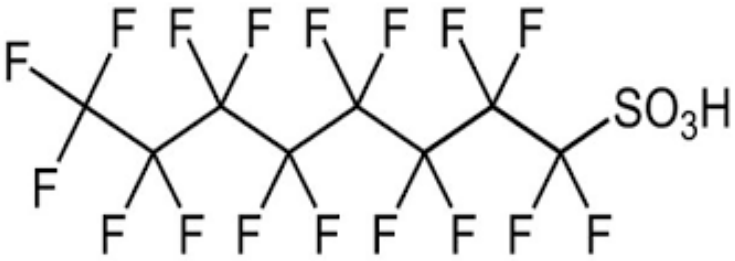
United States Environmental Protection Agency

Office of Water
Office of Science and Technology

EPA 822P24002
December 2024

DRAFT Human Health Ambient Water Quality Criteria:

Perfluorooctane Sulfonic Acid (PFOS) and Related Salts



Acknowledgements
This document was prepared by the Health and Ecological Criteria Division, Office of Science and Technology, Office of Water (OW) of the U.S. Environmental Protection Agency (EPA).

The OW scientists and managers who provided valuable contributions and direction in the development of these recommended water quality criteria are, from OST: Brandi Echols, PhD; Casey Lindberg, PhD; Czarina Cooper, MPH; Brittany Jacobs, PhD; Carlye Austin, PhD; Kelly Cunningham, MS (formerly OST); Erica Fleisig; Susan Euling, PhD; and Colleen Flaherty, MS; and, from the Office of Research and Development (ORD): Jason Lambert, PhD.

Table of Contents

1 Introduction: Background and Scope

The U.S. Environmental Protection Agency's national recommended ambient water quality criteria (AWQC) for human health are scientifically derived numeric values that define ambient water concentrations that are expected to protect human health from the adverse effects of individual pollutants in ambient water.

Section 304(a)(1) of the Clean Water Act (CWA) requires the EPA to develop and publish, and from time-to-time revise, recommended criteria for the protection of water quality that accurately reflect the latest scientific knowledge. Water quality criteria for human health developed under section 304(a) are based solely on data and scientific judgments about the relationship between pollutant concentrations and human health effects. Section 304(a) criteria do not reflect consideration of economic impacts or the technological feasibility of meeting pollutant concentrations in ambient water.

The EPA's recommended section 304(a) criteria provide technical information for states and authorized Tribes[a] to consider and use in adopting water quality standards that ultimately provide the basis for assessing water body health and controlling discharges of pollutants into waters of the United States. Under the CWA and its implementing regulations, states and authorized Tribes are required to adopt water quality criteria to protect the designated uses of waters (e.g., public water supply, aquatic life, recreational use, industrial use). The EPA's recommended water quality criteria do not substitute for the CWA or regulations, nor are they regulations themselves. Thus, the EPA's recommended criteria do not establish legal rights or obligations or impose legally binding requirements and are not final agency actions. States and authorized Tribes may adopt, where appropriate, other scientifically defensible water quality criteria that differ from these recommendations. EPA's water quality standards regulation at 40 CFR 131.20(a) requires states and authorized Tribes to consider any new or updated national section 304(a) recommended criteria as part of their triennial review process, and, if the state or authorized Tribe does not adopt new or revised criteria for parameters that correspond to those new or revised 304(a) criteria, to provide an explanation when it submits its triennial review to EPA. This requirement is to ensure that state or Tribal water quality standards reflect the current science and protect applicable designated uses.

The water quality criteria that are the subject of this document are draft national AWQC recommendations for human health issued under CWA section 304(a). Unless expressly indicated otherwise, all references to "human health criteria," "criteria," "water quality criteria," "ambient water quality criteria recommendations," or similar variants thereof are references to draft national AWQC recommendations for human health.

[a] Throughout this document, the term *states* means the 50 states, the District of Columbia, the Commonwealth of Puerto Rico, the Virgin Islands, Guam, American Samoa, and the Commonwealth of the Northern Mariana Islands. The term *authorized Tribe* or *Tribe* means an Indian Tribe authorized for treatment in a manner similar to a state under CWA section 518 for the purposes of section 303(c) water quality standards.

Perfluorooctane sulfonic acid (PFOS) is a member of the per- and polyfluoroalkyl substances (PFAS) class. PFAS are a large class of thousands of synthetic chemicals that have been in use in the United States and around the world since the 1940s (EPA, 2018). The ability for PFAS to withstand heat and repel water and stains makes them useful in a wide variety of consumer, commercial, and industrial products, and in the manufacturing of other products and chemicals. The current scientific evidence has shown the potential for harmful health effects after human exposure to certain PFAS. The persistence and resistance to hydrolysis, photolysis, metabolism, and microbial degradation of PFAS raise additional concerns about long-term exposure and human health effects.

The EPA developed the draft human health criteria (HHC) for PFOS to reflect the latest scientific information for input values including exposure factors (i.e., body weight [BW], drinking water intake [DWI] rate, and fish consumption rate [FCR]), bioaccumulation factors (BAFs), human health toxicity values (i.e., reference dose [RfD] or cancer slope factor [CSF]), and relative source contribution (RSC). The draft criteria are based on the EPA's current *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health* (2000a), which is referred to as the "2000 Methodology" in this document (EPA, 2000a).

2 Problem Formulation

Problem formulation provides a strategic framework for ambient water quality criteria development to systematically identify the major factors and chemical-specific scientific issues to be considered in the assessment (EPA, 2014a). The structure of this draft criteria document is intended to be consistent with general concepts of health assessments as described in the EPA's *Framework for Human Health Risk Assessment to Inform Decision Making* (EPA, 2014a).

In developing AWQC, the EPA follows the assessment method outlined in the 2000 Methodology (EPA, 2000a). The 2000 Methodology describes different approaches for addressing water and nonwater exposure pathways to derive human health AWQC depending on the toxicological endpoint of concern, the toxicological effect (noncarcinogenic or carcinogenic), and whether toxicity is considered a linear or threshold effect. Water sources of human exposure include both consuming drinking water and eating fish or shellfish from inland and nearshore water bodies that have been contaminated with pollutants. For pollutants that exhibit a threshold of exposure before deleterious human health effects occur, as is the case for noncarcinogens and nonlinear carcinogens, the EPA applies an RSC. The RSC is the percentage of the total exposure to a contaminant that is attributed to the combination of drinking water and eating freshwater and estuarine fish and shellfish, where the remainder of exposure is allocated to other sources of oral exposure and other routes of exposure. The RSC is calculated by examining the data for other sources of exposure (e.g., air, food, soil) and pathways of exposure following the exposure decision tree for calculation of an RSC described in the 2000 Methodology (EPA, 2000a).

For carcinogenic substances for which the cancer slope factor was quantified using linear low-dose extrapolation, only the exposures from drinking water and fish ingestion are reflected in human health AWQC; that is, nonwater sources are not explicitly included, and no RSC is

applied (EPA, 2000a). This is because in these situations, AWQC are derived with respect to the *incremental* lifetime cancer risk posed by the presence of a substance in ambient water, rather than an individual's total risk from all exposure sources. Therefore, the resulting AWQC represents the ambient water concentration that is expected to increase an individual's lifetime risk of cancer from exposure to the pollutant by no more than one chance in one million (10^{-6}) for the general population (male and female adults, 21 years and older; referred to as "general population" herein), regardless of the additional lifetime cancer risk due to exposure, if any, to that substance from other sources. The EPA calculates AWQC at a 10^{-6} cancer risk level for the general population (EPA, 2000a). The 2000 Methodology recommends that states set human health criteria cancer risk levels for the target general population at either 10^{-5} or 10^{-6} and also notes that states and authorized Tribes can choose a more stringent risk level, such as 10^{-7}.

For substances that are carcinogenic, the EPA takes an integrated approach by considering both cancer and noncancer effects when deriving AWQC (EPA, 2000a,b). Where sufficient data are available, the EPA first derives separate AWQC for both carcinogenic and noncarcinogenic toxicity endpoints and then selects the lower (more health protective) of the two values for the recommended AWQC.

PFOS may exist in multiple forms, such as isomers or associated salts and each form may have a separate Chemical Abstracts Service registry number [CASRN] or no CASRN at all. Additionally, these compounds have various names under different classification systems. PFOS is a strong acid that is generally present as the sulfonate anion at typical environmental pH values. Therefore, the conclusions in this document apply to all isomers of PFOS, as well as nonmetal salts of PFOS that would be expected to dissociate in aqueous solutions of pH ranging from 4 to 9. For the purpose of this assessment, "PFOS" will signify the ion, acid or any nonmetal salt of PFOS.

2.1 Uses and Sources of PFOS

PFAS are manufactured chemicals that have been widely used in industrial and consumer processes and products over the past several decades in the United States due to their repellant and surfactant properties. PFAS are persistent chemicals based on their physicochemical properties. Concerns about persistence of PFOS and other PFAS stem from the resistance of these compounds to hydrolysis, photolysis, metabolism, and microbial degradation.

PFOS has been used in a variety of products including surface treatments for soil and stain resistance, coating of paper, specialized applications such as firefighting foams and as a pesticide active ingredient for insect bait traps (NCBI, 2024). Most PFOS production in the United States was voluntarily phased out by its primary manufacturer (3M) between 2000 and 2002. In 2002 and 2007, the EPA took regulatory action under the Toxic Substances Control Act (TSCA) to require that EPA be notified prior to any future domestic manufacture or importation of PFOS and 270 related PFAS (EPA, 2024a).

Point sources of PFOS to the aquatic environment include both industrial facilities and municipal wastewater treatment plants (WWTPs). Additional sources may include surface water runoff from industrial use sites such as metal plating facilities, areas that have received aqueous film-forming foam (AFFF) applications, landfills, and contaminated soils.

2.2 Environmental Fate and Transport in the Environment

PFOS has low volatility in the ionized form, but long-range transport occurs through particulates and through the atmospheric transport of PFOS precursors, which results in PFOS detections in remote locations (Benskin et al., 2012; Butt et al., 2010; Young et al., 2007). PFOS is water soluble and has been found in surface water, groundwater, and drinking water. The sorption of PFOS to sediments and soils varies based on the amount of organic carbon present and many other site-specific conditions; in sediments, the range of log(K_d)[b] values reported in literature is −1.4 to 6.2 (EPA, 2024b; Ahrens, 2011; Beach et al., 2006; Giesy et al., 2010; Higgins and Luthy, 2006). In the water column, and other environmental compartments, the PFOS anion is stable and resistant to hydrolysis, photolysis, volatilization and biodegradation (Beach et al., 2006; OECD, 2002; EPA, 2024a,c). The persistence of PFOS has been attributed to the strong carbon-fluorine (C-F) bond. PFOS is considered highly persistent in the aquatic environment (Ahrens, 2011). Due to the surfactant properties of PFOS, it forms three layers when added to octanol and water in a standard test system used to measure an n-octanol-water partition coefficient (K_{ow}), therefore direct measurement is not possible (Giesy et al., 2010; OECD, 2002). Additionally, PFOS is thought to bind to proteins rather than partitioning into lipids (Giesy et al., 2010; OECD, 2002; EPA, 2024a,c).

PFOS is not expected to volatilize from aqueous solution based on its vapor pressure; Henry's law constant is not measurable (i.e., predicted to be $< 2.0 \times 10^{-6}$) (Beach et al., 2006). The Organization for Economic Co-Operation and Development (OECD) classified PFOS as a type 2, nonvolatile chemical that has a very low or possibly negligible volatility (Beach et al., 2006; Giesy et al., 2010; OECD, 2002). However, a potential source of PFOS is the indirect formation of PFOS through transformation of other PFAS, particularly volatile precursors (Butt et al. 2010; Wang et al. 2015; Young and Mabury, 2010).

2.3 Occurrence and Detection in Sources Relevant to Ambient Water Quality Criteria

PFOS has been detected in a variety of environmental matrices. The occurrence and detection of PFOS in sources relevant to ambient water quality criteria, including ambient water, fish and shellfish, is described below. Additional occurrence information for sources other than ambient water (e.g., air, food, soil) is summarized in Section 6.2 as part of the determination of the RSC.

2.3.1 Occurrence in Surface Water

Among the PFAS with established analytical methods for detection, PFOS is one of the dominant PFAS detected in ambient water both in the United States and worldwide (Ahrens, 2011; Benskin et al., 2012; Dinglasan-Panlilio et al., 2014; Nakayama et al., 2007; Remucal, 2019; Zareitalabad et al., 2013). Though it has a history of wide usage and is highly persistent in

[b] Log(Kd) is the logarithm of the equilibrium dissociation constant.

aquatic environments, current information on the distribution of PFOS in surface waters of the United States is somewhat limited; most published PFOS ambient water occurrence data focus on regions with known PFAS use or occurrence. These regions are primarily freshwater systems in eastern states, including the Mississippi River, Great Lakes, Cape Fear Drainage Basin, and waterbodies near Decatur, Alabama and in northern Georgia (Jarvis et al., 2021). Additional monitoring has been conducted in areas of known AFFF use.

Jarvis et al. (2021) found that concentrations of PFOS in global surface waters ranged over eight orders of magnitude, generally in picogram per liter (pg/L) to nanogram per liter (ng/L) concentrations, with reported maximum concentrations in the microgram per liter (µg/L) range (range: 0.074 ng/L–8,970,000 ng/L, arithmetic mean: 786.77 ng/L, geometric mean: 5.468 ng/L, median: 3.6 ng/L). Though these global calculated concentrations are not necessarily representative of PFOS concentrations in U.S. surface waters, the majority of PFOS concentrations reported across the U.S. (approximately 91%) are less than 300 ng/L.

2.3.2 Occurrence in Freshwater and Estuarine Fish and Shellfish

Studies have shown that PFOS bioaccumulates and biomagnifies with increasing trophic level in a variety of freshwater ecosystems (Kannan et al., 2005; Martin et al., 2004; Penland et al., 2020; Xu et al., 2014) and saltwater ecosystems (de Vos et al., 2008; Houde et al., 2006; Loi et al., 2011; Powley et al., 2008; Tomy et al., 2004) in North America, Europe, and Asia. PFOS is often the most abundant PFAS in aquatic organisms, and this high relative abundance is at least partially explained by the biotransformation of PFOS precursor chemicals into PFOS (Haukås et al., 2007; Kannan et al., 2005; Kelly et al., 2009; Martin et al., 2004; Tomy et al., 2004). Higher trophic level organisms have a greater capacity to metabolize PFOS precursor chemicals, which have been found in lower concentrations in increasing trophic level (Fang et al., 2014; Kannan et al., 2005; Martin et al., 2004).

Global distribution of PFAS, including PFOS, in tissues of aquatic species has been demonstrated in studies conducted in freshwater and marine environments across every continent, including remote regions far from direct sources, such as the high arctic, Antarctica, and oceanic islands (Giesy and Kannan, 2001; Houde et al., 2006).

The EPA collaborates with federal agencies, states, Tribes, and other partners to conduct freshwater fish contamination studies as part of a series of statistically based surveys to produce information on the condition of U.S. lakes, streams, rivers, and coastal waters (EPA, 2023a, 2024d). PFOS was detected in nearly all freshwater fish fillet samples collected during several national studies in rivers and the Great Lakes (Table 1). The 2022 National Lakes Assessment reported 86% of fish fillet samples had detectable PFOS, with a maximum concentration reported of 526 ng/g ww (EPA, 2024d).

Table 1. Summary of the EPA national fish tissue monitoring results for PFOS.

Reference	Most Commonly Sampled Species	Site Description	Results
2008–2009 National Rivers and Streams Assessment (NRSA) (Stahl et al., 2014)	Smallmouth bass Largemouth bass Channel catfish	162 urban river sites across the United States	PFOS was the most commonly detected out of 13 PFAS. PFOS was detected in 73% of fillet samples. Maximum detected concentration: 127 ng/g.
2013–2014 NRSA (EPA, 2020, 2023b)	Channel Catfish Largemouth bass Smallmouth bass	349 urban and nonurban river sites across the United States.	PFOS was the most commonly detected out of 13 PFAS. PFOS was detected in 99% of fillet samples. Maximum detected concentration: 283 ng/g.
2018–2019 NRSA (EPA, 2023a,c)	Channel catfish Smallmouth bass Largemouth bass	290 urban and nonurban river sites across the United States	PFOS was the most commonly detected out of 33 PFAS. PFOS detected in 91% of fillet samples. Maximum detected: concentration: 131 ng/g.
2010 National Coastal Condition Assessment (NCCA) Great Lakes Human Health Fish Tissue Study (Stahl et al., 2014)	Lake trout Smallmouth bass Walleye	157 nearshore sites along the U.S. shoreline of the Great Lakes	PFOS was the most commonly detected PFAS (out of 13 PFAS). PFOS was detected in 100% of fillet samples. Maximum detected concentration: 80 ng/g; median 15 ng/g.
2015 NCCA Great Lakes Human Health Fish Tissue Study (EPA, 2021, 2024c)	Lake Whitefish Yellow Perch Lake Trout Walleye	152 nearshore sites along the U.S. shoreline of the Great Lakes	PFOS was the most commonly detected PFAS (out of 13 PFAS). PFOS was detected in 100% of fillet samples. Maximum detected concentration: 64 ng/g; median 11 ng/g.
2022 National Lakes Assessment (EPA, 2024d)	Largemouth Bass Rainbow Trout Bluegill Yellow Perch Black Crappie	413 sampled lakes within the contiguous U.S. (excluding The Great Lakes, Great Salt Lake and lakes which are tidally influenced).	PFOS was the most commonly detected PFAS (out of 40 PFAS). PFOS was detected in 86% of fillet samples. Maximum detected concentration: 526 ng/g; median 3.17 ng/g.

Guo et al. (2012) measured PFOS in lake trout muscle tissue in Canadian waters of Lake Superior, Huron, Erie, and Ontario. Average PFOS concentrations correlated with increased watershed urbanization, and were 0.85 ng/g, 8.3 ng/g, 27 ng/g, and 46 ng/g wet weight (ww), respectively. Delinsky et al. (2010) measured PFOS in bluegill, black crappie, and pumpkinseed muscle tissue in (59 lakes in Minnesota, including four lakes in the Minneapolis–St. Paul metropolitan area. PFOS was detected in muscle tissues of fish collected in 13 of the 59 lakes, and concentrations ranged from 1.08 ng/g ww to 52.4 ng/g ww in lakes where it was detected. In the four lakes in the Minneapolis–St. Paul metropolitan area, PFOS concentrations in fish muscle tissues ranged from 4.39 ng/g ww to 47.3 ng/g ww.

Penland et al. (2020) measured PFAS concentrations in invertebrates and vertebrates along the Yadkin–Pee Dee River, in North Carolina and South Carolina in 2015. PFOS was measured in whole body tissues of snails (6.47 ng/g ww) but was not detected in whole body tissues of Asian clam, unionid mussels, or crayfish. The highest concentrations in invertebrates were measured in aquatic insect whole body samples (132.8 ng/g ww) which was hypothesized to result from dietary uptake of aquatic biofilms. PFOS was measured in muscle tissue of all 11 sampled fish species and ranged from 11.42 ng/g ww in channel catfish to 37.36 ng/g ww in whitefin shiner. The highest PFOS concentration that Penland et al. (2020) measured was 482.9 ng/g ww, from the eggs of a redhorse fish sample.

