



E C O S

Processes & Considerations for Setting State PFAS Standards

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Supported by & in conjunction with the ECOS PFAS Caucus

Executive Summary

In recent years, federal, state, and international authorities have established various health-based regulatory values and evaluation criteria for a number of specific per- and poly-fluoroalkyl substances (PFAS) in response to growing concerns with contamination. At this time, the U.S. has no federally enforceable PFAS standards, leaving individual states to navigate various avenues for addressing PFAS contamination. Some states have established legally enforceable values for certain PFAS in drinking water, groundwater, surface water, soil, or other environmental media (e.g., drinking water Maximum Contaminant Levels [MCLs]). Other states and regulatory agencies have opted for non-enforceable values such as guidance levels, screening numbers, or advisories that may apply to PFAS compounds for which promulgated standards do not exist.

The Environmental Council of the States (ECOS) in 2019 compiled information on state PFAS standards, advisories, and guidance values (hereinafter referred to as “guidelines”¹). Sharing data and regulatory approaches will help federal, state, and international authorities avoid unnecessary duplication of efforts, as well as understand and communicate about differences in guidelines. This paper outlines ECOS’ findings on state efforts and considerations for future regulatory activities on PFAS.

¹ For the purposes of this white paper, the term “guidelines” will apply to both regulatory (enforceable) standards and non-regulatory (non-enforceable) values.

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List of Acronyms

ACRONYM FULL PHRASE

ACGIH	American Conference of Governmental Industrial Hygienists
ACWA	Association of Clean Water Administrators
AFFF	Aqueous film-forming foam
APFO	Ammonium perfluorooctanoate
ASDWA	Association of State Drinking Water Administrators
ASTM	ASTM International (formerly American Society for Testing and Materials)
ATSDR	Agency for Toxic Substances and Disease Registry
BMDL	Benchmark dose (lower confidence limit)
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CSF	Cancer slope factor
CWA	Clean Water Act
DOD	U.S. Department of Defense
ECOS	Environmental Council of the States
EPA	U.S. Environmental Protection Agency
ESL	Effect Screening Level
FTE	Full-time employee
GAC	Granular activated carbon
HBV	Health-Based Value
HED	Human equivalent dose
HFPO-DA	Hexafluoropropylene oxide dimer acid; GenX
HRL	Health Risk Limit
ITRC	Interstate Technology and Regulatory Council
ITSL	Interim Threshold Screening Level
kg	Kilogram
L	Liter
LHA	U.S. EPA Lifetime Health Advisory
LOAEL	Lowest Observed Adverse Effect Level
MCL	Maximum Contaminant Level
mg	Milligram

MLA	Multi-linear array (SGS Axys method)
MPART	Michigan PFAS Action Response Team
MRL	Minimal risk level
NDAA	National Defense Authorization Act
NGO	Non-governmental organization
NOAEL	No Observed Adverse Effect Level
NPDES	National Pollutant Discharge Elimination System
NRWQC	National Recommended Water Quality Criteria
PFAS	Per- and polyfluoroalkyl substances
PFBA	Pentafluorobutanoic acid
PFBS	Perfluorobutanesulfonic acid
PFDA	Perfluorodecanoic acid
PFHpA	Perfluoroheptanoic acid
PFHxA	Perfluorohexanoic acid
PFHxS	Perfluorohexane sulfonic acid
PFIB	Perfluoroisobutylene
PFNA	Perfluorononanoic acid
PFOA	Perfluorooctanoic acid
PFOS	Perfluorooctane sulfonate
PFOSA	Perfluorooctanesulfonamide
POD	Point of Departure
ppm	Parts per million
ppt	Parts per trillion
PWS	Public water system
RCRA	Resource Conservation and Recovery Act
RfD	Reference Dose
RSC	Relative Source Contribution
RSL	Regional Screening Level
SDWA	Safe Drinking Water Act
SPLP	Synthetic precipitation leaching procedure
TOF	Total organic fluorine
TOP	Total oxidizable precursor
TSCA	Toxic Substances Control Act

Introduction

PFAS are a group of synthetic chemicals used in a wide array of consumer and industrial products since the 1940s. Several decades later, publicly available studies on certain PFAS risks indicated potential human health concerns related to these chemicals. In 2000, 3M announced a voluntary phase-out of certain legacy PFAS (e.g., perfluorooctanoic acid [PFOA], perfluorooctane sulfonate [PFOS], perfluorohexane sulfonic acid [PFHxS]). In 2006, the U.S. Environmental Protection Agency (EPA) initiated the PFOA Stewardship Program, which encouraged eight major chemical manufacturers to eliminate the use of PFOA and similar long-chain² PFAS in their products and in the emissions from their facilities.³ Despite these actions, U.S. manufacturers can still import PFOA, PFOS, and PFHxS for use in consumer goods, and some U.S. sites are legally required to keep PFAS-containing firefighting foams on-site for emergencies.

U.S. manufacturers have developed numerous PFAS chemicals (e.g., hexafluoropropylene oxide dimer acid [HFPO-DA; GenX], a Chemours [formerly DuPont] PFAS used as a PFOA replacement), to replace long-chain PFAS such as PFOA, PFOS, and PFNA. These replacement chemicals are part of the larger suite of nearly 5,000⁴ PFAS compounds, some of which the EPA has approved for manufacturing and use in the U.S. This is a problem on many fronts: PFAS do not break down or, in the case of precursors, are converted to terminal PFAS that do not break down. Therefore, there is a permanent “supply” of PFAS in the environment that maintain their chemical structures and potential toxicity, in contrast to many other organic compounds. In addition, regulators currently lack routinely available analytical methods for PFAS detection and measurement across most environmental media and have little, if any, toxicological data for the majority of PFAS (especially the precursors) to define risks to human and ecological receptors.

In 2016, the EPA updated its short-term Provisional Health Advisory values for PFOA (400 parts per trillion [ppt]) and PFOS (200 ppt) to a Lifetime Health Advisory (LHA) of 70 ppt for PFOA and PFOS, individually or in combination, in finished drinking water. The EPA states that this LHA was calculated “to provide Americans, including the most sensitive populations, with a margin of protection from a lifetime of exposure to PFOA and PFOS from drinking water.”⁵ The LHA is a non-regulatory and non-legally enforceable value, but is intended to provide guidance to federal, state, and municipal governments for addressing PFOA and PFOS contamination in public water systems and private potable wells. In February 2019, the EPA released its [PFAS Action Plan](#) in which the agency committed to make a “regulatory determination” under the Safe Drinking Water Act (SDWA) by the end of 2019. The EPA sent the regulatory determination to the Office of Management and Budget in December 2019 for interagency review, but as of February 13, 2020, the agency had not yet released the determination to the public. A regulatory determination is a formal decision on whether the EPA should initiate a process to develop a national primary drinking water regulation for a specific contaminant. The SDWA requires the EPA to make regulatory determinations for at least five contaminants from the most recent drinking water Chemical Candidate List⁶ within five years of the completion of the previous round of regulatory determinations. This determination may initiate the rulemaking process to establish an enforceable National Primary Drinking Water Regulation (i.e., MCL) for PFOA and PFOS. If the EPA develops an MCL under its existing guidance, the process is likely to take years due to the necessary technical evaluation, public comment, and rulemaking procedures.

² Long-chain PFAS are those with carbon chain lengths of 6 or higher for sulfonic acids like PFOS and PFHxS, and carbon chain lengths of 8 or higher for carboxylic acids like PFOA and perfluorononanoic acid (PFNA).

³ History and Use of Per- and Polyfluoroalkyl Substances (PFAS) [Fact Sheet](#), ITRC (2017).

⁴ [FDA PFAS](#)

⁵ [EPA Drinking Water Health Advisories for PFOA and PFOS](#)

⁶ The EPA’s [Chemical Candidate List](#) is a list of contaminants that are currently not subject to proposed or promulgated national primary drinking water regulations, but are known or anticipated to occur in public water systems.

In 2018, the U.S. Health and Human Services' Agency for Toxic Substances and Disease Registry (ATSDR) developed draft [minimal risk levels](#) (MRLs) for four PFAS compounds: PFOA, PFOS, PFHxS, and PFNA. MRLs are not regulatory values and are not intended to be used as public water standards. MRLs are screening tools to identify contaminants of concern at hazardous waste sites. If an exposure is below an MRL, it is not expected to result in adverse health effects, whereas an exposure exceeding an MRL warrants further investigation to determine if the exposure might harm human health. Additionally, MRLs are presented as dosage amounts (a measurement of exposure in units of milligrams/kilogram/day) and not in terms of concentration (the amount of a substance present in a particular media in units of parts per million [ppm] or ppt). These differences have resulted in public confusion and emphasize the need for improved risk communication, especially in the news media, to explain that MRLs and the EPA's LHAs are used in different situations and are not/should not be considered "equivalent."

Historically, many states relied on the promulgated standards from federal agencies to regulate chemicals, while other states have had the authority to develop their own standards for contaminants of concern for years. If no federal standard exists, states may rely on [EPA hierarchical values](#) or similar reference documents. Noting the complexity of the class of PFAS chemicals, the need for cross-media consideration, and the absence of a promulgated federal standard, states have taken alternative routes to actively address PFAS across a wide range of programs. At least 14 states⁷ have developed draft, proposed, or final health-based regulatory and/or guidance values for several PFAS compounds in drinking water, groundwater, or surface water.⁸ These guidelines may significantly differ from the EPA's LHA and from state-to-state given various legislative and scientific considerations. For example, states may have different mandates (e.g., regulations, policies) that direct them to interpret toxicity data (including considering exposures to sensitive life stages like infants or pregnant women) to develop risk assessments or require them to use EPA risk assessments as the bases for their guidelines. Several states have developed drinking water guidelines for PFOA and PFOS that are lower than the EPA's LHA due to considerations of more recent scientific information, more sensitive toxicological endpoints, and/or more stringent exposure parameters. Many of these states have also developed guidelines for various PFAS in addition to PFOA and PFOS. Other states have adopted the EPA's LHA for PFOA and PFOS in drinking water and/or groundwater to guide their efforts upon detection of contamination.⁹

With a growing body of science to inform standard development, an absence of a federally enforceable standard, and pressures from the public and legislative bodies to take regulatory action, it is important to know which states are setting guidelines, understand how the guidelines are developed, and be able to educate legislators on differences between state, federal, and other guidelines. This is essential so that states can make informed decisions when implementing their own regulations and/or risk communication practices.

[Overview of States' PFAS Guidelines](#)

ECOS surveyed states on their processes, rulemaking requirements, and other considerations for establishing PFAS guidelines (e.g., occurrence of specific PFAS in drinking water sources or other environmental media). ECOS and its working group of state environmental agency officials (the PFAS Caucus) examined responses from *23 states*

⁷ Several states in addition to those that completed the ECOS survey are known to have drafted, proposed, or finalized health-based regulatory and/or guidance values for PFAS in various environmental media. They are not included in the facts and figures outlined in this report.

⁸ See the Interstate Technology and Regulatory Council's [ITRC] [Sections 4 and 5 Tables](#) in its PFAS regulations fact sheet. ITRC is a subsidiary of ECOS.

⁹ The health basis for standards for other emerging contaminants may be as low as those for PFAS compounds, but the actual standards for other emerging contaminants are often higher because they are based on analytical limitations, while the PFAS standards can be set at the health-based levels.

(Alabama, Alaska, Arizona, California, Colorado, Florida, Indiana, Kansas, Massachusetts, Michigan, Minnesota, Missouri, Nebraska, New Hampshire, New Jersey, North Carolina, Oklahoma, Oregon, Tennessee, Texas, Vermont, Wisconsin, Wyoming).¹⁰ Below are findings and conclusions from the 23 states that completed the ECOS survey.

States without PFAS Guidelines

*Eight states (Alabama, Arizona, Kansas, Missouri, Nebraska, Oklahoma, Tennessee, Wyoming) indicated that they do not have state guidelines.*¹¹

Reasoning for Not Establishing State PFAS Guidelines:

- *Five states (Arizona, Indiana, Missouri, North Carolina, and Oklahoma)*¹² have restrictions that prohibit them from setting a drinking water or groundwater guideline more stringent (i.e., more protective) than a federal standard in at least one media. This could dissuade a state from setting a PFAS standard (at any level), or from setting a PFAS standard lower than the EPA's LHA in anticipation that a federal MCL may be enacted at a similar level, forcing the state to amend its guideline(s) in a way that appears to "weaken" it.
- Many states lack the capacity or resources to effectively and individually regulate PFAS. Barriers include lack of technical expertise needed for toxicity interpretation and standard development, labs certified to test for PFAS in the state, and legislative support and funding. Several states noted the need for more peer-reviewed science to make informed decisions on whether to establish guidance levels for some of the PFAS that have been found in their environmental media.

