PFASt and PFurious
Notable Updates in the PFAS World

K. Tyndall, G. Garvey, P. Goodrum, F. Becker, J. Wilhelm

Texas Environmental Superconference 2025 – Austin, Texas

"How Sweet It Is!" (Welcome to Candy Land)

TABLE OF CONTENTS

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EXHIBITS

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1.0 INTRODUCTION

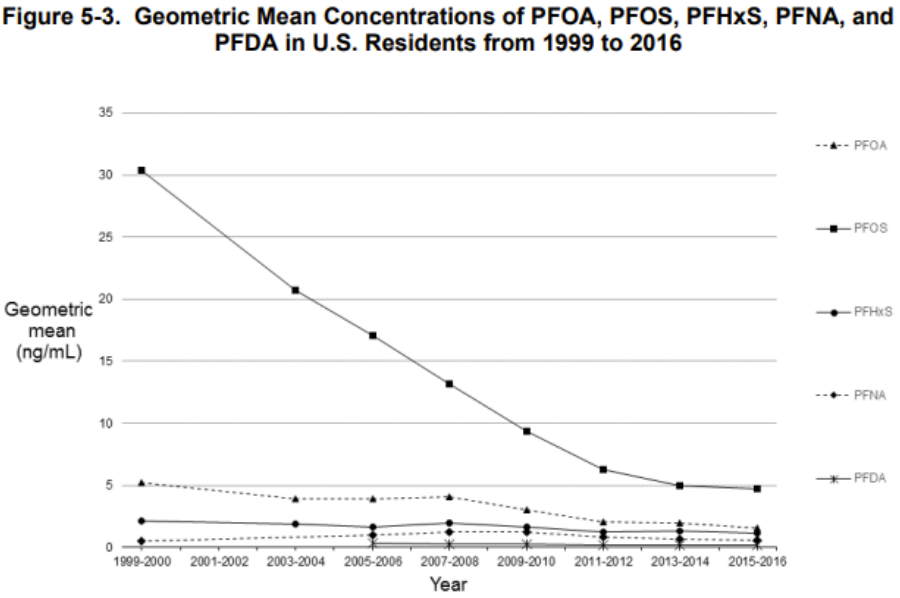
In 2024, the United States Environmental Protection Agency (EPA) enacted a flurry of per- and polyfluoroalkyl substances (PFAS) regulatory initiatives, scientific evaluations, and monitoring efforts as part of the PFAS Strategic Roadmap. In fact, in a document issued on January 14, 2025, EPA's Draft Sewage Sludge Risk Assessment for Perfluorooctanoic Acid (PFOA) CASRN 335-67-1 and Perfluorooctane Sulfonic Acid (PFOS) CASRN 1763-23-1[1], sixteen references were EPA PFAS-focused documents issued in 2024. Couple that with the significant actions that EPA enacted in 2024 (establishing Maximum Contaminant Levels, finalizing two analytical methods to be used for Clean Water Act permits, designating PFAS as a hazardous substance under the Comprehensive Environmental Response, Compensation, and Liability Act or CERCLA, etc.) and the many background documents, references, studies, and assessments that serve as the underpinnings to these actions, 2024 was a notable year at EPA. This paper will focus on the scientific evaluations used to support several of EPA's PFAS-specific rule-making initiatives.

The geometric mean serum PFOA and PFOS levels have decreased significantly in the general U.S. population, as shown below in **Exhibit 1**. Serum concentrations of other PFAS compounds have generally decreased or stayed at relatively consistent levels over the past two decades. This decrease is likely due to multiple factors including significant news coverage, increasing public awareness about potential health concerns related to PFAS levels in the environment, initiatives to phase out the use of PFOA and PFOS, and various regulations and voluntary actions aimed at reducing levels of PFAS in the environment, consumer goods, food packaging, etc. The Centers for Disease Control and Prevention (CDC) conducts the US biomonitoring program called the National Health and Nutrition Examination Survey (NHANES) from which these decreasing PFAS serum levels displayed in **Exhibit 1** are derived. Importantly, the CDC indicates that this data cannot be used to identify the source(s) of PFAS exposure, route of exposure, and/or likelihood of disease adverse effect and do not equate with a level of specific PFAS in air, water, food, soil or dust.[2]

[1] Draft Sewage Sludge Risk Assessment for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonic Acid (PFOS) | US EPA

[2] General Information "Interpretation of Report and Updated Tables Data: Important Considerations Section, pdf pages 18 and 19. Fourth National Report on Human Exposure to Environmental Chemicals. Updated Tables, March 2021: Volume One, NHANES 1999–2010

Exhibit 1. CDC's Summary of Serum Levels in the US Population over Time[3]



NHANES = National Health and Nutrition Examination Survey; PFDA = perfluorodecanoic acid; PFHxS = perfluorohexane sulfonic acid; PFNA = perfluorononanoic acid; PFOA = perfluorooctanoic acid; PFOS = perfluorooctane sulfonic acid