Houde et al. (2006) measured whole body PFOS in six fish species in Charleston Harbor, South Carolina, and whole body PFOS in zooplankton and five fish species in Sarasota Bay, Florida. Charleston Harbor was the more developed of the two sites and had higher overall PFOS concentrations. Average PFOS concentrations in Charleston Harbor ranged from 19 ng/g in pinfish to 92 ng/g in spot. In Sarasota Bay, PFOS concentrations averaged 0.2 ng/g in zooplankton, and ranged from 3.1 ng/g in pigfish to 8.8 ng/g in spotted seatrout, suggesting trophic biomagnification.

Ruffle et al. (2020) analyzed freshwater finfish and shellfish from four regions of the United States and seven countries with significant imports to the United States. The highest PFOS concentrations (1.2 ng/g ww to 19.1 ng/g ww) were detected in whitefish, walleye, and yellow perch from the Great Lakes region.

PFOS concentrations in aquatic biota tend to be higher in areas with known PFAS manufacturing, industrial use, and/or application of AFFF, which also tend to be more populated areas and where recreational and subsistence fishing is more common. Several states have developed fish consumption advisories for PFOS (e.g., Alabama, Wisconsin, Minnesota, Michigan) (EPA, 2024e).

3 Criteria Formulas: Analysis Plan

Human health AWQC for toxic pollutants may be necessary to protect designated uses of water bodies related to ingestion of water (i.e., public water supply or source water protection) and ingestion of freshwater/estuarine fish and shellfish. *See* CWA 303(c)(2)(A)–(B). Although the AWQC are based on chronic health effects data (both cancer and noncancer effects), the criteria are intended to also be protective against adverse effects that may reasonably be

expected to occur as a result of elevated acute or short-term exposures (EPA, 2000a). Human health AWQC are expected to provide adequate protection not only for the general population over a lifetime of exposure, but also for sensitive life stages and subpopulations who, because of high water or fish intake rates, or because of biological sensitivities, have an increased risk of receiving a dose that would elicit adverse effect (EPA, 2000a).

The derivation of human health AWQC requires information about both the toxicological endpoints of concern from exposure to water pollutants and human exposure pathways for those pollutants. The EPA considers only the following two primary pathways of human exposure to pollutants present in a particular water body when deriving human health 304(a) AWQC: (1) direct ingestion of drinking water obtained from the water body and (2) consumption of fish and shellfish obtained from the water body.

The equations for deriving human health AWQC are presented as Equations (Eqs.) 1 and 2 for noncancer and nonlinear carcinogenic effects, and Eqs. 3 and 4 for linear carcinogenic effects. The EPA derives two separate recommended human health AWQC based on 1) the consumption of both water and aquatic organisms (Eq. 1), called "water + organism"; and 2) the consumption of freshwater/estuarine fish and shellfish alone (Eq. 2), called "organism only." The use of one criterion over the other depends on the designated use of a particular water body or water bodies (i.e., drinking water source and/or fishable waters). The EPA recommends applying organism only AWQC (Eq. 2) to a water body where the designated use includes supporting fishable uses under section 101(a) of the CWA but the water body is not a drinking water supply source (e.g., nonpotable estuarine waters that support fish or shellfish for human consumption) (EPA, 2000a).

The EPA recommends including the drinking water exposure pathway for ambient surface waters where drinking water is a designated use for the following reasons: (1) drinking water is a designated use for surface waters under the CWA, and therefore, criteria are needed to ensure that this designated use can be protected and maintained; (2) although they are rare, some public water supplies provide drinking water from surface water sources without treatment; (3) even among the majority of water supplies that do treat surface waters, existing treatments might not be effective for reducing levels of particular contaminants; and (4) in consideration of the agency's goals of pollution prevention, ambient waters should not be contaminated to a level where the burden of achieving health objectives is shifted away from those responsible for pollutant discharges and placed on downstream users that must bear the costs of upgraded or supplemental water treatment (EPA, 2000a).

The equations for deriving the criteria values are as follows (EPA, 2000a):

Equations for Noncancer and Nonlinear Carcinogen HHC:
Consumption of water and organisms:

$$AWQC = \frac{RfD \times RSC \times BW \times 1{,}000^{c}}{DWI + \sum_{i=2}^{4}(FCR_i \times BAF_i)} \quad \text{(Eq. 1)}$$

For consumption of organisms only:

$$AWQC = \frac{RfD \times RSC \times BW \times 1{,}000^{c}}{\sum_{i=2}^{4}(FCR_i \times BAF_i)} \quad \text{(Eq. 2)}$$

Where:

AWQC = ambient water quality criteria, expressed in micrograms per liter (µg/L)
RfD = reference dose, expressed in milligrams per kilogram-day (mg/kg-d)
RSC = relative source contribution, unitless
BW = body weight, expressed in kg
DWI = drinking water intake, expressed in L/d
$\sum_{i=2}^{4}$ = summation of values for aquatic trophic levels (TLs), where the letter *i* stands for the TLs to be considered, starting with TL 2 and proceeding to TL 4
FCR_i = fish consumption rate for aquatic TLs (i) 2, 3, and 4, expressed in kg/d
BAF_i = bioaccumulation factor for aquatic TLs (i) 2, 3, and 4, expressed in L/kg

Equations for Linear Carcinogens HHC:
Consumption of water and organisms:

$$AWQC = \frac{RSD \times BW \times 1{,}000^{c}}{DWI + \sum_{i=2}^{4}(FCR_i \times BAF_i)} \quad \text{(Eq. 3)}$$

For consumption of organisms only:

$$AWQC = \frac{RSD \times BW \times 1{,}000^{c}}{\sum_{i=2}^{4}(FCR_i \times BAF_i)} \quad \text{(Eq. 4)}$$

Where:

AWQC = ambient water quality criteria, expressed in micrograms per liter (µg/L)
RSD = RSD = risk specific dose; the cancer risk level (i.e., a target risk for the population; 1 in 1 million or 10^{-6}) divided by the cancer slope factor (i.e., incidence of cancer relative to dose in units of $[mg/kg/day]^{-1}$), expressed in milligrams per kilogram-day (mg/kg-d)
BW = body weight, expressed in kg

[c] 1,000 µg/mg is used to convert the units of mass from milligrams to micrograms.

DWI = drinking water intake, expressed in L/d

$\sum_{i=2}^{4}$ = summation of values for aquatic trophic levels (TLs), where the letter *i* stands for the TLs to be considered, starting with TL 2 and proceeding to TL 4

FCR_i = fish consumption rate for aquatic TLs (i) 2, 3, and 4, expressed in kg/d

BAF_i = bioaccumulation factor for aquatic TLs (i) 2, 3, and 4, expressed in L/kg

The EPA rounds AWQC to the number of significant figures in the least precise parameter as described in the 2000 Methodology (EPA, 2000a, Section 2.7.3). The EPA used a rounding procedure that is consistent with the 2000 Methodology (EPA, 2000a) and the 2015 HHC update (https://www.epa.gov/wqc/human-health-water-quality-criteria-and-methods-toxics).

4 AWQC Input Parameters

4.1 Exposure Factor Inputs

National recommended HHC establish ambient concentrations of pollutants in waters of the United States which, if not exceeded, will protect the general population from adverse health impacts from those pollutants due to consumption of aquatic organisms (i.e., freshwater and estuarine fish and shellfish) and water (EPA, 2000a). It is the EPA's longstanding practice to set national recommended HHC at a level intended to be adequately protective of a human exposure over a lifetime (EPA, 2000a). To accomplish this, the EPA uses a combination of median values, mean values, and percentile estimates for the HHC inputs consistent with the EPA's 2000 Methodology. The EPA's assumptions afford an overall level of protection targeted at the high end of the general adult population (i.e., the target population or the criteria-basis population) (EPA, 2000a). This approach is reasonably conservative and appropriate to meet the goals of the CWA and the 304(a) criteria program (EPA, 2000a). If the EPA determines that another population or life stage (e.g., pregnant women and their fetuses, young children) is the target population, then exposure parameters for that target population or life stage could be considered in the derivation of the criteria (EPA, 2000a). Potentially sensitive life stages for PFOS are explored further in a comparative analysis in Appendix B.

4.1.1 Body Weight

The BW for the general adult population including males and females, ages 21 years and older, was selected for the PFOS HHC, consistent with the population selected in the agency's most recent major update to existing 304(a) HHC (EPA, 2015) and the EPA's 2000 Methodology (EPA, 2000a). The EPA used the mean weight for adults ages 21 and older of 80.0 kg, based on National Health and Nutrition Examination Survey (NHANES) data from 1999 to 2006 as reported in Table 8.1 of the EPA's *Exposure Factors Handbook* (EPA, 2011), the EPA's most recent publication of body weight exposure factors.

4.1.2 Drinking Water Intake Rate

For adults ages 21 and older, the EPA used an updated DWI of 2.3 L/d, rounded from 2.345 L/d. This DWI was estimated using the Food Commodity Intake Database consumption calculator (http://fcid.foodrisk.org[d]) which is based on NHANES 2005–2010 data used to develop the EPA's *Exposure Factors Handbook Update to Chapter 3, Ingestion of Water and Other Select Liquids* (EPA, 2019, Section 3.3.1.1). This rate represents the per capita estimate of combined direct and indirect community water[e] ingestion at the 90th percentile for adults, males and females, ages 21 and older. The EPA selected the per capita rate for the updated DWI because it represents the average daily dose estimates; that is, it includes both people who drank water during the survey period and those who did not, which is appropriate for a national-scale assessment such as the development of CWA section 304(a) national human health criteria development (EPA, 2019, Section 3.2.1). The updated DWI of 2.3 L/d reflects the latest scientific knowledge in accordance with CWA 304(a)(1).

The EPA's selection of the DWI of 2.3 L/d is consistent with the 2000 Methodology's selection of a rate based on per capita community water ingestion at the 86th percentile for adults surveyed in the U.S. Department of Agriculture's *1994–1996 Continuing Survey of Food Intake by Individuals (CSFII)* analysis (EPA, 2000a, Section 4.3.2.1).

4.1.3 Fish Consumption Rate

The FCR used for the general adult population is 22.0 g/d, or 0.0220 kg/d (EPA, 2014b, Table 9a). This FCR represents the 90th percentile per capita consumption rate of fish from inland and nearshore waters for U.S. adults ages 21 years and older based on NHANES data from 2003–2010. The 95% confidence interval (CI) of the 90th percentile per capita FCR is 19.1 g/d and 25.4 g/d, respectively.

As recommended in the 2000 Methodology, the EPA used TL-specific FCRs to better represent human dietary consumption of fish. An organism's trophic position in the aquatic food web can have an important effect on the magnitude of bioaccumulation of certain chemicals. The TL-specific FCRs are numbered 2, 3, and 4, and they account for different categories of fish and shellfish species based on their position in the aquatic food web: TL 2 accounts for benthic filter feeders; TL 3 accounts for forage fish; and TL 4 accounts for predatory fish (EPA, 2000a).

The EPA used the following TL-specific FCRs to derive the AWQC: TL 2 = 7.6 g/d (0.0076 kg/d) (95% CI [6.4, 9.1] g/d); TL 3 = 8.6 g/d (0.0086 kg/d) (95% CI [7.2, 10.2] g/d); and TL 4 = 5.1 g/d (0.0051 kg/d) (95% CI [4.0, 6.4] g/d). Each TL-specific FCR represents the 90th percentile per capita consumption rate of fish and shellfish from inland and nearshore waters from that particular TL for U.S. adults ages 21 years and older (EPA, 2014b, Tables 16a,

[d] Nutrition Examination Survey/What We Eat in America (NHANES/WWEIA) food intake and FCID recipes to estimate food commodity consumption for the purposes of pesticide dietary exposure assessment, as well as consumption estimates for EPA's *Exposure Factors Handbook* (EFH) users (University of Maryland, 2024).

[e] *Community water* includes direct and indirect use of tap water for household uses and excludes bottled water and other sources (EPA, 2019, Section 3.3.1.1). *Direct ingestion* is defined as direct consumption of water as a beverage, while *indirect ingestion* includes water added during food preparation (e.g., cooking, rehydration of beverages) but not water intrinsic to purchased foods (EPA, 2019, Section 3.1).

17a, and 18a). The sum of these three TL-specific FCRs is 21.3 g/d, which is within the 95% CI of the overall FCR of 22.0 g/d. The EPA recommends using the TL-specific FCRs when deriving AWQC; however, the overall FCR (22.0 g/d) may be used if a simplified approach is preferred.

4.2 Bioaccumulation Factor (BAF)

4.2.1 Approach

Several attributes of the bioaccumulation process are important to understand when deriving national BAFs for use in developing national recommended section 304(a) AWQC. First, the term *bioaccumulation* refers to the uptake and retention of a chemical by an aquatic organism from all surrounding media, such as water, food, and sediment. The term *bioconcentration* refers to the uptake and retention of a chemical by an aquatic organism from water only. In some cases, experiments conducted in a lab that measure *bioconcentration* can be used to estimate the degree of *bioaccumulation* expected in natural conditions. However, for many chemicals, particularly those that are highly persistent and hydrophobic, the magnitude of bioaccumulation by aquatic organisms can be substantially greater than the magnitude of bioconcentration. In these cases, an assessment of bioconcentration alone underestimates the extent of accumulation in aquatic biota. Accordingly, the EPA guidelines presented in the 2000 Methodology (EPA, 2000a) emphasize using, when possible, measures of *bioaccumulation* as opposed to measures of *bioconcentration* (EPA, 2000a).

The EPA estimated BAFs for the draft PFOS AWQC using the 2000 Methodology (EPA, 2000a) and the associated *Technical Support Document Volume 2: Development of National Bioaccumulation Factors* (Technical Support Document, Volume 2) (EPA, 2003). Specifically, these documents provide a framework for identifying alternative procedures to derive national TL-specific BAFs for a chemical based on the chemical's properties (e.g., ionization and hydrophobicity), metabolism, and biomagnification potential (EPA, 2000a, 2003). As described in the 2000 Methodology, the purpose of the EPA's national BAF is to represent the long-term, average bioaccumulation potential of a chemical in aquatic organisms that are commonly consumed by humans throughout the United States (EPA, 2000a). The EPA evaluated results from field BAF and laboratory bioconcentration factor (BCF) studies on aquatic organisms commonly consumed by humans in the United States for use in developing national trophic-level BAFs. National BAFs are not intended to reflect fluctuations in bioaccumulation over short periods (e.g., a few days) because human health AWQC are generally designed to protect humans from long-term (lifetime) exposures to waterborne chemicals (EPA, 2003).

The EPA followed the approach described in Figure 3-1 of the Technical Support Document, Volume 2 (EPA, 2003). The EPA used the best available data to classify each chemical according to this framework, and to derive the most appropriate BAFs following the 2000 Methodology (EPA, 2000a) and Technical Support Document, Volume 2 (EPA, 2003). Best available data consisted of 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 framework provides six procedures to calculate a national BAF based on the pollutant's physical and chemical properties (see Figure 1, Procedures 1–6). Each procedure contains a

hierarchy of the BAF derivation methods (listed below); however, this hierarchy should not be considered inflexible (EPA, 2000a). The four methods are:

1. **BAF Method.** This method calculates national TL-specific BAFs using water and fish and shellfish tissue concentration data obtained from field studies. Field-measured BAFs are calculated by dividing the concentration of a contaminant in an organism by the concentration of that contaminant in the surrounding water.

   For nonionic organic chemicals, BAFs are normalized to allow a common basis for averaging BAFs from several studies by adjusting for the water-dissolved portions of the chemical.

   In order to calculate representative TL-specific national BAFs used to calculate national recommended 304(a) criteria, the EPA averaged multiple field BAFs using a geometric mean of the normalized BAFs, first by species and then by TL, to calculate the TL baseline BAFs.

2. **BSAF Method.** This method uses biota-sediment accumulation factors (BSAFs) to estimate bioaccumulation. While BAFs are calculated by dividing the concentration of a contaminant in an organism by the concentration of the contaminant in water, BSAFs divide the concentration in the organism by the concentration in surrounding sediments. BSAFs are useful when calculating site-specific criteria for compounds that are highly hydrophobic—these compounds have the potential to cause bioaccumulation in aquatic organisms even when concentrations in the water column are below detection limits.

3. **BCF Method.** This method estimates BAFs from laboratory-measured BCFs. Experiments designed to calculate BCFs aim to measure bioconcentration resulting from an organism's exposure to contaminated water. Unlike BAFs measured in the field, BCF experiments do not capture bioaccumulation from other routes of exposure or biomagnification (the increase in bioaccumulation at higher levels of the food chain). However, BCFs may be used to estimate bioaccumulation if a contaminant's chemical and physical properties indicate that the compound is likely to primarily accumulate in the organism via the water exposure route, and there is no evidence that the contaminant biomagnifies in the food chain. If insufficient field-collected data are available to calculate a national BAF, then the EPA may also estimate bioaccumulation using laboratory measured BCFs and a food chain multiplier term, which accounts for biomagnification.

   A similar process to the one described in the BAF method description (above) for normalizing of water-dissolved portions of the chemical and particulate organic carbon content is used for calculating national BAFs from laboratory-measured BCF data. Ionic organic chemicals are normalized, then multiplied by the food chain multiplier if biomagnification is likely to occur. All available BCFs are averaged using a geometric mean across species and then across TL to compute baseline BAFs.

4. **K_{ow} Method.** This method predicts BAFs based on a chemical's octanol-water partition coefficient (K_{ow}), with or without adjustment using a food chain multiplier, as described in Section 5.4 of the Technical Support Document, Volume 2 (EPA, 2003).

4.2.2 Data Selection and Evaluation

The EPA conducted a systematic literature search in October 2022 of publicly-available literature sources to determine whether they contained information relevant to calculating national BAFs for human health AWQC, using the 2000 Methodology and Technical Support Document, Volume 2 (EPA, 2000a, 2003). The literature search for reporting the bioaccumulation of PFOS was implemented by developing a series of chemical-based search terms, consistent with the process for derivation of BAFs used in the development of the EPA's Final Aquatic Life Criteria for PFOA (EPA, 2024f) and PFOS (EPA, 2024g) and described in Burkhard (2021). These terms included chemical names and Chemical Abstracts Service Registry Number (CASRN or CAS), synonyms, tradenames, and other relevant chemical forms (i.e., related compounds). Databases searched were Current Contents, ProQuest CSA, Dissertation Abstracts, Science Direct, Agricola, TOXNET, and UNIFY (database internal to the EPA's ECOTOX database). The literature search (including literature published through the first two quarters of 2020) yielded > 37,000 citations that were further refined by excluding citations on analytical methods, human health, terrestrial organisms, bacteria, and where PFOS was not a chemical of study (Burkhard, 2021). The citations meeting the search criteria were reviewed for reported BAFs and/or reported concentrations in which BAFs could be calculated. Data from papers that met the inclusion and data quality screening criteria described below were extracted into the chemical dataset for PFOS.

Specifically, studies were evaluated for inclusion in the dataset used for calculating national BAFs for PFOS using the following evaluation criteria:

- Only BAF studies that included units for tissue, water, and/or BAFs were included.
- Mesocosm, microcosm, and model ecosystem studies were not selected for use in calculating BAFs.
- BAF studies in which concentrations in tissue and/or water were below the minimum level of detection were excluded.
- Only studies performed using freshwater or brackish water were included; high salinity values were excluded.
- Studies of organisms (e.g., damselfly, goby) and tissues (e.g., fish bladder) not commonly consumed by humans or not used as surrogate species for those commonly consumed by humans were excluded. Information on the ecology, physiology, and biology of the organism was used to determine whether an organism is a reasonable surrogate of a commonly consumed organisms.
- Studies in which the BAFs were not found to be at steady state were excluded.
- For pooled samples, averaging BAF data from multiple locations was only considered acceptable if corresponding tissue and water concentrations were available from matching locations (e.g., a BAF would not have been calculated using water and tissue samples collected from eight separate locations with tissue concentrations collected from only six of these corresponding locations). In addition to the evaluation criteria listed above, PFOS bioaccumulation data were also evaluated using five study quality criteria outlined in Burkhard 2021 (Table 2).

