpopulations. EPA's proposed HBWCs would permit unsafe levels of the HI PFAS to remain in drinking water and diminish the protectiveness of EPA's proposed MCLs. We urge EPA to strengthen the HBWCs in its final rule.

B. EPA Should Account for PFAS Detections Below the PQL When Determining MCL Compliance

To ensure compliance with the MCL and to protect communities from dangerous PFAS exposures, EPA must consider all PFAS detections when calculating MCL compliance. EPA has proposed determining initial MCL compliance based on a "running annual average," which considers a water provider's average PFOA concentration, PFOS concentration, or hazard index over four consecutive quarters to determine whether the provider has exceeded the MCL.¹⁸⁵ But when calculating the running annual average, EPA has proposed treating all PFAS detections below a chemical's PQL—4 ppt for PFOA, PFOS, and PFNA; 5 ppt for GenX; and 3 ppt for PFHxS and PFBS—as if they were non-detects.¹⁸⁶ This approach ignores measurable PFAS exposures and understates PFAS risks. For instance, if a water provider detected quarterly PFOA concentrations of 3.8 ppt, 3.8 ppt, 3.8 ppt, and 15 ppt, the mathematical average would be 6.6 ppt—more than 50 percent higher than the 4 ppt MCL. However, the average for the purpose of determining MCL compliance would be 3.75 ppt, since all of the 3.8 ppt detections would be replaced with zeros, meaning the provider would be considered in compliance with the MCL and

¹⁸⁴ Proposed Rule, 88 Fed. Reg. at 18,680.

¹⁸⁵ *Id.* at 18,667.

¹⁸⁶ *Id.* at 18,667; *see id.* at 18,680 (listing PQLs for different PFAS chemicals).

no additional treatment would be required. EPA's discounting of sub-PQL detections is scientifically unsupported, contrary to longstanding SDWA regulations, and inconsistent with other parts of EPA's proposed rule.

As EPA acknowledges, "almost all laboratories" can detect the PFAS at issue at levels below the chemicals' respective PQLs.¹⁸⁷ The PQL reflects the capacities of some of the least sophisticated laboratories; it is, according to EPA, the "minimum quantitation level that . . . can be achieved by capable analysts at 75 percent or more of the laboratories using a specified analytical method."¹⁸⁸ In a prior rulemaking, EPA found that "49 of the 54 laboratories seeking EPA approval" to test PFAS in drinking water "included a lowest PFAS calibration standard level at 1 ppt or lower, with the median lowest calibration level among all laboratories at 0.5 ppt."¹⁸⁹ Given that "the overwhelming majority of laboratories" can detect PFOA, PFOS, and other PFAS below their PQLs, there is no reason for EPA to disregard those detections and treat PFAS-contaminated water as if it were PFAS-free when determining MCL compliance.

EPA's proposed approach is contrary to longstanding EPA regulations, which consider all detections above the method detection limit ("MDL"), a level that is distinct from—and lower than—the PQL. The MDL reflects the "minimum measured concentration of a substance that can be reported with 99% confidence that the measured concentration is distinguishable from method blank results."¹⁹⁰ As a "general rule," EPA sets the PQL at a level that is 5–10 times greater than the MDL.¹⁹¹ EPA's SDWA regulations governing monitoring and analytical requirements for organic chemicals, such as PFAS, state that "[i]f a sample result is less *than the detection limit*, zero will be used to calculate the annual average."¹⁹² Similarly, EPA's SDWA regulations governing inorganic chemicals, such as arsenic and mercury, provide that "any sample *below the method detection limit* shall be calculated at zero for the purpose of determining the annual average."¹⁹³ EPA acknowledges that the consideration of sub-PQL detections is "consistent with EPA's [National Primary Drinking Water Regulations] related to other [synthetic organic chemicals] and has the potential to . . . increase the public health protection provided by this proposed regulation."¹⁹⁴ EPA should apply that same health-protective approach in its PFAS NPDWR.

¹⁸⁷ *Id.* at 18,667.

¹⁸⁸ *Id.* at 18,666.

¹⁸⁹ *Id.* at 18,667; *see also id.* (finding that "the overwhelming majority of laboratories with the necessary instrumentation to support PFAS monitoring have the capability to provide screening measurement results above . . . 1/3 of the MCL (i.e., 1.3 ppt for PFOS or PFOS).")

¹⁹⁰ 40 C.F.R. Part 136 App'x B; 40 C.F.R. § 141.2.

¹⁹¹ *Id.* at 18,666; National Primary Drinking Water Regulations; Volatile Synthetic Organic Chemicals, 50 Fed. Reg. 46, 902, 46,906 (Nov. 13, 1985) ("EPA believes that setting the PQLs in a range between 5 and 10 times the MDL achieved by the best laboratories is a fair expectation for most State and commercial laboratories.").

¹⁹² 40 C.F.R. § 141.24(f)(15)(v) (emphasis added).

¹⁹³ 40 C.F.R. § 141.23(i)(1) (emphasis added).

¹⁹⁴ Proposed Rule, 88 Fed. Reg. at 18,682–83.

EPA's justification for disregarding sub-PQL PFAS detections is unsupported and internally inconsistent. EPA claims that, even though most laboratories are able to detect PFAS below the PQL, "quantifying concentrations below the PQL for compliance purposes may decrease the precision and accuracy of the measured value."¹⁹⁵ But EPA's proposed approach is even less precise and accurate, since it would treat detectable PFAS levels as if they did not exist. Even if EPA has more confidence in PFAS detections above the PQL than below it, the relevant question is whether a detection above the MDL but below the PQL is more likely to reflect actual contamination or a false positive. EPA itself has acknowledged that "[f]or results between the detection limit and the PQL, EPA has determined that utilities would be able to reliably conclude analyte presence," meaning EPA's proposed approach of treating of all sub-PQL detections as zero understates real-world exposures and risks.¹⁹⁶ Moreover, elsewhere in its proposed rule, EPA considers detections at or below one-third of the PQL sufficiently reliable to "trigger . . . less frequent compliance monitoring."¹⁹⁷ In particular, EPA allows water systems to reduce their monitoring frequency from quarterly to once-every-three-years if their average PFAS concentrations are less than one-third of the MCL (i.e., 1.3 ppt for PFOA and PFOS and a hazard index of 0.33 for the HI PFAS).¹⁹⁸ In calculating that average, EPA considers PFAS detections that are well below the PQL. But there is no basis for considering sub-PQL detections to reduce water systems' monitoring obligations while ignoring those same detections when determining water systems' treatment obligations. For PFOA, PFOS, and the HI PFAS, EPA should instead consider all detections above the MDL to calculate a water system's annual running average and determine MCL compliance, including in hazard index calculations for HI PFAS.

C. EPA Should Strengthen the Proposed Monitoring Requirements to Comply with the SDWA and Enhance Public Health Protections

We urge EPA to strengthen the compliance monitoring requirements in the proposed rule by (1) relying on the MDL as the "trigger level" that can qualify a PWS for reduced monitoring where that value is lower than one-third of the PQL, and (2) requiring PWSs with consistent detections below the MDL to monitor annually for the regulated PFAS instead of triennially. EPA's proposed monitoring requirements are insufficient to ensure compliance with the proposed MCLs, would undermine the potential health benefits of the rule, and would deprive the public of vital information regarding exposures to PFAS in drinking water at levels that threaten human health.

At the outset, we support EPA's proposal to disallow monitoring waivers as part of the PFAS NPDWR in light of the "ubiquity, environmental persistence and transport abilities of PFAS."¹⁹⁹ But as explained below, these same factors—as well as the toxic effects of PFAS at very low concentrations—undermine EPA's proposal for reduced monitoring requirements for

¹⁹⁵ *Id.* at 18,682.

¹⁹⁶ *Id.* at 18,670.

¹⁹⁷ *Id.* at 18,681.

¹⁹⁸ *Id.*

¹⁹⁹ *Id.* at 18,683.

systems with initial PFAS detections below EPA’s proposed trigger level of one-third the relevant MCL.²⁰⁰

The SDWA requires NPDWRs to include monitoring requirements that will “[e]nsure compliance” with the relevant MCLs.²⁰¹ EPA’s proposal does not satisfy this requirement insofar as it gives primacy agencies discretion to reduce required compliance monitoring to just 1–2 monitoring events per three-year compliance period if a system’s initial year of monitoring documents PFAS concentrations below the proposed trigger level.²⁰² Indeed, EPA’s proposal does not attempt to demonstrate that this reduced monitoring scheme would be adequate to “insure” compliance with the MCLs,²⁰³ claiming instead that its proposal would “save resources” for purportedly “lower-risk water systems.”²⁰⁴ As explained below, existing PFAS monitoring data, as well as literature on PFAS toxicity and fluctuations in drinking water sources, indicate that EPA’s proposal must be strengthened to ensure compliance with the MCLs.

Available data from PWSs that have tested for PFAS on a quarterly or more frequent basis demonstrate significant variation in PFAS detections and measured concentrations, which indicates that consistent monitoring is needed to ensure that PFAS levels remain below the MCLs.²⁰⁵ For example, the Merrimack Village Water District reported non-detect results for PFOS at the Turkey Hill Road location within its drinking water distribution system during four sampling events between December 2, 2021, and July 27, 2022, then detected 15.20 ppt of PFOS during a subsequent sampling event on October 19, 2022, followed by another non-detect result on January 25, 2023.²⁰⁶ Monitoring data from industrial PFAS dischargers likewise demonstrates the potential for significant intra-annual variation in PFAS discharges to drinking water sources, which will in turn impact the levels of PFAS in finished drinking water from systems that have not installed PFAS treatment technology.²⁰⁷ Peer-reviewed literature also documents significant

²⁰⁰ *Id.* at 18,681.

²⁰¹ 42 U.S.C. § 300f(1)(D).

²⁰² *See* Proposed Rule, 88 Fed. Reg. at 18,681; Economic Analysis at 5-32.

²⁰³ 42 U.S.C. § 300f(1)(D).

²⁰⁴ Proposed Rule, 88 Fed. Reg. at 18,682.

²⁰⁵ *See, e.g.,* Merrimack Vill. Dist. Water Works, *Historical Charts for PFAS Water Sampling Test Results*, <https://www.mvdwater.org/historical-water-sampling-charts/>, and *PFAS Results, Distribution Sys.*, <https://www.mvdwater.org/wp-content/uploads/2023/03/PFAS-Distribution-System-02-15-2023.pdf> (distribution system notes); City of Ann Arbor, Mich., *Drinking Water Sampling Data*, <https://www.a2gov.org/departments/water-treatment/Documents/PFAS-forweb-RESERVIOR-031523.pdf> (updated Mar. 15, 2023); Orange Water and Sewer Auth., *Trends in PFAS Detections in Finished Drinking Water (Quarterly Sample Results)*, *Detections in Drinking Water*, <https://www.owasa.org/pfas-monitoring-program/> (scroll down to the “Complete Set of Historical PFAS Monitoring Data” and select “Raw Data.” Review table on upper left corner titled “Finished Drinking Water”).

²⁰⁶ Merrimack Vill. Water Dist. Water Works, *PFAS Results, Distribution Sys.*, at 8.

²⁰⁷ *See, e.g.,* Colo. Dep’t of Public Health & Env’t, *Suncor Water Quality Permits, Surface Water Suncor PFAS Data (Outfall 20)*, <https://cdphe.colorado.gov/suncor-water-quality-permits> (updated Apr. 2023) (scroll down to the section titled “Resources and Pollution Data” and select

intra-annual variation in PFAS concentrations in both source water and finished drinking water due to factors including variable flow rates, variation in industrial processes/production cycles, variable stormwater runoff from contaminated sites, mobilization of legacy PFAS contamination in sediment or groundwater, and the potential introduction of new sources of PFAS contamination.²⁰⁸ Taken together, this evidence indicates that allowing compliance monitoring as infrequently as 1–2 times per three-year compliance period may mask dangerous PFAS concentrations in monitored drinking water and potential violations of the MCLs.

Further, as EPA acknowledges, PFAS subject to the proposed NPDWRs can pose health risks at concentrations substantially lower than the proposed trigger values. Indeed, in the Proposed Rule EPA acknowledges that PFOA and PFOS can cause adverse health effects at “near zero” levels.²⁰⁹ While EPA has previously justified significant monitoring reductions on the grounds that “analytical results . . . below the MCL” for the contaminants at issue “do not pose a health threat,”²¹⁰ that logic is demonstrably inapposite for PFAS. Under EPA’s proposal, exceedances of one or more PFAS MCL(s) could persist for years before they are detected and treatment is required, posing significant health risks for people served by the affected water system. And in situations where a water system has detectable PFAS concentrations below the proposed trigger values, EPA’s proposal would deprive the public of information relevant to assessing health risks from consuming that PFAS-contaminated drinking water.²¹¹

Finally, as discussed above, EPA’s proposal to allow reduced monitoring based on PFAS detections below the trigger values, which in some cases are substantially below the relevant PQL, is inconsistent with EPA’s proposal to zero-out all detections below the PQL for purposes of demonstrating MCL compliance. This approach is also inconsistent with EPA’s assertion that detections “at the proposed rule trigger level” are “primarily useful in determining whether the contaminant is present in a sample . . . rather than to determine its specific concentration.”²¹²

To address these issues, EPA should modify its proposal to (1) set the trigger value at the MDL where that value is lower than one-third of the MCL, and (2) provide that systems with four consecutive quarters of non-detects for the 6 PFAS may reduce to *annual* monitoring.

“Toxic firefighting foam chemicals (PFAS)” from the drop down menu. Select and view “Surface water Suncor PFAS data (Outfall 20)”.

²⁰⁸ See Minh A. Nguyen et al. *Seasonal Trends of Per- and Polyfluoroalkyl Substances in River Water Affected by Fire Training Sites and Wastewater Treatment Plants*, 308 *Chemosphere* Art. No. 136467 (2022), <https://doi.org/10.1016/j.chemosphere.2022.136467>; M.-A. Pétré et al., *Per- and Polyfluoroalkyl Substances (PFAS) in River Discharge: Modeling Loads Upstream and Downstream of a PFAS Manufacturing Plant in the Cape Fear Watershed, North Carolina*, 831 *Sci. of the Total Env’t* Art. No. 154763 (2022), <https://doi.org/10.1016/j.scitotenv.2022.154763>.

²⁰⁹ Proposed Rule, 88 Fed. Reg. at 18,715.

²¹⁰ 56 Fed. Reg. at 3,526, 3,562.

²¹¹ See 42 U.S.C. 300j-4(g)(6) (providing for inclusion in public database of “information on the detection of [regulated] contaminant[s] at a quantifiable level in public water systems (including detection of the contaminant at levels not constituting a violation of the maximum contaminant level for the contaminant).”).

²¹² Proposed Rule, 88 Fed. Reg. at 18,681–82.

Lowering the trigger value would better align the proposal with EPA's Standardized Monitoring Framework for Synthetic Organic Compounds²¹³ as well as monitoring requirements in state-level PFAS MCLs.²¹⁴ Allowing annual, instead of triennial, monitoring for PWSs with consistent detections below the trigger value also would align the federal requirements with existing requirements in multiple states.²¹⁵

D. EPA's Proposed Tier 2 Designation for Violations of its PFAS MCLs Does Not Account for Acute Toxicity and Must be Amended

To “ensure[] that consumers will know if there is a problem with their drinking water,” the SDWA requires “each owner or operator of a public water system” to give notice to consumers of all violations of a NPDWR through public notice.²¹⁶ “The public notice requirements for each violation or situation,” “are determined by the tier to which [the violation or situation] is assigned.”²¹⁷

Tier 1 notice is required for violations and situations “with significant potential to have serious adverse effects on human health as a result of short-term exposure” and must be provided as soon as practical but no later than 24 hours after the system learns of the violation.²¹⁸ Tier 2 notice applies to all violations and situations not designated as Tier 1 but which have the “potential to have serious adverse effects on human health” and must be provided as soon as practical but no later than 30 days after the system learns of the violation.²¹⁹ Finally, Tier 3 notice is required for all NPDWR violations and situations not included in Tier 1 and Tier 2 and must be provided “not later than one year after the public water system learns of the violation or situation or begins operating under a variance or exemption.”²²⁰

In the Proposed Rule, EPA proposes designating violations of the MCLs for the Six PFAS as requiring “Tier 2” public notice.²²¹ This proposal ignores the acute toxicity of the Six PFAS, and EPA must modify its proposal to require Tier 1 public notice for violations of all the proposed MCLs so consumers can be informed of the potential for significant harm in a timely manner.

²¹³ See 40 C.F.R. § 141.24(f)(11)(i) (requiring quarterly monitoring for organic contaminants detected above 0.0005 mg/L).

²¹⁴ See N.J. Admin. Code § 7:10-5.2(a)(5)(i)(2), (ii)(2) (requiring quarterly monitoring for PFOA, PFOS, and PFNS when detected above 0.002 ppt); N.Y. Comp. Codes R. & Regs. tit. 10 § 5-1.52 (establishing MDL as trigger value for reduced PFAS monitoring).

²¹⁵ See N.J. Admin. Code § 7:10-5.2(a)(5)(ii)(3), (iii)(3); N.Y. Comp. Codes R. & Regs. tit. 10 § 5-1.52; Mich. Admin. Code R 325.10717d(9), (11).

²¹⁶ Proposed Rule, 88 Fed. Reg. at 18,684; 40 C.F.R. § 141.201(a), (c).

²¹⁷ 40 C.F.R. § 141.201(b).

²¹⁸ *Id.* §§ 141.202(a)(9), 141.202(b)(1).

²¹⁹ *Id.* §§ 141.201(b), 141.203(b).

²²⁰ *Id.* § 141.204(b).

²²¹ Proposed Rule, 88 Fed. Reg. at 18,684.

EPA’s proposal “that violations of the three MCLs . . . be designated as Tier 2” for purposes of the public notification rule ignores scientific evidence establishing a link between serious adverse health effects and short-term exposure to the Six PFAS.²²² EPA acknowledges that exposure to PFOA and PFOS “may have an adverse effect on the health of persons” and that PFHxS, GenX, PFNA, and PFBS “may individually and in a mixture each result in adverse health effects, including disrupting multiple biological pathways that result in common adverse effects on several biological systems including the endocrine, cardiovascular, developmental, immune, and hepatic systems.”²²³ However, while EPA acknowledges that these PFAS can cause serious harms, its analysis focuses significantly on chronic harms at the expense of a careful analysis of the harms these PFAS pose in the short-term.²²⁴ As a result of the short shrift EPA gave to acute toxicity studies, it has mis-designated the violation of the proposed MCLs for public notification purposes.

The Six PFAS all pose short-term health harms. Acute and short-term health effects for PFOA, PFOS, PFHxS, and PFNA are summarized in ATSDR’s Toxicological Profile for Perfluoroalkyls.²²⁵ Acute PFOA exposure is associated with liver, immunological, reproductive, and developmental effects.²²⁶ Acute PFOS exposure is associated with liver, immunological, and developmental effects.²²⁷ Acute PFNA exposure is associated with liver and immunological effects as well as changes in body weight.²²⁸

Acute exposure to PFOS has also been found to affect the plasticity of brain synapses, creating neurotoxic harm,²²⁹ and damage the liver.²³⁰ For PFOA, one study found that “cellular effects exerted after 24 h[our] exposure to perfluorooctanoic acid are non-reversible after a 48 h[our] recovery period.”²³¹ Another study found that acute exposure to PFOA can “disrupt[] key hormones in the pancreas” and “induce[] lipid accumulation in the liver.”²³² Acute exposure to

²²² *Id.* “The proposed rule also designates monitoring and testing procedure violations as Tier 3[.]” *Id.* at 18,699.

²²³ *Id.* at 18,644–45.

²²⁴ *Id.* at 18,645–46 (citing studies of mice dosed for 42–44 days or 53–64 days, for example).

²²⁵ ATSDR 2021 at 62, 84, 88–99.

²²⁶ *Id.* at 62.

²²⁷ *Id.* at 84.

²²⁸ *Id.* at 88–90.

²²⁹ Qian Zhang et al., *Effects Of Perfluorooctane Sulfonate and its Alternatives on Long-Term Potentiation in The Hippocampus CA1 Region if Adult Rats in vivo*, 5 Toxicology Rsch. 539, 539–546 (2016), doi: [10.1039/c5tx00184f](https://doi.org/10.1039/c5tx00184f).

²³⁰ Jiali Xing, *Toxicity Assessment Of Perfluorooctane Sulfonate Using Acute and Subchronic Male C57BL/6J Mouse Models*, 210 Env’t Pollution 388–96 (2016), <https://pubmed.ncbi.nlm.nih.gov/26807985/>.

²³¹ Peropadre et al., *An Acute Exposure to Perfluorooctanoic Acid Causes Non-Reversible Plasma Membrane Injury in Hela Cells*, 260 Env’t Pollution Art. No. 11400 (2020), <https://pubmed.ncbi.nlm.nih.gov/31995777/>.

²³² Xinmou Wu et al., *Effect of Acute Exposure to PFOA On Mouse Liver Cells In Vivo And In Vitro*, 24 Env’t Sci and Pollution Rsch. Int’l 24201, 24203 (2017), <https://pubmed.ncbi.nlm.nih.gov/28887612/>.

PFNA has been found to impair reproductive health,²³³ and negatively impact liver functionality in diabetic mice.²³⁴ A study on PFHxS found that “a single neonatal exposure to PFHxS can cause irreversible neurotoxic effects in mice.”²³⁵ PFHxS has also been found to pose similar brain development concerns as PFOS in response to acute exposure.²³⁶ PFBS has been found to have an effect on the liver which “may represent an acute response to the chemical at a high dose.”²³⁷ Additional acute studies of PFBS are identified in the Toxicity Assessment for PFBS.²³⁸

EPA’s Toxicity Assessment for GenX reviews 10 studies for acute toxicity and four studies for short term toxicity (seven-day dosing).²³⁹ Since the publication of that assessment, additional acute and short-term toxicity studies have been published. For example, Cannon et al. found that a single dose of GenX administered by oral gavage caused decreases in P-glycoprotein (P-gp) transport activity and breast cancer resistance protein (BCRP) transport

²³³ Shilpi Singh & Shio Kumar Singh, *Acute Exposure To Perfluorononanoic Acid in Prepubertal Mice: Effect on Germ Cell Dynamics and an Insight into the Possible Mechanisms of its Inhibitory Action on Testicular Functions*, 183 *Ecotoxicology Env’t Safety* Art No. 109499, 1667 (2019), <https://pubmed.ncbi.nlm.nih.gov/31398581/>; Shilpi Singh & Shio Kumar Singh, *Prepubertal Exposure to Perfluorononanoic Acid Interferes with Spermatogenesis and Steroidogenesis in Male Mice*, 170 *Ecotoxicology Env’t Safety* 590, 598 (2019), <https://pubmed.ncbi.nlm.nih.gov/30576894/>; Shilpi Singh & Shio Kumar Singh, *Effect of Gestational Exposure to Perfluorononanoic Acid On Neonatal Mice Testes*, 39 *J. of Applied Toxicology* 1663, 1665 (2019), <https://pubmed.ncbi.nlm.nih.gov/31389053/>; Yixing Feng et al., *Effects of PFNA Exposure on Expression of Junction-Associated Molecules and Secretory Function in Rat Sertoli Cells*, 30 *Reprod. Toxicology* 429–37 (2010), <https://pubmed.ncbi.nlm.nih.gov/20580666/>.

²³⁴ Fang X, *Perfluorononanoic Acid Disturbed the Metabolism of Lipid in the Liver of Streptozotocin-Induced Diabetic Rats*, 25 *Toxicology Mechanisms & Methods* 622, 626 (2015), <https://pubmed.ncbi.nlm.nih.gov/26056853/>.

²³⁵ Henrick Viberg et al., *Adult Dose-Dependent Behavioral and Cognitive Disturbances After a Single Neonatal PFHXS Dose*, 304 *Toxicology* 185–91 (2013) <https://pubmed.ncbi.nlm.nih.gov/23287389/>.

²³⁶ Qian Zhang et al., *Effects of Perfluorooctane Sulfonate and its Alternatives on Long-Term Potentiation in The Hippocampus CA1 Region of Adult Rats*, at 539 (“In addition, PFHxS and Cl-PFAES exhibited comparable potential to PFOS in disturbing LTP.”).

²³⁷ Lau et al., *Pharmacokinetic Profile of Perfluorobutane Sulfonate and Activation of Hepatic Nuclear Receptor Target Genes in Mice*, 441 *Toxicology* 152522 (2020), <https://pubmed.ncbi.nlm.nih.gov/32534104/>.

²³⁸ EPA, *Human Health Toxicity Values for Perfluorobutane Sulfonic Acid (CASRN 375-73-5) and Related Compound Potassium Perfluorobutane Sulfonate (CASRN 29420-49-3)* (2021) at 50–51.

