

# Groundwater and PFAS: State of Knowledge and Practice

## Human and Ecological Impacts Section 3

One of eight sections prepared by National Ground Water Association volunteers. Each section was prepared to stand independently, or to be integrated with the other seven sections.



## Human and Ecological Impacts

### INTRODUCTION

Poly- and perfluoroalkyl substances—collectively referred to as PFAS—are terms used to describe a group of organic fluorinated alkanes.<sup>1</sup> In the perfluoroalkyls, all hydrogen atoms attached to carbons in the aliphatic chain have been replaced by fluorines.<sup>2</sup> The polyfluoroalkyls have at least one hydrogen on the alkane that remains unfluorinated (Buck et al. 2011). The six PFAS addressed in this section are categorized as either perfluoroalkyl carboxylic acids (PFCAs) or perfluorosulfonic acids (PFSAs). The PFCAs perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), and perfluoroheptanoic acid (PFHpA) are perfluoroalkyls that contain a fully fluorinated carbon chain of seven, eight, or six carbons, respectively, and a terminal carboxylic acid functional group.<sup>3</sup> The PFSAs perfluorooctane sulfonate (PFOS), perfluorohexane sulfonate (PFHxS), and perfluorobutane sulfonate (PFBS) contain a perfluorinated alkyl carbon chain of eight, six, and four carbons, respectively, and a terminal sulfonate or sulfonic acid group. These six PFAS were selected based on their inclusion in the USEPA's Third Unregulated Contaminant Monitoring Rule (UCMR3), a program conducted under the Safe Drinking Water Act (SDWA) to develop information on the occurrence of unregulated contaminants in public water systems (USEPA 2012a).

Table 3.1 provides a list of the PFAS addressed in this section: their acronyms, chemical abstract service (CAS) registry numbers, and molecular formulas.

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### PHYSICAL AND CHEMICAL PROPERTIES

Many of the chemical and physical properties of PFAS that have made them commercially valuable as surfactants also determine their potential for partitioning, persistence, and accumulation in the environment and in biota. The PFAS listed in Table 3.1 have low pKa's<sup>4</sup>, and as a result, these PFAS tend to exist in their ionic (charged) forms in environmental and biotic media (Conder et al. 2008; CONCAWE 2016). These characteristics also contribute to their relatively high aqueous solubility. The fluorinated carbon chain of the PFCAs and PFSAs is hydrophobic and lipophobic, and in combination with the carboxylate and sulfonate functional groups, imparts amphiphilic and surfactant characteristics to these compounds.<sup>5</sup>

Because of the amphiphilic properties of many PFAS, they do not preferentially partition to lipids, but instead tend to bind to proteins. In humans, the highest concentrations have been detected in serum and liver, and to a lesser extent, the kidney and other organs. The carbon-fluorine bond is extremely strong and renders PFAS resistant to biotic and abiotic degradation; PFAS are not metabolized (ECHA 2013; CONCAWE 2016).

<sup>1</sup> An alkane is a molecule in which all of the carbon-carbon bonds are single. An alkyl is an alkane where at least one hydrogen has been replaced by a non-hydrogen atom or molecule. An alkyl has the general formula  $C_nH_{2n+1}$ ; the perfluoroalkyls have the basic formula  $C_nF_{2n+1}$  (Buck et al. 2011).

<sup>2</sup> Except those hydrogen atoms whose substitution would modify the nature of any functional groups present (Buck et al. 2011).

<sup>3</sup> A carboxylic acid functional group has the molecular formula  $COOH$ . A sulfonic acid functional group has the molecular formula  $SO_3H$ .

<sup>4</sup> The pKa is a measure of the tendency of an acid (in this context, the perfluorocarboxylic and perfluorosulfonic acids) to dissociate in solution (i.e., the aqueous environment). In general, a low pKa indicates that an acid dissociates fairly readily to form the corresponding positively and negatively charged chemical species. For example, PFOA will dissociate to form  $F(CF_2)_7COO^-$  and  $H^+$ .

<sup>5</sup> An amphiphilic compound has both hydrophilic and lipophilic properties, and as a result, does not fully solubilize in either medium. In biological systems, these compounds tend to partially partition to both aqueous and lipid compartments.

**Table 3.1. Name, acronyms, and molecular formulas of PFAS.**

Name	Acronym	CAS Registry Number	Molecular Formula
<b>Perfluoroalkyl Carboxylic Acids</b>			
<b>PFCAs</b>			
Perfluoroheptanoic Acid	PFHpA	375-85-9	F(CF <sub>2</sub> ) <sub>6</sub> COOH
Perfluorooctanoic Acid	PFOA	335-67-1	F(CF <sub>2</sub> ) <sub>7</sub> COOH
Perfluorononanoic Acid	PFNA	375-95-1	F(CF <sub>2</sub> ) <sub>8</sub> COOH
<b>Perfluoroalkyl Sulfonates/ Perfluoroalkyl Sulfonic Acids</b>			
<b>PFSA</b> s			
Perfluorobutane Sulfonate	PFBS	375-73-5	F(CF <sub>2</sub> ) <sub>4</sub> SO <sub>3</sub> H
Perfluorohexane Sulfonate	PFHxS	432-50-8	F(CF <sub>2</sub> ) <sub>6</sub> SO <sub>3</sub> H
Perfluorooctane Sulfonate	PFOS	1763-23-1	F(CF <sub>2</sub> ) <sub>8</sub> SO <sub>3</sub> H

### Bioaccumulation

Octanol-water partition coefficients ( $K_{ow}$ ) can be used as a predictor of bioaccumulation, as they provide a measure of the extent to which a chemical partitions between water and lipid. In general, partitioning to lipids is associated with the potential to persist and bioaccumulate, given the limited metabolic capacity and blood flow of lipid tissue. Given the amphiphilic nature of many PFAS, determination of  $K_{ow}$ 's is not experimentally feasible. Thus, while  $K_{ow}$ 's have been calculated for PFAS (see e.g., CONCAWE 2016), it is not clear that they provide a meaningful predictor of the potential of PFAS to accumulate or biomagnify (Conder et al. 2008; ECHA 2013; UNEP 2015; CONCAWE 2016).

The potential for bioaccumulation is typically assessed by empirical measurements of bioconcentration factors (BCFs); bioaccumulation factors (BAFs), biomagnification factors (BMFs), and trophic magnification factors (TMFs). These terms are defined in Appendix 1, which also provides a summary and interpretation of reported values for the PFAS addressed in this section.

The Stockholm Convention Persistent Organic Pollutants Review Committees for PFOS (OECD 2002) and PFOA (UNEP 2015) considered the weight of evidence sufficient to support conclusions that both PFOS and PFOA are bioaccumulative. Accumulation of PFNA in wildlife is well-documented (see section on Exposure of Ecological Receptors). Although there are little to no empirical data on the potential bioaccumulation of PFBS, PFHpA, and PFHxS, a review of published values indicates that in general, BAFs and BCFs increase with the number of fluorinated carbons (Conder et al. 2008).