Without their own state-based guidelines, several of these states are still taking non-regulatory actions to monitor for PFAS. Efforts include statewide sampling of Public Water Systems (PWSs) and surface water and groundwater intakes, conducting inventories of facilities that use or have used or produced PFAS, responding to drinking water and fish contamination, and forming interagency task forces to coordinate on addressing PFAS within the state.

States with PFAS Guidelines

*15 states (Alaska, California, Colorado, Florida, Indiana, Massachusetts, Michigan, Minnesota, New Hampshire, New Jersey, North Carolina, Oregon, Texas, Vermont, Wisconsin) have a guideline for at least one PFAS analyte in at least one environmental medium.*¹³

State guidelines specified in ECOS' survey have been incorporated into the ITRC's [Sections 4 and 5 Tables](#) in its PFAS regulations fact sheet. The tables define to which environmental medium each standard applies, as well as whether the values are promulgated or advisory. States may have slightly different definitions of each medium. For example, most states consider drinking water standards to be finished water from the PWSs, but a state may also include groundwater used as drinking water from a private residential well or similar source. ECOS compiled responses based on how the state categorized each medium in the survey and how it defines it generally for the public. For more detailed state-specific definitions, see [state PFAS websites](#).

¹⁰ Individual state PFAS websites can be found in the "Overview" section on ECOS' [PFAS Risk Communication Hub](#).

¹¹ These states may use the EPA's LHA of 70 ppt as guidance, remediation goals, action levels, or for regulatory oversight if PFAS contamination is detected. However, they will likely wait for a federal standard before enacting their own state guidelines.

¹² Indiana and North Carolina are included in this list because they have such a law. However, they have a guideline for at least one PFAS analyte, as indicated below.

¹³ These include promulgated rules and advisories (e.g., action and notification levels, cleanup target levels, initiation levels), and may be determined by the state or may be consistent with EPA's LHA of 70 ppt.

Of the states that responded to ECOS' survey, the following have different types of guidelines:

Regulatory Standards

- Drinking Water¹⁴: *Five states (Massachusetts [proposed], Michigan [proposed], New Hampshire, New Jersey, Vermont)*
- Groundwater: *Nine states (Alaska, Colorado, Massachusetts, Michigan, New Hampshire, New Jersey, North Carolina, Texas, Vermont)*
- Surface Water: *Two states (Michigan, Minnesota [site-specific criteria])*
- Soil: *Six states (Alaska, Massachusetts, Michigan, Texas, Vermont, Wisconsin)*
- Air: *Two states (Michigan, New Hampshire)*

Advisory Guidelines

- Drinking Water: *Six states (Alaska, California, Massachusetts, Minnesota, North Carolina, Vermont)*
- Groundwater: *Three states (Florida, Minnesota, Wisconsin)*
- Surface Water/Wastewater: *One state (Oregon)*
- Soil: *Three states (Florida, Indiana, Minnesota)*
- Air: *One state (Texas)*
- Water Interface: *One state (Alaska)*
- Fish or Wildlife Consumption Advisories: *Four states (Michigan [fish and deer], Minnesota, New Jersey, Wisconsin)*

States with a Final or Proposed MCL (Drinking Water Only)

- *Massachusetts* (Proposed for Six PFAS)
- *Michigan* (In Process for Seven PFAS)
- *New Hampshire* (Enacted for Four PFAS, Individually)
- *New Jersey* (Enacted for PFNA, Proposed for PFOA and PFOS)
- *Vermont* (In Process for Five PFAS)
- *Wisconsin* (In Process for PFOA and PFOS)

Grouping PFAS

Recently proposed congressional legislation suggested creating a federal MCL for a sum of total PFAS, derived by adding the concentration of each PFAS detected in a sample. This total PFAS concentration depends on which analytical methods researchers use, as different analytical methods detect different numbers of PFAS. Given that there are nearly 5,000 PFAS, most of which have little known information about their toxicities, many regulators and subject-matter experts advise against grouping PFAS as an entire class. Some states regulate PFOA and PFOS, individually or in combination, as EPA does in its LHA. Other state guidelines are based on the total concentration of PFOA, PFOS, and several additional long-chain PFAS analytes.

States' approaches for grouping PFAS, and the reasoning provided for grouping PFAS under each method, are as follows:

¹⁴ See States with a Final or Proposed MCL (Drinking Water Only) designation below.

Individual PFAS

- 12 states
 - *Alaska*: Soil and groundwater cleanup levels for PFOA, PFOS
 - *California*: Non-regulatory notification levels for PFOA, PFOS in drinking water
 - *Florida*: Provisional Soil Cleanup Target Levels for PFOA, PFOS; Provisional Irrigation Water Screening Levels for PFOA, PFOS, Surface Water Screening Levels for fish consumption for PFOA, PFOS
 - *Indiana*: Guidance Remediation Screening Levels for PFBS in soil
 - *Michigan*: Proposed MCLs for 7 PFAS (PFOA, PFOS, PFNA, PFHxA, PFHxS, PFBS, GenX); Surface Water Quality Standards for PFOA, PFOS; Soil criteria for PFOA, PFOS; Consumption advisories for PFOS in fish and deer tissue; Initial Threshold Screening Levels (ITSLs) for PFOA, PFOS in air; Michigan has developed and is currently developing ITSLs for several other PFAS including 6:2 fluorotelomer sulfonate and PFIB
 - *Minnesota*: Promulgated Health Risk Limits (HRLs) for PFOA, PFOS, PFBA, PFBS in groundwater¹⁵; Health-Based Values (HBVs) for PFOS, PFBS, PFHxS in groundwater; Rule-based Intervention Limits for PFOA, PFOS, PFBA, PFBS to protect surface water and groundwater at solid waste facilities; Soil Reference Values for PFOA, PFOS, PFBS, PFBA, PFHxS; Site-Specific Criteria for PFOA, PFOS in surface water; Fish Consumption Advice and Sediment Quality Target for PFOS
 - *New Hampshire*: MCLs and Ambient Groundwater Quality Standards for PFOA, PFOS, PFHxS, PFNA; Ambient Air Limit for APFO
 - *New Jersey*: MCL and Groundwater Quality Standard for PFNA; Interim Groundwater Quality Standards for PFOA, PFOS; Proposed MCLs and Groundwater Quality Standards for PFOA, PFOS; Fish Consumption Advisories for PFOS in some waterbodies
 - *North Carolina*: Groundwater Interim Maximum Allowable Concentration for PFOA; Non-Regulatory Drinking Water Health Goal for HPFO-DA, “GenX”
 - *Oregon*: Initiation levels for PFOA, PFOS, PFNA, PFHpA, PFOSA in municipal wastewater effluent
 - *Texas*: Health-Based Non-Carcinogenic Toxicity Factors and Cleanup Values for 16 PFAS (including PFOA and PFOS) in soil and groundwater; interim short- and long-term Effects Screening Levels (ESLs) for PFOA, PFOS in air permitting
 - *Wisconsin*: Regional Screening Levels (RSLs) for PFOA, PFOS, PFBS in Soil
- Reasoning:
 - Risk assessors evaluate PFAS analytes individually in the regulatory determination process. Regulations are therefore based on conclusions that human health effects, analytical limitations, and removal of drinking water contaminants vary by analyte.
 - Regulations vary based on the potential for each chemical to be found in a state, availability of chemical guidelines used for testing, and ability of available labs to test for and measure that analyte. States with more limited contamination potential and evaluations of health effects may be waiting to see whether the EPA develops a technical basis for grouping PFAS before summing or regulating additional analytes.
 - Toxicologists have more data on the terminal PFAS compounds (i.e., breakdown products/analytes), and less on the precursors which may be considered as PFAS in the same family.
 - Toxicological studies demonstrate differences in the potency and bioaccumulation (i.e., physiological half-lives) between individual PFAS.

¹⁵ Minnesota’s Health Risk Limits and Health-Based Values for groundwater are also used as guidance values for drinking water.

PFOA & PFOS, Summed

- *Five states*
 - *Alaska*: Drinking water action level for PFOA and PFOS
 - *Colorado*: Site-specific groundwater standard for PFOA and PFOS
 - *Florida*: Provisional Groundwater Cleanup Target Level for PFOA and PFOS, individually or combined
 - *Michigan*: Groundwater cleanup standard for PFOA and PFOS
 - *Wisconsin*: Recommended groundwater enforcement standard and recommended groundwater preventive action limit for PFOA and PFOS (individual and summed)
- Reasoning:
 - PFOA and PFOS are the most thoroughly studied of the long-chain PFAS compounds, with a large quantity of publicly available toxicity information available.
 - Regulating PFOA and PFOS aligns with the EPA's LHA. While the EPA has developed draft toxicity factors for a few other PFAS, PFOA and PFOS remain the only analytes with federal health advisories.
 - PFOA and PFOS (and other PFAS in a few states) may be considered hazardous substances or otherwise listed as a similar toxicant under a state law.

More than 2 PFAS, Summed

- *Three states*
 - *Massachusetts*: Proposed MCL and final groundwater cleanup standard for the sum of 6 PFAS (PFOA, PFOS, PFNA, PFHpA, PFHxS, PFDA)
 - *Minnesota*: MN's [Health Risk Limits Rules for Groundwater](#) require evaluation of exposure to multiple contaminants in groundwater. Hazard ratios are summed across contaminants that affect the same health endpoints. For example, PFOA, PFOS, PFHxS, and PFBA all affect liver and hazard ratios for each of these contaminants, and would therefore be added together to calculate a multiple contaminant health risk index.
 - *Vermont*: Proposed MCL and promulgated groundwater standard for the sum of 5 PFAS (PFOA, PFOS, PFNA, PFHpA, PFHxS)
- Reasoning: Many of the summed PFAS analytes are similar as indicated below:
 - They are long-chain compounds with similar chemical structures (+/- two carbons in chain length) to PFOA and PFOS.
 - They are often found together in the environment, and have characteristically similar bioaccumulative patterns and fate and transport mechanisms.
 - Human exposures to these PFAS often are correlated, making it difficult to differentiate the contributions of the individual PFAS to health effects observed in humans.
 - Their toxicity is assumed to be additive based on a substantial body of publicly available data indicating that they cause similar toxicological effects, have long serum half-lives in humans (long-chain PFAS only), and are associated with similar health effects in humans.¹⁶
 - They have similar limits for lab detection via EPA Method 537 (see Analytical Methods on page 17), and there is a minimal cost difference between analyzing a few or 24 compounds, so regulating and requiring

¹⁶ On the other hand, though similar, these PFAS do still present differences (e.g., different levels at which toxicity occurs, different toxicological effects and modes of action) that a state might acknowledge as a reason *not* to group the chemicals, but rather to regulate them individually.

testing for more analytes does not increase the cost and lessens the potential for the need to resample in the future.

- PFOA, PFOS, PFNA, PFHxS, PFHpA, and PFBS were the six PFAS included in the EPA's third round of the Unregulated Contaminant Monitoring Rule (UCMR3). These PFAS have been researched to the extent that they are regulated individually by some states. PFHpA has minimal toxicity data available and PFDA was not in UCMR3, but some states regulate both PFAS with the other six long-chain PFAS based on close structural similarity.
- Regulating more analytes can provide information on conceptual site model development and the potential for PFAS fingerprinting (chemical forensics).

Evaluating Differences among States' PFAS Guidelines

One of the most common questions that states are asked to address when communicating risks to the public and co-regulators is why guidelines vary from state-to-state. Many of the states' derived values typically differ within a factor of two to three, indicating that they are similarly protective; however, this is difficult to communicate with audiences who lack a background in the scientific and regulatory basis for the guidelines. Consequently, communicating the rationale for varying guidelines among state and federal entities remains a challenge.

States report that deviations among PFAS guidelines are driven by several main factors:

- Differences in professional judgments regarding the choice of the critical study and endpoint, the method for animal-to-human extrapolation, the uncertainty factors, and exposure parameters such as the Relative Source Contribution. Differences in any one of these choices (described in more detail in the State Trends for the Basis of Guidelines section on page 14) will result in different numerical values for the PFAS standard being developed.
- Differences in timing. *When* guidelines are developed and *when* a state looks at the available scientific information affects *what* the guidelines are. While many technically sound guidelines have been developed from older studies, toxicologists continue to conduct new PFAS research that will provide states with more referential data for deriving values. In this fast-paced field, short timeframes can change what studies relevant to PFAS standard development are available.
- Differences in state legislative or rulemaking requirements. The next section of this paper will explore differences in legislative procedures, but it should also be noted that beyond legislatures, state environmental and health agency programs (e.g., drinking water, surface water, and wastewater) have varying priorities or responsibilities in the standard-setting process.
- Differences in state regulatory processes and histories. States have different histories of developing standard methods, enacting regulations, and setting policy, all of which may direct toxicologists to use specific approaches and require protection of certain human life stages/vulnerable populations or other factors. Minnesota, for example, is required to evaluate risks to pregnant women and children in its exposure assumptions. These factors, coupled with how well a state's standard-setting methods reflect current and evolving science, can greatly affect how guidelines are calculated and what the resulting values are.