²³⁹ EPA, *Human Health Toxicity Values for Hexafluoropropylene Oxide (HFPO) Dimer Acid and Its Ammonium Salt (CASRN 13252-13-6 and CASRN 62037-80-3) Also Known as ‘GenX Chemicals’* at 35-37.

activity in the brain capillaries of exposed rats.²⁴⁰ Further, a pair of studies by Conley et al. exposed rats from gestation day 14–18 or gestation day 17–21 respectively (*i.e.*, 5 days of exposure) and found effects in both the dosed mothers and developing fetuses.²⁴¹ In animals exposed from gestation day 14–18, fetal and maternal livers had increased expression of genes in the PPAR signaling pathway, and rats exposed to GenX gained less weight during their pregnancy and had larger livers than unexposed animals.²⁴² Results were similar for animals exposed from gestation day 17–21.²⁴³ Additionally, Blake et al. (2023) exposed CD-1 mice from gestation day 1.5 to 11.5 or 17.5 (*i.e.* 10 days or 17 days of exposure) and found that exposed mothers gained more weight during the dosing period, had larger livers that showed “abnormal ultrastructure with enlarged hepatocytes,” had larger kidneys and altered blood clinical chemistry, including increased cholesterol, HDL, and ALT levels.²⁴⁴ GenX exposed fetuses had a larger embryo:placenta weight ratio and increased placental lesions.²⁴⁵

These health risks are significant and require swift public notice so consumers can take immediate action to protect themselves from exposure. Accordingly, EPA should adjust the proposed public notice designation for violations of the PFAS MCLs from Tier 2 to Tier 1 in the final rule.

Conclusion

For the millions of people with PFAS in their tap water, strong federal drinking water standards are essential and long overdue. We appreciate EPA’s leadership in developing this proposed rule, and we urge EPA to resist efforts to weaken its proposal. We further urge EPA to revise its proposed HBWCs and to incorporate the changes outlined above. Finally, EPA should move quickly to finalize this rule and to pursue s for the PFAS that are not addressed in the proposed rule.

²⁴⁰ Cannon et al., *Effect of GenX on P-Glycoprotein, Breast Cancer Resistance Protein, and Multidrug Resistance-Associated Protein 2 at the Blood-Brain Barrier*, 128 Env’t Health Persp. 37002 (March 2020), <https://doi.org/10.1289/EHP5884>.

²⁴¹ Conley et al., *Adverse Maternal, Fetal, and Postnatal Effects of Hexafluoropropylene Oxide Dimer Acid (GenX) from Oral Gestational Exposure in Sprague-Dawley Rats*, 127 Env’t Health Persp. 037008 (Mar 2019), <https://doi.org/10.1289/EHP4372> (“Conley 2019”); Conley et al., *Hexafluoropropylene Oxide-Dimer Acid (HFPO-DA or GenX) Alters Maternal and Fetal Glucose and Lipid Metabolism and Produces Neonatal Mortality, Low Birthweight, and Hepatomegaly in the Sprague-Dawley Rat*, 146 Env’t Int’t 106204 (Jan. 2021) <https://doi.org/10.1016/j.envint.2020.106204> (“Conley 2021”).

²⁴² Conley 2019 at 037008-6 to 037008-8.

²⁴³ Conley 2021 at 106204-4 to 106204-10.

²⁴⁴ Blake et al., *Evaluation of Maternal, Embryo, and Placental Effects in CD-1 Mice Following Gestational Exposure to Perfluorooctanoic Acid (PFOA) or Hexafluoropropylene Oxide Dimer Acid (HFPO-DA or GenX)*, 128 Env’t Health Persp. 128 (Feb. 2020), <https://doi.org/10.1289/EHP6233>.

²⁴⁵ *Id.*

If you have any questions about these comments, please contact Jon Kalmuss-Katz (jkalmusskatz@earthjustice.org) or Katherine O'Brien (kobrien@earthjustice.org) at Earthjustice or Erik Olson (eolson@nrdc.org), Anna Reade (areade@nrdc.org) or Katherine Pelch (kpelch@nrdc.org) at the Natural Resources Defense Council.

Respectfully submitted,

Alaska Community Action on Toxics
Alliance for the Great Lakes
Buckeye Environmental Network
Center for Biological Diversity
Clean Cape Fear
Clean Water Action
Delaware Riverkeeper Network
Earthjustice
Elevate Energy
Environmental Advocates NY
Environmental Defense Fund
Environmental Justice Task Force -Tucson
Environmental Working Group
Fight for Zero
Green Science Policy Institute
Holy Spirit Missionary Sisters, USA-JPIC
Lawyers for Good Government
Merrimack Citizens for Clean Water
Mountain Watershed Association
National PFAS Contamination Coalition
Natural Resources Defense Council
North Carolina Conservation Network
Ohio Environmental Council
Ohio River Foundation
Passaic River Coalition
People Over Petro Coalition
PfoaProject NY
Religious Coalition for the Great Lakes
Save The River, Upper St. Lawrence Riverkeeper
Save Our Sky Blue Waters
Sierra Club
Toxic Free NC
Union of Concerned Scientists
Vermont Natural Resources Council
Waterkeeper Alliance
Zero Waste Washington

Exhibit A



May 30, 2023

Via Regulations.gov

Assistant Administrator Radhika Fox
Office of Water
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, DC 20460

Re: PFAS National Primary Drinking Water Regulation Rulemaking, Docket No. EPA-HQ-OW-2022-0114

Dear Assistant Administrator Fox:

While EPA's proposed National Primary Drinking Water Standards acknowledge a broad range of adverse health effects from PFAS exposures, EPA has not fully accounted for those effects, or the corresponding benefits of the proposed regulation, in its analysis of the rule's economic impacts.¹ This document summarizes several of the health effects that EPA failed to quantify and provides resources and information that EPA should use to estimate additional benefits of the proposed drinking water standards.

Together, we have extensive experience reviewing the health and toxicological effects associated with PFAS exposure. As co-leads on the development of the PFAS-Tox Database, we have reviewed over 1,000 studies that evaluate the health impact of PFAS exposure.² We have also

¹ Preliminary Regulatory Determination and Proposed Rule, PFAS National Primary Drinking Water Regulation Rulemaking, 88 Fed. Reg. 18,638 (Mar. 29, 2023) (the “Proposed Rule”). The six PFAS covered by the Proposed Rule are perfluorooctanoic acid (“PFOA”), perfluorooctane sulfonic acid (“PFOS”), perfluorohexane sulfonic acid (“PFHxS”), hexafluoropropylene oxide dimer acid and its ammonium salt (“GenX”), perfluorononanoic acid (“PFNA”), and perfluorobutane sulfonic acid (“PFBS”) (collectively, the “Six PFAS”).

² Katherine E. Pelch, Anna Reade, Carol F. Kwiatkowski, Francheska M. Merced-Nieves, Haleigh Cavalier, Kim Schultz, Taylor Wolffe, and Julia Varshavsky, *The PFAS-Tox Database: A Systematic Evidence Map of Health Studies on 29 per- and Polyfluoroalkyl Substances*, Environment International 167 (September 1, 2022): 107408, <https://doi.org/10.1016/j.envint.2022.107408>; Katherine E. Pelch, Anna Reade, Taylor A. M. Wolffe, and Carol F. Kwiatkowski, *PFAS Health Effects Database: Protocol for a Systematic Evidence Map*, Environment International 130 (September 1, 2019): 104851,

provided public comment on ATSDR's "Toxicological Profile for Perfluoroalkyls", EPA's PFAS toxicity assessments for PFOA, PFOS, PFBA, PFHxA, GenX, and PFBS, and Health Canada's Draft Objective for Drinking Water.³ We have also critically evaluated and commented on health and risk assessment documents for PFAS developed by California, Illinois, Michigan, New Hampshire, New Jersey, New York, Vermont, and Washington.⁴ These activities inform

<https://doi.org/10.1016/j.envint.2019.05.045>; Katherine E. Pelch and Carol F. Kwiatkowski, *Invited Perspective: The Promise of Fit-for-Purpose Systematic Evidence Maps for Supporting Regulatory Health Assessment*, *Environmental Health Perspectives* 130, no. 5 (May 2022): 051303, <https://doi.org/10.1289/EHP10743>.

³ Anna Reade, *Comments on ATSDR Toxicological Profile on Perfluoroalkyls 2018 Draft*, September 6, 2018. https://www.nrdc.org/sites/default/files/comments-on-atsdr-toxicological-profile-on-perfluoroalkyls-2018-draft_2018-08-21.pdf; Katherine Pelch, *Technical comments to the Science Advisory Board on the U.S. Environmental Protection Agency external peer review draft: Proposed approaches to the derivation of a draft maximum contaminant level goal for perfluorooctanoic acid (PFOA) (CASRN 335-67-1) in drinking water and External peer review draft: proposed approaches to the derivation of a draft maximum contaminant level goal for perfluorooctane sulfonic acid (PFOS) (CASRN 1763-23-1) in drinking water*, December 23, 2021; Katherine Pelch and Anna Reade, *Comments on EPA's Draft Toxicological Review for Perfluorobutanoic Acid (PFBA)*, November 8, 2021; Katherine Pelch, *Comments on EPA's Draft Toxicological Review for Perfluorohexanoic Acid (PFHxA)*, April 4, 2022; Katherine E. Pelch, Anna Reade, Sonya Lunder, David Q. Andrews, and Ansje Miller, *Comments on EPA's Draft Toxicity Assessments for Perfluorobutane Sulfonic Acid (PFBS) and Hexafluoropropylene Oxide (or GenX Chemicals)*, January 22, 2019. <https://www.nrdc.org/sites/default/files/comments-assessments-of-pfbs-and-genx-01222019.pdf>; Katherine E. Pelch and Anna Reade, *RE: Draft Objective for per- and Polyfluoroalkyl Substances in Canadian Drinking Water*, April 12, 2023.

⁴ Anna Reade, Avinash Kar, and Katherine E. Pelch, *Technical Comments RE: Consideration of Perfluorooctane Sulfonic Acid (PFOS) and Its Salts and Transformation and Degradation Precursors for Possible Listing under Proposition 65 Based on Carcinogenicity*, November 2021; Anna Reade, Avinash Kar, and Andria Ventura, *Comments RE: Consideration of Perfluorononanoic Acid (PFNA) and Perfluorodecanoic Acid (PFDA) and Their Salts for Possible Listing under Proposition 65 Based on Developmental Reproductive Toxicity*, November 15, 2021; Anna Reade, Katherine E. Pelch, Nicole Saulsberry, and Iyana Simba, *Technical Comments Re 35 Ill. Adm. Code 620; Groundwater Quality Pre-Filing Public Comment Period. Joint Comments by Natural Resources Defense Council, Illinois Environmental Council and Sierra Club, Illinois Chapter*, June 5, 2021; Anna Reade, Tracy Quinn, Judith S Schreiber, and Schreiber Scientific, *Scientific and Policy Assessment for Addressing Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water*, March 15, 2019, <https://www.nrdc.org/sites/default/files/assessment-for-addressing-pfas-chemicals-in-michigan-drinking-water.pdf>; Anna Reade and Cyndi Roper, *Comments of the Natural Resources Defense Council on the Michigan Department of Environment, Great Lakes, and Energy's Proposed PFAS MCLs Pending Rule Set: 2019-35-EG*, January 31, 2020; Katherine E. Pelch and Carol F. Kwiatkowski, *Comments on New Hampshire Department of Environmental Services Proposed Rulemaking to Set Public Drinking Water and Groundwater Standards for PFOA, PFOS, PFNA, & PFHxS (Env-Dw 700-800 and Env-Or 603.03)*, November 7, 2018; Katherine E. Pelch, *Comments Re: Setting Public Drinking Water and Groundwater Standards for PFOA, PFOS,*

our additional comments regarding EPA’s proposed National Primary Drinking Water Standards, specifically identifying health effects that should have been better incorporated into the economic benefits analysis.

I. Lactation duration⁵

EPA has not fully considered the impact of PFAS exposure on mammary gland development and function, and specifically on lactation duration. Given the importance of breastfeeding and its association with many other health impacts, this is a major oversight. Breastfeeding is associated with short- and long-term health benefits for both mother and child, but <30% of mothers in the U.S. continue any breastfeeding until the American Academy of Pediatrics (AAP) recommended 12 months.⁶ The benefits of human milk for children are well described, with health benefits extending into adulthood.⁷ Potential health benefits of lactation for the mother are often described with the “reset” hypothesis, whereby the adverse cardiometabolic changes during gestation (insulin resistance, hyperlipidemia, and visceral fat of pregnancy) are ameliorated by breastfeeding. In contrast, without breastfeeding, these metabolic changes persist.⁸ Meta-

PFNA, & PFHxS (Env-Dw 700-800; Env-Or 603.03), April 12, 2019; Katherine E. Pelch and Carol F. Kwiatkowski, *Comments Re: Setting Public Drinking Water Standards for PFOA and PFOS*, December 21, 2018; Anna Reade, Tracy Quinn, and Judith S Schreiber, *Comments Re: Proposed Maximum Contaminant Level for Perfluorooctanoic Acid (PFOA) and Perfluorooctanesulfonic Acid (PFOS)*, DEP Dkt. No. 02-19-03, May 31, 2019, <https://www.nrdc.org/sites/default/files/pfas-comments-nj-05312019.pdf>; Anna Reade, Katie Pelch, Miriam Rotkin-Ellman, and Erik D. Olson, *Comments to New York State Governor Hochul and New York State Commissioner Bassett on the Need to Establish Comprehensive, and Health Protective, Drinking Water Standards for PFAS*, September 9, 2022; Anna Reade and Katherine E. Pelch, *Technical Comments Re: Advance Notice on the Regulation of Perfluoroalkyl, Polyfluoroalkyl Substances (PFAS) as a Class*, November 16, 2022, <https://www.nrdc.org/sites/default/files/pfas-class-technical-comments-20201116.pdf>; Erika Schreder and Katherine E. Pelch, *Comments on Washington Department of Health’s Draft Recommended State Action Levels for per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water: Approach, Methods and Supporting Information (Chapter 246-290 WAC)* January 2020.

⁵ We acknowledge helpful technical support and feedback from Dr. Megan Romano in the development of this section.

⁶ Arthur I. Eidelman, Richard J. Schanler, Margreete Johnston, Susan Landers, Larry Noble, Kinga Szucs, and Laura Viehmann, *Breastfeeding and the Use of Human Milk*, Pediatrics 12, no. 3 (March 2012): e827–41, <https://doi.org/10.1542/peds.2011-3552>; CDC, *Results: Breastfeeding Rates*, Centers for Disease Control and Prevention, April 4, 2023, https://www.cdc.gov/breastfeeding/data/nis_data/results.html.

⁷ Stanley Ip, Mei Chung, Gowri Raman, Priscilla Chew, Nombulelo Magula, Deirdre DeVine, Thomas Trikalinos, and Joseph Lau, *Breastfeeding and Maternal and Infant Health Outcomes in Developed Countries*, Evidence Report/Technology Assessment, no. 153 (April 2007): 1–186, <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4781366/>.

⁸ Alison M. Stuebe and Janet W. Rich-Edwards, *The Reset Hypothesis: Lactation and Maternal Metabolism*, American Journal of Perinatology 26, no. 1 (January 2009): 81–88, <https://doi.org/10.1055/s-0028-1103034>.

analyses with over 200,000 women confirmed relationships between breastfeeding for 12 months and protection against common adverse cardiometabolic health outcomes, including a 30% risk reduction for diabetes and a 13% risk reduction for hypertension.⁹

Importantly, shortened duration of breastfeeding has been associated with PFAS exposure in human studies. Six human studies, published between 2010 and 2022 were recently reviewed and evaluated in a meta-analysis.¹⁰ Four of the five included studies reported shortened total duration of breastfeeding with higher PFOS and PFOA exposure. The human epidemiological findings are consistent with findings from experimental animal studies. Despite these consistencies and the importance of breastfeeding duration on maternal and infant health, EPA failed to adequately review and consider shortened lactation duration in the 2023 Draft Toxicity Assessment and Proposed Maximum Contaminant Level Goal for Perfluorooctanoic Acid (PFOA) in Drinking Water. In that document, EPA reviewed the animal evidence for impacts on mammary gland development and function but did not evaluate the corresponding epidemiological evidence.¹¹

The 2023 Draft Toxicity Assessment and Proposed Maximum Contaminant Level Goal for Perfluorooctanoic Acid (PFOA) in Drinking Water cites but does not thoroughly discuss two of the epidemiological studies that are included in the recent meta-analysis on breastfeeding (Timmerman et al. 2017 and Romano et al. 2016)¹², and fails to cite or discuss the four additional studies, including those published before the review cut-off date (Fei et al., 2010, Nielsen et al.,

⁹ Rabel Misbah Rameez, Divyajot Sadana, Simrat Kaur, Taha Ahmed, Jay Patel, Muhammad Shahzeb Khan, Sarah Misbah, Marian T. Simonson, Haris Riaz, and Haitham M. Ahmed, *Association of Maternal Lactation With Diabetes and Hypertension: A Systematic Review and Meta-Analysis*, JAMA Network Open 2, no. 10 (October 16, 2019): e1913401, <https://doi.org/10.1001/jamanetworkopen.2019.13401>.

¹⁰ Amalie Timmermann, Oyemwenosa N. Avenbuan, Megan E. Romano, Joseph M. Braun, Janne S. Tolstrup, Laura N. Vandenberg, and Suzanne E. Fenton, *Per- and Polyfluoroalkyl Substances and Breastfeeding as a Vulnerable Function: A Systematic Review of Epidemiological Studies*, Toxics 11, no. 4 (April 2023): 325, <https://doi.org/10.3390/toxics11040325>.

¹¹ US EPA, *Public Comment Draft Toxicity Assessment and Proposed Maximum Contaminant Level Goal for Perfluorooctanoic Acid (PFOA) in Drinking Water*, Office of Water, March 14, 2023, https://www.epa.gov/system/files/documents/2023-03/MAIN_Proposed%20MCLG%20for%20PFOA%20in%20Drinking%20Water_3.9.23_For%20Proposal.pdf.

¹² Clara Amalie Gade Timmermann, Esben Budtz-Jørgensen, Maria Skaalum Petersen, Pál Weihe, Ulrike Steuerwald, Flemming Nielsen, Tina Kold Jensen, and Philippe Grandjean, *Shorter Duration of Breastfeeding at Elevated Exposures to Perfluoroalkyl Substances*, Reproductive Toxicology, Developmental Origins of Disease, 68 (March 1, 2017): 164–70, <https://doi.org/10.1016/j.reprotox.2016.07.010>; Megan E. Romano, Yingying Xu, Antonia M. Calafat, Kimberly Yoltson, Aimin Chen, Glenys M. Webster, Melissa N. Eliot, Cynthia R. Howard, Bruce P. Lanphear, and Joseph M. Braun, *Maternal Serum Perfluoroalkyl Substances during Pregnancy and Duration of Breastfeeding*, Environmental Research 149 (August 1, 2016): 239–46, <https://doi.org/10.1016/j.envres.2016.04.034>.

2022, Rosen et al., 2018, and Timmerman et al., 2021).¹³ None of the epidemiological studies are cited or discussed in the 2016 Drinking Water Health Advisory for Perfluorooctanoic Acid (PFOA).¹⁴ Though the animal literature is discussed in the document, the 2016 Drinking Water Health Advisory for PFOA did not consider any candidate reference doses (RfDs) based on mammary gland effects.

Perhaps EPA's failure to adequately consider mammary gland and lactational effects in the 2023 toxicity assessment is, in part, a result of mammary gland impacts being improperly diminished in earlier documents. Evidence for this can be seen in the conclusions EPA makes in the 2023 toxicity assessment. In the 2023 Draft Toxicity Assessment and Proposed Maximum Contaminant Level Goal for Perfluorooctanoic Acid (PFOA) in Drinking Water document EPA states, "no differences in response to a lactation challenge were seen in PFOA-exposed CD-1 mouse dams with delayed mammary gland development, and no significant effects on body weight gain were seen in pups nursing from dams with less fully developed mammary glands (White, 2011, 1276150)."¹⁵ Similarly, ATSDR stated "... the mammary gland effect did not result in an adverse effect on lactational support at maternal doses as high as 1 mg/kg/day, based on normal growth and survival in F2 pups (White et al. 2011). Given that milk production was adequate to support growth, the biological significance of the delayed development of the

¹³ Chunyuan Fei, Joseph K. McLaughlin, Loren Lipworth, and Jørn Olsen, *Maternal Concentrations of Perfluorooctanesulfonate (PFOS) and Perfluorooctanoate (PFOA) and Duration of Breastfeeding*, Scandinavian Journal of Work, Environment & Health 36, no. 5 (September 2010): 413–21, <https://doi.org/10.5271/sjweh.2908>; Christel Nielsen, Ying Li, Magdalena Lewandowski, Tony Fletcher, and Kristina Jakobsson, *Breastfeeding Initiation and Duration after High Exposure to Perfluoroalkyl Substances through Contaminated Drinking Water: A Cohort Study from Ronneby, Sweden*, Environmental Research 207 (May 1, 2022): 112206, <https://doi.org/10.1016/j.envres.2021.112206>; Emma M. Rosen, Anne Lise Brantsæter, Rachel Carroll, Line S. Haug, Alison B. Singer, Shanshan Zhao, and Kelly K. Ferguson, *Maternal Plasma Concentrations of Per- and Polyfluoroalkyl Substances and Breastfeeding Duration in the Norwegian Mother and Child Cohort*, Environmental Epidemiology (Philadelphia, Pa.) 2, no. 3 (September 2018): e027, <https://doi.org/10.1097/EE9.0000000000000027>; Clara Amalie Gade Timmermann, Marianne Skovsager Andersen, Esben Budtz-Jørgensen, Henriette Boye, Flemming Nielsen, Richard Christian Jensen, Signe Bruun, Steffen Husby, Philippe Grandjean, and Tina Kold Jensen, *Pregnancy Exposure to Perfluoroalkyl Substances and Associations With Prolactin Concentrations and Breastfeeding in the Odense Child Cohort*, The Journal of Clinical Endocrinology and Metabolism 107, no. 2 (September 13, 2021): e631–42, <https://doi.org/10.1210/clinem/dgab638>.

¹⁴ US EPA, *Drinking Water Health Advisory for Perfluorooctanoic Acid (PFOA)*, May 2016, https://www.epa.gov/sites/production/files/2016-05/documents/pfoa_health_advisory_final-plain.pdf.

¹⁵ US EPA, *Public Comment Draft Toxicity Assessment and Proposed Maximum Contaminant Level Goal for Perfluorooctanoic Acid (PFOA) in Drinking Water*, Office of Water, March 14, 2023, https://www.epa.gov/system/files/documents/2023-03/MAIN_Proposed%20MCLG%20for%20PFOA%20in%20Drinking%20Water_3.9.23_For%20Proposal.pdf.

mammary gland observed at very low doses is uncertain and was not considered a suitable basis for the MRL.”¹⁶

However, these summaries simplify the complex behaviors that are observed during lactation between a mother (dam) and offspring (pup). The lactation challenge in White et al. 2011 only evaluated the amount of milk passed from the dam to the pups in a single nursing event; it did not account for compensatory behaviors that may have been present in the pups.¹⁷ The study authors added this additional context, stating “[t]hese data suggest that nursing behavior of the neonates may have changed (i.e., increased number of nursing events per day or longer nursing per event) to compensate for the decreased potential in milk production by the F1 dam, but we did not evaluate these end points in this study.”¹⁸ We previously submitted comments to ATSDR highlighting the agency’s misinterpretation of the study by White et al. (2011), pointing out that “an estimated 3-6 million mothers each year are unable to produce milk or have difficulty breastfeeding. The cause of this remains unclear, however, exposure to toxic environmental chemicals are one candidate explanation for the inability to initiate and/or sustain breastfeeding.”¹⁹

Importantly, a 2009 workshop of experts in mammary gland biology and risk assessment came to the consensus that changes in mammary gland growth and differentiation, including changes in developmental timing, are a relevant human health concern.²⁰ Altered mammary gland development may lead to difficulty in breastfeeding and/or an increase in susceptibility to breast cancer later in life.

Although Michigan and New Jersey did not directly base their risk assessments for PFOA on mammary gland effects or changes in lactation duration, they did address the increased risk for this effect through their application of uncertainty factors. Michigan stated that mammary gland effects may not be considered adverse, but that they could be representative of endocrine

¹⁶ ATSDR, *Toxicological Profile for Perfluoroalkyls*, May 2021, <https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf>.

¹⁷ Sally S. White, Jason P. Stanko, Kayoko Kato, Antonia M. Calafat, Erin P. Hines, and Suzanne E. Fenton, *Gestational and Chronic Low-Dose PFOA Exposures and Mammary Gland Growth and Differentiation in Three Generations of CD-1 Mice*, *Environmental Health Perspectives* 119, no. 8 (August 2011): 1070–76, <https://ehp.niehs.nih.gov/doi/epdf/10.1289/ehp.1002741>.

¹⁸ *Id.*

¹⁹ Anna Reade, *Comments on ATSDR Toxicological Profile on Perfluoroalkyls 2018 Draft*, September 6, 2018, https://www.nrdc.org/sites/default/files/comments-on-atsdr-toxicological-profile-on-perfluoroalkyls-2018-draft_2018-08-21.pdf.

²⁰ Ruthann A. Rudel, Suzanne E. Fenton, Janet M. Ackerman, Susan Y. Euling, and Susan L. Makris, *Environmental Exposures and Mammary Gland Development: State of the Science, Public Health Implications, and Research Recommendations*, *Environmental Health Perspectives* 119, no. 8 (August 2011): 1053–61, <https://doi.org/10.1289/ehp.1002864>.