### HUMAN EXPOSURE

As previously discussed, PFAS, including PFOA and PFOS, are often characterized as persistent since they are extremely resistant to typical environmental degradation processes. They do not hydrolyze, photolyze, or biodegrade under environmental conditions (USEPA 2012b), resulting in widespread human exposure.

Human exposure to these chemicals can occur through the following pathways:

- Ingestion of PFAS-containing food, either directly contaminated (food grown on contaminated soils, fish caught from contaminated waters) or indirectly (food contaminated from PFAS-treated food paper wrappings such as pizza boxes, sandwich wrappers, popcorn bags, etc.)
- Ingestion of PFAS in drinking water
- Occupational exposure
- Direct contact through the use of consumer products containing PFAS
  - Teflon
  - Stainmaster carpets
  - Scotchgard
  - Gore-Tex
  - Personal care products (e.g., dental floss, cosmetics)
- Inhalation of ambient air and dust, or contact with PFAS-contaminated soils.

Currently, the relative importance of the human routes of exposure to these compounds is not well established.

Due to the ubiquitous use of PFAS in a variety of products and industrial applications, it is estimated that PFAS exposure is widespread throughout the U.S. population. Multiple biomonitoring studies have

estimated that greater than 95% of the general population have been exposed to PFAS (CDC 2009; Olsen et al. 2005, 2007, 2008, 2012; Biomonitoring California 2016).

In 2002, 3M Company, a principal manufacturer of PFOS at that time, discontinued production of PFOS and related chemicals. In 2006, the USEPA launched the PFOA Stewardship Program. The goals of the program for member companies were to commit to a 95% reduction in emissions of PFOA, precursor chemicals, and product content levels and to work toward eliminating these chemicals from emissions and products by 2016. Under the Stewardship Program and through the Toxic Substances Control Act (TSCA), as of 2015 EPA will have removed or restricted approximately 300 distinct PFAS from the market (USEPA 2016a).

Calafat et al. (2007) and Olson et al. (2005, 2007, 2008, 2012) have evaluated the temporal trend of PFAS concentrations in blood plasma. Data from the NHANES study (Calafat et al. 2007) and from Olson and coworkers' evaluation of PFAS serum concentrations of adult American Red Cross blood donors have shown that serum concentrations of PFOS, PFOA, and PFHxS have declined following reduction of PFOS and related chemical production. Olsen et al. (2012) determined the decline in PFOS serum concentrations suggest a population halving time of 4.3 years which is generally consistent with a PFOS excretion half-life of 4.5–7.4 years (Harada et al. 2005).

A discussion of the potential routes of human exposure and the relative importance of each route follows in the discussion below.

### Domestic Water Use

PFOS and PFOA have been widely detected in surface water including rivers, lakes, and streams (Boulanger et al. 2004, 2005; Kim and Kannan 2007; Nakayama et al. 2007; Lasier et al. 2011), and in groundwater in the United States (USEPA 2017, NJDWQI 2016; ATSDR 2016a). PFHpA and PFHxS were commonly detected in the few studies that analyzed surface water for these compounds (Kim and Kannan 2007; Nakayama et al. 2007; Simcik and Dorweiler 2005). Reported concentrations of PFAS in surface water samples are generally below 50 nanograms (ng)/L (ATSDR 2015).

Surface water contamination can occur as a result of wastewater discharge from PFAS manufactur-

ing (Davis et al. 2007; USEPA 2008), from municipal wastewater facilities (Boulanger et al. 2005; Sinclair and Kannan 2006), through the use of PFAS containing firefighting foam at military installations and firefighting training facilities (Moody and Field 1999; Moody et al. 2003), and through the land application of biosolids contaminated with PFAS (ATSDR 2016a; Hurley et al. 2016).

The USEPA uses the Unregulated Contaminant Monitoring Rule (UCMR) program to collect data for contaminants suspected to be in drinking water, but do not yet have health-based standards set under the Safe Drinking Water Act (SDWA). There is less information regarding the presence of other non-UCMR PFAS in surface water. The USEPA develops a new list of UCMR contaminants every five years. Under the third revision to UCMR (UCMR3), the USEPA began testing public water supplies (PWSs) in 2013 for six PFAS; the agency's data provides the most comprehensive population-based data set of PFAS occurrence in drinking water. Table 3.2 summarizes the UCMR data as of January 2017.

In 1035 samples of raw and finished PWS water in New Jersey, PFOA was the most frequently detected PFAS. It was detected in 65% of 72 public water supplies, with the highest detection in finished water of 100 ng/L, and at least one sample with concentrations exceeding 40 ng/L in 17% of the public water supplies tested (NJDWQI 2016).

One of the most robust studies investigating PFAS exposure due to groundwater contamination is the C8 Health Project which was created, authorized, and funded as part of the settlement agreement reached in the case of *Jack W. Leach et al. v. E.I. du Pont de Nemours & Company* (no. 01-C-608 W.Va., Wood County Circuit Court, filed 10 April 2002). Industrial discharges of PFOA to the atmosphere with subsequent deposition and leaching into groundwater as well as direct releases into the Ohio River led to groundwater contamination over six water districts (Frisbee et al. 2009). The C8 Health Project published a series of Probable Link Reports which, based upon the available scientific evidence, linked PFOA exposure to a number of human diseases including diagnosed high cholesterol, testicular and kidney cancer, ulcerative colitis, thyroid disease, pregnancy-induced hypertension, and reduced antibody titer rise (C8 Science Panel 2013; Looker et al. 2014).

**Table 3.2. Summary of UCMR3 data<sup>1</sup> (January 2017).**

Contaminant	MRL (µg/L)	# PWSs with Results > MRL	HA (µg/L)	# PWSs with Results > HA
PFOA	0.02	117/4920	0.07	13
PFOS	0.04	95/4920	0.07	46
PFNA	0.02	14/4920	No Value	Not Applicable
PFHxS	0.03	55/4920	No Value	Not Applicable
PFHpA	0.01	86/4920	No Value	Not Applicable
PFBS	0.09	8/4920	No Value	Not Applicable

<sup>1</sup> January 2017 UCMR3 Data Summary for Chemical Contaminants, USEPA 2017.