Section I. Legislative Considerations

Rulemaking Capacities

ECOS asked states to describe what authorities and processes they had to set PFAS guidelines. Responses indicate that most state guidelines are adopted/enacted through general rulemaking processes outlined in state

administrative policies or acts, while some states have bills or statutes specifically directed at PFAS. For example, the California Department of Toxic Substances Control's Safer Consumer Products Program lists PFAS as Candidate Chemicals and evaluates PFAS in consumer products like carpets in accordance with its Safer Consumer Products Regulations. Several states described their active PFAS bills prohibiting AFFF firefighting foams, regulating food packaging, and requiring PFAS sampling, among other actions. States active in PFAS regulation are typically backed by their legislators, Attorneys General, and other leadership entities that provide funding and direct the environmental agencies to take action on contamination. Such actions include forming task forces for improved coordination (e.g., Michigan), setting guidelines in different media by certain dates (e.g., Vermont), or initiating directives or lawsuits against PFAS manufacturers (e.g., New Jersey, Minnesota).

Enforcement of state regulations is typically a programmatic issue based on the contaminated medium and is conducted in accordance with rules or policies in effect for each regulatory program (e.g., Superfund and hazardous waste, Resource Conservation and Recovery Act [RCRA], SDWA). Consequently, enforcement efforts for PFAS in drinking water, groundwater, surface water, solid waste, biosolids, and other environmental media are led by the state agency with authority to administer the applicable rules, and would be conducted as directed by program rules, unless specific rules for PFAS have been adopted. Enforcement may occur if a regulatory standard is exceeded, the contamination is considered hazardous, or there is a requirement for assessment and remediation. Some states noted that PFAS enforcement is a challenge without having adequate toxicity data necessary to establish the criteria on which a permit limit or enforcement/remediation action is based.

Regulating PFAS as Hazardous

Nine states (Alaska, Florida, Indiana, Massachusetts, Minnesota, New Hampshire, Vermont, Wisconsin, and Wyoming) noted that they have emergency rulemaking powers in the event of a PFAS contamination event or if a PFAS compound is declared hazardous at the federal level.

New Jersey in 2018 [listed](#) PFNA as a hazardous substance and recently proposed adding PFOA and PFOS to the NJ Hazardous Substance List.

In its PFAS Action Plan, the EPA outlined its intent to explore hazardous substance definitions for PFOA and PFOS. Similarly, Congress recently considered a number of PFAS issues in its National Defense Authorization Act (NDAA), including a bill seeking to designate all PFAS as hazardous substances under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA). While these provisions were ultimately removed from NDAA for Fiscal Year 2020 ([Senate Bill 1790](#), which became law on December 20, 2019), several lawmakers stressed their intent to consider hazardous waste definitions in future rules.

Declaring PFAS (just PFOA and PFOS, or additional analytes) as hazardous under CERCLA would have some, though likely different, impacts on states. North Carolina notes that the declaration may provide more information to its rulemaking body, although its environmental agency is unsure if it will speed up the water quality criteria adoption process. Other states note that empowering them to act using existing regulatory CERCLA mechanisms allows for an expedited cleanup process and prevents draining already-strained funds for site investigation and characterization. Kansas said this definition is what it needs to regulate PFAS, as the state's definition of a hazardous substance is based on its inclusion as a CERCLA hazardous substance.

Intra-State PFAS Collaboration

States have varying procedures for designating who regulates PFAS. Many state environmental agencies are coordinating with their health, agriculture, and other state agency counterparts on the state's PFAS response. For

example, the Michigan PFAS Action Response Team (MPART) was created in 2017 through an executive directive to investigate sources and locations of PFAS and protect drinking water and public health. In 2019, MPART was signed into an executive order as an enduring advisory body of seven state agencies, led by the Michigan Department of Environment, Great Lakes, and Energy. Other states (e.g., Colorado, Connecticut, Maine, New York, Ohio, Pennsylvania, and Wisconsin) have formed similar task forces and action teams charged with recommending PFAS guidelines and conducting other statewide PFAS efforts.

Impacts of Federal Legislative Uncertainty

ECOS asked states that have already established guidelines how they think a federal MCL (as currently being considered by the EPA) or similarly enforceable federal PFAS standard would impact their regulations. A state may be required to modify its guidelines to be “no more stringent than” federal requirements, or a state may be required to “strengthen” its guidelines so that they are as protective as federal standards. North Carolina noted that a federal MCL could affect its groundwater programs, and another state noted its concern that a federal MCL may or may not adequately address protection for all populations and impacted communities because MCLs are not strictly risk-based. Should the EPA enact an enforceable drinking water standard, some states may need to make challenging management decisions regarding how to adjust their existing guidelines and PFAS response efforts.

Section II. Risk Assessment

State environmental and public health agencies use quantitative risk assessment to develop health-based criteria for PFAS guidelines. The processes for evaluating exposure and developing these criteria are described across several guidance documents produced by the EPA.¹⁷

At its core, risk assessment is used to develop the human health basis for guidance values or standards by considering the following:

$$\textit{Toxicity} \times \textit{Exposure} = \textit{Risk}$$

Risk is a function of the toxicity of a chemical and a person’s exposure to that chemical. The higher one’s exposure, the greater the risk; similarly, the more toxic a chemical is, the more risk there is at the same level of exposure. Both variables are fundamental to the resulting calculation of risk.

As described in more detail below, differences among state PFAS guidelines may arise from differences in toxicity factors, which include Reference Doses (RfDs) for non-cancer effects and Cancer Slope Factors (CSFs) for carcinogenic effects. These toxicity factors are developed based on toxicity studies in either humans or animals. Choices in scientific study and toxicity endpoint used, as well as choices made in developing an RfD or CSF from the selected study and endpoint, will result in differences in the numerical values of these toxicity factors.

Different guidelines may also result from variations in exposure factors, which include parameters relating to daily water ingestion, body weight of an individual, duration of exposure, and fraction of total exposure from the medium of concern (e.g., drinking water). As with toxicity factors, state agencies use evidence-based methods to characterize exposure factors.

¹⁷ Examples of these EPA guidance documents include the [Risk Assessment Guidelines](#), [Water Quality Standards Handbook](#), and [Exposure Factors Handbook](#) (2011).

Scientific Considerations, Professional Judgment, & Peer Review

In general, states prefer to use peer-reviewed, publicly available toxicity studies that meet risk assessment criteria (e.g., study duration, route of exposure) as the basis for their guidelines. In some cases, states will consider non-peer reviewed reports (e.g., contract lab reports or National Toxicology Program data tables). Regulators review studies to ensure that they were properly conducted and reported, and consider a study's results coupled with its relevance, degree of rigor, and importance to the question on hand. Some states routinely develop their own guidelines for chemicals of interest to their state; however, if the EPA completes this process first, states can review the agency's conclusions and decide whether to use them, saving the state the effort of doing it on its own. When EPA values are not available, some states refer to ATSDR's draft MRLs (like they would RfDs) or use health-protective values from other agencies like the American Conference of Governmental Industrial Hygienists (ACGIH).

Toxicity Criteria & Methodology

Regulatory agencies may rely on a chemical-by-chemical approach or grouping approaches for developing PFAS toxicity criteria (e.g., RfDs for non-carcinogens and CSFs for carcinogens). Most states conducting their own evaluations do not rely solely on EPA or ATSDR risk assessments, for which there are only published documents supporting the EPA's LHA for PFOA and PFOS, draft toxicity documents and RfDs for PFBS and GenX, and draft MRLs from ATSDR. Performing the scientific analysis needed to effectively regulate PFAS is time consuming and regulators lack toxicological data needed to develop criteria for some PFAS analytes detected in environmental media.

To develop health-based guidelines, agencies conduct risk assessments, which usually follow this sequence of events:

1. Review available studies (e.g., toxicological, epidemiological) to identify critical endpoints that are sensitive and relevant to humans.

While most scientists prefer human epidemiological information as the basis for guidelines when appropriate, the EPA and states have concluded that currently available human studies are not yet sufficient to use as the primary basis for PFAS guidelines. As such, all current federal and state PFAS guidelines are based on laboratory animal study data that are then translated.¹⁸ For PFOA and PFOS, the EPA and some states have identified developmental effects (e.g., decreased pup body weight, thyroid effects [PFOS]; accelerated puberty; delayed ossification, delayed mammary gland development, neurobehavioral and skeletal effects [PFOA]; hepatic [liver] toxicity, immune system suppression [PFOA, PFOS]) as critical endpoints. Critical endpoints can vary from state-to-state based on scientific judgments.

2. Determine a point of departure (POD), the spot on the dose-response curve from the animal study at which toxicologists begin to apply uncertainty factors (UFs). PODs can be a No Observed Adverse Effect Level (NOAEL), Lowest Observed Adverse Effect Level (LOAEL), or Benchmark Dose (lower confidence limit; BMDL). BMDL is the preferred POD when available, as it is less dependent on dose selection and sample size.

Toxicologists typically adjust the POD to account for the much slower excretion rate of PFAS in humans than animals (i.e., calculating human equivalent doses [HEDs] that will result in an equivalent internal dose [serum

¹⁸ This may not be true internationally, as the European Food Safety Authority has used epidemiological studies to develop acceptable intake rates of PFOA and PFOS in humans.

level] at the POD in animal studies). This dosimetric adjustment can be done using estimated human clearance values, or the ratio of estimated serum half-lives in humans and animals.¹⁹

3. Apply UFs to the HED to determine the RfD, an estimate of the daily oral dose at which humans are expected to be without risk from extended²⁰ exposure to a chemical, including PFAS. An RfD is expressed as mass of chemical per day adjusted for individual bodyweight ($\text{mg}_{\text{chemical}}/\text{kg}_{\text{body weight}}/\text{day}$).

Toxicologists apply the UFs of 1, square root of 10 (which rounds to 3 if a single such factor is applied; if two such factors are applied, the value equals 10), or 10 to reflect limitations of the data used. Limitations include sensitivity differences between people (intraspecies), extrapolating from animals to humans (interspecies), shorter duration of exposure in the study used, use of a LOAEL as the POD, and gaps in the toxicological database. Toxicologists multiply the UFs together to obtain the total UF, and then divide the selected (NOAEL, LOAEL, or BMDL) POD (or as adjusted, the HED) by that total to get the amount applied to the RfD (as shown in the equation below). The UFs are applied selectively for each chemical as appropriate for the toxicity data being used in the assessments.

$$\frac{HED}{\text{Total UFs}} \times \text{dosimetric adjustment factor} = RfD$$

4. Combine the RfD with selected exposure parameters to establish a concentration (i.e., standard or guidance value) for PFAS in a specific medium (e.g., drinking water) that is intended to be protective of human health.

Some states select exposure parameters to subgroups like pregnant women or children if they are sensitive for the toxicological effect of concern. Exposure parameters for health-based guidelines include the exposure rate (e.g., amount of drinking water, fish, or soil assumed to be ingested each day) and body weights for the target population. For drinking water guidelines (and groundwater guidelines based on drinking water exposure parameters), states consider the Relative Source Contribution (RSC), which is the percentage of the RfD allocated or allowed to come from drinking water. The default value for the RSC is 20 percent, but states can use chemical specific values from 20 to 80 percent if available data support them. For example, the EPA's LHA allows drinking water to contribute only 20 percent of the RfD and other sources can contribute 80 percent, so the RSC is 20 percent. Exposure assumptions vary among states and can result in different guidelines despite similar RfDs. Furthermore, scientists are still learning about PFAS sources and degrees/impacts of exposure; as such, states' assumptions about the RSC are likely to change in the future and affect PFAS guidelines.

State Trends on the Basis of Guidelines

ECOS examined states' calculations and factors applied to oral routes of exposure to PFAS that contributed to their standard setting processes.

¹⁹ The dosimetric adjustment factor measures external (oral) doses, and is how toxicologists account for PFAS bioaccumulation in risk assessment. It can be applied to develop the HED as described, or multiplied by the difference of the POD and Total UFs in the RfD equation below. Both methods are mathematically equivalent and the order of operations does not effect the final result.

²⁰ The length of exposure can vary depending on the chemical and regulatory agency. For example, in its draft toxicity values for [PFBS and GenX](#), the EPA characterizes exposure over a lifetime (chronic RfD) or less (subchronic RfD). For its LHA for [PFOA and PFOS](#), the RfD is defined by a lifetime of exposure. ATSDR uses the term MRL instead of RfD to describe the daily dose of a chemical that is not expected to pose a risk to human health. Its PFAS [MRLs](#) are derived for intermediate (14-364 days) exposure.