(hormone) effects at doses below Michigan's selected point of departure.²¹ Michigan therefore applied an additional uncertainty factor for database limitations regarding endocrine effects.²²

New Jersey stated that the mammary gland effect is “the most sensitive systemic endpoint for PFOA with data appropriate for dose-response modeling. It is a well-established toxicological effect of PFOA that is considered to be adverse and relevant to humans for the purposes of risk assessment.”²³ However, New Jersey also concluded that because altered mammary gland development had yet to be used as the basis for a risk assessment it would not select it as the critical effect, but did apply an uncertainty factor to protect for this more sensitive effect.²⁴

In finalizing the toxicity assessment and economic analysis, EPA should reconsider the effects of PFOA on mammary gland development and function, with specific attention to impacts on lactation duration. EPA could then quantify the number of people who may be impacted by the proposed regulation. For example, using data available from the meta-analysis by Timmerman et al. (2023), an attributable risk for shortened lactational duration could be calculated as follows:

$$AR = I_o (RR-1)^{25}$$

- where RR is the relative risk of the outcome of interest - In our case stopping any breastfeeding before 6 months. In this example we will use the data from Romano et al., 2016²⁶

²¹ Jamie C. DeWitt, Kevin Cox, and David A. Savitz, *Health Based Drinking Water Value Recommendations for PFAS in Michigan*, June 27, 2019, <https://www.michigan.gov/-/media/Project/Websites/PFAS-Response/Reports/2019-Health-Based-Drinking-Water-Value-Recommendations-PFAS-MI.pdf?rev=1779be946a5c41439f1db4f3eeaec4ec>.

²² *Id.*

²³ New Jersey Drinking Water Quality Institute, *Health-Based Maximum Contaminant Level Support Document: Perfluorooctanoic Acid (PFOA)*, February 15, 2017, <https://www.nj.gov/dep/watersupply/pdf/pfoa-appendixa.pdf>.

²⁴ Note that Texas has used altered mammary gland development in its PFAS risk assessment prior to NJ Drinking Water Quality Institute's comments. The report was formerly available from the Texas Commission on Environmental Quality (TCEQ) at <https://www.tceq.texas.gov/assets/public/implementation/tox/evaluations/pfcs.pdf>, but an update is now available: TCEQ, *Per- and Poly-Fluoroalkyl Substances (PFAS)*, February 14, 2023, <https://www.tceq.texas.gov/downloads/toxicology/pfc/pfcs.pdf>.

²⁵ Noel S. Weiss and Thomas D. Koepsell, eds, *Epidemiologic Methods: Studying the Occurrence of Illness*. Second edition. Oxford University Press, 2014, <https://academic.oup.com/book/24995>.

²⁶ Megan E. Romano, Yingying Xu, Antonia M. Calafat, Kimberly Yolton, Aimin Chen, Glenys M. Webster, Melissa N. Eliot, Cynthia R. Howard, Bruce P. Lanphear, and Joseph M. Braun, *Maternal Serum Perfluoroalkyl Substances during Pregnancy and Duration of Breastfeeding*, *Environmental Research* 149 (August 1, 2016): 239–46, <https://doi.org/10.1016/j.envres.2016.04.034>.

- where I_0 is the disease incidence in the unexposed - Here we estimate this based on data from the National Immunization Survey, which suggests that the prevalence of children who were breastfed at 6 months in the U.S. was 55.8% for 2019.²⁷
- This means that 44.2% of children are not breastfed at 6 months, which can be used as an estimate of cumulative incidence.²⁸

Calculation Inputs:

	PFOA	PFOS	PFHxS	PFNA
RR	1.41*	1.25*	1.22	1.13
I_0	0.442	0.442	0.442	0.442

*These RRs were statistically significant in the original paper, so there is more confidence in the precision of these estimates. The others were borderline significant, so though they are still reasonable, they are also possibly less precise.

Given the calculation inputs provided above, the attributable risk for stopping breastfeeding by 6 months can be calculated for those with the highest serum PFAS level in pregnancy (i.e., in the 4th quartile in the HOME Study) compared to those the lowest serum PFAS exposure (1st quartile in the HOME study). The HOME Study is the Health Outcomes and Measures of the Environment study) which was used in Romano et al., 2016.

The resulting attributable risks are:

- AR_{PFOA} = 18 per 100 mothers
- AR_{PFOS} = 11 per 100 mothers
- AR_{PFHxS} = 9 per 100 mothers
- AR_{PFNA} = 5 per 100 mothers

In other words, 18 additional mothers (per 100 mothers) stopped breastfeeding before 6 months of age in the highly exposed PFOA group versus the lowest exposed PFOA group.

More conservatively, one could assume that the incidence of stopping breastfeeding among truly unexposed women is $\frac{1}{4}$ that of the general population (11.05%). If that is the case, then the attributable risks are as follows.

- AR_{PFOA} = 5 per 100 mothers
- AR_{PFOS} = 3 per 100 mothers
- AR_{PFHxS} = 2 per 100 mothers
- AR_{PFNA} = 1 per 100 mothers

²⁷ CDC, *Results: Breastfeeding Rates*, Centers for Disease Control and Prevention, April 4, 2023. https://www.cdc.gov/breastfeeding/data/nis_data/results.html.

²⁸ Ideally the cumulative incidence is based on the disease incidence in an unexposed population. However, with PFAS, the general population is not a truly unexposed population. Therefore, the cumulative incidence, and consequently the attributable risk, may be overestimated. Therefore, an alternative analysis with a more conservative assumption of the cumulative incidence is also provided.

Monetization of the impact of shortened lactation duration is also possible.²⁹ An online tool that estimates the cost of not breastfeeding suggests that in the US there is an additional \$28 million in healthcare system treatment costs when children are not breastfed due to increased maternal and child infections, and additional costs due to cognitive losses and the need for households to purchase breastmilk substitutes.³⁰

II. Immunotoxicity

In its explanation of why immune effects were not selected for economic analysis, EPA states:

“While immune effects had indicative evidence of associations with exposure to PFOA and PFOS, EPA did not identify the necessary information to connect the measured biomarker responses (i.e., decrease in antibodies) to a clinical effect that could be valued in the economic analysis.”³¹

While it is difficult to quantitate the relationship between altered immune responses, such as decreases in antibody production, and frequency or severity of disease in inherently diverse human populations, these “subclinical” effects are associated with increased disease risk and economic cost and are therefore important to address. Small shifts within the range of normal clinical values can still have devastating population-level impacts. Specifically, the cellular and humoral immune response to vaccination is thought to be a sensitive indicator of immunosuppression.³² In a literature review from 2018 the authors conclude that, “[t]aken together, we find that results of epidemiological studies, supported by findings from toxicological studies, provide strong evidence that humans exposed to PFOA and PFOS are at risk for immunosuppression.”³³ In a more recent review, authors find that, “there is ample evidence illustrating PFAS affect multiple aspects of the immune system, which supports the overall conclusion that not only PFOA and PFOS, but also other members of the PFAS family

²⁹ Dylan D. Walters, Linh T H Phan, and Roger Mathisen, *The Cost of Not Breastfeeding: Global Results from a New Tool*, Health Policy and Planning 34, no. 6 (July 2019): 407–17, <https://doi.org/10.1093/heapol/czz050>.

³⁰ Alive & Thrive, *In the USA, Breastfeeding Impacts Families, Communities, and the Economy*, 2022, <https://www.aliveandthrive.org/en/country-stat/usa>.

³¹ US EPA, *Draft for Public Comment: Economic Analysis for the Proposed Per- and Polyfluoroalkyl Substances National Primary Drinking Water Regulation*, March 14, 2023, https://www.epa.gov/system/files/documents/2023-03/Proposed%20PFAS%20NPDWR%20EA_final_03_09_2023_0.pdf.

³² Ronald Glaser, Gary R. Pearson, Robert H. Bonneau, Brian A. Esterling, Cathie Atkinson, and Janice K. Kiecolt-Glaser, *Stress and the Memory T-Cell Response to the Epstein-Barr Virus in Healthy Medical Students*, Health Psychology 12, no. 6 (1993): 435–42, <https://doi.org/10.1037/0278-6133.12.6.435>.

³³ Jamie C. DeWitt, Sarah J. Blossom, and Laurel A. Schaidler, *Exposure to Per-Fluoroalkyl and Polyfluoroalkyl Substances Leads to Immunotoxicity: Epidemiological and Toxicological Evidence*, Journal of Exposure Science & Environmental Epidemiology 29, no. 2 (March 2019): 148–56, <https://doi.org/10.1038/s41370-018-0097-y>.

alter immune functions in humans.”³⁴ They go on to confirm that the “most reported immunotoxic effect in humans is immunosuppression, reflected by reduced vaccine antibody levels and increased risk of common infectious diseases.”³⁵

Importantly, immunosuppression has been defined by experts as “a reduced ability of the immune system to respond to a challenge from a level considered normal, regardless of whether clinical disease results.”³⁶ However, there are clinical consequences of mild-to-moderate chronic immunosuppression, including an increase in the incidence of infectious diseases. Even small changes in infectious disease frequency can have major social and economic impacts, particularly on vulnerable populations. The elderly experience age-related declines in immune function and factors that contribute to immunosuppression in this population can increase the risk of morbidity and mortality. Adults 65 and older make up approximately 90% of the total pneumonia and influenza-related deaths in the U.S.³⁷ Furthermore, vaccines are less effective in the elderly.³⁸ The young are particularly susceptible to infectious agents that require adult-level immune responses. For example, the ability to produce antibodies develops slowly, with infants (1-3 months old) starting off with approximately 30% of adult antibody levels and children (12-16 years old) still only producing around 70% of adult levels.³⁹ Agents that induce immunosuppression can exacerbate the inherent deficits in infants’ and children’s immature and still developing immune systems.⁴⁰

In a recent epidemiology study, authors looked directly at the link between PFAS exposure and persistent infections.⁴¹ They found that, “[e]ach PFAS was individually associated with

³⁴ Veronika Ehrlich, Wieneke Bil, Rob Vandebriel, Berit Granum, Mirjam Luijten, Birgitte Lindeman, Philippe Grandjean, et al, *Consideration of Pathways for Immunotoxicity of Per- and Polyfluoroalkyl Substances (PFAS)*, Environmental Health 22, no. 1 (February 22, 2023): 19, <https://doi.org/10.1186/s12940-022-00958-5>.

³⁵ *Id.*

³⁶ Jamie C. DeWitt, Dori R. Germolec, Robert W. Luebke, and Victor J. Johnson. *Associating Changes in the Immune System with Clinical Diseases for Interpretation in Risk Assessment*, Current Protocols in Toxicology 67 (February 1, 2016): 18.1.1-18.1.22, <https://doi.org/10.1002/0471140856.tx1801s67>.

³⁷ C. P. Mouton, O. V. Bazaldua, B. Pierce, and D. V. Espino, *Common Infections in Older Adults*, Health Care Food & Nutrition Focus 18, no. 3 (November 2001): 1, 3–7.

³⁸ Richard Aspinall, Giuseppe Del Giudice, Rita B. Effros, Beatrix Grubeck-Loebenstein, and Suryaprakash Sambhara, *Challenges for Vaccination in the Elderly*, Immunity & Ageing: I & A 4 (December 11, 2007): 9, <https://doi.org/10.1186/1742-4933-4-9>.

³⁹ E. R Stiehm and H. H. Fudenberg, *Serum Levels of Immune Globulins in Health and Disease: A Survey* Pediatrics 37, no. 5 (May 1966): 715–27.

⁴⁰ Jamie C. DeWitt, Dori R. Germolec, Robert W. Luebke, and Victor J. Johnson, *Associating Changes in the Immune System with Clinical Diseases for Interpretation in Risk Assessment*, Current Protocols in Toxicology 67 (February 1, 2016): 18.1.1-18.1.22, <https://doi.org/10.1002/0471140856.tx1801s67>.

⁴¹ Catherine M. Bulka, Vennela Avula, and Rebecca C. Fry, *Associations of Exposure to Perfluoroalkyl Substances Individually and in Mixtures with Persistent Infections: Recent*

significantly higher pathogen burdens and the most pronounced associations were observed in adolescents [e.g., among adolescents, a doubling of PFOS was associated with 30% (95% CI: 25–36%) higher pathogen burden]. Quantile g-computation revealed PFAS mixtures as a whole were also associated with higher pathogen burdens. Taken together, these results suggest PFAS exposure may increase susceptibility to and foster the clustering of persistent infections, particularly among adolescents.”⁴²

Impacts associated with mortality and morbidity from common pathogens (such as influenza and pneumonia) have been studied and estimated. These can serve as a basis for beginning to quantitate and even monetize the benefit of reducing the risk of immunotoxicity associated with PFAS exposure as these common pathogens are more likely to increase mortality and morbidity in those who are mildly to moderately immunosuppressed, i.e., the young, the elderly, and those with toxicant-induced PFAS immunosuppression. Relevant data includes:

Quantitative data on morbidity⁴³

- In 2010, influenza and pneumonia together were ranked the ninth leading cause of death in the U.S. for all ages.
- For infant deaths in 2010, influenza was ranked 46th and pneumonia was ranked 47th.
- For the elderly population, chronic lower respiratory disease was ranked 3rd and influenza-pneumonia was ranked 7th in leading causes of death in 2004.

Cost estimates

- Total cost of influenza and pneumonia was estimated to be \$40.2 billion in the U.S.⁴⁴
- Ear infections (otitis media) is the most common indication for antibiotic use and outpatient visits in children – \$2.88 billion in added annual health care expense in the U.S.⁴⁵
- Annual cost of treating RSV for children under 5 in 2000 was \$652 million⁴⁶

Taken together, the data support the identification of protecting against immune system effects, particularly immunosuppression, as a key benefit resulting from the proposed MCLs. The data also suggests that EPA has sufficient information to quantify this benefit. Examples include the number of infection-related deaths avoided, the number of infectious disease cases avoided, and the increased proportion of the population with successful responses to immunization. Finally,

Findings from NHANES 1999–2016, Environmental Pollution 275 (April 15, 2021): 116619, <https://doi.org/10.1016/j.envpol.2021.116619>.

⁴² *Id.*

⁴³ M. Heron, *Deaths: Leading causes for 2010*, National Vital Statistics Reports. 2013; 62.

⁴⁴ American Lung Association, *Influenza and pneumonia: State of lung disease in diverse communities*, 2010.

⁴⁵ Sameer Ahmed, Nina L. Shapiro, and Niel Bhattacharyya, *Incremental Health Care Utilization and Costs for Acute Otitis Media in Children*, The Laryngoscope 124 (2014): 301–5, <https://doi.org/10.1002/lary.24190>.

⁴⁶ L. Clark Paramore, Vincent Ciuryla, Gabrielle Ciesla, and Larry Liu, *Economic Impact of Respiratory Syncytial Virus-Related Illness in the US*, Pharmacoeconomics 22, no. 5 (April 1, 2004): 275–84, <https://doi.org/10.2165/00019053-200422050-00001>.

there is information detailing some of the costs related to common infectious diseases that can be used to monetize some of these benefits. In finalizing the economic analysis EPA should quantitate and monetize, where possible, the health benefits of reduced PFAS-related immunotoxicity that the proposed MCLs will provide.

III. Liver disease

We disagree with EPA's conclusions that the hepatic effects that have been observed are modest and unquantifiable. The reality is that the experimental literature and human literature are substantial, mutually reinforcing, consistent, and point to a problem of PFAS hepatotoxicity.⁴⁷ Few findings in environmental health are as consistent as the experimental and epidemiological evidence that PFAS, notably but not limited to PFOA and PFOS, are associated with liver damage.⁴⁸

Across species and toxicological studies, PFAS exposure causes increased ALT (alanine aminotransferase) levels and liver steatosis (fat accumulation), which is the starting point for NAFLD (nonalcoholic fatty liver disease). Yet, EPA has argued that 1) the connection from increased ALT to liver disease is lacking and 2) that the changes in ALT after PFAS exposure is modest (implying lacks importance).

1) Connecting increased ALT to liver disease:

Specifically, with respect to connecting increases in ALT to liver disease, EPA states, "Elevated ALT levels could be one of several contributors to the non-alcoholic fatty liver disease. Additionally, high ALT levels can be associated with alcohol consumption, heart failure, hepatitis (A, B, and C), medication use (e.g., Tylenol and statins), and obesity (Mayo Clinic, 2022) and this wide range of associations makes it difficult to model economic benefits of non-specific ALT level changes in response to reduced exposures."⁴⁹ Most health effects are associated with more than one risk factor. This does not mean that quantifying the benefit of reducing one of these multiple risk factors is not possible.

Multiple studies show that PFAS exposure causes liver toxicity and fatty liver disease across animal species, without known species exceptions so far. There is no reason to expect humans to be the exception.

⁴⁷ Elizabeth Costello, Sarah Rock, Nikos Stratakis, Sandrah P. Eckel, Douglas I. Walker, Damaskini Valvi, Dora Cserbik, et al, *Exposure to Per- and Polyfluoroalkyl Substances and Markers of Liver Injury: A Systematic Review and Meta-Analysis*, Environmental Health Perspectives 130, no. 4 (April 27, 2022): 046001, <https://doi.org/10.1289/EHP10092>.

⁴⁸ *Id.*

⁴⁹ US EPA, *Draft for Public Comment: Economic Analysis for the Proposed Per- and Polyfluoroalkyl Substances National Primary Drinking Water Regulation*, March 14, 2023, https://www.epa.gov/system/files/documents/2023-03/Proposed%20PFAS%20NPDWR%20EA_final_03_09_2023_0.pdf.

Evaluating environmental contributors to NAFLD in cross-sectional studies is difficult due to how and when the disease is diagnosed. NAFLD is seldom diagnosed in early stages in clinical practice and may not be coded as among the comorbidities for other diagnoses. Therefore, cross-sectional studies that have relied on medical record review (ICD-code verified) of NAFLD are likely to suffer from outcome misclassification. However newer studies, not reliant on ICD-code verification, but rather on imaging of livers, have linked PFAS exposure with deleterious effects on fatty liver findings.⁵⁰ In a biopsy-proven cohort of 105 patients with NAFLD, PFAS were found to be adversely associated with liver fat content, lipid metabolism, and bile acid metabolism.⁵¹

2) Addressing the biological significance of “modest” changes in ALT

EPA states that the effects of PFAS exposure on observed ALT levels are modest, but fails to recognize that more recently, professional societies have recommended more appropriate, physiologically-based cutoffs for what is considered a normal or abnormal ALT level based on the important societal need to address the NAFLD epidemic.⁵²

A large epidemiological study of more than 30,000 participants in a community with PFOA exposures ranging from national background levels to very high levels of contamination, concluded participants in the fifth quintile had 16% increased odds of having above-normal ALT (95% CI: odds ratio: 1.02, 1.33%).⁵³ There is a near monotonic increase in ALT with increasing PFOA, with the dose-response beginning at what are considered to be “background” levels of population exposure.

⁵⁰ Xincheng Wang, Xiaoqian Jin, Hancheng Li, Xianyu Zhang, Xi Chen, Kuan Lu, and Chenliang Chu, *Effects of Various Interventions on Non-Alcoholic Fatty Liver Disease (NAFLD): A Systematic Review and Network Meta-Analysis*, *Frontiers in Pharmacology* 14 (2023), <https://www.frontiersin.org/articles/10.3389/fphar.2023.1180016>.

⁵¹ Partho Sen, Sami Qadri, Panu K. Luukkonen, Oddny Ragnarsdottir, Aidan McGlinchey, Sirkku Jäntti, Anne Juuti, et al., *Exposure to Environmental Contaminants Is Associated with Altered Hepatic Lipid Metabolism in Non-Alcoholic Fatty Liver Disease*, *Journal of Hepatology* 76, no. 2 (February 1, 2022): 283–93, <https://doi.org/10.1016/j.jhep.2021.09.039>.

⁵² Naga, Chalasani, Zobair Younossi, Joel E. Lavine, Michael Charlton, Kenneth Cusi, Mary Rinella, Stephen A. Harrison, Elizabeth M. Brunt, and Arun J. Sanyal, *The Diagnosis and Management of Nonalcoholic Fatty Liver Disease: Practice Guidance from the American Association for the Study of Liver Diseases*, *Hepatology* 67, no. 1 (January 2018): 328, <https://doi.org/10.1002/hep.29367>; Jin Hwa Park, Jun Choi, Dae Won Jun, Sung Won Han, Yee Hui Yeo, and Mindie H Nguyen, *Low Alanine Aminotransferase Cut-Off for Predicting Liver Outcomes; A Nationwide Population-Based Longitudinal Cohort Study*, *Journal of Clinical Medicine* 8, no. 9 (September 11, 2019): 1445, <https://www.mdpi.com/2077-0383/8/9/1445>.

⁵³ Lyndsey A. Darrow, Alyx C. Groth, Andrea Winkquist, Hyeong-Moo Shin, Scott M. Bartell, and Kyle Steenland, *Modeled Perfluorooctanoic Acid (PFOA) Exposure and Liver Function in a Mid-Ohio Valley Community*, *Environmental Health Perspectives* 124, no. 8 (August 2016): 1227–33, <https://doi.org/10.1289/ehp.1510391>.

A reanalysis of the above data was performed using the updated physiologically-based cutoffs for ALT as recommended by the medical liver disease societies.⁵⁴ This reanalysis showed an increased association of PFOA to abnormal ALT and emphasized the near monotonic increases in ALT with increasing dose. For example, males in the 5th quintile of measured PFOA were 35% more likely and females 20% more likely to have abnormal ALT, with mean continuous increases of 9% per quintile for men and 4% for women.⁵⁵

Independent of PFAS, populations with higher biomarkers of liver distress such as ALT have worse outcomes for morbidity and mortality.⁵⁶

In finalizing the economic analysis EPA should reconsider the conclusions drawn regarding the literature exploring ALT and NAFLD in relationship to PFAS exposure and attempt to quantitate the number of people who would benefit from reduced risk of liver effects from the proposed MCLs. EPA should also attempt to monetize the health benefit from reduced NAFLD cases, as there are studies available that provide estimates of the economic burden associated with NAFLD.

For example, one study from 2016 estimated that in the U.S., “over 64 million people are projected to have NAFLD, with annual direct medical costs of about [\$103.3] billion (\$1,613 per patient)” but that the “burden is significantly higher when societal costs are included.”⁵⁷ The study estimated societal costs to be \$188.9 billion, yielding a total cost of \$292.2 billion. These cost estimates account for drugs, healthcare, and changes in quality of life, but they under-value other indirect costs such as lost productivity and federal benefits for disability. They also underestimate even the pharmaceutical costs, as they rely on formal diagnoses and it is clear that

⁵⁴ Alan Ducatman, Youran Tan, Brian Nadeau, and Kyle Steenland, *Perfluorooctanoic Acid (PFOA) Exposure and Abnormal Alanine Aminotransferase: Using Clinical Consensus Cutoffs Compared to Statistical Cutoffs for Abnormal Values*, *Toxics* 11, no. 5 (May 2023): 449, <https://doi.org/10.3390/toxics11050449>.

⁵⁵ *Id.*

⁵⁶ Paul Y. Kwo, Stanley M. Cohen, and Joseph K. Lim, *ACG Clinical Guideline: Evaluation of Abnormal Liver Chemistries*, *American Journal of Gastroenterology* 112, no. 1 (January 2017): 18–35, https://journals.lww.com/ajg/Fulltext/2017/01000/ACG_Clinical_Guideline_Evaluation_of_Abnormal.13.aspx; Naga Chalasani, Zobair Younossi, Joel E. Lavine, Michael Charlton, Kenneth Cusi, Mary Rinella, Stephen A. Harrison, Elizabeth M. Brunt, and Arun J. Sanyal, *The Diagnosis and Management of Nonalcoholic Fatty Liver Disease: Practice Guidance from the American Association for the Study of Liver Diseases*, *Hepatology* 67, no. 1 (January 2018): 328, <https://doi.org/10.1002/hep.29367>.

⁵⁷ Zobair M. Younossi, Deirdre Blissett, Robert Blissett, Linda Henry, Maria Stepanova, Youssef Younossi, Andrei Racila, Sharon Hunt, and Rachel Beckerman, *The Economic and Clinical Burden of Nonalcoholic Fatty Liver Disease in the United States and Europe*, *Hepatology* 64, no. 5 (2016): 1577–86, <https://doi.org/10.1002/hep.28785>.

NAFLD treatment, hospitalization, and indirect costs can precede the diagnosis and treatment.⁵⁸ Despite these limitations, EPA can still use this information to begin to monetize the benefit of reduced NAFLD cases from the proposed MCLs.

EPA failed to quantify several of the health effects associated with PFAS exposure. We have provided resources and information that EPA should use to estimate the proposed drinking water standards' additional benefits of protecting against PFAS-associated effects on mammary gland development, the liver and immune system.

