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Economic Analysis for the Final Per- and Polyfluoroalkyl Substances National Primary Drinking Water Regulation

Economic Analysis for the Final Per- and Polyfluoroalkyl Substances National Primary Drinking Water Regulation

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U.S. Environmental Protection Agency

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Disclaimer

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Acronyms and Abbreviations

ACS	American Community Survey
AFFF	Aqueous Film Forming Foam
AIX	Anion Exchange
ANSI	American National Standards Institute
AOC	Assimilable Organic Carbon
ARIC	Atherosclerosis Risk in Communities
ATSDR	Agency for Toxic Substances and Disease Registry
AWWA	American Water Works Association
BAT	Best Available Technology
BIL	Bipartisan Infrastructure Law
BLS	Bureau of Labor Statistics
BP	Blood Pressure
BV	Bed Volumes
CARDIA	Coronary Artery Risk Development in Young Adults
CBX	SafeWater Cost Benefit Model
CCL	Contaminant Candidate List
CCR	Consumer Confidence Report
CDC	Centers for Disease Control and Prevention
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	Code of Federal Regulations
CHMS	Canadian Health Measures Survey
COI	Cost of Illness
CPI	Consumer Price Index
CVD	Cardiovascular Disease
CWSs	Community Water Systems
CWSS	Community Water System Survey
DBP	Disinfection Byproduct
DHS	Department of Homeland Security
DWSRF	Drinking Water State Revolving Fund
EA	Economic Analysis
EBCT	Empty Bed Contact Time
ECEC	Employer Cost for Employee Compensation
ECI	Employment Cost Index
ECTT	Error Code Tracking Tool
EC-SDC	Emerging Contaminants in Small or Disadvantaged Communities
EJ	Environmental Justice
EP	Entry Point
EPA	U.S. Environmental Protection Agency
FR	Federal Register
GAC	Granular Activated Carbon
GDP	Gross Domestic Product
GHG	Greenhouse Gas

gpm	Gallons per Minute
GWUDI	Ground Water Under the Direct Influence
HDLC	High-Density Lipoprotein Cholesterol
HESD	Health Effects Support Document
HFPO-DA	Hexafluoropropylene Oxide Dimer Acid
HHS	Department of Health and Human Services
HI	Hazard Index
HRRCA	Health Risk Reduction and Cost Analysis
HTN	Hypertension
HUC	Hydraulic Unit Code
ICR	Information Collection Request
ICR TSD	Information Collection Rule Treatment Study Database
IHS	Indian Health Service
IPM	Integrated Planning Model
IRA	Inflation Reduction Act
IRFA	Initial Regulatory Flexibility Analysis
IS	Ischemic Stroke
IWG	Interagency Working Group
IX	Ion Exchange
LBW	Low Birth Weight
LDLC	Low-Density Lipoprotein Cholesterol
LRAA	Locational Running Annual Average
MCBC	Multi-Contaminant Benefit-Cost Model
MCLGs	Maximum Contaminant Level Goals
MCLs	Maximum Contaminant Levels
MCMC	Markov Chain Monte Carlo
MGD	Million Gallons Per Day
MHI	Median Household Income
MI	Myocardial Infarction
MRL	Minimum Reporting Level
NCHS	National Center for Health Statistics
NCWSs	Non-Community Water Systems
NDWAC	National Drinking Water Advisory Council
NF	Nanofiltration
NHANES	National Health and Nutrition Examination Survey
NOM	Natural Organic Matter
NPDWR	National Primary Drinking Water Regulation
NSF	National Sanitation Foundation
NTNCWSs	Non-Transient Non-Community Water Systems
NTTAA	National Technology Transfer and Advancement Act
O&M	Operation and Maintenance
OEHHA	California Environmental Protection Agency's Office of Environmental Health Hazard Assessment
OES	Occupational Employment Survey
OIRA	Office of Information and Regulatory Affairs

OMB	Office of Management and Budget
ORD	Office of Research and Development
OSHA	Occupational Safety and Health Administration
PAF	Population Attributable Fraction
PBPK	Physiological-Based Pharmacokinetic
PFAA	Perfluorinated Alkyl Acids
PFAS	Per- And Polyfluoroalkyl Substances
PFBS	Perfluorobutanesulfonic Acid
PFHpA	Perfluoroheptanoic Acid
PFHxS	Perfluorohexanesulfonic Acid
PFNA	Perfluorononanoic Acid
PFOA	Perfluorooctanoic Acid
PFOS	Perfluorooctane Sulfonatic Acid
PK	Pharmacokinetic
POU	Point-of-Use
POU RO	Point-of-Use Reverse Osmosis
PRA	Paperwork Reduction Act
PWS	Public Water System
PWSID	Public Water System Identifier
PWSS	Public Water System Supervision
Q	Design Flow
RCC	Renal Cell Carcinoma
RCRA	Resource Conservation and Recovery Act
RFA	Regulatory Flexibility Act
RIA	Regulatory Impact Analysis
RO	Reverse Osmosis
RO/NF	Reverse Osmosis/Nanofiltration
RSSCT	Rapid Small-Scale Column Test
SAB	Science Advisory Board
SBA	Small Business Administration
SBAR	Small Business Advocacy Review
SBREFA	Small Business Regulatory Enforcement Fairness Act
SDWA	Safe Drinking Water Act
SDWIS	Safe Drinking Water Information System
SEER	Surveillance, Epidemiology, And End Results
SER	Small Entity Representatives
SGA	Small for Gestational Age
SISNOSE	Significant Economic Impact on a Substantial Number of Small Entities
SOC	Synthetic Organic Compounds
SSCTs	Small System Compliance Technologies
T&C	Technologies and Costs
T3	Triiodothyronine
T4	Thyroxine
TC	Total Cholesterol

TDP	Technology Design Panel
THM4	Four Regulated Trihalomethanes
TNCWSs	Transient Non-Community Water Systems
TOC	Total Organic Carbon
TRI	Toxics Release Inventory
TSH	Thyroid Stimulating Hormone
UCMR	Unregulated Contaminant Monitoring Rule
UCMR 3	Third Unregulated Contaminant Monitoring Rule
UCMR 4	Fourth Unregulated Contaminant Monitoring Rule
UMRA	Unfunded Mandates Reform Act
VOCs	Volatile Organic Compounds
VSL	Value of a Statistical Life
WBS	Work Breakdown Structure
WIFIA	Water Infrastructure Finance and Innovation
WIIN	Water Infrastructure Improvements for the Nation Act

1 Executive Summary

Under the Safe Drinking Water Act (SDWA), the U.S. Environmental Protection Agency (EPA or "the agency") has the authority to set enforceable National Primary Drinking Water Regulations (NPDWRs) for drinking water contaminants and require monitoring of public water supplies. The EPA is finalizing a NPDWR for per-and polyfluoroalkyl substances (PFAS) (EPA-HQ-OW-2022-0114). The agency initiated the process for developing a NPDWR for PFAS compounds in March 2021, when the EPA published the fourth regulatory determination for contaminants on the fourth Contaminant Candidate List (CCL), which included a final determination to regulate perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS) in drinking water. Additionally, in the EPA's final regulatory determination for PFOA and PFOS, as well as its PFAS Strategic Roadmap, the agency committed to evaluating additional PFAS beyond PFOA and PFOS and considering actions to address groups of PFAS (86 FR 12272) (U.S. EPA, 2021b; U.S. EPA, 2021e). In March of 2023, the EPA made a preliminary regulatory determination for four additional PFAS and their mixtures: perfluorononanoic acid (PFNA), hexafluoropropylene oxide dimer acid (HFPO-DA) and its ammonium salt (also known as GenX chemicals)[1], perfluorohexanesulfonic acid (PFHxS), and perfluorobutanesulfonic acid (PFBS). Additionally, the EPA proposed a NPDWR and health-based Maximum Contaminant Level Goals (MCLGs) for PFOA, PFOS and these four additional PFAS and their mixtures (88 FR 18638). The final NPDWR is one of several actions consistent with the agency's commitment to address these long-lasting "forever chemicals" that occur in drinking water supplies and impact communities across the U.S.

The final PFAS NPDWR is a significant regulatory action that was submitted to the Office of Management and Budget (OMB) for review. An economic analysis (EA) is required for all significant rules under Executive Order (EO) 12866 (Regulatory Planning and Review). In addition, Section 1412(b)(3)(C) of the 1996 Amendments to the SDWA requires the EPA to prepare a Health Risk Reduction and Cost Analysis (HRRCA) in support of any NPDWRs that include a maximum containment level (MCL). This EA addresses these and other regulatory reporting requirements, including those that direct the EPA to conduct distributional and environmental justice analysis. With respect to the SDWA HRRCA requirements, this document provides the following:

- Quantifiable 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 as the result of compliance with each level of treatment (Chapter 6);
- Quantifiable 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 from reductions in co-occurring contaminants that may be attributed solely to compliance with the final MCL, excluding benefits resulting from compliance with other proposed or promulgated regulations (Chapter 6);

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[1] The EPA notes that the chemical HFPO-DA is used in a processing aid technology developed by DuPont to make fluoropolymers without using PFOA. The chemicals associated with this process are commonly known as GenX Chemicals and the term is often used interchangeably for HFPO-DA along with its ammonium salt.

- Quantifiable and nonquantifiable costs for which there is a factual basis in the rulemaking record to conclude that such costs are likely to occur solely as a result of compliance with the final MCL, including monitoring, treatment, and other costs, and excluding costs resulting from compliance with other proposed or promulgated regulations (Chapter 2);
- Incremental costs and benefits associated with each alternative MCL considered (Chapter 7);
- Effects of the contaminant on the general population and on groups within the general population, such as sub-populations identified as likely to be at greater risk of adverse health effects due to exposure to contaminants in drinking water than the general population (Chapters 6 and 8);
- Any increased health risk that may occur as the result of compliance, including risks associated with co-occurring contaminants (Chapter 6); and
- Other relevant factors, including the quality and extent of the information, uncertainties in the analysis, and factors related to the degree and nature of the risk (Chapters 5–7).

The final NPDWR will reduce PFAS concentrations in the drinking water distributed by public water systems (PWSs) from the current baseline to drinking water concentrations that are in compliance with MCLs of 4.0 parts per trillion (ppt; also expressed as ng/L) for PFOA, 4.0 ppt for PFOS, and a unitless hazard index (HI) of 1 for the group including PFNA, HFPO-DA , PFHxS, PFBS. Additionally, the EPA is finalizing individual MCLs for HFPO-DA, PFHxS, and PFNA at 10 ppt each. See Sections III and V of the PFAS NPDWR for further discussion (U.S. EPA, 2024h). These impacts are assessed in comparison to the baseline scenario, which reflects the PFAS occurrence and exposure conditions expected in the absence of finalizing a PFAS drinking water regulation. This EA presents the incremental costs and benefits associated with the final rule (PFOA, PFOS, HI, PFHxS, PFNA, and HFPO-DA MCLs) and three regulatory alternatives that only include MCLs for PFOA and PFOS. The regulatory alternative MCLs are referred to as Option 1a (MCL of 4.0 ppt for PFOA and 4.0 ppt for PFOS), Option 1b (MCL of 5.0 ppt for PFOA and 5.0 ppt for PFOS), and Option 1c (MCL of 10.0 ppt for PFOA and 10.0 ppt for PFOS). The regulatory alternative MCLs for PFOA and PFOS (Options 1a, 1b, and 1c) do not directly regulate additional PFAS, thereby limiting public health protection and benefits relative to the final rule.

In this EA, the EPA presents the quantified and nonquantifiable health benefits expected from reductions in PFAS exposures resulting from the final rule. Quantified benefits are assessed as avoided cases of illness and deaths (or morbidity and mortality, respectively) associated with exposure to PFAS contaminants. Adverse human health outcomes associated with PFAS exposure that cannot be quantified and monetized are assessed as nonquantifiable benefits. Additionally, this EA presents the costs associated with the final NPDWR. Costs presented include those expenses incurred by PWSs to (1) monitor for PFAS, (2) inform consumers, (3) install and operate treatment technologies, and (4) perform record-keeping and reporting to comply with the PFAS NPDWR; and the costs incurred by primacy agencies (typically states) with authority to implement and enforce SDWA regulations. The EPA presents annualized quantified benefits and costs discounted at a 2 percent discount rate, consistent with OMB guidance (OMB Circular A-4, 2023).

Quantified economic benefits analyses consider the strength of evidence for associations between PFAS exposure and each adverse health effect and the availability of data to quantify the morbidity and mortality impacts associated with that adverse health effect. To identify health effects that are associated with PFAS exposure, the EPA relied on the assessment of adverse health effects associated with PFOA and PFOS exposure in the final human health toxicity assessments for PFOA and PFOS (U.S. EPA, 2024f; U.S. EPA, 2024e). The EPA provides a national-level quantitative estimate of avoided morbidity and mortality related to cardiovascular disease (CVD; both PFOA and PFOS), low birth weight (both PFOA and PFOS), and renal cell carcinoma (RCC; PFOA only) associated with reductions in PFAS consistent with the final rule. Additional quantified benefits estimates for low birth weight (PFNA) and liver cancer (PFOS) are presented in sensitivity analyses in Appendix K and Appendix O, respectively.

As required by SDWA, the EPA also provides a qualitative assessment of potential benefits for adverse health effects that are associated with PFAS exposure but lack the economic or other information needed for a quantitative analysis. In this EA, a qualitative discussion is provided for other adverse health effects and potential avoided diseases associated with PFOA, PFOS, and the four PFAS compounds included in the HI group (PFHxS, PFNA, PFBS, and HFPO-DA). The agency anticipates that the nonquantifiable human health benefits associated with reductions in drinking water PFAS exposure are substantial and may reasonably exceed the benefits the agency was able to quantify for this final rule.

As part of its HRRCA, the EPA is directed by SDWA to evaluate quantifiable 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 from reductions in co-occurring contaminants that may be attributed solely to compliance with the final MCL (SDWA 1412(b)(3)(C)(II)). These co-occurring contaminants are expected to include additional PFAS contaminants not directly regulated by the final PFAS NPDWR, co-occurring chemical contaminants such as other synthetic organic compounds (SOCs), volatile organic compounds (VOCs), and disinfection byproduct (DBP) precursors. The EPA has quantified costs associated with reduction in DBP precursors, and has considered health risk reduction benefits for other PFAS, SOCs, and VOCs qualitatively.

The agency anticipates that because of the PFAS NPDWR, some community water systems (CWSs) and non-transient non-community water systems (NTNCWSs) will need to reduce their PFAS concentrations to comply with the rule. This EA describes the costs associated with activities PWSs are expected to undertake to comply with the final rule (e.g., installation of treatment technologies to remove PFAS), and the costs associated with primacy agency implementation and administration of the final rule. National quantified cost estimates are provided for PFOA, PFOS, and PFHxS treatment. In the national cost analysis, the EPA quantified the national treatment and monitoring costs for PFHxS individual MCL exceedances and HI MCL exceedances where PFHxS is present above its HBWC while one or more other HI PFAS is also present in that same mixture. In instances where concentrations of PFNA, PFBS, and HFPO-DA are high enough to cause or contribute to a HI exceedance when the concentrations of PFOA, PFOS, and PFHxS would not have already otherwise triggered treatment, the national quantified costs may be underestimated; however, these costs are considered quantitatively in a sensitivity analysis. Additional discussion of the methodology and results of this analysis can be found in Chapter 5, Section 5.3.1.4, and Appendix N.3. See section

XII.A.4 of the final rule preamble for more information about how EPA considered HI, PFNA, and HFPO-DA MCL costs.

The EPA identified effective treatment technologies as part of the NPDWR, and consistent with SDWA requirements found in Section 1412(b)(3)(C)(II) to consider benefits likely to occur from reductions in co-occurring compounds, the EPA estimated expected benefits from reductions in co-occurring compounds as a result of PFAS treatment. Moreover, the EPA developed a quantitative analysis for reductions in bladder cancer morbidity and mortality that stem from removal of DBP precursors. DBPs, specifically trihalomethanes, are formed when disinfectants interact with organic material in drinking water distribution systems. Since PFAS treatment has been demonstrated to remove DBP precursors, the agency anticipates that DBPs, including trihalomethanes, will be reduced with PFAS treatment. The EPA provides a qualitative discussion of benefits for other potential water quality improvements that stem from PFAS treatment, including those benefits associated with reductions in other co-occurring contaminants besides DBPs.

The tables below present quantified benefits and costs of the final NPDWR (“final rule”) and alternative MCLs considered. Compared to the economic analysis for the proposed PFAS NPDWR, which presented costs in 2021 dollars, the EPA presents costs for the final rule in 2022 dollars. Table ES-1 presents the total estimated national annualized benefits associated with the final rule and regulatory alternatives considered. Table ES-2 presents the total estimated national annualized costs associated with the final rule and regulatory alternatives considered. Quantitative estimates are presented using a 2 percent discount rate. Throughout this EA, benefits and costs are presented using mean (or “expected value”), 5th, and 95th percentile results to characterize key sources of uncertainty, including but not limited to PFAS baseline occurrence and health effect slope factor uncertainty, which is consistent with OMB and EPA guidance (OMB Circular A-4, 2003; U.S. EPA, 2010a). All significant limitations and uncertainties of this economic analysis are described in the pages that follow.

Table ES-1: Quantified Total National Annualized Benefits, All Options (Million $2022)

Option	2% Discount Rate[a]		
	5th Percentile[b]	Expected Value	95th Percentile[b]
Final rule[c]	$920.91	$1,549.40	$2,293.80
Option 1a[d]	$913.05	$1,542.74	$2,280.10
Option 1b[e]	$768.55	$1,296.84	$1,919.30
Option 1c[f]	$397.28	$664.45	$970.70

Notes: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates. Quantified total national annualized benefits do not include quantified sensitivity analysis results for PFNA effects on birth weight and PFOS effects on liver cancer, and as such, the quantified total national annualized benefits may be underestimated. See appendices K and O for PFNA birth weight and PFOS liver cancer sensitivity analysis results, respectively.

[a] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

[b] The 5th and 95th percentile range is based on modeled variability and uncertainty described in Section 6.1.2 and Table 6-1 for benefits. This range does not include the uncertainty described in Table 6-48 for benefits.

[c] The final rule sets PFOA and PFOS MCLs of 4.0 ppt each, an HI of 1, and MCLs for HFPO-DA, PFNA, and PFHxS of 10 ppt each.

[d] Option 1a sets PFOA and PFOS MCLs only, at 4.0 ppt each.

[e] Option 1b sets PFOA and PFOS MCLs only, at 5.0 ppt each.

[f] Option 1c sets PFOA and PFOS MCLs only, at 10.0 ppt each.

Table ES-2: Quantified Total National Annualized Costs, All Options (Million $2022)

Option	2% Discount Rate[a,b]		
	5th Percentile[c]	Mean	95th Percentile[c]
Final rule[d,e]	$1,435.70	$1,548.64	$1,672.10
Option 1a[f]	$1,423.60	$1,537.07	$1,660.30
Option 1b[g]	$1,102.60	$1,192.13	$1,291.40
Option 1c[h]	$462.87	$499.29	$540.68

Notes: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates.

[a] See Table 7-6 for a list of the nonquantifiable costs, and the potential direction of impact these costs would have on the estimated monetized total annualized costs in this table.

[b] PFAS-contaminated wastes are not considered regulatory under the Resource Conservation and Recovery Act (RCRA) or characteristic hazardous wastes at this time and therefore total costs reported in this table do not include costs associated with hazardous waste disposal of spent filtration materials. To address stakeholder concerns about potential costs for disposing PFAS-contaminated wastes as hazardous should they be regulated as such in the future, the EPA conducted a sensitivity analysis with an assumption of hazardous waste disposal for illustrative purposes only. See Appendix N, Section N.2 for additional detail.

[c] The 5th and 95th percentile range is based on modeled variability and uncertainty described in Section 5.1.2 and Table 5-1 for costs. This range does not include the uncertainty described in Table 5-21 for costs.

[d] Quantified national costs do not include quantified sensitivity analysis results for PFNA, PFBS, and HFPO-DA. Including the costs of treating for these compounds increases total annualized cost of the final rule to $1,631.05 million. These benefits and costs are considered quantitatively in the sensitivity analysis. See Appendix N.3 for more information.

[e] The final rule sets PFOA and PFOS MCLs of 4.0 ppt each, an HI of 1 and MCLs for HFPO-DA, PFNA, and PFHxS of 10 ppt each.

[f] Option 1a sets PFOA and PFOS MCLs of 4.0 ppt each.

[g] Option 1b sets PFOA and PFOS MCLs of 5.0 ppt each.

[h] Option 1c sets PFOA and PFOS MCLs of 10.0 ppt each.

2 Introduction

PFAS are a class of synthetic chemicals that have been manufactured and in use since the 1940s (AAAS, 2020; U.S. EPA, 2022h). PFAS are or were most commonly used to make products resistant to water, heat, and stains and are consequently found in industrial and consumer products like clothing, food packaging, cookware, cosmetics, carpeting, and fire-fighting foam (AAAS, 2020). PFAS manufacturing and processing facilities, facilities using PFAS in the production of other products, airports, and military installations have been associated with PFAS releases into the air, soil, and water (U.S. EPA, 2016b; U.S. EPA, 2016c). People may be exposed to PFAS by using certain consumer products, through occupational exposure, and/or through consuming contaminated food or contaminated drinking water (Domingo & Nadal, 2019; Fromme et al., 2009).

PFOS and PFOA are part of a subset of PFAS referred to as perfluorinated alkyl acids (PFAA) and are two of the most widely studied and longest-used PFAS. Due to their widespread use and persistence in the environment, most people have been exposed to PFAS, including PFOA and PFOS (U.S. EPA, 2016b; U.S. EPA, 2016c). PFOA and PFOS have been detected in up to 98 percent of blood serum samples taken in biomonitoring studies that are representative of the U.S. general population (CDC, 2019). Following the voluntary phase-out of PFOA by eight major chemical manufacturers and processors in the U.S. under the EPA's 2010/2015 PFOA Stewardship Program and reduced manufacturing of PFOS (last reported in 2002 under Chemical Data Reporting), serum concentrations have been declining. The National Health and Nutrition Examination Survey (NHANES) data exhibited that 95th-percentile serum PFOS concentrations have decreased over 75 percent, from 75.7 µg/L in the 1999-2000 cycle to 18.3 µg/L in the 2015-2016 cycle (CDC, 2019; Jain, 2018; Calafat et al., 2007; Calafat et al., 2019).

Despite voluntary phase-outs and reduced exposure to some PFAS chemicals, PFAS are still used in a wide range of consumer products and industrial applications. The EPA’s analysis of drinking water monitoring data shows widespread occurrence of PFAS compounds in multiple geographic locations. Most known exposures are relatively low, but some can be high, particularly when people are exposed to a concentrated source over long periods of time. Studies indicate that PFAS exposure above certain levels may result in adverse health effects, including developmental effects to fetuses during pregnancy or to breast-fed infants, cancer, and other immunologic-related effects.

Under SDWA, the EPA is regulating PFAS in drinking water distributed by all CWSs[2] and NTNCWSs. In 2021, the EPA determined that a NPDWR for PFAS would result in a meaningful opportunity to reduce health risks (U.S. EPA, 2021b). In March of 2023, the EPA proposed a NPDWR with health-based MCLGs and enforceable MCLs for PFOA, PFOS and four PFAS and their mixtures. Section 2.1 provides further detail on the final NPDWR for PFAS.

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[2] Systems that supply water to the same population year-round.

2.1 Summary of the Final PFAS Rule and Regulatory Alternatives

The EPA is regulating six PFAS in finished drinking water: (1) PFOS, (2) PFOA, (3) PFNA, (4) HFPO-DA and its ammonium salt (also known as GenX chemicals), (5) PFHxS, and (6) PFBS. The final regulation utilizes compound-specific MCLs for PFOA, PFOS, PFNA, HFPO-DA, and PFHxS and a group MCL based on a HI for PFNA, HFPO-DA, PFHxS, and PFBS. This regulatory approach utilizes the mixtures framework peer reviewed by the EPA's Science Advisory Board (SAB; U.S. EPA, 2022i) and builds a framework for inclusion of additional PFAS through future rulemaking as new data become available (U.S. EPA, 2024d). For more information on the HI approach, see the EPA's Framework for Estimating Noncancer Health Risks Associated with Mixtures of PFAS (U.S. EPA, 2024d).

Based on the best available scientific information on the health effects, the EPA is finalizing MCLGs of 0 ppt for PFOA and PFOS each, an MCLG of 1 for the HI, and MCLGs of 10 ppt for HFPO-DA, PFHxS, and PFNA each. The EPA has determined that it is feasible to set enforceable MCLs for PFOA and PFOS at 4.0 ppt each and MCLs for HFPO-DA, PFHxS, and PFNA at 10 ppt each. Additionally, the EPA has determined it is feasible to set an MCL for four PFAS with a HI limit of 1. As such, the EPA is finalizing enforceable MCLs of 4.0 ppt for PFOA, 4.0 ppt for PFOS, 10 ppt for HFPO-DA, 10 ppt for PFHxS, and 10 ppt for PFNA and a unitless HI of 1 for the group including PFNA, HFPO-DA, PFHxS, and PFBS. For additional details about the MCLGs and MCLs in the final rule, see the Federal Register Notice for this rulemaking.

Additionally, in this EA, the EPA presents benefits and costs for the final rule as well as three regulatory alternatives. For the proposed rule, the agency received comments on whether establishing traditional MCLGs and MCLs for PFHxS, HFPO-DA, PFNA, and PFBS instead of or in addition to the HI approach would change public health protection, improve clarity for the rule, or change costs. See Section V of the Federal Register Notice for further discussion of why the EPA added individual MCLs for HFPO-DA, PFHxS, and PFNA. For the final rule, the EPA has also included estimates of the marginal costs for the individual PFHxS, PFNA, and HFPO-DA MCLs in the absence of the HI (See Section 5.1.3 and Appendix N.4 for details). This analysis confirms that the treatment burden from the individual MCLs is fully considered in the HI cost estimates in Appendix N.3 (and as discussed above, the individual PFHxS, PFNA, and HFPO-DA MCL marginal costs are lower in the absence of the HI MCL).

The regulatory alternatives that the EPA evaluated present individual MCLG and enforceable MCL values for PFOA and PFOS. MCL values for PFOA and PFOS vary for each alternative considered: 4.0 ppt in Option 1a, 5.0 ppt in Option 1b, and 10.0 ppt in Option 1c. The EPA evaluated benefits and costs for Option 1a to determine the difference in costs between alternatives for PFOA and PFOS MCLs only versus MCLs for PFOA and PFOS and an HI for four additional PFAS. The EPA considered benefits and costs under Option 1b—MCLs of 5.0 ppt for PFOA and PFOS—because it is 25 percent above the compliance quantitation limit of 4.0 ppt established for the final rule. Lastly, the EPA considered benefits and costs of Option 1c—MCLs of 10.0 ppt for PFOA and PFOS—to provide information on whether the agency should consider utilizing its authority under Section 1412(b)(6) to set an alternative MCL at the level at which the benefits would justify the costs.

2.2 Economic Analysis Assumptions

2.2.1 Compliance Schedule and Period of Analysis for Final Rule

For purposes of this EA, the EPA assumes that the NPDWR will be promulgated in 2024. As the final rule will grant a 2-year nationwide extension of the date for MCL compliance, this analysis assumes that capital improvements (i.e., installation of treatment technologies) for systems taking action under the rule take effect five years after the date on which the regulation is promulgated, or in 2029. All other requirements, including initial monitoring, are assumed to be completed within three years of rule promulgation. In addition to this initial time window, the EPA's period of analysis includes the 80 years following the assumed compliance date.[3] This time span is based on an assumed median human lifespan of 80 years. In this EA, the EPA evaluates costs and benefits under the final rule for the period of analysis from 2024 through 2105. The EPA selected this period of analysis to estimate human health risk reduction to capture health effects from chronic illnesses that are typically experienced later in life (i.e., cardiovascular disease and cancer). Capital costs for installation of treatment technologies are spread over the useful life of the technologies. The EPA does not capture effects of compliance with the final rule beyond the year 2105.

2.2.2 Dollar Year and Discount Rates

The EPA presents estimated costs and benefits under the final rule in 2022 U.S. dollars. Appendix J provides additional details on the price indices used for inflation adjustments.

The final rule analysis estimates the annualized value of future benefits and costs using a 2 percent discount rate. The U.S. White House and OMB recently finalized and re-issued the A-4 and A-94 benefit-cost analysis guidance (see OMB Circular A-4, 2023), and the update includes new guidance to use a social discount rate of 2 percent. The updated OMB Circular A-4 states that the discount rate should equal the real (inflation-adjusted) rate of return on long-term U.S. government debt, which provides an approximation of the social rate of time preference. This rate for the past 30 years has averaged around 2.0 percent per year in real terms on a pre-tax basis. OMB arrived at the 2 percent discount rate figure by considering the 30-year average of the yield on 10-year Treasury marketable securities, and the approach taken by OMB produces a real rate of 1.7 percent per year, to which OMB added a 0.3 percent per-year rate to reflect inflation as measured by the personal consumption expenditure (PCE) inflation index. The OMB guidance states that Agencies must begin using the 2 percent discount rate for draft final rules that are formally submitted to the Office of Information and Regulatory Affairs (OIRA) after December 31, 2024. The updated OMB Circular A-4 guidance further states that "to the extent feasible and appropriate, as determined in consultation with OMB, agencies should follow this Circular's guidance earlier than these effective dates." Given the updated default social discount rate prescribed in the OMB Circular A-4 and also public input received on the discount rates considered by the EPA in the proposed NPDWR for this final rule (see response to comment

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[3] When calculating the present value of costs over the 82-year period of analysis, the EPA uses the useful life of the technology to determine when the capital components will need to be replaced. So, for example, if a PWS installs a technology in year 7 of the analysis that has an average useful life of 18 years, and costs $1M, the PWS accrues capital costs of $1M in each of the following years: 7, 25, 43, 61, and 79. It also accrues O&M costs every year of the analysis beginning in year 7.

document Section 13.2), the EPA estimated national benefits and costs at the 2 percent discount rate for the final rule and incorporated those results into the final economic analysis. Since the EPA proposed this NPDWR with the 3 and 7 percent discount rates based on guidance in the previous version of OMB Circular A-4, the EPA has kept the presentation of results using these discount rates in Appendix P. The Administrator reaffirms his determination that the benefits of the rule justify the costs. The EPA's determination is based on its analysis under in SDWA section 1412(b)(3)(C) of the quantifiable benefits and costs at the 2 percent discount rate, in addition to at the 3 and 7 percent discount rate, as well as the nonquantifiable benefits and costs. The EPA found that significant nonquantifiable benefits are likely to occur from the final PFAS NPDWR.

The same discount rate is used for both benefits and costs. All future cost and benefit values are discounted back to the initial year of the analysis, 2024, providing the present value of the cost or benefit.

2.2.3 Annualization

Consistent with the timing of the final rule and associated reductions in PFAS levels, the EPA uses the following equation to annualize the future costs and benefits:

Equation 1:

$$AV = \frac{r(PV)}{(1+r)[1-(1+r)^{-n}]}$$

Where AV is the annualized value, PV is the present value,[4] r is the discount rate (2%), and n is the number of years (82 years).

2.2.4 Population

To determine the number of people expected to benefit from actions under the final rule, the EPA uses population data from the Safe Drinking Water Information System Federal version (SDWIS/Fed) 2021 Quarter 4 (Q4) database (U.S. EPA, 2021h). The SDWIS/Fed data provide the population served by each PWS in the U.S. For analyses that rely on age-, sex-, and race/ethnicity-specific populations, the EPA uses county-level population proportions based on 2021 estimates from the U.S. Census Bureau (2020a). The EPA does not consider population growth during the period of analysis (2024–2105). For more information on the SDWIS/Fed and U.S. Census Bureau (2020a) data, see Appendix B.

2.2.5 Valuation

To estimate the economic value of avoided premature deaths, the EPA uses Value of Statistical Life estimates. The EPA follows *Guidelines for Preparing Economic Analyses* (U.S. EPA, 2010a) and approximates Value of Statistical Life growth using a compound annual growth rate of projected Value of Statistical Life values to obtain a Value of Statistical Life suitable for

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[4] The present value is the current value of a future sum of benefits given a specified discount rate. The present value represents the expected value of benefits determined at the date of valuation.

valuation of mortality risk reductions during the period of analysis, 2024-2105. As the base value, the EPA used the Value of Statistical Life estimate of $4.8 million ($1990, 1990 income year), which is the central tendency of the Value of Statistical Life distribution recommended for use in the EPA's regulatory impact analyses (U.S. EPA, 2010a). The base Value of Statistical Life estimate is adjusted for inflation and income growth as described in Appendix J. The Value of Statistical Life estimates employed in the EPA's analysis range from $11.6 million ($2022) in 2024 to $19.1 million ($2022) in 2105.[5]

To estimate the economic value of avoided morbidity (i.e., non-fatal heart attacks and ischemic strokes, birth weight decrements, and cancers), the EPA used the cost of illness (COI) valuation approach. The COI-based values used in this analysis reflect medical care expenditures and opportunity costs associated with managing/treating the condition. The health endpoint-specific morbidity valuation details are provided in Sections 6.4.4, 6.5.4, 6.6.4, and 6.7.2.5. The EPA received public comments on the proposed rule that recommended the EPA incorporate willingness to pay metrics in addition to COI in its final estimates of non-fatal health effects associated with reduced PFAS exposure. To address these comments, the EPA developed a sensitivity analysis in Appendix O to illustrate the impact to benefits results when using available willingness to pay information to monetize cancer morbidity.

2.3 Document Organization

The remainder of this EA is organized into the following chapters:

- Chapter 2: Introduction summarizes the final PFAS rule and regulatory alternatives, including the economic assumptions made in developing the rule.
- Chapter 3: Need for the Rule summarizes the statutory requirements, regulatory actions, and national EPA initiatives affecting PFAS in drinking water. It also explains the contributors to the PFAS rule, statutory authority, and the economic rationale for the regulatory approach.
- Chapter 4: Baseline Drinking Water System Conditions describes the systems subject to the final PFAS rule, PFAS water concentration levels, and data sources used to characterize the baseline before the EPA models estimated changes that result from complying with the final PFAS requirements.
- Chapter 5: Estimating Public Water System Costs provides a description of the estimated costs for the final regulatory changes affecting systems and Primary Agencies.
- Chapter 6: Benefits Analysis provides an estimate of the potential health benefits of the final PFAS rule and regulatory alternatives relative to the baseline, including quantification and monetization where possible.

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[5] Income growth projections from the U.S. Energy Information Administration (2021) are available through 2050. The EPA uses these projections to calculate annual VSL values in $2022 from years 2024 to 2050 as described in Equation J-1 in Appendix J. The EPA uses these calculated VSL values to estimate a compound annual growth rate (see Equation J-2 in Appendix J) and applies this growth rate to estimate annual VSL values beyond year 2050 (see Equation J-3 in Appendix J).

- Chapter 7: Comparison of Costs to Benefits provides a summary of costs and benefits associated with the provisions of the final PFAS rule.
- Chapter 8: Environmental Justice Analysis provides a description of how the final PFAS rule addresses Executive Order 12898: Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations.
- Chapter 9: Statutory and Administrative Requirements discusses analyses performed to evaluate the effects of the final PFAS rule and regulatory alternatives on different segments of the population in accordance with 12 federal mandates and statutory reviews, including but not limited to the Final Regulatory Flexibility Analysis/Small Business Regulatory Enforcement Fairness Act (RFA/SBREFA), Unfunded Mandates Reform Act (UMRA), and Executive Order 14008: Tackling the Climate Crisis at Home and Abroad.
- Chapter 10: References includes a list of references cited throughout the final PFAS rule economic analysis.

2.4 Supporting Documentation

This EA involves numerous detailed and complex analyses, and the following appendices are provided to help the reader understand how those analyses were conducted and their underlying data and assumptions:

- Appendix A: Framework of Bayesian Hierarchical Markov Chain Monte Carlo Occurrence Model
- Appendix B: Affected Population
- Appendix C: Cost Analysis Results
- Appendix D: PFOA and PFOS Serum Concentration-Birth Weight Relationship
- Appendix E: Effects of Reduced Birth Weight on Infant Mortality
- Appendix F: Serum Cholesterol Dose-Response Functions
- Appendix G: CVD Benefits Model Details and Input Data
- Appendix H: Cancer Benefits Model Details and Input Data
- Appendix I: Trihalomethane Co-Removal Model Details and Analysis
- Appendix J: Value of a Statistical Life Updating
- Appendix K: Benefits Sensitivity Analyses
- Appendix L: Uncertainty Characterization Details and Input Data
- Appendix M: Environmental Justice
- Appendix N: Supplemental Cost Analyses
- Appendix O: Supplemental Benefits Analyses
- Appendix P: Additional Model Outputs

- Appendix Q: Appendix References

3 Need for the Rule

This section provides the statutory and economic rationales for choosing a regulatory approach to address the public health consequences of PFAS contamination in drinking water. The EPA's statutory requirements, regulatory actions, and agency initiatives impacting PFAS in drinking water are discussed.

3.1 Previous EPA Nonregulatory and Regulatory Actions Potentially Affecting PFAS Drinking Water Management

This section provides a summary of actions and initiatives affecting PFAS in drinking water prior to the publication of the final NPDWR for PFAS. Additionally, states have begun proposing and promulgating their own regulatory and non-regulatory standards for PFAS in drinking water. For more information on these state actions, see the Environmental Council of the States' Processes & Considerations for Setting State PFAS Standards (ECOS, 2022).

3.1.1 PFAS Council and PFAS Strategic Roadmap

EPA Administrator Michael Regan established the EPA Council on PFAS in April 2021 and charged it to develop a bold, strategic, whole-of-EPA strategy to protect public health and the environment from the impacts of PFAS. The Council comprises senior technical and policy leaders from across EPA program offices and regions and is chaired by Assistant Administrator for Water Radhika Fox and Acting Region 1 Administrator Deb Szaro (U.S. EPA, 2021e).

On October 18, 2021, Administrator Regan announced the agency's PFAS Strategic Roadmap, developed by the PFAS Council to lay out the EPA's whole-of-agency approach to tackling PFAS. The PFAS Strategic Roadmap sets timelines by which the EPA plans to take specific actions and commits to bolder new policies to safeguard public health, protect the environment, and hold polluters accountable. Described in the Roadmap are key commitments the agency made toward addressing these contaminants in the environment. With this final rule, the EPA is delivering on a key commitment in the Roadmap to "establish a National Primary Drinking Water Regulation" (U.S. EPA, 2021e).

3.1.2 Final Regulatory Determinations on the Fourth Drinking Water Contaminant Candidate List

Section 1412(b)(1)(B)(i) of SDWA requires the EPA to publish the CCL every five years after public notice and an opportunity to comment. The CCL is a list of contaminants which are not subject to any final or promulgated NPDWRs but are known or anticipated to occur in PWSs and may require regulation under SDWA. SDWA Section 1412(b)(1)(B)(ii) directs the EPA to determine, after public notice and an opportunity to comment, whether to regulate at least five contaminants from the CCL every five years.

Under Section 1412(b)(1)(A) of SDWA, the EPA will regulate a contaminant in drinking water if the EPA Administrator determines that:

a) The contaminant may have an adverse effect on the health of persons;

b) The contaminant is known to occur or there is a substantial likelihood that the contaminant will occur in PWSs with a frequency and at levels of public health concern; and

c) In the sole judgment of the Administrator, regulation of such contaminant presents a meaningful opportunity for health risk reduction for persons served by PWSs.

If after considering public comment on a preliminary determination, the EPA decides to regulate a contaminant, the EPA will initiate the process to propose and promulgate a NPDWR. In that case, the statutory time frame provides for agency proposal of a regulation within 24 months and action on a final regulation within 18 months of proposal.

On March 10, 2020, the EPA published preliminary positive regulatory determinations for PFOS and PFOA (85 FR 14098) (U.S. EPA, 2020a). On March 3, 2021, the EPA published final regulatory determinations for PFOS and PFOA (86 FR 12272) (U.S. EPA, 2021b). In doing so, the EPA also committed to evaluating a broader range of PFAS, including new monitoring and occurrence data, and other information being developed by the EPA, other federal agencies, state governments, international organizations, industry groups, and other stakeholders (U.S. EPA, 2021b).

3.1.3 Proposed PFAS National Primary Drinking Water Rule and Regulatory Determinations for PFHxS, PFNA, HFPO-DA, PFBS, and their Mixtures.

On March 14th, 2023, the EPA announced the PFAS NPDWR and requested comments on all aspects of the proposed rule. This action included determinations to regulate PFHxS, HFPO-DA and its ammonium salt (also known as a GenX chemicals), PFNA, and PFBS, and mixtures of these PFAS as contaminants. A summary of major public comments and agency responses to those comments are presented in the preamble for the final rule (U.S. EPA, 2024h). The agency's detailed response to the comments received are presented in the document "Response to Comments on the EPA's Proposed PFAS NPDWR" which is available in the public docket for this rule. The EPA received approximately 122,000 comments on these regulatory determinations and proposed NPDWR and considered commenter input in finalizing the rule and this economic analysis.

3.1.4 Unregulated Contaminant Monitoring Rule

As part of its responsibilities under the SDWA, the EPA implements Section 1445(a)(2), Monitoring Program for Unregulated Contaminants. This section requires that once every five years, the EPA issues a list of no more than 30 unregulated contaminants to be monitored by PWSs. This monitoring is implemented through the Unregulated Contaminant Monitoring Rule (UCMR), which collects data from community water systems and NTNCWS. For each UCMR cycle, the EPA establishes a new list of contaminants for monitoring, specifies which systems are required to monitor, identifies the sampling locations, and defines the analytical methods to be used.

The third Unregulated Contaminant Monitoring Rule (UCMR 3) was published on May 2, 2012. UCMR 3 required monitoring for six PFAS: PFOA, PFOS, PFNA, PFHxS, PFBS, and perfluoroheptanoic acid (PFHpA). UCMR 3 data were used in the development of this economic analysis. See Sections 4.2.2 and 4.4 for further discussion of these data.

On December 17, 2021, the EPA Administrator Michael Regan signed the final Revisions to the Unregulated Contaminant Monitoring Rule (UCMR 5) for Public Water Systems, and the rule was subsequently published in the Federal Register on December 27, 2021 (86 FR 73131). The five-year UCMR 5 cycle spans from 2022 to 2026, with preparations in 2022, sample collection from 2023 to 2025, and completion of data reporting in 2026. UCMR 5 includes all 29 PFAS that are within the scope of EPA Methods 533 and 537.1 (U.S. EPA, 2021b). Initial sampling results for UCMR 5 are available at: https://www.epa.gov/dwucmr/occurrence-data-unregulated-contaminant-monitoring-rule#5 and discussed in *PFAS Occurrence & Contaminant Background Support Document* (U.S. EPA, 2024g). In addition to information on occurrence data from previous rounds of UCMR sampling, when completed, permanent results for UCMR 5 will be available at: https://www.epa.gov/dwucmr/occurrence-data-unregulated-contaminant-monitoring-rule.

3.2 Statutory Authority for Promulgating the Rule

Section 1412(b)(1)(A) of SDWA authorizes the EPA to establish NPDWRs for contaminants that may have an adverse public health effect, that are known to occur or that present a substantial likelihood of occurring in PWSs at a frequency and level of public health concern, and that present a meaningful opportunity for health risk reduction for persons served by PWSs.

Section 1445(a) of SDWA authorizes the EPA Administrator to establish monitoring, recordkeeping, and reporting regulations that the Administrator can use to establish regulations under the SDWA, determine compliance with SDWA, and advise the public of the risks of unregulated contaminants (42 U.S.C. § 300j-4(a)). In requiring a PWS to monitor under Section 1445(a), the Administrator may take into consideration the water system size and the contaminants likely to be found in the system's drinking water (42 U.S.C. § 300j-4(a)). Section 1445(a)(1)(C) of the SDWA provides that "every person who is subject to a national primary drinking water regulation" under Section 1412 must provide such information as the Administrator may reasonably require to assist the Administrator in establishing regulations under Section 1412 (42 U.S.C § 300j-4(a)(1)(C)).

Section 1413(a)(1) of the SDWA allows the EPA to grant a state primary enforcement responsibility ("primacy") for NPDWRs when the EPA has determined that the state has, among other things, adopted regulations that are no less stringent than the EPA's (42 U.S.C. § 300g-2(a)(1)). To obtain primacy for this rule, states must adopt comparable regulations within two years of the EPA's promulgation of the final rule, unless the EPA grants the state a two-year extension (40 CFR 142.12(b)). State primacy requires, among other things, adequate enforcement (including monitoring and inspections) and reporting. The EPA must approve or deny state primacy applications within 90 days of submission to the EPA (42 U.S.C. § 300g-2(b)(2)). In some cases, a state submitting revisions to adopt a NPDWR has interim primary enforcement authority for the new regulation while the EPA's decision on the revision is pending (42 U.S.C. § 300g-2(c)).

Section 1450 of the SDWA authorizes the Administrator to prescribe such regulations as are necessary or appropriate to carry out his or her functions under the Act (42 U.S.C § 300j-9).

3.3 Economic Rationale

Section 1(b) of Executive Order 12866, "The Principles of Regulation," provides that each agency, as applicable and permitted by law: "shall identify the problem that it intends to address (including, where applicable, the failures of private markets or public institutions that warrant new agency action) as well as assess the significance of that problem." This section describes the types of market failures that NPDWRs address.

In a perfectly competitive market, market forces guide buyers and sellers to attain the most efficient social outcome. A perfectly competitive market occurs when both buyers and sellers are price takers, usually when there are many producers and buyers of a product and both producers and buyers have complete knowledge about that product. Also, there must not be any barriers to entry into the industry, and existing producers in the industry must not have any advantage over potential new producers. Several factors in the public water supply industry preclude it from being a perfectly competitive market and lead to market failures that may require regulation.

First, it is not economically efficient to have multiple suppliers who would, for example, compete by building multiple systems of pipelines, reservoirs, wells, and other facilities. Instead, economic efficiency leads to a single firm or government entity performing these functions generally under public control. Under these monopoly conditions, consumers are provided only one level of service with respect to drinking water quality. If consumers do not believe that the quality of tap water is adequate, they cannot simply switch to another water utility. Consumers may purchase bottled water, but this option can be much more expensive due to the inefficiencies of bottling and transporting bottled water. Consumers may also install and operate home treatment systems, but this can also be considerably more expensive without the economies of scale of large, centralized water systems. Additionally, home treatment systems potentially can lead to increased health risks when not regularly maintained by the consumer.

Second, high information and transaction costs impede the public's understanding of health and safety issues concerning drinking water quality. The health risks potentially posed by trace quantities of drinking water contaminants requires the EPA to analyze and distill complex toxicological and health sciences data. The EPA promulgated the Consumer Confidence Report (CCR) rule to make water quality information more easily available to consumers. The CCR rule requires CWSs to mail their customers an annual report on local drinking water quality.

The report provides customers with information on levels of detected contaminants in their drinking water, limited health risk information associated with contaminant exposure when levels exceed MCLs, and utility contact information. Even if informed consumers can engage utilities regarding these health issues, the costs of such engagement, known as "transaction costs" (in this case measured in personal time and commitment), can be a barrier to efficient market outcomes.

SDWA regulations are intended to provide a level of protection from exposure to drinking water contaminants that would not otherwise occur in the existing market environment of public water supply. The regulations set minimum performance requirements for all public water supplies to reduce the risk confronted by all consumers from exposure to drinking water contaminants. SDWA regulations are not intended to restructure market mechanisms or establish competition in supply; rather, SDWA standards establish the level of service needed to better reflect the public's preference for safety. Federal regulations remove the high information and transaction costs by acting on behalf of all consumers in balancing the risk reduction and social costs of achieving this reduction.

4 Baseline Drinking Water System Conditions

4.1 Introduction

In its *Guidelines for Preparing Economic Analyses*, the EPA characterizes the baseline as a reference point that reflects the world without the regulation (U.S. EPA, 2010a); this baseline is the starting point for estimating the potential benefits and costs of the final PFAS NPDWR.

This chapter presents a characterization of PWSs and their current operations (i.e., the baseline) before changes are made to meet the final PFAS NPDWR. Section 4.2 identifies each major data source used to develop the baseline. Section 4.3 explains the derivation of each baseline characteristic and presents results in detailed tables. Section 4.4 describes the Bayesian model developed to estimate national PFAS occurrence in drinking water supplies. Section 4.5 summarizes limitations of the major data sources and uncertainties in the baseline characterization (both quantified and nonquantifiable) in table format.

4.2 Data Sources

The EPA used a variety of data sources to develop the baseline. Section 4.2.1 explains the relevant information provided in the federal version of the SDWIS/Fed and measures the EPA took to verify the data. Section 4.2.2 describes the purpose of UCMR 3 data. Section 4.2.3 describes the independent state sampling program data. Sections 4.2.4 and 4.2.5 describe two data sources used to develop key characteristics of system treatment plants. Section 4.2.6 explains the purpose of the 2006 Community Water System Survey (CWSS) and the representativeness of the data. Table 4-1 identifies each major data source and the baseline data element(s) derived from them.

Table 4-1: Data Sources Used to Develop the Water System Characteristics

Data Source	Baseline Data Derived from the Source
SDWIS/Fed fourth quarter 2021 Q4 “frozen” dataset[a]	• Water System Inventory (Section 4.3.1): PWS inventory, including system unique identifier, population served, number of service connections, source water type, and system type. • Population and Households Served (Section 4.3.2): PWS population served. • Treatment Plant Characterization (Section 4.3.3.1): Number of unique treatment plant facilities per system, which are used as a proxy for entry points (EPs) when UCMR 3 sampling site data are not available.
UCMR 3 (U.S. EPA, 2017)	• Treatment Plant Characterization (Section 4.3.3): Number of unique EP sampling sites, which are used as a proxy for EPs. • Treatment Plant Characterization (Section 4.3.3): PFAS concentration data collected as part of UCMR 3.
Independent state sampling programs	• Treatment Plant Characterization (Section 4.3.3): PFAS concentration data collected by states. These data supplemented the occurrence modeling for systems included in UCMR 3.
SYR4 ICR Occurrence Dataset (2012-2019)	• Treatment Plant Characterization (Section 4.3.3): TOC.
Geometries and Characteristics of PWSs (U.S. EPA, 2000)	• Treatment Plant Characterization (Section 4.3.3): Design and average daily flow per system.
2006 CWSS (U.S. EPA, 2009)	• PWS Labor Rates (Section 4.3.4): PWS labor rates.

Abbreviations: CWSS – Community Water System Survey; ICR – Information Collection Request; PFAS – per- and polyfluoroalkyl substances; PWS – public water system; SDWIS/Fed – Safe Drinking Water Information System/federal version; SYR – Six-Year Review; TOC – total organic carbon; UCMR 3 – Third Unregulated Contaminant Monitoring Rule.
Note:
[a] Contains information extracted on January 14, 2022.

4.2.1 SDWIS/Fed 2021

SDWIS/Fed (U.S. EPA, 2021h) is the EPA’s national regulatory compliance database for the drinking water program. It contains system inventory, treatment facility, violation, and enforcement information for PWSs as reported by primacy agencies, EPA regions, and EPA headquarters personnel. Primacy agencies report data quarterly to the EPA. The information presented in the EA is based on the fourth quarter 2021 “frozen” dataset that was extracted on January 14, 2022.

SDWIS/Fed contains information to characterize the inventory of PWSs, namely: system name and location; retail population served, source water type, and PWS type.

4.2.1.1 PWS Type

The EPA defines a PWS as a system that provides water for human consumption through pipes or other constructed conveyances to at least 15 service connections or regularly serves an average of at least 25 individuals per day for at least 60 days per year (U.S. EPA, 2021h). Systems are categorized as follows:

- CWSs are systems that supply water to the same population year-round.

- Non-community water systems (NCWSs) are systems that supply water to a varying population or one that is served less than year-round; these are sub-categorized as:
    - Non-transient non-community water systems (NTNCWSs) are systems that are not CWSs and that regularly supply water to at least 25 of the same people at least six months per year (e.g., schools).
    - Transient non-community water systems (TNCWSs) are NCWSs that do not meet the non-transient criterion; they provide water in places such as gas stations or seasonal campgrounds where people do not remain for long periods of time.

A final rule to limit PFAS in drinking water would not apply to TNCWSs. Therefore, system inventories in this analysis are classified into two categories: CWSs and NTNCWSs.

4.2.1.1.1 Population Served

Systems are also categorized by the number of people they serve.[6] The following nine categories of populations served by systems are used throughout this EA:

- ≤ 100
- 101–500
- 501–1,000
- 1,001–3,300
- 3,301–10,000
- 10,001–50,000
- 50,001–100,000
- 100,001–1,000,000 (1M)
- >1M

The EPA uses these system size categories based on distinctions in the way systems operate as the amount of water supplied and number of service connections increases. Systems within each size category can be expected to face similar implementation and cost challenges when complying with the new regulatory requirements for this final rule.

4.2.1.1.2 Source Water Type

SDWIS/Fed classifies system by source water using the following six categories:

- Ground water
- Ground water purchased

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[6] SDWIS/Fed classifies systems according to "retail" population that does not include the population served by other systems that purchase water from them.

- Ground water under the direct influence (GWUDI)[7]
- Ground water under the direct influence purchased (purchased GWUDI)
- Surface water
- Surface water purchased

For this analysis, the EPA broadly categorized systems as surface water if any of their sources are surface water, surface water purchased, GWUDI, or purchased GWUDI. Systems are classified as ground water if they exclusively used ground water or purchased ground water.[8]

4.2.1.1.3 Facilities

SDWIS/Fed provides additional information on system facilities, including the type of facility, its activity status, and a unique facility identification number.

4.2.1.2 Verification of SDWIS/Fed Data

The EPA routinely conducts program reviews to verify whether information in the primacy agencies' databases and files, such as inventory and violations for all regulations are correctly represented in SDWIS/Fed. Between 2006 and 2016, the EPA recorded the findings from these reviews in the national Error Code Tracking Tool (ECTT) (U.S. EPA, 2007b). The ECTT contains, as individual records, all actions assessed during each program review. The EPA identifies records as confirmed actions (correct compliance determinations and correct reporting to SDWIS/Fed), compliance determination discrepancies (incorrect compliance determinations), or data flow discrepancies (correct compliance determination but incorrect reporting). This section presents data from the ECTT from program reviews conducted from 2006 to 2016 related to system inventory.

It is important to note that treatment data (objective codes and process codes for plants in SDWIS/Fed) are not evaluated during program reviews and therefore have more uncertainty associated with the data as compared to inventory and compliance data.

4.2.1.2.1 System Inventory

From 2006 to 2016 the EPA evaluated inventory data for a total of 2,180 systems. Prior to August 2007, the program reviews evaluated eight inventory fields: system type, system status, activity status, source type, population, service connection, administrative contact, and administrative address. After August 2007, the reviews did not include administrative contact or address. In addition, in August 2007, the review policy changed so that discrepancies for

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[7] 40 CFR Section 141.2 defines ground water under the direct influence of surface water as "any water beneath the surface of the ground with significant occurrence of insects or other macroorganisms, algae, or large-diameter pathogens such as Giardia lamblia or Cryptosporidium, or significant and relatively rapid shifts in water characteristics such as turbidity, temperature, conductivity, or pH which closely correlate to climatological or surface water conditions."

[8] 23 CWS and 11 NTNCWS have an unknown primary water source. For purposes of this analysis, the EPA assigned these systems to the source type ground water.

inventory were only identified if they affected monitoring requirements (e.g., a change in population that would increase or decrease the minimum number of required samples).

Of the inventory fields evaluated from 2006 to 2016, only 82 (<1%) inventory discrepancies were identified. Furthermore, some of these discrepancies, such as those related to administrative contact and address, may not impact the PWS baseline characterization. The inventory data in ECTT indicate a high degree of completeness and accuracy in SDWIS/Fed as of 2016, and the EPA expects that the information is largely representative of the regulated PWS.

4.2.2 Unregulated Contaminant Monitoring Rule

Every five years, the EPA issues a new list of no more than 30 unregulated contaminants to be monitored by PWSs. UCMR 3 was published in 2012 and required monitoring for six PFAS from 2013-2015: PFOA, PFOS, PFBS, PFNA, PFHxS, and PFHpA. The final UCMR 3 dataset of analytical results was released in January 2017.

Under UCMR 3, all CWSs and NTNCWSs with more than 10,000 retail customers and a representative sample of 800 systems serving 10,000 or fewer retail customers were required to conduct assessment monitoring to collect occurrence data for the listed contaminants suspected to be present in drinking water but that do not have health-based standards set under the SDWA.

Systems conducted assessment monitoring over one consecutive 12-month period between January 2013 and December 2015. Ground water systems were required to monitor twice during that period, with sampling events occurring five to seven months apart. Surface water systems were required to monitor in four consecutive quarters, with sampling events occurring three months apart. For the PFAS compounds, sampling was conducted at the entry point (EP) to the distribution system post treatment.

The fifth UCMR (UCMR 5), published December 2021, requires sample collection and analysis for 29 PFAS to occur between January 2023 and December 2025 using analytical methods developed by the EPA and consensus organizations. In the Federal Register Notice for this rulemaking, the EPA describes the small subset (7%) of data released as of August 2023, the limitations with considering an incomplete dataset, and that findings from analyses of these data are generally confirmatory of the EPA's other occurrence analyses. Because of the partial nature of this dataset, the EPA has not used it to characterize baseline occurrence in this EA.

4.2.3 Independent State Sampling Programs

The EPA used state monitoring data from 20 states (Alabama, Colorado, Illinois, Indiana, Kentucky, Maine, Maryland, Massachusetts, Michigan, Minnesota, Missouri, New Hampshire, New Jersey, New York, North Dakota, Ohio, South Carolina, Tennessee, Vermont, and Wisconsin). These states conducted non-targeted monitoring (i.e., random sampling) of finished drinking water for one or more of the four PFAS in this analysis.

4.2.4 Six-Year Review Data

The EPA used information from the fourth Six-Year Review Information Collection Request (ICR) Dataset ("SYR4 ICR dataset") to characterize the total organic carbon (TOC) level for individual systems. The SYR4 ICR dataset is the most comprehensive and current national drinking water occurrence dataset, containing millions of records of water system compliance monitoring data and treatment technique information for regulated chemical, radiological, and microbiological contaminants collected from 2012 through 2019. The portion of the dataset containing the TOC information was made publicly available in August 2022.[9]

4.2.5 Geometries and Characteristics of Public Water Systems (2000)

An important factor in determining costs of treatment is average daily flow and design flow, measured in gallons per day or million gallons per day (MGD), at a treatment plant. The EPA estimated the average daily flow and design flow for each EP in the system based on the relationship between retail population and flow as derived in the EPA's *Geometries and Characteristics of Public Water Systems* report (U.S. EPA, 2000).

Utilizing data from the 1995 CWSS, the EPA conducted an extensive data-cleaning process[10] to develop a dataset of 1,734 records with paired responses for population and total average daily flow. These data were then weighted to account for non-responses to individual questions from the CWSS. The EPA used this dataset to develop regression equations that predict average daily flow based on retail population served (for both publicly-owned and privately-owned systems). The data show a strong correlation as indicated by a high R-squared value of 0.90. Additional information and background data are provided in Chapter 4 of the *Geometries and Characteristics of Public Water Systems* report (U.S. EPA, 2000).

4.2.6 Community Water System Survey (2006)

The EPA periodically conducts the CWSS to obtain data to support the agency's development and evaluation of drinking water regulations. The 2006 CWSS is the most recent survey. For this EA, the EPA relied on the national average estimates of unit labor from the 2006 CWSS to derive the unit labor rates.

The EPA selected the CWSS as a data source because it is based on a nationally representative sample of CWSs. The sample was drawn from SDWIS/Fed, which includes approximately 50,000 systems in the 50 states and the District of Columbia. The survey used a stratified random sample design to ensure the sample was representative. The EPA selected a survey sample of 2,210 systems, including all systems serving populations of 100,000 or more. In the 2006 CWSS, the agency took additional steps to improve response rates, ensure accurate responses, and reduce the burden of the survey on systems, especially systems serving 3,300 or fewer persons. The EPA sent water system experts to collect data from systems serving 3,300 or fewer persons. For systems serving more than 3,300 people, the agency mailed the survey, made available a spreadsheet and Web-based version of the questionnaire, and provided extensive assistance through e-mail and a toll-free telephone hotline. The survey was designed to collect data for the

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[9] Available at: https://www.epa.gov/dwsixyearreview/microbial-and-disinfection-byproduct-data-files-2012-2019-epas-fourth-six-year

[10] The EPA adjusted the dataset to remove non-zero values; adjusted flow if needed to represent retail flow only removing wholesale water flow; and adjusted for reporting discrepancies in population, flow, or service connections.

year 2006. Full-scale data collection occurred from June to December 2007. The overall response rate was 59 percent with a total of 1,314 systems responding; 95 percent of selected systems serving 3,300 or fewer persons (representing 571 of 600 systems sampled) participated in the survey (U.S. EPA, 2009).

4.3 Drinking Water System Baseline/Industry Profile

This section presents the following baseline characterizations for the purposes of estimating costs and benefits for the final rule. Section 4.3.1 provides a characterization of the inventory of systems subject to the final rule (CWSs and NTNCWSs). Section 4.3.2 includes the population served by CWSs and NTNCWSs and the number of households served by CWSs. Section 4.3.3 provides treatment plant characteristics used to determine treatment costs. Section 4.3.4 describes the derivation of PWS labor rates. Finally, Section 4.3.5 describes the cost of capital rates used to estimate household-level costs. Each section includes a characterization of the baseline for CWSs, followed by NTNCWSs, if applicable, and a characterization of data limitations and uncertainty. TNCWSs are not subject to the final rule.

4.3.1 Water System Inventory

A key component of the baseline is the inventory of systems—both CWSs and NTNCWSs—subject to the final rule. As shown in Table 4-2, approximately 81 percent of all CWSs serve 3,300 or fewer people (39,746 of the total systems), and those serving 500 or fewer account for about 54 percent of all CWSs (26,742 of the total systems). CWSs serving 3,301–50,000 people represent about 17 percent of all CWSs (8,422 of the total systems), and those serving more than 50,000 people account for only about 2 percent (1,025 of the total systems). Most CWSs (about 77 percent or 37,733 systems) use ground water as their primary source. Most systems serving more than 10,000 people, however, are classified as surface water systems (about 63 percent or 2,817 systems).

Table 4-2: Inventory of CWSs

System Size (Population Served)	CWSs[a]		
	Ground Water	Surface Water	Total
	A	B	C = A + B
≤ 100	10,654	739	11,393
101–500	13,037	2,042	15,079
501–1,000	4,132	1,179	5,311
1,001–3,300	5,503	2,460	7,963
3,301–10,000	2,784	2,223	5,007
10,001–50,000	1,385	2,030	3,415
50,001–100,000	162	417	579
100,001–1M	74	347	421
> 1M	2	23	25
TOTAL	37,733	11,460	49,193

Abbreviations: CWS – community water systems.
Note:
[a] Includes 23 CWSs serving 10,000 or fewer people for which no primary source water type was reported to SDWIS/Fed. The EPA assigned these systems to the source type of ground water.
Source: SDWIS/Fed fourth quarter 2021 "frozen" dataset that contains information reported through January 14, 2022. Includes all active CWSs.

As shown in Table 4-3, approximately 99 percent of all NTNCWSs serve 3,300 or fewer people (17,135 of the total). NTNCWSs serving 3,301 – 50,000 people account for about 1 percent of all NTNCWSs (200 of the total). Only two NTNCWSs serve more than 50,000 people, and none serve more than 1 million people. Most NTNCWSs (about 95 percent or 16,531 systems) use ground water as their primary source. Approximately 51 percent (21 systems) of those serving 10,001–100,000 people use surface water versus ground water and the one system serving 100,001–1 million people is classified as a surface water system.

Table 4-3: Inventory of NTNCWSs

System Size (Population Served)	NTNCWSs[a]		
	Ground Water	Surface Water	Total
	A	B	C=A+B
≤ 100	8,084	252	8,336
101–500	6,111	257	6,368
501–1,000	1,476	91	1,567
1,001–3,300	743	121	864
3,301–10,000	97	63	160
10,001–50,000	20	20	40
50,001–100,000	0	1	1
100,001–1M	0	1	1
> 1M	0	0	0
TOTAL	16,531	806	17,337

Abbreviations: NTNCWS – non-transient non-community water systems.

Notes:

[a] Includes 11 NTNCWSs serving 3,300 or fewer people for which no primary source type was reported to SDWIS/Fed. The EPA assigned these systems to the source water type of ground water.

Source: SDWIS/Fed fourth quarter 2021 "frozen" dataset that contains information reported through January 14, 2022. Includes all active NTNCWSs.

There is uncertainty in the approach used to assign source water type to the 23 CWSs and 11 NTNCWSs where no primary source type was reported to SDWIS/Fed. This analysis assumes that these systems have ground water as their primary source based on the preponderance of ground water systems in the inventory. This could result in an under- or overestimate of costs in those instances where the cost model inputs vary by source type (e.g., number of EPs per system); however, the EPA expects the impact to be low because the systems without a source type in SDWIS/Fed represent a small proportion of systems subject to the rule (23 of the total 49,193 CWSs and 11 of the total 17,337 NTNCWSs or 0.05 percent of all systems subject to the rule) and all serve fewer than 10,000 people.

4.3.2 Population and Households Served

It is necessary to have an accurate characterization of population served by water systems when assessing the potential benefits of a final regulation. Population is also an input for estimating treated water volumes and associated granular activated carbon (GAC) or ion exchange (IX) costs.

SDWIS/Fed tracks "retail" population served, meaning that it counts only the population that purchases water directly from the water system, not the population of a system's wholesale customers. The systems that purchase water appear in SDWIS/Fed as a separate system with a unique PWS identification (PWSID) number.

Table 4-4 and Table 4-5 show the total population served and average population served per system by size category for CWSs and NTNCWSs, respectively. Each exhibit is organized by source water type (surface water or ground water) and is based on the SDWIS/Fed fourth quarter 2021 "frozen" dataset that contains information reported by primacy agencies through January 14, 2022.

Because systems often pass some or all of their costs onto customers in the form of rate increases, the final rule cost analysis also includes analyses to assess the impact of the rule requirements on annual household expenditures. The EPA estimated the number of households served by affected CWSs by dividing the population for each system size category by the average number of people per household. For CWSs, the EPA assumed an average of 2.53 persons per household based on 2020 U.S. Census data (U.S. Census Bureau, 2020b). This information is also included in Table 4-4 by system size and source type. NTNCWSs do not serve households, thus, this information is not included in Table 4-5.

As shown in Table 4-4, although CWSs serving 3,300 or fewer people account for approximately 81 percent of all CWSs, they serve fewer than 8 percent of the population and households that receive their water from a CWS. Although CWSs serving more than 50,000 people account for only 2 percent of all CWSs, they serve more than half (59 percent) of the population and households that receive their water from a CWS.

Table 4-4: Population and Number of Households Served by CWSs

System Size (Population Served)	Ground Water[c]			Surface Water			TOTAL		
	Population Served	Average Population Per System	Number of Households Served	Population Served	Average Population Per System	Number of Households Served	Population Served	Average Population Per System	Number of Households Served
	A	B[a]	C = A/2.53[b]	D	E[a]	F = D/2.53[b]	G	H[a]	I = G/2.53[b]
≤ 100[d]	652,335	61	257,840	45,231	61	17,878	697,566	61	275,718
101–500	3,254,293	250	1,286,282	576,601	282	227,906	3,830,894	254	1,514,187
501–1,000	3,032,366	734	1,198,564	883,656	749	349,271	3,916,022	737	1,547,835
1,001–3,300	10,264,020	1,865	4,056,925	4,935,965	2,006	1,950,974	15,199,985	1,909	6,007,899
3,301–10,000	15,794,291	5,673	6,242,803	13,633,206	6,133	5,388,619	29,427,497	5,877	11,631,422
10,001–50,000	28,665,202	20,697	11,330,119	46,262,480	22,789	18,285,565	74,927,682	21,941	29,615,685
50,001–100,000	10,889,918	67,222	4,304,315	29,350,794	70,386	11,601,104	40,240,712	69,500	15,905,420
100,001–1M	15,082,760	203,821	5,961,565	84,675,709	244,022	33,468,660	99,758,469	236,956	39,430,225
> 1M	3,400,000	1,700,000	1,343,874	44,266,001	1,924,609	17,496,443	47,666,001	1,906,640	18,840,317
TOTAL[e]	91,035,185	2,413	35,982,287	224,629,643	19,601	88,786,420	315,664,828	6,417	124,768,707

Abbreviations: CWS – community water systems.

Notes:

[a] B, E, and H: Derived by dividing the population served by the number of systems presented in Table 4-2.

[b] C, F, and I: The average of 2.53 persons per household is from 2020 U.S. Census data (Table AVG1. Average Number of People per Household, by Race and Hispanic Origin/1, Marital Status, Age, and Education of Householder: 2020).

[c] CWSs with unreported primary source were assumed to be ground water systems. Thus, the ground water column reflects an additional 23 CWSs with unreported primary source type.

[d] The EPA removed any CWS wholesaler serving less than 25 people from the analysis and assumed that any remaining CWS had a minimum possible population of 25.

[e] Numbers may not sum to total because of rounding.

Source for A, D, and G: SDWIS/Fed fourth quarter 2021 "frozen" dataset that contains information reported through January 14, 2022.

As previously discussed, NTNCWSs serving 3,300 or fewer people account for approximately 99 percent of all NTNCWSs. As shown in Table 4-5, these systems serve approximately 70 percent of the population that receives their water from an NTNCWS. Those serving 3,301–50,000 people and more than 50,000 people serve approximately 26 percent and 4 percent of the population that receives water from an NTNCWS, respectively.

Table 4-5: Population Served by NTNCWSs

	Ground Water[b]		**Surface Water**		**TOTAL**	
System Size (Population Served)	**Population Served**	**Average Population Per System**	**Population Served**	**Average Population Per System**	**Population Served**	**Average Population Per System**
	A	**B[a]**	**D**	**E[a]**	**F**	**G[a]**
≤ 100[c]	452,516	56	12,534	50	465,050	56
101–500	1,513,562	248	69,046	269	1,582,608	249
501–1,000	1,049,638	711	68,235	750	1,117,873	713
1,001–3,300	1,241,973	1,672	239,516	1,979	1,481,489	1,715
3,301–10,000	511,494	5,273	377,219	5,988	888,713	5,554
10,001–50,000	397,246	19,862	414,099	20,705	811,345	20,284
50,001–100,000	0	0	71,963	71,963	71,963	71,963
100,001–1M	0	0	203,375	203,375	203,375	203,375
> 1M	0	0	0	0	0	0
TOTAL[d]	5,166,429	313	1,455,987	1,806	6,622,416	382

Abbreviations: NTNCWS – non-transient non-community water systems.
Notes:
[a] B, E, and G: Derived by dividing the population served by the number of systems presented in Table 4-3.
[b] NTCWSs with unreported primary source were assumed to be ground water systems. Thus, the "Ground Water" column reflects an additional 11 NTCWSs with unreported primary source type.
[c] The EPA assumed any non-wholesale NTNCWS had a minimum possible population of 25.
[d] Numbers may not sum to total because of rounding.
Source for A, D, and F: SDWIS/Fed fourth quarter 2021 "frozen" dataset that contains information reported through January 14, 2022.

As noted previously, the EPA consistently classifies systems in SDWIS/Fed according to the retail population served by the system and does not include the population served by wholesale customers. Wholesale customers who purchase water from another system and meet the PWS definition have their own unique PWSID, retail population, and associated regulatory requirements under SDWA. The EPA uses retail population to estimate design and average daily flow parameters, which are then used to estimate treatment costs associated with the rule. Use of retail population may overestimate aggregate costs by assuming that each system will have an individual treatment plant instead of the more common scenario of the seller having one large plant and selling treated water to their wholesale customers. Because of returns to scale in treatment capital costs, the cost of a single large plant will be less than the sum of the costs across several small plants treating the same aggregate flow.

In addition, given that some of the reported population values would create inconsistencies in the analysis, the EPA removed any CWS wholesaler serving less than 25 people from its analysis and assumed that any remaining CWS had a minimum possible population of 25. The EPA assumed any non-wholesale NTNCWS had a minimum possible population of 25.

4.3.3 Treatment Plant Characterization/Production Profile

This section explains the baseline inputs for the following treatment-related PWS characteristics. Section 4.3.3.1 discusses the EPs per system characterization. Section 4.3.3.2 discusses the EPA's TOC baseline assumptions and Section 4.3.3.3 presents the estimation method and the computed average daily flows and design flows by system type and size.

4.3.3.1 Entry Points Per System

EPs are the point of compliance for the final rule and systems can have multiple EPs. The EPA developed estimates of EPs per system using UCMR 3 unique sampling points, SDWIS/Fed facility data, and a modeled frequency distribution.

UCMR 3 required a subset of CWSs and NTNCWSs to conduct assessment monitoring for six PFAS compounds.[11] The data record a unique identifying number for the EP sample location(s) for each system. Given the information provided, the EPA assumes that the number of unique sample point IDs per system approximates the total number of EPs per system.

For systems without UCMR 3 occurrence data, the EPA developed estimates based on SDWIS/Fed facilities data. The SDWIS/Fed data include unique identification numbers for system facilities, as well as facility type and activity status. This analysis relies on active facilities identified as treatment plants. Using the assumption that treatment plants are associated with one EP, the SDWIS/Fed facility data provide an approximation for the number of EPs per system when a system does not have UCMR 3 occurrence data. The EPA considers the UCMR 3 sampling point data to be of higher quality than the SDWIS/Fed treatment facility data. If the SDWIS/Fed treatment facility data value for a system exceeded the maximum number found for the equivalent system size and source water combination in the UCMR 3 data, the EPA limited the system EP value to the UCMR3 maximum number of EPs.

For systems without UCMR 3 occurrence data or SDWIS/Fed facility data, the EPA relies on an estimate of the number of EPs. The estimated value for each system with missing EP count data was imputed from known EP counts for stratified SDWIS/Fed data. Within each stratum, defined by a combination of system size and source water, the EPA sampled from systems with known EP counts. Sampling was done with replacement after truncating the EP counts to the maximum recorded in UCMR 3. For reproducibility, the EPA performed this sample-based imputation in R using the 'base::sample' function (R Core Team, 2021).

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[11] UCMR 3 required all systems serving more than 10,000 people to collect and analyze samples for PFOA, PFOS, PFNA, PFHxS, PFBS, and PFHpA at each distribution system entry point. The EPA also identified a stratified random sample of 800 small systems serving up to 10,000 people to collect samples for these six PFAS.

Following this process, the EPA relies on sample point values recorded in UCMR 3 for 5,419 systems, SDWIS/Fed facility data for 43,563 systems, and imputed EP values for 17,523 systems. All systems have at least one EP. Among CWSs, the maximum number of EPs is 202, and the mean is 1.80. Among NTNCWSs, the maximum number of EPs is 22, and the mean is 1.31.

Table 4-6 summarizes the final frequency distribution of EP input ranges for each CWS stratum of size and source water combination. Table 4-7 summarizes the final frequency distribution of EP input ranges for each NTNCW stratum of size and source water combination. These distributions are used to proportionally assign numbers of EPs to systems in each system size and type category.[12]

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[12] The SDWIS/Fed data provide information on the PWS characteristics that typically define PWS categories, or strata, for which the EPA develops costs in rulemakings. These characteristics include system type (CWS, NTNCWS), number of people served by the PWS, PWS's primary raw water source (ground water or surface water), PWS's ownership type (public or private), and PWS state. For more information on the use of baseline and compliance characteristics to define model systems in the EPA's cost analysis, please see Section 5.2.

Table 4-6: Frequency Distribution of EP Inputs for CWSs

	Ground Water							Surface Water						
System Size	**1 EP**	**2–5 EP**	**6–10 EP**	**11–15 EP**	**16–20 EP**	**21–100 EP**	**> 100 EP**	**1 EP**	**2–5 EP**	**6–10 EP**	**11–15 EP**	**16–20 EP**	**21–100 EP**	**> 100 EP**
≤ 100	90%	10%	0.1%	0	0	0	0	87%	13%	0	0	0	0	0
101–500	76%	24%	0	0	0	0	0	84%	16%	0	0	0	0	0
501–1,000	62%	38%	0.5%	0	0	0	0	76%	23%	0.8%	0	0	0	0
1,001–3,300	48%	50%	1%	0	0	0	0	70%	30%	0.7%	0	0	0	0
3,301–10,000	32%	59%	8%	0.9%	0.1%	0	0	54%	43%	3%	0.5%	0.04%	0	0
10,001–50,000	3%	58%	28%	7%	3%	1%	0.07%	3%	82%	10%	2%	1%	0.6%	0
50,001–100,000	0	51%	25%	8%	8%	9%	0	0.2%	74%	13%	6%	2%	4%	0
100,001–1M	0	34%	22%	11%	8%	24%	1%	0.3%	67%	13%	4%	9%	6%	0.3%

Abbreviations: CWS – community water systems; EP – entry point.

Table 4-7: Frequency Distribution of EP Inputs for NTNCWSs

	Ground Water					Surface Water				
System Size	**1 EP**	**2–5 EP**	**6–10 EP**	**11–20 EP**	**> 20 EP**	**1 EP**	**2–5 EP**	**6–10 EP**	**11–20 EP**	**> 20 EP**
≤ 100	84%	16%	0.4%	0	0	82%	18%	0	0	0
101–500	81%	19%	0	0	0	74%	26%	0	0	0
501–1,000	0	0	0	0	0	0	0	0	0	0
1,001–3,300	68%	30%	2%	0	0	61%	31%	8%	0	0
3,301–10,000	53%	44%	2%	1%	0	35%	44%	14%	6%	0
10,001–50,000	10%	80%	0	10%	0	30%	40%	5%	20%	5%
50,001–100,000	0	0	0	0	0	0	100%	0	0	0
100,001–1M	0	0	0	0	0	0	100%	0	0	0

Abbreviations: NTNCWS – non-transient non-community water systems; EP – entry point.

4.3.3.2 Total Organic Carbon

The effectiveness of the GAC treatment process varies with the level of TOC in the influent water. There is no national dataset containing TOC values for every CWS or NTNCWS. Therefore, the EPA randomly assigned a TOC level to each system based on two distributions of TOC in 'finished' water. The agency developed distributions using TOC data voluntarily submitted by states in response to the SYR4 ICR drinking water regulations. Because TOC levels in ground water are lower on average than TOC levels in surface water, the EPA separated the data by system primary source water. TOC levels can also vary throughout a system. Source water TOC measurements can be higher than finished water estimates if a treatment process removes TOC. For each system, the EPA identified TOC measurements that best represented finished water quality. Using the resulting distribution of ground water or surface water estimates, the EPA identified decile midpoint values to randomly assign to each system.

4.3.3.3 Average Daily Production Flow and Design Flow

Average daily production flow and design flow per system are based on regression equations from the EPA's *Geometries and Characteristics of Public Water Supplies* report (U.S. EPA, 2000). The average daily flow and design flow are functions of the population served, with different equations for source water type (surface water or ground water). Table 4-8 presents these flow equations. The flow was then divided by the number of EPs to calculate the flow per treatment plant for the system (assuming each EP has one treatment plant). The EPA does not have comparable flow-population regression equations for NTNCWSs and, therefore, used the CWS relationships to estimate flow for NTNCWSs.

Table 4-8: Functions for Design and Average Daily Flow by System Types

Design Flow Functions (kgal)	
Surface water system	$\text{Design Flow} = 0.59028 \times \text{Population}^{0.94573}$ (or 2 x Average Flow, whichever is greater)
Ground water system	$\text{Design Flow} = 0.54992 \times \text{Population}^{0.95538}$ (or 2 x Average Flow, whichever is greater)
Average Daily Flow Functions (kgal)	
Surface water system	$\text{Average Flow} = 0.14004 \times \text{Population}^{0.99703}$
Ground water system	$\text{Average Flow} = 0.08575 \times \text{Population}^{1.05839}$

Abbreviations: kgal – 1000 gallons.

As an example, Table 4-9 shows the design flow and average daily flow results when applying the regression equations to the average population per system for each CWS system stratum. The results for NTNCWSs are in Table 4-10. Note that these results are examples only. In practice, the EPA applied the regression equations to the population served of individual systems, instead of the stratum average population. In addition, for systems serving more than 1 million people, the EPA obtained publicly available system-specific information on the average daily flow and design flow for each EP whenever possible (e.g., annual Consumer Confidence Reports).

Table 4-9: Design and Average Daily Flow for CWSs

System Size	Ground Water			Surface Water		
	Average Population	Design Flow (MGD)	Average Flow (MGD)	Average Population	Design Flow (MGD)	Average Flow (MGD)
≤ 100	61	0.028	0.007	61	0.029	0.008
101–500	250	0.107	0.030	282	0.123	0.039
501–1,000	734	0.301	0.093	749	0.309	0.103
1,001–3,300	1,865	0.733	0.248	2,006	0.784	0.275
3,301–10,000	5,673	2.121	0.806	6,133	2.255	0.837
10,001–50,000	20,697	7.305	3.171	22,789	7.804	3.098
50,001–100,000	67,222	22.512	11.031	70,386	22.671	9.535
100,001–1M	203,821	71.371	35.685	244,022	73.470	32.937

Abbreviations: CWS – community water systems; MGD – million gallons per day.

Table 4-10: Design and Average Daily Flow for NTNCWSs

System Size	Ground Water			Surface Water		
	Average Population	Design Flow (MGD)	Average Flow (MGD)	Average Population	Design Flow (MGD)	Average Flow (MGD)
≤ 100	56	0.026	0.006	50	0.024	0.007
101–500	248	0.107	0.029	269	0.117	0.037
501–1,000	711	0.292	0.089	750	0.309	0.103
1,001–3,300	1,672	0.660	0.221	1,979	0.774	0.271
3,301–10,000	5,273	1.978	0.746	5,988	2.205	0.817
10,001–50,000	19,862	7.023	3.035	20,705	7.127	2.815
50,001–100,000	Not applicable	Not applicable	Not applicable	71,963	23.151	9.748
100,001–1M	Not applicable	Not applicable	Not applicable	203,375	61.841	27.465

Abbreviations: NTNCWS – non-transient non-community water systems; MGD – million gallons per day.

4.3.4 Public Water System Labor Rates

The EPA recognizes that there may be variation in labor rates across all systems. However, for purposes of this EA, the EPA used national average estimates of unit labor from the 2006 CWSS, with a few modifications described below. Prior labor unit costs for managerial, technical, and clerical labor in the EPA's work breakdown structure[13] (WBS) were based on a review of data from three sources:

- The Occupational Employment Survey (OES), a semi-annual Bureau of Labor Statistics (BLS) survey that provides hourly wage estimates by occupation and industry (BLS, 2022b).

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[13] To estimate treatment costs, the EPA uses several engineering models using a bottom-up approach known as work breakdown structure (WBS). The WBS models derive system-level costs and provide the EPA with comprehensive, flexible and transparent tools to help estimate treatment costs.

- The Water Utility Compensation Survey, an annual American Water Works Association (AWWA) survey that provides hourly wage estimates for the water and wastewater industry by occupation. Data are in 2008 dollars.
- The 2006 CWSS, a periodic EPA survey that obtains employment information from a sample of CWSs.

There are more recent wage data from the OES and AWWA surveys, but there has not been a CWSS since 2006. A 2020 review of the WBS labor rates found that the WBS wage rates in 2019 dollars overstate labor costs for clerical labor hours as well as potentially overstate labor costs for technical labor hours (Abt Associates, 2020). Following these findings, the EPA adjusted the labor costs used in the WBS to reflect occupation-specific escalation factors rather than the seasonally adjusted employment cost index (ECI) for all civilian employees. The WBS labor costs for managerial hours were not clearly over- or understated compared to OES data but were consistently lower than the AWWA wage estimates (Abt Associates, 2020).

Table 4-11 presents the labor rate estimates used in the WBS in 2007 dollars. Labor rates were calculated for three occupation categories: technical, managerial, and clerical. The rates do not include benefits.

Table 4-11: Hourly Wage Rates Based on CWSS Data ($2007)

Occupation	≤ 500	501–3,300	3,301–10,000	10,001–50,000	50,001–100,000	> 100,000
Technical	$16.97	$16.97	$18.10	$19.11	$19.95	$23.32
Managerial	$24.06	$24.06	$27.52	$30.65	$35.76	$38.21
Clerical	$16.21	$16.21	$16.21	$20.93	$20.93	$20.93

Abbreviations: CWSS – Community Water System Survey.
Source: Abt Associates, 2020

A review of updated BLS Employer Cost for Employee Compensation (ECEC) data indicated that benefits account for a higher proportion of total compensation today than they did at the end of 2006 (Abt Associates, 2020). The WBS assumes a benefit multiplier of 1.45, which is the 2020 multiplier for all civilians working in service-producing industries (Abt Associates, 2020). The benefit-loaded wage rates are shown in Table 4-12.

Table 4-12: Hourly Labor Costs Including Wages Plus Benefits ($2007)

Occupation	≤ 500	501–3,300	3,301–10,000	10,001–50,000	50,001–100,000	> 100,000
Technical	$24.61	$24.61	$26.25	$27.71	$28.93	$33.81
Managerial	$34.89	$34.89	$39.90	$44.44	$51.85	$55.40
Clerical	$23.50	$23.50	$23.50	$30.35	$30.35	$30.35

Source: Abt Associates, 2020

Because the WBS relies on 2020 dollar values, the EPA escalated the CWSS values using the OES occupation-specific change in mean wage rate from 2007 to 2020 instead of the general civilian ECI escalation rate. The escalation for the technical rate is 35.2 percent and the escalation for the clerical rate is 36.3 percent. The WBS managerial wage rates are consistent with OES rates, but slightly lower than AWWA rates (Abt Associates, 2020). At the time of the analysis in 2020, the OES occupation-specific wage escalation rate for the managerial rate was comparable to the ECI rate (Abt Associates, 2020). Therefore, the WBS retains the ECI escalated managerial labor rates, which for 2020 is 41.4 percent. The national cost-benefit analysis method described in Section 5.2 presents all values in 2022 dollars. The method uses the gross domestic product (GDP) implicit price deflator to adjust values in other dollar years to 2022 dollars. Therefore, the labor costs including wages and benefits in 2022 dollars shown in Table 4-13 reflect an additional adjustment for dollar year. The EPA applied the same system labor rates to both CWSs and NTNCWSs.

Table 4-13: Hourly Labor Costs Escalated to $2022

Occupation	≤ 500	501–3,300	3,301–10,000	10,001–50,000	50,001–100,000	> 100,000
Technical	35.48	35.48	37.84	39.94	41.70	48.74
Managerial	52.60	52.60	60.16	67.02	78.19	83.55
Clerical	34.17	34.17	34.17	44.12	44.12	44.12

There is uncertainty in the derivation of labor rates that could result in an over- or underestimate of national costs of the final rule. The mean labor rate is based on findings of the 2006 CWSS. The labor rate mix may have changed since the time of the survey. The EPA accounted for general changes in cost of labor by adjusting 2007 values to 2020 using occupation-specific escalators and the ECI where appropriate. There is also uncertainty in assuming a 1.45 benefits multiplier; this may cause an under- or overestimation of cost of the final rule.

4.3.5 Cost of Capital

For the social cost-benefit analysis, the EPA uses a social discount rate of 2 percent to discount future values and annualize discounted present value over the period of analysis. This rates is in accordance with OMB Circular A-4 (OMB, 2023).

When evaluating the economic impacts on PWSs and households, however, the EPA uses estimated cost of capital to discount future costs and annualize the discounted present value over the analysis period. This rate best represents the actual costs of compliance that systems will incur over time. To estimate PWS cost of capital, the EPA used data from the 2006 CWSS. The CWSS defined the following categories of funding sources:

- Current revenue;
- Equity or other funds from private investors;
- Department of Homeland Security (DHS) grant;
- Other government grants;
- Drinking Water State Revolving Fund (DWSRF), including loans and Principal Repayment Forgiveness;
- Other borrowing from public sector sources; and
- Borrowing from private sectors sources.

The EPA calculated the overall weighted average cost of capital (across all funding sources and loan periods) for each size/ownership category, weighted by the percentage of funding from each source.[14] Table 4-14 shows the cost of capital for each CWS size category and ownership type. Similar cost of capital information is not available for NTNCWS. Therefore, the EPA used the CWS cost of capital when calculating the annualized cost per NTNCWS.

Table 4-14: Weighted Average Cost of Capital by PWS Ownership and Size Category

Size Category	Publicly Owned CWS	Privately Owned CWS
≤100	3.8%	7.8%
101–500	5.5%	8.2%
501–1,000	4.0%	8.6%
1,001–3,300	4.7%	7.1%
3,301–10,000	5.8%	7.0%
10,001–50,000	6.1%	7.0%
50,001–100,000	4.9%	6.9%
100,001–500,000	4.7%	3.9%
Over 500,000	3.7%	7.8%

Abbreviations: PWS – public water system; CWS – community water system.

Since the CWSS data collection, Congress established new programs and expanded funding for existing programs. These funding sources allow PWSs to lower their cost of capital. These include the DWSRF, the Water Infrastructure Finance and Innovation (WIFIA) program, the Water Infrastructure Improvements for the Nation Act of 2016 (WIIN Act), and the Bipartisan Infrastructure Law of 2021 (BIL).

Through the DWSRF Program, the EPA allocates annual capitalization grants to states. The grants, along with state matching monies, support a dedicated loan fund to finance eligible water system infrastructure improvement projects. States are permitted to use funding from their DWSRF to help PWS finance water treatment through low-interest loans. These loans range from zero percent to market rate. The weighted average interest rate across all signed DWSRF loans in the past ten fiscal years (2013 through 2023) has been below 2% each year, with the weighted average for the 2023 state fiscal year of 1.6%. EPA notes these weighted averages reflect the rates signed into final loans, not the range of possible rates offered during those years. The WIFIA program provides creditworthy PWSs access to low-interest direct federal loans for water treatment investment. The WIIN Act established a grant program to help small, underserved, and disadvantaged communities achieve compliance with drinking water standards. Additionally, the BIL (P.L. 117-58) authorizes $5 billion as part of the Emerging Contaminants in Small or Disadvantaged Communities (EC-SDC) grants program that can be used to reduce PFAS in drinking water in communities facing disproportionate impacts. BIL funds will be provided as grants and loan forgiveness associated with PFAS drinking water treatment capital expenditures. Overall, the actual cost of capital faced by some PWSs may be lower than those used in this analysis.

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[14] See "Cost of Capital Approach.doc" in the docket for details of how the cost of capital estimates were developed.

4.4 Occurrence of PFAS

The EPA's *PFAS Occurrence & Contaminant Background Support Document* provides estimates of the baseline PFAS occurrence in PWSs (U.S. EPA, 2024g). After reviewing the available data on PFAS in drinking water, the EPA determined that the data from the UCMR 3 are the best available nationally representative data to characterize the occurrence of multiple PFAS in drinking water. Consistent with the agency's commitment in the final regulatory determination for PFOA and PFOS and the EPA's PFAS Strategic Roadmap to present the best available occurrence information, the agency supplemented the UCMR 3 data with data collected by states that have made their data publicly available (U.S. EPA, 2021b; U.S. EPA, 2021e).

This section summarizes the EPA's PFAS occurrence analysis (U.S. EPA, 2024g). Section 4.4.1 provides an overview of UCMR 3 and its PFAS occurrence data. Section 4.4.2 provides an overview of state PFAS monitoring data. Section 4.4.4 summarizes the EPA's analysis of PFAS drinking water occurrence data. Section 4.4.5 summarizes the national PFAS occurrence estimates used in the cost and benefit analyses.

4.4.1 Overview of UCMR 3 Data

The UCMR is a national drinking water monitoring program administered by the EPA. The UCMR 3 monitoring cycle included a census of all large CWSs and NTNCWSs (i.e., those serving more than 10,000 people) and a statistical sample of 800 small CWSs and NTNCWSs (i.e., those serving 10,000 people or fewer). Monitoring under UCMR 3 occurred from 2013 to 2015. More information on the UCMR 3 study design and data analysis can be found in U.S. EPA (2012) and U.S. EPA (2019c).

The EPA collected the UCMR 3 data from PWSs in all 50 states and seven additional primacy agencies. UCMR 3 monitoring occurrence data are available for six PFAS: PFOS, PFOA, PFNA, PFHxS, PFHpA, and PFBS. For the individual PFAS contaminants, the EPA collected nearly 37,000 finished water samples from 4,920 PWSs.

Systems collected PFAS samples at each EP to their customer distribution system. EPs are the point of compliance for the final rule, and systems can have multiple EPs. The sampling frequency varied by source water: four quarterly samples in a one-year period for surface water systems, and two samples at least six months apart for ground water systems.

The EPA's *PFAS Occurrence & Contaminant Background Support Document* (U.S. EPA, 2024g) describes the data and analyses that the EPA used to develop national estimates of PFAS occurrence in public drinking water systems using UCMR 3 data.

4.4.2 Overview of State PFAS Data

Outside of the UCMR 3 data collection, many states have undertaken individual efforts to monitor for PFAS in both source and finished drinking water. The EPA collected data from 32 states that have made their data available and represents sampling conducted on or before May 2023. The EPA notes that this data collection cutoff was made to allow sufficient time for the agency to conduct analyses on the state information for the final NPDWR. Due to the limitations in representation and reporting of some of the available data, the EPA conducted technical analyses using a subset of the available state data from 20 states. These more recent state data, collected using improved analytical methods that have lower reporting limits than under UCMR 3, show widespread occurrence of PFOA, PFOS, PFNA, and PFHxS and co-occurrence of these four PFAS and PFBS in multiple geographic locations. These data also show that these PFAS occur with substantial frequency at lower concentrations than were analyzed under UCMR 3, as demonstrated within the EPA’s *PFAS Occurrence & Contaminant Background Support Document* (U.S. EPA, 2024g). Furthermore, these state data include results for more PFAS than were included in the UCMR 3, including HFPO-DA. Please see Sections III and VI of the Federal Register Notice for discussion about how these data enhanced and supported the EPA's occurrence analyses and were confirmatory of the EPA's findings and conclusions in the proposed PFAS NPDWR.

The EPA’s analysis of state PFAS data shows occurrence in multiple geographic locations consistent with what was observed during UCMR 3 monitoring. The agency notes that the data vary in terms of quantity and coverage; for example, some of these available data are from targeted sampling efforts (i.e., monitoring in areas of known or potential contamination) and thus may not be representative of levels found in all PWSs within the state. Summaries on the non-targeted state PFAS finished water data are available in Table 4-15 and Table 4-16. Specifically, a summary on the percent of samples in state datasets that were above reporting thresholds for select PFAS is provided in Table 4-15, and a summary on the number of systems in state datasets that had detections for select PFAS is available in Table 4-16. Comprehensive summaries of state data are available within the EPA’s *PFAS Occurrence & Contaminant Background Support Document* (U.S. EPA, 2024g).

Table 4-15: Non-Targeted State PFAS Finished Water Data – Summary of Samples with Detections of PFAS Included in Final Regulation

State	PFHxS[a]	PFNA[a]	PFBS[a]	HFPO-DA[a,b]
Colorado	10.8%	0.9%	11.0%	0.2%
Illinois	13.4%	0.6%	17.6%	0.0%
Indiana	1.5%	0.2%	5.6%	0.0%
Kentucky	8.6%	2.5%	12.3%	13.6%
Maine	3.0%	3.5%	10.1%	N/A
Maryland	18.2%	2.3%	19.3%	0.0%
Massachusetts	23.6%	2.9%	39.8%	0.1%
Michigan	4.3%	0.6%	7.5%	0.1%
Missouri	3.3%	0.0%	6.1%	0.0%
New Hampshire	16.8%	3.3%	32.1%	3.8%
New Jersey	26.2%	7.7%	28.1%	N/A
New York	21.6%	8.6%	28.8%	0.7%
North Dakota	5.3%	0.0%	8.8%	0.0%
Ohio	6.6%	0.3%	5.0%	0.1%
South Carolina	8.1%	0.1%	13.7%	1.3%
Tennessee	0.0%	0.0%	0.0%	N/A
Vermont	4.2%	2.5%	7.1%	0.2%
Wisconsin	27.2%	2.2%	28.0%	0.0%

Abbreviations: PFAS – per- and polyfluoroalkyl substances.

Note:

[a] 0.0 % indicates that monitoring data were available for the compound/state but there were no detections above minimum reporting limits. Detections are determined by individual state reporting limits which are not defined consistently across all states.

[b] N/A indicates that no data are available.

Table 4-16: Non-Targeted State PFAS Finished Water Data – Summary of Systems with Detections of Select PFAS

State	PFHxS[a]	PFNA[a]	PFBS[a]	HFPO-DA[a,b]
Colorado	13.4%	1.0%	13.4%	0.3%
Illinois	4.6%	0.5%	8.0%	0.0%
Indiana	1.3%	0.3%	6.5%	0.0%
Kentucky	9.5%	2.7%	13.5%	12.2%
Maine	2.8%	3.9%	10.3%	N/A
Maryland	12.7%	3.2%	12.7%	0.0%
Massachusetts	18.1%	4.4%	27.8%	0.3%
Michigan	4.1%	0.6%	7.9%	0.3%
Missouri	2.7%	0.0%	6.2%	0.0%
New Hampshire	22.5%	5.5%	38.1%	5.1%
New Jersey	32.9%	16.5%	35.2%	N/A
New York	25.0%	9.7%	36.7%	1.1%
North Dakota	5.4%	0.0%	9.0%	0.0%
Ohio	2.2%	0.3%	2.4%	0.1%
South Carolina	13.7%	0.3%	22.1%	2.0%
Tennessee	0.0%	0.0%	0.0%	N/A
Vermont	2.7%	0.9%	6.0%	0.5%
Wisconsin	31.8%	3.9%	33.9%	0.0%

Abbreviations: PFAS – per-and polyfluoroalkyl substances.

Note:

[a] 0.0 % indicates that monitoring data were available for the compound/state but there were no detections above minimum reporting limits. Detections are determined by individual state reporting limits which are not defined consistently across all states.

[b] N/A indicates that no data are available.

4.4.3 Overview of PFAS Co-Occurrence

Co-occurrence of multiple PFAS has been reported in drinking water, ambient surface waters, aquatic organisms, biosolids (sewage sludge), and other environmental media. PFOA and PFOS have historically been target analytes, which has partly contributed to their prevalence in environmental monitoring studies, although some recent monitoring studies have begun to focus on additional PFAS via advanced analytical instruments/methods and non-targeted analysis (McCord & Strynar, 2019; McCord et al., 2020).

The EPA's analysis on PFAS co-occurrence using UCMR 3 data found that 4 percent of PWSs reported results for which one or more of the six UCMR 3 PFAS were measured at or above their respective UCMR 3 minimum reporting levels (MRL). Additionally, several studies have demonstrated PFAS co-occurrence in finished drinking water (Adamson et al., 2017; Cadwallader et al., 2022; Guelfo & Adamson, 2018; Smalling et al., 2023). One study in particular used UCMR 3 data to demonstrate that two or more of the six PFAS monitored under UCMR 3 co-occurred in 48 percent (285/598) of sampling events with PFAS detected, and PFOA and PFOS co-occurred in 27 percent (164/598) of sampling events with two or more PFAS detected (Guelfo & Adamson, 2018).

For additional discussion and analysis on PFAS co-occurrence, reference the EPA's *PFAS Occurrence & Contaminant Background Support Document* (U.S. EPA, 2024g).

4.4.4 Summary of PFAS Occurrence Data Analysis

Identifying the systems and population exposed to PFAS exceeding the limits under the final rule and the three regulatory alternatives is a key step to estimating benefits and costs of the final NPDWR. The EPA used a Bayesian hierarchical Markov chain Monte Carlo (MCMC) occurrence model to estimate national PFAS occurrence in PWSs. The EPA used the MCMC occurrence model output to estimate the PWSs and EPs with PFAS occurrence exceeding the limits under the final rule and regulatory alternatives. The EPA assumed that the populations served by these PWSs were exposed to the PFAS concentration estimates generated by the MCMC occurrence model.

This section summarizes the occurrence model and the EPA's use of the model to identify the systems and EPs with PFAS occurrence exceeding the regulatory alternatives considered within the EA, as well as the corresponding populations exposed. Further details on the MCMC model are available in Appendix A, Cadwallader et al. (2022), and U.S. EPA (2024g).

Data collected under UCMR 3 served as the primary dataset for the MCMC occurrence model due to its nationally representative design. Additionally, the EPA incorporated state PFAS monitoring datasets to supplement UCMR 3 data in the occurrence model. These state datasets, for which the monitoring has been conducted more recently than UCMR 3, generally have lower reporting limits because the analytical methods have improved over the last 10 years, allowing laboratories to reliably measure PFAS at concentrations approximately 5 to 20 times lower than for UCMR 3. Thus, state datasets with lower reporting limits than those in UCMR 3 helped inform the model by enabling observation of PFAS occurrence at lower concentrations. State datasets also consist of more recent samples than UCMR 3, which broadened the temporal range of data used to fit the model. The supplemental state data were limited to samples collected from systems that were also in UCMR 3 to prevent biasing the dataset toward states for which the data from additional PWSs were available as well as maintain the nationally representative set of systems selected for UCMR 3. Using these criteria, 28 states were identified as having some state monitoring data to be included in fitting the national occurrence model.

The dataset used to fit the model included all data available in the final UCMR 3 dataset for PFOS, PFOA, PFHpA, and PFHxS. This amounted to 36,972 samples each for PFOS, PFOA, and PFHpA, and 36,971 samples for PFHxS. Of these four PFAS, 1,114 samples had results reported at or above the UCMR 3 MRLs. The additional state datasets included to supplement the UCMR 3 data contained 18,091 PFOS samples, 18,082 PFOA samples, 14,458 PFHpA samples, and 14,906 PFHxS samples collected at systems that were included in UCMR 3. Of these samples, 7,156 (40%) were reported values for PFOS, 8,257 (46%) were reported values for PFOA, 4,496 (31%) were reported values for PFHpA, and 5,041(34%) were reported values for PFHxS. The remainder were listed as being below their respective reporting limits. A summary of the state data used in the occurrence model, including system and sample counts, is available in Appendix A.

Some states have promulgated drinking water standards for PFAS since the UCMR 3 monitoring. The EPA reviewed state websites and identified states with enacted standards for the PFAS compounds considered within the regulatory alternatives discussed in the EA. Table 4-17 summarizes state regulations on PFAS in drinking water, which are current as of May 2023. The state PFAS regulation summary in Table 4-17 is reflective of only those states that have promulgated PFAS drinking water regulations and does not include information from states that have proposed PFAS drinking water regulations or issued guidance for PWSs.

Table 4-17: State PFAS Regulations

State	Regulated PFAS Levels (ppt)									
	PFOA	PFOS	PFBS	PFHpA	PFHxA	PFHxS	PFNA	PFDA	HFPO-DA	Sum
New Jersey	14	13					13			
Vermont[a]	*	*		*		*	*			20
New Hampshire	12	15				18	11			
Massachusetts[a]	*	*		*		*	*	*		20
Michigan	8	16	420		400,000	51	6		370	
New York	10	10								
Pennsylvania	14	18								
Wisconsin	70	70								
Rhode Island[a]	*	*		*		*	*	*		20

Abbreviations: PFAS – per-and polyfluoroalkyl substances.
Notes:
[a] Asterisks (*) indicate states that regulate PFAS compounds at an overall threshold value, as indicated in the Sum column.
Sources: State websites are as follows – New Jersey (https://www.nj.gov/health/ceohs/documents/pfas_drinking%20water.pdf), Vermont (https://dec.vermont.gov/water/drinking-water/water-quality-monitoring/pfas), New Hampshire (https://www.nhwwa.org/wp-content/uploads/NHWWA-Water-is-Essential-Seminar-Oct-20-2020-PFAS-Arsenic-Rule-Updates.pdf), Massachusetts (https://www.mass.gov/lists/development-of-a-pfas-drinking-water-standard-mcl#final-pfas-mcl-regulations-), Michigan (https://www.michigan.gov/pfasresponse/drinking-water/mcl), New York (https://www.health.ny.gov/environmental/water/drinking/docs/water_supplier_fact_sheet_new_mcls.pdf).

To estimate the costs and benefits of the final rule, the EPA assumed that all MCMC occurrence model estimates exceeding state limits are equivalent to the state-enacted limit. For these states, the EPA assumed that the state MCL is the maximum baseline PFAS occurrence value for all EPs in the state. This adjustment was made to the MCMC occurrence model PFAS estimates for PFOA, PFOS, and PFHxS in this EA. In the three states where PFAS is regulated at a combined threshold level (Vermont, Massachusetts, and Rhode Island), the EPA did not make any adjustment to the estimated PFAS occurrence values from the MCMC model. Since the final rule standards are more stringent than current state drinking water standards, systems in states with PFAS regulations are still expected to incur incremental costs to comply with the final rule, although the estimated compliance costs will be less compared to costs that do not adjust the MCMC occurrence data to reflect the state MCLs. Similarly, populations served by PWSs in the states with PFAS regulations are expected to benefit from further reductions in PFAS exposures, although the incremental benefits for these populations will be less compared to benefits that do not adjust the MCMC occurrence data to reflect the state MCLs.

The EPA used system-level distributions, as described in Cadwallader et al. (2022), to simulate EP concentrations and estimate PFAS occurrence relative to the regulatory alternatives and final rule limits. The EPA assumed EP concentrations were constant. Simulated sample data are composed of a set of 4,000 iterations with the number of simulated samples per system within each iteration equal to the number of EPs. The EPA estimated within system variation from all available samples within each system as part of the model fitting process. Although the data used to fit the model may have included multiple samples over time or EPs, this simulation strategy assumes that all within-system variability is across EPs.

For 4,920 systems with means fitted by the model (i.e., systems with PFAS data in UCMR 3), the EPA simulated system-specific samples based on the best-fit model. The EPA simulated from the high level multivariate normal distribution to produce means for each chemical at each non-UCMR system and then used those distributions to simulate system-specific samples. The agency then generated random samples from the multivariate distribution and the value of the fixed parameters for each iteration. The exception to this approach was systems serving more than 1 million people. For these systems, the EPA used UCMR 3 and more recent monitoring data to identify the EPs that might require PFAS removal. These relatively few very large systems have the potential to affect aggregate costs and, therefore, require more precision in baseline occurrence estimates.

4.4.5 Summary of National PFAS Occurrence

Using the MCMC occurrence model, the EPA estimated baseline occurrence to understand changes in occurrence and exposure for the final rule and the regulatory alternative MCLs under Options 1a – 1c. These estimates vary across the 4,000 MCMC occurrence model iterations, thereby characterizing baseline occurrence uncertainty. In addition, for PWSs in states with existing MCLs for PFOA, PFOS, and PFHxS, the EPA capped contaminant concentrations at the state MCLs. The EPA notes that the baseline occurrence estimates presented herein differ from those presented under the proposed rule, which is due to the EPA's incorporation of additional state data for the final rule. Additionally, the final rule requirements for the number of significant digits used to assess compliance also impacts the baseline occurrence estimates.

The estimated number of PWSs with at least one EP above the PFHxS MCL and, by definition the PFHxS HBWC are provided in Table 4-18 through Table 4-21, while the total estimated number of EPs above the MCLs are provided in Table 4-22 through Table 4-25. In Table 4-26 through Table 4-29, the EPA provides the population served by PWSs with at least one EP above the MCLs. The population served by EPs above the MCLs are provided in Table 4-30 through Table 4-33. Each table provides expected value estimates as well as 5th percentile and 95th percentile estimates that characterize the uncertainty of baseline PFAS occurrence.

Table 4-18: Total Systems Impacted, Final Rule (PFOA and PFOS MCLs of 4.0 ppt each, PFHxS, PFNA, HFPO-DA MCLs of 10 ppt each and HI of 1)

	5th Percentile	Mean	95th Percentile
Small Systems			
Total Number of PWSs	62,048	62,048	62,048
PWSs With PFOS Exceedance	1,929	2,854	3,942
PWSs With PFOA Exceedance	1,903	2,759	3,791
PWSs With PFHxS MCL and/or PFHxS HBWC Exceedance[a,b]	51	110	194
PWSs That Exceed One or More MCLs	2,797	3,872	5,217
Large Systems			
Total Number of PWSs	4,482	4,482	4,482
PWSs With PFOS Exceedance	912	969	1,025
PWSs With PFOA Exceedance	992	1,049	1,107
PWSs With PFHxS MCL and/or PFHxS HBWC Exceedance [a,b]	92	105	120
PWSs That Exceed One or More MCLs	1,207	1,266	1,328
All Systems			
Total Number of PWSs	66,530	66,530	66,530
PWSs With PFOS Exceedance	2,874	3,823	4,958
PWSs With PFOA Exceedance	2,924	3,808	4,825
PWSs With PFHxS MCL and/or PFHxS HBWC Exceedance [a,b]	154	215	297
PWSs That Exceed One or More Limits	4,023	5,139	6,427

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; PWS – public water system; MCL – maximum contaminant level; PFHxS - perfluorohexane sulfonate; HI – hazard index; HBWC - Health Based Water Concentration.

Note: Detail may not add exactly to total due to independent rounding.

[a] The national level exceedance estimates for PFHxS are reflective of both the total national PFHxS individual MCL exceedances and HI MCL exceedances where PFHxS is present above its HBWC while one or more other HI PFAS is also present in that same mixture. Total national exceedance values do not include the exceedances associated with the co-occurrence of HFPO-DA, PFBS, and PFNA. EPA has considered the additional HI and individual MCLs for PFNA and HFPO-DA exceedances associated with occurrence of HFPO-DA, PFBS, and PFNA in a quantified sensitivity analysis; see Appendix N, Section N.3 for the analysis and Section XII.A.4 of the final rule preamble for more information about how the EPA considered HI, PFNA, and HFPO-DA MCL costs.

[b] Exceedance of both the PFHxS MCL as well as the PFHxS HBWC is triggered by PFHxS occurrence estimates above 10 ppt from the MCMC occurrence model.

Table 4-19: Total Systems Impacted, Option 1a (PFOA and PFOS MCLs of 4.0 ppt)

	5th Percentile	Mean	95th Percentile
Small Systems			
Total Number of PWSs	62,048	62,048	62,048
PWSs With PFOS Exceedance	1,935	2,854	3,972
PWSs With PFOA Exceedance	1,903	2,759	3,800
PWSs That Exceed One or More MCLs	2,795	3,870	5,097
Large Systems			
Total Number of PWSs	4,482	4,482	4,482
PWSs With PFOS Exceedance	916	969	1,026
PWSs With PFOA Exceedance	987	1,049	1,109
PWSs That Exceed One or More MCLs	1,203	1,266	1,328
All Systems			
Total Number of PWSs	66,530	66,530	66,530
PWSs With PFOS Exceedance	2,875	3,823	4,952
PWSs With PFOA Exceedance	2,930	3,808	4,828
PWSs That Exceed One or More MCLs	4,018	5,136	6,441

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; PWS – public water system; MCL – maximum contaminant level.
Note: Detail may not add exactly to total due to independent rounding.

Table 4-20: Total Systems Impacted, Option 1b (PFOA and PFOS MCLs of 5.0 ppt)

	5th Percentile	Mean	95th Percentile
Small Systems			
Total Number of PWSs	62,048	62,048	62,048
PWSs With PFOS Exceedance	1,336	2,075	2,932
PWSs With PFOA Exceedance	1,217	1,867	2,636
PWSs That Exceed One or More MCLs	1,936	2,768	3,733
Large Systems			
Total Number of PWSs	4,482	4,482	4,482
PWSs With PFOS Exceedance	741	791	841
PWSs With PFOA Exceedance	779	827	877
PWSs That Exceed One or More MCLs	981	1,033	1,084
All Systems			
Total Number of PWSs	66,530	66,530	66,530
PWSs With PFOS Exceedance	2,142	2,865	3,753
PWSs With PFOA Exceedance	2,058	2,693	3,443
PWSs That Exceed One or More MCLs	2,945	3,801	4,809

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; PWS – public water system; MCL – maximum contaminant level.
Note: Detail may not add exactly to total due to independent rounding.

Table 4-21: Total Systems Impacted, Option 1c (PFOA and PFOS MCLs of 10.0 ppt)

	5th Percentile	Mean	95th Percentile
Small Systems			
Total Number of PWSs	62,048	62,048	62,048
PWSs With PFOS Exceedance	391	648	987
PWSs With PFOA Exceedance	250	421	645
PWSs That Exceed One or More MCLs	505	806	1,188
Large Systems			
Total Number of PWSs	4,482	4,482	4,482
PWSs With PFOS Exceedance	338	366	395
PWSs With PFOA Exceedance	300	323	347
PWSs That Exceed One or More MCLs	444	473	503
All Systems			
Total Number of PWSs	66,530	66,530	66,530
PWSs With PFOS Exceedance	750	1,014	1,348
PWSs With PFOA Exceedance	570	744	958
PWSs That Exceed One or More MCLs	977	1,279	1,658

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; PWS – public water system; MCL – maximum contaminant level.
Note: Detail may not add exactly to total due to independent rounding.

Table 4-22: Total Entry Points Impacted, Final Rule (PFOA and PFOS MCLs of 4.0 ppt each, PFHxS, PFNA, HFPO-DA MCLs of 10 ppt each and HI of 1)

	5th Percentile	Mean	95th Percentile
Small Systems			
Total Number of Entry Points	88,938	88,938	88,938
Entry Points With PFOS Exceedance	2,455	3,623	5,006
Entry Points With PFOA Exceedance	2,368	3,448	4,706
Entry Points With PFHxS MCL and/or PFHxS HBWC exceedance[a,b]	59	126	219
Entry Points That Exceed One or More MCLs	3,672	5,122	6,884
Large Systems			
Total Number of Entry Points	23,264	23,264	23,264
Entry Points With PFOS Exceedance	2,306	2,438	2,572
Entry Points With PFOA Exceedance	2,388	2,518	2,651
Entry Points With PFHxS MCL and/or PFHxS HBWC exceedance [a,b]	273	298	327
Entry Points That Exceed One or More MCLs	3,742	3,921	4,086
All Systems			
Total Number of Entry Points	112,202	112,202	112,202
Entry Points With PFOS Exceedance	4,852	6,061	7,520
Entry Points With PFOA Exceedance	4,856	5,966	7,248
Entry Points With PFHxS MCL and/or PFHxS HBWC exceedance [a,b]	349	425	524
Entry Points That Exceed One or More MCLs	7,546	9,043	10,759

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; PWS – public water system; MCL – maximum contaminant level; PFHxS - perfluorohexane sulfonate; HI – hazard index; HBWC - health based water concentration.

Note: Detail may not add exactly to total due to independent rounding.

[a] The national level exceedance estimates for PFHxS are reflective of both the total national PFHxS individual MCL exceedances and HI MCL exceedances where PFHxS is present above its HBWC while one or more other HI PFAS is also present in that same mixture. Total national exceedance values do not include the exceedances associated with the co-occurrence of HFPO-DA, PFBS, and PFNA. EPA has considered the additional HI and individual MCLs for PFNA and HFPO-DA exceedances associated with occurrence of HFPO-DA, PFBS, and PFNA in a quantified sensitivity analysis; see Appendix N, Section N.3 for the analysis and Section XII.A.4 of the final rule preamble for more information about how the EPA considered HI, PFNA, and HFPO-DA MCL costs.

[b] Exceedance of both the PFHxS MCL as well as the HBWC is triggered by PFHxS occurrence estimates above 10 ppt from the MCMC occurrence model.

Table 4-23: Total Entry Points Impacted, Option 1a (PFOA and PFOS MCLs of 4.0 ppt)

	5th Percentile	Mean	95th Percentile
Small Systems			
Total Number of Entry Points	88,938	88,938	88,938
Entry Points With PFOS Exceedance	2,456	3,623	5,007
Entry Points With PFOA Exceedance	2,368	3,448	4,709
Entry Points That Exceed One or More MCLs	3,666	5,115	6,858
Large Systems			
Total Number of Entry Points	23,264	23,264	23,264
Entry Points With PFOS Exceedance	2,305	2,438	2,572
Entry Points With PFOA Exceedance	2,386	2,518	2,651
Entry Points That Exceed One or More MCLs	3,701	3,878	4,056
All Systems			
Total Number of Entry Points	112,202	112,202	112,202
Entry Points With PFOS Exceedance	4,853	6,061	7,511
Entry Points With PFOA Exceedance	4,862	5,966	7,247
Entry Points That Exceed One or More MCLs	7,497	8,993	10,711

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; PWS – public water system; MCL – maximum contaminant level.

Note: Detail may not add exactly to total due to independent rounding.

Table 4-24: Total Entry Points Impacted, Option 1b (PFOA and PFOS MCLs of 5.0 ppt)

	5th Percentile	Mean	95th Percentile
Small Systems			
Total Number of Entry Points	88,938	88,938	88,938
Entry Points With PFOS Exceedance	1,735	2,620	3,794
Entry Points With PFOA Exceedance	1,532	2,321	3,234
Entry Points That Exceed One or More MCLs	2,567	3,643	4,967
Large Systems			
Total Number of Entry Points	23,264	23,264	23,264
Entry Points With PFOS Exceedance	1,821	1,928	2,043
Entry Points With PFOA Exceedance	1,784	1,884	1,982
Entry Points That Exceed One or More MCLs	2,900	3,038	3,185
All Systems			
Total Number of Entry Points	112,202	112,202	112,202
Entry Points With PFOS Exceedance	3,627	4,548	5,661
Entry Points With PFOA Exceedance	3,399	4,204	5,135
Entry Points That Exceed One or More MCLs	5,550	6,682	8,007

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; PWS – public water system; MCL – maximum contaminant level.

Note: Detail may not add exactly to total due to independent rounding.

Table 4-25: Total Entry Points Impacted, Option 1c (PFOA and PFOS MCLs of 10.0 ppt)

	5th Percentile	Mean	95th Percentile
Small Systems			
Total Number of Entry Points	88,938	88,938	88,938
Entry Points With PFOS Exceedance	475	809	1,221
Entry Points With PFOA Exceedance	308	520	780
Entry Points That Exceed One or More MCLs	676	1,051	1,547
Large Systems			
Total Number of Entry Points	23,264	23,264	23,264
Entry Points With PFOS Exceedance	787	842	903
Entry Points With PFOA Exceedance	609	649	693
Entry Points That Exceed One or More MCLs	1,177	1,244	1,312
All Systems			
Total Number of Entry Points	112,202	112,202	112,202
Entry Points With PFOS Exceedance	1,320	1,651	2,069
Entry Points With PFOA Exceedance	955	1,170	1,435
Entry Points That Exceed One or More MCLs	1,900	2,295	2,780

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; PWS – public water system; MCL – maximum contaminant level.

Note: Detail may not add exactly to total due to independent rounding.

Table 4-26: Total Population at PWSs Impacted, Final Rule (PFOA and PFOS MCLs of 4.0 ppt each, PFHxS, PFNA, HFPO-DA MCLs of 10 ppt each and HI of 1)

	5th Percentile	Mean	95th Percentile
Small Systems			
Total Population	58,607,697	58,607,697	58,607,697
Population Impacted by PFOS Exceedance	2,240,600	3,286,600	4,520,200
Population Impacted by PFOA Exceedance	2,362,000	3,309,200	4,393,900
Population Impacted by PFHxS MCL and/or PFHxS HBWC Exceedance[a,b]	83,044	177,250	296,240
Population Impacted by One or More MCL Exceedances	3,314,000	4,494,200	5,848,200
Large Systems			
Total Population	263,679,547	263,679,547	263,679,547
Population Impacted by PFOS Exceedance	51,819,000	56,096,000	60,482,000
Population Impacted by PFOA Exceedance	55,099,000	59,554,000	64,109,000
Population Impacted by PFHxS MCL and/or PFHxS HBWC Exceedance [a,b]	6,372,000	7,499,900	8,864,500
Population Impacted by One or More MCL Exceedances	67,160,000	71,789,000	76,869,000
All Systems			
Total Population	322,287,244	322,287,244	322,287,244
Population Impacted by PFOS Exceedance	54,945,000	59,383,000	64,025,000
Population Impacted by PFOA Exceedance	58,326,000	62,863,000	67,423,000
Population Impacted by PFHxS MCL and/or PFHxS HBWC Exceedance [a,b]	6,508,600	7,677,100	9,025,300
Population Impacted by One or More MCL Exceedances	71,354,000	76,283,000	81,397,000

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; PWS – public water system; MCL – maximum contaminant level; PFHxS - perfluorohexane sulfonate; HI – hazard index; HBWC - Heath Based Water Concentration.

Notes: Detail may not add exactly to total due to independent rounding.

[a]The national level exceedance estimates for PFHxS are reflective of both the total national PFHxS individual MCL exceedances and HI MCL exceedances where PFHxS is present above its HBWC while one or more other HI PFAS is also present in that same mixture. Total national exceedance values do not include the exceedances associated with the co-occurrence of HFPO-DA, PFBS, and PFNA. EPA has considered the additional HI and individual MCLs for PFNA and HFPO-DA exceedances associated with occurrence of HFPO-DA, PFBS, and PFNA in a quantified sensitivity analysis; see Appendix N, Section N.3 for the analysis and Section XII.A.4 of the final rule preamble for more information about how the EPA considered HI, PFNA, and HFPO-DA MCL costs.

[b]Exceedance of both the PFHxS MCL as well as the HI is triggered by PFHxS occurrence estimates above 10 ppt from MCMC occurrence model.

Table 4-27: Total Population at PWSs Impacted, Option 1a (PFOA and PFOS MCLs of 4.0 ppt)

	5th Percentile	Mean	95th Percentile
Small Systems			
Total Population	58,607,697	58,607,697	58,607,697
Population Impacted by PFOS Exceedance	2,268,500	3,286,700	4,520,100
Population Impacted by PFOA Exceedance	2,342,800	3,309,200	4,372,800
Population Impacted by One or More MCL Exceedances	3,176,300	4,489,900	5,816,300
Large Systems			
Total Population	263,679,547	263,679,547	263,679,547
Population Impacted by PFOS Exceedance	51,819,000	56,098,000	60,417,000
Population Impacted by PFOA Exceedance	55,205,000	59,554,000	64,109,000
Population Impacted by One or More MCL Exceedances	66,940,000	71,747,000	76,805,000
All Systems			
Total Population	322,287,244	322,287,244	322,287,244
Population Impacted by PFOS Exceedance	54,951,000	59,385,000	63,997,000
Population Impacted by PFOA Exceedance	58,313,000	62,863,000	67,420,000
Population Impacted by One or More MCL Exceedances	71,316,000	76,237,000	81,338,000

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; PWS – public water system; MCL – maximum contaminant level.
Note: Detail may not add exactly to total due to independent rounding.

Table 4-28: Total Population at PWSs Impacted, Option 1b (PFOA and PFOS MCLs of 5.0 ppt)

	5th Percentile	Mean	95th Percentile
Small Systems			
Total Population	58,607,697	58,607,697	58,607,697
Population Impacted by PFOS Exceedance	1,616,400	2,422,200	3,346,900
Population Impacted by PFOA Exceedance	1,557,200	2,294,100	3,119,500
Population Impacted by One or More MCL Exceedances	2,360,900	3,270,600	4,284,100
Large Systems			
Total Population	263,679,547	263,679,547	263,679,547
Population Impacted by PFOS Exceedance	42,546,000	46,436,000	50,371,000
Population Impacted by PFOA Exceedance	44,201,000	47,952,000	51,786,000
Population Impacted by One or More MCL Exceedances	55,498,000	59,542,000	64,103,000
All Systems			
Total Population	322,287,244	322,287,244	322,287,244
Population Impacted by PFOS Exceedance	44,997,000	48,858,000	52,916,000
Population Impacted by PFOA Exceedance	46,406,000	50,246,000	54,145,000
Population Impacted by One or More MCL Exceedances	58,436,000	62,812,000	67,277,000

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; PWS – public water system; MCL – maximum contaminant level.
Note: Detail may not add exactly to total due to independent rounding.