Appendices A-E of this report include tables of state toxicological information and exposure assumptions for setting guidelines in drinking water, groundwater, surface water, soil, and air. Some of the trends in the data are summarized below:

Critical Studies and Endpoints. This is a critical first step in the process, as it indicates the factor for which toxicologists are protecting (e.g., fetal/infant growth delays, thyroid dysfunction, infertility, alterations in liver function, and/or impaired immune function). *Five states* indicated that they use the EPA's preferred critical studies (e.g., Lau et al. [2006] for the PFOA LHA and Luebker et al. [2005] for the PFOS LHA) and pharmacokinetic model for developing a toxicity factor (i.e., modeled average animal serum levels at the POD). *Nine states* use a variety of critical studies and endpoints based on which PFAS compound they are evaluating. As discussed in the Human-to-Animal Extrapolation Methods section on page 16, state approaches may differ from the EPA methodology in that the POD is based on serum PFAS levels measured at the end of the animal study rather than serum levels predicted using the EPA pharmacokinetic model.

Points of Departure. The choice of POD depends on the dose response data for the critical endpoint being used as the basis for risk assessment. As previously mentioned, BMDL is the preferred POD when available as it is less dependent on the dose selection and sample size than the NOAEL or LOAEL. If a BMDL cannot be derived, the NOAEL is preferred. If there is no NOAEL in the study (i.e., effects occur at all doses), the LOAEL is used. *Four states* and the EPA use the LOAEL and NOAEL PODs for PFOA and PFOS in drinking water. Other states indicated that they use a combination of PODs depending on which PFAS they are examining, with LOAEL the most commonly used for PFOA and NOAEL the most commonly used for PFOS. *Four states* reported using a BMDL for various PFAS in drinking water.

Uncertainty Factors. States use a variety of combinations for UFs that differ based on the study used. However, most states reported applying a total UF of 300 for PFOA (with a UF of 3 for interspecies; 10 for intraspecies; and other UFs for extrapolation from LOAEL to NOAEL, database limitations, duration of exposure [i.e., subchronic to chronic extrapolation], and/or sensitive developmental endpoints), and a total UF of 30 (with a UF of 3 for interspecies and 10 for intraspecies) for PFOS.

Exposure Parameters:

- **Populations at Risk.** States including *Michigan, Minnesota, and New Hampshire* use Minnesota's model (Goeden et al. [2019]) to predict fetal and infant exposure from transplacental transfer, breastmilk, and prepared formula. This model applies the upper-percentile age-adjusted drinking water ingestion rates in the 95th percentile for pregnant women and formula-fed infants, and the upper-percentile ingestion rate for breast-fed infants. Other states account for populations that may be at increased risk by considering their higher intake rates, with infants and lactating women consuming more than typical adults when adjusted for body weight. Examples include a 0-1 year old body weight-adjusted drinking water intake rate of 0.175 L/kg/day (*Vermont*), a 10 kg body weight adjusted drinking water intake rate of 0.1 L/kg/day (*Wisconsin*), or a lifetime average drinking water intake rate of 0.053 L/kg/day that accounts for increased water consumption relative to body weight at young ages (*California*), as compared to the default adult water consumption rate (0.029 L/kg/day) (*New Jersey*). The EPA's LHA assumed the drinking water ingestion rate of the 90th percentile of lactating women to be 0.053 L/kg/day. Several states look at fish consumption rates as well when developing surface water quality criteria and fish consumption advisories; these advisories are more stringent for high risk populations (e.g., infants, children, pregnant and lactating women, women of childbearing age) in some states (e.g., *New Jersey*). Overall, target populations and RSCs differed among states, even if those states used the

same critical endpoint or had a similar RfD. The different exposure parameters resulted in different final guidelines.²¹

- **Relative Source Contributions.** Six states reported using the default value for the RSC of 20 percent (as the EPA does) for various PFAS analytes in drinking water, indicating that they allow 20 percent of the RfD to come from drinking water and 80 percent to come from other sources of exposure. Three states use a chemical-specific RSC of 50 percent in drinking water. No states reported using a less conservative RSC of 80 percent, which would allow 80 percent of the RfD to come from drinking water, allocating only 20 percent to exposure to all other sources like dust or consumer products. However, *Alaska and Wisconsin* do not use an RSC (i.e., an RSC of 100 percent) in groundwater; at that guideline, exposures from other sources would raise the intake above the RfD. Several states reported that the [EPA Decision Tree](#) (2000) is helpful in establishing an RSC.

Human Epidemiological Data. Eight states (*California, Florida, Massachusetts, Michigan, New Hampshire, New Jersey, North Carolina, Wisconsin*) reported considering both human and animal epidemiological data to support their selections of critical endpoints from animal toxicity studies and guide their risk assessments.²²

Human-to-Animal Extrapolation Methods. Human toxicity values for PFAS are primarily based on laboratory animal studies and rely on various approaches to account for the much longer half-lives in humans than in animals. Toxicologists consider the interspecies half-life difference in most PFAS risk assessments because the same daily dose of a PFAS results in a higher internal dose (blood serum PFAS level) in humans because of their slower excretion rate. In general, the serum PFAS levels from animal studies are converted to HEDs by applying a chemical-specific clearance factor (based on human half-life and volume of distribution) that relates serum levels to human-administered doses. The interspecies UF is reduced from the default value of 10 to 3 when these approaches are used since interspecies pharmacokinetic differences have already been accounted for.

Four states (*Alaska, Massachusetts, Vermont, Wisconsin*) reported using the EPA approach (used in its derivation of the LHA for PFOA and PFOS), which estimates the HED using modeled serum concentrations at the POD in the animal study as the internal dose metric. A few other states, including *New Jersey, New Hampshire, and California*, use measured serum concentrations at the end of the dosing period in the animal study as the POD.

Carcinogenicity. 11 states (*Alaska, California, Florida, Indiana, Massachusetts, Minnesota, New Hampshire, New Jersey, North Carolina, Vermont, Wisconsin*) reported that they consider carcinogenicity as well as non-cancer endpoints in their evaluations. Six of those states (*Alaska, California, Florida, New Jersey, Vermont, Wisconsin [PFOA only]*) quantify cancer risk with a slope factor and a cancer risk level of 1 in 100,000 (1×10^{-5}) or 1 in 1,000,000 (1×10^{-6}).²³ *California* uses cancer as the critical endpoint for PFOA (pancreatic and liver cancer in male rats) and PFOS (liver cancer in male rats).

Section III. Risk Management

Once their toxicologists assess potential health or ecological risks, states take steps to manage those risks and protect public health. This includes analyzing PFAS samples, establishing guidelines, and addressing resource issues.

²¹ Groundwater ingestion values, though different, are used in essentially the same way as drinking water ingestion rates. Therefore, other states (e.g., Texas) use similar processes for risk assessment.

²² As with any risk assessment, human epidemiology is considered, at a minimum, to support using an animal study. No state has relied on the human epidemiological data as the basis of an RfD derivation.

²³ Cancer risk levels used in risk assessments are policy choices that vary among states and may be specified in a state's legislation or regulation.

This could also include deciding whether to address PFAS individually or as a group (see Grouping PFAS section on page 8), deciding not to act based on their conclusions of the assessed risks, or looking at broader impacts of managing PFAS such as issuing discharge permits and availability of treatment removal technologies.

Analytical Methods & Limitations

States use a variety of methods to test PFAS samples in different media. The most widely used are [EPA Method 537](#) (2008, applies to 14 PFAS in drinking water) and [EPA Method 537.1](#) (2018, applies to 18 PFAS in drinking water). *Four states (Florida, Indiana, New Hampshire, Texas) use EPA Method 537 and six states (California, Michigan, Nebraska, North Carolina, Vermont, Wisconsin) use Method 537.1 in drinking water. Three states (Alaska, Massachusetts, New Jersey) reported using both.* EPA Method 537.1 analyzes the same 14 PFAS as the original method, which was used in sampling during UCMR3, and adds four other PFAS, including GenX. Both methods are designed for water with low total suspended or dissolved solids, and are performed using a solid phase extraction preparatory method before sample analysis.

Some labs perform modifications, like using isotope dilution, to these methods for use in other matrices to account for lower reporting limits or greater accuracy. For example, *five states (Alaska, California, Indiana, Texas, Vermont) reported that they use Method 537.1 for non-drinking water media.*

Other methods for PFAS analysis include:

- [EPA Method 8321](#): *Florida* uses for surface water, groundwater, wastewater, soil, and other solids.
- [EPA Method 8327](#): *Florida* uses for surface water, groundwater, and wastewater. This is a pending EPA method for 24 analytes, including all 18 target analytes from EPA Method 537.1. This method is not yet sufficient in low-level detection or for rigorous reporting quality, and 11 of the compounds have been reported by several states and other regulators as problematic. Thus, agencies such as the U.S. Department of Defense (DOD) advise its use for screening purposes only.
- [DOD Quality Systems Manual](#) Method 5.1 or later (i.e., 5.2): *California and North Carolina* use for consideration as additional guidance and quality control requirements, or performing analyses.
- Total Oxidizable Precursor (TOP) Assay: *Vermont* uses for soil and groundwater.
- [EPA Solid Waste Method 1312](#), Synthetic Precipitation Leaching Procedure (SPLP): *Vermont* uses for soil and sludge.
- SGS Axys Analytical, [MLA 110](#): *Vermont* uses for sludge; *Minnesota* uses for water/effluent, soil/sediment, biosolids, and tissue.
- [ASTM D7979-17](#): *Florida* uses for surface water and sludge.
- [ASTM D7968-17a](#): *Florida* uses for soil.
- State defers to each lab's preferred methods²⁴: *three states (Minnesota [drinking water], New Jersey, Wisconsin).*

Several methods are pending or were not final when ECOS conducted the survey, so it is unknown if or which states may already use them:

- EPA Solid Waste Method 8328: This method just began undergoing single-lab validation but, if approved, it would encompass the same 24 compounds as Method 8327 plus GenX, use isotope dilution for quantification,

²⁴ State agencies have method performance expectations that they use to approve labs and determine whether or not the lab's own method is considered suitable by state program standards.

and be applicable to complex matrices including soils and biosolids. A state noted that isotope dilution is the gold standard for quantitation and the only method that corrects results for potential matrix effects.

- **EPA Method 533:** Published in December 2019, this method targets short-chain²⁵ PFAS in drinking water and covers 25 PFAS, including 14 of Method 537 and 11 unique to this method.
- The EPA is developing a number of source emission methods for measurements from industrial and combustion/incineration sources. The EPA will apply what they learn in the source sampling (stack testing) efforts to ambient measurement techniques anticipated in 2022-2024.
- Some states are considering supplemental analysis (e.g., Total Organic Fluorine (TOF) and TOP assays) to more completely characterize total PFAS in various media including consumer and industrial products.

Challenges that confound PFAS analysis include:

- There are no regulatory-approved methods for most PFAS in water and all PFAS in solid media/air.
- Sample collection and analytical interference/contamination due to the presence of PFAS in common consumer products, sampling equipment, and lab materials can create challenges concerning quality control procedures in the laboratories.
- There are financial and time constraints of existing lab methods. The Minnesota Department of Health reports that the turnaround time for their samples is 45 days and each water sample costs more than \$300.
- There are different and sometimes inconsistent laboratory procedures for non-EPA approved methods. Not every state has a state lab, and some labs are government contracted or private. Each results in different costs, time constraints, and could vary sampling procedures. Each agency verifies labs for use based on their own criteria.

Establishing Guidelines

States consider the health-based criteria from risk assessment and other technical factors in the establishment of their guidelines. Some states' risk assessment approaches and conclusions have resulted in the development and adoption of PFAS guidelines that are lower than guidelines for most other contaminants. Scientific considerations that may contribute to these values include:

- PFAS cause toxicological effects at very low doses.
- Risk assessments account for the higher bioaccumulation of certain PFAS in humans than in animals. The same dose given to a human will result in a much higher blood serum level than in a lab animal.
- Low levels of certain PFAS in blood serum are associated with human health effects, and some states will consider how much a certain level in drinking water will increase blood serum PFAS levels. Low levels of PFAS in drinking water can cause considerable increases in blood serum PFAS levels.
- As mentioned in footnote 9, the health basis for standards for other emerging contaminants may be as low as those for PFAS, but the final guideline is set at the analytical quantitation levels, which may be up to several orders of magnitude higher than the health-based levels. For PFAS, analytical quantitation levels are very low, such that the final standard or guidance can be set at the health-based criterion.

Additionally, some states are required to perform a cost-benefit analysis in setting their final standards.

