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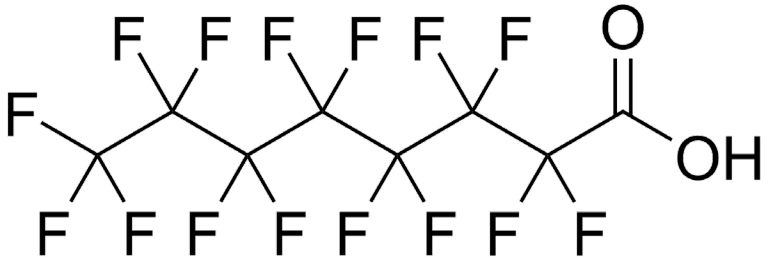
DRAFT Human Health Ambient Water Quality Criteria:

Perfluorooctanoic Acid (PFOA) and Related Salts



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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.

Perfluorooctanoic acid (PFOA) 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) PFOA 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* (EPA, 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.

PFOA 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. PFOA is a strong acid that is generally present as the perfluorooctanoate anion at typical environmental pH values. Therefore, this assessment applies to all isomers of PFOA, as well as nonmetal salts of PFOA that would be expected to dissociate in aqueous solutions of pH ranging from 4 to 9. For the purpose of this assessment, "PFOA" will signify the ion, acid or any nonmetal salt of PFOA.

2.1 Uses and Sources of PFOA

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 PFAS stem from the resistance of these compounds to hydrolysis, photolysis, metabolism, and microbial degradation.

PFOA is a synthetic, fully fluorinated, organic acid that is used in many types of consumer products and in the production of fluoropolymers (EPA, 2016a,b). PFOA is also formed by microbial, metabolic and abiotic degradation of many precursor chemicals. PFOA and its precursors have been used in flame repellents, cosmetics, paints, polishes, and processing aids used in the manufacture of nonstick coatings on cookware. It is one of a large group of perfluoroalkyl substances that are used in consumer and industrial products, etc. to improve their resistance to stains, grease, and water (Gaines, 2023). In 2006, EPA initiated the 2010/2015 PFOA Stewardship Program, resulting in major PFOA producers committing to a 95% reduction in PFOA facility emissions and product contents across the globe by 2010. The 2010/2015 PFOA Stewardship Program further aimed to eliminate PFOA emissions and product content by 2015 (EPA, 2006, 2023a). The EPA has found widespread PFOA contamination in

water, sediments, and soils. Exposure to PFOA can occur through food including fish and shellfish, house dust, air, and contact with consumer products.

2.2 Environmental Fate and Transport in the Environment

Under most environmental conditions PFOA in water rapidly dissociates into ionic components. In aquatic environments, the sorption of PFOA to sediments varies based on the amount of organic carbon present and other site-specific conditions; the range of log(Kd)[b] values reported in the literature for PFOA in sediments is −0.7 to 4.9 (EPA, 2024a). Because of its water solubility and preferential binding to proteins, once PFOA enters a waterbody it can remain dissolved in the water column, sorb to organic particulate matter, or be assimilated by organisms. In the water column, and other environmental compartments, PFOA is stable and resistant to hydrolysis, photolysis, volatilization, and biodegradation (NCBI, 2024; Lange et al. 2006). The persistence of PFOA has been attributed to the strong carbon-fluorine (C-F) bond.

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

PFOA has been detected in a variety of environmental matrices. The occurrence and detection of PFOA 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, PFOA (along with perfluorooctane sulfonic acid [PFOS]) is one of the dominant PFAS compounds detected in ambient water in the United States and worldwide (Ahrens, 2011a; Benskin et al., 2012; Dinglasan-Panlilio et al., 2014; Nakayama et al., 2007; Remucal, 2019; Zareitalabad et al., 2013). Most of the current, published PFOA occurrence studies have focused on a handful of broad geographic regions in the United States, often targeting sites with known manufacturing or industrial uses of PFAS such as the Great Lakes, the Cape Fear River, and waterbodies near Decatur, Alabama (Boulanger et al., 2004; Hansen et al., 2002; Konwick et al., 2008; Nakayama et al., 2007; 3M, 2000). PFOA concentrations in global surface waters range over seven orders of magnitude, generally in picogram per liter (pg/L) to nanogram per liter (ng/L) concentrations, but sometimes reaching microgram per liter (µg/L) levels (Jarvis et al., 2021; Zareitalabad et al., 2013).

PFOA concentrations in surface water tend to increase with increasing levels of urbanization. Across the Great Lakes region, PFOA was higher in the downstream lakes (Lake Erie and Lake Ontario), which are more heavily impacted by urbanization, and lower in the upstream lakes (Lakes Superior, Michigan, and Huron), which are located in relatively rural and forested areas (Remucal, 2019). Similarly, Zhang et al. (2016) found measured surface water PFOA concentrations in urban areas (urban average PFOA concentration = 10.17 ng/L; n = 20) to be more than three times greater than concentrations in rural areas (rural average PFOA concentration = 2.95 ng/L; n = 17) within New Jersey, New York, and Rhode Island. Seasonal variations in PFOA levels in U.S. surface waters remain largely unknown due to a lack of data.

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

2.3.2 Occurrence in Freshwater and Estuarine Fish and Shellfish

PFOA has been detected in freshwater fish fillet samples collected during several national studies in rivers and the Great Lakes; however, PFOA is reported at a lower frequency and at lower levels compared to other PFAS, including PFOS (Table 1). 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. The National Oceanic and Atmospheric Administration (NOAA) recorded 159 data points available for aquatic organisms in the National Status and Trends Data Portal for PFOA focusing on dreissenid mussel and other mussels, oyster, fish fillet, and fish liver samples. There were six detections reported, ranging from 0.33 ng/g wet weight (ww) to 75.1 ng/g ww; 153 were below the method detection limit (MDL) or not detected (NOAA, 2024).