Table 2. Bioaccumulation factor (BAF) study quality criteria based on Burkhard (2021).

Criteria	1	2	3
Number of water samples collected	> 3 samples	2–3 samples	1 sample
Number of organism samples collected	> 3 samples	2–3 samples	1 sample
Temporal coordination of water and biota samples	Concurrent collection of samples	Collected within a 1-year time frame	Collected > 1-year time frame
Spatial coordination of water and biota samples	Collected from same locations	Collected from reasonably close locations (1 kilometer (km)–2 km)	Significantly different sampling locations
General experimental design	Assigned a default value of zero for studies in which tissues from individual species were identified and analyzed		Assigned a value of 3 for studies in which tissues were from mixed species or reported as a taxonomic group.

Notes: The scores for each BAF were totaled and used to determine the overall confidence ranking for each individual BAF. The sum of quality values for the five criteria listed in Table 2 were classified as high quality (total score of 4 or 5), medium quality (total score of 5 or 6) or low quality (total score ≥ 7). Only high and medium quality data were included in final national BAFs calculations.

As noted in Burkhard (2021), study quality determinations based on temporal and spatial coordination were subjective and based on best professional judgement. In the absence of adequate quantifiable information regarding sample location (site coordinates for both water and tissue collection locations) or temporal coordination (specific dates of sample collection), BAF data were given a score of 2 or 3 for these categories.

4.2.3 BAFs for PFOS

Following the decision framework presented in Figure 1, the EPA selected one of the four methods to develop a national-level BAF for this chemical. Because PFOS is an organic chemical that predominantly exists in an anionic form in water (EPA, 2024a,e; NCBI, 2024), the BSAF and K_{ow} methods would not be applicable. The EPA selected the BAF estimate using the BAF method (i.e., based on a field-measured BAF) because sufficient field measured BAF data were available for PFOS.

The national-level BAF equation adjusts the TL baseline BAFs for nonionic organic chemicals by national default values for lipid content, as well as dissolved and particulate organic carbon content. The partitioning of PFOS is related to protein binding properties (EPA, 2016a,b); therefore, the EPA did not normalize measured BAF values for PFOS using lipid content when calculating baseline and national BAFs. The EPA selected the recommended 50th percentile dissolved and particulate organic carbon content for the national-level default values which is consistent with the goal of national BAFs (i.e., as central tendency estimates), as described in Section 6.3 of the Technical Support Document, Volume 2 (EPA, 2003). Adjustment for water-dissolved portions of PFOS is applied to TL baseline BAFs (EPA, 2000a) (see Appendix A).

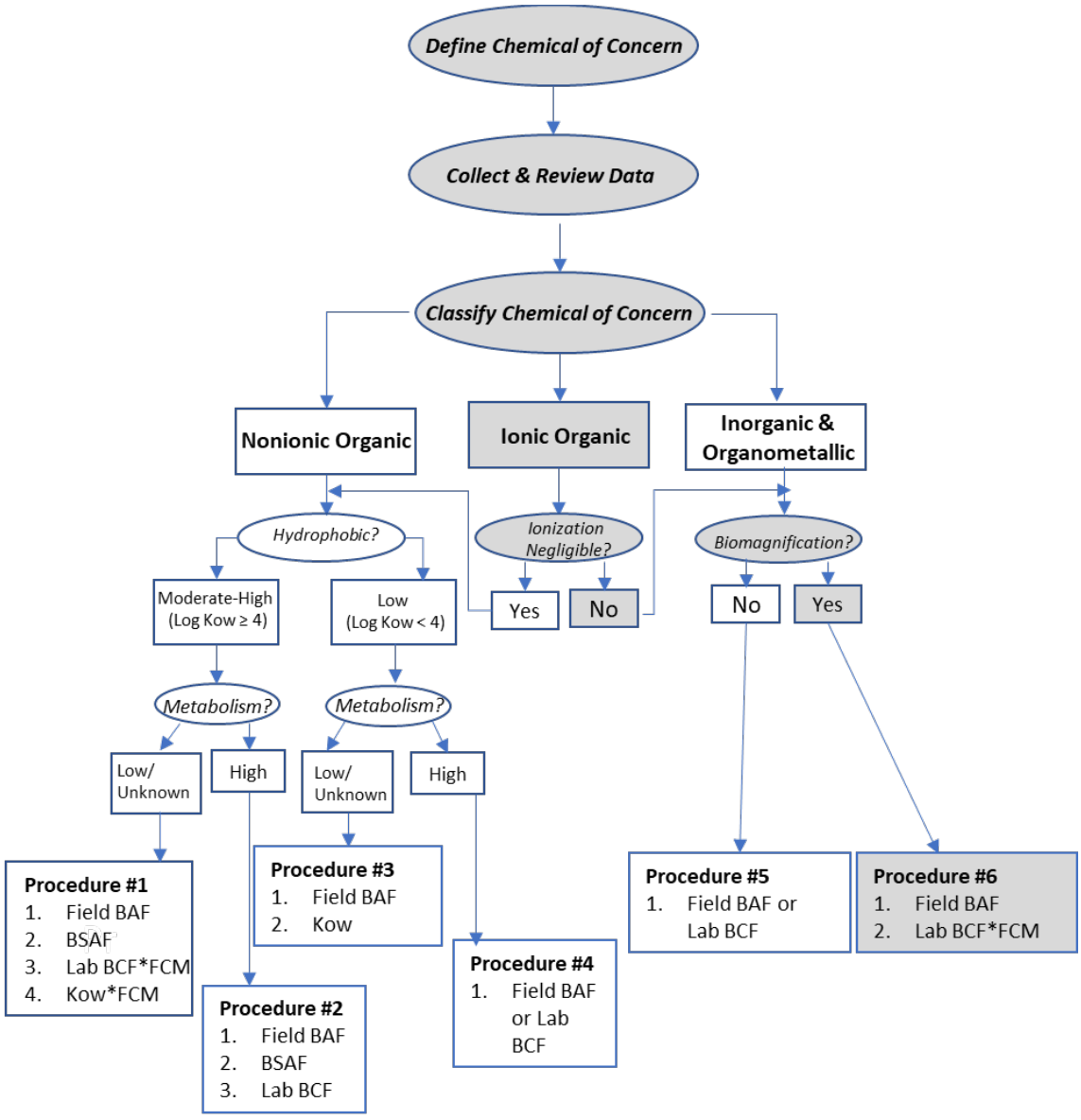


Figure 1. Application of the BAF framework for PFOS; gray boxes indicate steps followed based on available information for PFOS (EPA, 2000a).

The EPA followed the framework described in the Technical Support Document, Volume 2 (EPA, 2003), also presented in Figure 1 to select a procedure for estimating national BAFs for PFOS. Based on the characteristics of this chemical, the EPA selected Procedure 6 for deriving a national BAF value. PFOS has the following characteristics:

- Ionic organic chemicals, with ionization not negligible (EPA, 2024e; NCBI, 2024).
- Biomagnification likely (de Vos et al., 2008; EPA, 2016a; Houde et al., 2006; Kannan et al., 2005; Loi et al., 2011; Martin et al., 2004; Penland et al., 2020; Powley et al., 2008; Tomy et al., 2004; Xu et al., 2014).

The EPA was able to locate peer-reviewed, field-measured BAFs for TLs 2, 3, and 4 from the sources evaluated for which sufficient information was provided to indicate the quality and usability of the data; therefore, the EPA included only field BAF studies. The EPA used the BAF method to derive the national BAF values for PFOS:

- TL 2 = 420 L/kg
- TL 3 = 1,700 L/kg
- TL 4 = 860 L/kg

5 Selection of Toxicity Value

5.1 Approach

The EPA considered all available final toxicity values for both noncarcinogenic and carcinogenic toxicological effects after oral exposure to develop AWQC for PFOS. As described in the 2000 Methodology (EPA, 2000a), where data are available, the EPA derives AWQC for both noncarcinogenic and carcinogenic effects and selects the more protective value for the recommended AWQC (See Section 7, Criteria Derivation: Analysis).

For noncarcinogenic toxicological effects, the EPA uses chronic-duration oral reference values (RfVs; RfDs or equivalent) to derive human health AWQC. An RfV is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure of the human population to a substance that is likely to be without an appreciable risk of deleterious effects during a lifetime (EPA, 2002). An RfV may be derived from an animal toxicological study or a human epidemiological study, from which a point of departure (POD; i.e., a no-observed-adverse-effect level [NOAEL], lowest-observed-adverse-effect level [LOAEL], or benchmark dose [BMD]) can be derived. To derive the RfV, uncertainty factors are applied to the POD to reflect the limitations of the data in accordance with the EPA human health risk assessment methodology (EPA, 2002, 2014a, 2022a).

For carcinogenic toxicological effects, the EPA uses an oral CSF, when applicable and available, to derive human health AWQC. The oral CSF is an upper bound, approximating a 95% confidence limit, on the increased cancer risk from a lifetime oral exposure to a stressor. This value may also be derived from animal toxicological studies or human epidemiological studies.

In developing AWQC, the EPA conducts a systematic search of peer-reviewed, publicly available final toxicity assessments to obtain the toxicity value(s) (RfV and/or CSF) for use in developing AWQC. The EPA identified toxicological assessments by systematically searching the following EPA program offices, other national and international programs, and state programs in April 2024:

- EPA, Office of Research and Development
    - Integrated Risk Information System (IRIS) program (EPA, 2024h)
    - Provisional Peer-Reviewed Toxicity Values (PPRTV) (EPA, 2024i)
    - ORD Human Health Toxicity Values (EPA, 2024j)

- EPA, Office of Pesticide Programs (EPA, 2024k)
- EPA, Office of Pollution Prevention and Toxics (EPA, 2024l)
- EPA, Office of Water (EPA, 2024m)
    - Drinking Water Health Effects Support Documents
    - Toxicity Assessments
- U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR, 2024)
- Health Canada (HC, 2023)
- California Environmental Protection Agency, Office of Environmental Health Hazard Assessment (CalEPA, 2024)

After identifying and documenting all available final toxicity values, the EPA followed a systematic process to consider the identified toxicity values and select the toxicity value(s) to derive the AWQC for noncarcinogenic and carcinogenic effects. The EPA selected IRIS toxicity values to derive the draft AWQC if *any* of the following conditions were met:

1. The EPA's IRIS toxicological assessment was the only available source of a toxicity value.
2. The EPA's IRIS toxicological assessment was the most current source of a toxicity value.
3. The toxicity value from a more current toxicological assessment from a source other than the EPA's IRIS program was based on the same principal study and was numerically the same as an older toxicity value from the EPA IRIS program.
4. A more current toxicological assessment from a source other than the EPA's IRIS program was available, but it did not include the relevant toxicity value (chronic-duration oral RfV or CSF).
5. A more current toxicological assessment from a source other than the EPA's IRIS program was available, but it did not introduce new science (e.g., the toxicity value was not based on a newer principal study) or use a more current modeling approach compared to an older toxicological assessment from the EPA's IRIS program.

The EPA selected the toxicity value from a peer-reviewed, publicly available source other than the EPA IRIS program to derive the draft AWQC if *any* of the following conditions were met:

1. The chemical is currently used as a pesticide, and the EPA Office of Pesticide Programs had a toxicity value that was used in pesticide registration decision-making.
2. A toxicological assessment from a source other than the EPA's IRIS program was the only available source of a toxicity value.
3. A more current toxicological assessment from a source other than the EPA's IRIS program introduced new science (e.g., the toxicity value was based on a newer principal study) or used a more current modeling approach compared to an older toxicological assessment from the EPA's IRIS program.

5.2 Toxicity Value for PFOS

5.2.1 Reference Dose

After following the approach outlined in Section 5.1, the EPA identified the *Final Human Health Toxicity Assessment for Perfluorooctane Sulfonic Acid (PFOS) and Related Salts* (EPA, 2024a). This document is the most recent toxicity assessment identified for PFOS and used the best available science in the evaluation of noncancer risk. The EPA did not identify any other assessments that presented newer scientific information (i.e., unique RfVs) for PFOS.

The EPA's final human health toxicity assessment for PFOS (EPA, 2024a) considered all publicly available human epidemiological, animal toxicological, mechanistic and toxicokinetic evidence relevant to studies that evaluated health effects after oral PFOS exposure. Overall, the available *evidence indicates* that PFOS exposure is likely to cause hepatic, immunological, cardiovascular, and developmental effects in humans, given sufficient exposure conditions (e.g., at levels in humans as low as 0.57 to 5.0 ng/mL and doses in animals as low as 0.0017 to 0.4 mg/kg/day). These judgments are based on data from epidemiological studies of infants, children, adolescents, pregnant individuals, and non-pregnant adults, as well as short-term (28-day), subchronic (90-day), developmental (gestational), and chronic (2-year) oral-exposure studies in rodents.

PODs were developed following EPA's *Benchmark Dose Technical Guidance Document* (EPA, 2012) and converted to external POD human equivalent doses (POD_{HED}s) using pharmacokinetic modeling. Consistent with the recommendations presented in *A* Review *of the Reference Dose and Reference Concentration Processes* (EPA, 2002), the EPA applied uncertainty factors (UFs) to POD_{HED}s to address intraspecies variability, interspecies variability, extrapolation from a lowest observed adverse effect level (LOAEL) to no observed adverse effect level (NOAEL), extrapolation from a subchronic to a chronic exposure duration, and database deficiencies. The EPA derived and considered multiple candidate RfDs from both epidemiological and animal toxicological studies across the four noncancer health outcomes that the EPA determined had the strongest weight of evidence (i.e., immune, cardiovascular, hepatic, and developmental).

Decreased infant birth weight (Wikström et al., 2019) and increased total cholesterol in adults (Dong et al., 2019) were selected as the co-critical effects for the overall oral **RfD of 1×10^{-7} mg/kg/day** (EPA, 2024a). This RfD was derived by applying a total UF of 10 to account for intraspecies variability (UF_H). Critical effects observed during developmental periods (decreased birthweight) represent effects in susceptible subpopulations. The RfD based on these effects is considered protective of effects resulting from lifetime exposures to PFOS, as well as short-term risk assessment scenarios, as the observed developmental endpoints can potentially result from a short-term exposure during critical periods of development.

5.2.2 Cancer Slope Factor

Consistent with EPA's Guidelines for Carcinogen Risk Assessment (EPA, 2005a), the EPA's *Final Human Health Toxicity Assessment for Perfluorooctane Sulfonic Acid (PFOS) and Related Salts* (EPA, 2024a) reviewed the available data and conducted a weight of evidence evaluation across the human epidemiological and animal toxicological studies and concluded that PFOS is *Likely*

to Be Carcinogenic to Humans via the oral route of exposure. Epidemiological studies provided evidence of bladder, prostate, liver, kidney, and breast cancers in humans, although evidence was limited or mixed for some cancer types. Animal toxicological studies supported findings from human studies. Bioassays conducted in Sprague-Dawley rats reported hepatocellular tumors, pancreatic islet cell tumors, and thyroid follicular cell tumors after chronic oral exposure. Some studies observed multisite tumorigenesis (liver and pancreas) in male and female rats. PFOS exposure is associated with multiple key characteristics of carcinogenicity (Smith et al., 2016). Available mechanistic data suggest that multiple modes of action (MOAs) play a role in pancreatic and hepatic tumorigenesis associated with PFOS exposure in animal models (EPA, 2024a).

To derive a CSF for PFOS, the EPA followed agency guidelines and methodologies for risk assessment in deriving CSFs for PFOS (EPA, 2005a, 2012, 2024a). EPA conducted benchmark dose modeling and used a similar pharmacokinetic modeling approach as described for the derivation of noncancer RfDs. Data from human epidemiological studies were not suitable for CSF derivation (see EPA, 2024a for further details). The EPA used multistage models to derive and consider multiple candidate CSFs from an animal toxicological study in multiple sexes, tissue types, and organ systems (i.e., liver and pancreas). Multistage cancer models were used to predict the doses at which the selected BMR for tumor incidence would occur. BMDLs for each tumor type served as the PODs, which were then converted to POD_{HED}s by applying the human clearance value. CSFs were then calculated by dividing the selected BMR by the POD_{HED}s for each tumor type.

The oral slope factor of 39.5 (mg/kg/day)$^{-1}$ for combined hepatocellular adenomas and carcinomas in female rats from Butenhoff et al. (2012) and Thomford (2002) was selected as the basis of the overall CSF for PFOS. Per EPA's *Guidelines for Carcinogen Risk Assessment* and *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* (EPA, 2005a,b), age-dependent adjustment factors were not applied during CSF derivation, as a mutagenic mode of action (MOA) was not found for PFOS in available studies, and insufficient evidence was available to assess susceptibility to cancer following PFOS exposure during early life.

6 Relative Source Contribution) Derivation

6.1 Approach

The EPA applies an RSC to the RfD when calculating an AWQC based on noncancer effects or for carcinogens that are known to act through a nonlinear mode of action to account for the fraction of an individual's total exposure allocated to AWQC-related sources (EPA, 2000a). The purpose of the RSC is to ensure that the level of a chemical allowed by a criterion (e.g., the AWQC), when combined with other identified sources of exposure (e.g., diet excluding freshwater and estuarine fish and shellfish, ambient and indoor air) common to the population of concern, will not result in exposures that exceed the RfD. In other words, the RSC is the portion of total daily exposure equal to the RfD that is attributed to consumption of ambient water (directly or indirectly in beverages like coffee tea or soup, as well as from transfer to dietary items prepared with ambient water) and fish and shellfish from inland and nearshore

waters relative to other exposure sources; the remainder of the exposure equal to the RfD is allocated to other potential exposure sources. The EPA considers any potentially significant exposure source and route when deriving the RSC.

The RSC is derived by applying the Exposure Decision Tree approach published in the EPA's 2000 Methodology (EPA, 2000a). The Exposure Decision Tree approach allows flexibility in the RfD apportionment among sources of exposure and considers several characteristics of the contaminant of interest, including the adequacy of available exposure data, levels of the contaminant in relevant sources or media of exposure, and regulatory agendas (i.e., whether there are multiple health-based criteria or regulatory standards for the contaminant). The RSC is developed to reflect the exposure to the U.S. general population or a sensitive population within the U.S. general population, depending on the available data.

An RSC determination first requires "data for the chemical in question… representative of each source/medium of exposure and… relevant to the identified population(s)" (EPA, 2000a). The term "data" in this context is defined as ambient sampling measurements in the media of exposure, not internal human biomonitoring metrics. More specifically, the data must adequately characterize exposure distributions including the central tendency and high-end exposure levels for each source and 95% confidence intervals for these terms (EPA, 2000a). The EPA's approach recommends a "ceiling" RSC of 80% and a "floor" RSC of 20% to account for uncertainties including unknown sources of exposure, changes to exposure characteristics over time, and data inadequacies.

The EPA's Exposure Decision Tree approach states that when there are insufficient environmental monitoring and/or exposure intake data to permit quantitative derivation of the RSC, the recommended RSC is 20%. In the case of AWQC development, this means that 20% of the exposure equal to the RfD is allocated to the consumption of ambient water and fish and shellfish from inland and nearshore waters and the remaining 80% is reserved for other potential sources, such as diet (excluding fish and shellfish from inland and nearshore waters), air, consumer products, etc. This 20% RSC can be replaced if sufficient data are available to develop a scientifically defensible alternative value. If scientific data demonstrating that sources and routes of exposure other than drinking water are not anticipated for a specific pollutant, the RSC can be raised as high as 80% based on the available data, allowing the remaining 20% for other potential sources (EPA, 2000a). Applying a lower RSC (e.g., 20%) is a more health protective approach to public health and results in a lower AWQC.