Table 4-29: Total Population at PWSs Impacted, Option 1c (PFOA and PFOS MCLs of 10.0 ppt)

	5th Percentile	Mean	95th Percentile
Small Systems			
Total Population	58,607,697	58,607,697	58,607,697
Population Impacted by PFOS Exceedance	494,310	792,790	1,154,300
Population Impacted by PFOA Exceedance	345,510	566,290	841,210
Population Impacted by One or More MCL Exceedances	663,970	1,009,300	1,428,300
Large Systems			
Total Population	263,679,547	263,679,547	263,679,547
Population Impacted by PFOS Exceedance	19,723,000	22,216,000	24,811,000
Population Impacted by PFOA Exceedance	18,531,000	20,713,000	23,109,000
Population Impacted by One or More MCL Exceedances	26,477,000	29,287,000	32,179,000
All Systems			
Total Population	322,287,244	322,287,244	322,287,244
Population Impacted by PFOS Exceedance	20,510,000	23,009,000	25,642,000
Population Impacted by PFOA Exceedance	19,034,000	21,280,000	23,717,000
Population Impacted by One or More MCL Exceedances	27,545,000	30,296,000	33,118,000

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; PWS – public water system; MCL – maximum contaminant level.
Note: Detail may not add exactly to total due to independent rounding.

Table 4-30: Total Population at Entry Points Impacted, Final Rule (PFOA and PFOS MCLs of 4.0 ppt each, PFHxS, PFNA, HFPO-DA MCLs of 10 ppt each and HI of 1)

	5th Percentile	Mean	95th Percentile
Small Systems			
Total Population	58,607,697	58,607,697	58,607,697
Population Impacted by PFOS Exceedance	1,592,500	2,389,900	3,324,700
Population Impacted by PFOA Exceedance	1,553,100	2,282,900	3,157,000
Population Impacted by PFHxS MCL and/or Hazard Index Exceedance[a]	34,900	80,968	143,530
Population Impacted by One or More MCL Exceedances	2,423,800	3,394,500	4,582,500
Large Systems			
Total Population	263,679,547	263,679,547	263,679,547
Population Impacted by PFOS Exceedance	22,266,000	23,923,000	25,634,000
Population Impacted by PFOA Exceedance	24,109,000	25,766,000	27,448,000
Population Impacted by PFHxS MCL and/or Hazard Index Exceedance[a]	1,641,800	1,953,000	2,316,400
Population Impacted by One or More MCL Exceedances	35,505,000	37,817,000	40,155,000
All Systems			
Total Population	322,287,244	322,287,244	322,287,244
Population Impacted by PFOS Exceedance	24,476,000	26,313,000	28,238,000
Population Impacted by PFOA Exceedance	26,227,000	28,049,000	29,959,000
Population Impacted by PFHxS MCL and/or Hazard Index Exceedance[a]	1,723,000	2,034,000	2,388,100
Population Impacted by One or More MCL Exceedances	38,658,000	41,212,000	43,817,000

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; MCL – maximum contaminant level; PFHxS - perfluorohexane sulfonate; HI – hazard index.

Notes: Detail may not add exactly to total due to independent rounding.

[a] Exceedance of both the PFHxS MCL as well as the HI is triggered by PFHxS occurrence estimates above 10 ppt from the MCMC occurrence model.

Table 4-31: Total Population at Entry Points Impacted, Option 1a (PFOA and PFOS MCLs of 4.0 ppt)

	5th Percentile	Mean	95th Percentile
Small Systems			
Total Population	58,607,697	58,607,697	58,607,697
Population Impacted by PFOS Exceedance	1,595,900	2,390,000	3,320,100
Population Impacted by PFOA Exceedance	1,553,000	2,282,900	3,157,400
Population Impacted by One or More MCL Exceedances	2,422,500	3,389,700	4,576,500
Large Systems			
Total Population	263,679,547	263,679,547	263,679,547
Population Impacted by PFOS Exceedance	22,295,000	23,923,000	25,634,000
Population Impacted by PFOA Exceedance	24,014,000	25,765,000	27,504,000
Population Impacted by One or More MCL Exceedances	35,131,000	37,547,000	39,930,000
All Systems			
Total Population	322,287,244	322,287,244	322,287,244
Population Impacted by PFOS Exceedance	24,482,000	26,313,000	28,242,000
Population Impacted by PFOA Exceedance	26,221,000	28,048,000	29,959,000
Population Impacted by One or More MCL Exceedances	38,390,000	40,937,000	43,524,000

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; MCL – maximum contaminant level.

Note: Detail may not add exactly to total due to independent rounding.

Table 4-32: Total Population at Entry Points Impacted, Option 1b (PFOA and PFOS MCLs of 5.0 ppt)

	5th Percentile	Mean	95th Percentile
Small Systems			
Total Population	58,607,697	58,607,697	58,607,697
Population Impacted by PFOS Exceedance	1,128,900	1,725,500	2,455,000
Population Impacted by PFOA Exceedance	1,006,000	1,534,300	2,154,900
Population Impacted by One or More MCL Exceedances	1,661,300	2,411,500	3,279,500
Large Systems			
Total Population	263,679,547	263,679,547	263,679,547
Population Impacted by PFOS Exceedance	17,664,000	19,054,000	20,404,000
Population Impacted by PFOA Exceedance	17,229,000	18,563,000	19,877,000
Population Impacted by One or More MCL Exceedances	27,557,000	29,479,000	31,476,000
All Systems			
Total Population	322,287,244	322,287,244	322,287,244
Population Impacted by PFOS Exceedance	19,282,000	20,780,000	22,362,000
Population Impacted by PFOA Exceedance	18,650,000	20,097,000	21,605,000
Population Impacted by One or More MCL Exceedances	29,830,000	31,890,000	34,032,000

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; MCL – maximum contaminant level.
Note: Detail may not add exactly to total due to independent rounding.

Table 4-33: Total Population at Entry Points Impacted, Option 1c (PFOA and PFOS MCLs of 10.0 ppt)

	5th Percentile	Mean	95th Percentile
Small Systems			
Total Population	58,607,697	58,607,697	58,607,697
Population Impacted by PFOS Exceedance	315,170	531,480	797,030
Population Impacted by PFOA Exceedance	195,280	341,450	528,460
Population Impacted by One or More MCL Exceedances	434,870	691,810	1,007,800
Large Systems			
Total Population	263,679,547	263,679,547	263,679,547
Population Impacted by PFOS Exceedance	8,341,500	9,048,100	9,820,200
Population Impacted by PFOA Exceedance	5,758,200	6,399,500	7,097,400
Population Impacted by One or More MCL Exceedances	11,901,000	12,819,000	13,810,000
All Systems			
Total Population	322,287,244	322,287,244	322,287,244
Population Impacted by PFOS Exceedance	8,850,300	9,579,600	10,391,000
Population Impacted by PFOA Exceedance	6,089,500	6,741,000	7,435,600
Population Impacted by One or More MCL Exceedances	12,555,000	13,511,000	14,539,000

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; PWS – public water system; MCL – maximum contaminant level.
Note: Detail may not add exactly to total due to independent rounding.

4.5 Uncertainties in the Baseline and Compliance Characteristics of Systems

This section summarizes limitations and uncertainties of the baseline analysis. In the chapter, the EPA described how the quantitative analysis incorporates some sources of uncertainty. The agency also noted data limitations that introduce uncertainty because information is not available for the baseline analysis. Table 4-34 provides a summary of sources that have quantifiable uncertainty and data limitations.

The EPA notes that in most cases it is not possible to determine the extent to which a particular limitation or uncertainty can affect the magnitude of the baseline conditions. The EPA notes the potential direction of the impact on baseline inputs to the costs and/or benefits analysis when possible, but the agency does not prioritize the entries with respect to the impact magnitude.

Table 4-34: Limitations and Uncertainties that Apply to the Baseline Characteristics of Systems for the Final PFAS Rule

Uncertainty/ Assumption	Effect on Quantitative Analysis	Notes
The agency assigned ground water as the source to systems missing source water information.	Underestimate costs	The design and average flow equations for ground water systems result in lower flow estimates than the equations for surface water systems. If any of the systems assigned ground water source are in fact surface water systems, then the flow estimates used in the cost analysis will be underestimated. In addition, initial monitoring costs will be underestimated for small surface water systems that are assigned as a ground water source.
SDWIS/Fed retail populations used for baseline analysis	Overestimate costs	The EPA did not reallocate populations for purchased water systems to the wholesale suppliers. All systems are in the inventory with their respective retail populations. In general, this will result in extra systems with small populations in the analysis and smaller populations at the wholesale systems. Both results will tend to increase cost estimate because the cost curves reflect economies of scale.
SDWIS/Fed data quality	Uncertain impact on baseline number of systems and EPs	The EPA periodically reviews inventory information in SDWIS/Fed (U.S. EPA, 2021h) and has generally found a high level of completeness and accuracy. There is uncertainty, however, in some of the population and facility data reported per system. To address this, the EPA removed any CWS wholesaler serving fewer than 25 people from the analysis and assumed any remaining CWSs had a minimum possible population of 25. The EPA also assumed any non-wholesale NTNCWSs had a minimum possible population of 25. The maximum number of EPs per system was limited to the maximum number found for the equivalent system size and source water combination in the UCMR 3 data.
Flow relationships for CWS	Uncertain impact on flow inputs to cost analysis	The equations used to estimate design and average daily flow based on service population may over- or underestimate actual system flows. In general, average per capita household water consumption has declined since the source data were collected because of increased water efficiency.[a] The change in nonresidential consumption is unknown.
CWS flow curves applied to NTNCWS	Uncertain impact on flow inputs to cost analysis	The EPA applied the CWS population-flow equations to NTNCWSs. This approach may result in an over- or underestimate of flow, and therefore cost for NTNCWSs.
Uniform EP population distribution	Uncertain impact on flow inputs to cost analysis and population inputs to benefits analysis	The EPA assumed a uniform distribution of system population across system EPs. Actual EP population may be greater or lower than the modeled estimates.
System wage rates are based on old survey data	Uncertain impact on cost analysis	National average wage rates are based on CWSS data finalized in 2006. The EPA escalated the values to $2022 to reflect current national industry averages, but actual wage rates at affected systems may be greater or less than national averages.

Table 4-34: Limitations and Uncertainties that Apply to the Baseline Characteristics of Systems for the Final PFAS Rule

Uncertainty/ Assumption	Effect on Quantitative Analysis	Notes
Baseline occurrence based on MCMC occurrence model outputs	Uncertain effect on occurrence and exposure	The iterative MCMC approach (4,000 iterations) probabilistically estimates parameters for system-level distributions to capture uncertainty. The simulated EP concentrations then reflect the system-level distribution from which they are drawn across 4,000 iterations. Further details on the MCMC model are available in Cadwallader et al. (2022).
UCMR 3 data for PFBS and PFNA and no UCMR 3 data for HFPO-DA were available to incorporate into the Bayesian hierarchical occurrence model	Underestimate occurrence and exposure	Excluding occurrence estimates for PFNA, HFPO-DA, and PFBS underestimates the number of systems that would exceed the MCLs based on occurrence of these three compounds. Due to occurrence data limitations, cost estimates for PFNA, PFBS, and HFPO-DA are less precise relative to those for PFOA, PFOS, and PFHxS compounds, and as such, the EPA performed a quantitative sensitivity analysis of the national cost impacts associated with exceedances resulting from PFNA, PFBS, and HFPO-DA in Appendix N.3 to consider the potential magnitude of costs associated with treating these regulated PFAS.

Abbreviations: CWS – community water systems; CWSS– community water system survey; HI– hazard index; MCMC – Markov chain Monte Carlo; NTNCWS – non-transient, non-community water systems; PFAS – per- and polyfluoroalkyl substances; PFOA– perfluorooctanoic acid; PFOS– perfluorooctane sulfonate; SDWIS/Fed– safe drinking water information system federal version.

Note:

[a] There is uncertainty in using the equations from the EPA's *Geometries and Characteristics of Public Water Systems* report (U.S. EPA, 2000) to predict future average daily and design flow based on a system's retail population. Water use efficiency has increased substantially since the 1980s, with a major improvement between 2005 and 2010 (Rockaway et al., 2011). A 2016 Water Research Foundation study reported a 22 percent decline in indoor water use (Water Research Foundation, 2016). Several factors have contributed to increases in water efficiency. Technological changes, supported by policy, increased the efficiency of water use. For example, the Energy Policy Act of 1992 required water efficiency standards for fixtures, including shower heads, toilets, and washing machines. Water recycling and increased efficiency of power generation also reduces freshwater use. The economic downturn of 2008 contributed to the drop in water use and the increase in use of water-efficient fixtures and xeriscaping. Other demand-side management measures contributed to reduction in per capita use as well. The trend of lower residential water use could result in lower flow per population and lower treatment costs as compared to predicted values in this EA.

5 Cost Analysis

5.1 Introduction

In this chapter, the EPA presents its cost analysis for the final PFAS NPDWR (the final rule) and other alternative rule options considered by the agency as part of the rulemaking process (Options 1a through 1c). The contents include the national cost estimates for the final rule as well as options and the approach the EPA used to derive those estimates. The estimates include the cost that PWSs, households, and primacy agencies may incur in response to the final rule requirements.

5.1.1 Chapter Overview

This chapter has seven main sections including this introductory section. Section 5.2 provides an overview of the EPA's approach to estimate the cost of the final rule and options. In Section 5.3, the EPA provides the data and algorithms used to calculate the cost of activities PWSs will undertake to comply with the final rule. Section 5.4 provides the data and assumptions used to calculate the cost of activities primacy agencies will undertake to implement and administer the final rule. Sections 5.1.3, 5.5, and 5.6 provide the cost estimates at the national, PWS, and household level, respectively. As indicated below, some additional details on the approach and data used to calculate the costs of the final rule are in Appendix C.

5.1.2 Uncertainty Characterization

Many of the input values used to calculate the costs of drinking water regulations are not known with certainty. For example, estimated technology unit costs and contaminant occurrence values are uncertain to some degree given imperfect information. The EPA determined it does have enough information about the level or distribution of uncertainty to conduct a full Monte-Carlo based uncertainty analysis as part of the SafeWater Multi-Contaminant Benefit-Cost Model (MCBC). With respect to the cost analysis, the EPA modeled the sources of uncertainty summarized in Table 5-1.

Table 5-1: Quantified Sources of Uncertainty in Cost Estimates

Source	Description of Uncertainty
EP concentration of PFAS compounds	The concentration and co-occurrence at each PWS EP of each modeled compound is unknown. The cost analysis uses EP concentrations simulated with system level distributions produced by the Bayesian hierarchical MCMC occurrence model. The iterative MCMC approach (4,000 iterations) probabilistically estimates parameters for system-level distributions to capture uncertainty. The simulated EP concentrations then reflect the system-level distribution from which they are drawn across 4,000 iterations. Further details on the MCMC model are available in Cadwallader et al. (2022). For more information on the application of the model in this analysis, see Section 4.4 and Appendix A. For more information on the data and analyses that the EPA used to develop national estimates of PFAS occurrence in public drinking water systems see U.S. EPA (2024g).
TOC concentration	The TOC value assigned to each system is from a distribution derived from the fourth Six-Year Review Information Collection Request database (see Section 5.3.1.1)
Compliance technology unit cost curve selection	Cost curve selection varies with baseline PFAS concentrations and also includes a random selection from a distribution across feasible technologies (see Section 5.3.1.1), and a random selection from a triangular distribution of low-, mid-, and high-cost equipment (25%, 50%, and 25%, respectively).

Abbreviations: MCBC – Multi-Contaminant Benefit-Cost Model; PFAS – per- and polyfluoroalkyl substances; TOC – total organic carbon.

For each iteration, SafeWater MCBC assigned new values to the three sources of modeled uncertainty as described in Table 5-1, and then calculated costs for each of the model PWSs. This was repeated 4,000 times to reach an effective sample size for each parameter. At the end of the 4,000 iterations, SafeWater MCBC outputs the expected value as well as the 90 percent confidence interval for each cost metric (i.e., bounded by the 5th and 95th percentile estimates for each cost component). Detailed information on the data used to model uncertainty is provided in Appendix A and Appendix L.

5.1.3 Summary of Quantified National Cost Estimates of the Final Rule

In Table 5-2, the EPA summarizes the total annualized cost of the final rule at a 2 percent discount rate. The first three rows show the annualized PWS sampling costs, the annualized PWS implementation and administrative costs, and the annualized PWS treatment costs. The fourth row shows the sum of the annualized PWS costs. Expected annualized PWS costs are $1.54 billion. The quantified uncertainty range for annualized PWS costs is $1.43 billion to $1.67 billion. Finally, annualized primacy agency implementation and administrative costs are added to the annualized PWS costs to calculate the total annualized cost of the final rule. Expected total annualized cost of the final rule is $1.55 billion with an uncertainty range of $1.44 billion to $1.67 billion.

The difference in the costs between the final rule and Option 1a provides the marginal cost of the PFHxS standards. As shown in Table 4-18 and 4-22, the EPA estimates that 215 water systems (425 EPs) will exceed the PFHxS MCL of 10 ppt and by definition the HBWC of 10 ppt for the

HI.[15] Of the water systems estimated to exceed the PFHxS regulatory thresholds, many are also anticipated to exceed the PFOA and PFOS MCLs. The EPA estimates that 3 water systems with 50 EPs will be triggered into corrective action for PFHxS alone while 212 systems (375 EPs) will treat for PFHxS in addition to PFOA and/or PFOS, and the national annualized marginal costs of all PFHxS exceedances, including at systems with and without PFOA/PFOS exceedances, is $11.57 million dollars. This is the estimated contribution of costs from PFHxS to the overall costs of the rule, not in addition to the costs presented in Table 5-2. As discussed in U.S. EPA (2024g), PFHxS is observed to strongly cooccur with PFOA and PFOS; therefore, there are significantly more systems with PFHxS, PFOA, and PFOS present with two or more of these PFAS above their respective MCLs than systems with PFHxS above the MCL alone. Furthermore, this pattern is accentuated because the PFHxS MCL of 10 ppt is 2.5 times higher than either the PFOA or PFOS MCLs of 4.0 ppt. Additionally, since the PFHxS MCL is one significant figure, whereas PFOA and PFOS are two significant figures, for purposes of estimating compliance, PFHxS would not be deemed to be in exceedance until above 15 ppt. All told, this means that the PFHxS MCL (and its contributions to the HI) adds important public health protection for a modest additional cost.

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[15] Note that results above a single HBWC for a single PFAS does not constitute an HI exceedance (see Section V.B.III of the preamble for the final rule for more information).

Table 5-2: National Annualized Costs, Final Rule (PFOA and PFOS MCLs of 4.0 ppt each, PFHxS, PFNA, HFPO-DA MCLs of 10 ppt each and HI of 1) (Million $2022)

	2% Discount Rate		
	5th Percentile[a]	**Expected Value**	**95th Percentile[a]**
Annualized PWS Sampling Costs	$33.63	$36.23	$39.03
Annualized PWS Implementation and Administration Costs	$1.33	$1.33	$1.33
Annualized PWS Treatment Costs	$1,395.23	$1,506.44	$1,627.65
Total Annualized PWS Costs	**$1,431.00**	**$1,544.00**	**$1,667.10**
Primacy Agency Rule Implementation and Administration Cost	$4.35	$4.65	$4.97
Total Annualized Rule Costs[b,c,d]	**$1,435.70**	**$1,548.64**	**$1,672.10**

Abbreviations: PWS – public water system.

Notes: Detail may not add exactly to total due to independent rounding. See Appendix P, Section P.2 for results presented at 3 and 7 percent discount rates. 5th and 95th percentile values for total rule costs are not additive across cost category as the categories are not completely correlated.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty described in Section 5.1.2 and Table 5-1. This range does not include the uncertainty described in Table 5-21.

[b] See Table 7-6 for a list of the nonquantifiable costs, and the potential direction of impact these costs would have on the estimated monetized total annualized costs in this table.

[c] The national level cost estimates for PFHxS are reflective of both the total national cost for PFHxS individual MCL exceedances, and HI MCL exceedances where PFHxS is present above its HBWC while one or more other HI PFAS is also present in that same mixture. Total quantified national cost values do not include the incremental treatment costs associated with the co-occurrence of HFPO-DA, PFBS, and PFNA. EPA has considered the additional national costs of the HI and individual MCLs associated with HFPO-DA, PFNA, and PFBS occurrence in a quantified sensitivity analysis; See Appendix N, Section N.3 for the analysis and more information.

[d] PFAS-contaminated wastes are not considered RCRA regulatory or characteristic hazardous wastes at this time and therefore total costs reported in this table do not include costs associated with hazardous waste disposal of spent filtration materials. To address stakeholder concerns about potential costs for disposing PFAS-contaminated wastes as hazardous should they be regulated as such in the future, the EPA conducted a sensitivity analysis with an assumption of hazardous waste disposal for illustrative purposes only. See Appendix N, Section N.2 for additional detail.

In Table 5-3, Table 5-4, and Table 5-5 the EPA summarizes the total annualized cost of Options 1a, 1b, and 1c, respectively.

Table 5-3: National Annualized Costs, Option 1a (PFOA and PFOS MCLs of 4.0 ppt) (Million $2022)

	2% Discount Rate		
	5th Percentile[a]	**Expected Value**	**95th Percentile[a]**
Annualized PWS Sampling Costs	$33.37	$35.98	$38.77
Annualized PWS Implementation and Administration Costs	$1.33	$1.33	$1.33
Annualized PWS Treatment Costs	$1,383.33	$1,495.14	$1,616.15
Total Annualized PWS Costs	**$1,419.20**	**$1,532.44**	**$1,654.80**
Primacy Agency Rule Implementation and Administration Cost	$4.34	$4.63	$4.95
Total Annualized Rule Costs[b,c]	**$1,423.60**	**$1,537.07**	**$1,660.30**

Abbreviations: PWS – public water system.

Notes: Detail may not add exactly to total due to independent rounding. See Appendix P, Section P.2 for results presented at 3 and 7 percent discount rates. 5th and 95th percentile values for total rule costs are not additive across cost category as the categories are not completely correlated.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty described in Section 5.1.2 and Table 5-1. This range does not include the uncertainty described in Table 5-21.

[b] See Table 7-6 for a list of the nonquantifiable costs, and the potential direction of impact these costs would have on the estimated monetized total annualized costs in this table.

[c] PFAS-contaminated wastes are not considered RCRA regulatory or characteristic hazardous wastes at this time and therefore total costs reported in this table do not include costs associated with hazardous waste disposal of spent filtration materials. To address stakeholder concerns about potential costs for disposing PFAS-contaminated wastes as hazardous should they be regulated as such in the future, the EPA conducted a sensitivity analysis with an assumption of hazardous waste disposal for illustrative purposes only. See Appendix N, Section N.2 for additional detail.

Table 5-4: National Annualized Costs, Option 1b (PFOA and PFOS MCLs of 5.0 ppt) (Million $2022)

	2% Discount Rate		
	5th Percentile[a]	Expected Value	95th Percentile[a]
Annualized PWS Sampling Costs	$31.07	$33.29	$35.71
Annualized PWS Implementation and Administration Costs	$1.33	$1.33	$1.33
Annualized PWS Treatment Costs	$1,065.30	$1,153.31	$1,250.22
Total Annualized PWS Costs	**$1,098.40**	**$1,187.92**	**$1,286.50**
Primacy Agency Rule Implementation and Administration Cost	$3.98	$4.21	$4.47
Total Annualized Rule Costs[b,c]	**$1,102.60**	**$1,192.13**	**$1,291.40**

Abbreviations: PWS – public water system.

Notes: Detail may not add exactly to total due to independent rounding. See Appendix P, Section P.2 for results presented at 3 and 7 percent discount rates. 5th and 95th percentile values for total rule costs are not additive across cost category as the categories are not completely correlated.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty described in Section 5.1.2 and Table 5-1. This range does not include the uncertainty described in Table 5-21.

[b] See Table 7-6 for a list of the nonquantifiable costs, and the potential direction of impact these costs would have on the estimated monetized total annualized costs in this table.

[c] PFAS-contaminated wastes are not considered RCRA regulatory or characteristic hazardous wastes at this time and therefore total costs reported in this table do not include costs associated with hazardous waste disposal of spent filtration materials. To address stakeholder concerns about potential costs for disposing PFAS-contaminated wastes as hazardous should they be regulated as such in the future, the EPA conducted a sensitivity analysis with an assumption of hazardous waste disposal for illustrative purposes only. See Appendix N, Section N.2 for additional detail.

Table 5-5: National Annualized Costs, Option 1c (PFOA and PFOS MCLs of 10.0 ppt) (Million $2022)

	2% Discount Rate		
	5th Percentile[a]	**Expected Value**	**95th Percentile[a]**
Annualized PWS Sampling Costs	$26.11	$27.48	$28.97
Annualized PWS Implementation and Administration Costs	$1.33	$1.33	$1.33
Annualized PWS Treatment Costs	$431.37	$467.12	$507.50
Total Annualized PWS Costs	**$459.50**	**$495.93**	**$537.21**
Primacy Agency Rule Implementation and Administration Cost	$3.27	$3.37	$3.48
Total Annualized Rule Costs[b,c]	**$462.87**	**$499.29**	**$540.68**

Abbreviations: PWS – public water system.

Notes: Detail may not add exactly to total due to independent rounding. See Appendix P, Section P.2 for results presented at 3 and 7 percent discount rates. 5th and 95th percentile values for total rule costs are not additive across cost category as the categories are not completely correlated.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty described in Section 5.1.2 and Table 5-1. This range does not include the uncertainty described in Table 5-21.

[b] See Table 7-6 for a list of the nonquantifiable costs, and the potential direction of impact these costs would have on the estimated monetized total annualized costs in this table.

[c] PFAS-contaminated wastes are not considered RCRA regulatory or characteristic hazardous wastes at this time and therefore total costs reported in this table do not include costs associated with hazardous waste disposal of spent filtration materials. To address stakeholder concerns about potential costs for disposing PFAS-contaminated wastes as hazardous should they be regulated as such in the future, the EPA conducted a sensitivity analysis with an assumption of hazardous waste disposal for illustrative purposes only. See Appendix N, Section N.2 for additional detail.

5.2 Overview of SafeWater Multi-Contaminant Benefit Cost Model (MCBC)

The SafeWater Cost Benefit Model (SafeWater CBX) was designed to calculate the costs and benefits associated with setting a new or revised MCL. Since the final PFAS rule simultaneously regulates multiple PFAS contaminants, the EPA developed a new model version called the SafeWater MCBC to estimate the costs and benefits associated with regulating more than one contaminant. The following modifications were made to the SafeWater CBX model to create the SafeWater MCBC model:

1. Instead of tracking a single contaminant's level and comparing that to the MCL options to determine if the PWS must take compliance actions, SafeWater MCBC tracks each PWS's level of multiple PFAS contaminants and compares them against MCL options for each contaminant (or group of contaminants). The PWS will need to take corrective action if any of its EP's contaminant levels are above any of the MCLs. In this case the EP will incur treatment costs and will accrue health benefits.
2. The structure of the occurrence data input to the model was updated to not only handle multiple contaminants, but to incorporate all information from the PFAS occurrence model on the predicted co-occurrence of contaminants.
3. The model structure was also adjusted to allow for assignment of one or more compliance technologies that achieve all regulatory requirements and estimates costs and benefits

associated with multiple PFAS contaminant reductions and calculates before and after treatment concentrations of each contaminant for use in the estimation of benefits.

5.2.1 Modeling PWS Variability in SafeWater MCBC

The costs incurred by a PWS depend on water system characteristics. The data describing some of these characteristics for PWSs are in SDWIS/Fed. The SDWIS/Fed data provide information on the PWS characteristics that typically define PWS categories, or strata, for which the EPA develops costs in rulemakings:

- System type (CWS, NTNCWS);
- Number of people served by the PWS;
- PWS's primary raw water source (ground water or surface water);
- PWS's ownership type (public or private); and
- State in which PWS is located.

Because the EPA does not have complete PWS-specific data across the 49,193 CWSs and 17,337 NTNCWS in SDWIS/Fed for many of the baseline and compliance characteristics necessary to estimate costs and benefits, such as design and average daily flow rates, water quality characteristics, treatment in-place, and labor rates, the EPA adopted a "model PWS" approach. SafeWater MCBC creates model PWSs by combining the PWS-specific data available in SDWIS/Fed with data on baseline and compliance characteristics available at the PWS category level. In some cases, the categorical data are simple point estimates. In this case, every model PWS in a category is assigned the same value. In other cases, where more robust data representing system variability are available, the category-level data include a distribution of potential values. In the case of distributional information, SafeWater MCBC assigns each model PWS a value sampled from the distribution. These distributions are assumed to be independent. Table 5-6 provides a list of all the PWS characteristics that impact model PWS compliance costs. These data include inventory data specific to each system and categorical data for which randomly assigned values are based on distributions that vary by category (e.g., ground water and surface water TOC distributions or compliance forecast distributions that vary by system size category).

Table 5-6: Model PWS Variability Characteristics and Data Sources

PWS Characteristic	Data Type and Description
System Type	Known SDWIS/Fed Inventory
Primary Source Water	Known: SDWIS/Fed Inventory
Ownership	Known: SDWIS/Fed Inventory
Population Served	Known: SDWIS/Fed Inventory
Number of EPs	Known: UCMR 3, SDWIS/Fed Inventory, and modeled from SDWIS/Fed Inventory distribution (see Section 4.3.3.1)
PFAS Contaminant Concentration at each EP	Sampled from EPA Occurrence Model (see Section 4.3.3.2)
Influent TOC Level	Assigned from distribution derived from fourth Six-Year Review Information Collection Request database (see Section 5.3.1.1)
Compliance Technology Forecast at each EP	Assigned from distribution derived from full-scale compliance actions analyzed by the EPA (see Section 5.3.1.1)

Abbreviations: EPA – U.S. Environmental Protection Agency; PFAS – per-and polyfluoroalkyl substances; SDWIS/Fed – Safe Drinking Water Information System/Federal version; TOC – total organic carbon; UCMR 4 – Fourth Unregulated Contaminant Monitoring Rule.

As illustrated in Figure 5-1, once all the model PWSs are created and assigned baseline and compliance characteristics, SafeWater MCBC estimates the quantified costs and benefits of compliance for each model PWS under the final rule. Because of this model PWS approach, SafeWater MCBC does not output any results at the PWS-level. Instead, the outputs are cost and benefit estimates for 36 PWS categories, or strata. Each PWS category is defined by the system type (CWS and NTNCWS), primary water source (ground or surface), and size category (there are nine). Note the EPA does not report state specific strata although state location is utilized in the SafeWater MCBC model (e.g., current state level regulatory limits on PFAS in drinking water).

For each PWS category, the model then calculates summary statistics that describe the costs and quantified benefits associated with the final rule compliance. These summary statistics include total quantified costs of the final regulatory requirements, total quantified benefits of the final regulatory requirements, the variability in PWS-level costs (i.e., 10th, 25th, 50th, 75th and 90th percentile system costs), and the variability in household-level costs (i.e., 10th, 25th, 50th, 75th and 90th percentile household costs). In addition, SafeWater MCBC characterizes the uncertainty in the estimated costs and benefits by calculating the expected value and 90th percentile confidence interval (5th and 95th percentile values) for each output metric.

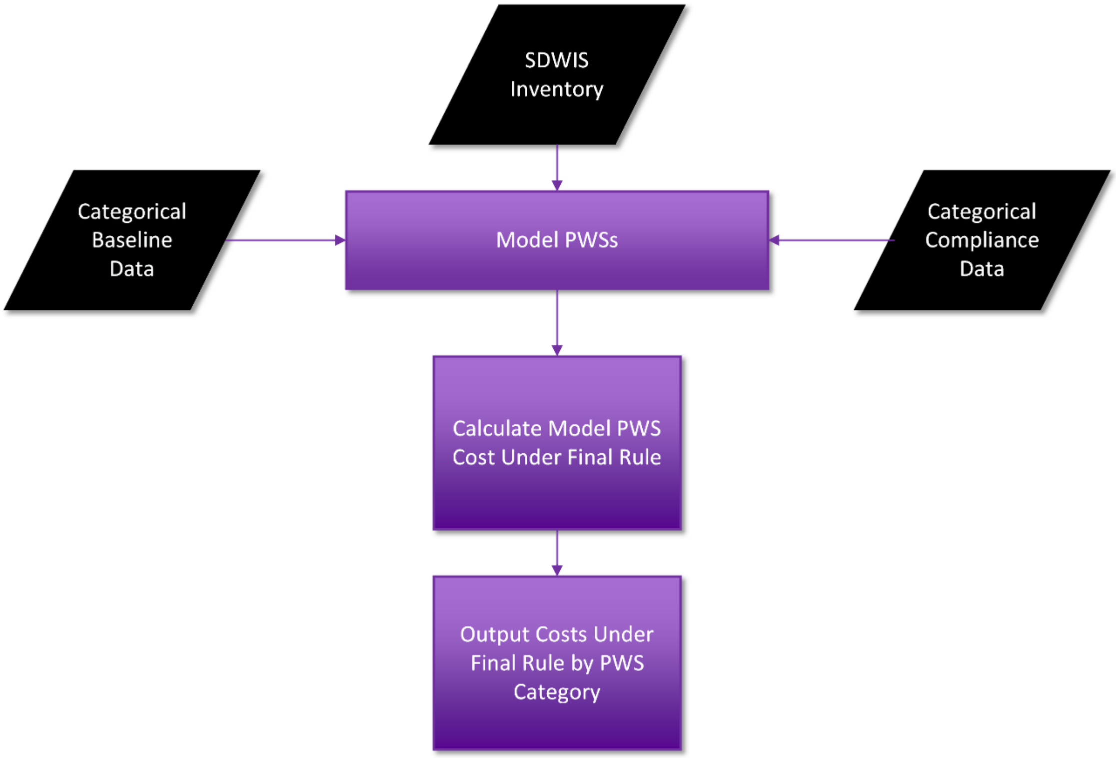

Figure 5-1: Approach Used by SafeWater MCBC to Model PWS Variability

5.3 Estimating Public Water System Costs

The EPA estimated PWS compliance activities that result in treatment costs and administrative and monitoring costs associated with the final rule. Each major regulatory component consists of required activities, which the EPA details here. The EPA presents the costs associated with treatment addition and nontreatment actions that could be taken in lieu of treatment in Section 5.3.1. The EPA presents the costs associated with the administrative and monitoring requirements of the final rule in Section 5.3.2.

5.3.1 PWS Treatment Costs

This section describes how the EPA estimated costs associated with:

- Engineering, installing, operating, and maintaining PFAS removal treatment technologies, including treatment media replacement and spent media destruction or disposal; and
- Nontreatment actions that some PWSs might take in lieu of treatment, such as constructing new wells in an uncontaminated aquifer or interconnecting with and purchasing water from a neighboring PWS.

The EPA used SafeWater MCBC to apply costs for one of these treatment technologies or nontreatment alternatives at each EP in a PWS estimated to be out of compliance with the regulatory option under consideration. First, for each affected EP, SafeWater MCBC selected from among the compliance alternatives using the decision tree procedure described in Section 5.3.1.1. Next, SafeWater MCBC estimated the cost of the chosen compliance alternative using inputs from the EPA's WBS cost estimating models. Specifically, SafeWater MCBC used cost equations generated from the following models:[16]

- The GAC WBS model;
- The PFAS-selective IX WBS model; and
- The nontreatment WBS model.

The national cost analysis reflects that PFAS-contaminated wastes are not considered Resource Conservation and Recovery Act (RCRA) regulatory or characteristic hazardous wastes. Additionally, this PFAS NPDWR does not require drinking water treatment residuals to be managed in any specific way. The EPA understands that the current practice for drinking water systems to manage their spent treatment media is generally to reactivate GAC and to dispose of ion exchange treatment residuals as non-hazardous waste. As shown below in Table 5-9, the EPA estimates that 52-89% of systems will use GAC and 11-48% of systems will use IX, depending on system size and water quality. The national cost analysis assumes the spent GAC media is reactivated off-site under current RCRA non-hazardous waste regulations. The WBS model uses a unit cost for reactivation that includes transportation to the reactivation facility and back to the treatment plant. To account for losses in the reactivation and replacement process, it also adds the cost of replacing 30 percent of the spent GAC with virgin media. The national cost analysis assumes the spent IX resin is incinerated off-site under current RCRA non-hazardous waste regulations. The WBS model uses a unit cost for non-hazardous incineration that includes transportation to the incineration facility. For purposes of the cost analysis, EPA does not assume any facilities will utilize Subtitle D Landfills. EPA notes that if the agency were to assume some or all facilities would utilize Subtitle D landfills to dispose of spent IX resin, estimated spent resin treatment residual disposal costs attributable to the PFAS NDPWR would have been lower. For more information on GAC and IX residuals management unit cost estimates for PFAS see Section 7.2 and 7.3 of the Technologies and Costs (T&C) document (U.S. EPA, 2024i).

The EPA proposed PFOA and PFOS be designated as Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) hazardous substances to require reporting of PFOA and PFOS releases, enhance the availability of data, and ensure agencies can recover cleanup costs (U.S. EPA, 2022). Stakeholders have expressed concern to the EPA that a hazardous substance designation for certain PFAS may limit their disposal options for drinking water treatment residuals (e.g., spent media, concentrated waste streams) and/or potentially increase costs. Designation of PFOA and PFOS as CERCLA hazardous substances would not require waste (e.g., biosolids, treatment residuals, etc.) to be treated in any particular fashion, nor disposed of at any specific particular type of landfill. The designation also would not restrict, change, or recommend any specific activity or type of waste at landfills. Although designating

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[16] At this time, the EPA is not including point-of-use (POU) devices in the national cost estimates because the final rule requires treatment to concentrations below the current NSF/ANSI certification standard for POU devices. However, POU treatment is reasonably anticipated to become a compliance option for small systems in the future if NSF/ANSI or other independent third-party certification organizations develop a new certification standard that mirrors the EPA's final regulatory standard. In the event POU treatment becomes a valid compliance option, national costs could be lower than estimated in this application of the SafeWater MCBC.

chemicals as hazardous substances under CERCLA would not result in new requirements for disposal of PFAS drinking water treatment residuals, to address stakeholder concerns, including those raised during the SBREFA process, the EPA conducted a sensitivity analysis with an assumption of hazardous waste disposal for illustrative purposes only. The EPA acknowledges that if in the future PFAS-contaminated wastes are required to be handled as hazardous wastes, the residuals management costs are expected to be higher. For a discussion of the findings from this sensitivity analysis, see Appendix N, Section N.2.

Section 5.3.1.2 describes the WBS models. Section 5.3.1.2.2 describes the form of the resulting cost equations and their application in SafeWater MCBC. The T&C document (U.S. EPA, 2024i) provides a comprehensive discussion of each of the treatment technologies, their effectiveness, and the WBS cost models. It also presents the cost equations themselves in tabular form. These models are available on the EPA's website at https://www.epa.gov/sdwa/drinking-water-treatment-technology-unit-cost-models as well as in the docket for this rulemaking.

5.3.1.1 Decision Tree for Technology Selection

For EPs at which baseline PFAS concentrations exceed regulatory thresholds, SafeWater MCBC selects a treatment technology or nontreatment alternative using a two-step process that:

1. Determines whether to include or exclude each alternative from consideration given the EP's characteristics and the regulatory option selected; and
2. Selects from among the alternatives that remain viable based on percentage distributions derived, in part, from data on recent PWS actions in response to PFAS contamination.

Inputs to SafeWater MCBC used in the Step 1 include the following:

- Influent concentrations of individual PFAS contaminants in ppt (ng/L);
- EP design flow in MGD; and
- TOC influent to the new treatment process in mg/L.

Section 4.4 describes the EPA's method for estimating PFAS influent concentrations and Section 4.3.3.3 describes how the EPA derived EP flow estimates. SafeWater MCBC selects influent TOC using the distribution shown in Table 5-7.

Table 5-7: Frequency Distribution to Estimate Influent TOC in mg/L

Percentile	Surface Water	Ground Water
0.05	0.65	0.35
0.15	1.1	0.48
0.25	1.38	0.5
0.35	1.6	0.5
0.45	1.85	0.58
0.5	1.97	0.69
0.55	2.14	0.75
0.65	2.54	1
0.75	3.04	1.39
0.85	3.63	2.01
0.95	4.81	3.8

Abbreviations: TOC – total organic carbon.
Source: The EPA's analysis of total organic carbon concentrations in the fourth Six-Year Review Information Collection Request database.

In Step 1, SafeWater MCBC uses these inputs to determine whether to include or exclude each treatment alternative from consideration in the compliance forecast. For the treatment technologies (GAC and IX), this determination is based on estimates of each technology's performance given available data about influent water quality and the regulatory option under consideration. Section 5.3.1.1.1 describes this process for GAC and IX.

The EPA assumes a small number of PWSs may be able to take nontreatment actions in lieu of treatment. The viability of nontreatment actions (interconnection with neighboring system or new wells) is likely to depend on the quantity of water being replaced. Therefore, SafeWater MCBC considers nontreatment only for EPs with design flows less than or equal to 3.536 MGD.

In Step 2, SafeWater MCBC selects a compliance alternative for each EP from among the alternatives that remain in consideration after Step 1. Table 5-8 shows the initial compliance forecast that is the starting point for this step. The percentages in Table 5-8 consider data presented in the T&C document (U.S. EPA, 2024i) on actions PWSs have taken in response to PFAS contamination.

Table 5-8: Initial Compliance Forecast Including POU RO

Compliance Alternative	Design Flow Less than 1 MGD		Design Flow 1 to Less than 10 MGD		Design Flow Greater than or Equal to 10 MGD	
	TOC Less than or Equal to 1.5 mg/L	TOC Greater than 1.5 mg/L	TOC Less than or Equal to 1.5 mg/L	TOC Greater than 1.5 mg/L	TOC Less than or Equal to 1.5 mg/L	TOC Greater than 1.5 mg/L
GAC	68%	53%	81%	52%	89%	52%
PFAS-selective IX	11%	26%	11%	40%	11%	48%
Central RO/NF	0%	0%	0%	0%	0%	0%
POU devices	13%	13%	0%	0%	0%	0%
Interconnection	6%	6%	6%	6%	0%	0%
New Wells	2%	2%	2%	2%	0%	0%

Abbreviations: GAC – granular activated carbon; PFAS – per-and polyfluoroalkyl substances; MGD – million gallons per day; IX – ion exchange; RO/NF – reverse osmosis/nanofiltration; POU – point-of-use; TOC – total organic carbon.
Source: The EPA's analysis of total organic carbon concentrations in the fourth Six-Year Review Information Collection Request database.

To date, the majority of PWSs for which data are available have installed GAC (U.S. EPA, 2024i). U.S. EPA (2024i) includes data for 52 systems, 34 of which (65%) have installed GAC. The first full-scale system treating drinking water using PFAS-selective IX began operation in 2017 (WWSD, 2018). The data in the T&C document (U.S. EPA, 2024i) also suggest that an increasing share of PWSs have selected IX in response to PFAS since that first installation. Specifically, for systems installed prior to 2017, 78% used GAC. The EPA expects this trend to continue, so the initial percentages include adjustments to account for this expectation. In addition, as discussed in Section 5.3.1.1.1, the performance of GAC is affected by the presence of TOC. Accordingly, the table includes adjusted distributions for systems with higher influent TOC.

While central reverse osmosis/nanofiltration (RO/NF) remains a best available technology (BAT) for the final rule, the EPA does not anticipate water systems will select this technology to comply with the rule, largely due to the challenges presented by managing the treatment residuals from this process.

The initial percentages in Table 5-8 reflect the fact that some small systems could choose point-of-use reverse osmosis (POU RO) as a compliance alternative. At this time, the EPA is not including POU devices in the national cost estimates because the regulatory options under consideration require treatment to concentrations below 70 ppt PFOA and PFOS summed, the current certification standard for POU devices.[17] Therefore, SafeWater MCBC excludes POU devices from consideration and proportionally redistributes the percentages among the other alternatives. Table 5-9 shows the final compliance forecast after this redistribution.

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[17] POU treatment might become a compliance option for small systems in the future if independent third-party certification organizations, such as NSF or ANSI develop a new certification standard that mirrors the EPA's proposed regulatory standard. In the event POU treatment becomes a valid compliance option, national costs could be lower than estimated here.

Table 5-9: Initial Compliance Forecast Excluding POU Devices

Compliance Alternative	Design Flow Less than 1 MGD		Design Flow 1 to Less than 10 MGD		Design Flow Greater than or Equal to 10 MGD	
	TOC Less than or Equal to 1.5 mg/L	TOC Greater than 1.5 mg/L	TOC Less than or Equal to 1.5 mg/L	TOC Greater than 1.5 mg/L	TOC Less than or Equal to 1.5 mg/L	TOC Greater than 1.5 mg/L
GAC	79%	62%	81%	52%	89%	52%
PFAS-selective IX	12%	29%	11%	40%	11%	48%
Central RO/NF	0%	0%	0%	0%	0%	0%
Interconnection	7%	7%	6%	6%	0%	0%
New Wells	2%	2%	2%	2%	0%	0%

Abbreviations: GAC – granular activated carbon; PFAS – per-and polyfluoroalkyl substances; MGD – million gallons per day; IX – ion exchange; RO/NF – reverse osmosis/nanofiltration; POU – point-of-use; TOC – total organic carbon.

If all the compliance alternatives (other than POU devices and Centralized RO) remain in consideration after Step 1, the decision tree uses the forecast shown in Table 5-9. If GAC or IX is not viable for a particular EP due to performance limitations (see Section 5.3.1.1.1), SafeWater MCBC proportionally redistributes the percentages among the remaining alternatives and uses the redistributed percentages.

5.3.1.1.1 Estimating GAC and IX Performance

The viability of GAC and IX depends on bed life, which is the length of time the technology can maintain a target removal percentage (e.g., 80 percent, 95 percent). Bed life can vary depending on factors including type of media used (GAC or IX), specific PFAS contaminants targeted, influent water quality, and removal performance required to meet regulatory option thresholds. Bed life determines media replacement frequency and, therefore, affects both the practicality and operation and maintenance (O&M) cost of these technologies. This analysis estimates bed life in bed volumes (BV), which is a measure of throughput: the volume of water treated during the bed life divided by the volume of the media bed.

The bed life estimates use linear equations derived as described in the T&C document (U.S. EPA, 2024i). The EPA estimated the equations based on pooled data from several studies of GAC as well as IX performance and reflect central tendency results under varying water quality conditions. As such, the EPA believes they represent the best approach currently available for use in a national cost estimation. However, they should not be used in lieu of site-specific engineering analyses or pilot studies to guide the design or operation of specific treatment systems.

The bed life equations are technology-specific and shown below:

Equation 2:

$$BV_{contam,GAC} = A_{TOC} \times TOC + A_{R,GAC} \times \%R_{contam} + B_{contam,GAC}$$

$$BV_{contam,IX} = A_{PFAS} \times PFAS_{total} + A_{R,IX} \times \%R_{contam} + B_{contam,IX}$$

Where:

$BV_{contam,tech}$ = bed life of the given technology for a given PFAS contaminant in BV; tech = GAC or IX

TOC = TOC influent to the new treatment process in mg/L

$PFAS_{total}$ = total influent concentration of all PFAS contaminants (regulated or unregulated) in ppt

$\%R_{contam}$ = target percent removal of a given PFAS as a decimal (e.g., 0.8, 0.95)

$B_{contam,tech}$ = constant; tech = GAC or IX

Table 5-10 shows the estimated values of the parameter coefficients A_{TOC}, A_{PFAS}, $A_{R,tech}$, and intercepts $BV_{contam,tech}$.

Table 5-10: Estimated Parameter Values for Technology-Specific Bed Life Equations

Parameter	GAC Model Value	IX Model Value
A_{TOC}	-37,932	Not applicable[a]
A_{PFAS}	Not applicable[a]	-6.04
A_R	-36,309	-198,242
$B_{HFPO-DA}$	113,034	Data not available
B_{PFHxA}	113,967	212,867
B_{PFBS}	129,357	439,515
B_{PFHpA}	129,357	319,511
B_{PFHxS}	129,357	439,515
B_{PFOA}	139,862	390,787
B_{PFOS}	143,731	439,515

Note:
[a] Total PFAS is not a significant parameter in GAC performance; TOC is not a significant parameter in IX performance.
Source: Technical Support Document - Technologies and Cost for Removing Per- and Polyfluoroalkyl Substances (PFAS) from Drinking Water (U.S. EPA, 2024i)

The bed life equations are only applicable over a specific range of water quality conditions (TOC up to 3.2 mg/L for GAC; total PFAS up to 7,044 ppt for IX). Data are not available to estimate performance beyond these limits. Therefore, SafeWater MCBC excludes GAC from consideration if an EP's influent TOC concentration is greater than 3.2 mg/L. It excludes IX if total influent PFAS is greater than 7,044 ppt. No PWS meets both of these exclusionary conditions.

If GAC and/or IX remain in consideration, SafeWater MCBC calculates the percent removal required for the regulatory option under consideration and uses the linear equations above to estimate bed life. These calculations vary depending on the regulatory option. Section 5.3.1.1.1.1 describes the calculations for PFOA and PFOS. Section 5.3.1.1.1.2 describes the calculations under the final rule (individual MCLs for PFOS, PFOA, PFNA, PFHxS, and HFPO-DA plus the group HI MCL).

Based on data presented in the T&C document (U.S. EPA, 2024i), specifically the maximum removal effectiveness values reported in EPA's Drinking Water Treatability Database plus the full set of removal data used to develop the bed life equations presented in Table 5-10, the EPA assumes the maximum PFAS removal achievable by GAC or IX is 99.5% percent. Therefore, if the relevant regulatory option requires removal at an EP greater than this maximum, SafeWater MCBC removes GAC and IX from consideration, as described in the sections below. Additionally, the EPA assumes that bed lives less than 5,000 BV for GAC and less than 20,000 BV for IX are impractical. These bed lives correspond to media replacement frequencies of two to five months depending on the average flow of the EP. If the relevant regulatory option results in a final operating bed life below these limits, SafeWater MCBC removes the corresponding technology from consideration. Finally, the EPA assumes that the maximum bed life for GAC is 75,000 BV and the maximum bed life for IX is 260,000 BV. While some water systems treating for PFAS may have performance that exceeds these values, the EPA included this assumption to more conservatively estimate operational costs. If the calculated bed life is greater than 75,000 BV for GAC or greater than 260,000 BV for IX, then SafeWater MCBC sets the bed life at 75,000 BV for GAC and 260,000 BV for IX. For EPs that ultimately select GAC or IX, the final operating bed life is also an input to the cost estimates (see Section 5.3.1.3) and the calculation of post-treatment PFAS concentrations used to estimate reduction in health risks).[18]

5.3.1.1.1.1 Bed Life for PFOA and PFOS

Under Options 1a-c, PWSs must meet individual MCLs for PFOS and PFOA. For these options, SafeWater MCBC calculates the percent removal required to meet each individual MCL in the following equation:

Equation 3:

$$\%R_{contam} = \frac{C_{0,contam} - MCL_{contam} \times SF}{C_{0,contam}}$$

Where:

$\%R_{contam}$ = target percent removal of a given PFAS as a decimal (e.g., 0.8, 0.95)

$C_{0,contam}$ = influent concentration of the given PFAS in ppt

MCL_{contam} = MCL for the given PFAS in ppt

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[18] As shown in Equation 2, bed life and percent removal are directly related. SafeWater uses the same equation to back-calculate final percent removal for each PFAS compound from final operating bed life. It then uses the final removal efficiency to calculate post-treatment concentrations.

SF = 0.8, a safety factor that assumes PWSs will design and operate treatment processes to achieve 80 percent of the MCL (i.e. to 20 percent below the MCL value).

SafeWater MCBC performs this calculation for each contaminant that occurs at an EP and has an MCL in the regulatory option, even if the contaminant occurs at a concentration below the MCL. Including contaminants that are below their respective MCLs helps to account for chromatographic peaking;[19] which is a concern in GAC along with IX and is discussed in greater detail in the T&C document (U.S. EPA, 2024i). The calculations here are designed to account for and avoid it.

If the percent removal required for any contaminant ($\%R_{contam}$) is greater than 0.99 (99 percent), SafeWater MCBC removes GAC and IX from consideration. If the technologies remain in consideration, SafeWater MCBC estimates the bed life for each contaminant using the linear equations presented in Section 5.3.1.1.1. The final operating bed life is the minimum of the individual contaminant-specific bed life estimates. If this final operating bed life is less than 5,000 BV for GAC or less than 20,000 BV for IX, SafeWater MCBC removes the corresponding technology from consideration.

5.3.1.1.1.2 Bed Life Under the Final Rule

The final rule utilizes compound-specific MCLs for PFOA, PFOS, PFNA, HFPO-DA, and PFHxS and an HI MCL for mixtures containing at least two or more of PFNA, HFPO-DA, PFHxS, and PFBS. Due to limitations in occurrence data, the national cost estimates summate costs only for the occurrence of PFOA, PFOS and PFHxS. The EPA notes that the costs for the HI MCL and the individual MCLs for PFNA and HFPO-DA, are included and considered in the Appendix N, Section N.3 sensitivity analysis. Therefore, for this option, SafeWater MCBC calculates the percent removal required to meet the individual health benchmark for PFHxS using the following equation:

Equation 4:

$$\%R_{PFHxS} = \frac{C_{0,PFHxS} - HB_{PFHxS} \times SF}{C_{0,PFHxS}}$$

Where:

$\%R_{PFHxS}$= target percent removal of PFHxS as a decimal (e.g., 0.8, 0.95)

$C_{0,PFHxS}$ = influent concentration of PFHxS in ppt

HB_{PFHxS} = heath benchmark for PFHxS in ppt

SF = 0.8, a safety factor that assumes PWSs will design and operate treatment processes to achieve 80 percent of the health benchmark.

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[19] Chromatographic peaking is a phenomenon in which less strongly sorbed contaminants are detached from sorbents by more strongly bound sorbents and the less tightly bound sorbent re-enters drinking water. Direct competition with stronger sorbing constituents can lead to effluent PFAS concentrations temporarily exceeding influent concentrations. Some PFAS species sorb more strongly than other PFAS species which can cause more weakly sorbed species to re-enter drinking water.

SafeWater MCBC performs this calculation even when PFHxS occurs at a concentration below its health benchmark. Including contaminants that are below their respective MCLs prevents the subsequent bed life calculations from selecting a bed life that results in a preferred PFAS displacing a less preferred PFAS from the treatment media to the extent that the less preferred PFAS periodically exceeds its MCL. This phenomenon is sometimes a concern in GAC as well as IX design and operation and is discussed in greater detail in the T&C document (U.S. EPA, 2024i). The calculations here are designed to account for and avoid it.

If the percent removal required to meet the MCL and health benchmark for PFHxS is greater than 0.99 (99 percent), SafeWater MCBC removes GAC and IX from consideration. If the technologies remain in consideration, SafeWater MCBC estimates the bed life for PFHxS using the linear equations presented in Section 5.3.1.1.1. It also calculates the bed lives necessary to meet the individual MCLs for PFOS and PFOA, as described in Section 5.3.1.1.1.1. The final operating bed life is the minimum of all the bed life estimates resulting from the calculations for all three contaminants (PFOS, PFOA, and PFHxS). If this final operating bed life is less than 5,000 BV for GAC or less than 20,000 BV for IX, SafeWater MCBC removes the corresponding technology from consideration. Finally, if the calculated bed life is greater than 75,000 for GAC, or greater than 260,000 for IX, then SafeWater MCBC sets the bed life at 75,000 for GAC and 260,000 for IX.

5.3.1.2 WBS Models

The WBS models are spreadsheet-based engineering models for individual treatment technologies, linked to a central database of component unit costs. The EPA developed the WBS model approach as part of an effort to address recommendations made by the Technology Design Panel (TDP), which convened in 1997 to review the agency's methods for estimating drinking water compliance costs (U.S. EPA, 1997). The TDP consisted of nationally recognized drinking water experts from the EPA, water treatment consulting companies, public as well as private water utilities along with suppliers, equipment vendors, and Federal along with State regulators in addition to cost estimating professionals.

In general, the WBS approach involves breaking a process down into discrete components for the purpose of estimating unit costs. The WBS models represent improvements over past cost estimating methods. By adopting a WBS-based approach to identify the components that should be included in a cost analysis, the models produce a more comprehensive, flexible, and transparent assessment of the capital and operating requirements for a treatment system.

Section 5.3.1.2.1 is a brief overview of the common elements of all the WBS models. Section 5.3.1.2.2 provides information on the anticipated accuracy of the models. Sections 5.3.1.2.3 through 5.3.1.2.5 identify technology-specific cost elements included in each model and discuss key inputs. The documentation for the individual WBS models (U.S. EPA, 2023i; U.S. EPA, 2023k; U.S. EPA, 2023j), provides more complete details on the structure, content, and use of each model.

5.3.1.2.1 Common Model Components and Inputs

Each WBS model contains the work breakdown for a particular treatment process and preprogrammed engineering criteria and equations that estimate equipment requirements for user-specified design requirements (e.g., system size and influent water quality). Each model also provides unit and total cost information by component (e.g., individual items of capital equipment) and totals the individual component costs to obtain a direct capital cost. Additionally, the models estimate add-on costs (e.g., permits and land acquisition), indirect capital costs, and annual O&M costs, thereby producing EPA's best estimates of complete compliance cost.

Primary inputs common to all the WBS models include design flow and average daily flow in MGD. Each WBS model has default designs (input sets) that correspond to specified categories of flow, but the models can generate designs for many other combinations of flows. To estimate costs for PFAS compliance, the EPA fit cost curves to the WBS estimates across a range of flow rates, as described in Section 5.3.1.3.

Another input common to all the WBS models is “component level” or “cost level.” This input drives the selection of materials for items of equipment that can be constructed of different materials. For example, a low-cost system might include fiberglass pressure vessels and polyvinyl chloride (PVC) piping. A high-cost system might include stainless steel pressure vessels and stainless-steel piping. The component level input also drives other model assumptions that can affect the total cost of the system, such as building quality and heating and cooling. The component level input has three possible values: low cost, mid cost, and high cost. To estimate costs for PFAS treatment, the EPA generated separate cost equations for each of the three component levels, thus creating a range of cost estimates for use in national compliance cost estimates.

The third input common to all the WBS models is system automation, which allows the design of treatment systems that are operated manually or with varying degrees of automation (i.e., with control systems that reduce the need for operator intervention). The cost equations described in Section 5.3.1.3 are for systems that are fully automated, minimizing the need for operator intervention and reducing operator labor costs.

The WBS models generate cost estimates that include a consistent set of capital, add-on, indirect, and O&M costs. Table 5-11 identifies these cost elements, which are common to all the WBS models and included in the cost estimates below. Sections 5.3.1.2.3 through 5.3.1.2.5 identify the technology-specific cost elements included in each model. The documentation for the WBS models (U.S. EPA, 2023i; U.S. EPA, 2023k; U.S. EPA, 2023l; U.S. EPA, 2023j) provide more information on the methods and assumptions used in the WBS models to estimate the costs for both the technology-specific and common cost elements.

Table 5-11: Cost Elements Included in All WBS Models

Cost Category	Components Included
Direct Capital Costs	• Technology-specific equipment (e.g., vessels, basins, pumps, treatment media, piping, valves) • Instrumentation and system controls • Buildings • Residuals management equipment
Add-on Costs	• Land • Permits • Pilot testing
Indirect Capital Costs	• Mobilization and demobilization • Architectural fees for treatment building • Equipment delivery, installation, and contractor's overhead and profit • Sitework • Yard piping • Geotechnical • Standby power • Electrical infrastructure • Process engineering • Contingency • Miscellaneous allowance • Legal, fiscal, and administrative • Sales tax • Financing during construction • Construction management
O&M Costs: Technology-specific	• Operator labor for technology-specific tasks (e.g., managing backwash and media replacement) • Materials for O&M of technology-specific equipment • Technology-specific chemical usage • Replacement of technology-specific equipment that occurs on an annual basis (e.g., treatment media) • Energy for operation of technology-specific equipment (e.g., mixers)
O&M Costs: Labor	• Operator labor for O&M of process equipment • Operator labor for building maintenance • Managerial and clerical labor
O&M Costs: Materials	• Materials for maintenance of booster or influent pumps • Materials for building maintenance
O&M Costs: Energy	• Energy for operation of booster or influent pumps • Energy for lighting, ventilation, cooling, and heating
O&M Costs: Residuals	• Residuals management operator labor, materials, and energy • Residuals disposal and discharge costs

Abbreviations: O&M – operation & maintenance; WBS – work breakdown structure.

5.3.1.2.2 WBS Model Accuracy

Costs for a given system can vary depending on site-specific conditions (e.g., raw water quality, climate, local labor rates, and location relative to equipment suppliers). The costs presented here are based on national average assumptions and include a range (represented by low-, mid-, and high-cost equations) intended to encompass the variation in costs that systems would incur to remove PFAS. To validate the engineering design methods used by the WBS models and increase the accuracy of the resulting cost estimates, the EPA has subjected the individual models to a process of external peer review by nationally recognized technology experts.

The GAC model underwent peer review in 2006. Two of the three reviewers expressed the opinion that resulting cost estimates would be in the range of budget estimates (+30 to -15 percent). The other reviewer did not provide a precise estimate of the model's accuracy range but commented that the resulting cost estimates were reasonable. The EPA made substantial revisions to the GAC model in response to the peer review.

The IX model underwent peer review in 2005, during an early stage of its development. One peer reviewer responded that resulting cost estimates were in the range of budget estimates (+30 to -15 percent). The other two reviewers thought the estimates were order of magnitude estimates (+50 to -30 percent), with an emphasis on the estimates being high. The IX model has since undergone extensive revision, both in response to the peer review and to adapt it for PFAS treatment using selective resin.

The EPA received peer review comments on the nontreatment model in May 2012. The first reviewer responded that cost estimates resulting from the nontreatment model were in the range of budget estimates (+30 to -15 percent). The second reviewer thought the cost estimates were order of magnitude estimates (+50 to -30 percent). The third reviewer felt the cost estimates were definitive (+15 to -5 percent), except for land costs, which were difficult to assess due to regional variations. The EPA revised the nontreatment model in response to the peer review recommendations.

5.3.1.2.3 GAC Model

Work Breakdown Structure-Based Cost Model for Granular Activated Carbon Drinking Water Treatment provides a complete description of the engineering design process used by the WBS model for GAC (U.S. EPA, 2023i). The model can generate costs for two types of design:

- Pressure designs where the GAC bed is contained in stainless steel, carbon steel, or fiberglass pressure vessel; and
- Gravity designs where the GAC bed is contained in open concrete basins.

Table 5-12 shows the technology-specific capital equipment and O&M requirements included in the GAC model. These items are in addition to the common WBS cost elements listed in Table 5-11.

Table 5-12: Technology-Specific Cost Elements Included in the GAC Model

Cost Category	Major Components Included
Direct Capital Costs	• Booster pumps for influent water • Contactors (either pressure vessels or concrete basins) that contain the GAC bed • Tanks and pumps for backwashing the contactors • GAC transfer and storage equipment • Spent GAC reactivation facilities (if on-site reactivation is selected) • Associated piping, valves and instrumentation
O&M Costs: Labor	• Operator labor for contactor maintenance (for gravity GAC designs) • Operator labor for managing backwash events • Operator labor for backwash pump maintenance (if backwash occurs weekly or more frequently) • Operator labor for GAC transfer and replacement
O&M Costs: Materials	• Materials for contactor maintenance (accounts for vessel relining in pressure designs, because GAC can be corrosive, and for concrete and underdrain maintenance in gravity designs) • Materials for backwash pump maintenance (if backwash occurs weekly or more frequently) • Replacement virgin GAC (loss replacement only if reactivation is selected)
O&M Costs: Energy	• Operating energy for backwash pumps
O&M Costs: Residuals	• Discharge fees for spent backwash • Fees for reactivating spent GAC (if off-site reactivation is selected) • Labor, materials, energy, and natural gas for regeneration facility (if on-site reactivation is selected) • Disposal of spent GAC (if disposal is selected)

Abbreviations: GAC – granular activated carbon; O&M – operation & maintenance; WBS – work breakdown structure.

For small systems (less than 1 MGD) using pressure designs, the GAC model assumes the use of package treatment systems that are pre-assembled in a factory, mounted on a skid, and transported to the site. These assumptions are based on common vendor practice for these technologies, for example, see Khera et al. (2013), which says "...small systems are often built as packaged, pre-engineered, or skid-mounted systems." The model estimates costs for package systems by costing all individual equipment line items (e.g., vessels, interconnecting piping and valves, instrumentation, and system controls) in the same manner as custom-engineered systems. This approach is based on vendor practices of partially engineering these types of package plants for specific systems (e.g., selecting vessel size to meet flow and treatment criteria). The model applies a variant set of design inputs and assumptions that are intended to simulate the use of a package plant and that reduce the size and cost of the treatment system. U.S. EPA (2023i) provides complete details on the variant design assumptions used for package plants.

To generate the cost equations discussed in Section 5.3.1.3, the EPA used the following key inputs in the GAC model:

- For pressure designs, two vessels in series with a minimum total empty bed contact time (EBCT) of 20 minutes;
- For gravity designs, contactors in parallel with a minimum total EBCT of 20 minutes; and
- Bed life varying over a range from 5,000 to 75,000 BV, estimated as discussed in Section 5.3.1.1.1.

The EPA generated separate cost equations for two spent GAC management scenarios:

- Off-site reactivation under current RCRA non-hazardous waste regulations; and
- Off-site disposal as a hazardous waste and replacement with virgin GAC (i.e., single use operation).

The T&C document (U.S. EPA, 2024i) provides a comprehensive discussion of these and other key inputs and assumptions.

5.3.1.2.4 PFAS-selective IX Model

Work Breakdown Structure-Based Cost Model for Ion Exchange Treatment of Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water provides a complete description of the engineering design process used by the WBS model for PFAS-selective IX (U.S. EPA, 2023j). Table 5-13 shows the technology-specific capital equipment and O&M requirements included in the model. These items are in addition to the common WBS cost elements listed in Table 5-11.

Table 5-13: Technology-Specific Cost Elements Included in the PFAS-Selective IX Model

Cost Category	Major Components Included
Direct Capital Costs	• Booster pumps for influent water • Pre-treatment cartridge filters • Pressure vessels that contain the resin bed • Tanks and pumps for initial rinse and (optionally) backwash of the resin bed • Tanks (with secondary containment), pumps and mixers for delivering sodium hydroxide for use in post-treatment corrosion control (optional) • Associated piping, valves, and instrumentation
O&M Costs: Labor	• Operator labor for pre-treatment filters • Operator labor for managing backwash/rinse events • Operator labor for backwash pump maintenance (only if backwash occurs weekly or more frequently) • Operator labor for resin replacement
O&M Costs: Materials	• Replacement cartridges for pre-treatment filters • Materials for backwash pump maintenance (only if backwash occurs weekly or more frequently) • Chemical usage (if post-treatment corrosion control is selected) • Replacement virgin PFAS-selective resin
O&M Costs: Energy	• Operating energy for backwash/rinse pumps
O&M Costs: Residuals	• Disposal of spent cartridge filters • Discharge fees for spent backwash/rinse • Disposal of spent resin

Abbreviations: IX – ion exchange; O&M – operation & maintenance; PFAS – per-and polyfluoroalkyl substances.

For small systems (less than 1 MGD), the PFAS-selective IX model assumes the use of package treatment systems that are pre-assembled in a factory, mounted on a skid, and transported to the site. The IX model estimates costs for package systems using an approach similar to that described for the GAC model, applying a variant set of inputs and assumptions that reduce the size and cost of the treatment system (see Section 5.3.1.2.3). U.S. EPA (2023j) provides complete details on the variant design assumptions used for IX package plants.

To generate the cost equations discussed in Section 5.3.1.3, the EPA used the following key inputs in the PFAS-selective IX model:

- Two vessels in series with a minimum total EBCT of 6 minutes; and
- Bed life varying over a range from 20,000 to 260,000 BV, estimated as discussed in Section 5.3.1.1.

The EPA generated separate cost equations for two spent resin management scenarios:

- Spent resin managed as non-hazardous and sent off-site for incineration; and

- Spent resin managed as hazardous and sent off-site for incineration.

The T&C document (U.S. EPA, 2024i) provides a comprehensive discussion of these and other key inputs and assumptions.

5.3.1.2.5 Nontreatment Model

Work Breakdown Structure-Based Cost Model for Nontreatment Options for Drinking Water Compliance provides a complete description of the engineering design process used by the WBS model for nontreatment actions (U.S. EPA, 2023k). The model can estimate costs for two nontreatment alternatives: interconnection with another system and drilling new wells to replace a contaminated source. Table 5-14 shows the technology-specific capital equipment and O&M requirements included in the model for each alternative. The interconnection alternative does not include any buildings. It includes all the indirect capital costs shown in Table 5-14 except for yard piping, site work, and architectural fees. The new well alternative includes a small shed or other low-cost building at the well site along with materials and labor for maintenance of this building. It includes all the indirect capital costs shown in Table 5-14 except for yard piping.

Table 5-14: Technology-Specific Cost Elements Included in the Nontreatment Model

Cost Category	Major Components Included for Interconnection	Major Components Included for New Wells
Direct Capital Costs	• Booster pumps or pressure reducing valves (depending on pressure at supply source) • Concrete vaults (buried) for booster pumps or pressure reducing valves • Interconnecting piping (buried) and valves	• Well casing, screens, and plugs • Well installation costs including drilling, development, gravel pack, and surface seals • Well pumps • Piping (buried) and valves to connect the new well to the system
O&M Costs: Labor	• Operator labor for O&M of booster pumps or pressure reducing valves (depending on pressure at supply source) and interconnecting valves	• Operator labor for operating and maintaining well pumps and valves
O&M Costs: Materials	• Cost of purchased water • Materials for maintaining booster pumps (if required by pressure at supply source)	• Materials for maintaining well pumps
O&M Costs: Energy	• Energy for operating booster pumps (if required by pressure at supply source)	• Energy for operating well pumps

Abbreviations: O&M – operation & maintenance.

To generate the cost equations discussed in Section 5.3.1.3, the EPA used the following key inputs in the nontreatment model for interconnection:

- An interconnection distance of 10,000 feet;
- Includes booster pumps designed to account for friction loss in interconnecting piping; and
- An average cost of purchased water of $3.35 per thousand gallons in 2022 dollars.

For new wells, the EPA used the following key inputs:

- A maximum well capacity of 500 gallons per minute (gpm), such that one new well is installed per 500 gpm of water production capacity required;
- A well depth of 250 feet; and
- 500 feet of distance between the new wells and the distribution system.

The T&C document (U.S. EPA, 2024i) provides a comprehensive discussion of these and other key inputs and assumptions.

5.3.1.3 WBS Cost Equations

The EPA developed the cost estimates for PFAS treatment using outputs from the WBS models. Outputs from these models are point estimates of total capital and O&M cost that correspond to a given set of inputs that include design flow and average daily flow in MGD. Separately for total capital and annual O&M cost, the EPA fit cost equations to the WBS outputs for up to 49 different flow rates. The EPA choose from among several possible equation forms: linear, quadratic, cubic, power, exponential, and logarithmic. For each equation, the EPA selected the form that resulted in the best correlation coefficient (R2), subject to the requirement that the equation must be monotonically increasing over the appropriate range of flow rates (i.e., within the flow rate category, the equation must always result in higher estimated costs for higher flow systems than for lower flow systems). The resulting cost equations take one of the following forms, identified by which coefficients (C1 through C10) are nonzero:

Equation 5:

$$\begin{aligned} \text{Cost} &= C1\ Q^{C2} \\ \text{or} &= C3\ \mathrm{Ln}(Q) + C4 \\ \text{or} &= C5\ e^{(C6\ Q)} \\ \text{or} &= C7\ Q3 + C8\ Q2 + C9\ Q + C10 \end{aligned}$$

In each case, Q is design flow in MGD for total capital costs, or average flow in MGD for annual O&M costs. The resulting costs are in 2022 dollars.

The equations are categorized by water source (surface water or ground water) and component level (low, mid, or high cost). The EPA developed separate equations for small, medium, or large systems. These equations apply as follows:

- Small system equations apply where design flow (Q) is less than 1 MGD;
- Medium system equations apply where design flow (Q) is 1 MGD or greater, but less than 10 MGD; and
- Large system equations apply where design flow (Q) is 10 MGD or greater.

SafeWater MCBC selects from among the small, medium, and large equations and applies the equations using the treated flow of the EP. For GAC, IX, and nontreatment alternatives, the treated flow is the entire flow of the EP.

For GAC and IX, the EPA developed separate equations that vary according to the estimated bed life. These equations are in increments of 5,000 BV for GAC and 20,000 BV for IX. Each bed life increment corresponds to a change in media replacement frequency of two to five months, depending on the average flow of the EP. For EPs using GAC or IX, SafeWater MCBC selects from among these equations based on the final operating bed life calculated as described in Section 5.3.1.1.1, rounded down to the nearest increment of 5,000 BV for GAC and 20,000 BV for IX.

For GAC, there are separate equations for pressure designs and gravity designs. For ground water EPs using GAC, the EPA assumed PWSs would always use pressure designs to maintain their existing pressure head. For surface water EPs using GAC, the EPA assumed PWSs would choose between pressure and gravity based on the design that results in the lower annualized cost.

In total, there are more than 2,600 individual cost equations across the categories of capital and O&M cost, water source, component level, flow, bed life (for GAC and IX), residuals management scenario (for GAC and IX), and design type (for GAC). The T&C document (U.S. EPA, 2024i) presents the equations in tabular form.

5.3.1.4 Incremental Treatment Costs of PFNA, PFBS, and HFPO-DA

The EPA has estimated the national level costs of the final rule associated with PFOA, PFOS and PFHxS. As discussed in Chapter 4 and detailed in the Technical Support Document for PFAS Occurrence and Contaminant Background Chapter 10.1 and 10.3, there are limitations with nationally representative occurrence information for the other compounds in the final rule (PFNA, HFPO-DA, and PFBS; U.S. EPA, 2024g). Specifically, HFPO-DA does not currently have a completed nationally representative dataset while PFNA and PFBS were not included in the national occurrence model because of limited results reported above the minimum reporting levels in UCMR 3. As described in the Technical Support Document for PFAS Occurrence and Contaminant Background Chapter 10.3 non-targeted state monitoring datasets were used for extrapolation of PFNA, HFPO-DA, and PFBS in lieu of a nationally representative dataset (U.S. EPA, 2024g). EPA used conservative assumptions in this extrapolation to generate conservative cost estimates. As demonstrated in this analysis, the HI, PFNA, and HFPO-DA MCLs meaningfully increase public health protection at modest additional costs.

Because of the increased uncertainty associated with PFNA, HFPO-DA and PFBS, the additional treatment cost from co-occurrence of PFNA, HFPO-DA, PFBS at systems already required to treat because of PFOA, PFOS, or PFHxS MCL and HI exceedances are not quantitatively assessed in the national cost estimates. These HI treatment costs are summarized here in this section and detailed in Appendix N, Section N.3. Likewise, treatment costs for systems that exceed the HI based on the combined occurrence of PFNA, HFPO-DA, PFBS, and PFHxS (where PFHxS itself does not exceed its HBWC of 10 ppt) are not included in the national monetized cost estimates and are also summarized in this section and detailed in Appendix N, Section N.3.

In the EA for the proposed PFAS NPDWR, the EPA used a model system approach to illustrate the potential incremental costs for removing PFAS not included in the national economic model. After considering public comments on the incremental cost analysis, the EPA decided to further explore the incremental costs associated with the HI and MCLs with a national level sensitivity analysis in the final rule.

When the modeled occurrence data for PFNA, HFPO-DA, PFBS is incorporated into the SafeWater MCBC model, the estimated number of EPs exceeding one or more MCLs, and therefore required to treat or use a different water source, increases to 9,471 from 9,043. This results in an increase in the expected national costs. Under the primary analyses, the expected total national cost is $1,549 million over EPA's period of analysis (2024-2105) for the PFOA, PFOS and PFHxS MCLs (which as discussed in Section XII.A.4 of the preamble for today's rule, accounts for a portion of the HI costs). When considering the additional incremental national cost impacts of the HI MCL based on occurrence of PFNA, HFPO-DA, and PFBS and individual MCLs for PFNA and HFPO-DA based on their individual occurrence the expected national costs of the final rule increase to $1,631 million, or approximately a 5 percent national cost increase.

For further detail on the assumptions and findings of the EPA’s analysis of incremental costs of other PFAS, see Appendix N, Section N.3.

5.3.2 Estimating PWS Administrative and Monitoring Costs

This section details how the EPA estimated the costs of compliance with system administrative and sampling activities associated with the final rule. In section 5.3.2, the EPA organizes and presents the cost information based on the series of activities that are required to comply with the final PFAS NPDWR, with tables for each data element used to calculate the final rule component costs. These tables include the data element name and a description of the data variable, as well as any relevant sources for the data. The EPA presents the costs categorized as follows:

- Administrative costs associated with implementation (Section 5.3.2.1)
- Sampling costs (Section 5.3.2.2)
- Administrative costs associated with treatment (Section 5.3.2.3)

Consistent with standard agency practice, the EPA assumes compliance with the rule throughout the economic analysis, and as a result, SafeWater MCBC does not accrue costs to any system for the Tier 2 and 3 public notifications. Nevertheless, the EPA presents a qualitative discussion of the public notification costs potentially associated with the final rule in Section 5.3.2.4.

5.3.2.1 Implementation Administration Costs

Systems conduct the following one-time actions to begin implementation of the rule:

- Reading and understanding the rule; and
- Attending training provided by primacy agencies.

The average unit costs for PWSs are based on the following burden assumptions: 1) The EPA anticipates that the majority of water systems will likely not read the entirety of the rule preamble (as they are not required to do so) but focus their time and attention on understanding the regulatory requirements through the Code of Federal Regulations (CFR) regulatory text, relevant portions of the preamble, the EPA provided fact sheets and small system guidance documents, and state provided summaries documents; 2) Additionally, the EPA anticipates that system staff will attend primacy agency PFAS rule trainings to reenforce the systems' understanding of the final rule. The EPA assumes that systems will conduct these activities during years one through three of the period of analysis. Table 5-15 lists the data elements and provides descriptions, values, and sources for these costs. The cost per system for each activity is the product of the hourly labor cost (labor_sys_rate) and the hours (hrs_sys_adopt_rule and hrs_sys_initial_ta), which vary by system size. The total cost is the sum of per-system costs.

Table 5-15: Implementation Administration Startup Costs ($2022)

Data Element Name	Data Element Description	Data Element Value	Data Element Source
labor_sys_rate	The labor rate per hour for systems	$36.43 (systems ≤3,300) $38.84 (systems 3,301-10,000) $41.00 (systems 10,001-50,000) $42.81 (systems 50,001-100,000) $50.03 (systems >100,000)	WBS Technical Labor Cost
hrs_sys_adopt_rule	The average hours per system to read and adopt the rule	4 hours per system	Arsenic in Drinking Water Rule Economic Analysis (EPA 815-R-00-026)
hrs_sys_initial_ta	The average hours per system to attend one-time training provided by primacy agencies	16 hours per system (systems ≤3,300) 32 hours per system (systems >3,300)	Arsenic in Drinking Water Rule Economic Analysis (EPA 815-R-00-026)

Abbreviation: WBS – work breakdown structure.

5.3.2.2 Sampling Costs

The final rule requires initial and long-term monitoring. As Table 5-16 shows, surface and ground water systems serving greater than 10,000 people will collect one sample each quarter, at each EP, during the initial 12-month monitoring period. Surface water systems serving 10,000 or fewer people are also required to collect a quarterly sample at each EP during the initial 12-month period. Ground water systems that serve 10,000 or fewer people will be required to sample once at each EP on a semi-annual basis for the first 12-month monitoring period.

Long-term monitoring schedules are based on specific EP sampling results (i.e., water systems can have different EPs within the system on different monitoring schedules). Long-term monitoring requirements differ based on whether a system can demonstrate during the initial monitoring period or once conducting long-term monitoring that an EP is below the trigger levels for regulated PFAS. The trigger levels are set as one-half each of the MCLs: 2.0 ppt for PFOA and PFOS 5 ppt for PFHxS, HFPO-DA, and PFNA and 0.5 for the HI. EPs below the trigger level values during the initial 12-month monitoring period and in future long-term monitoring periods may conduct triennial monitoring and collect one triennial sample at that EP. For EPs with concentration values at or above a trigger level, a quarterly sample must be taken at that EP following initial monitoring. EPs that demonstrate they are "reliably and consistently"[20] below the MCLs following four consecutive quarterly samples are eligible to conduct annual monitoring. After three annual samples at that EP showing no results at or above a trigger level, the location can further reduce to triennial monitoring.

For any samples that have a detection, the system will analyze the field reagent blank samples collected at the same time as the monitoring sample. Systems that have an MCL exceedance will collect one additional sample from the relevant EP to confirm the results (i.e., a confirmation sample) (U.S. EPA, 2004).

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[20] The definition of reliably and consistently below the MCL means that each of the samples contains regulated PFAS concentrations below the applicable MCLs. For the PFAS NPDWR, this demonstration of reliably and consistently below the MCL would include consideration of at least four quarterly samples at an EP below the MCL, but states will make their own determination as to whether the detected concentrations are reliably and consistently below the MCL.

Table 5-16: Modeled Initial and Long-Term Sampling Frequencies Per System Entry Point

Initial Monitoring		**Long Term Monitoring**[a]		
System Size Category	**Sample Number and Frequency**	**PFAS Detection ≥ MCLs**	**PFAS Detection ≥ Trigger Levels and < MCLs**[b]	**PFAS Detection < Trigger Levels**
≤ 10,000	Surface water: 1 sample every quarter Ground water: 1 sample every 6-month period	1 sample every quarter	1 sample every year (following four consecutive quarterly samples reliably and consistently below the MCL)	1 triennial sample
>10,000	Surface water and Ground water: 1 sample every quarter	1 sample every quarter	1 sample every year (following four consecutive quarterly samples reliably and consistently below the MCL)	1 triennial sample

Abbreviations: MCL– maximum contaminant level; PFAS – per-and polyfluoroalkyl substances.
Note:
[a] The EPA used the following thresholds to distinguish whether PFAS concentrations are reliably and consistently below the MCL: If after four consecutive quarterly samples, a system is below the MCLs (PFOA and PFOS – 4.0 ppt, PFHxS, HFPO-DA, PFNA – 10 ppt, HI – 1).
[b] Systems are not eligible for annual monitoring until after four consecutive quarterly samples are collected following initial monitoring.

For the national cost analysis, the EPA assumes that systems with either UCMR 5 data or monitoring data in the State PFAS Database will not conduct the initial year of monitoring (See Section 3.1.4). As a simplifying assumption for the cost analysis, the EPA assumes all systems serving a population of greater than 3,300 have UCMR 5 data and those with 3,300 or less do not. For the State PFAS Database, the EPA relied on the PWSIDs stored in the database and exempted those systems from the first year of monitoring in the cost analysis.

The EPA assumes that systems with an MCL exceedance will implement actions to comply with the MCL by the compliance date. As indicated in Section 5.3.1, the EPA assumes a treatment target, for systems required to treat for PFAS, that includes a margin of safety so finished water PFAS levels at these systems are 80 percent of the MCLs and HI. In the final rule, in order to reduce burden associated with monitoring, the EPA is adding an annual tier of sampling for any system with concentrations reliably and consistently below the MCL but not consistently below the trigger level. The EPA believes this tier would likely apply to most systems treating their water for regulated PFAS, at least for the first three years of treatment. Therefore, in the model, the EPA assumes EPs that have installed treatment will take one year of quarterly samples, then continue to sample on an annual basis after that. The final rule allows EPs showing no results at or above a trigger level after three annual samples to further reduce to triennial monitoring. In the national cost analysis, the EPA does not model this possibility nor does the EPA model instances where water systems are triggered back into quarterly monitoring after installing treatment.

For all systems, the activities associated with the sample collection in the initial 12-month monitoring period are the labor burden and cost for the sample collection and analysis, as well as a review of the sample results. Table 5-17 presents the data needs associated with the implementation monitoring period. The cost per EP for each sampling activity is the product of the hourly labor cost and the hours plus the laboratory analysis cost. The laboratory analysis cost will include the additional field blank cost when occurrence values exceed method detection limits. The total cost is the sum of per-EP costs.

Table 5-17: Sampling Costs ($2022)

Data Element Name	Data Element Description	Data Element Value	Data Element Source
labor_sys_rate	The labor rate per hour for systems	$36.43 (systems ≤3,300) $38.84 (systems 3,301-10,000) $41.00 (systems 10,001-50,000) $42.81 (systems 50,001-100,000) $50.03 (systems >100,000)	WBS Technical Labor Cost
numb_initial_samples	The number of samples per EP per monitoring round for the initial monitoring in Year 1	4 samples per system[a] 2 samples (ground water systems ≤ 10,000)	Final rule
numb_quarterly_samples	The number of samples per EP per long-term monitoring year for EPs with finished water concentrations > MCLs (i.e., Systems not reliably and consistently below the MCLs)	4 samples per year	Final rule
numb annual samples	The number of samples per EP per long-term monitoring year for EPs with finished water concentrations ≤ MCLs but ≥ the trigger levels for four consecutive quarterly samples	1 sample per year	
numb_trienniall_samples	The number of samples per EP per long-term monitoring round for EPs with finished water concentrations < the trigger levels	1 sample every 3 years	Final rule
hrs_samp	The hours per sample to travel to sampling locations, collect samples, record any additional information, submit samples to a laboratory, and review results	1 hour	UCMR5 ICR (EPA-HQ-OW-2020-0530-00141)
EPA537_cost	The laboratory analysis cost per sample for EPA Method 537.1[b]	$309	UCMR5 ICR (EPA-HQ-OW-2020-0530-0141)

Table 5-17: Sampling Costs ($2022)

Data Element Name	Data Element Description	Data Element Value	Data Element Source
EPA537_fieldblank_cost	The laboratory analysis cost per sample for the field reagent blank under EPA Method 537.1	$273[c]	

Abbreviations: EPA – U.S. Environmental Protection Agency; ICR – Information Collection Request; UCMR – Unregulated Contaminant Monitoring Rule; WBS – work breakdown structure.

Notes:

[a] Systems greater than 3,300 will rely on UCMR 5 data and a subset of other systems will rely on data in the State PFAS Monitoring Database.

[b] The EPA assumes that while both methods provide the required data to demonstrate compliance, water systems will select the least costly analytical method (which is Method 537.1).

[c] This incremental sample cost applies to all samples that exceed the method detection limit.

5.3.2.3 Treatment Administration Costs

As described in Section 5.3.1, any system with an MCL exceedance adopts either a treatment or nontreatment alternative to comply with final rule. The majority of systems are anticipated to install treatment technologies while a subset, described in Section 5.3.1.1, will choose alternative methods. The EPA assumes that systems will have administrative costs associated with obtaining permits for either the treatment or nontreatment methods. The costs vary depending on whether the system installs treatment or selects a nontreatment method. For the economic analysis, the EPA assumes that systems install treatment in the fifth year of the period of analysis. In addition, after installation of treatment, the EPA assumes that systems will spend an additional 2 hours per treating EP compiling data for and reviewing treatment efficacy with their primacy agency during their triennial sanitary survey.

Table 5-18 presents the data elements and sources for these costs. The cost per EP requiring treatment or changing water source is the product of the hourly labor cost and the hours per the relevant permit request and sanitary survey review. The total cost is the sum of per-EP costs.

Table 5-18: Treatment Administration Costs ($2022)

Data Element Name	Data Element Description	Data Element Value	Data Element Source
labor_sys_rate	The labor rate per hour for systems	\$36.43 (systems ≤3,300) \$38.84 (systems 3,301-10,000) \$41.00 (systems 10,001-50,000) \$42.81 (systems 50,001-100,000) \$50.03 (systems >100,000)	WBS Technical Labor Cost
hrs_sys_treat	The hours per EP for a system to notify, consult, and submit a permit request for treatment installation[a]	3 hours (systems ≤100) 5 hours (systems 101-500) 7 hours (systems 501-1,000) 12 hours (systems 1,001 - 3,300) 22 hours (systems 3,301-50,000) 42 hours (systems >= 50,001)	Lead and Copper Rule Revisions Support Material (EPA-HQ-OW-2017-0300-1701)
hrs_ss_increment	The additional hours per EP the system will spend every 3 years after PFAS-related treatment is installed during a sanitary survey.	2 hours per EP that installs treatment every 3 years post-installation	Lead and Copper Rule Revisions Support Material (EPA-HQ-OW-2017-0300-1701)
hrs_sys_source	The hours per EP for a system to notify, consult, and submit a permit request for source water change or alternative method[a]	6 hours	Lead and Copper Rule Revisions Support Material (EPA-HQ-OW-2017-0300-1700)

Abbreviations: WBS – work breakdown structure.
Note:
[a]The Lead and Copper Rule Revisions presents this burden per system, but the EPA applied the cost per EP for this economic analysis because the notification, consultation, and permitting process occurs for individual EPs.

5.3.2.4 Public Notification Costs

While the EPA assumes full compliance with the rule and does not include public notification costs in the cost estimates, there are public notification requirements in the final rule for systems with certain violations. The final rule designates MCL violations for PFAS as Tier 2, which requires systems to provide public notification as soon as practical, but no later than 30 days after the system learns of the violation. The system must repeat notice every three months if the violation or situation persists unless the primacy agency determines otherwise. At a minimum, systems must give repeat notice at least once per year.

The final rule designates monitoring and testing procedure violations as Tier 3, which requires systems to provide public notice not later than one year after the system learns of the violation.

The system must repeat the notice annually for as long as the violation persists. Community water systems may deliver Tier 3 PNs in their CCR if the timing, content, and delivery requirements are met according to 40 CFR 141.204(d). Using the CCR to deliver Tier 3 PNs can minimize the burden on systems by reducing delivery costs.

To provide an approximate estimate of the burden associated with the Tier 2 and 3 violations, the EPA reviewed the ICR for the Public Water System Supervision (PWSS) Program (U.S. EPA, 2011), which includes Tier 2 and 3 notifications. Table 5-19 presents the PWSS Program ICR burdens for the preparation and delivery of the Tier 2 and 3 public notifications.

Table 5-19: Public Notification Burden Estimate

Data Element[a]	Data Element Value	Data Element Source
Preparation of initial Tier 2 notices	3.5 hours	PWSS Program ICR (EPA-HQ-OW-2011-0433-0003)
Preparation of initial Tier 3 notices	3 hours (CWS) 3.5 hours (NTNCWS)	PWSS Program ICR (EPA-HQ-OW-2011-0433-0003)
Delivery of initial Tier 2 notices	9 hours (CWS ≤500) 30 hours (CWS >500) 9 hours (NTNCWS)	PWSS Program ICR (EPA-HQ-OW-2011-0433-0003)
Development and delivery of repeated Tier 2 and 3 notices	3 hours	PWSS Program ICR (EPA-HQ-OW-2011-0433-0003)

Abbreviations: CWS – community water systems; NTNCWS – non-transient non-community water systems; PWSS – public water systems supervision; ICR – information collection request.

Note:

[a] Delivery of Tier 3 notices must occur not later than one year after the system learns of the violation. The EPA assumes systems will include this notice with the Consumer Confidence Reports sent to all customers annually, therefore Tier 3 delivery costs are assumed to be zero.

5.4 Estimating Primacy Agency Costs

In addition to the PWS costs associated with the rule implementation, the EPA assumes primacy agencies will have upfront implementation costs as well as ongoing administrative costs and costs associated with the system actions related to sampling and treatment. The activities associated with primacy agencies under the final rule include:

- Reading and understanding the rule, providing internal primacy agency officials training for the rule implementation, updating sanitary survey standard operating procedures,
- Primacy package application, including making state regulatory changes to the federal rule where applicable
- Providing systems with training and technical assistance during the rule implementation;
- Reporting to the EPA on an ongoing basis any PFAS-specific information under 40 CFR 142.15 regarding violations as well as enforcement actions and general operations of public water supply programs;

- Performing inspection of PFAS related treatment during sanitary surveys every three years[21]
- Reviewing the sample results during the initial monitoring period and the long-term monitoring period; and
- Reviewing and consulting with systems on the installation of treatment technology or alternative methods, including source water change.

For the last three activities listed above, primacy agency burdens are incurred in response to an action taken by a system. For example, the cost to primacy agencies of reviewing any sample result depends on the number of samples taken at each EP by each system under the jurisdiction of the primacy agency. Table 5-20 presents the data elements and sources for all primacy agency costs. The data element descriptions indicate whether the cost is per primacy agency, per sample, per system, or per EP. In each instance, the primacy agency labor rate is multiplied by the number of relevant hours and the activity frequency.

Table 5-20: Primacy Agency Costs ($2022)

Data Element Name	Data Element Description	Data Element Value	Data Element Source
labor_pa_rate	The labor rate per hour for primacy agencies	$59.69	Loaded labor rate (including the cost of benefits) derived from the Bureau of Labor Statistics[a]
hrs_pa_adopt_rule	The average hours per primacy agency to read and understand the rule, update sanitary survey standard operating procedures, and train internal staff.	4,020 hours per primacy agency	ASDWA, 2023
hrs_pa_write_reg	The average hours for a primacy agency to develop state-level regulations	300 hours per primacy agency	ASDWA, 2023
hrs_pa_initial_ta	The average hours per primacy agency to provide initial training and technical assistance to systems	1,500 hours per primacy agency	ASDWA, 2023
hrs_sdwis	The average hours per primacy agency to report annually to the EPA information under 40 CFR 142.15 regarding violations, variances and exemptions, enforcement actions and general operations of State public water supply programs	0	The EPA assumes that the final PFAS rule will have no discernable incremental burden for quarterly or annual reports to SDWIS/Fed

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[21] Sanitary surveys are required for CWS every three years, except for CWS with outstanding performance based on prior sanitary surveys for which they are required every 5 years. Sanitary surveys are required for NCWS at least every 5 years. As a simplifying assumption in the national cost analysis, the EPA set the sanitary survey frequency to three years for all systems expected to install treatment to comply with the PFAS rule.

Table 5-20: Primacy Agency Costs ($2022)

Data Element Name	Data Element Description	Data Element Value	Data Element Source
hrs_pa_report_ep	The hours per sample for a primacy agency to review sample results	1 hour	Arsenic in Drinking Water Rule Economic Analysis (EPA 815-R-00-026)
hrs_pa_treat	The hours per EP for a primacy agency to review and consult on installation of a treatment technique[b]	80 hours (systems ≤3,300) 70 hours (systems serving 3,301 - 50,000) 50 hours (systems serving > 50,000)	ASDWA, 2023
hrs_pa_ss_increment	The additional hours per EP the primacy agency will spend every 3 years after PFAS-related treatment is installed during a sanitary survey.	2 hours per EP that installs treatment every 3 years post-installation.	Lead and Copper Rule Revisions Support Material (EPA-HQ-OW-2017-0300-1701)
hrs_pa_source	The hours per EP for a primacy agency to review and consult on a source water change[b]	4 hours	Lead and Copper Rule Revisions Support Material (EPA-HQ-OW-2017-0300-1700)

Abbreviations: PFAS – per-and polyfluoroalkyl substances; SDWIS/Fed – Safe Drinking Water Information System Federal Version; ASDWA – Association of State Drinking Water Administrators.

Notes:

[a] State employee wage rate of $33.91 from National Occupational Employment and Wage Estimates, United States, BLS SOC Code 19-2041, "State Government, excluding schools and hospitals - Environmental Scientists and Specialists, Including Health," hourly mean wage rate. May 2020 data (published in March 2021): https://www.bls.gov/oes/current/oes192041.htm. Wages are loaded using a factor of 62.2 from the BLS Employer Costs for Employee Compensation report, Table 3, March 2020. Percent of total compensation - Wages and Salaries - All Workers - State and Local Government Workers (https://www.bls.gov/news.release/archives/ecec_06182020.pdf). See worksheet BLS Table 3. The final loaded wage is adjusted for inflation.

[b] The Lead and Copper Rule Revisions present this burden per system, but the EPA has applied the cost per EP for this economic analysis because the notification, consultation, and permitting process occurs for individual EPs.

In addition to the costs described above, a primacy agency may also have to review the certification of any Tier 2 or 3 public notifications sent out by systems. The EPA assumes full compliance with the final rule but provides a brief discussion of the possible system costs associated with this component in Section 5.3.2.4. The public notification burden associated with primacy agencies is between 0.33 and 0.5 hours per system to review the system certification of the public notification. The burden is derived from the Lead and Copper Rule Revisions estimates for a similar activity.

5.5 PWS-Level Cost Estimates

PWS-level cost estimates for the final rule and other regulatory options are provided in Appendix C. PWS-level cost are provided for all PWSs by PWS-type, size category, primary source water type, and ownership. In addition, a second set of PWS-level costs are provided for PWSs that must take action to comply with the rule (treat or change water source).

5.6 Household-Level Cost Estimates

Household-level cost estimates for the final rule and other regulatory options are provided in Appendix C. Household-level cost are provided for all CWSs by size category, primary source water type, and ownership. In addition, a second set of household-level costs are provided for households served by CWSs that must take an action to comply with the rule (treat or change water source).[22]

5.7 Discussion of Data Limitations and Uncertainty

The preceding sections identify the nonquantifiable costs and the uncertainty information incorporated in the quantitative cost analysis. There are also data limitations that could not be incorporated in this analysis. Chapter 7 and Table 7-6 outline the nonquantifiable costs associated with the regulatory requirements of the final rule as well as Options 1a-c. Table 5-21 lists the data limitations and characterizes the impact on the quantitative cost analysis. The EPA notes that in most cases it is not possible to judge the extent to which a particular limitation or uncertainty could affect the cost analysis. The EPA provides the potential direction of the impact on the cost estimates when possible but does not prioritize the entries with respect to the impact magnitude.

Table 5-21: Limitations that Apply to the Cost Analysis for the Final PFAS Rule

Uncertainty/ Assumption	Effect on Quantitative Analysis	Notes
WBS engineering cost model assumptions and component costs	Uncertain	The WBS engineering cost models require many design and operating assumptions to estimate treatment process equipment and operating needs. Section 5.3.1 addressed the bed life assumption. The Technologies and Costs document (U.S. EPA, 2024i) and individual WBS models in the rule docket provide additional information. The component-level costs approximate national average costs, which can over- or under-estimate costs at systems affected by the final rule.
Compliance forecast	Uncertain	The forecast probabilities are based on historical full-scale compliance actions. Site-specific water quality conditions, changes in technology, and changes in market conditions can result in future technology selections that differ from the compliance forecast.

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[22] Note that the EPA does compute per household technology cost values in the separate national small system affordability determination analysis. These household values are distinct from the values generated in the national cost estimates as they include only small system compliance technology cost. For three small system size categories (systems serving 25-500, 501-3,300, and 3,301-10,000) The EPA estimates a per household treatment technology cost range including the minimum and maximum cost values. These cost estimates are based on system characteristics, contaminant reduction requirements, and technology efficacy, across the set of small system compliance technology options. See Chapter 9.12 for additional information on the national small system affordability determination.

Table 5-21: Limitations that Apply to the Cost Analysis for the Final PFAS Rule

Uncertainty/ Assumption	Effect on Quantitative Analysis	Notes
TOC concentration	Uncertain	The randomly assigned values from the two national distributions are based on a limited dataset. Actual TOC concentrations at systems affected by the final rule can be higher or lower than the assigned values.
Insufficient UCMR 3 data for PFBS and PFNA and no UCMR 3 data for HFPO-DA were available to incorporate into the Bayesian hierarchical occurrence model	Underestimate	The final rule regulates PFBS, PFNA, and HFPO-DA in addition to the PFAS modeled in the primary analysis (PFOA, PFOS and PFHxS). In instances when concentrations of PFBS, PFNA, and/or HFPO-DA are high enough to cause or contribute to HI exceedances or PFNA and/or HFPO-DA are high enough to cause individual MCL exceedances, the modeled costs in the primary analysis may be underestimated. If these PFAS occur in isolation at levels that affect treatment decisions, or if they occur in sufficient concentration to result in an exceedance when the concentration of PFHxS alone would be below the HBWC, then costs would be underestimated. Note that the EPA has conducted an analysis of and considered the potential changes in national level treatment cost associated with the occurrence of PFBS, PFNA, and HFPO-DA, which is discussed in detail in Appendix N, Section N.3.
POU not included in compliance forecast	Overestimate	If POU devices can be certified to meet concentrations that satisfy the final rule, then small systems may be able to reduce costs by using a POU compliance option instead of centralized treatment or source water changes.
Process wastes not classified as hazardous	Underestimate	The national cost analysis reflects the assumption that PFAS-contaminated wastes are not considered RCRA regulatory or characteristic hazardous wastes. To address stakeholder concerns, including those raised during the SBREFA process, the EPA conducted a sensitivity analysis with an assumption of hazardous waste disposal for illustrative purposes only. As part of this analysis, the EPA generated a second full set of unit cost curves that are identical to the curves used for the national cost analysis with the exception that spent GAC and spent IX resin are considered hazardous. The EPA acknowledges that if in the future PFAS-contaminated wastes require handling as hazardous wastes, the residuals management costs in the WBS treatment cost models are expected to be higher. See Appendix N, Section N.2 for a sensitivity analysis describing the potential increase in costs associated with hazardous waste disposal at 100 percent of systems treating for PFAS. The costs estimated in Appendix N, Section N.2 are consistent with EPA OLEM's "Interim Guidance on the Destruction and Disposal of Perfluoroalkyl and Polyfluoroalkyl Substances and Materials Containing Perfluoroalkyl and Polyfluoroalkyl Substances" (U.S. EPA, 2020b).

Table 5-21: Limitations that Apply to the Cost Analysis for the Final PFAS Rule

Uncertainty/ Assumption	Effect on Quantitative Analysis	Notes
Population served held constant over time	Uncertain	All PWS populations served were held constant over the period of analysis as not all locations have reliable information on population changes over time. If population served by affected PWSs increases (or decreases), then the estimated costs are likely underestimated (or overestimated).

Abbreviations: WBS – work breakdown structure; TOC – total organic carbon; HFPO-DA – hexafluoropropylene oxide dimer acid; PFAS – per and polyfluoroalkyl substances; PFBS – perfluorobutanesulfonic acid; PFNA – perfluorononanoic acid; PFHxS – perfluorohexanesulfonic acid; MCL – maximum contaminant level; HI – hazard index; HBWC- health based water concentration; POU – point-of-use; RCRA – Resource Conservation and Recovery Act; SBREFA – Small Business Regulatory Enforcement Fairness Act; GAC – granulated activated carbon; IX – ion exchange; OLEM – Office of Land Energy and Management.

6 Benefits Analysis

6.1 Introduction

This chapter discusses the potential quantified and nonquantifiable[23] benefits to human health resulting from changes in PFAS levels in drinking water due to implementation of the final rule, as well as several regulatory alternatives. The EPA's quantification of health benefits resulting from reduced PFAS exposure in drinking water was driven by PFAS occurrence estimates, pharmacokinetic (PK) model availability, information on exposure-response relationships, and economic data to monetize the impacts. The EPA either quantitatively assesses or qualitatively discusses health endpoints associated with exposure to PFAS. The EPA assesses potential benefits quantitatively if there is evidence of an association between PFAS exposure and health effects if it is possible to link the outcome to risk of a health effect, and if there is no overlap in effect with another quantified endpoint in the same outcome group. Only a subset of the avoided morbidity and mortality stemming from reduced PFAS levels in drinking water can be quantified and monetized. The monetized benefits evaluated in the economic analysis for the final rule include changes in human health risks associated with CVD and infant birth weight from reduced exposure to PFOA and PFOS in drinking water and RCC from reduced exposure to PFOA.[24] The EPA also quantified benefits from reducing bladder cancer risk due to the co-removal of non-PFAS pollutants via the installation of drinking water treatment, discussed in greater detail in Section 6.7. The EPA was not able to quantify or monetize other benefits, including those related to possible immune, hepatic, endocrine, metabolic, reproductive, musculoskeletal, or other outcomes. The EPA discusses these benefits qualitatively in more detail below in Section 6.2 of the economic analysis.

The EPA analyzes the quantified costs and benefits of the final rule MCLs of 4.0 ppt for PFOA, 4.0 ppt for PFOS, and a unitless HI of 1 for the group including PFNA, HFPO-DA, PFHxS, and PFBS. The analysis of costs and benefits associated with the HI also express the costs and benefits of the individual MCLs for PFNA, HFPO-DA, and PFHxS. Additionally, the EPA presents the incremental costs and benefits associated with three regulatory alternative MCLs for PFOA and PFOS at 4.0 ppt, 5.0 ppt, and 10.0 ppt, referred to as Options 1a through 1c respectively. As discussed in Section 2.1, the regulatory options include treatment thresholds that would reduce PFAS levels in finished drinking water by various amounts. The change in PFAS levels at a particular water system depends on baseline PFAS levels estimated using the occurrence model (Section 4.4) and the PFAS treatment threshold specified under each regulatory alternative.

The EPA notes that the quantified benefits alone of this analysis are a significant underestimate of the total benefits expected to result from this rule because the EPA was not able to quantitatively monetize all benefits. Hence, as mandated by SDWA Section 1412(b)(3)(C), the

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[23] Nonquantifiable benefits are discussed qualitatively.

[24] Benefits to human health in terms of reduced liver cancer incidence are described in Appendix O. This analysis is presented as a supplemental analysis for the final rule in response to public comments received on the proposed rule requesting that the EPA quantify additional health benefits.

EPA has considered both quantifiable and nonquantifiable benefits in informing its decision making that the costs of this rule are clearly justified by the benefits.

6.1.1 Chapter Overview

Section 6.2 provides an overview of the health benefit categories considered in the analysis of reductions of PFAS in drinking water. In addition to describing the benefits that the EPA is able to quantify, this section includes a robust qualitative discussion of nonquantifiable benefits. Because of the broad adverse health impacts of PFAS on many endpoints, the nonquantifiable benefits of this final rule are likely substantial. Section 6.3 describes the application of the EPA's PK models for PFAS to estimate changes in blood serum concentrations under each regulatory alternative. Section 6.4 presents the methodology and results of the impacts of the PFAS regulatory alternatives on a subset of developmental outcomes, namely infant birth weight. Section 6.5 presents the methodology and results of the impacts of the PFAS regulatory alternatives on CVD incidence. Section 6.6 presents the methodology and results of the impacts of the PFAS regulatory alternatives on the incidence of RCC, one of the cancers associated with PFOA exposure. Section 6.7 presents the methodology and results of the impacts of the PFAS regulatory alternatives on DBP formation and the associated incidence of bladder cancer. Finally, Section 6.8 describes limitations and uncertainties of the benefits analyses.

6.1.2 Uncertainty Characterization

The EPA characterizes sources of uncertainty in its analysis of potential quantified benefits resulting from changes in PFAS levels in drinking water. The analysis reports uncertainty bounds for benefits estimated in each health endpoint category modeled for the final rule. Each lower (upper) bound value is the 5th (95th) percentile of the category-specific benefits estimate distribution represented by 4,000 Monte Carlo draws. Table 6-1 provides an overview of the specific sources of uncertainty that the EPA quantified in this benefits analysis. In addition to these sources of uncertainty, reported uncertainty bounds also reflect the following upstream sources of uncertainty: baseline PFAS occurrence (Section 4.4), affected population size and demographic composition (Section 4.3), and the magnitude of PFAS concentration reductions (Section 4.4). These analysis-specific sources of uncertainty are further described in Appendix L.

Table 6-1: Quantified Sources of Uncertainty in Benefits Estimates

Source	Description of Uncertainty
Health effect slope factors	The slope factors that express the effects of serum PFOA, serum PFOS, and THM4 on health outcomes (birth weight, CVD,[a] RCC, and bladder cancer) are based either on the EPA meta-analyses or medium- or high-confidence studies that provide a central estimate and a confidence interval. To characterize uncertainty, the EPA assumed that these slope factors have a normal distribution with a mean set at the central estimate and the standard deviation set at the estimated standard error.
RCC risk reduction cap	The EPA implemented a cap on the cumulative RCC risk reductions due to reductions in serum PFOA based on the population attributable fraction (PAF) estimates for a range of cancers and environmental contaminants. This parameter is treated as uncertain; its uncertainty is characterized by a log-uniform distribution with a minimum set at the smallest PAF estimate identified in the literature and a maximum set at the largest PAF estimate identified in the literature. The central estimate for the PAF is the mean of this log-uniform distribution.

Abbreviations: PFAS – per- and polyfluoroalkyl substances; PFOA – perfluorooctanoic acid; PFOS – perfluorooctane sulfonic acid; RCC – renal cell carcinoma; PAF – population attributable fraction, THM4 – four regulated trihalomethanes.

Note:

[a] The slope factors contributing to the CVD benefits analysis include the relationship between total cholesterol and PFOA and PFOS, and the relationship between blood pressure and PFOS.

The EPA did not characterize the following sources of potentially quantifiable uncertainty in the national-level quantified benefits analysis: U.S. population life tables (see Section 6.1.4), annual all-cause and health outcome-specific incidence and mortality rates, coefficients of the CVD risk model linking total cholesterol (TC), high-density lipoprotein cholesterol (HDLC), and blood pressure (BP) to cardiovascular event incidence (Goff et al., 2014), CVD risk model predictors (e.g., share of smokers) estimated from health survey data, prevalence of CVD event history in the U.S. population, distribution of CVD events by type, the estimated infant mortality-birth weight slope factor (See Section 6.4.3.1), state-level distributions of infant births and infant deaths over discrete birth weight ranges, the 200-g cap on birth weight changes estimated under the rule, cost of illness estimates for all modeled non-fatal health outcomes, the Value of Statistical Life reference value, the Value of Statistical Life income elasticity value used to approximate the Value of Statistical Life income growth adjustment, and the gross domestic product per capita projection used for the Value of Statistical Life income growth adjustment (see Appendix J). The EPA expects that the sources listed in Table 6-1, in addition to uncertainty surrounding the estimates of PFAS occurrence, affected population size, and the magnitude of PFAS reduction, account for a substantial portion of the uncertainty in the benefits analysis.

6.1.3 Summary of Quantified National Benefits Estimates of the Final Rule

This section provides summary outputs for the benefits analysis of the final rule as well as Options 1a-c. Total annual benefits include human health risk reduction benefits for the health outcomes listed in Section 6.1.1. The EPA annualized benefit values for each endpoint at a 2 percent discount rate. Both the expected value and the 90% confidence interval (CI) are provided.

As discussed in Section 2.1, for purposes of this analysis, the EPA is considering the benefits analysis to be representative of the final rule utilizing individual MCLs for PFOA, PFOS, PFNA, HFPO-DA, and PFHxS and a group MCL based on a HI for PFNA, HFPO-DA, PFHxS, and PFBS.

Table 6-2: National Annualized Benefits, Final Rule (PFOA and PFOS MCLs of 4.0 ppt each, PFHxS, PFNA, and HFPO-DA MCLs of 10 ppt each and HI of 1) (Million $2022)

	2% Discount Rate		
	5th Percentile[a]	**Expected Value**	**95th Percentile[a]**
Annualized CVD Benefits	$140.66	$606.09	$1,069.40
Annualized Birth Weight Benefits	$124.85	$209.00	$292.78
Annualized RCC Benefits	$61.33	$353.90	$883.55
Annualized Bladder Cancer Benefits	$300.64	$380.41	$463.74
Total Annualized Rule Benefits[b]	**$920.91**	**$1,549.40**	**$2,293.80**

Abbreviations: CVD – cardiovascular disease; HI – hazard index; RCC – renal cell carcinoma.

Note: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates. 5th and 95th percentile values for total rule benefits are not additive across benefit category as the categories are not completely correlated. Quantifiable benefits are increased under final rule table results relative to the other options presented because of modeled PFHxS occurrence, which results in additional quantified benefits from co-removed PFOA and PFOS.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.

[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

When using willingness to pay instead of cost of illness values to monetize cancer morbidity impacts, annualized RCC benefits are $360.97 million, whereas annualized bladder cancer benefits are $456.28 million (see Appendix O). If used in the national benefits analysis, these willingness to pay estimates would result in approximately 83 million dollars additional quantified benefits from those presented in Table 6-2, resulting in an increase in quantified benefits of approximately 5.4%.

Additionally, in Appendix O, the EPA presents several sensitivity analyses, including an analysis evaluating liver cancer benefits. Quantified benefits associated with reduction of liver cancer from PFOS could increase total benefits from $1,549.40 million to $1,554.19 million (see Appendix O).

Table 6-3: National Annualized Benefits, Option 1a (PFOA and PFOS MCLs of 4.0 ppt) (Million $2022)

	2% Discount Rate		
	5th Percentile[a]	**Expected Value**	**95th Percentile**[a]
Annualized CVD Benefits	$140.12	$602.72	$1,059.60
Annualized Birth Weight Benefits	$124.82	$207.82	$291.00
Annualized RCC Benefits	$60.90	$351.79	$877.47
Annualized Bladder Cancer Benefits	$301.06	$380.41	$462.73
Total Annualized Rule Benefits[b]	**$913.05**	**$1,542.74**	**$2,280.10**

Abbreviations: CVD – cardiovascular disease; RCC – renal cell carcinoma.

Note: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates. 5th and 95th percentile values for total rule benefits are not additive across benefit category as the categories are not completely correlated.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.

[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

Table 6-4: National Annualized Benefits, Option 1b (PFOA and PFOS MCLs of 5.0 ppt) (Million $2022)

	2% Discount Rate		
	5th Percentile[a]	**Expected Value**	**95th Percentile**[a]
Annualized CVD Benefits	$119.18	$513.27	$900.13
Annualized Birth Weight Benefits	$107.34	$178.97	$250.00
Annualized RCC Benefits	$48.41	$290.72	$730.99
Annualized Bladder Cancer Benefits	$246.48	$313.88	$383.32
Total Annualized Rule Benefits[b]	**$768.55**	**$1,296.84**	**$1,919.30**

Abbreviations: CVD – cardiovascular disease; RCC – renal cell carcinoma.

Note: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates. 5th and 95th percentile values for total rule benefits are not additive across benefit category as the categories are not completely correlated.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.

[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

Table 6-5: National Annualized Benefits, Option 1c (PFOA and PFOS MCLs of 10.0 ppt) (Million $2022)

	2% Discount Rate		
	5th Percentile[a]	**Expected Value**	**95th Percentile[a]**
Annualized CVD Benefits	$66.97	$267.56	$469.05
Annualized Birth Weight Benefits	$60.24	$98.97	$137.75
Annualized RCC Benefits	$21.20	$137.30	$352.07
Annualized Bladder Cancer Benefits	$120.97	$160.62	$202.14
Total Annualized Rule Benefits[b]	**$397.28**	**$664.45**	**$970.70**

Abbreviations: CVD – cardiovascular disease; RCC – renal cell carcinoma.

Note: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates. 5th and 95th percentile values for total rule benefits are not additive across benefit category as the categories are not completely correlated.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.

[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

6.1.4 Life Table Modeling Background

The EPA uses a life table modeling approach to evaluate reductions in CVD and cancer risk. This approach allows for internally consistent estimation of the path-dependent health effects for regulatory alternatives, including annual incidence of CVD events or cancers among those without prior history of these conditions, which is dependent on the population prevalence of these chronic conditions and survival over time.

The life table is a statistical tool used to analyze the mortality experience of a population over time. Specifically, using data on the age-specific probability of death and the initial population size (e.g., 100,000 persons), the life table computes the number of persons surviving to a specific age, the number of deaths occurring at a given age, the number of person-years lived at a given age, the number of person-years lived beyond a given age, and age-specific life expectancy. The details of standard life table calculations can be found in Anderson (1999).

The life table modeling approach extends the standard life table calculations to characterize populations with respect to their chronic condition status and estimate transitions into the subpopulation affected by the chronic condition.[25] The EPA has previously used life table approaches in regulatory analyses, including the analysis of lead-associated health effects in the 2015 *Benefit and Cost Analysis for the Effluent Limitations Guidelines, Standards for the Steam*

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[25] For example, a benefits model that evaluates the impact of contaminant exposure on incidence of cancer—a chronic condition—would need to estimate the number of persons who are cancer free and, therefore, are eligible for the estimation of new cancer risk (i.e., the risk of transition into the subpopulation affected by the chronic condition).

Electric Power Generating Point Source Category (U.S. EPA, 2015), and $PM_{2.5}$-related health effects in revisions to the National Ambient Air Quality Standards for ground-level ozone (U.S. EPA, 2008). Other examples of the use of a life table approach among federal agencies include the EPA's analysis of Benefits and Costs of the Clean Air Act from 1990 to 2020 (U.S. EPA, 2011a) and the Occupational Safety and Health Administration (OSHA) assessment of lifetime excess lung cancer, nonmalignant respiratory disease mortality, and silicosis risks from exposure to respirable crystalline silica (OSHA, 2010; OSHA, 2016). Additionally, the agency sought advice from the EPA SAB on the use of the life table in this application and they supported this approach (U.S. EPA, 2022i). See Appendix G for details on application of the life table for the CVD benefits analysis. See Appendix H for details on application of the life table for cancer benefits analyses.

6.2 Overview of Benefit Categories

The EPA's decision to quantify health benefits resulting from reduced PFAS exposure in drinking water is driven by the availability of PFAS-related occurrence estimates, PK models, and information on exposure-response relationships. In this benefits analysis, the EPA either quantitatively assesses or qualitatively discusses the health endpoints associated with exposure to PFAS; the EPA assesses potential benefits quantitatively if (1) there is indicative evidence of a relationship between exposure and a health effect response, (2) it is possible to link the health outcome (e.g., CVD) to risk of a health effect (e.g., increased total cholesterol), and (3) there is no overlap in effect with another quantified endpoint in the same outcome group.

The EPA describes occurrence modeling information in Section 4.4. Table 6-6 presents an overview of the categories of health benefits expected to result from the implementation of treatment that reduces PFAS levels in drinking water. The PFAS compounds that the EPA identified as having indicative evidence linking exposure to a particular health endpoint, as well as compounds having reliable PK models estimating the distribution to PFAS compounds throughout the body, include PFOA, PFOS, and PFNA.[26]

As seen in Table 6-6, only a small subset of the potential health effects of reduced PFAS levels in drinking water can be quantified and monetized. The monetized benefits evaluated in the national-level quantified analysis for the final rulemaking include CVD, infant birth weight, and RCC. The EPA also quantified benefits from reducing bladder cancer risk due to the reduction of DBP formation as a result of the co-removal of organic carbon via the installation of additional treatment for PFAS (Cantor et al., 1998; Crittenden et al., 1993; Regli et al., 2015; Weisman et al., 2022). The EPA also quantified benefits associated with PFOS effects on liver cancer and PFNA effects on birth weight in sensitivity analyses, available in appendices O and K, respectively. The EPA notes that the agency anticipates additional benefits resulting from installing drinking water treatment for PFAS chemicals and the subsequent removal of co-occurring non-PFAS contaminants, including source water metals (e.g., chromium (VI)), organic regulated and unregulated contaminants, (e.g., cyanotoxins (Foreman et al., 2021)), and certain pesticides. The EPA was not able to quantify or monetize other benefits, including those related to possible immune, hepatic, endocrine, metabolic, reproductive, musculoskeletal, many cancers,

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[26] The EPA relies on the serum PFNA calculator from Lu and Bartell (2020). PFNA effects are described as part of a sensitivity analysis for birth weight-related benefits in Appendix K.

or other outcomes discussed in Section 6.1.2. The EPA discusses these benefits qualitatively in Sections 6.2.2.2 and 6.2.4.

Table 6-6: Overview of Health Benefits Categories Considered in the Analysis of Changes in PFAS Drinking Water Levels

Health Outcome		PFAS Compound[a,b,d]		Benefits Analysis	
Category	**Endpoint**	**PFOA**	**PFOS**	**Discussed Quantitatively**	**Discussed Qualitatively**
Lipids	Total cholesterol (TC)	X	X	X	
	High-density lipoprotein cholesterol (HDLC)	X[c]	X[c]	X	
	Low-density lipoprotein cholesterol (LDLC)	X	X		X
CVD	Blood pressure (BP)		X	X	
Developmental	Birth weight	X	X	X	
	Small for gestational age (SGA), non-birth weight developmental	X			X
Hepatic	Alanine transaminase (ALT)	X	X		X
Immune	Antibody response (tetanus, diphtheria)	X	X		X
Metabolic	Leptin	X			X
Musculoskeletal	Osteoarthritis, bone mineral density	X			X
Cancer	Renal Cell Carcinoma (RCC)	X		X	
	Liver		X	X[e]	
	Testicular	X			X

Abbreviations: PFAS – per- and polyfluoroalkyl substances; PFOA – perfluorooctanoic acid; PFOS – perfluorooctane sulfonic acid

Notes:

[a] Fields marked with "X" indicate the PFAS compound for which there is evidence of an association with a given health outcome in humans.

[b] Outcomes with indicative evidence of an association between a PFAS compound and a health outcome are assessed quantitatively unless (1) there is an overlap within the same outcome group (e.g., low density lipoprotein cholesterol overlaps with total cholesterol and small for gestational age overlaps with low birth weight), or (2) it is not possible to link the outcome to the risk of the health effect (e.g., evidence is inconclusive regarding the relationship between PFOS exposure, leptin levels and associated health outcomes). Such health outcomes are discussed qualitatively.

[c] Although evidence of associations between HDLC and PFOA and PFOS was mixed, certain individual studies reported robust associations in general adult populations (See Section 6.2.2.1.2 on Cardiovascular Effects). Based on comments and recommendations from the EPA SAB (U.S. EPA, 2022i), the EPA assessed HDLC in a sensitivity analysis (see Appendix K).

[d] Note that only PFOA and PFOS effects were modeled in the assessment of benefits under the final rule. For another PFAS in the rule, PFNA, the best available finalized analysis is based on studies published before 2018 (ATSDR, 2021). The EPA notes that new evidence since the release of the current, best available peer reviewed scientific assessment for PFNA (ATSDR, 2021) provides further justification for the EPA's analysis of potential economic benefits of PFNA exposure reduction and avoided birth weight effects. More recent epidemiological studies that evaluated PFNA and birth weight, including key studies modeled for PFOA and PFOS (Sagiv et al., 2018; Wikström et al., 2020), as well as a recently published meta-analysis of mean birth weight that indicates the birth weight results for PFNA are robust and consistent, even if associations in some studies may be small in magnitude (Wright et al., 2023). PFNA was modeled in a sensitivity analyses of birth weight benefits. This modeling relied on epidemiological studies published before 2018, representing the best available finalized human health analysis of PFNA (ATSDR, 2021) and the approach by Lu and Bartell (2020) was used for estimating PFNA blood serum levels resulting from PFNA exposures in drinking water (see Appendix K).

Table 6-6: Overview of Health Benefits Categories Considered in the Analysis of Changes in PFAS Drinking Water Levels

Health Outcome		PFAS Compound[a,b,d]		Benefits Analysis	
Category	**Endpoint**	**PFOA**	**PFOS**	**Discussed Quantitatively**	**Discussed Qualitatively**

[e]Liver cancer benefits are not included in the national-level quantified benefits analysis. See Appendix O for the liver cancer benefits analysis results.

In Table 6-7, the EPA presents an overview of the epidemiology and toxicology evidence regarding the effects of exposure to PFAS compounds on health outcomes that were examined in various EPA and Agency for Toxic Substances and Disease Registry's (ATSDR) assessments. Health outcomes are classified as having:

- No evidence of an association[27] (signified with a dot in the table);
- Evidence of an association noted as suggestive or slight (signified with an X in the table); or
- Indicative evidence of an association (signified with a green-highlighted X in the table).

Health outcomes that have indicative (likely) associations and that are quantified in the benefits analysis for the final rule are signified with X*. The EPA further describes the associations, and supporting evidence of associations, in Section 6.2.2 for PFOA and PFOS and in Section 6.2.4 for additional PFAS compounds.