²⁵ Short-chain PFAS are those with carbon chain lengths of 5 or lower for sulfonic acids like PFBS, and carbon chain lengths of 7 or lower for carboxylic acids like PFHxA.

PFAS Resource (Cost) Issues

Eight states (Alaska, California, Massachusetts, Michigan, New Jersey, North Carolina, New Hampshire, Wisconsin) have conducted, are required by state or federal law to conduct, or plan to consider costs or conduct cost-benefit analyses to define the economic impact of establishing guidelines for certain PFAS. Some states (e.g., North Carolina) require a cost-benefit analysis as part of their administrative procedures for developing MCLs or water quality criteria. Other states are not required to conduct a cost-benefit analysis prior to adopting guidelines into state regulation but plan to factor costs into decision-making. One state noted that the operations and management costs for treatment (e.g., Granular Activated Carbon [GAC]) are detrimental to its and others' budgets, especially for small public water systems that perform carbon changeouts regularly to ensure no arsenic MCL exceedances or other background factors when undergoing PFAS treatment procedures.²⁶

Four states (California, Michigan, Minnesota, New Jersey) have conducted cost-estimates for some PFAS efforts. Some actions may fall under a state's normal agency programmatic activity; others require more staff and time. For example, at the time that ECOS conducted this survey, Michigan had allocated \$1.7 million for testing its PWSs and three full-time employees (FTEs) for oversight of the testing and rulemaking, and estimated rulemaking costs to exceed \$250,000. New Jersey utilizes five FTEs for PFAS efforts. California has FTEs dedicated to enforcement of the regulation, but does not consider FTEs for rule development in its cost estimates. A couple of states noted that PFAS has required a somewhat swift and significant rebalancing of staff member projects; for example, a state may have difficulty hiring new employees to fill the previous positions of those now assigned to work on PFAS, or a state's other projects may fall by the wayside due to the demand of this issue.

Incurring costs extend beyond regulating PFAS and should factor in: expenditures for states to initially investigate whether and to what degree there are PFAS releases or contaminated media; removal methods for contaminated media; chemical analysis; liabilities; and tracking the fate and transport of PFAS once released from an active source to the environment, requiring (re)sampling and treatment. For example, Minnesota is still calculating its costs, but noted that an industrial facility in the state allocated about \$750,000 to retrofit its operations where PFAS were used and had contaminated a nearby waterbody. New Jersey estimates that the average cost for lab analysis is \$300 per PFAS sample at each point of entry, while PFAS-specific GAC treatment for a wastewater facility treating one million gallons per day (serving about 10,000 people) ranges from \$500,000 to \$1,000,000. Given PFAS ubiquity, the ability for precursors (e.g., fluorotelomers) to transform to perfluoroalkyl compounds and complicate site models, and complex transport mechanisms, especially at the air-water interface, states will need to use more resources to test process-based conceptual site models and fully understand the size and source of PFAS plumes.

States identified several cost implications of regulating PFAS:

- Resource availability is driven by dedicated government appropriations. For most states, resources to investigate and address PFAS come from existing program budgets (i.e., no new funds). Some states like Michigan have received funding from bills signed by their Governors, but this is state-specific and based on legislative priorities. Other states have received funding from settlements with PFAS manufacturers to use on regulation and/or restoration of contaminated sites.
- Resource disparity exists – States with the fewest resources to address PFAS may be more significantly impacted by PFAS than others. Similarly, they may only have resources to address PFAS-related risks that are most studied in existing science and most salient among the public, rather than addressing risks unique to that

²⁶ Small public water systems usually contain contaminants other than PFAS, including arsenic, manganese, nitrate, or bacteria that present health risks and are naturally occurring or originate from nearby land uses. Effectiveness of PFAS treatment will depend on how often filters are replaced and what levels of these other contaminants are present in the system. See more [here](#).

state. The complexities of PFAS scientific information also create a barrier to understanding risk in a public forum.

- Data gaps prevent confident decision-making on how resources are used to address PFAS. States want to develop regulations based on a sound understanding of the problem in their state, but various factors – the lack of information on the sources and fates of PFAS, how they can be removed from drinking water and aquifers, and resulting waste management issues – create barriers to state time and financial investment.

A few states identified the need for water quality-based effluent limits, as well as the need for a cost conversation through a national MCL or National Recommended Water Quality Criteria (NRWQC) processes, as many states do not have the resources to regulate PFAS on their own. These are SDWA and Clean Water Act (CWA) processes driven by the EPA and involving states as co-regulators, and are one example of how the EPA is assessing potential changes to its regulatory processes to better respond to emerging contaminants and be more inclusive of state priorities.²⁷

Conclusion

ECOS asked states to list considerations and unanswered questions that will affect their PFAS guidelines in the future. States noted that the greatest impacts on state PFAS regulations will be:

- How can regulators apply or develop guidelines to PFAS in less-explored media (e.g., food and agriculture, biosolids, landfills, foam, and air emissions), if at all? For example, a few states (e.g., Minnesota, Michigan, New Jersey) have guidelines or consumption advisories for fish tissue or deer meat.
- How can labs detect lower concentrations of PFAS for media other than drinking water?
- What new information on sensitive human subpopulations, bioaccumulation in fish and shellfish, etc. will affect PFAS regulation?
- How will shifting use and chemistries of PFAS that have yet to be addressed complicate the responses?
- How will developing information about PFAS migration from soil into animal feed, food crops, etc. affect the need for guidance values and state actions in response?
- What analytical approaches and health effects data will be available to develop guidelines for replacement PFAS?
- What will happen to current and pending state guidelines if federally enforceable standards (MCLs, NRWQCs) are enacted?
- What kinds of new science are needed to more effectively regulate PFAS?
- How will guidelines affect PFAS management/cleanup liability and other considerations? For example, what will be the impact of designating PFAS as hazardous substances or regulating discharges through the National Pollutant Discharge Elimination System (NPDES) and remediation programs? Who will pay for mitigation or remediation? What role does pollution prevention play in prohibiting PFAS in consumer goods from passing through regulated facilities and entering the environment?

PFAS pose complex challenges that are new (e.g., the carbon-fluorine bond) and especially daunting. Their unique characteristics include mobility; persistence in the environment and the human body; animal and health effects at low doses; a lack of toxicological data for most PFAS detected in the environment and used in commerce; ubiquitous detection in blood sampling; and technical obstacles for remediation. Regulatory and policy developments that vary by state and are uncertain at the federal level compound these challenges. There is also heightened public pressure

²⁷ For more information on states' recommendations for contaminants of emerging concern, see the Association of Clean Water Administrators (ACWA) and the Association of State Drinking Water Administrators (ASDWA) joint [Recommendations Report for Contaminants of Emerging Concern](#).

for swift risk management, encouraged through social media and reporting. For example, there have been high-profile lawsuits (e.g., \$850 million from 3M to Minnesota in 2018, \$671 million from DuPont to plaintiffs in West Virginia and Ohio in 2017). Groups have convened community events and produced films inspired by PFAS contamination in cities like Parchment, Michigan; Decatur, Alabama; and Parkersburg, West Virginia. And public data from the UCMR3 demonstrated that water suppliers serving 16.5 million people in the U.S. had detectable PFAS in their water and that more than six million people consuming water in 2015 had PFAS concentrations above the EPA's LHA.²⁸

A few states followed the emerging scientific information on, evaluated occurrence of, and developed guidelines for PFAS for many years before they were widely known to the public. Some states are actively responding to the recent events mentioned above by establishing programs and guidelines to regulate PFAS-contaminated sites. Other states are aware of PFAS as an emerging contaminant and addressing it as they can. Given these circumstances, risk communication is going to be an increasingly important function. Regulators need more transparency about the uses of existing PFAS, the ongoing development of new PFAS chemicals by industry, and PFAS approval by the EPA under statutes like the Toxic Substances Control Act (TSCA). As states seek to independently regulate PFAS, it is critical to coordinate with and learn from other states that have established and are establishing their own guidelines.

This compilation on state-developed PFAS guidelines is a moving target, as regulators are acting quickly to develop and/or update guidelines for PFAS in different media. Some states are waiting to set guidelines in the hopes that the EPA will establish a federally-enforceable MCL, and other states are establishing guidance at levels below the EPA's LHA and/or for PFAS other than PFOA and PFOS, indicating that some regulators and toxicologists view the federal approach²⁹ as insufficiently protective. As not all states completed the survey (including some states with known guidelines) and there will likely continue to be state standard setting at concentrations below the EPA's LHA and for PFAS other than PFOA and PFOS, ECOS hopes to compile additional information in the future.

This whitepaper is not intended to be a comprehensive compendium of state PFAS regulations. Rather, it aims to lay the foundation for states to dig deeper into the issue. ECOS hopes this paper will serve as a basis for future conversations, and encourages state-to-state, state-federal, and state-NGO partnerships and collaboration. ASDWA will soon publish a toolkit of modules on assessing state resources, characterizing health impacts, identifying treatment, analyzing costs and benefits, and other considerations surrounding PFAS in drinking water. ECOS encourages states to use this white paper in combination with ASDWA's report, the ITRC fact sheets, and other resources (including the forthcoming detailed ITRC Technical Regulatory Document) to fully understand the state of play on PFAS regulation.

State Agency Reports on PFAS Guidelines

These reports/resources were provided by state environmental and health agencies that responded to the ECOS survey. For a full list of individual state PFAS websites with information on how they developed their guidelines and on other PFAS efforts, see the "Overview" section on ECOS' [PFAS Risk Communication Hub](#).

- [California](#)
- [Colorado](#)
- [Florida](#)
- [Indiana](#)
- [Massachusetts](#)
- [Michigan](#)
- [Minnesota](#)
- [New Hampshire](#)
- [New Jersey](#)
- [Texas](#)
- [Vermont](#)

²⁸ Hu et al., 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 Letter*, vol. 3, no. 10, 2016, pp. 344-350. *ACS Publications*, <https://doi.org/10.1021/acs.estlett.6b00260>.

²⁹ I.e., its process as a whole, or in its choice of critical studies or factors for calculation.

Appendix A: State Drinking Water PFAS Guideline Criteria

State	PFAS Analyte(s)	Advisory Level (ug/L)	Toxicity Data	Critical Effect Study	Endpoint	RSC (%)	POD	HED (mg/kg/day)	UFs						RfD (mg/kg/day)	Drinking Water Intake Rate (L/day unless otherwise specified)	Exposure assumptions	Target Populations	Resources		
									Total	Interspecies	Intraspecies	LOAEL to NOAEL	Database Limitation	Duration of Exposure (i.e., Subchronic to Chronic)						Sensitive Developmental Endpoints	
CA	PFOA	0.0051 (based on health-based reference level of 0.1 ppt for cancer effects, 2 ppt for non-cancer effects [liver])	Animals (mice/liver, rats/cancer)	Li et al., 2017; NTP, 2018	Hepatotoxicity in female mice; Cancer (pancreatic and liver) in male rats	20	LOAEL (0.97 mg/L)		300	3	10	3				3	Lifetime average of 0.053 L/kg/day	Oral ingestion as significant route of exposure		https://www.waterboards.ca.gov/pfas/ https://oehha.ca.gov/water/notification-level/notification-level-recommendations-perfluorooctanoic-acid-pfoa https://www.waterboards.ca.gov/drinking_water/certlic/drinking_water/PFOA_PFOs.html	
	PFOS	0.0065 (based on health-based reference level of 0.4 ppt for cancer effects, 7 ppt for non-cancer effects [immune system])	Animals (mice/liver, rats/cancer)	Dong et al., 2009 Butenhoff et al., 2012	Immunotoxicity in male mice; Cancer (liver, structural similarity to PFOA) in male rats	20	NOAEL (0.674 mg/L)		30	3	10						Lifetime average of 0.053 L/kg/day				
MA	PFOS, PFOA, PFNA, PFHpA, PFHxS, PFDA	0.020*	Animals	Multiple	Based on multiple endpoints and evidence of effects below EPA PODs for PFOA and PFOS; including: immunotoxicity, hepatotoxicity, thyroid effects, developmental effects.	20; to account for dietary and other exposures to PFAS subgroup addressed as well as potentially higher infant exposures.	NOAEL for PFOS, LOAEL for PFOA, equivalent to EPA values.	Equivalent to EPA values for PFOA and PFOS	1000 for PFOA, 100 for PFOS	3	10	10 for PFOA	3 for both PFOA and PFOS				5x10 ⁻⁶ based on PFOS and PFOA value, which is applied to subgroup based on similarity in chemical structures, toxicities, long serum half-lives.	0.054 L/kg/day (same as EPA value used in LHA derivation)	Body weight and water intake of lactating women (same as EPA value used in LHA derivation)	Lactating and pregnant women; fetus; nursing infants	https://www.mass.gov/lists/development-of-a-pfas-drinking-water-standard-mcl
MI	PFOA	0.008	Animals (mice)	Onishchenko et al., 2011 and Koskela et al., 2016	Neurobehavioral effects and skeletal alterations	50	LOAEL		300	3	10	3	3	1			95th percentile, 50% RSC				https://dtmb.state.mi.us/ARS_Public/Transaction/RFRTransaction?TransactionID=29
	PFOS	0.016	Animals (mice)	Dong et al., 2009	Immunotoxicity and Hepatotoxicity	50	NOAEL		30	3	10	1	1	1			95th percentile, 50% RSC				