MRL = UCMR Minimum Reporting Level; HA = Health Advisory

### Drinking Water

Biomonitoring studies have shown drinking water can be a significant source of exposure among populations whose water is impacted with PFAS contamination (Frisbee et al. 2009; Hoffman et al. 2011; ATSDR 2013; MN DOH 2009; NH DHHS 2016), often resulting in PFAS serum concentrations above those measured in the general population (CDC 2009). Frisbee et al. (2009) and Hoffman et al. (2011) conducted biomonitoring as part of the C8 Health Project. Frisbee and co-workers evaluated data for 66,899 study participants and found that four PFAS (PFHxS, PFOS, PFOA, PFNA) were detectable in >97% of the serum samples. The geometric mean PFOA serum concentrations were approximately 8.5 times higher in the C8 study population as compared to the NHANES study population. Concentrations of PFHxS, PFOS, and PFNA were comparable between the two groups (Frisbee et al. 2009; CDC 2009). In a subset of the C8 Study Population, Hoffman and coworkers used a pharmacokinetic model to evaluate the relationship between drinking water and serum PFOA levels. The authors concluded that for each 1 µg/L increase in PFOA water concentration, there was a 141.5 µg/L (95% confidence interval, 134.9–148.1) increase in serum concentration. The steady-state serum:drinking water ratio from the pharmacokinetic model was calculated to be 114 (Hoffman et al. 2011).

Less is known however, as to whether drinking water is a significant route of PFAS exposure among the general population. Hurley et al. (2016) determined there was an association between PFOS and PFOA serum concentrations in women and concentrations of these compounds in public drinking water supplies. Study participants were linked to UCMR3

public water drinking supply PFAS results through residential zip code. It was determined that 40% of the combined concentrations of PFOS and PFOA exceeded the USEPA Drinking Water Health Advisory (HA) of 0.07 µg/L (USEPA 2016c, d). The investigators found median serum concentrations of PFOS and PFOA were 29% and 38% higher, respectively, among those with detectable levels of PFOS and PFOA in water compared to those without detectable levels.

### Showering/bathing

**Dermal:** Studies have shown very limited absorption of PFAS through the skin, and thus routine showering or bathing will not likely cause significant exposure to PFAS (ATSDR 2016b; MDDOH 2016). Although there is experimental evidence PFOA is dermally absorbed in experimental animals (Kennedy 1985; O'Malley and Ebbens 1981), it is not expected to be a significant route of exposure (Fasano et al. 2005). There are little to no data available on the dermal absorption of other PFAS but they are expected to be similar to PFOA.

**Incidental Ingestion:** There is no information regarding exposure to PFAS through incidental ingestion during showering, bathing, or brushing teeth. It has been recommended by various state agencies to avoid ingestion of PFAS-contaminated water during these activities in order to reduce exposure. However, the incidental ingestion of PFAS during showering, bathing, or brushing teeth is not expected to be a significant source of exposure due to the negligible amount of water ingested (NJDOH 2016; AKDHHS 2015; MNDOH 2016; VT ANR DOH 2016).

**Recreational Exposure:** There is no information regarding exposure to PFAS through incidental

ingestion or dermal exposure during recreational activities such as swimming. However, the incidental ingestion of PFAS during recreational activities such as swimming is not expected to be a significant source of exposure due to the negligible amount of water ingested. As discussed in the previous section, dermal absorption is also expected to be negligible and insignificant.

**Food:** Food is a significant source of exposure to PFAS (Trudel et al. 2008; ATSDR 2015; Lindstrom et al. 2011; Vestergren and Cousins 2009; ASTSWMO 2015), and modeling has indicated that 90% of human exposure to PFOS and PFOA may be through ingestion of contaminated food (Fromme et al. 2009). PFOA has been detected in a variety of foods including snack foods, vegetables, meat, dairy products, human breast milk, and fish. PFOS has been detected in eggs, milk, meat, fish, root vegetables, and human breast milk. Occurrence in food products can result from the use of contaminated water in processing and preparation; growth of food in contaminated soils (e.g., in areas that receive biosolids soil amendments from wastewater treatment plants); direct and indirect exposures of domestic animals to PFAS from drinking water and consumption of feed crops grown in contaminated soil; fish tissue bioaccumulation from contaminated waterways; and food packaging materials containing PFAS (Hag et al. 2011; Lindstrom et al. 2011; Sepulvado et al. 2011; Venkatesan and Halden 2013; Armstrong et al. 2016; ATSDR 2015; Fornis et al. 2015; USEPA 2016c, d). Studies have shown breast milk is a significant exposure route for infants to PFAS. Hag et al. (2011) have shown breast milk contributed greater than 94% of total PFOS exposure in 6-month-old infants, and greater than 83% of PFOA exposure.

Some studies suggest the consumption of fish from contaminated waters may be a major source of human exposure to PFOS (Trudel et al. 2008; Fromme et al. 2009; Domingo 2012). PFOS has been shown to bioaccumulate readily in fish, appears to have the highest bioaccumulation potential of the PFAS in food webs, and is the predominant PFAS detected in fish tissue (Houde et al. 2011). In a study of PFAS concentrations in fish and surface water in New York State, although PFOA was typically found at higher concentrations in water than PFOS, concentrations

of PFOS in fish tissue were approximately 9,000-fold greater than the levels in surface water (Sinclair et al. 2006). The USEPA national screening study of PFAS in freshwater fish determined 80% of samples of urban river fish tissue samples and 100% of Great Lake fish tissue samples contained some detectable PFAS. PFOS was the most commonly detected PFAS in both fish populations; the maximum detected PFOS concentrations were 127 ppb and 80 ppb in river fish and Great Lake fish tissues, respectively (Stahl et al. 2014).

## Exposure to PFAS-Containing Products

### Consumer Products

Although not a primary exposure route, use of PFAS-containing consumer products may result in potential exposure. Some examples are listed below (ATSDR 2015; USEPA 2016c, d).

- Stain/water repellants on clothing, bedding materials, upholstered furniture, carpets, and automobile interiors (Walters and Santillo 2006; Lindstrom et al. 2011). These materials can be a particularly important exposure route for infants and children because of their hand-to-mouth behaviors.
  - PFOA — Stainmaster™, Zonyl™, Nuva™, Unidyne™, Baygard™
  - PFOS — Scotchgard™
- Cooking surfaces (e.g., Teflon™) (PFOA)
- Toothpaste and dental floss, shampoos, cosmetics (PFOA)
- Polishes and waxes (PFOA)
- Paints, varnishes, sealants (PFOS, PFOA)
- Food containers and contact paper (PFOA and PFOS are impurities that can be found in some grease-proofing paper coatings [Begley et al. 2005])<sup>6</sup>
- Pesticides (PFOA)
- Textiles (e.g., Gore-Tex™) and leather (PFOS, PFOA)
- Plumbing tape (PFOA)
- Cleaning products (PFOS, PFOA).

### Occupational Exposure

Occupational use of PFAS-containing products may result in potential exposure. Some examples are listed as follows (USEPA 2016c, d).