Source: CDC 2018

2.0 PFAS UPDATES FOR TEXAS AND THE CANDY LAND DOWN UNDER

The Texas Commission for Environmental Quality (TCEQ) has derived toxicity values for PFOA, PFOS, PFBS and other PFAS[4] and is in the process of updating their Development Support Document (DSD) for PFOA and PFOS. **Exhibit 2** provides a summary of the values currently used for the three PFAS referred to in this paper (PFOA, PFOS, and PFBS). TCEQ expects to finalize their updated DSD for PFOA and PFOS and will put forward the draft for public review and comment by the end of 2025[5]. TCEQ has developed air inhalation values (inhalation reference concentrations or RfCs) which are novel when compared to other regulatory agencies. For comparative purposes, TCEQ, EPA and the Australian Government National Health and Medical Research Council (NHMRC) toxicity values are provided. NHMRC released their updates to these toxicity values on June 25, 2025. The large difference between these values highlights the variability in scientific and risk management decision making approaches used by different regulatory agencies. Undoubtedly, other state and international agencies have made differing assumptions and decisions that result in the derivation of divergent health risk-based toxicity values. Because of these underlying scientific assumptions, differences in policy decisions, and the variable use of safety factors, these risk-based toxicity values do not represent levels of exposure where scientific evidence would suggest that adverse health effects or harm would occur.

[3] Toxicological Profile for Perfluoroalkyls
[4] PFAS
[5] Personal communication with Joseph "Kip" Haney of the TCEQ Toxicology Division on June 27, 2025.

The chemical-specific toxicity values are tremendously important in all risk-based calculations. For non-cancer endpoints, the oral reference dose (RfD) or inhalation RfC is used in risk-based calculations for oral/dermal and inhalation exposure pathways, respectively. For cancer evaluations, the oral cancer slope factor (CSF) or inhalation unit risk factor (IUR) is used in risk-based calculations for oral/dermal and inhalation exposure pathways, respectively. These toxicity values are generally linearly related to the estimated risk-based concentration for a given media with higher RfDs and RfCs resulting in higher allowable media-specific risk-based concentrations.

Exhibit 2. TCEQ, EPA and AUS Toxicity Values[6]

	TCEQ			EPA			AUSTRALIA NHMRC		
	Reference Dose (RfD)	Reference Concentration (RFC)	Oral Cancer Slope Factor	Reference Dose (RfD)	Reference Concentration (RFC)	Oral Cancer Slope Factor	Reference Dose (RfD)	Reference Concentration (RFC)	Oral Cancer Slope Factor
	mg/kg-day	mg/m3	(mg/kg-day)-1	mg/kg-day	mg/m3	(mg/kg-day)-1	mg/kg-day	mg/m3	(mg/kg-day)-1
PFOA	1.20E-05	4.10E-06	--	3.00E-08	--	2.93E+04	6.00E-05	--	--
PFOS	2.30E-05	8.10E-05	--	1.00E-07	--	3.95E+01	2.40E-06	--	--
PFBS	1.40E-03	4.90E-03	--	3.00E-04	--	--	3.20E-04	--	--
Notes:									
TCEQ - Texas Commission on Environmental Quality									
EPA - Environmental Protection Agency									
NHMRC - National Health and Medical Research Council									

3.0 MAXIMUM CONTAMINANT LEVELS (MCLS) FOR PFOA AND PFOS

Final drinking water standards (maximum contaminant levels or MCLs) were promulgated on April 10, 2024 after several years of proposed values (March 2023), re-evaluation, etc. MCLs are enforceable under the Clean Water Act with compliance being determined by a running annual average at the sampling point for public water systems. EPA received 1,626 comment submissions on the proposed 2023 MCLs.[7] To support the derivation of the MCLs, the EPA Office of Water (EPA OW) derived the toxicity values that are shown in **Exhibit 1** outside of the more typical seven-step health effects evaluation process within EPA's Integrated Risk Information Systems (IRIS). On May 14, 2025, EPA announced that the current MCLs for PFOA and PFOS of 4 nanograms per liter (ng/L) or parts per trillion (ppt) will be maintained while it intends to rescind the values and approach for several other PFAS. It has been noted that the MCLs may be the

[6] While TCEQ and NHMRC do not consider PFOA and PFOS to be carcinogenic, the International Agency for Research on Cancer (IARC) released their cancer hazard evaluation for PFOA and PFOS as recently as March 2025. IARC concluded that PFOA is *carcinogenic to humans* (Group 1) on the basis of *sufficient evidence* for cancer in experimental animals and *strong* mechanistic evidence (for epigenetic alterations and immunosuppression) in exposed humans with *limited evidence* for cancer in humans (renal cell carcinoma and testicular cancer) and *strong* mechanistic evidence in human primary cells and experimental systems (for epigenetic alterations and immunosuppression, as well as several other key characteristics of carcinogens). IARC concluded that PFOS is *possibly carcinogenic to humans* (Group 2B) on the basis of *strong* mechanistic evidence across test systems, including in exposed humans (for epigenetic alterations and immunosuppression, as well as several other key characteristics of carcinogens). There was also *limited evidence* for cancer in experimental animals and *inadequate evidence* regarding cancer in humans. IARC (2025). Perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS). *IARC Monogr Identif Carcinog Hazards Hum*. 135:1–754. https://publications.iarc.who.int/636