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[27] No evidence of an association is listed in instances where an absence of evidence precludes definitive conclusions about the relationship between exposure and a given health effect or when there is evidence demonstrating that exposure does not result in a given health effect.

Table 6-7: Overview of Epidemiology and Toxicology Evidence of PFAS Effects on Health Outcomes

PFAS	Evidence Type	Health Outcomes																			Data Source(s)	Notes
		Lipids			CVD	Developmental		Hepatic	Immune	Endocrine	Metabolic	Renal	Reproductive	Musculoskeletal	Hematologic	Other non-cancer	Cancer					
		TC	HDLC	LDLC	BP[a] (human) Heart histopathology (animal)	Birth weight	SGA, non-birth weight developmental	ALT (human) Organ weight, cell death (animal)	AbR[a] (tetanus, diphtheria) (human) Various endpoints (animal)	Thyroid hormone disruption	Leptin, body weight (human)Body fat, body weight (animal)	Uric acid (human) Organ weight (animal)	Gestational hypertension/pre-eclampsia (human)Various endpoints (animal)	Osteoarthritis, bone mineral density	Vitamin D levels, hemoglobin levels, albumin levels	Other non-cancer	RCC[a]	Testicular	Liver	Other		
PFOA	Epi	X*	•	X	X	X*	X	X	X	X	X	•	X	X	•	•	X*	X[b]	•	X	U.S. EPA 2024b, 2024d; ATSDR 2021; NASEM, 2022	Other non-cancer: neurological effects, respiratory effects, gastrointestinal
	Tox	X	X	X	•	X*	X	X	X	X	•	•	X	X	•	X	•	•	X	•	U.S. EPA 2024b, 2024d; ATSDR 2021	Other non-cancer: neurological effects, respiratory effects, gastrointestinal
PFOS	Epi	X*	•	X	X	X*[c]	•	X	X	X	•	•	X[d]	•	•	•	X	•	X	X	U.S. EPA 2024a, 2024c; ATSDR 2021; NASEM, 2022	Other non-cancer: neurological effects, gastrointestinal
	Tox	•	•	•	•	X	X	X	X	X	•	•	X	•	•	X	•	•	X	X	U.S. EPA 2024a, 2024c; ATSDR 2021	Other non-cancer: neurological effects, gastrointestinal
PFBA	Epi	•	•	•	•	•	•	•		•		•	•								IRIS Assessment 2022; ATSDR 2021; NASEM, 2022	No associations in humans
	Tox	•			•		X	X	•	X	•	•	•	•	•	•					IRIS Assessment 2022; ATSDR 2021	Other non-cancer: ocular, respiratory (ATSDR)
PFNA	Epi	X	•	X	•	X[e]	•	•	•	•	•	•	•	•			•				ATSDR 2021; NASEM, 2022	Other non-cancer: respiratory effects
	Tox	X	•	•	•	•	X	X	X	X	•		•		X	•					ATSDR 2021	Other non-cancer: general toxicity
PFDA	Epi	X	•	X	•	X	X	X	X	•	X	•	•	•		X				•	ATSDR 2021; NASEM, 2022	
	Tox	•	•	•	•	X	X	X	X	•	•	•	X		•	X	•	•	•	•	ATSDR 2021	
PFHxS	Epi	•	•	•	•	X	•	•	X	•	•	•	•	•		•	•				ATSDR 2021; NASEM, 2022	
	Tox	X	•		•	X	•	X	•	X	•	•	•	•	X	•					ATSDR 2021	Other non-cancer: respiratory effects
PFHxA	Epi				•			•		•	•	•				•				•	IRIS Assessment 2023; ATSDR 2021; NASEM, 2022	No associations in humans
	Tox	•	•	•	•	X	•	X	•	X	•	•	•	•	X	•	•				IRIS Assessment 2023; ATSDR 2021	Other non-cancer: nervous (IRIS, ATSDR), respiratory (ATSDR)
PFBS	Epi	•	•	•	•						•	•	•								EPA Human Health Toxicity Study 2021; ATSDR 2021; NASEM, 2022	No associations in humans
	Tox	X	•	•	•	•	X	•	X	X	•	X	•	•	X	•					EPA Human Health Toxicity Study 2021; ATSDR 2021	Other non-cancer: respiratory effects (ATSDR)

PFAS	Evidence Type	Health Outcomes																			Data Source(s)	Notes
		Lipids			CVD	Developmental		Hepatic	Immune	Endocrine	Metabolic	Renal	Reproductive	Musculoskeletal	Hematologic	Other non-cancer	Cancer					
		TC	HDLC	LDLC	BP[a] (human) Heart histopathology (animal)	Birth weight	SGA, non-birth weight developmental	ALT (human) Organ weight, cell death (animal)	AbR[a] (tetanus, diphtheria) (human) Various endpoints (animal)	Thyroid hormone disruption	Leptin, body weight (human)Body fat, body weight (animal)	Uric acid (human) Organ weight (animal)	Gestational hypertension/pre-eclampsia (human)Various endpoints (animal	Osteoarthritis, bone mineral density	Vitamin D levels, hemoglobin levels, albumin levels	Other non-cancer	RCC[a]	Testicular	Liver	Other		
PFHpA	Epi	•	•	•	•	•	•	•	•							•	•			•	ATSDR 2021; NASEM, 2022	No associations in humans
	Tox																				ATSDR 2021	
PFUnA	Epi	•	•	•	•	X	•	•	•	•	•		•			•				•	ATSDR 2021; NASEM, 2022	
	Tox					X		•				•			•						ATSDR 2021	
PFDoDA	Epi	•	•	•	•	•	•	•	•	•	•	•	•				•			•	ATSDR 2021; NASEM, 2022	
	Tox	•			•	•	•	•			•	•	•		•						ATSDR 2021	
FOSA	Epi				•	•	•		•		•		•			•	•			•	ATSDR 2021; NASEM, 2022	
	Tox							•			•										ATSDR 2021	
HFPO-DA[f]	Epi																				EPA HFPO-DA 2021 final toxicity assessment	No data from epidemiology studies
	Tox	X		•		X		🟩 X	🟩 X	X		🟩 X	•		🟩 X				🟩 X		EPA HFPO-DA 2021 final toxicity assessment	

Notes:

•	Health outcomes examined, no evidence of associations (also noted as inadequate, or equivocal evidence).
X	Health outcomes examined, slight or suggestive evidence of associations.
🟩 X	Health outcomes examined, moderate or indicative evidence of associations (also noted as supports a hazard in IRIS assessments, evidence indicates, or evidence demonstrates).
X*	Health outcomes quantified in benefits analyses, indicative evidence of associations.
[Blank cell]	Health outcome was not examined.
[a]	AbR: antibody response; BP: blood pressure; Epi: epidemiology; Tox: toxicology; RCC: renal cell carcinoma.
[b]	Supported based on PFOA HESD (2016) and Bartell et al. (2021) meta-analysis.
[c]	Supported by Dzierlenga et al. (2020) meta-analysis.
[d]	Also supported by recent meta-analysis from Gao et al. (2021) (PFOS and preeclampsia risk).
[e]	Also supported by recent meta-analysis from Wright et al. (2023) (PFNA and birth weight).

6.2.1 Availability of Pharmacokinetic (PK) Models

PK models are tools for quantifying the relationship between external measures of exposure and internal measures of dose. The EPA evaluated existing PFOA and PFOS PK models for their utility in predicting internal doses for use in both cancer and non-cancer dose-response assessments (U.S. EPA, 2024e; U.S. EPA, 2024f). PFOA and PFOS PK models typically take one of three forms:

- **Classical compartment models**, where modelers define the body as a one- or two-compartment system with volumes and intercompartmental transfer fit specifically to the PFAS PK dataset. The most common approach for prediction of serum PFAS levels is to apply a simple single-compartment model.
- **Modified compartment models**, where modelers attempt to characterize absorption, distribution, metabolism, and/or excretion through protein-binding, cardiac output, and known renal elimination. These models also rely on fitting PFAS data to non-physiological parameters.
- **Physiological-based pharmacokinetic (PBPK) models**, where tissues and organs of the body are described as physiological-based compartments. In these models, transport between compartments is informed by measures of blood flow and tissue perfusion. These models are fit to time-course concentration data.

The EPA's *Final Human Health Toxicity Assessment for PFOA* (U.S. EPA, 2024f) and *Final Human Health Toxicity Assessment for PFOS* (U.S. EPA, 2024e)[28] describe existing PFOA and PFOS PK models and modifications made to existing PK models to derive points of departure in the assessments. Briefly, the EPA updated a modified single-compartment PK model for adult males and females to estimate blood serum PFOA and PFOS concentrations. These models are described in Section 4.1.3.2 of U.S. EPA (2024e; 2024f), and the application of these models in health risk benefits modeling is described in Section 6.3.

6.2.2 Benefits of PFOA and PFOS Exposure Reduction

This section provides an overview of the potential health benefits of reduced exposure to PFOA and PFOS in drinking water. These benefits are expected to be realized as avoided adverse health effects as a result of the final NPDWR, in addition to the benefits that the EPA has quantified. The EPA identified a wide range of potential health effects associated with exposure to PFOA and PFOS using five comprehensive federal government health effects assessments that summarize the recent literature on PFAS (mainly PFOA and PFOS, although many of the same health effects have been observed for the other PFAS in this rule) exposure and its health impacts: the EPA's Health Effects Support Document for PFOA and Health Effects Support Document for PFOS, hereafter referred to as the EPA HESDs (U.S. EPA, 2016e; U.S. EPA, 2016f); the EPA's Final Human Health Toxicity Assessments for PFOA and PFOS (U.S. EPA, 2024e; U.S. EPA, 2024f); and the U.S. Department of Health and Human Services ATSDR

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[28] For brevity, these documents are described throughout as the EPA's Final Human Health Toxicity Assessments for PFOA and PFOS.

Toxicological Profile for Perfluoroalkyls (ATSDR, 2021). Each source presents comprehensive literature reviews on adverse health effects associated with PFOA and PFOS.

The most recent literature reviews on PFAS exposures and health impacts, which are included in the EPA's Final Human Health Toxicity Assessments for PFOA and PFOS, describe the weight of evidence supporting PFOA and PFOS associations with health outcomes as either demonstrative, indicative (likely), suggestive, inadequate, or strong evidence supportive of no effect according to the evidence integration judgments outlined in the Office of Research and Development (ORD) Staff Handbook for Developing IRIS Assessments (U.S. EPA, 2022g; U.S. EPA, 2024e; U.S. EPA, 2024f). For the purposes of the reviews conducted to develop the Final Human Health Toxicity Assessments for PFOA and PFOS, an association is deemed demonstrative when there is a strong evidence base demonstrating that the chemical exposure causes a health effect in humans. The association is deemed indicative (likely) when the evidence base indicates that the chemical exposure likely causes a health effect in humans, although there might be outstanding questions or limitations that remain, and the evidence is insufficient for the higher conclusion level. The association is suggestive if the evidence base suggests that the chemical exposure might cause a health effect in humans, but there are very few studies that contributed to the evaluation, the evidence is very weak or conflicting, or the methodological conduct of the studies is poor. The association is inadequate if there is a lack of information or an inability to interpret the available evidence (e.g., findings across studies). The association supports no effect when extensive evidence across a range of populations and exposure levels has identified no effects/associations. Note that the EPA considered information available as of September 2023 for the analyses presented herein. Section 6.2.2.1 discusses PFOA and PFOS-related health effects that were considered quantitatively (modeled and monetized) in the benefits analysis, while Section 6.2.2.2 discusses PFOA and PFOS-related health effects that were considered only qualitatively in the benefits analysis. These sections specify whether evidence is based on animal (toxicology) or human (epidemiology) studies, or both.

6.2.2.1 Quantitative Benefits of PFOA and PFOS Exposure Reduction

In this section, the EPA discusses some of the health benefits expected to result from reduced exposure to PFOA and PFOS in drinking water. These benefits are expected to be realized as avoided adverse health effects as a result of the final NPDWR and are quantified in Sections 6.4, 6.5, and 6.6 respectively.

6.2.2.1.1 Developmental Effects

Exposure to PFOA and PFOS is linked to developmental effects such as decreased infant birth weight, birth length, head circumference at birth, and other effects (Steenland et al., 2018; Dzierlenga et al., 2020; Verner et al., 2015; U.S. EPA, 2016e; U.S. EPA, 2016f; Negri et al., 2017; Waterfield et al., 2020; U.S. EPA, 2024e; U.S. EPA, 2024f). Low birth weight (LBW) is an important health outcome because it is a significant factor in survival rates and medical care costs among infants (ATSDR, 2021). Infants are exposed prenatally to PFOA and PFOS through maternal serum via the placenta (U.S. EPA, 2024e, U.S. EPA, 2024f).

Because data on the cost of incremental changes in birth weight are available from Klein and Lynch (2018), the EPA selected decreased birth weight as a key developmental health effect when assessing the economic impacts of reduced PFOA and PFOS exposures. Epidemiology studies on PFOA were associated with an increased risk of decreased BW in infants with PFOA exposures (U.S. EPA, 2024f). Similarly, epidemiology studies on PFOS were associated with an increased risk of decreased BW in infants with increasing PFOS exposures (U.S. EPA, 2024e). As described in the toxicity assessments for PFOA and PFOS (see Section 3.4.4.1.4 of the final toxicity assessments; U.S. EPA, 2024e; U.S. EPA, 2024f), many epidemiology studies evaluating the association between maternal serum PFOA/PFOS and birth weight reported inverse associations (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).[29] Toxicology studies on PFOA further supported an association between decreased offspring weight and PFOA exposure; several studies conducted on rodents showed decreased fetal and pup weight with gestational PFOA exposure (U.S. EPA, 2024f). Toxicology studies also reported that increased exposure to PFOS was associated with decreased body weight in rodent fetuses and pups (U.S. EPA, 2024e). For additional details on developmental effects studies and their individual outcomes, see Chapter 3.4.4 (Developmental) in U.S. EPA (2024e) and U.S. EPA (2024f). See Section 6.4 for the EPA's analysis of avoided infant birth weight impacts estimated as attributable to reduced PFOA and PFOS exposure from the final rule.

6.2.2.1.2 Cardiovascular Effects

CVD is one of the leading causes of premature mortality in the U.S. (D'Agostino et al., 2008; Goff et al., 2014; Lloyd-Jones et al., 2017). As discussed in the EPA's Final Human Health Toxicity Assessments for PFOA and PFOS, exposure to PFOA and PFOS through drinking water contributes to increased serum PFOA and PFOS concentrations and elevated levels of TC, as well as suggestive changes in levels of HDLC and elevated levels of systolic BP (U.S. EPA, 2024e; U.S. EPA, 2024f). Changes in TC, HDLC, and BP are associated with changes in incidence of CVD events such as myocardial infarction (MI, i.e., heart attack), ischemic stroke (IS), and cardiovascular mortality occurring in populations without prior CVD event experience (D'Agostino et al., 2008; Goff et al., 2014; Lloyd-Jones et al., 2017).

Overall, epidemiology evidence indicated a positive association between PFOS/PFOA exposure and TC levels (i.e., increased exposure is associated with increased TC levels) (ATSDR, 2021; U.S. EPA, 2024e; U.S. EPA, 2024f). Epidemiology studies observed relatively consistent positive associations between PFOA and LDLC (U.S. EPA, 2024f). Most epidemiology studies on PFOS exposure reported a positive association between exposure and TC levels in the general population (ATSDR, 2021; U.S. EPA, 2024e). There was also some evidence of this association in children and pregnant women (U.S. EPA, 2024e). Consistent positive associations were also observed between PFOS and LDLC in general population adults. Toxicology studies often reported decreases in serum lipids from oral exposure to PFOA and PFOS (U.S. EPA, 2024e; U.S. EPA, 2024f). Although the biological significance of the decrease in various serum lipid

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[29] Recent evidence indicates that relationships between maternal serum PFOA/PFOS and birth weight may be impacted by changes in pregnancy hemodynamics, however exact patterns are not completely understood (Sagiv et al., 2018; Steenland et al., 2018).

levels observed in animal models regardless of species, sex, or exposure paradigm is unclear, these effects do indicate a disruption in lipid metabolism, which is consistent with effects observed in humans. For additional details on the TC studies and their individual outcomes, see Chapter 3.4.3 (Cardiovascular) in U.S. EPA (2024e) and U.S. EPA (2024f).

Existing epidemiology and toxicology studies provided inconsistent evidence of associations between PFOA and PFOS exposures and HDLC levels, with a mix of positive and some inverse associations in adult populations (ATSDR, 2021; U.S. EPA, 2024e; U.S. EPA, 2024f). Two studies reported a positive association between PFOA and HDLC in pregnant women (Starling et al., 2017; Dalla Zuanna et al., 2021). In children, prenatal exposure to PFOA was associated with lower HDLC in some studies, especially in boys, whereas childhood exposure was not consistently associated with higher HDLC (ATSDR, 2021; U.S. EPA, 2024f). Similarly, studies did not report consistent associations between PFOS and HDLC levels (ATSDR, 2021; U.S. EPA, 2024e). Most of the evidence in adults involved cross-sectional assessments, although associations between PFOS and lower HDLC were also observed in the cohort study by Lin et al. (2019). Studies examining PFOS and HDLC in pregnant women provided mixed evidence (U.S. EPA, 2024e). Although evidence of associations between PFOA and PFOS exposures and HDLC is mixed, certain individual studies reported robust associations in general adult populations. Based on comments and recommendations from the EPA SAB on the EPA's analysis of CVD risk reductions resulting from changes in PFOA/PFOS exposures (U.S. EPA, 2021a), the EPA assessed HDLC in a sensitivity analysis (see Appendix K). For additional details on the HDLC studies and their individual outcomes, see Chapter 3.4.3 (Cardiovascular) of U.S. EPA (2024e) and U.S. EPA (2024f).

Epidemiology studies observed inconsistent associations between PFOA exposure and BP (ATSDR, 2021; U.S. EPA, 2024f). In adults, some epidemiology studies reported positive associations between PFOA exposure and changes in BP or risk of hypertension (defined as elevated BP) (U.S. EPA, 2024f). Studies in children, adolescents, and pregnant women suggested no association between PFOA exposure and elevated BP (U.S. EPA, 2024f). In adults, there was consistent evidence of positive associations between PFOS exposure and BP, although the results were not always consistent between systolic BP and diastolic BP, and one study reported an inverse association (U.S. EPA, 2024e). However, there was overall consistent evidence of an association between PFOS and BP in studies conducted in general adult populations (U.S. EPA, 2024e). Evidence for associations between PFOS exposure and BP in children and adolescents was limited and did not suggest an association with elevated BP (U.S. EPA, 2024e). However, exposure duration was a limitation in these studies, and evidence of an association between PFOS and increased risk of hypertension, specifically, was limited and inconsistent (U.S. EPA, 2024e). Evidence of associations between BP and PFOS in animal toxicological studies was mixed (U.S. EPA, 2024e). For additional details on the BP studies and their individual outcomes, see Chapter 3.4.3 (Cardiovascular) in U.S. EPA (2024e) and U.S. EPA (2024f).

Given the breadth of evidence linking PFOA and PFOS exposure to effects on TC and BP in general adult populations, the EPA quantified public health impacts of changes in these well-established CVD risk biomarkers (D'Agostino et al., 2008; Goff et al., 2014; Lloyd-Jones et al., 2017) by estimating changes in incidence of several CVD events. Specifically, the EPA assumed that PFOA/PFOS-related changes in TC and BP had the same effect on the CVD risk as the changes unrelated to chemical exposure and used the Pooled Cohort Atherosclerotic

Cardiovascular Disease (ASCVD) model (Goff et al., 2014) to evaluate their impacts on the incidence of MI, IS, and cardiovascular mortality occurring in populations without prior CVD event experience (see Section 6.5). The EPA observed that the direct evidence of associations between PFOA/PFOS exposure and CVD risk was limited (U.S. EPA, 2024e; U.S. EPA, 2024f), with mixed findings reported by one high-quality longitudinal epidemiology study (Mattsson et al., 2015) and four medium-quality cross-sectional epidemiology studies (Huang et al., 2018; Shankar et al., 2012; Hutcheson et al., 2019; Fry & Power, 2017). However, inconclusive evidence of the direct association between PFOA/PFOS exposure and CVD effects from a limited collection of studies does not imply the absence of such an association. Future analyses of CVD effects using large longitudinal studies, such as the ones used to develop the ASCVD model (Goff et al., 2014), could help elucidate whether there is a consistent direct association between PFOA/PFOS and CVD risk. The EPA notes that the SAB review also supported this approach in consideration of impact of PFAS on CVD risk (U.S. EPA, 2022i). See Section 6.5 for EPA's analysis of reduced CVD impacts as a result of reduced PFOA and PFOS exposure from the final rule.

6.2.2.1.3 Cancer Effects

Data on the association between PFOA exposure and kidney cancer (i.e., RCC), particularly from epidemiological studies, indicate a positive association between exposure and increased risk of RCC (CalEPA, 2021; U.S. EPA, 2016f; ATSDR, 2021; U.S. EPA, 2024f). PFOA exposure effects on RCC were shown in two occupational population studies (Raleigh et al., 2014; Steenland & Woskie, 2012) and two high-exposure community studies (Vieira et al., 2013; Barry et al., 2013). A recent study of the relationship between PFOA and RCC in the U.S. general population found strong evidence of a positive association between exposure to PFOA and RCC in humans (Shearer et al., 2021). A meta-analysis of epidemiological literature also concluded that there was an increased risk of kidney cancer associated with increased PFOA serum concentrations (Bartell & Vieira, 2021). In the EPA's Final Human Health Toxicity Assessment for PFOA, the agency reviewed the weight of the evidence and determined that PFOA is Likely to Be Carcinogenic to Humans, as "the evidence is adequate to demonstrate carcinogenic potential to humans but does not reach the weight of evidence for the descriptor Carcinogenic to Humans" (U.S. EPA, 2005c; U.S. EPA, 2024f).[30] This determination is based on the evidence of kidney and testicular cancer in humans and Leydig cell tumors (LCTs), pancreatic acinar cell tumors (PACTs), and hepatocellular tumors in rats (U.S. EPA, 2024f). See Section 6.6 for the EPA's analysis of the benefits of reduced RCC as a result of reduced PFOA exposures from the final rule.

Evidence of the association between PFOS exposure and kidney cancer was inconclusive; the small number and limited scope of studies were inadequate to make definitive conclusions (U.S. EPA, 2016e; U.S. EPA, 2024e). One recent study observed an association between PFOS and an increased risk of RCC in the highest exposed quartile and per doubling of PFOS concentration (Shearer et al., 2021; U.S. EPA, 2024e). However, the association was no longer statistically

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[30] This determination is comparable to the International Agency for Research on Cancer (IARC) determination, which classified PFOA as "carcinogenic to humans" based on "sufficient" evidence for cancer in the toxicology literature and "strong" mechanistic evidence in the epidemiology literature. The IARC also determined that PFOS was classified as "possibly carcinogenic to humans" based on "strong" mechanistic evidence (Zahm et al., 2024).

significant after adjusting for other PFAS (Shearer et al., 2021). The EPA did not report any PFOA or PFOS toxicology studies specifically relating to RCC, although there was evidence of other cancer types in rodent models treated with PFOA or PFOS (U.S. EPA, 2024e; U.S. EPA, 2024f). The EPA did not quantify benefits associated with PFOS and RCC and the agency notes that the national quantifiable benefits analysis includes results for PFOA effects on RCC only. The EPA's benefits analysis for avoided RCC cases from reduced PFOA exposure is detailed in Section 6.6.

The EPA found evidence of a positive association between PFOS exposure and hepatocellular tumors in animal studies. Butenhoff et al. (2012)/Thomford (2002) reported a statistically significant increase in combined hepatocellular adenomas and carcinomas tumor incidence in female Sprague-Dawley rats exposed to PFOS. There was also a statistically significant increase in hepatocellular adenomas in males from the highest dose group. The study reported a statistically significant trend of increased incidence with increasing PFOS concentrations across dose groups in both sexes. Additionally, recently published studies reporting associations between PFOS exposure and hepatocellular carcinoma in humans (Goodrich et al., 2022; Cao et al., 2022) further strengthen these findings in rats and support the cancer classification of Likely to be Carcinogenic to Humans for PFOS (U.S. EPA, 2024e). Thomford (2002) also reported a statistically significant trend of increased incidence of pancreatic islet cell carcinomas with increasing PFOS doses. The EPA reviewed the weight of the evidence and determined that PFOS Is Likely to Be Carcinogenic to Humans, as "the evidence is adequate to demonstrate carcinogenic potential to humans but does not reach the weight of evidence for the descriptor Carcinogenic to Humans" (U.S. EPA, 2005c; U.S. EPA, 2024e). The EPA evaluated the effects of the final rule on liver cancer using relationships between PFOS exposure and liver cancer in female rats in Appendix O.

For additional details on cancer studies and their individual outcomes, see Chapter 3.5 (Cancer) in U.S. EPA (2024e) and U.S. EPA (2024f).

6.2.2.2 *Nonquantifiable Benefits of PFOA and PFOS Exposure Reduction*

In this section, the EPA qualitatively discusses the potential health benefits resulting from reduced exposure to PFOA and PFOS in drinking water. These nonquantifiable benefits are expected to be realized as avoided adverse health effects as a result of the final NPDWR, in addition to the benefits that the EPA has quantified. The EPA anticipates additional benefits associated with developmental, cardiovascular, liver, immune, endocrine, metabolic, reproductive, musculoskeletal, and carcinogenic effects beyond those benefits that the EPA has quantified. The evidence for these adverse health effects is briefly summarized below.

6.2.2.2.1 Developmental Effects

In addition to the infant birth weight impacts that the EPA has quantified (see Section 6.4), small for gestational age (SGA) is a developmental health outcome of interest when studying potential effects of PFOA/PFOS exposure, because infants who are SGA face increased health risks during pregnancy and delivery as well as post-delivery (Osuchukwu & Reed, 2022). The majority of epidemiology studies indicated increased risk of SGA with PFOA/PFOS exposure, although some studies reported null results (U.S. EPA, 2024e; U.S. EPA, 2024f). For instance, some studies suggested a potentially positive association between PFOA exposure and SGA (Govarts et al., 2018; Lauritzen et al., 2017; Y. Wang et al., 2016; Souza et al., 2020; Wikström et al., 2020; Chang et al., 2022; U.S. EPA, 2024f). In addition to decreases in offspring weight, toxicology studies on PFOA and PFOS exposures in rodents demonstrated relationships with multiple other developmental endpoints including increased offspring mortality, decreased maternal body weight and body weight change, skeletal and soft tissue effects, and delayed eye-opening (U.S. EPA, 2024e; U.S. EPA, 2024f). For additional details on developmental studies and their individual outcomes, see Chapter 3.4.4 (Developmental) in U.S. EPA (2024e) and U.S. EPA (2024f).

6.2.2.2.2 Cardiovascular Effects

In addition to the CVD effects that the EPA quantified associated with changes in TC and BP from exposure to PFOA and PFOS (see Section 6.5), available evidence suggests an association between exposure to PFOA and PFOS and increased LDLC (ATSDR, 2021; U.S. EPA, 2024e; U.S. EPA, 2024f). High levels of LDLC are known as the "bad" cholesterol because it can lead to the buildup of cholesterol in the arteries, which can raise the risk of heart disease and stroke. Epidemiology studies showed a positive association between PFOA and PFOS exposure and LDLC levels in adults and children (U.S. EPA, 2024e; U.S. EPA, 2024f). In particular, the evidence suggested positive associations between serum PFOA and PFOS levels and LDLC levels in adolescents ages 12–18, while positive associations between serum levels and LDLC levels in younger children were observed only for PFOA (ATSDR, 2021). Additionally, available evidence supports a relatively consistent positive association between PFOA or PFOS and LDLC in adults, especially those who are obese or prediabetic. Associations with other lipoprotein cholesterol known to increase cardiovascular risks were also positive, which increased confidence in the findings for LDLC. Available evidence regarding the impact of PFOA and PFOS exposure on pregnant women was too limited for the EPA to determine an association (U.S. EPA, 2024e; U.S. EPA, 2024f). Toxicology studies generally reported alterations in serum lipid levels in mice and rats following oral exposure to PFOA (U.S. EPA, 2024f) or PFOS (U.S. EPA, 2024e), indicating a disruption in lipid metabolism, which is coherent with effects observed in humans. For additional details on LDLC studies and their individual outcomes, see Chapter 3.4.3 (Cardiovascular) in U.S. EPA (2024e) and U.S. EPA (2024f).

6.2.2.2.3 Hepatic Effects

Several biomarkers can be used clinically to diagnose liver diseases, including alanine aminotransferase (ALT). Serum ALT measures are considered a reliable indicator of impaired liver function because increased serum ALT is indicative of leakage of ALT from damaged hepatocytes (Boone et al., 2005; Z. Liu et al., 2014; U.S. EPA, 2002). Additionally, evidence from both human epidemiological and animal toxicological studies indicates that increased serum ALT is associated with liver disease (Ioannou, Boyko, & Lee, 2006; Ioannou, Weiss, et al., 2006; Kwo et al., 2017; Roth et al., 2021). Human epidemiological studies have demonstrated that even low magnitude increases in serum ALT can be clinically significant (Mathiesen et al., 1999; J. H. Park et al., 2019). Additionally, numerous studies have demonstrated an association between elevated ALT and liver-related mortality (reviewed by Kwo et al., 2017). Furthermore, the American Association for the Study of Liver Diseases

(AASLD) recognizes serum ALT as an indicator of overall human health and mortality (W. R. Kim et al., 2008). Epidemiology data provides consistent evidence of a positive association between PFOS/PFOA exposure and ALT levels in adults (ATSDR, 2021; U.S. EPA, 2024e; U.S. EPA, 2024f). Studies of adults showed consistent evidence of a positive association between PFOA exposure and elevated ALT levels at both high exposure levels and exposure levels typical of the general population (U.S. EPA, 2024f). There is also consistent epidemiology evidence of associations between PFOS and elevated ALT levels. A limited number of studies reported inconsistent evidence on whether PFOA/PFOS exposure is associated with increased risk of liver disease (U.S. EPA, 2024e). It is also important to note that while evaluation of direct liver damage is possible in animal studies, it is difficult to obtain biopsy-confirmed histological data in humans. Therefore, liver injury is typically assessed using serum biomarkers of hepatotoxicity (Costello et al., 2022). Associations between PFOS/PFOA exposure and ALT levels in children were less consistent than in adults (U.S. EPA, 2024e; U.S. EPA, 2024f).

PFOA toxicology studies showed increases in ALT and other serum liver enzymes across multiple species, sexes, and exposure paradigms (U.S. EPA, 2024f). Toxicology studies on the impact of PFOS exposure on ALT also reported increases in ALT and other serum liver enzyme levels in rodents (U.S. EPA, 2024e). Several studies in animals also reported increases in the incidence of liver lesions or cellular alterations, such as hepatocellular cell death (U.S. EPA, 2024e; U.S. EPA, 2024f). For additional details on the ALT studies and their individual outcomes, see Chapter 3.4.1 (Hepatic) in U.S. EPA (2024e) and U.S. EPA (2024f).

6.2.2.2.4 Immune Effects

Proper antibody response helps maintain the immune system by recognizing and responding to antigens. The available evidence indicates a relationship between PFOA exposure and immunosuppression; epidemiology studies showed suppression of at least one measure of the antibody response for tetanus and diphtheria among people with higher prenatal and childhood serum concentrations of PFOA (ATSDR, 2021; U.S. EPA, 2024f). Data reporting on associations between PFOA exposure and antibody response to vaccinations other than tetanus and diphtheria (i.e., rubella and hand, foot, and mouth disease) are limited but supportive of associations between PFOA and decreased immune response in children (U.S. EPA, 2024f). Available studies supported an association between PFOS exposure and immunosuppression in children, where increased PFOS serum levels were associated with decreased antibody production in response to tetanus, diphtheria, and rubella vaccinations (U.S. EPA, 2024e). Studies reporting associations between PFOA or PFOS and immunosuppression in adults are less consistent, though this may be due to a lack of high confidence data (U.S. EPA, 2024e; U.S. EPA, 2024f). Toxicology evidence suggested that PFOA and PFOS exposure results in effects similarly indicating immune suppression, such as reduced response of immune cells to challenges (e.g., reduced natural killer cell activity and immunoglobulin production) (U.S. EPA, 2024e; U.S. EPA, 2024f). For additional details on immune studies and their individual outcomes, see Chapter 3.4.2 (Immune) in U.S. EPA (2024e) and U.S. EPA (2024f).

Because evidence indicates that PFOA or PFOS exposure results in immune effects, the EPA expects those effects to potentially impact immune response to other diseases. For instance, the coronavirus disease 2019 (COVID-19) caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) rapidly evolved into a global pandemic after its first report in

Wuhan, China, in December 2019. A few recent studies have considered the association between PFOA and PFOS exposure and COVID-19 infection, severity, or mortality (Catelan et al., 2021; Grandjean et al., 2020; Ji et al., 2021).

A case-control study in China (Ji et al., 2021) showed increased risks for COVID-19 infection with high urinary PFOS, PFOA, and total PFASs after adjusting for potential confounding factors including age, gender, diabetes, cardiovascular disease, and urine albumin-to-creatinine ratio. Adjusted odds ratios (ORs) were 1.94 (95% CI: 1.39, 2.96) for PFOS and 2.73 (95% CI: 1.71, 4.55) for PFOA. Using metabolome-wide association analysis, Ji et al. (2021) found that PFOA and PFOS exposure in COVID-19 patients was associated with metabolic disturbances in biochemical pathways involved in mitochondria stress signaling and the regulation of immune function, including fatty acid oxidation, tricarboxylic acid cycle, eicosanoid, and kynurenine pathways. One cross-sectional study in Denmark (Grandjean et al., 2020) observed no association between PFOA or PFOS concentrations and severity of COVID-19 development.[31] In a spatial ecological analysis, Catelan et al. (2021) showed higher mortality risk for COVID-19 in a population heavily exposed to PFAS (including PFOA, PFOS, PFHxS, PFBS, PFBA, PFPeA, PFHxA, and PFHpA) via drinking water in Veneto, Italy.

Although these studies provide a suggestion of possible associations, the body of evidence does not permit any conclusions about the relationship between COVID-19 and exposures to PFAS.

6.2.2.2.5 Endocrine Effects

Elevated circulating thyroid hormone levels can accelerate metabolism and cause irregular heartbeat; low levels of thyroid hormones can cause neurodevelopmental effects, tiredness, weight gain, and increased susceptibility to the common cold. There is suggestive evidence of a positive association between PFOA/PFOS exposure and thyroid hormone disruption (ATSDR, 2021; U.S. EPA, 2024e; U.S. EPA, 2024f). Epidemiology studies reported inconsistent evidence regarding associations between PFOA and PFOS exposure and general endocrine outcomes, such as thyroid disease, hypothyroidism, and hypothyroxinemia (U.S. EPA, 2024e; U.S. EPA, 2024f). However, for PFOA, epidemiological studies reported suggestive evidence of positive associations for serum levels of thyroid stimulating hormone (TSH) and the thyroid hormone triiodothyronine (T3) in adults, and the thyroid hormone thyroxine (T4) in children (U.S. EPA, 2024e; U.S. EPA, 2024f). For PFOS, epidemiological studies reported suggestive evidence of positive associations for TSH in adults, positive associations for T3 in children, and inverse associations for T4 in children (U.S. EPA, 2024e). Toxicology studies indicated that PFOA and PFOS exposure leads to decreases in serum thyroid hormone levels[32] and adverse effects to the endocrine system (ATSDR, 2021; U.S. EPA, 2024b; U.S. EPA, 2024e; U.S. EPA, 2024f). Overall, changes in serum thyroid hormone levels in animals indicate PFOS and PFOA toxicity potentially relevant to humans (U.S. EPA, 2024e; U.S. EPA, 2024f). For additional details on endocrine effects studies and their individual outcomes, see Appendix C.2 (Endocrine) in U.S. EPA (2024a) and U.S. EPA (2024b).

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[31] Note that the authors found that PFBA exposure was associated with increasing severity of COVID-19.

[32] Decreased thyroid hormone levels are associated with effects such as changes in thyroid and adrenal gland weight, hormone fluctuations, and organ histopathology, as well as adverse neurodevelopmental outcomes (ATSDR, 2021; U.S. EPA, 2024e).

6.2.2.2.6 Metabolic Effects

Leptin is a hormone that, along with adiponectin, can be a marker of adipose tissue dysfunction. Chronic high levels of leptin lead to leptin resistance that mirrors many of the characteristics associated with diet-induced obesity, including reduced leptin receptors and diminished signaling. Therefore, high leptin levels are associated with higher body fat mass, a larger size of individual fat cells, overeating, and inflammation (e.g., of adipose tissue, the hypothalamus, blood vessels, and other areas). Evidence suggests an association between PFOA exposure and leptin levels in the general adult population (ATSDR, 2021; U.S. EPA, 2024f). Based on a review of human epidemiology studies, evidence of associations between PFOS and metabolic outcomes appears inconsistent, but in some studies, positive associations were observed between PFOS exposure and leptin levels (U.S. EPA, 2024e). Studies examining newborn leptin levels did not find associations with maternal PFOA levels (ATSDR, 2021). Maternal PFOS levels were also not associated with alterations in leptin levels (ATSDR, 2021). For additional details on metabolic effect studies and their individual outcomes, see Appendix C.3 (Metabolic/Systemic) in U.S. EPA (2024a) and U.S. EPA (2024b).

6.2.2.2.7 Reproductive Effects

Studies of the reproductive effects from PFOA/PFOS exposure have focused on associations between exposure to these contaminants and increased risk of gestational hypertension and preeclampsia in pregnant women (ATSDR, 2021; U.S. EPA, 2024e; U.S. EPA, 2024f). Gestational hypertension (high BP during pregnancy) can lead to fetal problems such as poor growth and stillbirth. Preeclampsia—instances of gestational hypertension where the mother also has increased levels of protein in her urine—can similarly pose significant risks to both the fetus and mother. Risks to the fetus include impaired fetal growth due to the lack of oxygen and nutrients, stillbirth, preterm birth, and infant death (National Institutes of Health, 2017). Even if born full term, the infant may be at risk for later problems such as diabetes, high blood pressure, and congestive heart failure. Effects of preeclampsia on the mother may include kidney and liver damage, blood clotting problems, brain injury, fluid on the lungs, seizures, and mortality (National Institutes of Health, 2018). The epidemiology evidence yields mixed (positive and null) associations, with some suggestive evidence supporting positive associations between PFOA/PFOS exposure and both preeclampsia and gestational hypertension (ATSDR, 2021; U.S. EPA, 2024e; U.S. EPA, 2024f). For additional details on reproductive effects studies and their individual outcomes, see Appendix C.1 (Reproductive) in U.S. EPA (2024a) and U.S. EPA (2024b).

6.2.2.2.8 Musculoskeletal Effects

Adverse musculoskeletal effects such as osteoarthritis and decreased bone mineral density impact bone integrity and cause bones to become brittle and more prone to fracture. The available epidemiology evidence suggests that PFOA exposure may be linked to decreased bone mineral density, bone mineral density relative to bone area, height in adolescence, osteoporosis, and osteoarthritis (ATSDR, 2021; U.S. EPA, 2024f). Some studies found that PFOA/PFOS exposure was linked to osteoarthritis, in particular among women under 50 years of age (ATSDR, 2021). There is limited evidence from studies pointing to effects of PFOS on skeletal size (height), lean body mass, and osteoarthritis (U.S. EPA, 2024e). Evidence from some studies suggests that PFOS exposure has a harmful effect on bone health, particularly measures of bone mineral density, with more statistically significant effects occurring among females (U.S. EPA, 2024e). However, other reviews reported mixed findings on the effects of PFOS exposure including decreased risk of osteoarthritis, increased risk for some demographic subgroups, or no association (ATSDR, 2021). For additional details on musculoskeletal effects studies and their individual outcomes, see Appendix C.8 (Musculoskeletal) in U.S. EPA (2024a) and U.S. EPA (2024b).

6.2.2.2.9 Cancer Effects

In the EPA’s Final Human Health Toxicity Assessment for PFOA (U.S. EPA, 2024d), the agency evaluates the evidence for carcinogenicity of PFOA that has been documented in both epidemiological and animal toxicity studies. The evidence in epidemiological studies is primarily based on the incidence of kidney and testicular cancer, as well as potential incidence of breast cancer in genetically susceptible subpopulations or for particular breast cancer types. Other cancer types have been observed in humans, although the evidence for these is generally limited to low confidence studies. The evidence of carcinogenicity in animal models is provided in three chronic oral animal bioassays in Sprague-Dawley rats which identified neoplastic lesions of the liver, pancreas, and testes (U.S. EPA, 2024f). For more information on the EPA's cancer determination for PFOA, see Section 6.2.2.1.3.

In the EPA’s Final Human Health Toxicity Assessment for PFOS (U.S. EPA, 2024e), the agency evaluates the evidence for carcinogenicity of PFOS and found that several epidemiological studies and a chronic cancer bioassay comprise the evidence database for the carcinogenicity of PFOS (U.S. EPA, 2024e). The available epidemiology studies report elevated risk of liver cancer, consistent with increased incidence of liver tumors reported in male and female rats. There is also mixed but plausible evidence of bladder, prostate, kidney, and breast cancers in humans. The animal chronic cancer bioassay study also provides evidence of increased incidence of pancreatic islet cell tumors in male rats. For more information on the EPA's cancer determination for PFOS, see Section 6.2.2.1.3.

The EPA anticipates there are additional nonquantifiable benefits related to potential testicular, bladder, prostate, and breast cancer effects summarized above. Benefits associated with avoiding cancer cases not quantified in the EPA's analysis could be substantial. For example, a study by Obsekov et al. (2023) reports the number of breast cancer cases attributable to PFAS exposure ranges from 421 to 3,095 annually, with an estimated direct cost of 6-month treatment ranging from $27.1 to $198.4 million per year ($2022). This study also finds that approximately 5 (0.076%) annual testicular cancer cases are attributable to PFOA exposure with an estimated direct cost of treatment of $173,450 per year ($2022). Although the methods used by Obsekov et al. (2023) differ from those used to support the national quantified benefits of the rule, the information provided in the study is helpful in portraying the costs of cancers that are associated with PFAS exposures. For additional details on cancer studies and their individual outcomes, see Chapter 3.5 (Cancer) in U.S. EPA (2024e) and U.S. EPA (2024f).

6.2.3 Summary of Health Information Considered in the Economic Analysis

After assessing available health and economic information, the EPA was unable to quantify the benefits of avoided health effects discussed in Section 6.2.2.2 above. The agency prioritized health endpoints with the strongest weight of evidence conclusions and readily available data for monetization, namely cardiovascular effects, developmental effects, and carcinogenic effects. Several other health endpoints that had indicative or suggestive evidence of associations with exposure to PFOA and PFOS have not been selected for the economic analysis:

- While immune effects had indicative evidence of associations with exposure to PFOA and PFOS, the EPA did not identify the necessary information to connect the measured biomarker responses (i.e., decrease in antibodies) to a disease that could be valued in the economic analysis;
- Evidence indicates associations between PFOA and PFOS exposure and hepatic effects, such as increases in ALT. While increased ALT is considered an adverse effect, ALT can be one of several contributors to a variety of diseases, including liver disease, and it is difficult to therefore quantify the relationship between this biomarker and a disease that can be monetized. Similar challenges with the biomarkers representing metabolic effects (i.e., leptin) and musculoskeletal effects (i.e., bone density) prevented economic analysis of these endpoints;
- There is evidence of association between exposure to PFOA and testicular cancer in human and animal studies; however, the available slope factor in rats implied small changes in the risk of this endpoint. Because testicular cancer is rarely fatal and the Value of Statistical Life is the driver of economic benefits evaluated in the EA, the benefit of decreased testicular cancer expected with this rule was smaller in comparison and not quantified;
- There is evidence of association between exposure to PFOS and hepatic carcinogenicity in human and animal studies. The EPA quantified benefits associated with reduced liver cancer cases and deaths as part of a sensitivity analysis for the final rule in response to public comments received on the proposed rule requesting that the EPA quantify additional health benefits (see Appendix O);
- Finally, other health endpoints, such as small for gestational age and LDLC effects, were not modeled in the EA because they overlap with effects that the EPA did model. More specifically, SGA infants are often born with decreased birth weight or receive similar care to infants born with decreased birth weight. LDLC is a component of total cholesterol and could not be modeled separately as the EPA used total cholesterol as an input to the ASCVD model to estimate CVD outcomes.

6.2.4 Nonquantifiable Benefits of PFAS in Final Rule and PFAS Expected to be Co-Removed

The EPA also qualitatively summarized the potential health benefits resulting from reduced exposure to PFAS other than PFOA and PFOS in drinking water. The final rule and all regulatory alternatives are expected to result in additional benefits that have not been quantified. The final rule will reduce exposure to PFHxS, HFPO-DA, and PFNA to below their individual MCLs. It will also reduce exposure to mixtures of two or more of PFHxS, PFNA, HFPO-DA, and PFBS to below the HI MCLG and MCL of 1. Benefits from avoided cases of the adverse health effects discussed below are expected from the final rule due to co-occurrence of these contaminants in source waters containing PFOA and/or PFOS, as documented in detail in the *Per- and Polyfluoroalkyl Substances (PFAS) Occurrence & Contaminant Background Support Document* (U.S. EPA, 2024g). In addition, PFAS, including PFHxS, HFPO-DA, PFNA, and PFBS and their mixtures affect common target organs, tissues, or systems to produce dose-additive effects from their co-exposures with each other, as well as PFOA and PFOS (U.S. EPA, 2024d). The EPA expects that compliance actions taken under the final rule will remove additional unregulated co-occurring PFAS contaminants where present because the best available technologies have been demonstrated to co-remove additional PFAS. Treatment responses implemented to reduce PFOA and PFOS exposure under the final rule and Options 1a-c are likely to remove some amount of additional PFAS contaminants where they co-occur.

IX and GAC are effective at removing PFAS; there is generally a linear relationship between PFAS chain length and removal efficiency, shifted by functional group (McCleaf et al., 2017; Sörengård, 2020). Perfluoroalkyl sulfonates (PFSAs), such as PFOS, are removed with greater efficiency than corresponding perfluoroalkyl carboxylates (PFCAs), such as PFOA, of the same carbon backbone length (Appleman et al., 2014; Du, 2014; Eschauzier et al., 2012; Ochoa-Herrera & Sierra-Alvarez, 2008; Zaggia et al., 2016). Generally, for a given water type and concentration, PFSAs are removed approximately as effectively as PFCAs, which have two additional fully perfluorinated carbons in the carbon backbone. For example, PFHxS (i.e., sulfonic acid with a six-carbon backbone) is removed approximately as well as PFOA (i.e., carboxylic acid with an eight-carbon backbone) and PFHxA (i.e., carboxylic acid with a six-carbon backbone) is removed approximately as well as PFBS (i.e., sulfonic acid with a four-carbon backbone). Further, PFAS compounds with longer carbon chains display lower percentage decreases in average removal efficiency over time (McCleaf et al., 2017).

In cases where the six PFAS included in the final rule occur at concentrations above their respective regulatory standards, there is also an increased probability of co-occurrence of additional unregulated PFAS. Further, as the same technologies also remove other long-chain and higher carbon/higher molecular weight PFAS, the EPA expects that treatment will provide additional public health protection and benefits due to co-removal of unregulated PFAS that may have adverse health effects. While the EPA has not quantified these additional benefits, the agency expects that these important co-removal benefits will further enhance public health protection.

The EPA identified a wide range of potential health effects associated with exposure to PFAS other than PFOA and PFOS using documents that summarize the recent literature on exposure and associated health impacts: the ATSDR’s Toxicological Profile for Perfluoroalkyls (ATSDR, 2021); the EPA’s toxicity assessment of HFPO-DA (U.S. EPA, 2021c); publicly available IRIS assessments for PFBA and PFHxA (U.S. EPA, 2022e; U.S. EPA, 2023d); EPA's toxicity assessment of PFBS (U.S. EPA, 2021d); and the recent National Academies of Sciences, Engineering, and Medicine Guidance on PFAS Exposure, Testing, and Clinical Follow-up

(NASEM, 2022). Note that the determinations of associations between PFAS and associated health effects are based on information available as of September 2023.

Developmental effects: Toxicology and/or epidemiology studies observed evidence of associations between birth weight and/or other developmental effects and exposure to PFBA, PFDA, PFHxS, PFHxA, HFPO-DA, PFNA, PFUnA, and PFBS. Specifically, data from toxicology studies support this association for PFBS, PFBA, PFHxA, and HFPO-DA, while both toxicology and epidemiology studies support this association for PFHxS, PFDA, PFUnA, and PFNA (ATSDR, 2021; U.S. EPA, 2021c; U.S. EPA, 2022e; Wright et al., 2023). In general, epidemiological studies did not find associations between exposure and adverse pregnancy outcomes (miscarriage, preterm birth, or gestational age) for PFNA, PFUnA, and PFHxS (ATSDR, 2021; NASEM, 2022). Epidemiological studies support an association between PFNA, PFHxS or PFDA exposure and developmental effects such as decreases in infant birth weight and birth length, small for gestational age and increased risk of low birth weight (Valvi et al., 2017; C.C. Bach et al., 2016; Louis et al., 2018; Wright et al., 2023; Manzano-Salgado et al., 2017; Starling et al., 2017). Few epidemiologic studies also indicate that PFDA exposure is associated with developmental effects (Wikström et al., 2020; Valvi et al., 2017; Luo et al., 2021; Yao et al., 2021). The EPA has determined that evidence indicates that exposure to PFBA or PFHxA likely causes developmental effects, based on moderate evidence from animal studies and indeterminate evidence from human studies (U.S. EPA, 2022e; U.S. EPA, 2023d).

Cardiovascular effects: Epidemiology and/or toxicology studies observed evidence of associations between PFNA, PFDA, and PFHxS exposures and effects on total cholesterol, LDLC, and HDLC. Epidemiological studies report consistent associations between PFHxS and total cholesterol in adults (Cakmak et al., 2022; Dunder et al., 2022; Canova et al., 2020; Lin et al., 2019; G. Liu et al., 2020; Fisher et al., 2013). In an analysis based on studies published before 2018, evidence for associations between PFNA exposure and serum lipid levels in epidemiology studies was mixed; associations have been observed between serum PFNA levels and total cholesterol in general populations of adults but not in pregnant women, and evidence in children is inconsistent (ATSDR, 2021). Most epidemiology studies did not observe associations between PFNA and LDLC or HDLC. Epidemiological studies report consistent associations between PFDA and effects on total cholesterol in adults (Cakmak et al., 2022; Dunder et al., 2022; G. Liu et al., 2020; Dong et al., 2019). Positive associations between PFDA and other serum lipids, adiposity, cardiovascular disease, and atherosclerosis were observed in some epidemiology studies, but findings were inconsistent (Huang et al., 2018; Mattsson et al., 2015; Christensen et al., 2016). A single animal study observed decreases in cholesterol and triglyceride levels in rats at PFDA doses above 1.25 mg/kg/d for 28 days (National Toxicology Program, 2018b). There was no association between PFBA and serum lipids in a single epidemiology study and no animal studies on PFBA evaluated cardiovascular endpoints (U.S. EPA, 2022e). Other PFAS for which lipid outcomes were examined in toxicology or epidemiology studies showed limited to no evidence of associations. Studies have examined possible associations between various PFAS and blood pressure in humans or heart histopathology in animals. Epidemiological studies report positive associations between PFHxS and hypertension in adolescents and young adults (Averina et al., 2021; N. Li et al., 2021; Pitter et al., 2020), but not in other adults (P.-I. D. Lin et al., 2020; A. Chen et al., 2019; Christensen et al., 2018; G. Liu et al., 2018; Bao et al., 2017 ; Christensen et al., 2016) or children (Papadopoulou et al., 2021; Khalil et al., 2018; Manzano-Salgado et al., 2017). No evidence was observed of associations between PFHxS and cardiovascular diseases (Huang et al., 2018; Mattsson et al., 2015). Overall, studies did not find likely evidence of cardiovascular effects for other PFAS except for PFOS and PFOA (U.S. EPA, 2024e; U.S. EPA, 2024f).

Hepatic effects: Toxicology and/or epidemiology studies have reported associations between exposure to PFAS (PFBA, PFDA, PFUnA, PFDoDA, PFHxA, PFHxS, HFPO-DA, and PFBS) and hepatotoxicity. The results of the animal toxicology studies provide strong evidence that the liver is a sensitive target of PFHxS, PFNA, PFDA, PFUnA, PFBS, PFBA, PFDoDA, HFPO-DA and PFHxA toxicity. Observed effects in rodents include increases in liver weight, hepatocellular hypertrophy, hyperplasia, and necrosis (ATSDR, 2021; U.S. EPA, 2021c; U.S. EPA, 2022e; U.S. EPA, 2023d). Increases in serum enzymes (such as ALT) and decreases in serum bilirubin were observed in several epidemiological studies of PFNA and PFDA (Nian et al., 2019; Jain & Ducatman, 2019b; J.-J. Liu et al., 2022; Cakmak et al., 2022). Associations between exposure to PFHxS and effects on serum hepatic enzymes are less consistent (Cakmak et al., 2022; J.-J. Liu et al., 2022; Jain & Ducatman, 2019b; Salihovic et al., 2018; Gleason et al., 2015). Mixed effects were observed for serum liver enzymes in epidemiological studies for PFNA (ATSDR, 2021).

Immune effects: Epidemiology studies have reported evidence of associations between PFDA or PFHxS exposure and antibody response to tetanus or diphtheria (Grandjean et al., 2012; Grandjean, Heilmann, Nielsen, et al., 2017; Grandjean, Heilmann, Weihe, et al., 2017; Budtz-Jørgensen & Grandjean, 2018). There is also some limited evidence for decreased antibody response for PFNA, PFUnA, and PFDoDA, although there were notable inconsistencies across studies examining associations for these compounds (ATSDR, 2021). There is limited evidence for associations between PFHxS, PFNA, PFDA, PFBS, and PFDoDA and increased risk of asthma due to the small number of studies evaluating the outcome and/or inconsistent study results (ATSDR, 2021). The small number of studies investigating immunotoxicity in humans following exposure to PFHpA and PFHxA did not find associations (ATSDR, 2021; U.S. EPA, 2023d, NASEM, 2022). Toxicology studies have reported evidence of associations between HFPO-DA exposure and effects on various immune-related endpoints in animals (ATSDR, 2021; U.S. EPA, 2021c). No laboratory animal studies were identified for PFUnA, PFHpA, PFDoDA, or FOSA. A small number of toxicology studies evaluated the immunotoxicity of other perfluoroalkyls and most did not evaluate immune function. No alterations in spleen or thymus organ weights or morphology were observed in studies on PFHxS and PFBA. A study on PFNA found decreases in spleen and thymus weights and alterations in splenic lymphocyte phenotypes (ATSDR, 2021). Changes in spleen and thymus weights were reported in female mice and male/female rats in two 28-day gavage studies of PFDA, although the direction and dose-dependency of these changes in rats was inconsistent across studies (Frawley et al., 2018 ; National Toxicology Program, 2018b).

COVID-19: A cross-sectional study in Denmark (Grandjean et al., 2020) showed that PFBA exposure was associated with increasing severity of COVID-19, with an OR of 1.77 (95% CI: 1.09, 2.87) after adjustment for age, sex, sampling site, and interval between blood sampling and diagnosis. A case-control study showed increased risk of COVID-19 infection with high urinary PFAS (including PFOA, PFOS, PFHxA, PFHpA, PFHxS, PFNA, PFBS, PFDA, PFUnA, PFDoA, PFTrDA, PFTeDA) levels (Ji et al., 2021). Adjusted odds ratios were 1.94 (95% CI: 1.39, 2.96) for PFOS, 2.73 (95% CI: 1.71, 4.55) for PFOA, and 2.82 (95% CI: 1.97–3.51) for total PFAS (sum of 12 PFAS), while other PFAS were not significantly associated with COVID-19 susceptibility after adjusting for confounders. In a spatial ecological analysis, Catelan et al. (2021) showed higher mortality risk for COVID-19 in a population heavily exposed to PFAS (including PFOA, PFOS, PFHxS, PFBS, PFBA, PFPeA, PFHxA, and PFHpA) via drinking water. Overall, results suggested a general immunosuppressive effect of PFAS and/or increased COVID-19 respiratory toxicity due to a concentration of PFBA in the lungs. Although these studies provide a suggestion of possible associations, the body of evidence does not permit conclusions about the relationship between COVID-19 infection, severity, or mortality, and exposures to PFAS. In addition to the adverse health effects listed above, there was little or no evidence that exposure to the various PFAS is associated with the additional health effects summarized below.

Endocrine effects: Epidemiology studies have observed associations between serum PFHxS, PFNA, PFDA, and PFUnA and effects on thyroid stimulating hormone (TSH), triiodothyronine (T3), or thyroxine (T4) levels in serum or thyroid disease; however, there are notable inconsistencies across the studies identified in the available reports (ATSDR, 2021; NASEM, 2022). Toxicology studies have reported consistent associations between exposure to PFHxS, PFBA, PFHxA, and PFBS and effects on thyroid hormones, thyroid organ weight, and thyroid histopathology in animals; the endocrine system was a notable target of PFBS and PFHxS toxicity (ATSDR, 2021; U.S. EPA, 2021d; U.S. EPA, 2022e; U.S. EPA, 2023d; National Toxicology Program, 2018a; Ramhøj et al., 2018; Ramhøj et al., 2020; Butenhoff et al., 2009).

Metabolic effects: Epidemiology and toxicology studies have examined possible associations between various PFAS and metabolic effects, including leptin, body weight, or body fat in humans or animals (ATSDR, 2021). Exposure to PFDA has been associated with an increase in adiposity in adults (Blake et al., 2018; Christensen et al., 2018; G. Liu et al., 2018). However, evidence of associations was not suggestive or likely for any PFAS in this summary except for PFOA and PFOS (U.S. EPA, 2024a; U.S. EPA, 2024b; U.S. EPA, 2024e; U.S. EPA, 2024f). Evidence for changes such as maternal body weight gain, pup body weight, or other developmentally focused weight outcomes is strong but is considered under the Developmental effects category (ATSDR, 2021; NASEM, 2022).

Renal effects: A small number of epidemiology studies with inconsistent results evaluated possible associations between PFHxS, PFNA, PFDA, PFBS, PFDoDA, or PFHxA and renal function (including estimated glomerular filtration rate and increases in uric acid levels) (ATSDR, 2021; NASEM, 2022; U.S. EPA, 2023d). Toxicology studies have not observed impaired renal function or morphological damage following exposure to PFHxS, PFDA, PFUnA, PFBS, PFBA, PFDoDA, or PFHxA (ATSDR, 2021). Associations with kidney weight in animals were observed for PFBS and HFPO-DA and was a notable target for PFBS toxicity (ATSDR, 2021; U.S. EPA, 2021c; U.S. EPA, 2021d).

Reproductive effects: A small number of epidemiology studies with inconsistent results evaluated possible associations between reproductive hormone levels and PFHxS, PFNA, PFDA, PFUnA, PFDoDA, or PFHxA. Some associations between PFAS (PFHxS, PFHxA, PFNA, PFDA) exposures and sperm parameters have been observed, but often only one sperm parameter was altered. While there is suggestive evidence of an association between PFHxS or PFNA exposure and an increased risk of early menopause, this may be due to reverse causation since an earlier onset of menopause would result in a decrease in the removal of PFAS in menstrual blood. Epidemiological studies provide mixed evidence of impaired fertility (increased risks of longer time to pregnancy and infertility), with some evidence for PFHxS, PFNA, PFHpA, and PFBS but the results are inconsistent across studies or were only based on one study (ATSDR, 2021; Carlsen Bach et al., 2018; Vélez et al., 2015). Toxicology studies have evaluated the potential histological alterations in reproductive tissues, alterations in reproductive hormones, and impaired reproductive functions. No effect on fertility was observed for PFBS and PFDoDA, and no histological alterations were observed for PFBS and PFBA. One study found alterations in sperm parameters and decreases in fertility in mice exposed to PFNA, and one study for PFDoDA observed ultrastructural alterations in the testes (ATSDR, 2021). Decreased uterine weights, changes in hormone levels, and increased time spent in diestrus were observed in studies of PFDA or PFHxS exposures (National Toxicology Program, 2018b; Yin et al., 2021).

Musculoskeletal effects: Epidemiology studies observed evidence of associations between PFNA and PFHxS and musculoskeletal effects including osteoarthritis and bone mineral density, but data are limited to two studies (ATSDR, 2021; Khalil et al., 2016; Khalil et al., 2018). Toxicology studies reported no morphological alterations in bone or skeletal muscle in animals exposed to PFBA, PFDA, PFHxA, PFHxS, or PFBS, but evidence is based on a very small number of studies (NTP, 2018; ATSDR, 2021; U.S. EPA, 2022e; U.S. EPA, 2023d).

Hematological effects: A single uninformative epidemiological study reported on blood counts in pregnant women exposed to PFHxA (U.S. EPA, 2024e). Epidemiological data were not identified for the other PFAS (ATSDR, 2021). A limited number of toxicology studies observed alterations in hematological indices following exposure to relatively high doses of PFHxS, PFDA, PFUnA, PFBS, PFBA, or PFDoDA (ATSDR, 2021; U.S. EPA, 2022e; National Toxicology Program, 2018b; 3M Company, 2000; Frawley et al., 2018). Toxicology studies observed robust evidence of association between PFHxA or HFPO-DA exposure and hematological effects, including decreases in red blood cell (RBC) number, hemoglobin, and percentage of RBCs in the blood (U.S. EPA, 2021c; U.S. EPA, 2023d). A small number of toxicology studies observed slight evidence of associations between exposure to PFHxS, PFDA, or PFBA and decreases in multiple red blood cell parameters and in prothrombin time; however, effects were not consistent (U.S. EPA, 2022e; Butenhoff et al., 2009).

Other non-cancer effects: A limited number of epidemiology and toxicology studies have examined possible associations between various PFAS and dermal, ocular, and other non-cancer effects. However, the evidence does not support associations for any PFAS in this summary except for PFOA and PFOS (ATSDR, 2021; U.S. EPA, 2021d; U.S. EPA, 2022e; U.S. EPA, 2023d).

Cancer effects: A small number of epidemiology studies reported limited associations between multiple PFAS (i.e., PFHxS, PFDA, PFUnA, and FOSA) and cancer effects. No consistent associations were observed for breast cancer risk for PFHxS, PFHxA, PFNA, PFHpA, or PFDoDA; increased breast cancer risks were observed for PFDA and FOSA, but this was based on a single study (Bonefeld-Jorgensen et al., 2014), and one study observed non-significant increased risk for breast cancer risk and PFDA (Tsai et al., 2020). Exposure to PFHxS was associated with increased breast cancer risk in one study and with decreased breast cancer risk in two related studies (Bonefeld-Jorgensen et al., 2014; Ghisari et al., 2017; Tsai et al., 2020). No associations between PFHxS, PFNA, PFDA, or PFUnA and prostate cancer risk were observed. However, among men with a first-degree relative with prostate cancer, associations were observed for PFHxS, PFDA (Hardell et al., 2014), and PFUnA, but not for PFNA (ATSDR, 2021; U.S. EPA, 2022e; U.S. EPA, 2023d). A decreased risk of thyroid cancer was associated with exposure to PFHxS and PFDA in a single study (M. Liu et al., 2021). Epidemiological studies examining potential cancer effects were not identified for PFBS or PFBA (ATSDR, 2021; U.S. EPA, 2022e). No animal studies examined carcinogenicity of PFHxS or PFBA. Aside from a study that suggested an increased incidence of liver tumors in rats exposed to high doses of HFPO-DA, the limited number of available toxicology studies reported no evidence of associations between exposure to other PFAS (i.e., PFDA and PFHxA) and risk of cancer (ATSDR, 2021; U.S. EPA, 2021c; U.S. EPA, 2023d). At this time, there is inadequate information to assess carcinogenic potential for PFAS other than PFOA, PFOS, and HFPO-DA.

6.2.5 Sensitive Populations

SDWA Section 1412(b)(3)(C) establishes requirements for the EPA to develop a HRRCA that presents both quantifiable and nonquantifiable benefits and costs likely to occur as a result of compliance with the NPDWR. In developing this HRRCA, the EPA considered adverse health effects to sensitive populations and subpopulations.

Adverse health effects of PFAS such as cancer, developmental, hepatic, immune, and serum lipid effects (see Sections 6.2.2 and 6.2.4) have been observed in the general population, including women of reproductive age. Effects have been observed in vulnerable populations of groups who have relatively high exposures, for example workers and their families who worked at and/or lived near facilities that used PFOA (such as the C8 Health Project[33] populations). However, data for the elucidation of differential susceptibility dependent on life stage (e.g., developing embryo/fetus, or pregnant women) are very limited or not available. Children are frequently more vulnerable to contaminants than the average adult because of the differences in their behaviors and biology. These differences can result in greater exposure and/or unique windows of developmental susceptibility during the prenatal and postnatal periods for both the pregnant mother and the developing fetus.

When evaluating NPDWRs for any unregulated contaminant, the EPA considers the adverse health risks to infants/children, pregnant women, the elderly, individuals with a history of serious illness, and any subpopulation that are identifiable as being at greater risk due to exposure to contaminants in drinking water than the general population to ensure that the most sensitive population groups are protected. SDWA Section 1412(b)(3)(C)(i)(V). In conducting risk analyses and assessments, the EPA and other agencies and organizations consider subpopulations that may be sensitive to PFAS exposure to be pregnant women, infants/children, individuals who are immunologically compromised, and the elderly (U.S. EPA, 2024e; U.S. EPA, 2024f; ATSDR, 2021; CalEPA, 2021; Minnesota Pollution Control Agency, 2021). CalEPA (2021) and the Minnesota Pollution Control Agency (2021) also identify the timing of exposure to PFAS to be critical in the development of adverse health effects. There is evidence of associations with birth weight effects and exposure to PFDA, PFHxS, PFNA, PFOA, PFOS, or PFUnA (see Sections 6.2.2 and 6.2.4). There is some sex-specific variation in the toxicokinetics of PFOA in

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[33] The C8 Health Project studied over 60,000 individuals who had lived, worked, or attended school for more than one year in one of six water districts contaminated by PFOA between 1950 and 2004 (Frisbee et al., 2010).

humans and rodents, with females generally excreting PFOA faster than males (U.S. EPA, 2024f).

Overall, given that evidence of exposure and adverse health effects of PFAS is mostly reported in studies of the general population, not all potentially sensitive populations are quantified in developing this HRRCA. However, the modeled endpoints, including decreases in infant birth weight (Section 6.4), CVD (Section 6.5), and RCC (Section 6.6), are prevalent in sensitive populations (i.e., infants and the elderly).

6.2.6 Co-Removal of Additional Contaminants

Additional co-removal benefits can occur with the advanced treatment options for PFAS removal. Advanced treatment technologies including GAC, IX, as well as high-pressure membranes such as nanofiltration (NF) and reverse osmosis (RO) can remove many contaminants in addition to those specifically targeted by the final PFAS rule, including other contaminants that the EPA may regulate in the future (Chowdhury et al., 2013; de Abreu Domingos & da Fonseca, 2018; McNamara et al., 2018; Pramanik et al., 2015; Yu et al., 2012). For example, membrane technology (depending on pore size) can be used to lower DBP formation by the removal of organic carbon, and can also remove many microbial contaminants (e.g., bacteria and protozoans) of public health concern (S. K. Park et al., 2019).

Organic matter can also be removed by IX and GAC (Crittenden et al., 1993; W. H. Kim et al., 1997; Yapsakli & Çeçen, 2010; Dickenson & Higgins, 2016; Yuan et al., 2022). Removing TOC, which functions as a DBP precursor, may also help address DBP issues, including regulated and nonregulated DBPs. Epidemiological studies have shown that increased exposure to chlorinated DBPs is associated with higher risk of bladder cancer and other adverse health outcomes (Cantor et al., 1998; Freeman et al., 2017). Weisman et al. (2022) found that approximately 8,000 of the 79,000 annual bladder cancer cases in the U.S. were potentially attributable to chlorinated DBPs in drinking water systems.

In addition, TOC removal lowers disinfectant demand and could lower disinfectant dose requirements (Hooper & Allgeier, 2002). Membrane technology, IX, and GAC lower nutrient availability for bacterial growth, produce a more biologically stable finished water, and facilitate management of water quality in the distribution system. Lower organic matter concentration is also associated with lower assimilable organic carbon (AOC) and nutrient availability for biofilm growth, helping to maintain disinfectant residual in the distribution system and to reduce microbial risk (U.S. EPA, 2005b).

A major concern for drinking water systems is biofilm control in reducing microbial risk. One opportunistic pathogen of concern is Legionella, which can grow and multiply in amoeba that live in biofilms and sediments (National Academies of Sciences, 2020). Certain conditions in the distribution and plumbing systems can also support its proliferation, including low disinfectant residual (U.S. EPA, 2016i; LeChevallier, 2020). Legionella exposure can lead to legionellosis, Pontiac fever, or a form of pneumonia called Legionnaires' disease (National Academies of Sciences, 2020). Collier et al. (2021) estimated that in 2014 there were 11,000 cases of Legionnaires' disease due to waterborne exposure in the U.S., with an estimated one in 10 cases leading to death.

Since membrane technology and GAC also remove SOCs, these advanced treatment options provide additional protection from exposure to chemicals associated with accidental spills or environmental runoff. The EPA has previously used the term SOC to include volatile organic carbons, herbicides, pesticides, and other anthropogenic organic compounds (U.S. EPA, 1998d). One example of a volatile organic carbon that can be co-removed by GAC is dichloromethane (also known as methylene chloride), which has been linked to liver, neurological, and blood cell damage in addition to various cancers (U.S. EPA, 2014). The EPA also identified alachlor as a herbicide that can be removed by GAC and has been linked to liver, kidneys, and spleen damage (U.S. EPA, 1998a). Another SOC example that can be removed by GAC treatment is atrazine, a pesticide that targets the endocrine system and has been associated with adverse developmental reproductive effects (U.S. EPA, 2007a). Removal of any contaminants that may face current and/or future regulation could result in additional public health protection and cost savings to a water system. As public water systems move to advanced treatment, other non-health benefits are also anticipated including better-tasting and smelling water.

6.3 Blood Serum Concentration Modeling for PFAS

6.3.1 Introduction

The U.S. EPA implemented PK models to evaluate blood serum PFOA and PFOS levels in adults resulting from exposure to PFAS via drinking water. This section discusses the application of the PFOA and PFOS PK models in the context of the benefits analysis.

6.3.2 Application of PK Models to Benefits Analyses

The EPA used baseline and regulatory alternative PFOA/PFOS drinking water concentrations as inputs to its PK models to estimate blood serum PFOA/PFOS concentrations for adult males and females. In this analysis, the agency implemented the final PFOA/PFOS PK model version in SafeWater MCBC.[34] See the EPA’s Final Human Health Toxicity Assessments for PFOA and PFOS for further information on the model (U.S. EPA, 2024e; U.S. EPA, 2024f) and EPA's Github repository for pharmacokinetic modeling.[35] The PK models require total PFOA/PFOS dose in mg/kg of body weight per day to be provided as an input. The EPA multiplied PFOA/PFOS drinking water concentrations in mg/L by a water intake of 0.013 L/kg of body weight per day based on the EPA’s Exposure Factors Handbook (U.S. EPA, 2011b) in order to compute the PFOA/PFOS dose from drinking water sources.

The EPA acknowledges that sources or pathways of exposure other than drinking water consumption may contribute to an individual's total PFOA/PFOS exposure (see Section 6.3.3 for discussion of contributions from other sources). However, the assumed baseline exposure from drinking water sources does not affect the estimated changes in serum PFOA/PFOS, which is the key quantity of interest to the benefits estimation. For the PK model in humans, the EPA selected a “linear” approach in which the rates in the model are all proportional to concentration. In this

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[34] SafeWater MCBC was programmed for maximal computational efficiency. The implementation is mathematically consistent with what is described in the SAB documentation and associated R code, however, SafeWater performs a series of pre-calculations to reduce model runtime.

[35] https://github.com/USEPA/OW-PFOS-PFOA-MCLG-support-PK-models

type of model, predicted serum concentration is proportional to the dose, with a proportionality constant that is dependent on time, but not dose. Given the same model parameters, such as sampling age and exposure duration, doubling the dose will double the predicted serum concentration. Note that each simulation models an individual from birth through to the sampling age, with a default exposure scenario of constant lifetime exposure beginning at birth.[36] This implies that the change in predicted serum concentration is dependent only on the change in drinking water dose and independent of the dose from non-drinking water sources. The EPA additionally assumed that non-drinking water exposure is independent of the drinking water PFOA/PFOS concentration and estimated the total regulatory alternative dose as the sum of the baseline non-drinking water dose and the regulatory alternative drinking water dose.[37]

The EPA used the PK models to evaluate the following PWS EP-specific exposure scenarios in male and female subpopulations:

- **Lifetime baseline exposure scenario:** Lifetime exposure to baseline PFOA/PFOS drinking-water dose for cohorts of all ages alive at the start of the evaluation period in 2024 and cohorts born after 2024;
- **Lifetime regulatory alternative exposure scenario:** Lifetime exposure to regulatory alternative PFOA/PFOS drinking-water dose for cohorts born during or after 2029 (i.e., the year of full regulatory alternative implementation);
- **Partial lifetime treatment exposure scenario:** Exposure to baseline PFOA/PFOS drinking-water dose until age A–1 years and regulatory alternative PFOA/PFOS dose thereafter for cohorts aged A > 0 years in 2029.

The EPA selected the annual midpoint (the value on June 1 of each year) of the PK-modeled serum PFOA/PFOS concentration time series to represent the annual average serum PFOA/PFOS concentrations under the baseline and regulatory options. The EPA estimated changes in annual average serum PFOA/PFOS concentrations under the regulatory alternatives by subtracting baseline cohort-specific serum PFOA/PFOS concentrations from either full or partial lifetime cohort-specific serum PFOA/PFOS concentrations (as appropriate) under the regulatory alternatives. The EPA applied the PFOA/PFOS blood serum concentration time series estimated using the PK models to all benefits analyses that considered changes in PFOA/PFOS drinking water concentrations.

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[36] Specifically, let $C = \alpha \cdot D_t$, where C is serum concentration, α is a proportionality constant, and D_t is the total dose. This can be expanded to $C = \alpha \cdot D_t = \alpha \cdot (D_{dw} + D_o)$, where the total dose is the sum of the dose from drinking water, D_{dw}, and from other sources, D_o. The change in concentration due to a change in dose from drinking water is then $\Delta C = \alpha \cdot \Delta D_{dw} + \alpha \cdot \Delta D_o = \alpha \cdot \Delta D_{dw}$, given that the dose from other sources is constant, $\Delta D_o = 0$.

[37] The EPA used the fraction of exposure from drinking water under baseline conditions to estimate the total daily dose of PFOA/PFOS and the exposure from sources other than drinking water, which did not change upon implementation of the treatment scenario. While the total change in exposure is independent of the amount of exposure from other sources, the relative change in exposure does depend on the relative amount of exposure from non-drinking water sources. A greater fraction of exposure from drinking water sources will result in a greater relative change in total exposure upon implementation of the treatment scenario. The EPA also notes that, in reality, some portion of the non-drinking water exposure will be related to drinking water concentration (e.g., water used for cooking). This portion is difficult to estimate, and, depending on the relationship, there may be a time lag between the decrease in drinking water concentration and the decrease in the non-drinking water exposure.

The birth weight analysis focuses only on women of childbearing age defined by the Centers for Disease Control and Prevention (CDC) as those aged 15 to 44 (Ellington et al., 2020) and thus considers only maternal serum PFOA/PFOS levels. As described above, the PK models provide estimates of changes in serum PFOA/PFOS levels by PWS EP, age, and sex for each year during the period of analysis (2024 to 2105). The birth weight analysis requires a single estimate of change in maternal serum levels for each PFAS compound per year and location to evaluate potential changes in birth weight resulting from the regulatory alternatives. Therefore, the EPA used the race/ethnicity-specific distribution of populations of women of childbearing age during the period of analysis to estimate average annual race/ethnicity-specific change in PFOA/PFOS levels at each PWS EP and for each year. The EPA relied on the average age of race/ethnicity-specific women of childbearing age when determining PFOA/PFOS serum levels to reflect differences in maternal age across these groups. The population of women of childbearing age per PWS, race/ethnicity, age, and sex are based on population estimates for women aged 15 to 44 using county-level data from the U.S. Census (U.S. Census Bureau, 2020a; see Appendix B).[38]

6.3.3 Contributions from Other Sources

The regulatory alternatives considered in this economic analysis are based on potential reductions in PFOA/PFOS levels in drinking water. However, human exposures to PFOA and PFOS may also result from sources other than drinking water, including diet, ambient and indoor air, incidental soil/dust ingestion, consumer products, and others (U.S. EPA, 2024a; U.S. EPA, 2024b).

Following a systematic review of the PFOA and PFOS source contribution literature, the EPA identified ingestion of food as the dominant source of both PFOA and PFOS exposures in adults from the general population (U.S. EPA, 2024a; U.S. EPA, 2024b). This pathway is particularly dominant due to bioaccumulation of PFOA and PFOS in food from environmental emissions, large amounts of foods being consumed, and high gastrointestinal uptake. PFOA and PFOS may be present in food due to contact with non-stick cookware or grease-proofing agents in food packaging. PFOA and PFOS have also been shown to bioaccumulate in fish and shellfish. Consumer products, including certain cosmetics, textiles, and other household goods, are also a source of PFOA and PFOS exposure. While PFAS have been detected in ambient air globally, concentrations vary widely depending on location. PFAS have been detected in soils and dust from carpets and upholstered furniture. Incidental exposures from soils and dust are particularly important exposure routes for small children, who have a higher level of hand-to-mouth behavior compared to adults. PFAS levels in soils and surface water can also impact PFAS levels found in air particulates, fish, dairy products, meat/poultry, and produce (ATSDR, 2021; U.S. EPA, 2024a; U.S. EPA, 2024b).

6.4 Developmental Effects

Exposure to PFOA and PFOS is linked to developmental effects, including decreased infant birth weight (Steenland et al., 2018; Dzierlenga et al., 2020; Verner et al., 2015; Negri et al., 2017; ATSDR, 2018; ATSDR, 2021; Waterfield et al., 2020; U.S. EPA, 2016e; U.S. EPA, 2016f; U.S.

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[38] County-level population estimates are linked to PWSs based on the “counties served” field provided by the SDWIS/Fed 2021 Q4 database.

EPA, 2024e; U.S. EPA, 2024f). The route through which infants are exposed prenatally to PFOA and PFOS is maternal blood via the placenta. Most studies of the association between maternal serum PFOA/PFOS and birth weight report inverse relationships (Verner et al., 2015; Negri et al., 2017; Steenland et al., 2018; Dzierlenga et al., 2020).[39] This chapter outlines the overall methodology, assumptions, and data used for estimating changes in birth weight among infants whose mothers were exposed to PFOA and PFOS in drinking water during or prior to pregnancy.[40]

The EPA also considered the potential benefits from reduced exposure to PFNA that may be realized as a direct result of the final rule. The agency explored the birth weight impacts of PFNA in a sensitivity analysis based on epidemiological studies published before 2018 cited in the best available final human health analysis of PFNA (ATSDR, 2021), as well as a recently published meta-analysis of mean birth weight that indicates the birth weight results for PFNA are robust and consistent, even if associations in some studies may be small in magnitude (Wright et al., 2023). The EPA used a unit PFNA reduction scenario (i.e., 1 ppt change) and the PFAS serum calculator developed by Lu and Bartell (2020) to estimate PFNA blood serum levels resulting from PFNA exposures in drinking water. To estimate blood serum PFNA based on its drinking water concentration, the EPA used a first-order single-compartment model whose behavior was previously demonstrated to be consistent with PFOA pharmacokinetics in humans (Bartell et al., 2010). In addition to the PFOA-birth weight and PFOS-birth weight effects analyzed in the EA, the EPA examined the effect of inclusion of PFNA-birth weight effects using estimates from two studies (Lenters et al., 2016; Valvi et al., 2017). The EPA found that inclusion of a 1 ppt PFNA reduction could increase annualized birth weight benefits by a factor of 5.6 to 7.8, relative to the scenario that quantifies a 1 ppt reduction in PFOA and a 1 ppt reduction in PFOS only. The range of estimated PFNA-related increases in benefits is driven by the exposure-response, with smaller estimates produced using the slope factors from Lenters et al. (2016), followed by Valvi et al. (2017). The EPA notes that the PFNA slope factor estimates are orders of magnitude larger than the slope factor estimates used to evaluate the impacts of PFOA/PFOS reductions. The EPA also notes that the PFNA slope factor estimates in this analysis are not precise, with 95 percent CIs covering wide ranges that include zero (i.e., serum PFNA slope factor estimates are not statistically significant at 5 percent level). Caution should be exercised in making judgements about the potential magnitude of change in the national benefits estimates based on the results of these sensitivity analyses, although conclusions about the directionality of these effects can be inferred. The EPA did not include PFNA effects in the national benefits estimates for the final rulemaking because there was insufficient data above the UCMR 3 MRL to reasonably fit model parameters for PFNA (U.S. EPA, 2024g). For the EPA's PFNA sensitivity analysis, see Appendix K.

6.4.1 Overview of the Birth Weight Risk Reduction Analysis

Figure 6-1 provides an overview of the approach used to quantify and value the changes in birth weight-related risks associated with reductions in exposure to PFOA and PFOS via drinking water. Section 4.4 and Section 6.3 detail the PWS EP-specific PFOA/PFOS drinking water

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[39] Note that 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).

[40] The PK model assumes that mothers were exposed to PFOA/PFOS from birth to the year in which pregnancy occurred.

occurrence estimation and modeling of serum PFOA/PFOS concentrations, respectively. EP-specific time series of the differences between serum PFOA/PFOS concentrations under baseline and regulatory alternatives are inputs into this analysis. For each EP, evaluation of the changes in birth weight impacts involves the following key steps:

1. Estimating the changes in birth weight based on modeled changes in serum PFOA/PFOS levels and exposure-response functions for the effect of serum PFOA/PFOS on birth weight;
2. Estimating the difference in infant mortality probability between the baseline[41] and regulatory alternatives based on changes in birth weight under the regulatory alternatives and the association between birth weight and mortality;
3. Identifying the infant population affected by reduced exposure to PFOA/PFOS in drinking water under the regulatory alternatives;
4. Estimating the changes in the expected number of infant deaths under the regulatory alternatives based on the difference in infant mortality rates and the population of surviving infants affected by increases in birth weight due to reduced PFOA/PFOS exposure; and
5. Estimating the economic value of reducing infant mortality based on the Value of Statistical Life and infant morbidity based on reductions in medical costs associated with changes in birth weight for the surviving infants based on the cost of illness.

Section 6.4.2 discusses the exposure-response modeling for birth weight. Section 6.4.3 describes estimation of birth weight-related mortality and morbidity impacts in the affected population. Section 6.4.4 discusses the EPA’s valuation methodology for reductions in birth weight-related mortality and morbidity. Section 6.4.5 presents the results of the analysis.

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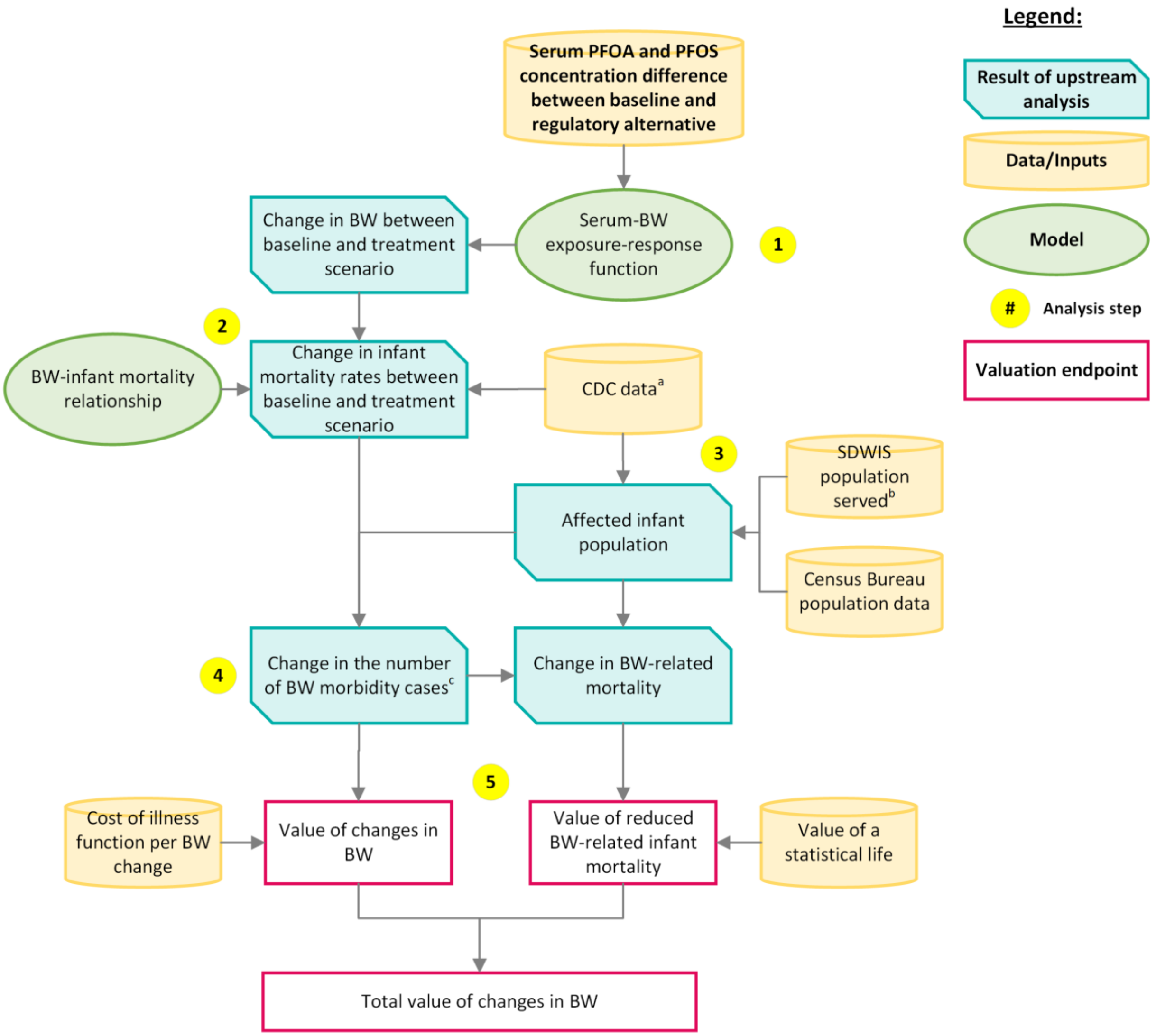
[41] Based on mortality rates per state and 500 g birth weight increment from the Centers for Disease Control and Prevention (CDC) from 2012 to 2018.



Notes:
SDWIS – Safe drinking water information system, CDC – Centers for Disease Control, BW – birth weight
[a]Includes baseline state-level birth rate and average BW (varies by 100-gm BW increment) and infant mortality rate (varies by 500-gm BW increment) data distributed based on national-level race/ethnicity-specific data. Baseline infant mortality rates, along with the BW-infant mortality relationship, are used to determine the change in infant mortality rate between the baseline and policy scenario. Birth rate and average BW data describe the affected population of infants.
[b]Includes both large and small surface water and ground water systems.
[c]Morbidity cases refer to the total affected population minus infant mortality cases under the regulatory alternatives.

Figure 6-1: Overview of Analysis of Birth Weight-Related Benefits

6.4.2 Estimation of Birth Weight Changes Between Baseline and Regulatory Alternatives

To estimate changes in birth weight resulting from reduced exposure to PFOA and PFOS under the regulatory alternatives, the EPA relied on the estimated time series of changes in serum

PFOA/PFOS concentrations specific to women of childbearing age and serum-birth weight exposure-response functions provided in recently published meta-analyses. The estimation of the time series of changes in serum PFOA/PFOS concentrations is explained in Section 6.3.2. The EPA reviewed five recent meta-analyses of PFAS-birth weight relationships in detail. As described in Table 6-8, two of the analyses used well-documented systematic review and risk of bias procedures to identify relevant studies in the literature (Johnson et al., 2014; Negri et al., 2017). The three other studies did not document risk of bias protocols and study quality evaluation criteria, however, the EPA evaluated most of the studies used in these meta-analyses for study quality (Verner et al., 2015; Dzierlenga et al., 2020; Steenland et al., 2018; U.S. EPA, 2024e; U.S. EPA, 2024f). As discussed below, there was extensive overlap in the studies used in the various meta-analyses. Two of the meta-analyses included exposure-response modeling for both PFOS and PFOA (Verner et al., 2015; Negri et al., 2017), while one addressed only PFOS (Dzierlenga et al., 2020) and the remaining two addressed only PFOA (Johnson et al., 2014; Steenland et al., 2018).

Table 6-8: Summary of Studies Relating PFOA or PFOS to Birth Weight

Author	PFOA	PFOS	Documented Risk of Bias Protocols
Johnson et al. (2014)	X		X
Verner et al. (2015)	X	X	
Negri et al. (2017)	X	X	X
Steenland et al. (2018)	X		
Dzierlenga et al. (2020)		X	

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctane sulfonic acid.

The EPA evaluated the applicability of these studies for use in the evaluation of birth weight changes resulting from reduced PFOS and PFOA exposure based on the following criteria: number of studies, homogeneity among studies, and sensitivity analyses. Based on these considerations, the agency selected results from Steenland et al. (2018) as the birth weight exposure-response function for PFOA and results from Dzierlenga et al. (2020) as the birth weight exposure-response function for PFOS.

Steenland et al. (2018) conducted a random effects meta-analysis based on 24 studies. The authors estimated a slope of −10.5 g birth weight per ng PFOA/mL with significant heterogeneity ($I^2 = 63\%$)[42] (p-value for heterogeneity <0.0001). The agency chose the results from this study for use in the risk assessment from exposure to PFOA and benefits analysis of reducing PFOA in drinking water because it is the most recent meta-analysis on PFOA-birth weight, and it included a large number of studies.

Dzierlenga et al. (2020) conducted a random effects meta-analysis based on 32 results from 29 studies. An EPA reanalysis of this study[43] estimated a slope of −3.0 g birth weight per ng PFOS/mL with significant heterogeneity ($I^2 = 58\%$) (p-value for heterogeneity <0.001). The agency chose the results from this study for use in the risk assessment from exposure to PFOS and benefits analysis of reducing PFOS in drinking water because it is the most recent meta-analysis on PFOS-birth weight and includes a large number of the most recent studies. While sensitivity analyses suggested that results may be sensitive to the timing of blood draw, the authors observed consistent inverse associations with birth weight among those with blood measurements in early pregnancy and in later pregnancy.

Changes in serum PFOA and PFOS concentrations are calculated for each PWS EP during each year in the analysis period. The EPA assumes that, given the long half-lives of PFOS and PFOA (with median half-lives of 2.7 and 3.5 years, respectively; Y. Li et al., 2018), any one-time measurement during or near pregnancy is reflective of a critical exposure window and not subject to considerable error. In other words, blood serum concentrations in a single year are expected to correlate with past exposures and are reflective of maternal exposures regardless of the timing of pregnancy. The mean change in birth weight per increment in long-term PFOA and PFOS exposure is calculated by multiplying each annual change in PFOA and PFOS serum concentration (ng/mL serum) by the PFOA and PFOS serum-birth weight exposure-response slope factors (g birth weight per ng/mL serum) provided in Table 6-9, respectively. The mean annual change in birth weight attributable to changes in both PFOA and PFOS exposure is the sum of the annual PFOA- and PFOS-birth weight change estimates. Appendix D provides additional details on the derivation of the exposure-response functions. Appendix K presents an analysis of birth weight risk reduction considering slope factors specific to the first trimester.

Table 6-9: Serum Exposure-Birth Weight Response Estimates

Compound	g Birth Weight/ng/mL Serum (95% CI)
PFOA[a]	−10.5 (−16.7, −4.4)
PFOS[b]	−3.0 (−4.9, −1.1)

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctane sulfonic acid; g – gram.
Notes:
[a] The serum-birth weight slope factor for PFOA is based on the main random effects estimate from Steenland et al. (2018).
[b] The serum-birth weight slope factor for PFOS is based on an EPA reanalysis of Dzierlenga et al. (2020).

The EPA places a cap on estimated birth weight changes in excess of 200 g based on existing studies that found that changes to environmental exposures result in relatively modest birth

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[42] I^2 represents the proportion of total variance in the estimated model due to inter-study variation.
[43] In the original Dzierlenga et al. (2020) estimate, the authors duplicated an estimate from M. H. Chen et al. (2017) in the pooled estimate. The EPA reran the analysis excluding the duplicated estimate.

weight changes (Windham & Fenster, 2008; Klein & Lynch, 2018; Kamai et al., 2019).[44,45] Modest changes in birth weight even as a result of large changes in PFOA/PFOS serum concentrations may be due to potential bias from studies only including live births (Liew et al., 2015). 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 evidence showing associations with PFOA/PFOS exposure and due to this uncertainty, these outcomes were not further assessed.

6.4.3 Estimation of Birth Weight Impacts

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 & Corman, 1991; J. R. Behrman & Rosenzweig, 2004; R. E. Behrman & Butler, 2007; Joyce et al., 2012; Kowlessar et al., 2013; Colaizy et al., 2016; Nicoletti et al., 2018; Klein & Lynch, 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). The EPA's analysis focuses on two categories of birth weight impacts that are amenable to monetization associated with incremental changes in birth weight: (1) medical costs associated with changes in infant birth weight and (2) the value of avoiding infant mortality at various birth weights.

The birth weight literature related to other sources of economic burden to society (e.g., parental and caregiver costs and productivity losses) is limited in geographic coverage, population size, and range of birth weights evaluated and therefore cannot be used in the economic analysis of birth weight effects from exposure to PFOA/PFOS in drinking water (ICF, 2021). The following sections summarize the relationship between infant mortality and birth weight as well as methods used to estimate changes in the number of infant deaths and the number of surviving infants whose birth weight is affected by reduced PFOA/PFOS exposures.

6.4.3.1 Impacts of Birth Weight on Infant Mortality

Infant mortality is defined as the deaths among infants who were delivered alive but passed before their first birthday. Birth weight is a significant factor in infant survival (Jacob, 2016). Epidemiology studies in the U.S. have reported relationships between birth weight and mortality. Most of these studies typically evaluate relationships between infant mortality and birth weight above or below various birth weight thresholds (e.g., McIntire et al., 1999; Lau et al., 2013). However, even small changes in birth weight could result in substantial avoided mortality benefits.

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[44] Klein et al. (2018) indicate that birth weight changes in response to reduced environmental exposures are likely to be small and simulated changes in birth weight up to 100 g. Kamai et al. (2019) found maximum changes in birth weight in response to reduced exposures to cigarette smoke of 150 g, while Windham et al. (2008) found a maximum decrement in mean birth weight of 200 g for infants of smokers.

[45] Under the final rule, the EPA estimates that the 200 g birth weight cap is triggered in 0.01 percent of affected infants.

Two studies showed statistically significant relationships between incremental changes in birth weight and infant mortality: Almond et al. (2005) and Ma and Finch (2010). Ma and Finch (2010) used 2001 National Center for Health Statistics (NCHS) linked birth/infant death data for singleton and multiple birth infants among subpopulations defined by sex and race/ethnicity to estimate a regression model assessing the associations between 14 key birth outcome measures, including birth weight, and infant mortality. They found notable variation in the relationship between birth weight and mortality across race/ethnicity subpopulations, with odds ratios for best-fit birth weight-mortality models ranging from 0.8-1 (per 100 g birth weight change). Almond et al. (2005) used 1989-1991 NCHS linked birth/infant death data for multiple birth infants to analyze relationships between birth weight and infant mortality within birth weight increment ranges. For their preferred model, they reported coefficients in deaths per 1,000 births per 1 g increase in birth weight that range from −0.420 to −0.002. However, the data used in these studies (Almond et al., 2005 and Ma & Finch, 2010) are outdated (1989-1991 and 2001, respectively). Given the significant decline in infant mortality over the last 30 years (ICF, 2020) and other maternal and birth characteristics that are likely to influence infant mortality (e.g., average maternal age and rates of maternal smoking), the birth weight-mortality relationship estimates from Almond et al. (2005) and Ma and Finch (2010) are likely to overestimate the benefits of birth weight changes.

Considering the discernible changes in infant mortality over the last 30 years, the EPA developed a regression analysis to estimate the relationship between birth weight and infant mortality using the Period/Cohort Linked Birth-Infant Death Data Files published by NCHS from the 2017 period/2016 cohort and the 2018 period/2017 cohort (CDC, 2017, 2018). These data provide information on infants who are delivered alive and receive a birth certificate.[46] The EPA selected variables of interest for the regression analysis, including maternal demographic and socioeconomic characteristics, maternal risk and risk mitigation factors (e.g., number of prenatal care visits, smoker status), and infant birth characteristics. The EPA included several variables used in Ma and Finch (2010) (maternal age, maternal education, marital status, and others – see Appendix E for the complete list) as well as additional variables to augment the set of covariates included in the analyses. In addition, the EPA developed separate models for different race/ethnicity categories (non-Hispanic Black, non-Hispanic White, and Hispanic) and interacted birth weight with categories of gestational age, similar to Ma and Finch (2010).[47] Appendix E provides details on model development and regression results.

Table 6-10 presents the resulting odds ratios and marginal effects (in terms of deaths per 1,000 births for every 1 g increase in birth weight) estimated for changes in birth weight among different gestational age categories in the mortality regression models for non-Hispanic Black, non-Hispanic White, and Hispanic race/ethnicity subpopulations. Marginal effects for birth weight among different gestational age categories indicate the change in the incidence of infant

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[46] These data do not include information on miscarriages or stillbirths.

[47] Note that Ma and Finch (2010) developed a model for infants with Mexican heritage, rather than the Hispanic population, and interacted birth weight with gestational age as a continuous interaction variable, rather than developing different birth weight variables per gestational age category. Ma and Finch (2010) did not consider the Hispanic paradox, a term for the epidemiological finding that Hispanic and Latino Americans often have lower risk of poor health outcomes compared to race/ethnicity groups with higher income and education levels..

mortality per 1 g increase in birth weight.[48] Marginal effects for birth weight among gestational age categories vary across different race/ethnicity subpopulations. As shown in Figure 6-2, the marginal effects for birth weight among different gestational age categories are higher in the non-Hispanic Black model than in the non-Hispanic White and Hispanic models, particularly for extremely and very preterm infants, indicating that LBW increases the probability of mortality within the first year more so among non-Hispanic Black infants than among non-Hispanic White and Hispanic infants.

The EPA relies on odds ratios estimated using the birth weight-mortality regression model to assess mortality outcomes of reduced exposures to PFOA/PFOS in drinking water under the regulatory alternatives. To obtain odds ratios specific to each race/ethnicity and 100 g birth weight increment considered in the birth weight benefits model,[49] the EPA averaged the estimated odds ratios for 1 g increase in birth weight over the gestational age categories using the number of infants (both singleton and multiple birth) that fall into each gestational age category as weights. Separate gestational age category weights were computed for each 100 g birth weight increment and race/ethnicity subpopulation within the 2017 period/2016 cohort and 2018 period/2017 cohort Linked Birth-Infant Death Data Files. The weighted birth weight odds ratios are then used in conjunction with the estimated change in birth weight and baseline infant mortality rates to determine the probability of infant death under the regulatory alternatives, as described further in Section 6.4.3.1.

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[48] All marginal effect values for birth weight among different gestational age categories are negative and decrease in magnitude with each higher gestational age category, indicating that the probability of mortality decreases as gestational age and birth weight increase. For example, using marginal effects from the non-Hispanic Black model, for extremely preterm infants a 100 g birth weight increase on average would translate to 20 fewer infant deaths per 1,000 births in this gestational age category or a 2% decrease in the probability of mortality within one year of birth. The same birth weight increase at a higher gestational age would still decrease mortality risk but to a lesser extent.

[49] The birth weight risk reduction model evaluates changes in birth weight in response to PFOA/PFOS drinking water level reductions for infants who fall into 100 g birth weight increments (e.g., birth weight 0-99 g, 100-199 g, 200-299 g… 8,000-8,099 g, 8,100-8,165 g).

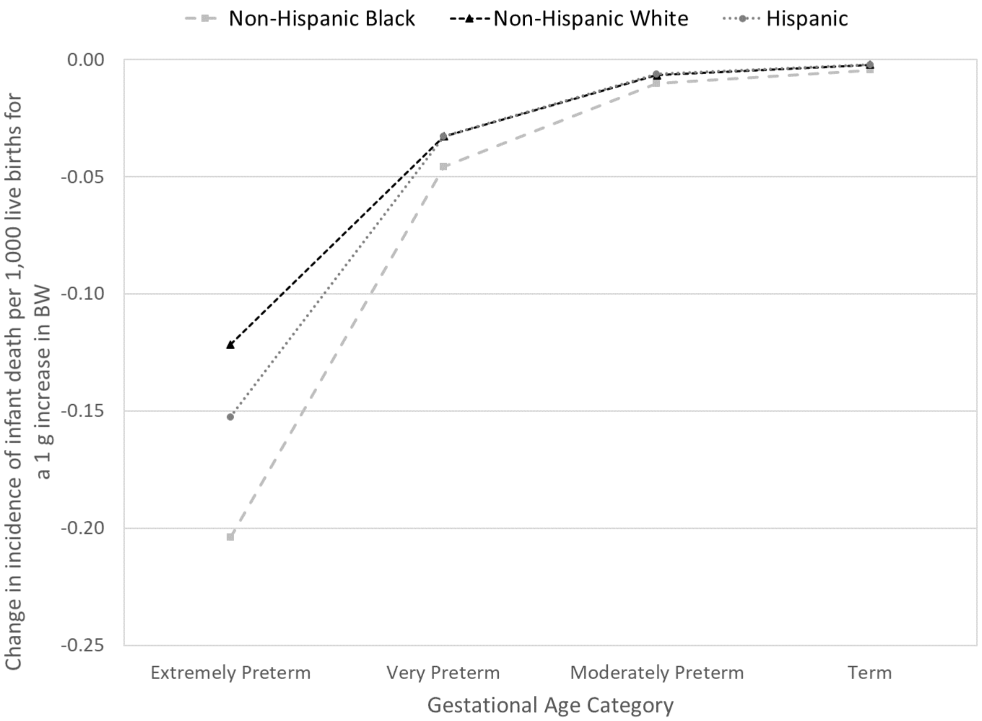

Figure 6-2: Comparison of Change in Incidence of Infant Death per 1 g Increase in Birth Weight by Gestational Age Category and Race/Ethnicity (Deaths per 1,000 Births)

Notes: Gestational age categories defined as extremely preterm (<=28 weeks), very preterm (>28 weeks and <=32 weeks), moderately preterm (>32 weeks and <=37 weeks), and term (>37 weeks). Data based on the 2016/17 and 2017/18 CDC Period Cohort Linked Birth-Infant Death Data Files obtained from NCHS/NVSS. Marginal effects and odds ratios are estimated using a regression model that also includes covariates representative of infant birth characteristics in addition to birth weight, maternal demographic characteristics, and maternal risk factors. Details are included in Appendix E.

Table 6-10: Race/Ethnicity- and Gestational Age-Specific Birth Weight Marginal Effects and Odds Ratios from the Mortality Regression Models

Race	Gestational Age Category[b]	Marginal Effect per 1,000 births (95% CI)	Odds Ratio (95% CI)
Non-Hispanic Black	Extremely Preterm	-0.20400 (-0.21910, -0.18890)	0.99817 (0.99802, 0.99832)
	Very Preterm	-0.04580 (-0.04820, -0.04340)	0.99816 (0.99804, 0.99827)
	Moderately Preterm	-0.01030 (-0.01080, -0.009850)	0.99852 (0.99846, 0.99857)
	Term	-0.00453 (-0.00472, -0.00434)	0.99856 (0.99851, 0.9986)
Non-Hispanic White	Extremely Preterm	-0.12160 (-0.13080, -0.11240)	0.99866 (0.99855, 0.99878)
	Very Preterm	-0.03290 (-0.03430, -0.03140)	0.9985 (0.99842, 0.99858)
	Moderately Preterm	-0.00677 (-0.00702, -0.00652)	0.99867 (0.99863, 0.99872)
	Term	-0.00228 (-0.00236, -0.00221)	0.99865 (0.99861, 0.99868)
Hispanic	Extremely Preterm	-0.15260 (-0.16770, -0.13750)	0.99835 (0.99817, 0.99853)
	Very Preterm	-0.03290 (-0.03510, -0.03070)	0.99846 (0.99835, 0.99858)
	Moderately Preterm	-0.00626 (-0.00659, -0.00592)	0.99856 (0.99849, 0.99862)
	Term	-0.00219 (-0.00229, -0.00208)	0.99849 (0.99844, 0.99855)

Notes:
[a] Data based on the 2016/17 and 2017/18 CDC Period Cohort Linked Birth-Infant Death Data Files obtained from NCHS/NVSS. Marginal effects and odds ratios are estimated using a regression model that also includes covariates representative of infant birth characteristics in addition to birth weight, maternal demographic characteristics, and maternal risk factors. All effects were statistically significant at the 5% level. Additional details are included in Appendix E.
[b] Gestational age categories defined as extremely preterm (<=28 weeks), very preterm (>28 weeks and <=32 weeks), moderately preterm (>32 weeks and <=37 weeks), and term (>37 weeks).

The EPA weighted the race/ethnicity-specific mortality odds ratios in Table 6-10 by the proportions of the infant populations who fell into each gestational age within a 100 g birth weight increment, based on the 2016/17 and 2017/18 period cohort data, to obtain a weighted mortality odds ratio estimate for each modeled race/ethnicity subpopulation and 100 g birth weight increment. The weighted mortality odds ratios are shown in Figure 6-3.[50]

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[50] Note that weighted mortality odds ratios for the Hispanic population at larger birth weight increments fluctuate between 0.99849 and 0.99856. Due to the small sample size of the Hispanic infant population within these birth weight increments, 100 percent of infants in a specific birth weight increment is associated with either moderately preterm or term gestational age categories. For instance, all Hispanic infants included in the analysis who were between 7,800 and 7,899 g were full-term, while all Hispanic infants who were between 7,900 and 7,999 g were moderately preterm. Therefore, the weighted mortality odds ratio for Hispanic infants between 7,800 and 7,899 g is equal to the full-term mortality odds ratio estimated for the Hispanic infant population, while the weighted mortality odds ratio for Hispanic infants between 7,900 and 7,999 g is equal to the moderately preterm mortality odds ratio estimated for the Hispanic infant population.

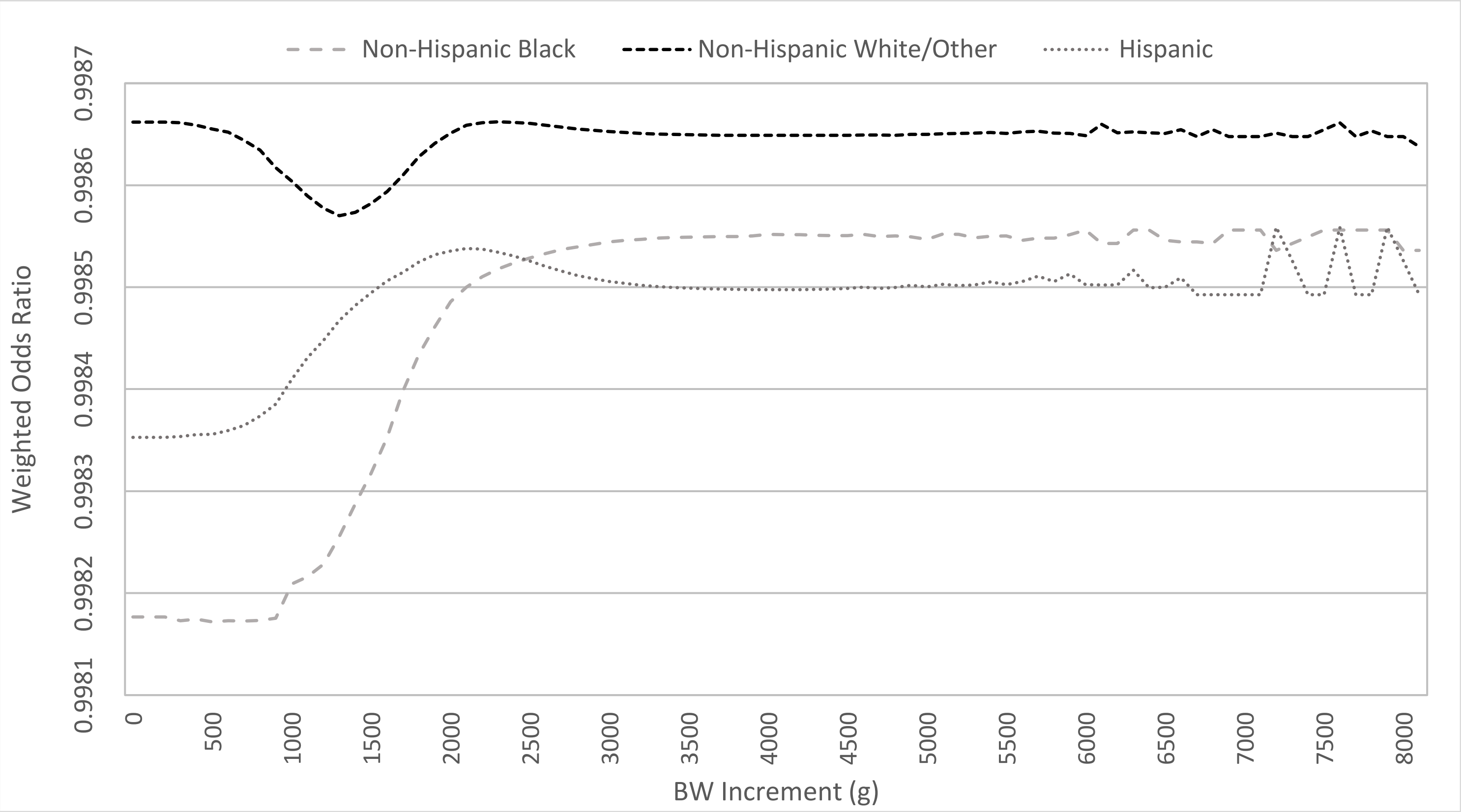

Figure 6-3: Weighted Mortality Odds Ratios Based on Populations of Infants Falling into 100 g Birth Weight Increments and Four Gestational Age Categories

Note: Weighted mortality odds ratios refer to the exponentiation of the sum of odds ratios estimated for each gestational age category and race/ethnicity-specific infant population multiplied by the proportions of the infant populations who fell into each gestational age within a 100 g birth weight increment, based on the 2016/17 and 2017/18 CDC Period Cohort Linked Birth-Infant Death Data Files obtained from NCHS/NVSS, to obtain a weighted odds ratio estimate for each modeled race/ethnicity and 100 g birth weight increment. The EPA applies the weighted mortality odds ratios estimated for the non-Hispanic White subpopulation to the “other” race/ethnicity subpopulation because of similarities in infant death rates from 2016 to 2018 among non-Hispanic White infants (4.75 deaths per 1,000) and non-Hispanic other infants (4.45 deaths per 1,000).

Note that the EPA did not model the relationship between birth weight and infant mortality for other race/ethnicity subpopulations because doing so for each individual race/ethnicity or combination of all "other" races/ethnicities is precluded by very low sample sizes (i.e., imprecise coefficients and imprecise marginal effects). The EPA applies the weighted mortality odds ratios estimated for the non-Hispanic White subpopulation to the "other" race/ethnicity subpopulation because of similarities in infant death rates from 2016 to 2018 among non-Hispanic White infants (4.75 deaths per 1,000) and non-Hispanic other infants (4.45 deaths per 1,000).

6.4.3.2 Estimating the Number of Infants Affected by Birth Weight Changes and Changes in Infant Mortality

Based on reduced serum PFOA/PFOS exposures under the regulatory alternatives and the estimated relationship between birth weight and infant mortality, the EPA estimates the subsequent change in birth weight for those infants affected by decreases in PFOA/PFOS and changes in the number of infant deaths. The EPA evaluates these changes at each PWS EP affected by the regulatory alternatives and the calculations are performed for each race/ethnicity group, 100 g birth weight category, and year of the analysis.

6.4.3.2.1 Changes in Birth Weight

The EPA combined estimated average annual changes in PFOA and PFOS serum levels for women of childbearing age (15 to 44 years old) by analysis year, race/ethnicity group, and PWS EP (see Section 6.3.2) with the serum PFOA/PFOS-birth weight exposure-response slope factors (see Table 6-9) to compute average annual changes in birth weight per newborn as follows:

Equation 6:

$$\Delta BW_{y,r,p} = \max\left(CAP, SF_{BW,PFOA} \cdot \Delta PFOA\_Serum_{y,r,p} + SF_{BW,PFOS} \cdot \Delta PFOS_{Serum_{y,r,p}}\right)$$

Where ΔBW is the change in birth weight under the regulatory alternatives, y is the analysis year, r is the race/ethnicity group, p is the PWS EP analyzed; $\Delta PFOA\_Serum$ is the change in PFOA serum for women of childbearing age under the regulatory alternatives; $\Delta PFOS\_Serum$ is the change in PFOS serum for women of childbearing age under the regulatory alternatives; $SF_{BW,PFOA}$ and $SF_{BW,PFOS}$ are the serum-birth weight exposure-response slope factors for PFOA and PFOS, respectively; and CAP is the 200 g cap placed on the birth weight changes.

6.4.3.2.2 Changes in Infant Death Rate

The EPA used average annual changes in birth weight under the regulatory alternatives (Equation 6) to estimate the associated infant mortality odds ratios, $OR_{y,i,r,p}$:

Equation 7:

$$OR_{y,i,r,p} = \exp(\Delta BW_{y,r,p} \cdot \ln(OR_{i,r}))$$

Where y is the analysis year, i is the 100 g birth weight increment, r is the race/ethnicity group, p is the PWS EP analyzed, and $OR_{i,r}$ is the weighted odds ratio for a 1 g birth weight increase associated with each 100 g birth weight increment for a given race/ethnicity category (see Section 6.4.3).

The EPA combined the result of Equation 7 with the baseline infant death rate to estimate the infant death rate under the regulatory alternatives, $DR_{Regulatory\ Alternative,y,i,r,p}$:

Equation 8:

$$DR_{Regulatory\ Alternative,y,i,r,p} = \frac{OR_{y,i,r,p} \cdot DR_{Baseline,y,i,r,p}}{1 + OR_{y,i,r,p} \cdot DR_{Baseline,y,i,r,p}}$$

Where $DR_{Baseline,y,i,r,p}$ is the baseline death rate per birth computed from 2012-2018 death rates per 500 g birth weight increment (CDC, 2020a),[51] y is the analysis year, i is 100 g birth weight increment, r is the race/ethnicity group, p is the PWS EP analyzed, and $OR_{y,i,r,p}$ is the mortality odds ratio associated with the annual change in birth weight under the regulatory alternatives.

6.4.3.2.3 Affected Infant Population Size

The annual race/ethnicity- and PWS EP-specific number of infants affected by changes in PFOA/PFOS drinking water levels is based on the 2021 retail population served at each PWS from the SDWIS/Fed and 2021 race/ethnicity-specific population estimates from the U.S. Census (U.S. Census Bureau, 2020a; see Appendix B). Because birth rates per race/ethnicity group and 100 g birth weight increment are often suppressed due to lack of data, the EPA multiplied state-level birth rates per race/ethnicity group from the Centers for Disease Control and Prevention (CDC) Linked Birth/Infant Death records from 2012 to 2018 (CDC, 2020a) by the ratio of infants falling within each 100 g birth weight increment per state (not specific to race/ethnicity) to the total number of infants per state to distribute the number of affected infants in each state. The EPA imputed state-level data that was missing from the 2012-2018 CDC Linked Birth/Infant Death records with data at the census region level. The EPA used the same approach to assign average birth weights per race/ethnicity group over the 100 g birth weight increments for use in COI data matching (See Section 6.4.4). Using the 2012-2018 imputed state-level birth rate data, the EPA computed the share of births that correspond to each 100 g birth weight increment (i), race/ethnicity (r), and PWS EP (p) as the ratio of race/ethnicity- and state-specific (s) birth rates[52] in a particular birth weight increment to the sum of birth rates associated with all birth weight increments:

Equation 9:

$$Share\ of\ Births_{i,r,p} = \frac{\left(BR_{2012-2018,i,r,s}\right)}{sum\left(BR_{2012-2018,i,r,s}\right)}$$

Next, the EPA assumed that the share of births within each 100 g birth weight increment (from Equation 9) would remain constant throughout the period of analysis and estimated the annual

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[51] The EPA assumed that the same death rate applies to infants in all 100 g birth weight increments falling in the 500 g birth weight range.

[52] In this analysis, the EPA applies state-specific birth rates that correspond to the state for which each PWS EP is located.

affected infant population size for each future analysis year (y), 100 g birth weight increment (i), race/ethnicity group (r), and PWS EP (p), $Births_{y,i,r,p}$ as follows:

Equation 10:

$$Births_{y,i,r,p} = Births_{y,r,p} \cdot Share\ of\ Births_{i,r,p}$$

6.4.3.2.4 Infant Deaths Avoided and the Number of Surviving Infants

The EPA used the estimated annual infant population size, $Births_{y,i,r,p}$, along with infant death rates, $DR_{Baseline,y,i,r,p}$ and $DR_{Regulatory\ Alternative,y,i,r,p}$, to compute the annual number of deaths expected at baseline (Equation 11) and the annual number of deaths expected under the regulatory alternatives (Equation 12):

Equation 11:

$$Deaths_{Baseline,y,i,r,p} = Births_{y,i,r,p} \cdot DR_{Baseline,y,i,r,p}$$

Equation 12:

$$Deaths_{Regulatory\ Alternative,y,i,r,p} = Births_{y,i,r,p} \cdot DR_{Regulatory\ Alternative,y,i,r,p}$$

The EPA estimated the annual number of avoided infant deaths, $Avoided\ Deaths_{y,i,r,p}$, as:

Equation 13:

$$Avoided\ Deaths_{y,i,r,p} = Deaths_{Baseline,y,i,r,p} - Deaths_{Regulatory\ Alternative,y,i,r,p}$$

The EPA computed the population of surviving infants whose birth weight would be affected by changes in PFOA/PFOS exposure ($Survivors_{Regulatory\ Alternative,y,i,r,p}$) as the number of births less the number of deaths under the regulatory alternatives. The EPA estimated the annual number of avoided infant deaths, $Avoided\ Deaths_{y,i,r,p}$, as:

Equation 14:

$$Survivors_{Regulatory\ Alternative,y,i,r,p} = Births_{y,i,r,p} \cdot (1 - DR_{Regulatory\ Alternative,y,i,r,p})$$

6.4.4 Valuation of Reduced Birth Weight Impacts

The EPA uses the Value of Statistical Life to estimate the benefits of reducing infant mortality and COI to estimate the economic value of increasing birth weight in the population of surviving infants born to mothers exposed to PFOA and PFOS in drinking water. Value of Statistical Life updating information is provided in Section 2.2.

The EPA's approach to monetizing benefits associated with incremental increases in birth weight resulting from reductions in drinking water PFOA/PFOS levels relies on avoided medical costs associated with various ranges of birth weight. Although the economic burden of treating infants at various birth weights also includes non-medical costs, very few studies to date have quantified such costs (Klein & Lynch, 2018; ICF, 2021). The EPA selected the medical cost function from Klein and Lynch (2018) to monetize benefits associated with the estimated changes in infant birth weight resulting from reduced maternal exposure to PFOA/PFOS.[53] The EPA selected the cost function from Klein and Lynch (2018) because it is based on recent data on birth weight, healthcare utilization, and healthcare costs that encompass a longer time period and a larger population than data used in other studies (e.g., Almond et al., 2005). Additional studies that the EPA reviewed provided only an incremental cost for LBW infants compared to normal birth weight infants (greater or equal to 2,500 g; e.g., Almond et al., 2010 and Malits et al., 2018). Klein and Lynch (2018), on the other hand, estimated incremental medical costs as a function of birth weight over the range from 900 to 4,500 g and used a continuous spline function (Figure 6-4), rather than allowing for a discontinuity at the very low birth weight level (i.e., < 1,500 grams). Table 6-11 summarizes the incremental cost changes associated with birth weight increases from Klein and Lynch (2018).

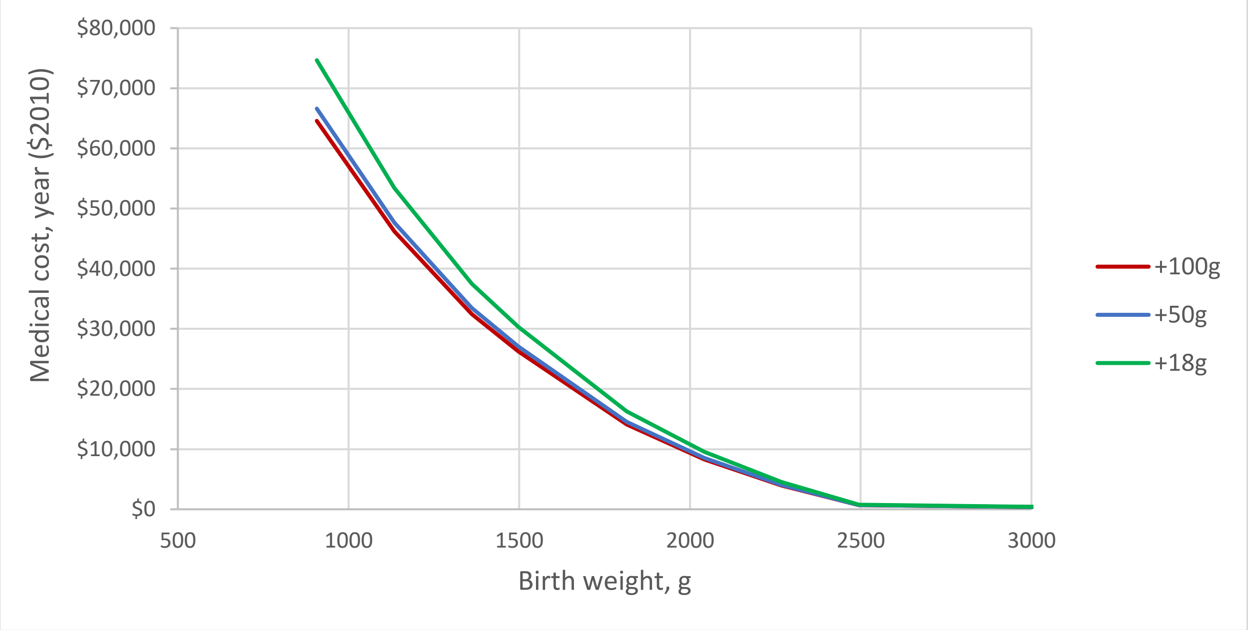

Figure 6-4: Piecewise Medical Cost Function Calculated by Klein and Lynch (2018) for Three Increments in Increased Birth Weight (18 g, 50 g, and 100 g)

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[53] The Klein and Lynch (2018) report was externally peer reviewed by three experts with qualifications in economics and public health sciences. The EPA's charge questions to the peer reviewers sought input on the methodology for developing medical cost estimates associated with changes in birth weight. The agency's charge questions and peer reviewer responses are available in the docket (see No. EPA-HQ-OW-2022-0114 at https://www.regulations.gov/docket/EPA-HQ-OW-2022-0114).