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

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	No PFOA detections reported.
2013–2014 NRSA (EPA, 2020, 2023b)	Channel catfish Largemouth bass Smallmouth bass	349 urban and nonurban river sites across the United States	PFOA detected in 4% of fillet samples. Maximum detected concentration 0.27 ng/g.
2018–2019 NRSA (EPA, 2023c,d)	Channel catfish Smallmouth bass Largemouth bass	290 urban and nonurban river sites across the United States	PFOA detected in 2% of fillet samples. Maximum detected concentration 0.354 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	PFOA detected in 12% of fillet samples. Maximum detected concentration 0.97 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	PFOA detected in 14% of fillet samples. Maximum detected concentration 1.93 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).	PFOA was detected in < 1% of samples (0.98%). Maximum detected concentration: 1.55 ng/g; median < MDL (0.152 ng/g).

In addition, Penland et al. (2020) measured PFAS concentrations in invertebrates and vertebrates along the Yadkin-Pee Dee River in North Carolina and South Carolina. PFOA was detected in whole body tissues of unionid mussels (7.41 ng/g ww) and aquatic insects (10.68 ng/g ww), but was not detected in Asian clam, snails, or crayfish. PFOA was measured in muscle tissue of 2 out of 11 sampled fish species: the channel catfish (21.19 ng/g ww) and notchlip redhorse (45.66 ng/g ww).

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 only considers 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 (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:

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

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

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

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 of life stage (e.g., pregnant women and their fetuses, young children) is the target 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 PFOA 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 PFOA 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 years 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) 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[d] 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 default 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.

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, 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

[d] *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).

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 PFOA 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. Each procedure contains a hierarchy of the BAF derivation methods (listed below); however, this hierarchy should not be considered inflexible (EPA, 2000). 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 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, 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 expected 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 PFOA 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, 2024e) and PFOS (EPA, 2024f) and published 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 PFOA 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 PFOA.

Specifically, studies were evaluated for inclusion in the dataset used for calculating national BAFs for PFOA 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.
- Studies in which the BAFs were not found to be at steady state were excluded.
- Initially, 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). After further review, for pooled samples, averaging data from multiple locations was considered acceptable if corresponding tissue and water concentrations were available from the overall spatial area of the study.

In addition to the evaluation criteria listed above, PFOA bioaccumulation data were also subsequently evaluated using the following study evaluation criteria outlined in Burkhard (2021) (Table 2).

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.

Table 2. Bioaccumulation factor (BAF) study quality criteria based on suggested criteria in 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.

4.2.3 BAFs for PFOA

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 PFOA is an organic chemical that predominantly exists in an anionic form in water (EPA, 2024g,h; 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 PFOA.

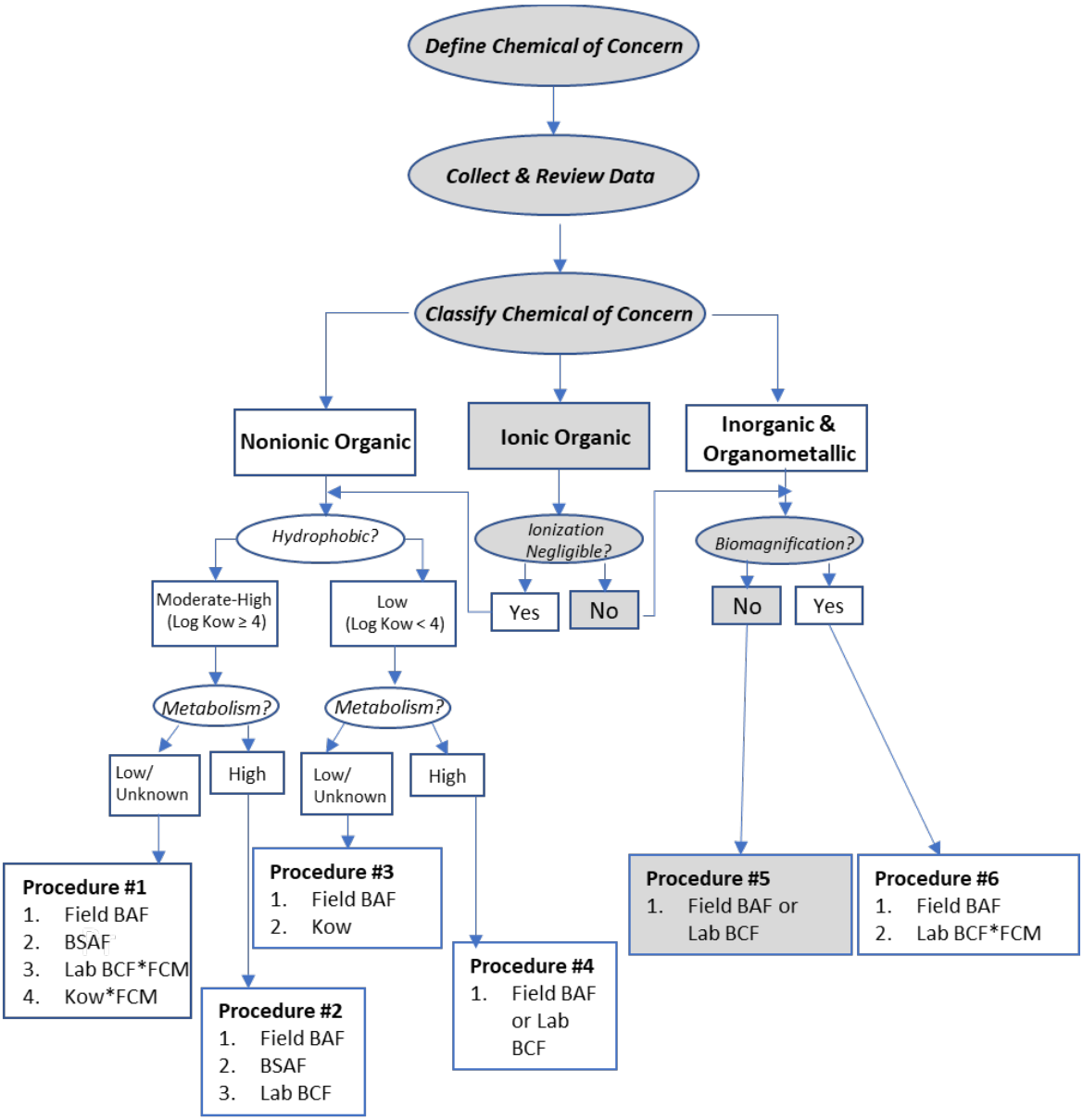

Figure 1. Application of the BAF framework for PFOA; gray boxes indicate steps followed based on available information for PFOA (EPA, 2000a).