To derive an RSC for PFOS, the EPA evaluated the exposure information identified through conducting prior systematic literature searches performed as part of the EPA's final human health toxicity assessment for PFOS (EPA, 2024e). To identify information on PFOS exposure routes and sources to inform RSC determination, the EPA considered primary literature published between 2003–2020 that was collected by the EPA's Office of Research and Development as part of an effort to evaluate evidence for pathways of human exposure to eight PFAS, including PFOS. This search was not date-limited and spanned information collected across the Web of Science, PubMed, and ToxNet/ToxLine (now ProQuest) databases. An

updated literature search was conducted and captured relevant literature published through March 2021. Literature captured by this search is housed in the EPA's HERO database (https://hero.epa.gov/). To supplement the primary literature database, the EPA also searched the following gray literature sources in February 2022 for information related to relative exposure of PFOS for all potentially relevant routes of exposure (oral, inhalation, dermal) and exposure pathways relevant to humans. The full description of methods used to identify and screen relevant literature is available in the EPA's *Final Appendix: Human Health Toxicity Assessment for Perfluorooctane Sulfonic Acid (PFOS) and Related Salts* (EPA, 2024e). The following description in Section 6.2 is a summary of the information provided in the Appendix of the final PFOS toxicity assessment.

6.2 Summary of Potential Exposure Sources of PFOS Other Than Water and Freshwater and Estuarine Fish/Shellfish

6.2.1 Dietary Sources

A number of studies support food ingestion as a major source of exposure to PFOS for the general population based on early studies that modeled the relative contributions of various sources among the general populations of North America and Europe (Fromme et al., 2009; Trudel et al., 2008; Vestergren and Cousins, 2009). The exposure to adults in western countries is typically estimated to be about 1 ng/kg-d (Domingo and Nadal, 2017; East et al., 2021). The dominance of the food ingestion pathway is attributed to bioaccumulation in food from environmental emissions, relatively large amounts of foods being consumed, and high GI uptake (Trudel et al., 2008). However, the estimates are highly uncertain due to limited data availability, relatively low detection frequencies, and relatively large differences in composition of diets across geographic locations (Domingo and Nadal, 2017; EFSA, 2020).

There is currently no comprehensive, nationwide Total Diet Study (TDS) for PFOS that can be used to draw conclusions about the occurrence and potential risk of PFOS in the U.S. food supply for the general population. In 2021, the FDA released PFAS testing results from their first survey of nationally distributed processed foods, including several baby foods, collected for the TDS (FDA, 2021a). Results of the survey showed that 164 of the 167 foods tested had no detectable levels of PFAS measured. Three food samples (fish sticks, canned tuna, and protein powder) had detectable levels of PFAS, including PFOS (FDA, 2021a). In another recent FDA study, PFOS was detected in one sample (baked cod: 98 ppt) out of 94 food samples collected nationally (FDA, 2021b). In a 2019 national survey of produce, meats, dairy and grain products, PFOS was detected in three of the 179 food samples tested (two samples of tilapia, one sample of ground turkey) (FDA, 2020a,b). PFOS was also detected in produce samples (collard greens and lettuce) in a 2018 focused study near a PFAS production plant in the Fayetteville, North Carolina area (FDA, 2018). PFOS was below the lower limit of quantification (LOQ; 4 ng/L) in all 30 samples analyzed in a study of domestic and imported carbonated water and noncarbonated bottled water (FDA, 2016). The sample size in all of these studies is limited, and thus, the results cannot be used to draw definitive conclusions about the levels of PFAS in the U.S. food supply more generally (FDA, 2023). In a 2010 study of 31 types of food collected from 5 grocery stores in Texas, PFOS was not detected in any of the samples (Schecter et al., 2010).

As a component of a scientific evaluation on the risks to human health related to PFAS in food, the European Food Safety Authority (EFSA) conducted an exposure assessment using consumption data from the EFSA Comprehensive Food Consumption Database and 69,433 analytical results for 26 PFAS in 1,528 samples of food and beverages obtained from 16 European countries (EFSA, 2020). Samples were collected between the years 2000 and 2016 (74% after 2008), mainly from Norway, Germany, and France. With 92% of the analytical results below the limit of detection (LOD) or LOQ, lower bound dietary exposure estimates were obtained by assigning zero to values below LOD/LOQ. Median chronic dietary exposures of PFOS for children and adults were estimated as 1.02 ng/kg-d and 0.58 ng/kg-d, respectively. The most important contributors for PFOS were "fish and other seafood[f]," "eggs and egg products," and "meat and meat products." It is unclear whether the contribution from food contact material is reflected in the data. The authors determined diet to be the major source of PFAS exposure for most of the population but noted that dust ingestion and indoor air inhalation may provide substantial contributions for some individuals.

The 2020 EFSA report highlighted a recent study of aggregate exposure to PFAS from diet, house dust, indoor air, and dermal contact among Norwegian adults (Poothong et al., 2020). Dietary exposures were estimated for 61 study participants using food diaries and data on concentrations from an extensive Norwegian database of concentrations in 68 different food and drinks (including drinking water). For PFOS, dietary intake was by far the greatest contributor to aggregate exposure (contributing 95% of total estimated PFOS intake), but intake from ingestion of house dust represented the dominant pathway for some of the top 20% most highly exposed individuals. The authors reported a significant positive correlation between the observed and modeled serum concentrations for PFOS ($r = 0.29$, $p < 0.05$). The correlation existed despite the model underestimating serum concentrations of PFOS by a factor of 4, which was attributed to the long half-life and decreased exposure over recent years. While the authors did not separately quantify intake from food and drinking water, an earlier article from the same research group (Papadopoulou et al., 2017) reported measured concentrations in duplicate diets with median estimated intake of PFOS approximately 150 times higher from solid food than from liquids.

De Felip et al. (2015) investigated correlations of blood concentrations of PFOS with dietary intake among Italian women. They estimated daily intake of PFOS based on the reported food consumption frequencies of specific food items and found strongly significant correlations of blood levels with consumption of beef, pork, and vegetables ($p < 0.01$), and moderate correlation with consumption of fish ($p < 0.05$).

Zafeiraki et al. (2019) analyzed samples of marine species of fish caught in Dutch waters or purchased at Dutch markets between 2012 and 2018. Of the 16 PFAS that were analyzed, PFOS was generally detected at a higher frequency and concentration across the tested species. Shrimp and seabass had the highest average concentrations of PFOS (each over 4 ng/g ww), but

[f] Some dietary studies use the term "seafood" to indicate fish and shellfish from ocean, freshwater, or estuarine water bodies. Information about the water bodies assessed in individual studies is reported in the articles.

was also detected in cod, mackerel, sole and common dab (100% detection, ranging from 1.10 to 2.5 ng/g ww).

In marine fish and shellfish samples collected for the FDA 2021–2022 seafood survey, Young et al. (2022), analyzed concentrations of 20 PFAS, including PFOS, in eight of the most highly consumed seafood products in the United States. PFOS was detected most frequently (100% of samples; n = 10) and at the highest average concentrations (422.9 ppt) in clams. The study also reported detections in crab (45.5% of samples; n = 11; 151.6 ppt average concentration in samples with detections), tuna (50% of samples; n = 10; 86.8 ppt average concentration in samples with detections), tilapia (20% of samples; n = 10; 57.5 ppt average concentration in samples with detections), and cod (60% of samples; n = 10; 62.5 ppt average concentration in samples with detections). In this study, PFOS was not detected above the method detection limits (MDLs) (39 ppt or 45 ppt) in salmon, shrimp, or pollock.

Based on the National Oceanic and Atmospheric Administration's (NOAA's) National Status and Trends Data Portal, PFOS concentrations (in ww) were not detected in mussels, oysters, and fish liver samples. However, PFOS was detected in marine fish fillet samples, up to 75.1 ppb (NOAA, 2024).

6.2.2 Food Contact Materials

The FDA has authorized the use of PFAS in food contact substances due to their non-stick and grease, oil, and water-resistant properties since the 1960s. There are four categories of products that may contain PFAS (FDA, 2020a,b):

- Nonstick cookware: PFAS may be used as a coating to make cookware nonstick.
- Gaskets, O-Rings, and other parts used in food processing equipment: PFAS may be used as a resin in forming certain parts used in food processing equipment that require chemical and physical durability.
- Processing aids: PFAS may be used as processing aids for manufacturing other food contact polymers to reduce build-up on manufacturing equipment.
- Paper/paperboard food packaging: PFAS may be used as grease-proofing agents in fast-food wrappers, microwave popcorn bags, take-out paperboard containers, and pet food bags to prevent oil and grease from foods from leaking through the packaging.

Paper products used for food packaging are often treated with PFAS for water and grease resistance. In previous testing, sandwich wrappers, french fry boxes, and bakery bags were all been found to contain PFAS (Schreder and Dickman, 2018). Older generation PFAS (e.g., PFOA, PFOS) were manufactured and used in products for decades, and the bulk of the information available on PFAS toxicity relates to the older compounds. However, because newer-generation PFAS are more mobile than their predecessors, they migrate more readily into food. In 2016, the FDA deauthorized the remaining uses of long-chain "C8" PFAS in food packaging, which are therefore, no longer used in food contact applications sold in the United States (FDA, 2020a,b). An analysis of popcorn bags, snack bags, and sandwich bags purchased in 2018 from international vendors and grocery stores in the United States found no evidence of PFOS at

concentrations above the LOD (0.63 ng/g paper) (Monge Brenes et al., 2019). The authors presented these results as evidence of a reduction in PFOS concentrations in microwave packaging between 2005 and 2018. In an analysis of microwave popcorn bags from around the world, Zabaleta et al. (2017) reported no measurable concentrations of any perfluorosulfonic acid (PFSA), including PFOS, in any of the samples. In a second study, Zabaleta et al. (2020) looked at PFAS in 25 paper- and paperboard packaging materials primarily collected in Spain. Again, no PFSAs, including PFOS, were found above the level of detection. The packaging materials with the largest number of detectable analytes was a popcorn bag from China and the inside paper lining from three individual pet food products, which contained a spectrum of C3 to C10 perfluorinated carboxylates. Similarly, in an analysis of 52 European products collected between 2014 and 2016, Borg and Ivarrson (2017) reported that PFSAs were rarely detected in the samples; PFOS was the only PFSA detected and was only present in one sample, a microwave popcorn bag. Kotthoff et al. (2015) reported PFOS was the most frequently and abundantly detected PFAS in paper-based cooking materials.

6.2.3 Consumer Product Uses

An early investigation of consumer exposure to PFOS by Trudel et al. (2008) used mechanistic modeling together with information on product-use habits to estimate exposures from mill-treated carpets and impregnated clothing. The authors concluded that contact with consumer products represents less than 1% of total exposure to PFOS, but also pointed out that because carpets have a relatively long lifetime, the exposure is expected to continue long after cessation of use of PFOS in carpet treatments. Liu et al. (2014) also investigated trends in PFAS content of household goods between 2007 and 2011. They reported a decrease in the availability of consumer products that contain PFOS but were still able to find products that contained PFOS.

In contrast, Kotthoff et al. (2015) reported broad detection of PFOS in a 2010 sampling effort that collected 115 European consumer products, which included carpets, leather, outdoor materials, and others. PFOS was detected in all but two sample types, often at the highest median concentration compared to other PFSAs. PFOS has also been detected in textile samples of outdoor apparel from Europe and Asia (Gremmel et al., 2016; van der Veen et al., 2020). PFOS was detected in one-third of the jackets tested by Gremmel et al. (2016) at relatively low concentrations ranging from 0.01 $\mu g/m^2$–0.59 $\mu g/m^2$.

6.2.4 Indoor Dust

Several studies suggest that PFOS and its precursors in indoor dust may be an important exposure source for some individuals (Shoeib et al., 2011; Gebbink et al., 2015; Gleason et al., 2017; Poothong et al., 2020). PFOS is generally a dominant ionic PFAS constituent in household dust, frequently occurring above detection limits and at relatively high concentrations in all or most samples (Shoeib et al., 2011; Kim et al., 2019; Wu et al., 2015; Poothong et al., 2020; Makey et al., 2017; Byrne et al., 2017; Fraser et al., 2013).

PFOS was measured at the second highest concentrations (geometric mean concentrations ranging from 29.0 ng/g to 34.6 ng/g) and frequencies (ranging from 85% to 87% detected) in dust sampled from Californian households. Similarly, PFOS was found at the highest levels

(mean concentration of 3.06 ng/g) of 15 PFAS measured in dust samples taken from households in Seoul, Republic of Korea (Kim et al., 2019). One study of Alaska Natives noted that PFOS was the predominant compound in dust samples (Byrne et al., 2017).

6.2.5 Ambient Air

Air concentrations of PFOS in the atmosphere vary widely across the globe. Areas near wastewater treatment facilities, waste incinerators, and landfills can be sources of PFOS to air (Ahrens et al., 2011). In an urban area in Albany, New York, perfluorinated acids were measured in air samples in both the gas and particulate phase in May and July 2006 (Kim and Kannan, 2007). PFOS in the gas phase had a mean concentration of 1.70 pg/m^3 (range: 0.94 pg/m^3–3.0 pg/m^3) and in the particulate phase had a mean concentration of 0.64 g/m^3 (range: 0.35 pg/m^3–1.16 pg/m^3). However, at Lake Ontario, concentrations of PFOS in the particulate phase measured in air samples over the lake were higher (Boulanger et al., 2005). The mean concentration of PFOS at Lake Ontario was 6.4 ± 3.3 pg/m^3. In an urban area in Minneapolis, Minnesota, PFOS was measured in both the particulate and gas phase (MPCA, 2008). PFOS in the particulate phase ranged from 2.1 pg/m^3 to 7.9 pg/m^3 and the gas phase ranged from 1.8 pg/m^3 to 5.0 pg/m^3 across the five samples.

The EPA's Toxics Release Inventory reported release data for PFOS in 2022 (EPA, 2024n). PFOS is not listed as a hazardous air pollutant under the Clean Air Act (EPA, 2024o). In Canada, PFOS air concentrations measured in 2009 showed widespread distribution with remote sites having similar concentrations to urban sites (ECCC, 2018). Using passive samplers, PFOS concentrations were detected in Toronto, Ontario (8 pg/m^3), an agricultural site in Saskatchewan (5 pg/m^3), Whistler, British Columbia (4 pg/m^3), and Alert, N Nunavut (2 pg/m^3) (ECCC, 2018).

Other reported concentrations of PFOS in air samples from Sydney, Florida (3.4 pg/m^3), Tudor Hill, Bermuda (6.1 pg/m^3), Malin Head, Ireland (3.3 pg/m^3), and Hilo, Hawaii (6.6 pg/m^3) are similar to the concentrations reported in Canada (ECCC, 2018) and Japan (Sasaki et al., 2003). The annual geometric mean concentration of PFOS in air samples collected monthly from 2001 to 2002 in the town of Oyamazaki and Fukuchiyama City were 5.3 pg/m^3 and 0.6 pg/m^3, respectively (Sasaki et al., 2003).

Across Europe, PFOS air concentrations were reported to be variable. In the particulate phase, PFOS concentrations ranged from < 1.8 pg/m^3 to 46 pg/m^3 (Martin et al., 2004). Most locations had low concentrations (~1 pg/m^3–2 pg/m^3) to less than the reported MDL and included Hazelrigg, United Kingdom, Kjeller, Norway, and Mace Head, Ireland (Barber et al., 2007). The highest concentrations were reported in Manchester, United Kingdom. Similarly, high concentrations (150 pg/m^3) were reported in Paris, France (ECCC, 2018).

Even in the Arctic, PFOS, its precursors, and degradation products, have been detected in air samples in Resolute Bay, Nunavut, Canada, during the summer of 2004 (Stock et al., 2007). PFOS in the filter samples were 1–2 orders of magnitude greater than other compounds, with a mean concentration of 5.9 pg/m^3. These concentrations are greater than PFOS concentrations measured in the particle phase of air samples measured in Zeppelinstasjonen, Svalbard, Norway (Butt et al., 2010). PFOS was measured in September and December 2006 and August and

December 2007, with mean concentrations of 0.11 pg/m^3 (range: 0.03 pg/m^3–0.50 pg/m^3) and 0.18 pg/m^3 (range: 0.02 pg/m^3–0.97 pg/m^3), respectively.

6.2.6 Summary and Recommended RSC for PFOS

As mentioned above, the scope of exposure sources considered for the draft recommended human health AWQC is limited to surface water used for drinking water and the consumption of freshwater/estuarine fish and shellfish (EPA, 2000a), consistent with previous human health AWQC (EPA, 2015). The EPA followed the Exposure Decision Tree approach to determine the RSC for PFOS (EPA, 2000a; see Figure 2).

To identify the population(s) of concern (Box 1, Figure 2), the EPA first identified potential subpopulations or lifestages based on the PFOS exposure intervals in the two co-critical studies from which the co-critical effects were selected for RfD derivation in the PFOS toxicity assessment (EPA, 2024a). Since the critical effects are the most sensitive adverse health effect that were identified from the available data of sufficient quality, then the exposure interval may be a sensitive window of exposure. Two co-critical effects were identified for PFOS (decreased infant birth weight and increased total cholesterol in adults) based on two human epidemiological studies; however, the specific critical window of exposure for each of the critical effects is not known. Based on epidemiological study design, the potentially sensitive life stages include women of childbearing age who may be or become pregnant, and pregnant women and their developing fetuses. However, limited information was available regarding specific PFOS exposure in these two potentially sensitive life stages from different environmental sources. Therefore, the EPA considered exposures in the general U.S. population, ages 21 years and older, which includes these two life stages.

Second, the EPA identified PFOS relevant exposure sources/pathways (Box 2, Figure 2), including dietary consumption, incidental oral, inhalation, or dermal exposure via dust, consumer products, and soil, and inhalation exposure via ambient air. Several of these may be potentially significant exposure sources.

Third, the EPA evaluated whether adequate data were available to describe the central tendencies and high-end exposures for all potentially significant exposure sources and pathways (Box 3, Figure 2). The EPA determined that there were inadequate quantitative data to describe the central tendencies and high-end estimates for all relevant exposure pathways. For example, studies from the U.S. indicate that dust may be a significant source of exposure to PFOS. Although several studies report PFOS detections in consumer products, most examined samples from specific locations that may not be nationally representative. Therefore, the agency does not have adequate quantitative data to describe the central tendency and high-end estimate of exposure for this potentially significant source in the U.S. population.