Table 6-11: Simulated Cost Changes for Birth Weight Increases ($2022) (Based on Klein and Lynch, 2018 Table 8)

Birth Weight[a]	Simulated Cost Changes for Birth Weight Increases, Dollars per Gram ($2022)[b]		
	+0.04 lb (+18 g)	+0.11 lb (+50 g)	+0.22 lb (+100 g)
2 lb (907 g)	-$131.66	-$117.44	-$113.82
2.5 lb (1,134 g)	-$98.72	-$88.07	-$85.35
3 lb (1,361 g)	-$74.03	-$66.04	-$64.00
3.3 lb (1,497 g)	-$62.29	-$55.56	-$53.85
4 lb (1,814 g)	-$41.63	-$37.13	-$35.99
4.5 lb (2,041 g)	-$31.21	-$27.84	-$26.98
5 lb (2,268 g)	-$23.41	-$20.88	-$20.23
5.5 lb (2,495 g)	-$0.97	-$0.88	-$0.87
6 lb (2,722 g)	-$0.95	-$0.86	-$0.86
7 lb (3,175 g)	-$0.92	-$0.83	-$0.83
8 lb (3,629 g)	-$0.89	-$0.81	-$0.80
9 lb (4,082 g)	$3.28	$2.99	$3.01
10 lb (4,536 g)	$3.69	$3.37	$3.39

Notes:
[a]Note that simulated medical costs increase, rather than decrease, in response to increased birth weight changes among high birth weight infants (those greater than 8 lb). Among high birth weight infants, there is a higher risk of birth trauma, metabolic issues, and other health problems (Klein & Lynch, 2018).
[b]Values scaled from $2010 to $2022 using the medical care Consumer Price Index (U.S. Bureau of Labor Statistics, 2022a).

Using the incremental cost changes from Klein and Lynch (2018), the EPA calculates the change in medical costs resulting from changes in birth weight among infants in the affected population who survived the first year following birth. To do so, the EPA linearly interpolates between the birth weight and cost values presented in Klein and Lynch (2018) to obtain a cost value for every 1 g birth weight increment, as shown in Figure 6-5. The EPA then matches this interpolated birth weight value to the nearest baseline average birth weight value in each 100 g birth weight increment to obtain the simulated cost change for birth weight increases that are estimated to be between zero and 18 g, between 19 and 50 g, and between 51 and 100 g or more.[54]

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[54] Note that the EPA caps birth weight changes at 200 g, as described in earlier sections. The EPA assumes that the cost of illness estimates for birth weight increases between 51 and 100 g apply to birth weight increases greater than 100 g.

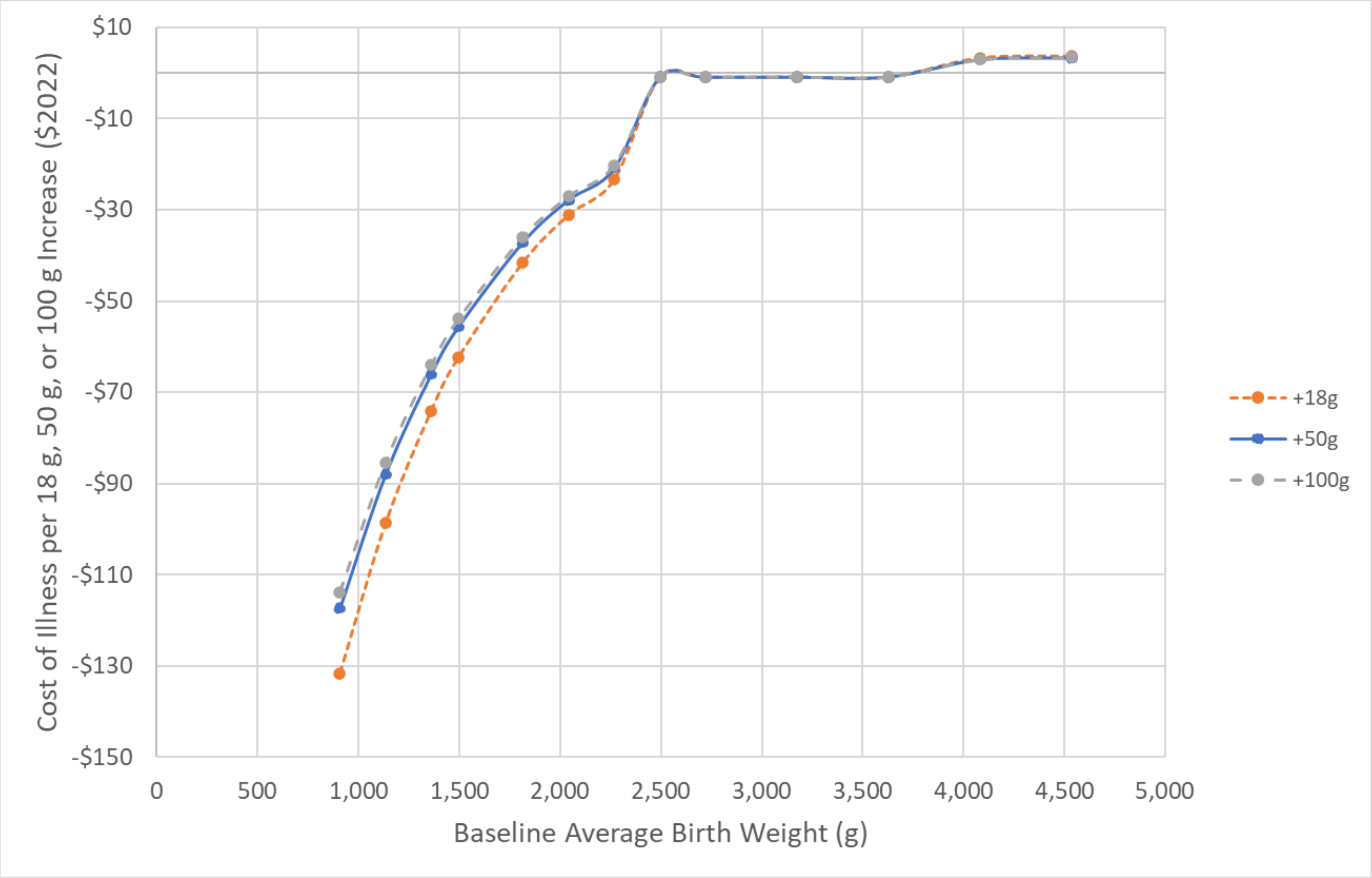

Figure 6-5. Interpolated Cost of Illness at Baseline Average Birth Weights, by Estimated Change in Birth Weight Under the Final Rule

6.4.5 Results

Table 6-12 to Table 6-15 provide the health effects avoided and valuation associated with birth weight impacts.

Table 6-12: National Birth Weight Benefits, Final Rule (PFOA and PFOS MCLs of 4.0 ppt each, PFHxS, PFNA, and HFPO-DA MCLs of 10 ppt each and HI of 1) (Million $2022)

Benefits Category	2% Discount Rate		
	5th Percentile[a]	Expected Value	95th Percentile[a]
Increase in Birth Weight (millions of grams)	129.6	216.8	304.1
Number of Birth Weight-Related Deaths Avoided	781.9	1,301.7	1,823.6
Total Annualized Birth Weight Benefits (Million $2022)[b]	**$124.85**	**$209.00**	**$292.78**

Note: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates. Quantifiable benefits are increased under final rule table results relative to the other options presented because of modeled PFHxS occurrence, which results in additional quantified benefits from co-removed PFOA and PFOS.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.

[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

Table 6-13: National Birth Weight Benefits, Option 1a (PFOA and PFOS MCLs of 4.0 ppt) (Million $2022)

Benefits Category	2% Discount Rate		
	5th Percentile[a]	Expected Value	95th Percentile[a]
Increase in Birth Weight (millions of grams)	128.8	215.6	302.1
Number of Birth Weight-Related Deaths Avoided	777.4	1,294.4	1,812.9
Total Annualized Birth Weight Benefits (Million $2022)[b]	**$124.82**	**$207.82**	**$291.00**

Note: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.

[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

Table 6-14: National Birth Weight Benefits, Option 1b (PFOA and PFOS MCLs of 5.0 ppt) (Million $2022)

Benefits Category	2% Discount Rate		
	5th Percentile[a]	Expected Value	95th Percentile[a]
Increase in Birth Weight (millions of grams)	111.3	185.6	260.3
Number of Birth Weight-Related Deaths Avoided	668.9	1,114.7	1,561.2
Total Annualized Birth Weight Benefits (Million $2022)[b]	**$107.34**	**$178.97**	**$250.00**

Note: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.

[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

Table 6-15: National Birth Weight Benefits, Option 1c (PFOA and PFOS MCLs of 10.0 ppt) (Million $2022)

Benefits Category	2% Discount Rate		
	5th Percentile[a]	Expected Value	95th Percentile[a]
Increase in Birth Weight (millions of grams)	62.1	102.0	142.4
Number of Birth Weight-Related Deaths Avoided	375.8	616.6	859.1
Total Annualized Birth Weight Benefits (Million $2022)[b]	**$60.24**	**$98.97**	**$137.75**

Note: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.

[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

6.5 Cardiovascular Disease

6.5.1 Overview of the Cardiovascular Disease Risk Analysis

Figure 6-6 provides an overview of the approach used to quantify and value the changes in CVD risk associated with reductions in exposure to PFOA and PFOS via drinking water. Section 4.4 details the PWS EP-specific PFOA/PFOS drinking water occurrence estimation and Section 6.3 describes modeling of serum PFOA/PFOS concentrations. EP-specific time series of the differences between serum PFOA/PFOS concentrations under baseline and regulatory alternatives are inputs into this analysis. For each EP, evaluation of the changes in CVD risk involves the following key steps:

1. Estimation of annual changes in TC[55] and BP levels using exposure-response functions for the potential effects of serum PFOA/PFOS on these biomarkers;
2. Estimation of the annual incidence of fatal and non-fatal first hard CVD events,[56] defined as fatal and non-fatal myocardial infarction (MI; i.e., heart attack), fatal and non-fatal IS, or other coronary heart disease (CHD) death occurring in populations without prior CVD event experience (D'Agostino et al., 2008; Goff et al., 2014; Lloyd-Jones et al., 2017), and post-acute CVD mortality corresponding to baseline and regulatory alternative TC and BP levels in all populations alive during or born after the start of the evaluation period; and
3. Estimation of the economic value of reducing CVD mortality and morbidity from baseline to regulatory alternative levels, using the Value of Statistical Life and COI measures, respectively.

Section 6.5.2 discusses the exposure-response models for TC and BP. Section 6.5.3 details the estimated CVD risk reductions using the Pooled Cohort ASCVD risk model (Goff et al., 2014) and the life table approach. Section 6.5.4 discusses the EPA's valuation methodology for fatal and non-fatal CVD events. Section 6.5.5 presents the results of the analysis.

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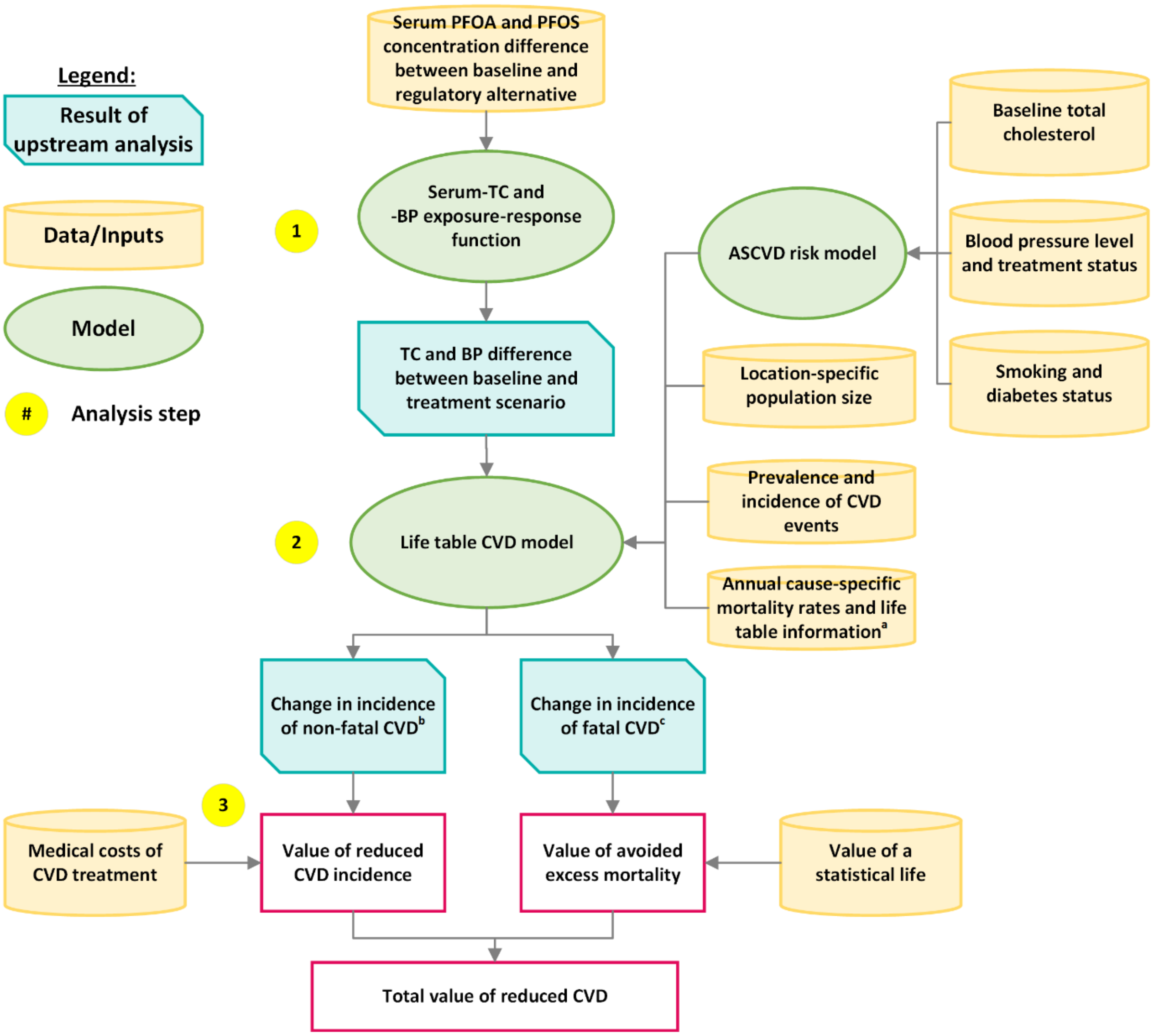
[55] The EPA discusses the relationship between PFOA/PFOS exposure and other forms of cholesterol in Appendix F.

[56] Hard CVD events include fatal and non-fatal myocardial infarction, fatal and non-fatal stroke, and other coronary heart disease mortality.



Abbreviations: PFOA – perfluorooctanoic acid, PFOS – perfluorooctanesulfonic acid, TC – total cholesterol, BP – blood pressure, CVD – cardiovascular disease, ASCVD – atherosclerotic cardiovascular disease, MI – myocardial infarction, IS – ischemic stroke, CHD – coronary heart disease

Notes:

[a] Data from the Centers for Disease Control (CDC) and Prevention.

[b] Non-fatal CVD includes non-fatal first MI and non-fatal first IS.

[c] Fatal CVD includes fatal first MI, fatal first IS, other fatal first CHD events, and post acute CVD mortality among survivors of the first MI and the first IS.

Figure 6-6: Overview of the CVD Risk Model

6.5.2 Cardiovascular Disease Exposure-Response Analyses

6.5.2.1 Estimation of Cholesterol Changes

The ASCVD model includes TC as a predictor of first hard CVD events. The EPA did not identify any readily available relationships for PFOA or PFOS and TC that were specifically relevant to the age group of interest (40-89 years, the years for which the ASCVD model estimates the probability of a first hard CVD event). Therefore, the agency developed a meta-analysis of studies reporting associations between serum PFOA or PFOS and TC in general populations (e.g., populations that are not a subset of workers or pregnant women). Statistical analyses that combine the results of multiple studies, such as meta-analyses, are widely applied to investigate the associations between contaminant levels and associated health effects. Such analyses are suitable for economic assessments because they can improve precision and statistical power (Engels et al., 2000; Deeks, 2002; Rücker et al., 2009). Appendix F provides details on the studies selection criteria, meta-data development, meta-analysis results, and discussion of the uncertainty and limitations inherent in the EPA's exposure-response analysis.

The EPA identified studies for inclusion in the meta-analysis using data from literature reviews, including those performed by the ATSDR in the development of their Toxicological Review Public Comment Draft (ATSDR, 2018), which included literature through mid-2017, and those performed for developing the EPA's Final Human Health Toxicity Assessment for PFOA and PFOS (U.S. EPA, 2024e; U.S. EPA, 2024f), which included studies published from 2016 through September 2020. The EPA included studies in the meta-analysis if they reported quantitative estimates (e.g., regression coefficients) and measures of uncertainty (e.g., standard errors, confidence intervals) of associations between serum PFOA or PFOS and TC or HDLC in general population adults aged 20 years and older. The EPA included a total of 14 studies in the meta-analysis. Of these, 12 studies were used to develop exposure-response relationships for serum PFOA or PFOS and TC (i.e., not all relevant studies report the effects for both PFOA and PFOS). The unit in the meta-analysis was the change in TC (or HDLC) in mg/dL per increases in serum PFOA or PFOS.

Table 6-16 summarizes the 14 studies that the EPA identified from literature reviews and used to derive slope estimates for PFOA and PFOS associations with serum TC levels.[57] Six of the studies that the EPA retained for use in the meta-analysis were based on serum PFAS and serum TC measurements from the U.S. general population (National Health and Nutrition Examination Survey [NHANES]) (Dong et al., 2019; Fan et al., 2020; He et al., 2018; Jain & Ducatman, 2019a; H.-S. Liu et al., 2018; Nelson et al., 2010); there were also general population studies from Canada (Fisher et al., 2013), Sweden (Y. Li et al., 2020), Taiwan (Yang et al., 2018; C. Y. Lin et al., 2020), and Henan Province, China (Fu et al., 2014). Château-Degat et al. (2010) reported on the association between PFOS and TC in a Canadian Inuit population. The EPA also retained the results from a study of a highly exposed population in the U.S. (the C8 cohort) (Steenland et al., 2009) and from a study using participants in a U.S. diabetes prevention program (Lin et al., 2019). The EPA retained results from Steenland et al. (2009) because serum levels in the examined cohort were only modestly elevated compared to less exposed populations (e.g., the median serum PFOA concentration in this cohort was 27 ng/mL, with an interquartile

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[57] For this effort, the EPA focused on PFOA and PFOS, since these are by far the most well-studied perfluorinated compounds.

range of 13.1 to 67 ng/mL). The EPA retained results from Lin et al. (2019) because the examined cohort included pre-diabetic adults enrolled in a diabetes prevention program; thus, this cohort was representative of a large portion of the U.S. adult population.

Table 6-16: Studies Selected for Inclusion in the Meta-Analyses

Author and Year	Title	TC and Serum PFAS Relationship Evaluated in Study	
		PFOA	PFOS
Steenland et al., 2009[a,d]	Association of Perfluorooctanoic Acid and Perfluorooctane Sulfonate With Serum Lipids Among Adults Living Near a Chemical Plant	X	X
Château-Degat et al., 2010[a,d]	Effects of Perfluorooctanesulfonate Exposure on Plasma Lipid Levels in the Inuit Population of Nunavik (Northern Quebec)		X
Nelson et al., 2010[a,d]	Exposure to Polyfluoroalkyl Chemicals and Cholesterol, Body Weight, and Insulin Resistance in the General U.S. Population	X	X
Fisher et al., 2013 [a,d]	Do Perfluoroalkyl Substances Affect Metabolic Function and Plasma Lipids? —Analysis of the 2007–2009, Canadian Health Measures Survey (CHMS) Cycle 1	X	X
Fu et al., 2014[a,d]	Associations Between Serum Concentrations of Perfluoroalkyl Acids and Serum Lipid Levels in a Chinese Population	X	X
He et al., 2018[c]	PFOA is Associated with Diabetes and Metabolic Alteration in US Men: National Health and Nutrition Examination Survey 2003-2012	X	X
Liu et al., 2018[c]	Association Among Total Serum Isomers of Perfluorinated Chemicals, Glucose Homeostasis, Lipid Profiles, Serum Protein and Metabolic Syndrome in Adults: NHANES, 2013–2014	X	X
Dong et al., 2019[b]	Using 2003–2014 U.S. NHANES Data to Determine the Associations Between Per- and Polyfluoroalkyl Substances and Cholesterol: Trend and Implications	X	X
Jain et al., 2019[b]	Roles of Gender and Obesity in Defining Correlations Between Perfluoroalkyl Substances and Lipid/Lipoproteins	X	X
P.-I. D. Lin et al., 2019[b]	Per- and Polyfluoroalkyl Substances and Blood Lipid Levels in Pre-Diabetic Adults—Longitudinal Analysis of the Diabetes Prevention Program Outcomes Study	X	X
Fan et al., 2020[b]	Serum Albumin Mediates the Effect of Multiple Per- and Polyfluoroalkyl Substances on Serum Lipid Levels	X	X
Y. Li et al., 2020[b]	Associations Between Perfluoroalkyl Substances and Serum Lipids in a Swedish Adult Population With Contaminated Drinking Water	X	X

Abbreviations: TC – total cholesterol; PFOS – perfluorooctane sulfonic acid; PFOA – perfluorooctanoic acid; PFAS – per-and polyfluoroalkyl substances.

Notes:

[a] Studies identified based on ATSDR literature review.

[b] Studies identified based on the EPA's literature review.

[c] Studies available in both assessments.

[d] Studies available in PFOA and/or PFOS health effects support documents (U.S. EPA, 2016e; U.S. EPA, 2016f).

The EPA developed exposure-response relationships between serum PFOA/PFOS and TC for use in the CVD analysis using the meta-analyses restricted to studies of adults in the general population reporting similar models. The EPA used untransformed serum PFOA/PFOS to reduce bias due to back-transformations of effect estimates. For studies that provided results only for log-transformed serum PFOA/PFOS (five studies) or log-transformed outcomes (two studies), or both log-transformed serum PFOA/PFOS and outcomes (two studies), the EPA approximated the results for an untransformed analysis using the approach outlined by Rodríguez-Barranco et al. (2017) and Dzierlenga et al. (2020). When using studies reporting linear associations between TC and serum PFOA or PFOS, the EPA estimated a positive increase in TC of 1.57 (95% CI: 0.02, 3.13) mg/dL per ng/mL serum PFOA (p-value = 0.048), and of 0.08 (95% CI: -0.01, 0.16) mg/dL per ng/mL serum PFOS (p-value = 0.064). The EPA selected the pooled slope estimate based on the studies using linear models to ease interpretability and to reduce bias due to back-transformations of effect estimates with log-transformed outcomes or exposures (see Appendix F for details). While the association for PFOS and TC is not significant at the 0.05 confidence level, it is significant at the 0.10 confidence level (p-value = 0.064). Furthermore, the literature provides sufficient support of a positive association (e.g., Château-Degat et al., 2010; Dong et al., 2019; U.S. EPA, 2024e; U.S. EPA, 2024f). The studies are large with more than 700 and 8,900 participants, respectively (Château-Degat et al., 2010; Dong et al., 2019) and have low risk of bias. In addition, the estimated values are supported by sensitivity analyses and by the estimates from potential candidate studies from exposure-response modeling for ongoing agency efforts (Dong et al., 2019). Based on the systematic literature review of epidemiologic studies published through February 2023 for developing the EPA's Final Human Health Toxicity Assessments for PFOA and PFOS, the available evidence supports a positive association between PFOS and TC in the general population (U.S. EPA, 2024e; U.S. EPA, 2024f). For more information on the systematic review and results, see the EPA's Final Human Health Toxicity Assessments for PFOA and PFOS (U.S. EPA, 2024e; U.S. EPA, 2024f).

Note that the EPA sought comments from the EPA SAB on the cardiovascular disease exposure-response approach (U.S. EPA, 2022i). The SAB recommended that the EPA evaluate how the inclusion of HDLC effects would influence results. The EPA evaluated the inclusion of HDLC effects in a sensitivity analysis, described in Appendix K.

6.5.2.2 Estimation of BP Changes

PFOS exposure has been linked to other cardiovascular outcomes, such as systolic BP and hypertension (Liao et al., 2020; U.S. EPA, 2024e). Because systolic BP is another predictor used by the ASCVD model, the EPA included the estimated changes in BP from reduced exposure to PFOS in the CVD analysis. The EPA selected the slope from the Liao et al. (2020) study — a high confidence study conducted based on U.S. general population data from NHANES cycles 2003-2012. Liao et al. (2020) estimated an increase of 1.35 (95% CI: 0.18, 2.53) in mmHg systolic BP per log10(ng/mL) PFOS among those not using antihypertensive medications. For the purposes of this analysis, the EPA converted this slope to 0.044 (95% CI: 0.006, 0.083) mmHg per ng/mL. The evidence on the associations between PFOA and BP is not as consistent as for PFOS (see Section 6.2.2.1.2). Therefore, the EPA is not including effect estimates for the serum PFOA-BP associations in the CVD analysis.

6.5.3 Estimation of Cardiovascular Disease Risk Reductions

The EPA relies on the life table-based approach to estimate CVD risk reductions because (1) changes in serum PFOA/PFOS in response to changes in drinking water PFOA/PFOS occur over multiple years, (2) CVD risk, relying on the ASCVD model, can be modeled only for those older than 40 years without prior CVD history, and (3) individuals who have experienced non-fatal CVD events have elevated mortality implications immediately and within at least five years of the first occurrence.[58] Recurrent life table calculations are used to estimate a PWS EP-specific annual time series of CVD event incidence for a population cohort characterized by sex, race/ethnicity, birth year, age at the start of the PFOA/PFOS evaluation period (i.e., 2024), and age- and sex-specific time series of changes in TC and BP levels obtained by combining serum PFOA/PFOS concentration time series (Section 6.3) with exposure-response information (Section 6.5.3). Baseline and regulatory alternatives are evaluated separately, with regulatory alternative TC and BP levels estimated using baseline information on these biomarkers from external statistical data sources and modeled changes in TC and BP due to conditions under the regulatory alternatives (see Appendix G for detailed information on data sources used in CVD modeling).

The EPA estimated the incidence of first hard CVD events based on TC serum and BP levels using the ASCVD model (Goff et al., 2014), which predicts the 10-year probability of a hard CVD event to be experienced by a person without a prior CVD history (see Section 6.5.3.2).[59] The EPA adjusted the modeled population cohort to exclude individuals with pre-existing conditions, as the ASCVD risk model does not apply to these individuals. For BP effects estimation, the EPA further restricts the modeled population to those not using antihypertensive medications for consistency with the exposure-response relationship (see Section 6.5.3.2 for detail). Modeled first hard CVD events include fatal and non-fatal MI, fatal and non-fatal IS, and other CHD mortality. The EPA has also estimated the incidence of post-acute CVD mortality among survivors of the first MI or IS within 6 years of the initial event (Section 6.5.3.3).

The estimated CVD risk reduction resulting from reducing serum PFOA and serum PFOS concentrations is the difference in annual incidence of CVD events (i.e., mortality and morbidity associated with first-time CVD events and post-acute CVD mortality) under the baseline and regulatory alternatives. Appendix G provides detailed information on all CVD model components, computations, and sources of data used in modeling.

6.5.3.1 Life Table Calculations

The CVD model integrates the ASCVD model predictions and post-acute CVD mortality rates in the series of recurrent calculations that produce a life table estimate for the affected population cohort (e.g., non-Hispanic White females aged 70 years at the beginning of the evaluation period). For each PWS EP, the EPA evaluates population cohorts defined by a combination of birth year, age, sex (males and females), and race/ethnicity (non-Hispanic White, non-Hispanic

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[58] The EPA notes that elevated mortality for hard CVD event survivors may persist beyond five years of the initial event. However, the EPA did not identify U.S. based studies with sufficiently long follow-up to quantify mortality impacts beyond five years of the initial event.

[59] The EPA did not identify studies that found statistically significant associations between the modeled biomarkers (TC, BP) and CVD events in populations with prior CVD history. Discussion of the relevant literature is provided in Appendix G.

Black, Hispanic, Other). In addition to the key standard life table components (i.e., the number of persons surviving to a specific age and the number of all-cause deaths occurring at a given age) for ages 40 years or older, the CVD model estimates the number of surviving persons with and without a history of hard CVD events, the number of persons experiencing hard CVD events at a given age, and the deaths from CVD and non-CVD causes at a given age.

Figure 6-7 summarizes the CVD model calculations for a population cohort age 0 at the start of the evaluation period.[60] The CVD model calculations are identical across race/ethnicity and sex demographic subgroups but use subgroup-specific parameters.[61] For cohorts born prior to or in 2024, the CVD model is initialized using the PWS-specific number of persons estimated to be alive at the beginning of 2024. For cohorts born after 2024 (i.e., 2025–2105), the CVD model is initialized using the PWS EP-, race/ethnicity-, sex, and scenario-specific number of persons who died in the previous calendar year of the analysis, thereby ensuring that the size of the modeled population remains constant throughout the analysis period. Additional PWS EP- and sex, race/ethnicity, and age-specific population estimation assumptions are provided in Section 2.2; additional details are included in Appendix B.

Once the model is initialized, the following types of calculations occur for each year within the simulation period:[62]

- Recurrent standard life table calculations that rely on the all-cause, age-specific annual mortality rates to evaluate the number of deaths among persons of a specific integer age and the number of survivors to the beginning of the next integer age.[63] These calculations are executed whenever the current cohort age is in the 0–39 range. They are represented by the navy-blue segment of the timeline shown in Figure 6-7.
- Recurrent life table calculations that separately track subpopulations with and without a history of hard CVD events, including estimation of the number of annual CVD and non-CVD deaths (in either subpopulation), as well as the number of annual post-acute CVD deaths experienced by survivors of the first hard CVD events that occurred, at most, 5 years ago. These calculations are executed whenever the current cohort age is 40 years or older.[64] These calculations are represented by the blue segment of the timeline. Figure 6-7 and Figure 6-8 further illustrates the year-specific calculations required for explicit tracking of subpopulations with and without a hard CVD event history.

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[60] This initial population cohort age is chosen because it allows for illustration of the full set of calculation types used in the CVD model.

[61] There are different ASCVD model coefficients for non-Hispanic White and non-Hispanic Black males and females. The figure shows the generalized approach of the CVD model.

[62] The EPA notes that the simulation period is the lifespan of individuals relevant to the analysis. The simulation period is distinct from the period of analysis in that some parts of the simulation period may fall outside the period of analysis. For example, for a person aged 40 years at the start of the analysis period, the period of analysis will not capture the first 40 years of simulation results.

[63] Life table calculations are based on the present-day information about life expectancy, disease, environmental exposure, and other factors.

[64] People 85 years or older, are treated as a single cohort in the model. The mortality rate for this cohort is assumed to be the average mortality rate for those age 85-100 years. This cohort also used serum PFOA/PFOS values at age 85.

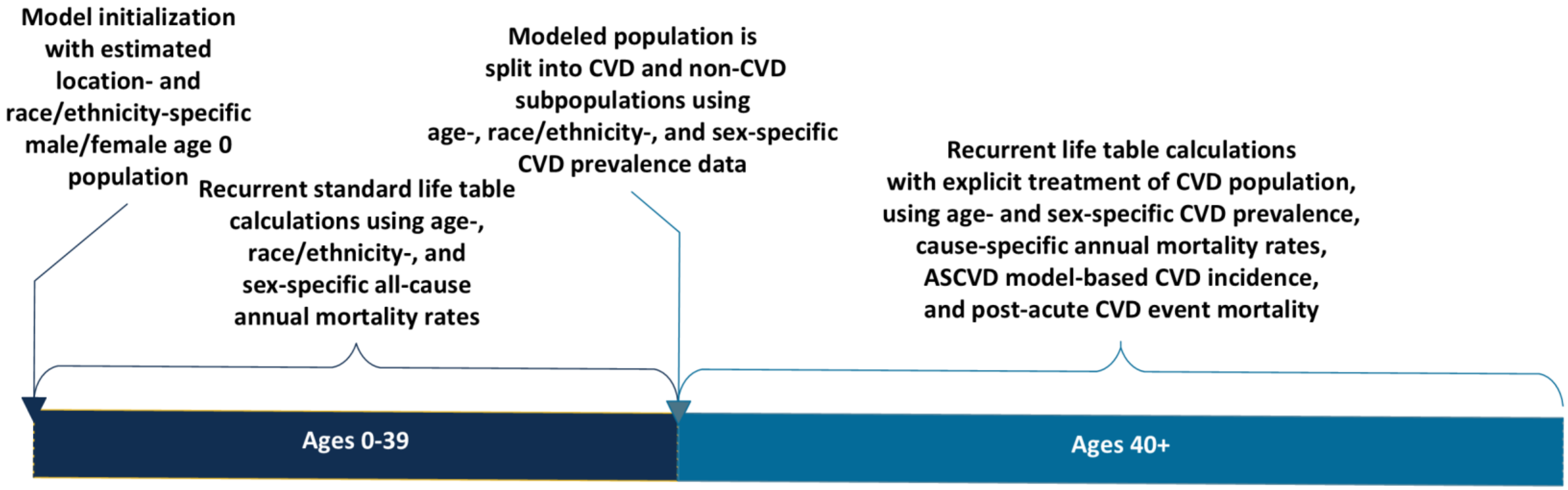

Figure 6-7: Overview of Life Table Calculations in the CVD Model

Note: The figure illustrates the model for population cohort age 0 years at the beginning of the evaluation period (i.e., calendar year 2024). The model is initialized using the age 0 PWS EP-specific population (see Appendix B for PWS population estimation details).

Figure 6-8 provides additional information on the post-acute CVD mortality estimation. Each person included in the surviving current age-specific incident CVD subpopulation[65] (corresponding to the group F result in Figure 6-8) is tracked for 5 additional years to estimate the number of CVD deaths occurring in that timeframe. The recurrent estimates rely on age-specific non-CVD mortality rates, estimated based on the CDC's life table data and annual CVD mortality rates, and on post-acute CVD mortality rates, estimated based on Thom et al. (2001) and S. Li et al. (2019).

Further details of the life table calculations are provided in Appendix G. The outputs of the life table calculations and application of the ASCVD model are the PWS EP-specific estimates of the annual number of persons experiencing their first non-fatal MI or IS event and the number of deaths among those who have experienced their first hard CVD event, at most, 6 years ago. Note that the ASCVD model does not predict risks separately by type of first hard CVD event (i.e., non-fatal MI, non-fatal IS, and fatal CVD). The distribution of these events by type is estimated using data publicly available on CVD prevalence, incidence, and hospital mortality statistics as described in Section 6.5.3.2 and integrated into the overall CVD impacts modeling.

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[65] For example, persons who experienced their first non-fatal MI or IS at age 70 and survived through the first post-event year.

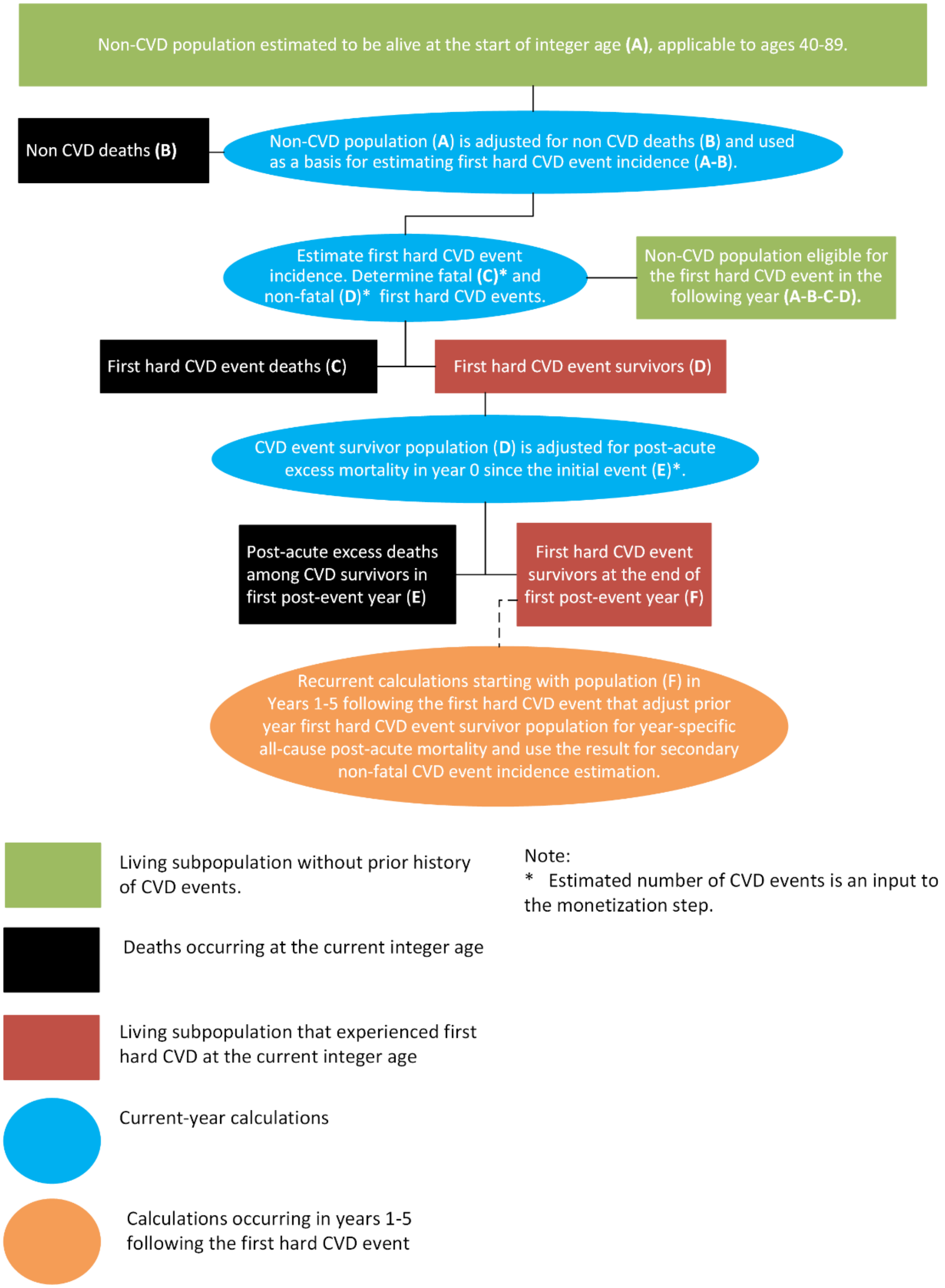

Figure 6-8: CVD Model Calculations for Ages 40+ Tracking CVD

6.5.3.2 Risk and Distribution of First Hard Cardiovascular Disease Event

The first hard CVD event incidence estimates are generated by the Pooled Cohort ASCVD model (Goff et al., 2014). The ASCVD model is commonly used in clinical practice to estimate CVD risk for those between ages 40 and 80, as well as for overall population risk management (Lloyd-Jones et al., 2017). The ASCVD model predicts the 10-year probability of a hard CVD event—fatal and non-fatal MI, fatal and non-fatal IS, or CHD death—to be experienced by a person without a prior history of MI, IS, congestive heart failure, percutaneous coronary intervention, coronary bypass surgery, or atrial fibrillation. The ASCVD model is a survival model that links predictor levels at the start of the 10-year follow-up period to the first hard CVD event incidence during the follow-up period; the modeling does not account for changes in CVD risk predictors over time.

Four large longitudinal community-based epidemiologic cohort studies were combined to develop a geographically and racially diverse dataset used for the ASCVD model estimation.[66] The predictors of the ASCVD model include age, TC and HDLC concentrations, systolic BP, current smoking, diagnosed diabetes, and whether the participant is undergoing treatment for high BP. The model was fit separately to four population subgroups: non-Hispanic White females, non-Hispanic Black females, non-Hispanic White males, and non-Hispanic Black males.

Several studies assessed predictive performance of the ASCVD risk model in racial and ethnic groups other than other non-Hispanic White and non-Hispanic Black populations, as well as in various sociodemographic subgroups in the U.S. Two studies concluded that the ASCVD risk model overestimated CVD risk among Asian and Hispanic groups, while noting that these groups were not included in the development and validation of the ASCVD model (Mongraw-Chaffin et al., 2018; Rodriguez et al., 2019). Five studies acknowledged limitations for the ASCVD risk model in terms of performance among individuals with high levels of CVD risk, diabetes, older adults with frailty and multimorbidity, smokers, and women (Muntner et al., 2014; Leigh et al., 2019; Mora et al., 2018; Q. D. Nguyen et al., 2020; Raghavan et al., 2020). Overall, the literature across different sociodemographic subgroups concluded that the ASCVD risk model tended to overestimate risk but suggested the model may improve through additional input variables and recalibration given contemporary ASCVD prevalence, especially if the prevalence differs significantly across geographic locations to which the model is applied (Mora et al., 2018; (Muntner et al., 2014). Extended discussion of ASCVD risk model performance and availability of alternative CVD risk prediction models for national analysis is provided in ICF (2022a).

In light of these findings, the EPA does not follow the Goff et al. (2014) recommendation that the ASVCD risk model for non-Hispanic White populations be used for other race/ethnicity groups. In the development and parameterization of the CVD model for Hispanic, Asian American, and American Indian/Alaska Native populations, the EPA applies the model for non-Hispanic Black populations based on the ASCVD model validation relative to reported CVD

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[66] These studies include the Atherosclerosis Risk in Communities (ARIC) study (Williams, 1989) and the Cardiovascular Health Study (Fried et al., 1991), along with applicable data from the Coronary Artery Risk Development in Young Adults (CARDIA) study (Friedman et al., 1988) and the Framingham Original and Offspring cohort data (D'Agostino et al., 2008).

prevalence and mortality statistics (the EPA analysis based on Medical Expenditure Panel Surveys from 2010–2017), as described in Appendix G. The results of this validation exercise showed that the ASCVD model coefficients for the non-Hispanic Black model are more consistent with data on CVD prevalence and mortality for Hispanic and non-Hispanic other race subpopulations than the ASCVD model coefficients for the non-Hispanic White model. The all-cause and CVD mortality was obtained from CDC's National Vital Statistics System, whereas CVD prevalence was estimated using agency for Healthcare Research and Quality survey data (see Appendix G for details). As explained in Appendix G, race/ethnicity and sex-specific CVD incidence consistent with these reported statistics was compared with the incidence estimated using the ASCVD model, where the baseline race/ethnicity- and sex-specific values for the ASCVD model predictors were obtained from CDC's public health surveys (see Appendix G for details).

The ASCVD model generates predictions of the 10-year probability of the first hard CVD event without differentiation across CVD event types. The specifics of annual first hard CVD event probability derivation, which is needed for the life table calculations in Section 6.5.3.1, are provided in Appendix G. As is also detailed in Appendix G, the EPA combined the Medical Expenditure Panel Survey (MEPS) 2010–2017 data and the Healthcare Cost and Utilization Project (HCUP) 2017 data to derive the ASCVD event distribution over the following event types: non-fatal MI, non-fatal IS, and fatal CVD events. The fatal CVD events include fatal MI, fatal IS, and other fatal CHD events. The EPA used the MEPS data to identify the subpopulation of persons without a prior CVD event history and estimate the rate of new CVD events by type (i.e., MI, IS, and other CHD) in this subpopulation. The probabilities of in-hospital death for MI, IS, and other CHD were obtained from HCUP.

Table 6-17 shows the derived race/ethnicity-, sex-, and age group-specific shares of first hard CVD events for the following event types: non-fatal MI, fatal MI, non-fatal IS, fatal IS, other non-fatal CHD, and other fatal CHD. For males, looking across race/ethnicity and age categories, the share of non-fatal MI events is 4.9 percent to 28 percent, the share of non-fatal IS events is 9.4 percent to 38 percent, and the share of other non-fatal CHD events is 44 percent to 78 percent. For females, across race/ethnicity and age categories, the share of non-fatal MI events is 6.4 percent to 19 percent, the share of non-fatal IS events is 8.7 percent to 29 percent, and the share of other non-fatal CHD events is 51 percent to 76 percent. For both sexes, shares of all fatal events increase with age. The share of fatal CVD events is largest for Hispanic and non-Hispanic other race subpopulations of both sexes. Table 6-17 also shows derived race/ethnicity-, sex-, and age group-specific shares of first hard CVD events over ASCVD event types (i.e., non-fatal MI, non-fatal IS, and fatal CVD). Note that these shares were re-normalized to sum to 100 percent after exclusion of other non-fatal CHD not predicted by the ASCVD model. The CVD model relies on the re-normalized shares to allocate the total number of first hard CVD events predicted by the ASCVD model.

Table 6-17: Estimated Shares of Fatal and Non-Fatal First Hard CVD Events Based on MEPS and HCUP Data

Sex	Age (in years)	Race/ Ethnicity	Non-Fatal CVD (%)			Fatal CVD (%)		
			Non-Fatal MI (%)	Non-Fatal IS (%)	Other Non-Fatal CHD (%)	Fatal MI (%)	Fatal IS (%)	Other Fatal CHD (%)
Shares of First Hard CVD Events								
Males	18–44	NH White	14	9.4	77	0.19	0.17	0
	45–64	NH White	16	15	69	0.39	0.34	0.44
	65–84	NH White	13	20	64	0.71	0.75	0.76
	85 or older	NH White	13	20	63	1.3	1.4	1.9
	18–44	NH Black	4.9	17	78	0.067	0.31	0
	45–64	NH Black	11	38	50	0.28	0.88	0.32
	65–84	NH Black	8.9	22	67	0.48	0.8	0.79
	85 or older	NH Black	8.5	21	66	0.87	1.5	2
	18–44	Hispanic	23	17	59	0.31	0.31	0
	45–64	Hispanic	19	29	51	0.48	0.67	0.32
	65–84	Hispanic	20	17	60	1.1	0.65	0.71
	85 or older	Hispanic	19	17	59	2	1.2	1.8
	18–44	NH Other	26	30	44	0.35	0.54	0
	45–64	NH Other	28	19	52	0.71	0.43	0.33
	65–84	NH Other	13	25	60	0.71	0.92	0.71
	85 or older	NH Other	12	24	59	1.3	1.7	1.8
Females	18–44	NH White	8.1	19	72	0.13	0.41	0
	45–64	NH White	6.9	20	72	0.2	0.55	0.54
	65–84	NH White	11	28	58	0.68	1.2	0.82
	85 or older	NH White	10	27	57	1.2	2.3	2.1
	18–44	NH Black	15	8.7	76	0.23	0.18	0
	45–64	NH Black	10	27	61	0.29	0.74	0.46
	65–84	NH Black	6.7	29	62	0.42	1.2	0.87
	85 or older	NH Black	6.4	28	61	0.76	2.3	2.2
	18–44	Hispanic	8.8	18	73	0.14	0.38	0
	45–64	Hispanic	13	27	59	0.37	0.73	0.45
	65–84	Hispanic	19	26	52	1.2	1.1	0.73
	85 or older	Hispanic	18	25	51	2.1	2.1	1.9
	18–44	NH Other	11	13	75	0.17	0.27	0
	45–64	NH Other	14	29	55	0.42	0.78	0.42
	65–84	NH Other	12	28	58	0.74	1.2	0.81
	85 or older	NH Other	11	27	56	1.3	2.3	2.1
Shares of First Hard CVD Event Categories Predicted by the ASCVD Model[a]								
Males	18–44	NH White	58	40	–	1.5		
	45–64	NH White	50	47	–	3.7		
	65–84	NH White	37	57	–	6.2		
	85 or older	NH White	34	53	–	13		
	18–44	NH Black	22	77	–	1.7		
	45–64	NH Black	22	75	–	2.9		
	65–84	NH Black	27	66	–	6.4		
	85 or older	NH Black	25	62	–	13		
	18–44	Hispanic	56	42	–	1.5		
	45–64	Hispanic	38	59	–	3.0		

Table 6-17: Estimated Shares of Fatal and Non-Fatal First Hard CVD Events Based on MEPS and HCUP Data

Sex	Age (in years)	Race/ Ethnicity	Non-Fatal CVD (%)			Fatal CVD (%)		
			Non-Fatal MI (%)	Non-Fatal IS (%)	Other Non-Fatal CHD (%)	Fatal MI (%)	Fatal IS (%)	Other Fatal CHD (%)
	65–84	Hispanic	50	44	–		6.1	
	85 or older	Hispanic	47	41	–		12	
	18–44	NH Other	46	53	–		1.6	
	45–64	NH Other	58	39	–		3.1	
	65–84	NH Other	33	62	–		5.8	
	85 or older	NH Other	30	58	–		12	
Females	18–44	NH White	29	69	–		1.9	
	45–64	NH White	24	71	–		4.6	
	65–84	NH White	26	67	–		6.5	
	85 or older	NH White	24	63	–		13	
	18–44	NH Black	62	36	–		1.7	
	45–64	NH Black	26	70	–		3.9	
	65–84	NH Black	18	76	–		6.7	
	85 or older	NH Black	16	70	–		14	
	18–44	Hispanic	32	66	–		1.9	
	45–64	Hispanic	31	65	–		3.8	
	65–84	Hispanic	40	54	–		6.4	
	85 or older	Hispanic	37	51	–		12	
	18–44	NH Other	45	53	–		1.8	
	45–64	NH Other	32	64	–		3.6	
	65–84	NH Other	28	66	–		6.5	
	85 or older	NH Other	26	61	–		13	

Abbreviations: CVD – cardiovascular disease; CHD – coronary heart disease; fatal CVD – includes fatal MI, fatal IS, and fatal other coronary heart disease events; HCUP – Healthcare Cost and Utilization Project; IS – ischemic stroke; MEPS – Medical Expenditure Panel Survey; MI – myocardial infarction; NH – non-Hispanic.
Note:
[a]The distribution is derived by (1) excluding the other non-fatal CHD category; (2) aggregating fatal MI, fatal IS, and other fatal CHD categories into the fatal CVD category; and (3) re-normalizing the data to sum to 100%.

6.5.3.3 Risk of Post-Acute Cardiovascular Disease Mortality

Persons who have experienced non-fatal MI and non-fatal IS have an elevated risk of post-acute CVD mortality and morbidity (Roger et al., 2012). Studies focusing on secondary hard CVD events point to an elevated risk of these events among survivors of the first hard CVD event (e.g., Beatty et al., 2015; S. Li et al., 2019; Thom et al., 2001), but do not support the link between these risks and TC/BP levels (Beatty et al., 2015). (See Appendix G for details.) Therefore, the CVD model evaluates post-acute CVD mortality among survivors of the initial MI/IS event under baseline and regulatory alternatives using the baseline post-acute mortality rates that do not depend on the levels of modeled biomarkers. The CVD model does not explicitly evaluate secondary CVD morbidity because available first non-fatal MI/IS valuation measures (e.g., O'Sullivan et al., 2011) incorporate incidence of these secondary events.

For survivors of the first hard CVD event at ages 40–65, the EPA uses estimates of sex- and race/ethnicity-specific all-cause post-acute mortality for MI survivors at 1- and 5-year follow-up from Thom et al. (2001). Because Thom et al. (2001) reports all-cause post-acute mortality rates, the EPA adjusted these rates to exclude deaths from non-CVD causes. To this end, the EPA used general population integer age- and sex-specific all-cause mortality from U.S. Life Tables, 2017 (Arias & Xu, 2019), U.S. CVD mortality rates (CDC, 2020b), and U.S. Life Tables Eliminating Certain Causes of Death, 1999–2000 (Arias et al., 2013). Appendix G provides additional estimation details. Although the EPA was unable to identify comparable post-acute mortality statistics for non-fatal IS, an analysis of the Medicare population by S. Li et al. (2019) suggests that post-acute MI mortality is a reasonable approximation for post-acute IS mortality.[67] Table 6-18 shows estimated post-acute CVD mortality rates for survivors of the first MI or IS at ages 40–65 that are used to parameterize the CVD model.

For survivors of the first hard CVD event at ages 66 or older, the EPA uses the results from S. Li et al. (2019) to estimate the number of post-acute CVD deaths within 6 years of the initial event. Because S. Li et al. (2019) reports only all-cause post-acute mortality rates, the EPA adjusted these rates to exclude deaths from non-CVD causes. Integer age- and sex-specific probability of death from non-CVD causes was derived from U.S. Life Tables, 2017 (Arias & Xu, 2019), U.S. CVD mortality rates (CDC, 2020b), and U.S. Life Tables Eliminating Certain Causes of Death, 1999–2000 (Arias et al., 2013). The sex-specific probabilities of death from non-CVD causes were average using the demographic information for the cohorts analyzed by S. Li et al. (2019). See Appendix G for additional estimation details. Table 6-18 shows estimated post-acute CVD mortality rates for survivors of the first MI and survivors of the first IS at ages 66 years or older that are used to parameterize the CVD model.[68]

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[67] For those age 65 or older, S. Li et al. (2019) have estimated the probability of death within 1 year after non-fatal IS to be 32.07 percent and the probability of death within 1 year after non-fatal MI to be 32.09 percent.

[68] These rates are applied to all those aged 66 or older in the SafeWater MCBC implementation of the model.

Table 6-18: Estimated Risk of Post-Acute CVD Mortality Following the First Non-Fatal Hard CVD Event

Type of First Non-Fatal Hard CVD Event	Demographic Group	Post-Acute CVD Mortality Rate per 100,000 by Integer Year Since the First Non-Fatal Hard CVD Event					
		0	1	2	3	4	5
Source: Thom et al. (2001)							
MI, IS[a]	Non-Hispanic White[b] males aged 45–65 years	4,500	910	860	820	760	–
	Non-Hispanic Black males aged 45–65 years	12,000	1,200	1,100	1,100	1,000	–
	Non-Hispanic White[b] females aged 45–65 years	8,600	1,900	1,900	1,900	1,800	–
	Non-Hispanic Black females aged 45–65 years	7,700	4,300	4,200	4,100	4,100	–
Source: S. Li et al. (2019)							
MI	Persons aged 66 years or older	27,000	11,000	9,600	9,040	8,600	8,040
IS	Persons aged 66 years or older	28,000	9,900	10,000	9,800	8,900	8,030

Abbreviations: CVD – cardiovascular disease; IS – ischemic stroke (International Classification of Disease Ninth Revision [ICD9] = 433, 434; International Classification of Disease Tenth Revision [ICD10] = I63), MI – myocardial infarction (ICD9 = 410; ICD10 = 121).

Notes:

[a] Thom et al. (2001) reported data for the first MI survivors only for aged 45–64 years. The CVD model applies these rates to both the first MI and first IS survivors.

[b] Estimates for non-Hispanic White populations are applied to other race/ethnicity-specific populations.

6.5.4 Valuation of Cardiovascular Disease Risk Reductions

The EPA uses the Value of Statistical Life to estimate the benefits of reducing mortality associated with hard CVD events in the population exposed to PFOA and PFOS in drinking water. Value of Statistical Life updating information is provided in Section 2.2. The EPA relies on COI-based valuation that represents the medical costs of treating or mitigating non-fatal first hard CVD events (MI, IS) during the three years following an event among those without prior CVD history, adjusted for post-acute mortality.

The annual medical expenditure estimates for MI and IS are based on O'Sullivan et al. (2011). The estimated expenditures do not include long-term institutional and home health care. For non-fatal MI, O'Sullivan et al. (2011) estimated medical expenditures are $53,246 ($2022)[69] for the initial event and then $33,162, $14,635, $13,078 annually within 1, 2, and 3 years after the initial event, respectively. For non-fatal IS, O'Sullivan et al. (2011) estimated medical expenditures are $16,503 ($2022) for the initial event and then $11,988, $788, $1,868 annually within 1, 2, and 3 years after the initial event, respectively. Annual estimates within 1, 2, and 3 years after the initial event include the incidence of secondary CVD events among survivors of first MI and IS events.

To estimate the present discounted value of medical expenditures within 3 years of the initial non-fatal MI, the EPA combined O'Sullivan et al. (2011) MI-specific estimates with post-acute

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[69] Original values from the source were inflated to $2022 using the medical care Consumer Price Index (U.S. Bureau of Labor Statistics, 2021).

survival probabilities based on Thom et al. (2001) (for MI survivors aged 40-64) and S. Li et al. (2019) (for MI survivors aged 65 or older). To estimate the present discounted value of medical expenditures within 3 years of the initial non-fatal IS, the EPA combined O'Sullivan et al. (2011) IS-specific estimates with post-acute survival probabilities based on Thom et al. (2001) (for IS survivors aged 40-64, assuming post-acute MI survival probabilities reasonably approximate post-acute IS survival probabilities) and S. Li et al. (2019) (for IS survivors aged 65 or older). The EPA did not identify post-acute IS mortality information in this age group, but instead applied post-acute MI mortality estimates for IS valuation.[70] Table 6-19 presents the resulting MI and IS unit values.

Table 6-19: Cost of Illness of Non-Fatal First CVD Event Used in Modeling

Type of First Non-fatal Hard CVD Event	Age Group	Present Discounted Value of 3-Year Medical Expenditures ($2022, 2% Discount Rate)[a,b] Adjusted for Post-Acute Mortality[c]
MI	40-64 years	$110,040
	65 years or older	$96,626
IS	40-64 years	$30,373
	65 years or older	$27,954

Abbreviations: CVD – cardiovascular disease; MI – myocardial infarction (ICD9 = 410; ICD10 = 121, IS – ischemic stroke (ICD9 = 433, 434; ICD10 = I63).

Notes:

[a] Estimates of annual medical expenditures are from O'Sullivan et al. (2011).

[b] Original values from O'Sullivan et al. (2011) were inflated to $2022 using the medical care Consumer Price Index (U.S. Bureau of Labor Statistics, 2022a).

[c] Post-acute MI mortality data for those aged 40-64 years is from Thom et al. (2001); probabilities to survive 1 year, 2 years, and 3 years after the initial event are 0.93, 0.92, and 0.90, respectively. The EPA applies these mortality values to derive the IS value in this age group. Post-acute MI mortality data and post-acute IS mortality data for persons aged 65 years and older are from S. Li et al. (2019). For MI, probabilities to survive 1 year, 2 years, and 3 years after the initial event are 0.68, 0.57, and 0.49, respectively. For IS, probabilities to survive 1 year, 2 years, and 3 years after the initial event are 0.67, 0.57, and 0.48, respectively.

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[70] Post-acute mortality estimates for IS and MI were very close in the Medicare population (S. Li et al., 2019). For those ages 65 years or older, S. Li et al. (2019) have estimated probability of death within 1 year after non-fatal IS to be 32.07 percent and probability of death within 1 year after non-fatal MI to be 32.09 percent. Therefore, reliance on the post-acute mortality for MI to approximate the same for stroke is reasonable.

6.5.5 Results

Table 6-20 to Table 6-23 provide the health effects avoided and valuation associated with cardiovascular disease.

Table 6-20: National CVD Benefits, Final Rule (PFOA and PFOS MCLs of 4.0 ppt each, PFHxS, PFNA, and HFPO-DA MCLs of 10 ppt each and HI of 1) (Million $2022)

Benefits Category	2% Discount Rate		
	5th Percentile[a]	Expected Value	95th Percentile[a]
Number of Non-Fatal MI Cases Avoided	1,407.7	6,333.1	11,189.0
Number of Non-Fatal IS Cases Avoided	2,074.8	9,247.6	16,279.0
Number of CVD Deaths Avoided	845.5	3,715.8	6,555.6
Total Annualized CVD Benefits (Million $2022)[b]	**$140.66**	**$606.09**	**$1,069.40**

Abbreviations: CVD – cardiovascular disease, MI – myocardial infarction, IS – Ischemic Stroke.

Notes: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates. Quantifiable benefits are increased under final rule table results relative to the other options presented because of modeled PFHxS occurrence, which results in additional quantified benefits from co-removed PFOA and PFOS.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.

[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

Table 6-21: National CVD Benefits, Option 1a (PFOA and PFOS MCLs of 4.0 ppt)

Benefits Category	2% Discount Rate		
	5th Percentile[a]	Expected Value	95th Percentile[a]
Number of Non-Fatal MI Cases Avoided	1,400.8	6,296.0	11,115.0
Number of Non-Fatal IS Cases Avoided	2,065.0	9,194.8	16,203.0
Number of CVD Deaths Avoided	839.9	3,695.1	6,484.4
Total Annualized CVD Benefits (Million $2022)[b]	**$140.12**	**$602.72**	**$1,059.60**

Abbreviations: CVD – cardiovascular disease, MI – myocardial infarction, IS – Ischemic Stroke.

Notes: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.

[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

Table 6-22: National CVD Benefits, Option 1b (PFOA and PFOS MCLs of 5.0 ppt)

Benefits Category	2% Discount Rate		
	5th Percentile[a]	Expected Value	95th Percentile[a]
Number of Non-Fatal MI Cases Avoided	1,209.2	5,352.0	9,417.5
Number of Non-Fatal IS Cases Avoided	1,778.3	7,826.9	13,778.0
Number of CVD Deaths Avoided	733.1	3,146.8	5,518.0
Total Annualized CVD Benefits (Million $2022)[b]	**$119.18**	**$513.27**	**$900.13**

Abbreviations: CVD – cardiovascular disease, MI – myocardial infarction, IS – Ischemic Stroke.
Notes: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates.
[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.
[b] See Table 7-6 for a list of the nonquantifiable costs, and the potential direction of impact these costs would have on the estimated monetized total annualized costs in this table.

Table 6-23: National CVD Benefits, Option 1c (PFOA and PFOS MCLs of 10.0 ppt)

Benefits Category	2% Discount Rate		
	5th Percentile[a]	Expected Value	95th Percentile[a]
Number of Non-Fatal MI Cases Avoided	673.7	2,776.5	4,872.8
Number of Non-Fatal IS Cases Avoided	987.0	4,079.2	7,145.6
Number of CVD Deaths Avoided	411.6	1,640.9	2,878.1
Total Annualized CVD Benefits (Million $2022)[b]	**$66.97**	**$267.56**	**$469.05**

Abbreviations: CVD – cardiovascular disease, MI – myocardial infarction, IS – Ischemic Stroke.
Notes: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates.
[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.
[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

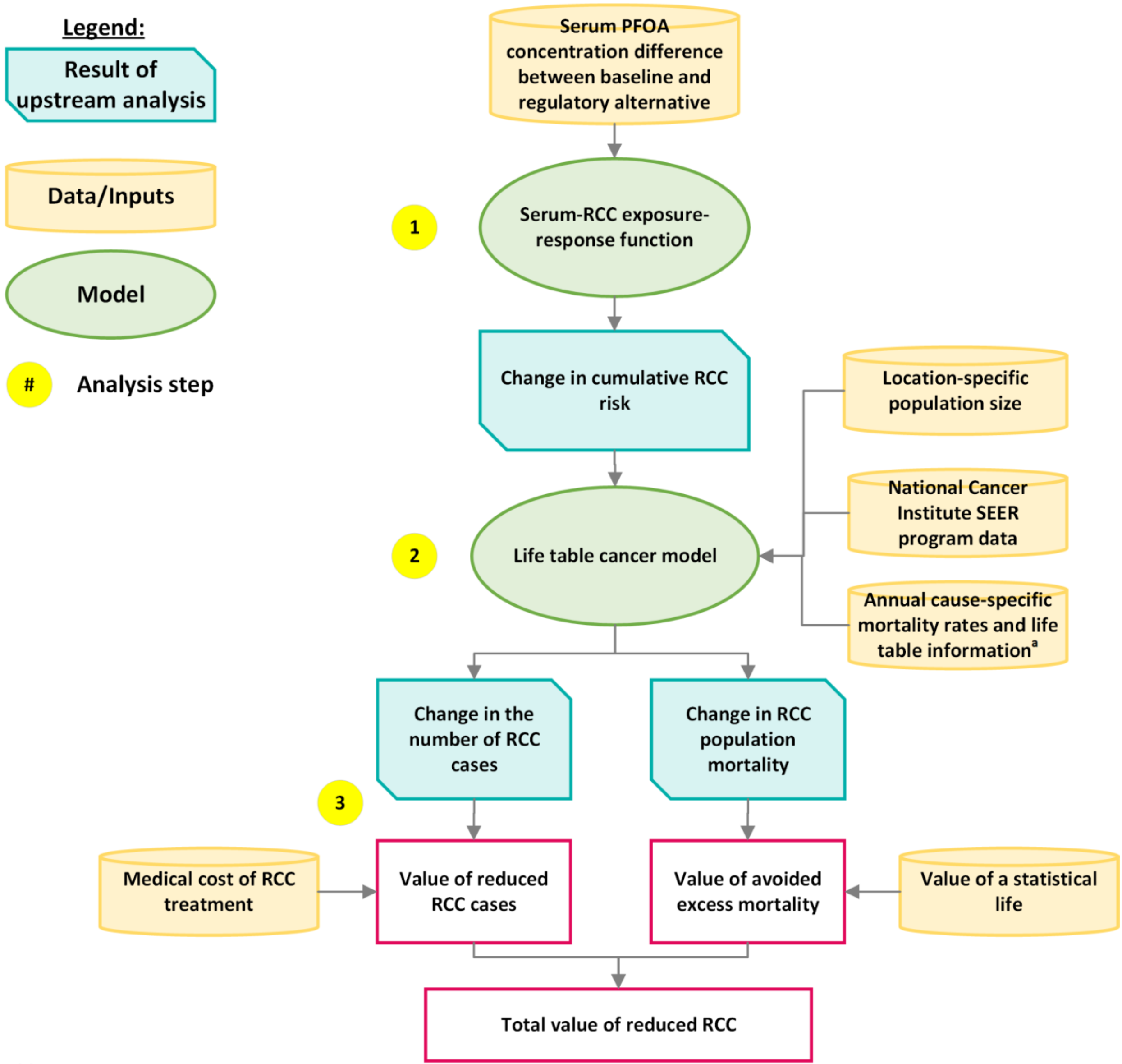
6.6 Renal Cell Carcinoma

6.6.1 Overview of the RCC Risk Reduction Analysis

Figure 6-9 illustrates the approach used to quantify and value the changes in RCC risk associated with lowered serum PFOA levels from reductions in drinking water PFOA concentrations under the regulatory alternatives. Section 4.4 and Section 6.3 detail the PWS EP-specific PFOA drinking water occurrence estimation and modeling of serum PFOA concentrations, respectively. PWS EP-specific time series of the differences between serum PFOA concentrations under baseline and regulatory alternatives are inputs into this analysis. For each PWS EP, evaluation of the changes in RCC impacts involves the following key steps:

1. Estimating the changes in RCC risk based on modeled changes in serum PFOA levels and the exposure-response function for the effect of serum PFOA on RCC;
2. Estimating the annual incidence of RCC cases and excess mortality among those with RCC in all populations corresponding to baseline and regulatory alternative RCC risk levels, as well as estimating the regulatory alternative-specific reduction in cases relative to the baseline; and
3. Estimating the economic value of reducing RCC mortality from baseline to regulatory alternative levels, using the Value of Statistical Life and COI measures, respectively.

Section 6.6.2 discusses the exposure-response modeling for RCC. Section 6.6.3 summarizes the life table-based approach for estimation of RCC risk reductions. Section 6.6.4 discusses the EPA's valuation methodology for RCC mortality and morbidity. Section 6.6.5 presents the results of the analysis.



Abbreviations:
PFOA – perfluorooctanoic acid, RCC – renal cell carcinoma, SEER - Surveillance, Epidemiology, and End Results program
Notes:
(a) Data from the Centers for Disease Control (CDC) and Prevention.

Figure 6-9: Overview of Analysis of Reduced RCC Risk

6.6.2 RCC Exposure-Response Modeling

To identify an exposure-response function, the EPA reviewed studies highlighted in the HESD for PFOA (U.S. EPA, 2016f) and a recent study discussed in both the California Environmental Protection Agency's Office of Environmental Health Hazard Assessment (OEHHA) PFOA Public Health Goals report (CalEPA, 2021) and the EPA's Final Human Health Toxicity Assessment for PFOA (U.S. EPA, 2024f). Steenland and Woskie (2012) observed an increase in kidney cancer deaths among workers with high exposures to PFOA. Vieira et al. (2013) found that kidney cancer was positively associated with "high" and "very high" PFOA exposures. Barry et al. (2013) found a slight trend in cumulative PFOA serum exposures and kidney cancer among the C8 Health Project population.[71] In a large case-control general population study of the relationship between PFOA and kidney cancer in 10 locations across the U.S., Shearer et al. (2021) found evidence that exposure to PFOA is associated with RCC, the most common form of kidney cancer, in humans.

To evaluate changes between baseline and regulatory alternative RCC risk resulting from reduced exposure to PFOA, the EPA relied on the estimated time series of changes in serum PFOA concentrations (Section 6.3) and the serum-RCC exposure-response function provided by Shearer et al. (2021): 0.00178 (95% CI: 0.00005, 0.00352) per ng/mL. The analysis reported in Shearer et al. (2021) was designed as a case-control study with population controls based on 10 sites within the U.S. population. Shearer et al. (2021) accounted for age, sex, race, ethnicity, study center, year of blood draw, smoking, and hypertension in modeling the association between PFOA and RCC. Results showed a strong and statistically significant association between PFOA and RCC. The EPA selected the exposure-response relationship from Shearer et al. (2021) because it included exposure levels typical in the general population and the study was found to have a low risk of bias when assessed in the EPA's *Final Human Health Toxicity Assessment for PFOA* (U.S. EPA, 2024f).

The linear slope factor developed by the agency (see Section 4.2 of U.S. EPA, 2024f) based on Shearer et al. (2021) enables estimation of the changes in the lifetime RCC risk associated with reduced lifetime serum PFOA levels:

Equation 15:

$$LR(x) = LR(z) + 0.00178 \cdot (x - z)$$

Where $LR(x)$ is the probability of lifetime RCC incidence for an individual exposed to a lifetime average serum PFOA concentration of x ng/mL, and $LR(z)$ is the probability of lifetime RCC at the baseline lifetime average serum PFOA concentration of z ng/mL.

Because baseline RCC incidence statistics are not readily available from the National Cancer Institute public use data, the EPA used kidney cancer statistics in conjunction with an assumption that RCC comprises 90 percent of all kidney cancer cases to estimate baseline lifetime probability of RCC (U.S. EPA, 2024f; American Cancer Society, 2020). The EPA estimated the baseline lifetime RCC incidence for males at 1.89 percent and the baseline lifetime

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[71] The C8 Health Project collected data to ascertain the amount of C8 (otherwise known as PFOA) in blood among Mid-Ohio Valley communities from 2005-2013. Mean PFOA at enrollment was 24 ng/mL.

RCC incidence for females at 1.05 percent. Details of these calculations are provided in Appendix H. Because the Shearer et al. (2021) slope factor is not sex-specific, the EPA averaged sex-specific baseline lifetime RCC estimates to obtain $LR(z) = 0.0147$ for use in the estimation of annual RCC risk changes.

To enable annual RCC risk estimation, the EPA further assumed that the relative risk relationship implied by Equation 15, i.e., $RR(x,z) = LR(x)/LR(z) = 1 + 0.00178 \cdot (x - z)/LR(z) = 1 + 0.00178 \cdot (x - z)/0.0147$, also holds for the cumulative RCC risk and cumulative average exposure to serum PFOA from birth to a specific age.

A person's cumulative serum PFOA exposure by age a—denoted by x_a—is defined as:

Equation 16:

$$x_a = \frac{1}{a}\sum_{i=0}^{a-1} serum\ PFOA_i\,, x_0 = 0$$

The EPA estimated the relative risk of RCC by a particular age from a change in average serum PFOA experienced by this age as follows:

Equation 17:

$$RR(x_a, z_a) = max\left(1 - PAF, 1 + \frac{0.00178\ \cdot (x_a - z_a)}{0.0147}\right)$$

Where $RR(x_\mathrm{a}, z_\mathrm{a})$ is the relative cumulative risk of RCC by age a associated with a change from baseline cumulative exposure z_a to treatment cumulative exposure x_a and PAF is the environmental exposure-related population attributable fraction of RCC incidence set at 0.0394. As such, this equation implies that the EPA caps the magnitude of PFOA-related cumulative RCC risk reduction at the PAF of 3.94 percent to ensure plausibility of the estimated RCC benefits size. The EPA developed this PAF estimate based on its review of literature on environmental contaminant-attributable risk estimates for cancers (ICF, 2022b). In calculations of the annual RCC risk changes, the EPA continued to assume that RCC comprises 90 percent of annual kidney cancer incidence.

6.6.3 Estimation of RCC Risk Reductions

The EPA relies on the life table approach to estimate RCC risk reductions because:

- Changes in serum PFOA in response to changes in drinking water PFOA occur over multiple years;
- Annual risk of new RCC should be quantified only among those not already experiencing this chronic condition;
- RCC has elevated mortality implications.

The EPA used recurrent life table calculations to estimate PWS EP-specific time series of RCC incidence for a population cohort characterized by sex, race/ethnicity, birth year, and age at the beginning of the evaluation period (i.e., 2024) under the baseline scenario and the regulatory alternatives. The life table analysis accounts for the gradual changes in lifetime exposures to PFOA following implementation of treatment under the regulatory alternatives compared to the baseline.[72] Details of the life table calculations are provided in Appendix H. The outputs of the life table calculations are the PWS EP-specific estimates of the annual change in the number of RCC cases and the annual change in RCC population mortality.

Although the change in PFOA exposure likely affects the risk of developing RCC beyond the end of the analysis period (the majority of RCC cases manifest during the latter half of the average individual lifespan; see Appendix H), the EPA does not capture effects after the end of the period of analysis, 2105. Individuals alive after the end of the period of analysis likely benefit from lower lifetime exposure to PFOA. Lifetime health risk model data sources include SDWIS/Fed; age-, sex-, and race/ethnicity-specific population estimates from the U.S. Census Bureau (U.S. Census Bureau, 2020a); the Surveillance, Epidemiology, and End Results (SEER) program database (National Cancer Institute),[73] and the CDC NCHS.[74] Appendix H provides additional detail on the data sources and information used in this analysis as well as baseline kidney cancer statistics. Appendix B describes estimation of the affected population.

6.6.4 Valuation of RCC Risk Reductions

The EPA uses the Value of Statistical Life to estimate the benefits of reducing mortality associated with RCC in the population exposed to PFOA in drinking water. Section 2.2 provides information on updating Value of Statistical Life for inflation and income growth. The EPA uses the COI-based valuation to estimate the benefits of reducing morbidity associated with RCC.

The EPA used the medical cost information from a recent RCC cost-effectiveness study by Ambavane et al. (2020) to develop COI estimates for RCC morbidity. Ambavane et al. (2020) used a discrete event simulation model to estimate the lifetime treatment costs of several RCC treatment sequences, which included first and second line treatment[75] medication costs, medication administration costs, adverse effect management costs, and disease management costs on- and off-treatment. To this end, the authors combined RCC cohort data from a CheckMate 214 clinical trial and recent US-based healthcare cost information assembled from multiple sources (see supplementary information from Ambavane et al. (2020)).

The EPA received public comments on the economic analysis for the proposed rule related to the EPA's use of cost of illness information for morbidity valuation. Specifically, some commenters recommended that the EPA use willingness to pay information (instead of cost of illness information) when valuing the costs associated with non-fatal illnesses, stating that willingness to pay information better accounts for lost opportunity costs (e.g., lost productivity and pain and suffering) associated with non-fatal illnesses. To better account for these opportunity costs, the

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[72] As described above, the EPA models PFAS changes under the regulatory alternatives as being in effect for the years 2024 through 2105, with nonzero PFAS changes first occurring in 2029, the year when all PWSs are assumed to comply with PFAS treatment requirements.

[73] For cancer incidence and stage distribution data, the EPA relies on SEER 21 (2009-2018); for cancer survival data, the EPA relies on SEER 18 (2000-2017).

[74] CDC WONDER data on 1999-2019 all-cause and kidney cancer mortality by age and sex.

[75] Second line cancer treatment is a treatment implemented after the failure of the initial treatment (i.e., first line treatment). The first line treatment may fail because it stops working or has side effects that are not tolerated.

EPA used recently available willingness to pay values in a sensitivity analysis for morbidity associated with RCC. The sensitivity analysis results show that when willingness to pay values are used in RCC benefits analysis, morbidity benefits are increased by 2.0 percent. See Appendix O for full details and results on the willingness to pay sensitivity analyses.

Table 6-24 summarizes RCC morbidity COI estimates derived by the EPA using Ambavane et al. (2020)-reported disease management costs on- and off-treatment along with medication, administration, and adverse effect management costs for the first line treatment that initiated the most cost-effective treatment sequences as identified by Ambavane et al. (2020), i.e., the nivolumab and ipilimumab drug combination. This is a forward-looking valuation approach in that it assumes that the clinical practice would follow the treatment recommendations in Ambavane et al. (2020) and other recent studies cited therein. The EPA notes that the second line treatment costs are not reflected in the EPA's COI estimates, because Ambavane et al. (2020) did not report information on the expected durations of the treatment-free interval (between the first line treatment discontinuation and the second line treatment initiation) and the second line treatment phase, conditional on survival beyond discontinuation of the second line treatment. As such, the EPA valued RCC morbidity at $261,175 ($2022) during year 1 of the diagnosis, $198,705 ($2022) during year 2 of the diagnosis, and $1,661 ($2022) starting from year 3 of the diagnosis. Additionally, the EPA assumed that for individuals with RCC who die during the specific year, the entire year-specific cancer treatment regimen is applied prior to the death event. This may overestimate benefits if a person does not survive the entire year.

Table 6-24: RCC Morbidity Valuation

Time Interval	First Line Medication ($2018)[a]	First Line Administration ($2018)[a]	First Line Adverse Effect Management ($2018)[a,c]	Disease Management ($2018)[a]	Total ($2018)	Total ($2022)[d]
Monthly cost, month 1-3 from diagnosis[a,e]	32,485	516	78	73	33,152	37,382
Monthly cost, month 4-24 from diagnosis[b,f]	13,887	647	78	73	14,685	16,559
Monthly cost, month 25+ from diagnosis[g]	-	-	-	123	123	139
Annual cost, year 1 from diagnosis	222,438	7,371	934	878	231,621	261,175
Annual cost, year 2 from diagnosis	166,644	7,764	934	878	176,220	198,705
Annual cost, year 3+ from diagnosis	-	-	-	1,473	1,473	1,661

Abbreviations: RCC – renal cell carcinoma.
Notes:
[a] Ambavane et al. (2020) Table 1;
[b] Ambavane et al. (2020) p. 41, a maximum treatment duration assumption of 2 years;
[c] The adverse effect management costs of $1,868 in Ambavane et al. (2020) Table 1 were reported for the treatment duration. The EPA used the treatment duration of 24 months (i.e., 2 years) to derive monthly costs of $77.83.
[d] To adjust for inflation, the EPA used U.S. Bureau of Labor Statistics Consumer Price Index for All Urban Consumers: Medical Care Services in U.S. (City Average).
[e] First line treatment induction
[f] First line treatment maintenance
[g] Treatment-free interval

6.6.5 Results

Table 6-25 to Table 6-28 provide the health effects avoided and valuation associated with renal cell carcinoma.

Table 6-25: National RCC Benefits, Final Rule (PFOA and PFOS MCLs of 4.0 ppt each, PFHxS, PFNA, and HFPO-DA MCLs of 10 ppt each and HI of 1) (Million $2022)

Benefits Category	2% Discount Rate		
	5th Percentile[a]	**Expected Value**	**95th Percentile[a]**
Number of Non-Fatal RCC Cases Avoided	1,091.5	6,964.2	17,937.0
Number of RCC-Related Deaths Avoided	320.4	2,028.8	5,206.5
Total Annualized RCC Benefits (Million $2022)[b,c]	**$61.33**	**$353.90**	**$883.55**

Abbreviations: RCC – renal cell carcinoma.

Notes: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates. Quantifiable benefits are increased under final rule table results relative to the other options presented because of modeled PFHxS occurrence, which results in additional quantified benefits from co-removed PFOA and PFOS.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.

[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

[c] When using willingness to pay metrics to monetize morbidity benefits, total annualized RCC benefits are increased by $7.1 million (see Appendix O).

Table 6-26: National RCC Benefits, Option 1a (PFOA and PFOS MCLs of 4.0 ppt)

Benefits Category	2% Discount Rate		
	5th Percentile[a]	**Expected Value**	**95th Percentile[a]**
Number of Non-Fatal RCC Cases Avoided	1,082.0	6,922.4	17,870.0
Number of RCC-Related Deaths Avoided	319.1	2,016.7	5,190.9
Total Annualized RCC Benefits (Million $2022)[b]	**$60.90**	**$351.79**	**$877.47**

Abbreviations: RCC – renal cell carcinoma.

Notes: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.

[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

Table 6-27: National RCC Benefits, Option 1b (PFOA and PFOS MCLs of 5.0 ppt)

Benefits Category	2% Discount Rate		
	5th Percentile[a]	Expected Value	95th Percentile[a]
Number of Non-Fatal RCC Cases Avoided	851.9	5,696.1	14,906.0
Number of RCC-Related Deaths Avoided	251.6	1,663.8	4,328.4
Total Annualized RCC Benefits (Million $2022)[b]	**$48.41**	**$290.72**	**$730.99**

Abbreviations: RCC – renal cell carcinoma.
Notes: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates.
[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.
[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

Table 6-28: National RCC Benefits, Option 1c (PFOA and PFOS MCLs of 10.0 ppt)

Benefits Category	2% Discount Rate		
	5th Percentile[a]	Expected Value	95th Percentile[a]
Number of Non-Fatal RCC Cases Avoided	372.1	2,648.1	6,967.4
Number of RCC-Related Deaths Avoided	111.5	782.8	2,057.3
Total Annualized RCC Benefits (Million $2022)[b]	**$21.20**	**$137.30**	**$352.07**

Abbreviations: RCC – renal cell carcinoma.
Notes: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates.
[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.
[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

6.7 Benefits from Co-Removal of Disinfection Byproducts

As part of its health risk reduction and cost analysis, the EPA is directed by SDWA to evaluate quantifiable 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 from reductions in co-occurring contaminants that may be attributed solely to compliance with the maximum contaminant level (SDWA 1412(b)(3)(C)(II)). These co-occurring contaminants are expected to include additional PFAS contaminants not directly regulated by the final PFAS NPDWR, co-occurring chemical contaminants such as SOCs, VOCs, and DBP precursors. In this section, the

EPA presents a quantified estimate of the reductions in DBP formation potential that are likely to occur as a result of compliance with the final PFAS NPDWR.[76]

6.7.1 Overview of Reduced Disinfection Byproduct Formation

DBPs are formed when disinfectants react with naturally occurring materials in water. Under the Stage 2 Disinfectants and Disinfection Byproducts Rule (Stage 2 DBP Rule, U.S. EPA, 2006b), the EPA regulates 11 individual DBPs from three subgroups: four trihalomethanes, five haloacetic acids, and two inorganic compounds (bromate and chlorite). Under the Stage 2 DBP Rule, compliance is based on a locational running annual average (LRAA) calculation, where the annual average at each sampling location in the distribution system is used to determine compliance with the MCL of 0.08 mg/L for THM4 (regulated as TTHM, bromodichloromethane, bromoform, chloroform, and dibromochloromethane). There is a substantial body of literature on DBP precursor occurrence and THM4 formation mechanisms in drinking water treatment. The formation of THM4 in a particular drinking water treatment plant is a function of several factors including disinfectant type, disinfectant dose, bromide concentration, organic material type and concentration, temperature, pH, and system residence times. Epidemiology studies have shown that THM4 exposure, a surrogate for chlorinated drinking water, is associated with an increased risk of bladder cancer, among other diseases (Cantor et al., 1998; Cantor et al., 2010; Costet et al., 2011; Freeman et al., 2017; King & Marrett, 1996; Regli et al., 2015; Villanueva et al., 2004; Villanueva et al., 2006; U.S. EPA, 2019d). These studies considered THM4 as surrogate measures for DBPs formed from the use of chlorination that may co-occur. Reductions in exposure to THM4 is expected to yield significant public health benefits (Regli et al., 2015). In what Richardson (2022) describes as the “largest risk assessment of DBPs in the U.S. to date, focusing on bladder cancer cases associated with chlorinated drinking water”, Weisman et al. (2022) estimated that 8,000 of 79,000 national cases of bladder cancer are attributable to DBPs in drinking water.

The EPA used the following data sources for the DBP co-removal analysis (see Table 6-29).

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[76] The methodology detailed in Section 6.7.1 on estimated DBP reductions was externally peer reviewed by three experts in GAC treatment for PFAS removal and DBP formation potential. The external peer reviewers supported the EPA’s approach and edits based on their recommendations for clarity and completeness are reflected in the following analysis and discussion. Please see “Response to Letter of Peer Review for Disinfectant Byproduct Reduction " (U.S. EPA, 2023b) for discussion of the peer review and the EPA’s responses to peer reviewed comments.

Table 6-29: Data Sources and How the Information Derived from each Source is Used in the DBP Co-Removal Analysis

Data Source	Acronym	How Specific Data were Used in Analysis
Consumer Confidence Reports	CCR	• Identify GAC treatment start date/year. • Identify intended purpose for GAC treatment. • Estimate baseline THM4 (four regulated trihalomethanes) concentrations at systems when SYR4 data were unavailable. • Calculate THM4 reduction at systems when SYR4 data were unavailable.
DBP Information Collection Rule Treatment Study Database	DBP ICR TSD	• Estimate changes in THM4 levels based on implementing GAC treatment.
DBP ICR Aux 1 (1998)	Aux 1	• Evaluate changes in DBP precursor occurrence over time by comparing TOC data to SYR3 TOC data.
Six-Year Review 3, Information Collection Rule (2011)	SYR3 ICR	• Evaluate raw water TOC data.
Six-Year Review 4, Information Collection Rule (2019)	SYR4 ICR	• Evaluate raw water TOC data. • Estimate baseline THM4 concentrations. • Calculate THM4 reductions.
Unregulated Contaminant Monitoring Rule 3	UCMR 3	• Inform a Bayesian occurrence model to identify PWSs expected to implement treatment under the NPDWR. • Identify PWSIDs that had a detectable level of PFOA and/or PFOS to identify systems used in trihalomethane reduction comparison.
Unregulated Contaminant Monitoring Rule 4	UCMR 4	• Identify plants that indicated GAC treatment. • Inform disinfectant type.

Abbreviations: THM4 – four regulated trihalomethanes; DBP – disinfection byproduct; NPDWR – National Primary Drinking Water Regulation; PWS – public water system; PWSID – public water system identifier; SYR – Six-Year Review; GAC – granular activated carbon; TOC – total organic carbon; PFOS – perfluorooctane sulfonic acid; PFOA – perfluorooctanoic acid.

6.7.1.1 Overview of PFAS Treatment with Disinfection Byproduct Reduction

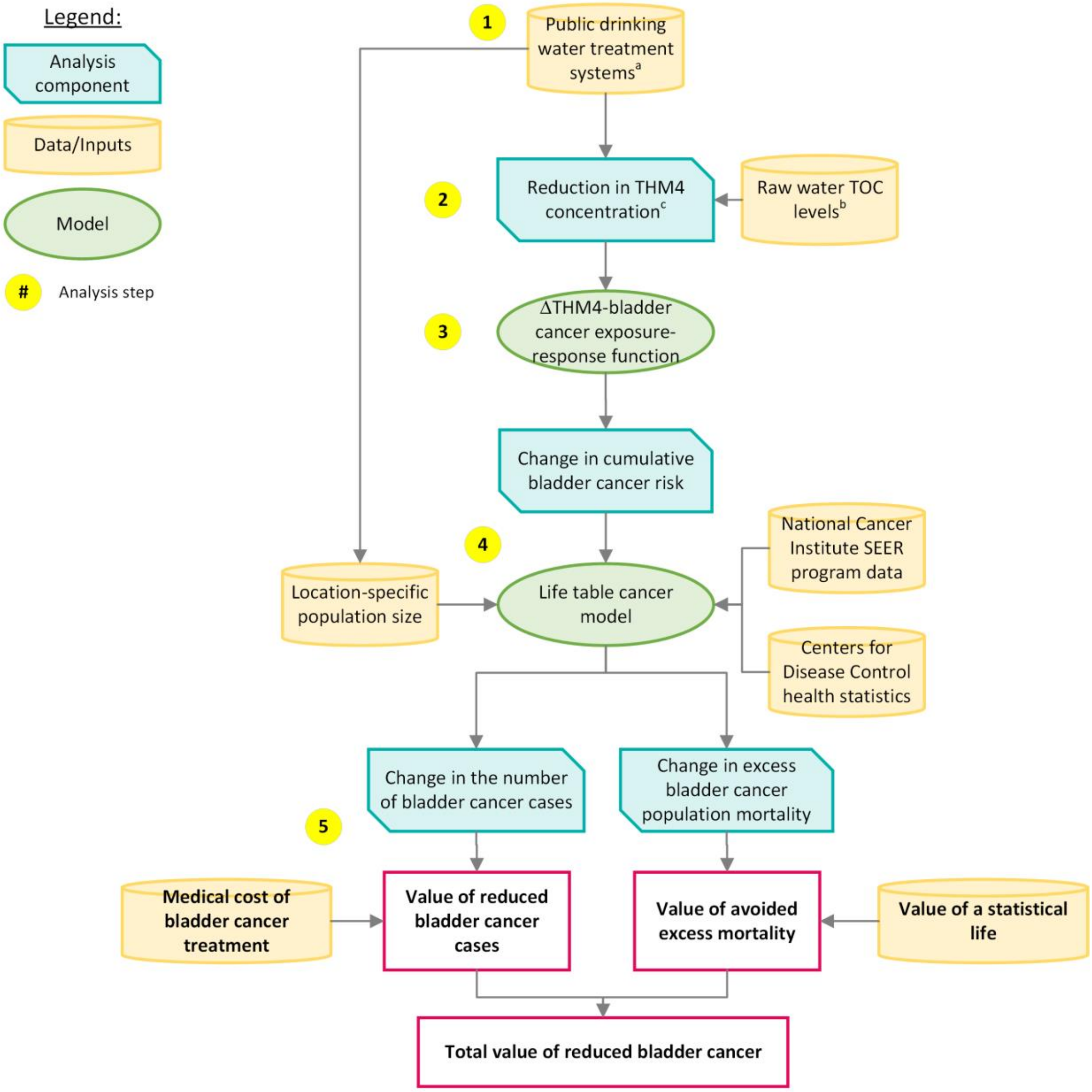
GAC adsorption has been used to remove synthetic organic chemicals, taste and odor compounds, and natural organic matter (NOM) during drinking water treatment (Chowdhury et al., 2013). Recently, many water utilities have installed or are considering installing GAC and/or other advanced technologies as a protective or mitigation measure to remove various contaminants of emerging concern, such as PFAS (Dickenson & Higgins, 2016). Because NOM often exists in a much higher concentration (in mg/L) than trace organics (in µg/L or ng/L) in water, NOM, often measured as TOC, can interfere with the adsorption of trace organics by outcompeting the contaminants for adsorption sites and by general fouling (blockage of adsorption pores) of the GAC.

NOM and inorganic matter are precursors for the formation of THMs and other DBPs when water is disinfected using chlorine and other disinfectants to control microbial contaminants in finished drinking water. Removal of DBP precursors through adsorption onto GAC has been included as a treatment technology for compliance with the existing DBP Rules and is a BAT for the Stage 2 DBP Rule. Dissolved organic matter can be removed by GAC through adsorption and biodegradation (Crittenden et al., 1993; W. H. Kim et al., 1997; Yapsakli & Çeçen, 2010). Upon startup, the initial removal is via adsorption of the DBP precursors; GAC is well-established for removal of THM and HAA precursors (Dastgheib et al., 2004; Cheng et al., 2005; Iriarte-Velasco et al., 2008; Summers et al., 2013; Cuthbertson et al., 2019; L. Wang et al., 2019). However, biodegradation becomes the predominant mechanism over time as adsorption capacity is exhausted and microbial growth within the GAC column establishes itself (Speitel Jr et al., 1989; Velten et al., 2007). In addition to removal of organic DBPs, GAC also exhibits some capacity for removal of inorganic DBPs such as bromate and chlorite (Kirisits et al., 2000; Sorlini & Collivignarelli, 2005) and removal of preformed organic DBPs via adsorption and biodegradation (Jiang et al., 2017; Terry & R.S., 2018). Further, GAC may offer limited removal of dissolved organic nitrogen (Chili et al., 2012).

Based on an extensive review of published literature in sampling studies where both contaminant groups (PFAS and DBPs) were sampled, there is limited information about PFAS removal and co-occurring reductions in DBPs, specifically THMs. To help inform its economic analysis, the EPA relied on the DBP Information Collection Rule Treatment Study Database and DBP formation studies to estimate reductions in THM4 (ΔTHM4) that may occur when GAC is used to remove PFAS. Subsequently, these results were compared to THM4 data from PWSs that have detected PFAS and have indicated use of GAC.

The objective of the co-removal benefits analysis is to determine the reduction in bladder cancer cases associated with the decrease of regulated THM4 in treatment plants due to the installation of GAC for PFAS removal. Figure 6-10 illustrates the EPA's approach for quantifying the human health benefits of reducing THM4 levels in drinking water. The analysis entails:

1. Estimating the number of systems expected to install GAC treatment in compliance with the final PFAS NPDWR and affected population size;
2. Estimating changes in THM4 levels that may occur when GAC is installed for PFAS removal based on influent TOC levels;
3. Estimating changes in the cumulative risk of bladder cancer using an exposure-response function linking lifetime risk of bladder cancer to THM4 concentrations in residential water supply (Regli et al., 2015);
4. Estimating annual changes in the number of bladder cancer cases and mortality in the bladder cancer population corresponding to changes in THM4 levels under the final rule and regulatory alternatives in all populations alive during or born after the start of the evaluation period;
5. Estimating the economic value of reducing bladder cancer morbidity and mortality from baseline to the final rule and regulatory alternative levels, using COI measures and the Value of Statistical Life, respectively.



Abbreviations: THM4 = Four Regulated Species of Trihalomethanes; SEER = Surveillance, Epidemiology, and End Results; TOC = Total Organic Carbon

Notes:

[a] Systems expected to be triggered into PFAS treatment using either granular activated carbon (GAC) or ion exchange (IX) treatment technologies.

[b] Based on median raw water TOC annual system-means for non-purchased water systems.

[c] Based on THM4 reductions due to GAC installation from the disinfection byproduct (DBP) information collection rule (ICR) treatment studies. Reductions dependent on empty bed contact time (EBCT) and source water type (surface water or groundwater).

Figure 6-10: Overview of Analysis of Co-Removal Benefits

6.7.1.2 Baseline Information on DBP Precursors and Trihalomethane Formation

DBP precursors are the chemical constituents that are reactants or intermediates in the formation of DBPs. Precursors can be characterized by their origin and the nature of their chemistry (inorganic vs. organic). Precursors include NOM and anthropogenic organic matter (i.e., wastewater) from watersheds, organic matter contaminants within treatment processes, and biofilm growth within the distribution system. Additional precursors include inorganic matter present in source water from anthropogenic and natural sources, or chemical additives introduced during treatment. The presence of DBP precursors is site-specific and dependent on many factors such as, but not limited to, environment, location, watershed, and treatment.

The EPA evaluated raw water TOC data included in the SYR3 and SYR4 ICR datasets (U.S. EPA, 2016j; U.S. EPA, 2022f). The fourth Unregulated Contaminant Monitoring Rule (UCMR 4) TOC data were not used since that dataset did not include THM4 information. In addition, the EPA compared the DBP ICR Aux 1 TOC data (pre-Stage 1 DBP Rule[77]) to the SYR3 ICR TOC data to evaluate changes in DBP precursor occurrence over time. PWSs (specifically subpart H systems[78]) are required to achieve a certain percentage of TOC removal; occurrence estimates for TOC are typically evaluated at the plant-level. The SYR3 ICR dataset contains TOC data for 33 states and systems of all sizes. The SYR4 ICR dataset contains TOC data for 49 states/tribes and systems of all sizes. To be consistent with SYR3 and SYR4 data management protocols, non-detections of TOC were assigned a value of 0.0 mg/L for all plant-mean calculations (U.S. EPA, 2016a).

In U.S. EPA (2005b), the EPA reviewed the raw water TOC levels for ground water plants included in the DBP ICR Aux 1 data. The results shown in Table 6-30 represent the distribution of ground water plant-mean data as calculated using ICR Aux 1 monthly data from the year 1998. Only plants with reported data for at least 9 of the 12 months are included in this summary table. Note that the table does not include results for blended, mixed, or purchased water plants. Table 6-31 shows the distribution of plant-mean TOC concentrations in raw water for non-purchased surface water plants. Segmenting the plants with raw water TOC means provides some indication of the percentage of plants that would be within each THM4 reduction category outlined in Section 6.7.1.3. The levels in ground water plants tended to be lower compared to concentrations in surface water plants (Table 6-30 and Table 6-32 compared to Table 6-31 and Table 6-33). As mentioned above, TOC non-detections were assumed to be zero for plant-mean calculations.

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[77] Stage 1 Disinfectants and Disinfection Byproducts Rule was promulgated by the EPA in December 1998 (U.S. EPA, 1998e).

[78] Subpart H systems are defined as public water systems using surface water or ground water under the direct influence of surface water as a source that are subject to the requirements of subpart H of the National Primary Drinking Water Regulations (U.S. EPA, 2006a).

Table 6-30: DBP ICR (1998), SYR3 ICR (2011), and SYR4 ICR (2019) – Summary of Raw Water TOC Annual System Means for Ground Water Systems

Data Source (Year)[a]	Source Water Type	Count of Systems	Median (mg/L)	Mean (mg/L)	90th Percentile (mg/L)	Range of System-Means[b]
DBP ICR (1998)	Ground Water	103	0.19	1.46	3.36	0.0 - 16.1
SYR3 ICR (2011)	Ground Water	68	2.19	3.33	5.85	0.42 – 17.0
SYR4 ICR (2019)	Ground Water	80	1.50	2.54	7.11	0.0 – 15.73

Notes:
Abbreviations: DBP – disinfection byproduct; ICR – information collection rule; SYR – Six-Year Review; TOC – total organic carbon.
[a] Using SYR3 cutoff values, values > 100 mg/L were excluded from calculations.
[b] Values below the MRL were converted to 0.0 mg/L to calculate system-means.
Source: ICR AUX1 database; table extracted from Exhibit 3.6 of U.S. EPA (2005b).

Table 6-31: DBP ICR (1998), SYR3 ICR (2011), and SYR4 ICR (2019) – Summary of Raw Water TOC Annual System Means for Surface Water Systems

Data Source (Year)[a]	Source Water Type	Count of Systems	Median (mg/L)	Mean (mg/L)	90th Percentile (mg/L)	Range of System-Means[b]
DBP ICR (1998)	Surface Water	307	2.71	3.14	5.29	0.0 – 21.4
SYR3 ICR (2011)	Surface Water	756	2.89	3.45	6.45	0.0 – 29.3
SYR4 ICR (2019)	Surface Water	802	3.29	3.88	6.93	0.0 – 38.9

Abbreviations: ICR – information collection rule; SYR – Six-Year Review; TOC – total organic carbon.
Notes:
[a] Using SYR3 cutoff values, values > 100 mg/L were excluded from calculations.
[b] Values below the MRL were converted to 0.0 mg/L to calculate system-means.

The EPA reviewed the finished water TOC levels included in SYR3 ICR and SYR4 ICR data. The results shown in Table 6-32 represent the distribution of TOC concentrations for ground water plants. Note that ground water plants are not federally required to report finished water TOC data. In addition, the EPA reviewed finished water TOC levels for surface water plants included in SYR3 and SYR4 ICR data. Table 6-33 displays the distribution of TOC levels in finished water for surface water plants. Similar to the raw water comparison, TOC levels tended to be higher among surface water plants compared to ground water plants.

Table 6-32: SYR3 ICR (2011) and SYR4 ICR (2019) – Summary of Finished Water TOC Annual System Means for Ground Water Systems

Data Source (Year)[a]	Source Water Type	Count of Systems	Median (mg/L)	Mean (mg/L)	90th Percentile (mg/L)	Range of System-Means[b]
SYR3 ICR (2011)	Ground Water	78	1.86	2.30	4.53	0.0 – 11.4
SYR4 ICR (2019)	Ground Water	113	0.73	2.77	3.63	0.0 – 93.0

Abbreviations: ICR – information collection rule; SYR – Six-Year Review; TOC – total organic carbon.
Notes:
[a] Using SYR3 cutoff values, values > 100 mg/L were excluded from calculations.
[b] Values below the MRL were converted to 0.0 mg/L to calculate system-means.

Table 6-33: SYR3 ICR (2011) and SYR4 ICR (2019) – Summary of Finished Water TOC Annual System Means for Surface Water Systems

Data Source (Year)[a]	Source Water Type	Count of Systems	Median (mg/L)	Mean (mg/L)	90th Percentile (mg/L)	Range of System-Means[b]
SYR3 ICR (2011)	Surface Water	756	1.93	2.32	3.99	0.0 – 25.1
SYR4 ICR (2019)	Surface Water	802	1.89	2.24	3.90	0.0 – 74.4

Abbreviations: ICR – information collection rule; SYR – Six-Year Review; TOC – total organic carbon.
Notes:
[a] Using SYR3 cutoff values, values > 100 mg/L were excluded from calculations.
[b] Values below the MRL were converted to 0.0 mg/L to calculate system-means.

The EPA compared the levels of raw water TOC between the DBP ICR and SYR3 ICR to evaluate the changes in TOC occurrence over time (U.S. EPA, 2016g). The EPA used 1998 data from the DBP ICR Aux 1 database and 2011 data from the SYR3 ICR dataset and included only the data from systems that were found in both datasets (referred to as “common systems”). The evaluation of TOC changes over time was limited to large surface water systems (≥100,000 population served) because the DBP ICR only covered large systems.

Table 6-34 below presents plant-level summary statistics for finished water TOC from common systems in the Aux 1 database and SYR3 ICR. The common systems were distributed across 14 states (Alabama, Alaska, Illinois, Indiana, Iowa, Kentucky, Nevada, New Jersey, North Carolina, Oklahoma, Pennsylvania, South Carolina, Virginia, and West Virginia). The comparison of data for large surface water supplies between 1998 and 2011 shows a small decrease in treated water TOC levels. The median finished water TOC concentrations at large systems were 1.76, 1.75, and 1.51 mg/L in the Aux 1 database, SYR3 ICR dataset, and SYR4 ICR dataset, respectively.

Table 6-34: DBP ICR (Aux 1; 1998), SYR3 ICR (2011), and SYR4 ICR (2019) – Finished Water Annual System Mean TOC; Common Surface Water Systems

Data Source (Year)	Count of Systems[a]	Median (mg/L)	Mean (mg/L)	90th Percentile (mg/L)	95th Percentile (mg/L)	% Means > 2 mg/L	% Means > 3 mg/L
DBP ICR (1998)		1.76	1.77	2.90	3.23	34%	8%
SYR3 ICR (2011)	80	1.75	1.74	2.78	3.24	30%	8%
SYR4 ICR (2019)	80	1.51	1.49	2.44	2.81	21%	5%

Abbreviations: DBP – disinfection byproduct; ICR – information collection rule; SYR – Six-Year Review; TOC – total organic carbon.

Note:

[a] Some systems included data for multiple plants.

Source: Table extracted from Exhibit 6.11 of U.S. EPA (2016g)

Table 6-35 summarizes THM4 baselines under DBP ICR, which represents pre-Stage 1 and Stage 2 DBP Rules. Prior to evaluating the SYR4 ICR THM4 data, the EPA removed values greater than 10 times the MCL (800 µg/L) due to potential data entry errors. Additionally, the EPA converted values below the MRL (10 µg/L) to 0 µg/L, which is consistent with previous SYR data analysis (U.S. EPA, 2016a). Average THM4 values were higher for surface water plants compared to ground water plants across the two datasets. Within the DBP ICR dataset, representing PWSs serving populations ≥100,000, 82 ground water plants had a median THM4 concentration of 6.8 µg/L with a range of 0-123 µg/L. For the 213 surface water plants in the DBP ICR, the median THM4 concentration was 40 µg/L with a range of 0 to 117 µg/L. In comparison, post-Stage 1 and 2 DBP Rules SYR4 ICR data show median THM4 concentrations of 5.0 µg/L and 41.4 µg/L and mean THM4 concentrations of 13.4 µg/L and 41.1 µg/L in ground water and surface water, respectively. Plant means ranged from 0 to 371.4 µg/L and from 0 to 263.8 µg/L for ground water and surface water, respectively. Note that the SYR4 dataset was from voluntary submissions and includes data from systems of all sizes. The SYR4 ICR reduced dataset, limited to PWSs serving populations ≥100,000, shows median THM4 concentrations of 24.4 and 36.1 µg/L and mean THM4 concentrations of 25.0 and 35.1 µg/L for ground water and surface water, respectively. Plant means ranged from 0 to 66.6 µg/L and from 0 to 62.0 µg/L for ground water and surface water, respectively.

Table 6-35: Summary of THM4 Baseline Comparing DBP ICR and SYR4 ICR

Data Source	Source Water Type	Count of Systems[c]	THM4 Median (µg/L)	THM4 Mean (µg/L)	90th Percentile (µg/L)	Range of System-Means[d]
DBP ICR (1998)[a]	Ground Water	82	6.8	15.4	37	0–123
DBP ICR (1998)[a]	Surface Water	213	40	42	70	0–117
SYR4 ICR Reduced (2012-2019)[b,e,f]	Ground Water	84	24.4	25.0	53.1	0–66.6
SYR4 ICR Reduced (2012-2019)[b,e,f]	Surface Water	291	36.1	35.1	50.2	0–62.0
SYR4 ICR (2012-2019)[b,e]	Ground Water	26,243	5.0	13.4	38.5	0–371.4
SYR4 ICR (2012-2019)[b,e]	Surface Water	9,618	41.4	41.1	64.1	0–263.8

Abbreviations: DBP – disinfection byproduct; ICR – information collection rule; SYR – Six-Year Review; THM4 – four regulated trihalomethanes.

Notes:

[a] Stage 2 DBP Rule Economic Analysis (U.S. EPA, 2005b), screened data from Exhibit 3.15 and 3.20

[b] Using SYR3 cutoff values, values > 10 times the MCL were excluded from calculations.

[c] NA values and blanks were removed prior to calculations.

[d] Values below the MRL were converted to 0.0 µg/L to calculate system-means.

[e] SYR4 data collected from 2012 to 2019. All years were included in calculations.

[f] SYR4 reduced dataset included only PWSs serving populations ≥100,000

In the Economic Analysis for the Stage 2 DBP Rule, the EPA estimated a combined average THM4 reduction for all systems of 7.8 percent, with surface water systems ranging from 9.2 percent (systems serving ≥10,000) to 7.2 percent (systems serving <10,000), and ground water systems ranging from 1.4 percent (systems serving ≥10,000) to 2.0 percent (systems serving <10,000) (U.S. EPA, 2005b). Comparisons of the DBP ICR THM4 baseline data and the SYR4 data that reflects Stage 1 and Stage 2 DBP Rule changes indicate that the Stage 2 EA slightly overestimated the ΔTHM4 for surface water systems (40 to 41.4 µg/L, 3.5% increase) and underestimated the ΔTHM4 for ground water systems (6.8 to 5.0 µg/L, 26.5% reduction). Comparing all systems (surface water and ground water) serving ≥100,000, no statistically significant difference (p-value = 0.2) was observed between the DBP ICR and SYR4 dataset means. Comparing ground water systems in the DBP ICR dataset to those in the reduced SYR4 dataset showed a statistically significant difference (p-value = 0.0003) in THM4 means, with THM4 increasing in the more recent years (SYR4). Comparing surface water systems in the DBP ICR dataset to those in the reduced SYR4 dataset showed no statistically significant difference (p-value = 0.3) in THM4 means. The lack of statistically significant differences in THM4 means between the DBP ICR and SYR4 datasets for surface water systems indicates that TOC and THM4 trends support the use of the DBP ICR dataset to predict ΔTHM4 resulting from GAC treatment. For large ground water systems (populations ≥100,000), reductions in THM4 mean concentrations may be underestimated due to the increase in THM4 baseline concentrations observed from data reported in the DBP ICR to the SYR4 ICR. Based on the TOC and THM4 trends over time and the percent differences observed between the DBP ICR and SYR4 dataset means, the EPA determined that using the DBP ICR Treatment Study Database results for ΔTHM4 to predict future ΔTHM4 resulting from GAC treatment was justified and reasonable. Additionally, with this focus on GAC treatment and the reduction of THM4, it is important to note that the DBP ICR treatment study required systems to conduct DBP precursor removal studies (Treatment Study Database), which contains the most extensive amount of data on GAC treatment and DBP formation potentials (U.S. EPA, 1996; L. Wang et al., 2019).

Larger datasets, such as SYR ICRs, do not include data on both disinfectant type and DBP formation. The DBP ICR collected this information in addition to other source and water quality parameters. Table 6-36 shows mean THM4 concentrations in the DBP ICR per disinfectant type and source water type.

Table 6-36: DBP ICR (Aux 1) Summary of THM4 Concentrations Based on Disinfectant and Source Water Type

Disinfectant Type	Source Water Type	Count of Plants / Facilities	Mean THM4 concentration (µg/L)
Chloramine	Ground Water	15	29.2
	Surface Water	77	43.2
Free Chlorine	Ground Water	34	21.3
	Surface Water	164	45.0
Free Chlorine + Chloramine (DS)	Ground Water	1	18.7
	Surface Water	20	53.2

Abbreviations: DBP – disinfection byproduct; THM4 – four regulated trihalomethanes; DS – distribution system.

Despite the significant public health improvements provided by the EPA's Stage 2 DBP Rule, DBPs are still estimated to cause approximately 8,000 cases of drinking water-attributable bladder cancer cases every year (Weisman et al., 2022). Hence, there are still public health benefits to be realized when DBPs are reduced when feasible. Where systems install activated carbon, the PFAS rule will, for many systems, further reduce DBP concentrations because of precursor removal. While the Stage 1 and Stage 2 DBP Rules were effective at reducing THM4, there are remaining risks associated with DBP exposure that could be further reduced as shown in the baseline analysis above. The Stage 2 DBP Rule was promulgated in 2006 and since the rule implementation there have been numerous peer-reviewed studies that have shown an increased weight of evidence supporting an association between chlorination DBPs and bladder cancer with updated estimates on attributable cases (Weisman et al., 2022; Regli et al., 2015). Additionally, there is an increased understanding of the role of genetically susceptible populations and exposure routes for THMs (i.e., oral, inhalation, and dermal) that impact risk assessments. This comparison between the SYR4 ICR (2019) and DBP ICR (1998) showed that the DBP ICR THM4 data were still relevant for the post Stage 2 DBP Rule baselines for both TOC (i.e., DBP precursors) and THM4. Because the baseline was pre-Stage 1 (DBP ICR), the EPA took the low-end estimate for THM4 reduction to reduce possible overestimation. Further reduction in TOC concentrations in finished water could be achieved if additional treatment is added (i.e., PFAS removal using GAC treatment).

6.7.1.3 Estimation of Trihalomethane Reduction using Treatment Models

6.7.1.3.1 DBP Information Collection Rule Treatment Study Database

The Information Collection Rule Treatment Study Database (ICR TSD) contains results of the most extensive GAC study conducted on a national scale. The ICR TSD contains treatment study data submitted by systems required to conduct DBP precursor removal studies under the DBP ICR (U.S. EPA, 1996). The systems included in the ICR TSD were considered "challenged" in their ability to achieve compliance with potential Stage 2 DBP rule revision MCLs. The participating systems included surface water systems (and ground water systems under the direct influence of surface water) serving 100,000 or more people and having ≥ 4 mg/L of TOC in source water, and ground water systems serving 50,000 or more people and having ≥ 2 mg/L of TOC in finished water. Both free chlorine and chloramine systems were included in the treatment study (U.S. EPA, 1996; L. Wang et al., 2019).

Data from the ICR TSD study from these "challenged systems" can be used to identify conservative estimates of TOC reduction and associated ΔTHM4. Due to upstream pollution, drought, and/or climate change, individual drinking water sources may be as challenged as when the ICR TSD data were collected (Hashempour et al., 2020; McDonough et al., 2020). While the GAC treatment dataset dates are from 1998, the physical/chemical relationships observed have only improved with the current application of GAC being at least as effective for THM4 as was observed in the ICR TSD (Yuan et al., 2022). While source water parameters and treatments at individual plants have changed over time, as seen in the baseline characterization in Section 6.7.1.2, the EPA determined the ICR TSD was still appropriate to inform estimates of ΔTHM4 formation potential given the lack of available data to directly inform ΔTHM4 from PFAS adsorption studies and the low percent difference in TOC changes on a national scale between the DBP ICR and SYR4 collection efforts.

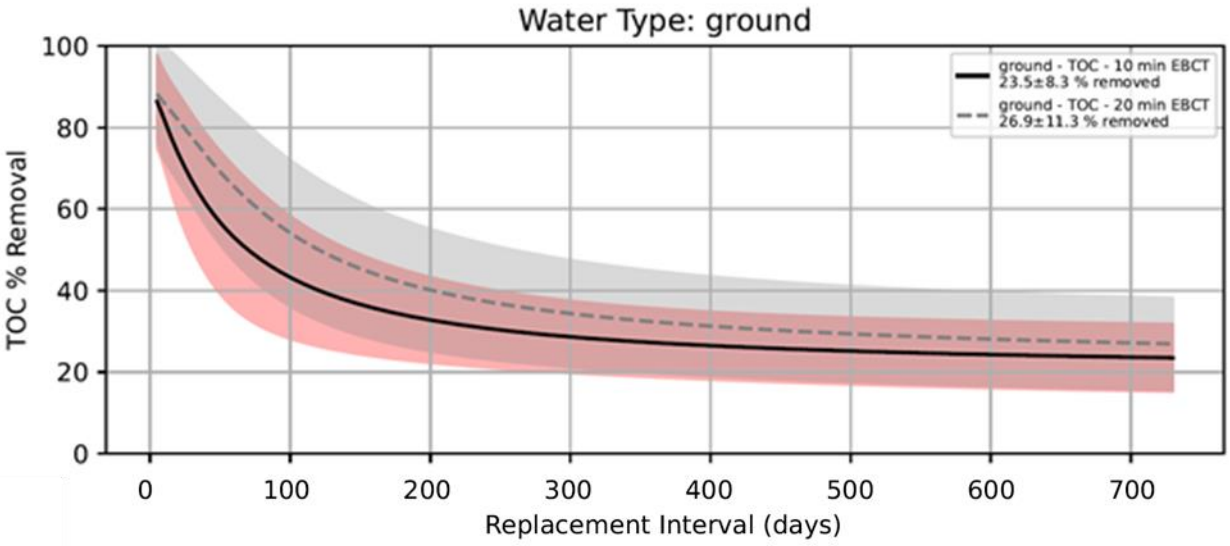
From the 63 GAC systems included in the ICR TSD, a total of 182 pilot/rapid small-scale column test (RSSCT) studies were conducted to develop breakthrough curves of TOC and DBP formation changes. Two EBCTs, 10 and 20 min, were evaluated for treated water that had passed through any full-scale treatment processes previously in place at each drinking water treatment plant to remove precursors (i.e., coagulation/flocculation, sedimentation, filtration) but before any disinfectant was added. To determine the effect of GAC treatment on DBP formation, these studies evaluated TOC removal and THM4 formation potential for the treated water before and after GAC treatment. Uniform formation condition procedures were standardized across each study, with a reaction time of 24 ± 1 hours, temperature of 20.0 ± 1.0°C, buffered pH at 8.0 ± 0.2, and 24-hr chlorine residual of 1.0 ± 0.4 mg/L as Cl_2 (U.S. EPA, 1996; Summers et al., 1996).

The pilot/RSSCT studies were timed to account for seasonal changes and an "averaging" approach was used to remove temporal variations. This approach was consistent with analysis used to characterize different GAC options for compliance with the MCLs under the Stage 2 DBP Rule (Hooper & Allgeier, 2002). Additional details on the GAC study design specifications under the ICR TSD are available in the "ICR Manual for Bench and Pilot Scale Treatment Studies" (U.S. EPA, 1996).

For drinking water systems in the ICR TSD that used chloramines (n = 123 pilots/RSSCTs) in their distribution system, free chlorine was still used in the DBP formation tests, therefore the pilot and RSSCT systems were not compared based on disinfectant type used by the individual treatment system. For reference, a summary of the THM4 estimates by disinfectant type is provided in Appendix I Table I-1. Additionally, if the comparison categories were further parsed by source water type, disinfectant type, and TOC concentrations, then the number of systems in each bin would not provide sufficient studies to compare the ΔTHM4 estimates. Therefore, the EPA analyzed the THM4 reductions based on raw-water TOC.

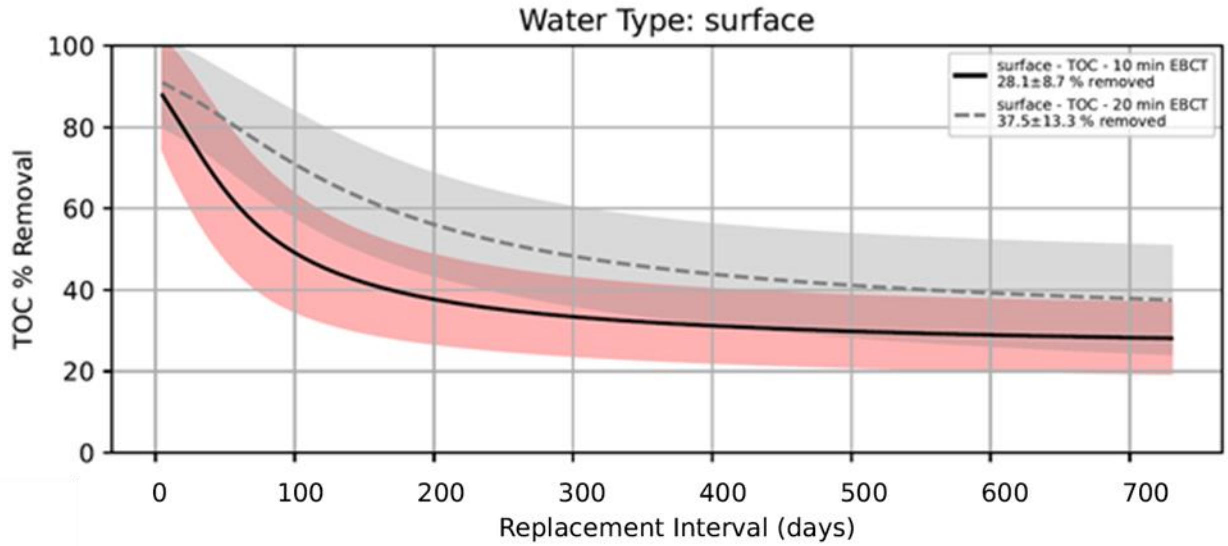
The TOC and THM4 formation potential reductions data from the ICR TSD were modeled with a logistic equation using results from 182 pilot plant/RSSCT studies. The EPA fit the logistic function parameters for each EBCT and did not consider feed water quality parameters. Results were categorized by TOC level and source water type. Further subdivision of these or additional categories would have resulted in very small numbers of systems in bins and some bins not being filled (see Appendix I Table I-1 for example of “disinfection type” added as a category). The model calculated individual system TOC removal for the EBCT and results were averaged for each subset of systems for the GAC replacement interval. The model was not intended to simulate the dynamics of TOC removal by GAC or the formation of THM4, but it simulated the TOC ranges within the pilot/RSSCT studies and the changes in THM4 due to the reduction in TOC observed in the ICR TSD. The EPA used Python to individually fit data from each pilot or RSSCT study in the ICR TSD to a logistic equation and the performance was then averaged. Additional details on the data model are included in Appendix I.

To conservatively estimate national scale THM4 reduction due to GAC treatment to reduce levels of PFAS compounds, the EPA chose a 2-year GAC replacement time. The EPA assumes that this is the longest amount of time before replacement would be required and percent removals are approximately at their long-term removal level with minimal further changes. The PFAS NPDWR will likely result in some systems replacing GAC media more frequently than 2 years, which the EPA expects would result in a greater average TOC reduction since TOC removal decreases over time with GAC treatment (see Figure 6-11 and Figure 6-12 for ground water and surface water respectively). The overall trends seen in Figure 6-11 and Figure 6-12 show greatest TOC removal in the first 200 days of use, after which the predicted TOC removal becomes consistent for ground water with 26.9 percent (EBCT 20 min) and surface water with 37.5 percent (EBCT 20 min).



Abbreviations: TOC – total organic carbon; GAC – granular activated carbon; EBCT – empty bed contact times.
Notes:
Pink shaded area represents ±1 standard deviation for ground water TOC with a GAC EBCT of 10 min
Gray shaded area represents ±1 standard deviation for ground water TOC with a GAC EBCT of 20 min

Figure 6-11: Estimated TOC Percent Removal in Ground Water Using GAC Based on Logistic Equation Model



Abbreviations: TOC – total organic carbon; GAC – granular activated carbon; EBCT – empty bed contact time.
Notes:
Pink shaded area represents ±1 standard deviation for surface water TOC with a GAC EBCT of 10 min
Gray shaded area represents ±1 standard deviation for surface water TOC with a GAC EBCT of 20 min

Figure 6-12: Estimated TOC Percent Removal in Surface Water Using GAC Based on Logistic Equation Model

To estimate the TOC reduction, the ICR TSD pilot/RSSCT studies (n = 182) were partitioned into five potential bins based on TOC concentrations in raw water (Very Low ≤1 mg/L, Low >1 to ≤2 mg/L TOC, Mid >2 to ≤3.5mg/L, High-Mid >3.5 to ≤5mg/L, High TOC >5mg/L TOC). There were no systems in the ICR TSD that fell into very low TOC bin. Based on the logistic equation for TOC reduction, higher raw water TOC concentrations yield greater TOC reductions (in absolute value) following GAC treatment. Table 6-37 shows the TOC reduction for all waters (both ground water and surface water) for a 20 min EBCT.

Table 6-37: TOC Reduction for All Waters (Both Surface Water and Ground Water) with GAC EBCT of 20 Min and a 2-year Replacement Time

TOC Bin	Number of Studies in GAC Treatment Dataset	TOC Reduction ± 1-Standard Deviation (%)	TOC Reduction (mg/L)
TOC 1–2 mg/L	20	41.9 ± 23.2	0.75 ± 0.39
TOC 2–3.5 mg/L	103	37.1 ± 11.6	1.06 ± 0.36
TOC 3.5–5 mg/L	44	32.0 ± 9.6	1.31 ± 0.39
TOC above 5mg/L	15	26.3 ± 14.2	1.83 ± 0.91

Abbreviations: TOC – total organic carbon; GAC – granular activated carbon; EBCT – empty bed contact time.
Note: The model calculated individual system TOC removal and results were averaged for each influent TOC bin for a two-year GAC replacement interval and the standard deviation was calculated for each subset average.

Using the same raw water TOC bins, the EPA estimated national scale ΔTHM4 values resulting from GAC treatment. The selected ΔTHM4 estimate was based on a conservative approach (mean concentration minus one standard deviation), since the DBP ICR systems included in the treatment studies were “challenged systems” (i.e., systems that had difficulty meeting regulatory compliance requirements) that may experience increased TOC reduction due to GAC installation.

The analysis here assumes operation of GAC with a replacement interval of 730 days (2 years). Although some systems will operate with longer replacement intervals, after 730 days, the modeled TOC reduction due to GAC shows consistent removal when further extending the replacement interval (Figure 6-11 and Figure 6-12). While systems may replace GAC at shorter time intervals, the 2-year replacement assumption also approximates blended systems (i.e., multiple GAC treatment trains in parallel with varying replacement intervals) and TOC long term removal by adsorption since GAC treatment for PFAS uses adsorption rather than biodegradation (Kempisty et al., 2022). Therefore, the estimated TOC reduction at 730 days should also be representative for systems with longer replacement intervals or systems with intermittent use. If GAC replacement occurred more frequently due to PFAS treatment needs, higher average TOC removal would occur, resulting in greater THM4 reduction as shown in the 6-month GAC replacement time-steps (1/2, 1, 1 ½, and 2 years) shown in Appendix I (Tables I-2, I-3, I-4, and I-5). Using the longer replacement time of 2 years is consistent with the EPA’s conservative approach to estimating ΔTHM4 even when the presence of PFAS compounds and other source water conditions may affect GAC replacement frequency.