State	PFAS Analyte(s)	Advisory Level (ug/L)	Toxicity Data	Critical Effect Study	Endpoint	RSC (%)	POD	HED (mg/kg/day)	UFs					RfD (mg/kg/day)	Drinking Water Intake Rate (L/day unless otherwise specified)	Exposure assumptions	Target Populations	Resources		
									Total	Interspecies	Intraspecies	LOAEL to NOAEL	Database Limitation						Duration of Exposure (i.e., Subchronic to Chronic)	Sensitive Developmental Endpoints
MI	PFNA	0.006	Animals (mice)	Das et al., 2015	Reduced pup body weight	50	NOAEL		300	3	10	1	10	1		95th percentile, 50% RSC				
	PFHxA	400	Animals (rats)	Klaunig et al., 2015	Renal effects	20	BMDL		300	3	10	1	10	1		95th percentile, 20% RSC				
	PFHxS	0.051	Animals (rats)	NTP 2018 Tox-96 Report	Thyroid effects	50	BMDL		300	3	10	1	10	1		95th percentile, 50% RSC				
	PFBS	0.42	Animals (mice)	Feng et al., 2017	Thyroid effects	20	BMDL		300	3	10	1	10	1		95th percentile, 20% RSC				
	Gen X	0.37	Animals (mice)	DuPont 18405-1037, 2010	Reduced pup body weight, Hepatotoxicity	20	BMDL		300	3	10	1	3	3		95th percentile, 20% RSC				
MN	PFOA (Short-term, Subchronic and chronic)	0.035	Animals (mice)	Lau et al., 2006	Developmental and liver effects, kidney effects, Immunotoxicity	50	38 mg/L serum concentration	0.0053	300	3	10	3	3			1.8x10 ⁻⁵	95th percentile	Half-life 840 days; placental transfer 87%, 5.2% breastmilk transfer	Fetus and Breastfeeding Infants	https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfoa.pdf
	PFOS (Short-term, Subchronic and chronic)	0.015	Animals (mice)	Dong et al., 2011	Immunotoxicity, adrenal, developmental effects, liver effects, thyroid effects	20 for older children and adults, 50 for infants/ young children	2.36 mg/L serum concentration	0.000307	100	3	10		3			3.1x10 ⁻⁶	95th percentile	Half-life 1241 days; placental transfer 40%; 1.7% breastmilk transfer	Fetus and Breastfeeding Infants	https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfos.pdf
	PFBA (Short-term, Subchronic and chronic)	7	Animals (rats)	NOTOX, 2007 and Butenhoff, 2007	Liver effects, Thyroid effects	50	3.01 mg/kg/day	0.38	100	3	10		3			3.8x10 ⁻³	95th percentile	Half-life 72 hrs; placental transfer ND; breastmilk transfer ND	Infants and Adults	https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfba2summ.pdf
	PFBS (Short-term and Subchronic)	3	Animals (mice)	Feng, 2017	Developmental effects, Thyroid effects, Reproduction	50	50 mg/kg/day	0.158	100	3	10		3			1.6x10 ⁻³	95th percentile	Half-life 665 hrs; placental transfer ND; breastmilk transfer ND	Infants and Adults	https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfbssummary.pdf
	PFBS (Chronic)	2	Animals (rats)	Lieder, 2009 and York, 2003	Kidney	20	45 mg/kg/day	0.129	300	3	10		3	3		4.3x10 ⁻⁴	95th percentile	Half-life 665 hrs; placental transfer ND; breastmilk transfer ND	General Population	https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfbssummary.pdf

State	PFAS Analyte(s)	Advisory Level (ug/L)	Toxicity Data	Critical Effect Study	Endpoint	RSC (%)	POD	HED (mg/kg/day)	UFs						RfD (mg/kg/day)	Drinking Water Intake Rate (L/day unless otherwise specified)	Exposure assumptions	Target Populations	Resources			
									Total	Interspecies	Intraspecies	LOAEL to NOAEL	Database Limitation	Duration of Exposure (i.e., Subchronic to Chronic)						Sensitive Developmental Endpoints		
MN	PFHxS (Short-term, Subchronic and chronic)	0.047	Animals (rats)	NTP, 2018	Thyroid effects, Liver effects	20 for older children and adults, 50 for infants/ young children	32.4 mg/L	0.00292	300	3	10		10					9.7x10 ⁻⁶	95th percentile	Half-life 1935 days; placental transfer 70%; breastmilk transfer 1.4%	Fetus and Breastfeeding Infants	https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfahs.pdf
NH	PFOA	0.012	Animals (mice)	Loveless et al., 2007	Hepatotoxicity		BMDL10		100	3	10		3					95th percentile	MDH Model	Fetus and Breastfeeding Infants		
	PFOS	0.015	Animals (mice)	Dong et al., 2011	Immunosuppression		NOAEL		100	3	10		3					95th percentile	MDH Model	Fetus and Breastfeeding Infants		
	PFNA	0.011	Animals (mice)	Das et al., 2015	Hepatotoxicity		BMDL10		100	3	10		3									
	PFHxS	0.018	Animals (mice)	Chang et al., 2018 and Ali et al.	Infertility		BMDLSD (under peer review)		300	3	10		3	3								
NJ	PFOA	0.014	Animals (mice)	Loveless et al., 2006	Hepatotoxicity	20	BMDL		30	3	10				10			2 (70 kg body wt)			Infants	
	PFOS	0.013	Animals (mice)	Dong et al., 2009	Immunotoxicity	20	NOAEL		30	3	10							2 (70 kg body wt)			Infants	
	PFNA	0.013	Animals (mice)	Das et al., 2015	Hepatotoxicity	50	BMDL		1000	3	10		3	10	3				200:1 serum: drinking water ratio			
VT	PFOA, PFOS, PFHxS, PFHpA, PFNA	0.02*	Animals (mice)	EPA (2016)	EPA (2016)	20	EPA (2016)		EPA (2016)									0.175 L/kg/day			0-1 year old	

*= Advisory level is based on the total of more than one PFAS

Appendix B: State Groundwater PFAS Guideline Criteria

State	PFAS Analyte(s)	Advisory Level (ug/L)	Toxicity Data	Critical Effect Study	Endpoint	RSC (%)	POD	HED (mg/kg/day)	UFs						RfD (mg/kg/day)	Drinking Water Intake Rate (L/day unless otherwise specified)	Exposure assumptions	Target Populations	Resources & Notes
									Total	Interspecies	Intraspecies	LOAEL to NOAEL	Database Limitation	Duration of Exposure (i.e., Subchronic to Chronic)					
AK	PFOA	0.4	Animals (mice)	Lau et al., 2006	Decreased ossification of pup proximal phalanges, accelerated preputial separation	100	EPA (2016)		EPA (2016)						EPA (2016)	0.78	Residential exposure for 6 yrs old child receptor	Child	http://dec.alaska.gov/media/7543/201802_01_pccl.pdf
	PFOS	0.4	Animals (mice)	Luebker et al., 2005	Reduced pup body weight	None (but does not include an RSC in cleanup level calculations, so essentially use an RSC of 100)	EPA (2016)		EPA (2016)						EPA (2016)	0.78	Residential exposure for 6 yrs old child receptor	Child	http://dec.alaska.gov/media/7543/201802_01_pccl.pdf
CO	PFOA, PFOS	0.07*	Animals (mice)	EPA (2016)	EPA (2016)	20	EPA (2016)		EPA (2016)						EPA (2016)	EPA (2016)	EPA (2016)	EPA (2016)	
FL	PFOA	0.07	Animals (mice)	Lau et al., 2006	Decreased ossification of pup proximal phalanges, accelerated preputial separation	20	EPA (2016)		300	3		10			2x10 ⁻⁵	0.054 L/kg/day		Pregant/lactating women	
	PFOS	0.07	Animals (mice)	Luebker et al., 2005	Decreased offspring body weight	20	EPA (2016)		30	3					2x10 ⁻⁵	0.054 L/kg/day		Pregant/lactating women	
MA	PFOS, PFOA, PFNA, PFHpA, PFHxS, PFDA	0.020*	Animals	Multiple	Based on multiple endpoints and evidence of effects below EPA PODs for PFOA and PFOS; including: immunotoxicity, hepatotoxicity, thyroid effects, developmental effects.	20; to account for dietary and other exposures to PFAS subgroup addressed as well as potentially higher infant exposures.	NOAEL for PFOS, LOAEL for PFOA, equivalent to EPA values.	Equivalent to EPA values for PFOA and PFOS	1000 for PFOA, 100 for PFOS						5x10 ⁻⁶ based on PFOS and PFOA value, which is applied to subgroup based on similarity in chemical strutures, toxicities, long serum half-lives.	0.054 L/kg/day (same as EPA value used in LHA derivation)	Body weight and water intake of lactating women (same as EPA value used in LHA derivation)	Lactating and pregnant women; fetus; nursing infants	https://www.mass.gov/lists/development-of-a-pfas-drinking-water-standard-mcl

State	PFAS Analyte(s)	Advisory Level (ug/L)	Toxicity Data	Critical Effect Study	Endpoint	RSC (%)	POD	HED (mg/kg/day)	UFs						RfD (mg/kg/day)	Drinking Water Intake Rate (L/day unless otherwise specified)	Exposure assumptions	Target Populations	Resources & Notes	
									Total	Interspecies	Intraspecies	LOAEL to NOAEL	Database Limitation	Duration of Exposure (i.e., Subchronic to Chronic)						Sensitive Developmental Endpoints/ Subpopulations
MI	PFOA	0.07 (combined)*	Animals (mice)	Lau et al., 2006		20	EPA (2016)		300	3	10	10						Pregant/lactating women		
	PFOS	0.07 (combined)*	Animals (rats)	Luebker et al., 2005		20	EPA (2016)		30	3	10							Pregant/lactating women		
	PFOA (GSI for drinking water source)	0.42	Animals (primates)	Butenhoff et al., 2002	Hepatotoxicity	n/a	LOAEL		3000	3	10	10		10					https://www.michigan.gov/egle/0,9429,7-135-3311_4109-251790--00.html	
	PFOA (GSI)	12	Animals (primates)	Butenhoff et al., 2002	Hepatotoxicity	n/a	LOAEL		3000	3	10	10		10						
	PFOS (GSI for drinking water source)	0.011	Animals (primates)	Seacat et al., 2002	Decreased body weight, hepatotoxicity, thyroid toxicity	n/a	NOAEL		30	3	10									
	PFOS (GSI)	0.012	Animals (primates)	Seacat et al., 2002	Decreased body weight, hepatotoxicity, thyroid toxicity	n/a	NOAEL		30	3	10									
MN	PFOA (Short-term, Subchronic and chronic)	0.035	Animals (mice)	Lau et al., 2006	Developmental and liver effects, kidney effects, Immunotoxicity	50	38 mg/L serum concentration	0.0053	300	3	10	3	3					Half-life 840 days; placental transfer 87%, 5.2% breastmilk transfer	Fetus and Breastfeeding Infants	https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfoa.pdf
	PFOS (Short-term, Subchronic and chronic)	0.015	Animals (mice)	Dong et al., 2011	Immunotoxicity, adrenal, developmental effects, liver effects, thyroid effects	20 for older children and adults, 50 for infants/young children	2.36 mg/L serum concentration	0.000307	100	3	10		3					Half-life 1241 days; placental transfer 40%; 1.7% breastmilk transfer	Fetus and Breastfeeding Infants	https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfos.pdf
	PFBA (Short-term, Subchronic and chronic)	7	Animals (rats)	NOTOX, 2007 and Butenhoff, 2007	Liver effects, Thyroid effects	50	3.01 mg/kg/day	0.38	100	3	10		3					Half-life 72 hrs; placental transfer ND; breastmilk transfer ND	Infants and Adults	https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfba2summ.pdf
	PFBS (Short-term and Subchronic)	3	Animals (mice)	Feng, 2017	Developmental effects, Thyroid effects, Reproduction	50	50 mg/kg/day	0.158	100	3	10		3					Half-life 665 hrs; placental transfer ND; breastmilk transfer ND	Infants and Adults	https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfbssummary.pdf