<sup>6</sup>In January 2016, the Food and Drug Administration amended their food additive regulations to no longer allow for the use of perfluoroalkyl ethyl containing food-contact substances as oil and water repellants for paper and paperboard for use in contact with aqueous and fatty foods (USEPA 2016c, d).

- Metal plating and finishing (continuing use, PFOS)
- Photograph development (continuing use, PFOS)
- Aviation fluids (continuing use, PFOS)
- Lubricants/surfactants/emulsifiers (continuing use, PFOA)
- Aqueous film forming foams (continuing use; used for firefighting) (PFOS, PFOA)
- Semiconductor industry (PFOS)
- Oil and mining (PFOS)
- Electronics (PFOA)
- Flame repellants (PFOS, PFOA).

### Ambient air and dust

PFOA and PFOS are not volatile and airborne fractions are primarily bound to aerosol particles (Trudel et al. 2008; USEPA 2016c, d; ATSDR 2015). These compounds can be released into the atmosphere from fluoropolymer manufacturing and industrial and municipal waste incinerators (ATSDR 2015; USEPA 2016c, d). They can be transported long distances through the atmosphere and have been detected globally at low concentrations (Jahnke et al. 2007). PFOS has been detected in samples collected over the Atlantic Ocean east of southern Africa. Shoeib et al. (2006) detected PFAS concentrations crossing the North Atlantic and the Canadian Arctic Archipelago. Given the widespread commercial and industrial use of PFOA and PFOS and their physical properties, air is a potential source of exposure.

PFAS have been measured in indoor dust in residential, commercial, and office settings because of their use in carpets, textiles, paint, furniture, and other consumer products. Strynar and Lindstrom (2008) detected nine PFAS in indoor dust samples collected from homes and daycare centers in Ohio and North Carolina. PFOS and PFOA were the most commonly detected (94.6% and 96.4% of samples, respectively) with maximum detections of 12,100 ng/g for PFOS and 35,700 ng/g for PFOA. Incidental exposure from indoor dust is an important exposure route, particularly for small children because of their hand-to-mouth behaviors (USEPA 2016c, d).

### Soil

#### Incidental Ingestion

Incidental ingestion of soils represents a potential exposure route for PFAS. There is little information regarding background concentrations of PFAS in soil

(ATSDR 2015) and the one limited study found concentrations to be very low or below detection limits (Washington et al. 2009). Soil contamination tends to occur at manufacturing sites of producers and users of PFAS (3M 2007, 2008) or where disposal of treated products has occurred (e.g., landfills) (USEPA 2016c, d), and potentially where biosolids containing PFAS are applied (Washington et al. 2009; Sepulvado et al. 2011; Venkatesan and Halden 2013; Armstrong et al. 2016). Contaminated soils also can be transported offsite via water and wind.

The use of biosolids as a soil amendment has resulted in the contamination of soils and in the indirect contamination of produce and domestic animal feed grown on contaminated soils when wastewater plants received PFAS-containing industrial waste (USEPA 2011; Yoo et al. 2009; Yoo et al. 2011). PFAS have been detected in biosolids samples (Sepulvado et al. 2011; Venkatesan and Halden 2013; Armstrong et al. 2016). Sepulvado et al. (2011) measured the occurrence and fate of PFAS in soil following the land application of municipal sludge biosolids. PFOS was the dominant PFAS detected in both the biosolids and the amended soils. They also found trace levels of shorter-chain PFAS in soil cores from biosolids-amended soils to depths of 120 cm, suggesting potential movement of these compounds within the soil profile over time and confirming the higher transport potential for short-chain PFAS in soils amended with municipal biosolids. Venkatesan and Halden (2013) and Armstrong et al. (2016) also found PFOS was the most abundant PFAS identified in biosolids. Concentrations in biosolids are expected to decline because of the phaseout of the use of PFOS and PFOA in manufacturing and industrial processes.

#### Dermal

As discussed in the previous sections, the dermal absorption of PFOS is slow and not significant. Although there are little to no data on other PFAS, dermal absorption is also expected to be insignificant.

#### Inhalation of re-suspended soil particulates

There is no information available regarding exposure through the inhalation of re-suspended soil particulates. However, this pathway is not expected to be a relevant exposure pathway to the general population unless located near contaminated soil sites or near agricultural fields where PFAS-contaminated biosolids were applied as a soil amendment.

## Conclusions

As discussed, PFAS have been used in a variety of products and industrial applications, resulting in widespread exposure of the general population. Bio-monitoring studies have estimated more than 95% of the U.S. population have been exposed to PFAS and have measurable concentrations in their blood. However, it should be noted these concentrations have been demonstrably decreased since the discontinuation of production of these chemicals in 2002. The greatest portion of chronic intake is likely from the ingestion of contaminated foods and drinking water. Consumer products, recreational activities, ambient air, and soil exposures contribute to a lesser extent to the total chronic intake. Small children, however, experience higher exposure due to hand-to-mouth transfer of chemicals from treated carpets and indoor dust.

## EXPOSURE OF ECOLOGICAL RECEPTORS

PFAS have been detected in the tissues of invertebrates, fish, birds, and mammals around the globe (Kelly et al. 2009; Butts et al. 2010; Houde et al. 2011; CONCAWE 2016). Much of the published tissue monitoring data are for PFCAs and PFSA in fish-eating Arctic marine mammals (e.g., seals, dolphins, whales) and polar bears. PFOS is the most frequently detected PFAS; has generally been measured at the highest concentrations; and is the dominant PFAS found in all species and locations around the world (Butts et al. 2010; Houde et al. 2011). PFOA, PFHpA, PFNA, and PFHxS have also been detected (Kelly et al. 2009; Houde et al. 2011; CONCAWE 2016). In the Arctic, PFNA (and perfluoroundecanoate) levels are generally second only to PFOS, with elevated levels of other longer-chain PFCAs observed in seabirds (Butts et al. 2010).

PFAS may discharge to the marine environment via groundwater seepage and surface water flow; however, the prevalence of PFOS and other PFAS in Arctic species such as polar bears, seals, and beluga whales is attributed to precursor volatilization and atmospheric degradation to these terminal stable species, followed by deposition to marine and terrestrial environments and food chain transfer (Houde et

al. 2011; Government of Canada 2013).<sup>7</sup> The recent review by Houde et al. (2011) noted PFAS accumulation in wildlife is common; long-chain PFCAs (C7-C12), especially the C8 PFOS and the C9 PFNA, have accumulated in Russian seals, Arctic seabirds, and polar bears. These patterns have been observed in Europe and Asia as well; in general, lower PFAS levels have been measured in wildlife from the Southern Hemisphere.