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 PFOA is related to protein binding properties (ATSDR, 2021); therefore, the EPA did not normalize measured BAF values for PFOA 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). Adjustments for water-dissolved portions of PFOA is applied to TL baseline BAFs (EPA, 2000a) (see Appendix A).

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 PFOA. Based on the characteristics of this chemical, the EPA selected Procedure 5 for deriving a national BAF value. PFOA has the following characteristics:

- Ionic organic chemicals, with ionization not negligible (NCBI, 2024).
- Biomagnification unlikely (Houde et al., 2011; Du et al., 2021; Munoz et al., 2022).

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 PFOA:

- TL 2 = 22 L/kg
- TL 3 = 49 L/kg
- TL 4 = 31 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 PFOA. 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 a 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 a 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 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 websites of 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, 2024i)
    - Provisional Peer-Reviewed Toxicity Values (PPRTV) (EPA, 2024j)
    - ORD Human Health Toxicity Values (EPA, 2024k)
- EPA, Office of Pesticide Programs (EPA, 2024l)
- EPA, Office of Pollution Prevention and Toxics (EPA, 2024m)
- EPA, Office of Water Drinking Water Health Effects Support Documents (EPA, 2024n)
- 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 RfD 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 PFOA

5.2.1 Reference Dose

After following the approach outlined in Section 5.1, the EPA identified the final *Human Health Toxicity Assessment for Perfluorooctanoic Acid (PFOA) and Related Salts* (EPA, 2024g). This document is the most recent toxicity assessment identified for PFOA 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 PFOA.

The EPA's final human health toxicity assessment for PFOA (EPA, 2024g) considered all publicly available human epidemiological, animal toxicological, mechanistic and toxicokinetic evidence relevant to studies that evaluated health effects after oral PFOA exposure. Overall, the available *evidence indicates* that PFOA 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 1.1 to 5.2 ng/mL and doses in animals as low as 0.3 to 1.0 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 serum anti-tetanus and anti-diphtheria antibody concentrations in children (Budtz-Jorgensen and Grandjean, 2018), decreased infant birth weight (Wikström et al., 2020), and increased total cholesterol in adults (Dong et al., 2019) were selected as the co-critical effects

for the overall oral **RfD of 3 × 10^{-8} mg/kg/day** (EPA, 2024g). 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 antibody concentrations in children, decreased birth weight) represent effects in susceptible subpopulations. The RfD based on these effects is considered protective of effects resulting from lifetime exposures to PFOA, 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 *Human Health Toxicity Assessment for Perfluorooctanoic Acid (PFOA) and Related Salts* (EPA, 2024g) reviewed the weight of the evidence across epidemiological, animal toxicological, and mechanistic studies and concluded that PFOA is *Likely to Be Carcinogenic to Humans* via the oral route of exposure. Epidemiological studies provided evidence of kidney and testicular cancer in humans and some evidence of breast cancer in susceptible subpopulations. Chronic oral animal toxicological studies in Sprague-Dawley rats reported Leydig cell tumors (LCT), pancreatic acinar cell tumors (PACT), and hepatocellular tumors. PFOA exposure is associated with multiple key characteristics of carcinogenicity (Smith, 2016). Available mechanistic data suggest that multiple MOAs could be involved in the renal, testicular, pancreatic, and hepatic tumorigenesis associated with PFOA exposure in humans and animal models.

To derive a CSF for PFOA, the EPA followed agency risk assessment guidelines and methodologies (EPA, 2005a, 2012, 2022c). EPA conducted benchmark dose modeling and used a similar pharmacokinetic modeling approach as described for the derivation of noncancer RfDs above (see Section 5.2.1). EPA derived and considered multiple candidate CSFs from both epidemiological and animal toxicological studies across multiple tissue types and organ systems (i.e., kidney, liver, pancreas, testes). CSFs were derived for epidemiological data on renal cell carcinoma (RCC) and kidney cancer using weighted linear regressions to calculate quartile-specific relative kidney cancer risks. Relative risks were then converted to the absolute risk scale, yielding an internal CSF, which represents the excess cancer risk associated with each ng/mL increase in serum PFOA. The internal serum CSF was then divided by the selected clearance value and converted to an external dose CSF. For animal toxicological studies, 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 (EPA, 2024g).

The **oral slope factor of 0.0293 (ng/kg/day)$^{-1}$** (29,300 (mg/kg/day)$^{-1}$) for RCC in human males from Shearer et al. (2021) was selected as the basis of the overall CSF for PFOA (EPA, 2024g). 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 determined for PFOA from a review of the available studies, and evidence did not support increased susceptibility to cancer following PFOA exposure during early life.

6 Relative Source Contribution (RSC) 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 PFOA, 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 PFOA (EPA, 2024g). To identify information on PFOA 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 PFOA. 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 PFOA 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 Perfluorooctanoic Acid (PFOA) and Related Salts (EPA, 2024h). The following description in Section 6.2 is a summary of the information provided in the Appendix of the final PFOA toxicity assessment.

6.2 Summary of Potential Exposure Sources of PFOA 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 PFOA 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 the U.S. population is typically estimated to be about 2 ng/kg-d to 3 ng/kg-d (Gleason et al., 2017). 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 gastrointestinal uptake (Trudel et al., 2008). However, the estimates are highly uncertain due to analytical methods with poor sensitivity, relatively few food items with detectable levels, and levels that can vary greatly depending on sources or location (Gleason et al., 2017).

There is currently no comprehensive, nationwide Total Diet Study (TDS) for PFOA that can be used to draw conclusions about the occurrence and potential risk of PFOA 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 but did not include PFOA (FDA, 2021b). PFOA was not

detected in any of the food samples analyzed in the FDA TDS samples of produce, meats, dairy and grain products in 2019 or 2021 (FDA, 2020a,b, 2021c). In a 2018 focused study near a PFAS production plant in the Fayetteville, North Carolina area, PFOA was detected in several produce samples (cabbage, collard greens, kale, mustard greens, swiss chard, and lettuce) (FDA, 2018). In bottled water, PFOA was below the lower limit of quantification (LOQ; 4 ng/L) in all (30) analyzed samples of domestic and imported carbonated water and noncarbonated bottled water (FDA, 2016). The sample size in these studies is limited, and thus, the results cannot be used to draw definitive conclusions about the general levels of PFAS in the U.S. food supply (FDA, 2023). In a 2010 study, PFOA was detected in food samples collected from five grocery stores in Texas (Schecter et al., 2010); based on the results from this study and on dietary intakes from the 2007 USDA food availability data set, the estimated daily exposure to PFOA per capita was 60 ng/day (EPA, 2016a).