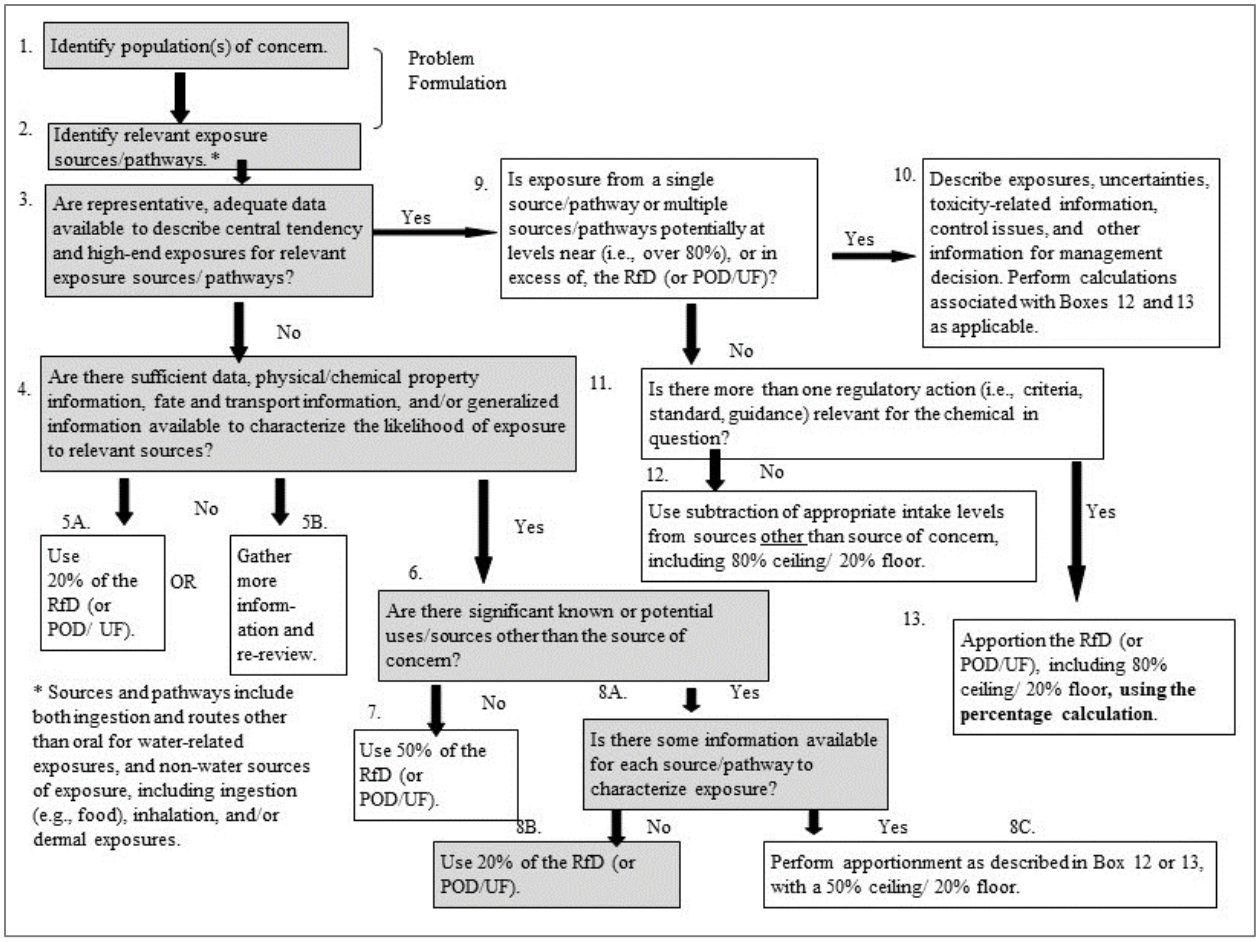


Figure 2. RSC exposure decision tree framework for PFOS; figure adapted from EPA (2000a) with gray boxes indicating key decision points for this chemical.

Fourth, the EPA determined whether there were sufficient data on the physical/chemical properties, fate and transport and generalized information characterizing the likelihood of exposure to PFOS from relevant sources (Box 4, Figure 2). Sufficient information for PFOS was available to characterize the likelihood of exposure. The agency relied on the studies summarized above to determine if there are potential uses/source of PFOS other than AWQC-related sources (Box 6, Figure 2). There are significant known or potential uses/sources of PFOS other than AWQC-related sources (Box 6, Figure 2). For example, diet from sources other than freshwater/estuarine fish and shellfish, such as marine fish, are potentially significant sources of PFOS. Based on this information, the next step was to determine if adequate information was available on PFOS to characterize each source/pathway of exposure (Box 8a, Figure 2). The EPA determined there is not enough information available on each source to make a quantitative characterization of exposure among exposure sources, such as dust, air and consumer products. Therefore, the data are insufficient to allow for quantitative characterization of the different exposure sources. The EPA's Exposure Decision Tree approach states that when there is insufficient environmental and/or exposure data to permit quantitative derivation of the RSC, the recommended RSC for the general population is 20% (U.S. EPA, 2000a). Thus, the

EPA recommend an RSC of 20% (0.20) for PFOS for both the water plus organism AWQC as well as the organism only AWQC (Box 8b, Figure 2).

7 Criteria Derivation: Analysis

Table 3 summarizes the input parameters used to derive the draft recommended human health AWQC that are protective of exposure to PFOS from consuming drinking water and/or eating fish and shellfish (organisms) from inland and nearshore waters. The criteria calculations are presented below. These criteria recommendations are based on the 2000 Methodology (EPA, 2000a) and the toxicity and exposure assumptions described above (see Section 4, AWQC Input Parameters; Section 5, Selection of Toxicity Value; and Section 6, Relative Source Contribution Derivation).

Table 3. Input parameters for the human health AWQC for PFOS.

Input Parameter		Value
RfD		1×10^{-7} mg/kg/day
CSF		$39.5\ (mg/kg/day)^{-1}$
RSC		0.20
BW		80.0 kg
DWI		2.3 L/d
FCR	TL 2	0.0076 kg/d
	TL 3	0.0086 kg/d
	TL 4	0.0051 kg/d
BAF	TL 2	420 L/kg
	TL 3	1,700 L/kg
	TL 4	860 L/kg

Notes: RfD = reference dose; CSF = cancer slope factor; RSC = relative source contribution; BW = bodyweight; DWI = drinking water intake; FCR = fish consumption rate; TL = trophic level; BAF = bioaccumulation factor.

7.1 AWQC for Noncarcinogenic Toxicological Effects

For consumption of water and organisms:

$$\text{AWQC } (\mu g/L) = \frac{\text{RfD (mg/kg-d)} \times \text{RSC} \times \text{BW (kg)} \times 1{,}000\ (\mu g/mg)}{\text{DWI (L/d)} + \sum_{i=2}^{4} (FCR_i\ (kg/d) \times BAF_i\ (L/kg))}$$

$$= \frac{0.0000001\ mg/kg\text{-}d \times 0.20 \times 80.0\ kg \times 1{,}000\ \mu g/mg}{2.3\ L/d + ((0.0076\ kg/d \times 420\ L/kg) + (0.0086\ kg/d \times 1{,}700\ L/kg) + (0.0051\ kg/d \times 860\ L/kg))}$$

$$= 0.00006531\ \mu g/L$$

$$= 0.00006\ \mu g/L \text{ (rounded)}$$

For consumption of organisms only:

$$\text{AWQC } (\mu g/L) = \frac{\text{RfD (mg/kg-d)} \times \text{RSC} \times \text{BW (kg)} \times 1{,}000\ (\mu g/mg)}{\sum_{i=2}^{4} (FCR_i\ (kg/d) \times BAF_i\ (L/kg))}$$

$$= \frac{0.0000001\ mg/kg\text{-}d \times 0.20 \times 80.0\ kg \times 1{,}000\ \mu g/mg}{(0.0076\ kg/d \times 420\ L/kg) + (0.0086\ kg/d \times 1{,}700\ L/kg) + (0.0051\ kg/d \times 860\ L/kg)}$$

$$= 0.00007208\ \mu g/L$$

$$= 0.00007\ \mu g/L \text{ (rounded)}$$

7.2 AWQC for Carcinogenic Toxicological Effects

The EPA derives cancer-based HHC for contaminants that have been determined to be *Carcinogenic to Humans* or *Likely to Be Carcinogenic to Humans* (EPA, 2000a,c). Since PFOS was determined to be *Likely to Be Carcinogenic to Humans* (EPA, 2024a,e), the EPA derived AWQC for carcinogenic toxicological effects.

Consumption of water and organisms:

$$\text{AWQC} = \frac{\text{RSD} \times \text{BW} \times 1{,}000^{g}}{\text{DWI} + \sum_{i=2}^{4} (FCR_i \times BAF_i)}$$

$$= \frac{(10^{-6} / 39.5)\ mg/kg\text{-}d \times 80.0\ kg \times 1{,}000\ \mu g/mg}{2.3\ L/d + ((0.0076\ kg/d \times 420\ L/kg) + (0.0086\ kg/d \times 1{,}700\ L/kg) + (0.0051\ kg/d \times 860\ L/kg))}$$

$$= 0.00008267\ \mu g/L$$

$$= 0.000083\ \mu g/L \text{ (rounded)}$$

For consumption of organisms only:

$$\text{AWQC} = \frac{\text{RSD} \times \text{BW} \times 1{,}000^{g}}{\sum_{i=2}^{4} (FCR_i \times BAF_i)}$$

$$= \frac{(10^{-6} / 39.5)\ mg/kg\text{-}d \times 80.0\ kg \times 1{,}000\ \mu g/mg}{(0.0076\ kg/d \times 420\ L/kg) + (0.0086\ kg/d \times 1{,}700\ L/kg) + (0.0051\ kg/d \times 860\ L/kg)}$$

$$= 0.00009124\ \mu g/L$$

$$= 0.000091\ \mu g/L \text{ (rounded)}$$

[g] 1,000 µg/mg is used to convert the units of mass from milligrams to micrograms.

7.3 AWQC Summary for PFOS

The EPA derived the draft recommended AWQC for PFOS using both noncarcinogenic and carcinogenic toxicity endpoints. The human health AWQC for noncarcinogenic effects for PFOS are **0.00006 µg/L** (0.06 ng/L) for consumption of water and organisms and **0.00007 µg/L** (0.07 ng/L) for consumption of organisms only (Table 4). The human health AWQC for carcinogenic effects (at a 10^{-6} cancer risk level) for PFOS are **0.000083 µg/L** (0.083 ng/L) for consumption of water and organisms and **0.000091 µg/L** (0.091 ng/L) for consumption of organisms only (Table 4). EPA recommends the lower AWQC, based on the noncarcinogenic effects of PFOS, as the human health AWQC. The EPA evaluated the use of exposure factors relevant to sensitive subpopulations based on the critical effect(s) used to derive the RfD (Appendix B). Based on the results of this evaluation, the criteria based on exposure factors for the general adult (≥ 21 years of age) population are the most health protective.

Under the EPA's recently finalized Method 1633 (EPA, 2024p) for aqueous samples, the level of quantification (LOQ) representing the observed LOQs in the multi-laboratory validation study, range from 1 to 4 ng/L for PFOS. The pooled MDL for PFOS is 0.63 ng/L. The pooled MDL value is derived from the multi-laboratory validation study using MDL data from eight laboratories and represents the sensitivity that should be achievable in a well-prepared laboratory but may not represent the actual MDL used for data reporting or data quality assessments (EPA, 2024p). The MDLs and ranges presented here provide a reference for comparison of analytical concentrations and recommended criteria.

Table 4. Summary of the EPA's recommended human health AWQC for PFOS chemicals.

	Human Health AWQC for Noncarcinogenic Effects
Water and Organism	0.00006 µg/L (0.06 ng/L)
Organism Only	0.00007 µg/L (0.07 ng/L)

8 Consideration of Noncancer Health Risks from PFAS Mixtures

The EPA recently released its final *Framework for Estimating Noncancer Health Risks Associated with Mixtures of Per- and Polyfluoroalkyl Substances (PFAS)* (referred to here as the PFAS mixtures framework; EPA, 2024q). The PFAS mixtures framework describes three flexible, data-driven approaches that facilitate practical component-based mixtures evaluation of two or more PFAS based on dose additivity, consistent with the EPA*'s Guidelines for the Health Risk Assessment of Chemical Mixtures* (EPA, 1986) and *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures* (EPA, 2000d). The approaches described in the EPA PFAS mixtures framework may support interested federal, state, and Tribal partners, as well as public health experts and other stakeholders to assess the potential noncancer human health hazards and risks associated with PFAS mixtures. The EPA is providing an illustration of one approach which could be applied to PFAS mixture HHC derivation. The PFAS mixtures framework underwent peer review by the EPA Science Advisory Board (EPA, 2022b) and public review and the EPA responded to comments (EPA, 2024r). The public comment period ended on May 30, 2023. The public docket can be accessed at www.regulations.gov under Docket ID: EPA-HQ-OW-2022-0114.

Dose additivity means that the combined effect of the component chemicals in a mixture is equal to the sum of the individual doses or concentrations scaled for potency. As noted in the PFAS mixtures framework, exposure to a number of individual PFAS has been shown to elicit the same or similar profiles of adverse effects in various organs and systems. Many toxicological studies of PFAS as well as other classes of chemicals support the health-protective conclusion that chemicals that elicit the same or similar observed adverse effects following individual exposure should be assumed to act in a dose-additive manner when in a mixture unless data demonstrate otherwise (EPA, 2024q). Importantly, few studies have examined the toxicity of PFAS mixtures, particularly with component chemical membership and proportions that are representative of the diverse PFAS mixtures that occur in the environment. Mixtures assessments for chemicals that share similar adverse health effects, and therefore assume dose additivity, typically apply component-based assessment approaches.

The Hazard Index (HI) approach is one of the component-based mixtures assessment approaches described in the PFAS mixtures framework. In order to support states and Tribes interested in addressing potential noncancer risks of PFAS mixtures, the application of the HI approach for deriving HHC for mixtures is described below. States and authorized Tribes may choose to adopt this approach to derive HHC for PFAS mixtures. Use of the HI approach to assess risks associated with PFAS mixtures was supported by the EPA Science Advisory Board (EPA, 2022b).

In the HI approach (see PFAS mixtures framework; EPA, 2024q), a hazard quotient (HQ) is calculated as the ratio of human exposure (E) to a human health-based toxicity value (e.g., reference value [RfV]) for each mixture component chemical (i) (EPA, 1986). The HQs for the component chemicals are then summed to derive a mixture-specific HI (for the specified exposure route/medium). Since the HI is unitless, the E and the RfV inputs to the HI formula must be expressed in the same dose units (e.g., mg/L) (Eq. 5). For example, in the context of the human health criteria, HQs for each individual PFAS are calculated by dividing the measured ambient water concentration of each component PFAS (e.g., expressed as µg/L) by its corresponding human health criterion (e.g., expressed as µg/L), and the resulting component PFAS HQs are summed to yield the PFAS mixture HI (Eqs. 5-7). Either water-plus-organism or organism-only HHC can be used in the PFAS mixtures HI approach; however, the type of HHC selected for HI calculation should be consistent. Because cancer data are lacking for most PFAS, the HI approach is currently recommended for PFAS HHC based on noncancer effects.

A hypothetical example is included below to illustrate using the HI approach to derive an HHC for a mixture of three PFAS. A PFAS mixture HI exceeding 1 indicates that co-occurrence of two or more PFAS in a mixture in ambient water exceeds the health-protective level(s), indicating potential health risks. Some individual PFAS have HHC below the analytical MDLs (e.g., PFOA, PFOS). If one such PFAS is included as a component PFAS in the HI approach, then any detectable level of that component PFAS in surface water will result in a component HQ greater than 1, and thus, an HI greater than 1 for the PFAS mixture.

$$HI = \sum_{i=1}^{n} HQ_i = \sum_{i=1}^{n} \frac{E_i}{HHC_i} \quad \text{(Eq. 5)}$$

$$HI = HQ_{PFAS_X} + HQ_{PFAS_Y} \quad \text{(Eq. 6)}$$

$$HI = \left(\frac{[PFAS_{X,ambient\ water}]}{[PFAS_{X,HHC}]}\right) + \left(\frac{[PFAS_{Y,ambient\ water}]}{[PFAS_{Y,HHC}]}\right) \quad \text{(Eq. 7)}$$

Where:

HI = hazard index
n = the number of component (i) PFAS
HQ_i = hazard quotient for component (i) PFAS
E_i = human exposure for component (i) PFAS
HHC_i = human health criterion for component PFAS (i)
HQ_{PFAS} = hazard quotient for a given individual PFAS
$PFAS_X$ = Hypothetical PFAS
$PFAS_Y$ = Hypothetical PFAS
$[PFAS_{ambient\ water}]$ = concentration of a given PFAS in ambient water
$[PFAS_{HHC}]$ = water-plus-organism HHC or organism-only HHC for a given PFAS

9 Chemical Name and Synonyms

- Perfluorooctane Sulfonic Acid (PFOS) (CASRN 1763-23-1)
- 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-1-octanesulfonic acid
- perfluorooctane sulfonic acid
- heptadecafluoro-1-octane sulfonic acid
- PFOS acid

10 References

Ahrens, L. 2011. Polyfluoroalkyl compounds in the aquatic environment: A review of their occurrence and fate. *Journal of Environmental Monitoring* 13:20–31. https://doi.org/10.1039/C0EM00373E.

Ahrens, L., M. Shoeib, T. Harner, S.C. Lee, R. Guo, and E.J. Reiner. 2011. Wastewater treatment plant and landfills as sources of polyfluoroalkyl compounds to the atmosphere. *Environmental Science & Technology* 45:8098–8105.

ATSDR (Agency for Toxic Substances and Disease Registry). 2024. *Toxicological Profiles*. U.S. Department of Health and Human Services, ATSDR, Atlanta, GA. Accessed January 2024. https://www.atsdr.cdc.gov/toxicological-profiles/about/index.html.

Barber, J.L., U. Berger, C. Chaemfa, S. Huber, A. Jahnke, C. Temme, and K.C. Jones. 2007. Analysis of per- and polyfluorinated alkyl substances in air samples from Northwest Europe. *Journal of Environmental Monitoring* 9(6):530–541. https://doi.org/10.1039/B701417A.

Beach, S.A., J.L. Newsted, K. Coady and J.P. Giesy. 2006. Ecotoxicological evaluation of perfluorooctanesulfonate (PFOS). *Reviews of Environmental Contamination and Toxicology* 186:133–174.

Benskin, J.P., D.C.G. Muir, B.F. Scott, C. Spencer, A.O. De Silva, H. Kylin, J.W. Martin, A. Morris, R. Lohmann, G. Tomy, B. Rosenberg, S. Taniyasu, and N. Yamashita. 2012. Perfluoroalkyl acids in the Atlantic and Canadian Arctic Oceans. *Environmental Science & Technology* 46:5815–5823. https://doi.org/10.1021/es300578x.

Borg, D., and J. Ivarsson. 2017. *Analysis of PFASs and TOF in Products*. TemaNord 2017:543. Nordic Council of Ministers. Accessed February 2024. https://norden.diva-portal.org/smash/get/diva2:1118439/FULLTEXT01.pdf.

Boulanger, B., A.M. Peck, J.L. Schnoor, and K.C. Hornbuckle. 2005. Mass budget of perfluorooctane surfactants in Lake Ontario. *Environmental Science & Technology* 39(1):74–79. https://doi.org/10.1021/es049044o.

Burkhard, L.P. 2021. Evaluation of published bioconcentration factor (BCF) and bioaccumulation factor (BAF) data for per- and polyfluoroalkyl substances across aquatic species. *Environmental Toxicology and Chemistry* 40(6):1530–1543. https://setac.onlinelibrary.wiley.com/doi/10.1002/etc.5010.

Butenhoff, J.L., S.C. Chang, G.W. Olsen, and P.J. Thomford. 2012. Chronic dietary toxicity and carcinogenicity study with potassium perfluorooctanesulfonate in Sprague Dawley Rats. *Toxicology* 293:1–15.

Butt, C.M., U. Berger, R. Bossi, and G.T. Tomy. 2010. Levels and trends of poly- and perfluorinated compounds in the arctic environment. *Science of the Total Environment* 408:2936–2965. https://doi.org/10.1016/j.scitotenv.2010.03.015.