Sincerely,



Anna Reade, PhD
Senior Scientist
Natural Resources Defense Council



Katherine Pelch, PhD
Scientist
Natural Resources Defense Council

⁵⁸ Myriam Alexander, A. Katrina Loomis, Jolyon Fairburn-Beech, Johan van der Lei, Talita Duarte-Salles, Daniel Prieto-Alhambra, David Ansell, et al., *Real-World Data Reveal a Diagnostic Gap in Non-Alcoholic Fatty Liver Disease*, BMC Medicine 16, no. 1 (August 13, 2018): 130. <https://doi.org/10.1186/s12916-018-1103-x>.

Exhibit B

Memorandum

To: Earthjustice

From: Dennis Guignet, PhD.

Subject: Review of the Economic Analysis for the Proposed PFAS NPDWR

Date: May 26, 2023

Purpose

The purpose of this memorandum is to evaluate the U.S. Environmental Protection Agency's Economic Analysis of the proposed Per- and Polyfluoroalkyl Substances (PFAS) National Primary Drinking Water Regulation, and to identify ways the economic analysis (EA) can be further improved.

As a trained economist with a PhD in Agricultural and Resource Economics, and over a decade of experience, I am qualified to identify analytical strengths and weaknesses in the EA, and recognize potential areas where the EA can be further strengthened. I served as a research economist in the National Center for Environmental Economics at the U.S. Environmental Protection Agency (EPA) for seven years (from 2011 to 2018). During that time, I helped develop, review, and revise EAs of regulatory actions. I have taught environmental economics courses at the University of Maryland and American University, as well as a graduate and undergraduate-level course in benefit-cost analysis at Appalachian State University, where I am currently employed as an Assistant Professor of Economics. My research focuses on applied quantitative analysis, benefit-cost analysis of environmental policies, and the estimation of environmental and human health benefits, with a particular focus on toxic chemicals. I have 25 publications in peer-reviewed journals, and significant related experience that adds to my qualifications (see the attached Exhibit 1 for my full curriculum vitae).

Overall, based on my professional experience, the EA that has been developed for the proposed PFAS National Primary Drinking Water Standards (NPDWS) is one of the most well-organized, thorough, and transparent EAs I have reviewed. The quantified benefits entail reduced mortality and non-fatal health outcomes from decreased exposures to PFAS, including reduced risks of cardiovascular disease, low infant birthweight, and renal cell carcinoma. Additionally, reduced risk of bladder cancer due to the co-removal of non-PFAS pollutants is a co-benefit that is quantified. For all four health outcomes, the number of avoided fatal and non-fatal cases that result from each regulatory option, relative to the baseline, are estimated. These quantified benefits are monetized based on the Value of a Statistical Life (VSL) for reduced fatal cases (i.e., reduced mortality risks), and based on avoided cost-of-illness (COI) estimates for reduced non-fatal cases (i.e., reduced morbidity risks). Both are common and well-accepted approaches for monetizing (i.e., assigning a dollar value to) human health-related benefits in policy analysis.

The quantified costs mainly include public water system (PWS) costs for implementing treatment and non-treatment technologies when the proposed maximum contaminant levels (MCLs) are exceeded, as well as monitoring and reporting costs, and administrative costs to PWSs and implementing agencies.

This memorandum is organized as follows. First, key strengths of the EA are highlighted. Then I discuss potential areas where the analysis could be improved.

Strengths of the Economic Analysis.

There are several strengths of the EA that are worth highlighting. These features should be maintained and potentially built upon for the final rule EA.

1. The entire EA is centered on a detailed, data-driven Monte Carlo simulation model that has been calibrated based on existing federal and state data on point-of-entry water concentrations of PFOS, PFOA, and other PFAS.

This carefully laid approach relies on existing data to project baseline and policy scenarios under the various regulatory options. Point-of-entry pollutant concentrations are predicted across PWSs, while at the same time accounting for various system-specific factors. A particularly advantageous feature of this approach is in enabling data-driven, comprehensive sensitivity analyses. The analysis utilizes Monte Carlo simulations entailing 4,000 iterations. This allows the Agency to derive a distribution of benefit and cost estimates, and in turn quantitatively and simultaneously account for key points of analytical uncertainty by providing a range of possible net benefit calculations (e.g., a 90% confidence interval). Monte Carlo simulations like this are a common and defensible approach to account for uncertainty (EPA 2014), and are the most thorough way to simultaneously account for numerous sources of uncertainty in economic analyses (Boardman et al. 2018; OMB 2003).

As outlined in the Office of Management and Budget's Circular A-4, the Executive Branch's seminal guidance for conducting regulatory analysis, there are basically three broad approaches for accounting for analytical uncertainties (OMB 2003). In terms of increasing levels of complexity and rigor, these approaches include:

- A qualitative discussion of the main uncertainties;
- A quantitative sensitivity analysis, such as a "partial sensitivity analysis" or "worst- and best-case analysis"; and
- A probabilistic quantitative analysis using Monte Carlo simulations.

In cases where the necessary data and information are not available to assess the likelihood and outcomes of various contingencies under alternative assumptions, then a qualitative discussion of the key uncertain assumptions and potential implications for the results of the EA should be included. And in cases where the available information is limited, EPA does in fact include such qualitative assessments; for example, see Table 5-22 and Tables 6-48 through 6-53 in Chapters 5 and 6, respectively.

Other more quantitative sensitivity analyses are recommended when some quantitative information is available, but where information on the probability of alternative contingencies (or outcomes) is not. For example, “partial sensitivity analysis” is when one varies one assumption at a time, and assesses how the estimated net benefits vary across alternative assumptions (Boardman et al. 2018). This approach has two main drawbacks. First, varying only one assumption at a time may not fully bound the plausible range of results, especially in cases where there may be interactive or cascading effects across assumptions. Although one could vary multiple sets of assumptions at a time, there are only so many distinct scenarios an analyst can reasonably estimate. The second weakness is that even with a large menu of distinct scenarios, one can say nothing about the likelihood of any one scenario or results of central tendency (e.g., an average), due to the lack of an estimated probability distribution.

An alternative type of quantitative sensitivity analysis sometimes labelled “worst- and best-case analysis” (Boardman et al. 2018), allows analysts to bound the plausible range of net benefits. This is done by estimating a worst-case scenario, where for all key analytical decision points, model parameters, etc., the “worst-case” plausible assumptions are made that would result in the highest estimates of the costs and lowest estimates for the benefits. Then on the other extreme, a best-case scenario is estimated, where the underlying key assumptions are set such that the analysis will yield the highest plausible estimates for the benefits and lowest estimates of the costs. This overcomes the first weakness described above by varying all key assumptions at once, and thus providing upper- and lower-end bounds on the net benefits, and it accounts for interactive and cascading effects across assumptions. The second weakness, however, is still not addressed. One still would not know where the eventually realized benefits and costs are most likely to fall within those bounds. In other words, “worst- and best-case analysis” can reasonably bound the range of plausible net benefits, but tells us nothing about how likely one scenario is over another, or where within that range we are most likely to fall.

This final weakness is overcome by the third, and most thorough type of analysis of uncertainty – Monte Carlo simulations. Detailed Monte Carlo simulations form the underlying foundation for the entire EA for the proposed PFAS NPDWR. By taking advantage of existing data on PFAS concentrations in drinking water sources, as well as past treatment decisions of PWSs (discussed below in comment #2), the Agency was able to estimate a range of benefits and costs, as well as a probability associated with each potential realization of these estimates. In short, this yields several advantages by allowing the Agency to (i) assess the simultaneous impacts across multiple key assumptions, (ii) account for the likelihood of those assumptions and the resulting estimates of the benefits and costs that would be realized, and finally (iii) account for a range based on, for example, the 90% confidence interval, as well as the estimates of the benefits and costs that will be realized, on average. The Agency went through great efforts to gather the necessary data to not only pursue this most rigorous approach to account for analytical uncertainty, but they did so in a way that accounts for numerous points of uncertainty, and that relies squarely on existing data (as opposed to, for example, best professional judgment).

2. EPA uses an equally thorough, data-driven approach for estimating what treatment and non-treatment technologies would be adopted to comply with the rule.

EPA recognizes that there would likely be variation in what technologies PWSs adopt to comply with the proposed rule, and as such they did not simply assume all PWSs with PFAS levels above the proposed MCLs would adopt the seemingly cheapest technology. Instead, based on system and source water-specific parameters, EPA estimated systems' choices of treatment and non-treatment technologies based on data of past technology adoption choices. More specifically, a decision tree was developed to determine what treatment technologies or non-treatment approaches are feasible based on entry point, water source, and system characteristics, as well as the regulatory option being considered. Then among the feasible options, the estimated compliance approach is randomly chosen for each PWS based on the probability distribution of recently observed choices made by PWSs. In other words, holding all system and water-source characteristics constant, if a certain technology has tended to be chosen more often in practice, then that increased likelihood is accounted for in EPA's technology adoption models and subsequent cost estimates.

EPA then developed over 3,500 individual cost equations to estimate the costs of the chosen technologies based on key factors, including the corresponding technology bed life (i.e., the length of time that a technology can maintain a target pollutant removal percentage), water source, flow, etc.

Overall, this detailed, data-driven approach to estimate treatment and non-treatment approaches towards compliance, and the subsequent costs, strengthens the analysis because the cost estimates are based on empirical evidence (i.e., observed choices made by PWSs), rather than theoretical assumptions and subjective best judgements. The randomization of PWS compliance choices and subsequent costs based on the observed distribution of PWS adoption choices is layered on top of the Monte Carlo simulations discussed in comment #1, and thus further exhibits all the advantages of such an approach that are discussed above.

3. EPA correctly excludes any incremental costs to PWSs that would result from the need to dispose of spent filtration materials as hazardous waste.

The PFAS addressed by EPA's proposed NPDWRs—PFOS, PFOA, PFHxS, HFPO-DA and its ammonium salt, PFNA, and PFBS—are not currently regulated as hazardous materials under federal law, and therefore additional disposal costs that would be required by federal law for the disposal of hazardous materials should not be applied in this EA of the proposed NPDWS. In my professional experience, it is standard practice to only account for other regulations in the baseline if those regulations have been promulgated as of the time of the EA. Accounting for future potential regulations in the baseline is uncommon, and trying to account for all potential future contingencies in this regard would quickly make any EA unmanageable. If EPA later determines that PFOS, PFOA, and other regulated PFAS are hazardous materials, then any incremental increase in disposal costs for such materials over normal spent filtration material

disposal costs would be a cost of that future regulation, and not of the currently proposed NPDWS.

Overall, EPA's choice to not incorporate in its cost analysis the expense of treating spent filtration materials as hazardous waste is well-grounded in economic theory and common practice. Nonetheless, in response to stakeholder comments EPA did include such costs in an illustrative sensitivity analysis. Although beyond standard practice, this illustrative exercise demonstrates the Agency's responsiveness and desire for full transparency in the regulatory development process. As discussed in comment #11 in the next section of this memorandum, if EPA maintains this illustrative cost analysis, then the Agency should also estimate the corresponding health and environmental benefits of treating spent filtration materials as hazardous waste. This is necessary in order to make this sensitivity analysis comprehensive and balanced.

4. EPA correctly accounts for existing state-level drinking water standards for PFAS in the baseline.

EPA undertook a detailed and thorough approach to account for existing state-level standards that limit the allowable levels of certain PFAS in drinking water. These states include NJ, VT, NH, MA, MI and NY. When baseline PFAS concentration levels for system points-of-entry in these states are estimated to exceed existing state-specific MCLs, then the estimated concentrations are instead assumed to equal the state-level MCLs – i.e., it is assumed that PWSs are in full compliance with any existing state standards for PFAS in drinking water. In making this adjustment, any incremental improvements already achieved by the state programs would be correctly accounted for in the baseline, and not falsely contribute to the estimated benefits and costs of the proposed federal PFAS NPDWS. This accounting of existing state-level standards in the baseline correctly results in a reduction in both benefits and costs compared to a baseline that would incorrectly disregard existing state regulations.

5. The inclusion of co-benefits (or ancillary benefits) in benefit-cost analyses is well-grounded in economic theory.

Roughly 14% to 18% of the estimated quantified benefits of the PFAS NPDWS are due to reduced bladder cancer risks, which in the current context is a co-benefit or ancillary benefit – i.e., a benefit that results from a regulatory action but that is not the direct intent of that action (OMB 2003). More specifically, the proposed PFAS NPDWS is anticipated to also reduce disinfection byproducts, and in turn reduce the risks of bladder cancer associated with exposure to those byproducts. The inclusion of such benefits in benefit-cost analysis is directed under the Safe Drinking Water Act (SDWA) and is well-grounded in economic theory (EPA 2014, OMB 2003).

6. EPA provides a balanced and detailed qualitative accounting of the benefits and costs that could not be quantified.

Ideally, all benefits and costs of a regulatory action would be quantified and monetized, but analysts are often limited by the available information, as well as resource constraints when conducting an EA. EPA prioritizes the quantification of benefits, for example, based on the contaminants and endpoints (i.e., adverse health outcomes) where (i) the weight of evidence linking the contaminant to key biomarkers is strongest, (ii) it is possible to link the contaminant or related biomarkers to a health endpoint (e.g., cardiovascular disease) that can be monetized (i.e., valued in dollar terms based on available economic literature and practices), and (iii) the endpoint does not overlap with another benefit category. Based on my professional experience, the first two criteria are standard practice when prioritizing analytical efforts given the practical constraints in conducting empirical analyses. The third criterion helps EPA avoid “double counting.” As stated in EPA’s Guidelines for Preparing Economic Analyses, when estimating the effects separately for each health endpoint “it is important to avoid double counting benefits across effects as much as possible” (EPA, 2014, pg. 7-3). Additionally, given limited resources, EPA prioritizes quantification of benefits and costs that are anticipated to be the largest. In my professional experience, prioritizing in this manner is sometimes necessary, and it is an appropriate way to prioritize given the objective of providing the best and most comprehensive information possible to inform the regulatory development process.

It is important to emphasize that the lack of quantification does not imply that any unquantified benefits and costs are not relevant. Following standard practice (EPA 2014, OMB 2003), EPA goes to great lengths to detail benefits and costs that could not be quantified, but that are still relevant for the EA and the ultimate determination of whether the benefits of the proposed PFAS NPDWS exceed the costs. A systematic summary is provided in Table 7-5 in Chapter 7, and further details are discussed in Chapters 5 and 6. Based on my own professional experience, the qualitative discussion in this EA is more rigorous than others I have reviewed. The Agency goes into a lot more detail, combs the literature more thoroughly, and touches on a larger number of potential health endpoints.

Although the net benefits for the central estimates of the proposed regulatory option are negative in some scenarios (e.g., under an assumed 7% discount rate), the Agency lays out convincing evidence that the net benefits are likely positive. The net benefits are positive under an assumed 3% discount rate (see Table 7-1), and the qualitative evidence and sheer number of unquantified benefit endpoints suggest that the benefits likely do exceed the costs. Furthermore, although consideration of alternative 3% and 7% discount rates is currently the standard practice for EAs of federal regulations (OMB 2003), the lower 3% discount rate may be more appropriate in this context. See comment #15 below for details.

In any case, the reliance of the key conclusions of this EA on qualitatively discussed health benefits is not atypical. A review by Petrolia et al. (2021) of EAs for all major EPA rules from 2008 to 2019 revealed that of the 43 analyses that included non-fatal health outcomes, nine (21%) *only* included unquantified health benefits (see Figure 2 in Petrolia et al. 2021); and additional qualitative health benefits were included in the other EAs that did quantify at least one

health endpoint. In OMB’s recently proposed revised guidance for economic analysis, it is emphasized that:

“relying on materially incomplete monetized BCA [net benefits] does not offer an adequate summary of evidence intended to inform determination of the most beneficial alternative, and such reliance could even be misleading. You [(analysts)] should exercise professional judgement in identifying the importance of non-quantified factors and assess as best you can how they might change the ranking of alternatives based on estimated net benefits” (OMB 2023, pg. 5).

Potential Areas to Improve the Economic Analysis.

Although the EA for the proposed PFAS NPDWS is quite thorough and transparent, there are several areas where the EA can be further improved. The below comments entail suggestions on where further clarification or support would be helpful, as well as a few cases where additional or revised analysis should be considered.

7. The analysis assumes that population is held constant based on 2021 levels for the entire 80-year study period.

If population is projected to increase over this time, then this constant population assumption would result in an underestimate of the benefits and of the costs. The EA would be improved if EPA considers available projections and/or current population trends when estimating future benefits and costs. EPA recognizes this area for improvement, and in Chapter 6 (Table 6-48) EPA discusses how they intend to account for population trends in the final rule EA. I encourage the Agency to pursue such revisions for the final rule EA.

8. Concentrations of PFOS, PFOA, and other PFAS at system entry points are simulated/projected based on data of past occurrences, and as such EPA is assuming that entry point PFAS concentrations are constant over time.

This assumption is explicitly stated on page 4-23, but the validity of this assumption is unclear. There are assumptions being made here in terms of stock PFAS concentrations in water sources, and implicitly the future use and releases of PFAS into drinking water sources. The high degree of persistence of PFAS, along with various confounding trends over time that the EA mentions (e.g., voluntary phaseout programs, industry trends, and trends in human exposure¹) make it difficult to assess the validity of this assumption. Put plainly, how representative are the data of current and recently observed PFAS concentrations compared to those expected in the future under the baseline (i.e., business as usual) scenario? To the extent that baseline stock concentration levels and the use of PFOS, PFOA, and other PFAS chemicals in industrial processes and consumer products, and subsequent releases into the environment, have been

¹ For example, on page 2-1 of the EA it is noted that that PFOA human blood levels have been decreasing.

decreasing, then both benefits and costs in the EA would be overestimated. The opposite is true if current baseline trends suggest an increase in future concentration levels of these chemicals at PWS points-of-entry.

The EA would be improved by providing additional support for the assumption that future baseline PFAS concentrations will be similar to recently observed concentrations, and/or by adding discussion of the resulting uncertainties regarding this assumption to Table 4-34.

9. Do the occurrence and concentrations of PFAS in water at PWS points-of-entry vary depending on whether the source is surface water or groundwater?

The Monte Carlo simulations used to predict baseline and policy scenario levels of these pollutants is very thorough in accounting for key factors that may lead to differences in pollutant levels (e.g., system size). However, it does not seem that the water source was accounted for when estimating point-of-entry concentration levels. If observed concentrations in the past vary across surface versus ground water sources in the data used to calibrate the Monte Carlo simulations, then this is another source of heterogeneity that should be explicitly accounted for to improve those models.

The agency was careful to make distinctions in the technology cost curves based on water source, so perhaps such heterogeneity is also important for modelling the occurrence and concentrations of PFAS at PWS points-of-entry.

10. Are the state-level water quality data used to supplement the federal data when calibrating the Monte Carlo simulation models representative?

EPA relies primarily on federal water quality data to estimate point-of-entry concentrations of PFOA, PFOS, PFNA, HFPO-DA and its ammonium salt, PFHxS, and PFBS. EPA took an extra step and supplemented the federal data with state-level data from 11 states on point-of-entry concentrations. The state-level data are described as being useful because of the lower detection limits for identifying PFAS concentrations (pg. 4-20). At the same time, the EA would be strengthened with additional discussion regarding the representativeness of these state-level data. In particular, is there a potential selection bias to consider?

Additional qualitative discussion of this potential lack of representativeness may be sufficient. Or perhaps additional descriptive statistics can be provided to demonstrate the representativeness of the state-level data from these 11 states compared to the rest of the U.S.; perhaps by comparing PFAS concentrations across states with versus without state-level data based on the more widely available UCMR 3 federal data.

11. EPA provides a supplemental, illustrative analysis of the costs of compliance with the proposed NPDWS if spent filtration materials were regulated as hazardous waste, but to be fully objective, this illustrative analysis should also demonstrate the benefits of such an action.

As noted in comment #3 in the above section, in the main analysis EPA correctly excludes any incremental increase in PWS disposal costs that are specific to hazardous materials. The Agency does, however, provide an illustrative analysis of the disposal costs if PFAS were regulated as a hazardous material.

In my professional opinion, it is misleading to present this illustrative exercise for just the costs, without also discussing the benefits. To be fully transparent and balanced, it would be informative to provide a companion illustrative analysis of the corresponding environmental and health benefits that would result from treating spent filtration materials as a hazardous waste.

12. Any environmental benefits anticipated to result from the proposed regulatory action should be included in the EA.

The quantified and qualitative benefits identified in the EA focus solely on human health. Given the persistence of PFAS pollutants, PFAS that are left untreated by a PWS in the baseline likely would be passed through the entire system and eventually released back into the environment. If this is the case, then the proposed MCLs would reduce the concentrations of PFAS in the public water before it is eventually discharged into the environment, and thus reduce any adverse impacts to ecosystem services and other environmental endpoints. EPA should consult with environmental risk assessors and ecotoxicologists, but if reasonable, then such benefits at the very least deserve a qualitative discussion and consideration in the EA.

13. EPA should reconsider whether additional health benefits can be quantified and/or monetized.

As described in comment #6, EPA went to impressive lengths to identify and qualitatively discuss potential health benefits that it asserted could not be quantitatively estimated. However, in developing the final rule EA, EPA should consider whether there is adequate information to support quantification and/or monetization of additional benefits. As per EPA's (2014) own economic guidelines, analysts should try to get as far as possible in first *identifying* all key benefit and cost categories. The next step (when possible) is to then *quantify* the projected change in each benefit and cost outcome that is expected to result from the policy option, relative to the baseline. Quantifying in this case means to measure the change in terms of some quantitative metric, such as the number of lives saved, number of cases prevented, etc. The final step is to *monetize* the quantified change, meaning that a dollar value is assigned. EPA spent significant effort in monetizing benefits when possible, and then qualitatively identifying and discussing other benefits, but the Agency should consider whether there is adequate information to at least quantify additional benefit categories in the final EA.

As suggested by OMB's recently proposed revisions to Circular A4 (OMB 2023), EPA should consider highlighting the expected magnitude of some of the qualitatively discussed benefits. For example, quantified estimates of the number of individuals potentially exposed, and/or that are susceptible to the increased risks and that would therefore benefit from the proposed regulatory options, would provide further insight and aid the qualitative discussion. In other words, perhaps the quantification step can be taken to some degree for some benefit categories, even if fully monetizing a benefit category is not possible.

Additionally, there may be health outcomes where quantification is not possible, but perhaps monetary cost-of-illness (COI) or willingness to pay (WTP) estimates exist. In such cases, as suggested by the Agency's own economic guidelines (EPA 2014), EPA should consider illustrative analyses or perhaps even a full-blown "break-even" analysis, to provide further insight to the potential magnitude of the qualitatively discussed benefits and plausibility that those unquantified benefits may further result in positive net benefits. Such analyses are recommended in cases where unquantified benefits could be meaningful (OMB 2003, 2023).

Finally, EPA should thoroughly evaluate information submitted during the public comment period for the proposed rule and assess whether additional health benefits can be identified, quantified, and ideally monetized in the EA for the final rule. For example, the comments submitted by Earthjustice and the Natural Resource Defense Council (NRDC) include estimates of lactation duration effects attributable to increased exposures to PFOA, PFOS, PFHxS, and PFNA. Impacts of PFAS exposure on breastfeeding, and the resulting impacts to infant and maternal health are not currently discussed in the EA. At the very least, this is a benefit category that deserves qualitative discussion, and as the commenters' analysis suggests, perhaps quantification and even monetization of these benefits is possible. As another example, on page 6-21 of the EA, EPA states that hepatic effects were not quantified or monetized because PFAS exposure could not be linked to a health endpoint (i.e., an increased incidence of disease). However, more recent studies discussed in the comments submitted by Earthjustice and NRDC may allow for quantification, and perhaps even monetization of this important benefit category.

14. The EA includes several alternative regulatory options, but all options are less stringent than the proposed option. Why were no more stringent regulatory options considered?

OMB's Circular A4 (2003) and EPA's (2014) economic guidelines suggest analyzing an array of alternative regulatory options, with at least one more stringent and one less stringent than the proposed option. The proposed PFAS NPDWR EA only includes less stringent alternative regulatory options. To strengthen the EA, EPA should evaluate a more stringent regulatory option or explicitly describe the rationale for not including a more stringent option. For example, Circular A4 (OMB 2003) states that more stringent regulatory alternatives are not required in cases where the proposed option is near or at the limits of what is technically feasible.

15. EPA should further consider whether the central EA results should focus more on the lower 3% discount rate.

Although equal consideration of alternative 3% and 7% discount rates is currently the standard practice for EAs of federal regulations (OMB 2003), EPA should consider whether the lower 3% discount rate is more appropriate in the current context. This could be the case for several reasons, including the long 80-year time period for the analysis, the fact that future generations are impacted, and the considerable uncertainties in the magnitude of the future health benefits.² Additionally, EPA states on page 2-3 of the EA that “OMB’s Circular A-4 indicates that a 3 percent discount rate represents the rate that an average saver uses to discount future consumption and *is therefore more appropriate for this rulemaking.*” [Emphasis added.] Given this rationale, discussions in the literature (e.g., Howard and Schwartz 2022), and the recently proposed revised guidance from OMB (2023) regarding discount rates, EPA should consider focusing the central analysis primarily on an assumed 3% (or even lower) discount rate, rather than treating the 3% and 7% assumptions as being equally valid in the main analysis.

16. Uncertainty around the cost of illness (COI) estimates for each non-fatal health endpoint that was quantified is not accounted for.