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 LOD or LOQ, lower bound dietary exposure estimates were obtained by assigning zero to values below LOD/LOQ. Median chronic dietary exposures of PFOA for children and adults were estimated as 0.30 and 0.18 ng/kg body weight per day, respectively. The most important contributor was "fish and other seafood[e]," followed by "eggs and egg products," "meat and meat products," and "fruit and fruit products." "Vegetables and vegetable products" and "drinking water" were also found to be important contributors to dietary PFOA exposure. It is unclear whether or not the contribution from food contact material is reflected in the data.

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 PFOA, dietary intake was by far the greatest contributor to aggregate exposure (contributing 92% of total estimated PFOA intake), but intake from ingestion of house dust represented the dominant pathway for some of the top 20% most highly exposed individuals. On average, measured serum concentrations of PFOA were similar to modeled concentrations based on intakes. It is notable that while the authors reported significant positive correlations between PFOA concentrations in serum and estimated intakes based on surface dust and vacuum cleaner bag dust samples, correlations with estimated dietary intakes were not significant, which the authors attributed to temporal variations in dietary intakes over several years. While the authors did not separately quantify intake from food and drinking water, an earlier article from the same research group

[e] 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.

(Papadopoulou et al., 2017) reported measured concentrations in duplicate diets with median estimated intake of PFOA approximately three times higher from solid food than from liquids.

Zafeiraki et al. (2019) analyzed about 250 samples of marine fish, farmed fish, crustaceans, bivalves and European eel, caught in Dutch waters or purchased at Dutch markets between 2012 and 2018. Samples were analyzed for 16 PFAS, including PFOA. Brown crab and shrimp had the highest average concentrations of PFOA (0.78 ng/g ww and 0.43 ng/g ww, respectively). PFOA was also detected in farmed fish including eel and trout, and marine fish species including cod, haddock, and sole.

In seafood samples collected for the FDA 2021–2022 seafood survey (FDA, 2022), Young et al. (2022), analyzed concentrations of 20 PFAS, including PFOA, in eight of the most highly consumed marine seafood products in the United States. PFOA was detected most frequently (100% of samples; n = 10) and at the highest average concentrations (8,334 parts per trillion [ppt]) in clams and was also detected in 100% of crab samples (n = 11; 300.9 ppt average concentration). The study reported detections in cod (20% of samples; n = 10; 103.5 ppt average concentration in samples with detections). PFOA was not detected above the MDL (68 ppt or 90 ppt) in tuna, salmon, shrimp, or pollock.

6.2.2 Food Contact Materials

The FDA has authorized the use of PFAS in food contact substances due to their nonstick 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, takeout 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).

Schaider et al. (2017) collected 407 samples of food contact papers, beverage containers, and paperboard boxes from locations throughout the United States. Twenty fast food packaging samples of the 407 total samples were selected for more extensive PFAS specific analysis. PFOA, was among the PFAS with the highest detection rates and was detected in six out of 20 samples.

An analysis of popcorn bags, snack bags, and sandwich bags purchased in 2018 from international vendors and grocery stores in the United States found little evidence of PFOA, with only two popcorn bags with content above the limit of quantitation of 5.11 ng per gram (ng/g) of paper (Monge Brenes et al., 2019). The authors presented these results as evidence of a reduction in PFOA 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 PFOA in the two bags from the United States, levels typically at about 4 ng/g in those from several European countries, and levels around 50 ng/g in bags from China.

Yuan et al. (2016) analyzed 25 food contact materials purchased in Columbus, Ohio for PFAS as compared to 69 products purchased in China. In food packaging materials from China, of the 15 detected perfluorinated carboxylic acids, PFOA was the most frequently detected (90%) and was detected with the highest median concentration (1.72 ng/g). The authors also report a migration efficiency of PFOA from paper bowl packaging into food stimulants of 1.58%. This is a relatively low efficiency compared to several of the fluorotelomer alcohols (FTOHs) which the authors reported to migrate with greater than 90% efficiency.

Zabaleta et al. (2020) also monitored migration of the PFAS carboxylates (C6 to C10) from packaging materials into cereal, rice, or milk. For each PFAS studied the percent migration to milk exceeded that to rice with the lowest percent migration being that to cereal. The migration percentage of PFOA into cereal, rice, and milk powder products over six months ranged from 1.4%–5.6%.

6.2.3 Consumer Product Uses

A targeted analysis of 29 U.S. and Canadian cosmetic products with high fluorine content (Whitehead et al., 2021) found high concentrations of FTOH, including 8:2 FTOH, commonly present in the formulations. A fraction of 8:2 FTOH is believed to undergo metabolic transformation into PFOA. In addition to direct contact with personal care products, products and articles (and the use of these) may be sources in the indoor environment that manifest as measured occurrence in house dust and indoor air. An earlier investigation of consumer exposure to PFOA by Trudel et al. (2008) used mechanistic modeling together with information on product use habits to estimate oral and dermal exposures from clothes, carpet, upholstery, and food contact materials. Noting that PFOA may be contained as a contaminant in older and in new products, the authors estimated exposure via both mill-treated and home-treated carpets. The authors concluded that contact with consumer products is not a significant contributor to total exposure, but that since PFOA may be a contaminant in even new products, consumer exposure may continue to occur, particularly via both mill-treated and home-treated carpets. The authors also point out that carpet and other textiles are likely to be continuing

sources of PFOA in house dust. In contrast, in an analysis of 116 articles of commerce from the United States, the EPA (2009) identified carpets and related products as potentially the most significant source of perfluorinated carboxylic acids (PFCAs) out of 13 total product categories analyzed. PFOA was detected in all 13 product types. Other important indoor sources of PFCAs include floor wax/sealant and home textiles, upholstery, and apparel. In a similar analysis of 52 European products collected between 2014–2016, Borg and Ivarrson (2017) reported that PFOA was the most commonly detected PFAS and was detected in all samples except those that did not contain any detectable levels of PFAS. Notably, the authors specifically targeted products that were known or suspected to contain PFAS in their analyses.