The direct discharge of PFAS to the ocean from groundwater or surface water is partially mitigated by dilution and dispersion associated with tidal fluctuation, wave action, and currents. Additionally, the salinity of ocean water reduces the solubility of PFAS (USEPA 2014) and promotes its adhesion to sediments (Chen 2012), thus reducing its concentration in marine water. Consequently, emerging surface water criteria for marine waters are less stringent than for freshwater.

## Contamination Profiles

The Canadian government has monitored concentrations of “PFOS and precursors” and “PFCAs and precursors” in Arctic biota since the early 1990s. For PFCAs and precursors (most recently analyzed in 2011), significant increasing trends in concentrations have been documented in lake trout and landlocked char (select locations), and polar bears. For other monitoring locations, these and other species of wildlife exhibited either no significant change, a declining concentration trend, or a significant increase in the early 2000s but with concentrations currently stable or declining. For all monitored species and locations, concentration trends of PFOS and precursors are either stable or declining; no significant concentration increases are currently evident (Government of Canada 2013).

Hong et al. (2014) detected PFAS in fish, bivalves, crabs, gastropods, shrimp, starfish, and polychaetes off the coast of South Korea. Of these species, tissue samples from bivalves, gastropods, shrimp, and fishes exhibited PFOS concentrations in excess of the draft Canadian Federal Environmental Quality Guideline for PFOS in Mammalian Wildlife Diet (i.e., food/tissue) of 4.6 ng/g wet weight (ww) (Environment Canada 2013). Marine gastropods from the Solberg Scandinavian AFFF site in Norway exhibit a PFOS tissue concentra-

<sup>7</sup> Buck et al. (2011) defines PFAS precursors as large functional derivatives and polymers that contain a perfluoroalkyl moiety and degrade in the environment to form PFOS, PFOA, and similar substances that are stable and resistant to further degradation.

tion of 23 ng/g (wet weight) where a stream with a PFOS concentration of 68.8 µg/L discharges to the ocean (Norwegian Pollution Control Authority 2008).

### Wildlife Effects

Wildlife tissue measurements have primarily been made on samples collected from free-ranging wildlife species, with few correlations made between tissue levels and adverse effects. The exceptions to this include Kelly et al. (2009) who calculated nursing beluga whale calves in Hudson Bay were exposed to PFOS via maternal transfer at concentrations ( $2.7 \times 10^{-5}$  to  $8 \times 10^{-4}$  mg/kg-d) that could be toxicologically significant, and Kannan et al. (2006) who established the mean concentrations of PFOA (69 ng/g) and PFOS (65 ng/g) in California sea otters were significantly associated with mortality from disease. Dietary exposures of mallard and quail to PFBS (100–900 mg/kg) had no effect on mortality, organ histology, or reproductive endpoints such as egg production, viability, hatchability, and hatchling success and survival (Newsted et al. 2008). In contrast, PFOS caused mortality when fed to mallards and quail at 50 and 150 mg/kg in the diet, and 10 mg/kg PFOS affected egg hatchability and survival of quail (Newsted et al. 2007). Substantially lower concentrations of PFOS (1 to 5 mg/kg) injected into the eggs of chickens elicited a spectrum of adverse effects including increases in spleen and liver weights, and developmental and immunologic effects such as shorter wings, brain asymmetry, and lowered antibody levels. Average serum PFOS levels (154 ng/g) in these chickens were within levels observed in wild birds, indicating environmental expo-

sure of birds have the potential to cause biologically significant effects.

Marine gastropods and bivalves occur at the marine interface where groundwater and/or surface water discharge to the ocean. As certain PFAS are bioaccumulative, these shellfish are effectively “trap samplers” that can be collected and analyzed for PFAS. They have a limited range and their locations can be mapped. Such samples are not confounded by tides, currents, and wave action that make it difficult to obtain a meaningful and representative open water sample. Moreover, marine gastropods and bivalves are located lower in the food web. Sampling and tissue analysis of marine gastropods and bivalves may help map the nature and extent of PFAS impacts at the marine interface while serving as a screening measure for ecological health.

### TOXICOKINETICS

For the general population, ingestion is likely the primary route by which humans are exposed to PFOA and PFOS (Trudel et al. 2008; Fromme et al. 2009); oral exposure may also be a significant exposure route for PFHpA, PFHxS, PFBS, and PFNA, but this has not been unequivocally established.

Following oral exposure, PFOA, PFOS, PFHxS, and PFBS are absorbed across the gastrointestinal tract and bind to albumin and other serum proteins. PFOA and PFOS are known to distribute from serum to organs via the action of several different classes of transporter proteins. The distribution of these transporter proteins—and thus the distribution of PFAS—varies between organs. The liver typically accumu-

**Table 3.3. Elimination half-lives for PFAS.**

Species	Chemical					
	PFOS	PFOA	PFNA	PFHxS	PFHpA	PFBS
Human	4.5-7.4 years	2.3-10.1 years	2.5-4.3 years (estimated mean)	8.5 years	—	27.7 days
Primates	110-170 days	20.1-32.6 days	—	87-141 days	—	8-95.2 hours
Mice	731-1027 hours	408-456 hours	624-1656 hours	597-732 hours	—	—
Rats	125-1968 hours	1.9-322 hours	58.6-1128 hours	39-688 hours	—	0.64-7.42 hours

Source: PFNA data from Tatum-Gibbs et al. (2011); Ohmori et al. (2003); and NJDWQI (2015). All other values from ATSDR 2015 except for estimated elimination half-life of PFOA in mice (Lau 2015).

lates the greatest concentrations of PFAS, with lesser amounts in the kidneys and other organs (ATSDR 2015; Lau 2015; USEPA 2016f, g).

Due to the strength and stability of the carbon-fluorine bond, PFAS are not metabolized. Their elimination varies with PFAS carbon chain length, species, and gender, with the longest half-lives for PFASs > C4 and PFCAs > C6 in humans. Primates also eliminate PFAS slowly, and rodents tend to clear PFAS more quickly (Table 3.3). In general, males eliminate PFAS more slowly than females. PFAS are eliminated in the urine, with minor amounts also eliminated in the bile. Significant reabsorption from the kidney occurs in humans and primates, and to a much more limited extent in rodents, and is thought to be a key factor in the species-specific differences in elimination half-lives (ATSDR 2015; Lau 2015; USEPA 2016f, g).

## TOXICOLOGICAL EFFECTS OF PFAS

PFOA and PFOS have been linked to a multiplicity of adverse effects including hepatic toxicity, reproductive and developmental toxicity, suppression of the immune system, and cancer. The data for PFNA, PFHxS, and PFBS are much more limited, but suggest these compounds also affect the liver. PFNA caused developmental toxicity that was qualitatively similar to that associated with PFOS and PFOA; available data indicate PFBS and PFHxS do not cause reproductive or developmental effects. PFCAs and PFASs do not appear to be genotoxic or mutagenic (USEPA 2016f, g; Butenhoff et al. 2014).