Based on common treatment designs, the EPA expects GAC treatment parameters for PFAS removal to be 20 min EBCTs (U.S. EPA, 2024i). Table 6-38 and Table 6-39 provide estimates of THM4 reductions in the modeled 182 pilot/RSSCT systems considering a 20 min EBCT broken out by surface water versus ground water. The number of GAC systems included in each TOC bin is provided along with the average ΔTHM4 and the “conservative” ΔTHM4 (defined as average ΔTHM4 minus 1-standard deviation) with a GAC replacement time of 2 years.

Table 6-38: Estimation of ΔTHM4 in Surface Water with a 20 Min EBCT, and a 2-year GAC Replacement Time

Raw Water TOC Bin	Number of Studies in GAC Treatment Dataset	Average ΔTHM4 ± 1-Standard Deviation (µg/L)	Conservative ΔTHM4 (µg/L)
TOC 1–2 mg/L	12	14.23 ± 7.34	6.9
TOC 2–3.5 mg/L	89	31.54 ± 24.02	7.5
TOC 3.5–5 mg/L	37	48.55 ± 31.81	16.7
TOC above 5mg/L	7	67.2 ± 18.3	48.9

Abbreviations: EBCT – empty bed contact time; TOC – total organic carbon; THM4 – four regulated trihalomethanes; GAC – granular activated carbon.

Table 6-39: Estimation of ΔTHM4 in Ground Water with a 20 Min EBCT, and a 2-year GAC Replacement Time

Raw Water TOC Bin	Number of Studies in GAC Treatment Dataset	Average ΔTHM4 ± 1-Standard Deviation (µg/L)	Conservative ΔTHM4 (µg/L)
TOC 1–2 mg/L	8	15.14 ± 8.98	6.2
TOC 2–3.5 mg/L	14	22.02 ± 17.48	4.5
TOC 3.5–5 mg/L	7	27.46 ± 8.33	19.1
TOC above 5 mg/L	8	56.66 ± 38.69	18.0

Abbreviations: EBCT – empty bed contact time; TOC – total organic carbon; THM4 – four regulated trihalomethanes; GAC – granular activated carbon.

For the Low (1–2 mg/L), Mid (2–3.5 mg/L), and High-Mid (3.5–5 mg/L) TOC bins, the conservative ΔTHM4 estimates were reasonable compared to the mean concentrations reported into the SYR4 baseline occurrence data. Conservative ΔTHM4 estimates in the High TOC bin (>5 mg/L) were higher due to the greater reduction in TOC. For the THM4 reduction observed in the High TOC bin (>5 mg/L), the conservative THM4 reduction estimates were higher due to the greater reduction in TOC and may not be plausible based on the baseline occurrence information and if it is assumed that all systems are currently in compliance (THM4 <80 µg/L). However, based on SDWIS/Fed violations, not all systems are currently in compliance with the Stage 2 DBP Rule. The EPA assumes that these larger ΔTHM4 estimates would be observed only in the 90th percentile of TOC data. Ground water systems in the High TOC bin may also be mischaracterized during the ICR TSD and should be more accurately described as GWUDI of surface water (Brunke & Gonser, 1997; Chin & Qi, 2000). The GWUDI provisions of the 1989 Surface Water Treatment Rule instituted the concept of ground water that is so closely connected to surface water that public water supply wells should be regulated as surface water rather than as ground water (U.S. EPA, 1989). If one or more ground water systems were mischaracterized, then this could overestimate the ΔTHM4 estimate since these systems make act more like a surface water system in terms of TOC removal.

Since these conservative ΔTHM4 estimates were based on the longest assumed time period for GAC use (i.e., GAC replacement time) in the current regulatory options, the EPA assumes that the estimated ΔTHM4 values are conservative, considering that shorter replacement times would increase the average TOC removal during that operation time.

Since these surface water and ground water systems have already been identified as “challenged” in the ICR TSD (pre-Stage 1 and Stage 2 DBP Rules), this indicates the specific advantages of using GAC to reduce THM4 precursors in comparison to conventional treatment (i.e., coagulation/flocculation followed by sedimentation and filtration). When GAC treatment is used for additional contaminant removal such as PFAS, TOC reduction benefits will also be observed. Since there is a lack of PFAS and TOC co-removal data, the ICR TSD can provide the largest dataset on TOC reduction and THM4 formation changes in drinking water to provide a national estimate of ΔTHM4.

The limitations and uncertainties for using this method to quantify ΔTHM4 due to GAC treatment for PFAS are listed in Section 6.8. One major limitation of using the ICR TSD was that this dataset only used chlorine as a disinfectant and does not capture THM reduction in chloraminating systems. This limitation may lead to an overestimate of THMs formed in systems that used chloramines in the distribution system since THM4 can continue to form within the distribution system and formation tends to be lower when chloramines are used in comparison to free chlorine (Hua & Reckhow, 2008). Most chlorinating systems use free chlorine as a primary disinfectant followed by the addition of ammonia to form chloramines for the secondary disinfectant. Of the 9,838 ground water EPs to distribution systems included in UCMR 3, chlorine disinfection was used 8.8 times more often than chloramine (n = 7,881 for chlorine exclusively and n = 896 for chloramines or both chlorine and chloramines) (U.S. EPA, 2016g). For the 3,179 surface water EPs to distribution systems in UCMR 3, chlorine was used 1.9 times more than chloramine (n = 1,648 for chlorine exclusively and n = 879 for chloramines or both chlorine and chloramines) (U.S. EPA, 2016g).

By assuming the use of free chlorine only, the estimates of ΔTHM4 from pilot/RSSCTs studies may provide an overestimation when factoring in use of both free chlorine and chloramines. Thus, using the conservative free chlorine THM4 formation potential (average ΔTHM4 minus 1-standard deviation) rather than the average ΔTHM4, the EPA attempted to address the overestimation and provide a reasonable national estimate of ΔTHM4.

In a separate DBP formation study under the ICR TSD, individual DBP formation conditions were selected to represent simulated distribution systems for each individual plant that accounted for the disinfectant differences (i.e., chlorine versus chloramine) by using only chlorine as the disinfectant and varying the reaction times. The simulated distribution system studies were not included in the estimated ΔTHM4 provided in this document since including them would have further increased the uncertainty error for systems using chloramine due to the longer reaction times.

6.7.1.3.2 Trihalomethane Reduction Comparison to Fourth Six-Year Review PFAS Plants with GAC Treatment

The EPA compared ΔTHM4 estimates from the ICR TSD to the SYR4 data for PFAS-associated plants that have installed GAC. The objective of this analysis was to compare the ICR TSD modeled predictions of ΔTHM4 to the observed ΔTHM4 concentrations from PWSs that installed GAC for PFAS treatment.

The EPA identified systems that had detectable levels of PFOA and/or PFOS in UCMR 3. Subsequently, the EPA used UCMR 4 data to identify which systems indicated use of GAC treatment. Finally, the EPA used CCRs for all systems that detected PFAS and specified GAC treatment for PFAS to approximate the year that GAC treatment was installed and the purpose for installation. While this approach limited the number of systems available for comparison (n = 7), it allowed the EPA to pinpoint, approximately, which samples were taken before and after GAC installation. The EPA obtained THM4 compliance monitoring data through the SYR4 ICR, based on data collected between 2012 and 2019. The EPA calculated the ΔTHM4 values based on observed THM4 levels before and after GAC installation.

The EPA identified plants using the following criteria (see Table 6-40):

1. Detectable level of PFAS in UCMR 3 (i.e., detections of PFOA and/or PFOS above their respective MRL values).
2. GAC installed as indicated in UCMR 4 and confirmed for PFAS treatment by using CCR information.
3. Ability to identify the year GAC was installed using CCR information.
4. THM4 data available from SYR4 (CCR THM4 data were used as an alternative when SYR4 data were unavailable).

Table 6-40: Selected Distribution Systems from SYR4 Based on Outlined Criteria

PWSID	Source Water Type	Disinfectant Type	Year GAC Began
AL0000577	Surface Water	Free Chlorine[a]	2018
AL0001092	Surface Water	Free Chlorine, Chlorine Dioxide	2016
AZ0407046	Ground Water	Free Chlorine	2017
MI0005370	Surface Water	Free Chlorine	2018
NY3503549	Surface Water	Free Chlorine	2018
OH2903412	Ground Water	Free Chlorine	2017
PA1090069	Ground Water	Free Chlorine	2017

Abbreviations: PWSID – public water system identifier; SYR – Six-Year Review; GAC – granular activated carbon.

Note:

[a] Free chlorine includes gaseous chlorine, offsite generated hypochlorite, or onsite generated hypochlorite.

The EPA chose sampling years to represent conditions before and after GAC treatment based on the following criteria:

- If source water type was surface water, used one year before and one year after the year in which GAC treatment began.
- If source water type was ground water, used two years before and two years after the year in which GAC treatment began. Since ground water plants have fewer samples, this was done to offset the lower sample number. (Note that ground water quality typically has fewer fluctuations than surface water quality, so the EPA expects fewer changes in year-to-year data for ground water systems.)

The EPA extracted and matched sampling point IDs for the years that represent before and after GAC treatment (see Appendix I). Only sampling point IDs with the same number of samples for before and after GAC treatment were used to determine THM4 averages. The seasonality and quantity of samples were considered, and the EPA found that samples were taken consistently and remained at the same frequency throughout the years selected to represent before and after GAC treatment.

The EPA calculated ΔTHM4 concentrations for each system at matched sampling point locations using THM4 data collected before and after GAC installation. The EPA also estimated ΔTHM4 concentrations at the broader plant level by aggregating all THM4 locational sampling data collected before and after GAC installation (see Table 6-41).

Table 6-41: Information on Selected Distribution System and Corresponding ΔTHM4 Values

PWSID	Source Water Type	Disinfectant Type	Sampling Point ID[a]	Average THM4 (Before) (µg/L)	Average THM4 (After) (µg/L)	ΔTHM4 (µg/L)[b]	Average ΔTHM4 (µg/L)[c]
AL0000577	Surface Water	Free Chlorine	12975	16.5	10.9	5.7	9.8
AL0001092	Surface Water	Free Chlorine, Chlorine Dioxide	23592	16.6	6.4	10.2	15.7
AZ0407046	Ground Water	Free Chlorine	33997	28.8	21.6	7.3	4.8
MI0005370	Surface Water	Free Chlorine	CCR	84.9	66.4	18.5	18.5
NY3503549	Surface Water	Free Chlorine	334940	39.1	7.6	31.5	31.5
OH2903412	Ground Water	Free Chlorine	541452	8.9	7.0	1.9	-4.1
PA1090069	Ground Water	Free Chlorine	892902	21.0	21.3	-0.3	-10.7

Abbreviations: THM4 – four regulated trihalomethanes.

Notes:

[a] Sampling point IDs that have a sampling point type of EP were used when available. When unavailable, the first listed sampling point ID was used.

[b] ΔTHM4 = THM4 Average (Before) – THM4 Average (After).

[c] Average delta of pairwise changes in THM4 for each location in the entire distribution system.

Based on available data, the EBCT for the seven plants from SYR4 is unknown. The EPA used TOC values from SYR4 when available and used CCR TOC data as an alternative when TOC data were missing from SYR4. TOC values for SYR4 ground water plants were missing from the SYR4 dataset and corresponding CCRs, and due to this limitation, the EPA did not use raw water TOC bins, but instead used a range of ΔTHM4 values for comparison between SYR4 and ICR TSD.

The EPA compared ΔTHM4 values from the SYR4 to the ICR TSD dataset conservative approach (see Table 6-42). Among SYR4 ground water plants, ΔTHM4 values ranged from -10.7 µg/L to 4.8 µg/L. ICR TSD ground water ΔTHM4 values ranged from 3.5 µg/L to 67.2 µg/L. SYR4 ground water averages were between -7.2 µg/L to 62.4 µg/L lower than ICR TSD surface water averages.

Table 6-42: Comparison Between ICR TSD Conservative ΔTHM4 and SYR4 ΔTHM4 for Surface Water Systems

Raw Water TOC Bin[a]	Surface Water		
	ICR TSD Conservative ΔTHM4 (µg/L)	PWSID	SYR4 ΔTHM4 (µg/L)[b]
TOC 0-1 mg/L	No available data	AL0000577, AL0001092	5.7, 15.7
TOC 1-2 mg/L	6.9	NY3503549	31.5
TOC 2-3.5 mg/L	7.5	MI0005370	18.5
TOC 3.5-5 mg/L	16.7	No available data	No available data
TOC >5 mg/L	48.9	No available data	No available data

Abbreviations: TOC – total organic carbon; THM4 – four regulated trihalomethanes; ICR – information collection rule; TSD – treatment study database.

Notes:

[a] Three of the seven surface water PWSs had no TOC measurements.[b] 20 min EBCT was used to determine best-case and conservative ΔTHM4 values.

Two of the three ground water systems showed increased THM4 formation after the installation of GAC. Possible reasons for increased formation may include source water changes (i.e., increased sediment runoff or spore concentration fluctuations in ground water), operational challenges of the GAC treatment, changes to other treatments within the PWS, or changes in retention time within the distribution system. The four surface water systems had ΔTHM4 values ranging from 5.7 to 31.5 µg/L.

Three out of the seven plants had no available TOC data in SYR4 or CCRs. TOC data for the SYR4 THM4 analysis were only available for surface water plants. SYR4 surface water plants with influent TOC concentrations between 1–2 mg/L had an average ΔTHM4 of 31.5 µg/L compared to the ICR TSD conservative ΔTHM4 estimate of 6.9 µg/L. For SYR4 surface water plants with influent TOC concentrations between 2–3.5 mg/L, the EPA observed an average ΔTHM4 of 18.5 µg/L compared to the ICR TSD conservative ΔTHM4 estimate of 7.5 µg/L. Both comparisons of TOC bins for surface water show that the conservative estimates for THM4 reduction are plausible. Note that this finding is based on a small subset of systems (n = 4) and may not be representative of systems nationally.

Due to lack of TOC data for SYR4 ground water plants, the EPA compared ground water plants to the lowest TOC bin (1–2 mg/L) with ICR TSD data available. SYR4 ground water plants had an average ΔTHM4 between ICR TSD ground water plants with influent TOC concentrations between 1–2 mg/L had an average change in THM4 between -10.7 µg/L to 4.8 µg/L compared to the ICR TSD conservative THM4 reduction estimate of 4.5 µg/L. Limitations on the comparison between the ICR TSD ΔTHM4 estimates and the SYR4 THM changes are described in Section 6.8.

6.7.2 Estimation of Bladder Cancer Risk Reductions

Evaluation of the expected reductions in bladder cancer risk resulting from treatment of PFAS in drinking water involves five steps listed in Section 6.7.1.1. Section 6.7.1.3.2 provides details on the estimation of changes in THM4, while Section 6.7.2.1 provides details on selecting the changes in THM4 specific to the modeled scenarios.[79]

6.7.2.1 Application of Changes in THM4 to PFAS PWSs

The EPA expects PWSs that exceed the PFAS regulatory threshold to consider both treatment and nontreatment options to achieve compliance with the drinking water standard. The EPA assumes that the populations served by systems with EPs expected to install GAC based on the compliance forecast detailed in Section 5.3 will receive the DBP exposure reduction benefits. The EPA notes that other compliance actions included in the compliance forecast could result in DBP exposure reductions, including installation of RO. However, these compliance actions are not included in the DBP benefits analysis because this DBP exposure reduction function is specific to GAC. Switching water sources may or may not result in DBP exposure reductions, therefore the EPA assumed no additional DBP benefits for an estimated percentage of systems that elect this compliance option. Also, the EPA assumed no change in DBP exposure at water systems that install IX, as that treatment technology is not expected to remove a substantial amount of DBP precursors. Finally, the EPA also assumed that PWSs included in this analysis use chlorine only for disinfection and have conventional treatment in place prior to GAC installation.

As described in Section 6.7.1.3, the EPA used the relationship between median raw water TOC levels and changes in THM4 levels estimated in the 1998 DBP ICR to estimate changes in THM4 concentrations in the finished water of PWSs fitted with GAC treatment. The EPA applied changes in THM4 levels to PWS treating for PFAS using the following steps:

1. Identifying the PWSs expected to be triggered into PFAS treatment under various thresholds and the associated PWS populations served by source water type: surface water and ground water;

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[79] The benefits analyses described herein relied on methodology implemented in R software (R Core Team, 2021) and differ slightly from SafeWater MCBC methods. Specifically, SafeWater performs a set of pre-calculations to maximize computational efficiency and, as such, the order of analytical steps across R and SafeWater models differs. However, results across models are mathematically consistent.

2. Estimating the TOC levels associated with each source water type, based on median raw water TOC data collected among non-purchased surface water and ground water systems from the 2019 SYR4 dataset; and
3. Identifying the associated THM4 reduction value based on relationships between raw water TOC levels and changes in THM4 levels estimated in the 1998 DBP ICR.

As shown in the Section 6.7.1.3 tables, the EPA estimated changes in THM4 levels that vary based on the following characteristics:

- **Replacement time:** Assumed to be 730 days;
- **EBCT:** 20 min;
- **Source water type**: Surface Water, Ground Water;
- **THM4 change scenario:** Conservative (mean DBP ICR THM4 reduction minus one standard deviation per TOC bin).

For the DBP risk reduction modeling, the EPA focused on the following treatment scenario (See Table 6-38 and Table 6-39):

- **PWS treatment threshold:** PFOA or PFOS mean concentration exceeds threshold defined by regulatory alternatives;
- **EBCT:** 20 min;
- **Source water type:** Surface Water, Ground Water;
- **THM4 change scenario:** Conservative.

As described in Section 2.2.4, the EPA models a scenario where reduced exposures to THM4 begin in 2029. Therefore, the EPA assumed that the population affected by reduced THM4 levels resulting from implementation of GAC treatment is exposed to baseline THM4 levels prior to actions to comply with the rule (i.e., prior to 2029) and to reduced THM4 levels from 2029 through 2105.

6.7.2.2 *Affected Population*

Information on PWS attributes required for estimating changes in population-level bladder cancer is obtained from the EPA's 2021 Q4 SDWIS/Fed database (U.S. EPA, 2021h). This information includes data on PWS primary sources of water (e.g., whether a PWS relies primarily on ground water or surface water for their source water), operational status, and population served. Some PWSs have multiple EPs delivering drinking water to the distribution network. As discussed in Section 6.7.2.1, the analysis assumes that PWSs will reduce PFAS levels by fitting individual EPs for either GAC or IX treatment and therefore changes in NOM and THM4 will also be specific to EPs.

Rather than modeling individual locations (e.g., PWS), the EPA evaluates changes in bladder cancer cases among the aggregate population per treatment scenario and source water type that is expected to install GAC treatment to reduce PFAS levels. Because of this aggregate modeling approach, the EPA used national-level population estimates to distribute the SDWIS/Fed populations based on single-year age and sex and to extrapolate the age- and sex-specific populations to future years. Section 5.3 describes the decision tree for GAC technology selection. Appendix B provides additional details on estimation of the affected population.

6.7.2.3 *Bladder Cancer Exposure-Response Modeling*

The relationship between exposure to DBPs, specifically trihalomethanes and other halogenated compounds resulting from water chlorination, and bladder cancer has been the subject of multiple epidemiology studies (Cantor et al., 2010; U.S. EPA, 2016g; NTP, 2018c), meta-analyses (Villanueva et al., 2003; Costet et al., 2011), and a pooled analysis (Villanueva et al., 2004). The EPA used the relationship between THM4 levels and bladder cancer in the Villanueva et al. (2004) study to support the benefits analysis for the Stage 2 DBP Rule[80] which specifically aimed to reduce the potential health risks from DBPs (U.S. EPA, 2005b).

Regli et al. (2015) analyzed the potential lifetime bladder cancer risks associated with increased bromide levels in surface source water resulting in increased THM4 levels in finished water.[81] To account for variable levels of uncertainty across the range of THM4 exposures from the pooled analysis of Villanueva et al. (2004), they derived a weighted mean slope factor from the odds ratios reported in Villanueva et al. (2004). They showed that, while the original analysis deviated from linearity, particularly at low concentrations, the overall pooled exposure-response relationship for THM4 could be well-approximated by a linear slope factor that predicted an incremental lifetime cancer risk of 1 in ten thousand exposed individuals (10^{-4}) per 1 µg/L increase in THM4. The linear slope factor developed by Regli et al. (2015) is 0.00427 per 1 µg/L. Using a fixed effects meta-analysis model assumed by Regli et al. (2015), the EPA estimated a 95% confidence interval for the estimated slope of 0.00331–0.00522 per 1 µg/L. This

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[80] See DBP Rule documentation at https://www.epa.gov/dwreginfo/stage-1-and-stage-2-disinfectants-and-disinfection-byproducts-rules

[81] The Regli et al. (2015) slope factor was utilized in the recently peer-reviewed Weisman et al. (2022) study, which estimates that 8,000 of 79,000 US bladder cancer cases are attributable to bladder cancer. Among other things, the authors found that there is a stronger weight of evidence linking DBPs and bladder cancer since the promulgation of the 2006 Stage 2 DBP regulations and even since publication of Regli et al. (2015).

slope enables estimation of the changes in the lifetime bladder cancer risk associated with lifetime exposures to reduced THM4 levels:

Equation 18:

$$Odds(x) = Odds(0) \cdot exp(0.00427 * x)$$

Where $Odds(x)$ are the odds of lifetime bladder cancer incidence for an individual exposed to a lifetime average THM4 concentration in residential water supply of x µg/L and $Odds(0)$ are the odds of lifetime bladder cancer in the absence of exposure to THM4 in residential water supply. The relationship (Equation 18) has the advantage of being independent from the baseline THM4 exposure level, which is highly uncertain for most affected individuals due to lack of historical data.

To enable annual bladder cancer risk estimation, the EPA assumed that the relationship (Equation 18) also holds for the cumulative bladder cancer risk and cumulative average exposure to residential water THM4 from birth to a specific age. A person's cumulative THM4 exposure from drinking water by age a—denoted by x_a—is defined as:

Equation 19:

$$x_a = \frac{1}{a}\sum_{i=0}^{a-1} THM4_i\,, x_0 = 0$$

The EPA estimated the relative risk of bladder cancer by a particular age from a change in average THM4 experienced by this age as follows:

Equation 20:

$$RR(x_a, z_a) = \frac{exp(0.00427 * [x_a - z_a])}{exp(0.00427 * [x_a - z_a]) * LR(z_a) + 1 - LR(z_a)}$$

Where $RR(x_a, z_a)$ is the relative cumulative risk of bladder cancer associated with a change from baseline cumulative exposure z_a to treatment cumulative exposure x_a. This calculation requires an estimate of baseline cumulative bladder cancer risk $LR(z_a)$ which is described in Appendix H.

6.7.2.4 Estimation of Bladder Cancer Risk Reductions

The EPA estimated changes in annual bladder cancer cases and annual mortality in the bladder cancer population due to estimated reductions in lifetime THM4 exposure using a life table-based approach. This approach was used because (1) annual risk of new bladder cancer should be quantified only among those not already experiencing this chronic condition, and (2) bladder cancer has elevated mortality implications.

The EPA used recurrent life table calculations to estimate a water source type-specific time series of bladder cancer incidence for a population cohort characterized by sex, birth year, and age at the beginning of the PFOA/PFOS evaluation period under the baseline scenario and the GAC regulatory alternative described in Section 6.7.2.1. The estimated risk reduction from lower exposure to DBPs in drinking water is calculated based on changes in THM4 levels used as inputs to the Regli et al. (2015)-based health impact function, as shown in Section 6.7.2.3. The life table analysis accounts for the gradual changes in lifetime exposures to THM4 following implementation of GAC treatment under the regulatory alternative compared to the baseline.[82] Details of the life table calculations are provided in Appendix H. The outputs of the life table calculations are the water source type-specific estimates of the annual change in the number of bladder cancer cases and the annual change in bladder cancer population mortality.

Although the change in THM4 exposure likely affects the risk of developing bladder cancer beyond the end of the analysis period (the majority of cancer cases manifest during the latter half of the average individual life span; Hrudey et al., 2015), the EPA does not capture effects after the end of the period of analysis, 2105. Individuals alive after the end of the period of analysis likely benefit from lower lifetime exposure to THM4. Lifetime health risk model data sources include; the SDWIS/Fed; age- and sex-specific population estimates from the U.S. Census Bureau (U.S. Census Bureau, 2020a); the SEER program database (National Cancer Institute),[83] and the CDC National Center for Health Statistics.[84] Appendix H provides additional detail on the data sources and information used in this analysis as well as baseline bladder cancer statistics. Appendix B provides additional details on the estimation of the affected population.

6.7.2.5 Valuation of Bladder Cancer Risk Reductions

The EPA uses the Value of Statistical Life to estimate the benefits of reducing mortality associated with bladder cancer in the affected population. Section 2.2 provides information on updating Value of Statistical Life for inflation and income growth. The EPA uses COI-based valuation to estimate the benefits of reducing morbidity associated with bladder cancer. Specifically, the EPA used bladder cancer treatment-related medical care and opportunity cost[85] estimates from Greco et al. (2019). Table 6-43 shows the original COI estimates from Greco et al. (2019) which were reported in $2010, along with the values updated to $2022 used in this analysis. The EPA further notes that the estimates for non-invasive bladder cancer subtype were used to value local, regional, and unstaged bladder cancer morbidity reductions, while the estimates for the invasive bladder cancer subtype were used to value distant bladder cancer morbidity reductions.[86]

The EPA received public comments on the economic analysis for the proposed rule related to the EPA's use of cost of illness information for morbidity valuation. Specifically, a couple commenters recommended that the EPA use willingness to pay information (instead of cost of illness information) when valuing the costs associated with non-fatal illnesses, stating that

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[82] As described above, the EPA models THM4 changes under the treatment scenario as being in effect for the years 2024 through 2105, with nonzero THM4 changes first occurring in 2029, the year when all PWS are assumed to comply with PFAS treatment requirements.

[83] For cancer incidence and stage distribution data, the EPA relies on SEER 21 (2009-2018); for cancer survival data, the EPA relies on SEER 18 (2000-2017).

[84] CDC Wonder data on 1999-2019 all-cause and bladder cancer mortality by age and sex.

[85] Opportunity (or indirect) costs modeled by this study were represented by the value of time needed to undergo the cancer treatment, which could otherwise have been dedicated to work or leisure activities.

[86] Local cancer is a malignant cancer confined entirely to the organ where the cancer began. Remote cancer refers to cancer that has grown beyond the original (primary) tumor to nearby lymph nodes or organs and tissues. Distant cancer refers to cancer that has spread from the original (primary) tumor to distant organs or distant lymph nodes; it is also called a distant metastasis. Unstaged cancer is a cancer whose subtype is unknown.

willingness to pay information better accounts for lost opportunity costs (e.g., lost productivity and pain and suffering) associated with non-fatal illnesses. To better account for these opportunity costs, the EPA used recently available willingness to pay values in a sensitivity analysis for morbidity associated with bladder cancer. The sensitivity analysis results show that when willingness to pay values are used in bladder cancer benefits analysis, morbidity benefits are increased by 19.9 percent. See Appendix O for full details and results on the willingness to pay sensitivity analyses.

Table 6-43: Bladder Cancer Morbidity Valuation

Bladder Cancer Subtype[a]	Type of Cost	Cost in First Year ($2010)[b]	Cost in Subsequent Years ($2010)[b]	Cost in First Year ($2022)[c]	Cost in Subsequent Years ($2022)[c]
Non-invasive	Medical care	$9,133	$916	$12,851	$1,289
	Opportunity cost	$4,572	$24	$6,212	$33
	Total cost	$13,705	$941	$19,062	$1,321
Invasive	Medical care	$26,951	$2,455	$37,922	$3,454
	Opportunity cost	$10,513	$77	$14,283	$105
	Total cost	$37,463	$2,532	$52,205	$3,559

Notes:

[a] The estimates for non-invasive bladder cancer subtype were used to value local, regional, and unstaged bladder cancer morbidity reductions, while the estimates for the invasive bladder cancer subtype were used to value distant bladder cancer morbidity reductions.

[b] The estimates come from Greco et al. (2019).

[c] To adjust for inflation, the EPA used the U.S. Bureau of Labor Statistics Consumer Price Index for All Urban Consumers: Medical Care Services in U.S. (City Average).

6.7.3 Results

Table 6-44 to Table 6-47 provide the health effects avoided and valuation associated with bladder cancer.

Table 6-44: National Bladder Cancer Benefits, Final Rule (PFOA and PFOS MCLs of 4.0 ppt each, PFHxS, PFNA, and HFPO-DA MCLs of 10 ppt each and HI of 1) (Million $2022)

Benefits Category	2% Discount Rate		
	5th Percentile[a]	**Expected Value**	**95th Percentile[a]**
Number of Non-Fatal Bladder Cancer Cases Avoided	5,781.0	7,313.0	8,912.7
Number of Bladder Cancer-Related Deaths Avoided	2,029.6	2,567.8	3,129.9
Total Annualized Bladder Cancer Benefits (Million $2022)[b,c]	**$300.64**	**$380.41**	**$463.74**

Notes: See Appendix P for results presented at 3 and 7 percent discount rates. Quantifiable benefits are increased under final rule table results relative to the other options presented because of modeled PFHxS occurrence, which results in additional quantified benefits from co-removed PFOA and PFOS.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.

[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

[c] When using willingness to pay metrics to monetize morbidity benefits, total annualized bladder cancer benefits are increased by $75.87 million (see Appendix O).

Table 6-45: National Bladder Cancer Benefits, Option 1a (PFOA and PFOS MCLs of 4.0 ppt)

Benefits Category	2% Discount Rate		
	5th Percentile[a]	**Expected Value**	**95th Percentile[a]**
Number of Non-Fatal Bladder Cancer Cases Avoided	5,789.3	7,312.9	8,896.0
Number of Bladder Cancer-Related Deaths Avoided	2,032.5	2,567.8	3,123.2
Total Annualized Bladder Cancer Benefits (Million $2022)[b]	**$301.06**	**$380.41**	**$462.73**

Notes: See Appendix P for results presented at 3 and 7 percent discount rates.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.

[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

Table 6-46: National Bladder Cancer Benefits, Option 1b (PFOA and PFOS MCLs of 5.0 ppt)

Benefits Category	2% Discount Rate		
	5th Percentile[a]	Expected Value	95th Percentile[a]
Number of Non-Fatal Bladder Cancer Cases Avoided	4,739.4	6,034.0	7,367.1
Number of Bladder Cancer-Related Deaths Avoided	1,664.0	2,118.7	2,587.1
Total Annualized Bladder Cancer Benefits (Million $2022)[b]	**$246.48**	**$313.88**	**$383.32**

Notes: See Appendix P for results presented at 3 and 7 percent discount rates.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.

[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized benefits in this table.

Table 6-47: National Bladder Cancer Benefits, Option 1c (PFOA and PFOS MCLs of 10.0 ppt)

Benefits Category	2% Discount Rate		
	5th Percentile[a]	Expected Value	95th Percentile[a]
Number of Non-Fatal Bladder Cancer Cases Avoided	2,326.9	3,087.9	3,885.3
Number of Bladder Cancer-Related Deaths Avoided	816.8	1,084.3	1,364.3
Total Annualized Bladder Cancer Benefits (Million $2022)[b]	**$120.97**	**$160.62**	**$202.14**

Notes: See Appendix P for results presented at 3 and 7 percent discount rates.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty. This range does not include the uncertainty described in Table 6-48.

[b] See Table 7-6 for a list of the nonquantifiable benefits, and the potential direction of impact these benefits would have on the estimated monetized total annualized costs in this table.

6.8 Limitations and Uncertainties of the Benefits Analysis

This section describes limitations of the quantified benefits analysis, along with uncertainties that could not be modeled quantitatively as part of the national benefits analysis. The sources of uncertainty characterized quantitatively are presented in Section 6.1.2. In the tables below, the EPA summarizes limitations and uncertainties that apply to:

- All quantitative benefits analyses implemented for the final PFAS rule (Table 6-48);
- Application of PK models for blood serum PFAS concentration estimation (Table 6-49);
- Developmental effects (i.e., infant birth weight) modeling (Table 6-50);

- CVD impacts modeling (Table 6-51);
- RCC impacts modeling (Table 6-52); and
- Modeling of bladder cancer impacts from GAC treatment related THM4 reductions (Table 6-53).

The EPA notes that in most cases it is not possible to judge the extent to which a particular limitation or uncertainty could affect the magnitude of the estimated benefits. Therefore, in each table below, the EPA notes the potential direction of the impact on the quantified benefits (e.g., a source of uncertainty that tends to underestimate quantified benefits indicates expectation for larger quantified benefits) but does not prioritize the entries with respect to the impact magnitude.

Table 6-48: Limitations and Uncertainties that Apply to Benefits Analyses Considered for the Final PFAS Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
The EPA has quantified benefits for three health endpoints for PFOA (birth weight, CVD, and RCC) and two health endpoints for PFOS (birth weight and CVD).	Underestimate	For various reasons, the EPA has not quantified the benefit of removing PFOA and PFOS from drinking water for most of the health endpoints PFOA and PFOS are expected to impact. See discussion in Section 6.2.2 for more information about these nonquantifiable benefits.
The EPA has quantified benefits for one co-removed contaminant group (THM4).	Underestimate	Treatment technologies that remove PFAS can also remove numerous other contaminants, including some other PFAS compounds, additional regulated and unregulated DBPs, heavy metals, organic contaminants, pesticides, among others. These co-removal benefits may be significant, depending on co-occurrence, how many facilities install treatment and which treatment option they select.
The EPA has not quantified national benefits for any health endpoint for the PFAS that make up the Hazard Index (PFHxS, PFNA, PFBS, and HFPO-DA).	Underestimate	PFHxS, PFNA, PFBS, and HFPO-DA each have substantial health impacts on multiple health endpoints. However, the effects of PFNA on birth weight are evaluated as part of a sensitivity analysis in Appendix K.
The analysis does not explicitly consider changes in PFOA/PFOS and THM4 concentrations for systems that purchase their drinking water from other PWSs.	Uncertain	Many PWSs purchase their primary source water from PWSs that are likely to implement treatment under the rule. The SDWIS/Fed inventory of PWSs includes these systems with their retail populations instead of allocating those populations to the wholesale systems. The MCMC occurrence analysis outputs for the wholesale system and purchasing system may vary from one another, resulting in either an under- or over-estimate of affected population in any iteration. The net effect on total benefits is uncertain.
The analysis does not account for populations that consume bottled water as their primary drinking water source.	Uncertain	Studies indicate that between 13 percent and 33 percent of the U.S. population consumes bottled water as their primary drinking water source (Z. Hu et al., 2011; Rosinger et al., 2018; Vieux et al., 2020). The benefits models do not consider these populations. This could result

Table 6-48: Limitations and Uncertainties that Apply to Benefits Analyses Considered for the Final PFAS Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
		in an overestimate of avoided cases of health effects and associated benefits. However, bottled water consumers can also be CWS customers and may still be exposed to PFAS by using water for cooking etc., and therefore, would benefit from PFAS removal. (U.S. FDA, 2022; Aquafina, 2022). Finally, the benefits may also be underestimated because those using bottled water as a primary drinking water source may switch to CWS supply as a result of the final NPDWR; the EPA did not model this behavioral response and hence the benefits do not account for the potential cost savings to those consuming bottled water at baseline.
The analysis considers PFOA/PFOS concentrations from NTNCWSs.	Overestimate	SDWIS/Fed population served estimates for NTNCWSs represent both the population that has regular exposure to the NTNCWS' drinking water (e.g., the employees at a location) and the peak day transient population (e.g., customers) who have infrequent exposure to the NTNCWS' drinking water. Estimating the demographic distribution and the share of daily drinking water consumption for these two types of NTNCWS populations would be difficult across many of the industries which operate NTNCWSs. The inclusion of NTNCWS results is an overestimate of benefits because daily drinking water consumption for these populations is also modeled at their residential CWS.
The EPA assumes that the effects of PFOA and PFOS exposures are independent.	Uncertain	The exposure-response functions used in benefits analyses assume that the effects of serum PFOA/PFOS on the health outcomes considered are independent and therefore additive. This assumption is consistent with the *Framework for Estimating Noncancer Health Risks Associated with Mixtures of Per- and Polyfluoroalkyl Substances (PFAS)* (U.S. EPA, 2024d). Due to limited evidence, the EPA does not consider synergies or antagonisms in PFOA/PFOS exposure-response.
The derivation of PFOA/PFOS exposure-response functions for the relationship between PFOA/PFOS serum and associated health outcomes assumes that there are no threshold serum concentrations below which effects do not occur.	Overestimate	The EPA's *Final Human Health Toxicity Assessments* indicate that the levels at which adverse health effects could occur are much lower than previously understood when the EPA issued the 2016 health advisories for PFOA and PFOS (70 ppt) – including near zero for certain health effects. Therefore, the exposure-response functions used in benefits analyses assume that there are no threshold serum concentrations below which effects do not occur. This could result in a slight overestimate of benefits for noncancer health endpoints.
Causality is assumed for all health effects for which exposure-response functions are used to estimate risk.	Overestimate	Analyses evaluating the evidence on the associations between PFAS exposure and health outcomes are ongoing and the EPA has not conclusively determined causality. As described in Section 6.2, the EPA modeled health risks from PFOA/PFOS exposure for endpoints for which the evidence of association was found to be likely. These

Table 6-48: Limitations and Uncertainties that Apply to Benefits Analyses Considered for the Final PFAS Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
		endpoints include birth weight, TC, and RCC. While the evidence supporting causality between DBP exposure and bladder cancer has increased since the EPA's Stage 2 DBP Rule (NTP, 2021; Weisman et al., 2022), causality has not yet been conclusively determined (Regli et al., 2015).
The analysis assumes that quantified benefits categories are additive.	Uncertain	The EPA did not model birth weight, CVD, RCC, and bladder cancer benefits jointly, in a competing risk framework. Therefore, reductions in health risk in a specific benefits category do not influence health risk reductions in another benefits category. For example, lower risk of CVD and associated mortality implies a larger population that could benefit from cancer risk reductions, because cancer incidence grows considerably later in life (see Tables G-3 through G-6 in Appendix G).
The scope of the analysis does not include intra- or international migration throughout the evaluation period.	Uncertain	Throughout the analysis period people may migrate from one place to another. If persons migrate to locations with larger decreases in PFOA/PFOS under the regulatory alternative, the EPA would be underestimating the impacts. The opposite is true if persons migrate to locations with smaller decreases in PFOA/PFOS under the regulatory alternative.
The analysis does not take into account population growth and other changes in long-term trends.	Underestimate	The benefits analysis does not reflect the effects of growing population that may benefit from reduction in PFOA/PFOS exposure, which is expected to result in underestimated benefits. The EPA uses present-day information on life expectancy, disease, environmental exposure, and other factors, which are likely to change in the future.
The analysis does not include the impacts of COVID-19 on future population health and economic growth.	Uncertain	Impacts of the COVID-19 pandemic have had resulting effects on conception, pregnancy, and birth rates (Aassve et al., 2021; McLaren Jr et al., 2021; Ullah et al., 2020). Some studies suggest that the economic recession caused by the COVID-19 pandemic may impose long-term impacts on fertility rates (McLaren Jr et al., 2021; Ullah et al., 2020). Such impacts are not accounted for in this benefits analysis.
For PWSs with multiple EPs, the analysis assumes a uniform population distribution across the EPs.	Uncertain	Data on the populations served by each EP are not available and the EPA therefore uniformly distributes system population across EPs. Effects of the regulatory alternative may be greater or smaller than estimated, depending on actual populations served by affected EPs. For one large system serving more than one million customers the EPA has sufficient data on EP flow to proportionally assign effected populations.
Valuation of mortality risk reductions assumes that per capita income will grow at the constant rate.	Uncertain	The EPA uses Value of Statistical Life adjusted for income growth to estimate economic value of the premature mortality avoided in the future. Per capita income growth projections were available through 2050. The EPA estimated the compound annual growth rate in per capita

Table 6-48: Limitations and Uncertainties that Apply to Benefits Analyses Considered for the Final PFAS Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
		income during 2023-2050 and applied it to project Value of Statistical Life over the analysis period 2024-2105.
The EPA does not characterize uncertainty associated with the Value of Statistical Life reference value or Value of Statistical Life elasticity.	Uncertain	The EPA did not quantitatively characterize the uncertainty for the Value of Statistical Life reference value and income elasticity. Because the economic value of avoided premature mortality comprises the majority of the overall benefits estimate, not considering uncertainty surrounding the Value of Statistical Life is a limitation.
Process wastes are not classified as hazardous.	Underestimate	The national economic analysis reflects the assumption that PFAS-contaminated wastes are not considered RCRA regulatory or characteristic hazardous wastes. The EPA acknowledges that if Federal authorities later determine that PFAS-contaminated wastes require handling as hazardous wastes, there will be additional benefits to public health and the environment from reduced exposures to PFAS that have not been quantified as part of this analysis.

Abbreviations: COVID-19 – coronavirus disease 2019; CVD – cardiovascular disease; CWS – community water system; DBP – disinfection byproduct; MCLG - maximum contaminant level goal; PFOA – perfluorooctanoic acid; PFOS – perfluorooctane sulfonic acid; PWS – public water system; RCC – renal cell carcinoma; RO – reverse osmosis; UCMR – unregulated contaminant monitoring rule.

Table 6-49: Limitations and Uncertainties in the PK Model Application

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
The benefits analysis assumes that there are no reductions in PFOA/PFOS exposure from other sources associated with treatment-related reductions in PFOA/PFOS drinking water concentrations.	Underestimate	Some portion of the non-drinking water PFOA/PFOS exposure could be related to drinking water concentration (e.g., food affected by water contamination). This portion is difficult to estimate, and, depending on the relationship, there may be a time lag between the decrease in drinking water concentration and the decrease in the non-drinking water exposure.
The birth weight analysis uses the adult PK model to estimate changes in female serum PFOA/PFOS from changes in drinking water PFOA/PFOS.	Overestimate	Evidence from epidemiology studies connects birth weight to serum PFOA/PFOS levels throughout pregnancy: The serum PFOS-birth weight slope factor in the birth weight benefits module comes from the meta-analysis of 29 studies by Dzierlenga et al. (2020). Table 1 in Dzierlenga et al. (2020) summarizes the timing of the serum samples for the contributing studies, including pre-pregnancy (2 studies), first trimester (6 studies), second trimester (5 studies), third trimester (5 studies), and cord blood samples/delivery (11 studies).[a] The serum PFOA-birth weight slope factor comes from the meta-analysis of 24 studies by Steenland et al. (2018). Steenland et al. (2018) summarizes the timing of the serum samples for the contributing studies, including pre-pregnancy (2 studies), first trimester (4 studies), straddling first and second trimester (1 study), second trimester (2 studies), straddling second and third trimester (2 studies), third trimester (4 studies), and cord blood samples/delivery (9 studies).[b] Because the slope factors included epidemiological evidence throughout pregnancy, a developmental version of the PK model may be a more appropriate choice. A developmental PK model would allow the observed decrease in serum levels that occurs during pregnancy to be captured by accounting for maternal physiological changes. For example, Glynn et al. (2012) found a mean decrease of 16 percent for PFOA and 11 percent for PFOS between serial measurements taken in the 1st trimester and 3rd trimester of pregnancy. This decrease is associated with increases in maternal plasma volume and transfer of the chemicals to the placenta and fetus. The EPA expects that the use of the adult PK model overestimates the additive difference in serum concentrations between baseline and regulatory alternative (and, therefore, the birth weight benefits of the regulatory alternative) because of the expected larger volume of distribution for pregnant females and, therefore, proportionally lower serum concentrations.

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctane sulfonic acid; PK – pharmacokinetic.
Notes:

[a] For PFOS, the EPA used 4 high confidence studies (Chu et al., 2020; Sagiv et al., 2018; Starling et al., 2017; and Wikström et al., 2019) with a variety of PFOS exposure measures across the fetal and neonatal window. Sagiv et al. (2018) collected maternal samples in trimester 1, while Wikström et al. (2020) collected them in trimesters 1 and 2. The samples from Starling et al. (2017) were from trimesters 2 and 3, while Chu et al. (2020) collected exclusively in trimester 3. Of these studies, only Sagiv et al. (2018) and Starling et al. (2017) were part of the Dzierlenga et al. (2020) meta-analysis.

[b] For PFOA, the EPA used 5 high confidence studies (Chu et al., 2020; Govarts et al., 2016; Sagiv et al., 2018; Starling et al., 2017; and Wikström et al., 2020) with a variety of PFOA exposure measures across the fetal and neonatal window. Sagiv et al. (2018) collected maternal samples in trimester 1, while Wikström et al. (2020) collected them in trimesters 1 and 2. The samples from Starling et al. (2017) were from trimesters 2 and 3, while Chu et al. (2020) collected exclusively in trimester 3. The samples in the Govarts et al. (2016) study were collected from umbilical cords. None of these studies were part of the Negri et al. (2017) meta-analysis.

Table 6-50: Limitations and Uncertainties in the Analysis of Birth Weight Benefits Under the Final Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
Characterizing the Exposed Population		
The analysis does not consider the effects of PFOA/PFOS exposure on fertility rates.	Uncertain	Studies have shown that exposure to PFAS may lead to reduced fertility rates among women (Fei et al., 2009; Waterfield et al., 2020), while the evidence supporting PFAS effects on the male reproductive system is inconclusive (Cathrine Carlsen Bach et al., 2016; U.S. EPA, 2024e; U.S. EPA, 2024f). The birth weight risk reduction analysis does not account for any potential differences in birth rates among the baseline and treatment scenario due to PFAS-related changes in fertility.
The EPA uses state-specific birth rate data, distributed based on census region-level race/ethnicity-specific birth rates, to determine the share of infants born to women of childbearing age at each PWS and within each 100 g birth weight increment.	Uncertain	County-level birth rates from CDC by 100 g birth weight increment are often tagged as "unreliable" by CDC in cases where there are low infant counts per birth weight increment. State-specific 100 g increment-specific birth rates may not reflect the number of infants born in each 100 g birth weight increment in PWS service area that is affected by PFOA/PFOS through the pregnant mother's ingestion of drinking water. Using state-specific birth rates may over- or underestimate the number of infants falling into each 100 g birth weight increment born to mothers who experience PWS specific changes in drinking water PFOA/PFOS levels. This in turn may over- or underestimate benefits associated with changes in PFOA/PFOS levels.
The EPA uses state-specific death rate data, distributed based on national-level race/ethnicity-specific infant mortality rates, as the baseline infant mortality rate (i.e., number of deaths per 1,000 births)	Uncertain	State-specific death rates may not reflect the baseline number of infants who die in each PWS that is affected by PFOA/PFOS in mother's drinking water. Using state-specific baseline death rates may over- or underestimate the post-regulation death rates determined using the birth weight-mortality relationship and changes in birth weight, and result in an over- or underestimate of benefits associated with changes in PFOA/PFOS levels.

Table 6-50: Limitations and Uncertainties in the Analysis of Birth Weight Benefits Under the Final Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
of infants born to women of childbearing age at each PWS.		
Baseline infant death rates per location are held constant throughout the years of the analysis.	Uncertain	Although changes in infant death rates may not be consistent across race/ethnicity and location in the US, medical advances in infant care will likely reduce infant mortality in future years.
The EPA uses county-specific percentages of the population that fall within four race/ethnicity categories (non-Hispanic White, non-Hispanic Black, Hispanic, and other) to separate total PWS-specific populations into race categories for application of the birth weight-mortality marginal effects estimates.	Uncertain	County-specific population percentages may not accurately represent the race/ethnicity makeup of PWS-level populations served. PWS populations served may span multiple counties or may represent a portion of a single county.
Modeling Changes in Health Risks		
The analysis does not model variability in pregnancy stage-specific serum PFOA/PFOS concentrations and exposure-response relationships.	Overestimate	The studies estimating the link between maternal serum PFOA/PFOS and infant birth weight use serum PFOA/PFOS measurements from various stages of pregnancy. The EPA used a constant, adult PK model-based estimate of serum PFOA/PFOS concentration to represent exposure during pregnancy, which is more consistent with early pregnancy exposures and likely overestimates the reduction in serum PFOA/PFOS exposure later in pregnancy. In a sensitivity analysis (Appendix K), the EPA estimated birth weight benefits using exposure-response functions that evaluated the association between early pregnancy serum PFOA/PFOS and birth weight. The EPA found that using an early pregnancy-based exposure-response function would result in approximately a 60 percent reduction in birth weight benefits.

Table 6-50: Limitations and Uncertainties in the Analysis of Birth Weight Benefits Under the Final Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
The analysis assumes that birth weight changes resulting from changes in PFAS serum levels will not exceed 200 g.	Underestimate	The EPA places a cap on estimated birth weight changes in excess of 200 g based on existing studies that found that changes to environmental exposures result in relatively modest birth weight changes (Windham & Fenster, 2008; Klein & Lynch, 2018; Kamai et al., 2019). Under the final rule, this birth weight threshold is exceeded in only 0.01 percent of affected infants.
Economic Valuation of Changes in Health Risk		
Some possible benefits from increased birth weight in infants are omitted from the analysis.	Underestimate	Omitted benefit categories include reduction in IQ loss, special education costs, early intervention costs, and labor market productivity losses associated with specific developmental diseases, among others (National Academies of Sciences, 2023). The EPA's analysis omitted these categories because the available studies documenting relationships between birth weight and non-medical effects either did not identify methods for determining the associated economic burden of such effects or had other limitations such as older (pre-2000s) data, limited geographical coverage, small sample sizes, small ranges of birth weight evaluated, performed outside of the U.S., or lack of statistical significance. See ICF (2021) for additional details.
The analysis does not monetize medical treatment costs for infants who die within 1 year of birth.	Underestimate	This limitation likely results in an underestimate of total benefits. The magnitude of this underestimate is likely to be small because the number of infants who do not survive represent a small percentage of the total number of LBW infants. In addition, the medical cost function is based on estimated treatment expenses over a two-year period after birth and thus the EPA would have to scale down medical costs to account for the distribution of infant death timing within 1-year (e.g., within 28 days of birth or 3 months). Based on the 2016-2018 NCHS/NVSS data, approximately 50 percent of LBW infant deaths occur within the first 28 days of birth. Thus, it is likely that only a small portion of medical costs from Klein et al. (2018) is applicable to infants who die within 1 year of birth.
Simulated medical cost changes from Klein and Lynch (2018) do not reflect	Uncertain	Preliminary modeling indicates that reductions in PFOA/PFOS concentrations based on the regulatory alternatives may lead to birth weight changes greater

Table 6-50: Limitations and Uncertainties in the Analysis of Birth Weight Benefits Under the Final Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
birth weight changes greater than 100 g.		than 100 g. Although the EPA caps birth weight change estimates at 200 g, the EPA uses the COI estimates associated with a 100 g change in birth weight for all birth weight changes between 100 and 200 g to avoid extrapolation outside of the data range.

Abbreviations: CDC – Centers for Disease Control and Prevention; COI – cost of illness; g – gram; LBW – low birth weight; NCHS – National Center for Health Statistics; NTNCWS - non-transient non-community water system; NVSS – National Vital Statistics System; PFAS – per-and polyfluoroalkyl substances; PFOA – perfluorooctanoic acid; PFOS – perfluorooctane sulfonic acid; PWS – public water system; SDWIS/Fed - Safe Drinking Water Information System Federal Version.

Table 6-51: Limitations and Uncertainties in the Analysis of CVD Benefits Under the Final Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
Characterizing the Exposed Population		
The analysis uses national-level estimates of CVD prevalence and incidence rates, life tables, and ASCVD model inputs (e.g., prevalence of treated and untreated hypertension, diabetes, smoking).	Uncertain	Using national-level baseline health data may over- or underestimate the effects of regulatory alternatives on CVD morbidity and mortality overall and in specific PWSs.
The effects of statin use on changes in CVD risk were not modeled in this analysis.	Uncertain	Because statin medications lower LDLC, statin use may impact the relationship between serum PFOA/PFOS levels and TC and, ultimately, the estimated changes in CVD risk. The EPA did not model population variability with respect to this factor for two reasons. First, as described in Appendix F, not all studies modeling serum PFOA/PFOS levels and TC consider and/or control for statin use. Exclusion of persons who rely on statins for LDLC control from the modeled population would underestimate CVD benefits if serum PFOA/PFOS-TC effect represents an average across statin user and non-user groups. Second, there are challenges in estimating statin use prevalence. Depending on age, sex, race/ethnicity, and disease status, approximately 20 percent-40 percent of the U.S. population relies on statins (Robinson & Booth, 2010). Factors such as overt CVD, healthcare, and demographics are significantly associated with statin use (Leino et al., 2020; Electricwala et al., 2020). While statin therapy is intended to be permanent, many individuals who are prescribed statins take them irregularly (Colantonio, 2019; Lewey et al., 2013; Ellis et al., 2004; Goldstein et al., 2016; Toth et al., 2019); Toth

Table 6-51: Limitations and Uncertainties in the Analysis of CVD Benefits Under the Final Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
		et al. (2019) found a <25 percent rate of adherence 5 years after initiation of therapy.
Modeling Changes in Health Risks		
The analysis assumes that there is no lag between changes in serum PFOA/PFOS concentrations and changes in TC and BP. Likewise, the analysis assumes that there is no lag between changes in TC/BP and changes in CVD risk.	Overestimate	The studies estimating the link between serum PFOA/PFOS and TC/BP and the ASCVD model are not dynamic, and hence do not provide insights into whether TC/BP may respond gradually to changes in serum PFOA/PFOS and/or if CVD risk may respond gradually to changes in TC/BP. The analysis assumes immediate adjustment, which may overestimate impacts to the exposed population. Note, however, that reductions in TC/BP and CVD risk do not instantaneously follow the reductions in PFOA/PFOS drinking water concentrations, because the reductions in serum PFOA/PFOS are gradual, as predicted by the PK model.
The derivation of PFOA/PFOS exposure-response functions for the relationship between PFOA/PFOS serum and TC levels assumes that the studies used in the meta-analysis represent the PFOA/PFOS effects on serum TC levels in general population adults.	Uncertain	The exposure-response function was developed based on six general population studies reporting linear serum PFAS-TC level associations. Four of these studies were high quality as reflected by the lower risk of bias evaluations. These studies may not capture all possible relationships between PFOA/PFOS and serum TC levels.
The analysis excludes exposure-response relationships between serum PFOA/PFOS and HDLC.	Uncertain	The relationship between serum PFOA/PFOS and HDLC is uncertain. As shown in Section 6.5.2 and Appendix F, the meta-analysis-based estimate of the effect of serum PFOA/PFOS on HDLC concentration is positive but not statistically significant. Single-study analyses of this relationship have generated both positive (Dong et al. (2019) serum PFOS-HDLC relationship) and inverse (Dong et al. (2019) serum PFOA-HDLC relationship, Lin et al. (2019) serum PFOA-HDLC and serum PFOS-HDLC relationship) effect estimates that were not statistically significant. To better understand the impact of incorporating HDLC in the CVD risk model, the EPA has implemented a sensitivity analysis (see details in Appendix K). The EPA found that, using the meta-analysis results, inclusion of HDLC would decrease benefits by approximately 23-25%.
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	Uncertain	While the CVD risk impacts of changes in TC/BP from behavioral and medical interventions is well documented (Lloyd-Jones et al., 2017), there is no information on whether changes in serum PFOS/PFOA leading to changes in these biomarkers would result in similar outcomes.

Table 6-51: Limitations and Uncertainties in the Analysis of CVD Benefits Under the Final Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
biomarkers due to other reasons such as behavioral changes or medication.		
The CVD risk analysis assumes that person’s TC/BP level history does not have an impact on changes in CVD risk due to changes in the levels of these biomarkers.	Uncertain	The ASCVD model links TC/BP levels at the start of the 10-year follow-up period to first hard CVD event incidence during the follow-up period. The modeling does not account for TC/BP changes over time, which could have an impact on the CVD event risk.
The ASCVD model was not recalibrated for the contemporary CVD incidence and prevalence.	Overestimate	Assessments of ASCVD risk model performance across different sociodemographic subgroups (Asian populations, Hispanic populations, persons with high levels of CVD risk, diabetes, older adults with frailty and multimorbidity, smokers, and women) indicated that the model tended to overestimate risk but suggested that the model may improve through additional input variables and recalibration given contemporary CVD incidence and prevalence (Mora et al., 2018; Muntner et al., 2014).
The analysis uses the ASCVD model developed for non-Hispanic Black populations to assess potential CVD risks for race/ethnicity groups other than non-Hispanic Black and non-Hispanic White populations.	Uncertain	The ASCVD model documentation encourages the use of equations for non-Hispanic White populations for other race/ethnicity categories, specifying that estimated risks may be biased upward, especially for Hispanic and Asian American populations. The EPA’s model validation analysis detailed in Appendix G shows that the non-Hispanic Black model is a better fit for these race/ethnicity groups. However, the ultimate impact of this assumption is uncertain.
The EPA uses the fraction of the population who smokes and has diabetes as inputs into the ASCVD model.	Underestimate	The ASCVD model uses binary values to indicate whether a person is a current smoker or has diabetes. The EPA simplifies calculations by using the fraction of the population who smokes and has diabetes as inputs to the ASCVD model. The EPA has implemented a targeted evaluation of the effect of this assumption and confirmed that this simplification likely underestimates impacts by approximately 5 percent to 10 percent, depending on the age group, due to the non-linearity of the estimated model.
The analysis assumes that the threshold for high BP is a systolic/diastolic measurement of 140/90.	Underestimate	In November 2017, the threshold defined for high BP was reduced to 130/80 (Whelton et al., 2018). The analysis relies on high BP prevalence data and treated, untreated, and normal BP measurements that are based on NHANES surveys from 2011 to 2016. Therefore, the EPA adheres to the pre-2017 threshold. Furthermore, the ASCVD model was developed prior to the change in high BP definition. Adhering to the pre-2017 threshold may affect the number of people sorted into the high BP population category, potentially underestimating CVD risk.

Table 6-51: Limitations and Uncertainties in the Analysis of CVD Benefits Under the Final Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
The analysis assumes independence among the prevalence of high BP, smoking, and diabetes.	Overestimate	Smoking and high BP are often related, and smoking is a risk factor for Type 2 diabetes. Assuming independence among the prevalence of high BP, smoking, and diabetes may result in overestimated CVD risk impacts.
The analysis assumes that deaths from causes other than hard CVD events occur first.	Underestimate	By assuming that deaths from causes other than hard CVD events occur first, the EPA underestimates the eligible population (e.g., population without CVD history) evaluated for the first hard CVD event estimation.
The analysis does not account for survivors of first hard CVD events that are neither MI nor IS. The analysis does not account for persons who were younger than 40 years at the time of their first hard CVD event.	Underestimate	The ASCVD model captures risk of non-fatal MI, non-fatal IS, and fatal CVD; however, it does not capture other non-fatal CHD. The ASCVD model can be used to predict the annual probability of a first hard CVD event for persons aged 40–89 years; the EPA applied this model to populations aged 40 years and older. The prevalence of CVD history before age 40 is low (<7% based on estimates from the Medical Expenditure Panel Survey) and likely includes persons whose CVD arises from genetic factors (Zhang et al., 2019). Early life PFAS exposures and TC are inconclusively associated for PFOA and positively associated for PFOS (U.S. EPA, 2024e; U.S. EPA, 2024f). TC later in life is highly positively correlated with early TC as seen in Pletcher et al. (2016) and Zhang et al. (2019). This analysis does not directly capture effects of early life increases in TC due to PFAS exposures. The analysis does capture the effects of early life TC indirectly to the extent that early and later in life TC levels are correlated.
The analysis does not capture post-acute CVD mortality beyond 5 years of the first MI or IS for those ages 40–65 at the time of the initial event nor does it capture post-acute CVD mortality beyond 6 years of the first MI or IS for those ages 66–89 at the time of the initial event.	Underestimate	The risk of post-acute CVD mortality was estimated based on Thom et al. (2001) for those aged 40–65 years and on S. Li et al. (2019) for those older than 65 years. Neither study reported post-acute mortality information for a longer follow-up period. The reported information does not support complete post-acute mortality risk elimination beyond the longest follow-up period. The EPA did not identify U.S. population-based MI/IS survivor studies that had a longer follow-up time and, thus, has no reliable quantitative basis to estimate post-acute mortality impacts beyond 6 years of the initial event.
The analysis assumes that post-acute CVD mortality for survivors of IS at ages 40–65 is the same as post-acute CVD mortality for survivors of MI at ages 40–65.	Uncertain	Post-acute mortality estimates for IS and MI were very close in the Medicare population (S. Li et al., 2019). For those aged 65 years or older, S. Li et al. (2019) have estimated the probability of death within 1 year after non-fatal IS to be 32.07 percent and the probability of death within 1 year after non-fatal MI to be 32.09 percent. Therefore, reliance on the post-acute mortality for MI to approximate the same for stroke is reasonable.
The analysis models the 85 year or older group jointly	Uncertain	The effect of this modeling approximation on the CVD benefits is not certain because the integer age-specific

Table 6-51: Limitations and Uncertainties in the Analysis of CVD Benefits Under the Final Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
and applies average mortality rate for those aged 85 or older in this age group.		mortality rates may be above or below the average mortality rate.
The analysis models the 85 year or older group jointly and uses serum PFOA/PFOS estimates for age 85 in initiate calculations in this age group.	Underestimate	Because the impacts of changes in PFOA/PFOS drinking water concentrations on serum PFOA/PFOS levels increase over time, the use of serum PFOA/PFOS concentrations at 85 years to model the 85 or older age group will underestimate the CVD risk impacts in this group.
The analysis applies the ASCVD model to those older than 80 years.	Overestimate	The ASCVD model evaluates first hard CVD event risk for adults aged 40-80. Applying the predicted hard CVD event risk for those aged 80 years or older results in an overestimate of benefits.
The EPA does not characterize uncertainty associated with ASCVD model parameters.	Uncertain	The EPA treats the coefficients of the ASCVD risk model as certain. However, uncertainty surrounding race/ethnicity- and sex-specific ASCVD model parameters could be characterized by multivariate normal distribution using the ASCVD model coefficient estimates, and the variance-covariance matrix shared by the ASCVD model authors. Assuming that ASCVD model parameters are certain is a limitation of this analysis.
Economic Valuation of Changes in Health Risk		
The analysis monetized changes in non-fatal first MI/IS risk using medical expenditures that do not cover long-term institutional or at-home care. Furthermore, the COI estimates do not include lost productivity. Finally, the COI-based approach does not account for the pain and suffering associated with non-fatal CVD events.	Underestimate	This analysis likely understates morbidity benefits since hard CVD events, particularly IS, require a longer rehabilitation period. According to HCUP 2017 data, 65 percent of IS survivors and 33 percent of MI survivors are discharged to a long-term care facility or to a home healthcare setting. Lost productivity impacts are also likely (Cropper & Krupnick, 2000; Skolarus et al., 2014). MI/IS survivors also experience significant reductions in the health-related quality of life (Bach et al., 2011; Kirchberger et al., 2020; Martino Cinnera et al., 2020; Mollon & Bhattacharjee, 2017).

Abbreviations: ASCVD – Atherosclerotic cardiovascular disease; BP – blood pressure; CVD – cardiovascular disease; HDLC – high-density lipoprotein; IS – ischemic stroke (ICD9 = 433, 434; ICD10 = I63); MI – myocardial infarction (ICD9 = 410; ICD10 = 121); NHANES – National Health and Nutrition Examination Survey; PFOA – perfluorooctanoic acid; PFOS – perfluorooctane sulfonic acid; TC – total cholesterol.

Table 6-52: Limitations and Uncertainties in the Analysis of RCC Benefits Under the Final Rule

Uncertainty/Assumption	Effect on Benefits Estimate	Notes
Characterizing the Exposed Population		
The analysis uses national-level estimates of kidney cancer incidence, prevalence, stage distribution, and relative survival data, as well as national-level life tables.	Uncertain	Using national-level baseline health data may over- or underestimate the effects of regulatory alternatives on RCC morbidity and mortality in specific PWSs and well as overall.
The EPA assumed that RCC comprises 90 percent of kidney cancer incidence.	Uncertain	Because baseline RCC incidence statistics are not readily available from the National Cancer Institute public use data, the EPA used kidney cancer statistics in conjunction with an assumption that RCC comprises 90 percent of all kidney cancer cases to estimate baseline lifetime probability of RCC. This assumption was used in RCC exposure-response modeling by U.S. EPA (2024f).
RCC risks are estimated for populations for which reductions in PFOA exposures relative to baseline exposures start at different ages, including children.	Uncertain	The relative cancer potency of PFOA in children is unknown, which may bias benefits estimates either upward or downward. Because RCC incidence in children is very small, we assess any bias to be negligible.
Modeling Changes in Health Risks		
The analysis assumes that the magnitude of RCC risk reductions resulting from reductions in serum PFOA levels will not exceed a PAF of 3.94 percent.	Uncertain	The EPA placed a cap of 3.94 percent on the magnitude of the estimated cumulative RCC risk reduction resulting from reductions in serum PFOA levels, based on its analysis of PAF values found in the literature on environmental contaminants and cancers (ICF, 2022b). This review found that changes in environmental exposures result in relatively modest PAFs (between 0.2 percent and 17.9%); however, few of the studies provided PAFs related specifically to RCC or kidney cancer. The EPA characterized the uncertainty surrounding this parameter using a log-uniform distribution with a minimum of 0.2 percent and a maximum of 17.9 percent. For the central estimate of RCC benefits, the EPA used a PAF of 3.94 percent, which is the mean of the PAF uncertainty distribution. As such, the EPA assumed that RCC risk reduction estimates in excess of the PAF are unreasonable even as a result of large changes in serum PFOA concentrations. Because this PAF cap is not based on RCC studies specifically, it is uncertain whether the RCC impacts are under- or overestimated.

Table 6-52: Limitations and Uncertainties in the Analysis of RCC Benefits Under the Final Rule

Uncertainty/Assumption	Effect on Benefits Estimate	Notes
The analysis assumes that there is no lag between changes in serum PFOA concentrations and changes in RCC incidence.	Overestimate	The studies estimating the link between serum PFOA and RCC are not dynamic, and hence do not provide insights into whether RCC incidence may respond gradually to changes in serum PFOA. The PK model estimates daily serum levels, which are averaged annually for the purposes of modeling gradual serum changes for the RCC risk reduction analysis. The RCC risk reduction analysis assumes immediate RCC incidence adjustment within each year, which may overestimate impacts to the exposed population.
The analysis relies on public-access SEER 18 10-year relative kidney cancer survival data to model mortality patterns in the kidney cancer population.	Uncertain	Reliance on these data generates both a downward and an upward bias. The downward bias is due to the short, 10-year excess mortality follow-up window. Survival rates beyond 10 years following the initial diagnosis are likely to be lower. The upward bias comes from the inability to determine how many of the excess deaths were deaths from kidney cancer.
The analysis assumes that RCC incidence patterns and survival are reasonably approximated by the kidney cancer statistics.	Uncertain	The exposure-response function provides information on changes in RCC risk, while detailed race/ethnicity-, sex-, and age-specific cancer incidence, stage, and survival information is available for kidney cancer only. For consistency with the RCC exposure-response modeling (U.S. EPA, 2024f), the EPA assumed that RCC comprises 90 percent of kidney cancer cases. In absence of RCC-specific detailed information, the model relies on patterns based on kidney cancer statistics.
The analysis models the 85 years or older group jointly and applies the average mortality rate for those aged 85 or older in this age group.	Uncertain	The effect of this modeling approximation on the RCC benefits is not certain because integer age-specific mortality rates may be above or below the average mortality rate.
The analysis models the 85 years or older group jointly and uses serum PFOA estimates for those aged 85 to initiate calculations in this age group.	Underestimate	Because the impacts of changes in PFOA drinking water concentrations on serum PFOA levels increase over time, the use of serum PFOA concentrations at 85 years to model the 85 or older age group will underestimate the RCC risk impacts in this group.

Table 6-52: Limitations and Uncertainties in the Analysis of RCC Benefits Under the Final Rule

Uncertainty/Assumption	Effect on Benefits Estimate	Notes
Economic Valuation of Changes in Health Risk		
RCC morbidity valuation is based on medical costs associated with the first line treatment that resulted in the most cost-effective treatment sequences, as reported in Ambavane et al. (2020).	Uncertain	The valuation is biased downward because it does not account for (1) the second line treatments that may also be applied; (2) lost productivity by the person experiencing RCC and family caregivers; and (3) the pain and suffering associated with experiencing RCC and/or adverse effects of RCC treatment. The valuation is biased upward because (1) the full year-specific cancer treatment is assumed to occur prior to the year-specific cancer population death; and (2) the first line treatment may be discontinued prior to the assumed maximum treatment duration of 2 years. The effect of using costs associated with the most cost-effective treatment from Ambavane et al. (2020) rather than costs for treatments currently prevalent in clinical practice is uncertain. The EPA could not assess the impact of this assumption because the EPA is not aware of publicly available information on the frequency of various kidney cancer treatments in the U.S. population. To assess the impact of using a willingness to pay based valuation approach, the EPA performed a sensitivity analysis using willingness to pay values for non-fatal unspecified cancer to value reductions in risk of RCC morbidity (See Appendix O).

Abbreviations: PFOA – perfluorooctanoic acid; PFOS – perfluorooctane sulfonic acid; PK – pharmacokinetic; RCC – renal cell carcinoma.

Table 6-53: Limitations and Uncertainties in the Analysis of DBP Quantified Benefits Under the Final Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
Modeling Reduced THM4 in PWSs		
Reductions in THM4 formation depend only on the relationship between raw water TOC levels and THM4 levels as estimated in the 1998 DBP ICR. Other source water quality parameters were not modeled.	Uncertain	The EPA assumes that PWSs affected by implementation of PFAS treatment technologies have similar characteristics as those evaluated in the 1998 DBP ICR. Source water parameters and treatments at individual plants may have changed over time.

Table 6-53: Limitations and Uncertainties in the Analysis of DBP Quantified Benefits Under the Final Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
The EPA uses available TOC data to estimate reduced THM4 concentration.	Uncertain	Due to the lack of site-specific information on factors affecting THM4 formation at each potentially affected drinking water treatment plant, the EPA uses relationships between TOC levels and changes in THM4 levels among GAC-treating systems from the 1998 DBP ICR and median raw water TOC levels for each source water type from the 2019 SYR4 dataset. Actual changes in THM4 concentrations for a given change in treatment at any specific PWS could be higher or lower than that estimated using the EPA's approach.
The EPA assigned TOC values at the system level based on ground water or surface water distributions.	Uncertain	Because the TOC levels for all systems are not available, the EPA used TOC data provided by states in response to the fourth Six-Year Review to derive TOC probability distributions for influent into a PFAS treatment process; one distribution for ground water systems and another for surface water systems. The EPA randomly assigned values from these distributions to each ground water or surface water system, respectively. The actual TOC values may be higher or lower than the assigned values. For systems using GAC for PFAS removal, the corresponding impact would be under-stating or over-stating costs.
The EPA estimates THM4 reduction based on free chlorine formation potential but does not estimate the reduction based on chloramine use.	Overestimate	The 1998 DBP ICR TSD provided information for systems that only used free chlorine as a disinfectant and did not capture THM4 reduction in chloraminating systems. This limitation likely leads to an overestimate of THM4 formed in systems that used chloramines in the distribution system because THM4 formation within the distribution system is lower when chloramines are used, compared to when free chlorine is used (Hua & Reckhow, 2008). Based on SYR3 data, 36 percent of surface water systems and 4 percent of ground water systems use chloramination (U.S. EPA, 2016j). Chloramines may produce greater amounts of genotoxic and carcinogenic DBPs, but a reduction in the TOC prior to disinfection will also yield a reduction in DBP formation (Cuthbertson et al., 2019).

Table 6-53: Limitations and Uncertainties in the Analysis of DBP Quantified Benefits Under the Final Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
THM4 is assumed to be a surrogate for other chlorination DBPs, some of which are more genotoxic and cytotoxic than THMs.	Uncertain	The EPA's analysis relies on the slope factor from Regli et al. (2015), which links lifetime risk of bladder cancer to THM4 concentrations in finished water. Regli et al. (2015) did not explicitly account for brominated or nitrogenous DBPs, but instead used THM4 as a surrogate for the broad suite of chlorination DBPs. This is consistent with the approach used in numerous epidemiolocal studies (Costet et al., 2011; Freeman et al., 2017) since insufficient data are available to estimate the co-occurrence and co-removal of specific genotoxic or cytotoxic DBPs.
The EPA estimates THM4 reduction based on GAC use but does not estimate the reduction in individual THM4 species.	Uncertain	GAC has been shown to shift the speciation among THM4 and can result in a relatively larger fraction of brominated species (THM3) compared to chloroform. However, studies show that even as speciation shifts, the absolute concentrations of each species are reduced (Cuthbertson et al., 2019; L. Wang et al., 2019).
The EPA assumed a GAC replacement frequency.	Underestimate	A GAC replacement frequency of 730 days was assumed based on the estimated percent removal of TOC curves (see Figures 6-11 and 6-12). After 730 days of GAC use the modeled TOC removal remained consistent for both ground water and surface water models. If the GAC was replaced more frequently based on PFAS removal needs, then increased average TOC removal would be observed further reducing DBP precursors.
The logistic model uses pilot/RSSCT results to predict ΔTHM4.	Overestimate	RSSCTs may overpredict full-scale adsorption capacity of GAC (Kempisty et al., 2022; Zachman & Summers, 2010)
SYR4 Comparison		
Estimates of reductions in THM4 formation assume that GAC treatment is the only treatment change in a distribution system.	Uncertain	Uncertainty exists if other changes (i.e., new source water, chemical dosing, other treatments added such as pre-chlorination, existing treatments changed such as new filter media) that could have been made in public water systems beyond GAC treatment could potentially over- or underestimate THM4 reduction.
The EPA analyzed only systems that were sampled under UCMR 3 and indicated GAC treatment under UCMR 4.	Uncertain	Assessing only UCMR GAC systems limited the sample to PWS serving ≥ 10,000 people. Therefore, the EPA was unable to compare THM4 reduction estimates to measured data for small systems.

Table 6-53: Limitations and Uncertainties in the Analysis of DBP Quantified Benefits Under the Final Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
The EPA relied on available CCRs to estimate the GAC treatment start date to determine before and after treatment years.	Uncertain	Available CCRs were used to inform the GAC start date. When CCRs were unavailable, the EPA searched the web to identify information about the timeline of treatment for individual PWSs. While installation dates were found, the exact date for when the GAC systems went into full-scale use was not always specified.
The EPA obtained THM4 values from multiple data sources.	Uncertain	For PWSs that met criteria outlined in Section 6.7.1.3.2 but had no THM4 data available in SYR4, the EPA relied on CCR THM4 data. Reporting on THM4 levels is inconsistent across CCRs. If a CCR listed “Amount Detected” instead of the THM4 average, then the EPA used the “Amount Detected” value to represent the THM4 average.
Characterizing the Exposed Population		
Analysis assumes that systems implementing IX do not accrue benefits associated with bladder cancer risk reductions.	Underestimate	Systems using IX for PFAS removal will also benefit from some TOC removal, but the removal will be limited in comparison to GAC treatment because PFAS-selective IX can show preferential removal of PFAS over organic matter (de Abreu Domingos & da Fonseca, 2018).
The analysis does not model location-specific demographics.	Uncertain	Because the EPA models impacts to aggregate populations based on systems triggered into treatment under various scenarios, the EPA relies on national-level demographic and bladder cancer data. The impact of this limitation is uncertain. For instance, populations with a large portion of elderly or male individuals will be more sensitive to changes in THM4 levels due to the high baseline bladder cancer incidence among elderly and male populations, compared to younger and female populations.
The analysis does not model variability by race/ethnicity.	Uncertain	Because the EPA models impacts based on a national-level distribution of finished water TOC levels, specific TOC levels at actual PWSs are not available. Therefore, these impacts were not included in the EPA’s DBP analysis. Accordingly, the EPA did not pursue race/ethnicity-specific modeling of health risk because it would not provide meaningful insight into distributional effects.

Table 6-53: Limitations and Uncertainties in the Analysis of DBP Quantified Benefits Under the Final Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
Bladder cancer risks are estimated for populations for which reductions in THM4 exposures relative to baseline exposures start at different ages, including children.	Uncertain	The relative cancer potency of THM4 in children is unknown, which may bias estimates either upward or downward. Past reviews found no clear evidence that children are at greater risk of adverse effects from bromoform or dibromochloromethane exposure (U.S. EPA, 2005a), although certain modes of action and health effects may be associated with exposure to THM4 during childhood (U.S. EPA, 2016g). Because bladder cancer incidence in children is very small, the EPA assesses any bias to be negligible.
Modeling Changes in Health Risks		
Analysis assumes an immediate and full reduction in bladder cancer risk following THM4 exposure reduction.	Overestimate	The EPA did not model the transitional dynamics in relative annual risk of bladder cancer following the THM4 exposure reduction. The EPA considered age-specific cohort cumulative exposures to THM4. Therefore, while drinking water concentrations are assumed to be reduced upon compliance with the rulemaking, the changes in cumulative average exposure are much more gradual. The EPA has not identified any studies on bladder cancer-specific risk cessation lag. Regli et al. (2015) do not provide pertinent information; as such, this is a cross-sectional analysis quantifying the relationship between lifetime cancer risk and lifetime average exposure. Existing cancer risk cessation lag studies focused on smoking and arsenic exposure (e.g., Hrubec & McLaughlin, 1997, Hartge et al., 1987, and C. W. Chen & Gibb, 2003); show that, annual cancer risk drops within the first 25 years after exposure cessation, yet it may never reach the annual cancer risk of persons who were always exposed to the treatment contaminant levels. In the EPA's modeling this issue pertains to those alive at the start of the evaluation period who have been exposed to the pre-treatment THM4 levels for a considerable amount of time, such as persons older than 60 years at the start of the evaluation period. This subpopulation comprises approximately 20 percent of the affected population alive in 2023.

Table 6-53: Limitations and Uncertainties in the Analysis of DBP Quantified Benefits Under the Final Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
The analysis relies on public-access SEER 18 10-year relative bladder cancer survival data to model mortality patterns in the bladder cancer population.	Uncertain	Reliance on these data generates both a downward and an upward bias. The downward bias is due to the short, 10-year excess mortality follow-up window. Survival rates beyond 10 years following the initial diagnosis are likely to be lower. The upward bias comes from the inability to determine how many of the excess deaths were deaths from bladder cancer.
The relationship from Regli et al. (2015) is a linear approximation of the odds ratios reported in Villanueva et al. (2004).	Uncertain	Given the uncertainty about the historical, location-specific THM4 baselines, Regli et al. (2015) provides a reasonable approximation of the risk. However, depending on the baseline THM4 exposure level, the impact computed based on Regli et al. (2015) may be larger or smaller than the impact computed using the Villanueva et al. (2004)-reported odds ratios directly.
The analysis assumes that the magnitude of DBP risk reductions resulting from reductions in serum PFOA levels will not exceed a PAF of 3.94 percent.	Uncertain	The EPA placed a cap of 3.94 percent on the magnitude of the estimated cumulative bladder cancer risk reduction resulting from reductions in THM4 levels, based on its analysis of PAF values found in the literature on environmental contaminants and cancers (ICF, 2022b). This review found that changes in environmental exposures result in relatively modest PAFs (between 0.2 and 17.9 percent); however, few of the studies provided PAFs related specifically to bladder cancer. For the estimate of bladder cancer benefits, the EPA used a PAF of 3.94 percent, which is the mean of the PAF uncertainty distribution. As such, the EPA did not quantify bladder cancer risk reduction estimates in excess of the PAF that are predicted to occur as a result of changes in THM4 concentrations. Because this PAF cap is not based on bladder cancer studies specifically, it is uncertain whether the bladder cancer impacts are under- or overestimated. Because the PAF is rarely binding in the bladder cancer analysis, the influence of PAF uncertainty on the analysis is likely negligible.

Table 6-53: Limitations and Uncertainties in the Analysis of DBP Quantified Benefits Under the Final Rule

Uncertainty/ Assumption	Effect on Benefits Estimate	Notes
Economic Valuation of Changes in Health Risk		
Bladder cancer morbidity valuation is based on medical costs and indirect/time costs (by cancer stage), as reported in Greco et al. (2019).	Uncertain	The valuation is biased downward because it does not account for (1) lost productivity by the family caregivers and volunteers; (2) broader labor market participation effects for those experiencing bladder cancer and/or providing care; and (3) the pain and suffering associated with experiencing bladder cancer and/or adverse effects of bladder cancer treatment. The valuation is biased upward because (1) the full year-specific cancer treatment is assumed to occur prior to the year-specific cancer population death; and (2) the treatment may be discontinued if it is no longer effective. To assess the impact of using a willingness to pay based valuation approach, the EPA performed a sensitivity analysis using willingness to pay values for non-fatal bladder cancer to value reductions in risk of bladder cancer morbidity (See Appendix O).

Abbreviations: CCR – consumer confidence reports; DBP – disinfection byproduct; GAC – granular activated carbon; ICR – information collection request; PFAS – per-and polyfluoroalkyl substances; PFOA – perfluorooctanoic acid; PFOS – perfluorooctane sulfonic acid; PWS – public water system; SYR – Six-Year Review; THM4 – four regulated trihalomethanes; TOC – total organic carbon; TSD – treatment study database; UCMR – Unregulated Contaminant Monitoring Rule, PAF – population attributable fraction.

7 Comparison of Costs to Benefits

This chapter provides a comparison of the incremental costs and benefits of the final rule, as described in Chapter 5 and Chapter 6.[87] The incremental cost is the difference between costs that will be incurred if the final rule is enacted over current baseline conditions. Incremental benefits reflect the avoided future adverse health outcomes attributable to PFAS reductions and co-removal of additional contaminants due to actions undertaken to comply with the final rule. This chapter also provides benefits and costs for the alternatives to the final rule that the EPA considered. Results for the final rule precede estimates for the alternatives. The EPA notes that under SDWA, the EPA must consider whether the costs of the rule are justified by the benefits based on all statutorily-prescribed costs and benefits, not just the quantified costs and benefits (see SDWA 1412(b)(3)(c)(i)).

Table 7-1 provides the incremental quantified costs and benefits of the final rule at a 2 percent discount rate in 2022 dollars. The top row shows total monetized annualized costs including total PWS costs and primacy agency costs. The second row shows total monetized annualized benefits including all endpoints that could be quantified and valued. For both, the estimates are the expected (mean) values and the 5th percentile and 95th percentile quantified estimates from the uncertainty distribution. These percentile estimates come from the distributions of annualized quantified costs and annualized quantified benefits generated by the 4,000 iterations of SafeWater MCBC, as described in Sections 5.1.2 and 6.1.2. Therefore, these distributions reflect the joint effect of the multiple sources of variability and uncertainty for quantified costs identified in Section 5.1.2 and for quantified benefits identified in Section 6.1.2 as well as the baseline uncertainties discussed throughout Chapter 4 such as baseline PFAS occurrence. The third row shows net quantified benefits (benefits minus costs). The net annual quantified incremental benefits are $760,000. Because of the variation associated with the use of statistical models such as SafeWater MCBC, the modeled quantified net benefits are nearly at parity. The uncertainty range for net quantified benefits is negative $622 million to $725 million. Additional uncertainties are presented in Table 7-6.

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[87] The cost-benefit analysis results for each option reflect the variability and uncertainties that could be quantified given the best available scientific data. There are many factors that the EPA could not quantify because of data limitations. For example, benefits will be underestimated if the PFOA and PFOS reductions result in avoided adverse health outcomes that cannot be quantified and valued. Chapters 0 and 0 identify these limitations and the potential effect on the cost or benefit estimates, respectively.

Table 7-1: Annualized Quantified National Costs and Benefits, Final Rule (PFOA and PFOS MCLs of 4.0 ppt each, PFHxS, PFNA, HFPO-DA MCLs of 10 ppt each, and HI of 1) (Million $2022)

	2% Discount Rate		
	5th Percentile[a]	**Mean**	**95th Percentile**[a]
Total Annualized Rule Costs	$1,435.70	$1,548.64	$1,672.10
Total Annualized Rule Benefits	$920.91	$1,549.40	$2,293.80
Total Net Benefits[b,c,d]	**-$621.99**	**$0.76**	**$725.07**

Notes: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates. Quantifiable benefits are increased under final rule table results relative to the other options presented because of modeled PFHxS occurrence, which results in additional quantified benefits from co-removed PFOA and PFOS.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty described in Section 5.1.2 and Table 5-1 for costs and Section 6.1.2 and Table 6-1 for benefits. This range does not include the uncertainty described in Table 5-21 for costs and Table 6-48 for benefits.

[b] See Table 7-6 for a list of the nonquantifiable benefits and costs, and the potential direction of impact these benefits and costs would have on the estimated monetized total annualized benefits and costs in this table.

[c] The national level cost estimates for PFHxS are reflective of both the total national cost for PFHxS individual MCL exceedances, and HI MCL exceedances where PFHxS is present above its HBWC while one or more other HI PFAS is also present in that same mixture. Total quantified national cost values do not include the incremental treatment costs associated with the co-occurrence of HFPO-DA, PFBS, and PFNA. EPA has considered the additional national costs of the HI and individual MCLs associated with HFPO-DA, PFNA, and PFBS occurrence in a quantified sensitivity analysis; see Appendix N, Section N.3 for the analysis and more information.

[d] PFAS-contaminated wastes are not considered RCRA regulatory or characteristic hazardous wastes at this time and therefore total costs reported in this table do not include costs associated with hazardous waste disposal of spent filtration materials. To address stakeholder concerns about potential costs for disposing PFAS-contaminated wastes as hazardous should they be regulated as such in the future, the EPA conducted a sensitivity analysis with an assumption of hazardous waste disposal for illustrative purposes only. See Appendix N, Section N.2 for additional detail.

Table 7-2 to Table 7-4 summarize the monetized total annualized costs and benefits for Options 1a, 1b, and 1c, respectively.

Table 7-2: Annualized Quantified National Costs and Benefits, Option 1a (PFOA and PFOS MCLs of 4.0 ppt) (Million $2022)

	2% Discount Rate		
	5th Percentile[a]	**Mean**	**95th Percentile[a]**
Total Annualized Rule Costs	$1,423.60	$1,537.07	$1,660.30
Total Annualized Rule Benefits	$913.05	$1,542.74	$2,280.10
Total Net Benefits[b,c]	**-$613.79**	**$5.67**	**$722.09**

Notes: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty described in Section 5.1.2 and Table 5-1 for costs and Section 6.1.2 and Table 6-1 for benefits. This range does not include the uncertainty described in Table 5-21 for costs and Table 6-48 for benefits.

[b] See Table 7-6 for a list of the nonquantifiable benefits and costs, and the potential direction of impact these benefits and costs would have on the estimated monetized total annualized benefits and costs in this table.

[c] PFAS-contaminated wastes are not considered RCRA regulatory or characteristic hazardous wastes at this time and therefore total costs reported in this table do not include costs associated with hazardous waste disposal of spent filtration materials. To address stakeholder concerns about potential costs for disposing PFAS-contaminated wastes as hazardous should they be regulated as such in the future, the EPA conducted a sensitivity analysis with an assumption of hazardous waste disposal for illustrative purposes only. See Appendix N, Section N.2 for additional detail.

Table 7-3: Annualized Quantified National Costs and Benefits, Option 1b (PFOA and PFOS MCLs of 5.0 ppt) (Million $2022)

	2% Discount Rate		
	5th Percentile[a]	**Mean**	**95th Percentile[a]**
Total Annualized Rule Costs	$1,102.60	$1,192.13	$1,291.40
Total Annualized Rule Benefits	$768.55	$1,296.84	$1,919.30
Total Net Benefits[b,c]	**-$414.34**	**$104.71**	**$710.38**

Notes: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty described in Section 5.1.2 and Table 5-1 for costs and Section 6.1.2 and Table 6-1 for benefits. This range does not include the uncertainty described in Table 5-21 for costs and Table 6-48 for benefits.

[b] See Table 7-6 for a list of the nonquantifiable benefits and costs, and the potential direction of impact these benefits and costs would have on the estimated monetized total annualized benefits and costs in this table.

[c] PFAS-contaminated wastes are not considered RCRA regulatory or characteristic hazardous wastes at this time and therefore total costs reported in this table do not include costs associated with hazardous waste disposal of spent filtration materials. To address stakeholder concerns about potential costs for disposing PFAS-contaminated wastes as hazardous should they be regulated as such in the future, the EPA conducted a sensitivity analysis with an assumption of hazardous waste disposal for illustrative purposes only. See Appendix N, Section N.2 for additional detail.

Table 7-4: Annualized Quantified National Costs and Benefits, Option 1c (PFOA and PFOS MCLs of 10.0 ppt) (Million $2022)

	2% Discount Rate		
	5th Percentile[a]	Mean	95th Percentile[a]
Total Annualized Rule Costs	$462.87	$499.29	$540.68
Total Annualized Rule Benefits	$397.28	$664.45	$970.70
Total Net Benefits[b,c]	**-$96.42**	**$165.16**	**$468.54**

Notes: Detail may not add exactly to total due to independent rounding. See Appendix P for results presented at 3 and 7 percent discount rates.

[a] The 5th and 95th percentile range is based on modeled variability and uncertainty described in Section 5.1.2 and Table 5-1 for costs and Section 6.1.2 and Table 6-1 for benefits. This range does not include the uncertainty described in Table 5-21 for costs and Table 6-48 for benefits.

[b] See Table 7-6 for a list of the nonquantifiable benefits and costs, and the potential direction of impact these benefits and costs would have on the estimated monetized total annualized benefits and costs in this table.

[c] PFAS-contaminated wastes are not considered RCRA regulatory or characteristic hazardous wastes at this time and therefore total costs reported in this table do not include costs associated with hazardous waste disposal of spent filtration materials. To address stakeholder concerns about potential costs for disposing PFAS-contaminated wastes as hazardous should they be regulated as such in the future, the EPA conducted a sensitivity analysis with an assumption of hazardous waste disposal for illustrative purposes only. See Appendix N, Section N.2 for additional detail.

The EPA notes that these quantified benefits are estimated using a COI approach (see Chapter 6 for further discussion). In the sensitivity analysis, the EPA also calculated quantified benefits using a willingness to pay approach instead of COI information, for non-fatal RCC and bladder cancer illnesses. In this case, the estimated expected quantified annualized costs are $1,548.64 million and the estimated expected quantified annualized benefits increase to $1,632.34 million (see Appendix O), resulting in $83.7 million in expected annualized net benefits.

The EPA further notes that the quantified benefit-cost results above are not representative of all benefits and costs anticipated under the final NPDWR. Due to occurrence, health, and economic data limitations, there are several adverse health effects associated with PFAS exposure and costs associated with treatment that the EPA could not estimate quantitatively.

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. Based on the available data at rule proposal and submitted by public commenters, the EPA is only able to quantify three PFOA- and PFOS-related health endpoints (i.e., changes in birth weight, CVD, and RCC) in the national analysis.

The EPA also evaluated the impacts of PFNA on birth weight and PFOS on liver cancer in quantitative sensitivity analyses (see Appendix K and Appendix O, respectively). Those analyses demonstrate that there are potentially significant other quantified benefits not included in the national quantified benefits above. For example, the EPA's quantitative sensitivity analysis for PFNA (Appendix K) found that inclusion of a 1 ppt PFNA reduction could increase annualized birth weight benefits 5.6-7.8-fold in a model system serving 100,000 people, relative to a scenario that quantifies a 1 ppt reduction in PFOA and a 1 ppt reduction in PFOS only. In the case of PFOS impacts on liver cancer, the EPA has estimated additional benefits of up to $4.79 million via the reduction in liver cancer cases anticipated to be realized by the final rule. All regulatory alternatives are expected to produce substantial additional benefits from all the other adverse health effects avoided, but that cannot be quantified at this time. Treatment responses implemented to remove PFOA and PFOS under Options 1a-c are likely to remove some amount of additional PFAS contaminants where they co-occur. Co-occurrence among PFAS compounds has been observed frequently as discussed in the PFAS Occurrence & Contaminant Background Support Document (U.S. EPA, 2024g). The final rule is expected to produce the greatest reduction in exposure to PFAS compounds as compared to the three regulatory alternative MCLs because it includes PFHxS, HFPO-DA, PFNA, and PFBS in the regulation. Inclusion of the HI will trigger more systems to treat (as shown in Section 4.4.4) and provides enhanced public health protection by ensuring reductions of these additional compounds when present above the HI of 1. For further discussion of the quantitative and qualitative benefits associated with the final rule, see Section 6.2.

The EPA also expects that the final rule will result in additional nonquantifiable costs. As noted above, the HI and individual MCLs are expected to trigger more systems into more frequent monitoring and treatment. In the national cost analysis, the EPA quantified the national treatment and monitoring costs associated with the PFHxS individual MCL and the HI associated costs based on PFHxS occurrence only. Due to occurrence data limitations, cost estimates for PFNA, PFBS, and HFPO-DA are less precise relative to those for PFOA, PFOS, and PFHxS compounds, and as such, the EPA performed a quantitative sensitivity analysis of the national cost impacts associated with HI exceedances resulting from PFNA, PFBS, and HFPO-DA and the PFNA and HFPO-DA MCLs to understand and consider the potential magnitude of costs associated with treating these three PFAS. The EPA found that in addition to the costs associated with PFHxS exceedances, which are included in the national cost estimate, the HI and individual MCLs for PFNA and HFPO-DA could cost an additional $82.4 million per year. In cases where these compounds co-occur at locations where PFAS treatment is implemented because of nationally modeled PFOA, PFOS, and PFHxS occurrence, treatment costs are likely to be marginally higher as treatment media estimated bed-life is shortened. In instances where concentrations of HFPO-DA, PFNA, and PFBS are high enough to cause or contribute to an HI exceedance when the concentrations of PFOA, PFOS, and PFHxS would not have already otherwise triggered treatment, the national modeled costs may be underestimated. If these PFAS occur in isolation at levels that affect treatment decisions, or if these PFAS occur in combination with PFHxS when PFHxS concentrations were otherwise below the its respective HBWC in isolation (i.e., less than 10 ppt) then the quantified costs underestimate the impacts of the final rule. See Appendix N.3 for a sensitivity analysis of additional treatment costs at systems with HI exceedances. See Appendix N.4 for a sensitivity analysis of the marginal costs of HFPO-DA and PFNA MCLs. For further discussion of how EPA considered the costs of the five individual MCLs and the HI MCL, see Section XII.A.4 of the preamble for the final rule.

The EPA has proposed designating PFOA and PFOS as CERCLA hazardous substances (U.S. EPA, 2022b). Stakeholders have expressed concern to the EPA that a hazardous substance designation for certain PFAS may limit their disposal options for drinking water treatment residuals (e.g., spent media, concentrated waste streams) and/or potentially increase costs. Designation of PFOA and PFOS as CERCLA hazardous substances would not require waste (e.g., biosolids, treatment residuals, etc.) to be treated in any particular fashion, nor disposed of at any specific particular type of landfill. The designation also would not restrict, change, or recommend any specific activity or type of waste at landfills. In its estimated national costs, the EPA has maintained the assumption that disposal does not have to occur in accordance with hazardous waste standards thus national costs may be underestimated. The EPA has conducted a sensitivity analysis that assumes hazardous waste disposal at all systems treating for PFAS to assess the potential increase in costs (see Appendix N). Table 7-5 summarizes the benefits and costs that are quantified and nonquantified under the final NPDWR.

Table 7-5: Summary of Quantified and Nonquantified Benefits and Costs in the National Analysis

Category	Quantified	Non-quantified	Methods (Report Section where Analysis is Detailed)
Costs			
PWS treatment costs[a]	✓		Section 5.3.1
PWS sampling costs	✓		Section 5.3.2.2
PWS implementation and administration costs	✓		Section 5.3.2.1
Primacy agency rule implementation and administration costs	✓		Section 5.3.2
Hazardous waste disposal for treatment media		✓	Section 5.6
POU not in compliance forecast		✓	Section 5.6
Benefits			
PFOA and PFOS birth weight effects	✓		Section 6.4
PFOA and PFOS cardiovascular effects	✓		Section 6.5
PFOA and PFOS renal cell carcinoma	✓		Section 6.6
Health effects associated with disinfection byproducts, specifically bladder cancer	✓		Section 6.7
Other PFOA and PFOS health effects[b]		✓	Section 6.2.2.2
Health effects associated with HI compounds HFPO-DA, PFNA, PFBS, and PFHxS		✓	Section 6.2
Health effects associated with other PFAS		✓	Section 6.2

Abbreviations: HFPO-DA – hexafluoropropylene oxide dimer acid; PFAS – per and polyfluoroalkyl substances; PFBS – perfluorobutanesulfonic acid; PFHxS – perfluorohexane sulfonate; PFNA – perfluorononanoic acid; PFOA – Perfluorooctanoic Acid; PFOS– Perfluorooctane Sulfonate; POU – point-of-use; PWS– public water system

Notes:

[a] The national level cost estimates for PFHxS are reflective of both the total national cost for PFHxS individual MCL exceedances, and HI MCL exceedances where PFHxS is present above its HBWC while one or more other HI PFAS is also present in that same mixture. Total quantified national cost values do not include the incremental treatment costs associated with the co-occurrence of HFPO-DA, PFBS, and PFNA. EPA has considered the additional national costs of the HI and individual MCLs associated with HFPO-DA, PFNA, and PFBS occurrence in a quantified sensitivity analysis; see Appendix N, Section N.3 for the analysis and more information.

[b] Effects of PFOS on liver cancer are summarized as a national-level sensitivity analysis in Appendix O.

Table 7-6 provides a summary of the likely impact of nonquantifiable benefit-cost categories. In each case, the EPA notes the potential direction of the impact on costs and/or benefits. For example, benefits are underestimated if the PFOA and PFOS reductions result in avoided adverse health outcomes that cannot be quantified and valued. Sections 5.7 and 6.8 identify the key methodological limitations and the potential effect on the cost or benefit estimates, respectively.

Table 7-6: Potential Impact of Nonquantifiable Benefits and Costs

Source	Final Rule	Option 1a	Option 1b	Option 1c
Nonquantifiable PFOA and PFOS health endpoints	B: underestimate	B: underestimate	B: underestimate	B: underestimate
Limitations with nationally representative HFPO-DA, PFNA, and PFBS occurrence data	B&C: underestimate	N/A	N/A	N/A
Nonquantifiable HFPO-DA, PFNA, PFHxS, and PFBS health endpoints	B: underestimate	N/A	N/A	N/A
Limitations with nationally representative occurrence data for additional PFAS compounds	B&C: underestimate	B&C: underestimate	B&C: underestimate	B&C: underestimate
Removal of co-occurring non-PFAS contaminants	B&C: underestimate	B&C: underestimate	B&C: underestimate	B&C: underestimate
POU not in compliance forecast	C: overestimate	C: overestimate	C: overestimate	C: overestimate
Unknown future hazardous waste management requirements for PFAS	B&C: underestimate	B&C: underestimate	B&C: underestimate	B&C: underestimate

Abbreviations: B – benefits; C – costs; POU – point-of-use; PFAS – per-and polyfluoroalkyl substances.

Table 7-1 through Table 7-6 summarize the results of this final rule analysis. As indicated in Section 2.2.2 of this EA, the EPA discounted the estimated monetized cost and benefit values using a 2 percent discount rate, consistent with OMB Circular A-4 (OMB, 2003; OMB, 2023) guidance. The U.S. White House and OMB recently finalized and re-issued the A-4 and A-94 benefit-cost analysis guidance (see OMB Circular A-4, 2023), and the update includes new guidance to use a social discount rate of 2 percent. The updated OMB Circular A-4 states that the discount rate should equal the real (inflation-adjusted) rate of return on long-term U.S. government debt, which provides an approximation of the social rate of time preference. This rate for the past 30 years has averaged around 2.0 percent per year in real terms on a pre-tax basis. OMB arrived at the 2 percent discount rate figure by considering the 30-year average of the yield on 10-year Treasury marketable securities, and the approach taken by OMB produces a real rate of 1.7 percent per year, to which OMB added a 0.3 percent per-year rate to reflect inflation as measured by the personal consumption expenditure (PCE) inflation index. The OMB guidance states that Agencies must begin using the 2 percent discount rate for draft final rules that are formally submitted to OIRA after December 31, 2024. The updated OMB Circular A-4 guidance further states that "to the extent feasible and appropriate, as determined in consultation with OMB, agencies should follow this Circular's guidance earlier than these effective dates." Given the updated default social discount rate prescribed in the OMB Circular A-4 and also public input received on the discount rates considered by the EPA in the proposed NPDWR, for this final rule, the EPA estimated national benefits and costs at the 2 percent discount rate for the final rule and incorporated those results into the final economic analysis. Since the EPA proposed this NPDWR with the 3 and 7 percent discount rates based on guidance in the previous version of OMB Circular A-4, the EPA has kept the presentation of results using these discount rates in Appendix P. The Administrator reaffirms his determination that the benefits of the rule justify the costs. The EPA's determination is based on its analysis under in SDWA section 1412(b)(3)(C) of the quantifiable benefits and costs at the 2 percent discount rate, in addition to at the 3 and 7 percent discount rate, as well as the nonquantifiable benefits and costs. The EPA found that significant nonquantifiable benefits are likely to occur from the final PFAS NPDWR.

The quantified analysis is limited in its characterization of uncertainty. In Table 7-1, the EPA provides 5th and 95th percentile values for net benefits. These values represent the quantified, or modeled, potential range in the expected net benefit values associated with the uncertainty resulting from the following variables: the baseline PFAS occurrence; the affected population size; the compliance technology unit cost curves, which are selected as a function of baseline PFAS concentrations and population size, the distribution of feasible treatment technologies, and the three alternative levels of treatment capital costs; the concentration of total organic carbon in a system's source water (which impacts GAC O&M costs); the demographic composition of the system's population; the magnitude of PFAS concentration reductions; the health effect-serum PFOA and PFOS slope factors that quantify the relationship between changes in PFAS serum level and health outcomes for birth weight, CVD, and renal cell carcinoma; and the cap placed on the cumulative renal cell carcinoma risk reductions due to reductions in serum PFOA. These modeled sources of uncertainty are discussed in more detail in Sections 5.1.2 and 6.1.2. The quantified 5th and 95th percentile values do not include a number of factors that impact both costs and benefits but for which the agency did not have sufficient data to include in the quantification of uncertainty. The factors influencing the final rule cost estimates that are not quantified in the uncertainty analysis are detailed in Table 5-21. These uncertainty sources include: the specific design and operating assumptions used in developing treatment unit cost; the use of national average costs that may differ from the geographic distribution of affected systems; the possible future deviation from the compliance technology forecast; and the degree to which actual TOC source water values differ from the EPA's estimated distribution. The EPA has no information to indicate a directional influence of the estimated costs with regard to these uncertainty sources. To the degree that uncertainty exists across the remaining factors, it would most likely influence the estimated 5th and 95th percentile range and not significantly impact the expected value estimate of costs.

Table 6-48 discusses the sources of uncertainty affecting the estimated benefits not captured in the estimated 5th and 95th reported values. The modeled values do not capture the uncertainty in: the exposure that results from daily population changes at NTNCWSs or routine population shifting between PWSs, for example spending working hours at a NTNCWS or CWS and home hours at a different CWS; the exposure-response functions used in the benefits analyses assume that the effects of serum PFOA/PFOS on the health outcomes considered are independent, additive, and that there are no threshold serum concentrations below which effects (cardiovascular, developmental, and renal cell carcinoma) do not occur; the distribution of population by size and demographics across EPs within modeled systems and future population size and demographic changes, and the Value of Statistical Life reference value or income elasticity used to update the VSL. Given information available to the agency, four of the listed uncertainty sources would not affect the benefits expected value but the dispersion around that estimate. They are the unmodeled movements of populations between PWSs with potentially differing PFAS concentrations; the independence and additivity assumptions with regard to the effects of serum PFOA/PFOS on the health outcomes; the uncertainty in the population and demographic distributions among EPs within individual systems; and the VSL value and the income elasticity measures. Two of the areas of uncertainty not captured in the analysis would tend to indicate that the quantified benefits numbers are overestimates. First, the data available to the EPA with regard to population size at NTNCWSs, while likely capturing peaks in populations utilizing the systems, does not account for the variation in use and population and would tend to overestimate the exposed population. The second source of uncertainty, which definitionally would indicate overestimates in the quantified benefits values, is the assumption that there are no threshold serum concentrations below which health effects (cardiovascular, developmental, and renal cell carcinoma) do not occur. One source of possible underestimation of benefits not accounted for in the quantified analysis is the impact of general population growth over the extended period of analysis.

In addition to the quantified cost and benefit expected values, the modeled uncertainty associated within the 5th and 95th percentile values, and the un-modeled uncertainty associated with a number of factors listed above, there are also significant nonquantifiable costs and benefits, which are important to the overall weighing of costs and benefits. Table 7-6 provides a summary of these nonquantifiable cost and benefit categories along with an indication of the directional impact each category would have on total costs and benefit. Table 5-21 and Table 6-48 also provide additional information on a number of these nonquantifiable categories.

For the nonquantifiable costs, the EPA had insufficient nationally representative data to precisely characterize occurrence of HFPO-DA, PFNA, and PFBS at the national level and therefore could not include complete treatment costs associated with: the co-occurrence of these PFAS at systems already required to treat as a result of estimated PFOA, PFOS, or PFHxS levels, which would shorten the filtration media life and therefore increase operation costs; and the occurrence of HFPO-DA, PFNA, and/or PFBS at levels high enough to cause systems to exceed the individual MCLs for PFNA and HFPO-DA or the HI and have to install PFAS treatment. The EPA expects that the quantified national costs, which do not include HFPO-DA, PFNA, and PFBS treatment costs are marginally underestimated (on the order of 5%) as a result of this lack of sufficient nationally representative occurrence data. In an effort to better understand and consider the costs associated with treatment of the PFNA and HFPO-DA MCLs and potentially co-occurring HFPO-DA, PFNA, and PFBS at systems both with and without PFOA, PFOS and PFHxS occurrence in exceedance of the MCLs the EPA performed a quantitative sensitivity analysis of the national cost impacts associated with HI MCL exceedances resulting from HFPO-DA, PFNA, and PFBS and/or individual MCL exceedances of PFNA and HFPO-DA. The analysis is discussed in Section 5.3.1.4 and Appendix N.3. Two additional nonquantifiable cost impacts stemming from insufficient co-occurrence data could also potentially shorten filtration media life and increase operation costs. The co-occurrence of other PFAS and other non-PFAS contaminants not regulated in the final rule could both increase costs to the extent that they reduce media life. The EPA did not include POU treatment in the compliance technology forecast because current POU units are not certified to remove PFAS to the standards required in the final rule. Once certified, this technology may be a low-cost treatment alternative for some subset of small systems. Not including POU treatment in this analysis has resulted in a likely overestimate of costs.

Appendix N.2 contains a sensitivity analysis that estimates possible additional national annualized costs of $99 million, which would accrue to systems if the waste filtration media from GAC and IX were handled as RCRA regulatory or characteristic hazardous waste. This sensitivity analysis includes only disposal costs and does not consider other potential environmental benefits and costs associated with the disposal of the waste filtration media.

There are significant nonquantifiable sources of benefits that were not captured in the quantified benefits estimated for the proposed rule. While the EPA was able to monetize some of the PFOA and PFOS benefits related to cardiovascular disease, infant birth weight, and renal cell carcinoma effects, the agency was unable to quantify additional reductions in negative health impacts in the national quantitative analysis. In addition to the national analysis for the final rule, the agency developed a sensitivity analysis assessing liver cancer impacts, which is detailed in Appendix O. The EPA did not quantify PFOA and PFOS benefits related to health endpoints including developmental, cardiovascular, hepatic, immune, endocrine, metabolic, reproductive, musculoskeletal, and other types of carcinogenic effects. Section 6.2.2 provides additional information on the nonquantifiable impacts of PFOA and PFOS. Further, the agency did not quantify any health endpoint benefits associated with the potential reductions in HI PFAS, which include PFHxS, HFPO-DA, PFNA, and PFBS, or other co-occurring non-regulated PFAS which would be removed due to the installation of required filtration technology at those systems that exceed the final MCLs. The nonquantifiable benefits impact categories associated with PFHxS, HFPO-DA, PFNA, and PFBS include developmental, cardiovascular, immune, hepatic, endocrine, metabolic, reproductive, musculoskeletal, and carcinogenic effects. In addition, the EPA did not quantify the potential developmental, cardiovascular, immune, hepatic, endocrine, metabolic, reproductive, musculoskeletal, and carcinogenic impacts related to the removal of other co-occurring non-regulated PFAS. See Section 6.2.4 for additional information on the nonquantifiable impacts of PFHxS, HFPO-DA, PFNA, and PFBS, and other non-regulated co-occurring PFAS.

The treatment technologies installed to remove PFAS can also remove numerous other non-PFAS drinking water contaminants which have negative health impacts including additional regulated and unregulated DBPs (the quantified benefits assessment does estimate benefits associated with THM4), heavy metals, organic contaminants, and pesticides, among others. The removal of these co-occurring non-PFAS contaminants could have additional positive health benefits. In total these nonquantifiable benefits are anticipated to be significant and are discussed qualitatively in Section 6.2.

To fully weigh the costs and benefits of the action, the agency considered the totality of the monetized values, the potential impacts of the nonquantifiable uncertainties described above, the nonquantifiable costs and benefits, and public comments received by the agency related to the quantification and qualitative assessment of the costs and benefits. In the final rule, the EPA is reaffirming the Administrator’s determination made at proposal that the quantified and nonquantifiable benefits of the rule justify its quantified and nonquantifiable costs (88 FR 18638).

8 Environmental Justice Analysis

8.1 Introduction

The EPA defines environmental justice (EJ) as "the fair treatment and meaningful involvement of all people regardless of race, color, national origin, or income with respect to the development, implementation, and enforcement of environmental laws, regulations, and policies" (U.S. EPA, 2016h). The concept of fair treatment includes not just the distribution of burdens across populations but also the distribution of risk reduction from the EPA's actions. The EPA reviews potential EJ concerns regarding minority populations, low-income populations, and/or indigenous peoples (U.S. EPA, 2016h).

The framework used to evaluate the anticipated EJ impacts of the final rule for per– and polyfluoroalkyl substances (PFAS) comes from the Technical Guidance for Assessing Environmental Justice in Regulatory Analysis (U.S. EPA, 2016h), which provides the following guiding questions:

- Are there potential EJ concerns associated with environmental stressors affected by the regulatory action for population groups of concern in the baseline?
- Are there potential EJ concerns associated with environmental stressors affected by the regulatory action for population groups of concern for the regulatory options under consideration?
- For the regulatory options under consideration, are potential EJ concerns created or mitigated compared to the baseline?

Contextualizing these questions for the final PFAS rule, the EPA evaluated the following questions:

- Are population groups of concern (i.e., people of color and low-income populations) disproportionately exposed to PFAS compounds in drinking water delivered by PWSs?
- Are population groups of concern disproportionately affected by the final rule and regulatory alternatives under consideration for the final PFAS NPDWR?
- If any disproportionate impacts are identified, do they create or mitigate baseline EJ concerns?

As part of the proposal process for the PFAS NPDWR, the EPA conducted the EJ analyses in this chapter to assess the demographic distribution of baseline PFAS drinking water exposure and impacts that are anticipated to result from the final rule. The EPA conducted two separate analyses to address the research questions presented above. To inform the first question, the EPA conducted an analysis using the agency's EJSCREENbatch R package, which utilizes data from EJScreen, the agency's Environmental Justice Screening and Mapping Tool and from the U.S. Census Bureau's American Community Survey (ACS) 2015–2019 five-year sample (U.S. EPA, 2019a). To inform the second and third questions above, the EPA conducted an EJ analysis of the EPA's final regulatory option and regulatory alternatives using SafeWater MCBC.

Section 8.2 provides an overview of the EPA's EJ literature review. Sections 8.3 and 8.4 describe the EJ analyses the EPA conducted. Section 8.5 presents the conclusions from the EPA's EJ analyses.

8.2 Literature Review

The EPA conducted a literature review to develop a broad understanding of current research at the intersection of drinking water quality, PFAS exposure, and communities with related EJ concerns. The literature covered a range of specific topics including the likelihood of exposure based on proximity to sites of contamination, sociodemographic characteristics of communities exposed to PFAS in region-specific studies and understanding the sociodemographic distribution of health outcomes associated with exposure to PFAS. The EPA's literature review also examined the relationship between PFAS exposure via drinking water in overburdened communities and a range of health outcomes.

8.2.1 Methods

The EPA conducted its literature review to evaluate and synthesize findings from studies that explored associations between PFAS exposure via drinking water in overburdened communities and associated health outcomes, including those health endpoints the EPA quantified as part of its benefits analysis: changes in infant birth weight, CVD, and kidney cancer.

The EPA applied a variety of search terms for the literature review, including: CVD; disparities; disproportionate exposure; disproportionate impact; drinking water quality/contamination; environmental justice; equity; forever chemicals; inequity; infant birth weight; kidney cancer; low-income; minority; over-burdened; people of color; PFAS; PFAS interactions; PFC(s); PFOA; PFOS; race differences in health effects after PFAS exposure; race disparities in health effects, immune effects, and PFAS exposure; race ethnicity and health effects of PFAS exposure and interactions; sociodemographic differences in health effects after PFAS exposure; social justice; and tribal.

From the literature review, the EPA found that there are a limited number of studies that focus on the association between disproportionate exposure to PFAS via drinking water and health outcomes for overburdened communities on a national level. The agency excluded studies that examined exposure routes apart from drinking water and/or did not evaluate race/ethnicity within their participant demographics. Of the studies that the EPA identified as part of its literature review, all but two studies were published in peer reviewed journals (with the remaining two studies appearing in gray literature).

8.2.2 Findings

To contextualize its analysis of EJ impacts related to PFAS in drinking water, the EPA reviewed studies that evaluate overall EJ concerns related to environmental contamination. In 1987, the EPA reported in a nationwide study that roughly twice as many people of color resided in proximity to a commercial hazardous waste facility compared to communities without a facility (U.S. EPA, 1994). Later research indicated that communities of low socioeconomic status are more likely to reside in proximity to environmental hazardous facilities, thereby potentially facing a disproportionate impact of exposure to toxic chemicals than communities of higher socioeconomic status (Brown, 1995; Brulle & Pellow, 2006). A 2010 study showed 63 percent of large polluters in a North Carolina county were operating in census tracts with per capita income below $21,000, as identified in the EPA's Toxics Release Inventory (TRI) (Banzhaf et al., 2019).

When specifically examining studies related to PFAS in drinking water, available literature showed associations between PFAS contamination in drinking water and proximity to sites including those critical for transportation infrastructure, industry, and national defense (Black et al., 2021; X. C. Hu et al., 2016; Johnston & Cushing, 2020; Sunderland et al., 2019). Researchers noted that identifiable sources of PFAS are often prevalent at aforementioned locations and are more frequently located in overburdened communities (Black et al., 2021; X. C. Hu et al., 2016; Stoiber et al., 2020).

The characteristics of PFAS, such as high aqueous solubility and persistence within the environment, allow them to travel readily between ecological zones (ATSDR, 2021; X. C. Hu et al., 2016; Kotlarz et al., 2020). As such, PFAS contamination can negatively impact drinking water sources downstream from an original contamination site, putting residents in communities surrounding known sources of PFAS at a disproportionate risk of exposure. A 2019 study in Michigan by Desikan et al. (2019) evaluated the proportion of low-income households and households with people of color in communities within five miles of PFAS-contaminated sites compared to census projections for those areas. The study found that 38,962 more low-income households and 294,591 more households with people of color reside within five miles of a site contaminated with PFAS than expected, based on U.S. Census data.

In California, Lee et al. (2021) demonstrated that overburdened communities are more likely to be served by PWSs with higher levels of PFAS. PFAS data were integrated with results from CalEnviroScreen 3.0, a statewide EJ screening tool (OEHHA, 2016). Of the 7,896 PWSs in the state, about 3 percent (n = 248) had been monitored for PFAS, serving 42 percent of California's total population. Results from the study showed that PFAS was detected in 160 of 248 PWSs, or roughly 65 percent of systems monitored. Lee, Kar, and Reade (2021) overlaid the upper 25 percent of disadvantaged communities as identified by CalEnviroScreen 3.0 with water systems experiencing the highest levels of PFAS contamination. Among the communities in the top quartile for people of color and low-income demographic groups, 69 percent had PFAS detected in their water system. Further, PWSs in 20 percent of overburdened communities with PFAS contamination fell within the highest quartile of PFAS concentration levels in the state of California, suggesting that PFAS occurrence is disproportionately higher in drinking water serving already overburdened communities. Only 2 of the 10 water systems with the highest PFAS concentrations fell below the state average for all relevant demographic indicators included in the study (people of color, education level, unemployment, poverty, and housing burden).

A 2023 study by Liddie, Schaider, and Sunderland examined sociodemographic disparities in exposures to PFAS via drinking water based on potential exposure source. Community water systems were geocoded within 8-digit hydrologic codes where the exact coordinates of water source regions were unavailable, and the study refers to these areas as watersheds or CWS watersheds. In examining data from 18 states with the most robust PFAS monitoring and reporting infrastructure, Liddie et al. (2023) found that watersheds with higher concentrations of PFAS also had higher concentrations of the potential PFAS exposure sources that the researchers examined. Sources of potential exposure (based on correlation of proximity) included industrial sites, wastewater treatment plants, municipal solid waste landfills, military fire training areas, and civilian airports. Additionally, watersheds that served Hispanic/Latino communities and non-Hispanic Black communities were found to have significantly greater odds of containing PFAS sources. Further, CWS watersheds with PFAS concentrations above 5 ppt or above the lowest state-level MCL served communities with greater proportions of Hispanic/Latino and non-Hispanic Black populations than CWS watersheds with concentrations below these limits (Liddie et al., 2023).

At least two studies identified the use of aqueous film forming foam (AFFF) as a predictor of PFAS concentrations in U.S. drinking water (Johnston & Cushing, 2020; Sunderland et al., 2019). Using nationally representative PFAS occurrence data from UCMR 3, a study from X. C. Hu et al. (2016) found that the presence of a military fire training area using AFFF within a watershed's eight-digit hydraulic unit code (HUC) increased the frequency of exposure to at least one PFAS analyte in drinking water from 10.4 percent to 28.2 percent . For each additional military site within a HUC, drinking water samples with detectable levels of PFAS found a 20 percent increase in PFHxS, a 10 percent increase in both PFHpA and PFOA, and a 35 percent increase in PFOS.

The EPA also sought to characterize literature that discusses potential pathways of PFAS exposure for communities in proximity to waste disposal and destruction sites. The EPA is unaware of any literature which specifically discusses PFAS exposure for communities with potential environmental justice concerns due to disposal of PFAS-contaminated drinking water treatment residuals. Therefore, the EPA has reviewed literature which discusses the siting of waste facilities in general as well as the pathways of exposure for other contaminants. It is also important to note that there are uncertainties associated with the potential pathways of exposure for communities with potential environmental justice concerns regarding the destruction and disposal of PFAS in drinking water. For information related to the destruction and disposal of PFAS, please see the EPA's Interim Guidance on the Destruction and Disposal of Perfluoroalkyl and Polyfluoroalkyl Substances and Materials Containing Perfluoroalkyl and Polyfluoroalkyl Substances, version 2 (U.S. EPA, 2020c.

Waste facilities are often disproportionately located near communities with potential environmental justice concerns (Martuzzi et al., 2010). Additionally, in a national-level study of the demographic characteristics of communities collocated with waste management facilities, racial composition was found to be an independent predictor of waste management facility siting, controlling for other socioeconomic variables (Mohai & Saha, 2015). As such, communities with potential EJ concerns may experience adverse health effects that result from these disproportionate exposures to PFAS due to proximity to waste sites if PFAS are released from these sites (Desikan et al., 2019).

Martin et al. (2023) found that communities with hazardous waste incinerators that regularly receive PFAS shipments have demographic characteristics that indicate that potential exposures, if any, that result from incineration may affect individuals that reside in communities with lower incomes and less education than the US average. However, there is uncertainty regarding exposures from PFAS destruction at these hazardous waste incinerators. PFAS residuals from drinking water are likely carbon, which is likely to be reactivated. As described in the EPA's Interim Guidance on the Destruction and Disposal of Perfluoroalkyl and Polyfluoroalkyl

Substances and Materials Containing Perfluoroalkyl and Polyfluoroalkyl Substances, version 2 (U.S. EPA, 2020c), carbon reactivation systems have the potential to remove PFAS from the reactivated carbon and destroy PFAS. Additionally, Distefano et al. (2022) showed >99.99 percent destruction of measured PFAS at a full-scale commercial reactivation facility. The EPA believes when proper guidance, such as that from the EPA's Interim Guidance on the Destruction and Disposal of Perfluoroalkyl and Polyfluoroalkyl Substances and Materials Containing Perfluoroalkyl and Polyfluoroalkyl Substances, is followed, the destruction and disposal of drinking water treatment residuals can be suitably managed in a way which can minimize risk.

For more information and further discussion on this topic, please see Section 4 (Considerations for Potentially Vulnerable Populations Living Near Likely Destruction or Disposal Sites) in *Interim Guidance on the Destruction and Disposal of Perfluoroalkyl and Polyfluoroalkyl Substances and Materials Containing Perfluoroalkyl and Polyfluoroalkyl Substances*, version 2 (U.S. EPA, 2020c).

To remain consistent with the health endpoints associated with PFAS exposure that are monetized as part of the final PFAS NPDWR's benefits analysis, the health outcomes of focus in this literature review included CVD, kidney cancer, and impacts on infant birth weight. For more information on the EPA's quantified benefits analysis, see Chapter 60.

Literature showed that overburdened communities experience relatively higher adverse health outcomes compared to communities with fewer people of color (Driscoll & Gregory, 2021; Fryar et al., 2017; Pinheiro et al., 2021). Literature also showed that risk of CVD, kidney cancer, and changes in infant birth weight are associated with PFAS exposure (Almond et al., 2005; Barry et al., 2013; Goff et al., 2014; Ma & Finch, 2010; Raleigh et al., 2014; Steenland & Woskie, 2012; Vieira et al., 2013; U.S. EPA, 2016d; U.S. EPA, 2016h; U.S. EPA, 2021a; U.S. EPA, 2024e; U.S. EPA, 2024f), discussed in more detail in Chapter 60.

The Centers for Disease Control and Prevention (CDC) identified hypertension (HTN) as a substantial risk factor for CVD (Fryar et al., 2017). Using the 140/90 mmHg threshold for HTN diagnosis, the CDC reported that African American adults reported a higher burden of HTN (40.3%) compared to White (27.8%), Asian (25.0%), or Hispanic (27.8%) adults (Fryar et al., 2017). Additionally, a comprehensive narrative literature review by Graham (2015) found disproportionate rates of CVD among minority subpopulations in the U.S., particularly the African American population. African American subpopulations were found to have higher incidence of myocardial infarction, heart failure, stroke, among other cardiovascular events and experience the highest overall death rate from CVD among various minority population groups.

With regards to cancer, a study by Uche et al. (2021) showed statistically significantly greater cumulative cancer risk was identified in communities in which small and large CWSs serve higher proportions of Hispanic/Latino and Black/African American residents in Texas and California.[88] In Texas, greater cumulative cancer risk was statistically significantly greater for small and medium CWSs serving relatively higher proportions of Hispanic/Latino community members. Additionally, small CWSs serving relatively higher proportions of Black/African

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[88] A CWS was defined as small if it served 501-3,300 people, medium if it served 3,301-10,000 people, large if it served 10,001-100,000 people, and very large if it served more than 100,000 people.