[7] Hua M, McCauley K, Brew D, Heywood J, Siracusa J, Stevens M, Paustenbach D. United States Environmental Protection Agency's Perfluorooctanoic Acid, Perfluorooctane Sulfonic Acid, and Related Per- and Polyfluoroalkyl Substances 2024 Drinking Water Maximum Contaminant Level: Part 1 - Analysis of Public Comments. Crit Rev Toxicol. 2025;55(3):321-367. doi: 10.1080/10408444.2024.2415893. Epub 2025 May 20. PMID: 40391661.

costliest environmental regulation to date given the extremely low MCLs, the widespread occurrence of PFOA and PFOS, and their recalcitrance in the environment.

There are significant concerns within the scientific community regarding the evaluations used to derive the PFOA and PFOS toxicity values that serve as the basis for the MCLs. We recognize that EPA has derived and approved the PFOA and PFOS toxicity values; however, the consequences of using irreproducible scientific methods – such as untested, poorly controlled, and novel epidemiological approaches – instead of well-established, reproducible toxicological evaluations are severe. Additionally, because the assumptions that EPA used are overly conservative and unsupported and, as such, these toxicity values are suspect, we feel compelled to identify areas of significant uncertainty and scientific disagreement. Our concern is amplified given the widespread influence these values have on all risk-based analyses for PFOA and PFOS, regardless of the media of concern, the pathway under consideration or the vast uncertainty and variability that the values inherently include. For brevity, we have only identified several high-level items for each toxicity value derived by the EPA OW for PFOA and PFOS.

3.1 PFOA Cancer Slope Factor

As shown in **Exhibit 1**, EPA OW CSF for PFOA is 29,300 $[mg/kg\text{-}day]^{-1}$. Of the 249 chemicals for which EPA lists a CSF in its Regional Screening Level table[8], only one chemical, dioxin, has a higher CSF. It is important to note that, unlike PFAS compounds that have many critical uses in various industries, dioxin was a by-product of combustion with no real or productive commercial use. The CSF for PFOA appears highly irregular and inconsistent with the potency of other known or suspected human carcinogens such as benzene, trichloroethylene, or benzo[a]pyrene. In addition, the determination of the potential carcinogenicity of PFOA relies on an epidemiology study (Shearer et al. (2021))[9] with several questionable scientific shortcomings described below. It is interesting to note that IARC classified PFOA as *carcinogenic to humans* (Group 1) largely because of animal and mechanistic studies but indicated that there was *limited evidence* for cancer in humans (renal cell carcinoma and testicular cancer), yet the renal cell carcinoma endpoint from one epidemiological study was the critical study chosen to derive the CSF for PFOA. By relying on one study to make such a determination, IARC and EPA discount the significant and disparate findings of other well-designed studies.

As previously mentioned, the PFOA CSF was derived based on kidney cancer in one study, Shearer et al. (2021)[10]. There are several concerns about this study: 1) the study considered serum concentrations at a single point in time, which is a poor representation for long-term average exposure, since the CSF is an estimate of the increase in cancer risk over a lifetime per unit increase in the lifetime average daily dose; 2) the study that EPA relied upon to calculate the CSF likely overestimates the half-life (and therefore, also the CSF) because it does not account for multiple sources of potential exposure to PFOA[11]; 3) several other studies are available that provide a more robust human exposure assessment compared to the single blood sample measurement in the Shearer et al. (2021) study; and 4) the positive association between lower kidney function and higher serum levels of PFOA may be biased by reverse causation, meaning

[8] Regional Screening Levels (RSLs) - Generic Tables | US EPA; November 2024 table update, accessed July 3, 2025.
[9] Shearer, JJ, et al. 2021. Serum concentrations of per- and polyfluoroalkyl substances and risk of renal cell carcinoma. Journal of the National Cancer Institute 113: 580-587. http://dx.doi.org/10.1093/jnci/djaa143.
[10] Shearer, JJ, et al. 2021.
[11] Sun Q, et al. 2018. Plasma Concentrations of Perfluoroalkyl Substances and Risk of Type 2 Diabetes: A Prospective Investigation among U.S. Women. Environ Health Perspect.126(3):037001.

that the change in kidney function triggers a lower elimination rate (and higher serum concentration) of PFOA.

Ultimately, an overarching concern with EPA's evaluation is that the CSF is inconsistent with other comprehensive evaluations. Based on the CSF and EPA point-of-departure of a one in a million excess lifetime cancer risk, the risk-based intake threshold is calculated to be 0.000034 ng/kg-day. By contrast, an independent international consortium assembled by the Alliance for Risk Assessment (ARA) completed a detailed, comprehensive evaluation of PFOA epidemiology and animal toxicity studies, and determined that limitations in both the study by Shearer et al. as well as animal toxicity studies on PFOA-induced liver tumors preclude the use of a cancer endpoint to derive a risk-based safe dose for PFOA. Based on a range of non-cancer endpoints, the authors determined a safe human dose for PFOA ranges between 10 to 70 ng/kg-day[12]. EPA's risk-based dose for PFOA is between 300,000 and 2 million times lower than the value derived by ARA, which highlights the extreme differences that changing assumptions, models and scientific decision points can have when establishing toxicity values.