Liu et al. (2014) investigated trends in PFAS content of household goods between 2007 and 2011. They reported that while PFOA concentrations displayed an overall downward temporal trend with significant reductions observed in nearly all product categories, PFOA was still detected in many products. Kotthoff et al. (2015) similarly reported broad detection of PFOA in a 2010 sampling effort that collected 115 European consumer products, including carpets, leather, outdoor textiles, cooking materials, and others. PFOA was detected in all but one sample type, often at the highest median concentration compared to other PFCAs. The product samples with the highest concentrations of PFOA included ski wax (median concentration of 15.5 μg/kg), leather products (median concentration of 12.4 $\mu g/m^2$), and outdoor materials (median concentration of 6 $\mu g/m^2$). PFOA has also been detected in textile samples of outdoor apparel from Europe and Asia (Gremmel et al., 2016; van der Veen et al., 2020). PFOA was detected in jackets ranging from concentrations of 0.02–4.59 $\mu g/m^2$ (Gremmel et al., 2016). Interestingly, the level of almost all individual PFAS, including PFOA, and total PFAS increased when the textiles were subjected to weathering (i.e., increased ultraviolet [UV] radiation, temperature, and humidity for 300 hours to mimic the average lifespan of outdoor apparel) (van der Veen et al., 2020).

6.2.4 Indoor Dust

Several studies suggest that PFOA and its precursors in indoor air and/or house dust may be an important exposure source for some individuals (Shoeib et al., 2011; Schlummer et al., 2013; Gebbink et al., 2015; Poothong et al., 2020). PFOA is generally a dominant ionic PFAS constituent in indoor air and 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).

PFOA was measured at the highest concentrations (geometric mean concentrations ranging from 41.4–45.0 ng/g) and frequencies (ranging from 89%–91% detected) in dust sampled from Californian households (Wu et al., 2015). Similarly, PFOA was found at the second highest levels (mean concentration of 1.98 ng/g) of 15 PFAS measured in dust samples taken from households in Seoul, South Korea (Kim et al., 2019). PFOA was detected in all dust samples from that study. Makey et al. (2017) measured PFOA and PFOA precursors in dust and found weak correlations between concentrations in dust and serum PFOA concentrations in pregnant Canadian participants. One study in Alaska Natives found no correlation between dust and serum PFOA concentrations (Byrne et al., 2017).

6.2.5 Ambient Air

Perfluoroalkyl chemicals have been found in ambient air globally, with the highest concentrations observed or expected in urban areas and nearest to industrial facilities, areas where AFFF firefighting foams are used, wastewater treatment plants, waste incinerators, and landfills (Ahrens et al., 2011b). Perfluorinated acids were measured in Albany, New York air samples (gas mean concentration of 3.16 pg/m^3 and particulate phase mean concentration of 2.03 pg/m^3) (Kim and Kannan, 2007). In Minneapolis, Minnesota, PFOA in the particulate phase ranged from 1.6 pg/m^3 to 5.1 pg/m^3 and from 1.7 pg/m^3 to 16.1 pg/m^3 in the gas phase (MPCA, 2008). Even remote areas far from urban centers have previously reported PFOA concentrations in air samples; PFOA has been detected in Resolute Bay, Nunavut, Canada (Stock et al., 2007), as well as other Arctic environments (Butt et al., 2010).

The EPA's Toxics Release Inventory reported release data for PFOA in 2022 (EPA, 2024o). PFOA is not listed as a hazardous air pollutant under the Clean Air Act (EPA, 2024p). However, two states (New York and Michigan) have set enforceable air emissions limits. Ambient air is a possible source of exposure to PFOA for the general population; however, the contribution of air to total exposure is likely low. For example, De Silva et al. (2021) estimated that less than 1% of PFOA exposure to humans in the United States is from inhalation.

6.2.6 Summary and Recommended RSC for PFOA

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 PFOA (EPA, 2000a; see Figure 2).

To identify the population(s) of concern (Box 1, Figure 2), the EPA first identified potential subpopulations or life stages based on the PFOA exposure interval in the critical studies from which the critical effect was selected for RfD derivation in the PFOA toxicity assessment (EPA, 2024d). Since the critical effects are the most sensitive adverse health effects that were identified from the available data of sufficient quality, then the exposure intervals may be sensitive windows of exposure. Three co-critical effects were identified for PFOA in three human epidemiological studies (decreased serum anti-tetanus and anti-diphtheria antibody concentrations in children, decreased infant birth weight, and increased total cholesterol in adults); however, the specific critical windows of exposure for each of the critical effects is not known. However, based on epidemiological study design, potentially sensitive life stages include women of childbearing age who may be or become pregnant, pregnant women and their developing fetuses, lactating women, and early childhood. Limited information was available regarding specific PFOA exposure in these life stages from different environmental sources. Therefore, the EPA considered exposures in the general U.S. population, ages 21+, which includes some of these potentially sensitive life stages (i.e., women of childbearing age, pregnant women and their developing fetuses, and lactating women).