The quantified examination of analytical uncertainty is quite thorough throughout the EA with respect to other “upstream” analytical steps, and in points where it is not, EPA is still transparent in describing any shortcomings. Nonetheless, the reliance on just central COI estimates is a weakness that could be addressed. For example, a sensitivity analysis could be conducted based on the statistical distribution of the COI estimates from the literature, or perhaps other bounding values if the primary studies did not estimate the COI values using statistical methods. That said, perhaps such an exercise is not worth the additional effort if this source of uncertainty is expected to be trumped by numerous other points of uncertainty that are addressed in the Monte Carlo simulations. If this is expected to be the case, then EPA should make such assertions explicit in the EA.

17. Is it possible to incorporate broader opportunity costs into the cost-of-illness (COI) estimates for reduced risks of low birth weight, cardiovascular disease, and renal cancer?

The Agency correctly includes avoided direct expenditures for medical care, as well as broader opportunity costs – namely the opportunity cost of time (e.g., missed work days) – in the COI estimates for reduced risks of bladder cancer. But these broader opportunity costs are not accounted for in the COI estimates for reduced risks of low birthweight, cardiovascular disease, and renal cancer. The COI estimates for these three non-fatal health outcomes only account for

² See the “Discount Rates” section in the proposed revisions to Circular A-4 (OMB 2023), for example, for further details.

avoided medical expenditures. EPA recognizes this shortcoming in various parts of Chapter 6, and points out that these exclusions are due to the lack of available estimates in the literature.

Nonetheless, this omission likely results in a significant underestimate of the benefits. For example, Table 6-43 shows that for bladder cancer (the one non-fatal health outcome where broader opportunity cost estimates are included), the opportunity cost of time beyond medical expenditures makes up 27% to 32% of the total COI estimates for the first year. If a similar proportional scaling is applicable to the other non-fatal health outcomes, then the missing portion of the COI-based benefits for non-fatal cardiovascular disease, renal cancer, and low birthweight cases would be substantial. In the final rule EA, the Agency should consider whether it is possible to incorporate these broader opportunity costs into their COI estimates for these three non-fatal health outcomes.

Even if the inclusion of the broader opportunity costs into these COI estimates is legitimately not possible given the available literature, EPA should still consider a bounding exercise where the available COI estimates for low birth weight, cardiovascular disease, and renal cancer are scaled-up based on the COI estimates for bladder cancer, or perhaps for more similar non-fatal health endpoints where the available literature does provide more comprehensive COI estimates. Such a bounding exercise would require considerable judgement, but at the same time, we know that assuming the opportunity cost of time associated with these adverse health outcomes is zero (as the EPA currently does) is not correct.

18. Did EPA consider using willingness to pay (WTP) estimates to monetize reductions in non-fatal human health outcomes?

The use of COI estimates to monetize the benefits of reduced risks of non-fatal health outcomes, as EPA does, is standard (EPA 2014), and in my professional experience is generally deemed acceptable for policy analysis. Nonetheless, a theoretically more appropriate approach (when available) is to use estimates of WTP (OMB 2003). For example, WTP estimates better account for “pain and suffering and other quality-of-life effects” (OMB 2023, pg. 48). It is unclear whether such estimates are available in the peer-reviewed literature, but EPA should include a review of the literature and discussion of why available WTP estimates are or are not appropriate to use in the EA.

I would also like to point out that the Organisation for Economic Co-operation and Development (OECD) has implemented stated preference studies that estimate the public’s WTP for reductions in the risks of various non-fatal health outcomes that result from reduced exposure to toxic chemicals (OECD, n.d.). Their research includes reductions in the risks of adult asthma, chronic kidney disease, fertility loss, and of particular relevance to the current EA, cases of low birth weight. It is unclear whether the results of the OECD’s studies are yet available, but if they are available now or by the time of the final rule EA is being conducted, then the Agency should consider using such estimates (when applicable) in lieu of the current COI estimates.

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Exhibit 1:
Curriculum Vitae

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EDUCATION:

- Ph.D. Agricultural and Resource Economics, University of Maryland, College Park, MD, 2011.
- M.S. Agricultural and Resource Economics, University of Maryland, College Park, MD, 2009.
- B.S. Environmental and Renewable Resource Economics, The Pennsylvania State University, State College, PA, 2006. *Highest Distinction*. Minor in Geographical Information Systems (GIS).

EMPLOYMENT:

Assistant Professor, Department of Economics, Appalachian State University, Boone, NC.	2018 - present
Research Economist, National Center for Environmental Economics, US Environmental Protection Agency, Washington, DC.	2011 – 2018
Lecturer, Department of Agricultural and Resource Economics, University of Maryland, College Park, MD.	2017 – 2018
Adjunct Assistant Professor, Department of Economics, American University, Washington, DC.	2016 – 2017
Research Assistant, Fondazione Eni Enrico Mattei (FEEM), Milan, Italy.	2010 – 2011
Research Assistant, Department of Agricultural and Resource Economics, University of Maryland, College Park, MD.	2006 – 2010
Research Assistant, Department of Agricultural Economics and Rural Sociology, The Pennsylvania State University, State College, PA.	2005 – 2006

TEACHING:

- Appalachian State University
- ECO 2030: Principles of Microeconomics. 2018-2022.
- ECO 4660/5660: Benefit-Cost Analysis. 2019-2021.
- ECO 4810: Seminar in Economics (Senior Capstone Course). 2022.

TEACHING (cont.):

University of Maryland

AREC 240: Introduction to Economics and the Environment. 2017.

American University

ECON 379: Economics of Environmental Policy. 2016-2017.

Teaching Assistant

AREC 200: The Chesapeake Bay Ecosystem: Intersection of Science, Economics, and Policy. University of Maryland. 2010-2011.

CHEM 12: Chemical Principles. The Pennsylvania State University Berks-Lehigh Valley. 2003.

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“The External Costs of Industrial Chemical Accidents: A Nationwide Property Value Study,” US Department of Agriculture Economic Research Service Seminar Series, Kansas City, MO, September 2022.

“The Impacts of Mitigating Hazardous Chemical Exposure on Infant Health,” Association of Environmental and Resource Economists’ Annual Summer Conference, Miami, FL, June 2022.

“The Property Value Impacts of Industrial Chemical Accidents,” 2022 Appalachian Experimental and Environmental Economics Workshop, Appalachian State University, Boone, NC, April 2022.

“The Property Value Impacts of Industrial Chemical Accidents,” Centre for Environmental and Resource Economics Seminar Series, Umeå University and the Swedish University of Agricultural Sciences, Umeå, Sweden, September 2021.

“Hazardous Waste and Home Values: An Analysis of Treatment and Disposal Sites in the U.S.,” 2021 PLACES Webinar: Land, Water, and ZTRAX, Boston, MA, June 2021.

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“The Property Value Impacts of Groundwater Contamination: Agricultural Runoff and Private Wells,” presented at the Northeastern Agricultural and Resource Economics Association’s Water Quality Economics Workshop, Newport, RI, June 2015.

“Do Home Values Respond to Underground Storage Tank Releases? An Examination of High-Profile Cases Across the US,” presented at the Northeastern Agricultural and Resource Economics Association’s and the Canadian Agricultural Economic Society’s Joint Annual Meeting, Newport, RI, June 2015.

“A Stated Preference Study of the Chesapeake Bay and Watershed Lakes,” presented at the Allied Social Science Associations’ (ASSA) 2015 Meeting, Boston, MA, January 2015.

“The Implicit Price of Aquatic Grasses,” presented at Resources for the Future’s (RFF) Academic Seminar Series, Washington, DC, October 2014.

“The Implicit Price of SAV: A Hedonic Study of the Chesapeake Bay,” presented at the Northeast Agricultural and Resource Economics Association’s Annual Meeting, Morgantown, WV, June 2014.

“To Sell or Not To Sell: The Impacts of Pollution on Home Transactions,” presented at the 6th Annual Conference of the Society for Benefit Cost Analysis, Washington, DC, March 2014.

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“To Sell or Not To Sell: The Impacts of Pollution on Home Transactions,” presented in Sponsored Session at the Association of Environmental and Resource Economists’ 3rd Annual Summer Conference, Banff, Canada, June 2013.

“Can Property Values Capture Changes in Environmental Health Risks: Evidence from a Stated Preference Study in Italy and the UK,” presented at the Association of Environmental and Resource Economists’ 2nd Annual Summer Conference, Asheville, NC, June 2012.

“What do Property Values Really Tell Us? A Hedonic Study of Pollution from Underground Storage Tanks,” presented at the Annual Conference of the Society for Benefit Cost Analysis, Washington, DC, October 2011.

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“A Hedonic Analysis of the Impact of LUST Sites on House Prices,” presented at the Association of Environmental and Resource Economists’ Inaugural Summer Conference, Seattle, WA, June 2011.

“Environmental Health Risks and Home Values: A Revealed and Stated Preference Analysis,” presented in the Research Sketches Session at the National Bureau of Economic Research (NBER) Environmental and Energy Economics Workshop, Cambridge, MA, July 2010.

“The Effects of LUSTs on Home Values: Is Proximity Enough?,” presented at the 4th World Congress of Environmental and Resource Economists, Montreal, Canada, July 2010.

“LUSTs as Contaminated Sites: What are the Effects on Property Values?,” presented at the 17th Annual Conference of the European Association of Environmental and Resource Economists, Amsterdam, The Netherlands, June 2009.

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Program Committee Member: Association of Environmental and Resource Economists' sponsored sessions at the Southern Economic Association Annual Conference, 2019.

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Session Chair: Benefit-Cost Analysis session sponsored by the Association of Environmental and Resource Economists at the Southern Economic Association's Annual Conference, Washington, DC, 2018.

Session Chair: Hedonic Price Studies and BCA session at the 10th Annual Conference of the Society for Benefit Cost Analysis, Washington, DC, 2018.

Book Reviewer: *Environmental & Natural Resource Economics* (11th ed.) by Tom Tietenberg & Lynne Lewis, Taylor & Francis, 2017.

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Moderator: The Northeastern Agricultural and Resource Economics Association's and the Canadian Agricultural Economic Society's Joint Annual Meeting, Newport, RI, 2015.

Member: Selected Papers and Symposia Committee, Northeastern Agricultural and Resource Economics Association's 2016 Annual Conference, 2015-2016.

Moderator: US Environmental Protection Agency sponsored workshop on the Economics of Water Quality Improvement in Chesapeake Bay, Washington, DC, 2011.

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Exhibit C

Analysis of the USEPA Proposed PFAS National Primary Drinking Water Regulation Treatment Costs and Comparison to the AWWA National PFAS Cost Model Report

May 30, 2023

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Analysis of the USEPA Proposed PFAS National Primary Drinking Water Regulation Treatment Costs and Comparison to the AWWA National PFAS Cost Model Report

Prepared by Elin Betanzo, Safe Water Engineering, LLC

Executive Summary

The presence of per- and polyfluoroalkyl substances (PFAS) in drinking water sources has emerged as a pressing environmental and public health concern over the last decades. These persistent and bioaccumulative chemicals, commonly found in firefighting foam, nonstick cookware, and numerous other consumer products, have been linked to various adverse health effects. As a result, the United States Environmental Protection Agency (EPA) has undertaken the task of proposing regulations to limit the concentration of PFAS in drinking water.

The proposed regulation would establish enforceable maximum contaminant levels (MCLs) of PFAS in drinking water supplies. This analysis, requested and paid for by the Natural Resources Defense Council, focuses on the potential financial burden of installing treatment to comply with the EPA's proposed PFAS regulation. This analysis also includes a comparison with the American Water Works Association (AWWA) PFAS National Cost Model Report (AWWA, 2023), which provides an industry perspective on the costs associated with PFAS treatment.

These analyses seek to understand realistic treatment costs that would be triggered by the regulation. Furthermore, comparing the EPA's cost estimates to those provided by the AWWA National PFAS Cost Model Report will offer valuable insights into the potential variations and discrepancies between regulatory projections and industry-based assessments. This exploration of the intricacies of these analyses will help inform the overall Economic Analysis to ensure costs are assigned to the rule that will realistically allow water systems to install treatment and achieve the public health benefits anticipated for the new requirements.

Although the USEPA analyzed complete compliance costs for the proposed rule, including sampling and state oversight, this present analysis focuses only on the cost of treatment installation and annualized operations and maintenance (O&M). When AWWA published their analysis, the published proposed rule was not yet available. AWWA estimated the cost of 3 potential compliance options. Although the first AWWA compliance scenario, MCLs of 4 ppt for PFOA and PFOS, does not match the EPA proposed rule (which also includes a Hazard Index of 1.0), it does match USEPA's Option 1a. Because the same modeling methods are used in all options presented in each report, this analysis compares EPA's Option 1a to AWWA's first compliance scenario.

Table 1: EPA and AWWA Modeled Annualized Water System Treatment Costs for Achieving 4.0 ppt for PFOA and PFOS

	EPA	AWWA
Modeled Annualized WS Treatment Costs for achieving 4.0 ppt for PFOA and PFOS	\$658,510,000	\$3,803,926,000

Estimating national costs for drinking water rules requires applying assumptions and professional judgement, especially when there are limitations in availability of occurrence data. Although it is a tricky task, it is a necessary task to estimate the necessary funding for protecting public health. Both cost estimates inform and help move the conversation forward. This memo attempts to identify the sources of the \$3.1 billion difference between these two cost estimates and identify which modeling assumptions are more likely to reflect a realistic compliance forecast and reasonable engineering judgement.

The analysis that follows shows that the \$3.1 billion dollar difference in annualized cost can be explained by the following primary factors:

1. **Discount rate used.** EPA presented cost estimates using both a 3% and 7% discount rate, which is consistent with current OMB guidance. AWWA presented costs for only the 7% rate. In the Economic Analysis, EPA notes that the lower, consumption-based discount rate is more appropriate for this rulemaking (Economic Analysis at 2-3), a conclusion that is supported by the economics literature (Howard and Schwartz, 2002). This factor alone explains \$1 billion of the cost difference (Appendix A). The AWWA approach in this case is an overestimate. Factoring other cost considerations described below, in addition to the excess discount rate, would increase the magnitude of impact of the different discount rates. Further, OMB recently published draft revisions to its guidance (White House Office of Management and Budget, 2023) that favor a substantially lower consumption-based discount rate of 1.7%, consistent with Howard and Schwartz (2002). EPA's assertion that the lower, consumption-based discount rate is more appropriate for this rulemaking is supported by the literature and OMB's proposed update to its guidance. Both indicate that even the 3% consumption-based discount rate EPA used here may be significantly higher than appropriate.
2. **Screening of occurrence datasets.** Both EPA and AWWA relied on UCMR3 data collected by water utilities across the country. In addition, both approaches also used state data. EPA, as documented in Cadwallader et al., 2022, screened state data to only look at finished water samples and limited to UCMR3 water systems so occurrence samples would not be biased high due to non-PWS PFAS samples or PWS samples collected by water systems investigating known PFAS contamination sites (not necessarily used as drinking water sources). The inclusion of non-PWS samples resulted in higher median PFOA and PFOS data in the AWWA analysis and explains AWWA's 4,709 small systems required to comply vs EPA's 3,251 (Appendix B). The potential bias in the AWWA method indicates either the EPA approach is more appropriate, or the actual answer is somewhere between the two estimates. On the other hand, AWWA found a lower occurrence rate in large systems compared to EPA. This may be due to reliance on the UCMR3 data with higher detection limits for large system sampling. A model was described in Cadwallader et al., 2022 to fill in the non-detect median PFOA and PFOS levels. Developing a model may have been beyond the scope of the AWWA 2023 estimate, resulting in the difference in large systems exceeding the MCLs.
3. **Treatment of Entry Points.** EPA calculates treatment and O&M cost per entry point to the distribution system (EPTDS) that exceeds the MCL using modeled system and flow characteristics. Some PWSs have more than one entry point that requires treatment; costs are assigned per entry point that exceeds the MCL. AWWA calculates the number of PWSs that

exceed the MCL, assumes that every PWS that exceeds the MCL has the average number of entry points per system for a system in that size category, and assumes every entry point in that system will require treatment regardless of whether a given entry point exceeds the MCL. This assumption results in 3,645 more entry points with treatment installation in the AWWA analysis that may not actually require treatment, and impacts both capital and O&M costs. A conservative estimate of the overall impact of this assumption, using the number of PWSs that AWWA estimated exceed the MCL along with EPA's metric of 1.3 entry points exceeding the MCL per PWS for small systems and 3.1 for large systems, is an overestimate of \$1.4 billion (Appendix C).

4. **Non-treatment compliance options.** AWWA assumes all PWSs that exceed the MCL will install treatment at every entry point, and EPA assumes that small systems will explore interconnections with other complying systems and new sources before installing treatment. As one example, Michigan Department of Environment, Great Lakes, and Energy (EGLE) personnel have indicated that it is policy to investigate safe water options before considering installation of treatment on a source that exceeds an MCL (Smith, Personal Communication 2023). Michigan's analysis exploring the existing Michigan MCLs and the proposed EPA rule indicates that EPA's estimates of systems opting for non-treatment are reasonable and may even be low (Smith, Personal Communication 2023). Michigan anticipates that up to 26% of PWSs requiring treatment may be able to establish a new connection with another PWS that already meets the MCLs. In this case, both the EPA and the AWWA cost estimate for treatment may be high. Using EPA's assumptions for establishing new interconnections or new sources would result in at least \$159 million in savings compared to AWWA. (Appendix D).
5. **Differences in flow calculations.** EPA and AWWA use different average flow assumptions. EPA uses inventory data, and AWWA assumes 150 gpd per person with published populations to calculate average flow. According to the USGS (Dieter et al., 2018), the AWWA assumption of 150 gpd per person is high, compared to their estimate of 82 gpd. This indicates that EPA flows may be more appropriate. The AWWA estimate also does not reflect regional differences in water use. Even though essentially the same peaking factors are used, AWWA ends up with higher design flows for systems serving <10,000 compared to EPA (Appendix E). The higher AWWA flows result in larger capital and O&M costs relative to actual flow requirements and are magnified even further when applied to the overestimate of entry points requiring treatment in small systems. On the other hand, Appendix E shows that for ground water CWSs serving $\geq 10,000$ EPA flows are higher than AWWA estimated flows. This is not an apples-to-apples comparison because the EPA estimate does not include surface water CWSs, but it means there may not be a differential impact in costs for systems serving $\geq 10,000$. The total magnitude of the net impact of differences in flow between the two estimates is unclear because equivalent datasets are not available. However, the AWWA excess flows assumed for small systems likely results in net larger magnitude costs in AWWA compared to EPA.
6. **Cumulative impact of cost assumptions for small systems (serving <10,000).** The EPA model assumes that package plants, at a lower cost point, are available for entry points <1 MGD. Package plants have been an option for reducing costs for small systems for decades (National Drinking Water Clearinghouse, 1997), but this option is not described as incorporated in the AWWA estimate. The EPA estimate for cost savings from package plants may be conservative as

some package plants may be available up to 6 MGD flows and PWSs typically seek the lowest cost option available for compliance. The cumulative impact of the points raised here: lack of package plants, higher than actual design flows, treatment of unnecessary entry points, and lack of non-treatment options means that AWWA does not provide a realistic cost estimate for small systems.

7. **Magnitude of treatment cost inputs.** EPA's Economic Analysis (2023a), Technologies and Costs document (2023b), and Work Breakdown Structure Model documents (e.g., 2021a) provide hundreds of pages of documentation of the EPA cost analysis process whereas AWWA 2023 provides 36 pages with no references. The difference in documentation makes it impossible to compare cost inputs one to one, but it is possible to insert AWWA's flow assumptions into the EPA model to explore the net magnitude of underlying cost assumptions by comparing average systems. Appendix F shows that for small system capital costs, AWWA estimates treatment for a single entry point up to two times higher than EPA (e.g., \$2.2 million compared to \$1.1 million per entry point for systems serving 500-1,000 people). For large system capital costs, the EPA model for midlevel ground water CWS costs generates larger costs than the AWWA published averages. In Appendix G, application of the EPA model using AWWA average flows generates annual O&M cost estimates that are the same or larger than AWWA's average cost estimates, indicating that AWWA O&M cost inputs may be lower than EPA's. AWWA does not provide a treatment forecast showing the percent of systems expected to implement any given treatment. This analysis provides limited insight on determining the cumulative difference of cost inputs between the EPA and AWWA analyses. The relative magnitude of underlying costs is unknown, but nonetheless has a major impact on the net outcome of total national costs.

The evidence provided in this memo demonstrates that the EPA cost estimate is robust. While there are several items that could not be directly compared to the AWWA cost model, there is no evidence that EPA is consistently underestimating occurrence or costs. According to calculations shown in Appendix G, if AWWA's O&M costs are accurate (\$30,000-125,000 per entry point), this could mean that EPA's O&M cost estimates (\$27,000-2,515,000) are larger than necessary and may be lower in practice. If Michigan's rate of non-treatment options is relevant nationwide, the total EPA cost would fall even further.

The EPA cost estimate of \$658.5 million appears to be the more realistic result based on the calculations and findings presented here.

While professional judgement must be used in applying cost modeling assumptions, it appears that several of the assumptions in the AWWA cost model are too conservative. These assumptions consistently result in higher capital and O&M costs for treatment, especially for systems serving fewer than 10,000. As shown in Table 2, the analyses presented here demonstrate the AWWA estimate includes at least \$2.6 billion in excess costs. Many of these overestimates have cascading effects that could not be modeled with available data. The cumulative impact of these corrections is likely even larger than estimated here. Subtracting the excess costs from AWWA's total estimate would result in a maximum annual cost of \$1.2 billion for treatment installation and O&M.

Table 2: Total Magnitude of Quantified Excess Costs in the AWWA Cost Model

Cost Element	Magnitude of Excess Costs
Discount rate	\$1,000,000,000
Overestimate for entry points requiring treatment	\$1,400,000,000
Non-treatment compliance options	\$159,000,000
O&M cost correction	\$37,000,000
Total	\$2,596,000,000

Analysis

Tables are presented below that compare the two cost modeling approaches and outcomes. These evaluate occurrence data, public water system (PWS) inventory data, capital cost data and analytical approaches, and O&M.

Table 3: Occurrence Factors

Cost element	EPA Cadwallader et al., 2022	AWWA Seidel and Samson, 2022 AWWA, 2023	Approach Generating Larger Cost Estimate	Discussion and Observations
Occurrence data	Only uses finished water samples. Investigative sampling excluded	No screening of finished water samples described; investigative samples included. Dataset Includes non-PWS sources: surface and ground water, landfills, military bases, etc. Magnitude of this contribution is unclear.	AWWA	As described in Seidel and Samson, 2022, AWWA results may be biased high.
Occurrence data	Only uses samples from PWS that participated in UCMR3 to prevent biasing toward states where data from additional PWS was available	Assumed existing occurrence data is representative of national occurrence, although potential for bias acknowledged	AWWA	Could result in underestimation from EPA while also biasing AWWA high. Net impact unclear.
Number of UCMR3 samples included in analysis	36,972	36,972	n/a	
Number of State samples included in analysis	6,645 PFOS 6,656 PFOA 4,715 PFHpA 5,114 PFHxS	19,791 PFOS 20,149 PFOA 15,138 PFHpA 17,649 PFHxS	AWWA	No screening of AWWA results for finished water samples is described, potentially biasing results high.

Cost element	EPA Cadwallader et al., 2022	AWWA Seidel and Samson, 2022 AWWA, 2023	Approach Generating Larger Cost Estimate	Discussion and Observations
Percent systems with detections	33% PFOS 40% PFOA 20% PFHpA 25% PFHxS	39% PFOS 34% PFOA 33%PFHpA 28% PFHxS	Unclear	Same percent exceedance of combined PFOS and PFOA. Costs for treatment of multiple species were included in AWWA analysis but not explicitly shown.
Medians of state data	1.76 PFOS 1.58 PFOA 0.66 PFHpA 1.09 PFHxS	2.0 PFOS 2.0 PFOA 1.7 PFHpA 1.7 PFHxS	AWWA	Cost impact is two-fold: Increase in number of systems that require treatment Increase in treatment cost to remove larger magnitude of contaminant.
Number of Systems serving <=10,000 Exceeding PFOS and PFOA 4 ppt	Table 4-19 3251	Table 6-1 minus the systems which have already addressed treatment due to state requirements (Table 6-3): 4709	AWWA	Nearly 1500 more small systems are expected to require treatment per AWWA. With AWWA cost estimates for small systems greatly exceeding EPA's, this has a significant impact on the bottom line.
Number of Systems serving >10,000 Exceeding PFOS and PFOA 4 ppt	Table 4-19 1060	Table 6-1 minus the systems which have already addressed treatment due to state requirements (Table 6-3): 277	EPA	EPA expects about 783 more large systems will need to install treatment to comply with the rule. EPA and AWWA costs per large system are closer to each other than for small systems. Cadwallader et al., 2022 used a model to fill in the non-detect median PFOA and PFOS levels for large systems, which may explain the difference in large systems exceeding the MCLs

Cost element	EPA Cadwallader et al., 2022	AWWA Seidel and Samson, 2022 AWWA, 2023	Approach Generating Larger Cost Estimate	Discussion and Observations
Number of System Entry Points serving <=10,000 Exceeding PFOS and PFOA 4 ppt	Table 4-23 in the Economic Analysis estimates: 4,327	Table 6-1 minus the entry points which have already addressed due to state requirements (Table 6-3): 10,049	AWWA	AWWA assumes all entry points at a water system with one MCL exceedance will install treatment. This overestimate is compounded multiple times throughout the cost estimate.
Number of System Entry Points serving >10,000 Exceeding PFOS and PFOA 4 ppt	Table 4-23 in the Economic Analysis estimates: 3,238	Table 6-1 minus the entry points which have already addressed due to state requirements (Table 6-3): 1,161	EPA	It's not clear why EPA estimates so many more large systems, and consequently, entry points, will exceed the MCLs compared to the AWWA estimate.
State data included in occurrence analysis	Colorado Illinois Kentucky Massachusetts Michigan New Hampshire New Jersey North Dakota Ohio South Carolina Vermont	Colorado Kentucky Massachusetts New Hampshire New Jersey South Carolina Vermont Alaska Arizona California Delaware Minnesota Missouri North Carolina New Mexico New York Pennsylvania Rhode Island West Virginia	Unclear	States with some of the lower detection rates (particularly for non-targeted PFAS data) were not included in the AWWA analysis (e.g., Illinois, Michigan, North Dakota, Ohio, Vermont); reason for not including is not clear. The AWWA dataset covers more geographies.