3.2 PFOA Reference Dose

EPA OW's RfD for PFOA is 0.03 ng/kg-day. This value is based on multiple endpoints, including immunosuppression of antibody titers for tetanus and diphtheria as well as low birth weight and increased total cholesterol. EPA's use of these epidemiological endpoints has been controversial since the clinical significance of these endpoints is debatable, and most toxicity values used by EPA for risk-based criteria development are based on laboratory animal studies where there is some precision in dosing and study design. A criticism of using epidemiological studies for deriving an RfD is that epidemiological studies often have a poor understanding of dose and limitations with the strength of association. In addition, these endpoints have not been associated with any increases in actual adverse health outcomes (e.g., increased incidences of diphtheria and tetanus in a population or the clinical significance of a slight increase in liver enzymes). This is highlighted by Antoniou et al. (2022),[13] which found that a reduced antibody response is not a predictor of immune response, nor does it necessarily relate to increased disease or adverse health outcomes.

An international panel of experts also shared similar conclusions after participating in a double-blind review process to evaluate PFAS exposure and immunotoxicity (Garvey et al. 2023)[14]. This panel concluded that while epidemiological data can be a useful metric to inform the potential immunomodulation of chemical exposures, it is not suitable for deriving toxicity values upon which regulatory values are based. The panel identified several factors that could influence the results of such studies, including vaccine types and schedules, age, gender, socioeconomic factors and co-morbidities. Additionally, more recent meta-analyses of PFAS and immunotoxicity have not found any associations between PFAS and antibody responses in other populations[15].

[12] Burgoon, LD, et al. 2023.
[13] Antoniou, E, et al. 2022. Immunomodulation and exposure to per- and polyfluoroalkyl substances: an overview of the current evidence from animal and human studies. Arch Toxicol 96:2261–2285.
[14] Garvey, GJ, et al. 2023. Weight of evidence evaluation for chemical-induced immunotoxicity for PFOA and PFOS: findings from an independent panel of experts. Crit Rev Toxicol 53(1):34-51.
[15] Crawford L *et al.* Systematic review and meta-analysis of epidemiologic data on vaccine response in relation to exposure to five principal perfluoroalkyl substances, Environ Intern 172:107734.

3.3 PFOS Cancer Slope Factor

As shown in **Exhibit 1**, EPA OW CSF for PFOS is 39.5 [mg/kg-day]$^{-1}$. A significant flaw with this CSF is that the critical study relies on a single laboratory rat study for liver effects[16] that may have little relevance to humans due to potential species-specific mode of action considerations (non-human relevant mechanisms involving xenobiotic nuclear receptors, such as PPARα) and no clear dose response was noted. Based on EPA's own cancer risk assessment guidelines[17], the animal data evaluating PFOS carcinogenicity are "suggestive", at best, and are not definitive or supportive of a "likely" classification for human cancer risk. IARC considered the animal data as being *limited evidence of carcinogenicity*. In fact, in 2016, EPA OW concluded that "the weight of evidence for the carcinogenic potential to humans was judged to be too limited to support a quantitative cancer assessment."[18] In summary, the decision by EPA to classify PFOS as a carcinogen and derive an associated CSF is not supported by a credible evaluation of the scientific evidence for this compound and results in an unreliable CSF.

3.4 PFOS Reference Dose

The EPA OW RfD for PFOS is 0.1 ng/kg-day. It was derived from a statistical evaluation of the U.S. NHANES database looking at cholesterol and various PFAS compounds in serum (Dong et al., 2019)[19]. This kind of correlation analysis can identify large trends and associations but cannot establish causality, as specifically acknowledged by the study authors: "The NHANES data are capable of examining the association but cannot address the issue of causality. Similar to other cross-sectional studies, this study cannot answer whether: 1) exposure to PFASs elevates the cholesterol level; 2) high cholesterol levels allow the storage of PFASs easier; or 3) joint factors simultaneously affect both PFASs and cholesterol." The study of cholesterol effects is impacted by many variables. One such variable, diet, was excluded and not controlled for in this study, with the justification that "A previous study indicated other factors (such as diet) had little effect on the associations between serum PFAS and cholesterol." A review of the cited study, however, did not confirm this conclusion[20]. For this reason and others, it is our opinion that the Dong et al., 2019 study is not suitable to provide the basis for deriving an RfD for PFOS.