American residents had statistically significantly greater cumulative cancer risk. In California, cumulative cancer risk was statistically significantly greater for very large CWSs serving relatively higher proportions of Black/African American community members, followed by small CWSs serving relatively higher proportions of Hispanic/Latino residents.

Pinheiro et al. (2021) studied kidney cancer rates in White, Black, Asian/Pacific Islander (API), American Indian, all non-Hispanic, and Hispanic populations of any race by using reported cancer deaths in California and Florida (2008–2018) and New York (2008–2017). This study's methodology directly compared results for specific race/ethnicity groups to White populations. Results indicated that American Indian individuals experience the highest mortality (54%), and mortality is 53 percent higher for men and women when compared to mortality among White participants (Pinheiro et al., 2021). Conversely, API populations showed significantly lower mortality than White populations, with 45 percent lower mortality among males and 43 percent lower mortality among females. Kidney cancer mortality among Black populations and all-combined Hispanic populations (i.e., Cuban, Puerto Rican, and Mexican) was also significantly lower than among White populations, but by smaller margins: mortality was 12 percent and 16 percent lower for Black males and females and 11 percent and 8 percent lower for Hispanic males and females, respectively.

Additionally, the CDC's National Vital Statistics Reports used the 2020 birth file from the National Vital Statistic System to display distributions in prepregnancy body mass index (BMI), including three classes of obesity, by maternal race and Hispanic origin for women who gave birth in 2020 (Driscoll & Gregory, 2021). Infants born to non-Hispanic Black women had the highest rate of low birth weight (14.19%), followed by infants of Hispanic women (7.40%). Infants of non-Hispanic White women had the lowest rate of low birth weight (6.84%) (Driscoll & Gregory, 2021).

Furthermore, the EPA reviewed studies that examine blood serum levels of PFAS across various demographic groups. Studies analyzing biomarker data indicate some demographic disparities that exist in blood serum levels across certain PFAS analytes (Boronow et al., 2019; Calafat et al., 2007; Eick et al., 2021; C. Y. Lin et al., 2020; Nelson et al., 2012; V. K. Nguyen et al., 2020; S. K. Park et al., 2019). Specifically, blood serum levels of PFNA and PFOS were found to be elevated in Black adults (Boronow et al., 2019; Calafat et al., 2007; Eick et al., 2021; C. Y. Lin et al., 2020; Nelson et al., 2012; S. K. Park et al., 2019). PFNA was also found to be elevated in Asian American mothers, when compared to all other races (Eick et al., 2021). Additionally, PFDA was found to be elevated in Asian American women, when compared to non-Hispanic White populations (V. K. Nguyen et al., 2020). Finally, Me-FOSAA was found to be elevated in Black women at some but not all study sites analyzed (S. K. Park et al., 2019).

However, many studies indicate lower average blood serum PFAS levels among people of color. Three studies in particular demonstrated that non-Hispanic White populations had the highest concentrations of PFAS across all analytes (Barton et al., 2020; Kato et al., 2014; Kingsley et al., 2018). It should be noted, however, that the study design for Barton et al. (2020), Kato et al. (2014), and Kingsley et al. (2018) each had majority non-Hispanic White participant demographics of 75 percent, 63 percent, and 61 percent of study participants, respectively. The literature also indicates that higher socioeconomic status (e.g., income) is associated with higher PFAS blood serum levels (Buekers et al., 2018).

8.2.3 Discussion and Limitations

The EPA's purpose in conducting its literature review was to examine the relationship between PFAS exposure via drinking water in overburdened communities and health outcomes related to CVD, changes in infant birth weight, and kidney cancer. Presented studies indicate that higher percentages of low-income and minority communities reside near a range of PFAS-contaminated sites. Such contamination is also shown to occur at higher levels in low-income and minority communities. Further, the EPA's literature review analysis indicates that PFAS contamination occurs more often and/or at higher levels in overburdened communities.

It should be noted there are substantial gaps in current literature on PFAS exposure and health outcomes in overburdened communities. One substantial gap in the available literature is a dearth of studies that examine differential impacts of health outcomes associated with PFAS exposure, as reported by race or ethnicity. Potential gaps in understanding also relate to determining whether the rate of developed risk for one or more of the aforementioned health endpoints is related to exposure to PFAS contamination in drinking water rather than other exposure pathways.

The blood serum PFAS studies evaluated as part of this literature review have their limitations in extrapolating to the potential disproportionate impacts of PFAS drinking water exposure given their focus on overall PFAS exposure across many exposure routes rather than drinking water-specific exposures. Wilder et al. (2017) note that national average PFAS blood serum levels are influenced by a variety of major exposure pathways, including diet and consumer products in addition to exposure via drinking water. As such, this limits conclusions that can be drawn about the demographic breakdown of PFAS blood serum levels due to drinking water exposure alone. Additional information on exposure via drinking water alone is necessary to better understand the impacts of PFAS drinking water contamination on PFAS blood serum levels within overburdened communities.

Another limitation of these blood serum-based studies is their inequitable representation of study participants by race. The participant demographic makeup of three published studies that examined PFAS blood serum levels was highly biased toward the non-Hispanic White population, resulting in an incomplete understanding of people of color's exposure to PFAS. Statisticians can adjust the results if certain participant demographic groups are disproportionately represented. However, these adjustments are based on assumptions about the underlying demographic makeup of the study population.

8.3 EJ PFAS Exposure Analysis

This section describes the data sources and approach the EPA used to characterize the demographic distribution of PFAS exposure in drinking water. This analysis is designed to answer the question posed in the beginning of the chapter: Are population groups of concern (i.e., people of color and low-income populations) disproportionately exposed to PFAS compounds in drinking water delivered by PWSs? This analysis estimates anticipated exposure rates above various PFAS concentrations for four PFAS analytes, where occurrence of these is used as a proxy for co-occurrence of many other PFAS compounds. In some cases, the thresholds that the EPA uses in this analysis overlap with regulatory alternatives considered by the EPA in the final regulatory action. This analysis does not evaluate the anticipated costs and benefits of the final rule and regulatory alternatives. The EPA's analysis of the anticipated demographic distribution of costs and benefits of the final rule and regulatory alternatives can be found in Section 8.4.

The EPA estimated the sociodemographic characteristics of populations that the EPA anticipates are exposed to levels higher than various threshold concentrations of four PFAS analytes (PFOA, PFOS, PFHxS, and PFHpA). For this analysis, the EPA had sufficient information on PFAS occurrence and PWS service area boundaries in the sample population, which was a subset of PWSs.[89] PWSs were first categorized by available data (Section 8.3.1), using availability of UCMR 3 sampling data, state sampling data, and availability of service area boundary information (Table 8-1).

The EPA used PWS service area data in conjunction with the EJSCREENbatch R package to obtain sociodemographic characteristics of the populations served by PWSs (U.S. EPA, 2022a). The EJSCREENbatch R package allows analysts to conduct EJ screening analyses for multiple geographies using environmental and sociodemographic data from EJScreen and the American Community Survey. The EPA estimated the rate of exposure to PFAS across demographic groups using PFAS occurrence data and the sociodemographic characteristics of populations served with designated service area boundaries. The EPA conducted this analysis using several thresholds: hypothetical trigger levels set above Method 537.1 detection limits and intended to reflect a water system's margin of safety below the MCL values (also referred to as baseline occurrence level for this analysis), UCMR 5 MRLs, and 10.0 ppt. This analysis serves as an estimate of possible exposure to PFAS levels over these thresholds, as the EPA cannot confirm that these populations consumed the water at the time of elevated PFAS occurrence at each PWS.

8.3.1 Data Sources and Approach

8.3.1.1 Categorization of Public Water Systems

The EPA designated distinct categories for PWSs based on data availability for PFAS occurrence and estimated PWS service area boundaries. The agency used two types of PFAS occurrence data sources in this analysis: (1) simulated PFAS occurrence data for PWSs with sampled PFAS occurrence data under UCMR 3; and (2) state-collected PFAS occurrence data for PWSs not sampled under UCMR 3 (U.S. EPA, 2017). PWS service area boundary data are distinguished by three types: (1) those with predelineated PWS service area boundaries, (2) those where zip codes served by PWSs were used as a proxy to approximate and delineate PWS service area boundaries, and (3) those with no available PWS service area boundary information. Table 8-1 describes the characteristics of each of the six distinct PWS categories examined in this analysis.

For the EJ exposure analysis, the EPA focused on reporting results for PWSs in categories 1 and 2, which were sampled for PFAS under UCMR 3. The PWSs in categories 4 and 5 include systems with state PFAS occurrence data, and the EPA has summarized the results for these categories in Appendix M. The EPA used data from EJScreen (U.S. EPA, 2022a) and the

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[89] PWS service area boundaries are defined as the spatial extent of the geographic area served by a PWS.

American Community Survey along with PWS service area boundary data to characterize the sociodemographic characteristics of PWSs.

PWSs in categories 1 and 2 account for 252.2 million people served (n = 4,743 PWSs), and PWSs in categories 4 and 5 account for approximately 2 million people served (n = 736 PWSs). PWSs in categories 3 and 6 were not included in the EJ exposure analysis, as PWS service area boundaries or zip codes served by the PWS were unavailable.

Table 8-1: Categorizing of PWSs Based on Data Availability for PFAS Occurrence and PWS Service Area Boundaries

	PWS Included in UCMR 3	**PWS State PFAS Occurrence Data Available and Not Included in UCMR 3**
PWS Service Areas Available	Category 1	Category 4
PWS Service Area Boundary Estimates from Zip Codes	Category 2	Category 5
No PWS Service Area Information Available	Category 3	Category 6

Abbreviations: PWS – public water system; UCMR – Unregulated Contaminant Monitoring Rule.

8.3.1.2 Data Sources

8.3.1.2.1 PFAS Occurrence

The two data source categories used to derive PFAS occurrence estimates for this analysis are described in more detail below. All PFAS occurrence data are presented in parts per trillion (ppt).

Generally, if a system was sampled for PFAS under UCMR 3, the EPA used simulated occurrence data that were based on system-specific results. For PWSs in categories 1 and 2 (n = 4,743 PWSs), the EPA simulated PFAS occurrence data using a hierarchical Bayesian model that was optimized with PFAS occurrence data from UCMR 3 and, where available, state data (see Cadwallader et al., 2022, and Section 4.44.4 for further description). The EPA calculated the system-level geometric mean occurrence value for each PWS from the simulated water sample concentrations. All simulated values (i.e., simulated samples for PWSs in categories 1 and 2) were above zero because the occurrence model assumes a log-normal distribution for water concentration. The system-level geometric mean occurrence values for the category 1 and 2 PWSs ranged from 0.01 to 254.65 ppt.

For other systems, the EPA used state sampling data. The EPA used state monitoring data from 12 states[90], which generally conducted nontargeted monitoring (i.e., random sampling) of finished drinking water for one or more of the four PFAS in this analysis. PWSs that had state sampling data but were not sampled under UCMR 3 fell into categories 4 and 5 (n = 736). The EPA calculated the system-level geometric means of measured PFAS water sample concentrations to characterize PFAS occurrence for each PWS. For this dataset, the agency did not pursue Bayesian estimation of non-detection concentrations due to a limited sample size and non-standardized sampling regime. Instead, for these data, the EPA set non-detections to a small constant, 10 percent of the lowest analyte sample value (i.e., 0.02 ppt for each analyte), before calculating the system-level geometric mean.[91]

Among the 12 state occurrence datasets used in this analysis to characterize PFAS occurrence for category 4 and 5 PWS service areas, the EPA noted that different states utilized various reporting, quantification, and/or detection limits when analyzing and presenting data, and for some states, no clearly defined limits were publicly provided as part of the dataset. Further, the limits often varied within the data for each state depending on the specific PFAS analyte. In some cases, states reported detection, quantification, or reporting limits and/or presented data at concentrations below the EPA’s final rule detection limits and/or practical quantitation limits provided in the Federal Register Notice for this final regulatory action. In addition to variable reporting limits and PFAS analytes evaluated, sample collection routines across state datasets also lacked uniformity. For more information on the collection and analysis of occurrence data, see U.S. EPA (2024g).

For both simulated occurrence data and state-sampled occurrence data, system-level geometric means were calculated to represent a typical concentration of a single sample for each PFAS analyte in a system. The concentrations of samples are log-normally distributed for all four PFAS analytes (PFOA, PFOS, PFHxS, PFHpA), meaning that while most samples have low concentrations, some may have much higher concentrations.

8.3.1.2.2 PWS Service Area Boundaries

For CWSs and NTNCWSs that had PFAS occurrence data sampled under UCMR 3 or PFAS occurrence data collected by states, the EPA acquired or estimated service area boundaries. Since TNCWSs have changing populations throughout the year, they were not included in this analysis. Data were categorized by the availability of PWS service areas, those with predelineated PWS service areas (categories 1 and 4), and those where zip codes served by PWSs were used to approximate PWS service area boundaries (categories 2 and 5). When available, predelineated PWS service areas were prioritized over zip code-approximated PWS service area boundaries. The EPA used the federal version of the SDWIS/Fed to inform the type of water system (e.g., CWS, NTNCWS), population served, identify Native American-owned

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[90] States include: Alabama, Colorado, Illinois, Kentucky, Massachusetts, Michigan, New Hampshire, New Jersey, North Dakota, Ohio, South Carolina, and Vermont.

[91] The EPA evaluated the difference between using 10 percent (0.02 ppt) and 50 percent (1 ppt) of the minimum reported sample concentration for all analytes. The difference in population estimates from this change was less than 0.5 percent for all analytes. 10 percent of the minimum reported value was used in the analysis (0.02 ppt).

PWSs, and determine activity status for PWSs included in the analysis. Only active systems, as identified in SDWIS/Fed fourth quarter 2021, were included.

For predelineated PWS service area boundaries, the EPA aggregated spatial data from a variety of sources spanning multiple file formats into one ESRI file geodatabase.[92] Data sources are provided in Table 8-2.

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[92] File formats included: ESRI ArcGIS Online (AGOL) layers, shapefiles, and GeoJSON.

Table 8-2: Data Sources for Predelineated PWS Service Areas

Accessed Through State Sources or EPA Correspondence

State	Source Name	Link	Date
CO	State of Colorado – Water District Boundaries	https://data.colorado.gov/Water/Water-District-Boundaries/82ke-q8t2	Accessed 1/26/2022
CA	State of California – Division of Drinking Water, California Water Resources Control Board	https://gispublic.waterboards.ca.gov/portal/home/item.html?id=fbba842bf134497c9d611ad506ec48cc	Accessed 1/31/2022
NJ	EPA correspondence	EPA Office of Ground Water and Drinking Water	Accessed 1/31/2022
NM	State of New Mexico – water data	https://catalog.newmexicowaterdata.org/dataset/5d069bbb-1bfe-4c83-bbf7-3582a42fce6e/resource/037d915d-4a28-4c39-9922-3556ec492698/download/nm_pws_areas.zip	Accessed 1/26/2022
NY	State of New York – Department of Health	https://water.ny.gov/doh2/applinks/waterqual/assets/PWS_GeoJson3.json	Accessed 1/31/2022
OK	State of Oklahoma – Water Resources Board	https://www.owrb.ok.gov/maps/data/layers/Water%20Supply/ws_system_service_areas.htm; https://owrb.maps.arcgis.com/apps/webappviewer/index.html?id=68c5f3fd492a43ee8386f39a80f88afb	Accessed 1/26/2022
PA	State of Pennsylvania – Department of Environmental Protection	https://newdata-padep-1.opendata.arcgis.com/datasets/public-water-systems-public-water-supplier-service-areas/explore?location=40.917958%2C-77.621150%2C8.24	Accessed 1/12/2022
RI	EPA correspondence	EPA Office of Ground Water and Drinking Water	Accessed 1/31/2022

Accessed through EPA ArcGIS Online Portal

State	Source	Link	Date
AR	EPA ArcGIS – Portal	https://epa.maps.arcgis.com/home/item.html?id=59eb7810caa044678f1e26e637b4fa79	Accessed 12/7/2021
AZ	EPA ArcGIS – Portal		
CT	EPA ArcGIS – Portal		
KS	EPA ArcGIS – Portal		
MO	EPA ArcGIS – Portal		
MS	EPA ArcGIS – Portal		
TX	EPA ArcGIS – Portal		
UT	EPA ArcGIS – Portal		
NC	EPA ArcGIS – Portal	https://www.nconemap.gov/search?groupIds=9eb59a7bdc8e4bdf8cbe2488c8584552	Accessed 1/10/2021

Under UCMR 3 and 4, PWSs sampled were asked to report U.S. Postal Service zip code(s) for all areas being served water by a PWS. As such, when pre-delineated PWS service area boundaries were unavailable, the EPA used zip codes served by PWSs to delineate approximated boundaries using the following steps:

- The EPA joined zip codes served—as specified for PWSs in UCMR 3 (U.S. EPA, 2017) and UCMR 4 (U.S. EPA, 2022c)—to a zip code polygon layer that represented postal service delivery areas.
- The EPA projected zip codes served by PWSs.
- In cases where zip codes did not have polygons (i.e., zip codes for post offices and large volume mail customers), to map these zip codes as approximate service areas, the EPA selected and overlaid zip code points for each service area with zip code polygons to select the polygon at that location. Then, the EPA merged and dissolved all zip codes (both point- and polygon-based) to map each service area.
- The EPA aggregated all zip code polygons served by each PWS into one boundary representative of PWS service area boundaries.
- In instances where one zip code was served by multiple PWSs, the EPA included the zip code boundary in all corresponding PWS service area boundaries. For example, if one zip code was served by two PWSs, both PWS service area boundaries would contain the same zip code region represented in their boundaries. In some cases, this resulted in the EPA referencing the same population demographic composition for multiple systems; however, the populations were not double-counted because population-served data were obtained from SDWIS/Fed and were unique to each PWS.

PWSs with pre-delineated PWS service areas (categories 1 and 4), account for 38.4 percent of all PWSs included in the analysis. PWSs with zip code delineated boundaries (categories 2 and 5), account for 61.6 percent of all PWSs included in the analysis.

Because there is greater accuracy with the predelineated PWS service areas, and to reduce double-counting of affected populations, the EPA removed the portion of the zip code boundaries that were already accounted for within the predelineated PWS service area boundaries.

For example, in rural areas, the zip code boundaries can be relatively large and therefore overlap with predelineated PWS service area boundaries. To avoid redundancy and reduce bias from potentially counting populations outside a service area in the demographic composition of a system, the EPA used the following approach:

- The EPA used predelineated PWS service area boundaries (including overlap[93]) when available.

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[93] For PWSs with predelineated PWS service area boundaries, the EPA conducted a sensitivity analysis of the results of the EPA's EJ exposure analysis to evaluate the impact of retaining PWS boundaries including overlapping areas versus removing overlapping boundaries. The impact on the results of the EPA's EJ exposure analysis showed very few differences across the two approaches. As such, the EPA used service area boundaries with overlapping areas included.

- If predelineated PWS service areas were not available, the EPA used zip code-approximated PWS service area boundaries (as provided in UCMR 3 and UCMR 4).

The EPA carved out or removed predelineated PWS service area boundaries from the zip code-approximated PWS service area boundaries to reduce the risk of double-counting the demographic composition of the populations served.

The EPA used predelineated PWS service area boundaries and zip code-approximated PWS service area boundaries as inputs to the EJSCREENbatch R package to estimate the sociodemographic characteristics of PWS service areas included in the analysis (see Section 8.3.1.2.3 for more detail on this process) (U.S. EPA, 2022a). The population served counts were obtained from SDWIS/Fed for each PWS. Further description of the population-served data and sociodemographic characteristics of the population served by PWS service areas is provided in Section 8.3.2.1 and in Appendix M.

8.3.1.2.2.1 Categories 1 and 2

Categories 1 and 2 contained PWSs that had sampled PFAS occurrence data from UCMR 3. Category 1 (n = 1,707 PWSs) comprised PWSs that had predelineated PWS service area boundaries, whereas category 2 (n = 3,036 PWSs) comprised PWSs that had zip code-approximated PWS service area boundaries.

The exposure analysis included service areas for 1,707 category 1 PWSs and 3,036 category 2 PWSs, for a total of 4,743 PWSs. There were 4,920 PWSs that conducted PFAS sampling under UCMR 3, and categories 1 and 2 PWSs accounted for approximately 96 percent of all PWSs that participated in UCMR 3. Of the 4,920 PWSs that participated in UCMR 3, 10 PWSs did not have predelineated PWS service area boundaries or zip code-served data available to approximate PWS service area boundaries. Systems were excluded from the analysis if they were classified as "inactive" in SDWIS/Fed (67 PWSs). Additionally, PWSs could not be evaluated if there were errors processing the EJSCREENbatch R package (100 PWSs). The majority of these systems are located in US territories.[94] In such instances, the EJSCREENbatch R package did not provide sociodemographic characteristics for a given PWS service area.

Category 1 and 2 PWSs account for 252.2 million people served, or approximately 76 percent of the U.S. population. However, the subset of category 1 and 2 PWSs captured in the analysis represented roughly 3 percent of active PWSs.[95]

8.3.1.2.2.2 Categories 4 and 5

The EPA used state PFAS occurrence data for PWSs in categories 4 and 5 because these systems did not monitor for PFAS under UCMR 3. Category 4 (n = 440 PWSs) included PWSs that had predelineated PWS service areas, whereas category 5 (n = 296 PWSs) included PWSs that had zip code-approximated PWS service area boundaries.

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[94] These included 69 PWSs in Puerto Rico, 3 PWSs in the Virgin Islands, 2 PWSs in Guam, 1 PWS in American Samoa, and 1 system in the Northern Mariana Islands.

[95] The number of active public water systems was retrieved from SDWIS/Fed fourth quarter 2021.

The EJ exposure analysis includes PWS service areas for 440 category 4 PWSs and 296 category 5 PWSs. Category 4 and 5 PWSs account for approximately 5 percent of all PWSs with state PFAS sample occurrence data. 1,143 PWSs with state PFAS occurrence data have PFAS occurrence data available in UCMR 3, and therefore are included in the analysis under categories 1 and 2. In addition, the EPA included PWSs with state PFAS occurrence data in the analysis only if finished water samples were available for at least one of the four PFAS analytes. The agency could not include many of the PWSs with state PFAS occurrence data because predelineated PWS service areas or zip code approximated PWS service area boundaries were not available.

Category 4 and 5 PWSs account for 2 million people served, or approximately 0.6 percent of the U.S. population. The EPA summarized the results for these PWSs in Appendix M.

8.3.1.2.2.3 Categories 3 and 6

The EPA did not include category 3 and 6 PWSs in the EJ exposure analysis because predelineated PWS service areas and information containing zip codes served by PWSs were both unavailable.

8.3.1.2.3 Sociodemographic Data

The EPA used version 2.0.1 of the agency's EJSCREENbatch R package to characterize the sociodemographic makeup of populations living in PWS service areas, as described in Section 8.3.1.2.2 (U.S. EPA, 2022a). The EJSCREENbatch R package offers functions to extract and process Census block group EJScreen data within user-provided geographies. This analysis relies on 2021 EJScreen data, which corresponds to demographic estimates from the U.S. Census Bureau's ACS 2015–2019 five-year sample (U.S. EPA, 2022a). EJScreen data are input into a function that spatially apportions (i.e., using areal apportionment) data to service areas using a 1 km resolution raster population dataset from NASA's Socioeconomic Data and Applications Center.

The EPA used the following data outputted from the EJSCREENbatch package on the race, ethnicity, and poverty status of populations served by the PWSs:

- Race: Percent non-Hispanic American Indian or Alaska Native; percent non-Hispanic Asian; percent non-Hispanic Black or African American; percent non-Hispanic White, and percent non-Hispanic Pacific Islander.[96]
- Ethnicity: Percent Hispanic.
- Income: Percent of the population below twice the Federal poverty level; percent of the population above twice the Federal poverty level.

In addition, the agency identified PWSs that are Native American-owned and within the EPA's tribal primacy program using SDWIS/Fed data (U.S. EPA, 2021h).

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[96] In an effort to avoid double counting populations, race/ethnicity categories reported here do not account for people who selected "some other race alone" or "two or more races" in the ACS.

Note that sociodemographic information used for the EPA's EJ exposure analysis includes additional demographic groups from those used in the EPA's benefits analysis, which relies on SDWIS/Fed and race/ethnicity-specific population estimates from the U.S. Census Bureau (2020a). Population estimates from the U.S. Census Bureau are available at the county level, but more granular location-specific population and demographic information was needed for the EPA's EJ exposure analysis. In particular, this analysis presents non-Hispanic American Indian or Alaska Native, non-Hispanic Asian, and non-Hispanic Pacific Islander instead of the Other demographic category employed in Section 8.4. Both analyses include the demographic categories non-Hispanic Black, Hispanic, and non-Hispanic White. For further information on the use of U.S. Census Bureau population proportions in the EPA's benefits analysis, see Appendix B.

Based on public comment provided on this proposed rule, the EPA has disaggregated the Asian and Pacific Islander demographic group into two separate categories for the final rule analysis; one representing Asian populations and the other Pacific Islander populations. The EPA disaggregated these demographic groups to ensure the prior aggregated category for Asian and Pacific Islander populations would not mask exposures and impacts specific to various ethnic subpopulations that fall under the broader Asian and Pacific Islander designation. These subpopulations vary in language, culture, and historic, social, economic, and environmental experiences; these differences contribute to unique social determinants of health, which could lead to disparate environmental exposures, impacts, and health outcomes (Look et al, 2020; Bhakta 2022). An aggregate Asian and Pacific Islander demographic group that encapsulates these various subpopulations may obscure possible disparities that exist across subpopulations (Quint et al, 2021). For the final rule analysis, the EPA disaggregated this group to investigate such possible disparities among the diverse subpopulations.

8.3.1.3 EJ Exposure Analytic Approach

The EPA conducted a baseline analysis of populations served by PWS service areas in categories 1 and 2 to evaluate the demographic characteristics of systems exposed to PFAS concentrations above a baseline set of thresholds and two hypothetical regulatory thresholds.

For purposes of this baseline analysis, the EPA assumed the following baseline thresholds are intended to reflect trigger levels of one-half of the MCL values for PFOA, and PFOS. For consistency, the EPA also applied these baseline thresholds for PFHxS and PFHpA. Note that the following values are slightly higher than Method 537.1 detection limits (U.S. EPA, 2018):[97,98,99]

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[97] There are no detection limits reported for Method 533 (U.S. EPA, 2019b).

[98] The EPA used these detection limits solely as baseline thresholds for purposes of its EJ analysis. The EPA has defined the Rule Detection Limit for purposes of consideration of monitoring data to determine monitoring schedules as 1/2 the MCL for PFOA and PFOS, or 1.3 ppt. Refer to Sections VI, VIII, and IX of the federal register notice for this proposed regulatory action for further discussion on the EPA's analytical methods and the determination of practical quantitation limits (PQLs).

[99] As noted in Section 8.3.1.2.1, different states utilized various reporting, quantification, and/or detection limits when analyzing and presenting data, and for some states, no clearly defined limits were publicly provided as part of the dataset. Further, the limits often varied within the data for each state depending on the specific PFAS analyte. In some cases, states reported detection, quantification, or reporting limits and/or presented data at concentrations below the EPA's proposed rule detection limits and/or practical quantitation limits provided in the federal register notice for this proposed regulatory action. For more information on the collection and analysis of occurrence data, see U.S. EPA (2022h).

- PFHpA: 2 ppt
- PFHxS: 2 ppt
- PFOS: 2 ppt
- PFOA: 2 ppt

The EPA also evaluated the rate of exposure using two hypothetical regulatory thresholds: (1) the UCMR 5 MRL values for each PFAS analyte, and (2) 10.0 ppt. For the purpose of this analysis, these values are assumed to be individual regulatory thresholds for each contaminant. The EPA notes that while these thresholds are not exactly set at the final or regulatory alternative MCL values, the EPA began this analysis prior to refinement of those regulatory options. This analysis is not intended to determine the demographic breakdown of costs and benefits expected to result from the final rule and alternatives; rather, this analysis determines whether overburdened communities are disproportionately exposed to PFAS over baseline conditions and these hypothetical thresholds. The UCMR 5 MRL values for PFOA, PFOS, PFHpA, and PFHxS are as follows:

- PFHpA: 3 ppt
- PFHxS: 3 ppt
- PFOS: 4 ppt
- PFOA: 4 ppt

The EPA compared the estimated population served in each demographic group anticipated to experience reductions in PFAS exposure under each hypothetical regulatory threshold to the total population served across all demographic groups. This analysis seeks to answer the following question: When PFAS occurs in drinking water over a certain threshold, will overburdened communities be disproportionately exposed to PFAS compared to the total population that is exposed to PFAS over the same threshold?

As described above, the EPA's EJ exposure analysis for the final rule uses data from EJScreen and the American Community Survey to examine anticipated exposure above set baseline and theoretical regulatory thresholds using system-level mean occurrence data. As the literature shows, the degree to which a community experiences PFAS exposure above a specific threshold can vary. As such, the EPA also characterized population-weighted mean concentrations of PFAS to evaluate the extent to which the levels of potential exposure correlate with community characteristics.

8.3.2 EJ Exposure Analysis Results

This section describes the demographic characterization of category 1 and 2 PWS service areas in the baseline as well as the results of the analysis exploring the EJ implications of two hypothetical regulatory thresholds. The EPA focused on category 1 and 2 PWS service areas due to the availability of spatial boundaries (from both predelineated PWS service area boundaries and zip code-approximated PWS service area boundaries) and PFAS occurrence data from UCMR 3. Results from categories 4 and 5 are reported in Appendix M.

8.3.2.1 Demographic Profile of PWS Service Areas

Table 8-3 summarizes the breakdown of category 1 and 2 PWS service areas by state and by size, where small systems are those serving fewer than or equal to 10,000 people. In total, these PWSs account for roughly 252 million people served, or approximately 76 percent of the U.S. population. Category 1 and 2 PWSs span all states in the continental U.S. Category 1 and 2 PWSs included in this analysis capture roughly 3 percent of active PWSs. Among the 3 percent of active PWSs captured by the EPA's analysis (i.e., category 1 and 2 PWSs), there are 26 PWSs within the EPA's tribal primacy program, serving a population of approximately 306,000 people. Additionally, approximately 17 percent of the systems are defined as small (serving fewer than 10,000 people), accounting for 1.3 percent of the total population served.

Table 8-4 summarizes the demographic profile for category 1 and 2 PWS service areas and compares it to the demographic characteristics of the overall U.S. population. There are slight differences in the demographic characteristics of the population served by PWS service areas included in the EPA's analysis compared to the overall U.S. population, with percent differences all being less than +/- 4.1 percent. The population served by these PWSs has slightly higher percentages of Asian (+0.8%) and Black (+1.5%) populations compared to the overall U.S. population. The percentage of American Indian or Alaska Native and Pacific Islander populations is consistent with the percent of these populations across the U.S. The Hispanic population served by category 1 and 2 PWSs is slightly higher (+2.3%) and the non-Hispanic White population is lower (-4.1%) than that of the overall U.S. population. When examining income demographics, Table 8-4 shows that category 1 and 2 PWSs have a slightly higher percentage of populations with income below twice the Federal poverty level (+1.4%) and a slightly lower percentage of population with income above twice the Federal poverty level (-1.4%) compared to the overall U.S. population.

Table 8-3: Number of Category 1 and 2 PWSs and Populations Served by Size and State

State	Number of Total Service Areas	Number of Small Service Areas	Total Population Served[a]	Population Served in Small Systems[a]	Population Served in Medium and Large Systems
Tribal Service Areas	26	12	305,846	36,235	269,611
Alabama	124	19	4,488,042	86,106	4,401,936
Arizona	75	14	5,897,987	52,559	5,845,428
Arkansas	63	18	1,786,895	81,217	1,705,678
California	451	38	36,995,867	149,032	36,846,835
Colorado	81	13	5,298,922	54,590	5,244,332
Connecticut	42	6	2,457,248	13,799	2,443,449
Delaware	13	3	642,261	13,535	628,726
District of Columbia	3	0	676,068	0	676,068
Florida	259	28	19,366,933	111,293	19,255,640
Georgia	124	20	8,752,508	77,382	8,675,126
Idaho	26	6	991,096	16,854	974,242
Illinois	252	32	9,702,346	120,173	9,582,173
Indiana	101	20	3,792,604	63,428	3,729,176
Iowa	57	15	1,810,021	52,241	1,757,780
Kansas	45	14	1,999,477	50,363	1,949,114
Kentucky	119	26	3,599,670	172,624	3,427,046
Louisiana	88	24	3,363,018	84,804	3,278,214
Maine	16	3	411,385	16,456	394,929
Maryland	39	8	4,980,513	20,084	4,960,429
Massachusetts	171	15	6,236,022	74,117	6,161,905
Michigan	158	25	5,895,618	122,403	5,773,215
Minnesota	98	14	3,478,561	40,952	3,437,609
Mississippi	77	24	1,399,379	86,698	1,312,681
Missouri	86	21	3,879,698	87,393	3,792,305
Montana	15	6	416,576	10,070	406,506
Nebraska	21	7	1,136,091	12,642	1,123,449

Table 8-3: Number of Category 1 and 2 PWSs and Populations Served by Size and State

State	Number of Total Service Areas	Number of Small Service Areas	Total Population Served[a]	Population Served in Small Systems[a]	Population Served in Medium and Large Systems
Nevada	16	4	2,826,471	10,200	2,816,271
New Hampshire	23	5	570,449	10,907	559,542
New Jersey	173	17	8,123,044	54,089	8,068,955
New Mexico	28	5	1,442,144	7,457	1,434,687
New York	169	32	15,965,142	98,790	15,866,352
North Carolina	147	21	7,307,497	82,447	7,225,050
North Dakota	12	3	425,637	4,903	420,734
Ohio	184	28	8,971,538	113,929	8,857,609
Oklahoma	66	16	2,533,092	57,411	2,475,681
Oregon	65	11	2,875,275	33,730	2,841,545
Pennsylvania	174	34	9,402,219	130,731	9,271,488
Rhode Island	17	2	934,307	12,485	921,822
South Carolina	80	9	3,475,385	46,773	3,428,612
South Dakota	18	5	458,464	17,065	441,399
Tennessee	137	16	6,143,130	86,951	6,056,179
Texas	383	92	21,617,805	370,158	21,247,647
Utah	62	8	2,595,756	32,847	2,562,909
Vermont	12	6	142,888	23,438	119,450
Virginia	80	13	6,263,605	48,692	6,214,913
Washington	132	20	6,304,525	70,712	6,233,813
West Virginia	32	8	844,387	30,705	813,682
Wisconsin	92	18	2,920,851	82,496	2,838,355
Wyoming	11	2	268,828	3,341	265,487
TOTAL	4,743	806	252,173,091	3,137,307	249,035,784

Abbreviations: PWS – public water system.

Note:

[a] Population served by PWSs was obtained from SDWIS/Fed fourth quarter 2021. Small systems include those serving fewer than or equal to 10,000 people. Medium and large systems serve populations more than 10,000 people.

Table 8-4: Population Served by Category 1 and 2 PWSs Compared to Percent of U.S. Population by Demographic Group

	Race/Ethnicity						Income		
	Non-Hispanic American Indian or Alaska Native	Non-Hispanic Asian	Non-Hispanic Black	Non-Hispanic Pacific Islander	Hispanic	Non-Hispanic White	Below Twice the Poverty Level	Above Twice the Poverty Level	Total Population Served
Population Served	1,140,247	16,168,317	34,581,847	366,278	51,710,333	141,147,967	78,625,592	173,547,499	252,173,091
Percent of Total Population Served	0.5%	6.4%	13.7%	0.10%	20.50%	56.00%	31.2%	68.8%	100.0%
U.S. Population Percent by Demographic Group[a]	0.6%	5.6%	12.2%	0.20%	18.20%	60.10%	29.8%	70.2%	-
Percent Difference Between Population Served and U.S. Population	-0.1%	0.8%	1.5%	-0.10%	2.30%	-4.10%	1.4%	-1.4%	-

Note:

[a] U.S. population estimates were obtained from the U.S. Census Bureau's American Community Survey 2016–2020 five-year estimates.

8.3.2.2 Exposure Analysis Results

8.3.2.2.1 Baseline Scenario

To evaluate impacts of the final rule on population groups of concern, the percent of a specific demographic group with modeled PFAS above baseline thresholds needs to be presented in relation to another group, typically referred to as a comparison group. The way in which the comparison group is defined can have important implications for identifying differences in potential exposure across population groups of concern in an EJ analysis. The agency's *Technical Guidance for Assessing Environmental Justice in Regulatory Analysis* notes that the comparison group can be defined as individuals with similar socioeconomic characteristics across different areas in the state, region or nation (i.e., within-group comparison) or as affected individuals with different socioeconomic characteristics (i.e., across-group comparison) (U.S. EPA, 2016h).

For this final regulatory action, the EPA examines individuals served by PWSs with modeled PFAS occurrence above the baseline concentration threshold or a specific hypothetical alternative policy threshold. The EPA presents the total affected population as a possible metric of comparison, noting however that each affected demographic group is reflected also within the total affected population. It is possible that the EPA understates the magnitude of disproportionate baseline exposure to PFAS for populations of concern by using the total affected population as the basis of comparison. For this reason, the EPA also makes comparisons between affected population groups of concern and the mutually exclusive affected non-Hispanic White population or the affected population with income above twice the Federal poverty level.

As currently defined, race and ethnicity classifications are presented in a disaggregated form such that racial categories include individuals who identify as non-Hispanic, while the Hispanic category includes individuals of any race who identify with Hispanic ethnicity. In aggregate, those who identify (1) as a race other than White and/or (2) identify with Hispanic ethnicity are considered "people of color" when considering potential EJ concerns. The EPA has therefore included the category non-Hispanic White in the analysis, as this category does not include individuals who identify as a race or ethnicity included within "people of color".

The results of the EPA's analysis of baseline exposure are shown in Table 8-5 and Table 8-6. Table 8-5 summarizes the population served by category 1 and 2 PWSs with modeled PFAS occurrence above baseline thresholds based on a trigger level of 2 ppt for each PFAS analyte, which is slightly above the Method 537.1 detection limits. The second set of rows in Table 8-5 summarizes the percentage of each demographic group with modeled PFAS occurrence above these baseline thresholds. Table 8-6 shows average population-weighted PFAS concentrations across demographic groups. In Table 8-5, percentages are bolded and italicized when the percentage of the population in a specific demographic group with modeled PFAS above the baseline threshold is greater than the percentage of the total population across all demographic groups exposed to modeled PFAS above this threshold (right-hand column). In, the highlighted numbers represent where percentages of the population served in a particular demographic group are more than 1 percentage point greater than percentages of the total population. In Table 8-6, highlighted cells represent whether the average concentration for a given demographic group is higher than the average for the total population served across all demographic groups (right-hand column). Higher percentages or concentrations indicate higher PFAS exposure for a given demographic group compared to the percentage of the population served across all demographic groups. Between 4.7 percent and 10.8 percent of the total population served by category 1 and 2 PWS service areas, depending on the analyte, are exposed to modeled PFAS occurrence above baseline thresholds based on a trigger level of 2 ppt for each PFAS analyte.

The following are findings from the EPA's baseline EJ exposure analysis:[100]

- The percentage of Hispanic populations served with exposure to PFAS above baseline thresholds is higher across all four PFAS analytes compared to the percentage of the total population served across all demographic groups with anticipated PFAS exposure above baseline thresholds. All percentages are more than 1 percentage point greater than percentages exposed across the total population, ranging from 1.3 - 2.6 percentage points higher. These percentages are also higher than those of non-Hispanic White populations by 2.1 - 3.5 percentage points.
- The percentage of non-Hispanic Black populations served with exposure to PFAS above baseline thresholds is higher across all four PFAS analytes compared to the percentage of the total population served across all demographic groups. Exposure is at least one percentage point greater for PFOA and PFOS and less than 1 percentage point greater for PFHxS and PFHpA, with a range of 0.3 - 1.6 percentage points difference. The percentage of non-Hispanic Black populations exposed is also greater than the percentage of non-Hispanic White populations for all four PFAS analytes. The difference in percentage exposed between Black and non-Hispanic White populations ranges from 0.9 - 2.4 percentage points.
- The percentage of non-Hispanic American Indian or Alaska Native populations served have greater PFHxS exposure above its baseline threshold compared to the total population served across all demographic groups, and exposures to PFOS, PFHpA, and PFOA are similar to or less than the percentages exposed across all demographic groups. Exposure to PFHxS, PFHpA, and PFOA exposure above the baseline thresholds is higher for non-Hispanic American Indian or Alaska Native populations in comparison to the non-Hispanic White population by 0.3 to 1.8 percentage points.
- The percentage of non-Hispanic Asian populations served with exposure above baseline thresholds is comparable or less than the percentages of the population served across all demographic groups. When compared to non-Hispanic White populations, the percentage of non-Hispanic Asian populations served with exposure above baseline thresholds is 1 percentage point higher for PFOS but less than the exposure for non-Hispanic White populations for PFHxS, PFHpA, and PFOA.
- Other demographic groups, including non-Hispanic Pacific Islanders and those representing relative income status, are anticipated to experience percentages of PFAS occurrence above baseline thresholds similar to (within 0.5%) or less than the percentage of the population served across all demographic groups facing exposure above baseline thresholds.

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[100] Although differences in anticipated exposure between a particular demographic group and the entire sample population are <5%, all results are reported in the EPA's summary of results regardless of magnitude.

Table 8-6 characterizes population-weighted mean concentrations of PFAS by demographic group. In addition to having a higher percentage of populations served by PWSs with concentrations of PFAS above baseline thresholds, Hispanic and non-Hispanic Black populations are also exposed to higher mean concentrations than is typical for the total population served and average population-weighted exposures for non-Hispanic White populations. On average, Hispanic populations are exposed to 0.1-0.2 ppt more of each of the four PFAS analytes examined than non-Hispanic White populations served. Differences in average exposure between non-Hispanic Black populations and non-Hispanic White populations are close to or less than 0.1 ppt.

The results also suggest that non-Hispanic American Indian and Alaska Native as well as non-Hispanic Pacific Islander populations have greater exposure to PFHxS and PFOA in comparison to the total population served and the non-Hispanic White population. Non-Hispanic Pacific Islander populations have the highest average exposures to PFHxS and PFOA of any demographic group, while Hispanic populations are the most highly exposed to PFOS and PFHpA. The findings of differential population-weighted average exposure for non-Hispanic American Indian or Alaska Native as well as Pacific Islander populations was not observed in Table 8-5 except with respect to PFHxS for non-Hispanic American Indian or Alaska Native populations; this difference in results suggests that these populations may be exposed to higher average PFHxS and PFOA concentrations when exposure does occur, however these populations are not always more likely to be served by public water systems with above baseline concentrations of PFAS.

In addition, low-income populations are exposed to higher average concentrations of PFHxS, PFHpA, and PFOA in comparison to the total population served and to populations with income above twice the Federal poverty level, although differences are all less than 0.1 ppt.

Table 8-5: Baseline Scenario: Population Served by Category 1 and 2 PWS Service Areas Above Baseline Thresholds and as a Percent of Total Population Served

PFAS	Race/Ethnicity						Income		Total Population Served
	Non-Hispanic American Indian or Alaska Native	Non-Hispanic Asian	Non-Hispanic Black	Non-Hispanic Pacific Islander	Hispanic	Non-Hispanic White	Below Twice the Poverty Level	Above Twice the Poverty Level	
Population Served Above Baseline Threshold									
PFOS	96,464	1,761,960	4,130,816	25,193	6,263,677	14,156,536	8,035,262	19,075,300	27,110,562
PFHxS	79,130	802,126	2,303,033	20,823	4,458,586	7,184,715	5,095,233	10,132,796	15,228,029
PFHpA	52,579	602,837	1,732,707	12,312	3,459,309	5,729,697	3,670,395	8,188,555	11,858,950
PFOA	88,674	1,069,233	3,423,882	19,851	5,202,088	10,561,078	6,651,205	14,211,140	20,862,345
Population Served Above Baseline Threshold as a Percent of Total Population Served									
PFOS	8.5%	***10.9%***	***11.9%***	6.9%	***12.1%***	10.0%	10.2%	***11.0%***	10.8%
PFHxS	***6.9%***	5.0%	***6.7%***	5.7%	***8.6%***	5.1%	***6.5%***	5.8%	6.0%
PFHpA	4.6%	3.7%	***5.0%***	3.4%	***6.7%***	4.1%	4.7%	4.7%	4.7%
PFOA	7.8%	6.6%	***9.9%***	5.4%	***10.1%***	7.5%	***8.5%***	8.2%	8.3%

Abbreviations: PFHpA – perfluoroheptanoic acid; PFHxS – perfluorohexanesulfonic acid; PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid.

Table 8-6: Modeled Average PFAS Concentrations (ppt) by Demographic Group in the Baseline, Category 1 and 2 PWS Service Areas

	Race and Ethnicity						Income		
PFAS	**Non-Hispanic American Indian or Alaska Native**	**Non-Hispanic Asian**	**Non-Hispanic Black**	**Non-Hispanic Pacific Islander**	**Hispanic**	**Non-Hispanic White**	**Below Twice the Poverty Level**	**Above Twice the Poverty Level**	**Total Population Served**
PFOS	0.97	1.01	1.05	0.90	1.15	0.96	1.01	1.02	1.01
PFHxS	0.81	0.58	0.64	0.86	0.75	0.59	0.64	0.62	0.63
PFHpA	0.53	0.50	0.55	0.51	0.64	0.50	0.54	0.53	0.53
PFOA	1.05	0.85	1.03	1.14	1.11	0.89	0.99	0.94	0.96

Abbreviations: PFHpA – perfluoroheptanoic acid; PFHxS – perfluorohexanesulfonic acid; PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid.

8.3.2.2.2 Hypothetical Regulatory Scenario #1: UCMR 5 MRLs

Table 8-7 and Table 8-8 summarize the results for population served by category 1 and 2 PWSs with PFAS occurrence above UCMR 5 MRL values. For this hypothetical regulatory scenario, the EPA assumed that PWSs with PFAS system-level means above the MRL value will reduce PFAS levels to comply with the final rule. The first set of rows in Table 8-7 summarizes populations served by category 1 and 2 PWS service areas with modeled PFAS occurrence above the UCMR 5 MRLs. The second set of rows provides these estimates as a percentage of the total population served by PWSs included in the EPA's analysis. Table 8-8 summarizes the population-weighted average reductions in PFAS assuming all PWSs reduce their concentrations to UCMR 5 MRL levels.

In Table 8-7, percentages are bolded and italicized when the percentage of the population in a specific demographic group with PFAS occurrence above the MRL value is greater than the percentage of the total population across all demographic groups with PFAS occurrence above the MRL (right-hand column). In Table 8-7, the highlighted numbers represent where percentages of the population served in a particular demographic group are more than 1 percent greater than percentages of the total population. In Table 8-8, highlighted cells represent whether the average reduction in PFAS concentrations for a given demographic group is higher than the average for the total populations served across all demographic groups (right-hand column). The percentages that are bolded, italicized, or highlighted indicate higher PFAS exposure above the MRL for a given demographic group; the EPA anticipates that relatively higher reductions in PFAS exposure will accrue to these demographic groups under this hypothetical regulatory scenario compared to the percentage of the population across all demographic groups. The EPA provides additional details on anticipated exposure above UCMR 5 MRL values in Appendix M.

Between 3 percent and 5.4 percent of the population served by category 1 and 2 PWS service areas, depending on the PFAS analyte, are exposed to modeled PFAS concentrations above the UCMR 5 MRL values for PFOS, PFOA, PFHpA, and PFHxS. Under this hypothetical regulatory scenario, where MCLs are assumed to be equal to UCMR 5 MRL values, the EPA expects these populations to experience reductions in PFAS exposure to below the hypothetical regulatory thresholds. The EPA's analysis of the demographic distribution of anticipated health benefits and household costs due to reductions in PFAS exposure resulting from the final PFAS rule and regulatory alternatives is discussed in Section 8.4.2.

Based on this analysis, non-Hispanic American Indian or Alaska Native, non-Hispanic Black, Hispanic, and low-income populations are estimated to face higher rates of system-level mean PFAS exposure above UCMR 5 MRL values compared to rates of exposure over these thresholds for the total population served across all demographic groups. The differences are even greater when compared to the rates of exposure over these thresholds for non-Hispanic White populations. Specifically, non-Hispanic American Indian or Alaska Native populations served have higher exposure above the UCMR 5 MRL values for PFOA, PFHxS, and PFHpA compared to the percent of the population served across all demographic groups by 0.5 to 1.1 percentage points. These differences in exposure are 1.1 to 2.1 percentage points greater when compared to non-Hispanic White populations. Non-Hispanic Black populations served have higher exposure above the UCMR 5 MRL values for all four PFAS analytes compared to the percent of the total population served across all demographic groups, although these differences are all less than 0.5 percentage points. In comparison to the non-Hispanic White population, non-Hispanic Black populations have higher exposure above the UCMR 5 MRLs for all PFAS analytes examined, with a range of 0.7 to 1.2 percentage points greater exposure. Hispanic populations served have higher exposure above the UCMR 5 MRL values across all four PFAS analytes compared to the percent of the total population served across all demographic groups. The percent of Hispanic populations served with exposure above the UCMR 5 MRL values is at least double the percent of non-Hispanic White populations with exposure above the UCMR 5 MRL values for PFHxS and PFHpA and at least 2 percentage points greater for all PFAS analytes. This is the most notable difference in exposure for a single demographic group. The percent differences observed suggest that, in this analysis, Hispanic populations are estimated to face the highest baseline levels of exposure to all four PFAS analytes compared to the entire sample population across all demographic groups. As such, these Hispanic populations could also be expected to experience the greatest reductions in PFAS exposure under this hypothetical regulatory scenario. Populations served with income less than twice the Federal poverty level have higher rates of PFAS exposure above the UCMR 5 MRL values across all four PFAS analytes compared to the percent of the population served across all demographic groups. Exposure percentages for populations served with income less than twice the Federal poverty level are also higher than exposure for populations with income above twice the Federal poverty level for all four PFAS analytes, however differences are generally relatively small at between 0.2 - 0.6 percentage points.

Table 8-8 displays population-weighted reductions in PFAS exposure in a hypothetical regulatory scenario where system-level means are reduced to UCMR 5 MRLs to comply with the final rule. Hispanic populations see the greatest reductions in concentrations for PFOS and PFHpA in this hypothetical regulatory scenario, which is consistent with higher levels of exposure for this group observed in Table 8-7. However, despite having a lower percentage of populations affected than Hispanic populations, non-Hispanic Pacific Islander populations see the greatest reduction in PFOA and PFHxS of any demographic group in this hypothetical regulatory scenario. Similarly, non-Hispanic American Indian or Alaska Native populations see relatively large reductions in PFHxS and PFOA in comparison to both the total population served and non-Hispanic White populations. Non-Hispanic Black populations see higher reductions in PFOA and PFHpA than the average across the total population served, which is consistent with the percentage of non-Hispanic Black individuals with exposure above UCMR 5 MRLs being slightly higher than percentage of the total population served across all demographic groups as observed in Table 8-7.

Table 8-7: Hypothetical Regulatory Scenario #1: Demographic Breakdown of Population Served by Category 1 and 2 PWS Service Areas Above UCMR 5 MRLs and as a Percent of Total Population Served

PFAS	**Race and Ethnicity**						**Income**		**Total Population Served**
	Non-Hispanic American Indian or Alaska Native	**Non-Hispanic Asian**	**Non-Hispanic Black**	**Non-Hispanic Pacific Islander**	**Hispanic**	**Non-Hispanic White**	**Below Twice the Poverty Level**	**Above Twice the Poverty Level**	
Population Served Above UCMR5 MRL									
PFOS	49,319	688,558	1,795,216	11,297	3,575,722	5,792,353	3,911,510	8,272,754	12,184,264
PFHxS	62,140	515,410	1,544,025	14,361	3,706,494	4,703,878	3,667,141	7,131,026	10,798,167
PFHpA	39,582	371,042	1,083,498	7,783	2,656,914	3,326,278	2,587,679	5,071,760	7,659,439
PFOA	67,366	706,541	2,026,879	11,952	3,925,638	6,642,157	4,409,705	9,273,284	13,682,989
Population Served Above UCMR 5 MRL as a Percent of Total Population Served									
PFOS	4.3%	4.3%	**5.2%**	3.1%	**6.9%**	4.1%	**5.0%**	4.8%	4.8%
PFHxS	**5.4%**	3.2%	**4.5%**	3.9%	**7.2%**	3.3%	**4.7%**	4.1%	4.3%
PFHpA	**3.5%**	2.3%	**3.1%**	2.1%	**5.1%**	2.4%	**3.3%**	2.9%	3.0%
PFOA	**5.9%**	4.4%	**5.9%**	3.3%	**7.6%**	4.7%	**5.6%**	5.3%	5.4%

Abbreviations: MRL – minimum reporting level; PFHpA – perfluoroheptanoic acid; PFHxS – perfluorohexanesulfonic acid; PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; UCMR – Unregulated Contaminant Monitoring Rule.

Table 8-8: Reductions in Average PFAS Concentrations (ppt) by Demographic Group in a Hypothetical Regulatory Scenario with Maximum Contaminant Level at the UCMR 5 MRLs, Category 1 and 2 PWS Service Areas

	Race and Ethnicity						Income		
PFAS	**Non-Hispanic American Indian or Alaska Native**	**Non-Hispanic Asian**	**Non-Hispanic Black**	**Non-Hispanic Pacific Islander**	**Hispanic**	**Non-Hispanic White**	**Below Twice the Poverty Level**	**Above Twice the Poverty Level**	**Total Population Served**
PFOS	0.18	0.17	0.19	0.16	0.26	0.18	0.19	0.19	0.19
PFHxS	0.29	0.10	0.13	0.37	0.15	0.14	0.15	0.14	0.14
PFHpA	0.03	0.02	0.06	0.05	0.06	0.04	0.05	0.04	0.04
PFOA	0.43	0.22	0.35	0.58	0.40	0.29	0.35	0.30	0.32

Abbreviations: PFHpA – perfluoroheptanoic acid; PFHxS – perfluorohexanesulfonic acid; PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid.

8.3.2.2.3 Hypothetical Regulatory Scenario #2: 10.0 ppt

Table 8-9 and Table 8-10 summarize the results of the population served by category 1 and 2 PWS service areas with modeled PFAS occurrence above 10.0 ppt. The first set of rows in Table 8-9 summarizes populations served by PWSs with PFAS occurrence above 10.0 ppt. The second set of rows displays these estimates as a percent of the total population served for PWSs included in the EPA's analysis. Table 8-10 shows the population-weighted average reduction in PFAS concentrations assuming all PWSs reduce their concentrations to 10.0 ppt.

In Table 8-9, percentages are bolded and italicized when the percentage of the population in a specific demographic group with PFAS occurrence above 10.0 ppt is greater than the percentage of the total population served across all demographic groups with PFAS occurrence above 10.0 ppt (right-hand column). In Table 8-10, highlighted cells represent whether the average reduction in PFAS concentrations for a given demographic group is higher than the average for the total populations served across all demographic groups (right-hand column). The percentages that are bolded, italicized, or highlighted indicate greater PFAS exposure above 10.0 ppt for a given demographic group compared to the total population served across all demographic groups; the EPA anticipates potentially relatively higher reductions in PFAS exposure to accrue to these demographic groups under this hypothetical regulatory scenario compared to the percentage of population across all demographic groups. Unlike the results from the EPA's exposure analysis where UCMR 5 MRLs are used as hypothetical MCL values, percentages in particular demographic groups are less than 1 percent greater than percentages across the total population. Between 0.1 percent and 1.3 percent of the population served by category 1 and 2 PWS service areas, depending on the PFAS analyte, is exposed to PFAS occurrence above 10.0 ppt.

The following are findings from the EJ exposure analysis for PFAS occurrence above 10.0 ppt:

- Non-Hispanic American Indian or Alaska Native, non-Hispanic Asian, non-Hispanic Black, and low-income populations have slightly higher PFAS exposure above 10.0 ppt for some PFAS analytes compared to the population served across all demographic groups. These results are essentially unchanged when comparing exposures above 10.0 ppt to non-Hispanic White populations.
- The most notable difference is for PFOA and PFHxS exposure for non-Hispanic American Indian or Alaska Native populations served. PFOA and PFHxS population exposure percentages are 2.5 and 1.3 percent, respectively, for non-Hispanic American Indian or Alaska Native populations served compared to 1.3 and 0.6 percent of the total population served across all demographic groups.
- Non-Hispanic Asian populations also have elevated exposure to PFOS over 10.0 ppt at 1.3 percent, whereas the percent of the total population served with PFOS above 10.0 ppt is 0.9 percent.

Table 8-10 characterizes population-weighted average reductions of PFAS by demographic group in a hypothetical regulatory scenario where system-level means are reduced to 10.0 ppt. This analysis provides similar evidence with respect to PFOA and PFHxS exposures above 10.0 ppt for non-Hispanic American Indian or Alaska Native populations as was summarized in Table 8-9. Similarly, reductions in PFOA exposure for non-Hispanic Black populations are greater than for the total population served. Notably, we observe that non-Hispanic Pacific Islander populations see greater reductions than the total population served for all PFAS analytes despite having similar percentages of the population exposed above 10.0 ppt. Reductions of PFHxS and PFOA for non-Hispanic Pacific Islander populations are at least three times greater than reductions in these PFAS for both the total population served and non-Hispanic White populations. Low-income populations see slightly greater reductions in PFOA in comparison to the total population served as well as those with income above twice the Federal poverty level.

Table 8-9: Hypothetical Regulatory Scenario #2: Demographic Breakdown of Population Served by Category 1 and 2 PWS Service Areas Above 10.0 ppt and as a Percent of Total Population Served

PFAS	**Race and Ethnicity**						**Income**		**Total Population Served**
	Non-Hispanic American Indian or Alaska Native	**Non-Hispanic Asian**	**Non-Hispanic Black**	**Non-Hispanic Pacific Islander**	**Hispanic**	**Non-Hispanic White**	**Below Twice the Poverty Level**	**Above Twice the Poverty Level**	
Population Served Above 10.0 ppt									
PFOS	6,398	202,496	223,186	2,212	504,833	1,379,440	666,066	1,703,438	2,369,504
PFHxS	14,318	77,674	204,849	2,500	237,828	1,041,439	493,350	1,136,891	1,630,241
PFHpA	1,674	11,466	74,657	722	52,522	218,417	119,836	251,455	371,291
PFOA	28,563	178,353	535,779	5,607	716,412	1,775,254	1,143,134	2,185,557	3,328,691
Population Served Above 10.0 ppt as a Percent of Total Population Served									
PFOS	0.6%	**1.3%**	0.6%	0.6%	**1.0%**	**1.0%**	0.8%	**1.0%**	0.9%
PFHxS	**1.3%**	0.5%	0.6%	**0.7%**	0.5%	**0.7%**	0.6%	**0.7%**	0.6%
PFHpA	0.1%	0.1%	**0.2%**	**0.2%**	0.1%	0.2%	**0.2%**	0.1%	0.1%
PFOA	**2.5%**	1.1%	**1.5%**	**1.5%**	**1.4%**	1.3%	**1.5%**	1.3%	1.3%

Abbreviations: PFHpA – perfluoroheptanoic acid; PFHxS – perfluorohexanesulfonic acid; PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; ppt – parts per trillion.

Table 8-10: Reductions in Average PFAS Concentrations (ppt) by Demographic Group in a Hypothetical Regulatory Scenario with Maximum Contaminant Level at 10.0 ppt, Category 1 and 2 PWS Service Areas

	Race and Ethnicity						**Income**		
PFAS	**Non-Hispanic American Indian or Alaska Native**	**Non-Hispanic Asian**	**Non-Hispanic Black**	**Non-Hispanic Pacific Islander**	**Hispanic**	**Non-Hispanic White**	**Below Twice the Poverty Level**	**Above Twice the Poverty Level**	**Total Population Served**
PFOS	0.04	0.03	0.04	0.07	0.03	0.05	0.04	0.04	0.04
PFHxS	0.09	0.04	0.05	0.30	0.06	0.07	0.06	0.06	0.06
PFHpA	0.01	0.00	0.01	0.03	0.01	0.01	0.01	0.01	0.01
PFOA	0.20	0.09	0.16	0.45	0.13	0.15	0.16	0.14	0.14

Abbreviations: PFHpA – perfluoroheptanoic acid; PFHxS – perfluorohexanesulfonic acid; PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid.

8.3.2.3 Comparison of Results by PWS Size

8.3.2.3.1 Demographic Profile of PWS Service Areas

Table 8-11 and Table 8-12 summarize the demographic profile for category 1 and 2 PWS service areas by system size for large and small PWS service areas, respectively. Small systems are defined as systems serving fewer than or equal to 10,000 people while large systems serve more than 10,000 people. Table 8-11 and Table 8-12 also provide a comparison to the demographic characteristics of the overall U.S. population.

Table 8-11 shows that the population served by large category 1 and 2 PWS service areas has slight differences in demographic characteristics compared to the overall U.S. population, with percent differences all being less than +/- 4.3 percent. The population served by large category 1 and 2 PWS service areas has lower percentages of non-Hispanic White (-4.3%) populations and populations with income above twice the Federal poverty level (-1.4%) compared to the overall U.S. population. Additionally, the population served by large category 1 and 2 PWS service areas have higher percentages of non-Hispanic Black (+1.6%), Hispanic (+2.4%), and non-Hispanic Asian populations (+0.9%) populations and populations with income below twice the Federal poverty level (+1.4%) compared to the overall U.S. population. The percentage of non-Hispanic American Indian or Alaska Native populations and non-Hispanic Pacific Islander populations is relatively consistent with the percent of these populations across the U.S.

Table 8-12 shows that the population served by small category 1 and 2 PWS service areas has greater differences in the demographic characteristics of the population served compared to the overall U.S. population, with percent differences being generally greater than +/- 2 percent, and the greatest difference being +12.2 percent. The population served by small category 1 and 2 PWS service areas has lower percentages of non-Hispanic Asian (-3.62%), non-Hispanic Black (-2.13%), and Hispanic (-6.58%) populations and populations with income above twice the Federal poverty level (-4.02%) compared to the overall U.S. population. Additionally, the population served by small category 1 and 2 PWS service areas has higher percentages of non-Hispanic American Indian or Alaska Native (+1%), non-Hispanic White (+12.23%) populations, and populations with income below twice the Federal poverty level (+4.02%) compared to the overall U.S. population.

Table 8-11: Population Served by Category 1 and 2 PWSs and Percent of U.S. Population by Demographic Group, Large Systems

	Race and Ethnicity						Income		
	Non-Hispanic American Indian or Alaska Native	**Non-Hispanic Asian**	**Non-Hispanic Black**	**Non-Hispanic Pacific Islander**	**Hispanic**	**Non-Hispanic White**	**Below Twice the Poverty Level**	**Above Twice the Poverty Level**	**Total Population Served**
Population Served	1,089,683	16,106,131	34,265,828	363,866	51,345,649	138,878,798	77,564,529	171,471,255	249,035,784
Percent of Total Population Served	0.44%	6.47%	13.76%	0.15%	20.62%	55.77%	31.15%	68.85%	100.00%
U.S. Population Percent	0.6%	5.6%	12.2%	0.2%	18.2%	60.1%	29.8%	70.2%	
Percent Difference Between Population Served Percent and U.S. Percent	-0.16%	0.87%	1.56%	-0.05%	2.42%	-4.33%	1.35%	-1.35%	

Table 8-12: Population Served by Category 1 and 2 PWSs and Percent of U.S. Population by Demographic Group, Small Systems

	Race and Ethnicity						Income		
	Non-Hispanic American Indian or Alaska Native	**Non-Hispanic Asian**	**Non-Hispanic Black**	**Non-Hispanic Pacific Islander**	**Hispanic**	**Non-Hispanic White**	**Below Twice the Poverty Level**	**Above Twice the Poverty Level**	**Total Population Served**
Population Served	50,564	62,185	316,020	2,412	364,684	2,269,169	1,061,063	2,076,244	3,137,307
Percent of Total Population Served	1.61%	1.98%	10.07%	0.08%	11.62%	72.33%	33.82%	66.18%	100.00%
U.S. Population Percent	0.6%	5.6%	12.2%	0.2%	18.2%	60.1%	29.8%	70.2%	
Percent Difference between Population Served Percent and U.S. Percent	1.01%	-3.62%	-2.13%	-0.12%	-6.58%	12.23%	4.02%	-4.02%	

8.3.2.3.2 Baseline Scenario

Table 8-13 and Table 8-14 summarize the populations served by large and small category 1 and 2 PWS service areas with modeled PFAS occurrence above baseline thresholds based on a trigger level of 2 ppt for each PFAS analyte, which is slightly above the Method 537.1 detection limits for each PFAS analyte. The second set of rows in Table 8-13 and Table 8-14 summarize the percentage of the total population served by demographic group with modeled PFAS occurrence above baseline thresholds. Percentages are bolded and italicized when the percentage of the population in a specific demographic group with modeled PFAS above baseline thresholds is greater than the percentage of the total population across all demographic groups exposed to modeled PFAS above the baseline thresholds. Additionally, percentages are highlighted when the percentage of the population in a specific demographic group with modeled PFAS above the baseline threshold represents greater than a 1 percentage point difference compared to the total population across all demographic groups. Table 8-15 characterizes population-weighted average PFAS concentrations across demographic groups in large and small category 1 and 2 PWSs. Highlighted cells represent whether the average concentration for a given demographic group is higher than the average concentration for the total population served across all demographic groups (right-hand column).

Depending on the PFAS analyte, between 4.8 percent and 10.8 percent of the total population served by large category 1 and 2 PWS service areas are exposed to modeled PFAS occurrence above baseline thresholds based on a trigger level of 2 ppt. Depending on the PFAS analyte, between 0.5 percent and 3.7 percent of the total population served by small category 1 and 2 PWS service areas is exposed to modeled PFAS occurrence above baseline thresholds based on baseline thresholds of 2 ppt.

For large systems, the percentage of Hispanic populations served by category 1 and 2 PWS service areas is higher across all four PFAS analytes compared to the percentage of the total population served across all demographic groups with anticipated PFAS exposure above baseline thresholds. Depending on the PFAS analyte, the percentage of Hispanic populations served by large systems with exposure above baseline thresholds is 1.3 percent to 2.6 percentage points higher than percentages of the total population served across all demographic groups, or 2.6 to 3.5 percentage points higher than for non-Hispanic White populations. Non-Hispanic Black populations are also exposed to all PFAS analytes above baseline levels to a greater extent than the total population served, with 1.6 and 1.2 percentage point higher exposure levels to PFOA and PFOS, respectively. In comparison to non-Hispanic White populations, non-Hispanic Black populations have 1 to 2.4 percentage points greater exposure depending on the PFAS analyte. The percent of non-Hispanic American Indian or Alaska Native populations served with exposure above baseline thresholds to PFHxS is 1.2 percentage points greater than the share of the total population served, or 2.1 percentage points higher than the percent of non-Hispanic White populations exposed. For large systems, significant differences in exposure to any PFAS for non-Hispanic Asian, non-Hispanic Pacific Islander, or low-income populations were not observed.

For small systems, the percent of non-Hispanic Asian populations served by category 1 and 2 PWS service areas with PFAS above baseline thresholds is higher for PFOA and PFOS in comparison to the total population served, with a range of 1.5 to 2.2 percentage points more of the population served. The percentage of non-Hispanic Black populations served by category 1 and 2 PWS service areas with PFAS above baseline thresholds is 0.8 percentage points higher for PFHxS compared to the percentage of the total population served across all demographic groups with anticipated PFAS exposure above baseline thresholds. The EPA also observed that non-Hispanic White populations and those with income above twice the Federal poverty level have slightly higher percentages of the population exposed across all PFAS analytes examined. Given the data gaps in occurrence information among small systems, extrapolating these results to small systems across the country is not possible. For example, the EPA observed only 2,400 individuals who identify as non-Hispanic Pacific Islander.

Table 8-15 provides detail on average concentrations across these demographic groups for large and small water systems, respectively. The first panel of Table 8-15 supports the previous findings in Table 8-13 that, for large PWSs, non-Hispanic American Indian or Alaska Native, non-Hispanic Black, and Hispanic populations served have greater exposure across at least two PFAS analytes in comparison to exposure for the total population served across all demographic groups. Hispanic and non-Hispanic Black populations served have greater exposure to all four PFAS in comparison to the total population served.

In addition, Table 8-15 demonstrates that non-Hispanic Pacific Islander populations are served by water systems with higher average concentrations of PFHxS and PFOA in comparison to average concentrations of these PFAS analytes for the total population served by large PWSs, even though non-Hispanic Pacific Islander populations have similar or even lower share of the population exposed in comparison to the total population served, as shown in Table 8-13. Non-Hispanic White and non-Hispanic Asian populations generally have lower average concentrations, on average, across all four PFAS analytes in comparison to the total population served. Further, populations with income less than twice the Federal poverty level are on average served by large PWSs with higher concentrations of three PFAS analytes in comparison to populations with income above twice the Federal poverty level or the total population served across all large PWSs.

The second panel of Table 8-15 shows that non-Hispanic Asian, non-Hispanic Black, and non-Hispanic White populations have greater potential exposure to specific PFAS analytes in comparison to the total population served across all demographic groups served by small PWSs. Non-Hispanic Asian populations served by small PWSs have elevated concentrations of PFOS and PFHpA in comparison to the total population served by small PWSs. Non-Hispanic Black populations served by small PWSs have greater exposure to PFOS, PFHxS, and PFOA in comparison to the total population served by small PWSs. Non-Hispanic White populations also see greater exposure to PFHpA in comparison to the total population served in small PWSs, although this difference is small (0.01 ppt).

Table 8-13: Baseline Scenario: Demographic Breakdown of Population Served by Category 1 and 2 PWS Service Areas Above Baseline Thresholds and as a Percent of Total Population Served, Large Systems

	Race and Ethnicity						Income			
PFAS	**Non-Hispanic American Indian or Alaska Native**	**Non-Hispanic Asian**	**Non-Hispanic Black**	**Non-Hispanic Pacific Islander**	**Hispanic**	**Non-Hispanic White**	**Below Twice the Poverty Level**	**Above Twice the Poverty Level**	**Total Population Served**	**System Count**
Population Served Above Baseline Threshold										
PFOS	96,211	1,758,287	4,120,087	25,192	6,253,084	14,069,015	8,013,031	18,982,735	26,995,766	339
PFHxS	79,002	802,016	2,296,946	20,822	4,457,379	7,156,640	5,083,638	10,107,999	15,191,637	186
PFHpA	52,475	602,507	1,732,401	12,295	3,458,779	5,714,325	3,666,804	8,175,133	11,841,937	143
PFOA	88,407	1,066,286	3,413,512	19,849	5,195,766	10,480,694	6,630,748	14,129,410	20,760,158	296
Population Served Above Baseline Threshold as a Percentage of Total Population Served										
PFOS	8.83%	***10.92%***	***12.02%***	6.92%	***12.18%***	10.13%	10.33%	***11.07%***	10.84%	-
PFHxS	***7.25%***	4.98%	***6.70%***	5.72%	***8.68%***	5.15%	***6.55%***	5.89%	6.10%	-
PFHpA	***4.82%***	3.74%	***5.06%***	3.38%	***6.74%***	4.11%	4.73%	***4.77%***	4.76%	-
PFOA	8.11%	6.62%	***9.96%***	5.46%	***10.12%***	7.55%	***8.55%***	8.24%	8.34%	-
Total Population Served in Sampled Population										
	1,089,683	16,106,131	34,265,828	51,345,649	138,878,798	363,866	77,564,529	171,471,255	249,035,784	-

Abbreviations: PFHpA – perfluoroheptanoic acid; PFHxS – perfluorohexanesulfonic acid; PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid.

Table 8-14: Baseline Scenario: Demographic Breakdown of Population Served by Category 1 and 2 PWS Service Areas Above Baseline Thresholds and as a Percent of Total Population Served, Small Systems

	Race and Ethnicity						**Income**			
PFAS	**Non-Hispanic American Indian or Alaska Native**	**Non-Hispanic Asian**	**Non-Hispanic Black**	**Non-Hispanic Pacific Islander**	**Hispanic**	**Non-Hispanic White**	**Below Twice the Poverty Level**	**Above Twice the Poverty Level**	**Total Population Served**	**System Count**
Population Served Above Baseline Threshold										
PFOS	253	3,672	10,730	1	10,593	87,520	22,231	92,565	114,796	22
PFHxS	128	111	6,087	1	1,207	28,075	11,595	24,797	36,392	9
PFHpA	104	330	306	17	530	15,372	3,591	13,422	17,013	4
PFOA	267	2,947	10,369	1	6,322	80,383	20,456	81,731	102,187	20
Population Served Above Baseline Threshold as a Percentage of Total Population Served										
PFOS	0.50%	***5.90%***	3.40%	0.04%	2.90%	***3.86%***	2.10%	***4.46%***	3.66%	-
PFHxS	0.25%	0.18%	***1.93%***	0.04%	0.33%	***1.24%***	1.09%	***1.19%***	1.16%	-
PFHpA	0.21%	0.53%	0.10%	***0.70%***	0.15%	***0.68%***	0.34%	***0.65%***	0.54%	-
PFOA	0.53%	***4.74%***	***3.28%***	0.04%	1.73%	***3.54%***	1.93%	***3.94%***	3.26%	-
Total Population Served in Sampled Population										
	50,564	62,185	316,020	364,684	2,269,169	2,412	1,061,063	2,076,244	3,137,307	-

Abbreviations: PFHpA – perfluoroheptanoic acid; PFHxS – perfluorohexanesulfonic acid; PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid.

Table 8-15: Modeled Average PFAS Concentrations (ppt) by Demographic Group and System Size in the Baseline, Category 1 and 2 PWS Service Areas

	Race and Ethnicity						Income		
PFAS	**Non-Hispanic American Indian or Alaska Native**	**Non-Hispanic Asian**	**Non-Hispanic Black**	**Non-Hispanic Pacific Islander**	**Hispanic**	**Non-Hispanic White**	**Below Twice the Poverty Level**	**Above Twice the Poverty Level**	**Total Population Served**
Large Systems									
PFOS	0.99	1.02	1.05	0.90	1.16	0.96	1.02	1.02	1.02
PFHxS	0.84	0.58	0.64	0.86	0.75	0.59	0.65	0.63	0.63
PFHpA	0.54	0.50	0.55	0.51	0.64	0.50	0.55	0.53	0.54
PFOA	1.09	0.85	1.04	1.14	1.12	0.90	0.99	0.95	0.96
Small Systems									
PFOS	0.46	0.68	0.59	0.44	0.54	0.57	0.50	0.60	0.57
PFHxS	0.20	0.24	0.26	0.19	0.23	0.23	0.23	0.24	0.24
PFHpA	0.22	0.26	0.24	0.24	0.23	0.25	0.23	0.25	0.24
PFOA	0.32	0.45	0.57	0.29	0.38	0.46	0.42	0.48	0.46

Abbreviations: PFHpA – perfluoroheptanoic acid; PFHxS – perfluorohexanesulfonic acid; PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid.

8.3.2.3.3 Hypothetical Regulatory Scenario #1: UCMR 5 MRLs

Table 8-16, Table 8-17, and Table 8-18 summarize the results for populations served by large and small category 1 and 2 PWS service areas with PFAS occurrence above UCMR 5 MRL values, respectively. The EPA assumed that PWS service areas with PFAS system-level means above the UCMR 5 MRL value will reduce PFAS levels to comply with the final rule.

The first set of rows in Table 8-16 and Table 8-17 summarize populations served by large and small category 1 and 2 PWSs with modeled PFAS occurrence above the UCMR 5 MRLs, respectively. The second set of rows provides these estimates as a percentage of the total population served by PWS service areas included in the EPA's analysis. In Table 8-16 and Table 8-17, percentages are bolded and italicized when the percentage of the population in a specific demographic group with PFAS occurrence above the MRL value is greater than the percentage of the total population across all demographic groups with PFAS occurrence above the MRL. Additionally, percentages are highlighted when the percentage of the population in a specific demographic group with modeled PFAS above the MRL represents greater than a 1 percentage point difference compared to the total population across all demographic groups exposed to modeled PFAS above the MRL value. Table 8-17 characterizes population-weighted average reductions in PFAS concentrations across demographic groups in large and small category 1 and 2 PWSs. Highlighted cells represent whether the average reduction for a given demographic group is higher than the average reduction for the total population served across all demographic groups (right-hand column). The percentages that are bolded, italicized, or highlighted indicate more PFAS exposure above the MRL for a given demographic group; the EPA anticipates relatively higher reductions in PFAS exposure will accrue to these demographic groups under this hypothetical regulatory scenario compared to the percentage of the population across all demographic groups.

Depending on the PFAS analyte, between 3.1 percent and 5.5 percent of the total population served by large category 1 and 2 PWS service areas are exposed to at least one of the modeled four PFAS occurrences above UCMR 5 MRL values. For small category 1 and 2 PWS service areas, depending on the PFAS analyte, between 0.3 percent and 1.9 percent of the total population served is exposed to modeled PFAS occurrence above UCMR 5 MRL values.

Findings for large systems are as follows:

- Non-Hispanic American Indian or Alaska Native populations served have higher exposure above UCMR 5 MRL values for PFOA, PFHpA, and PFHxS compared to the percent of the population served across all demographic groups. Non-Hispanic American Indian or Alaska Native populations also have higher exposure than the non-Hispanic White population across all PFAS analytes. The differences in percent of non-Hispanic American Indian or Alaska Native populations exposed range from 0.6 - 1.4 percentage points in comparison to the total population served across all demographic groups.
- Non-Hispanic Black populations served have higher exposure above the UCMR 5 MRL for all PFAS analytes compared to the percent of the total population served across all demographic groups, however the differences are all relatively small in magnitude (<0.5 percentage points). Exposure to PFOA, PFOS, and PFHxS for non-Hispanic Black

populations is at least 1 percentage point higher than percent of the non-Hispanic White population exposed to these PFAS analytes.

- Hispanic populations served by large PWSs have higher exposure above the UCMR 5 MRL values for all four PFAS analytes compared to the percent of the total population served across all demographic groups. Differences in percent of Hispanic populations exposed are consistently at least 2 percentage points higher in comparison to the total population served across all demographic groups. Hispanic populations have at least double the exposure above UCMR 5 MRL values in comparison to non-Hispanic White populations for PFHxS and PFHpA.
- Non-Hispanic Asian and non-Hispanic Pacific Islander populations served have comparable or lower levels of exposure above UCMR 5 MRL values for all PFAS analytes compared to both the percent of the total population served across all demographic groups and the non-Hispanic White population.
- Low-income populations have higher PFAS exposure above UCMR 5 MRL values for all PFAS analytes compared to the total population served across all demographic groups. These differences are all relatively small at less than 0.4 percentage points, but disparate exposures are larger for each PFAS analyte when compared to populations with income above twice the Federal poverty level.

Findings for small systems are as follows:

- Non-Hispanic Asian populations served have higher exposure above UCMR 5 MRL values for PFOS and PFHpA compared to the percent of the total population served across all demographic groups, with PFOS exposure 1 percentage point higher than the percent of the total population or non-Hispanic White population served that is exposed to PFOS.
- Non-Hispanic Black populations served have higher exposure above the UCMR 5 MRL values for PFOS, PFHxS, and PFOA compared to the percent of the total population served across all demographic groups, with PFOA exposure over 1 percentage point higher than the percent of the total population or non-Hispanic White population served. The differences in population exposed range from 0.9 - 1.3 percentage points when comparing exposure for non-Hispanic Black populations to the total population served.
- Non-Hispanic White populations served experience slightly higher exposure above the UCMR 5 MRL value for PFHpA compared to the percent of the total population served across all demographic groups.
- Populations with income above twice the Federal poverty level have higher exposure above the UCMR 5 MRL values across all PFAS analytes compared to the percent of the total population served across all demographic groups.

Table 8-18 characterizes population-weighted average reductions in PFAS exposures anticipated to occur for large and small PWSs in a hypothetical regulatory scenario where system-level means are reduced to UCMR 5 MRL values. For large systems, as in Table 8-16, Hispanic populations have higher exposures above UCMR 5 MRL values for all PFAS analytes in comparison to the total population served among large PWSs. The results also show that non-

Hispanic Black populations have higher average exposures to PFHpA and PFOA than the total population served across all demographic groups in large PWSs. On average, non-Hispanic American Indian or Alaska Native populations also see large reductions in PFHxS and PFOA in comparison to the total population served, which is consistent with elevated percentages of the population exposed as shown in Table 8-16. Non-Hispanic Pacific Islander populations served by large systems see the greatest reductions in PFHxS and PFOA of any demographic group for large systems, with reductions of each that are roughly twice the average reduction observed for the total population served by large systems. In large systems, the EPA also observed elevated population-weighted concentrations of all PFAS analytes for low-income populations in comparison to the total population served, and low-income populations are also more exposed to all PFAS analytes in comparison to populations with income above twice the Federal poverty level. For small systems, non-Hispanic Asian, non-Hispanic Black, and Hispanic populations have larger reductions in particular PFAS than the total population served across all demographic groups. In general, however, differences in PFAS reductions across demographic groups are slight for small systems.

It should be noted that the sample size of small PWS service areas included in categories 1 and 2 with PFAS exposure above UCMR 5 MRL values is limited and could meaningfully impact the results presented in this analysis. The population served by small category 1 and 2 PWS service areas included in this analysis captures roughly 1 percent of the total U.S. population. Given that approximately 20 percent of the U.S. population is served by small systems, this subset of systems may not be representative of small systems across the U.S. As such, results from this analysis cannot be extrapolated to be representative of small systems nationwide. Additionally, the population served by the subset of small systems in categories 1 and 2 is disproportionately non-Hispanic White, with 12.2 higher percentage point representation compared to the overall U.S. population. The population served is also less Hispanic, with representation of this group being 6.58 percentage points lower than the overall U.S. population. Further evaluation is needed to demonstrate whether the sample population served by small category 1 and 2 PWS service areas is representative of the demographic breakdown of all small systems nationwide.

Table 8-16: Hypothetical Regulatory Scenario #1: Demographic Breakdown of Population Served by Category 1 and 2 PWS Service Areas Above UCMR 5 MRLs and as a Percent of Total Population Served, Large Systems

	Race and Ethnicity						Income			
PFAS	**Non-Hispanic American Indian or Alaska Native**	**Non-Hispanic Asian**	**Non-Hispanic Black**	**Non-Hispanic Pacific Islander**	**Hispanic**	**Non-Hispanic White**	**Below Twice the Poverty Level**	**Above Twice the Poverty Level**	**Total Population Served**	**System Count**
Population Served Above UCMR 5 MRL										
PFOS	49,110	686,780	1,786,279	11,297	3,571,565	5,750,090	3,898,651	8,227,263	12,125,914	190
PFHxS	62,128	515,390	1,539,938	14,361	3,706,392	4,695,697	3,663,223	7,122,201	10,785,424	111
PFHpA	39,572	370,784	1,083,401	7,783	2,656,626	3,318,167	2,586,693	5,063,861	7,650,554	81
PFOA	67,138	705,879	2,017,982	11,950	3,922,241	6,608,160	4,397,699	9,237,285	13,634,984	175
Population Served Above UCMR 5 MRL as a Percentage of Total Population Served										
PFOS	4.51%	4.26%	***5.21%***	3.10%	***6.96%***	4.14%	***5.03%***	4.80%	4.87%	-
PFHxS	***5.70%***	3.20%	***4.49%***	3.95%	***7.22%***	3.38%	***4.72%***	4.15%	4.33%	-
PFHpA	***3.63%***	2.30%	***3.16%***	2.14%	***5.17%***	2.39%	***3.33%***	2.95%	3.07%	-
PFOA	***6.16%***	4.38%	***5.89%***	3.28%	***7.64%***	4.76%	***5.67%***	5.39%	5.48%	-
Total Population Served in Sampled Population										
	1,089,683	16,106,131	34,265,828	51,345,649	138,878,798	363,866	77,564,529	171,471,255	249,035,784	-

Abbreviations: MRL – minimum reporting level; PFHpA – perfluoroheptanoic acid; PFHxS – perfluorohexanesulfonic acid; PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; UCMR – Unregulated Contaminant Monitoring Rule.

Table 8-17: Hypothetical Regulatory Scenario #1: Demographic Breakdown of Population Served by Category 1 and 2 PWS Service Areas Above UCMR 5 MRLs and as a Percent of Total Population Served, Small Systems

	Race and Ethnicity						Income			
PFAS	**Non-Hispanic American Indian or Alaska Native**	**Non-Hispanic Asian**	**Non-Hispanic Black**	**Non-Hispanic Pacific Islander**	**Hispanic**	**Non-Hispanic White**	**Below Twice the Poverty Level**	**Above Twice the Poverty Level**	**Total Population Served**	**System Count**
Population Served Above UCMR 5 MRL										
PFOS	209	1,778	8,937	0	4,157	42,263	12,859	45,491	58,350	13
PFHxS	12	21	4,087	0	103	8,181	3,918	8,825	12,743	4
PFHpA	9	258	97	0	287	8,111	986	7,899	8,885	2
PFOA	228	663	8,897	1	3,397	33,997	12,006	35,999	48,005	10
Population Served Above UCMR 5 MRL as a Percentage of Total Population Served										
PFOS	0.41%	***2.86%***	***2.83%***	0.00%	1.14%	1.86%	1.21%	***2.19%***	1.86%	-
PFHxS	0.02%	0.03%	***1.29%***	0.00%	0.03%	0.36%	0.37%	***0.43%***	0.41%	-
PFHpA	0.02%	***0.41%***	0.03%	0.00%	0.08%	***0.36%***	0.09%	***0.38%***	0.28%	-
PFOA	0.45%	1.07%	***2.82%***	0.04%	0.93%	1.50%	1.13%	***1.73%***	1.53%	-
Total Population Served in Sampled Population										
	50,564	62,185	316,020	364,684	2,269,169	2,412	1,061,063	2,076,244	3,137,307	-

Abbreviations: MRL – minimum reporting level; PFHpA – perfluoroheptanoic acid; PFHxS – perfluorohexanesulfonic acid; PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; UCMR – Unregulated Contaminant Monitoring Rule.

Table 8-18: Reductions in Average PFAS Concentrations (ppt) by Demographic Group in a Hypothetical Regulatory Scenario with Maximum Contaminant Levels at the UCMR 5 MRLs, Category 1 and 2 PWS Service Areas

PFAS	Race and Ethnicity						Income		Total Population Served
	Non-Hispanic American Indian or Alaska Native	Non-Hispanic Asian	Non-Hispanic Black	Non-Hispanic Pacific Islander	Hispanic	Non-Hispanic White	Below Twice the Poverty Level	Above Twice the Poverty Level	
Large Systems									
PFOS	0.19	0.17	0.19	0.16	0.26	0.18	0.20	0.19	0.19
PFHxS	0.31	0.10	0.14	0.38	0.16	0.15	0.15	0.14	0.15
PFHpA	0.04	0.02	0.06	0.05	0.06	0.04	0.05	0.04	0.04
PFOA	0.44	0.22	0.36	0.58	0.41	0.29	0.35	0.31	0.32
Small Systems									
PFOS	0.01	0.09	0.05	0.00	0.03	0.06	0.03	0.07	0.06
PFHxS	0.00	0.02	0.01	0.00	0.03	0.02	0.02	0.02	0.02
PFHpA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PFOA	0.02	0.05	0.17	0.00	0.05	0.10	0.09	0.10	0.10

Abbreviations: PFHpA – perfluoroheptanoic acid; PFHxS – perfluorohexanesulfonic acid; PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid.

Note:

[a]The demographic group people of color includes individuals who identify as Hispanic and/or a race other than White. It is calculated from EJScreen's percent minority indicator and is non-duplicative across race and ethnicity categories.

8.3.2.3.4 Hypothetical Regulatory Scenario #2: 10.0 ppt

Table 8-19 summarizes results for populations served by large category 1 and 2 PWS service areas with PFAS occurrence above 10.0 ppt. Table 8-20 summarizes results for populations served by small category 1 and 2 PWS service areas with PFAS occurrence above 10.0 ppt. Table 8-21 characterizes population-weighted average reductions in PFAS exposures anticipated to occur for large and small PWSs in a hypothetical regulatory scenario where system-level means are reduced to 10.0 ppt. The first set of rows in Table 8-19 and Table 8-20 summarizes populations served by large and small category 1 and 2 PWSs with modeled PFAS occurrence above the 10.0 ppt, respectively. The second set of rows provides these estimates as a percentage of the total population served by these PWS service areas.

In Table 8-19 and Table 8-20, percentages are bolded and italicized when the percent of the population in a specific demographic group with PFAS occurrence above 10.0 ppt is greater than the percentage of the total population across all demographic groups with PFAS occurrence above 10.0 ppt. In Table 8-21, highlighted cells represent whether the average reduction for a given demographic group is higher than the reductions for the total population served across all demographic groups in large and small PWSs (right-hand column). The percentages that are bolded, italicized, or highlighted indicate more PFAS exposure above 10.0 ppt for a given demographic group; the EPA anticipates relatively higher reductions in PFAS exposure will accrue to these demographic groups under this hypothetical regulatory scenario compared to the percentage of the population across all demographic groups.

For large systems, a greater percent of non-Hispanic American Indian or Alaska Native populations experience exposure to PFHxS and PFOA in comparison to the total population served, with differences ranging from 0.7 to 1.3 percentage points. Non-Hispanic Asian populations are exposed to PFOS to a greater extent than the total population served, with a difference of 0.3 percentage points. The percent of the non-Hispanic Black population with PFHpA and PFOA above 10.0 ppt is also elevated, although differences are relatively small in comparison to both the total population served or non-Hispanic White populations (<0.2 percentage points). Non-Hispanic Pacific Islander populations served by large systems have elevated exposure above 10.0 ppt for PFHpA, PFOA and PFHxS compared to the total population served across all demographic groups, although differences are again relatively small (<0.2 percentage points). Hispanic populations are slightly more likely to be served by large PWSs with PFOA and PFOS concentrations above 10.0 ppt (<0.1 percentage points). Populations with income below twice the Federal poverty level are also slightly more likely to have PFOS and PFHxS above 10.0 ppt in comparison to the total population served.

For small systems, non-Hispanic Asian populations are slightly more likely to be served by PWSs with PFOS above 10.0 ppt than the total population served by small systems. Non-Hispanic Black populations are more likely to be served by small systems with PFOA above 10.0 ppt, with a difference in percent of the population exposed of 0.9 percentage points in comparison to the total population served or non-Hispanic White populations. Non-Hispanic White populations have slightly elevated exposure above 10.0 ppt for PFOS compared to the population served across all demographic groups. The average reductions by demographic group, shown in Table 8-21, largely confirm the findings of Table 8-19 and Table 8-20, with greater average reductions generally accruing to populations with a higher percentage of potentially exposed individuals for large and small PWSs.

As previously noted, the sample size of small PWS service areas included in categories 1 and 2 is limited, with population served capturing roughly 1 percent of the total U.S. population. Additionally, the population served by the subset of small systems in categories 1 and 2 is disproportionately White and non-Hispanic compared to the overall U.S. population, as previously discussed. Further evaluation is needed to demonstrate whether the sample population served by small category 1 and 2 PWS service areas is representative of the demographic breakdown of all small systems nationwide.

Table 8-19: Hypothetical Regulatory Scenario #2: Demographic Breakdown of Population Served by Category 1 and 2 PWS Service Areas Above 10.0 ppt and as a Percent of Total Population Served, Large Systems

	Race and Ethnicity						Income			
PFAS	**Non-Hispanic American Indian or Alaska Native**	**Non-Hispanic Asian**	**Non-Hispanic Black**	**Non-Hispanic Pacific Islander**	**Hispanic**	**Non-Hispanic White**	**Below Twice the Poverty Level**	**Above Twice the Poverty Level**	**Total Population Served**	**System Count**
Population Served Above 10.0 ppt										
PFOS	6,388	202,238	223,089	2,212	504,545	1,371,329	665,080	1,695,539	2,360,619	48
PFHxS	14,317	77,668	204,840	2,500	237,779	1,041,289	493,256	1,136,755	1,630,011	32
PFHpA	1,673	11,460	74,648	722	52,474	218,268	119,742	251,319	371,061	8
PFOA	28,551	178,333	531,691	5,607	716,309	1,767,073	1,139,216	2,176,732	3,315,948	62
Population Served Above 10.0 ppt as a Percentage of Total Population Served										
PFOS	0.59%	***1.26%***	0.65%	0.61%	***0.98%***	***0.99%***	0.86%	***0.99%***	0.95%	-
PFHxS	***1.31%***	0.48%	0.60%	***0.69%***	0.46%	***0.75%***	0.64%	***0.66%***	0.65%	-
PFHpA	0.15%	0.07%	***0.22%***	***0.20%***	0.10%	***0.16%***	0.15%	0.15%	0.15%	-
PFOA	***2.62%***	1.11%	***1.55%***	***1.54%***	***1.40%***	1.27%	***1.47%***	1.27%	1.33%	-
Total Population Served in Sampled Population										
	1,089,683	16,106,131	34,265,828	51,345,649	138,878,798	363,866	77,564,529	171,471,255	249,035,784	-

Abbreviations: MRL – minimum reporting level; PFHpA – perfluoroheptanoic acid; PFHxS – perfluorohexanesulfonic acid; PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; UCMR – Unregulated Contaminant Monitoring Rule.

Table 8-20: Hypothetical Regulatory Scenario #2: Demographic Breakdown of Population Served by Category 1 and 2 PWS Service Areas Above 10.0 ppt and as a Percent of Total Population Served, Small Systems

	Race and Ethnicity						**Income**			
	Non-Hispanic American Indian or Alaska Native	**Non-Hispanic Asian**	**Non-Hispanic Black**	**Non-Hispanic Pacific Islander**	**Hispanic**	**Non-Hispanic White**	**Below Twice the Poverty Level**	**Above Twice the Poverty Level**	**Total Population Served**	**System Count**
Population Served Above 10.0 ppt										
PFOS	9	258	97	0	287	8111	986	7899	8,885	2
PFHxS	1	7	9	0	48	149	94	136	230	1
PFHpA	1	7	9	0	48	149	94	136	230	1
PFOA	12	21	4087	0	103	8181	3918	8825	12,743	3
Population Served Above 10.0 ppt as a Percentage of Total Population Served										
PFOS	0.02%	***0.41%***	0.03%	0.00%	0.08%	***0.36%***	0.09%	***0.38%***	0.28%	-
PFHxS	0.00%	***0.01%***	0.00%	0.00%	***0.01%***	0.01%	0.01%	0.01%	0.01%	-
PFHpA	0.00%	***0.01%***	0.00%	0.00%	***0.01%***	0.01%	0.01%	0.01%	0.01%	-
PFOA	0.02%	0.03%	***1.29%***	0.00%	0.03%	0.36%	0.37%	***0.43%***	0.41%	-
Total Population Served in Sampled Population										
	50,564	62,185	316,020	364,684	2,269,169	2,412	1,061,063	2,076,244	3,137,307	-

Abbreviations: PFHpA – perfluoroheptanoic acid; PFHxS – perfluorohexanesulfonic acid; PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid; ppt – parts per trillion.

Table 8-21: Reductions in Average PFAS Concentrations (ppt) by Demographic Group in a Hypothetical Regulatory Scenario with Maximum Contaminant Levels at 10.0 ppt, Category 1 and 2 PWS Service Areas

PFAS	Race and Ethnicity						Income		Total Population Served
	Non-Hispanic American Indian or Alaska Native	Non-Hispanic Asian	Non-Hispanic Black	Non-Hispanic Pacific Islander	Hispanic	Non-Hispanic White	Below Twice the Poverty Level	Above Twice the Poverty Level	
Large Systems									
PFOS	0.04	0.03	0.04	0.07	0.03	0.05	0.04	0.04	0.04
PFHxS	0.10	0.04	0.05	0.30	0.06	0.07	0.06	0.06	0.06
PFHpA	0.01	0.00	0.01	0.03	0.01	0.01	0.01	0.01	0.01
PFOA	0.21	0.09	0.16	0.45	0.13	0.15	0.16	0.14	0.15
Small Systems									
PFOS	0.00	0.02	0.00	0.00	0.01	0.01	0.00	0.01	0.01
PFHxS	0.00	0.02	0.01	0.00	0.03	0.01	0.02	0.01	0.02
PFHpA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PFOA	0.01	0.03	0.06	0.00	0.03	0.05	0.05	0.05	0.05

Abbreviations: PFHpA – perfluoroheptanoic acid; PFHxS – perfluorohexanesulfonic acid; PFOA – perfluorooctanoic acid; PFOS – perfluorooctanesulfonic acid.

8.4 SafeWater EJ Analysis of Final Rule and Regulatory Alternatives

8.4.1 Methodology

In addition to analyzing EJ exposure using the EJSCREENbatch R package, the EPA also conducted an EJ analysis of the final rule and regulatory alternatives using the SafeWater MCBC. The EPA's final rule sets MCLs of 4.0 ppt for PFOA and PFOS each, MCLs of 10 ppt each for PFHxS, PFNA, and HFPO-DA and an HI of 1.0 for PFNA, HFPO-DA, PFHxS, and PFBS. Options 1a, 1b, and 1c set MCL values for PFOA and PFOS at 4.0 ppt, 5.0 ppt, and 10.0 ppt, respectively.

SafeWater MCBC was used to analyze the distribution of anticipated health benefits and household costs associated with the final PFAS NPDWR across race/ethnicity groups and income level. For more information on SafeWater MCMC and its application in the EPA's analysis of national quantified benefits and costs associated with the final PFAS NPDWR, see Section 5.2.

Using SafeWater MCBC, the EPA estimated the quantified health benefits and household costs expected to accrue to specific race/ethnicity and income level groups for category 1 and 2 PWS service areas. As previously described in Section 8.3.1, category 1 and 2 PWS service areas include systems that have sampled PFAS occurrence data from UCMR 3 and have predelineated service area boundaries or those estimated using zip code served information (n = 4,723). The subset of category 1 and 2 PWSs captured in the analysis represents roughly 3 percent of active PWSs.[101]

Results are presented across four race/ethnicity groups, consistent with the subpopulation definitions used to estimate the national quantified benefits for the final PFAS NPDWR (see Section 8.1). These race/ethnicity groups include: non-Hispanic Black, Hispanic, non-Hispanic White, and Other.[102] Race/ethnicity categories examined in the EPA's analysis using SafeWater MCBC differ from the demographic groups presented in the exposure analysis discussed previously in this chapter due to the availability of demographic information utilized in the EPA's quantified benefits analysis. For more information on the selection of data inputs to the EPA's benefit analysis, see Chapter 6.

The total sample population captured by the EPA's analysis using SafeWater MCBC is roughly 196 million people, with a breakdown by race/ethnicity and income group as follows:

- Non-Hispanic Black: 25.1 million (~13%)
- Hispanic: 32.6 million (~17%)
- Other: 12.2 million (~6%)

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[101] The number of active PWSs was retrieved from SDWIS/Fed fourth quarter 2021.

[102] The "Other" race/ethnicity category includes any race/ethnicity populations that are not non-Hispanic Black, Hispanic, or non-Hispanic White.

- Non-Hispanic White: 125.9 million (~64%)
- Income below twice the poverty level: 61.6 million (~32%)
- Income above twice the poverty level: 133.9 million (~68%)

When compared to the breakdown of the total U.S. population by these same race/ethnicity groups, the makeup of the sample population in the EPA's analysis is generally representative of the overall U.S. population. Non-Hispanic Black, Hispanic, and Other race/ethnicity groups (making up ~13 percent, ~19 percent, and ~8 percent of the U.S. population, respectively) are slightly underrepresented, while the non-Hispanic White race/ethnicity group (making up ~60% of the U.S. population) is slightly overrepresented in the EPA's analysis (U.S. Census Bureau, 2020a).

Because demographic proportion information utilized in the EPA's benefits analysis was available at the county level, the EPA utilized the following step-by-step approach to identify the number of people in each race/ethnicity and income group within a given PWS service area. Specifically, in this order, the EPA utilized the following stepwise approach:

1. Overlayed census block groups with PWS service area boundaries;
2. Calculated the area of each census block group and PWS service area boundary;
3. Calculated the percent of each census block group overlapping each PWS service area boundary;
4. Multiplied the population of the census block group by the percent of each census block overlapping each PWS service area boundary;
5. Summed across census block groups to calculate the population in each PWS service area boundary that lives in each county;
6. Calculated the percent of the population in each county (PWS_county_weight) for each PWS; and
7. Estimated the number of people served by a PWS for each subpopulation as follows:

Equation 21:

$$SubPop = \sum_{c} PWS\_county\_weight_c \ x \ PWS\_Pop \ x \ Subpop\_share_c$$

Where:

$SubPop$ = number of people in each subpopulation served by a PWS

$PWS\_County\_weight_c$ = the percentage of the PWS population in each county (c)

PWS_Pop = Number of people served by PWS from SDWIS/Fed inventory

$Subpop\_share_c$ = The share of county (c) population consisting of the subpopulation from the U.S. Census

As part of its national analysis of quantified benefits and costs using SafeWater MCBC, the EPA accounted for states that have enacted enforceable MCLs for PFAS contaminants. For these states, the EPA assumed that the state MCL is the maximum baseline PFAS occurrence value for all EPs in the state. For more information on this assumption and on state-enacted MCLs, see Section 4. The EPA has applied this assumption as part of its EJ analysis conducted in SafeWater MCBC.

8.4.2 SafeWater EJ Analysis Results

8.4.2.1 Health Benefits

To determine if there are disproportionate health impacts borne by any race/ethnicity subpopulation or income group under the final rule or regulatory alternatives, the EPA estimated the annual avoided cases of mortality and morbidity per 100,000 people, as shown in Table 8-22 through Table 8-25.

For the analysis conducted in SafeWater MCBC, the EPA reports the estimated avoided cases of mortality and morbidity by race/ethnicity and income groups for the following health endpoints:

- CVD: Non-fatal MI, non-fatal IS, CVD deaths
- RCC: Non-fatal RCC cases avoided, fatal RCC cases avoided
- Birth weight: Birth weight gain (total grams), birth weight-related deaths avoided

Baseline incidence associated with these health endpoints varies by demographic group, and disparities in underlying incidence by demographic group likely influence the distribution of quantified health benefits expected under the final PFAS NPDWR. For example, non-fatal MI incidence is generally most prevalent among non-Hispanic White males, while non-fatal IS incidence is generally most prevalent among non-Hispanic Black males. Additionally, low income and poverty are linked to higher cancer mortality rates. Survival after a cancer diagnosis is shorter for people of all races who have a lower socio-economic status (National Cancer Institute, 2020). The demographic distribution of quantified health benefits presented here incorporates differing incidence in baseline health outcomes by race/ethnicity. However, the demographic distribution of quantified health benefits that the EPA reports here have not been adjusted for income. For a detailed breakdown of incidence associated with the effects of reduced birth weight on infant mortality, CVD events, and RCC by race/ethnicity, see Appendices E, G, and H, respectively.

The EPA did not analyze the demographic breakdown of bladder cancer cases avoided that are expected to result from the co-removal of PFAS and DBP precursors (discussed in Section 6.7). The EPA models bladder cancer impacts based on a national-level distribution of finished water TOC levels; because specific TOC levels at actual PWSs are not available, the EPA did not include these impacts in the portion of its EJ analysis conducted in SafeWater MCBC.

Table 8-22 summarizes the number of avoided cases of morbidity and mortality per 100,000 people per year for all health endpoints evaluated under the EPA's final regulatory option. Table 8-23 through Table 8-25 summarize the number of avoided cases of morbidity and mortality per

100,000 people per year for all health endpoints evaluated under the EPA's regulatory alternatives.

For the final rule and all regulatory alternatives, benefits are anticipated to be realized across all health endpoints and demographic groups (i.e., race/ethnicity and income) evaluated. A summary of benefits anticipated for each health endpoint is included below. In general, when comparing benefits under the final rule to those across regulatory alternatives, the distribution of quantified health benefits for a given demographic group is relatively similar. Variation exists between the final rule and regulatory alternatives with respect to the total amount of health benefits anticipated. Additionally, across all health endpoints evaluated and across all race/ethnicity groups, the greatest benefits are anticipated under the final rule.

Below is a summary of quantified health benefits categorized by endpoint, with results presented across the final rule and regulatory alternatives and across demographic groups.

Cardiovascular Disease

Non-Fatal MI Cases Avoided – Under the final rule and all alternatives and across all race/ethnicity and income groups, values range from 1.07 to 3.78 cases avoided per 100,000 people per year. Under the final rule and all alternatives, the EPA anticipates the greatest benefit to accrue to the Hispanic race/ethnicity group and the lowest benefit to accrue to the non-Hispanic Black race/ethnicity group. The number of MI cases avoided per 100,000 people per year is similar across income groups (e.g., for the final rule, 3.09 cases avoided per 100,000 people for populations with income below twice the poverty level vs. 2.99 cases avoided per 100,000 people for populations with income above twice the poverty level).

Non-Fatal IS Cases Avoided – Under the final rule and all alternatives and across all race/ethnicity and income groups, values range from 1.58 to 7.48 cases avoided per 100,000 people per year. Under the final rule and all alternatives, the EPA anticipates the greatest benefit to accrue to the non-Hispanic Black race/ethnicity group. Under the final rule, the EPA anticipates the lowest benefit to accrue to the non-Hispanic White race/ethnicity group, though this is not the case across all regulatory alternatives evaluated.[103] The number of IS cases avoided per 100,000 people per year is similar across income groups (e.g., for the final rule, 4.68 cases avoided per 100,000 people for populations with income below twice the poverty level vs. 4.45 cases avoided per 100,000 people for populations with income above twice the poverty level).

CVD Deaths Avoided – Under the final rule and all alternatives and across all race/ethnicity and income groups, values range from 0.53 to 3.90 deaths avoided per 100,000 people per year. Under the final rule and all alternatives, the EPA anticipates the greatest benefit to accrue to the non-Hispanic Black race/ethnicity group. The lowest benefit is anticipated to accrue to the non-Hispanic White race/ethnicity group under the final rule and Options 1a and 1b, whereas the Other race/ethnicity group is anticipated to experience the lowest benefit under Option 1c. The number of deaths avoided per 100,000 people per year is similar across income groups (e.g., for the final rule, 1.72 deaths avoided per 100,000 people for populations with income below twice

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[103] The non-Hispanic White race/ethnicity group is anticipated to experience the lowest benefit related to non-fatal IS cases avoided under the final rule and under Options 1a and 1b. Under Option 1c, the Other race/ethnicity group is anticipated to experience the lowest benefit for non-fatal IS cases avoided, (i.e., 1.58 cases avoided per 100,000 people).

the poverty level vs. 1.62 deaths avoided per 100,000 people for populations with income above twice the poverty level).

Renal Cell Carcinoma

Non-Fatal RCC Cases Avoided – Under the final rule and all alternatives and across all race/ethnicity and income groups, values range from 0.98 to 4.04 cases avoided per 100,000 people per year. Under the final rule and all alternatives, the EPA anticipates the greatest benefit to accrue to Hispanic race/ethnicity groups and the lowest benefit to accrue to the non-Hispanic White race/ethnicity group. The number of cases avoided per 100,000 people per year is similar across income groups (e.g., for the final rule, 3.09 cases avoided per 100,000 people for populations with income below twice the poverty level vs. 3.02 cases avoided per 100,000 people for populations with income above twice the poverty level).

Fatal RCC Cases Avoided – Under the final rule and all alternatives and across all race/ethnicity and income groups, values range from 0.26 to 1.44 deaths avoided per 100,000 people per year. Under the final rule and all alternatives, the EPA expects the greatest benefit to accrue to the Hispanic race/ethnicity group and the lowest benefit to accrue to the non-Hispanic White race/ethnicity group. The number of deaths avoided per 100,000 people per year is similar across income groups (e.g., for the final rule, 0.91 deaths avoided per 100,000 people for populations with income below twice the poverty level vs. 0.88 deaths avoided per 100,000 people for populations with income above twice the poverty level).

Birth Weight

Birth Weight Gain (total grams) – Under the final rule and all alternatives and across all race/ethnicity and income groups, values range from 32,431 grams to 167,846 grams of birth weight gain per 100,000 people per year. Under the final rule and all alternatives, the EPA expects the largest benefit to accrue to the Hispanic race/ethnicity group and the lowest benefit to accrue to the non-Hispanic White race/ethnicity group. The EPA also expects slightly larger benefits to accrue to populations with income below twice the poverty level (100,943 grams of birth weight gain per 100,000 people per year under the final rule) compared to populations with income above twice the poverty level (93,366 grams of birth weight gain per 100,000 people per year under the final rule).

Birth Weight-Related Deaths Avoided – Under the final rule and all alternatives and across all race/ethnicity and income groups, values range from 0.19 to 1.00 birth weight-related deaths avoided per 100,000 people per year. Under the final rule and all alternatives, the EPA anticipates the greatest benefit to accrue to the non-Hispanic Black race/ethnicity group and the lowest benefit to accrue to the non-Hispanic White race/ethnicity group. The number of birth weight-related deaths avoided per 100,000 people per year is similar across income groups (e.g., for the final rule, 0.62 deaths avoided per 100,000 people for populations with income below twice the poverty level vs. 0.55 deaths avoided per 100,000 people for populations with income above twice the poverty level).

Table 8-22: Annualized Cases Avoided per 100,000 People by Race/Ethnicity and Income Group, Final Rule (PFOA and PFOS MCLs of 4.0 ppt each, PFHxS, PFNA, HFPO-DA MCLs of 10 ppt each and HI of 1)

Health Endpoint	Race and Ethnicity				Income	
	Non-Hispanic Black	Hispanic	Other	Non-Hispanic White	Below Twice the Poverty Level	Above Twice the Poverty Level
Non-Fatal MI Cases Avoided	2.34	3.78	3.52	2.91	3.09	2.99
Non-Fatal IS Cases Avoided	7.48	5.33	3.87	3.78	4.68	4.45
CVD Deaths Avoided	3.90	1.57	1.29	1.26	1.72	1.62
Non-Fatal RCC Cases Avoided	3.31	4.04	3.04	2.73	3.09	3.02
Fatal RCC Cases Avoided	0.96	1.44	0.86	0.74	0.91	0.88
Birth Weight Gain (total grams)	122,024	167,846	102,190	71,201	100,943	93,366
Birth Weight-Related Deaths Avoided	1.00	0.93	0.47	0.41	0.62	0.55

Abbreviations: CVD – cardiovascular disease; MI – myocardial infarction; IS – ischemic stroke; RCC – renal cell carcinoma.

Table 8-23: Annualized Cases Avoided per 100,000 People by Race/Ethnicity and Income Group, Option 1a (PFOA and PFOS MCLs of 4.0 ppt)

Health Endpoint	Race and Ethnicity				Income	
	Non-Hispanic Black	Hispanic	Other	Non-Hispanic White	Below Twice the Poverty Level	Above Twice the Poverty Level
Non-Fatal MI Cases Avoided	2.32	3.76	3.50	2.90	3.07	2.97
Non-Fatal IS Cases Avoided	7.44	5.30	3.85	3.76	4.65	4.42
CVD Deaths Avoided	3.88	1.56	1.28	1.26	1.71	1.62
Non-Fatal RCC Cases Avoided	3.29	4.02	3.02	2.72	3.07	3.00
Fatal RCC Cases Avoided	0.96	1.43	0.85	0.73	0.90	0.87
Birth Weight Gain (total grams)	121,470	166,945	101,591	70,745	100,345	92,829
Birth Weight-Related Deaths Avoided	0.99	0.92	0.47	0.41	0.62	0.55

Abbreviations: CVD – cardiovascular disease; MI – myocardial infarction; IS – ischemic stroke; RCC – renal cell carcinoma.

Table 8-24: Annualized Cases Avoided per 100,000 People by Race/Ethnicity and Income Group, Option 1b (PFOA and PFOS MCLs of 5.0 ppt)

Health Endpoint	Race and Ethnicity				Income	
	Non- Hispanic Black	Hispanic	Other	Non-Hispanic White	Below Twice the Poverty Level	Above Twice the Poverty Level
Non-Fatal MI Cases Avoided	2.00	3.30	2.96	2.46	2.64	2.54
Non-Fatal IS Cases Avoided	6.40	4.66	3.26	3.19	4.01	3.78
CVD Deaths Avoided	3.34	1.37	1.09	1.07	1.47	1.38
Non-Fatal RCC Cases Avoided	2.75	3.48	2.50	2.22	2.57	2.49
Fatal RCC Cases Avoided	0.80	1.23	0.70	0.60	0.76	0.73
Birth Weight Gain (total grams)	105,756	147,990	86,953	60,483	87,588	80,186
Birth Weight-Related Deaths Avoided	0.86	0.82	0.40	0.35	0.54	0.48

Abbreviations: CVD – cardiovascular disease; MI – myocardial infarction; IS – ischemic stroke; RCC – renal cell carcinoma.

Table 8-25: Annualized Cases Avoided per 100,000 People by Race/Ethnicity and Income Group, Option 1c (PFOA and PFOS MCLs of 10.0 ppt)

Health Endpoint	Race and Ethnicity				Income	
	Non- Hispanic Black	Hispanic	Other	Non-Hispanic White	Below Twice the Poverty Level	Above Twice the Poverty Level
Non-Fatal MI Cases Avoided	1.07	1.93	1.43	1.26	1.45	1.32
Non-Fatal IS Cases Avoided	3.41	2.73	1.58	1.64	2.21	1.97
CVD Deaths Avoided	1.78	0.81	0.53	0.55	0.81	0.72
Non-Fatal RCC Cases Avoided	1.35	1.99	1.13	0.98	1.28	1.17
Fatal RCC Cases Avoided	0.39	0.71	0.32	0.26	0.38	0.35
Birth Weight Gain (total grams)	59,981	89,583	44,661	32,431	50,809	44,150
Birth Weight-Related Deaths Avoided	0.49	0.49	0.21	0.19	0.32	0.26

Abbreviations: CVD – cardiovascular disease; MI – myocardial infarction; IS – ischemic stroke; RCC – renal cell carcinoma.

8.4.2.2 Household Costs

For category 1 and 2 PWS service areas, the EPA used SafeWater MCBC to estimate the distribution of annualized incremental household costs across race/ethnicity and income groups. The results are provided by system size category in Table 8-26 through Table 8-29. In addition to presenting annualized incremental household costs for each race/ethnicity group in Table 8-26 and Table 8-28, the EPA also presents household costs across "All" race/ethnicity groups to provide a basis for comparison. Table 8-28 and Table 8-29 present annualized incremental household costs by income group.

In estimating annualized incremental household costs of the final PFAS NPDWR, SafeWater MCBC first divided each PWS's total compliance costs by the PWS's average daily flow to determine the cost of compliance per 1,000 gallons of daily flow. Next, this cost was multiplied by the average household consumption from the Community Water System Survey (CWSS) to calculate the average household cost of compliance for the PWS. To calculate the average household cost for each race/ethnicity group by PWS system size strata, for each PWS included in the subset of systems in the EPA's EJ analysis, the EPA calculated a weighted average household cost by using the number of people in each race/ethnicity or income group served by each PWS as the weight. In addition to estimating the demographic breakdown of annualized incremental household costs of the final PFAS NPDWR for all systems included in the EPA's EJ analysis, the EPA also estimated the demographic breakdown of annualized incremental household costs for just the subset of PWSs that are anticipated to install treatment to comply with the rule.[104]

Below is a summary of the demographic distribution of incremental household costs, categorized by system size, for the final rule and regulatory alternatives. Results are presented both for the entire subset of PWSs included in the EPA's EJ analysis and just those anticipated to install treatment under the rule. Note that an analysis of household costs served by systems serving fewer than 3,300 people could not be completed due to limited sample size. In general, across all demographic groups and system size categories, the final rule is anticipated to have the highest associated costs and Option 1c is anticipated to have the lowest associated costs.

8.4.2.2.1 Incremental Household Costs for All PWSs

System size 3,300 to 10,000 – Annualized incremental household costs range from $5.88 to $29.26 per year across the final rule and regulatory alternatives and across race/ethnicity groups. When comparing household costs borne by particular race/ethnicity groups to those borne by the overall population served by systems in this size category, the non-Hispanic Black and Other race/ethnicity groups bear minimally elevated household costs under the final rule and all regulatory alternatives. Additionally, the Hispanic race/ethnicity group bears minimally elevated household costs under the final rule and Options 1a and 1b. The magnitude of household cost differences between each of these race/ethnicity groups and the overall population is small,

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[104] For additional detail on treatment technology selection among systems anticipated to install treatment under the proposed rule, see Section 5.3.1.1.

ranging from $0.03 to $4.04 per year across race/ethnicity groups and across the final rule and regulatory alternatives. The Other race/ethnicity group bears the highest household costs under the final rule and Options 1a and 1b, whereas the non-Hispanic Black race/ethnicity group bears the highest household costs under Option 1c. The non-Hispanic White race/ethnicity group bears the lowest household costs under the final rule and Options 1a and 1b, whereas the Hispanic race/ethnicity groups bears the lowest household costs under Option 1c. When comparing incremental household costs across income groups, costs range from $4.99 to $26.93 per year across the final rule and regulatory alternatives. Populations with income above twice the poverty level bear higher incremental household costs compared to populations with income below twice the poverty level, with the cost difference ranging from $1.76 to $5.00.

System size 10,000 to 50,000 – Annualized incremental household costs range from $4.34 to $16.41 per year across the final rule and regulatory alternatives and across race/ethnicity groups. When comparing household costs borne by particular race/ethnicity groups to those borne by the overall population served by systems in this size category, the Other race/ethnicity group bears minimally elevated household costs under the final rule and all regulatory alternatives. The Hispanic race/ethnicity group also bears minimally elevated household costs under Option 1c. The magnitude of household cost differences between each of these race/ethnicity groups and the overall population is very small, ranging from $0.02 to $1.74 per year across race/ethnicity groups and across the final rule and regulatory alternatives. Under the final rule and all regulatory alternatives, the Other race/ethnicity group bears the highest household costs, whereas the non-Hispanic Black race/ethnicity group bears the lowest household costs. Under Option 1b, both the non-Hispanic Black and non-Hispanic White race/ethnicity groups bear the lowest household costs. When comparing incremental household costs across income groups, costs range from $4.13 to $15.07 per year across the final rule and regulatory alternatives. Populations with income above twice the poverty level bear slightly higher incremental household costs compared to populations with income below twice the poverty level, with the cost difference ranging from $0.45 to $1.32.

System size 50,000 to 100,000 – Annualized incremental household costs range from $3.29 to $13.67 per year across the final rule and regulatory alternatives and across race/ethnicity groups. When comparing household costs borne by particular race/ethnicity groups to those borne by the overall population served by systems in this size category, the Hispanic and Other race/ethnicity groups bear minimally elevated household costs under the final rule and all regulatory alternatives. Additionally, the non-Hispanic Black race/ethnicity group bears minimally elevated household costs under Option 1c. The magnitude of household cost differences between each of these race/ethnicity groups and the overall population is very small, ranging from $0.10 to $1.00 per year across race/ethnicity groups and across the final rule and regulatory alternatives. The Other race/ethnicity group bears the highest household costs under the final rule and Options 1a and 1b, whereas the Hispanic race/ethnicity group bears the highest household cost under Option 1c. The non-Hispanic Black race/ethnicity group bears the lowest household costs under the final rule and Option 1a, whereas the non-Hispanic White race/ethnicity group bears the lowest household costs under Options 1b and 1c. When comparing incremental household costs across income groups, costs range from $3.42 to $12.87 per year across the final rule and regulatory alternatives. Populations with income below twice the poverty level bear slightly higher incremental household costs compared to populations with income above twice the poverty level. However, the magnitude of these cost differences is small, ranging from $0.14 to $0.31.

System size 100,000 to 1,000,000 – Annualized incremental household costs range from $3.59 to $13.46 per year across the final rule and regulatory alternatives and across race/ethnicity groups. When comparing household costs borne by particular race/ethnicity groups to those borne by the overall population served by systems in this size category, the non-Hispanic Black, Hispanic, and Other race/ethnicity groups bear minimally elevated household costs under the final rule and all regulatory alternatives. As in other system size categories, the magnitude of household cost differences between each of these race/ethnicity groups and the overall population is small, ranging from $0.11 to $1.18 per year across race/ethnicity groups and across the final rule and regulatory alternatives. The Hispanic race/ethnicity group bears the highest household costs under the final rule and Options 1a and 1b, whereas the Other race/ethnicity group bears the highest household costs under Option 1c. The non-Hispanic White race/ethnicity group bears the lowest household costs. When comparing incremental household costs across income groups, costs range from $3.73 to $12.44 per year across the final rule and regulatory alternatives. Populations with income below twice the poverty level bear slightly higher incremental household costs compared to populations with income above twice the poverty level. However, the magnitude of these cost differences is very small, ranging from $0.22 to $0.31.

The EPA's comparison of annualized incremental household costs across system size categories reveals that, in general, as system size increases, incremental household costs decrease under the final rule and all regulatory alternatives and across all demographic groups. One exception to this trend is Option 1c among systems serving 100,000 to 1,000,000 people, where costs are marginally higher than costs for systems serving 50,000 to 100,000 people.

The highest incremental household costs under the final rule and all regulatory alternatives are realized for the smallest systems (i.e., systems serving 3,300 to 10,000 people). Across race/ethnicity groups examined, the range of household costs within this system size category is $5.88 to $29.26 per year, and the EPA anticipates the highest cost ($29.26 per year) under the final rule for the Other race/ethnicity group. When comparing costs across income groups, populations with income above twice the poverty level bear the highest costs ($26.93) within this system size category under the final rule. The lowest incremental household costs under the final rule and all regulatory alternatives are realized for systems serving 50,000 to 100,000 people. Across race/ethnicity groups examined, the range of household costs within this system size category is $3.29 to $13.67, with the non-Hispanic White race/ethnicity group having the lowest cost of $3.29 under Option 1c. When comparing costs across income groups within this system size category, populations with income above twice the poverty level bear the lowest costs ($3.42) within this system size category under Option 1c.

Comparing the magnitude of household costs anticipated across system size categories illustrates the role that system size plays in household costs anticipated under the final PFAS rule. This is an expected result due to economies of scale and the impact that a smaller customer and tax base has on costs per household for funding and financing capital and operational infrastructure investments. Further, this analysis includes the estimated household costs for all systems impacted by the rule, not just the systems expected to install and operate treatment after exceeding the final MCLs. Households served by water systems triggered into treatment are expected to face greater cost increases than those presented here. The EPA presents the demographic breakdown of estimated household costs for those systems anticipated to install treatment under the final rule below in Section 8.4.2.2.2. Additionally, the EPA assesses the impact of treatment technology costs specifically on small system households in the small system affordability analysis. For more information, see the EPA's assessment of small system affordability in Section 9.13.

Table 8-26: Annualized Population Weighted Household Cost by PWS Size Category and Race/Ethnicity Group ($2022)

System Size[a]	Race/Ethnicity Group	Final Rule[b]	Option 1a[c]	Option 1b[d]	Option 1c[e]
3,300 to 10,000	All	$25.22	$25.15	$18.78	$6.15
3,300 to 10,000	Non-Hispanic Black	$28.24	$28.17	$21.49	$7.89
3,300 to 10,000	Hispanic	$27.20	$27.15	$20.38	$5.88
3,300 to 10,000	Other	$29.26	$29.17	$21.69	$6.18
3,300 to 10,000	Non-Hispanic White	$24.30	$24.23	$18.01	$5.94
10,000 to 50,000	All	$14.67	$14.59	$11.32	$4.44
10,000 to 50,000	Non-Hispanic Black	$14.55	$14.48	$11.23	$4.34
10,000 to 50,000	Hispanic	$14.64	$14.57	$11.29	$4.46
10,000 to 50,000	Other	$16.41	$16.33	$12.90	$5.28
10,000 to 50,000	Non-Hispanic White	$14.57	$14.49	$11.23	$4.40
50,000 to 100,000	All	$12.67	$12.56	$9.51	$3.46
50,000 to 100,000	Non-Hispanic Black	$12.30	$12.22	$9.41	$3.66
50,000 to 100,000	Hispanic	$12.93	$12.80	$9.78	$3.84
50,000 to 100,000	Other	$13.67	$13.51	$10.27	$3.81
50,000 to 100,000	Non-Hispanic White	$12.55	$12.46	$9.38	$3.29
100,000 to 1,000,000	All	$12.28	$12.13	$9.41	$3.83
100,000 to 1,000,000	Non-Hispanic Black	$12.39	$12.24	$9.63	$4.01
100,000 to 1,000,000	Hispanic	$13.46	$13.27	$10.26	$4.27
100,000 to 1,000,000	Other	$13.25	$13.11	$10.24	$4.28
100,000 to 1,000,000	Non-Hispanic White	$11.77	$11.63	$8.98	$3.59

Notes:

[a] The number of systems serving fewer than 3,300 people represented in the UMCR 3 occurrence data is too limited to accurately estimate average population-weighted household costs by subpopulation. Therefore, results for these small systems are omitted. Also, household costs in this exhibit are population-weighted and will not match average household costs by size category shown in other exhibits in the economic analysis document that are not population-weighted.