State	PFAS Analyte(s)	Advisory Level (ug/L)	Toxicity Data	Critical Effect Study	Endpoint	RSC (%)	POD	HED (mg/kg/day)	UFs						RfD (mg/kg/day)	Drinking Water Intake Rate (L/day unless otherwise specified)	Exposure assumptions	Target Populations	Resources & Notes
									Total	Interspecies	Intraspecies	LOAEL to NOAEL	Database Limitation	Duration of Exposure (i.e., Subchronic to Chronic)					
MN	PFBS (Chronic)	2	Animals (rats)	Lieder, 2009 and York, 2003	Kidney	20	45 mg/kg/day	0.129	300	3	10		3	3	4.3x10 ⁻⁴	95th percentile	Half-life 665 hrs; placental transfer ND; breastmilk transfer ND	General Population	https://www.health.state.mn.us/environment/risk/docs/guidance/gw/pfbssummary.pdf
	PFHxS (Short-term, Subchronic and chronic)	0.047	Animals (rats)	NTP, 2018	Thyroid effects, Liver effects	20 for older children and adults, 50 for infants/young children	32.4 mg/L	0.00292	300	3	10		10		9.7x10 ⁻⁶	95th percentile	Half-life 1935 days; placental transfer 70%; breastmilk transfer 1.4%	Fetus and Breastfeeding Infants	https://www.health.state.mn.us/environment/risk/docs/guidance/gw/pfhxs.pdf
NC	PFOA	2	Animals (rats)	York et al., 2002, Butenhoff et al., 2004	Reduced pup body weight	20	LOAEL		3000	10	10	10	3	1		Assumed body weight and water consumption of adult	Daily exposure to human population	Adults	
NH	PFOA	0.012	Animal (mice)	Loveless et al., 2007	Hepatotoxicity	50	BMDL10		100	3	10		3		95th percentile	MDH Model	Fetus and Breastfeeding Infants		
	PFOS	0.015	Animal (mice)	Dong et al., 2011	Immunosuppression	50	NOAEL		100	3	10		3		95th percentile	MDH Model	Fetus and Breastfeeding Infants		
	PFNA	0.011	Animal (mice)	Das et al., 2015	Hepatotoxicity	50	BMDL10		100	3	10		3						
	PFHxS	0.018	Animal (mice)	Chang et al., 2018 and Ali et al.	Infertility	50	BMDLSD (under peer review)		300	3	10		3	3					
NJ	PFOA	0.014	Animals (mice)	Loveless et al., 2006	Hepatotoxicity	20	BMDL		30	3	10				2 (70 kg body wt)			Infants	
	PFOS	0.013	Animals (mice)	Dong et al., 2009	Immunotoxicity	20	NOAEL		30	3	10				2 (70 kg body wt)			Infants	
	PFNA	0.013	Animals (mice)	Das et al., 2015	Hepatotoxicity	50	BMDL		1000	3	10		3	10	3		200:1 serum: drinking water ratio		
TX	PFBA	71	Animals (mice)	MDH	Hepatotoxicity		NOAEL (6.9 mg/kg/d)		2400	1	10		10	3	2.9x10 ⁻³				Note: oral dose, 0.5 acre source area) (Res GWGWIng PCLs) https://www.tceq.texas.gov/assets/public/implementation/tox/evaluations/pfcs.pdf
	PFBuS	34	Animals (mice)	Leider et al., 2009, York et al., 2002	Systemic Toxicity		NOAEL (60 mg/kg/d)		42600	1	10		10	3	1.4x10 ⁻³				

State	PFAS Analyte(s)	Advisory Level (ug/L)	Toxicity Data	Critical Effect Study	Endpoint	RSC (%)	POD	HED (mg/kg/day)	UFs						RfD (mg/kg/day)	Drinking Water Intake Rate (L/day unless otherwise specified)	Exposure assumptions	Target Populations	Resources & Notes
									Total	Interspecies	Intraspecies	LOAEL to NOAEL	Database Limitation	Duration of Exposure (i.e., Subchronic to Chronic)					
TX	PFPeA	0.093	Animals (mice)	Surrogate: PFHxS	Hematotoxicity		NOAEL (0.3 mg/kg/d)	78900	1	10	3	10			3.8x10 ⁻⁶				
	PFHxS	0.093	Animals (mice)	Hoberman and York, 2003	Hematotoxicity		NOAEL (0.3 mg/kg/d)	78900	1	10	3	10			3.8x10 ⁻⁶				
	PFHxA	0.093	Animals (mice)	Surrogate: PFHxS	Hematotoxicity		NOAEL (0.3 mg/kg/d)	78900	1	10	3	10			3.8x10 ⁻⁶				
	PFHpA	0.56	Animals (mice)	Surrogate: PFOS	Neurodevelopment		NOAEL (0.6 mg/kg/d)	26300	1	10	10	1			2.3x10 ⁻⁵				
	PFOS	0.56	Animals (mice)	Zeng et al., 2011	Neurodevelopment		NOAEL (0.6 mg/kg/d)	26300	1	10	10	1			2.3x10 ⁻⁵				
	PFOA	0.29	Animals (mice)	Macon et al., 2011	Mammary gland development		NOAEL (0.3 mg/kg/d)	24300	1	10	30	1			1.2x10 ⁻⁵				
	PFOSA	0.29	Animals (mice)	Surrogate: PFOA	Mammary gland development		NOAEL (0.3 mg/kg/d)	24300	1	10	30	1			1.2x10 ⁻⁵				
	PFNA	0.29	Animals (mice)	Fang et al., 2010	Spleen Cell Death		NOAEL (1 mg/kg/d)	81000	1	10		10	10		1.2x10 ⁻⁵				
	PFDeA	0.37	Animals (mice)	Kawashima et al., 1995	Hepatotoxicity		NOAEL (1.2 mg/kg/d)	81000	1	10		10	10		1.5x10 ⁻⁵				
	PFDS	0.29	Animals (mice)	Surrogate: PFDaA	Reduced Body Weight		NOAEL (1 mg/kg/d)	81000	1	10		10	10		1.2x10 ⁻⁵				
	PFUA	0.29	Animals (mice)	Surrogate: PFDaA	Reduced Body Weight		NOAEL (1 mg/kg/d)	81000	1	10		10	10		1.2x10 ⁻⁵				
	PFDaA	0.29	Animals (mice)	Shi et al., 2007	Reduced Body Weight		NOAEL (1 mg/kg/d)	81000	1	10		10	10		1.2x10 ⁻⁵				
	PFTrDA	0.29	Animals (mice)	Surrogate: PFDaA	Reduced Body Weight		NOAEL (1 mg/kg/d)	81000	1	10		10	10		1.2x10 ⁻⁵				
	PFTeDA	0.29	Animals (mice)	Surrogate: PFDaA	Reduced Body Weight		NOAEL (1 mg/kg/d)	81000	1	10		10	10		1.2x10 ⁻⁵				
VT	PFOA, PFOS, PFHxS, PFHpA, PFNA	0.02*	Animals (mice)	EPA (2016)	EPA (2016)	20	EPA (2016)	EPA (2016)								0.175 L/kg/day		0-1 year old	
WI	PFOA	0.02 (combined)*	Animals (mice)	Lau et al., 2006	Developmental (reduced ossification)	100	LOAEL	300	10	3	10							https://www.dhs.wisc.gov/water/gws.htm	
	PFOS	0.02 (combined)*	Animals (mice)	Luebker et al., 2005	Reduced pup body weight	100	NOAEL	30	3	10			10		1 (10 kg body wt)	Gestation and infancy (including breastfeeding)			

*= Advisory level is based on the total of more than one PFAS

Appendix C: State Surface Water PFAS Guideline Criteria

State	PFAS Analyte(s)	Advisory Level (ug/L)	Toxicity Data	Critical Effect Study	Endpoint	POD	UFs					RfD (mg/kg/day)	Drinking Water Intake Rate (L/day)	Resources & Notes
							Total	Interspecies	Intraspecies	LOAEL to NOAEL	Duration of Exposure (i.e., Subchronic to Chronic)			
MI	PFOA (drinking water source)	0.42	Animals (primates)	Butenhoff et al., 2002	Hepatotoxicity	LOAEL	3000	3	10	10	10	2×10^{-5}	2	https://www.michigan.gov/egle/0,9429,7-135-3313_3681_3686_3728-11383--,00.html
	PFOA	12	Animals (primates)	Butenhoff et al., 2002	Hepatotoxicity	LOAEL	3000	3	10	10	10	2×10^{-5}	0.01	
	PFOS (drinking water source)	0.011	Animals (primates)	Seacat et al., 2002	Decreased body weight, hepatotoxicity, thyroid toxicity	NOAEL	30	3	10			1.3667×10^{-5}	2	
	PFOS	0.012	Animals (primates)	Seacat et al., 2002	Decreased body weight, hepatotoxicity, thyroid toxicity	NOAEL	30	3	10			1.3667×10^{-5} mg/kg/day	0.01	
MN	PFOA, PFOS													<p>MN developed surface water criteria for PFOA and PFAS between 2007-2013, but these values are outdated and should not be used.</p> <p>MN is updating its surface water criteria for PFOA and PFOS, and developing criteria for PFHxS, PFBA, and PFBS. These criteria are expected to be available in early 2020. Note that these are site-specific criteria for the protection of human health (fish consumption and recreation).</p>
OR	PFOA	24												<p>Note: The Oregon wastewater initiation levels were adopted into rule (OAR 340-045-0100, Table A) in 2011. The PFAS are 5 chemicals on a list of 118 persistent priority pollutants for water that Oregon DEQ developed in response to state legislation. Municipal wastewater treatment plants with effluent exceeding initiation levels are required to develop a pollution prevention plan that becomes a part of their NPDES permit. The list and associated initiation levels were developed in consultation with a science advisory committee.</p>
	PFOS	300												
	PFNA	1												
	PFOSA	0.2												
	PFHpA	300												

*= Advisory level is based on the total of more than one PFAS

Appendix D: State Soil PFAS Guideline Criteria

State	PFAS Analyte(s)	Advisory Level (mg/kg, unless otherwise specified)	Toxicity Data	Critical Effect Study	Endpoint	RSC (%)	POD	UFs						RfD (mg/kg/day)	Drinking Water Intake Rate (L/day unless otherwise specified)	Exposure assumptions	Target Populations	Resources & Notes
								Total	Interspecies	Intraspecies	LOAEL to NOAEL	Database Limitation	Duration of Exposure (i.e., Subchronic to Chronic)					
AK	PFOA	2.2 in Arctic Zone, 1.6 under 40" zone, 1.3 over 40" zone, 0.003 migration to groundwater	Animals (mice)	Lau et al., 2006	Decreased ossification of pup proximal phalanges, accelerated preputial separation	100	EPA (2016)	EPA (2016)								Residential exposure for 6 yrs old child receptor	Child	http://dec.alaska.gov/media/7543/20180201_pcccl.pdf
	PFOS	2.2 in Arctic Zone, 1.6 under 40" zone, 1.3 over 40" zone, 0.0017 migration to groundwater	Animals (mice)	Luebker et al., 2005	Reduced pup body weight	100	EPA (2016)	EPA (2016)								Residential exposure for 6 yrs old child receptor	Child	http://dec.alaska.gov/media/7543/20180201_pcccl.pdf
FL	PFOA	1.3 (residential), 25 (industrial/commercial), 0.002 (leachability) Soil Cleanup Target Levels	Animals (mice)	Lau et al., 2006	Decreased ossification of pup proximal phalanges, accelerated preputial separation	20	5.3×10^{-3} mg/kg/day	300	3		10		10	2×10^{-5}	0.054 L/kg/day	Children- 200 mg/day, worker- 50 mg/day, oral	Children ages 0-6	
	PFOS	1.3 (residential), 25 (industrial/commercial), 0.007 (leachability) Soil Cleanup Target Levels	Animals (mice)	Luebker et al., 2005	decreased weight	20	5.1×10^{-4} mg/kg/day	30	3				10	2×10^{-5}	0.054 L/kg/day	Risk target level of 10^{-6} and hazard quotient of 1	Children ages 0-6	
IN	PFBS	1800 (residential), 16000 (commercial/industrial), 34000 (evacuation worker)	Animals (mice)	EPA RSL Tables		100										Direct contact exposure duration of 250 days/year, or 100000 mg/kg (10% by weight)		