3.5 Working Toward More Useable Toxicity Value Evaluation for PFOA and PFOS

Given the many uncertainties and limitations of the available epidemiology data and toxicity assessments conducted by the EPA OW, it is our opinion that the toxicity values for PFOA and PFOS should be based on robust experimental animal data that is consistent, reproducible, biologically plausible in humans, and demonstrates a clear dose-response relationship. As enumerated above, the PFOA and PFOS human data are generally only useful as a qualitative line of evidence and to help with identifying hazards. The many confounding factors associated with most epidemiology studies cannot be adequately accounted for and thereby restrict their usefulness for quantifying exposure and "safe" risk-based levels. Given the enormous implications the toxicity values for PFOA and PFOS will have, from which their respective MCLs are loosely

[16]Butenhoff *et al.* Chronic dietary toxicity and carcinogenicity study with potassium perfluorooctanesulfonate in Sprague Dawley rats. *Toxicol* 293(1-3):1-15 (2012).
[17] EPA. 2005. "Guidelines for Carcinogen Risk Assessment." Risk Assessment Forum, U.S. Environmental Protection Agency, Washington, DC.
[18] USEPA PFOS HESD 2016, at ES-2.
[19] Dong, Z, et al. 2019.
[20] Nelson, JW, et al. 2010. Exposure to polyfluoroalkyl chemicals and cholesterol, body weight, and insulin resistance in the general U.S. population. Environ. Health Perspect. 11:197-202.

based on, the PFOA and PFOS toxicity evaluations would benefit from a more balanced, thorough and defensible assessment.

4.0 HUMAN HEALTH AMBIENT WATER QUALITY CRITERIA

EPA published Draft National Recommended Ambient Water Quality Criteria (AWQC) for the Protection of Human Health for Perfluorooctanoic Acid (PFOA), Perfluorooctane Sulfonic Acid (PFOS), and Perfluorobutane Sulfonic Acid (PFBS) (the "Draft PFAS Human Health Criteria" or the "Draft PFAS HHC") on December 26, 2024 (90 Fed. Reg. 105041). EPA issued a separate document, one for each PFAS and largely followed the standardized approach previously described by EPA[21]. Thus, because of the similar approach used by EPA, discussion provided in this paper generally applies to all three documents. The original comment deadline for these documents was February 24, 2025 although that was later extended to April 24, 2025 (and extended again to April 29, 2025 due to a planned outage of *Regulations.gov*). EPA received 781 comments on the Draft PFAS HHC documents. The Draft PFAS HHCs derived in the criteria documents[22] are shown in **Exhibit 3**:

Exhibit 3. Summary of EPA's HHCs for PFOA, PFOS and PFBS

Table 1. Draft Human Health Criteria (HHC) for Three PFAS.

PFAS	Water + Organism HHC (ng/L; ppt)[1]	Organism Only HHC (ng/L; ppt)[1]
PFOA	0.0009	0.0036
PFOS	0.06	0.07
PFBS	400	500

[1] Values are provided in ng/L units to aid in comparison to method detection limit (MDL).

Our concern regarding these very low HHCs as proposed in the Draft PFAS HHC documents is that these values are overly conservative, do not accurately predict real human health risks, and may unnecessarily result in many or all waters of the United States being designated as "impaired" which could significantly and unnecessarily curtail the use and enjoyment of these waters. In fact, per Tables 5-12 and 5-13 of the Toxicological Profile for PFAS[23], detectable levels of PFOA and PFNA were found at all surface water bodies sampled/surveyed, and PFHxS, PFOS, and PFDA were detectable in >90% of the surface water bodies. Given the low HHCs for PFOA and PFOS, these water bodies would likely be designated as "impaired" even though there may not be a point source and there is likely no remedial action or technology that could be deployed to reduce PFOA and PFOS concentrations in surface water below these proposed levels.

As presented by EPA, the Draft HHC are estimated from a standard risk-based model that include the assumed adverse health effects associated with the contaminant (either carcinogenic or non-carcinogenic), the extent of contaminant bioaccumulation into fish, the assumed relative source contribution (RSC) used to account for other potential sources of exposure not related to the surface water body, and the assumed rate of fish and/or water ingestion. These assumptions,

[21] EPA. 2000. *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000)*. EPA 822-B-00-004. Available on the internet at: https://www.epa.gov/sites/default/files/2018-10/documents/methodology-wqc-protection-hh-2000.pdf.

[22] Technical Fact Sheet: Draft National Recommended Human Health Ambient Water Quality Criteria for PFOA, PFOS, and PFBS

[23] Toxicological Profile for Perfluoroalkyls

which directly affect the estimated Draft HHC in a linear fashion, are discussed in greater detail in the following subsections.

4.1 Toxicity Values Used to Calculate the AWQ Human Health Criteria

The toxicity value used to calculate the HHC is one of the most important aspects of the HHC calculation. As discussed in greater detail in Section 3, the RfDs for PFOA and PFOS are some of the lowest values derived by EPA. Likewise, the CSF for PFOA is one of the highest CSF values, second only to dioxin. It should be noted that higher CSFs and lower RfDs result in lower risk-based criteria such as the HHCs. These toxicity values directly impact the HHC, resulting in unnecessarily low recommended concentrations.