Byrne, S., S. Seguinot-Medina, P. Miller, V. Waghiyi, F.A. von Hippel, C.L. Buck, and D.O. Carpenter. 2017. Exposure to polybrominated diphenyl ethers and perfluoroalkyl substances in a remote population of Alaska Natives. *Environmental Pollution* 231:387–395. http://dx.doi.org/10.1016/j.envpol.2017.08.020.

CalEPA (California Environmental Protection Agency). 2024. *Public Health Goals (PHGs)*. CalEPA, Office of Environmental Health Hazard Assessment. Accessed January 2024. https://oehha.ca.gov/water/public-health-goals-phgs.

De Felip, E., A. Abballe, F.L. Albano, T. Battista, V. Carraro, M. Conversano, S. Franchini, L. Giambanco, N. Iacovella, A.M. Ingelido, A. Maiorana, F. Maneschi, V. Marra, A. Mercurio, R. Nale, B. Nucci, V. Panella, F. Pirola, M.G. Porpora, E. Procopio, N. Suma, S. Valentini, L. Valsenti, and V. Vecchiè. 2015. Current exposure of Italian women of reproductive age to PFOS and PFOA: A human biomonitoring study. *Chemosphere* 137:1–8. https://doi.org/10.1016/j.chemosphere.2015.03.046.

Delinsky, A.D., M.J. Strynar, P.J. McCann, J.L. Varns, L. McMillan, S.F. Nakayama, and A.B. Lindstrom. 2010. Geographical distribution of perfluorinated compounds in fish from Minnesota lakes and Rivers. *Environmental Science & Technology* 44(7):2549–2554. https://doi.org/10.1021/es903777s.

de Vos, M.G., M.A.J. Huijbregts, M.J. van den Heuvel-Greve, A.D. Vethaak, K.I. Van de Vijver, P.E.G. Leonards, S.P.J. van Leeuwen, P. de Voogt, and A.J. Hendriks. 2008. Accumulation of perfluorooctane sulfonate (PFOS) in the food chain of the Western Scheldt estuary: Comparing field measurements with kinetic modeling. *Chemosphere* 70(10):1766–1773. https://doi.org/10.1016/j.chemosphere.2007.08.038.

Dinglasan-Panlilio, M.J., S.S. Prakash, and J.E. Baker. 2014. Perfluorinated compounds in the surface waters of Puget Sound, Washington and Clayoquot and Barkley Sounds, British Columbia. *Marine Pollution Bulletin* 78:173–180. https://doi.org/10.1016/j.marpolbul.2013.10.046.

Domingo, J.L., and M. Nadal. 2017. Per- and polyfluoroalkyl substances (PFASs) in food and human dietary intake: A review of the recent scientific literature. *Journal of Agricultural and Food Chemistry* 65(3):533–543. https://doi.org/10.1021/acs.jafc.6b04683.

Dong, Z; Wang, H; Yu, YY; Li, YB; Naidu, R; Liu, Y. (2019). Using 2003–2014 U.S. NHANES data to determine the associations between per- and polyfluoroalkyl substances and cholesterol: Trend and implications. *Ecotoxicology and Environmental Safety* 173:461–468. https://hero.epa.gov/hero/index.cfm/reference/details/reference_id/5080195.

East, A., P.P. Egeghy, E.A. Cohen Hubal, R. Slover, and D.A. Vallero. 2021. Computational estimates of daily aggregate exposure to PFOA/PFOS from 2011 to 2017 using a basic intake model. *Journal of Exposure Science & Environmental Epidemiology* 33:56–68. https://doi.org/10.1038/s41370-021-00374-w.

ECCC (Environment and Climate Change Canada). 2018. *Canadian Environmental Protection Act, 1999 Federal Environmental Quality Guidelines Perfluorooctane Sulfonate (PFOS).* ECCC, Canada. Accessed February 2024. https://www.canada.ca/en/environment-climate-change/services/evaluating-existing-substances/federal-environmental-quality-guidelines-perfluorooctane-sulfonate.html.

EFSA (European Food Safety Authority Panel on Contaminants in the Food Chain). 2020. Risk to human health related to the presence of perfluoroalkyl substances in food. *EFSA Journal* 18(9):ej06223. https://doi.org/10.2903/j.efsa.2020.6223.

EPA (Environmental Protection Agency). 1986. *Guidelines for the Health Risk Assessment of Chemical Mixtures*. EPA/630/R-98/002. EPA, Risk Assessment Forum, Washington, DC. https://www.epa.gov/risk/guidelines-health-risk-assessment-chemical-mixtures.

EPA (Environmental Protection Agency). 2000a. *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000)*. EPA-822-B-00-004. EPA, Office of Water, Office of Science and Technology, Washington, DC. Accessed January 2024. https://www.epa.gov/sites/default/files/2018-10/documents/methodology-wqc-protection-hh-2000.pdf.

EPA (Environmental Protection Agency). 2000b. *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000), Technical Support Document. Vol. 1: Risk Assessment*. EPA-822-B-00-005. EPA, Office of Water, Office of Science and Technology, Washington, DC. Accessed January 2024. https://www.epa.gov/sites/default/files/2018-12/documents/methodology-wqc-protection-hh-2000-volume1.pdf.

EPA (Environmental Protection Agency). EPA. 2000c. Revisions to the Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000); Notice. *Federal Register*, Nov. 3, 2000, 65:66444. https://www.govinfo.gov/content/pkg/FR-2000-11-03/pdf/00-27924.pdf.

EPA (Environmental Protection Agency). 2000d. *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures*. EPA/630/R-00/002. EPA, Risk Assessment Forum, Washington, DC. https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=20533.

EPA (Environmental Protection Agency). 2002. *A Review of the Reference Dose and Reference Concentration Processes*. EPA/630/P-02/002F. EPA, Risk Assessment Forum, Washington, DC. Accessed March 2024. https://www.epa.gov/sites/default/files/2014-12/documents/rfd-final.pdf.

EPA (Environmental Protection Agency). 2003. *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000), Technical Support Document Volume 2: Development of National Bioaccumulation Factors*. EPA-822-R-03-030. EPA, Office of Water, Office of Science and Technology, Washington, DC. Accessed January 2024. https://www.epa.gov/sites/default/files/2018-10/documents/methodology-wqc-protection-hh-2000-volume2.pdf.

EPA (Environmental Protection Agency). 2005a. *Guidelines for Carcinogen Risk Assessment*. EPA-630-P-03-001F. EPA, Risk Assessment Forum, Washington, DC. Accessed January 2024. https://www.epa.gov/sites/default/files/2013-09/documents/cancer_guidelines_final_3-25-05.pdf.

EPA (Environmental Protection Agency). 2005b. *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens*. EPA/630/R-03/003F. EPA, Risk Assessment Forum, Washington, DC. Accessed January 2024. https://www.epa.gov/sites/default/files/2013-09/documents/childrens_supplement_final.pdf.

EPA (Environmental Protection Agency). 2011. Body Weight Studies. Chapter 8 in *Exposure Factors Handbook*. EPA/600/R-09/052F. EPA, National Center for Environmental Assessment, Office of Research and Development, Washington, DC. Accessed January 2024. https://www.epa.gov/sites/default/files/2015-09/documents/efh-chapter08.pdf.

EPA (Environmental Protection Agency). 2012. *Benchmark dose technical guidance*. EPA/100/R-12/001. EPA, Risk Assessment Forum, Washington, DC. Accessed March 2024. https://hero.epa.gov/hero/index.cfm/reference/details/reference_id/1239433.

EPA (Environmental Protection Agency). 2014a. *Framework for Human Health Risk Assessment to Inform Decision Making.* EPA/100/R-14/001. EPA, Office of the Science Advisor, Risk Assessment Forum. Accessed January 2024. https://www.epa.gov/sites/default/files/2014-12/documents/hhra-framework-final-2014.pdf.

EPA (Environmental Protection Agency). 2014b. *Estimated Fish Consumption Rates for the U.S. Population and Selected Subpopulations (NHANES 2003–2010)*. EPA-820-R-14-002. EPA. Accessed January 2024. https://www.epa.gov/sites/default/files/2015-01/documents/fish-consumption-rates-2014.pdf.

EPA (Environmental Protection Agency). 2015. *Human Health Ambient Water Quality Criteria: 2015 Update*. EPA 820-F-15-001. EPA, Office of Water, Washington, DC. https://www.epa.gov/sites/default/files/2015-10/documents/human-health-2015-update-factsheet.pdf.

EPA (Environmental Protection Agency). 2016a. *Drinking Water Health Advisory for Perfluorooctane Sulfonate (PFOS)*. EPA-822-R-16-004. EPA, Office of Water, Washington, DC. Accessed January 2024. https://www.epa.gov/sites/default/files/2016-05/documents/pfos_health_advisory_final-plain.pdf.

EPA (Environmental Protection Agency). 2016b. *Health Effects Support Document for Perfluorooctane Sulfonate (PFOS)*. EPA 822-R-16-002. EPA, Office of Water, Health and Ecological Criteria Division, Washington, DC. Accessed February 2024. https://www.epa.gov/sites/default/files/2016-05/documents/pfos_hesd_final_508.pdf.

EPA (Environmental Protection Agency). 2018. *Basic Information on PFAS*. https://19january2021snapshot.epa.gov/pfas/basic-information-pfas_.html.

EPA (Environmental Protection Agency). 2019. *Update for Chapter 3 of the Exposure Factors Handbook, Ingestion of Water and Other Select Liquids*. EPA/600/R-18/259F. EPA, National Center for Environmental Assessment, Office of Research and Development Washington, DC. Accessed January 2024. https://www.epa.gov/sites/default/files/2019-02/documents/efh_-_chapter_3_update.pdf.

EPA (Environmental Protection Agency). 2020. *National Rivers and Streams Assessment 2013–2014 Technical Support Document.* EPA 843-R-19-001. EPA, Office of Water and Office of Research and Development, Washington, DC. Accessed January 2024. https://www.epa.gov/sites/default/files/2020-12/documents/nrsa_2013-14_final_tsd_12-15-2020.pdf.

EPA (Environmental Protection Agency). 2021. *National Coastal Condition Assessment 2015 Technical Support Document*. EPA-841-R-20-002. EPA, Office of Water, Office of Wetlands Oceans and Watersheds and EPA Office of Research and Development, Washington, DC. Accessed February 2024. https://www.epa.gov/system/files/documents/2022-07/NCCA%202015%20TSD%20FINAL.20210901.pdf.

EPA (Environmental Protection Agency). 2022a. *ORD Staff Handbook for Developing IRIS Assessments*. EPA 600-R22-268. EPA, Office of Research and Development, Center for Public Health and Environmental Assessment, Washington, DC. https://cfpub.epa.gov/ncea/iris_drafts/recordisplay.cfm?deid=356370.

EPA (Environmental Protection Agency). 2022b. Transmittal of the Science Advisory Board Report titled, "Review of EPA's Analyses to Support EPA's National Primary Drinking Water Rulemaking for PFAS." EPA-22-008. https://sab.epa.gov/ords/sab/r/sab_apex/sab/advisoryreports.

EPA (Environmental Protection Agency). 2023a. *2018–2019 National Rivers and Streams Assessment Fish Tissue Study*. EPA, Office of Water, Washington, DC. Accessed February 2024. https://www.epa.gov/choose-fish-and-shellfish-wisely/2018-2019-national-rivers-and-streams-assessment-fish-tissue-study#fish.

EPA (Environmental Protection Agency). 2023b. *2013–2014 National Rivers and Streams Assessment Fish Tissue Study*. EPA, Office of Water, Washington, DC. Accessed February 2024. https://www.epa.gov/choose-fish-and-shellfish-wisely/2013-2014-national-rivers-and-streams-assessment-fish-tissue-study#fish.

EPA (Environmental Protection Agency). 2023c. *National Rivers and Streams Assessment 2018–2019 Technical Support Document*. EPA 841-R-22-005. EPA, Office of Water, Office of Wetlands, Oceans and Watersheds, and EPA, Office of Research and Development, Washington, DC. Accessed February 2024. https://www.epa.gov/system/files/documents/2023-11/nrsa-2018-19-tsd-final-11072023.pdf.

EPA (Environmental Protection Agency). 2024a. *Final: Human Health Toxicity Assessment for Perfluorooctane Sulfonic Acid (PFOS) and Related Salts*. EPA 815-R-24-007. EPA, Office of Water, Washington, DC. https://www.epa.gov/system/files/documents/2024-04/main_final-toxicity-assessment-for-pfos_2024-04-09-refs-formatted_508c.pdf.

EPA (Environmental Protection Agency). 2024b. *Draft Sewage Sludge Risk Assessment for Perfluorooctanoic Acid (PFOA) CASRN 335-67-1 and Perfluorooctane Sulfonic Acid (PFOS) CASRN 1763-23-1*. EPA-820-P-24-001.

EPA (Environmental Protection Agency). 2024c. *2015 Great Lakes Human Health Fish Fillet Tissue Study*. EPA, Office of Water, Washington, DC. Accessed February 2024. https://www.epa.gov/choose-fish-and-shellfish-wisely/2015-great-lakes-human-health-fish-fillet-tissue-study#fish.

EPA (Environmental Protection Agency). 2024d. *National Lakes Assessment 2022. Technical Support Document*. EPA 841-R-24-006. EPA, Office of Wetlands, Oceans and Watersheds, Office of Research and Development, Washington, DC.

EPA (Environmental Protection Agency). 2024e. *Final: Appendix: Human Health Toxicity Assessment for Perfluorooctane Sulfonic Acid (PFOS) and Related Salts*. EPA 8125-R-24-009. EPA, Office of Water, Washington, DC. https://www.epa.gov/system/files/documents/2024-04/appendix_final-toxicity-assessment-for-pfos_2024-04-09-refs-formatted.pdf.

EPA (Environmental Protection Agency). 2024f. Final Freshwater Aquatic Life Ambient Water Quality Criteria and Acute Saltwater Aquatic Life Benchmark for Perfluorooctanoic Acid (PFOA). EPA-842-R-24-002. EPA, Office of Water, Office of Science and Technology, Washington, DC. https://www.epa.gov/system/files/documents/2024-09/pfoa-report-2024.pdf.

EPA (Environmental Protection Agency). 2024g. Final Freshwater Aquatic Life Ambient Water Quality Criteria and Acute Saltwater Aquatic Life Benchmark for Perfluorooctane Sulfonate (PFOS). EPA-842-R-24-003. EPA, Office of Water, Office of Science and Technology, Washington, DC. https://www.epa.gov/system/files/documents/2024-09/pfos-report-2024.pdf.

EPA (Environmental Protection Agency). 2024h. *Integrated Risk Information System*. EPA, Office of Research and Development, Washington, DC. Accessed January 2024. https://www.epa.gov/iris.

EPA (Environmental Protection Agency). 2024i. *Provisional Peer-Reviewed Toxicity Values (PPRTVs)*. EPA, Office of Research and Development, Center for Public Health and Environmental Assessment, Washington, DC. Accessed January 2024. https://www.epa.gov/pprtv.

EPA (Environmental Protection Agency). 2024j. *Risk Assessment*. EPA, Office of Research and Development, Washington, DC. Accessed January 2024. https://www.epa.gov/risk.

EPA (Environmental Protection Agency). 2024k. *Pesticide Chemical Search*. EPA, Office of Pesticide Programs, Washington, DC. Accessed January 2024. https://ordspub.epa.gov/ords/pesticides/f?p=chemicalsearch:1.

EPA (Environmental Protection Agency). 2024l. *TSCA Chemical Substance Inventory*. EPA, Office of Pollution Prevention and Toxics, Washington, DC. Accessed January 2024. https://www.epa.gov/tsca-inventory.

EPA (Environmental Protection Agency). 2024m. *Water Topics*. EPA, Office of Water, Washington, DC. Accessed April 2024. https://www.epa.gov/environmental-topics/water-topics.

EPA (Environmental Protection Agency). 2024n. *Toxics Release Inventory (TRI) Explorer—Release Reports: Release Chemical Report Page*. 2022 Updated dataset (released October 2023). EPA, Washington, DC. Accessed March 2024. https://enviro.epa.gov/triexplorer/tri_release.chemical.

EPA (Environmental Protection Agency). 2024o. *Initial List of Hazardous Air Pollutants with Modifications*. EPA, Air Toxics Assessment Group, Research Triangle Park, NC. Accessed January 2022. https://www.epa.gov/haps/initial-list-hazardous-air-pollutants-modifications#mods.

EPA (Environmental Protection Agency). 2024p. *Method 1633. Analysis of Per- and Polyfluoroalkyl Substances (PFAS) in Aqueous, Solid, Biosolids, and Tissue Samples by LC-MS/MS*. EPA 821-R-24-001. EPA, Office of Water, Washington, DC. https://www.epa.gov/cwa-methods/cwa-analytical-methods-and-polyfluorinated-alkyl-substances-pfas.

EPA (Environmental Protection Agency). 2024q. *Final Framework for Estimating Noncancer Health Risks Associated with Mixtures of Per-and Polyfluoroalkyl Substances (PFAS)*. EPA-815-R-24-003. EPA, Office of Water, Washington, DC. https://www.epa.gov/system/files/documents/2024-04/final-pfas-mix-framework-3.25.24_final-508.pdf.

EPA (Environmental Protection Agency). 2024r. Responses to Public Comments on Per- and Polyfluoroalkyl Substances (PFAS) National Primary Drinking Water Regulation Rulemaking. EPA-815R24005. EPA, Office of Water, Washington, DC. https://www.epa.gov/system/files/documents/2024-04/pfas-comment-response-document_final-508_v2.pdf.

Fang, S., X. Chen, S. Zhao, Y. Zhang, W. Jiang, L. Yang, and L. Zhu. 2014. Trophic magnification and isomer fractionation of perfluoroalkyl substances in the food web of Taihu Lake, China. *Environmental Science & Technology* 48(4):2173–2182. https://doi.org/10.1021/es405018b.

FDA (Food and Drug Administration). 2016. *Analytical Results for PFAS in 2016 Carbonated Water and Non-Carbonated Bottled Water Sampling (Parts Per Trillion)*. U.S. Department of Health and Human Services, FDA, Silver Spring, MD. Accessed January 2024. https://www.fda.gov/media/127848/download.

FDA (Food and Drug Administration). 2018. *Analytical Results for PFAS in 2018 Produce Sampling (Parts Per Trillion)*. U.S. Department of Health and Human Services, FDA, Silver Spring, MD. Accessed January 2024. https://www.fda.gov/media/127848/download.

FDA (Food and Drug Administration). 2020a. *Analytical Results for PFAS in 2019 Total Diet Study Sampling (Parts Per Trillion)—Dataset 1*. U.S. Department of Health and Human Services, FDA, Silver Spring, MD. Accessed January 2024. https://www.fda.gov/media/127852/download.

FDA (Food and Drug Administration). 2020b. *Analytical Results for PFAS in 2019 Total Diet Study Sampling (Parts Per Trillion)—Dataset 2*. U.S. Department of Health and Human Services, FDA, Silver Spring, MD. Accessed January 2024. https://www.fda.gov/media/133693/download.

FDA (Food and Drug Administration). 2021a. *Analytical Results for PFAS in 2021 Total Diet Study Sampling (Parts Per Trillion)—Dataset 4*. U.S. Department of Health and Human Services, FDA, Silver Spring, MD. Accessed January 2024. https://www.fda.gov/media/151574/download.