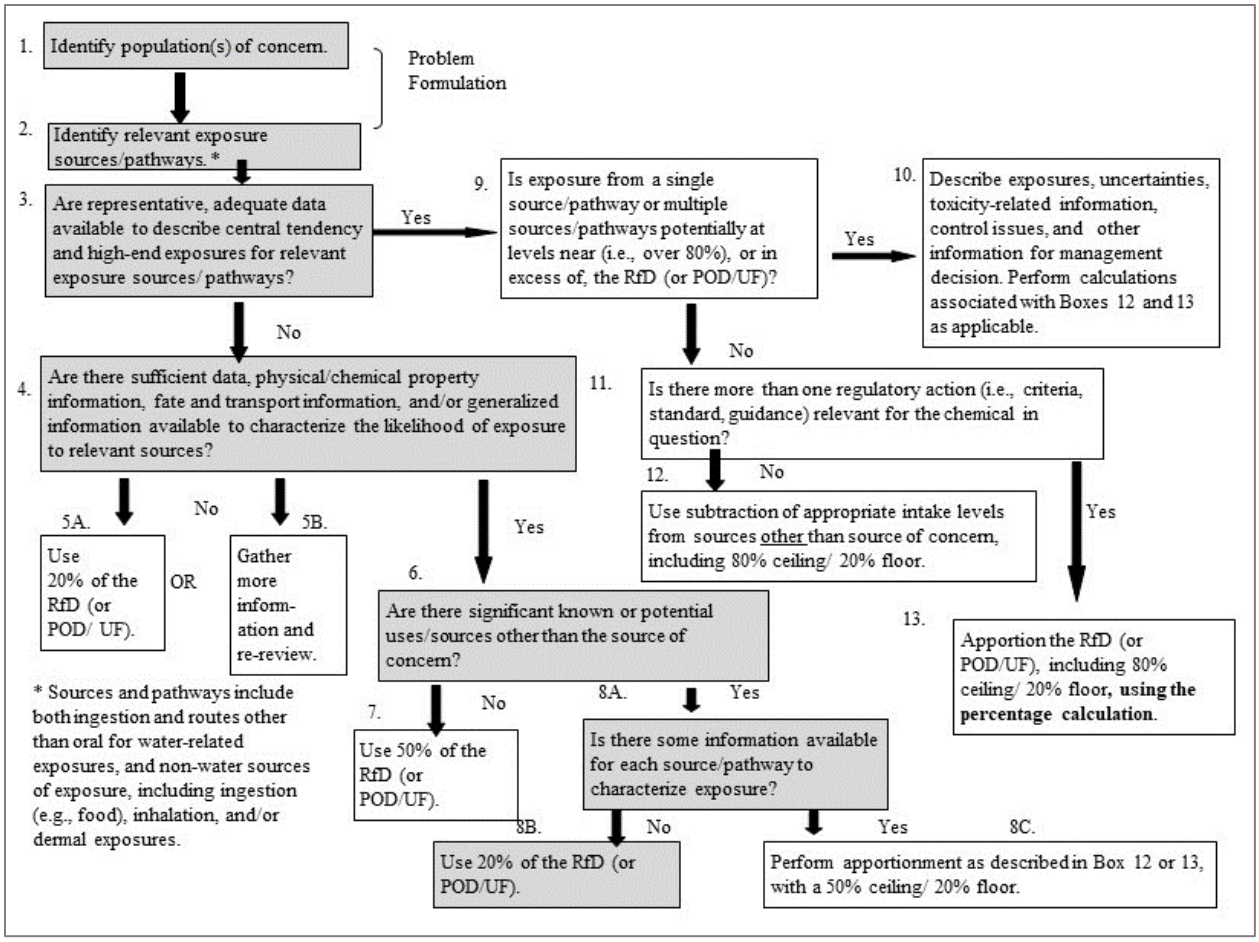

Figure 2. RSC exposure decision tree framework for PFOA; figure adapted from EPA (2000a) with gray boxes indicating key decision points for this chemical.

Second, the EPA identified PFOA-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 of the potentially significant sources. For example, studies from the United States, Canada and Europe indicate that consumer products may be significant sources of exposure to PFOA. Although several studies report PFOA detections in consumer products, most examined very few samples (i.e., n = 1–5) of only a few types of media. 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.

Fourth, the agency determined whether there were sufficient data, physical/chemical property information, fate and transport information, and/or generalized information available to characterize the likelihood of exposure to relevant sources (Box 4, Figure 2). Sufficient information for PFOA was available to characterize the likelihood of exposure. To determine if there are potential uses/source of PFOA other than AWQC-related sources (Box 6, Figure 2), the agency relied on the studies summarized in Section 6 (this document). There are potential other uses/sources of PFOA. PFOA has been detected in soils, dust in carpets and upholstered furniture in homes, offices, and vehicles. Incidental exposure from soils and dust is an important exposure route, particularly for small children because of their increase level of hand-to-mouth behaviors compared to adults. Also, the levels in soils and surface waters can affect the concentrations in local produce, meat/poultry, dairy products and particulates in the air. Based on this information, the next step was to determine if adequate information was available on PFOA 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. 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% (EPA, 2000a). Thus, the EPA recommends an **RSC of 20% (0.20)** for PFOA (Box 8b, Figure 2) for both the water plus organism AWQC as well as the organism only AWQC.

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 PFOA 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 PFOA.

Input Parameter		Value
RfD		0.00000003 mg/kg-d
CSF		29,300 $[mg/kg\text{-}d]^{-1}$
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	22 L/kg
	TL 3	49 L/kg
	TL 4	31 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:

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

$$= \frac{0.00000003\ \text{mg/kg-d} \times 0.20 \times 80.0\ \text{kg} \times 1{,}000\ \mu g/mg}{2.3\ \text{L/d} + ((0.0076\ \text{kg/d} \times 22\ \text{L/kg}) + (0.0086\ \text{kg/d} \times 49\ \text{L/kg}) + (0.0051\ \text{kg/d} \times 31\ \text{L/kg}))}$$

$$= 0.0001575\ \mu g/L$$

$$= 0.0002\ \mu g/L \text{ (rounded)}$$

For consumption of organisms only:

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

$$= \frac{0.00000003\ \text{mg/kg-d} \times 0.20 \times 80.0\ \text{kg} \times 1{,}000\ \mu g/mg}{(0.0076\ \text{kg/d} \times 22\ \text{L/kg}) + (0.0086\ \text{kg/d} \times 49\ \text{L/kg}) + (0.0051\ \text{kg/d} \times 31\ \text{L/kg})}$$

$$= 0.0006428\ \mu g/L$$

$$= 0.0006\ \mu g/L \text{ (rounded)}$$

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; EPA, 2000d). Since PFOA was determined to be *Likely to Be Carcinogenic to Humans* (EPA, 2024b,c), the EPA derived AWQC for carcinogenic toxicological effects.