4.2 Fish Consumption Rate Used to Calculate the AWQ Human Health Criteria

One of the critical parameters in the HHC calculations is the fish consumption rate. EPA's assumed fish consumption rate used in the HHC calculation represents a 90th percentile consumption rate of 22 g/day (from a short-term 2-day dietary recall survey) or roughly 6.6 ounces of fish per week and assumes all fish consumed are sourced from one water body. This consumption rate overestimates exposure for the majority of people *especially* considering that the majority of fish consumed in the United States are purchased and not locally sourced.[24] Even EPA (EPA, 2011, Section 10.3.1.1)[25] recognizes the uncertainty and challenges in using short-term consumption survey data and indicates that these data may not represent longer term consumption rates stating, "[B]ecause the increased variability of the short-term distribution, the short-term upper percentiles shown here may overestimate the corresponding percentiles of the long-term distribution."

When subsistence fishing is the concern for a given water body, a better policy would be to simply evaluate those situations on a case-by-case basis, which is often done without the presumption that most people catch and consume locally caught fish at a 90th percentile consumption rate and allows for waterbody-specific information to be considered. Furthermore, it should be noted that just because a waterbody is considered fishable, many smaller water bodies that are under the jurisdiction of the Clean Water Act are not sufficiently productive fisheries, which means that they could not provide the quantity of fish to support the consumption rate used in the HHC calculations for a large population of people.

4.3 Bioaccumulation Factors Used to Calculate the AWQ Human Health Criteria

EPA stated in the Draft National Recommended Ambient Water Quality Criteria for the Protection of Human Health documents for PFOA, PFOS and PFBS that they followed the approach described in Figure 3-1 of the Technical Support Document, Volume 2.[26] The EPA claims to have used the best available data to classify each chemical according to this framework, and to derive the most appropriate BAFs following the 2000 Methodology[27] and Technical Support Document, Volume 2.[28] The EPA conducted a systematic literature search in October 2022 of publicly

[24] U.S. Department of Agriculture, Economic Research Service (USDA). (n.d.). Aquaculture. Last update: 1/6/2025. Available at https://www.ers.usda.gov/topics/animal-products/aquaculture
[25] EPA. 2011. Exposure Factors Handbook, Office of Research and Development, EPA/600/R-090/052F.
[26] EPA. 2003. Technical Support Document Volume 2: Development of National Bioaccumulation Factors
[27] EPA. 2000. Guidance for assessing chemical contaminant data for use in fish advisories. Volume 1: fish sampling and analysis. EPA/823/B-00/007.
[28] EPA. 2003. Technical Support Document Volume 2: Development of National Bioaccumulation Factors

available literature sources and identified peer-reviewed literature sources, government reports, and professional society proceedings, when sufficient information was provided to indicate the quality and usability of the data.

The data collated by EPA for the BAF derivations also includes non-edible tissues that are not relevant for establishing HHCs. In its 2000 guidance,[29] EPA states that "BAFs should be based on concentrations in the edible tissue(s) of the biota unless it is demonstrated that whole-body BAFs are similar to edible tissue BAFs". The Draft HHC documents for PFOA, PFOS and PFBS do not include any evaluation of which whole body data are appropriate to include/exclude for human health and even includes at least one study where the fish tissue is identified by EPA as "presumed whole body (not specified)." The BAFs used in the calculation of the HHCs likely result in an unjustified lowering of the HHCs.

4.4 Relative Source Contribution Used to Calculate the AWQ Human Health Criteria

The EPA uses a "relative source contribution" (RSC) value to reflect "background exposures" for chemical-specific exposures in some regulatory programs. RSCs are most often used when setting public drinking water standards. EPA's RSC typically ranges from 20–80% depending on the chemical. Using an RSC in this manner assumes that a portion of exposure for the RfD is derived from exposure sources not related to ambient surface water and fish consumption (from the surface water body). In essence, the RSC is an additional safety factor used to ensure that a person's exposure from all potential sources does not exceed the RfD. The derived HHC is directly related to the RSC, with a lower RSC resulting in a lower HHC.

For PFOA, PFOS, and PFBS, EPA chose to use an RSC of 20% because "the EPA determined there is not enough information available on each source to make a quantitative characterization of exposure among exposure scenarios"[30] using the Exposure Decision Tree Framework[31] and the EPA assumes that 80% of a person's exposure comes from other sources. Exposure to PFAS, especially PFOA and PFOS, has been well studied for over a decade so this conclusion is debatable. In fact, the EPA provides numerous studies for each category describing PFAS levels in dietary sources, food contact materials, consumer product uses, indoor dust, and ambient air. It is unclear why these data are not adequate for estimating exposure levels since an RSC value derived with some delimited uncertainty is far better than a default value, especially for well-studied compounds like PFOA, PFOS and PFBS. There have been several studies of dietary, dust, and inhalation exposure to PFOA and PFOS, none of which suggest that exposures other than drinking water are likely to add up to 80% of the allowable daily intake.[32,33] Given the low dietary levels of PFAS in the United States, significant decreases in exposure of PFOA and PFOS over the last two decades as demonstrated with biomonitoring data as shown in **Exhibit 1**, and regulatory programs designed to reduce the likelihood of significant background exposure, a