[b] The final rule sets PFOA and PFOS MCLs of 4.0 ppt each, MCLs of 10 ppt for HFPO-DA, PFHxS, and PFNA each, and an HI of 1.

[c] Option 1a sets PFOA and PFOS MCLs of 4.0 ppt.

[d] Option 1b sets PFOA and PFOS MCLs of 5.0 ppt.

[e] Option 1c sets PFOA and PFOS MCLs of 10.0 ppt.

Table 8-27: Annualized Population Weighted Household Cost by PWS Size Category and Income Level ($2022)

System Size[a]	Income	Final Rule[b]	Option 1a[c]	Option 1b[d]	Option 1c[e]
3,300 to 10,000	Below twice the poverty level	$21.93	$21.87	$15.95	$4.99
3,300 to 10,000	Above twice the poverty level	$26.93	$26.87	$20.25	$6.75
10,000 to 50,000	Below twice the poverty level	$13.75	$13.68	$10.55	$4.13
10,000 to 50,000	Above twice the poverty level	$15.07	$15.00	$11.66	$4.58
50,000 to 100,000	Below twice the poverty level	$12.87	$12.78	$9.73	$3.56
50,000 to 100,000	Above twice the poverty level	$12.58	$12.47	$9.42	$3.42
100,000 to 1,000,000	Below twice the poverty level	$12.44	$12.28	$9.56	$4.04
100,000 to 1,000,000	Above twice the poverty level	$12.21	$12.06	$9.33	$3.73

Notes:

[a] The number of systems serving fewer than 3,300 people represented in the UMCR 3 occurrence data is too limited to accurately estimate average population-weighted household costs by subpopulation. Therefore, results for these small systems are omitted. Also, household costs in this exhibit are population-weighted and will not match average household costs by size category shown in other exhibits in the economic analysis document that are not population-weighted.

[b] The final rule sets PFOA and PFOS MCLs of 4.0 ppt each, MCLs of 10 ppt for HFPO-DA, PFHxS, and PFNA each, and an HI of 1.

[c] Option 1a sets PFOA and PFOS MCLs of 4.0 ppt.

[d] Option 1b sets PFOA and PFOS MCLs of 5.0 ppt.

[e] Option 1c sets PFOA and PFOS MCLs of 10.0 ppt.

8.4.2.2.2 Incremental Household Costs for Treating PWSs

System size 3,300 to 10,000 – Annualized incremental household costs for systems anticipated to install treatment range from $120.94 to $181.78 per year across the final rule and regulatory alternatives and across race/ethnicity and income groups. When comparing household costs borne by particular race/ethnicity groups to those borne by the overall population served by systems in this system size category, the Hispanic and Other race/ethnicity group bears minimally elevated household costs under the final rule and Option 1a. Additionally, the non-Hispanic Black race/ethnicity group bears minimally elevated household costs under Options 1b and 1c and the non-Hispanic White race/ethnicity group bears minimally elevated household costs under Option 1c. The magnitude of household cost differences between each of these race/ethnicity groups and the overall population ranges from $0.21 to $30.12 per year across race/ethnicity groups and across the final rule and regulatory alternatives. The Other race/ethnicity group bears the highest household costs under the final rule and Option 1a, whereas the non-Hispanic Black race/ethnicity group bears the highest household costs under Options 1b and 1c. The non-Hispanic Black race/ethnicity group bears the lowest household costs under the final rule and Option 1a, whereas the Hispanic race/ethnicity group bears the lowest household costs under Options 1b and 1c. Populations with income below twice the poverty level bear lower incremental household costs compared to populations with income above twice the poverty level under the final rule and all regulatory alternatives. The magnitude of household cost differences between the two income groups ranges from $0.65 to $15.96 per year across the final rule and regulatory alternatives.

System size 10,000 to 50,000 – Annualized incremental household costs for systems anticipated to install treatment range from $39.05 to $51.82 per year across the final rule and regulatory alternatives and across race/ethnicity and income groups. When comparing household costs borne by particular race/ethnicity groups to those borne by the overall population served by systems in this system size category, the Other and non-Hispanic White race/ethnicity groups bear minimally elevated household costs under the final rule and all regulatory alternatives. Additionally, the non-Hispanic Black race/ethnicity group bears minimally elevated household costs under Option 1c. The magnitude of household cost differences between each race/ethnicity group and the overall population is small, ranging from $0.05 to $2.55 per year across race/ethnicity groups and across the final rule and regulatory alternatives. The Other race/ethnicity group bears the highest household costs under the final rule and Options 1a and 1b, whereas the non-Hispanic Black race/ethnicity group bears the highest household costs under Option 1c. The Hispanic race/ethnicity group bears the lowest household costs under the final rule and all regulatory alternatives. Populations with income below twice the poverty level bear slightly lower incremental household costs compared to populations with income above twice the poverty level under the final rule and Options 1a and 1b; populations with income above twice the poverty level bear slightly lower incremental household costs compared to populations with income below twice the poverty level under Option 1c. The magnitude of household cost differences between the two income groups is very small, ranging from $0.22 to $1.31 per year across the final rule and regulatory alternatives.

System size 50,000 to 100,000 – Annualized incremental household costs for systems anticipated to install treatment range from $31.53 to $43.84 per year across the final rule and regulatory alternatives and across race/ethnicity and income groups. When comparing household costs borne by particular race/ethnicity groups to those borne by the overall population served by systems in this system size category, the non-Hispanic Black and non-Hispanic White race/ethnicity group bears minimally elevated costs under the final rule and all regulatory alternatives. Additionally, the Other race/ethnicity group bears minimally elevated household costs under the final rule and Options 1a and 1b. The magnitude of household cost differences between each of these race/ethnicity groups and the overall population is very small, ranging from $0.10 to $2.04 per year across race/ethnicity groups and across the final rule and regulatory alternatives. The non-Hispanic Black race/ethnicity group bears the highest household costs under the final rule and all regulatory alternatives. The Hispanic race/ethnicity group bears the lowest household costs under the final rule and all regulatory alternatives. Populations with income below twice the poverty level bear slightly lower incremental household costs compared to populations with income above twice the poverty level under the final rule and all regulatory alternatives. The magnitude of household cost differences between the two income groups is very small, ranging from $0.24 to $0.98 per year across the final rule and all regulatory alternatives.

System 100,000 to 1,000,000 – Annualized incremental household costs for systems anticipated to install treatment range from $21.63 to $32.92 per year across the final rule and regulatory alternatives and across race/ethnicity and income groups. When comparing household costs borne by particular race/ethnicity groups to those borne by the overall population served by systems in this size category, the non-Hispanic Black race/ethnicity group bears minimally elevated costs under the final rule and all regulatory alternatives. Additionally, the Hispanic race/ethnicity group bears minimally elevated costs under the final rule and Option 1a, whereas the non-Hispanic White race/ethnicity group bears minimally elevated costs under Options 1b and 1c. The magnitude of household cost differences between each of these race/ethnicity groups and the overall population is small, ranging from $0.02 to $1.73 per year across race/ethnicity groups and across the final rule and regulatory alternatives. The non-Hispanic Black race/ethnicity group bears the highest household costs under the final rule and all regulatory alternatives. The Other race/ethnicity group bears the lowest household costs under the final rule, Option 1a, and Option 1b, whereas the Hispanic race/ethnicity group bears the lowest household costs under Option 1c. Populations with income below twice the poverty level bear slightly higher incremental household costs compared to populations with income above twice the poverty level under the final rule and all regulatory alternatives. The magnitude of household cost differences between the two income groups is very small, ranging from $1.01 to $1.74 per year across the final rule and all regulatory alternatives.

Consistent with the EPA’s findings for incremental household costs across all systems, the EPA’s comparison of incremental household costs across system size categories for just treating systems reveals that, in general, as system size increases, annualized incremental household costs decrease under the final rule and all regulatory alternatives and across all race/ethnicity and income groups.

The highest annualized incremental household costs for treating systems under the final rule and all regulatory alternatives are realized for the smallest systems, with the range of incremental household costs for systems serving 3,300 to 10,000 people ranging from $120.94 to $181.78 per year across race/ethnicity groups examined. The EPA anticipates the highest cost ($181.78 per year) under Option 1a for the Other race/ethnicity group. Among the two income groups, the EPA anticipates the highest cost ($180.70) under the final rule for populations with income above twice the poverty level. Systems serving 100,000 to 1,000,000 people bear the lowest annualized incremental household costs for treating systems under the final rule and all options.

This analysis provides an opportunity to understand the demographic breakdown of incremental household costs anticipated to be incurred due to treatment installation needed to comply with the final PFAS NPDWR. Annualized incremental household costs for systems required to install treatment are higher for all size categories and across all demographic groups compared to incremental household costs across all systems. These differences are expected, as treatment installation costs are higher than other compliance costs (i.e., monitoring and reporting). In some cases, such as for communities served by the smallest systems (i.e., systems serving 3,300 to 10,000 people), the annual incremental household costs isolated among only systems anticipated to install treatment are over $100 higher than annual incremental household costs averaged across all systems.

Table 8-28: Annualized Population-Weighted Household Cost for Treating PWSs by Size Category and Race/Ethnicity Group

System Size[a]	Race/Ethnicity Group	Final Rule[b]	Option 1a[c]	Option 1b[d]	Option 1c[e]
3,300 to 10,000	All	$175.66	$175.56	$167.04	$151.06
3,300 to 10,000	Non-Hispanic Black	$174.17	$173.99	$169.33	$163.10
3,300 to 10,000	Hispanic	$177.67	$177.49	$166.11	$120.94
3,300 to 10,000	Other	$181.64	$181.78	$166.46	$123.78
3,300 to 10,000	Non-Hispanic White	$175.25	$175.17	$166.83	$156.09
10,000 to 50,000	All	$50.92	$50.69	$48.07	$41.03
10,000 to 50,000	Non-Hispanic Black	$50.78	$50.56	$48.02	$41.42
10,000 to 50,000	Hispanic	$48.37	$48.16	$45.57	$39.05
10,000 to 50,000	Other	$51.82	$51.60	$48.92	$41.38
10,000 to 50,000	Non-Hispanic White	$51.39	$51.16	$48.53	$41.36
50,000 to 100,000	All	$43.08	$42.74	$40.51	$32.81
50,000 to 100,000	Non-Hispanic Black	$43.84	$43.58	$41.86	$34.85
50,000 to 100,000	Hispanic	$41.34	$40.96	$38.71	$31.53
50,000 to 100,000	Other	$43.50	$43.04	$40.61	$31.72
50,000 to 100,000	Non-Hispanic White	$43.43	$43.11	$40.82	$33.01
100,000 to 1,000,000	All	$32.62	$32.24	$29.63	$23.34
100,000 to 1,000,000	Non-Hispanic Black	$32.92	$32.54	$30.34	$25.07
100,000 to 1,000,000	Hispanic	$32.77	$32.31	$29.29	$21.63
100,000 to 1,000,000	Other	$32.07	$31.76	$28.95	$22.48
100,000 to 1,000,000	Non-Hispanic White	$32.56	$32.20	$29.65	$23.69

Notes:

[a] The number of systems serving fewer than 3,300 people represented in the UMCR 3 occurrence data is too limited to accurately estimate average population-weighted household costs by subpopulation. Therefore, results for these small systems are omitted. Also, household costs in this exhibit are population-weighted and will not match average household costs by size category shown in other exhibits in the economic analysis document that are not population-weighted.

[b] The final rule sets PFOA and PFOS MCLs of 4.0 ppt each, MCLs of 10 ppt for HFPO-DA, PFHxS, and PFNA each, and an HI of 1.

[c] Option 1a sets PFOA and PFOS MCLs of 4.0 ppt.

[d] Option 1b sets PFOA and PFOS MCLs of 5.0 ppt.

[e] Option 1c sets PFOA and PFOS MCLs of 10.0 ppt.

Table 8-29: Annualized Population Weighted Household Cost for Treating PWSs by PWS Size Category and Income Level ($2022)

System Size[a]	Income	Final Rule[b]	Option 1a[c]	Option 1b[d]	Option 1c[e]
3,300 to 10,000	Below twice the poverty level	$164.78	$164.65	$158.28	$150.58
3,300 to 10,000	Above twice the poverty level	$180.70	$180.61	$170.88	$151.23
10,000 to 50,000	Below twice the poverty level	$49.99	$49.77	$47.38	$41.19
10,000 to 50,000	Above twice the poverty level	$51.30	$51.07	$48.35	$40.97
50,000 to 100,000	Below twice the poverty level	$42.86	$42.58	$40.23	$32.15
50,000 to 100,000	Above twice the poverty level	$43.18	$42.82	$40.64	$33.13
100,000 to 1,000,000	Below twice the poverty level	$33.33	$32.92	$30.45	$24.51
100,000 to 1,000,000	Above twice the poverty level	$32.28	$31.91	$29.24	$22.77

Notes:

[a] The number of systems serving fewer than 3,300 people represented in the UMCR 3 occurrence data is too limited to accurately estimate average population-weighted household costs by subpopulation. Therefore, results for these small systems are omitted. Also, household costs in this exhibit are population-weighted and will not match average household costs by size category shown in other exhibits in the economic analysis document that are not population-weighted.

[b] The final rule sets PFOA and PFOS MCLs of 4.0 ppt each, MCLs of 10 ppt for HFPO-DA, PFHxS, and PFNA each, and an HI of 1.

[c] Option 1a sets PFOA and PFOS MCLs of 4.0 ppt.

[d] Option 1b sets PFOA and PFOS MCLs of 5.0 ppt.

[e] Option 1c sets PFOA and PFOS MCLs of 10.0 ppt.

8.5 Conclusions

This section provides a summary of the EJ analyses for estimating the demographic distribution of baseline PFAS exposure and exposure over several thresholds as well as the cost and benefits of the final PFAS NPDWR.

8.5.1 EJ PFAS Exposure Analysis

The EPA's analysis of demographic groups with PFAS exposure over baseline thresholds based on trigger levels of 2 ppt demonstrates that certain communities of color experience elevated baseline PFAS drinking water exposures compared to the entire sample population. For example, the percentage of non-Hispanic Black and Hispanic populations with PFAS drinking water exposure above baseline thresholds is greater than the percentage of the total populations served across all PFAS analytes considered in this analysis. Similarly, when these results are further filtered by system size, for large systems, non-Hispanic Black and Hispanic populations have higher baseline PFAS drinking water exposure compared to the percentage of the total population served across all demographic groups. For small systems, non-Hispanic Asian and non-Hispanic Black populations served have higher baseline PFAS drinking water exposure compared to the percentage of the total population served across all demographic groups for particular PFAS analytes.

Across all hypothetical regulatory thresholds, elevated exposure—and thus expected reductions in exposure under the hypothetical regulatory scenarios—is anticipated to occur in communities of color and/or low-income populations. The EPA estimates the most notable differences in anticipated reductions in exposure are for Hispanic populations, specifically when using UCMR 5 MRL values as hypothetical regulatory thresholds in the analysis. The results from the EPA's analysis indicate that Hispanic populations are estimated to experience at least two percentage points higher rates of exposure to all PFAS analytes examined in this analysis. Hispanic populations are therefore also anticipated to experience greater reductions in exposure compared to the entire sample population. In addition, under hypothetical regulatory thresholds set at the UCMR 5 MRL values, the EPA anticipates some of the largest reductions in exposure to PFOA and PFHxS to occur for non-Hispanic Native American or Alaska Native and non-Hispanic Pacific Islander populations due to relatively high concentration levels when these PFAS are detected at PWSs serving these groups.

These findings are supported by literature that indicates that communities of lower socioeconomic status are more likely to live near environmentally hazardous facilities and face disproportionate impacts of exposure to toxic chemicals than communities of relatively higher socioeconomic status (Brown, 1995; Brulle & Pellow, 2006; Banzhaf et al., 2019; U.S. EPA, 1994). The literature also indicates that people of color and low-income populations are more likely to be served by water systems with higher PFAS occurrence or reside in proximity to a PFAS contamination site, thereby increasing baseline exposure (Black et al., 2021; Lee, Kang, et al., 2021; Desikan et al., 2019).

8.5.2 SafeWater EJ Analysis of Regulatory Options

The EPA's analysis of the demographic distribution of health benefits and household costs anticipated to result from the final PFAS NPDWR and regulatory alternatives evaluated demonstrates that, in general, across demographic groups, the EPA's final rule offers the greatest quantified benefits when compared to benefits anticipated to result under the regulatory alternatives. Additionally, in general, when compared to regulatory alternatives evaluated, the EPA's final rule will result in the highest household costs.

Under the final rule, across all health endpoints evaluated, communities of color (i.e., Hispanic, non-Hispanic Black, and/or Other race/ethnicity groups) are anticipated to experience the greatest reductions in adverse health effects associated with PFAS exposure, resulting in the greatest quantified benefits associated with the final rule. For instance, non-Hispanic Black populations are expected to experience 7.48 avoided non-fatal IS cases and 3.90 avoided CVD deaths per 100,000 people per year, as compared to 3.78 avoided non-fatal IS cases and 1.26 avoided CVD deaths per 100,000 people per year for non-Hispanic White populations. Additionally, under the final rule, while in most cases the difference in cases of illnesses and deaths avoided across income groups is small, quantified health benefits are higher for low-income communities (i.e., populations with income below twice the poverty level) across all health endpoints evaluated, compared to populations with income above twice the poverty level.

The EPA's findings could be driven by disparities in baseline exposure to PFAS and underlying disparities in death and/or disease incidence by race/ethnicity. This potential explanation is supported by literature demonstrating that overburdened communities continue to experience elevated rates of morbidity and mortality (Uche et al., 2021; Driscoll & Gregory, 2021; Fryar et al., 2017). Additionally, evidence in the literature indicates that people of color and low-income populations are more likely to be served by water systems with higher PFAS occurrence or reside in close proximity to a PFAS contamination site, which also supports this finding (Black et al., 2021; Lee, Kang, et al., 2021; Desikan et al., 2019).

When examining costs anticipated to result from the final rule, the EPA found that cost differences across demographic groups are typically small, with no clear unidirectional trend in cost differences based on demographic group. In some cases, the EPA found that communities of color and low-income communities are anticipated to bear minimally increased costs but in other cases, costs to communities of color and low-income communities are anticipated to be lower than those across all race/ethnicity groups or populations with income above twice the poverty level, respectively.

Additionally, incremental household costs to all race/ethnicity and income groups generally decrease as system size increases, which is expected due to economies of scale. This is especially true if systems serving these communities are required to install treatment to comply with the PFAS NPDWR. For example, systems serving 3,300 to 10,000 people that will be required to install treatment to comply with the final rule have substantially higher costs than systems in all larger size categories, irrespective of demographic group.

8.5.3 Overall Environmental Justice Conclusion

The EPA conducted the EJ analyses presented in this chapter on populations served by a subset of PWSs to assess the demographic distribution of exposure to PFAS and the EJ impacts that are anticipated to result from the final PFAS NPDWR. The EPA conducted two separate analyses to address the following questions:

1. Are population groups of concern (i.e., people of color and low-income populations) disproportionately exposed to PFAS compounds in drinking water delivered by PWSs?
2. Are population groups of concern disproportionately affected by the final rule?
3. If any disproportionate impacts are identified, do they create or mitigate baseline EJ concerns?

When examined collectively, results from these analyses identify communities of color and low-income communities as being disproportionately exposed to PFAS in drinking water under baseline conditions. In one hypothetical regulatory scenario, non-Hispanic American Indian or Alaska Native, non-Hispanic Black, and Hispanic populations face elevated exposure across nearly all PFAS analytes examined when compared to the total population served. In some cases, these communities experience twice the rate of PFAS exposure in drinking water in comparison to non-Hispanic White populations. When quantifying the race/ethnicity distribution of quantified health benefits anticipated to result from the final PFAS NPDWR, the EPA found that of the race/ethnicity groups evaluated, communities of color are anticipated to experience the greatest health benefits under the final rule and all regulatory alternatives.

When comparing benefits across the final rule and regulatory alternatives, quantified health benefits were generally the highest for communities of color under the final rule. This finding could be influenced by the fact that elevated baseline exposure rates for these populations translate to higher benefits associated with the final rule, as greater reductions in exposure are anticipated to occur as a result of implementing the final PFAS NPDWR.

To alleviate potential cost disparities identified by the EPA's analysis, there may be an opportunity for many communities to utilize BIL (P.L. 117-58) funding to provide financial assistance for addressing emerging contaminants. BIL funding has specific allocations for both disadvantaged and/or small communities and emerging contaminants, including PFAS.

9 Statutory and Administrative Requirements

As part of the rulemaking process, the EPA is required to address the burden that the final rule may place on certain types of governments, businesses, and populations. This chapter presents analyses performed by the EPA in accordance with the following federal mandates and statutory requirements:

1. Executive Order 12866: Regulatory Planning and Review and Executive Order 13563 (2011): Modernizing Regulatory Review.
2. Paperwork Reduction Act (PRA) (U.S. EPA, 2010b).
3. The Regulatory Flexibility Act (RFA) of 1980, as amended by the Small Business Regulatory Enforcement Fairness Act (SBREFA) of 1996.
4. Unfunded Mandates Reform Act (UMRA) of 1995.
5. Executive Order 13132: Federalism.
6. Executive Order 13175: Consultation and Coordination with Indian Tribal Governments.
7. Executive Order 13045: Protection of Children from Environmental Health and Safety Risks.
8. Executive Order 13211: Actions That Significantly Affect Energy Supply, Distribution, or Use.
9. National Technology Transfer and Advancement Act of 1995 (NTTAA).
10. Executive Order 12898: Federal Action to Address Environmental Justice in Minority Populations and Low-Income Populations and Executive Order 14096: Revitalizing our Nation’s Commitment to Environmental Justice for All.
11. Consultations with the Science Advisory Board (SAB), National Drinking Water Advisory Council (NDWAC), and the Department of Health and Human Services.
12. SDWA Section 1412(b)(4)(E) National Small System Affordability Determination.

Many of the statutory requirements and executive orders listed above call for an explanation of why the final requirements are necessary, the statutory authority for the final requirements, and the primary objectives that the final requirements are intended to achieve (see Chapter 3 for additional information regarding the need for the final rule). Others are designed to assess the financial and health effects of the final regulatory requirements on sensitive, low-income, and tribal populations as well as on small systems and governments.

9.1 Executive Order 12866: Regulatory Planning and Review and Executive Order 14094: Modernizing Regulatory Review

Executive Order 12866, 1993 (58 FR 51735, October 4, 1993), as amended by Executive Order 14094 (88 FR 21879, April 6, 2023) gives OMB the authority to review regulatory actions that are categorized as “significant” under section 3(f) of Executive Order 12866. The Order defines “significant regulatory action” as one that is likely to result in a rule that may:

1. Have an annual effect on the economy of $200 million or more (adjusted every 3 years by the Administrator of OIRA for changes in gross domestic product); or adversely affect in a material way the economy, a sector of the economy, productivity, competition, jobs, the environment, public health or safety, or state, local, or tribal governments or communities;
2. Create a serious inconsistency or otherwise interfere with an action taken or planned by another agency;
3. Materially alter the budgetary impact of entitlements, grants, user fees, or loan programs or the rights and obligations of recipients thereof; or
4. Raise legal or policy issues for which centralized review would meaningfully further the President's priorities or the principles set forth in this Executive order, as specifically authorized in a timely manner by the Administrator of OIRA in each case.

This action is an economically significant regulatory action that was submitted to the OMB for review. Any changes made in response to OMB recommendations have been documented in the docket. The analysis in Chapter 7 compares the annual estimated incremental costs and the annual incremental benefits of the final rule. In addition to the monetized costs and benefits of the final regulation, a number of non-monetized impacts exist. See Sections 5.7, 6.2.2, and 6.2.3 of this EA for greater detail on the non-monetized impacts of the final regulation.

9.2 Additional Analysis Pursuant to EO 12866

The EPA is committed to understanding and addressing climate change impacts in carrying out the agency's mission of protecting human health and the environment. Pursuant to EO 12866, the EPA has estimated the carbon dioxide (CO_2) emissions associated with the operation of the best available treatment technologies the EPA expects will be used to comply with the PFAS NPDWR.

The EPA estimated the climate disbenefits of changes in CO_2 emissions expected from the final PFAS rule using estimates of the social cost of carbon (SC-CO_2) that reflect recent advances in the scientific literature on climate change and its economic impacts and incorporate recommendations made by the National Academies of Science, Engineering, and Medicine (National Academies, 2017). The EPA presented these estimates in the regulatory impact analysis (RIA) of the EPA’s December 2023 Final Rulemaking, “Standards of Performance for New, Reconstructed, and Modified Sources and Emissions Guidelines for Existing Sources: Oil and Natural Gas Sector Climate Review”. The EPA solicited public comment on the methodology and use of these estimates in the RIA for the agency’s December 2022 supplemental proposed Oil and Gas rulemaking, and has conducted an external peer review of these estimates, as described further below.

The SC-CO_2 is the monetary value of the net harm to society from emitting a metric ton of CO_2 into the atmosphere in a given year, or the benefit of avoiding that increase. In principle, SC-CO_2 is a comprehensive metric that includes the value of all future climate change impacts (both negative and positive), including changes in net agricultural productivity, human health effects, property damage from increased flood risk, changes in the frequency and severity of natural disasters, disruption of energy systems, risk of conflict, environmental migration, and the value of ecosystem services. The SC-CO_2, therefore, reflects the societal value of reducing CO_2 emissions by one metric ton and is the theoretically appropriate value to use in conducting benefit-cost analyses of policies that affect CO_2 emissions. In practice, data and modeling limitations restrain the ability of SC-CO_2 estimates to include all physical, ecological, and economic impacts of climate change, implicitly assigning a value of zero to the omitted climate damages. The estimates are, therefore, a partial accounting of climate change impacts and likely underestimate the marginal benefits of abatement (and marginal damages from emissions).

Since 2008, the EPA has used estimates of the social cost of various greenhouse gases (i.e., social cost of carbon (SC-CO_2), social cost of methane (SC-CH_4), and social cost of nitrous oxide (SC-N_2O)), collectively referred to as the "social cost of greenhouse gases" (SC-GHG), in analyses of actions that affect GHG emissions. The values used by the EPA from 2009 to 2016, and since 2021 have been consistent with those developed and recommended by the Interagency Working Group (IWG) on the SC-GHG; and the values used from 2017 to 2020 were consistent with those required by E.O. 13783, which disbanded the IWG. During 2015–2017, the National Academies conducted a comprehensive review of the SC-CO_2 and issued a final report in 2017 recommending specific criteria for future updates to the SC-CO_2 estimates, a modeling framework to satisfy the specified criteria, and both near-term updates and longer-term research needs pertaining to various components of the estimation process (National Academies, 2017). The IWG was reconstituted in 2021 and E.O. 13990 directed it to develop a comprehensive update of its SC-GHG estimates, recommendations regarding areas of decision-making to which SC-GHG should be applied, and a standardized review and updating process to ensure that the recommended estimates continue to be based on the best available economics and science going forward.

The EPA is a member of the IWG and is participating in the IWG's work under E.O. 13990. While that process continues, as noted in previous EPA RIAs, the EPA is continuously reviewing developments in the scientific literature on the SC-GHG, including more robust methodologies for estimating damages from emissions, and looking for opportunities to further improve SC-GHG estimation going forward.[105] In the December 2022 RIA for the Standards of Performance for New, Reconstructed, and Modified Sources and Emissions Guidelines for Existing Sources: Oil and Natural Gas Sector Climate Review, the agency included a sensitivity analysis of the climate benefits of the Supplemental Proposal using a new set of SC-GHG estimates that incorporates recent research addressing recommendations of the National Academies (2017) in addition to using the interim SC-GHG estimates presented in the Technical Support Document: Social Cost of Carbon, Methane, and Nitrous Oxide Interim Estimates under Executive Order 13990 (IWG, 2021) that the IWG recommended for use until updated estimates that address the National Academies' recommendations are available.

The EPA solicited public comment on the sensitivity analysis and the accompanying draft technical report, *EPA Report on the Social Cost of Greenhouse Gases: Estimates Incorporating Recent Scientific Advances*, which explains the methodology underlying the new set of estimates, in the December 2022 Supplemental Proposal.[106] Please see the response to comments document

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[105] EPA strives to base its analyses on the best available science and economics, consistent with its responsibilities, for example, under the Information Quality Act.

[106] See https://www.epa.gov/environmental-economics/scghg for a copy of the final report and other related materials.

for that rulemaking for summaries and responses to public comments. The response to comments document can be found in the docket for the Standards of Performance for New, Reconstructed, and Modified Sources and Emissions Guidelines for Existing Sources: Oil and Natural Gas Sector Climate Review.

To ensure that the methodological updates adopted in the technical report are consistent with economic theory and reflect the latest science, the EPA also initiated an external peer review panel to conduct a high-quality review of the technical report, completed in May 2023. The peer reviewers commended the agency on its development of the draft update, calling it a much-needed improvement in estimating the SC-GHG and a significant step towards addressing the National Academies' recommendations with defensible modeling choices based on current science. The peer reviewers provided numerous recommendations for refining the presentation and for future modeling improvements, especially with respect to climate change impacts and associated damages that are not currently included in the analysis. Additional discussion of omitted impacts and other updates have been incorporated in the technical report to address peer reviewer recommendations. Complete information about the external peer review, including the peer reviewer selection process, the final report with individual recommendations from peer reviewers, and the EPA's response to each recommendation is available on the EPA's website.[107]

For an overview of the methodological updates incorporated into the SC-GHG estimates applied in the EA for the final PFAS NPDWR, see Section 3.2 of the RIA for the Standards of Performance for New, Reconstructed, and Modified Sources and Emissions Guidelines for Existing Sources: Oil and Natural Gas Sector Climate Review (U.S. EPA, 2023g). A more detailed explanation of each input and the modeling process is provided in the technical report, Supplementary Material for the RIA: EPA Report on the Social Cost of Greenhouse Gases: Estimates Incorporating Recent Scientific Advances (U.S. EPA, 2023h), included in the docket for the Standards of Performance for New, Reconstructed, and Modified Sources and Emissions Guidelines for Existing Sources: Oil and Natural Gas Sector Climate Review, and included in the docket for this action.

Table 9-1 summarizes the resulting averaged certainty-equivalent SC-CO_2 estimates under each near-term discount rate that are used to estimate the climate disbenefits of the changes in CO_2 emissions expected to result from the final PFAS rule. These estimates are reported in 2020 dollars and are identical to those presented in U.S. EPA (2023h). The SC-CO_2 increases over time within the models — i.e., the societal harm from one metric ton emitted in 2030 is higher than the harm caused by one metric ton emitted in 2025 — because future emissions produce larger incremental damages as physical and economic systems become more stressed in response to greater climatic change, and because GDP is growing over time and many damage categories are modeled as proportional to GDP. The full results generated from the updated methodology for carbon dioxide and other greenhouse gases (SC-CO2, SC-CH4, and SC-N2O) for emissions years 2020 through 2080 are provided in U.S. EPA (2023h).

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[107] https://www.epa.gov/environmental-economics/scghg-tsd-peer-review

Table 9-1: Estimates of the Social Cost of CO_2, 2020-2080 (2020$ per metric ton CO_2)

Year	Near-Term Ramsey Discount Rate		
	2.50%	2.00%	1.50%
2020	120	190	340
2021	120	200	340
2022	120	200	350
2023	130	200	350
2024	130	210	360
2025	130	210	370
2026	130	220	370
2027	140	220	370
2028	140	220	380
2029	140	230	380
2030	140	230	380
2031	150	230	390
2032	150	240	390
2033	150	240	400
2034	160	250	400
2035	160	250	410
2036	160	250	410
2037	160	260	420
2038	170	260	420
2039	170	260	430
2040	170	270	430
2041	180	270	440
2042	180	280	440
2043	180	280	450
2044	190	280	450
2045	190	290	460
2046	190	290	460
2047	200	300	470
2048	200	300	470
2049	200	300	480
2050	210	310	480
2051	210	310	490
2052	210	320	490
2053	210	320	500
2054	220	320	500
2055	220	330	510
2056	220	330	510
2057	230	330	510
2058	230	340	520
2059	230	340	520

Table 9-1: Estimates of the Social Cost of CO_2, 2020-2080 (2020$ per metric ton CO_2)

Year	Near-Term Ramsey Discount Rate		
	2.50%	2.00%	1.50%
2060	230	350	530
2061	240	350	530
2062	240	350	540
2063	240	350	540
2064	240	360	540
2065	250	360	550
2066	250	360	550
2067	250	370	550
2068	250	370	560
2069	260	370	560
2070	260	380	570
2071	260	380	570
2072	260	380	570
2073	270	390	580
2074	270	390	580
2075	270	390	580
2076	270	390	590
2077	280	400	590
2078	280	400	590
2079	280	400	600
2080	280	410	600

Note: This table displays the values rounded to two significant figures. The annual unrounded values used in the calculations in this EA are available in Appendix A.5 of U.S. EPA (2023g) and at: www.epa.gov/environmental-economics/scghg

The methodological updates described in U.S. EPA (2023h) represent a major step forward in bringing SC-GHG estimation closer to the frontier of climate science and economics and address many of the National Academies' (2017) near-term recommendations. Nevertheless, the resulting SC-GHG estimates, including the SC-CO_2 estimates presented in Table 9-1, still have several limitations, as would be expected for any modeling exercise that covers such a broad scope of scientific and economic issues across a complex global landscape. There are still many categories of climate impacts and associated damages that are only partially or not reflected yet in these estimates and sources of uncertainty that have not been fully characterized due to data and modeling limitations. Please see Section 3.2 of U.S. EPA (2023h) for further discussion.

All of the EPA's peer reviewed WBS models include the consumption of purchased electricity. The EPA has used the WBS models to estimate the electricity consumed annually by operating each technology at the entry-point level. For more information on WBS estimation of energy usage, see the EPA's Work Breakdown Structure-Based Cost Models documents for GAC, IX, and RO, specifically Appendix E General Assumptions for Operating and Maintenance Costs.

Table 9-2 below provides a summary of the electricity consumption at the entry-point level by system size and treatment technology.

Table 9-2: Entry Point Level Electricity Consumption Range by System Size and Technology (MWh/year)

Treatment Technology	Minimum Electricity Use (MWh/year)	Maximum Electricity Use (MWh/year)
GAC		
<100 to 3,300	0	1
3,301 to 10,000	6	7
10,000 to 100,000	8	233
100,000 and above	33	653
IX		
<100 to 3,300	0	0
3,301 to 10,000	2	2
10,000 to 100,000	2	4
100,000 and above	7	14

The EPA uses the WBS estimates of MWh by system size and source and the estimates of the number of water systems anticipated to select each technology based on the decision tree (presented in Chapter 5 of this document) to estimate the total electricity used nationally to operate treatment technologies to comply with the rule. Table 9-3 below shows the annual national electricity use anticipated by system size and technology used. The EPA estimates the total national annual electricity use to be 229,179 MWh per year.

Table 9-3: National Electricity Use (MWh/year) by Technology and System Size.

Treatment Technology	Total Electricity Use (MWh/year)
GAC	
<100 to 3,300	839
3,301 to 10,000	3,216
10,000 to 100,000	55,341
100,000 and above	163,214
IX	
<100 to 3,300	38
3,301 to 10,000	398
10,000 to 100,000	2,806
100,000 and above	3,326
Total	229,179

To convert this estimated increase in electricity use nationally into national CO_2 emissions through 2080, the EPA used the latest reference case from the EPA's peer-reviewed Integrated Planning Model (IPM). The IPM is a multi-regional, dynamic, deterministic linear programming model of the U.S. electric power sector. It provides projections of least-cost capacity expansion, electricity dispatch, and emission control strategies for meeting energy demand and environmental, transmission, dispatch, and reliability constraints (U.S. EPA, 2023e). The EPA uses the IPM to analyze the projected impact of environmental policies on the electric power sector, and it also provides projections of CO_2 emissions from the power sector through 2055. The latest reference case, "Post-IRA 2022 reference case" was published in April of 2023 and reflects the impacts of the Inflation Reduction Act (IRA).

Although the U.S. electricity grid continues to decrease its reliance on coal combustion in favor of natural gas and renewable alternatives, electricity consumption continues to be associated with GHG emissions across the entire system of production and delivery. Combustion of fossil fuels releases CO_2, CH_4, and N_2O; sulfur hexafluoride (SF_6) and perfluorocarbons (PFCs) are used in electricity transmission and distribution equipment; and additional GHG emissions are associated with the manufacture and installation of equipment as well the extraction and delivery of fossil fuels (U.S. EPA, 2023c). An exact accounting of all these emissions categories would yield the most precise estimate of electricity sector climate-related impacts. However, CO_2 emissions from fossil fuel combustion comprise the vast majority of the electricity sector GHG emissions. Therefore, accounting for combustion emissions of CO_2 is sufficient for the purposes of estimating the approximate magnitude of the climate-related disbenefits of increased electricity consumption. For example, in 2021, the EPA estimates total electricity sector emissions of 1,584 million metric ton (MMT) of CO_2-equivalent GHGs from fossil fuel combustion, waste incineration, process emissions, and electricity transmission and distribution. Over 97 percent of this total consists of CO_2 emissions from fossil fuel combustion.[108] Even accounting for upstream coal mining and natural gas systems, the share of electricity sector GHG emissions that are from fossil fuel combustion release of CO_2 is still at least 90 percent.[109,110] Note that the non-GHG emissions impacts associated with changes in electricity consumption are not accounted for in this analysis. For a more complete description of non-GHG impacts from the electricity sector, including ozone- and PM2.5-attributable premature mortality and illness as well as discussion of various unquantified health and welfare impacts, see recent the EPA regulatory impact analyses for air pollution regulations and the utilities sector in particular (U.S. EPA, 2023f).

From IPM reference case summary outputs, the EPA calculated projections of annual national-average CO_2 emissions per MWh of electricity generation over the model time horizon. The EPA mapped non-model years to calendar years following the IPM documentation guidance (U.S. EPA, 2023a).[111] After calendar year 2059, through the end of the period of analysis (2080), the EPA assumes that national-average electricity emission factors remain constant, which may lead to overstating the disbenefits of this rule. Table 9-4 below shows the IPM summary outputs and implied national-average CO_2 emissions factors for each IPM model year.

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[108] Ibid. See Table 2-11. (1,540.9 MMT CO_2 from fossil fuel combustion)/(1,584.1 MMT CO_2 eq. total) = 97.3 percent in 2021.

[109] 92 percent = share of coal consumed by electricity sector in 2022. See U.S. Energy Information Administration, Monthly Energy Review, Table 6.2.
38 percent = share of natural gas consumed by electricity sector in 2022. See U.S. Energy Information Administration, Monthly Energy Review, Table 4.3.
90.1 percent = (1,540.9 MMT CO_2 from fossil fuel combustion in EPA GHGI Table 2-11)/[(1,584.1 MMT CO_2 eq. total in EPA GHGI Table 2-11) + (217.5 MMT CO_2 eq. from natural gas systems in EPA GHGI Table 3-65)*(38 percent) + (44.7 MMT CH_4 in CO_2 eq. from coal mining in EPA GHGI Table 3-34)*(92 percent) + (2.5 MMT CO_2 in CO_2 eq. from coal mining in EPA GHGI Table 3-36)*(92 percent)].

[110] Coal and especially gas are inputs to other sectors of the economy, so decreasing electricity sector demand for these fuels does not necessarily preclude their extraction and use elsewhere.

[111] The EPA mapped the calendar year 2028 to model run year 2028, calendar years 2029-31 to run year 2030, calendar years 2032-37 to run year 2035, calendar years 2038-42 to run year 2040, calendar years 2043-47 to run year 2045, calendar years 2048-52 to run year 2050, and calendar years 2053-80 to run year 2055.

Table 9-4: CO_2 Emissions per MWh Calculated from Post-IRA 2022 IPM Reference Case

IPM Model Year	CO_2 Emissions (Million Metric Tons/year)[a]	Grand Total Electricity Generated (Billions MWh/year)[a]	CO_2 Emissions (mt/MWh/year)
2028	1,222	4.409	0.28
2030	972	4.545	0.21
2035	608	4.891	0.12
2040	481	5.265	0.09
2045	406	5.628	0.07
2050	357	6.071	0.06
2055	391	6.454	0.06

[a]Source: Post IRA Reference Case SSR.xlsx available at: https://www.epa.gov/power-sector-modeling/post-ira-2022-reference-case.

The EPA estimates the CO_2 emissions per model year associated with PFAS compliance emissions by multiplying the total annual electricity use associated with the rule per year by the annual national-average CO_2 emissions per MWh from Table 9-4 above. This methodology using national-average emission factors assumes that the geographic locations of these technologies and timing of their operations is similar to average U.S. electricity demand. The EPA believes that these assumptions are a reasonable approximation in this analysis where the treatment technologies are geographically widespread with fairly continuous operations. At this time, the EPA does not have sufficient information about the exact locations of units and timing of operations for a more refined methodology but expects this would have a minimal impact on the quantified results.

Table 9-5: CO_2 emissions per Year from Operating Treatment Technologies to Comply with the PFAS NPDWR

IPM Model Year	Period of Analysis Year[a]	CO_2 Emission (mt/year)
2028	2028	0
2030	2029-2031	49,004
2035	2032-2037	28,505
2040	2038-2042	20,954
2045	2043-2047	16,520
2050	2048-2052	13,466
2055	2053-2080	13,897

Note:
[a] The EPA's analysis assumes the rule is promulgated in 2024 and per the final rule requirements, systems must be in compliance with the final NPDWR by 2029. Therefore, the EPA models emissions associated with electricity use to operate treatment technologies beginning in 2029. Please see Chapter 1 for additional discussion on compliance timelines.

Table 9-6 presents the monetized climate disbenefits associated with operation of PFAS removal treatment technologies under the final PFAS NPDWR. The EPA multiplied the projected CO_2 emissions each year (shown in Table 9-5) by the SC-CO_2 estimate for that year (from Table 9-1) and annualized these results over the 2024-2080 analysis period. Monetized climate effects are presented under a 1.5 percent, 2 percent, and 2.5 percent near-term Ramsey discount rate, consistent with the EPA's updated estimates of the SC-CO_2. As described in U.S. EPA (2023h), the SC-CO_2 estimates rely on a dynamic discounting approach that provides over the constant discount rate framework used for SC-GHG estimation in EPA RIAs to date. Specifically, it provides internal consistency within the modeling and a more complete accounting of uncertainty consistent with economic theory and the National Academies' (2017) recommendation to employ a more structural, Ramsey-like approach to discounting that explicitly recognizes the relationship between economic growth and discounting uncertainty. This approach is also consistent with the National Academies' (2017) recommendation to use three sets of Ramsey parameters that reflect a range of near-term certainty-equivalent discount rates and are consistent with theory and empirical evidence on consumption rate uncertainty. See U.S. EPA (2023h) for a more detailed discussion of the entire discounting module and methodology used to value risk aversion in the SC-GHG estimates. The results presented in Table 9-6 are not directly comparable to the economic analyses prepared in the HRRCA analysis presented in Chapters 1-7 of this EA because climate disbenefits were assessed over a shorter period of analysis[112] and at different discount rates[113]. The EPA estimates a range of climate disbenefits associated with this rule from $8.8 million dollars per year (at a 1.5 percent discount

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[112] The final rule analysis evaluates costs and benefits under the final rule for the period of analysis from 2024 through 2105. For more information see Chapter 2.2.1.

[113] The final rule analysis estimates the annualized value of future benefits and costs using a 2 percent discount. For more information see Chapter 2.2.2.

rate) to $3.6 million dollars per year (at a 2.5 percent discount rate), which constitute less than 0.6% of the quantified benefits at a 2 percent discount rate.

Table 9-6: Annualized Monetized Climate Disbenefits Associated with Operating Treatment Technologies to Comply with the Final PFAS NPDWR ($2022)

Ramsey Near Term Discount Rate	Annualized Value ($2022)[a]
2.5 percent	$3,600,000
2 percent	$5,516,000
1.5 percent	$8,771,000

Note:
[a] Results were annualized over the 2024-2080 period of analysis.

9.3 Paperwork Reduction Act

The information collection requirements for the final rule will be submitted for approval to OMB under the Paperwork Reduction Act (PRA), 44 U.S.C. 3501 et seq. The ICR supporting statement prepared by the EPA has been assigned the EPA ICR number 2732.01 and is available in the docket at https://www.regulations.gov/docket/EPA-HQ-OW-2022-0114.

The PRA requires the EPA to estimate the burden, as defined in 5 CFR 1320.3(b), on PWSs and primacy agencies of complying with the rule. The information collected as a result of the final rule should allow primacy agencies and the EPA to determine appropriate requirements for specific systems and evaluate compliance with the final rule. Burden is defined at 5 CFR 1320.3(b) and means the total time, effort, and financial resources required to generate, maintain, retain, disclose, or provide information to or for a federal agency. The burden includes the time needed to conduct primacy agency and system activities during the first three years after promulgation, as described below.

9.3.1 Primacy Agency Activities

The EPA anticipates primacy agencies will be involved in the following activities for the first three years after publication of the final rule:

- Startup activities – read and understand the rule, adopt regulatory change, and provide internal and system staff with training and technical assistance;
- Review the initial monitoring event results, including confirmation sample results for MCL exceedances; and
- Review the results of standard monitoring from systems.

9.3.2 Public Water System Activities

The EPA anticipates systems will be involved in the following activities for the first three years after publication of the final rule:

- Startup activities – read and understand the rule and attend initial training from the primacy agency;
- Conduct initial monitoring including confirmation sampling for MCL exceedances; and
- Conduct standard monitoring, as needed; the EPA assumed that sampling for annual and triennial monitoring would not occur until after the three-year ICR period.

For the first three years after publication of the rule in the Federal Register, information requirements apply to an average of 33,594 respondents annually, including 33,538 PWSs and 56 primacy agencies. The burden associated with the final rule over the three years covered by the ICR is 2.1 million hours, for an average of 684,119 hours per year. The total cost over the three-year period is $176.8 million, for an average of $58.9 million per year (simple average over three years). The average burden per response (i.e., the amount of time needed for each activity that requires a collection of information) is 2.6 hours for PWSs and 2.6 hours for primacy agencies; the average cost per response is $247 for PWSs and $154 for primacy agencies. The collection requirements are mandatory under SDWA (42 U.S.C. 300g-7). Details on the calculation of the final rule information collection burden and costs can be found in the ICR for the final rule and Chapter 5 of this EA. A summary of the average annual burden and costs of the collection is presented in Table 9-7. The burdens and costs reflect labor and laboratory analysis costs.

Table 9-7: Average Annual Burden, Costs, and Responses for the Final Rule Information Collection Request

Item	Burden (Hours in Thousands)[a]	Costs (Million $2022)[a]	Responses
Systems	506	$48.3	195,739
Primacy agencies	178	$10.6	69,056
Total[b]	684	$59.0	264,795
Average per response – systems (hours or dollars)	2.6	$247.0	Not applicable
Average per response – primacy agencies (hours or dollars)	2.6	$154.0	Not applicable

Notes:
[a] Different units indicated for the estimates of burden and cost average per response.
[b] Detail may not add to totals because of independent rounding.
Source: ICR Supporting Statement, available in the docket at https://www.regulations.gov/docket/EPA-HQ-OW-2022-0114.

The estimates of total responses, burden, and cost for system and primacy agency startup activities are provided in Table 9-8.

Table 9-8: Total Burden, Costs, and Responses for Each Required Activity

Item	Burden (Thousand Hours)	Costs (Million $2022)	Responses
System startup activities	1,312	$48.5	133,060
Systems collect initial samples	207	$96.5	454,158
System subtotal	**1,519**	**$145.0**	**587,218**
Primacy agency startup activities	326	$19.5	112
Primacy agency review initial monitoring data	207	$12.4	207,056
Primacy agency subtotal[a]	**533**	**$31.8**	**207,168**
Combined systems and primacy agency[a]	**2,052**	**$176.8**	**794,386**

Note:
[a] Detail may not add to totals because of independent rounding.
Source: ICR Supporting Statement, available in the docket at https://www.regulations.gov/docket/EPA-HQ-OW-2022-0114.

An agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number. The OMB control numbers for the EPA's regulations in 40 CFR are listed in 40 CFR part 9. The control number for this action is ICR OMB Control No. 2040-0307.

The information collection activities in this final rule have been submitted for approval to the OMB under the PRA. The ICR document that the EPA prepared has been assigned the EPA ICR number 2732.01. You can find a copy of the ICR in the docket for this rule at https://www.regulations.gov/docket/EPA-HQ-OW-2022-0114. When OMB approves this ICR, the agency will announce that approval in the Federal Register and publish a technical amendment to 40 CFR part 9 to display the OMB control number for the approved information collection activities contained in this final rule.

9.4 The Final Regulatory Flexibility Analysis

The RFA of 1980, amended by the SBREFA of 1996, requires regulators to assess the effects of regulations on small entities including businesses, nonprofit organizations, and governments. RFA/SBREFA generally requires an agency to prepare an initial regulatory flexibility analysis (IRFA) of any rule subject to notice and comment rulemaking requirements under the Administrative Procedure Act or any other statute unless the agency certifies that the rule will not have a significant economic impact on a substantial number of small entities (SISNOSE). Small entities include small businesses, small organizations, and small governmental jurisdictions. Under the RFA, the Final Regulatory Flexibility Analysis (FRFA) must include:

1. A statement of the need for, and objectives of, the rule;
2. A statement of the significant issues raised by the public comments in response to the initial regulatory flexibility analysis, a statement of the assessment of the agency of such issues, and a statement of any changes made in the proposed rule as a result of such comments;
3. The response of the agency to any comments filed by the Chief Counsel for Advocacy of the Small Business Administration in response to the proposed rule, and a detailed

statement of any change made to the proposed rule in the final rule as a result of the comments;

4. A description of and an estimate of the number of small entities to which the rule will apply or an explanation of why no such estimate is available;
5. A description of the projected reporting, recordkeeping and other compliance requirements of the rule, including an estimate of the classes of small entities which will be subject to the requirement and the type of professional skills necessary for preparation of the report or record;
6. A description of the steps the agency has taken to minimize the significant economic impact on small entities consistent with the stated objectives of applicable statutes, including a statement of the factual, policy, and legal reasons for selecting the alternative adopted in the final rule and why each one of the other significant alternatives to the rule considered by the agency which affect the impact on small entities was rejected.

The RFA provides default definitions for each type of small entity. Small entities are defined as: (1) a small business as defined by the Small Business Administration's (SBA) regulations at 13 CFR 121.201; (2) a small governmental jurisdiction that is a government of a city, county, town, school district, or special district with a population of less than 50,000; and (3) a small organization that is any "not-for-profit enterprise which is independently owned and operated and is not dominant in its field." The RFA also authorizes an agency to use alternative definitions for each category of small entity, "which are appropriate to the activities of the agency" after proposing the alternative definition(s) in the Federal Register and taking comment (5 USC 601(3)-(5)). In addition, to establish an alternative small business definition, agencies must consult with SBA's Chief Counsel for Advocacy.

For purposes of assessing the impacts of the final rule on small entities, the EPA considered small entities to be systems serving 10,000 people or fewer. This is the threshold specified by Congress in the SDWA 1996 Amendments for small system flexibility provisions. As required by the RFA, the EPA proposed using this alternative definition in the Federal Register (FR) (63 FR 7620, February 13, 1998), requested public comment, consulted with the SBA, and finalized the alternative definition in the agency's Consumer Confidence Reports regulation (U.S. EPA, 1998c, 63 FR 44524, August 19, 1998). As stated in that final rule, the alternative definition would be applied to all future drinking water regulations.

The EPA notes that the Infrastructure Investment and Jobs Act (also known as the BIL, P.L. 117-58) invests over $11.7 billion in the DWSRF General Supplemental fund; $4 billion in the DWSRF Emerging Contaminants fund; and $5 billion in the EC-SDC grants program. Together, these funds will reduce people's exposure to PFAS and other emerging contaminants through their drinking water. The BIL funding will prioritize investment in local communities that are on the frontlines of PFAS contamination and that have few options to finance solutions through traditional programs and help them meet their obligations under this regulation.

9.4.1 Need for, Objectives, and Legal Basis of the Rule

The need for the rule, the objectives of the rulemaking, the stakeholder outreach conducted, and the statutory authority the EPA is utilizing to finalize the rule are described in detail in Chapter 3.

See Section 3.1 for detailed information on the need for the rule, Chapter 9 for information on stakeholder outreach during the rulemaking process, and Section 3.2 for additional detail on the statutory authority for the promulgation of the PFAS regulation. In summary, SDWA authorizes the EPA to establish NPDWRs for contaminants that may have an adverse public health effect, that are known to occur or that present a substantial likelihood of occurring in PWSs at a frequency and level of public health concern, and that present a meaningful opportunity for health risk reduction for persons served by PWSs. As a result, the EPA is finalizing an NPDWR for six PFAS including PFOA, PFOS, PFNA, PFHxS, HFPO-DA, and PFBS. Additionally, under the SDWA, the EPA Administrator is authorized to establish monitoring, recordkeeping, and reporting regulations that the Administrator can use to establish regulations under the SDWA, determine compliance with SDWA, and advise the public of the risks of unregulated contaminants.

The EPA is also addressing PFAS through several of its statutory authorities other than SDWA, including the CERCLA, RCRA, Toxic Substances Control Act (TSCA), Clean Water Act, Clean Air Act, and Emergency Planning and Community Right-to-Know Act. For example, as part of the EPA PFAS Strategic Roadmap, in 2022, the EPA has proposed to designate PFOA and PFOS as CERCLA hazardous substances to require reporting of PFOA and PFOS releases, enhance the availability of data, and ensure agencies can recover cleanup costs. The EPA recognizes that future actions under some of these statutes may have direct or indirect impacts for drinking water treatment facilities and could impact the compliance requirements related to disposal of PFAS treatment residuals that are generated by water systems. The EPA has also committed to restrict PFAS discharges from industrial sources through a multi-faceted Effluent Limitations Guidelines program to proactively establish national technology-based regulatory limits. Additionally, the EPA is seeking to proactively use National Pollutant Discharge Elimination System (NPDES) authorities to reduce discharges of PFAS at the source and obtain more comprehensive information through monitoring on the sources of PFAS discharges and quantity of PFAS discharged by these sources. The EPA notes that these actions may prevent or reduce PFAS entering into sources of drinking water in the future. More information on these statutory authorities and PFAS-related EPA activities can be found in the PFAS Strategic Roadmap.

9.4.2 Summary of the SBAR Comments and Recommendations

A Small Business Advocacy Review Panel (SBAR Panel or Panel) was convened to review the planned proposed rulemaking on the Proposed PFAS NPDWR. In addition to the EPA's Small Business Advocacy Chairperson, the Panel consists of the Director of the Standards and Risk Management Division of the EPA OGWDW, the Administrator of the Office of Information and Regulatory Affairs within the OMB, and the Chief Counsel for Advocacy of the Small Business Administration. The panel consulted with and reported on the comments of small entity representatives (SERs) and made findings on issues related to elements of an IRFA under Section 603 of the RFA. The SERs were presented with information related to PFAS background (such as health and occurrence, the SDWA regulatory development process and the EPA's actions to address PFAS in drinking water potential monitoring and reporting rule compliance considerations, treatment and feasibility considerations, potential public notification and education rule compliance considerations, and preliminary economic impacts to small systems. The EPA also provided to SERs that the agency's final regulatory determination for PFOA and PFOS outlined avenues that the agency considered to further evaluate additional PFAS chemicals, other than PFOA and PFOS, and consider groups of PFAS as supported by use of the best available science. Additionally, as part of the EPA's PFAS Strategic Roadmap, the EPA reaffirmed its commitment to evaluate additional PFAS and consider regulatory actions to address additional PFAS or groups of PFAS as it develops the NDPWR. Further, the EPA provided to SERs that as the EPA considers whether to include additional PFAS as part of this regulation, the agency would consider several factors, including whether the same treatment approaches co-remove certain PFAS contaminants and how different PFAS are anticipated to be removed as part of the treatment process, the likelihood that the PFAS co-occur, the similarity of health effects and chemical structures, the environmental persistence characteristics, and the availability of accepted and approved analytical methods or indicators with comparable costs to those currently identified by the EPA to evaluate PFAS removal from drinking water, among other considerations.

In light of the SERs' comments, the Panel considered the regulatory flexibility issues and elements of the IRFA specified by RFA/SBREFA and developed the findings and discussion summarized in the SBAR report. For example, the SBAR Panel recommended several flexibilities in monitoring requirements for small systems, including the use of existing monitoring data (such as the UCMR 5) for initial monitoring purposes, as well as reduced initial monitoring requirements specifically for small ground water systems. Regarding public comment requests, the Panel recommended that the EPA request this for a few areas, such as laboratory capacity for monitoring, additional treatment technologies other than those identified in the proposed rule that have been shown to reduce levels of PFAS to the proposed regulatory standards, additional monitoring flexibilities, and PFAS disposal considerations. Moreover, specific to PFAS disposal, the Panel recommended that the EPA continue to evaluate the potential impacts related to the disposal of PFAS treatment residuals and potential implications from other EPA statutory authorities. This recommendation included presenting the costs of both non-hazardous and hazardous waste disposal of treatment residuals as a part of the proposed rule. To address stakeholder concerns, including those raised during the SBREFA process, the EPA conducted a sensitivity analysis with an assumption of hazardous waste disposal for illustrative purposes only. As part of this analysis, the EPA generated a second full set of unit cost curves that are identical to the curves used for the national cost analysis with the exception that spent GAC and spent IX resin are considered hazardous. The EPA acknowledges that if federal authorities later determine that PFAS-contaminated wastes require handling as hazardous wastes, the residuals management costs are expected to be higher. The EPA incorporated all Panel recommendations, as well as others, in the proposed and final rule.

The Panel also recommended the EPA to consider rule implementation delays for potential laboratory capacity-related challenges if those challenges potentially impact the ability of water systems to monitor for PFAS and reasonably comply with the NPDWR. As described in the proposed rule preamble (Section XII.D.), in accordance with SDWA 1412(b)(10), a state or the EPA may grant an extension of up to two additional years to comply with an NPDWR's MCL if the state or the EPA determines a system needs additional time for capital improvements. In the rule proposal, the EPA indicated that the agency did not intend to provide a two-year extension nationwide. However, the EPA noted in the proposal that under SDWA 1412(b)(10) or 1416 States may provide such extension on an individual system basis which may address compliance issues associated with treatment, laboratory, and disposal capacity. Additionally, the EPA notes that in the proposed rule preamble (Section IX.F) the agency sought public comment on the proposed initial monitoring timeframe, particularly for NTNCWS or all systems serving 3,300 or fewer.

The report includes a number of other observations and recommendations to meet the statutory obligations for achieving small-system compliance through flexible regulatory compliance options. The report was finalized on August 1, 2022 and transmitted to the EPA Administrator for consideration. Detailed information on the overall panel process, including the comprehensive comments of the SERs and full description of Panel recommendations, can be found in the panel report titled, Final Report of the Small Business Advocacy Review Panel on the EPA's Planned Proposed Rule Per- and Polyfluoroalkyl Substances National Primary Drinking Water Regulation and can be found in the rulemaking docket at: https://www.regulations.gov/document/EPA-HQ-OW-2022-0114-0048.

9.4.3 Summary of the Final Rule and Public Comments on the Impacts to Small Entities

The EPA is regulating six PFAS in finished drinking water: (1) PFOS, (2) PFOA, (3) PFNA, (4) HFPO-DA and its ammonium salt (also known as GenX chemicals)), (5) PFHxS, and (6) PFBS. The final regulation utilizes compound-specific MCLs for PFOA, PFOS, PFNA, HFPO-DA and PFHxS and an MCL based on a HI for combinations of PFNA, HFPO-DA, PFHxS, and PFBS in mixtures. With this action, the EPA finalizes monitoring, reporting, public notification, and Consumer Confidence Report requirements for PWSs and primacy agencies to comply with the NPDWR.

In the proposal, the EPA evaluated three significant alternatives to minimize significant economic impacts on small PWSs that serve 10,000 or fewer people. The proposed and final rule would also allow water systems to select the most financially and technologically viable strategy that is effective in reducing PFAS in drinking water. The EPA evaluated the following significant alternatives for the proposed rule: 1) use of previously collected monitoring data, 2) a provision for small ground water systems to collect two, rather than four, quarterly samples over a one-year period for initial monitoring, or 3) installation and maintenance of POU treatment devices.

In response to the IRFA included as part of the proposal, the EPA received one comment specifically on the analytical approach used in the IRFA. The commenter states that "[d]etailed analysis on the impacts to NTNCWSs should be conducted to inform the cost/benefit analysis. For example, treating PFAS with GAC at the low levels proposed is much more costly than current treatment for currently regulated contaminants, and a 2008 study is not a reliable indicator of future costs. Lack of both actual data on occurrence in these systems and reliable information on cost of compliance makes finalizing the MCL as to NTNCWSs too uncertain." The EPA disagrees that the agency has not analyzed the impacts of the PFAS NPDWR on NTNCWS. The EPA has used both actual data on occurrence at NTNCWSs from UCMR3 and state data, as well as reliable information on costs to NTNCWSs using the WBS treatment cost models to assess the impact of the rule on NTNCWSs. As the EPA stated in the proposal, the EPA lacks information on the revenues of NTNCWS, therefore the agency does not take the same approach used for CWSs in the SISNOSE screening analysis where costs are compared to 1 and 3% of revenues. Instead, the EPA used the best available data, the EPA's Assessment of the Vulnerability of Noncommunity Water Systems to SDWA Cost Increases (SAIC, 1998) to find that NTNCWSs are less vulnerable to SDWA related increases than a typical CWS. The EPA proceeded with the SBAR Panel process, as detailed in this chapter.

Additionally, the EPA received many comments, including from the SBA Office of Advocacy, specific to various small system and IRFA-related topics including lack of funding availability for small water systems, the EPA's estimation of the impacts of the rule on small systems, the EPA's estimation and characterization on federal funding to defray compliance costs for small water systems, and "other factors that will further deter timely compliance" such as personnel shortages, supply chain disruptions, limited lab and disposal capacity, and availability of treatment technologies. For the EPA's response to SBA and other comments on funding availability, please see Section I of the preamble. For the EPA's response to SBA and other comments on the estimated costs to small water systems, please see Section XII of the preamble. For the EPA's response to SBA and other comments on lab capacity, see Section V and VIII of the preamble. For the EPA's response to SBA and other comments on technology and disposal capacity, see Section X of the preamble. For responses to SBA's and other commenters' recommendations to the EPA to provide burden-reducing flexibilities for small water systems, including finalizing one of the regulatory alternatives and phasing in the MCL, as well as providing additional time for compliance see Section V of the preamble. For response to SBA and other commenters concerned about the EPA's concurrent preliminary determination and proposed regulation for four PFAS, see Section III of the preamble.

9.4.4 Number and Description of Small Entities Affected

The EPA used SDWIS/Fed data from the fourth quarter of 2021 to identify 62,048 small PWSs, which represent 93% of all systems that may be impacted by the final PFAS regulation. A small PWS serves between 25 and 10,000 people. These water systems include 44,753 CWSs that serve year-round residents and 17,295 NTNCWSs that serve the same persons over six months per year (e.g., a PWS that is an office park or church). The final NPDWR will not affect TNCWSs as those systems will not be subject to the rule requirements. Additional information on the characteristics of these small drinking water systems along with a discussion of uncertainty in the dataset used to derive the estimated number of small systems impacted by the final PFAS regulation can be found in Section 4.3.1.

Table 9-9 and Table 9-10 show the number of affected small CWSS and NTNCWs respectively.

Table 9-9: Inventory of Small CWSs

System Size (Population Served)	CWSs[a]		
	Ground Water	Surface Water	Total
	A	B	C = A + B
≤ 100	10,654	739	11,393
101–500	13,037	2,042	15,079
501–1,000	4,132	1,179	5,311
1,001–3,300	5,503	2,460	7,963
3,301–10,000	2,784	2,223	5,007
TOTAL	36,110	6,601	44,753

Abbreviations: CWS – community water systems.
Note:
[a] Includes 23 CWSs serving 10,000 or fewer people for which no primary source water type was reported to SDWIS/Fed. The EPA assigned these systems to the source type of Ground Water.
Source: SDWIS/Fed fourth quarter 2021 "frozen" dataset that contains information reported through January 14, 2022. Includes all active CWSs.

Table 9-10: Inventory of Small NTNCWSs

System Size (Population Served)	NTNCWSs[a]		
	Ground Water	SW	Total
	A	B	C = A+B
≤ 100	8,084	252	8,336
101–500	6,111	257	6,368
501–1,000	1,476	91	1,567
1,001–3,300	743	121	864
3,301–10,000	97	63	160
TOTAL	16,551	784	17,295

Abbreviations: NTNCWS – non-transient non-community water systems.
Note:
[a] Includes 11 NTNCWSs serving 3,300 or fewer people for which no primary source type was reported to SDWIS/Fed. The EPA assigned these systems to the source water type of Ground Water.
Sources: SDWIS/Fed fourth quarter 2021 "frozen" dataset that contains information reported through January 14, 2022. Includes all active NTNCWSs.

9.4.5 Description of Compliance Requirements of the Final Rule

For a detailed description of the regulatory requirements under the final PFAS regulation see Section 2.1. The final rule requires PWSs subject to the rule to conduct initial monitoring. Related to this initial monitoring requirement, the final NPDWR includes a provision, made available to PWSs of all sizes, including CWSs and NTNCWs serving 10,000 or fewer people, to use qualified previously collected monitoring data to demonstrate levels of regulated PFAS in their water system to satisfy the initial monitoring requirement. The EPA assessed the extent to which this significant alternative minimizes the economic impact on small PWSs specifically in Section 9.4.7.1 below. Additionally, the EPA has included a provision in the final NPDWR where ground water systems serving a population of 10,000 or fewer may collect two quarterly samples over a one-year period for the purpose of initial monitoring, rather than collecting four quarterly samples. The EPA assessed the extent to which this regulatory flexibility minimizes the economic impact on small PWSs in Section 9.4.7.2 below.

Based on initial monitoring results, systems will be required to conduct ongoing monitoring at least every three years or as often as four times per year. Details on the monitoring frequency requirements of the final NPDWR can be found in Section VIII of the Federal Register Notice for the final rule.

PWSs that exceed the drinking water standard are required to choose between treatment and nontreatment compliance options. The EPA identified the following Small System Compliance Technologies (SSCTs) GAC, Anion Exchange (AIX), and High-pressure Membranes (RO and NF). POU RO is not currently listed as a compliance option because the final rule requires treatment to concentrations below the current National Sanitation Foundation (NSF) International/American National Standards Institute (NSF/ANSI) certification standard for POU device removal of PFAS. However, POU treatment is reasonably anticipated to become a compliance option for small systems in the future if NSF/ANSI or other independent third-party certification organizations develop a new certification standard that mirrors the EPA's proposed regulatory standard. Details on SSCTs and costs can be found in Section 5.3.1 and *Best Available Technologies and Small System Compliance Technologies for Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water* (U.S. EPA, 2024c).

9.4.6 Analysis of Impact of Regulatory Options on Small System Costs

The EPA limited the quantitative cost impact analysis to small CWSs because small NTNCWSs operate in numerous industries and the EPA does not have information on NTNCWSs' revenues. The EPA's decision to limit its cost impact analysis to CWSs is supported by the EPA's Assessment of the Vulnerability of Noncommunity Water Systems to SDWA Cost Increases (1998). In this study, the EPA examined the burden of SDWA rule costs in comparison to the average revenues of various categories of NTNCWSs. All the NTNCWS categories reviewed were less vulnerable to SDWA-related increases than a typical CWS. The report notes that in some categories of businesses, costs are more easily passed on to the customer base than in others. In each NTNCWS category, however, total expenditures on water were found to be a relatively small percentage of total revenues. Water expenditures (including expenditures for sewer service and miscellaneous other utilities) totaled less than one percent of total revenues in nearly all cases and were not more than 1.3 percent of total revenues for any category. The implication is that an increase in water costs would similarly be less than one percent of revenue. This report included several caveats such as one that considered the potential for underestimating the impact to golf courses, which were grouped in with other recreational entities whose use of water was less significant to the core business than the golf courses. The EPA notes, however, that irrigation water for golf courses would not need to meet the final rule; only water used for human consumption would need to be treated. Despite the significant caveats listed, the report strongly suggested that NTNCWSs should not be considered particularly vulnerable to operating cost increases resulting from SDWA rulemakings.

To indicate the potential economic impact on small CWSs, the EPA divided annual costs by annual revenues and converted the decimal values to percentages and identified the number and percent of CWSs for which the impact percentages exceeded thresholds of one percent and three percent. For each system, the EPA estimated annual revenue using each system's average daily flow and the average revenue per thousand gallons delivered from the CWSS (U.S. EPA, 2009). For annual costs, the EPA estimated annual average monitoring costs based on system size and baseline PFAS occurrence. Annual costs also included annual treatment costs when baseline PFAS concentrations exceeded the PFAS limits of the final rule or options. Annual treatment costs are the sum of annual operating and maintenance costs and annualized capital costs.

Table 9-11 shows the number and proportion of CWSs incurring annual costs that exceed 1 percent and 3 percent of annual revenue at the commercial rate of capital for the final rule. Under the final rule, 16,542 small CWSs (37 percent of small CWSs) could incur annual costs greater than 1 percent of annual revenue and 8,199 small CWSs (18 percent of small CWSs) could incur annual costs greater than 3 percent of annual revenue. These potential impacts are high enough to preclude a finding of no SISNOSE. Details on treatment costs curves can be found in Section 5.3.1 and *Best Available Technologies and Small System Compliance Technologies for Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water* (U.S. EPA, 2024c). For the EPA's estimates of treatment costs by system size, see Appendix C.1. For information on federal financial assistance available to small systems for the installation of PFAS treatment technology, see Section 9.13.2.2.

Table 9-11: Cost-Revenue Ratio for Small CWSs, Final Rule (PFOA and PFOS MCLs of 4.0 ppt each, PFHxS, PFNA, HFPO-DA MCLs of 10 ppt each and HI of 1) (Commercial Cost of Capital)

Ownership	Source Water	Population Served Size Category	Number of CWSs	Number of CWSs with Cost Revenue Ratio > 1%	Number of CWSs with Cost Revenue Ratio > 3%	Percent of CWS with Cost Revenue Ratio > 1%	Percent of CWS with Cost Revenue Ratio > 3%
Private	Ground	Less than 100	9,260	9,260	4,967	100%	54%
Private	Ground	100 to 500	8,225	2,892	890	35%	11%
Private	Ground	500 to 1,000	1,313	110	88	8%	7%
Private	Ground	1,000 to 3,300	1,048	80	77	8%	7%
Private	Ground	3,300 to 10,000	347	29	29	8%	8%
Private	Surface	Less than 100	399	398	196	100%	49%
Private	Surface	100 to 500	770	206	67	27%	9%
Private	Surface	500 to 1,000	244	19	15	8%	6%
Private	Surface	1,000 to 3,300	278	18	18	6%	6%
Private	Surface	3,300 to 10,000	184	15	14	8%	8%
Public	Ground	Less than 100	1,394	745	213	53%	15%
Public	Ground	100 to 500	4,812	1,153	395	24%	8%
Public	Ground	500 to 1,000	2,819	245	186	9%	7%
Public	Ground	1,000 to 3,300	4,455	341	326	8%	7%
Public	Ground	3,300 to 10,000	2,437	222	221	9%	9%
Public	Surface	Less than 100	340	184	51	54%	15%
Public	Surface	100 to 500	1,272	255	93	20%	7%
Public	Surface	500 to 1,000	935	72	58	8%	6%
Public	Surface	1,000 to 3,300	2,182	143	140	7%	6%
Public	Surface	3,300 to 10,000	2,039	155	155	8%	8%
Total			**44,753**	**16,542**	**8,199**	**37%**	**18%**

Abbreviations: CWS – community water system

Note:

The commercial cost of capital is the weighted average cost for PWSs to raise capital or borrow to pay for compliance activities. Please see Section 4.3.5 for additional details on how the cost of capital for different CWSs was calculated. The CWS compliance costs were annualized using the cost of capital and then compared to the average revenue of the CWS size and ownership category.

9.4.7 The EPA's Steps to Minimize the Significant Economic Impact of the Final Rule on Small Systems

Significant alternatives are described below. The EPA evaluated the minimized economic impact for small systems for each of these alternatives. In the final rule, the EPA elected to allow use of previously collected PFAS monitoring data to satisfy initial monitoring requirements and retain the provision to allow for reduced initial monitoring for small ground water systems serving a population of 10,000 or fewer. After considering public comments on the proposal, the EPA has included a provision in the final rule to allow for an annual compliance monitoring frequency, raised the trigger level which determine when more frequency monitoring is required, and is also exercising its authority under SDWA Section 1412(b)(10) to implement a nationwide two-year capital improvement extension to comply with MCL. Finally, the EPA notes that should POU devices become certified to meet the final NPDWR standard, this could minimize the economic impact of the final regulation on small PWSs, particularly on water systems in the smallest size category (e.g., those serving between 25 and 500 people).

9.4.7.1 Use of Previously Collected PFAS Monitoring Data

The EPA has included a provision in the final NPDWR where PWSs of all sizes may use previously collected monitoring data if it meets stated criteria to satisfy the initial monitoring requirement. This significant alternative is expected to offer substantial cost savings to small PWSs, particularly those serving a population between 3,301 and 10,000 that participate in UCMR 5. For the national cost analysis, the EPA assumes that systems with either UCMR 5 data or monitoring data in the State PFAS Database (U.S. EPA, 2024g) will not need to conduct the initial year of monitoring. As a simplifying assumption for the cost analysis, the EPA assumes all systems serving a population of greater than 3,300 have UCMR 5 data and those serving 3,300 or less do not. The EPA notes that this assumption is conservative and will likely overestimate costs for systems serving a population less than 3,300 as many state monitoring programs and other efforts will have collected monitoring data that can be used as initial monitoring data for these systems, thus offsetting those costs. Under these assumptions, the EPA estimates that this provision will reduce the economic burden on small systems nationally by $7 million dollars per year for three years.

9.4.7.2 Reduced Monitoring for Small Ground Water Systems

The EPA has included a provision in the final NPDWR where ground water systems serving a population of 10,000 or fewer may collect two quarterly samples over a one-year period for the purpose of initial monitoring, rather than collecting four quarterly samples. The EPA estimates that this provision will reduce the economic burden on small systems nationally by $21 million per year for three years.

9.4.7.3 Annual Monitoring for Systems "Reliably and Consistently" below the MCLs

Upon consideration of information submitted by commenters, the EPA has included a provision in the final rule to allow for annual compliance monitoring for all sized systems that are deemed to be "reliably and consistently"[114] below the MCLs, but still above the trigger levels. These systems would not be required to remain on quarterly monitoring, as proposed, and would instead be allowed to monitor annually once meeting the requirements of being deemed "reliably and consistently" below the MCLs. The introduction of annual monitoring has the potential to significantly reduce monitoring burden for water systems, including small systems, from taking 4 samples per year to taking 1 sample per year per EP. As most small systems have one EP, this

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[114] The definition of reliably and consistently below the MCL means that each of the quarterly samples contains regulated PFAS concentrations below the applicable MCLs. For the PFAS NPDWR, this demonstration of reliably and consistently below the MCL would include consideration of at least four quarterly samples at an EP below the MCL, but states will make their own determination as to whether the detected concentrations are reliably and consistently below the MCL.

requirement would save small water systems that are deemed "reliably and consistently" below the MCL a minimum approximately $930 per year.[115]

The EPA estimates that approximately 4,300 to 7,000 small PWSs may have regulated PFAS occurrence between the trigger levels and the MCLs, and therefore may be eligible for annual monitoring following four consecutive quarterly samples demonstrating they are "reliably and consistently" below the MCLs. Further, the EPA believes that most systems treating their water for regulated PFAS would likely be eligible for this compliance monitoring tier. Therefore, the EPA estimates that 2,900 to 5,400 small water systems in addition to the 4,300 to 7,000 small water systems above, may be eligible for annual monitoring, instead of quarterly monitoring, after taking action to comply with the rule.

9.4.7.4 Increased Trigger Levels

Upon consideration of information submitted by commenters, the EPA is finalizing higher trigger levels for the rule. In the proposal the EPA included trigger levels at 1/3 the MCLs: 1.3 ppt for PFOA and PFOS, 0.5 for the HI. For the final rule, the EPA has set trigger levels at 1/2 of the MCLs: 2.0 ppt for PFOA and PFOS, 5 ppt for PFHxS, GenX and PFNA, and 0.5 for the HI. As the trigger levels determine when more frequent monitoring is required, an increase in these levels will result in a burden reduction compared to the proposed rule for all water systems, including small water systems with compliance monitoring results between 1/2 and 1/3 of the MCLs.

9.4.7.5 MCL Compliance Period Extension

Upon consideration of information submitted by commenters, the EPA is exercising its authority under SDWA § 1412(b)(10) to implement a nationwide capital improvement extension to comply with MCL. All systems have 5 years to achieve compliance with the MCLs under the final rule. However, all systems must comply with the initial monitoring requirements by three years following rule promulgation, and all other requirements of the NPDWR, other than the MCL, starting three years following rule promulgation (e.g., compliance monitoring, reporting, and recordkeeping).

The agency notes that SDWA § 1416(a) and (b)(2)(C) describe how primacy agencies may also grant an exemption for systems meeting specified criteria that provides an additional period for compliance. PWSs that meet the minimum criteria outlined in the SDWA§ 1416 may be eligible for an exemption of up to three years. Exemptions for smaller water systems (≤3,300 population), meeting certain specified criteria may be renewed for one or more 2-year periods, but not to exceed six years. States exercising primacy enforcement responsibility must have adopted the 1998 Variance and Exemption Regulation for a water system to be eligible for an exemption in that State.

The EPA anticipates this will significantly reduce the burden of the final rule on all water systems, including small water systems. For more information see Section XI of the FRN.

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[115] The laboratory analysis cost per sample for EPA Method 537.1 is $309 ($2022). The cost of three avoided samples equals $927.

9.4.7.6 Point-of-Use (POU) Technologies as Small System Compliance Technologies (SSCTs)

In the *Best Available Technologies and Small System Compliance Technologies for Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water* (U.S. EPA, 2024c), the EPA discusses POUs technologies and notes that the current certification standard is 70 ppt, which would not ensure these devices are able to meet the MCLs of the final rule. The EPA notes that based on the technologies used in many POU devices (e.g., RO), the agency anticipates devices are or will be capable of meeting the MCLs in this final rulemaking. If POU certifications are updated and do meet the SSCT criteria in the final NPDWR, this could minimize the economic impact of the final regulation on small PWSs, particularly on water systems in the smallest size category (e.g., those serving between 25 and 500 people). In particular, NTNCWS that control all of their potable taps (e.g., schools, gas stations, churches) may find the use of POU devices to be a particularly attractive option. The EPA has not estimated the potential national economic burden reduction because the current certification prevents POU devices from meeting the SSCT criteria for the final NPDWR. However, the EPA notes there is a potential for significant burden reduction particularly for very small water systems if POU certifications are updated and POU devices meet the SSCT criteria for the final NPDWR in the future.

9.5 Unfunded Mandates Reform Act

The UMRA (1995) seeks to protect state, local, and tribal governments from the imposition of unfunded federal mandates. In addition, the Act seeks to strengthen the partnership among the federal government and state, local, and tribal governments.

Title II of UMRA establishes requirements for federal agencies to assess the effects of their regulatory actions on state, local, and tribal governments, and the private sector. Under Section 202 of UMRA, the EPA generally must prepare a written statement, including a cost-benefit analysis, for proposed and final rules with “federal mandates” that may result in expenditures by state, local, and tribal governments, in the aggregate, or by the private sector, of $100 million or more in any one year, adjusted for inflation. The EPA has calculated the cost of the rule in 2022 dollars, therefore, the UMRA requirements are triggered if expenditures exceed $168 million in one year (escalation based on GDP deflator).

Section 205 of UMRA generally requires the EPA to identify and consider a reasonable number of regulatory alternatives and adopt the least costly, most cost-effective, or least burdensome option that achieves the objectives of the rule. The provisions of Section 205 do not apply when they are inconsistent with applicable law. Note that in the case of NPDWRs, the UMRA Section 205 requirement to adopt the least costly, most cost-effective or least burdensome option is inconsistent with SDWA regulatory development requirements. SDWA section 1412(b)(4)(B) states that each national primary drinking water regulation for a contaminant for which a maximum contaminant level goal is established under this subsection shall specify a maximum contaminant level for such contaminant which is as close to the maximum contaminant level goal as is feasible, with feasible defined in section 1412(b)(4)(B)(5) as “feasible with the use of the best technology, treatment techniques and other means which the Administrator finds, after examination for efficacy under field conditions and not solely under laboratory conditions, are available (taking cost into consideration).” Moreover, Section 205 allows the EPA to adopt an alternative other than the least costly, most cost-effective, or least burdensome alternative if the Administrator publishes with the rule an explanation of why that alternative was not adopted. The EPA’s analysis of regulatory alternatives (Options 1a through 1c) found that they are less costly and lower burden options compared to the final rule. However, these options do not meet EPA’s statutory requirement to, as stated above, set the MCL for a contaminant(s) as close to the MCLG as is feasible, taking costs into consideration. EPA has determined that the final rule is feasible, taking costs into consideration; see discussion in Section V of the preamble. Finally, as detailed in Chapter 7, the Administrator has reaffirmed the SDWA required (Section 1412(b)(4)(C)) determination made at proposal that the quantifiable and nonquantifiable benefits of the rule justify the quantifiable and nonquantifiable costs, which provides further justification for why EPA did not select the least burdensome option.

Before the EPA establishes any regulatory requirements that may significantly or uniquely affect small governments, including tribal governments, it must have developed under Section 203 of UMRA a small government agency plan. The plan must provide for notifying potentially affected small governments, enabling officials of affected small governments to have meaningful and timely input in the development of the EPA regulatory proposals with significant federal intergovernmental mandates, and informing, educating, and advising small governments on compliance with the regulatory requirements. Section 204 of UMRA requires EPA, to the extent permitted by law, develop an effective process to permit elected officials of state, local, and tribal governments to provide meaningful and timely input in the development of regulatory proposals containing significant Federal intergovernmental mandates. Options being considered for the proposed rule also met the consultation requirements of Federalism, therefore the EPA elected to engage the UMRA (Sections 203 and 204) and Federalism stakeholders in the same consultation as there are overlapping interests, and a discussion of potential options for the development of the proposed rule was more effectively communicated simultaneously. For more information on the consultation, refer to the Summary Report on Federalism and Unfunded Mandates Reform Act Consultation for the Development of the Proposed PFAS NPDWR in the public docket at https://www.regulations.gov/document/EPA-HQ-OW-2022-0114-0706.

The final rule contains a federal mandate that may result in expenditures to state, local, and tribal governments, in the aggregate, or to the private sector, of $168 million or more in any one year. For the final rule, the highest annual incremental cost over the analysis period occurs in the 6th year after rule promulgation. In this year PWSs are expected to have undiscounted incremental costs of $15.5 billion and Primacy Agencies will have undiscounted incremental costs of $5 million. Therefore, the final rule has costs in a single year of $15.5 billion and, therefore, is subject to the requirements of Sections 202 and 205 of UMRA. As discussed in Section II.E of the preamble for the final rule, the EPA anticipates that significant federal funding available through BIL and other sources will assist many disadvantaged communities, small systems, and others with the costs of addressing emerging contaminants, like PFAS.

The annualized costs of the final rule, that are borne by public, private, and tribal PWSs are provided in Table 9-12. As the exhibit shows, public entities bear most of the costs (but may pass them on to consumers). As discussed in Chapter 2, in addition to these PWS costs, primacy agencies will incur annualized incremental administrative costs of $4.7 million under the final rule.

Table 9-12: Annual Costs by PWS Size and Ownership, Final Rule (Million $2022) (Commercial Cost of Capital)

	Public Water Systems Serving < 10,000 People	All Public Water Systems
Publicly-Owned Public Water Systems	$189.6	$1,284.6
Privately-Owned Public Water Systems	$161.3	$247.0
Tribal-Owned Public Water Systems	$4.4	$9.0

Abbreviations: PWS – public water system; PFAS – per- and polyfluoroalkyl substances; PFOS – perfluorooctanesulfonic acid; MCL – maximum contaminant level; HI – hazard index.

9.6 Executive Order 13132: Federalism

Executive Order 13132 (1999), entitled "Federalism" (64 FR 43255, August 10, 1999), requires the EPA to develop an accountable process to ensure "meaningful and timely input by state and local officials in the development of regulatory policies that have federalism implications." "Policies that have federalism implications" are defined in the Executive Order to include regulations that have "substantial direct effects on the states, on the relationship between the national government and the states, or on the distribution of power and responsibilities among the various levels of government."

To fulfill requirements of Executive Order 13132 Section 6, the EPA held a Federalism consultation with state and local government officials as well as their representative associations to solicit input on key areas to inform the development of the proposed rule. Options considered for the proposed rule also met the consultation requirements of UMRA, therefore the EPA elected to engage the UMRA stakeholders in the same consultation because there are overlapping interests, and a discussion of potential options for the development of the proposed rule was more effectively communicated simultaneously. For more information on the consultation, refer to the Summary Report on Federalism and Unfunded Mandates Reform Act Consultation for the Development of the Proposed PFAS NPDWR in the public docket at https://www.regulations.gov/document/EPA-HQ-OW-2022-0114-0706. The EPA also received public comments from some of these organizations during the public comment period following the rule proposal. These individual organization comments are available in the Docket. The EPA considered all comments provided by individual states and state organizations provided during the public comment period and used these comments to inform the final rule.

This action has federalism implications due to the substantial direct compliance costs on state or local governments. The net change in annualized primacy agency related cost for state, local, and tribal governments in the aggregate is estimated to be $4.7 million. Also see Table 9-12 for annual costs to publicly-owned water systems, which are estimated to be $1,284.6 million. Please see Section XIII.E of the preamble for the final rule for the EPA's federalism summary impact statement.

9.7 Executive Order 13175: Consultation and Coordination with Indian Tribal Governments

Executive Order 13175 (2000), entitled “Consultation and Coordination with Indian Tribal Governments” (65 FR 67249, November 9, 2000), requires the EPA to develop an accountable process to ensure “meaningful and timely input by tribal officials in the development of regulatory policies that have tribal implications.” The Executive Order defines “policies that have tribal implications to include regulations that have “substantial direct effects on one or more Indian tribes, on the relationship between the federal government and the Indian tribes, or on the distribution of power and responsibilities between the federal government and Indian tribes.”

Under Executive Order 13175, the EPA may not issue a regulation that has tribal implications, that imposes substantial direct compliance costs, and that is not required by statute, unless the federal government provides the funds necessary to pay the direct compliance costs incurred by tribal governments, or the EPA consults with tribal officials early in the process of developing the proposed regulation and develops a tribal summary impact statement.

The EPA has identified 998 public water systems serving tribal communities, 84 of which are federally owned. The EPA estimates that tribal governments will incur public water system compliance costs of $9.0 million per year attributable to monitoring, treatment or nontreatment actions to reduce PFAS in drinking water, and administrative costs, and that these estimated impacts will not fall evenly across all tribal systems. The final PFAS NPDWR does offer regulatory relief by providing flexibilities for all water systems to potentially utilize pre-existing monitoring data in lieu of initial monitoring requirements and for ground water CWSs and NTNCWSs serving 10,000 or fewer people to reduce initial monitoring from quarterly monitoring during a consecutive 12-month period to only monitoring twice during a consecutive 12-month period. These flexibilities may result in implementation cost savings for many tribal systems since 98 percent of tribal CWSs and 94 percent of NTNCWs serve 10,000 or fewer people.

The EPA has concluded that the final rule has Tribal implications, because it will impose direct compliance costs on Tribal governments, and the federal government will not provide funds necessary to pay those direct compliance costs. However, the EPA notes that the federal government will provide a potential source of funds necessary to offset some of those direct compliance costs. The Infrastructure Investment and Jobs Act (also known as the BIL, P.L. 117-58) invests over $11.7 billion in the DWSRF General Supplemental fund; $4 billion in the DWSRF Emerging Contaminants fund; and $5 billion in the EC-SDC grants program. The EPA has reserved a portion of the EC-SDC program for EPA Regions to provide direct support to Tribes, similar to support under the Small, Underserved, and Disadvantaged Communities Tribal program that was enacted under the WIIN Act. Together, these funds will reduce people’s exposure to PFAS and other emerging contaminants through their drinking water. Additionally, the EPA partners closely with the Indian Health Service (IHS) Areas to identify infrastructure needs and to implement drinking water infrastructure projects. Additionally, the EPA partners with IHS to provide technical assistance to support compliance with regulatory requirements.

Consistent with the EPA's Policy on Consultation and Coordination with Indian Tribes (May 4, 2011), the EPA consulted with tribal officials early in the process of developing the proposed regulation to gain an understanding of tribal views on key areas of the proposed PFAS NPDWR and provide tribal officials an opportunity to have meaningful and timely input on its development. For more information on the consultation with tribes, refer to the Summary Report on Tribal Consultation: Development of the Proposed PFAS NPDWR in the public docket at https://www.regulations.gov/document/EPA-HQ-OW-2022-0114-0704.

9.8 Executive Order 13045: Protection of Children from Environmental Health and Safety Risks

Executive Order 13045 (1997), entitled "Protection of Children from Environmental Health and Safety Risks" (62 FR 19885; April 23, 1997) applies to any rule initiated after April 21, 1998, that (1) is determined to be "economically significant" as defined under Executive Order 12866; and (2) concerns an environmental, health, or safety risk that the EPA has reason to believe may have a disproportionate effect on children. If the regulatory action meets both criteria, the EPA must evaluate the environmental, health, or safety effects of the planned rule on children, and explain why the planned regulation is preferable to other potentially effective and reasonably feasible options considered by the EPA.

The final rule is subject to Executive Order 13045 because it is economically significant as defined in Executive Order 12866. This action's health and risk assessments are contained in Section 6.2.2, and the associated appendices. The EPA expects that the final rule would provide additional protection to both children and adults who consume drinking water supplied by the affected systems. The EPA also expects that the benefits of the final rule, including reduced health risk, will provide significant benefits to infants and children. As detailed in the Final Human Health Toxicity Assessments for PFOA and PFOS (U.S. EPA, 2024e; U.S. EPA, 2024f), the ATSDR *Toxicological Profile for Perfluoroalkyls* (ATSDR, 2021), and the toxicity assessments for HFPO-DA and PFBS (U.S. EPA, 2021c; U.S. EPA, 2021d), there is evidence for adverse effects of PFAS for several developmental and reproductive endpoints, as well as evidence for adverse endocrine, and immune effects in infants or children. The EPA discusses the qualitative benefits from avoided adverse health effects of PFOA, PFOS, and other PFAS, including effects on infants and children in Section 6.2.2.2. In Section 6.2.2.2.1, the EPA quantifies the avoided morbidity and mortality associated with reductions in infant birth weight from reduced maternal PFOA and PFOS exposure in drinking water. The EPA also assesses the potential benefits of reduced PFNA on infant birth weight in a sensitivity analysis found in Appendix K.

This rulemaking finalizes the MCLGs for PFOA and PFOS as zero based on cancer effects. This MCLG is protective of the adverse effects observed in infants and children (e.g., decreased birth weight). This rulemaking also finalizes individual MCLGs for HFPO-DA, PFNA, and PFHxS, as well as the HI MCLG for mixtures of PFBS, HFPO-DA, PFNA, and PFHxS. The chronic toxicity values (i.e., chronic oral reference dose and equivalents) used to develop these MCLGs U.S. EPA, 2024e; U.S. EPA, 2024fprovide an estimate of a daily oral exposure to the human population (including sensitive subpopulations) that is likely to be without an appreciable risk of deleterious non-cancer effects during a lifetime.

9.9 Executive Order 13211: Actions That Significantly Affect Energy Supply, Distribution, or Use

Executive Order 13211 (2001), "Actions Concerning Regulations That Significantly Affect Energy Supply Distribution, or Use," provides that agencies shall prepare and submit to the Administrator of the Office of Information and Regulatory Affairs, OMB, a Statement of Energy Effects for certain actions identified as "significant energy actions." Section 4(b) of Executive Order 13211 defines "significant energy actions" as "any action by an agency (normally published in the Federal Register) that promulgates or is expected to lead to the promulgation of a final rule or regulation, including notices of inquiry, advance notices of proposed rulemaking, and notices of proposed rulemaking: (1)(i) that is a significant regulatory action under Executive Order 12866 or any successor order, and (ii) is likely to have a significant adverse effect on the supply, distribution, or use of energy; or (2) that is designated by the Administrator of the Office of Information and Regulatory Affairs as a significant energy action."

The final rule is not a "significant energy action" as defined in Executive Order 13211. The EPA estimates that the PFAS NPDWR will result in an increased electricity use of approximately 229 GWh per year, for more information see Section 9.2. Total U.S. electricity consumption in 2022 was about 4.05 million GWh (U.S. EIA, 2023). The electricity consumed as a result of the PFAS NPDWR represents approximately 0.005% of total U.S. electricity consumption. This rule is a significant regulatory action under Executive Order 12866; however, it is not likely to have a significant adverse effect on the supply, distribution, or use of energy, for the reasons described as follows.

9.9.1 Energy Supply

The final rule does not regulate power generation, either directly or indirectly, and public and private systems subject to the proposed rule do not, as a general rule, generate power. Further, the energy cost increases borne by customers of systems as a result of the final rule is a low percentage of the total cost of water. Therefore, power generation utilities that purchase water as part of their operations are unlikely to face any significant effects as a result of the final rule.

9.9.2 Energy Distribution

The final rule does not regulate any aspect of energy distribution and systems that are regulated by the proposed rule already have electrical service. The rule is not expected to increase peak electricity demand for systems because of the small amount of electricity used (see above). Therefore, the EPA assumes that the existing connections are adequate and that the final rule has no discernible adverse effect on energy distribution.

9.9.3 Energy Use

The EPA has determined that the incremental energy used to implement water treatment at drinking water systems in response to the final regulatory requirements is minimal. Therefore, the EPA does not expect any noticeable effect on the national levels of power generation in terms of average and peak loads.

9.10 National Technology Transfer and Advancement Act

Section 12(d) of the NTTAA of 1995 directs the EPA to use voluntary consensus standards in its regulatory activities unless to do so would be inconsistent with applicable law or otherwise impractical. Voluntary consensus standards are technical standards (e.g., materials specifications, test methods, sampling procedures, and business practices) that are developed or adopted by voluntary consensus standards bodies. NTTAA directs the EPA to provide Congress, through OMB, explanations when the EPA decides not to use available and applicable voluntary consensus standards.

The EPA's approved monitoring and sampling protocols generally include voluntary consensus standards developed by agencies such as the American National Standards Institute (ANSI) and other such bodies wherever the EPA deems these methodologies appropriate for compliance monitoring.

9.11 Executive Order 12898: Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations, Executive Order 14096: Revitalizing our Nation's Commitment to Environmental Justice for All

Executive Order 12898 (1994), "Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations" (59 FR 7629, February 16, 1994) established federal executive policy on environmental justice. Its main provision directs federal agencies, to the greatest extent practicable and permitted by law, to make environmental justice part of their mission. Agencies must do this by identifying and addressing as appropriate any disproportionately high and adverse human health or environmental effects of their programs, policies, and activities on minority populations and low-income populations in the U.S. Executive Order 14096 (2023), "Revitalizing our Nation's Commitment to Environmental Justice for All" (88 FR 25251, April 21, 2023) builds upon and strengthens its commitment to environmental justice outlined in Executive Order 12898, directing the federal government to identify, analyze, and address disproportionate and adverse human health or environmental effects of agency actions on communities with environmental justice concerns. For information on the EPA's Environmental Justice Analysis, see Chapter 8.

On March 2, 2022 and April 5, 2022, the EPA held public stakeholder meetings related to EJ and the development of the proposed NPDWR. The meetings provided an opportunity for the EPA to share information and for communities to offer input on EJ considerations related to the development of the proposed rule. The EPA received public comment on topics including establishing an MCL for PFAS and regulating PFAS as a class, affordability of PFAS abatement options and responsibility for remediation, limiting industrial discharge of PFAS, and the EPA's relationship with community groups. For more information on the EJ stakeholder meetings, refer to the EJ Considerations for the Development of the Proposed PFAS Drinking Water Regulation Public Meeting Summaries in the public docket at https://www.regulations.gov/document/EPA-HQ-OW-2022-0114-0009 and https://www.regulations.gov/document/EPA-HQ-OW-2022-0114-0026. Additionally, the written public comments from this pre-rule proposal engagement are included within the public docket.

9.12 Consultations with the Science Advisory Board, National Drinking Water Council, and the Secretary of Health and Human Services

9.12.1 Science Advisory Board

As required by Section 1412(e) of the SDWA, in 2021-2022, the EPA asked SAB to evaluate the current scientific data on the following: The EPA's Proposed Approaches to the Derivation of a Draft Maximum Contaminant Level Goal for PFOA and PFOS in Drinking Water (U.S. EPA, 2021f; U.S. EPA, 2021g); a draft framework for estimated noncancer health risks associated with mixtures of PFAS; and the EPA's methodology for evaluating reduced cardiovascular disease risks. The EPA sought SAB comment on whether the analyses provided in these documents are scientifically supported, clearly described, and informative toward supporting the EPA's proposed National Primary Drinking Water Rulemaking effort (U.S. EPA, 2022j). The SAB PFAS Review Panel deliberated and sought input from public meetings held in December 2021, January 2022, and May 2022. The SAB Chartered Body conducted a quality review of the draft panel report July 2022. The SAB's final report, titled "EPA's Analyses to Support EPA's National Primary Drinking Water Rulemaking for PFAS" was transmitted to the EPA Administrator on August 22, 2022. See SAB website at for more information on the SAB review.[116] For information on the EPA responses to SAB's review, see U.S. EPA (2022i).

9.12.2 National Drinking Water Advisory Council

In accordance with Section 1412 (d) of the SDWA, the EPA consulted with NDWAC, on the proposed rule. The EPA consulted with NDWAC in a public meeting on April 19, 2022, on key areas of the proposed rule including monitoring, treatment, public notification, and PFAS mixtures. For more information on the consultation with the NDWAC, refer to the NDWAC Virtual Public Meeting Summary in the public docket at https://www.regulations.gov/document/EPA-HQ-OW-2022-0114-0705.

On August 8, 2023, the EPA consulted with the NDWAC prior to the final rule during a virtual meeting where the EPA presented the proposed PFAS NPDWR, including the proposed MCLs, monitoring and public notification requirements, and treatment and economic considerations. The EPA reiterated that the PFAS NPDWR was developed with extensive consultation from state, local and tribal partners to identify avenues that would reduce PFAS in drinking water and reaffirmed its commitment to working with these partners on rule implementation. The EPA carefully considered the information provided by the NDWAC during the development of a final PFAS NPDWR. A summary of the NDWAC input from that meeting is available in the National Drinking Water Advisory Council Meeting Summary Report (NDWAC, 2023).

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[116] https://sab.epa.gov/ords/sab/f?p=100:18:10311539418988:::18:P18_ID:2601#charge

9.12.3 Secretary of Health and Human Services

In accordance with Section 1412 (d) of the SDWA, on September 28, 2022, the EPA consulted with the Department of Health and Human Services (HHS). The EPA provided information to HHS officials on the draft proposed NPDWR and considered HHS input as part of the interagency review. A summary of this meeting is available in the docket at EPA-HQ-OW-2022-0114 at www.regulations.gov.

On November 2nd, 2022, the EPA consulted with HHS on the final rule. Like with the proposed rule, the EPA provided information to HHS officials on the final NPDWR and considered HHS input as part of the interagency review. A summary of this meeting is available in the docket at EPA-HQ-OW-2022-0114 at www.regulations.gov.

9.13 Affordability Analyses

The SDWA, as amended in 1996, requires that the EPA list technologies for small systems [Section 1412(b)(4)(E)(ii)]:

> The Administrator shall include in the list any technology, treatment technique, or other means that is affordable, as determined by the Administrator in consultation with the States, for small public water systems serving -
> (I) a population of 10,000 or fewer but more than 3,300;
> (II) a population of 3,300 or fewer but more than 500; and
> (III) a population of 500 or fewer but more than 25;
> and that achieves compliance with the MCL or treatment technique, including packaged or modular systems and point-of-entry or POU treatment units.

The EPA’s long-standing methodology for determining whether there are affordable compliance technologies for a new drinking water standard for small systems compares the cumulative cost of providing drinking water that complies with the new standard to an affordability threshold equal to 2.5 percent of median household income (63 FR 42032). Should the EPA determine there are no affordable SSCTs, the SDWA Section 1412(b)(15)(B) requires the EPA to identify variance technologies that may not achieve compliance with the drinking water standard but achieve the maximum reduction or inactivation efficiency that is affordable considering the size of the system and the quality of the source water.

In addition to the required analysis for small system affordability, the EPA is using alternative expenditure margins and other changes to the national level affordability methodology to better understand the cost impacts of new standards on low income and disadvantaged households served by small drinking water systems. As part of this analysis, the EPA is utilizing a number of recommendations from the SAB, NDWAC, and other stakeholders such as the AWWA. The agency conducted supplemental affordability analyses using alternative metrics suggested to the EPA by these advisory bodies and stakeholders to demonstrate the potential affordability implications of the proposed NPDWR on the determination of affordable technologies for small systems at the national level of analysis.

The EPA’s national small system affordability determination can be found in Section 9.13.1. The EPA’s supplementary affordability analyses can be found in Section 9.13.2.

9.13.1 National Small System Affordability Determination

The EPA determined that there are several affordable treatment technologies for small systems. The determination, documented in *Best Available Technologies and Small System Compliance Technologies for Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water* (U.S. EPA, 2024c), compared the estimated incremental treatment costs per household with a baseline expenditure margin that equals 2.5 percent of median household income minus baseline drinking water utility cost per household. Table 9-13 shows which technologies satisfy the affordability criterion for three small system size categories. Where the EPA does not consider the substantial financial assistance for capital costs available as part of the Bipartisan Infrastructure Law and other mechanisms, for systems serving between 25-500 people IX and POU devices are affordable technologies, GAC is affordable in some cases, and centralized RO is not. In this scenario, for the smallest system size category, upper bound estimated annual household treatment costs for GAC exceed the expenditure margin. This exceedance is primarily driven by capital costs and attributable to the use of high-cost materials (e.g., stainless steel) in the upper bound estimates. Systems using low-cost materials, but with source water characteristics otherwise set to the upper bound (e.g., influent PFAS at approximately 7,000 ppt, influent TOC at 2 mg/L), would fall below the expenditure margin. As discussed in Section 9.13.2.2 below, where available financial assistance for capital costs is considered, GAC, IX, and POU devices are affordable technologies (see Section 9.13.2.2 below). For systems serving 501-3,300 people, where The EPA does not consider financial assistance, GAC, IX, and POU devices are affordable technologies, and RO is affordable in some cases. For systems serving 3,301-10,000 people GAC, IX and centralized RO are affordable technologies, and POU treatment is not applicable to systems of that size category.[117]

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[117] Note, the results shown in Table 9-13 and discussed in this section are dependent on the estimated annual household technology costs reported in Table 9-15 which assumes costs associated with standard waste management of spent GAC and spent IX resin using current typical management practices (reactivation for GAC and incineration for resin). Future changes to regulations might result in classification of spent GAC or spent IX resin as hazardous waste. The EPA estimated annual cost per household if systems are required to dispose of these residuals as hazardous waste and conducted the same national level affordability analysis using the higher hazardous waste handling treatment costs. The agency found the increased treatment costs for both GAC and IX did not change the affordability conclusions. See Table 9-16 for annualized cost per household assuming hazardous waste disposal and U.S. EPA (2024c) for the complete analysis.

A technology must be both effective and affordable to be designated as an SSCT. Technologies that meet the effectiveness criterion include those designated as BATs for the final rule are: GAC, IX, and RO. This section also presents preliminary affordability results for POU devices. POU devices are not currently evaluated as a compliance option because the regulatory options under consideration require treatment to concentrations below the current certification standard for POU devices. However, POU treatment is anticipated to become a compliance option for small systems in the future should NSF/ANSI or another accredited third-party certification entity develop a new certification standard that mirrors the EPA's regulatory standard. More information is available in Section XI of the preamble the EPA does not anticipate additional costs for water systems associated with the certification updating process. To evaluate affordability, the EPA compared incremental costs per household for each technology against an expenditure margin. Table 9-14 shows the expenditure margins for each system size category. It also shows how the EPA derived the expenditure margins, beginning with estimates of median household income (MHI), which vary by system size category. The annual affordability threshold for household expenditures on drinking water is 2.5 percent of MHI. The EPA deducted estimates of baseline or current water bills from the affordability threshold to obtain the expenditure margin estimates.

Table 9-13: SSCT Affordability Analysis Results – Technologies that Meet Effectiveness Criterion

System Size (Population Served)	GAC	IX	RO	POU[a]
25 to 500	In some cases[b]	Yes	No	Yes
501 to 3,300	Yes	Yes	No[b]	Yes
3,301 to 10,000	Yes	Yes	Yes	Data Unavailable[c]

Abbreviations: GAC – granular activated carbon; IX – ion exchange; POU – point-of-use treatment; RO – reverse osmosis; SSCT – small system compliance technology.

Notes:

[a] POU devices are not currently a compliance option because the final rule requires treatment to concentrations below the current certification standard for POU devices. However, POU treatment is anticipated to become a compliance option for small systems in the future should NSF/ANSI or another accredited third-party certification entity develop a new certification standard that mirrors (or is demonstrated to treat to concentrations lower than) the EPA's proposed regulatory standard. The affordability conclusions presented here should be considered preliminary because they reflect the costs of devices certified under the current standard, not a future standard. More information is in Section XI of the preamble for the final rule.

[b] Upper bound estimates of annual household treatment costs exceed expenditure margin. Lower bound estimates of annual household treatment costs do not exceed the expenditure margin. This exceedance is primarily driven by capital costs and attributable to the use of high-cost materials (e.g., stainless steel) in the upper bound estimates. Systems using low-cost materials, but with source water characteristics otherwise set to the upper bound (e.g., influent PFAS at approximately 7,000 ppt, influent TOC at 2 mg/L), would fall below the expenditure margin.

[c] For evaluating costs for this PFAS rulemaking, the EPA's WBS model for POU treatment does not cover systems serving more than 3,300 people (greater than 1 MGD design flow).

Table 9-14: Expenditure Margins for SSCT Affordability Analysis

System Size (Population Served)	Median Household Income[a]	Affordability Threshold[b]	Baseline Water Cost[c]	Expenditure Margin
	A	B = 2.5% x A	C	D = B - C
25 to 500	$62,950	$1,574	$551	$1,022
501 to 3,300	$60,926	$1,523	$638	$885
3,301 to 10,000	$66,746	$1,669	$666	$1,002

Abbreviations: SSCT – small system compliance technology.

Notes:

[a] MHI based on U.S. Census Bureau's American Community Survey five-year estimates (U.S. Census Bureau, 2010) stated in 2010 dollars, adjusted to 2022 dollars using the CPI (for all items) for areas under 2.5 million persons.

[b] Affordability threshold equals 2.5 percent of MHI.

[c] Household water costs derived from 2006 Community Water System Survey (U.S. EPA, 2009), based on residential revenue per connection within each size category, adjusted to 2022 dollars based on the CPI for All Urban Consumers: Water and Sewer and Trash Collection Services in U.S. City Average.

Table 9-15 provides ranges of per-household costs for each technology and system size category. The ranges indicate minimum and maximum costs, for further information on SSCT costs, see U.S. EPA (2024c).

Table 9-15: Total Annual Cost per Household for Candidate Technologies

System Size (Population Served)	GAC	IX	RO	POU[a]
25 to 500	$607 to $1,241	$563 to $990	$4,332 to $5,224	$345 to $357
501 to 3,300	$203 to $484	$171 to $351	$721 to $1,324	$327 to $327
3,301 to 10,000	$178 to $417	$145 to $284	$388 to $544	Data unavailable[b]

Abbreviations: GAC – granular activated carbon; IX – ion exchange; POU – point-of-use treatment; RO – reverse osmosis; SSCT – small system compliance technology.

Notes:

[a] POU devices are not currently a compliance option because the final rule requires treatment to concentrations below the current certification standard for POU devices. However, POU treatment is anticipated to become a compliance option for small systems in the future should NSF/ANSI or another accredited third-party certification entity develop a new certification standard that mirrors (or is demonstrated to treat to concentrations lower than) the EPA's proposed regulatory standard. Costs presented here should be considered preliminary estimates because they reflect the costs of devices certified under the current testing standard, not a future standard. More information is in Section XI of the preamble for the final rule.

[b] For evaluating costs for this PFAS rulemaking, the EPA's WBS model for POU treatment does not cover systems serving more than 3,300 people (greater than 1 MGD design flow).

The results discussed above assume management of spent GAC and spent IX resin using current typical management practices (reactivation for GAC and incineration for resin). The EPA has proposed some PFAS be designated as hazardous substances under CERCLA and is in the process of proposing some PFAS be listed as hazardous constituents under the RCRA. If finalized, neither of these actions would result in new requirements as to how PFAS containing waste, including spent GAC or resin, is required to be managed. However, waste management facilities may, at their own discretion, refuse to accept PFAS-containing materials or drinking water treatment operations may choose to send spent GAC and resin containing PFAS to facilities permitted to treat and/or dispose of hazardous wastes. To consider the implications of this possibility, the EPA has developed an assessment of the current unit costs for disposing spent treatment materials and the costs associated with their disposal as hazardous waste. Table 9-16 shows the resulting cost per household if systems dispose of these residuals as hazardous waste. For the smallest system size category not considering available financial assistance, upper bound estimated annual household treatment costs for GAC and IX exceed the expenditure margin in Table 9-8. This exceedance is primarily driven by capital costs and attributable to the use of high-cost materials (e.g., stainless steel) in the upper bound estimates. Systems using low-cost materials, but with source water characteristics otherwise set to the upper bound (e.g., influent PFAS at approximately 7,000 ppt, influent TOC at 2 mg/L), would fall below the expenditure margin, even under a hazardous waste scenario. Technologies are affordable for all small systems when the technologies do not use high-cost materials. Technologies that do not use high-cost materials are available for small systems. Although costs increase in this scenario, the increases are not significant enough to change the conclusions about affordability.

Table 9-16: Total Annual Cost per Household Assuming Hazardous Waste Disposal

System Size (Population Served)	GAC	IX
25 to 500	$630 to $1,369	$586 to $1,027
501 to 3,300	$211 to $520	$176 to $360
3,301 to 10,000	$185 to $438	$148 to $289

Abbreviations: GAC – granular activated carbon; IX – ion exchange.

9.13.2 Supplemental Affordability Analyses

In 2002, Congress required the EPA to re-evaluate small system variance policy because of the concern with the high cost of arsenic treatment in small communities. In response, in 2003, the EPA consulted with NDWAC and SAB. The SAB and NDWAC made a number of recommendations regarding the method by which the EPA evaluates the affordability of compliance with drinking water standards.

Some key recommendations made by both the SAB and the NDWAC include:

- The EPA should consider the household cost of each new regulation on an incremental basis rather than a total cost of all water treatment regulations, and
- The EPA should consider reducing the current affordability threshold, and
- Financial assistance should be incorporated in the affordability calculations if the financial support is generally available to all systems (nationwide).

In addition to the SAB and NDWAC recommendations, several additional reports by stakeholders have offered recommendations on the improvement of the EPA's affordability methodology, including:

- The National Academy of Public Administration (NAPA) report, Developing a New Framework for Community Affordability of Clean Water Services (NAPA, 2017),
- The National Association of Clean Water Agencies, American Water Works Association, and Water Environment Federation report, Developing a New Framework for Household Affordability and Financial Capability Assessment in the Water Sector (Raucher et al., 2019), and

- The American Water Works Association expert panel report, Improving the Evaluation of Household-Level Affordability in SDWA Rulemaking: New Approaches (AWWA, 2021).

The recommendations in these reports point to the need to further assess the impacts of new regulatory costs across income groups with a particular focus on low income and disadvantaged communities and individuals within water systems. In particular, the American Water Works Association (2021) expert panel report stressed that the agency also assess the affordability impacts to low-income households by setting the per household expenditure margin based on the lowest quintile (20th percentile) of the income distribution.

The EPA has estimated the impact of some potential changes to National Level Affordability Criteria and analysis based on suggested changed from the SAB, NDWAC, and AWWA's expert panel. In the following subsections, the EPA estimated small system affordability based on; (1) an incremental approach with expenditure margins of 1.0 percent of annual MHI and 2.5 percent of the lowest quintile of annual household income, and no additional adjustment for total current annual water expenditures, and (2) taking into account nationally available financial assistance when assessing affordability.

9.13.2.1 Small System Affordability Analysis with Potential Additional Expenditure Margins

As part of the EPA's consideration of additional annual expenditure margins to improve the assessment of affordability impacts to low income and disadvantaged communities, two incremental cost analyses are conducted utilizing alternative potential expenditure margins. Given the recommendations from the NDWAC, the first expenditure margin threshold is based on 1.0 percent of annual MHI. The second expenditure margin threshold is set equal to 2.5 percent of the lowest quintile of annual household income and is based on the American Water Works Association (2021) expert panel report. These expenditure margins are estimated for each of the small system size categories: 25 to 500, 501 to 3,300, and 3,301 to 10,000 people served. As this is an incremental analysis no additional adjustments are made to the values to account for current annual drinking water cost. Table 9-17 shows the calculated annual expenditure margins by system size.

Table 9-17: Potential Annual Expenditure Margins for SSCT Affordability Analysis

System Size (Population Served)	1.0% of Median Household Income[a]	2.5% of Lowest Quintile Income[b]
	A	B
25 to 500	$629	$731
501 to 3,300	$609	$714
3,301 to 10,000	$667	$774

Abbreviations: SSCT – small system compliance technology.
Notes:
[a] MHI is based on U.S. Census Bureau's American Community Survey five-year estimates (U.S. Census Bureau, 2010) stated in 2010 dollars, adjusted to 2022 dollars using the CPI (for all items) for areas under 2.5 million persons.
[b] Lowest quintile (20th percentile) household income is based on U.S. Census 2010 American Community Survey 5-year estimates (U.S. Census Bureau, 2010) stated in 2010 dollars, adjusted to 2022 dollars using the CPI (for all items) for areas under 2.5 million persons.

Given these alternative annual expenditure margins the remainder of the assessment process is the same as the EPA's current small system affordability methodology. The estimated total annual household costs for each of the deemed efficient treatment technologies presented in Table 9-15 are compared against the estimated annual expenditure margin thresholds from Table 9-17 for each system size category. Table 9-18 presents the affordability results using the 1.0 percent of annual MHI expenditure margin and Table 9-19 provides the information when the 2.5 percent of the lowest quintile of annual household income is used as the threshold.

Table 9-18: Affordability Analysis Results Using a 1.0% of Annual Median Household Income Expenditure Margin

System Size (Population Served)	GAC	IX	RO	POU[a]
25 to 500	In some cases[b]	In some cases[b]	No	Yes
501 to 3,300	Yes	Yes	No	Yes
3,301 to 10,000	Yes	Yes	Yes	Data unavailable[c]

Abbreviations: GAC – granular activated carbon; IX – ion exchange; POU– point-of-use treatment; and RO – reverse osmosis.
Notes:
[a] POU devices are not currently a compliance option because the final rule requires treatment to concentrations below the current certification standard for POU devices. However, POU treatment is anticipated to become a compliance option for small systems in the future should NSF/ANSI or another accredited third-party certification entity develop a new certification standard that mirrors (or is demonstrated to treat to concentrations lower than) the EPA's final regulatory standard. The affordability conclusions presented here should be considered preliminary because they reflect the costs of devices certified under the current standard, not a future standard. More information is in Section XI of the preamble for the final rule.
[b] Upper bound estimates of annual household treatment costs exceed expenditure margin. Lower bound estimates of annual household treatment costs do not exceed the expenditure margin. This exceedance is primarily driven by capital costs and attributable to the use of high-cost materials (e.g., stainless steel) in the upper bound estimates. Systems using low-cost materials, but with source water characteristics otherwise set to the upper bound (e.g., influent PFAS at approximately 7,000 ppt, influent TOC at 2 mg/L), would fall below the expenditure margin.
[c] For evaluating costs for this PFAS rulemaking, the EPA's WBS model for POU treatment does not cover systems serving more than 3,300 people (greater than 1 MGD design flow).

Table 9-19: Affordability Analysis Results Using a 2.5% of Lowest Quintile of Annual Household Income Expenditure Margin

System Size (Population Served)	GAC	IX	RO	POU[a]
25 to 500	In some cases[b]	In some cases[b]	No	Yes
501 to 3,300	Yes	Yes	No	Yes
3,301 to 10,000	Yes	Yes	Yes	Data unavailable[c]

Abbreviations: GAC – granular activated carbon; IX – ion exchange; POU – point-of-use treatment; and RO – reverse osmosis.

Notes:

[a] POU devices are not currently a compliance option because the final rule requires treatment to concentrations below the current certification standard for POU devices. However, POU treatment is anticipated to become a compliance option for small systems in the future should NSF/ANSI or another accredited third-party certification entity develop a new certification standard that mirrors (or is demonstrated to treat to concentrations lower than) the EPA's proposed regulatory standard. The affordability conclusions presented here should be considered preliminary because they reflect the costs of devices certified under the current standard, not a future standard. More information is in Section XI of the preamble for the final rule.

[b] Upper bound estimated annual household treatment costs exceed expenditure margin. Lower bound estimated annual household treatment costs do not exceed the expenditure margin. This exceedance is primarily driven by capital costs and attributable to the use of high-cost materials (e.g., stainless steel) in the upper bound estimates. Systems using low-cost materials, but with source water characteristics otherwise set to the upper bound (e.g., influent PFAS at approximately 7,000 ppt, influent TOC at 2 mg/L), would fall below the expenditure margin.

[c] For evaluating costs for this PFAS rulemaking, the EPA's WBS model for POU treatment does not cover systems serving more than 3,300 people (greater than 1 MGD design flow).

The results in both Table 9-18 and Table 9-19, which utilize the supplemental expenditure margins, of 1.0 percent of annual MHI and 2.5 percent of the lowest quintile of annual household income, and the results of the EPA's national level affordability analysis in Table 9-9, which utilizes a household expenditure margin estimated by adjusting 2.5 percent of median household income minus baseline median annual drinking water costs, differ in the case of IX for systems serving 25 to 500 people. As indicated by the "In some cases" reported in Table 9-18 and Table 9-19 for GAC and IX the upper bound annual household treatment cost for both these technologies exceed both the 1.0 percent of annual MHI and 2.5 percent of the lowest quintile of annual household income expenditure margins, however, the estimated lower bound annual household treatment costs do not exceed the expenditure margins. The alternative expenditure margins also changed the affordability results for RO in the 501–3,300 system size category. In the national affordability analysis using the 2.5 percent of MHI with baseline adjustment upper bound RO annual household cost estimates exceed the expenditure margin but the lower bound costs do not. When using both the 1.0 percent of annual MHI and 2.5 percent of the lowest quintile of annual household income potential criteria both the high and low bound estimated annual household treatment costs exceed the expenditure margins.[118]

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[118] Note, the results shown in Table 9-18 and Table 9-19 and discussed in this section are dependent on the estimated annual household technology costs reported in Table 9-15 which assumes costs associated with standard waste management of spent GAC and spent IX resin using current typical management practices (reactivation for GAC and incineration for resin). Future changes to regulations might result in classification of spent GAC or spent IX resin as hazardous waste. The EPA estimated annual cost per household if systems are required to dispose of these residuals as hazardous waste and conducted the same national level affordability analyses with the 1.0 percent of MHI and 2.5 percent of the lowest quintile of annual household income expenditure margins and using the higher hazardous waste handling treatment costs. The agency found the increased treatment costs for both GAC and IX did not change the affordability conclusions.

9.13.2.2 Small System Affordability Analysis When Accounting for Financial Assistance

The SAB and NDWAC recommended to the EPA that the national level affordability analysis should include the impact of financial assistance if the financial support is generally available to all systems (nationwide). The recommendations themselves indicate a two-step process; (1) determine if and how much financial assistance is available to small systems on a national level for compliance with a specific rule, in this case the PFAS drinking water rule, and (2) calculate the potential impact of the financial assistance on the estimated per household treatment costs for each of the small system size categories.

On the national level, significant financial assistance is available to small systems for the installation of PFAS treatment technology. One critical and long-established source of this assistance is available through the EPA's DWSRF Program that was authorized by Congress as part of the 1996 Amendments to the SDWA. The DWSRF's purpose is to provide a source of financial assistance to water systems and states to help them achieve the public health protection objectives of SDWA. A unique feature of the DWSRF Program is that it is state based. The EPA awards capitalization grants to states who provide a 20 percent match, creating a dedicated fund from which loans are made to water systems and into which the loan repayments (and interest) are deposited so they can be loaned out again. Within some broad statutory constraints contained in SDWA, the states have considerable flexibility to tailor the DWSRF Program to their own unique needs and circumstances.

The SDWA established three criteria at the core of the process used by states in ranking projects in priority order to receive funding. States are required, to the maximum extent practicable, to give priority for the use of DWSRF funds to projects that:

1. Address the most serious risk to human health;
2. Are necessary to ensure compliance with SDWA requirements; and
3. Assist systems most in need on a per household basis according to state affordability criteria.

Thus, system level affordability, according to state affordability criteria, is a central consideration in ranking projects eligible to receive DWSRF assistance. Each state has developed, and the EPA has approved, a project priority ranking procedure. The specific weight given to affordability considerations vis-à-vis public health and SDWA compliance considerations varies from state to state. States are required to include their project priority ranking system as part of the Intended Use Plan they are required to develop in support of their application for each capitalization grant. The Intended Use Plan must contain both the project priority ranking system and the priority list of projects eligible for DWSRF assistance. The state must provide notice and opportunity for public comment on the priority list of projects.

Under the core DWSRF Program, the state may establish an interest rate between zero percent and the market rate. The lower the interest rate, the greater the subsidy provided to the borrower. SDWA requires states to establish a Disadvantaged Communities Program within their DWSRF under which communities considered disadvantaged according to state developed affordability criteria could receive additional subsidies beyond a zero percent loan. These additional subsidies often take the form of principal forgiveness (i.e., loan forgiveness) or grants. There is no limit to the amount of additional subsidy that can be provided to a particular project except for an overall limit on the total amount of additional subsidy of 35 percent of the state's annual capitalization grant.

This additional subsidization could be directed entirely to a few projects, essentially making the assistance those projects receive equivalent to a 100 percent grant; or the additional subsidization could be distributed among a larger number of projects and combined with zero or low-interest loans. States may also offer communities they consider disadvantaged[119] a loan term of 40 years rather than the base period of 20 to 30 years. Notably, the loan term cannot extend beyond the design life of the capital improvement constructed via the DWSRF loan.