### Reproductive and Developmental Effects

PFAS cross the placenta of humans and animals, and can also be transferred to offspring during lactation (Karrman et al. 2007; Fenton et al. 2009; Gutzcow et al. 2012).

**PFOA.** PFOA is one of the PFAS known to cross the mammalian placenta, and exposures during gestation have been tied to a range of reproductive and developmental effects (USEPA 2016f). Epidemiological studies of individuals exposed to PFOA-contaminated drinking water from industrial discharges (“highly exposed populations”) have demonstrated an association between PFOA and pregnancy-induced hypertension (Darrow et al. 2013; USEPA 2016f). Although PFOA has not been linked to low birth weight in these highly exposed populations, there is some evidence associating PFOA exposure in the general population

to small decreases in birth weight (Verner et al. 2015), decreases in fecundity (Velez et al. 2015) and fertility (Fei et al. 2009). In mice, PFOA has a significantly longer half-life than in rats—a fact that likely contributes to the greater sensitivity of mice to the reproductive and developmental effects of PFOA (Lau 2015). In mice, PFOA ( $\geq 1$  mg/kg) has caused decreases in pup body weight and decreased neonatal survival, whereas higher doses ( $> 5$  mg/kg) increased litter resorption and numbers of still births. Rats also experience developmental effects of PFOA, albeit at larger doses than mice. Decreased body weight gain, delayed sexual maturity, and decreased viability were observed in rat offspring after gestational exposure to 30 mg/kg (Butenhoff et al. 2004). The USEPA identified reduced ossification of the fore- and hindlimbs in male and female mouse pups, and accelerated puberty in male mice pups administered 1 mg/kg PFOA for derivation of the reference dose (RfD) (USEPA 2016f).

**PFOS.** In human populations, fetal PFOS exposure has been linked with lower average birth weights and with low infant body weight. Elevated serum PFOS levels have also been associated with decreased fertility and fecundity, manifested as greater time to pregnancy (USEPA 2016g). Several studies have shown effects of PFOS on semen quality, although the majority of studies that examined sperm parameters have not found a significant effect (USEPA 2016g). Additional evidence has linked serum PFOS with pre-gestational diabetes, and pregnancy-induced hypertension (USEPA 2016g). In rodents, PFOS has caused increased pup mortality, decreased body weight, and developmental delays. Exposure of rodents during gestation and lactation has been associated with abnormal glucose regulation in the offspring at maturity (USEPA 2016g). The EPA identified decreased body weights in newborn rats as the most sensitive endpoint observed from toxicity studies, and used the no observed adverse effect level (NOAEL) of 0.1 mg/kg-d as the basis of the RfD (USEPA 2016g).

**PFNA.** In mice, PFNA caused qualitatively similar developmental toxicity to PFOA and PFOS, with oral PFNA exposure causing dose-related effects in the number of live pups at birth and survival to weaning (Wolf et al. 2010) and neonatal mortality (Das et al. 2015). Surviving pups (1, 3, and 5 mg/kg) exhibited delayed eye opening and a delay in the onset of puberty (Das et al. 2015).

**Other PFAS.** Limited data indicate neither PFHxS nor PFBS are reproductive or developmental toxins, in that orally administered PFBS (30–1000 mg/kg) did not affect fertility, reproduction, or multiple indices of developmental toxicity in rats including litter size, mean pup birth weight, or pup survival (Lieder et al. 2009a). Similarly, PFHxS did not cause reproductive or developmental effects in rats given 0.3 to 10 mg/kg prior to mating, and during gestation and lactation (Butenhoff et al. 2009). However, increasing serum concentrations of PFHxS have been correlated to small decrements in human fecundity (Velez et al. 2015). There are no data on the reproductive or developmental effects of PFHpA.

### Liver and Lipoproteins

**PFOA.** In PFOA-exposed workers and nearby “highly exposed” community members exposed via drinking water, multiple epidemiology studies have linked elevated PFOA serum concentrations with increases in total cholesterol and low-density lipoproteins (serum PFOA levels of 0.4 to 12 micrograms per milliliter [ $\mu\text{g}/\text{mL}$ ] in workers, and an average of 0.08  $\mu\text{g}/\text{mL}$  in community members). PFOA serum levels of 0.002 to 0.007  $\mu\text{g}/\text{mL}$  were associated with elevated levels of triglycerides (USEPA 2016f). In animals, PFOA (and PFOS) tend to lower levels of total cholesterol, triglycerides, and lipoproteins (USEPA 2016f). Epidemiology studies of PFOA-exposed workers and residents of communities affected by industrial discharges of PFOA have consistently shown a relationship between serum PFOA levels and the liver enzymes alanine aminotransferase and gamma-glutamyl transaminase. These results notwithstanding, epidemiology data have not provided evidence of PFOA-induced liver damage. Rodents exposed to PFOA frequently exhibit increased liver weight and hepatocellular hypertrophy. Unless there is concomitant necrosis or cellular degeneration however, these effects are generally not considered biologically significant as they tend to be reversible once exposure ends (USEPA 2016f).

**PFOS.** Data from epidemiology studies have demonstrated an association between serum PFOS levels (0.0084  $\mu\text{g}/\text{mL}$  to 0.022  $\mu\text{g}/\text{mL}$ ) and increases in total cholesterol (USEPA 2016g). Multiple species of animals exposed to PFOS (0.098 to 1.56 mg/kg) have exhibited a similar spectrum of hepatic effects to those induced by PFOA, including cellular hypertro-

phy, increased liver weight, and vacuolation (USEPA 2016g).

**Other PFAS.** Limited data indicate the liver is also a target for PFNA, PFBS, and PFHxS, with hepatocellular hypertrophy, increased liver weight, and decreased lipoprotein synthesis observed. PFBS exposure elicited increased liver weight and adaptive hepatic hypertrophy in rats following doses of 300–1000 mg/kg (Lieder et al. 2009a). PFHxS caused increased liver weight and hypertrophy in rats (0.3–10 mg/kg); decreased triglyceride levels (rats, 3–10 mg/kg; mice, 30 mg/kg), and decreased levels of cholesterol and various lipoproteins in mice (6 mg/kg) were also features of PFHxS exposure (Butenhoff et al. 2009; Bijland et al. 2011). While PFBS exposure (30 mg/kg) has also been associated with impaired lipoprotein synthesis, the effects were limited to triglycerides (Bijland et al. 2011).

There are no data on the potential effects of PFHpA on the liver.