Table 4: Capital Treatment Selection and Costs

Cost element	EPA	AWWA	Approach Generating Larger Cost Estimate	Discussion and Observations
PWS Inventory for considering treatment	Uses standard PWS designs to estimate costs for a median sized system in each category.	Considers each PWS with occurrence data	Unclear	
Number of Entry Points to the Distribution System	Combination of UCMR3 and SDWIS/Fed facility data presented in Economic Analysis Table 4-6: GW <=100: 90% have only 1 EPTDS	AWWA Letter to Congressional Budget Office Re: S.1507 - PFAS Release Disclosure Act, dated August 8, 2019, which incorporated updates to information originally collected by EPA's Community Water System Survey. Range from 2.4-14.5.	AWWA	The relative magnitude and origin of the underlying numbers is unclear; they become significant with AWWA's assumption that all entry points must be treated (see below).
Average Flows	Geometries and Characteristics of PWSs (USEPA, 2000)	150 gpdpc	AWWA for smaller systems	Dieter et al. (2018) found average water use per capita at 82 gallons per day. Basing flow on population data doesn't account for regional differences in water use and the actual flow at the water system. Appendix E shows AWWA estimates are high for small systems compared to EPA flow data.
Peak Flows	Geometries and Characteristics of PWSs (USEPA, 2000)	Peaking Factor	same	Average/Design flow ratios are not substantially different (Appendix E).
Package Plants	EPA uses them <1 MGD, resulting in lower costs	Does not describe	AWWA	Impact is large on Size Categories 1-3.

Cost element	EPA	AWWA	Approach Generating Larger Cost Estimate	Discussion and Observations
Non-treatment options - new connections or wells included as compliance options	Non-treatment is considered only for entry points with design flows less than or equal to 3.536 MGD.	No non-treatment options are included in the analysis.	AWWA	Significant bias in AWWA cost. Using EPA's compliance forecast (appendix D), which may be low according to Michigan data (indicates up to 26% of systems may be able to interconnect), this assumption increases the AWWA estimate by at least \$159 million.
Number of treatment points per PWS	SafeWater MCBC is used to apply costs for a treatment technology or nontreatment alternative only at each entry point in a PWS estimated to be out of compliance with the regulatory option under consideration.	If one EPTDS exceeds the regulatory option under consideration, assumes installation of a treatment system at each EPTDS regardless of contaminant levels. If a PWS is assumed to exceed an MCL at any EPTDS, the EPTDS treatment cost is multiplied by the average number of EPTDS.	AWWA	AWWA cost is biased higher in two ways: 1) treatment costs are assumed at locations that may not require treatment and 2) EPTDS that meet the MCL might be usable for blending to bring down treatment costs at the EPTDS that do exceed the MCL.
Treatment selection	Screens for viable options first, then option selected based on estimated cost, using cost equations for up to 49 different flow rates.	Treatment technology with lowest life-cycle cost selected. Costs were independently calculated for IX, GAC vessels, GAC basins, and RO. Does not include impact of TOC on selection or cost of treatment. Monte Carlo analysis is used to estimate the range of varying TOC, sulfate, pH, and alkalinity on treatment cost.	AWWA	It is possible that the high cost of EPA non-viable options might exclude these options from the AWWA estimate, which is based on lowest cost. However, EPA's inclusion of non-treatment options results in a lower capital cost estimate.

Cost element	EPA	AWWA	Approach Generating Larger Cost Estimate	Discussion and Observations
Compliance Forecast (% systems installing what treatment)	50-85% of entry points will install GAC	Not Provided	Unclear	Unable to compare EPA's method of excluding non-viable treatment options vs AWWA method of selecting the lowest cost treatment option and the lack of non- treatment options.
Safety Factor	Target effluent set at 80% of regulatory limit. Accounts for contaminants below MCL to account for chromatographic peaking.	Target effluent set at 80% of regulatory limit.	EPA	EPA assumption is more conservative than AWWA.
Technology exclusions	GAC is excluded if EPTDS influent TOC > 3.2 mg/L. IX is excluded if total influent PFAS > 7,044 ppt. GAC and IX are excluded if PFAS removal > 99% is required. GAC is excluded for bed lives <5,000 and IX excluded for bed lives <20,000.	None described	Unclear	Not clear if the increased cost of these options would automatically exclude them in the AWWA analysis.

Cost element	EPA	AWWA	Approach Generating Larger Cost Estimate	Discussion and Observations
Reverse Osmosis Costs	Although some systems will blend treated with untreated water, EPA uses the conservative assumption that blending is only used when less than 95% removal is required. More blending scenarios are likely during implementation.	No blending assumptions are described.	AWWA	Even though the EPA assumption is conservative and may result in a higher than actual compliance cost, the AWWA assumption of no blending results in an even higher Capital and O&M cost for RO.
Automation	Cost equations are for fully automated systems, minimizing the need for operator intervention and reducing operator labor costs.	Emphasis on automation in treatment selection to decrease O&M cost not described.	not clear	May increase EPA capital costs but decrease EPA O&M cost

Cost element	EPA	AWWA	Approach Generating Larger Cost Estimate	Discussion and Observations
Quantified Uncertainty	Monte-Carlo uncertainty analysis with the SafeWater MCBC. Applied to TOC value and compliance technology unit cost curve selection: random selection from a triangular distribution of low-, mid-, and high- cost equipment (25%, 50%, and 25% respectively). At the end of 4,000 iterations, SafeWater MCBC outputs the expected value as well as 90% confidence interval for each cost metric (bounded by 5th and 95th percentiles).	Monte Carlo analysis using major factors for GAC, ion exchange, and reverse osmosis/nanofiltration. Triangular distributions where assigned for all factors except RO recovery. For each modeled scenario, each of the modeled costs (10th percentile, 90th percentile, and most probable) was stored as a modeled output for each system represented in the occurrence database for use in determining the overall national cost of compliance with the modeled limit.	Not clear	Two similar but different procedures are described. Net result is unclear.

Cost element	EPA	AWWA	Approach Generating Larger Cost Estimate	Discussion and Observations
Estimate class and/or peer review	Peer review of GAC model indicated budget estimates would be +30 to -15% Peer review of IX model indicated estimates in range of +50 to -30, with an emphasis on the high range but has been revised since then. Non-treatment model peer review: costs ranged widely with the biggest unknown being land costs that vary regionally.	Capital costs generated for individual systems represent a Class 5 Association for the Advancement of Cost Engineering (AACE) estimate, at approximately 1 to 2 percent maturity level of deliverable definition. According to AACE, a class 5 estimate is more likely to be a high estimate than a low estimate, with the 80 percent confidence interval ranging at the low end from -20% to -50% to +30 to +100%.	AWWA	The AWWA estimate could range as high as twice the actual cost, whereas EPA estimate could be as high as 1.5 times the actual cost.
Cost curves	National database used for decades.	Empirically derived cost curves as a function of size using several decades of infrastructure project design and delivery at Black and Veatch to estimate cost for these major components.	Unclear	Net result is unclear.

Cost element	EPA	AWWA	Approach Generating Larger Cost Estimate	Discussion and Observations
Capital cost markups	Contingencies based on Project budget ranging from 4-7%. The WBS models assume contingency costs incurred only in high-cost scenarios (see Section 2.3). For low and medium cost estimates, none is incurred. EPA includes line items not in described in the AWWA model: • Residuals management equipment • Pilot testing • Geotechnical • Standby power • Sales tax • Financing	Includes 30% contingency for all treatment installations. The AWWA Contingency does not vary based on project dollar value.	AWWA	Although the EPA contingency may be an underestimate, the AWWA 30% contingency is on top of the Level 5 assessment. A realistic value may be between the two estimates.
Systems that have already installed treatment to comply	The EPA model accounts for systems and states that have already installed treatment to comply with PFAS MCLs.	Only accounts for systems in states with existing MCLs. For example, does not account for treatment installed at Cape Fear Public Utility Authority that is used for some treatment estimates.	AWWA	The cost to install treatment already in place should not be attributed to the new PFAS regulation.

Cost element	EPA	AWWA	Approach Generating Larger Cost Estimate	Discussion and Observations
Cost Inputs	Multiple technical documents are provided describing complex models built in Excel workbooks. Treatment forecasts are available, but discrete cost points are not.	High level approaches are described but no treatment forecast, discrete cost values or cost breakouts by treatment alternatives are provided.	Unclear	Calculations in Appendix F using EPA's formulas and AWWA inputs indicate AWWA cost inputs may exceed EPA by 1-2 times as much for small systems, but AWWA costs for large systems may be lower than EPA's.
NTNCWs	Includes cost of NTNCWS compliance.	Does not include NTNCWS compliance.	EPA	

Table 5: Operations and Maintenance Costs

Cost element	EPA	AWWA	Approach Generating Larger Cost Estimate	Discussion and Observations
Flows used to calculate O&M costs	Average Flows (modeled)	Average Flows (based on per capita consumption estimates)	AWWA	As described in Table 3, AWWA average flows appear high for small systems.
O&M Correction	N/A	Table A-2 shows that AWWA estimates lower O&M costs to comply with a more stringent standard for systems in categories 1 and 5. If it costs less to comply with a more protective standard, a PWS would select this alternative. Correcting for this assumption would result in additional \$37 million in savings.	AWWA	See Appendix H for the calculation.
Media Life	Bed life estimates using linear equations based on pooled data from several studies of GAC and IX performance and reflect central tendency results under varying water quality conditions. Increments of 5,000 BV for GAC and 20,000 BV for IX, resulting in media replacement every 2-5 months depending on average flow at EPTDS.	Clark, 1987 non-linear model for GAC and IX. Values for GAC were derived from a pilot study for CFPUA. Values for IX were derived from CFPUA and LHHCWD pilot data.	Unclear	EPA uses a more representative dataset compared to the AWWA model that only uses data from two PWSs. The AWWA approach uses values from only two pilot studies to extrapolate to the entire country. It is unlikely that these two PWSs are representative of the average PWS nationwide.

Cost element	EPA	AWWA	Approach Generating Larger Cost Estimate	Discussion and Observations
Disposal Costs	Assumes PFAS-contaminated wastes are not considered hazardous wastes, but accounts for waste disposal costs.	Waste disposal is included in O&M Costs.	Unclear	Unclear
Cost Inputs	Multiple technical documents are provided describing complex models built in Excel workbooks. Discrete cost points are not easily extracted for comparison purposes.	Discrete cost values are available in Table 5-8 but the net difference in input costs and methodology are difficult to identify and calculate due to the very different methods in each approach.	EPA	In Appendix G, application of the EPA model using AWWA average flows generates O&M estimates that are the same or larger than AWWA's average cost estimates, indicating that AWWA O&M cost inputs are lower than EPA's.

A case study for actual costs incurred can be identified from the AWWA analysis. Table 6-3 quantifies one PWS serving >1,000,000 that installed treatment in response to state PFAS MCLs with an AWWA estimated capital cost of \$407.5 million and an annualized cost of \$47 million. The water utility was identified via UCMR3 monitoring data. Information on this water system (O'Connell and Kilcommons, 2023) states the following: "Through a mix of blending, GAC treatment, and taking wells offline that have contaminant detections over the MCL that they are currently in compliance with the state requirements. This PWS anticipates additional treatment may be needed to meet the proposed federal requirements." To date, they have spent:

- \$15.0 million from 2016 – 2022 on PFAS related work
- \$36.2 million on Emerging Contaminant Work
- \$21.2 million on 1,4-Dioxane work

Although this is not the complete capital cost for the work (as more is pending), and some of the emerging contaminant and 1,4-Dioxane work may result in PFAS reduction benefits, the cost to date is significantly less than the estimated capital cost of \$407.5 million presented in AWWA Table 6-3. Even if the \$15 million is the actual annualized cost of PFAS treatment alone, it would be less than one third of the annualized cost shown in AWWA Table 6-3.

Unmodeled sources of uncertainty in both cost estimates

There are some unmodeled sources of uncertainty that were not included in both estimates. One factor for which inclusion would increase cost would be the potential for competition for supplies and supply chain issues driving up prices if thousands of PWS nationwide try to install treatment at the same time. On the other hand, there are sources of uncertainty not included in both estimates that would have the impact of decreasing overall costs, including:

- Innovation in the marketplace
- Innovation/new technology for PFAS destruction/disposal
- Future options for point-of-use (POU) compliance
- Increasing the number of PWS that consolidate with other PWS that meet MCLs instead of installing treatment.

In the case of this last option, according to EGLE, state primacy agencies focus on finding a safe source prior to exploring treatment options. Michigan expects up to 33% of PWS and 26% of CWS to connect to another system before installing treatment, indicating that EPA's estimate of 6-7% of systems with <3.536 MGD design flow is not only reasonable, but possibly low (Smith, personal communication May 23, 2023). This omission of non-treatment options by AWWA is the source of at least \$159 million in difference between the AWWA and EPA cost estimates. If Michigan's results are found to hold true nationwide, up to \$362 million in savings could be realized in comparison to the AWWA cost estimate.

Conclusion

As discussed throughout this memo, assumptions used in AWWA cost modeling of the proposed PFAS drinking water rule result in higher annualized capital and O&M costs for treatment, especially for

systems serving fewer than 10,000. While AWWA cost inputs for O&M may be lower than EPA's inputs, the larger number of entry points installing treatment per AWWA result in cumulative increased O&M costs. Although several details that are not provided cannot be quantified, the source of \$2.6 billion in excess costs are clearly identified in this analysis and in Table 2. Many of these overestimates have cascading effects that are not readily calculated without access to the underlying cost models, so the cumulative impact of these corrections is likely even larger than estimated here. Subtracting the overestimates from the AWWA cost estimate results in a revised AWWA annual estimate of \$1.2 billion for treatment and O&M to comply with the proposed MCLs. While there is a possibility that the actual result lies between the EPA and AWWA cost estimates, the calculations here indicate it is more likely to be closer to the EPA estimate.

The evidence provided in the executive summary and Tables 3-5 demonstrate that the EPA cost estimate is robust. While there are several items that could not be directly compared between the EPA and AWWA cost models, there is no evidence that EPA is consistently underestimating occurrence or costs. When looking toward which cost estimate is likely to better reflect future compliance decisions, the EPA cost estimate appears to be more realistic based on the calculations and findings presented here.

If AWWA's apparent lower O&M cost inputs are accurate, this could mean that EPA's O&M cost estimates are higher than necessary and will be lower during implementation. If Michigan's rate of non-treatment options is relevant nationwide, the total EPA estimated cost would fall even further. The EPA \$658 million annual treatment cost projection is realistic, and there are several opportunities for actual costs to turn out even lower upon implementation.

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Appendix A: Recalculation at 3% discount rate

AWWA 4 ppt PFOA and PFOS at 3% plus corrected O&M										Discount Rate	0.03		
PWS Size	Population Range Annualized	Average CAPEX/PWS	Average O&M/PWS	Annualized PWS Cost	Estimated Number of Impacted PWSs	National C	Column1	Net Impacted PWS (Impacted PWS - State MCL Impacted PWS)					
1	<100	\$ 1,920,000	\$ 72,000	\$ 177,000	2167	\$ 383,559,000		1406					
2	101-500	\$ 3,400,000	\$ 60,000	\$ 289,000	2469	\$ 713,541,000		1660					
3	501-1,100	\$ 4,620,000	\$ 63,000	\$ 374,000	609	\$ 227,766,000		359	Small systems	4709			
4	1,001-3,300	\$ 5,510,000	\$ 57,000	\$ 427,000	858	\$ 366,366,000		746					
5	3,301-10,000	\$ 11,000,000	\$ 176,000	\$ 893,000	781	\$ 697,433,000		538	Large Systems	277			
6	10,001-50,000	\$ 24,490,000	\$ 372,000	\$ 2,018,000	255	\$ 514,590,000		156					
7	50,001-100,000	\$ 45,510,000	\$ 512,500	\$ 3,571,000	64	\$ 228,544,000		58					
8	100,001-1,000,000	\$ 110,880,000	\$ 891,000	\$ 8,344,000	71	\$ 592,424,000		60					
9	>1,000,000	\$ 507,500,000	\$ 3,045,000	\$ 37,157,000	4	\$ 148,628,000		3					
All Systems					7278	\$3,872,851,000		4986					
State Costs in the report, recalculated at 3%.													
PWS Size Category	Population Range	% Impacted	Average CAPEX/PWS	Average O&M/PWS	Annualized PWS Cost	Estimated Number of Impacted PWSs	Annualized Total Cost	Present Value of Lifecycle Cost					
1	<100	7%	\$1,920,000	\$48,000	\$177,000	761	\$ 134,697,000						
2	101-500	5%	\$3,400,000	\$60,000	\$289,000	809	\$ 233,801,000						
3	501-1,100	5%	\$4,620,000	\$63,000	\$374,000	250	\$ 93,500,000						
4	1,001-3,300	1%	\$5,510,000	\$76,000	\$446,000	112	\$ 49,952,000						
5	3,301-10,000	5%	\$10,560,000	\$132,000	\$842,000	243	\$ 204,606,000						
6	10,001-50,000	3%	\$24,180,000	\$310,000	\$1,935,000	99	\$ 191,565,000						
7	50,001-100,000	1%	\$43,050,000	\$594,500	\$3,488,000	6	\$ 20,928,000						
8	100,001-1,000,000	3%	\$98,340,000	\$1,848,000	\$8,458,000	11	\$ 93,038,000						
9	>1,000,000	4%	\$407,450,000	\$8,555,000	\$35,942,000	1	\$ 35,942,000						
All Systems		4%				2292	\$ 1,058,029,000						

Appendix B: Number of impacted systems and entry points

Table 4-19: Total Systems Impacted, Option 1a (PFOA and PFOS MCLs of 4.0 ppt)				Table 6-1 AWWA Systems Impacted Minus Impacted State PWSs of Table 6-3	4-23: Total Entry points Impacted, Option 1a	AWWA Table 6-1 minus impacted State EPTDS of Table 6-3
	5th Percentile	Mean	95th Percentile		Mean	Nationally impacted entry points
Small Systems <10,000						
Total Number of PWSs	61,463	61,463	61,463			
PWSs With PFOS Exceedance	1,801	2,905	4,260			
PWSs With PFOA Exceedance	836	1,520	2,422			
PWSs That Exceed One or More MCLs	2,111	3,251	4,676	4709	4,327	10,049
Large Systems >10,000						
Total Number of PWSs	4,433	4,433	4,433			
PWSs With PFOS Exceedance	721	791	868			
PWSs With PFOA Exceedance	803	878	959			
PWSs That Exceed One or More MCLs	975	1,060	1,145	277	3,238	1,161
All Systems						
Total Number of PWSs	65,896	65,896	65,896			
PWSs With PFOS Exceedance	2,522	3,696	5,128			
PWSs With PFOA Exceedance	1,639	2,399	3,381			
PWSs That Exceed One or More MCLs	3,086	4,310	5,821		7,564	11,211

Appendix C: Estimate of impact of excess EPTDS

Population Range	Impacted PWS	State Impacted PWS	Net PWS	Average EPTDS	AWWA EPTDS Requiring treatment	Annualized PWS Cost	Annualized cost per EPTDS	EPTDS Requiring Treatment Using EPA Rate	AWWA cost of EPTDS Requiring Treatment	EPA Cost of EPA EPTDS Requiring Treatment
<100	2167	761	1406	2.4	3374.4	\$ 253,000	\$ 105,417	1871	\$ 355,718,000	\$ 197,271,581
101-500	2469	809	1660	2.0	3320.0	\$ 381,000	\$ 190,500	2209	\$ 632,460,000	\$ 420,894,251
501-1,100	609	250	359	2.1	753.9	\$ 499,000	\$ 237,619	478	\$ 179,141,000	\$ 113,539,146
1,001-3,300	858	112	746	1.9	1417.4	\$ 577,000	\$ 303,684	993	\$ 430,442,000	\$ 301,530,304
3,301-10,000	781	243	538	2.2	1183.6	\$ 1,173,000	\$ 533,182	716	\$ 631,074,000	\$ 381,792,623
10,001-50,000	255	99	156	3.1	483.6	\$ 2,684,000	\$ 865,806	477	\$ 418,704,000	\$ 412,587,813
50,001-100,000	64	6	58	4.1	237.8	\$ 4,808,000	\$ 1,172,683	177	\$ 278,864,000	\$ 207,768,438
100,001-1,000,000	71	11	60	6.6	396.0	\$ 11,357,000	\$ 1,720,758	183	\$ 681,420,000	\$ 315,385,643
>1,000,000	4	1	3	14.5	43.5	\$ 50,949,000	\$ 3,513,724	9	\$ 152,847,000	\$ 32,200,298
			4986	Sum EPTDS	11,210			SUM	\$ 3,760,670,000	\$ 2,382,970,098
							Estimate of impact of excess EPTDS =			\$ 1,377,699,902
		EPA Small System Impacted EPTDS/Impacted PWS ratio	1.3	AWWA Small system impacted EPTDS/impacted PWS ratio	2.1					
		EPA Large System Impacted EPTDRS/Impacted PWS ratio	3.1	AWWA Large system Impacted EPTDS/impacted PWS ratio	4.2					

Appendix D: Cost Savings of non-treatment options

Cost Savings via non-treatment options												
Size Category	Population	MGD @150gpd WITAF 56 Average flows	Design Flows	Mid Cost Results for New Well T&C Document Figure 7-6	Mid Cost Results for New Well O&M T&C Document Figure 7-6	Annualized Costs Using EPA average cost and AWWA top of range flows	Impacted PWS	Impacted EPTDS	Estimated Number of New Well PWS (low = 2%)	Estimated Number of Impacted PWS (high = 10%)	Annualized National Cost (Low)	Annualized National Cost (High)
1	100	0.015	0.0645	\$ 360,682.04	\$ 4,173.14	\$ 28,416.64	1406	3374	67	67	\$ 1,917,782	\$ 1,917,782
2	500	0.075	0.27	\$ 458,211.74	\$ 6,638.54	\$ 37,437.56	1660	3320	66	66	\$ 2,485,854	\$ 2,485,854
3	1,000	0.15	0.495	\$ 538,379.66	\$ 9,550.52	\$ 45,738.09	359	754	15	15	\$ 689,639	\$ 689,639
4	3,300	0.495	1.386	\$ 1,121,295.27	\$ 33,402.34	\$ 108,770.99	746	1417	28	28	\$ 3,083,440	\$ 3,083,440
5	10,000	1.345	3.536	\$ 2,564,950.50	\$ 91,341.58	\$ 263,746.55	538	1184	24	24	\$ 6,243,408	\$ 6,243,408
										Cost of New Wells	\$ 14,420,124	\$ 14,420,124
										AWWA treatment for same number of systems	\$ 94,020,838	\$ 94,020,838
										\$ Saved with New Well assumption	\$ 79,600,714	\$ 79,600,714
Size Category	Population	MGD @150gpd WITAF 56 Average flows	Design Flows	Mid Cost Results for Interconnection T&C Document Figure 7-7	Mid Cost Results for Interconnection O&M T&C Document Figure 7-7	Annualized Costs Using EPA average cost and AWWA top of range flows	Impacted PWS	Impacted EPTDS	Estimated Number of New Well PWS (low = 6.7%)	Estimated Number of Impacted PWS (high = 25%)	Annualized National Cost (Low)	Annualized National Cost (High)
1	100	0.015	0.0645	\$ 382,781.01	\$ 18,072.28	\$ 43,801.18	1406	3374	236	844	\$ 10,346,189	\$ 36,950,673
2	500	0.075	0.27	\$ 420,653.92	\$ 90,354.24	\$ 118,628.79	1660	3320	232	830	\$ 27,569,330	\$ 98,461,893
3	1,000	0.15	0.495	\$ 452,132.73	\$ 180,706.68	\$ 211,097.10	359	754	53	188	\$ 11,140,227	\$ 39,786,526
4	3,300	0.495	1.386	\$ 543,407.09	\$ 596,401.41	\$ 632,926.90	746	1417	99	354	\$ 62,797,741	\$ 224,277,648
5	10,000	1.345	3.536	\$ 682,442.58	\$ 1,620,464.91	\$ 1,666,335.77	538	1184	83	296	\$ 138,059,252	\$ 493,068,756
										Sum	\$ 249,912,739	\$ 892,545,495
										AWWA treatment for same number of systems	\$ 329,072,933	\$ 1,175,260,475
										\$ Saved with Interconnection assumption	\$ 79,160,194	\$ 282,714,980
										Sum of cost savings via new wells and interconnections	\$ 158,760,908	\$ 362,315,694

Appendix E: Average and Design Flows

EPA			AWWA			
Design Flow (MGD)	Average Flow (MGD)	Peaking Factor	Design Flow (MGD)	Average Flow (MGD)	Peaking Factor	Delta EPA-WITAF
			0.007	0.0015	4.7	
			0.022	0.0054	4.1	
0.030	0.007	4.3	0.037	0.0095	3.9	0.4
			0.091	0.025	3.6	
0.124	0.035	3.5				
			0.18	0.054	3.3	
			0.27	0.084	3.2	
0.305	0.094	3.2	0.36	0.11	3.3	0.0
0.740	0.251	2.9	0.68	0.23	3.0	0.0
			1	0.3	3.3	
2.152	0.819	2.6	2	0.77	2.6	0.0
			3.5	1.4	2.5	
7.365	3.200	2.3	7	3	2.3	0.0
			17	7.8	2.2	
22.614	11.087	2.0	22	11	2.0	0.0
75.072	37.536	2.0	76	38	2.0	0.0

System Size	Table 4-9: Design and Average Daily Flow for CWSs EPA Ground Water			AWWA Equivalent			AWWA to EPA Design Flow Ratio
	Average Population	Design Flow (MGD)	Average Flow (MGD)	Design Flow (MGD)	Average Flow (MGD)	Peaking Factor	
≤ 100	61	0.028	0.007	0.043	0.009	4.7	1.54
101–500	250	0.107	0.030	0.169	0.038	4.5	1.58
501–1,000	734	0.301	0.093	0.363	0.110	3.3	1.21
1,001–3,300	1,865	0.733	0.248	0.895	0.280	3.2	1.22
3,301–10,000	5,673	2.121	0.806	2.127	0.851	2.5	1.00
10,001–50,000	20,697	7.305	3.171	7.140	3.105	2.3	0.98
50,001–100,000	67,222	22.512	11.031	20.167	10.083	2.0	0.90
100,001–1M	203,821	71.371	35.685	61.146	30.573	2.0	0.86

Abbreviations: CWS – community water systems; MGD – million gallons per day.