[29] EPA. 2000. Guidance for assessing chemical contaminant data for use in fish advisories. Volume 1: fish sampling and analysis. EPA/823/B-00/007. See pp. 5-60.
[30] PFOS and PFOA HH AWQC documents, Section 6.2.6; PFBS HH AWQC document, Section 6.2.7.
[31] Figures 2 of the Draft HHC documents for FPOA, PFOS and PFBS provide the Decision Tree Framework that was adapted from Figure 4-1 of USEPA, 2000, https://www.epa.gov/sites/default/files/2018-10/documents/methodology-wqc-protection-hh-2000.pdf
[32] Lorber, M and Egeghy, PP. 2011. Simple intake and pharmacokinetic modeling to characterize exposure of Americans to perfluorooctanoic acid, PFOA. Environ Sci Technol 45(19): 8006-14.
[33] Sunderland et al. 2019. A review of the pathways of human exposure to poly- and perfluoroalkyl substances (PFASs) and present understanding of health effects. J Exp Sci and Environ Epidemiol 29(2): 131-147.

higher background intake assumption (RSC value) could be used and would result in a higher, more realistic HHC.

4.5 Working Toward a More Useable HHC for PFOA, PFOS and PFBS

Compounded conservatism occurs when high-end and unrealistic exposure assumptions are used in conjunction with toxicity values that may not be grounded in sound science. Compounded conservatism results in unusable and unattainable standards that do not provide additional protection to human health and the environment because they are orders of magnitude lower than available scientific evidence would suggest is necessary but for the reliance of high-end estimates of exposure and toxicity. We believe that it is possible to use EPA's general approach with more realistic assumptions and a more robust analysis of available peer-reviewed studies to derive scientifically defensible criteria that sufficiently protect human health and the environment. To date, EPA has not finalized these Draft National Recommended Ambient Water Quality Criteria for the Protection of Human Health or issued a response to comments.

5.0 PFAS IN BIOSOLIDS

On January 14, 2025, EPA issued a Draft Sewage Sludge Risk Assessment for Perfluorooctanoic Acid (PFOA) CASRN 335-67-1 and Perfluorooctane Sulfonic Acid (PFOS) CASRN 1763-23-1 (USEPA, 2025) (hereafter referred to as the SSRA). The comment period was originally set for March 17, 2025, but the comment period was extended to August 14, 2025. In 1987, the US Congress passed the Water Quality Act which required the EPA to 1) Establish numerical limits and management practices that protect public health and the environment from the reasonably anticipated adverse effects of toxic pollutants in sewage sludge; and 2) Periodically review existing regulations for the purpose of identifying additional toxic pollutants that may be present in sewage sludge and assesses whether those pollutants may adversely affect public health or the environment based on their toxicity, persistence, concentration, mobility, and potential for exposure[34]. Pollutant limits were established for pathogens and ten inorganic compounds in 1993 using a multi-pathway analysis that considered various land disposal scenarios. Over time, EPA added additional compounds, including organic compounds, to the list of compounds potentially in biosolids for consideration and evaluation.

EPA acknowledges that the purpose of this SSRA is to inform future risk mitigation efforts for PFOA and PFOS under the Clean Water Act and that this document is not intended to be enforceable (i.e., it is not a rule and does not compel any actions). Additionally, EPA indicates that the risk derived in this model for PFOA and PFOS is not representative of the general population and does not contain pollutant limits. However, it is noted in the SSRA that potential risks are linear (higher or lower) from the 1 part per billion (ppb) proxy concentration used to represent PFOA and PFOS concentrations in biosolids. The estimated cancer risks and non-cancer hazard quotients using the 1 ppb proxy concentration, modeled uptake and exposure pathways, and toxicity values derived by the EPA OW were higher for almost all of the scenarios and pathways evaluated than EPA's "acceptable" risk and hazard goals.[35] The highly conservative and hypothetical nature of this SSRA results in categorical risks that strongly

[34] Sewage Sludge Laws and Regulations | US EPA

[35] EPA's indicated that the SSRA contains a refined risk assessment using central tendency assumptions (not the high-end deterministic screening level evaluation that was originally performed). Despite the central tendency exposure assumptions, estimated risks and hazards were unacceptable for many pathways/scenarios evaluated by EPA.

suggest reusing biosolids is harmful under the scenarios evaluated and calls into question the validity and usefulness of the SSRA.

5.1 Toxicity Values Used in the Biosolids PFAS Risk Assessment

Similar to the HHC calculation discussed in the preceding section, the toxicity values used to calculate the risks and hazards are one of the most important aspects of the biosolids risk assessment. **Exhibit 1** provides a summary of the toxicity values for PFOA and PFOS. Carcinogenic and non-carcinogenic risks and hazards were calculated for both PFOA and PFOS in the SSRA using these toxicity values and, as discussed previously in Section 3, there is significant uncertainty associated with these values. As such, there is considerable uncertainty in the estimated risks/hazards of the SSRA.