### Immune System

**PFOA and PFOS.** Evidence from both human and animal studies indicate PFOA and PFOS adversely affect multiple components of the immune system and as a result, can alter immune function (National Toxicology Program [NTP] 2016). Both PFOA and PFOS suppressed the antibody response and increased hypersensitivity-related outcomes in humans and animals. In humans, there is also evidence PFOA decreased resistance to infectious disease and increased autoimmune disease (NTP 2016). Animal data also indicate PFOS suppresses disease resistance and natural killer (NK) cell activity (NTP 2016).

**Other PFAS.** PFNA was cytotoxic to the spleen and thymus of mice (key organs of the immune system), reducing populations of splenic T cells, impairing cell replication in the thymus, and impairing the production of cell-signaling molecules (Fang et al. 2008).

There are no data on the effects of PFHpA, PFBS, or PFHxS to the immune system.

### Carcinogenicity

**PFOA and PFOS.** Available data indicate neither PFOA nor PFOS are genotoxic or mutagenic (USEPA 2016e, f); however, both PFOA and PFOS induce tumors in rodents after long-term high-level exposure. The USEPA Office of Water determined that there is

“suggestive evidence of carcinogenic potential” for PFOA (USEPA 2016c) and PFOS (USEPA 2016d). The International Agency for Research on Cancer (IARC 2016) found PFOA to be “possibly carcinogenic.” Neither agency nor independent scientists have evaluated the genotoxicity, mutagenicity, or carcinogenicity of other PFAS.

Epidemiology studies from highly exposed populations exposed to PFOA in drinking water found associations between serum PFOA serum levels and kidney and testicular cancers (C8 Science Panel 2013). In rats, PFOA exposure led to liver, testicular, and pancreatic tumors (reviewed in USEPA 2016d). Based on the rat testicular data for PFOA, the USEPA (2016d) derived a cancer slope factor (CSF) of 0.07 (mg/kg-d<sup>-1</sup>).

The evidence regarding the potential carcinogenicity of PFOS is more limited than for PFOA, and consists of somewhat equivocal epidemiologic evidence linking human exposure to PFOS to bladder and prostate cancer. Animal data support an association between PFOS exposure and an increased incidence of several tumor types, with the strongest evidence for liver tumors (USEPA 2016e). The USEPA (2016e) found the PFOS tumor data insufficient to derive a CSF.

**Other PFAS.** The carcinogenicity of PFNA, PFHpA, PFBS, and PFHxS has not been evaluated.

## RISK ASSESSMENT OF PFAS

Many PFAS are environmentally persistent, bioaccumulate in living organisms, and have demonstrated toxicity in laboratory animals and humans. The C8 Health Project, one of the most robust studies of PFOA exposure, provided data on approximately 70,000 exposed Ohio and West Virginia residents. Drinking water exposure to PFOA was linked to serum PFOA concentrations and a range of specific adverse health outcomes. Additionally, national biomonitoring studies have shown that the majority of the U.S. population has been exposed to PFAS. It is therefore prudent to assess and potentially mitigate human and/or environmental exposures.

### Available Toxicity Criteria and Screening Levels

#### Toxicity Criteria

**Non-Cancer RfD:** USEPA developed updated oral Reference Doses of 2E-05 mg/kg/day, applicable to both PFOS and PFOA. The RfDs were derived from developmental toxicity studies in mice (PFOA) (USEPA

2016e) and rats (PFOS) (USEPA 2016f). The RfD is an estimated daily exposure level for the human population (including sensitive subpopulations) that is likely to be without an appreciable risk of deleterious effects during a lifetime (USEPA 1989). USEPA recommends this value for both short-term (sub-chronic) and chronic (lifetime) exposures. USEPA has derived a provisional sub-chronic RfD (0.2 mg/kg/day) and a provisional chronic RfD (0.02 mg/kg/day) for perfluorobutane sulfonate (PFBS) based upon a sub-chronic and reproductive study in rats (USEPA 2014b).

At this time, there are insufficient data available to calculate an inhalation Reference Concentration (RfC) for PFOS and PFOA (USEPA 2016e, f).

**Cancer Slope Factor:** PFOA and PFOS have been classified as having *Suggestive Evidence of Carcinogenic Potential* in humans by USEPA (USEPA 2016e, f), while PFOA has also been classified as a Group 2B carcinogen (*possibly carcinogenic to humans*) by the International Agency for Research on Cancer (IARC) (IARC 2016). An oral cancer slope factor of 0.07 (mg/kg/day<sup>-1</sup>) has been developed for PFOA by USEPA, based upon testicular cancer (Leydig Cell tumors) in rodents (USEPA 2016e). An oral cancer slope factor has not been calculated for PFOS since the available tumor data were determined to be inadequate for quantitative assessment (USEPA 2016f). PFBS is provisionally classified as “*Inadequate Information to Assess Carcinogenic Potential*” (USEPA 2014b). Cancer slope factors provide an upper-bound estimate of the probability of a response (i.e., cancer) per unit intake of a chemical over a lifetime (USEPA 1989).

#### Drinking Water Health Advisory

Currently there are no federal regulations under the Safe Drinking Water Act (SDWA) or national recommended ambient water quality criteria under the Clean Water Act (CWA) for PFAS. In 2009, USEPA developed interim, provisional HAs for short-term exposure to PFOS and PFOA of 0.2 and 0.4 µg/L, respectively (USEPA 2009). HAs identify the concentration of a contaminant in drinking water at which adverse health effects are not anticipated to occur over specific exposure durations (e.g., 1 day, 10 days, a lifetime). HAs serve as informal technical guidance to assist federal, state, and local officials and managers of public or community water systems in protecting public health when emergency spills or other contamination situations occur (USEPA 2016c, d).

**Table 3.4. Soil screening levels for PFOS and PFOA (mg/kg).**

Entity	Resident		Industrial Worker		Recreational User		Outdoor Commercial Worker		Excavation or Construction Worker	
	PFOS	PFOA	PFOS	PFOA	PFOS	PFOA	PFOS	PFOA	PFOS	PFOA
USEPA (USEPA 2016g)	1.26	1.26	16.4	16.4	NE	NE	18.2 <sup>1</sup>	18.2 <sup>1</sup>	NE	NE
Maine (ME DEH 2014)	11	0.80	NE	NE	18	1.3	82	6.2	19	1.4
US Navy (NAVFAC 2013)	4.9	12	49	123	NE	NE	NE	NE	NE	NE
Delaware (DNREC 2016)	6	16	NE	NE	NE	NE	NE	NE	NE	NE
Iowa (IDNR 2016)	1.8	1.2	NE	NE	NE	NE	NE	NE	NE	NE
Minnesota (MPCA 2009)	2.1	2.1	14	13	2.6	2.5	NE	NE	NE	NE
Texas (TRRP 2016)	1.5/1.5 <sup>2</sup>	0.6/0.49 <sup>2</sup>	15/14 <sup>2</sup>	2.4/1.4 <sup>2</sup>	NE	NE	NE	NE	NE	NE