State	PFAS Analyte(s)	Advisory Level (mg/kg, unless otherwise specified)	Toxicity Data	Critical Effect Study	Endpoint	RSC (%)	POD	UFs						RfD (mg/kg/day)	Drinking Water Intake Rate (L/day unless otherwise specified)	Exposure assumptions	Target Populations	Resources & Notes
								Total	Interspecies	Intraspecies	LOAEL to NOAEL	Database Limitation	Duration of Exposure (i.e., Subchronic to Chronic)					
MA	PFOA	0.720 ug/kg	Based on soil background data; 90th percentile.															Note: Method 1 standards. Based on 90th percentile value of soil background data set from Vermont soils.
	PFOS	2.000 ug/kg	Based on soil background data; 90th percentile.															
	PFNA	0.320 ug/kg	Based on soil background data; 90th percentile.															
	PFHxS	0.300 ug/kg	Based on soil background data; 90th percentile.															
	PFHpA	0.500 ug/kg	Based on soil background data; 90th percentile.															
	PFDA	0.30 ug/kg	Based on soil background data; 90th percentile.															
MI	PFOA	10 (ug/kg)	Animals (primates)	Butenhoff et al., 2002	Hepatotoxicity		LOAEL (3 mg/kg/day)	3000	3	10	10			10	2x10 ⁻⁵	0.01		https://www.michigan.gov/egle/0,9429,7-135-3311_4109-251790--,00.html
	PFOA (drinking water source)	0.35 (ug/kg)	Animals (primates)	Butenhoff et al., 2002	Hepatotoxicity		LOAEL (3 mg/kg/day)	3000	3	10	10			10	2x10 ⁻⁵	2		https://www.michigan.gov/egle/0,9429,7-135-3311_4109-251790--,00.html

State	PFAS Analyte(s)	Advisory Level (mg/kg, unless otherwise specified)	Toxicity Data	Critical Effect Study	Endpoint	RSC (%)	POD	UFs						RfD (mg/kg/day)	Drinking Water Intake Rate (L/day unless otherwise specified)	Exposure assumptions	Target Populations	Resources & Notes	
								Total	Interspecies	Intraspecies	LOAEL to NOAEL	Database Limitation	Duration of Exposure (i.e., Subchronic to Chronic)						Sensitive Developmental Endpoints
MI	PFOS	0.00024 (ug/kg)	Animals (primates)	Seacat et al., 2002	Decreased body weight, hepatotoxicity, thyroid toxicity		NOAEL (0.03 mg/kg/day)	30	3	10					1.3667x10 ⁻⁵	0.01		https://www.michigan.gov/egle/0,9429,7-135-3311_4109-251790--,00.html	
	PFOS (drinking water source)	0.00022 (ug/kg)	Animals (primates)	Seacat et al., 2002	Decreased body weight, hepatotoxicity, thyroid toxicity		NOAEL (0.03 mg/kg/day)	30	3	10					1.3667x10 ⁻⁵	2		https://www.michigan.gov/egle/0,9429,7-135-3311_4109-251790--,00.html	
MN	PFOA	0.24, 3.2 (ug/kg)	Animals (mice)	Numerous	Hepatotoxicity, Kidney Effects, Immunotoxicity, Developmental Effects	0.2 (combined HQ/RSC)	38 mg/L serum concentration	300	3	10	3	3			1.8x10 ⁻⁵		Resident, Industrial	Children, adults	https://www.pc.state.mn.us/waste/risk-based-site-evaluation-guidance
	PFOS	0.041, 0.56 (ug/kg)	Animals (mice)	Numerous	Hepatotoxicity, Thyroid effects, Immunotoxicity, Developmental Effects	0.2 (combined HQ/RSC)	2.36 ug/L serum concentration	100	3	10		3			3.1x10 ⁻⁶		Resident, Industrial	Children, adults	https://www.pc.state.mn.us/waste/risk-based-site-evaluation-guidance
	PFBA	38, 520 (ug/kg)	Animals (rats)	Numerous	Hepatotoxicity, Thyroid Effects	0.2 (combined HQ/RSC)	6.9 mg/kg/day	300	3	10		10			2.9x10 ⁻³		Resident, Industrial	Children, adults	https://www.pc.state.mn.us/waste/risk-based-site-evaluation-guidance
	PFBS	5.7, 77 (ug/kg)	Animals (mice)	Numerous	Developmental effects, Thyroid effects, Reproduction	0.2 (combined HQ/RSC)	60 mg/kg/day	300	3	10		3	3		1.4x10 ⁻³		Resident, Industrial	Children, adults	https://www.pc.state.mn.us/waste/risk-based-site-evaluation-guidance
	PFHxS	0.13, 1.7 (ug/kg)	Animals (rats)	Numerous	Hepatotoxicity, Thyroid Effects	0.2 (combined HQ/RSC)	32.4 ug/mL	300	3	10		10			9.7x10 ⁻⁶		Resident, Industrial	Children, adults	https://www.pc.state.mn.us/waste/risk-based-site-evaluation-guidance

State	PFAS Analyte(s)	Advisory Level (mg/kg, unless otherwise specified)	Toxicity Data	Critical Effect Study	Endpoint	RSC (%)	POD	UFs						RfD (mg/kg/day)	Drinking Water Intake Rate (L/day unless otherwise specified)	Exposure assumptions	Target Populations	Resources & Notes
								Total	Interspecies	Intraspecies	LOAEL to NOAEL	Database Limitation	Duration of Exposure (i.e., Subchronic to Chronic)					
TX	PFBA	0.2	Animals (mice)	MDH	Hepatotoxicity		NOAEL (6.9 mg/kg/d)	2400	1	10		10	3	2.9x10 ⁻³				Note: oral dose, 0.5 acre source area) (Res GWSoilng PCLs) https://www.tceq.texas.gov/assets/public/implementation/tox/evaluations/pfcs.pdf
	PFBuS	0.11	Animals (mice)	Leider et al., 2009, York et al., 2002	Systemic Toxicity		NOAEL (60 mg/kg/d)	42600	1	10		10	3	1.4x10 ⁻³				
	PFPeA	0.00032	Animals (mice)	Surrogate: PFHxS	Hematotoxicity		NOAEL (0.3 mg/kg/d)	78900	1	10	3	10		3.8x10 ⁻⁶				
	PFHxS	0.002	Animals (mice)	Hoberman and York, 2003	Hematotoxicity		NOAEL (0.3 mg/kg/d)	78900	1	10	3	10		3.8x10 ⁻⁶				
	PFHxA	0.00048	Animals (mice)	Surrogate: PFHxS	Hematotoxicity		NOAEL (0.3 mg/kg/d)	78900	1	10	3	10		3.8x10 ⁻⁶				
	PFHpA	0.0046	Animals (mice)	Surrogate: PFOS	Neurodevelopment		NOAEL (0.6 mg/kg/d)	26300	1	10	10	1		2.3x10 ⁻⁵				
	PFOS	0.05	Animals (mice)	Zeng et al., 2011	Neurodevelopment		NOAEL (0.6 mg/kg/d)	26300	1	10	10	1		2.3x10 ⁻⁵				
	PFOA	0.003	Animals (mice)	Macon et al., 2011	Mammary gland development		NOAEL (0.3 mg/kg/d)	24300	1	10	30	1		1.2x10 ⁻⁵				
	PFOSA	0.92	Animals (mice)	Surrogate: PFOA	Mammary gland development		NOAEL (0.3 mg/kg/d)	24300	1	10	30	1		1.2x10 ⁻⁵				
	PFNA	0.0031	Animals (mice)	Fang et al., 2010	Spleen Cell Death		NOAEL (1 mg/kg/d)	81000	1	10		10	10	1.2x10 ⁻⁵				
	PFDeA	0.022	Animals (mice)	Kawashima et al., 1995	Hepatotoxicity		NOAEL (1.2 mg/kg/d)	81000	1	10		10	10	1.5x10 ⁻⁵				
	PFDS	0.04	Animals (mice)	Surrogate: PFDoA	Reduced Body Weight		NOAEL (1 mg/kg/d)	81000	1	10		10	10	1.2x10 ⁻⁵				
	PFUA	0.018	Animals (mice)	Surrogate: PFDoA	Reduced Body Weight		NOAEL (1 mg/kg/d)	81000	1	10		10	10	1.2x10 ⁻⁵				
	PFDoA	0.034	Animals (mice)	Shi et al., 2007	Reduced Body Weight		NOAEL (1 mg/kg/d)	81000	1	10		10	10	1.2x10 ⁻⁵				
	PFTTrDA	0.061	Animals (mice)	Surrogate: PFDoA	Reduced Body Weight		NOAEL (1 mg/kg/d)	81000	1	10		10	10	1.2x10 ⁻⁵				
	PFTeDA	0.11	Animals (mice)	Surrogate: PFDoA	Reduced Body Weight		NOAEL (1 mg/kg/d)	81000	1	10		10	10	1.2x10 ⁻⁵				

State	PFAS Analyte(s)	Advisory Level (mg/kg, unless otherwise specified)	Toxicity Data	Critical Effect Study	Endpoint	RSC (%)	POD	UFs					RfD (mg/kg/day)	Drinking Water Intake Rate (L/day unless otherwise specified)	Exposure assumptions	Target Populations	Resources & Notes
								Total	Interspecies	Intraspecies	LOAEL to NOAEL	Database Limitation					
VT	PFOA, PFOS, PFHxS, PFHpA, PFNA	1.22*	Animals (mice)	EPA (2016)	EPA (2016)	20	EPA (2016)	EPA (2016)						0.175 L/kg/day			
WI	PFOA	1.26 (residential), 16.4 (composite [industrial] worker)		EPA RSL Tables									26 yrs, 350 days/yr, 24 hrs (residential), 25 yrs, 250 days/yr, 8 hrs (composite worker)			Vary through life (residential), 80 kg wt, 100 mg/day intake (composite worker) THQ=1, cancer risk 1x10 ⁻⁶ , other default assumptions	Residential, Composite Worker EPA RSL calculator
	PFOS	1.26 (residential), 16.4 (composite [industrial] worker)		EPA RSL Tables									26 yrs, 350 days/yr, 24 hrs (residential), 25 yrs, 250 days/yr, 8 hrs (composite worker)			Vary through life (residential), 80 kg wt, 100 mg/day intake (composite worker) THQ=1, cancer risk 1x10 ⁻⁶ , other default assumptions	Residential, Composite Worker EPA RSL calculator
	PFBS	1260 (residential), 16400 (composite [industrial] worker)		EPA RSL Tables									26 yrs, 350 days/yr, 24 hrs (residential), 25 yrs, 250 days/yr, 8 hrs (composite worker)			Vary through life (residential), 80 kg wt, 100 mg/day intake (composite worker) THQ=1, cancer risk 1x10 ⁻⁶ , other default assumptions	Residential, Composite Worker EPA RSL calculator

*= Advisory level is based on the total of more than one PFAS

Appendix E: State Air PFAS Guideline Criteria

State	PFAS Analyte(s)	Advisory Level ($\mu\text{g}/\text{m}^3$)	Toxicity Data	Critical Effect Study	Endpoint	POD	HED (mg/kg/day)	UFs					RfD (mg/kg/day)	Route-to-Route Extrapolation	Exposure Parameters	Target Populations	Resources
								Total	Interspecies	Intraspecies	LOEL to NOAEL	Duration of Exposure (i.e., Subchronic to Chronic)					
MI	PFOA (initial threshold screening level; ITSL)	0.07	Animals (mice)	EPA, 2016; Butenhoff et al., 2004; Lau, 2006	Reproductive, Developmental	EPA (2016)	0.0053; 0.0064	300	3	10	10	2 generations +developmental	2×10^{-5}	Air Value (ITSL) = RfD x 70kg/20m ³	Continuous over time period= 24 hours	Sensitive individuals	http://www.deq.state.mi.us/aps/downloads/ATSL/335-67-1/335-67-1_24hr_ITSL.pdf
	PFOS (initial threshold screening level; ITSL)	0.07	Animals (rats)	EPA, 2016; Luebker et al., 2005	Reproductive, Developmental	EPA (2016)	0.00051	30	10	3		2 generations +developmental	2×10^{-5}	Air Value (ITSL) = RfD x 70kg/20m ³	Continuous over time period= 24 hours	Sensitive individuals	http://www.deq.state.mi.us/aps/downloads/ATSL/1763-23-1/1763-23-1_24hr_ITSL.pdf
NH	APFO (CAS #3825-26-1; 24-hr Ambient Air Limit)	Regulatory Level 0.05	Animals (rats)	ACGIH TLV	Acute, Reproductive/ Developmental												
	APFO (CAS #3825-26-1; Annual Ambient Air Limit)	Regulatory Level 0.024	Animals (rats)	ACGIH TLV	Acute, Reproductive/ Developmental												
TX	PFOA (ESL) (CAS #335-67-1; based on annual average)	0.005		Republic of Germany DFG Maximum Concentration at the Workplace				1000							Occupational Exposure Limit		
	PFOS (ESL) (CAS #1763-23-1; based on annual average)	0.01		Republic of Germany DFG Maximum Concentration at the Workplace				100							Occupational Exposure Limit		

*= Advisory level is based on the total of more than one PFAS