Consumption of water and organisms:

$$\text{AWQC} = \frac{\text{RSD} \times \text{BW} \times 1{,}000^{f}}{\text{DWI} + \sum_{i=2}^{4} (\text{FCR}_i \times \text{BAF}_i)}$$

$$= \frac{(10^{-6} / 29{,}300)\ \text{mg/kg-d} \times 80.0\ \text{kg} \times 1{,}000\ \mu g/mg}{2.3\ \text{L/d} + ((0.0076\ \text{kg/d} \times 22\ \text{L/kg}) + (0.0086\ \text{kg/d} \times 49\ \text{L/kg}) + (0.0051\ \text{kg/d} \times 31\ \text{L/kg}))}$$

$$= 0.000000896175\ \mu g/L$$

$$= 0.00000090\ \mu g/L \text{ (rounded)}$$

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

For consumption of organisms only:

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

$$= \frac{(10^{-6} / 29{,}300)\ \text{mg/kg-d} \times 80.0\ \text{kg} \times 1{,}000\ \mu\text{g/mg}}{(0.0076\ \text{kg/d} \times 22\ \text{L/kg}) + (0.0086\ \text{kg/d} \times 49\ \text{L/kg}) + (0.0051\ \text{kg/d} \times 31\ \text{L/kg})}$$

$$= 0.00000365659\ \mu\text{g/L}$$

$$= 0.0000036\ \mu\text{g/L (rounded)}$$

7.3 AWQC Summary for PFOA

The EPA derived the draft recommended AWQC for PFOA using both noncarcinogenic and carcinogenic toxicity endpoints. The human health AWQC for noncarcinogenic effects for PFOA are **0.0002 µg/L** (0.2 ng/L) for consumption of water and organisms and **0.0006 µg/L** (0.6 ng/L) for consumption of organisms only for the general population (≥ 21 years old) (Table 4). The EPA also evaluated the use of exposure factors relevant to sensitive subpopulations based on the critical effect(s) used to derive the noncarcinogenic RfD (Appendix B). For children 1 to < 3 years old, the criteria calculated for illustrative purposes based on noncarcinogenic effects are slightly lower than for the general population (≥ 21 years old), 0.0001 µg/L (0.1 ng/L) for consumption of water and organisms and 0.0005 µg/L (0.5 ng/L) for consumption of organisms only. The human health AWQC for carcinogenic effects (at a 10^{-6} cancer risk level) for PFOA are **0.00000090 µg/L** (0.0009 ng/L) for consumption of water and organisms and **0.0000036 µg/L** (0.0036 ng/L) for consumption of organisms only (Table 4). The EPA recommends the lower AWQC, based on the carcinogenic effects of PFOA, as the national recommended human health AWQC because they are protective of the general population, including potentially sensitive subpopulations.

Under the EPA's recently finalized Method 1633 (EPA, 2024q) 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 PFOA. The pooled MDL for PFOA is 0.54 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, 2024q). 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 PFOA chemicals.

	Human Health AWQC for Carcinogenic Effects
Water and Organism	0.00000090 µg/L (0.0009 ng/L)
Organism Only	0.0000036 µg/L (0.0036 ng/L)

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

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, 2024r). 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, 2000c). 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, 2024s). 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, 2024r). 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, 2024r), 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.

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

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

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

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

- Perfluorooctanoic Acid (PFOA) (CASRN 335-67-1)
- PFOA
- 2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-pentadecafluorooctanoic acid
- pentadecafluoro-1-octanoic acid
- pentadecafluoro-n-octanoic acid
- octanoic acid, pentadecafluoro-

- perfluorocaprylic acid
- pentadecafluorooctanoic acid
- perfluoroheptanecarboxylic acid

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Appendix A: Bioaccumulation Factor (BAF) Supporting Information

BAF Calculation Description for PFOA

The 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 PFOA based on chemical's properties (e.g., ionization, hydrophobicity), metabolism, and biomagnification potential (see Figure 1 this document). The EPA followed the guidelines provided in Section 5.5 of the EPA's 2000 Methodology (EPA, 2000), to assess the occurrence of cationic and anionic forms of PFOA at typical environmental pH ranges. Based on the dissociation constant (pK_a) information provided in the Hazardous Substance Data Bank (HSDB) for PFOA, it was determined that ionization of PFOA was significant at typical environmental pH ranges (NCBI, 2023; EPA, 2024a,b).

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 PFOA. Based on information in the peer-reviewed literature, it was determined that biomagnification of PFOA was unlikely (Houde et al. 2011; Du et al., 2021; Munoz et al., 2022). Based on the characteristics of PFOA, EPA selected Procedure 5 for deriving national BAF values for this chemical.

As described in Section 4.2.1, for a given procedure, the 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 laboratory 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 PFOA from the peer-reviewed literature sources for which sufficient information was provided to determine the quality and usability of the data. Therefore, the EPA used the Field BAF method (EPA, 2003) to derive the national BAF values for this chemical.

Calculating Baseline BAFs

The EPA calculated baseline BAFs for PFOA using a procedure analogous to the baseline BAF calculation for nonionic organic chemicals to account for the physical and chemical properties of PFOA. 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 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 peer-reviewed literature; these BAF data converted from total to dissolved

were 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 ATSDR (2021), the partitioning of PFOA is related to protein binding properties (ATSDR, 2021). The EPA considered protein-normalizing 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 PFOA to various proteins in aquatic organisms. Because of this lack of data on the relationship between protein content and PFOA bioaccumulation, attempts to normalize BAFs based on protein content would likely introduce greater uncertainty into BAF averages.

Consistent with the 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 PFOA. As described in the 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 the 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 unique physical-chemical properties of PFOA, K_{ow} cannot be reliably measured for these compounds and therefore cannot be used to estimate K_{poc} or K_{doc} (ATSDR, 2021).

Using the K_{oc} information in Higgins and Luthy (2006), the 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 PFOA to DOC, nor on the bioavailability of PFOA partitioned to DOC. In addition, Higgins and Luthy (2006) included DOC-bound PFOA in the aqueous phase of their calculations. Thus, the amount of PFOA 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 (EPA, 2000)) is used in baseline BAF calculations, unless this value is reported in the BAF source.
- K_{oc} = PFOA log K_{oc} = 2.06 (Higgins and Luthy, 2006).