NE = Not Established

<sup>1</sup> Calculated with USEPA's RSL Calculator

<sup>2</sup> Total Soil Combined Protective Concentration Level for 0.5-acre/30-acre source area

**Table 3.5. Drinking water/groundwater screening levels (µg/L) for PFOS and PFOA.**

Entity	PFOS	PFOA
USEPA (USEPA 2016g). States that adopted USEPA HA: Alabama (ADEM 2016), Arizona (Liberty Utilities 2016), Colorado (CO DPHE 2016), Connecticut (CT DPH 2016), Delaware (DNREC 2016), Iowa (IDNR 2016), Massachusetts (MA DEP 2016), Maine (ME DEP 2016), New Hampshire (NHDES 2016), New York (NYS DOH 2016), West Virginia (WV DHHR 2016)	0.07	0.07
Alaska (AKDNC 2016)	0.4	0.4
Illinois (ASTSWMO 2015); Kentucky (NKWD 2016)	0.2	0.4
Michigan (MI DEQ 2016)	0.011	0.42
Minnesota (MN DOH 2017)	0.027	0.035
New Jersey (NJDEP 2007)	NE	0.04
North Carolina (NC DENR 2013)	NE	2
Texas (TRRP 2016)	0.56	0.29
Vermont (VT DOH 2016)	0.02 (combined)	0.02 (combined)

NE = Not Established

**Table 3.6. Recreational sediment and surface water screening levels for PFOS and PFOA.**

Entity	Recreational Sediment (mg/kg)		Recreational Surface Water (µg/L)	
	PFOS	PFOA	PFOS	PFOA
USEPA (USEPA 2016f)	NE	NE	NE	NE
Maine (ME DEH 2014)	20	1.5	1.2	0.05
Michigan (MI DEQ 2016)	NE	NE	0.012	12

NE = Not Established

As of May 2016, the lifetime drinking water HA for PFOA and PFOS is 0.07 µg/L, which is applicable to either chemical individually or collectively. The HA is based upon the RfD of  $2 \times 10^{-5}$  mg/kg discussed previously. Since the HA was developed based upon sensitive developmental effects, it is also protective of adverse effects seen in adults (liver and kidney toxicity). Because the HA is lower than the concentration derived for carcinogenic effects of PFOA, it is also protective of carcinogenic effects for the general population (USEPA 2016c, d).

### Screening Levels

A summary of available screening levels for PFOS and PFOA are provided in Tables 3.4 (Soil), 3.5 (Drinking Water/Groundwater) and 3.6 (Recreational Sediment and Surface Water). Additional screening levels for PFBS and a number of other PFAS are available from USEPA (USEPA 2016g) and from other state agencies including Delaware (DNREC 2016), Indiana (IDEM 2016), Minnesota (MPCA 2009; MN 2016), New Jersey (NJDEP 2015, 2016), Nevada (NDEP 2015), North Carolina (NC DENR 2016), and Texas (TRRP 2016).

### Relative Source Contribution in the Derivation of PFAS Risk-Based Criteria

A Relative Source Contribution (RSC) factor takes into account drinking water and non-drinking water exposures to a contaminant including food, soil, air, water, and consumer product use. The RSC is applied in the USEPA HA calculation to ensure an individual's total exposure from a contaminant (i.e., PFOA) does not exceed the RfD. The RSC accounts for the portion of the RfD attributed to drinking water (directly or indirectly

in beverages like coffee, tea, or soup); the remainder of the RfD is allocated to other potential exposure sources. In the case of PFOA, other potential sources include ambient air, foods, incidental soil/dust ingestion and consumer products.

The USEPA's default RSC value is 20%, which means 20% of the total exposure is assumed to come from drinking water and 80% from non-drinking water sources. The USEPA determined there were insufficient data to develop a chemical-specific RSC for PFOA and PFAS, and therefore the default value of 20% was used to calculate the HA (USEPA 2016c, d).

### Unique Receptors and/or Exposure Factors Used in the Derivation of PFAS Risk-Based Criteria

The lifetime HAs for PFOS and PFOA were calculated using drinking water intake and body weight parameters for lactating women in order to be protective of this critical exposure period for infants. Studies have shown significant exposure to infants through the ingestion of breast milk (Hag et al. 2011; Forns et al. 2015); consistent with this fact, studies have also shown breast feeding to be a potential excretion route for mothers (Mondal et al. 2014). The lactating woman provides the more protective scenario over the pregnant woman, given an increased water intake is needed to support milk production. The use of these exposure factors to calculate the lifetime HA are specific to the most sensitive population and therefore are protective of pregnant and lactating women as well as the general population (USEPA 2016c, d).

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## APPENDIX 1

### PFAS Bioaccumulation Potential

Factor	Definition	Reported Values	Interpretation	References
Bioconcentration factor (BCF)	Ratio of chemical concentration in an organism to the concentration in water (at steady-state). Values > 2000 indicate bioconcentration	PFOA 1.8-27 (fish)	PFOA. Does not bioconcentrate in fish	ECHA 2013; CONCAWE 2016
		PFOS 830-26,000 (fish)	PFOS. Bioconcentrates in fish	
		PFBS <1 (fish)	PFBS. Does not bioconcentrate in fish	
Bioaccumulation factors (BAFs)	Ratio of chemical concentration in an organism to the concentration from all sources (water, diet, soil, air). Values > 2000 indicate bioaccumulation	PFOA. 0.9-292 (fish, mussels, phytoplankton)	PFOA. Does not bioaccumulate in fish, mussels, phytoplankton	ECHA 2013; CONCAWE 2016
		PFOS 240 (zooplankton); 1200 (mussels); 16,000-95,000 (fish)	PFOS. Bioaccumulates in fish	
Biomagnification factors (BMFs)	Ratio of chemical concentration in an organism to the concentration in the diet. Values >1 indicate biomagnification	PFOA. 0.02-7.2 (fish); 1.8-125 (dolphins, polar bears); 0.1-2.7 (whales, seals); 0.9-11 (wolves, caribou)	PFOA. Biomagnifies in multiple species	ECHA 2013; CONCAWE 2016
		PFOS. 4.6-8.7 (fish); 2.0-9.1 (caribou); 0.8-4.5 (wolves); 0.9 (dolphins); 4.0-8.4 (whales).	PFOS. Biomagnifies in fish, caribou, dolphins, whales	
Trophic magnification factors (TMFs)	The increase in chemical concentration in an organism with an increase of one trophic level. Values >1 indicate trophic magnification	PFOA 0.37-0.53 (fish); 1.2-13 (seals, dolphins); 0.3-2.64 (whales); 1.1-1.3 (wolves, caribou)	PFOA. Trophic magnification occurs in air-breathing aquatic mammals, not in gill-breathers	ECHA 2013