Per- and Poly-Fluorinated Alkyl Substances Chemical Action Plan (PFAS CAP) – 2019 Updates

Updated Ecological Toxicology Chapter

In 2017, the Washington State departments of Ecology and Health shared draft PFAS CAP chapters with external parties for review and comment. Comments received are available online. This document is either an update of a 2017 draft or a new 'chapter.' Ecology and Health are sharing chapters with interested parties prior to the March 2019 PFAS CAP webinar. Updates will be discussed during the March webinar. We expect to publish the entire Draft PFAS CAP around June 2019 followed by a 60-day comment period.

In March 2019, Ecology and Health will host a PFAS CAP webinar to:

- Review updated/new chapters – comments to be accepted during the 2019 public comment period (anticipated summer 2019).
- Discuss draft Recommendations – requesting comments and suggestions from interested parties – due April 5th.

Quick summary of PFAS CAP efforts:

- PFAS CAP Advisory Committee and interested parties meetings in 2016, 2017 and 2018.
- September 2017 Draft PFAS CAP chapters posted:

Intro/Scope	Environment
Biosolids	Health
Chemistry	Regulations
Ecological Toxicology	Uses/Sources

- March of 2018, Ecology and Health published the Interim PFAS CAP.
- The 2019 updated PFAS CAP "chapters" to be posted (in the order we expect to post on the PFAS CAP website):

Biosolids	Chemistry
Ecological Toxicology	Health
Environment	*Analytical methods (new)*
Regulations	*Fate and Transport (new)*
Uses/Sources	*Economic analysis (new)*
	Recommendations (new)

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This document is posted on the PFAS CAP Website - https://www.ezview.wa.gov/?alias=1962&pageid=37105

Per- and PolyFluoroalkyl Substances (PFAS): Persistence, Bioaccumulation, Toxicokinetics, and Toxicological Response for Ecological Receptors

1.0 Purpose, Applicability, and Terminology

This chapter summarizes the potential risk that per- and polyfluoroalkyl substances (PFAS) pose to ecological receptors. To address this objective, a range of PFAS compounds are included in order to evaluate several bioaccumulation and toxicity endpoints on representative aquatic and upland receptors.

PFAS terminology proposed by Buck, et al. (2011) is used as overall guidance in this chapter. However, terminology specific to cited articles is not altered to avoid translation errors. In some cases, this conflicts with acronyms recommended by Buck, et al. (2011). For example, in the wildlife study authored by Reiner and Place (2015), the PFAS acronym appears to denote perfluoroalkyl acids (PFAA), which include perfluoroalkyl carboxylic acids (PFCA) and perfluoroalkyl sulfonic acids (PFSA). Additionally, terminology presented by Kelly, et al. (2009) and Houde, et al. (2006) include perfluoroalkyl contaminant (PFC) and polyfluoroalkyl substance (PFS), respectively, which both appear to denote PFAS. Other PFAS terms are introduced throughout this chapter, and a list of acronyms is provided in Section 7.0, following the reference list.

2.0 Summarizing Ecological Risk

The potential risk chemicals pose to ecological receptors is dependent on:

- Distribution of the chemical in the environment.
- Persistence of the chemical in the environment.
- Persistence and bioaccumulation of the chemical within the organism.
- Toxicokinetics[1].
- Resulting toxicological response of the organism to the chemical.

Distribution and persistence of PFAS in the environment have been described in an earlier chapter of the CAP. As a result, the foci of this chapter are on the persistence and bioaccumulation within organisms, toxicokinetics, and the resulting toxicological responses of the organism to PFAS compounds.

[1] Toxicokinetics is the determination of the fate of the chemical administered to a living organism (i.e. where does it move within the organism). The term includes the basic kinetics concepts of absorption, distribution, metabolism, and excretion (ADME) of chemicals.

2.1 Assessing Ecological Risk Based on Grouping (Short vs. Long-Chain PFAS)

An early step in the assessment of evaluating the potential risk of PFAS is to group the short- and long-chain substances based on the number of associated perfluorinated carbons.

Short-Chain PFAS

Short-chain PFAS contain up to five perfluorinated carbons terminating with a sulfonate group, or up to six perfluorinated carbons terminating with a carboxyl group (Buck, et al. 2011). While resistant to degradation, these substances do not appear to be highly bioaccumulative or to have significant toxicological effects on ecological receptors (IMAP 2017a, 2017b, and 2017c; USEPA 2017). These short-chain substances include:

- Short-chain PFCAs and their direct and indirect precursors.
- Direct and indirect precursors of PFBS[2].
- Short-chain polyfluoroalkyl acrylic polymer[3] based on 6:2 fluorotelomer chemistry (Methacrylate Polymer), intermediates and degradation products.
- Short-chain polyfluoroalkyl acrylic polymer based on perfluorobutane sulfonamide chemistry, intermediates and degradation products (that may be precursors to PFBS and PFBA).