FDA (Food and Drug Administration). 2021b. *Analytical Results for PFAS in 2021 Total Diet Study Sampling (Parts Per Trillion)—Dataset 3*. U.S. Department of Health and Human Services, FDA, Silver Spring, MD. Accessed January 2024. https://www.fda.gov/media/150338/download.

FDA (Food and Drug Administration). 2023. *Analytical Results of Testing Food for PFAS from Environmental Contamination*. FDA, Silver Spring, MD. Accessed January 2024. https://www.fda.gov/food/chemical-contaminants-food/analytical-results-testing-food-pfas-environmental-contamination.

Fraser, A.J., T.F. Webster, D.J. Watkins, M.J. Strynar, K. Kato, A.M. Calafat, V.M. Vieira, and M.D. McClean. 2013. Polyfluorinated compounds in dust from homes, offices, and vehicles as predictors of concentrations in office workers' serum. *Environment International* 60:128–136. https://doi.org/10.1016/j.envint.2013.08.012.

Fromme, H., S.A. Tittlemier, W. Völkel, M. Wilhelm, and D. Twardella. 2009. Perfluorinated compounds-exposure assessment for the general population in Western countries. *International Journal of Hygiene and Environmental Health* 212(3):239–270. https://doi.org/10.1016/j.ijheh.2008.04.007.

Gebbink, W.A., U. Berger, and I.T. Cousins. 2015. Estimating human exposure to PFOS isomers and PFCA homologues: The relative importance of direct and indirect (precursor) exposure. *Environment International* 74:160–169. https://doi.org/10.1016/j.envint.2014.10.013.

Giesy, J.P., and K. Kannan. 2001. Global distribution of perfluorooctane sulfonate in wildlife. *Environmental Science & Technology* 35(7):1339–1342. https://doi.org/10.1021/es001834k.

Giesy, J. P., J. E. Naile, J. S. Khim, P. D. Jones and J. L. Newsted. 2010. Aquatic toxicology of perfluorinated chemicals. *Reviews of Environmental Contamination and Toxicology* 202:1–52.

Gleason, J.A., K.R. Cooper, J.B. Klotz, G.B. Post, and G. Van Orden. 2017. Appendix A in *Health-based Maximum Contaminant Level Support Document: Perfluorooctanoic Acid (PFOA)*. New Jersey Drinking Water Quality Institute Health Effects Subcommittee, NJ. Accessed February 2024. https://www.nj.gov/dep/watersupply/pdf/pfoa-appendixa.pdf.

Gremmel, C., T. Frömel, and T.P. Knepper. 2016. Systematic determination of perfluoroalkyl and polyfluoroalkyl substances (PFASs) in outdoor jackets. *Chemosphere* 160:173–180. http://dx.doi.org/10.1016/j.chemosphere.2016.06.043.

Guo, R., E.J. Reiner, S.P. Bhavsar, P.A. Helm, S.A. Mabury, E. Braekevelt, and S.A. Tittlemier. 2012. Determination of polyfluoroalkyl phosphoric acid diesters, perfluoroalkyl phosphonic acids, perfluoroalkyl phosphinic acids, perfluoroalkyl carboxylic acids, and perfluoroalkane sulfonic acids in lake trout from the Great Lakes region. *Analytical and Bioanalytical Chemistry* 404:2699–2709. https://doi.org/10.1007/s00216-012-6125-1.

Haukås, M., U. Berger, H. Hop, B. Gulliksen, and G.W. Gabrielsen. 2007. Bioaccumulation of per- and polyfluorinated alkyl substances (PFAS) in selected species from the Barents Sea food web. *Environmental Pollution* 148(1):360–371. https://doi.org/10.1016/j.envpol.2006.09.021.

HC (Health Canada). 2023. *Health Canada*. HC, Ottawa, Ontario, Canada. Accessed January 2024. https://www.canada.ca/en/health-canada.html.

Higgins, C.P., and R.G. Luthy. 2006. Sorption of perfluorinated surfactants on sediments. *Environmental Science & Technology* 40(23):7251–7256.

Houde, M., J.W. Martin, R.J. Letcher, K.R. Solomon, and D.C.G. Muir. 2006. Biological monitoring of polyfluoroalkyl substances: A review. *Environmental Science & Technology* 40(11):3463–3473. https://doi.org/10.1021/es052580b.

Jarvis, A.L., J.R. Justice, M.C. Elias, B. Schnitker, and K. Gallagher. 2021. Perfluorooctane sulfonate in US ambient surface waters: A review of occurrence in aquatic environments and comparison to global concentrations. *Environmental Toxicology and Chemistry* 40:2425–2442. https://doi.org/10.1002/etc.5147.

Kannan, K., L. Tao, E. Sinclair, S.D. Pastva, D.J. Jude, and J.P. Giesy. 2005. Perfluorinated compounds in aquatic organisms at various trophic levels in a Great Lakes food chain. *Archives of Environmental Contamination and Toxicology* 48:559–566. https://pubmed.ncbi.nlm.nih.gov/15883668/.

Kelly, B.C., M.G. Ikonomou, J.D. Blair, B. Surridge, D. Hoover, R. Grace, and F.A.P.C. Gobas. 2009. Perfluoroalkyl contaminants in an Arctic marine food web: Trophic magnification and wildlife exposure. *Environmental Science & Technology* 43(11):4037–4043. https://doi.org/10.1021/es9003894.

Kim, S.K., and K. Kannan. 2007. Perfluorinated acids in air, rain, snow, surface runoff, and lakes: Relative importance of pathways to contamination of urban lakes. *Environmental Science & Technology* 41:8328–8334. https://doi.org/10.1021/es072107t.

Kim, D.H., J.H. Lee, and J.E. Oh. 2019. Assessment of individual-based perfluoroalkyl substances exposure by multiple human exposure sources. *Journal of Hazardous Materials* 365:26–33. https://doi.org/10.1016/j.jhazmat.2018.10.066.

Kotthoff, M., J. Müller, H. Jürling, M. Schlummer, and D. Fiedler. 2015. Perfluoroalkyl and polyfluoroalkyl substances in consumer products. *Environmental Science and Pollution* Research 22:14546–14559. https://link.springer.com/article/10.1007/s11356-015-4202-7.

Liu, X., Z. Guo, K.A. Krebs, R.H. Pope, and N.F. Roache. 2014. Concentrations and trends of perfluorinated chemicals in potential indoor sources from 2007 through 2011 in the US. *Chemosphere* 98:51–57. http://dx.doi.org/10.1016/j.chemosphere.2013.10.001.

Loi, E.I.H., L.W.Y. Yeung, S. Taniyasu, P.K.S. Lam, K. Kannan, and N. Yamashita. 2011. Trophic magnification of poly- and perfluorinated compounds in a subtropical food web. *Environmental Science & Technology* 45(13):5506–5513. https://doi.org/10.1021/es200432n.

Makey, C.M., T.F. Webster, J.W. Martin, M. Shoeib, T. Harner, L. Dix-Cooper, and G.M. Webster. 2017. Airborne precursors predict maternal serum perfluoroalkyl acid concentrations. *Environmental Science & Technology* 51:7667–7675. https://doi.org/10.1021/acs.est.7b00615.

Martin, J.W., M.M. Smithwick, B.M. Braune, P.F. Hoekstra, D.C.G. Muir, and S.A. Mabury. 2004. Identification of long-chain perfluorinated acids in biota from the Canadian Arctic. *Environmental Science & Technology* 38:373–380. https://pubs.acs.org/doi/10.1021/es034727%2B.

Monge Brenes, A.L., G. Curtzwiler, P. Dixon, K. Harrata, J. Talbert, and K. Vorst. 2019. PFOA and PFOS levels in microwave paper packaging between 2005 and 2018. *Food Additives and Contaminants: Part B Surveillance* 12:191–198. https://doi.org/10.1080/19393210.2019.1592238.

MPCA (Minnesota Pollution Control Agency). 2008. *PFCs in Minnesota's Ambient Environment: 2008 Progress Report*. MPCA, MN. Accessed February 2024. https://www.pca.state.mn.us/sites/default/files/c-pfc1-02.pdf.

Nakayama, S., M.J. Strynar, L. Helfant, P. Egeghy, X. Ye, and A.B. Lindstrom. 2007. Perfluorinated compounds in the Cape Fear Drainage Basin in North Carolina. *Environmental Science & Technology* 41:5271–5276. https://doi.org/10.1021/es070792y.

NCBI (National Center for Biotechnology Information). 2024. PubChem Compound Summary for CID 74483, Perfluorooctanesulfonic Acid. U.S. National Library of Medicine, National Center for Biotechnology Information, Bethesda, MD. Accessed February 2024. https://pubchem.ncbi.nlm.nih.gov/compound/74483.

NOAA (National Oceanic and Atmospheric Administration). 2024. *NOAA's National Status and Trends Data Page. NCCOS Data Collections*. U.S. Department of Commerce, NOAA, National Centers for Coastal Ocean Science, Silver Spring, MD. Accessed March 2024. https://products.coastalscience.noaa.gov/nsandt_data/data.aspx.

OECD (Organization for Economic Co-Operation and Development). 2002. *Hazard Assessment of Perfluorooctane Sulfonate (PFOS) and its Salts*. ENV/JM/RD(2002)17/FINAL.

Papadopoulou, E., S. Poothong, J. Koekkoek, L. Lucattini, J.A. Padilla-Sánchez, M. Haugen, D. Herzke, S. Valdersnes, A. Maage, I.T. Cousins, P.E.G. Leonards, and L. Småstuen Haug. 2017. Estimating human exposure to perfluoroalkyl acids via solid food and drinks: Implementation and comparison of different dietary assessment methods. *Environmental Research* 158:269–276. https://doi.org/10.1016/j.envres.2017.06.011.

Penland, T.N., W.G. Cope, T.J. Kwak, M.J. Strynar, C.A. Grieshaber, R.J. Heise, and F.W. Sessions. 2020. Trophodynamics of per- and polyfluoroalkyl substances in the food web of a large Atlantic slope river. *Environmental Science & Technology* 54:6800–6811. https://doi.org/10.1021/acs.est.9b05007.

Poothong, S., E. Papadopoulou, J.A. Padilla-Sánchez, C. Thomsen, and L.S. Haug. 2020. Multiple pathways of human exposure to poly- and perfluoroalkyl substances (PFASs): From external exposure to human blood. *Environment International* 134:105244. https://doi.org/10.1016/j.envint.2019.105244.

Powley, C.R., S.W. George, M.H. Russell, R.A. Hoke, and R.C. Buck. 2008. Polyfluorinated chemicals in a spatially and temporally integrated food web in the Western Arctic. *Chemosphere* 70(4):664–672. https://doi.org/10.1016/j.chemosphere.2007.06.067.

Remucal, C.K. 2019. Spatial and temporal variability of perfluoroalkyl substances in the Laurentian Great Lakes. *Environmental Science: Process & Impacts* 21:1816–1834. https://doi.org/10.1039/C9EM00265K.

Ruffle, B., U. Vedagiri, D. Bogdan, M. Maier, C. Schwach, and C. Murphy-Hagan. 2020. Perfluoroalkyl substances in U.S. market basket fish and shellfish. *Environmental Research* 190:109932. https://doi.org/10.1016/j.envres.2020.109932.

Sasaki, K., K. Harada, N. Saito, T. Tsutsui, S. Nakanishi, H. Tsuzuki, and A. Koizumi. 2003. Impact of airborne perfluorooctane sulfonate on the human body burden and the ecological system. *Bulletin of Environmental Contamination and Toxicology* 71:0408–0413. https://doi.org/10.1007/s00128-003-0179-x.

Schecter, A., J. Colacino, D. Haffner, K. Patel, M. Opel, O. Päpke, and L. Birnbaum. 2010. Perfluorinated compounds, polychlorinated biphenyls, and organochlorine pesticide contamination in composite food samples from Dallas, Texas, USA. *Environmental Health Perspectives* 118(6):796–802. https://ehp.niehs.nih.gov/doi/10.1289/ehp.0901347.

Schreder, E., and J. Dickman. 2018. *Take Out Toxics: PFAS Chemicals in Food Packaging*. Safer Chemicals Healthy Families, Washington, DC, and Toxic-Free Future, Seattle, WA. Accessed February 2024. https://toxicfreefuture.org/wp-content/uploads/2019/05/Take-Out-Toxics-Full-Report.pdf.

Shoeib, M., T. Harner, G.M. Webster, and S.C. Lee. 2011. Indoor sources of poly- and perfluorinated compounds (PFCS) in Vancouver, Canada: Implications for human exposure. *Environmental Science & Technology* 45:7999–8005. https://doi.org/10.1021/es103562v.

Smith, M.T., K.Z. Guyton, C.F. Gibbons, J.M. Fritz, C.J. Portier, I. Rusyn, D.M. DeMarini, J.C. Caldwell, R.J. Kavlock, P-F. Lambert, S.S. Hecht, J.R. Bucher, B.W. Stewart, R. Baan, V.J. Cogliano, and K. Straif. 2016. Key characteristics of carcinogens as a basis for organizing data on mechanisms of carcinogenesis. *Environmental Health Perspectives* 124:713–721.

Stahl, L.L., B.D. Snyder, A.R. Olsen, T.M. Kincaid, J.B. Wathen, and H.B. McCarty. 2014. Perfluorinated compounds in fish from U.S. urban rivers and the Great Lakes. *Science of the Total Environment* 499:185–195. https://doi.org/10.1016/j.scitotenv.2014.07.126.

Stock, N.L., V.I. Furdui, D.C. Muir, and S.A. Mabury. 2007. Perfluoroalkyl contaminants in the Canadian Arctic: Evidence of atmospheric transport and local contamination. *Environmental Science & Technology* 41:3529–3536. https://doi.org/10.1021/es062709x.

Thomford, P.J. 2002. 104-Week Dietary Chronic Toxicity and Carcinogenicity Study with Perfluorooctane Sulfonic Acid Potassium Salt (PFOS; T-6295) in Rats. Final Report. Volumes I-IX. Covance Study No. 6329-183. 3M Company, St. Paul, MN.

Tomy, G.T., W. Budakowski, T. Halldorson, P.A. Helm, G.A. Stern, K. Friesen, K. Pepper, S.A. Tittlemier, and A.T. Fisk. 2004. Fluorinated organic compounds in an eastern Arctic marine food web. *Environmental Science & Technology* 38(24):6475–6481. https://doi.org/10.1021/es049620g.

Trudel, D., L. Horowitz, M. Wormuth, M. Scheringer, I.T. Cousins, and K. Hungerbuhler. 2008. Estimating consumer exposure to PFOS and PFOA. *Risk Analysis* 28:251–269. https://doi.org/10.1111/j.1539-6924.2008.01017.x.

University of Maryland. 2024. *What We Eat in America—Food Commodity Intake Database 2005–10*. University of Maryland, College Park, MD, and EPA Office of Pesticide Programs, Washington, DC. Accessed February 2024. https://fcid.foodrisk.org/.

van der Veen, I., A.C. Hanning, A. Stare, P.E.G. Leonards, J. de Boer, and J.M. Weiss. 2020. The effect of weathering on per- and polyfluoroalkyl substances (PFASs) from durable water repellent (DWR) clothing. *Chemosphere* 249:126100. https://doi.org/10.1016/j.chemosphere.2020.126100.

Vestergren, R., and I.T. Cousins. 2009. Tracking the pathways of human exposure to perfluorocarboxylates. *Environmental Science & Technology* 43(15):5565–5575. https://pubs.acs.org/doi/abs/10.1021/es900228k.

Wang, Z., I.T. Cousins, M. Scheringer, and K. Hungerbuehler. 2015. Hazard assessment of fluorinated alternatives to long-chain perfluoroalkyl acids (PFAAs) and their precursors: status quo, ongoing challenges and possible solutions. *Environment International* 75:172–179.

Wikström, S., P. Lin, C.H. Lindh, H. Shu, and C. Bornehag. Maternal serum levels of perfluoroalkyl substances in early pregnancy and offspring birth weight. *Pediatric Research* 87:1093–1099. https://doi.org/10.1038/s41390-019-0720-1.

Wu, X.M., D.H. Bennett, A.M. Calafat, K. Kato, M. Strynar, E. Andersen, R.E. Moran, D.J. Tancredi, N.S. Tulve, and I. Hertz-Picciotto. 2015. Serum concentrations of perfluorinated compounds (PFC) among selected populations of children and adults in California. *Environmental Research* 136:264–273. https://doi.org/10.1016/j.envres.2014.09.026.

Xu, J., C. Guo, Y. Zhang, and W. Meng. 2014. Bioaccumulation and trophic transfer of perfluorinated compounds in a eutrophic freshwater food web. *Environmental Pollution* 184:254–261. https://doi.org/10.1016/j.envpol.2013.09.011.

Young, C.J., V.I. Furdui, J. Franklin, R.M. Koerner, D. Muir, and S.A. Mabury. 2007. Perfluorinated acids in arctic snow: new evidence for atmospheric formation. *Environmental Science & Technology* 41(10):3455–3461.

Young, C.J., and S.A. Mabury. 2010. Atmospheric perfluorinated acid precursors: chemistry, occurrence, and impacts. *Reviews of Environmental Contamination and Toxicology* 208:1–109.

Young, W., S. Wiggins, W. Limm, C.M. Fisher, L. DeJager, and S. Genualdi. 2022. Analysis of per- and poly(fluoroalkyl) substances (PFASs) in highly consumed seafood products from U.S. markets. *Journal of Agricultural and Food Chemistry* 70:13545–13553. https://doi.org/10.1021/acs.jafc.2c04673.

Zabaleta, I., N. Negreira, E. Bizkarguenaga, A. Prieto, A. Covaci, and O. Zuloaga. 2017. Screening and identification of per- and polyfluoroalkyl substances in microwave popcorn bags. *Food Chemistry* 230:497–506. https://doi.org/10.1016/j.foodchem.2017.03.074.

Zabaleta, I., L. Blanco-Zubiaguirre, E.N. Baharli, M. Olivares, A. Prieto, O. Zuloaga, and M.P. Elizalde. 2020. Occurrence of per- and polyfluorinated compounds in paper and board packaging materials and migration to food simulants and foodstuffs. *Food Chemistry* 321:126746. https://doi.org/10.1016/j.foodchem.2020.126746.

Zafeiraki, E., W.A. Gebbink, R. Hoogenboom, M. Kotterman, C. Kwadijk, E. Dassenakis, and S.P.J. van Leeuwen. 2019. Occurrence of perfluoroalkyl substances (PFASs) in a large number of wild and farmed aquatic animals collected in the Netherlands. *Chemosphere* 232:415–423. https://doi.org/10.1016/j.chemosphere.2019.05.200.

Zareitalabad, P., J. Siemens, M. Hamer, and W. Amelung. 2013. Perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS) in surface waters, sediments, soils and wastewater—A review on concentrations and distribution coefficients. *Chemosphere* 91:725–732. https://doi.org/10.1016/j.chemosphere.2013.02.024.

Appendix A: Bioaccumulation Factor (BAF) Supporting Information

BAF Calculation Description for PFOS

EPA used the decision framework presented in the *Technical Support Document, Volume 2: Development of National Bioaccumulation Factors* (Technical Support Document, Volume 2) (EPA, 2003) to identify procedures to derive national trophic level-specific BAFs for PFOS based on that chemical's properties (e.g., ionization, hydrophobicity), metabolism, and biomagnification potential (see Figure 1). EPA followed the guidelines provided in Section 5.5 of EPA's 2000 Methodology (EPA, 2000), to assess the occurrence of cationic and anionic forms of PFOS at typical environmental pH ranges. Based on the dissociation constant (pK_a) information provided in the Hazardous Substance Data Bank (HSDB) sources for PFOS, it was determined that ionization of PFOS was significant at typical environmental pH ranges (NCBI, 2024; EPA, 2024).