The SDWA provided the EPA with the authority to publish information to assist states in establishing affordability criteria for purposes of a disadvantaged community program. The agency worked with a group of expert stakeholders and published "Information for States on Developing Affordability Criteria for Drinking Water" (document number 816-R-98-002) in February 1998 (U.S. EPA, 1998b). The agency provided additional information to assist states' affordability criteria development in the "Implementation of the Clean Water and Drinking Water State Revolving Fund Provisions of the Bipartisan Infrastructure Law" memorandum in March 2022 (U.S. EPA, 2022d).

PFAS drinking water treatment loans and grants have been and will continue to be available to systems of all sizes under the traditional DWSRF program funding and allocation structure. In addition to these funding sources, on November 15, 2021, the Infrastructure Investment and Jobs Act (IIJA), often referred to as the Bipartisan Infrastructure Law or BIL (P.L. 117-58), appropriated $4 billion over 5 years ($800,000,000 per year) for projects that are DWSRF eligible whose primary purpose must be to address emerging contaminants, with a focus on PFAS. The EPA expects to establish a NPDWR for PFOA and PFOS. The agency is also evaluating additional PFAS and groups of PFAS. Given stated Congressional intent of this appropriation, PFAS-focused projects will be eligible for funding under this appropriation regardless of whether the EPA has established a NPDWR for that particular PFAS or group of PFAS. These BIL funds must be distributed to communities entirely as forgivable loans or grants, and states are not required to provide matching funds as with most DWSRF projects. 25 percent of this BIL funding is targeted toward disadvantaged communities and/or communities fewer than or equal to 25,000 people.

In addition to the DWSRF BIL funds, as part of a government-wide effort to confront PFAS pollution, the BIL authorizes $5 billion as part of the EC-SDC grants program that can be used to reduce PFAS in drinking water in communities facing disproportionate impacts. The goal of the EC-SDC grants program is for states to provide grants to public water systems in small or disadvantaged communities to address emerging contaminants, including PFAS. Funding will be provided to participating states and territories to benefit small or disadvantaged communities in scoping, planning, testing, and remediating emerging contaminants in drinking and source water.

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[119] Disadvantaged community is defined as the service area of a public water system that meets affordability criteria established after public review and comment by the State in which the public water system is located.

These funds can be used in small or disadvantaged communities to address emerging contaminants like PFAS in drinking water through actions such as technical assistance, water quality testing, contractor training, and installation of centralized treatment technologies and systems. On June 15, 2022, the EPA announced that it is making $1 billion available in FY2022 of a total of $5 billion for fiscal years 2022–2026.

Given the BIL emerging contaminant funding being made available through the DWSRF and the EC-SDC grants program, the EPA expects that most small systems will have access to financial assistance for PFAS related capital expenditures. The EPA estimates that the total amount of initial capital treatment technology expenditures for small systems nationally ranges between approximately $1.8 and $3.5 billion. The EPA expects funding from BIL to be more than sufficient to cover the capital costs for small systems. Hence, it seems reasonable to consider these funds for the purposes of illustrating the potential impact of including financial assistance in the calculation of the national level affordability assessment for small system compliance technologies. Because BIL funds are limited to providing grants and loan forgiveness associated with PFAS drinking water treatment capital expenditures, the EPA in this example zeroed out only the capital cost of the candidate effective technologies. The annual per household treatment cost ranges presented in Table 9-15 represent operations and maintenance costs for the technologies by small system size category. Comparing the cost ranges in Table 9-20 with unadjusted cost ranges in Table 9-15 demonstrates the potential large decrease in technology cost when financial assistance is considered. The decreases across technologies and system size categories range from 52 percent to 84 percent for the centralized technologies, and approximately 30 percent for POU technologies.

Table 9-20: Annual Cost per Household for Candidate Technologies Assuming 100% Financial Assistance for Technology Capital Costs

System Size (Population Served)	GAC	IX	RO	POU[a]
25 to 500	$134 to $230	$140 to $161	$1,160 to $1,242	$244 to $256
501 to 3,300	$57 to $141	$58 to $78	$281 to $338	$228 to $228
3,301 to 10,000	$66 to $147	$60 to $86	$186 to $219	Data unavailable[b]

Abbreviations: GAC – granular activated carbon; IX – ion exchange; POU – point-of-use treatment; and RO – reverse osmosis.

Notes:

[a] POU devices are not currently a compliance option because the final rule requires treatment to concentrations below the current certification standard for POU devices. However, POU treatment is anticipated to become a compliance option for small systems in the future should NSF/ANSI or another accredited third-party certification entity develop a new certification standard that mirrors (or is demonstrated to treat to concentrations lower than) the EPA's proposed regulatory standard. The affordability conclusions presented here should be considered preliminary because they reflect the costs of devices certified under the current standard, not a future standard. More information is in Section XI of the preamble for the final rule.

[b] For evaluating costs for this PFAS rulemaking, the EPA's WBS model for POU treatment does not cover systems serving more than 3,300 people (greater than 1 MGD design flow).

Table 9-21, Table 9-22, and Table 9-23 below show the affordability results utilizing the 2.5 percent of annual MHI minus the baseline median annual drinking water cost, the incremental 1.0 percent of annual MHI, and using the 2.5 percent of the lowest quintile of annual household income expenditure margins, respectively. Given the significant reduction in estimated per household annual treatment costs for GAC and IX, the technologies were found to satisfy the national level affordability criterion for the three statutorily mandated small system size categories.[120] Centralized RO with high per household operations and maintenance costs, of $1,081 to $1,153, in the system size category of 25–500 people served was found to be unaffordable in that system size category across all alternative expenditure margins, but economies of scale reduce per household costs in systems serving between 501 and 10,000 people sufficiently to approve the technology as affordable under the three alternative expenditure margins. POU treatment was also found to be affordable at the national level of analysis for systems serving 25 to 500 and 501 to 3,300 people across the three presented expenditure margins. POU treatment is not applicable to systems serving more than 3,300 people given the increasing complexity of managing POU programs at such large scales.

Table 9-21: Affordability Analysis Results Using a 2.5% of Annual Median Household Income Minus the Baseline Median Annual Drinking Water Cost Expenditure Margin and Assuming 100% Financial Assistance for Technology Capital Costs

System Size (Population Served)	GAC	IX	RO	POU[a]
25 to 500	Yes	Yes	No	Yes
501 to 3,300	Yes	Yes	Yes	Yes
3,301 to 10,000	Yes	Yes	Yes	Data unavailable[b]

Abbreviations: GAC – granular activated carbon; IX – ion exchange; POU – point-of-use treatment; and RO – reverse osmosis.

Notes:

[a] POU devices are not currently a compliance option because the final rule requires treatment to concentrations below the current certification standard for POU devices. However, POU treatment is anticipated to become a compliance option for small systems in the future should NSF/ANSI or another accredited third-party certification entity develop a new certification standard that mirrors (or is demonstrated to treat to concentrations lower than) the EPA’s proposed regulatory standard. The affordability conclusions presented here should be considered preliminary because they reflect the costs of devices certified under the current standard, not a future standard. More information is available in Section XI of the preamble for the final rule.

[b] For evaluating costs for this PFAS rulemaking, the EPA’s WBS model for POU treatment does not cover systems serving more than 3,300 people (greater than 1 MGD design flow).

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[120] Note, the results shown in Table 9-21, Table 9-22, and Table 9-23 and discussed in this section are dependent on the estimated annual household technology costs reported in Table 9-20 which assumes operations and maintenance costs associated with standard waste management of spent GAC and spent IX resin using current typical management practices (reactivation for GAC and incineration for resin). Future changes to regulations might result in classification of spent GAC or spent IX resin as hazardous waste. The EPA estimated annual operations and maintenance cost per household if systems are required to dispose of these residuals as hazardous waste and conducted the same national level affordability analyses using the three alternative expenditure margins using the higher hazardous waste handling treatment costs. The agency found the increased treatment costs for both GAC and IX did not change the affordability conclusions.

Table 9-22: Affordability Analysis Results Using a 1.0% of Annual Median Household Income Expenditure Margin and Assuming 100% Financial Assistance for Technology Capital Costs

System Size (Population Served)	GAC	IX	RO	POU[a]
25 to 500	Yes	Yes	No	Yes
501 to 3,300	Yes	Yes	Yes	Yes
3,301 to 10,000	Yes	Yes	Yes	Data unavailable[b]

Abbreviations: GAC – granular activated carbon; IX – ion exchange; POU– point-of-use treatment; and RO – reverse osmosis.
Notes:

[a] POU devices are not currently a compliance option because the final rule requires treatment to concentrations below the current certification standard for POU devices. However, POU treatment is anticipated to become a compliance option for small systems in the future should NSF/ANSI or another accredited third-party certification entity develop a new certification standard that mirrors (or is demonstrated to treat to concentrations lower than) the EPA's proposed regulatory standard. The affordability conclusions presented here should be considered preliminary because they reflect the costs of devices certified under the current standard, not a future standard. More information is available in Section XI of the preamble for the final rule.

[b] For evaluating costs for this PFAS rulemaking, the EPA's WBS model for POU treatment does not cover systems serving more than 3,300 people (greater than 1 MGD design flow).

Table 9-23: Affordability Analysis Results Using a 2.5% of Lowest Quintile of Annual Household Income Expenditure Margin and Assuming 100% Financial Assistance for Technology Capital Costs

System Size (Population Served)	GAC	IX	RO	POU[a]
25 to 500	Yes	Yes	No	Yes
501 to 3,300	Yes	Yes	Yes	Yes
3,301 to 10,000	Yes	Yes	Yes	Data unavailable[b]

Abbreviations: GAC – granular activated carbon; IX – ion exchange; POU – point-of-use treatment; and RO – reverse osmosis.

Notes:

[a] POU devices are not currently a compliance option because the final rule requires treatment to concentrations below the current certification standard for POU devices. However, POU treatment is anticipated to become a compliance option for small systems in the future should NSF/ANSI or another accredited third-party certification entity develop a new certification standard that mirrors (or is demonstrated to treat to concentrations lower than) the EPA's proposed regulatory standard. The affordability conclusions presented here should be considered preliminary because they reflect the costs of devices certified under the current standard, not a future standard. More information is available in Section XI of the final rule.

[b] For evaluating costs for this PFAS rulemaking, the EPA's WBS model for POU treatment does not cover systems serving more than 3,300 people (greater than 1 MGD design flow).

10 References

3M Company. (2000). *Sanitized report: Exploratory 28-day oral toxicity study with telomer alcohol, telomer acrylate, PFHS and PFOS (positive control) by daily gavage in the rat followed by a 14/28-day recovery period [TSCA Submission]. (FYI-0500-01378 B; DCN 84000000018). St. Paul, MN*. Retrieved from https://yosemite.epa.gov/oppts/epatscat8.nsf/ReportSearchView/05ADDF3067814C978525703B004B4119

Aassve, A., Cavalli, N., Mencarini, L., Plach, S., & Sanders, S. (2021). Early assessment of the relationship between the COVID-19 pandemic and births in high-income countries. *Proceedings of the National Academy of Sciences, 118*(36).

Abt Associates. (2020). *Labor Costs for National Drinking Water Rules. Submitted to Rajiv Khera, U.S. Environmental Protection Agency, Office of Ground Water and Drinking Water. EPA Contract No. EP-B16C-0001*.

Adamson, D. T., Piña, E. A., Cartwright, A. E., Rauch, S. R., Anderson, R. H., Mohr, T., & Connor, J. A. (2017). 1, 4-Dioxane drinking water occurrence data from the third unregulated contaminant monitoring rule. *Science of the total environment, 596*, 236-245.

Agency for Toxic Substances and Disease Registry. (2018). *Toxicological Profile for Perfluoroalkyls*.

Agency for Toxic Substances and Disease Registry. (2021). *Toxicological Profile for Perfluoroalkyls*.

Almond, D., Chay, K. Y., & Lee, D. S. (2005). The costs of low birth weight. *The Quarterly journal of economics*, *120*(3), 1031-1083. doi:10.1093/qje/120.3.1031

Almond, D., Doyle Jr, J. J., Kowalski, A. E., & Williams, H. (2010). Estimating marginal returns to medical care: Evidence from at-risk newborns. *The Quarterly journal of economics, 125*(2), 591-634.

Ambavane, A., Yang, S., Atkins, M. B., Rao, S., Shah, A., Regan, M. M., . . . Michaelson, M. D. (2020). Clinical and economic outcomes of treatment sequences for intermediate- to poor-risk advanced renal cell carcinoma. *Immunotherapy*, *12*(1), 37-51. doi:10.2217/imt-2019-0199

American Association for the Advancement of Science. (2020). Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water. Retrieved from https://www.aaas.org/epi-center/pfas

American Cancer Society. (2020). *Cancer facts and figures - 2019 key statistics about kidney cancer*. Retrieved from https://www.cancer.org/cancer/types/kidney-cancer/about/key-statistics.html

American Water Works Association. (2021). Improving the Evaluation of Household-Level Affordability in SDWA Rulemaking: New Approaches.

Anderson, R. N. (1999). *Method for constructing complete annual US life tables* (Vol. 129): National Ctr for Health Statistics.

Appleman, T. D., Higgins, C. P., Quiñones, O., Vanderford, B. J., Kolstad, C., Zeigler-Holady, J. C., & Dickenson, E. R. (2014). Treatment of poly-and perfluoroalkyl substances in US full-scale water treatment systems. *Water research, 51*, 246-255.

Aquafina. (2022). Aquafina FAQ. Retrieved from https://www.aquafina.com/en-US/faq.html#:~:text=Aquafina%20originates%20from%20public%20water,can%20affect%20a%20water's%20taste

Arias, E., Heron, M., & Tejada-Vera, B. (2013). United States life tables eliminating certain causes of death, 1999-2001.

Arias, E., & Xu, J. (2019). *United States Life Tables, 2017.*

Association of State Drinking Water Administrators. (2023). *PFAS Cost of State Transactions (PCoSTS) Study*. Retrieved from https://www.asdwa.org/wp-content/uploads/2023/05/PCoSTS-Final.pdf

Averina, M., Brox, J., Huber, S., & Furberg, A.-S. (2021). Exposure to perfluoroalkyl substances (PFAS) and dyslipidemia, hypertension and obesity in adolescents. The Fit Futures study. *Environmental research, 195,* 110740.

Bach, C. C., Bech, B. H., Nohr, E. A., Olsen, J., Matthiesen, N. B., Bonefeld-Jorgensen E.C., . . . Henriksen, T. B. (2016). Perfluoroalkyl acids in maternal serum and indices of fetal growth: the Aarhus Birth Cohort. *Environmental health perspectives, 124*(6), 848-854. doi:10.1289/ehp.1510046.

Bach, C. C., Vested, A., Jørgensen, K. T., Bonde, J. P. E., Henriksen, T. B., & Toft, G. (2016). Perfluoroalkyl and polyfluoroalkyl substances and measures of human fertility: a systematic review. *Critical reviews in toxicology, 46*(9), 735-755.

Bach, J. P., Riedel, O., Pieper, L., Klotsche, J., Dodel, R., & Wittchen, H. U. (2011). Health-related quality of life in patients with a history of myocardial infarction and stroke. Cerebrovascular Diseases. *31(1),* 68-76.

Banzhaf, S., Ma, L., & Timmins, C. (2019). Environmental Justice: The Economics of Race, Place, and Pollution. *The Journal of Economic Perspectives: A Journal of the American Economic Association, 33(1), 185–208.*

Bao, W. W., Qian, Z. M., Geiger, S. D., Liu, E., Wang, S. Q., Lawrence, W. R., . . . Dong, G. H. (2017). Gender-specific associations between serum isomers of perfluoroalkyl substances and blood pressure among Chinese: isomers of C8 health project in China. *Science of the total environment, 607-608,* 1304-1312. doi:10.1016/j.scitotenv.2017.07.124

Barry, V., Winquist, A., & Steenland, K. (2013). Perfluorooctanoic Acid (PFOA) Exposures and Incident Cancers Among Adults Living Near A Chemical Plant. *Environmental health perspectives, 121*(11-12), 1313–1318. doi:https://doi.org/10.1289/ehp.1306615

Bartell, S. M., Calafat, A. M., Lyu, C., Kato, K., Ryan, P. B., & Steenland, K. (2010). Rate of decline in serum PFOA concentrations after granular activated carbon filtration at two public water systems in Ohio and West Virginia. *Environmental health perspectives, 118*(2), 222-228.

Bartell, S. M., & Vieira, V. M. (2021). Critical review on PFOA, kidney cancer, and testicular cancer. *J Air Waste Manag Assoc, 71*(6), 663-679. doi:10.1080/10962247.2021.1909668

Barton, K. E., Starling, A. P., Higgins, C. P., McDonough, C. A., Calafat, A. M., & Adgate, J. L. (2020). Sociodemographic and Behavioral Determinants of Serum Concentrations of Per- and Polyfluoroalkyl Substances in A Community Highly Exposed to Aqueous Film-Forming Foam Contaminants in Drinking Water. *Int J Hyg Environ Health, 223*(1), 256-266. doi:10.1016/j.ijheh.2019.07.012

Beatty, A. L., Ku, I. A., Bibbins-Domingo, K., Christenson, R. H., DeFillippi, C. R., Ganz, P., . . . Whooley, M. A. (2015). Traditional risk factors versus biomarkers for prediction of secondary events in patients with stable Coronary Heart Disease: from the Heart and Soul Study. *Journal of the American Heart Association, 4*. doi:10.1161/JAHA.114.001646

Behrman, J. R., & Rosenzweig, M. R. (2004). Returns to birthweight. *Review of Economics and Statistics, 86*(2), 586-601.

Behrman, R. E., & Butler, A. S. (2007). Preterm birth: causes, consequences, and prevention. *Institute of Medicine of the National Academies*.

Black, J., Moreland, A., McNeill Ransom, M., & Sanchez, E. (2021). Perfluoroalkyl and Polyfluoroalkyl Substances: Using Law and Policy to Address These Environmental Health Hazards in the United States. *Health Matrix, 31,* 340.

Blake, B. E., Pinney, S. M., Hines, E. P., Fenton, S. E., & Ferguson, K. K. (2018). Associations between longitudinal serum perfluoroalkyl substance (PFAS) levels and measures of thyroid hormone, kidney function, and body mass index in the Fernald Community Cohort. *Environmental pollution, 242*, 894-904.

Bonefeld-Jorgensen, E. C., Long, M., Fredslund, S. O., Bossi, R., & Olsen, J. (2014). Breast cancer risk after exposure to perfluorinated compounds in Danish women: A case-control study nested in the Danish National Birth Cohort. *Cancer Causes Control, 25*(11), 1439-1448.

Boone, L., Meyer, D., Cusick, P., Ennulat, D., Bolliger, A. P., Everds, N., . . . Bounous, D. (2005). Selection and interpretation of clinical pathology indicators of hepatic injury in preclinical studies. *Veterinary clinical pathology, 34*(3), 182-188.

Boronow, K. E., Brody, J. G., Schaider, L. A., Peaslee, G. F., Havas, L., & Cohn, B. A. (2019). Serum concentrations of PFASs and exposure-related behaviors in African American and non-Hispanic white women. *Journal of exposure science & environmental epidemiology, 29*(2), 206-217.

Brown, P. (1995). Race, class, and environmental health: A review and systematization of the literature. *Environmental research, 69(1), 15–30.* doi:https://doi.org/10.1006/enrs.1995.1021

Brulle, R. J., & Pellow, D. N. (2006). Environmental justice: Human health and environmental inequalities. *Annual Review of Public Health, 27, 103–124.* doi:https://doi.org/10.1146/annurev.publhealth.27.021405.102124

Brunke, M., & Gonser, T. (1997). The ecological significance of exchange processes between rivers and groundwater. *Freshwater biology, 37*(1), 1-33.

Budtz-Jørgensen, E., & Grandjean, P. (2018). Application of benchmark analysis for mixed contaminant exposures: Mutual adjustment of perfluoroalkylate substances associated with immunotoxicity. *PLoS One, 13*(10), e0205388.

Buekers, J., Colles, A., Cornelis, C., Morrens, B., Govarts, E., & Schoeters, G. (2018). Socio-Economic Status and Health: Evaluation of Human Biomonitored Chemical Exposure to Per- and Polyfluorinated Substances across Status. *International journal of environmental research and public health, 15*(12). doi:10.3390/ijerph15122818

Butenhoff, J. L., Chang, S.-C., Ehresman, D. J., & York, R. G. (2009). Evaluation of potential reproductive and developmental toxicity of potassium perfluorohexanesulfonate in Sprague Dawley rats. *Reproductive Toxicology, 27*(3-4), 331-341.

Butenhoff, J. L., Chang, S. C., Olsen, G. W., & Thomford, P. J. (2012). Chronic dietary toxicity and carcinogenicity study with potassium perfluorooctanesulfonate in Sprague Dawley rats. *Toxicology, 293*, 1-15. doi:10.1016/j.tox.2012.01.003

Cadwallader, A., A. Greene, H. Holsinger, A. Lan, M. Messner, M. Simic, & Albert, R. (2022). A Bayesian hierarchical model for estimating national PFAS drinking water occurrence. *AWWA Water Science, e1284.*

Cakmak, S., Lukina, A., Karthikeyan, S., Atlas, E., & Dales, R. (2022). The association between blood PFAS concentrations and clinical biochemical measures of organ function and metabolism in participants of the Canadian Health Measures Survey (CHMS). *Science of the total environment, 827*, 153900.

Calafat, A. M., Kato, K., Hubbard, K., Jia, T., Botelho, J. C., & Wong, L.-Y. (2019). Legacy and alternative per- and polyfluoroalkyl substances in the U.S. general population: Paired serum-urine data from the 2013–2014 National Health and Nutrition Examination Survey. *Environment international, 131*, 105048.

Calafat, A. M., Wong, L.-Y., Kuklenyik, Z., Reidy, J. A., & Needham, L. L. (2007). 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. *Environmental health perspectives, 115*(11), 1596–1602.

California Environmental Protection Agency. (2021). *Proposed Public Health Goals for Perfluorooctanoic Acid and Perfluorooctane Sulfonic Acid in Drinking Water*.

Canova, C., Barbieri, G., Jeddi, M. Z., Gion, M., Fabricio, A., Dapra, F., . . . Pitter, G. (2020). Associations between perfluoroalkyl substances and lipid profile in a highly exposed young adult population in the Veneto Region. *Environment international, 145*, 106117.

Cantor, K. P., Lynch, C. F., Hildesheim, M. E., Dosemeci, M., Lubin, J., Alavanja, M., & Craun, G. (1998). Drinking water source and chlorination byproducts. I. Risk of bladder cancer. *Epidemiology (Cambridge, Mass.), 9*(1), 21-28. doi:http://dx.doi.org/10.1097/00001648-199801000-00007

Cantor, K. P., Villanueva, C. M., Silverman, D. T., Figueroa, J. D., Real, F. X., Garcia-Closas, M., . . . Tardon, A. (2010). Polymorphisms in GSTT1, GSTZ1, and CYP2E1, disinfection by-products, and risk of bladder cancer in Spain. *Environmental health perspectives, 118*(11), 1545-1550.

Carlsen Bach, C., Bjerregård Matthiesen, N., Olsen, J., & Brink Henriksen, T. (2018). Conditioning on parity in studies of perfluoroalkyl acids and time to pregnancy: an example from the Danish National Birth Cohort. *Environmental health perspectives, 126*(11), 117003.

Catelan, D., Biggeri, A., Russo, F., Gregori, D., Pitter, G., Da Re, F., . . . Canova, C. (2021). Exposure to perfluoroalkyl substances and mortality for COVID-19: A spatial ecological analysis in the Veneto region (Italy). *International journal of environmental research and public health, 18*(5), 2734.

Centers for Disease Control and Prevention. (2017). *User Guide to the 2017 Period/2016 Cohort Linked Birth/Infant Death Public Use File*.

Centers for Disease Control and Prevention. (2018). *User Guide to the 2018 Period/2017 Cohort Linked Birth/Infant Death Public Use File*.

Centers for Disease Control and Prevention. (2019). *Fourth National Report on Human Exposure to Environmental Chemicals: Updated Tables, January 2019, Volume One*. Retrieved from https://stacks.cdc.gov/view/cdc/75822

Centers for Disease Control and Prevention. (2020a). *National Vital Statistics System, Linked Birth / Infant Death Records 1995-2019 on CDC WONDER Online Database. Data are from the Linked Birth / Infant Deaths Records 1995-2019, as compiled from data provided by the 57 vital statistics jurisdictions through the Vital Statistics Cooperative Program.* Retrieved from: https://wonder.cdc.gov/lbd-current.html

Centers for Disease Control and Prevention. (2020b). *Underlying Cause of Death, 1999-2020 on CDC WONDER Online Database, released in 2020. Data are from the Multiple Cause of Death Files, 1999-2019, as compiled from data provided by the 57 vital statistics* *jurisdictions through the Vital Statistics Cooperative Program.* Retrieved from: http://wonder.cdc.gov/ucd-icd10.html

Chaikind, S., & Corman, H. (1991). The impact of low birthweight on special education costs. *Journal of Health Economics, 10*(3), 291-311.

Chang, C.-J., Barr, D. B., Ryan, P. B., Panuwet, P., Smarr, M. M., Liu, K., . . . Ly, V. (2022). Per-and polyfluoroalkyl substance (PFAS) exposure, maternal metabolomic perturbation, and fetal growth in African American women: a meet-in-the-middle approach. *Environment international, 158*, 106964.

Château-Degat, M.-L., Pereg, D., Dallaire, R., Ayotte, P., Dery, S., & Dewailly, É. (2010). Effects of perfluorooctanesulfonate exposure on plasma lipid levels in the Inuit population of Nunavik (Northern Quebec). *Environmental research, 110*(7), 710-717.

Chatterji, P., Kim, D., & Lahiri, K. (2014). Birth weight and academic achievement in childhood. *Health Economics, 23*(9), 1013-1035.

Chen, A., Jandarov, R., Zhou, L., Calafat, A. M., Zhang, G., Urbina, E. M., . . . Bockor, L. (2019). Association of perfluoroalkyl substances exposure with cardiometabolic traits in an island population of the eastern Adriatic coast of Croatia. *Science of the total environment, 683*, 29-36.

Chen, C. W., & Gibb, H. (2003). Procedures for calculating cessation lag. *Regulatory Toxicology and Pharmacology, 38*(2), 157-165.

Chen, M. H., Ng, S., Hsieh, C. J., Lin, C. C., Hsieh, W. S., & Chen, P. C. (2017). The impact of prenatal perfluoroalkyl substances exposure on neonatal and child growth. *Science of the total environment, 607-608*, 669-675. doi:10.1016/j.scitotenv.2017.06.273

Cheng, W., Dastgheib, S. A., & Karanfil, T. (2005). Adsorption of dissolved natural organic matter by modified activated carbons. *Water research, 39*(11), 2281-2290.

Chili, C. A., Westerhoff, P., & Ghosh, A. (2012). GAC removal of organic nitrogen and other DBP precursors. *Journal-American Water Works Association, 104*(7), E406-E415.

Chin, D. A., & Qi, X. (2000). Ground water under direct influence of surface water. *Journal of Environmental Engineering, 126*(6), 501-508.

Chowdhury, Z. Z., Zain, S. M., Rashid, A. K., Rafique, R. F., & Khalid, K. (2013). Breakthrough Curve Analysis for Column Dynamics Sorption of Mn(II) Ions from Wastewater by Using <i>Mangostana garcinia</i> Peel-Based Granular-Activated Carbon. *Journal of Chemistry, 2013*, 959761. doi:10.1155/2013/959761

Christensen, K. Y., Raymond, M., & Meiman, J. (2018). Perfluoralkyl substances and metabolic syndrome. *International journal of hygiene and environmental health, 222*(1), 147-153. doi:10.1016/j.ijheh.2018.08.014

Christensen, K. Y., Raymond, M., Thompson, B., & Anderson, H. (2016). Perfluoroalkyl substances in older male anglers in Wisconsin. *Environment international, 91*, 312-318.

Chu, C., Zhou, Y., Li, Q.-Q., Bloom, M. S., Lin, S., Yu, Y.-J., . . . Yang, B.-Y. (2020). Are perfluorooctane sulfonate alternatives safer? New insights from a birth cohort study. *Environment international, 135*, 105365.

Colaizy, T. T., Bartick, M. C., Jegier, B. J., Green, B. D., Reinhold, A. G., Schaefer, A. J., . . . Jobe, A. H. (2016). Impact of optimized breastfeeding on the costs of necrotizing enterocolitis in extremely low birthweight infants. *The Journal of Pediatrics, 175*, 100-105. e102.

Colantonio, L. D. M., PhD; Rosenson, Robert S. MD; Deng, Luqin PhD; Monda, Keri L. PhD; Dai, Yuling MS, MSPH; Farkouh, Michael E. MD, MSc; Safford, Monika M. MD;

Philip, Kiran MD, MBA; Mues, Katherine E. PhD, MPH; Muntner, Paul PhD. (2019). Adherence to Statin Therapy Among US Adults Between 2007 and 2014. *Journal of the American Heart Association, 8*(1). doi:https://doi.org/10.1161/JAHA.118.010376

Collier, S. A., Deng, L., Adam, E. A., Benedict, K. M., Beshearse, E. M., Blackstock, A. J., . . . Fullerton, K. E. (2021). Estimate of burden and direct healthcare cost of infectious waterborne disease in the United States. *Emerging infectious diseases, 27*(1), 140.

Costello, E., Rock, S., Stratakis, N., Eckel, S. P., Walker, D. I., Valvi, D., . . . Kohli, R. (2022). Exposure to per-and polyfluoroalkyl substances and markers of liver injury: a systematic review and meta-analysis. *Environmental health perspectives, 130*(4), 046001.

Costet, N., Villanueva, C. M., Jaakkola, J. J. K., Kogevinas, M., Cantor, K. P., King, W. D., . . . Cordier, S. (2011). Water disinfection by-products and bladder cancer: is there a European specificity? A pooled and meta-analysis of European case–control studies. *Occupational and environmental medicine, 68*(5), 379-385.

Crittenden, J. C., Vaitheeswaran, K., Hand, D. W., Howe, E. W., Aieta, E. M., Tate, C. H., . . . Davis, M. K. (1993). Removal of dissolved organic carbon using granular activated carbon. *Water research, 27*(4), 715-721. doi:https://doi.org/10.1016/0043-1354(93)90181-G

Cropper, M. L., & Krupnick, A. J. (2000). *Social costs of chronic heart and lung disease*.

Cuthbertson, A. A., Kimura, S. Y., Liberatore, H. K., Summers, R. S., Knappe, D. R., Stanford, B. D., . . . Richardson, S. D. (2019). Does granular activated carbon with chlorination produce safer drinking water? From disinfection byproducts and total organic halogen to calculated toxicity. *Environmental Science & Technology, 53*(10), 5987-5999.

D'Agostino, R. B., Vasan, R. S., Pencina, M. J., Wolf, P. A., Cobain, M., Massaro, J. M., & Kannel, W. B. (2008). General cardiovascular risk profile for use in primary care. *Circulation, 117*(6), 743-753.

Dalla Zuanna, T., Savitz, D. A., Barbieri, G., Pitter, G., Jeddi, M. Z., Daprà, F., . . . Canova, C. (2021). The association between perfluoroalkyl substances and lipid profile in exposed pregnant women in the Veneto region, Italy. *Ecotoxicology and environmental safety, 209*, 111805.

Darrow, L. A., Stein, C. R., & Steenland, K. (2013). Serum Perfluorooctanoic Acid and Perfluorooctane Sulfonate Concentrations in Relation to Birth Outcomes in the Mid-Ohio Valley, 2005–2010. *Environ. Health Perspect., 121*(10), 1207-1213. doi:10.1289/ehp.1206372

Dastgheib, S. A., Karanfil, T., & Cheng, W. (2004). Tailoring activated carbons for enhanced removal of natural organic matter from natural waters. *Carbon, 42*(3), 547-557.

de Abreu Domingos, R., & da Fonseca, F. V. (2018). Evaluation of adsorbent and ion exchange resins for removal of organic matter from petroleum refinery wastewaters aiming to increase water reuse. *Journal of environmental management, 214*, 362-369.

Deeks, J. J. (2002). Issues in the selection of a summary statistic for meta-analysis of clinical trials with binary outcomes. *Statistics in medicine, 21*(11), 1575-1600.

Desikan, A., Carter, J., Kinser, S., & Goldman, G. (2019). Abandoned Science, Broken Promises: How the Trump Administration's Neglect of Science Is Leaving Marginalized Communities Further Behind.

Dickenson, E., & Higgins, C. (2016). Treatment Mitigation Strategies for Poly- and Perfluorinated Chemicals. *Water Research Foundation*.

Dobson, K. G., Ferro, M. A., Boyle, M. H., Schmidt, L. A., Saigal, S., & Van Lieshout, R. J. (2018). How do childhood intelligence and early psychosocial adversity influence income attainment among adult extremely low birth weight survivors? A test of the cognitive reserve hypothesis. *Development and psychopathology*, *30*(4), 1421-1434.

Domingo, J. L., & Nadal, M. (2019). Human exposure to per-and polyfluoroalkyl substances (PFAS) through drinking water: A review of the recent scientific literature. *Environmental research, 177*, 108648.

Dong, Z., Wang, H., Yu, Y. Y., Li, Y. B., Naidu, R., & Liu, Y. (2019). Using 2003–2014 US NHANES data to determine the associations between per-and polyfluoroalkyl substances and cholesterol: Trend and implications. *Ecotoxicology and environmental safety, 173*, 461-468.

Driscoll, A. K., & Gregory, E. C. W. (2021). Prepregnancy Body Mass Index and Infant Outcomes by Race and Hispanic Origin: United States, 2020. *Natl Vital Stat Rep, 70*(16), 1-8.

Du, Z., Deng, S., Bei, Y., Huang, Q., Wang, B., Huang, J., & Yu, G. 2014. . (2014). Adsorption behavior and mechanism of perfluorinated compounds on various adsorbents—A review. *Journal of hazardous materials, 274*, 443-454.

Dunder, L., Lind, P. M., Salihovic, S., Stubleski, J., Kärrman, A., & Lind, L. (2022). Changes in plasma levels of per-and polyfluoroalkyl substances (PFAS) are associated with changes in plasma lipids-A longitudinal study over 10 years. *Environmental research, 211*, 112903.

Dzierlenga, M., Crawford, L., & Longnecker, M. (2020). Birth weight and perfluorooctane sulfonic acid: a random-effects meta-regression analysis. *Environmental Epidemiology, 4*(3).

Eick, S. M., Enright, E. A., Geiger, S. D., Dzwilewski, K. L., DeMicco, E., Smith, S., . . . Morello-Frosch, R. (2021). Associations of maternal stress, prenatal exposure to per-and polyfluoroalkyl substances (PFAS), and demographic risk factors with birth outcomes and offspring neurodevelopment: an overview of the ECHO. CA. IL prospective birth cohorts. *International journal of environmental research and public health, 18*(2), 742.

Elder, T., Figlio, D., Imberman, S., & Persico, C. (2020). The role of neonatal health in the incidence of childhood disability. *American Journal of Health Economics, 6*(2), 216-250.

Electricwala, B., Wassel, C. L., Amari, D. T., & Cristino, J. (2020). PCV73 Risk Factors Associated with Statin Use Among Adult Nhanes Participants with Ascvd. *Value in Health, 23,* S499. doi:10.1016/j.jval.2020.08.564

Ellington, S., Strid, P., Tong, V. T., Woodworth, K., Galang, R. R., Zambrano, L. D., . . . Gilboa, S. M. (2020). Characteristics of women of reproductive age with laboratory-confirmed SARS-CoV-2 infection by pregnancy status—United States, January 22–June 7, 2020. *Morbidity and Mortality Weekly Report*, *69*(25), 769.

Ellis, J. J., Erickson, S. R., Stevenson, J. G., Bernstein, S. J., Stiles, R. A., & Fendrick, A. M. (2004). Suboptimal statin adherence and discontinuation in primary and secondary prevention populations. *Journal of General Internal Medicine, 19*(6), 638-645. doi:10.1111/j.1525-1497.2004.30516.x

Engels, E. A., Schmid, C. H., Terrin, N., Olkin, I., & Lau, J. (2000). Heterogeneity and statistical significance in meta-analysis: an empirical study of 125 meta-analyses. *Statistics in medicine, 19*(13), 1707-1728.

Environmental Council of the States. (2022). Processes and Considerations for Setting State PFAS Standards.

Eschauzier, C., Beerendonk, E., Scholte-Veenendaal, P., & De Voogt, P. (2012). Impact of treatment processes on the removal of perfluoroalkyl acids from the drinking water production chain. *Environmental Science & Technology, 46*(3), 1708-1715.

Executive Order 12866. 1993. Regulatory Planning and Review. 58 FR 51735, October 4, 1993. Available at https://www.reginfo.gov/public/jsp/Utilities/EO_12866.pdf, (1993).

Executive Order 12898. 1994. Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations. 59 FR 7629; February 16, 1994. Available at https://www.archives.gov/files/federal-register/executive-orders/pdf/12898.pdf, (1994).

Executive Order 13045. 1997. Protection of Children from Environmental Health Risks and Safety Risks. 62 FR 19885, April 23, 1997. Available at https://www.gpo.gov/fdsys/pkg/FR-1997-04-23/pdf/97-10695.pdf, (1997).

Executive Order 13132. 1999. Federalism. 64 FR 43255, August 10, 1999. Available at https://www.gpo.gov/fdsys/pkg/FR-1999-08-10/pdf/99-20729.pdf, (1999).

Executive Order 13175. 2000. Consultation and Coordination with Indian Tribal Governments. 65 FR 67249, November 9, 2000. Available at https://www.gpo.gov/fdsys/pkg/FR-2000-11-09/pdf/00-29003.pdf, (2000).

Executive Order 13211. 2001. Actions Concerning Regulations That Significantly Affect Energy Supply, Distribution, or Use. 66 FR 28355, May 22, 2001. Available at https://www.gpo.gov/fdsys/pkg/FR-2001-05-22/pdf/01-13116.pdf, (2001).

Executive Order 13563. 2011. Improving Regulation and Regulatory Review. 76 FR 3821, January 21, 2011. Available at https://www.gpo.gov/fdsys/pkg/FR-2011-01-21/pdf/2011-1385.pdf, (2011).

Executive Order 14096. 2023. Revitalizing Our Nation’s Commitment to Environmental Justice for All. Federal Register. 88 FR 25251. Available at https://www.whitehouse.gov/briefing-room/presidential-actions/2023/04/21/executive-order-on-revitalizing-our-nations-commitment-to-environmental-justice-for-all/, (2023).

Fan, Y., Li, X., Xu, Q., Zhang, Y., Yang, X., Han, X., . . . Lu, C. (2020). Serum albumin mediates the effect of multiple per-and polyfluoroalkyl substances on serum lipid levels. *Environmental pollution, 266*, 115138.

Fei, C., McLaughlin, J. K., Lipworth, L., & Olsen, J. (2009). Maternal levels of perfluorinated chemicals and subfecundity. *Human reproduction, 24*(5), 1200-1205. doi:10.1093/humrep/den490

Fisher, M., Arbuckle, T. E., Wade, M., & Haines, D. A. (2013). Do perfluoroalkyl substances affect metabolic function and plasma lipids?—Analysis of the 2007–2009, Canadian Health Measures Survey (CHMS) Cycle 1. *Environmental research, 121*, 95-103.

Foreman, K., Vacs Renwick, D., McCabe, M., Cadwallader, A., Holsinger, H., Kormondy, C., & Albert, R. (2021). Effects of harmful algal blooms on regulated disinfection byproducts: Findings from five utility case studies. *AWWA Water Science, 3*(3), e1223.

Frawley, R. P., Smith, M., Cesta, M. F., Hayes-Bouknight, S., Blystone, C., Kissling, G. E., . . . Germolec, D. (2018). Immunotoxic and hepatotoxic effects of perfluoro-n-decanoic acid (PFDA) on female Harlan Sprague–Dawley rats and B6C3F1/N mice when administered by oral gavage for 28 days. *Journal of Immunotoxicology, 15*(1), 41-52.

Freeman, L. E., Cantor, K. P., Baris, D., Nuckols, J. R., Johnson, A., Colt, J. S., . . . Silverman, D. T. (2017). Bladder Cancer and Water Disinfection By-product Exposures through

Multiple Routes: A Population-Based Case-Control Study (New England, USA). *Environmental health perspectives, 125*(6).

Fried, L. P., Borhani, N. O., Enright, P., Furberg, C. D., Gardin, J. M., Kronmal, R. A., . . . Newman, A. (1991). The cardiovascular health study: design and rationale. *Annals of epidemiology, 1*(3), 263-276.

Friedman, G. D., Cutter, G. R., Donahue, R. P., Hughes, G. H., Hulley, S. B., Jacobs Jr, D. R., . . . Savage, P. J. (1988). CARDIA: study design, recruitment, and some characteristics of the examined subjects. *Journal of clinical epidemiology, 41*(11), 1105-1116.

Frisbee, S. J., Shankar, A., Knox, S. S., Steenland, K., Savitz, D. A., Fletcher, T., & Ducatman, A. M. (2010). Perfluorooctanoic acid, perfluorooctanesulfonate, and serum lipids in children and adolescents: results from the C8 Health Project. *Archives of pediatrics & adolescent medicine, 164*(9), 860-869.

Fromme, H., Tittlemier, S. A., Völkel, W., Wilhelm, M., & Twardella, D. (2009). Perfluorinated compounds–exposure assessment for the general population in Western countries. *International journal of hygiene and environmental health, 212*(3), 239–270.

Fry, K., & Power, M. C. (2017). Persistent organic pollutants and mortalityin the United States, NHANES 1999–2011. *Environmental Health, 16*(105), 1-12. doi:10.1186/s12940-017-0313-6

Fryar, C. D., Ostchega, Y., Hales, C. M., Zhang, G., & Kruszon-Moran, D. (2017). Hypertension Prevalence and Control Among Adults: United States, 2015-2016. *NCHS Data Brief*(289), 1-8.

Fu, Y., Wang, T., Fu, Q., Wang, P., & Lu, Y. (2014). Associations between serum concentrations of perfluoroalkyl acids and serum lipid levels in a Chinese population. *Ecotoxicology and environmental safety, 106*, 246-252.

Ghisari, M., Long, M., Røge, D. M., Olsen, J., & Bonefeld-Jørgensen, E. C. (2017). Polymorphism in xenobiotic and estrogen metabolizing genes, exposure to perfluorinated compounds and subsequent breast cancer risk: A nested case-control study in the Danish National Birth Cohort. *Environmental research, 154*, 325–333. doi:10.1016/j.envres.2017.01.020

Gleason, J. A., Post, G. B., & Fagliano, J. A. (2015). Associations of perfluorinated chemical serum concentrations and biomarkers of liver function and uric acid in the US population (NHANES), 2007–2010. *Environmental research, 136*, 8-14.

Glynn, A., Berger, U., Bignert, A., Ullah, S., Aune, M., Lignell, S., & Darnerud, P. O. (2012). Perfluorinated Alkyl Acids in Blood Serum from Primiparous Women in Sweden: Serial Sampling during Pregnancy and Nursing, And Temporal Trends 1996–2010. *Environmental Science & Technology, 46*(16), 9071-9079. doi:10.1021/es301168c

Goff, D. C., Lloyd-Jones, D. M., Bennett, G., Coady, S., D'Agostino, R. B., Gibbons, R., . . . Wilson, P. W. F. (2014). 2013 ACC/AHA guideline on the assessment of cardiovascular risk: A report of the American College of Cardiology/American Heart Association task force on practice guidelines. *Circulation, 129*(25 suppl 2), 49-73.

Goldstein, K. M., Zullig, L. L., Bastian, L. A., & Bosworth, H. B. (2016). Statin Adherence: Does Gender Matter? *Current Atherosclerosis Reports, 18*(11), 63. doi:10.1007/s11883-016-0619-9

Govarts, E., Iszatt, N., Trnovec, T., De Cock, M., Eggesbø, M., Murinova, L. P., . . . Koppen, G. (2018). Prenatal exposure to endocrine disrupting chemicals and risk of being born small for gestational age: Pooled analysisof seven European birth cohorts. *Environment International, 115*, 267-278. doi:10.1016/j.envint.2018.03.01

Govarts, E., Remy, S., Bruckners, L., Hond, E. D., Sioen, I., Nelen, V., . . . Schoeters, G. (2016). Combined Effects of Prenatal Exposures to Environmental Chemicals on Birth Weight. *International journal of environmental research and public health, 13*(495), 1-19. doi:10.3390/ijerph13050495

Graham, G. (2015). Disparities in cardiovascular disease risk in the United States. *Current cardiology reviews, 11*(3), 238-245.

Grandjean, P., Andersen, E. W., Budtz-Jørgensen, E., Nielsen, F., Molbank, K., Weihe, P., & Heilmann, C. (2012). Serum Vaccine Antibody Concentrationsin Children Exposed to Perfluorinated Compounds. *Jama, 307*(4), 391-397.

Grandjean, P., Heilmann, C., Nielsen, F., Mogensen, U. B., Timmermann, A. G., & Budtz-Jørgensen, E. (2017). Estimated exposures to perfluorinated compounds in infancy predict attenuated vaccine antibody concentrations at age 5-years. *Journal of Immunotoxicolgy, 14*(1), 188-195. doi:10.1080/1547691X.2017.1360968

Grandjean, P., Heilmann, C., Weihe, P., Nielsen, F., Mogensen, U. B., & Budtz-Jørgensen, E. (2017). Serum Vaccine Antibody Concentrations in Adolescents Exposed to PerfluorinatedCompounds. *Environmental health perspectives*. doi:10.1289/EHP275

Grandjean, P., Timmermann, C. A. G., Kruse, M., Nielsen, F., Vinholt, P., Boding, L., . . . Mølbak, K. (2020). Severity of COVID-19 at elevated exposure to perfluorinated alkylates. *PLoS One, 15*(12), e0244815.

Greco, S. L., Belova, A., Haskell, J., & Backer, L. (2019). Estimated burden of disease from arsenic in drinking water supplied by domestic wells in the United States. *Journal of water and health, 17*(5), 801-812.

Guelfo, J. L., & Adamson, D. T. (2018). Evaluation of a National Data set for Insights into Sources, Composition, and Concentrations of Per- and Polyfluoroalkyl Substances (PFASs) in U.S. Drinking Water. *Environmental pollution, 236*, 505-513. doi:https://doi.org/10.1016/j.envpol.2018.01.066

Hardell, E., Karrman, A., van Bavel, B., Bao, J., Carlberg, M., & Hardell, L. (2014). Case–control study on perfluorinated alkyl acids (PFAAs) and the risk of prostate cancer. *Environment international, 63*, 35-39. doi:10.1016/j.envint.2013.10.005

Hartge, P., Silverman, D., Hoover, R., Schairer, C., Altman, R., Austin, D., . . . Marrett, L. D. (1987). Changing cigarette habits and bladder cancer risk: a case-control study. *Journal of the National Cancer Institute, 78*(6), 1119-1125.

Hashempour, Y., Nasseri, M., Mohseni-Bandpei, A., Motesaddi, S., & Eslamizadeh, M. (2020). Assessing vulnerability to climate change for total organic carbon in a system of drinking water supply. *Sustainable Cities and Society, 53*, 101904. doi:https://doi.org/10.1016/j.scs.2019.101904

He, X., Liu, Y., Xu, B., Gu, L., & Tang, W. (2018). PFOA is associated with diabetes and metabolic alteration in US men: National Health and Nutrition Examination Survey 2003–2012. *Science of the total environment, 625*, 566-574.

Hines, C. T., Padilla, C. M., & Ryan, R. M. (2020). The effect of birth weight on child development prior to school entry. *Child development, 91*(3), 724-732.

Hooper, S. M., & Allgeier, S. C. (2002). GAC and membrane treatment studies Chapter 21 in Information Collection Rule Data Analysis. *Water Research Foundation and AWWA.*

Hrubec, Z., & McLaughlin, J. K. (1997). Former cigarette smoking and mortality among US veterans: a 26-year follow-up, 1954-1980. *Changes in cigarette-related disease risks and their implication for prevention and control. Bethesda, MD: US Government Printing Office*, 501-530.

Hrudey, S. E., Backer, L. C., Humpage, A. R., Krasner, S. W., Michaud, D. S., Moore, L. E., . . . Stanford, B. D. (2015). Evaluating evidence for association of human bladder cancer with drinking-water chlorination disinfection by-products. *Journal of Toxicology and Environmental Health, Part B, 18*(5), 213-241.

Hu, X. C., Andrews, D. Q., Lindstrom, A. B., Bruton, T. A., Schaider, L. A., Grandjean, P., . . . Sunderland, E. M. (2016). Detection of Poly- and Perfluoroalkyl Substances (PFASs) in U.S. Drinking Water Linked to Industrial Sites, Military Fire Training Areas, and Wastewater Treatment Plants. *Environmental Science & Technology Letters, 3*(10), 344-350. doi:10.1021/acs.estlett.6b00260

Hu, Z., Morton, L. W., & Mahler, R. L. (2011). Bottled water: United States consumers and their perceptions of water quality. *International journal of environmental research and public health, 8*(2), 565-578. doi:10.3390/ijerph8020565

Hua, G., & Reckhow, D. A. (2008). DBP formation during chlorination and chloramination: effect of reaction time, pH, dosage, and temperature. *Journal-American Water Works Association, 100*(8), 82-95.

Huang, M., Jiao, J., Zhuang, P., Chen, X., Wang, J., & Zhang, Y. (2018). Serum polyfluoroalkyl chemicals are associated with risk of cardiovascular diseases in national US population. *Environment international, 119*, 37-46.

Hutcheson, R., Innes, K., & Conway, B. (2019). Perfluoroalkyl substances and likelihood of stroke in persons with and without diabetes. *Diabetes and Vascular Disease Research, 17*(1), 1479164119892223. doi:10.1177/1479164119892223

ICF. (2020). *Memorandum re: Trends in Infant Mortality by Birth Weight Categories, EPA Contract No. 68HE0C18D0001, TO 68HERC20F0107. Submitted to U.S. Environmental Protection Agency Office of Groundwater and Drinking Water July 30, 2020*. Retrieved from https://www.regulations.gov/docket/EPA-HQ-OW-2022-0114

ICF. (2021). *Memorandum re: Summary of Literature Review of Non-Medical Effects and Economic Burden Related to Low Birth Weight Infants. Submitted to U.S. Environmental Protection Agency Office of Groundwater and Drinking Water October 5, 2021*. Retrieved from https://www.regulations.gov/docket/EPA-HQ-OW-2022-0114

ICF. (2022a). *Memorandum re: Literature Review of ASCVD Risk Models and Limitations. Submitted to U.S. Environmental Protection Agency Office of Groundwater and Drinking Water April 4, 2022*. Retrieved from https://www.regulations.gov/docket/EPA-HQ-OW-2022-0114

ICF. (2022b). *Memorandum re: Literature Review of Population Attributable Fraction Estimates and Next Steps for Renal Cell Carcinoma PFAS Benefits Modeling, EPA Contract No. 68HE0C18D0001, TO 68HERC22F0262. Submitted to U.S. Environmental Protection Agency Office of Groundwater and Drinking Water July 28, 2022*. Retrieved from https://www.regulations.gov/docket/EPA-HQ-OW-2022-0114

Ioannou, G. N., Boyko, E. J., & Lee, S. P. (2006). The prevalence and predictors of elevated serum aminotransferase activity in the United States in 1999–2002. *Official journal of the American College of Gastroenterology| ACG, 101*(1), 76-82.

Ioannou, G. N., Weiss, N. S., Boyko, E. J., Mozaffarian, D., & Lee, S. P. (2006). Elevated serum alanine aminotransferase activity and calculated risk of coronary heart disease in the United States. *Hepatology, 43*(5), 1145-1151.

Iriarte-Velasco, U., I. Álvarez-Uriarte, J., Chimeno-Alanis, N., & R. González-Velasco, J. (2008). Natural organic matter adsorption onto granular activated carbons: implications in the molecular weight and disinfection byproducts formation. *Industrial & engineering chemistry research, 47*(20), 7868-7876.

Jacob, J. A. (2016). U.S. infant mortality rate declines but still exceeds other developed countries. *Journal of the American Medical Association, 315*(5), 451-452.

Jain, R. B. (2018). Time trends over 2003–2014 in the concentrations of selected perfluoroalkyl substances among U.S. adults aged >20 years: Interpretational issues. *Science of the total environment, 645*, 946–957. doi:10.1016/j.scitotenv.2018.07.198

Jain, R. B., & Ducatman, A. (2019a). Roles of gender and obesity in defining correlations between perfluoroalkyl substances and lipid/lipoproteins. *Science of the total environment, 653*, 74-81.

Jain, R. B., & Ducatman, A. (2019b). Selective associations of recent low concentrations of perfluoroalkyl substances with liver function biomarkers: NHANES 2011 to 2014 data on US adults aged≥ 20 years. *Journal of occupational and environmental medicine, 61*(4), 293-302.

Jelenkovic, A., Mikkonen, J., Martikainen, P., Latvala, A., Yokoyama, Y., Sund, R., . . . Kim, J. (2018). Association between birth weight and educational attainment: an individual-based pooled analysis of nine twin cohorts. *J Epidemiol Community Health, 72*(9), 832-837.

Ji, J., Song, L., Wang, J., Yang, Z., Yan, H., Li, T., . . . Li, J. (2021). Association between urinary per-and poly-fluoroalkyl substances and COVID-19 susceptibility. *Environment international, 153*, 106524.

Jiang, J., Zhang, X., Zhu, X., & Li, Y. (2017). Removal of intermediate aromatic halogenated DBPs by activated carbon adsorption: a new approach to controlling halogenated DBPs in chlorinated drinking water. *Environmental Science & Technology, 51*(6), 3435-3444.

Johnson, P. I., Sutton, P., Atchley, D. S., Koustas, E., Lam, J., Sen, S., . . . Woodruff, T. J. (2014). The Navigation Guide—Evidence-Based Medicine Meets Environmental Health: Systematic Review of Human Evidence for PFOA Effects on Fetal Growth. *Environmental health perspectives, 122*(10), 1028-1039. doi:10.1289/ehp.1307893

Johnston, J., & Cushing, L. (2020). Chemical Exposures, Health, and Environmental Justice in Communities Living on the Fenceline of Industry. *Current Environmental Health Reports, 7*(1), 48-57. doi:10.1007/s40572-020-00263-8

Joyce, C., Goodman-Bryan, M., & Hardin, A. (2012). *Preterm Birth and Low Birth Weight*. Retrieved from The Urban Child Institute: http://www.urbanchildinstitute.org/sites/all/files/2010-10-01-PTB-and-LBW.pdf

Kamai, E. M., McElrath, T. F., & Ferguson, K. K. (2019). Fetal growth in environmental epidemiology: mechanisms, limitations, and a review of associations with biomarkers of non-persistent chemical exposures during pregnancy. *Environmental Health, 18*(1), 43.

Kato, K., Wong, L.-Y., Chen, A., Dunbar, C., Webster, G. M., Lanphear, B. P., & Calafat, A. M. (2014). Changes in serum concentrations of maternal poly-and perfluoroalkyl substances over the course of pregnancy and predictors of exposure in a multiethnic cohort of Cincinnati, Ohio pregnant women during 2003–2006. *Environmental Science & Technology, 48*(16), 9600-9608.

Kempisty, D., Arevalo, E., Spinelli, A., Edeback, V., Dickenson, E., Husted, C., . . . Knappe, D. (2022). Granular activated carbon adsorption of perfluoroalkyl acids from ground and surface water. *AWWA Water Science, 4(1)e1269*. doi:https://doi.org/10.1002/aws2.1269

Khalil, N., Chen, A., Lee, M., Czerwinski, S., Ebert, J. R., DeWitt, J., & Kannan, K. (2016). Association of perfluoroalkyl substances, bone mineral density, and osteoporosis in the US population in NHANES 2009–2010. *Environmental health perspectives, 124*(1), 81-87.

Khalil, N., Ebert, J. R., Honda, M., Lee, M., Nahhas, R. W., Koskela, A., . . . Kannan, K. (2018). Perfluoroalkyl substances, bone density, and cardio-metabolic risk factors in obese 8–12 year old children: A pilot study. *Environmental research, 160*, 314-321. doi:10.1016/j.envres.2017.10.014

Khera, R., Ransom, P., & Speth, T. F. (2013). Using work breakdown structure models to develop unit treatment costs. *Journal-American Water Works Association, 105*(11), E628-E641.

Kim, W. H., Nishijima, W., Shoto, E., & Okada, M. (1997). Competitive removal of dissolved organic carbon by adsorption and biodegradation on biological activated carbon. *Water Science and Technology, 35*(7), 147-153. doi:https://doi.org/10.1016/S0273-1223(97)00125-X

Kim, W. R., Flamm, S. L., Di Bisceglie, A. M., & Bodenheimer, H. C. (2008). Serum activity of alanine aminotransferase (ALT) as an indicator of health and disease. *Hepatology, 47*(4), 1363-1370.

King, W. D., & Marrett, L. D. (1996). Case-control study of bladder cancer and chlorination by-products in treated water (Ontario, Canada). *Cancer causes & control: CCC, 7*(6), 596–604. doi:https://doi.org/10.1007/BF00051702

Kingsley, S. L., Eliot, M. N., Kelsey, K. T., Calafat, A. M., Ehrlich, S., Lanphear, B. P., . . . Braun, J. M. (2018). Variability and predictors of serum perfluoroalkyl substance concentrations during pregnancy and early childhood. *Environmental research, 165*, 247-257. doi:10.1016/j.envres.2018.04.033

Kirchberger, I., Burkhardt, K., Heier, M., Thilo, C., & Meisinger, C. (2020). Resilience is strongly associated with health-related quality of life but does not buffer work-related stress in employed persons 1 year after acute myocardial infarction. *Quality of Life Research, 29*(2), 391-401. doi:10.1007/s11136-019-02306-6

Kirisits, M. J., Snoeyink, V. L., & Kruithof, J. C. (2000). The reduction of bromate by granular activated carbon. *Water research, 34*(17), 4250-4260.

Klein, R., & Lynch, M. (2018). *Development of Medical Cost Estimates for Adverse Birth Outcomes*. Prepared for U.S. EPA National Center for Environmental Economics

Kotlarz, N., McCord, J., Collier, D., Lea, C. S., Strynar, M., Lindstrom, A. B., . . . Hoppin, J. A. (2020). Measurement of Novel, Drinking Water-Associated PFAS in Blood from Adults and Children in Wilmington, North Carolina. *Environ Health Perspect, 128*(7), 77005. doi:10.1289/ehp6837

Kowlessar, N. M., Jiang, H. J., & Steiner, C. (2013). Hospital stays for newborns, 2011: statistical brief# 163.

Kwo, P. Y., Cohen, S. M., & Lim, J. K. (2017). ACG clinical guideline: evaluation of abnormal liver chemistries. *Official journal of the American College of Gastroenterology| ACG, 112*(1), 18-35.

Lau, C., Ambalavanan, N., Chakraborty, H., Wingate, M. S., & Carlo, W. A. (2013). Extremely low birth weight and infant mortality rates in the United States. *Pediatrics, 131*(5), 855-860.

Lauritzen, H. B., Larose, T. L., Øien, T., Sandanger, T. M., Odland, J. O., van der Bor, M., & Jacobsen, G. W. (2017). Maternal serum levels of perfluoroalkyl substances and organochlorines and indices of fetal growth: a scandinavian case–cohort study. *Pediatric research, 81*(1), 33-42. doi:10.1038/pr.2016.187

LeChevallier, M. W. (2020). Managing Legionella pneumophila in Water Systems. *Journal: American Water Works Association, 112*(2).

Lee, S., Kang, D., Kim, Y., Kim, Y. J., & Kim, S. Y. (2021). Activity-Based Exposure Levels and Cancer Risk Assessment Due to Naturally Occurring Asbestos for the Residents Near Abandoned Asbestos Mines in South Korea. *Int J Environ Res Public Health, 18*(10). doi:10.3390/ijerph18105225

Lee, S., Kar, A., & Reade, A. (2021). *Dirty Water: Toxic "Forever" PFAS Chemicals Are Prevalent in the Drinking Water of Environmental Justice Communities*. Retrieved from https://www.nrdc.org/sites/default/files/dirty-water-pfas-ej-communities-report.pdf.

Leigh, A., McEvoy, J. W., Garg, P., Carr, J. J., Sandfort, V., Oelsner, E. C., . . . Yeboah, J. (2019). Coronary artery calcium scores and atherosclerotic cardiovascular disease risk stratification in smokers. *JACC: Cardiovascular Imaging, 12*(5), 852-861.

Leino, A. D., Dorsch, M. P., & Lester, C. A. (2020). Changes in Statin Use Among U.S. Adults With Diabetes: A Population-Based Analysis of NHANES 2011-2018. *Diabetes Care, 43*(12), 3110-3112. doi:10.2337/dc20-1481

Lenters, V., Portengen, L., Rignell-Hydbom, A., Jonsson, B. A. G., Lindh, C. H., Piersma, A. H., . . . Vermeulen, R. (2016). Prenatal Phthalate, Perfluoroalkyl Acid, and Organochlorine Exposures and Term Birth Weight in Three Birth Cohorts: Multi-Pollutant Models Based on Elastic Net Regression. *Environmental health perspectives, 124,* 365-372. doi:10.1289/ehp.1408933

Lewey, J., Shrank, W. H., Bowry, A. D. K., Kilabuk, E., Brennan, T. A., & Choudhry, N. K. (2013). Gender and racial disparities in adherence to statin therapy: A meta-analysis. *American Heart Journal, 165*(5), 665-678. doi:https://doi.org/10.1016/j.ahj.2013.02.011

Li, N., Liu, Y., Papandonatos, G. D., Calafat, A. M., Eaton, C. B., Kelsey, K. T., . . . Lanphear, B. P. (2021). Gestational and childhood exposure to per-and polyfluoroalkyl substances and cardiometabolic risk at age 12 years. *Environment international, 147*, 106344.

Li, S., Peng, Y., Wang, X., Qian, Y., Xiang, P., Wade, S. W., . . . Handelsman, Y. (2019). Cardiovascular events and death after myocardial infarction or ischemic stroke in an older Medicare population. *Clinical cardiology, 42*(3), 391-399.

Li, Y., Barregard, L., Xu, Y., Scott, K., Pineda, D., Lindh, C. H., . . . Fletcher, T. (2020). Associations between perfluoroalkyl substances and serum lipids in a Swedish adult population with contaminated drinking water. *Environmental Health, 19*(1), 1-11.

Li, Y., Fletcher, T., Mucs, D., Scott, K., Lindh, C. H., Tallving, P., & Jakobsson, K. (2018). Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water. *Occupational and environmental medicine, 75*(1), 46-51.

Liao, S., Yao, W., Cheang, I., Tang, X., Yin, T., Lu, X., . . . Li, X. (2020). Association between perfluoroalkyl acids and the prevalence of hypertension among US adults. *Ecotoxicology and environmental safety, 196*, 110589.

Liddie, J. M., Schaider, L. A., & Sunderland, E. M. (2023). Sociodemographic Factors Are Associated with the Abundance of PFAS Sources and Detection in U.S. Community Water Systems. *Environmental Science & Technology, 57(21)*, 7902–7912.

Liew, Z., Olsen, J., Cui, X., Ritz, B., & Arah, O. A. (2015). Bias from conditioning on live birth in pregnancy cohorts: an illustration based on neurodevelopment in children after prenatal exposure to organic pollutants. *International Journal of Epidemiology, 44*(1), 345-354.

Lin, C. Y., Lee, H. L., Hwang, Y. T., & Su, T. C. (2020). The association between total serum isomers of per-and polyfluoroalkyl substances, lipid profiles, and the DNA oxidative/nitrative stress biomarkers in middle-aged Taiwanese adults. *Environmental research, 182*, 109064.

Lin, P.-I. D., Cardenas, A., Hauser, R., Gold, D. R., Kleinman, K. P., Hivert, M.-F., . . . Oken, E. (2020). Per-and polyfluoroalkyl substances and blood pressure in pre-diabetic adults—cross-sectional and longitudinal analyses of the diabetes prevention program outcomes study. *Environment international, 137*, 105573.

Lin, P.-I. D., Cardenas, A., Hauser, R., Gold, D. R., Kleinman, K. P., Hivert, M.-F., . . . Horton, E. S. (2019). Per-and polyfluoroalkyl substances and blood lipid levels in pre-diabetic adults—longitudinal analysis of the diabetes prevention program outcomes study. *Environment international, 129*, 343-353.

Liu, G., Dhana, K., Furtado, J. D., Rood, J., Zong, G., Liang, L., . . . Coull, B. (2018). Perfluoroalkyl substances and changes in body weight and resting metabolic rate in response to weight-loss diets: a prospective study. *PLoS medicine, 15*(2), e1002502.

Liu, G., Zhang, B., Hu, Y., Rood, J., Liang, L., Qi, L., . . . Grandjean, P. (2020). Associations of perfluoroalkyl substances with blood lipids and apolipoproteins in lipoprotein subspecies: the POUNDS-lost study. *Environmental Health, 19*(1), 1-10.

Liu, H.-S., Wen, L.-L., Chu, P.-L., & Lin, C.-Y. (2018). Association among total serum isomers of perfluorinated chemicals, glucose homeostasis, lipid profiles, serum protein and metabolic syndrome in adults: NHANES, 2013–2014. *Environmental pollution, 232*, 73-79.

Liu, J.-J., Cui, X.-X., Tan, Y.-W., Dong, P.-X., Ou, Y.-Q., Li, Q.-Q., . . . Qin, S.-J. (2022). Per-and perfluoroalkyl substances alternatives, mixtures and liver function in adults: A community-based population study in China. *Environment international, 163*, 107179.

Liu, M., Zhang, G., Meng, L., Han, X., Li, Y., Shi, Y., . . . Jiang, G. (2021). Associations between novel and legacy per-and polyfluoroalkyl substances in human serum and thyroid cancer: a case and healthy population in Shandong Province, East China. *Environmental Science & Technology, 56*(10), 6144-6151.

Liu, Z., Que, S., Xu, J., & Peng, T. (2014). Alanine aminotransferase-old biomarker and new concept: a review. *International journal of medical sciences, 11*(9), 925.

Lloyd-Jones, D. M., Huffman, M. D., Karmali, K. N., Sanghavi, D. M., Wright, J. S., Pelser, C., . . . Goff Jr, D. C. (2017). Estimating longitudinal risks and benefits from cardiovascular preventive therapies among Medicare patients: the million hearts longitudinal ASCVD risk assessment tool: a special report from the American heart association and American College of cardiology. *Circulation, 135*(13), e793-e813.

Louis, G. M. B., Zhai, S., Smarr, M. M., Grewal, J., Zhang, C., Grantz, K. L., . . . Honda, M. (2018). Endocrine disruptors and neonatal anthropometry, NICHD fetal growth studies-singletons. *Environment international, 119*, 515-526.

Lu, S., & Bartell, S. M. (2020). *Serum PFAS Calculator for Adults*. Retrieved from www.ics.uci.edu/~sbartell/pfascalc.html

Luo, D., Wu, W., Pan, Y., Du, B., Shen, M., & Zeng, L. (2021). Associations of prenatal exposure to per-and polyfluoroalkyl substances with the neonatal birth size and hormones in the growth hormone/insulin-like growth factor axis. *Environmental Science & Technology, 55*(17), 11859-11873.

Ma, S., & Finch, B. K. (2010). Birth outcome measures and infant mortality. *Population research and policy review, 29*(6), 865-891.

Malits, J., Blustein, J., Trasande, L., & Attina, T. M. (2018). Perfluorooctanoic acid and low birth weight: Estimates of US attributable burden and economic costs from 2003 through 2014. *International journal of hygiene and environmental health, 221*(2), 269-275. doi:10.1016/j.ijheh.2017.11.004

Manzano-Salgado, C. B., Casas, M., Lopez-Espinosa, M.-J., Ballester, F., Iñiguez, C., Martinez, D., . . . Basterretxea, M. (2017). Prenatal exposure to perfluoroalkyl substances and cardiometabolic risk in children from the Spanish INMA Birth Cohort Study. *Environmental health perspectives, 125*(9), 097018.

Martin, K. V., Hilbert, T. J., Reilly, M., Christian, W. J., Hoover, A., Pennell, K. G., . . . Haynes, E. N. (2023). PFAS soil concentrations surrounding a hazardous waste incinerator in East Liverpool, Ohio, an environmental justice community. *Environmental science and pollution research international, 30(33)*, 80643–80654.

Martino Cinnera, A., Bonnì, S., Pellicciari, M. C., Giorgi, F., Caltagirone, C., & Koch, G. (2020). Health-related quality of life (HRQoL) after stroke: Positive relationship between lower extremity and balance recovery. *Topics in Stroke Rehabilitation, 27*(7), 534-540. doi:10.1080/10749357.2020.1726070

Martuzzi, M., Mitis, F., & Forastiere, F. (2010). Inequalities, inequities, environmental justice in waste management and health. *European Journal of Public Health, 20*(1), 21-26. doi:https://doi.org/10.1093/eurpub/ckp216

Mathiesen, U., Franzen, L., Frydén, A., Foberg, U., & Bodemar, G. (1999). The clinical significance of slightly to moderately increased liver transaminase values in asymptomatic patients. *Scandinavian journal of gastroenterology, 34*(1), 85-91.

Mattsson, K., Rignell-Hydbom, A., Holmberg, S., Thelin, A., Jönsson, B. A., Lindh, C. H., . . . Rylander, L. (2015). Levels of perfluoroalkyl substances and risk of coronary heart disease: Findings from a population-based longitudinal study. *Environmental research, 142*, 148-154.

McCleaf, P., Englund, S., Östlund, A., Lindegren, K., Wiberg, K., & Ahrens, L. (2017). Removal efficiency of multiple poly-and perfluoroalkyl substances (PFASs) in drinking water using granular activated carbon (GAC) and anion exchange (AE) column tests. *Water research*(120), 77-87.

McCord, J., & Strynar, M. (2019). Identification of per-and polyfluoroalkyl substances in the Cape Fear River by high resolution mass spectrometry and nontargeted screening. *Environmental Science & Technology, 53*(9), 4717-4727.

McCord, J., Strynar, M., Washington, J., Bergman, E., & Goodrow, S. (2020). Emerging chlorinated polyfluorinated polyether compounds impacting the waters of southwestern New Jersey identified by use of nontargeted analysis. *Environmental Science & Technology Letters, 7*(12), 903-908.

McDonough, L. K., Santos, I. R., Andersen, M. S., O'Carroll, D. M., Rutlidge, H., Meredith, K., . . . Lapworth, D. J. (2020). Changes in global groundwater organic carbon driven by climate change and urbanization. Nature communications. *11(1)*, 1-10.

McIntire, D. D., Bloom, S. L., Casey, B. M., & Leveno, K. J. (1999). Birth weight in relation to morbidity and mortality among newborn infants. *New England Journal of Medicine, 340*(16), 1234-1238.

McLaren Jr, R. A., Trejo, F. E., Blitz, M. J., Bianco, A., Limaye, M., Brustman, L., . . . Minkoff, H. (2021). COVID-related "lockdowns" and birth rates in New York. *American journal of obstetrics & gynecology MFM, 3*(6), 100476.

McNamara, J. D., Franco, R., Mimna, R., & Zappa, L. (2018). Comparison of activated carbons for removal of perfluorinated compounds from drinking water. *Journal-American Water Works Association, 110*(1), E2-E14.

Minnesota Pollution Control Agency. (2021). *Minnesota's PFAS Blueprint*. (p-gen1-22). Retrieved from https://www.pca.state.mn.us/sites/default/files/p-gen1-22.pdf

Mohai, P., & Saha, R. (2015). Which came first, people or pollution? A review of theory and evidence from longitudinal environmental justice studies. *Environmental research letters, 10(12)*, 125011.

Mollon, L., & Bhattacharjee, S. (2017). Health related quality of life among myocardial infarction survivors in the United States: a propensity score matched analysis. *Health and Quality of Life Outcomes, 15*(1), 235. doi:10.1186/s12955-017-0809-3

Mongraw-Chaffin, M., Gujral, U. P., Kanaya, A. M., Kandula, N. R., Carr, J. J., & Anderson, C. A. (2018). Relation of ectopic fat with atherosclerotic cardiovascular disease risk score in South Asians living in the United States (from the Mediators of Atherosclerosis in South Asians Living in America [MASALA] Study). *The American journal of cardiology, 121*(3), 315-321.

Mora, S., Wenger, N. K., Cook, N. R., Liu, J., Howard, B. V., Limacher, M. C., . . . Paynter, N. P. (2018). Evaluation of the pooled cohort risk equations for cardiovascular risk prediction in a multiethnic cohort from the Women's Health Initiative. *JAMA Internal Medicine, 178*(9), 1231-1240.

Muntner, P., Colantonio, L. D., Cushman, M., Goff, D. C., Jr., Howard, G., Howard, V. J., . . . Safford, M. M. (2014). Validation of the atherosclerotic cardiovascular disease Pooled Cohort risk equations. *Jama, 311*(14), 1406-1415. doi:10.1001/jama.2014.2630

National Academies of Sciences, E., and Medicine,. (2020). *Management of Legionella in Water Systems*. Washington, DC.: The National Academies Press

National Academies of Sciences, E., and Medicine,. (2022). *Guidance on PFAS Exposure, Testing, and Clinical Follow-Up (2022)*. Washington, DC: The National Academies Press.

National Academies of Sciences, E., and Medicine,. (2023). *Low Birth Weight Babies and Disability*. Retrieved from Washington, DC: https://nap.nationalacademies.org/catalog/27375/low-birth-weight-babies-and-disability

National Academy of Public Administration. (2017). Developing a New Framework for Community Affordability of Clean Water Services.

National Cancer Institute. (2020). *Persistent Poverty Linked to Increased Risk of Dying from Cancer*. Retrieved from https://www.cancer.gov/news-events/cancer-currents-blog/2020/persistent-poverty-increased-cancer-death-risk

National Drinking Water Advisory Council. (2023). *National Drinking Water Advisory Council Meeting Summary Report*.

National Institutes of Health. (2017). What are the risks of preeclampsia & eclampsia to the fetus? Retrieved from https://www.nichd.nih.gov/health/topics/preeclampsia/conditioninfo/risk-fetus

National Institutes of Health. (2018). What are the risks of preeclampsia & eclampsia to the mother? Retrieved from https://www.nichd.nih.gov/health/topics/preeclampsia/conditioninfo/risk-mother

National Technology Transfer and Advancement Act. 1995. Public Law No. 104-113, 110 Stat. 783. 104th Congress. Available at https://www.gpo.gov/fdsys/pkg/PLAW-104publ113/pdf/PLAW-104publ113.pdf., (1995).

National Toxicology Program. (2018a). 28-day evaluation of the toxicity (C06100) of perfluorohexane sulfonate potassium salt (PFHSKslt) (3871-99-6) on Harlan Sprague-Dawley rats exposed via gavage.

National Toxicology Program. (2018b). *28-day evaluation of the toxicity (C20615) of perfluorodecanoic acid (PFDA) (335-76-2) on Harlan Sprague-Dawley rats exposed via gavage [NTP]. U.S. Department of Health and Human Services.* Retrieved from http://dx.doi.org/10.22427/NTP-DATA-002-02652-0004-0000-1

National Toxicology Program. (2018c). *Report on Carcinogens: Monograph on Haloacetic Acids Found as Water Disinfection By-Products*. Research Triangle Park, NC.

National Toxicology Program. (2021). Report on Carcinogens, Fifteenth Edition.; Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service.

Negri, E., Metruccio, F., Guercio, V., Tosti, L., Benfenati, E., Bonzi, R., . . . Moretto, A. (2017). Exposure to PFOA and PFOS and fetal growth: a critical merging of toxicological and epidemiological data. *Critical reviews in toxicology, 10.1080/10408444.2016.1271972,* 489-515. doi:10.1080/10408444.2016.1271972

Nelson, J. W., Hatch, E. E., & Webster, T. F. (2010). Exposure to polyfluoroalkyl chemicals and cholesterol, body weight, and insulin resistance in the general US population. *Environmental health perspectives, 118*(2), 197-202.

Nelson, J. W., Scammell, M. K., Hatch, E. E., & Webster, T. F. (2012). Social disparities in exposures to bisphenol A and polyfluoroalkyl chemicals: a cross-sectional study within NHANES 2003-2006. *Environmental Health, 11*(1), 1-15.

Nguyen, Q. D., Odden, M. C., Peralta, C. A., & Kim, D. H. (2020). Predicting risk of atherosclerotic cardiovascular disease using pooled cohort equations in older adults with frailty, multimorbidity, and competing risks. *Journal of the American Heart Association, 9*(18), e016003.

Nguyen, V. K., Kahana, A., Heidt, J., Polemi, K., Kvasnicka, J., Jolliet, O., & Colacino, J. A. (2020). A comprehensive analysis of racial disparities in chemical biomarker concentrations in United States women, 1999–2014. *Environment international, 137,* 105496.

Nian, M., Li, Q.-Q., Bloom, M., Qian, Z. M., Syberg, K. M., Vaughn, M. G., . . . Gurram, N. (2019). Liver function biomarkers disorder is associated with exposure to perfluoroalkyl acids in adults: Isomers of C8 Health Project in China. *Environmental research, 172,* 81-88.

Nicoletti, C., Salvanes, K. G., & Tominey, E. (2018). Response of parental investments to child's health endowment at birth. In *Health Econometrics*: Emerald Publishing Limited.

O'Sullivan, A. K., Rubin, J., Nyambose, J., Kuznik, A., Cohen, D. J., & Thompson, D. (2011). Cost estimation of cardiovascular disease events in the US. *Pharmacoeconomics, 29*(8), 693-704.

Obsekov, V., Kahn, L. G., & Trasande, L. (2023). Leveraging systematic Reviews to Explore disease burden and costs of per-and polyfluoroalkyl substance exposures in the United States. *Exposure and Health, 15*(2), 373-394.

Occupational Safety and Health Administration. (2010). *Occupational Exposure to Respirable Crystalline Silica -- Review of Health Effects Literature and Preliminary Quantitative Risk Assessment*.

Occupational Safety and Health Administration. (2016). *Occupational Exposure to Respirable Crystalline Silica*. (81 FR 16285 2016-04800). Federal Register Retrieved from https://www.federalregister.gov/documents/2016/03/25/2016-04800/occupational-exposure-to-respirable-crystalline-silica

Ochoa-Herrera, V., & Sierra-Alvarez, R. (2008). Removal of perfluorinated surfactants by sorption onto granular activated carbon, zeolite and sludge. *Chemosphere, 72*(10), 1588-1593.

Office of Environmental Health Hazard Assessment. (2016). CalEnviroScreen 3.0.

Osuchukwu, O., & Reed, D. (2022). Small for Gestational Age. *StatPearls [Internet]*.

Papadopoulou, E., Stratakis, N., Basagana, X., Brantsæter, A. L., Casas, M., Fossati, S., . . . Maitre, L. (2021). Prenatal and postnatal exposure to PFAS and cardiometabolic factors and inflammation status in children from six European cohorts. *Environment international, 157*, 106853.

Park, J. H., Choi, J., Jun, D. W., Han, S. W., Yeo, Y. H., & Nguyen, M. H. (2019). Low alanine aminotransferase cut-off for predicting liver outcomes; a nationwide population-based longitudinal cohort study. *Journal of clinical medicine, 8*(9), 1445.

Park, S. K., Peng, Q., Ding, N., Mukherjee, B., & Harlow, S. D. (2019). Determinants of per-and polyfluoroalkyl substances (PFAS) in midlife women: evidence of racial/ethnic and geographic differences in PFAS exposure. *Environmental research, 175*, 186-199.

Pinheiro, P. S., Medina, H. N., Callahan, K. E., Koru-Sengul, T., Sharma, J., Kobetz, E. N., & Penedo, F. J. (2021). Kidney cancer mortality disparities among Hispanics in the US. *Cancer Epidemiology, 72*, 101938. doi:https://doi.org/10.1016/j.canep.2021.101938

Pitter, G., Zare Jeddi, M., Barbieri, G., Gion, M., Fabricio, A. S., Daprà, F., . . . Canova, C. (2020). Perfluoroalkyl substances are associated with elevated blood pressure and hypertension in highly exposed young adults. *Environmental Health, 19*(1), 1-11.

Pletcher, M. J., Vittinghoff, E., Thanataveerat, A., Bibbins-Domingo, K., & Moran, A. E. (2016). Young adult exposure to cardiovascular risk factors and risk of events later in life: The Framingham Offspring Study. *PLoS One, 11*(5), e0154288.

Pramanik, B. K., Pramanik, S. K., & Suja, F. (2015). A comparative study of coagulation, granular-and powdered-activated carbon for the removal of perfluorooctane sulfonate and perfluorooctanoate in drinking water treatment. *Environmental technology, 36*(20), 2610-2617.

R Core Team. (2021). R: A language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing.

Raghavan, S., Ho, Y.-L., Vassy, J. L., Posner, D., Honerlaw, J., Costa, L., . . . Cho, K. (2020). Optimizing Atherosclerotic Cardiovascular Disease Risk Estimation for Veterans With Diabetes Mellitus. *Circulation. Cardiovascular quality and outcomes, 13*(9), 120006528. doi:10.1161/circoutcomes.120.006528

Raleigh, K. K., Alexander, B. H., Olsen, G. W., Ramachandran, G., Morey, S. Z., Church, T. R., . . . Allen, E. M. (2014). Mortality and cancer incidence in ammonium perfluorooctanoate production workers. *Occupational Environmental Medicine, 71*, 500-506.

Ramhøj, L., Hass, U., Boberg, J., Scholze, M., Christiansen, S., Nielsen, F., & Axelstad, M. (2018). Perfluorohexane sulfonate (PFHxS) and a mixture of endocrine disrupters reduce thyroxine levels and cause antiandrogenic effects in rats. *Toxicological Sciences, 163*(2), 579-591.

Ramhøj, L., Hass, U., Gilbert, M. E., Wood, C., Svingen, T., Usai, D., . . . Axelstad, M. (2020). Evaluating thyroid hormone disruption: investigations of long-term neurodevelopmental effects in rats after perinatal exposure to perfluorohexane sulfonate (PFHxS). *Scientific reports, 10*(1), 2672.

Raucher, R., Clements, J., Rothstein, E., Mastracchio, J., & Green, Z. (2019). Developing a New Framework for Household Affordability and Financial Capability Assessment in the Water Sector.

Regli, S., Chen, J., Messner, M., Elovitz, M. S., Letkiewicz, F. J., Pegram, R. A., . . . Wright, J. M. (2015). Estimating potential increased bladder cancer risk due to increased bromide concentrations in sources of disinfected drinking waters. *Environmental Science & Technology, 49*(22), 13094-13102.

Richardson, S. D. (2022). Invited Perspective: Existing Rules for Disinfection By-Products Are Good, but They Are Not Enough. *Environmental health perspectives, 130*(8), 081302.

Robinson, J. G., & Booth, B. (2010). Statin use and lipid levels in older adults: National Health and Nutrition Examination Survey, 2001–2006. *Journal of Clinical Lipidology, 4*(6), 483–490. doi:10.1016/j.jacl.2010.10.002

Rockaway, T. D., Coomes, P. A., Rivard, J., & Kornstein, B. (2011). Residential water use trends in North America. *Journal-American Water Works Association, 103*(2), 76-89.

Rodríguez-Barranco, M., Tobías, A., Redondo, D., Molina-Portillo, E., & Sánchez, M. J. (2017). Standardizing effect size from linear regression models with log-transformed variables for meta-analysis. *BMC medical research methodology, 17*(1), 1-9.

Rodriguez, F., Chung, S., Blum, M. R., Coulet, A., Basu, S., & Palaniappan, L. P. (2019). Atherosclerotic cardiovascular disease risk prediction in disaggregated Asian and Hispanic subgroups using electronic health records. *Journal of the American Heart Association, 8*(14), e011874.

Roger, V. L., Go, A. S., Lloyd-Jones, D. M., Benjamin, E. J., Berry, J. D., Borden, W. B., . . . Ford, E. S. (2012). Heart disease and stroke statistics—2012 update: a report from the American Heart Association. *Circulation, 125*(1), e2.

Rosinger, A. Y., Herrick, K. A., Wutich, A. Y., Yoder, J. S., & Ogden, C. L. (2018). Disparities in plain, tap and bottled water consumption among US adults: National Health and Nutrition Examination Survey (NHANES) 2007-2014. *Public health nutrition, 21*(8), 1455-1464. doi:10.1017/S1368980017004050

Roth, K., Yang, Z., Agarwal, M., Liu, W., Peng, Z., Long, Z., . . . Petriello, M. C. (2021). Exposure to a mixture of legacy, alternative, and replacement per-and polyfluoroalkyl substances (PFAS) results in sex-dependent modulation of cholesterol metabolism and liver injury. *Environment international, 157*, 106843.

Rücker, G., Schwarzer, G., Carpenter, J., & Olkin, I. (2009). Why add anything to nothing? The arcsine difference as a measure of treatment effect in meta-analysis with zero cells. *Statistics in medicine, 28*(5), 721-738.

Sagiv, S. K., Rifas-Shiman, S. L., Fleisch, A. F., Webster, T. F., Calafat, A. M., Ye, X., . . . Oken, E. (2018). Early-pregnancy plasma concentrations of perfluoroalkyl substances and birth outcomes in Project Viva: confounded by pregnancy hemodynamics? *American journal of epidemiology, 187*(4), 793-802.

Salihovic, S., Stubleski, J., Kärrman, A., Larsson, A., Fall, T., Lind, L., & Lind, P. M. (2018). Changes in markers of liver function in relation to changes in perfluoroalkyl substances-a longitudinal study. *Environment international, 117*, 196-203.

Science Applications International Corporation. (1998). *An Assessment of the Vulnerability of Non-Community Water Systems to SDWA Cost Increases. Submitted to U.S. Environmental Protection Agency September 29, 1998. EPA Contract No. 68-C.-0059, WA No. I-26.*

Shankar, A., Xiao, J., & Ducatman, A. (2012). Perfluorooctanoic acid and cardiovascular disease in US adults. *Archives of Internal Medicine, 172*(18), 1397-1403.

Shearer, J. J., Callahan, C. L., Calafat, A. M., Huang, W. Y., Jones, R. R., Sabbisetti, V. S., . . . Hofmann, J. N. (2021). Serum concentrations of per- and polyfluoroalkyl substances and risk of renal cell carcinoma. *Journal of the National Cancer Institute, 113*(5), 580-587. doi:10.1093/jnci/djaa143

Skolarus, L. E., Burke, J. F., Brown, D. L., & Freedman, V. A. (2014). Understanding stroke survivorship: expanding the concept of poststroke disability. *Stroke, 45*(1), 224-230.

Smalling, K. L., Romanok, K. M., Bradley, P. M., Morriss, M. C., Gray, J. L., Kanagy, L. K., . . . Jones, D. K. (2023). Per-and polyfluoroalkyl substances (PFAS) in United States tapwater: Comparison of underserved private-well and public-supply exposures and associated health implications. *Environment international*, 108033.

Sörengård, M., Östblom, E., Köhler, S., & Ahrens, L. (2020). Adsorption behavior of per-and polyfluoralkyl substances (PFASs) to 44 inorganic and organic sorbents and use of dyes as proxies for PFAS sorption. *Journal of Environmental Chemical Engineering, 8*(3), 103744.

Sorlini, S., & Collivignarelli, C. (2005). Chlorite removal with granular activated carbon. *Desalination, 176*(1-3), 255-265.

Souza, M. C. O., Saraiva, M. C. P., Honda, M., Barbieri, M. A., Bettiol, H., Barbosa, F., & Kannan, K. (2020). Exposure to per-and polyfluorinated alkyl substances in pregnant Brazilian women and its association with fetal growth. *Environmental research, 187*, 109585.

Speitel Jr, G. E., Turakhia, M. H., & Lu, C.-J. (1989). Initiation of micropollutant biodegradation in virgin GAC columns. *Journal (American Water Works Association)*, 168-176.

Starling, A. P., Adgate, J. L., Hamman, R. F., Kechris, K., Calafat, A. M., Ye, X., & Dabelea, D. (2017). Perfluoroalkyl substances during pregnancy and offspring weight and adiposity at birth: examining mediation by maternal fasting glucose in the Healthy Start Study. *Environ Health Perspect, 125*, 067016-067011. doi:10.1289/EHP641

Steenland, K., Barry, V., & Savitz, D. (2018). Serum Perfluorooctanoic Acid and Birthweight: An Updated Meta-analysis With Bias Analysis. *Epidemiology, 29*(6), 765-776. doi:10.1097/EDE.0000000000000903

Steenland, K., Tinker, S., Frisbee, S., Ducatman, A., & Vaccarino, V. (2009). Association of perfluorooctanoic acid and perfluorooctane sulfonate with serum lipids among adults living near a chemical plant. *American journal of epidemiology, 170*(10), 1268-1278.

Steenland, K., & Woskie, S. (2012). Cohort mortality study of workers exposed to perfluorooctanoic acid. *American journal of epidemiology, 176*, 909–917.

Stoiber, T., Evans, S., & Naidenko, O. V. (2020). Disposal of products and materials containing per- and polyfluoroalkyl substances (PFAS): A cyclical problem. *Chemosphere, 260*, 127659. doi:https://doi.org/10.1016/j.chemosphere.2020.127659

Summers, R. S., Hooper, S., Shukairy, H., Solarik, G., & Owen, D. M. (1996). Assessing DBP Yield: Uniform Formation Conditions. *J. Amer. Water Works Assoc., 88(6)*, 80-93.

Summers, R. S., Kim, S. M., Shimabuku, K., Chae, S. H., & Corwin, C. J. (2013). Granular activated carbon adsorption of MIB in the presence of dissolved organic matter. *Water research, 47(10)*, 3507-3513.

Sunderland, E. M., Hu, X. C., Dassuncao, C., Tokranov, A. K., Wagner, C. C., & Allen, J. G. (2019). A review of the pathways of human exposure to poly- and perfluoroalkyl substances (PFASs) and present understanding of health effects. *J Expo Sci Environ Epidemiol, 29*(2), 131-147. doi:10.1038/s41370-018-0094-1

Temple, J. A., Reynolds, A. J., & Arteaga, I. (2010). Low birth weight, preschool education, and school remediation. *Education and urban society, 42*(6), 705-729.

Terry, L. G., & R.S., S. (2018). Biodegradable organic matter and rapid-rate biofilter performance: A review. *Water research, 128*, 234-245.

Thom, T., Kannel, W., Silbershatz, H., & D'Agostino, R. (2001). Cardiovascular diseases in the United States and prevention approaches. *Hurst's the heart, 1*, 3-17.

Thomford, P. (2002). 104-week dietary chronic toxicity and carcinogenicity study with perfluorooctane sulfonic acid potassium salt (PFOS; T-6295) in rats.

Toth, P. P., Granowitz, C., Hull, M., Anderson, A., & Philip, S. (2019). Long-term statin persistence is poor among high-risk patients with dyslipidemia: a real-world administrative claims analysis. *Lipids in Health and Disease, 18*(1). doi:10.1186/s12944-019-1099-z

Tsai, M., Chang, S., Kuo, W., Kuo, C., Li, S., Wang, M., . . . Chen, P. (2020). A case-control study of perfluoroalkyl substances and the risk of breast cancer in Taiwanese women. *Environment international*. doi:10.1016/j.envint.2020.105850

U.S Environmental Protection Agency. (2011). *PWSS Program ICR*.

U.S Environmental Protection Agency. (2022). *Designation of Perfluorooctanoic Acid (PFOA) and Perfluorooctanesulfonic Acid (PFOS) as CERCLA Hazardous Substances.*

U.S. Bureau of Labor Statistics. (2021). Medical care in U.S. city average, all urban consumers, not seasonally adjusted, 1980-2021 CUUR0000SAM.

U.S. Bureau of Labor Statistics. (2022a). Medical care in U.S. city average, all urban consumers, not seasonally adjusted, 1980-2022 CUUR0000SAM.

U.S. Bureau of Labor Statistics. (2022b). Table 1. Employment Cost Index for total compensation, by occupational group and industry. Employment Cost Index Historical Listing – Volume III. Retrieved from https://www.bls.gov/web/eci/eci-current-nominal-.dollar.pdf

U.S. Census Bureau. (2010). *American Community Survey Five-Year Estimates. American Community Survey Demographic and Housing Estimates*. Retrieved from https://www.census.gov/programs-surveys/acs/technical-documentation/table-and-geography-changes.2010.html

U.S. Census Bureau. (2020a). *2019 Population Estimates by Age, Sex, Race and Hispanic Origin*. Retrieved from https://www.census.gov/newsroom/press-kits/2020/population-estimates-detailed.html

U.S. Census Bureau. (2020b). *Average Number of People per Household, by Race and Hispanic Origin, Marital Status, Age, and Education of Householder*. Retrieved from https://www2.census.gov/programs-surveys/demo/tables/families/2020/cps-2020/tabavg1.xls

U.S. Energy Information Administration. (2021). Annual Energy Outlook 2021. Retrieved August 10, 2021 https://www.eia.gov/outlooks/aeo/

U.S. Environmental Protection Agency. (1989). *Surface Water Treatment Rule*.

U.S. Environmental Protection Agency. (1994). *EPA09: Establish a Blueprint for Environmental Justice Throughout EPA's Operations*. Retrieved from https://govinfo.library.unt.edu/npr/library/reports/EPA9.html

U.S. Environmental Protection Agency. (1996). *DBP/ICR Analytical Methods Manual*. Cincinnati, Ohio

U.S. Environmental Protection Agency. (1997). *Discussion Summary: EPA Technology Design Workshop*. Retrieved from https://downloads.regulations.gov/EPA-HQ-OW-2018-0780-0197/content.pdf

U.S. Environmental Protection Agency. (1998a). *Alachlor Technical Fact Sheet. https://www3.epa.gov/pesticides/chem_search/reg_actions/reregistration/fs_PC-090501_1-Dec-98.pdf*.

U.S. Environmental Protection Agency. (1998b). Information for States on Developing Affordability Criteria for Drinking Water. EPA 816-R-98-002.

U.S. Environmental Protection Agency. (1998c). *National Primary Drinking Water Regulations: Regulatory Flexibility Act. Federal Register 63(30):7620. February 13, 2008.*

U.S. Environmental Protection Agency. (1998d). *Primary Drinking Water Regulations, Code of Federal Regulations Title 40 Chapter I Subchapter D Part 141 https://www.ecfr.gov/current/title-40/chapter-I/subchapter-D/part-141*.

U.S. Environmental Protection Agency. (1998e). *Stage 1 Disinfectants and Disinfection Byproducts Rule*. Retrieved from https://www.govinfo.gov/content/pkg/FR-1998-12-16/pdf/98-32887.pdf

U.S. Environmental Protection Agency. (2000). *Geometries and Characteristics of Public Water Systems*. (EPA-815-R-00-24).

U.S. Environmental Protection Agency. (2002). *Hepatocellular hypertrophy. HED guidance document #G2002.01*.

U.S. Environmental Protection Agency. (2004). *The Standardized Monitoring Framework: A Quick Reference Guide*. (EPA-816-F-04-010). Retrieved from https://www.epa.gov/sites/default/files/2020-05/documents/smf_2020_final_508.pdf

U.S. Environmental Protection Agency. (2005a). *Drinking Water Criteria Document for Brominated Trihalomethanes*. (EPA-822-R-05-011).

U.S. Environmental Protection Agency. (2005b). *Economic Analysis for the Final Stage 2 Disinfectants and Disinfection Byproducts Rule*. (EPA 815-R-05-010).

U.S. Environmental Protection Agency. (2005c). *Guidelines for Carcinogen Risk Assessment.* Washington, D.C. Retrieved from https://www3.epa.gov/airtoxics/cancer_guidelines_final_3-25-05.pdf

U.S. Environmental Protection Agency. (2006a). *National Primary Drinking Water Regulations: Long Term 2 Enhanced Surface Water Treatment Rule; Final Rule, EPA 815-Z-06-001*. (EPA 815-Z-06-001).

U.S. Environmental Protection Agency. (2006b). *National Primary Drinking Water Regulations: Stage 2 Disinfectants and Disinfection Byproducts Rule; Final Rule. 71 FR 388.* Retrieved from https://www.federalregister.gov/documents/2006/01/04/06-3/national-primary-drinking-water-regulations-stage-2-disinfectants-and-disinfection-byproducts-rule

U.S. Environmental Protection Agency. (2007a). *Atrazine Chemical Summary*. *https://archive.epa.gov/region5/teach/web/pdf/atrazine_summary.pdf*.

U.S. Environmental Protection Agency. (2007b). Office Ground Water and Drinking Water's Error Code Tracking Tool [for SDWIS/Fed]. *Developed 2007*.

U.S. Environmental Protection Agency. (2008). *Final Ozone NAAQS Regulatory Impact Analysis*. (EPA-452/R-08-003). Research Triangle Park, North Carolina Retrieved from https://www3.epa.gov/ttn/ecas/docs/ria/naaqs-o3_ria_final_2008-03.pdf

U.S. Environmental Protection Agency. (2009). *Community Water System Survey, Volume II: Detailed Tables and Survey Methodology. EPA 815-R-09-002.* Retrieved from http://nepis.epa.gov/Exe/ZyPDF.cgi?Dockey=P1009USA.txt

U.S. Environmental Protection Agency. (2010a). *Guidelines for Preparing Economic Analyses*.

Paperwork Reduction Act, as amended. 44 USC 3501–44 USC 3521. Available at https://www.gpo.gov/fdsys/pkg/USCODE-2015-title44/pdf/USCODE-2015-title44-chap35-subchapI-sec3501.pdf, (2010b).

U.S. Environmental Protection Agency. (2011a). *The Benefits and Costs of the Clean Air Act from 1990 to 2020. Final Report - Rev. A.* Retrieved from https://www.epa.gov/sites/default/files/2015-07/documents/fullreport_rev_a.pdf

U.S. Environmental Protection Agency. (2011b). *Exposure Factors Handbook, 2011 Edition (Final)*. (EPA-600-R-09-025F). U.S. Environmental Protection Agency, Washington, DC

U.S. Environmental Protection Agency. (2012). *Revisions to the Unregulated Contaminant Monitoring Regulation (UCMR 3) for Public Water Systems; Final Rule*. Federal Register Retrieved from https://www.federalregister.gov/documents/2012/05/02/2012-9978/revisions-to-the-unregulated-contaminant-monitoring-regulation-ucmr-3-for-public-water-systems

U.S. Environmental Protection Agency. (2014). *Dichloromethane Technical Fact Sheet. https://archive.epa.gov/water/archive/web/pdf/archived-technical-fact-sheet-on-dichloromethane.pdf*.

U.S. Environmental Protection Agency. (2015). *Benefit and Cost Analysis for the Effluent Limitations Guidelines and Standards for the Steam Electric Power Generating Point Source Category*. (EPA-821-R-15-005). US EPA, Office of Water, Washington DC 20460

U.S. Environmental Protection Agency. (2016a). *The Data Management and Quality Assurance/Quality Control Process for the Third Six-Year Review Information Collection Rule Dataset*. (EPA 810-R-16-015).

U.S. Environmental Protection Agency. (2016b). *Drinking Water Health Advisory for Perfluorooctane Sulfonate (PFOS)*. (EPA822-R-16-004).

U.S. Environmental Protection Agency. (2016c). *Drinking Water Health Advisory for Perfluorooctanoic Acid (PFOA)*. (EPA 822-R-16-005).

U.S. Environmental Protection Agency. (2016d). *Fact Sheet: PFOA& PFOS Drinking Water Health Advisories*. (EPA 800-F-16-003). Retrieved from https://www.epa.gov/sites/default/files/2016-06/documents/drinkingwaterhealthadvisories_pfoa_pfos_updated_5.31.16.pdf

U.S. Environmental Protection Agency. (2016e). *Health effects support document for Perfluorooctane Sulfonate (PFOS)*. (EPA 822-R-16-002). Retrieved from https://www.epa.gov/sites/production/files/2016-05/documents/pfos_hesd_final_508.pdf

U.S. Environmental Protection Agency. (2016f). *Health effects support document for Perfluorooctanoic Acid (PFOA)*. (EPA 822-R-16-003). Retrieved from https://www.epa.gov/sites/production/files/2016-05/documents/pfoa_hesd_final-plain.pdf

U.S. Environmental Protection Agency. (2016g). *Six-Year Review 3 Technical Support Document for Disinfectants/Disinfection Byproducts Rules*. (EPA-810-R-16-012). Retrieved from https://www.epa.gov/sites/production/files/2016-12/documents/810r16012.pdf

U.S. Environmental Protection Agency. (2016h). *Technical Guidance for Assessing Environmental Justice in Regulatory Analysis*. Retrieved from https://www.epa.gov/sites/default/files/2016-06/documents/ejtg_5_6_16_v5.1.pdf

U.S. Environmental Protection Agency. (2016i). *Technologies for Legionella Control in Premise Plumbing Systems: Scientific Literature Review. EPA 810-R-16-001.*

U.S. Environmental Protection Agency. (2016j). *Third Six-Year Review of National Primary Drinking Water Standards*. Retrieved from https://www.epa.gov/sites/default/files/2016-12/documents/six_year_3_review_frn_12-21-16.pdf

U.S. Environmental Protection Agency. (2017). *Third Unregulated Contaminant Monitoring Rule: Occurrence Data*. Retrieved from https://www.epa.gov/dwucmr/occurrence-data-unregulated-contaminant-monitoring-rule#3

U.S. Environmental Protection Agency. (2018). *Method 537.1: Determination of Selected Per- and Polyfluorinated Alkyl Substances in Drinking Water by Solid Phase Extraction and Liquid Chromatography/Tandem Mass Spectrometry (LC/MS/MS), Version 1.0.* (EPA/600/R-18/352). Retrieved from https://cfpub.epa.gov/si/si_public_record_Report.cfm?dirEntryId=343042&Lab=NERL

U.S. Environmental Protection Agency. (2019a). EJSCREEN Technical Documentation.

U.S. Environmental Protection Agency. (2019b). *Method 533: Determination of Per- and Polyfluoroalkyl Substances in Drinking Water by Isotope Dilution Anion Exchange Solid Phase Extraction and Liquid Chromatography/Tandem Mass Spectrometry*. (EPA 815-B-19-020). Retrieved from https://www.epa.gov/sites/default/files/2019-12/documents/method-533-815b19020.pdf

U.S. Environmental Protection Agency. (2019c). *Occurrence Data from the Third Unregulated Contaminant Monitoring Rule (UCMR 3)*. Retrieved from https://www.epa.gov/dwucmr/occurrence-data-unregulated-contaminant-monitoring-rule#3

U.S. Environmental Protection Agency. (2019d). Supplemental Environmental Assessment for Proposed Revisions to Effluent Limitations Guidelines and Standards for the Steam Electric Power Generating Point Source Category (EA). EPA-821-R-19-010.

U.S. Environmental Protection Agency. (2020a). *Announcement of Preliminary Regulatory Determinations for Contaminants on the Fourth Drinking Water Contaminant Candidate List. EPA-HQ-OW-2019-0583 FRL-10005-88-OW. 85 FR 14098.* Retrieved from https://www.federalregister.gov/documents/2020/03/10/2020-04145/announcement-of-preliminary-regulatory-determinations-for-contaminants-on-the-fourth-drinking-water

U.S. Environmental Protection Agency. (2020b). *Interim Guidance on the Destruction and Disposal of Perfluoroalkyl and Polyfluoroalkyl Substances and Materials Containing Perfluoroalkyl and Polyfluoroalkyl Substances*. Washington, D.C. Retrieved from https://www.epa.gov/system/files/documents/2021-11/epa-hq-olem-2020-0527-0002_content.pdf

U.S. Environmental Protection Agency. (2020c). *Interim Guidance on the Destruction and Disposal of Perfluoroalkyl and Polyfluoroalkyl Substances and Materials Containing Perfluoroalkyl and Polyfluoroalkyl Substances. EPA-HQ-OLEM-2020-0527-0002.* Retrieved from https://www.epa.gov/system/files/documents/2021-11/epa-hq-olem-2020-0527-0002_content.pdf

U.S. Environmental Protection Agency. (2021a). *Analysis of Cardiovascular Disease Risk Reduction as a Result of Reduced PFOA and PFOS Exposure in Drinking Water*. (EPA 815-D-21-001). Retrieved from https://sab.epa.gov/ords/sab/f?p=100:19:9687145615924:::RP,19:P19_ID:963

U.S. Environmental Protection Agency. (2021b). *Announcement of Final Regulatory Determinations for Contaminants on the Fourth Drinking Water Contaminant Candidate List*. (86 FR 12272). Retrieved from https://www.federalregister.gov/documents/2021/03/03/2021-04184/announcement-of-final-regulatory-determinations-for-contaminants-on-the-fourth-drinking-water

U.S. Environmental Protection Agency. (2021c). *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". EPA-8232-PR-1821-001010.* Retrieved from https://www.epa.gov/system/files/documents/2021-10/genx-chemicals-toxicity-assessment_tech-edited_oct-21-508.pdf

U.S. Environmental Protection Agency. (2021d). *Human Health Toxicity Values for Perfluorobutane Sulfonic Acid (CASRN 375-73-5) and Related Compound Potassium Perfluorobutane Sulfonate (CASRN 29420-49-3). EPA/600/R-20/345F*. Washington, DC

U.S. Environmental Protection Agency. (2021e). *PFAS Strategic Roadmap: EPA's Commitments to Action 2021–2024*. (EPA-100-K-21-002).

U.S. Environmental Protection Agency. (2021f). *Proposed Approaches to the Derivation of a Draft Maximum Contaminant Level Goal for Perfluorooctane Sulfonic Acid (PFOS) (CASRN 1763-23-1) in Drinking Water*. (EPA 822D21002).

U.S. Environmental Protection Agency. (2021g). *Proposed Approaches to the Derivation of a Draft Maximum Contaminant Level Goal for Perfluorooctanoic Acid (PFOA) (CASRN 335-67-1) in Drinking Water*. (EPA 822D21001). Washington, DC

U.S. Environmental Protection Agency. (2021h). *Safe Drinking Water Information System 2021 Quarter 4 Database*. Retrieved from https://www.epa.gov/ground-water-and-drinking-water/safe-drinking-water-information-system-sdwis-federal-reporting

U.S. Environmental Protection Agency. (2022a). *2022 EJSCREENBatch R package*. Retrieved from: https://github.com/USEPA/EJSCREENBatch.

U.S. Environmental Protection Agency. (2022b). *Comprehensive Environmental Response, Compensation, and Liability Act Hazardous Substances: Designation of Perfluorooctanoic Acid and Perfluorooctanesulfonic Acid. EPA-HQ-OLEM-2019-0341-0001*. Washington, D.C. Retrieved from https://www.regulations.gov/document/EPA-HQ-OLEM-2019-0341-0001

U.S. Environmental Protection Agency. (2022c). *The Fourth Unregulated Contaminant Monitoring Rule Occurrence Data. Office of Ground Water and Drinking Water. January 2022*. Retrieved from https://www.epa.gov/dwucmr/occurrence-data-unregulated-contaminant-monitoring-rule#4

U.S. Environmental Protection Agency. (2022d). *Implementation of the Clean Water and Drinking Water State Revolving Fund Provisions of the Bipartisan Infrastructure Law*. Retrieved from https://www.epa.gov/dwsrf/bipartisan-infrastructure-law-srf-memorandum

U.S. Environmental Protection Agency. (2022e). *IRIS Toxicological Review of Perfluorobutanoic Acid (PFBA) and Related Salts (Final Report, 2022). EPA/635/R-22/277F*. Retrieved from https://www.epa.gov/chemical-research/iris-toxicological-review-perfluorobutanoic-acid-pfba-and-related-salts-final

U.S. Environmental Protection Agency. (2022f). *Microbial and Disinfection Byproduct Data Files (2012-2019) from EPA's Fourth Six-Year Review Information Collection Request*. Retrieved from https://www.epa.gov/dwsixyearreview/microbial-and-disinfection-byproduct-data-files-2012-2019-epas-fourth-six-year

U.S. Environmental Protection Agency. (2022g). *ORD staff handbook for developing IRIS assessments [EPA Report]. (EPA 600/R-22/268). Washington, DC: U.S. Environmental Protection Agency, Office of Research and Development, Center for Public Health and Environmental Assessment.*

U.S. Environmental Protection Agency. (2022h). *PFAS Explained*. Retrieved from https://www.epa.gov/pfas/pfas-explained

U.S. Environmental Protection Agency. (2022i). *Review of EPA's Analyses to Support EPA's National Primary Drinking Water Rulemaking for PFAS. EPA-SAB-22-008*. Washington, D.C. Retrieved from https://sab.epa.gov/ords/sab/f?p=100:18:16490947993:::RP,18:P18_ID:2601#report

U.S. Environmental Protection Agency. (2022j). SAB PFAS Review Panel meeting. Retrieved from https://sab.epa.gov/ords/sab/f?p=100:19:7661444876021:::19:P19_ID:963#charge

U.S. Environmental Protection Agency. (2023a). Documentation for Post-IRA 2022 Reference Case.

U.S. Environmental Protection Agency. (2023b). *EPA Response to Letter of Peer Review for Disinfectant Byproduct Reduction as a Result of Granular Activated Carbon Treatment for PFOA and PFOS in Drinking Water: Benefits Analysis Related to Bladder Cancer*. Retrieved from EPA-815-B23-001

U.S. Environmental Protection Agency. (2023c). *Inventory of U.S. Greenhouse Gas Emissions and Sinks 1990-2021*. Retrieved from https://www.epa.gov/system/files/documents/2023-04/US-GHG-Inventory-2023-Main-Text.pdf

U.S. Environmental Protection Agency. (2023d). *IRIS Toxicological Review of Perfluorohexanoic Acid [PFHxA, CASRN 307-24-4] and Related Salts*. (EPA/635/R-

23/027Fa). Retrieved from https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0704tr.pdf

U.S. Environmental Protection Agency. (2023e). Power Sector Modeling.

U.S. Environmental Protection Agency. (2023f). Regulatory Impact Analyses for Air Pollution Regulations.

U.S. Environmental Protection Agency. (2023g). *Regulatory Impact Analysis for Standards of Performance for New, Reconstructed, and Modified Sources and Emissions Guidelines for Existing Sources: Oil and Natural Gas Sector Climate Review*. Washington, D.C. Retrieved from https://www.epa.gov/system/files/documents/2023-12/eo12866_oil-and-gas-nsps-eg-climate-review-2060-av16-ria-20231130.pdf

U.S. Environmental Protection Agency. (2023h). *Supplementary Material for the Regulatory Impact Analysis for the Final Rulemaking, "Standards of Performance for New, Reconstructed, and Modified Sources and Emissions Guidelines for Existing Sources: Oil and Natural Gas Sector Climate Review": EPA Report on the Social Cost of Greenhouse Gases: Estimates Incorporating Recent Scientific Advances*. Washington, D.C. Retrieved from https://www.epa.gov/system/files/documents/2023-12/epa_scghg_2023_report_final.pdf

U.S. Environmental Protection Agency. (2023i). *Work Breakdown Structure-Based Cost Model for Granular Activated Carbon Drinking Water Treatment. EPA-822-P-23-012.* Retrieved from https://www.epa.gov/system/files/documents/2022-03/gac-documentation-.pdf_0.pdf

U.S. Environmental Protection Agency. (2023j). *Work Breakdown Structure-Based Cost Model for Ion Exchange Treatment of Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water. EPA-822-P-23-013.*

U.S. Environmental Protection Agency. (2023k). *Work Breakdown Structure-Based Cost Model for Nontreatment Options for Drinking Water Compliance. EPA-822-P-23-014.* Retrieved from https://www.epa.gov/sites/default/files/2019-07/documents/wbs-nontreatment-documentation-june-2019.pdf

U.S. Environmental Protection Agency. (2023l). *Work Breakdown Structure-Based Cost Model for Reverse Osmosis/Nanofiltration Drinking Water Treatment. EPA-822-P-23-015.*

U.S. Environmental Protection Agency. (2024a). *Appendix - Office of Water Final Human Health Toxicity Assessment for Perfluorooctane Sulfonic Acid (PFOS). EPA-815-R-24-007.* Washington, D.C.

U.S. Environmental Protection Agency. (2024b). *Appendix - Office of Water Final Human Health Toxicity Assessment for Perfluorooctanoic Acid (PFOA). EPA-815-R-24-006.* Washington, D.C.

U.S. Environmental Protection Agency. (2024c). *Best Available Technologies and Small System Compliance Technologies for Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water. EPA-815-R-24-011.*

U.S. Environmental Protection Agency. (2024d). *Framework for Estimating Noncancer Health Risks Associated with Mixtures of Per- and Polyfluoroalkyl Substances (PFAS). EPA-815-R-24-003.* Washington, D.C.

U.S. Environmental Protection Agency. (2024e). *Office of Water Final Human Health Toxicity Assessment for Perfluorooctane Sulfonic Acid (PFOS). EPA-815-R-24-009.* Washington, D.C.

U.S. Environmental Protection Agency. (2024f). *Office of Water Final Human Health Toxicity Assessment for Perfluorooctanoic Acid (PFOA). EPA-815-R-24-008.* Washington, D.C.

U.S. Environmental Protection Agency. (2024g). Per- and Polyfluoroalkyl Substances (PFAS) Occurrence & Contaminant Background Support Document for the Final PFAS National Primary Drinking Water Regulation. EPA-815-R-24-013.

U.S. Environmental Protection Agency. (2024h). *PFAS National Primary Drinking Water Regulation Rulemaking Federal Register Notice*. Retrieved from https://www.govinfo.gov/content/pkg/FR-2023-03-29/pdf/2023-05471.pdf

U.S. Environmental Protection Agency. (2024i). *Technical Support Document - Technologies and Cost for Removing Per- and Polyfluoroalkyl Substances (PFAS) from Drinking Water. EPA-815-R-24-012.*

U.S. Food and Drug Administration. (2022). *Bottled Water Everywhere: Keeping it Safe*. Retrieved from https://www.fda.gov/consumers/consumer-updates/bottled-water-everywhere-keeping-it-safe

U.S. Office of Management and Budget. (2003). *Circular A-4: Regulatory Analysis*. Washington, DC: OMB

U.S. Office of Management and Budget. (2023). *Circular A-4: Regulatory Analysis*. Washington, DC: OMB Retrieved from https://www.whitehouse.gov/wp-content/uploads/2023/11/CircularA-4.pdf

Uche, U. I., Evans, S., Rundquist, S., Campbell, C., & Naidenko, O. V. (2021). Community-Level Analysis of Drinking Water Data Highlights the Importance of Drinking Water Metrics for the State, Federal Environmental Health Justice Priorities in the United States. *International journal of environmental research and public health, 18*(19), 10401.

Ullah, M., Moin, A. T., Araf, Y., Bhuiyan, A. R., Griffiths, M. D., & Gozal, D. (2020). Potential effects of the COVID-19 pandemic on future birth rate. *Frontiers in public health, 8*, 893.

Unfunded Mandates Reform Act of 1995. Public Law 104-4. 110 Stat. 48. 104th Congress. Available at https://www.gpo.gov/fdsys/pkg/PLAW-104publ4/pdf/PLAW-104publ4.pdf., (1995).

United States Energy Information Administration. (2023). *Electricity Explained: Use of Electricity*. Retrieved from https://www.eia.gov/energyexplained/electricity/use-of-electricity.php

Valvi, D., Oulhote, Y., Weihe, P., Dalgård, C., Bjerve, K. S., Steuerwald, U., & Grandjean, P. (2017). Gestational diabetes and offspring birth size at elevated environmental pollutant exposures. *Environment international, 107*, 205-215.

Vélez, M., Arbuckle, T., & Fraser, W. (2015). Maternal exposure to perfluorinated chemicals and reduced fecundity: the MIREC study. *Human reproduction, 30*(3), 701-709.

Velten, S., Hammes, F., Boller, M., & Egli, T. (2007). Rapid and direct estimation of active biomass on granular activated carbon through adenosine tri-phosphate (ATP) determination. *Water research, 41*(9), 1973-1983.

Verner, M. A., Loccisano, A. E., Morken, N. H., Yoon, M., Wu, H., McDougall, R., . . . Longnecker, M. P. (2015). Associations of Perfluoroalkyl Substances (PFAS) with Lower Birth Weight: An Evaluation of Potential Confounding by Glomerular Filtration Rate Using a Physiologically Based Pharmacokinetic Model (PBPK). *Environmental health perspectives, 123*(12), 1317-1324. doi:10.1289/ehp.1408837

Vieira, V. M., Hoffman, K., Shin, H., Weinberg, J. M., Webster, T. F., & Fletcher, T. (2013). Perfluorooctanoic acid exposure and cancer outcomes in a contaminated community: A geographic analysis. *Environmental health perspectives, 121*(3), 318-323.

Vieux, F., Maillot, M., Rehm, C. D., Barrios, P., & Drewnowski, A. (2020). Trends in tap and bottled water consumption among children and adults in the United States: analyses of NHANES 2011–16 data. *Nutrition Journal, 19*(1), 10. doi:10.1186/s12937-020-0523-6

Villanueva, C. M., Cantor, K. P., Cordier, S., Jaakkola, J. J. K., King, W. D., Lynch, C. F., . . . Kogevinas, M. (2004). Disinfection byproducts and bladder cancer: a pooled analysis. *Epidemiology, 15*(3), 357-367.

Villanueva, C. M., Cantor, K. P., King, W. D., Jaakkola, J. J., Cordier, S., Lynch, C. F., . . . Kogevinas, M. (2006). Total and specific fluid consumption as determinants of bladder cancer risk. *International Journal of Cancer, 118*(8), 2040–2047. doi:https://doi.org/10.1002/ijc.21587

Villanueva, C. M., Fernández, F., Malats, N., Grimalt, J. O., & Kogevinas, M. (2003). Meta-analysis of studies on individual consumption of chlorinated drinking water and bladder cancer. *J Epidemiol Community Health, 57*(3), 166-173. doi:10.1136/jech.57.3.166

Wang, L., Vacs Renwick, D., & Regli, S. (2019). Re-assessing effects of bromide and granular activated carbon on disinfection byproduct formation. *AWWA Water Science, 1*(4), e1147. doi:https://doi.org/10.1002/aws2.1147

Wang, Y., Adgent, M., Su, P.-H., Chen, H.-Y., Chen, P.-C., Hsiung, C. A., & Wang, S.-L. (2016). Prenatal exposure to perfluorocarboxylic acids (PFCAs) and fetal and postnatal growth in the Taiwan Maternal and Infant Cohort Study. *Environmental Health Perspectives, 124*(11), 1794-1800.

Water Research Foundation. (2016). *Residential End Uses of Water, Version 2: Executive Report*. Retrieved from https://www.circleofblue.org/wp-content/uploads/2016/04/WRF_REU2016.pdf

Waterfield, G., Rogers, M., Grandjean, P., Auffhammer, M., & Sunding, D. (2020). Reducing exposure to high levels of perfluorinated compounds in drinking water improves reproductive outcomes: evidence from an intervention in Minnesota. *Environmental Health, 19*, 1-11.

Weisman, R. J., Heinrich, A., Letkiewicz, F., Messner, M., Studer, K., Wang, L., & Regli, S. (2022). Estimating national exposures and potential bladder cancer cases associated with chlorination DBPs in US drinking water. *Environmental Health Perspectives, 130*(8), 087002. doi:https://doi.org/10.1289/EHP9985

Whelton, P. K., Carey, R. M., Aronow, W. S., Casey, D. E., Collins, K. J., Dennison Himmelfarb, C., . . . Jones, D. W. (2018). 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Journal of the American College of Cardiology, 71*(19), e127-e248.

Widefield Water and Sanitation District. (2018). *From Pilot to Full-Scale: A Case Study for the Treatment of Perfluorinated Compounds (PFCs) with Ion Exchange*. Paper presented at the AWWA Water Quality Technology Conference, Toronto, Ontario, Canada.

Wikström, S., Lin, P.-I., Lindh, C. H., Shu, H., & Bornehag, C.-G. (2020). Maternal serum levels of perfluoroalkyl substances in early pregnancy and offspring birth weight. *Pediatric research, 87*(6), 1093-1099. doi:10.1038/s41390-019-0720-1

Wikström, S., Lindh, C. H., Shu, H., & Bornehag, C.-G. (2019). Early pregnancy serum levels of perfluoroalkyl substances and risk of preeclampsia in Swedish women. *Scientific reports, 9*(1), 1-7.

Wilder, L., Worley, R., & Breysse, P. (2017). DIRECT FROM ATSDR: Community Exposures to Per-and Polyfluoroalkyl Substances in Drinking Water: A National Issue. *Journal of Environmental Health, 80*(2), 38-41.

Williams, O. (1989). The Atherosclerosis Risk in Communities (ARIC) study-design and objectives. *American journal of epidemiology, 129*(4), 687-702.

Windham, G., & Fenster, L. (2008). Environmental contaminants and pregnancy outcomes. *Fertility and sterility, 89*(2), e111-e116.

Wright, J., Larsen, A., Rappazzo, K., Ru, H., Radke, E., & Bateson, T. (2023). Systematic review and meta-analysis of birthweight and PFNA exposures. *Environmental research*, 115357.

Yang, Q., Guo, X., Sun, P., Chen, Y., Zhang, W., & Gao, A. (2018). Association of serum levels of perfluoroalkyl substances (PFASs) with the metabolic syndrome (MetS) in Chinese male adults: A cross–sectional study. *Science of the total environment, 621*, 1542-1549. doi:10.1016/j.scitotenv.2017.10.074

Yao, Q., Gao, Y., Zhang, Y., Qin, K., Liew, Z., & Tian, Y. (2021). Associations of paternal and maternal per-and polyfluoroalkyl substances exposure with cord serum reproductive hormones, placental steroidogenic enzyme and birth weight. *Chemosphere, 285*, 131521.

Yapsakli, K., & Çeçen, F. (2010). Effect of type of granular activated carbon on DOC biodegradation in biological activated carbon filters. *Process Biochemistry, 45*(3), 355-362. doi:https://doi.org/10.1016/j.procbio.2009.10.005

Yin, X., Di, T., Cao, X., Liu, Z., Xie, J., & Zhang, S. (2021). Chronic exposure to perfluorohexane sulfonate leads to a reproduction deficit by suppressing hypothalamic kisspeptin expression in mice. *Journal of Ovarian Research, 14*, 1-13.

Yu, J., Lv, L., Lan, P., Zhang, S., Pan, B., & Zhang, W. (2012). Effect of effluent organic matter on the adsorption of perfluorinated compounds onto activated carbon. *Journal of hazardous materials, 225*, 99-106.

Yuan, J., Passeport, E., & Hofmann, R. (2022). Understanding adsorption and biodegradation in granular activated carbon for drinking water treatment: A critical review. *Water research, 210*, 118026. doi:https://doi.org/10.1016/j.watres.2021.118026

Zachman, B. A., & Summers, R. S. (2010). Modeling TOC breakthrough in granular activated carbon adsorbers. *Journal of Environmental Engineering, 136*(2), 204-210.

Zaggia, A., Conte, L., Falletti, L., Fant, M., & Chiorboli, A. (2016). Use of strong anion exchange resins for the removal of perfluoroalkylated substances from contaminated drinking water in batch and continuous pilot plants. *Water research, 91*, 137-146.

Zahm, S., Bonde, J. P., Chiu, W. A., Hoppin, J., Kanno, J., Abdallah, M., . . . Dorman, D. C. (2024). Carcinogenicity of perfluorooctanoic acid and perfluorooctanesulfonic acid. *The Lancet Oncology, 25*(1), 16-17.

Zhang, Y., Vittinghoff, E., Pletcher, M. J., Allen, N. B., Zeki Al Hazzouri, A., Yaffe, K., . . . Ives, D. G. (2019). Associations of Blood Pressure and Cholesterol Levels During Young

Adulthood with Later Cardiovascular Events. *Journal of the American College of Cardiology, 74*(3), 330-341.