AWWA uses essentially the same peaking factors as EPA. The mean for AWWA average flows ≤ 0.37 MGD is 4.2, a difference of 0.1. This may have the impact of lower EPA cost estimates for the lowest flows, however the EPA consideration of less expensive package plants for flows < 1 MGD likely results in an overestimate of costs in the AWWA report.

Appendix F: Capital Cost Input Estimate

Size Category	Assumed Population	MGD @150gpd (AWWA Assumed Average flow)	Design Flow (AWWA peaking factor)	Mid Cost Results for Removal of PFAS from Groundwater Using Pressure GAC T&C Document Figure 7.2	Mid cost results for removal of PFAS from Groundwater using IX T&C Document Figure 7.3	Mid cost Results for Removal of PFAS from Groundwater using RO T&C Document Figure 7.4	Average of Midrange EPA Groundwater treatment costs using AWWA design flows for the top population of each size category	AWWA Average Capital cost per entry point (Table 6-1)	AWWA to EPA cost ratio
1	100	0.015	0.0645	\$ 686,746.50	\$ 144,418.66	\$ 1,423,072.73	\$ 751,412.63	\$ 800,000.00	1.1
2	500	0.075	0.27	\$ 778,085.22	\$ 258,496.39	\$ 1,733,665.26	\$ 923,415.62	\$ 1,700,000.00	1.8
3	1,000	0.15	0.495	\$ 940,576.52	\$ 376,607.25	\$ 2,016,864.60	\$ 1,111,349.45	\$ 2,200,000.00	2.0
4	3,300	0.495	1.386	\$ 2,159,302.43	\$ 1,489,043.77	\$ 3,401,507.67	\$ 2,349,951.29	\$ 2,900,000.00	1.2
5	10,000	1.5	3.75	\$ 3,561,947.46	\$ 2,845,388.48	\$ 5,693,520.69	\$ 4,033,618.88	\$ 4,800,000.00	1.2
6	50,000	7.5	16.5	\$ 9,631,776.59	\$ 9,374,281.37	\$ 15,117,104.00	\$ 11,374,387.32	\$ 7,900,000.00	0.7
7	100,000	15	30	\$ 14,846,302.37	\$ 16,003,911.78	\$ 23,659,330.75	\$ 18,169,848.30	\$ 11,100,000.00	0.6
8	1,000,000	150	270	EPA mid-cost GW model does not cover design flow >200 MGD					
9	>1,000,000	350	520						

Appendix G: O&M Cost Input Estimate

Size Category	Assumed Population	MGD @150gpd (AWWA Assumed Average flow)	O&M Mid Cost Results for Removal of PFAS from Groundwater Using Pressure GAC T&C Document Figure 7.2	O&M Mid cost results for removal of PFAS from Groundwater using IX T&C Document Figure 7.3	O&M Mid cost Results for Removal of PFAS from Groundwater using RO T&C Document Figure 7.4	Average of Midrange EPA Groundwater treatment costs using WITAF 56 design flows for the top end of each size category	AWWA Average O&M cost ((Table A-1 Column 4)/ (Average EPTDS))	AWWA to EPA cost ratio
1	100	0.015	\$ 28,243.53	\$ 6,376.71	\$ 48,097.57	\$ 27,572.60	\$30,000	1.1
2	500	0.075	\$ 33,673.53	\$ 15,800.31	\$ 69,954.98	\$ 39,809.61	\$30,000	0.8
3	1,000	0.15	\$ 43,510.21	\$ 27,296.39	\$ 93,801.74	\$ 54,869.45	\$30,000	0.5
4	3,300	0.495	\$ 116,883.10	\$ 75,656.86	\$ 233,169.69	\$ 141,903.22	\$30,000	0.2
5	10,000	1.5	\$ 228,798.78	\$ 229,263.22	\$ 628,114.08	\$ 362,058.69	\$80,000	0.2
6	50,000	7.5	\$ 812,108.79	\$ 1,154,598.53	\$ 2,114,215.21	\$ 1,360,307.51	\$120,000	0.1
7	100,000	15	\$ 1,460,783.53	\$ 2,270,651.60	\$ 3,812,647.18	\$ 2,514,694.10	\$125,000	0.0
8	1,000,000	150	EPA model does not cover average flow >100 MGD					
9	>1,000,000	350						

Appendix H: O&M Correction

Original AWWA Table A-1								
PWS Size	Population Range Ann	Average CAPEX/PWS	Average O&M/PWS	Annualized PWS Cost	Estimate	National Cost	Discount Rate	0.07
1 <100		\$1,920,000	\$72,000	\$253,000	2167	\$548,251,000		
2 101-500		\$3,400,000	\$60,000	\$381,000	2469	\$940,689,000		
3 501-1,100		\$4,620,000	\$63,000	\$499,000	609	\$303,891,000		
4 1,001-3,300		\$5,510,000	\$57,000	\$577,000	858	\$495,066,000		
5 3,301-10,000		\$10,560,000	\$176,000	\$1,173,000	781	\$916,113,000		
6 10,001-50,000		\$24,490,000	\$372,000	\$2,684,000	255	\$684,420,000		
7 50,001-100,000		\$45,510,000	\$512,500	\$4,808,000	64	\$307,712,000		
8 100,001-1,000,000		\$110,880,000	\$891,000	\$11,357,000	71	\$806,347,000		
9 >1,000,000		\$507,500,000	\$3,045,000	\$50,949,000	4	\$203,796,000		
All Systems					7278	\$ 5,206,285,000		
Substitute lower A-2 costs per system size into Table A-1 (if it costs less to meet a higher standard, why not always choose that technology option?)								
PWS Size	Population Range Annua	Average CAPEX/PWS	Average O&M/PWS	Annualized PWS Cost	Estimated I	National Cost		
1 <100		\$1,920,000	\$48,000	\$ 229,000	2167	\$496,243,000		\$ 229,234
2 101-500		\$3,400,000	\$60,000	\$ 381,000	2469	\$940,689,000		\$ 380,936
3 501-1,100		\$4,620,000	\$63,000	\$ 499,000	609	\$303,891,000		\$ 499,095
4 1,001-3,300		\$5,510,000	\$57,000	\$ 577,000	858	\$495,066,000		\$ 577,105
5 3,301-10,000		\$11,000,000	\$154,000	\$ 1,192,000	781	\$930,952,000		\$ 1,192,322
6 10,001-50,000		\$24,490,000	\$372,000	\$ 2,684,000	255	\$684,420,000		\$ 2,683,683
7 50,001-100,000		\$45,510,000	\$512,500	\$ 4,808,000	64	\$307,712,000		\$ 4,808,322
8 100,001-1,000,000		\$110,880,000	\$891,000	\$ 11,357,000	71	\$806,347,000		\$ 11,357,288
9 >1,000,000		\$507,500,000	\$3,045,000	\$ 50,949,000	4	\$203,796,000		\$ 50,949,410
All Systems					7278	\$ 5,169,116,000		
			average annual	\$ 8,075,111				
							O&M correction savings=	\$ 37,169,000

Corr, Elizabeth

From: Chris Moody <CMoody@awwa.org>
Sent: Tuesday, August 8, 2023 4:49 PM
To: Lisa Daniels
Cc: Corr, Elizabeth
Subject: Clarification of AWWA's Cost Analysis for the PFAS Rule
Attachments: Appendix C - Supplemental Figures Comparison Costs for EPA WBS BV Model and Case Studies.pdf

Good Afternoon Ms. Daniels –

This afternoon's NDWAC conversation about the EPA's Economic Analysis suggested that there may be some misunderstanding among the Council's membership about the approach that the American Water Works Association used to estimate the national and system-level cost of drinking water treatment to comply with the proposed PFAS standards.

To ensure that the NDWAC has an accurate understanding of the data that underpins AWWA's comments to NDWAC this morning as well as our submissions to the rulemaking record, I offer the following clarification.

AWWA's comments relied on an analysis by Black & Veatch which estimated capital and O&M costs for each treatment technology (GAC, IX, and RO) and for each system in the occurrence database projecting costs based on the treatment with the lowest life cycle cost. The estimates provided rely on as much of an apples-to-apples comparison with EPA's analysis as possible with the data provided by EPA in the record. The analysis features identical discount rates (3% and 7%) and EPA's projected occurrence of PFAS in drinking water systems (and subsequently entry points impacted) across system sizes.

To provide a more complete evaluation of the EPA's cost analysis for different treatment options, there is also a comparative assessment of cost projections by EPA and Black & Veatch against a collection of case study data (more than 100 data points) on a treatment capacity (design MGD) basis. This approach ensured a comparative assessment that minimized the impact of varying assumptions. The case study comparison to each model (EPA and Black & Veatch) is provided in Appendix C of our comment letter and is attached for your convenience.

Thank you for time and consideration in this matter,

Chris Moody, PE

Regulatory Technical Manager
American Water Works Association

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Appendix C

Supplemental Figures Comparing Case Study Data with EPA and BV Cost Models

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- Figure A 2: Comparison of GAC Operating Costs for Small Systems (<2.5 MGD)
- Figure A 3: Comparison of GAC Capital Costs for Medium Systems (<10 MGD)
- Figure A 4: Comparison of GAC Operating Costs for Medium Systems (<10 MGD)
- Figure A 5: Comparison of IX Capital Costs for Small Systems (<2.5 MGD)
- Figure A 6: Comparison of IX Operating Costs for Small Systems (<2.5 MGD)
- Figure A 7: Comparison of IX Capital Costs for Medium Systems (<10 MGD)
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- Figure A 9: Comparison of RO Capital Costs for Small Systems (<2.5 MGD)
- Figure A 10: Comparison of RO Operating Costs for Small Systems (< 2.5 MGD)
- Figure A 11: Comparison of RO Capital Costs for Medium Systems (<10 MGD)
- Figure A 12: Comparison of RO Operating Costs for Medium Systems (<10 MGD)

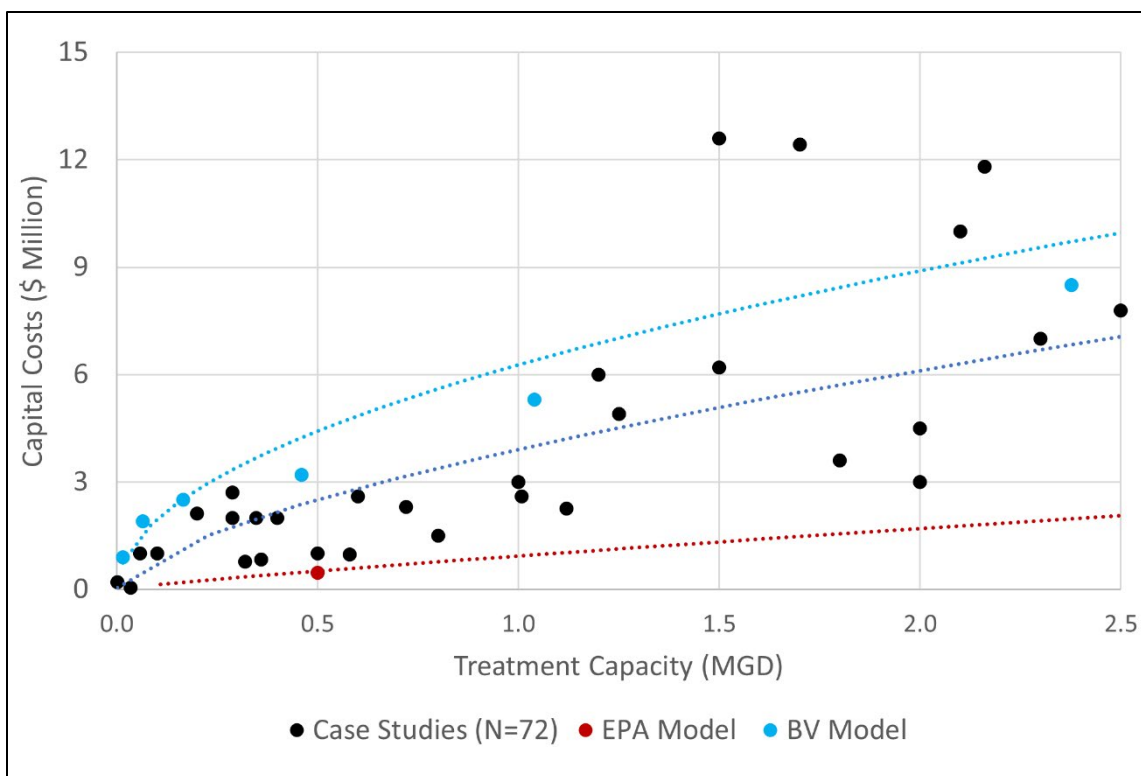


Figure A 13: Comparison of GAC Capital Costs for Smaller Systems (<2.5 MGD)

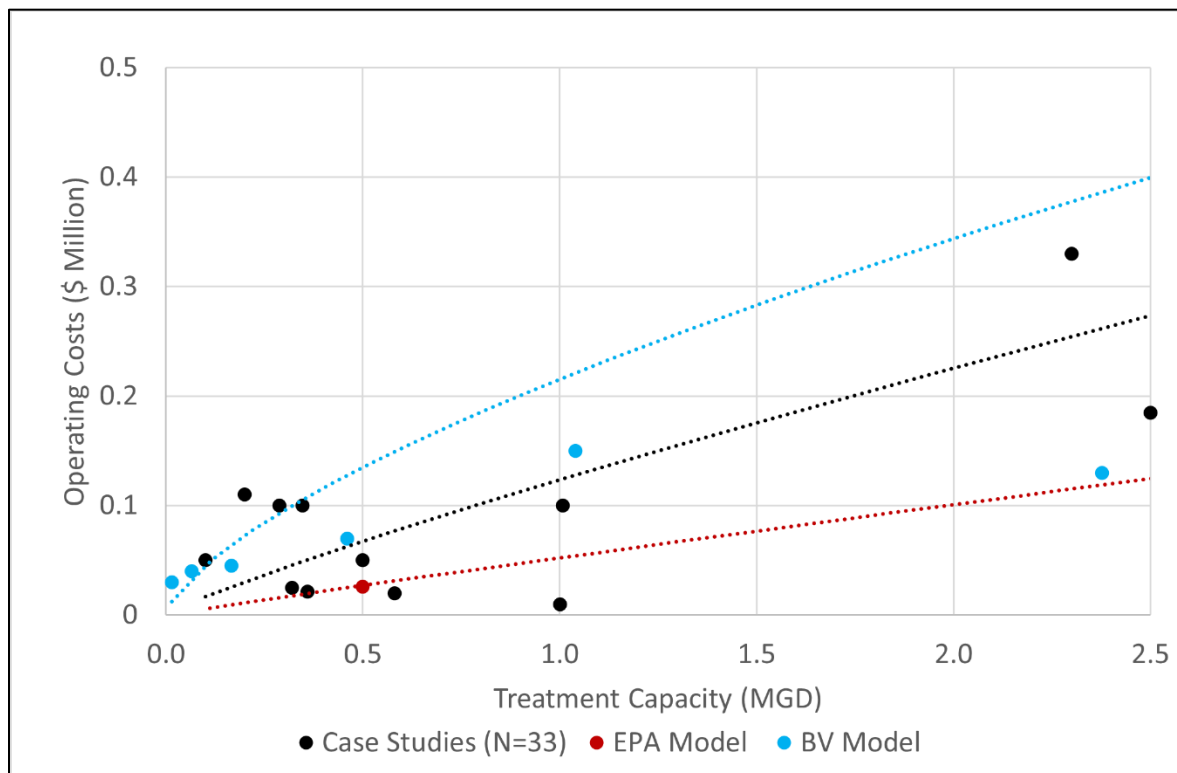


Figure A 14: Comparison of GAC Operating Costs for Small Systems (<2.5 MGD)

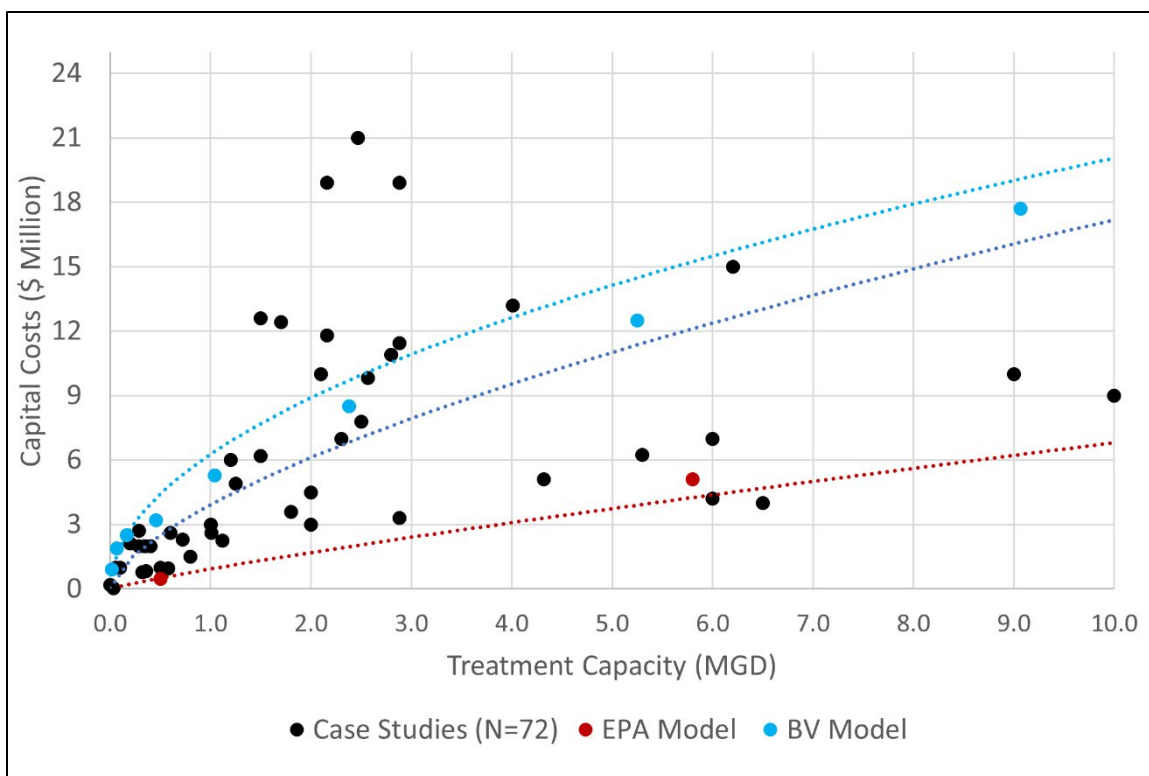


Figure A 15: Comparison of GAC Capital Costs for Medium Systems (<25 MGD)

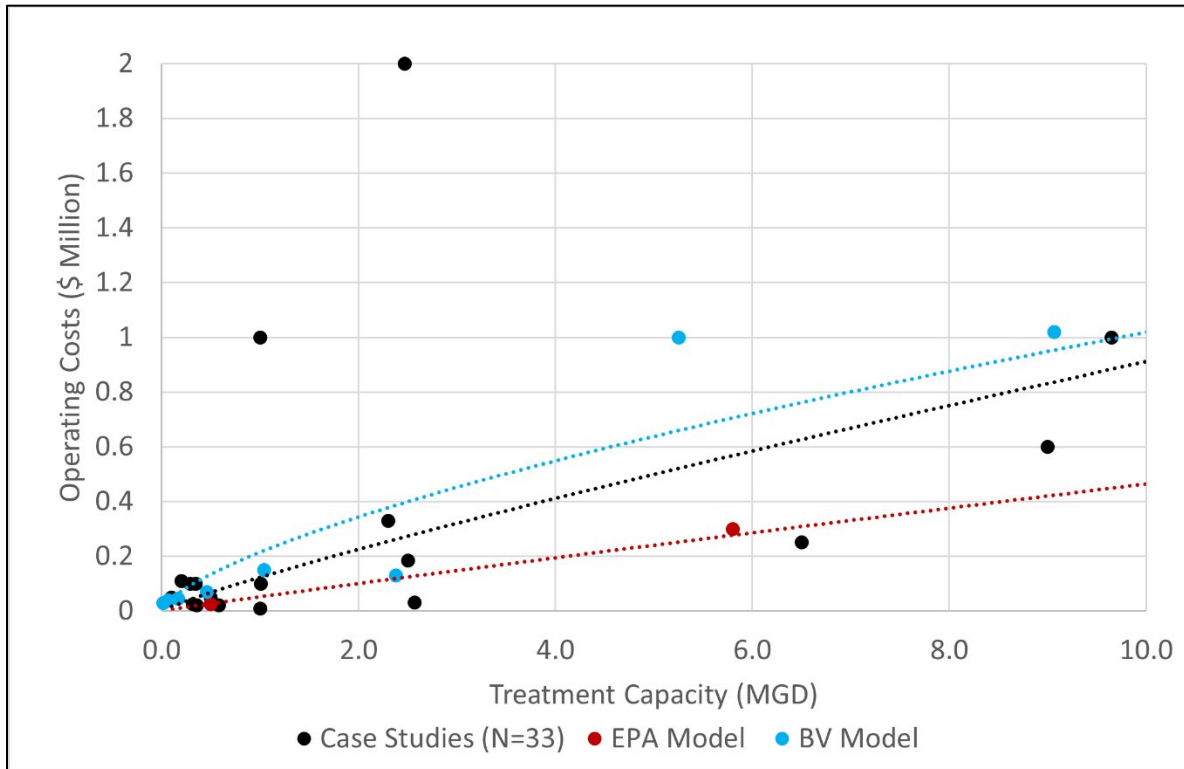


Figure A 16: Comparison of GAC Operating Costs for Medium Systems (<10 MGD)