5.2 Biosolids Risk Assessment Was Conducted Without an Understanding of Data from a National Sewage Sludge Survey

PFAS have been measured in domestic wastewater and sewage sludge (i.e., biosolids) for over two decades[36] so this is not an emergent issue. National Sewage Sludge Surveys (NSSS) were conducted in 1988, 2001, and 2006 with a new survey currently underway[37] to specifically evaluate the presence of PFAS in biosolids. However, some "background" data were presented by EPA in the Appendices of the SSRA, but EPA mostly discounted these data as representing 'highly contaminated" biosolids. With previous biosolid risk evaluations, EPA used data from the NSSS for various compounds and, in some cases, such as with arsenic, used the 98th percentile concentration as the pollutant limit since risk-based limits were lower than concentrations typically measured in biosolids. Having the benefit of the NSSS data that is currently being collected, or a recent accounting of the state of the science related to PFAS in biosolids in the US, and a better understanding about the magnitude and frequency of PFAS concentrations in biosolids would be very beneficial to understanding the validity and potential national impacts of the SSRA.

5.3 Exposure Assessment Assumptions Used in SSRA

We noted several general conservative assumptions used in the model that cause the evaluation to likely overestimate potential risks and limit the usefulness of the evaluation. The SSRA Executive Summary provides an overview of the rationale for several modeled "non-conservative" assumptions. Among these assumptions, the agency lists using 1 part per billion (ppb) PFOA or PFOS concentration for biosolids and the calculation of 50th percentile intake rates for specific agricultural use pathways as a way to convey median exposure conditions. The report also notes that the modeling efforts did not account for PFAS additivity, transformation, or non-sewage sludge exposure sources, and thus likely underestimates their occurrence and behavior in both environmental media and the human body. It should be noted that exposure to the general public was not considered, so only high-end use scenarios (e.g., self-sustaining family farm or ranch where fishing also occurs in an area impacted by runoff) were targeted.

The methodology described in the report states that it targets median exposure conditions. However, EPA instead applies a variety of conservative assumptions that are unrealistic in nature such as: 1) outdated food intake rates in humans, 2) assuming 100% of the persons crop intake

[36] Venkatesan AK; Halden RU 2013. National Inventory of Perfluoroalkyl Substances in Archived U.S. Biosolids from the 2001 EPA National Sewage Sludge Survey. J. Hazard. Mater 252–253, 413–418. https://doi.org/10.1016/j.jhazmat.2013.03.016

[37] Sewage Sludge Surveys | US EPA

is grown/raised on PFAS-impacted soil, 3) not a using robust enough evaluation to estimate bioconcentration factors (BCFs) for estimating PFOA and PFOS concentrations in crops and livestock to accurately depict the range of BCF values possible, 4) not considering PFAS loss during food preparation and cooking (for which there are conflicting study results), 5) assuming that there is no tilling of soil during biosolids application in the animal pasture scenario, and 6) the parameters used to model runoff and infiltration scenarios, among others. The adoption of different values for model parameterization such as using the mean residence time (and consequently period of exposure) of 10 years for the farming family is unusual, compared to the 26 years used in typical EPA calculations that reflect a 90th percentile risk estimation, and 20 years in the original EPA sewage sludge pollutant limit calculations authored by the agency in 1993. We believe that the "cherry-picking" of exposure assumptions highlights the challenges of performing a risk assessment when some of the science is unreliable and poorly sourced, in the absence of robust analysis of empirical biosolids data.

5.4 Overall Conclusions of EPA's Sewage Sludge Risk Assessment

As with all models, the evaluation is limited by the assumptions contained therein. We contend that the SSRA for PFOA and PFOS is constructed on too many conservative assumptions that likely overestimate exposure and potential risk/hazards, and it ultimately does not provide a reliable assessment to inform biosolids management. Since the only "acceptable" risk scenario for disposing of biosolids was shown to be disposal in a composite lined landfill, the conclusion of the study suggests that using biosolids to amend soil for agricultural purposes or in areas where it may migrate to groundwater or surface water is unsafe, even at levels as low as 1 ppt, provides few reasonable or realistic risk management options.

6.0 CONCLUSIONS

Risk-based evaluation tools were developed to provide management options that allowed for safe levels of compounds to be left in place and to avoid scenarios where "background levels" became the regulatory standard or cleanup criteria for naturally-occurring compounds. We are concerned that choosing to always err on the side of conservatism results in 1) overly protective decisions that are inconsistent with available science and cause background concentrations of inorganic or ubiquitous organic compounds like PFAS to become the *de facto* criteria because the risk-based standards are artificially low; 2) tremendous cost implications with no added health protection or net societal benefit; and 3) unintended consequences such as unnecessarily restricting the use of precious natural resources like groundwater and surface water bodies, increasing the amount of biosolids that are incinerated (without the technology to capture PFAS air emissions), or vast amounts of waste produced to treat water, soil and other media to exceedingly low concentrations (some of which are below the limits of analytical detection). The range of toxicity values and risk-based concentrations between various agencies and programs for PFOA and PFOS highlights that there is a range of plausible, defensibly safe options that are sufficiently protective of human health and the environment.