As explained in Section 5.5 of EPA's 2000 Methodology (EPA, 2000), when a significant fraction of the total chemical concentration is expected to be present as the ionized species in water, procedures for deriving the national BAF rely on empirical (measured) methods (i.e., Procedures 5 and 6) in Figure 1. EPA followed the guidelines in Section 3.2.1 of the Technical Support Document, Volume 2, to evaluate the biomagnification potential of PFOS. Based on the information in the *ATSDR Toxicological Profile for Perfluoroalkyls* (ATSDR, 2021), it was determined that biomagnification was likely. Based on the characteristics of PFOS, EPA selected Procedure 6 for deriving national BAF values for this chemical.

As described in Section 4.1.1, for a given procedure, EPA selected the method that provided BAF estimates for all three TLs (TL 2–TL 4) in the following priority:

- BAF estimates using the BAF method (i.e., based on field-measured BAFs) if possible.
- BAF estimates using the BCF method if (a) the BAF method did not produce estimates for all three TLs and (b) the BCF method produced national-level BAF estimates for all three TLs.

The EPA was able to locate field-measured BAFs for TLs 2, 3, and 4 for PFOS from the peer-reviewed literature sources for which sufficient information was provided to determine the quality and usability of the data. Therefore, EPA used the Field BAF method (EPA, 2003) to derive the national BAF values for this chemical.

Calculating Baseline BAFs

EPA calculated baseline BAFs for PFOS using a procedure analogous to the baseline BAF calculation for nonionic organic chemicals to account for the physical and chemical properties of PFOS. Dissolved field measured BAFs were considered to be 100 percent bioavailable for the purposes of the baseline BAF calculation. Field measured BAFs reported in total concentrations were converted to dissolved BAFs using national default K_{poc} values (the equilibrium partition coefficient of the chemical between the particulate organic carbon [POC] phase and the freely dissolved phase of water), from the EPA's (2000) Methodology; these BAF data were converted

from total to dissolved and then added to the dissolved field measured BAF data set and used to calculate baseline BAFs for TLs 2, 3, and 4.

Methods for calculating baseline BAFs ($(\text{Baseline BAF})_{\text{TL n}}$) involves normalizing the field-measured BAF, which are based on total concentrations in tissue and water, by the lipid content in the organism and the freely dissolved concentration in the study water (EPA, 2000, 2003). As described in EPA's (2016) Drinking Water Health Advisory for PFOS, partitioning of PFOS is related to protein binding properties. The EPA considered protein-normalizing the measured BAF values in the baseline BAF equation; however, insufficient data were available from the scientific literature on protein content of aquatic organisms and on the binding efficiencies of PFOS to various proteins in aquatic organisms. Because of this lack of data on the relationship between protein content and PFOS bioaccumulation, attempts to normalize BAFs based on protein content would likely introduce greater uncertainty into BAF averages.

Consistent with EPA's 2000 Methodology (EPA, 2000), a procedure analogous to the one used to adjust for the water-dissolved portions of a nonionic organic chemical is applied to measured BAFs for PFOS. As described in EPA's (2003) Technical Support Document, Volume 2, the K_{poc} is approximately equal to the K_{ow} of a hydrophobic organic chemical. It is further described in EPA's (2003) Technical Support Document, Volume 2, that K_{doc} (the equilibrium partition coefficient of the chemical between the dissolved organic carbon (DOC) phase and the freely dissolved phase of water) is directly proportional to the K_{ow} of a hydrophobic organic chemical, and that K_{doc} is less than the K_{ow}. Due to the physical-chemical properties of PFOS, its K_{ow} cannot be reliably measured for these compounds and therefore cannot be used to estimate K_{poc} or K_{doc} (ATSDR, 2021; Hidalgo and Mora-Diez, 2016; EPA, 2003, 2024; Xiang et al. 2018)**.**

Using the K_{oc} information in the study of Higgins and Luthy (2006), EPA determined that the K_{oc} values were applicable to POC but there is no indication that they would be applicable to DOC. Currently, information is not available on the partitioning of PFOS to DOC, nor on the bioavailability of PFOS partitioned to DOC. In addition, Higgins and Luthy (2006) included DOC-bound PFOS in the aqueous phase of their calculations. Thus, the amount of PFOS partitioned to DOC was presumed to be part of the aqueous fraction of the f_{fd} equation, resulting in the following formula (Equation 1):

$$f_{fd} = \frac{1}{[1 + (POC \cdot K_{OC})]} \qquad \text{(Eq. 1)}$$

Where:

- f_{fd} = fraction of the total concentration of chemical in water that is freely dissolved.
- POC = national default value of 0.5 mg/L (refer to page 5-44 of EPA's 2000 Methodology (U.S. EPA, 2000)) is used in baseline BAF calculations, unless this value is reported in the BAF source.
- K_{oc} = PFOS log K_{oc} = 2.57 (Higgins and Luthy, 2006).

Because the measured BAFs for PFOS are not adjusted for lipid or protein content, the baseline BAF equation (refer to Equation 5-10 on pages 5-24 and 5-25 of EPA's 2000 Methodology [EPA, 2000]) is adjusted (as shown below in Equation 2) to determine the freely dissolved concentration of PFOS BAFs in water:

$$\text{Baseline BAF} = \frac{\text{Measured BAF}}{f_{fd}} - 1 \qquad \text{(Eq. 2)}$$

EPA used this equation to calculate baseline BAFs from field measured BAFs based on total concentrations.

Dissolved PFOS Baseline BAFs

EPA included results from several field BAF studies for PFOS reported as dissolved (i.e., filtered) concentrations in its baseline BAF calculations. Because these dissolved PFOS data are presumed to represent the freely-dissolved (non-particulate) fraction, the f_{fd} term in Equation 2 is set to 1. Also, as described above, the measured BAFs for PFOS are not being adjusted for lipid or protein content to calculate baseline BAFs for PFOS. Thus, Equation 3 is used to calculate the freely dissolved concentration of PFOS for "baseline BAFs" using field-measured dissolved PFOS BAFs:

$$\text{Baseline BAF} = \text{Measured (dissolved) BAF} - 1 \qquad \text{(Eq. 3)}$$

Calculating National BAFs

Final baseline BAFs were used to compute national BAFs for PFOS. Equation 4 (an equation analogous to the equation used for nonionic organic chemicals in EPA's 94 chemical criteria updates (EPA, 2015) for calculating national BAFs (see Equation 5-28 on Page 5-42 of EPA's 2000 Methodology [U.S. EPA, 2000]) is used to convert the baseline BAF to a national BAF for each trophic level:

$$\text{National BAF}_{(\text{TL n})} = [(\text{Final Baseline BAF}^{fd})_{\text{TL n}} + 1] \cdot (f_{fd}) \qquad \text{(Eq. 4)}$$

Where:

- National BAF = national BAF (L/kg-tissue)
- $(\text{Final Baseline BAF})_{\text{TL n}}$ = mean baseline BAF for TL "n" (L/kg-lipid)
- f_{fd} = fraction of the total concentration of chemical in water that is freely dissolved

In summary, for PFOS, the baseline BAFs are calculated using Equation 2 (for field BAFs calculated from total water concentrations) and Equation 3 (for field BAFs calculated from dissolved water concentrations) for each TL. National BAFs are then calculated from TL baseline BAFs using Equation 4.

National Trophic level BAF calculations:

$$\text{National BAF PFOS}_{(\text{TL }2)} = [(425.0)_{\text{TL }2} + 1] \times (0.999814267)$$

= 425.9 L/kg

= 420 L/kg (rounded)

$$\text{National BAF PFOS}_{(\text{TL }3)} = [(1663.4)_{\text{TL }3} + 1] \times (0.999814267)$$

= 1664.1 L/kg

= 1700 L/kg (rounded)

$$\text{National BAF PFOS}_{(\text{TL }4)} = [(859.2)_{\text{TL }4} + 1] \times (0.999814267)$$

= 860.0 L/kg

= 860 L/kg (rounded)

The corresponding values for TL 2, TL 3 and TL 4 were computed as 425.9 L/kg, 1664.1 L/kg and 860.0 L/kg, respectively. Rounding the values to two significant figures yields national BAF values of 420, 1,700 and 860 L/kg for TLs 2, 3, and 4, respectively.

References

ATSDR (Agency for Toxic Substances and Disease Registry). 2021. *Toxicological Profile for Perfluoroalkyls*. U.S. Department of Health and Human Services, ATSDR, Atlanta, GA. https://www.atsdr.cdc.gov/ToxProfiles/tp200.pdf.

EPA (Environmental Protection Agency). 2000. *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000)*. EPA-822-B-00-004. EPA, Office of Water, Office of Science and Technology, Washington, DC. https://nepis.epa.gov/Exe/ZyPDF.cgi/20003D2R.PDF?Dockey=20003D2R.PDF.

EPA (Environmental Protection Agency). 2003. Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000), Technical Support Document. Vol. 2, Development of National Bioaccumulation Factors. EPA-822-R-03-030. EPA, Office of Water, Office of Science and Technology, Washington, DC. https://www.epa.gov/sites/default/files/2018-10/documents/methodology-wqc-protection-hh-2000-volume2.pdf.

EPA (Environmental Protection Agency). 2015. *Development of National Bioaccumulation Factors: Supplemental Information for EPA's 2015 Human Health Criteria Update*. EPA 822-R-16-001. https://www.epa.gov/sites/default/files/2016-01/documents/national-bioaccumulation-factors-supplemental-information.pdf.

EPA (Environmental Protection Agency). 2016. *Drinking Water Health Advisory for Perfluorooctane Sulfonate (PFOS)*. EPA 822-R-16-004. EPA, Office of Water, Health and Ecological Criteria Division. Washington, DC. https://www.epa.gov/sites/default/files/201605/documents/pfos_health_advisory_final_508.pdf.

EPA (Environmental Protection Agency). 2024. *Final: Human Health Toxicity Assessment for Perfluorooctane Sulfonic Acid (PFOS) and Related Salts*. EPA 815-R-24-007. EPA, Office of Water, Washington, DC. https://www.epa.gov/system/files/documents/2024-04/main_final-toxicity-assessment-for-pfos_2024-04-09-refs-formatted_508c.pdf.

Hidalgo, H., and N. Mora-Diez. 2016. Novel approach for predicting partition coefficients of linear perfluorinated compounds. *Theoretical Chemistry Accounts* 135(18):1–11.

Higgins, C., and R. Luthy. 2006. Sorption of perfluorinated surfactants on sediments. *Environmental Science & Technology* 40(23):7251–7256.

NCBI (National Center for Biotechnology Information). 2024. *PubChem Compound Summary for Perfluorooctane Sulfonic Acid*. U.S. National Library of Medicine, NCBI, Bethesda, MD. https://pubchem.ncbi.nlm.nih.gov/compound/Perfluorooctanesulfonic-acid/.

Xiang, Q. G. Shan, W. Wu, H. Jin, and L. Zhu. 2018. Measuring log K_{ow} coefficients of neutral species of perfluoroalkyl carboxylic acids using reversed-phase high-performance liquid chromatography. *Environmental Pollution* 242:1283–1290.

Appendix B: Comparative Analysis for Potentially Sensitive Populations for PFOS

The EPA evaluated several exposure scenarios for PFOS to determine whether the national recommended criteria for the general population (male and female adults ≥ 21 years old) are sufficiently protective of potentially sensitive subpopulations. To accomplish this, the EPA considered three additional exposure scenarios, as supported by data from the EPA *Exposure Factors Handbook* (EFH; EPA, 2011) and the Human Health Methodology (EPA, 2000). Specifically, the EPA evaluated exposure parameters for "all ages" as well as two potentially sensitive life stages associated with the critical effects used to derive the chronic RfD, i.e., decreased infant birth weight and increased total cholesterol in adults as the co-critical effects. Based on this exposure interval in the critical study, the potentially sensitive subpopulations in humans include women of childbearing age who may be or become pregnant and pregnant women (EPA, 2024; Table B-1, this document).

For the body weight exposure parameter, a mean bodyweight of 75 kg for pregnant women (all trimesters) was identified in the EFH (2011, Ch. 8, Table 8-29). A representative body weight for the "all ages" scenario was not specifically presented in the EFH (EPA, 2011). To address this data limitation, for this exercise, the EPA assumed that the average body weight for "all ages" was 71.6 kg based on the sum of the time-weighted averages of the mean male and female combined body weights from 1 year up to 80 years old from the NHANES (1999–2006) (EPA, 2011, Table 8-3). A body weight average of 67 kg for women of childbearing age was identified in the Human Health Methodology (EPA, 2000); however, this average is based on an older NHANES dataset (NHANES III; WESTAT 2000). More recent NHANES data (1999–2006) suggest that the mean body weight for women of childbearing age ranges from 65.9 kg for 16 to < 21-year-olds to 77.1 kg for 40 to < 50-year-olds (EPA, 2011, Table 8-5). Using these data, the EPA assumed a time-weighted average body weight of 73.4 kg for women of childbearing age (EPA, 2011, Table 8-5).

Drinking water intake values were available for all populations (Table B-1, this document).

The EPA encountered several data limitations for trophic level specific fish consumption rates for some of these potentially sensitive populations. The EPA's national criteria are typically derived using trophic-level specific fish consumption rates (FCRs), paired with trophic-level specific bioaccumulation factors (BAFs) to account for the potential bioaccumulation of some chemicals in aquatic food webs and the broad physiological differences between trophic levels which may influence bioaccumulation (EPA, 2000). Trophic level specific FCRs for women of childbearing age were identified (Table B-1). However, trophic level specific FCRs are not available for two of the potentially sensitive life stages—all ages and pregnant women. Therefore, criteria could not be calculated for these two populations. However, in all cases with available data, the total FCR for the alternative scenarios is lower than the FCR for the general population. Because bodyweights are similar for all of the considered populations (see above and Table B-1), the FCR is likely to be the main determinant of the criteria value, with a larger FCR resulting in a lower, more health protective criterion. Therefore, criteria based on the general population are expected to be protective of the identified potentially sensitive life stages (Table B-1). Separately, paired bodyweight adjusted FCRs are not available for specific trophic levels which precludes the use of body-weight adjusted DWI rates to derive ambient water quality criteria.

Table B-1. Comparison of noncancer-based HHC values for different candidate sensitive populations identified from the critical effect and study.

Population	Bodyweight (kg)	Drinking Water Intake (L/day)	Fish Consumption Rate (g/day)				Criteria (µg/L)	
			Total	TL 2	TL 3	TL 4	W + O	OO
General, adult (≥ 21 years)	**80[a]**	**2.3[b]**	**22[c]**	**7.6[c]**	**8.6[c]**	**5.1[c]**	**0.00006**	**0.00007**
Women of childbearing Age (13–49 years)	73.4[d]	2.1[e]	15.8[c]	5.6[c]	6.0[c]	2.9[c]	0.00008	0.0001
All Ages (Birth to 80 years)	71.6[f]	2.0[b]	19.3[g]	NA	NA	NA	ND	ND
Pregnant Women	75[h]	2.1[e]	10[i]	NA	NA	NA	ND	ND

Notes: g/day = grams of fish consumed per day; L/day = liters of water per day; NA = not available; ND = not determined; OO = organism only; W + O = water plus organism.
Bold values indicate draft national recommended criteria.
Gray highlighting indicates most health protective HHC based on noncancer effects.
[a] EPA, 2011, *Exposure Factors Handbook*, Ch. 8, Table 8-1, NHANES 1999–2006. Recommended mean bodyweight for adults.
[b] Estimated using the FCID calculator (University of Maryland, 2024; https://fcid.foodrisk.org/), NHANES 2005–2010, community water, 90th percentile per capita rate.
[c] EPA, 2014; NHANES 2003–2010 survey data, 90th percentile per capita rate, freshwater and estuarine fish and shellfish edible portion, adults ≥ 21 years.
[d] Time weighted average of combined bodyweights for women ages 16 to < 50 years, NHANES 1999–2006 (EPA, 2011; Table 8-5).
[e] EPA, 2019, *Exposure Factors Handbook*; Update Ch. 3., Table 3-62, Community water, 90th percentile, per capita rate.
[f] Time weighted average of mean male and female combined body weights from 1 year up to 80 years, NHANES 1999–2006 (EPA, 2011; Table 8-3).
[g] Estimated using the FCID calculator (University of Maryland, 2024; https://fcid.foodrisk.org/), NHANES 2005–2010; freshwater and estuarine fish and shellfish combined, 90th percentile per capita rate; male and female, all ages included.
[h] EPA, 2011, *Exposures Factors Handbook*, Ch 8, mean, NHANES 1999–2006, Table 8-29.
[i] Estimated using the FCID calculator (University of Maryland, 2024; https://fcid.foodrisk.org/), NHANES 2005–2010; freshwater and estuarine fish and shellfish combined, 90th percentile per capita rate pregnant females only.

For illustrative purposes, the EPA calculated criteria based on the exposure parameters for women of childbearing age. As demonstrated in Table B-1, criteria based on the exposure inputs for the general population result in the more health protective criteria and thus are protective of the potentially susceptible life stage of women of childbearing age (Table B-1). Overall, when bodyweight averages are similar, the resulting criteria are driven predominantly by the FCR; thus, a higher FCR results in a more health protective criteria.

References

EPA (Environmental Protection Agency). 2000. *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000)*. EPA-822-B-00-004. EPA, Office of Water, Office of Science and Technology, Washington, DC. Accessed January 2024. https://www.epa.gov/sites/default/files/2018-10/documents/methodology-wqc-protection-hh-2000.pdf.

EPA (Environmental Protection Agency). 2011. Body Weight Studies. Chapter 8 in *Exposure Factors Handbook*. EPA/600/R-09/052F. EPA, National Center for Environmental Assessment, Office of Research and Development, Washington, DC. Accessed August 2024. https://www.epa.gov/sites/default/files/2015-09/documents/efh-chapter08.pdf.

EPA (Environmental Protection Agency). 2014. *Estimated Fish Consumption Rates for the U.S. Population and Selected Subpopulations (NHANES 2003–2010)*. EPA-820-R-14-002. EPA. Accessed August 2024. https://www.epa.gov/sites/default/files/2015-01/documents/fish-consumption-rates-2014.pdf.

EPA (Environmental Protection Agency). 2019. *Update for Chapter 3 of the Exposure Factors Handbook, Ingestion of Water and Other Select Liquids*. EPA/600/R-18/259F. EPA, Office of Research and Development, National Center for Environmental Assessment, Washington, DC. Accessed August 2024. https://www.epa.gov/sites/default/files/2019-02/documents/efh_-_chapter_3_update.pdf.

EPA (Environmental Protection Agency). 2024. *Final: Human Health Toxicity Assessment for Perfluorooctane Sulfonic Acid (PFOS) and Related Salts*. EPA 815-R-24-007. Office of Water, Washington, DC. https://www.epa.gov/system/files/documents/2024-04/main_final-toxicity-assessment-for-pfos_2024-04-09-refs-formatted_508c.pdf.

WESTAT. 2000. *Memorandum on Body Weight Estimates Based on NHANES III Data, Including Data Tables and Graphs*. Analysis Conducted and Prepared by WESTAT under EPA Contract No. 68-C-99-242. March 3, 2000.

University of Maryland. 2024. *What We Eat in America—Food Commodity Intake Database 2005–10*. University of Maryland, College Park, MD, and EPA Office of Pesticide Programs, Washington, DC. Accessed August 2024. https://fcid.foodrisk.org/.