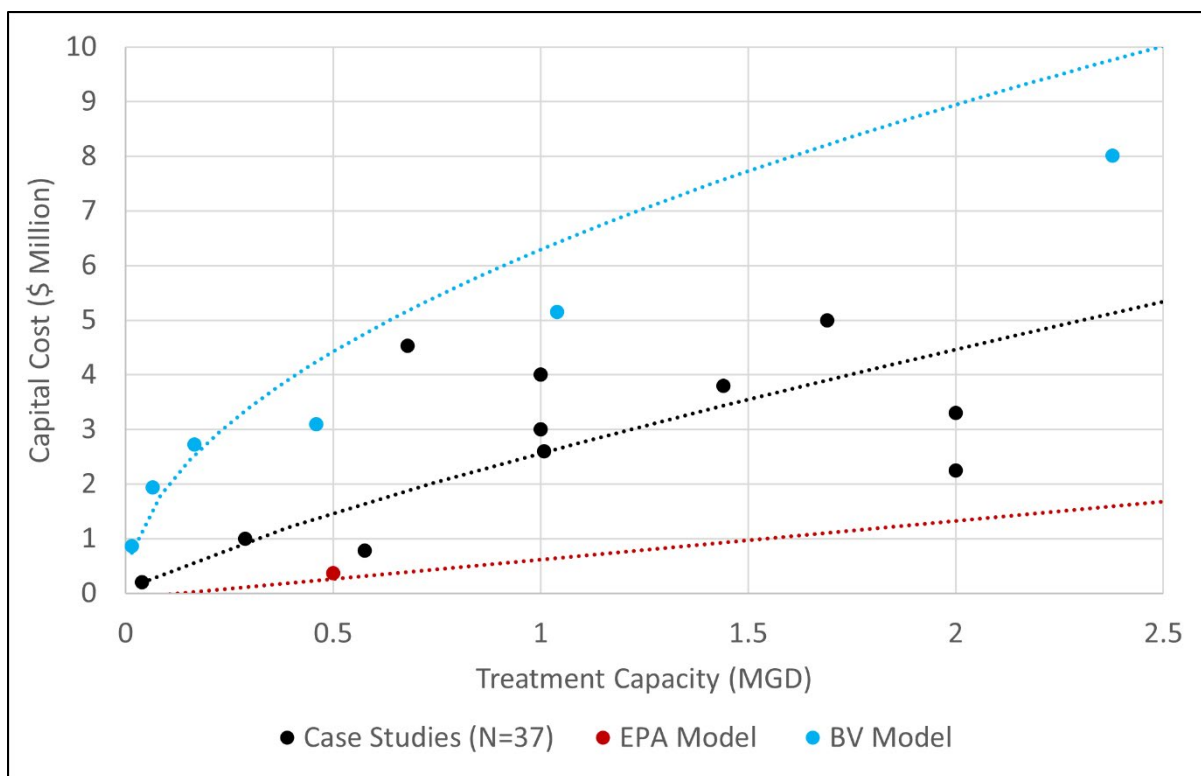


Figure A 17: Comparison of IX Capital Costs for Small Systems (<2.5 MGD)

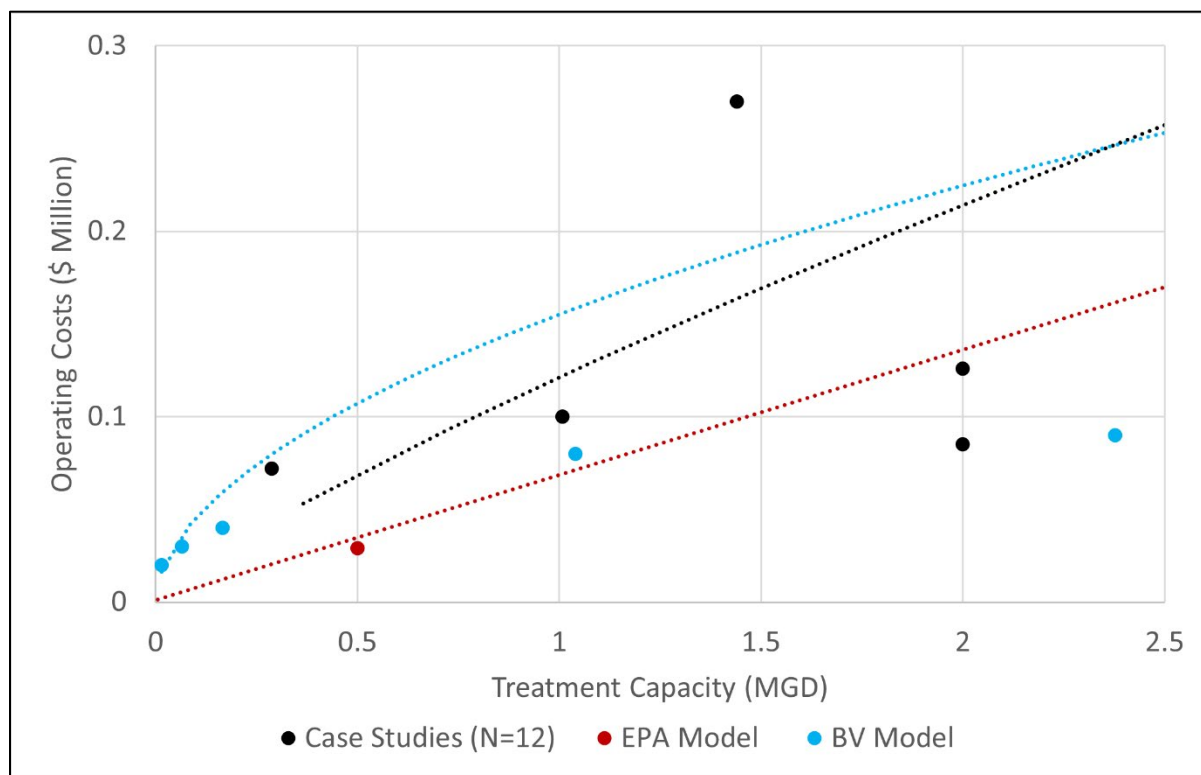


Figure A 18: Comparison of IX Operating Costs for Small Systems (<2.5 MGD)

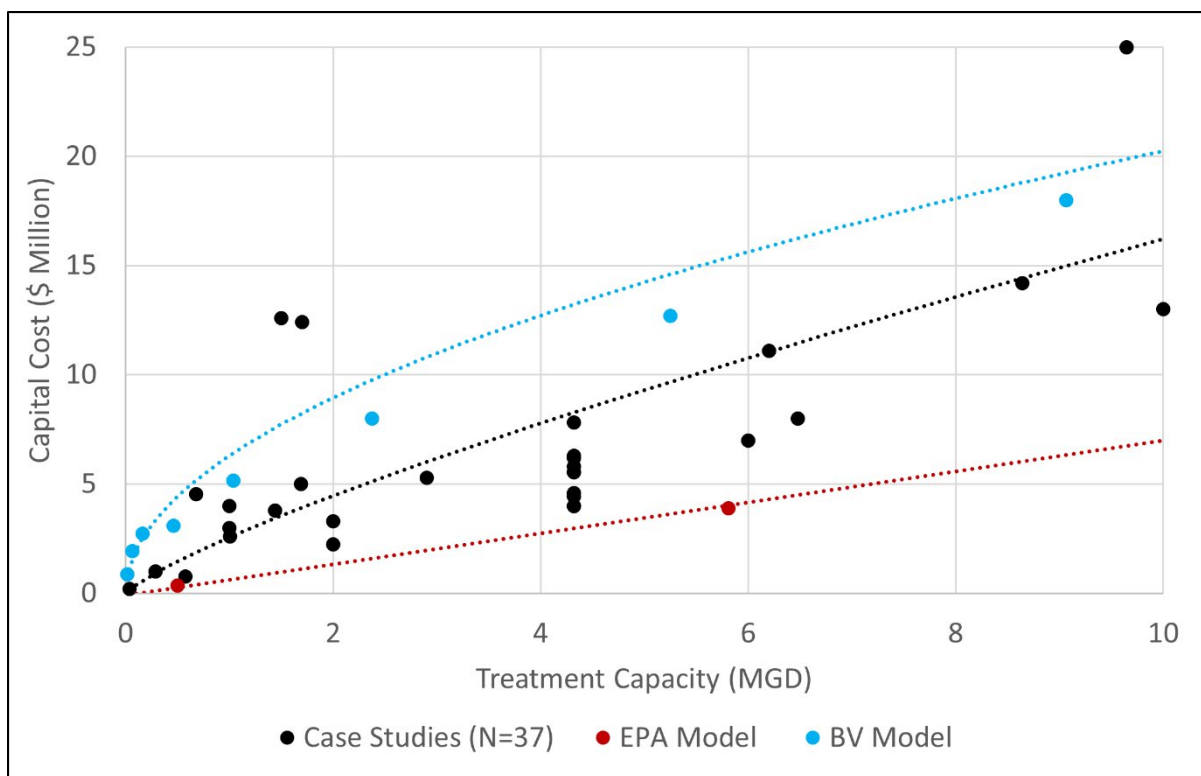


Figure A 19: Comparison of IX Capital Costs for Medium Systems (<10 MGD)

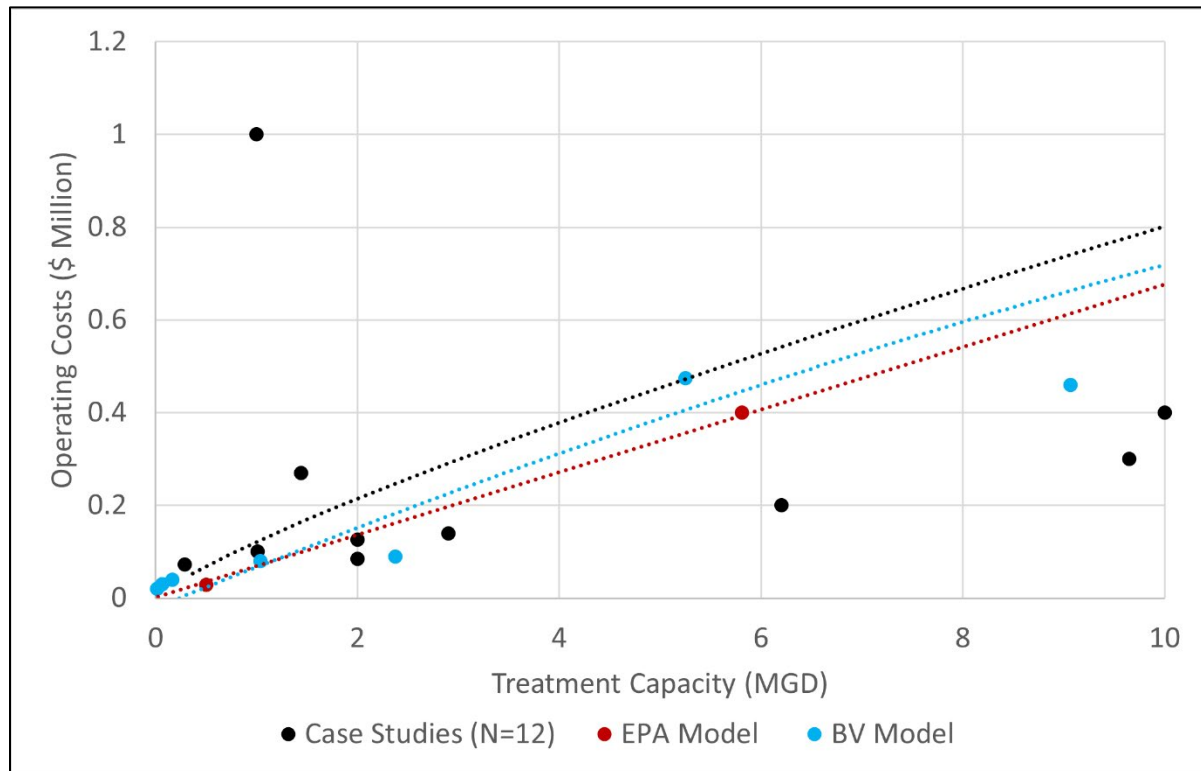


Figure A 20: Comparison of IX Operating Costs for Medium Systems (<10 MGD)

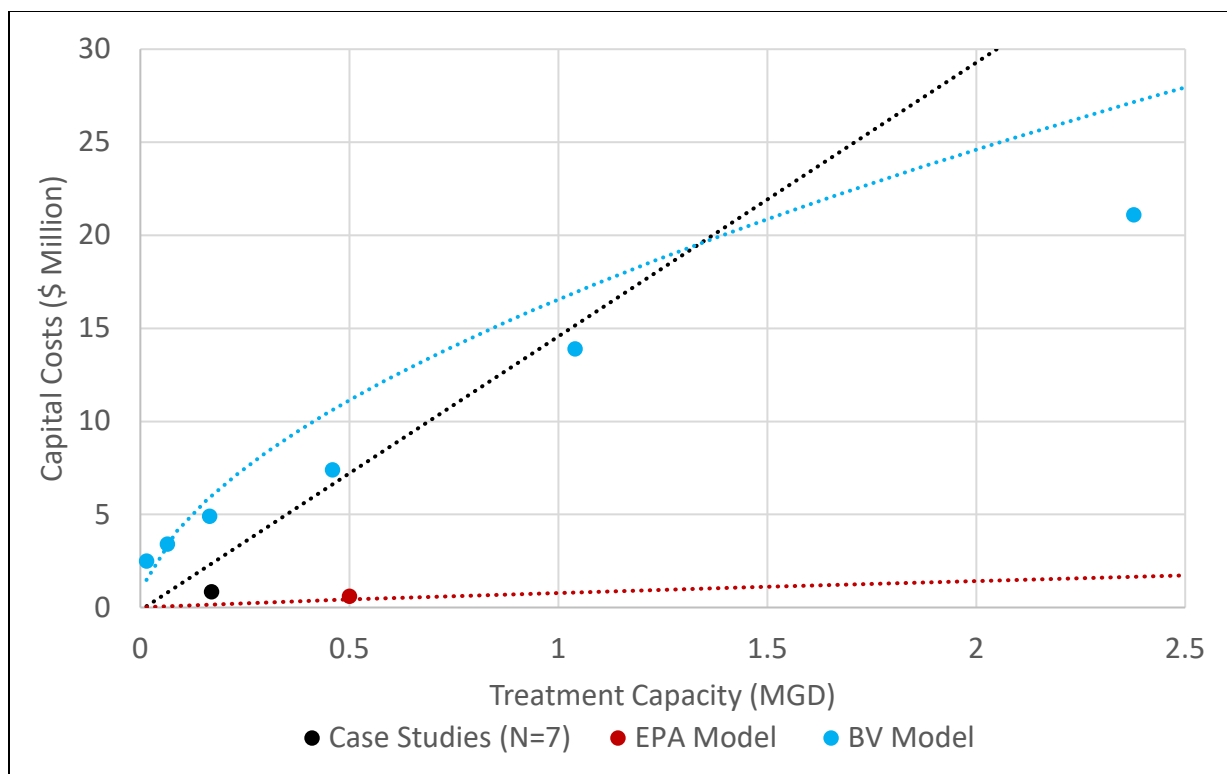


Figure A 21: Comparison of RO Capital Costs for Small Systems (<2.5 MGD)

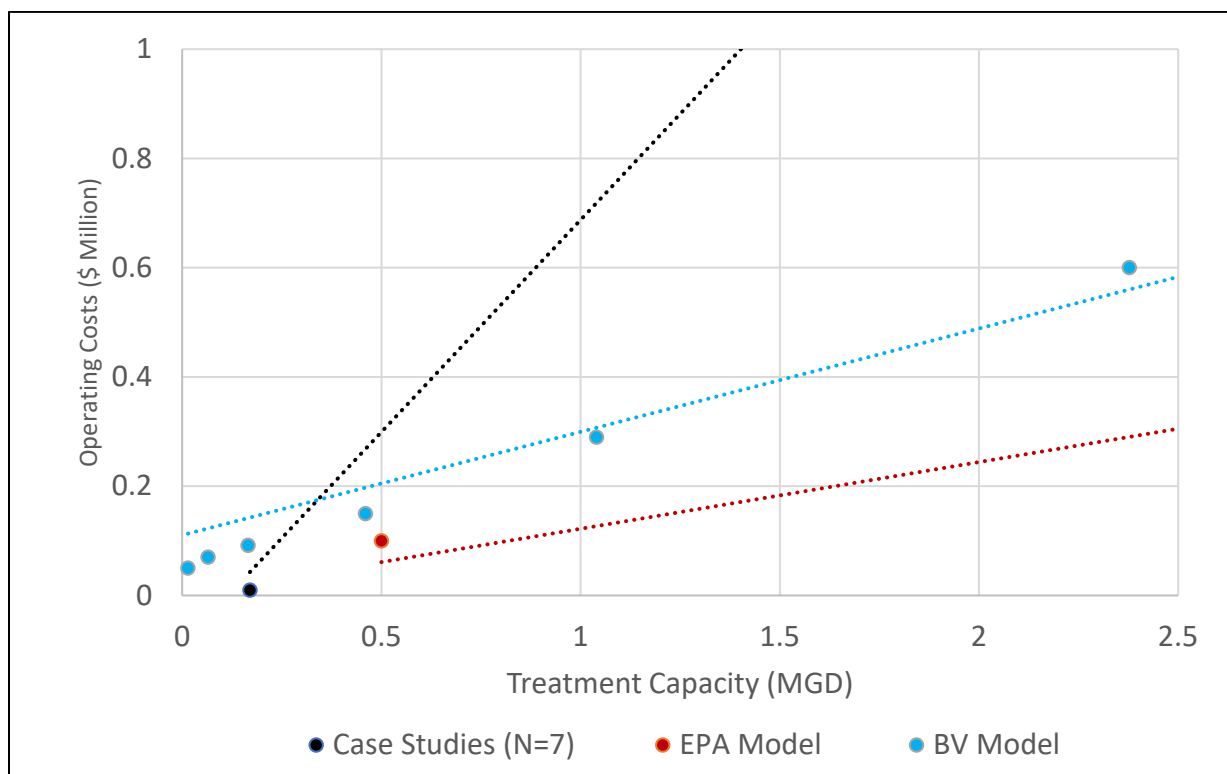


Figure A 22: Comparison of RO Operating Costs for Small Systems (< 2.5 MGD)

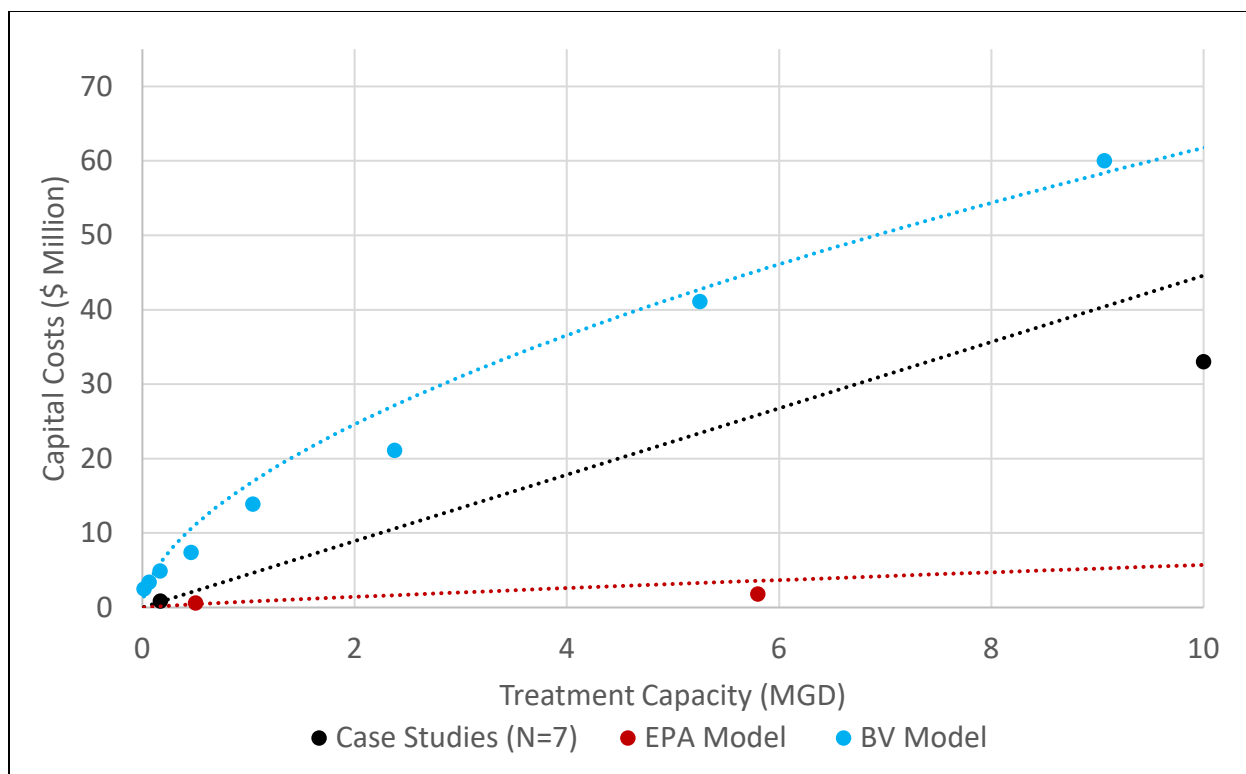


Figure A 23: Comparison of RO Capital Costs for Medium Systems (<10 MGD)

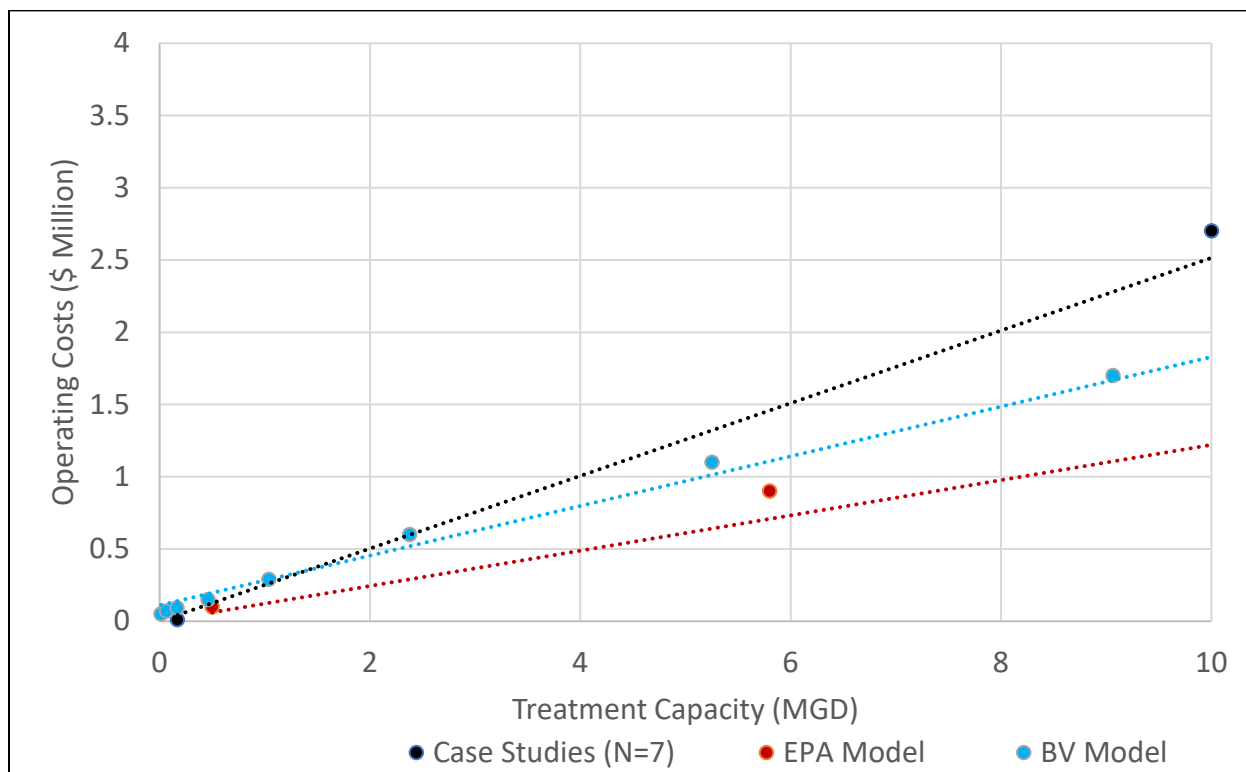


Figure A 24: Comparison of RO Operating Costs for Medium Systems (<10 MGD)

[received 3.17.2023]

Dear NDWAC:

The latest version of the NTP Report was released on Wednesday because of our fluoride lawsuit against the US EPA. How is EPA NDWAC going to proceed with the information that fluoride is neurotoxic to the brain of the developing fetus and infant? The report is linked in these articles that came out this week.

https://www.prnewswire.com/news-releases/government-report-finds-no-safe-level-of-fluoride-in-water-fluoridation-policy-threatened-301774635.html?tc=eml_cleartime

https://www.prnewswire.com/news-releases/suppressed-government-report-finding-fluoride-can-reduce-childrens-iq-made-public-under-epa-lawsuit-301773074.html?tc=eml_cleartime

Fluoride is not required by the EPA, which is prohibited by the Safe Drinking Water Act from requiring the addition of any substance to drinking water for preventive health care purposes.

Sincerely,
Brenda Staudenmaier

NDWAC Meeting Participants
August 8, 2023

National Drinking Water Advisory Council Members

Yolanda Barney
Elin Betanzo
Scott Borman
Alexandra Campbell-Ferrari
Shellie Chard
Lisa Daniels
Steve Elmore
Eagle Jones
Jana Littlewood
Jennifer Peters
Jim Proctor
Nancy Quirk
Alex Rodriguez
Jeffrey Szabo

Centers for Disease Control Liaisons

Arthur Chang

U.S. Environmental Protection Agency Meeting Participants

Elizabeth Corr, NDWAC Designated Federal Officer, Office of Ground Water and Drinking Water
Yu-Ting Guilaran, Deputy Director, Office of Ground Water and Drinking Water
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