Because the measured BAFs for PFOA 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 the EPA's 2000 Methodology [EPA, 2000]) is adjusted (as shown below in Equation 2) to determine the freely dissolved concentration of PFOA BAFs in water:

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

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

Dissolved PFOA Baseline BAFs

The EPA included results from several field BAF studies for PFOA reported as dissolved (i.e., filtered) concentrations in its baseline BAF calculations. Because these dissolved PFOA data are presumed to represent the freely-dissolved (non-particulate) fraction, the ffd term in Equation 2 is set to 1. Also, as described above, the measured BAFs for PFOA are not being adjusted for lipid or protein content to calculate baseline BAFs for PFOA. Thus, Equation 3 is used to calculate the freely dissolved concentration of PFOA for "baseline BAFs" using field-measured dissolved PFOA 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 PFOA. Equation 4 (an equation analogous to the equation used for nonionic organic chemicals for calculating national BAFs (see Equation 5-28 on Page 5-42 of the EPA's 2000 Methodology (EPA, 2000)) is used to convert the baseline BAF to a national BAF for each trophic level:

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

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 PFOA, 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:

$$\text{National BAF PFOA}_{(\text{TL 2})} = [(21.2)_{\text{TL 2}} + 1] \times (0.999942596)$$

= 22.2 L/kg

= 22 L/kg (rounded)

$$\text{National BAF PFOA}_{(\text{TL 3})} = [(47.9)_{\text{TL 3}} + 1] \times (0.999942596)$$
= 48.9 L/kg
= 49 L/kg (rounded)

$$\text{National BAF PFOA}_{(\text{TL 4})} = [(30.0)_{\text{TL 4}} + 1] \times (0.999942596)$$
= 30.9 L/kg
= 31 L/kg (rounded)

The corresponding values for TL 2, TL 3 and TL 4 were computed as 22.2 L/kg, 48.9 L/kg and 30.9 L/kg, respectively. Rounding the values to two significant figures yields national BAF values of 22, 49 and 31 L/kg for TLs 2, 3, and 4, respectively.

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Appendix B: Comparative Analysis for Potentially Sensitive Populations for PFOA

The EPA evaluated several exposure scenarios for PFOA to determine whether the national recommended criteria based on carcinogenic effects are sufficiently protective of potentially sensitive subpopulations related to the noncancer health effects. To accomplish this, the EPA considered four 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 four potentially sensitive life stages associated with the critical effects used to derive the PFOA chronic RfD, i.e., co-critical effects of decreased serum anti-tetanus and anti-diphtheria antibody concentrations in children (PFOA concentration measured at age 5 and antibody concentration measured at age 7), decreased infant birth weight, and increased total cholesterol in adults. 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, pregnant women, lactating women, and early childhood (ages 1 to < 3 years and 3 to < 6 years) (EPA, 2024; Table B-1, this document). The age ranges for early childhood were selected because they are relevant to the exposure in the critical study (e.g., children were exposed through infancy to age five) and based on data availability (e.g., trophic level specific fish consumption rates).

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). Representative body weights for the "all ages" scenario and lactating women populations were 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) (Table 8-3; EPA, 2011). 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 (Table 8-5; EPA, 2011). Using these data, the EPA assumed a time-weighted average body weight of 73.4 kg for women of childbearing age (Table 8-5; EPA, 2011). The EPA also used this body weight for women of childbearing age as a proxy for lactating women, in the absence of other data. For children 1 to < 3 years, an average bodyweight of 11.4 kg for children 1 to < 2 years was used as a proxy for children 1 to < 3 years (EPA, 2011, Table 8-1). For children 3 to < 6 years, a mean of 18.6 kg was used (EPA, 2011, Table 8-1).

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

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]	0.0002	0.0006
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]	0.0002	0.0009
Children 1 to < 3 years	11.4[f]	0.507[g]	4.7[h]	1.2[h]	1.4[h]	1.2[h]	0.0001	0.0005
Children 3 to < 6 years	18.6[i]	0.588[j]	5.8[h]	1.7[h]	2.5[h]	1.1[h]	0.0001	0.0006
All Ages (Birth to 80 years)	71.6[k]	2.0[b]	19.3[l]	NA	NA	NA	ND	ND
Pregnant Women	75[m]	2.1[e]	10[n]	NA	NA	NA	ND	ND
Lactating Women	73.4[d]	2.7[e]	7.2[o, p]	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.

[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] EPA, 2011, *Exposure Factors Handbook*, Ch. 8, Table 8-1, NHANES 1999–2006. Recommended mean bodyweight ages 1 to < 2 years.

[g] Estimated using the FCID calculator (University of Maryland, 2024; https://fcid.foodrisk.org/), NHANES 2005–2010, community water, 90th percentile per capita rate, age 1 to < 3 years.

[h] EPA, 2014. NHANES 2003–2010 survey data, 90th percentile per capita rate, freshwater and estuarine fish and shellfish edible portion, Tables 27a, 28a, 29a, ages 1 to < 3 years.

[i] EPA, 2011, *Exposure Factors Handbook*, Ch. 8, Table 8-1, NHANES 1999–2006. Recommended mean bodyweight ages 3 to < 6 years.

[j] Estimated using the FCID calculator (University of Maryland, 2024; https://fcid.foodrisk.org/), NHANES 2005–2010, community water, 90th percentile per capita rate, ages 3 to < 6 years.

[k] 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).

[l] 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.

[m] EPA, 2011, *Exposures Factors Handbook*, Ch 8, mean, NHANES 1999–2006, Table 8-29

[n] 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.

[o] 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, breastfeeding females only.

[p] Estimates are less statistically reliable based on guidance published in the Joint Policy on Variance Estimation and Statistical Reporting Standards on NHANES III and CSFII Reports.

childbearing age and children were identified (Table B-1). However, trophic level specific FCRs are not available for three of the potentially sensitive life stages—all ages, pregnant women, or lactating women. Therefore, criteria could not be calculated for these three life stages. However, in these cases with available data, the total FCR for the alternative scenarios is lower than the FCR for the general population. Because bodyweights for all ages, pregnant women, and lactating women are similar to the general population (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.

For illustrative purposes, the EPA calculated noncancer-based criteria based on the exposure parameters for women of childbearing age, children ages 1 to < 3 years, and children ages 3 to < 6 years. As demonstrated in Table B-1, criteria based on the exposure inputs for children 1 to < 3 years result in a slightly more health protective noncancer criteria as compared to the general population; however, the national recommended criteria for PFOA (0.00000060 µg/L W + O and 0.0000036 µg/L OO) are based on the carcinogenic toxicological endpoint (CSF), which results in the most health protective criteria overall. Therefore, the criteria based on carcinogenic effects of PFOA is protective of the noncancer-based criteria derived for the potentially sensitive populations and life stages.

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