Additional literature suggests that:

- 6:2 FTOH, 6:2 FTAC, and 6:2 FTMAC (considered short-chain PFAS) would not meet the criteria for persistence, bioaccumulation, or toxicity based on the Stockholm Convention on Persistent Organic Pollutants (Ramboll Environ. 2016).
- Research findings suggest (see Table 1 below) that biomagnification and bioaccumulation increase as the number of fluorinated carbons also increase (Conder, et al. 2008).

Table 1: Example of the bioaccumulation potential of some PFAS, as related to the number of fluorinated carbons comprising each compound's molecular structure (Conder, et al. 2008).

# Fluorinated Carbons	Compound	Frequency of Detection	BAV/BCF Significant Values (L/Kg)	Biomagnification	Bioaccumulative
Perfluoroalkyl Sulfonates					
4	PFBS	Not detected	< 1	No	No

[2] The degradation of PFBS is very slow compared with its rate of formation from degradation of the precursors and PFBS will be the final degradant from multiple precursors. Therefore, the amount of PFBS in the environment (general or local) is expected to be higher than that of any of the precursors. It is therefore assumed for the purposes of the cited (IMAP) assessment that the primary risk posed by the chemicals in this group results from the release of PFBS to the environment.

[3] Polymers may be precursors to short-chain substances, but they are not themselves short-chain substances. The side chains may be based on short-chain monomers.

# Fluorinated Carbons	Compound	Frequency of Detection	BAV/BCF Significant Values (L/Kg)	Biomagnification	Bioaccumulative
6	PFHxS	Detected in some wildlife	10	No	No
8	PFOS	Detected in most wildlife	18 - 11000	Possibly	Yes
Perfluorocarboxylates					
4 - 6	PFPn, PFHx, PFHp	Not detected or infrequently detected	< 1	No	No
7	PFO	Detected in some wildlife	2 – 570	No	No
8 – 13	PFN, PFD, PFU, PFDo, PFTri, PFT	Detected in most wildlife	100 - 23000	Possibly	Possibly

Long-Chain PFAS

Long-chain PFAS contain seven or more perfluorinated carbons terminating with a carboxylate group, or six or more perfluorinated carbons terminating with a sulfonate group (Buck, et al. 2011; OECD 2013; Wang, et al. 2017). These chemicals also resist degradation. Data in Table 1 (Conder, et al. 2008) limit bioaccumulation to PFAS with eight or more fluorinated carbons. However, in contrast to the short-chain substances, more recent data confirm that long-chain PFAS tend to be more bioaccumulative and produce adverse toxicological effects to both upland and aquatic ecological receptors even at relatively low contaminant levels (IMAP 2017d, 2017e, 2017f, and 2017g). These long-chain substances include:

- PFHxS and related perfluoroalkylcyclohexane sulfonates.
- PFOA and its direct precursors.
- PFOS and its direct precursors.
- Direct precursors to:
    - PFHpS.
    - PFHxS.
    - PFPeS.

Additional literature suggests that long chain PFAS refer to the following (OECD 2015):

- PFCAs with 7 and more perfluoroalkyl carbons, such as:
    - PFOA or C_8 PFCA; with 8 carbons.
    - PFNA or C_9 PFCA; with 9 carbons.
- PFSAs with 6 and more perfluoroalkyl carbons, such as:

  - PFHxS or C_6 PFSA; with 6 perfluoroalkyl carbons.
  - PFOS or C_8 PFSA; with 8 perfluoroalkyl carbons.
- Precursors that have the potential to transform to long-chain PFCAs or PFSAs in the environment or biota, such as PASF- and FT based substances.

Summary
Relative to long-chain PFAS, it is important to note that there appears to be less research available on short-chain PFAS. Although bioaccumulation may be lower, short-chain PFAS are more water soluble and show greater mobility in the environment, relative to long-chain PFAS (Guelfo and Higgins 2013; Wang, et al. 2015; ITRC 2018). However, the information presented above indicates the potential risk of these short-chain PFAS substances (e.g., PFBS, PFPn, PFHx, PFHp) is generally less than that of the long-chain substances (e.g., PFOA, PFOS) to both aquatic and upland ecological receptors. As a result, this review will focus on evaluating the potential risks of long-chain PFAS on ecological receptors.

2.2 PFAS Representative Substances

As mentioned earlier, the potential risk for ecological receptors is much greater for the general class of chemicals known as long-chain PFAS. It is important to note that most of the information presented in this review is derived from the most extensively produced of the long-chain PFAS—PFOA and PFOS. The rationale for using these two specific chemicals as representative of the general class of long-chain PFAS chemicals is:

- PFOA and PFOS are the most widely studied of the long-chain PFAS.
- These chemicals are structurally related, in that one of the defining characteristics that differentiates the chemicals within this class from other classes is chain length (or number of carbon atoms in the molecule).
- The carbon-fluorine bonds are among the strongest in organic chemistry which renders them practically non-biodegradable and persistent in the environment (Key, et al. 1997; Presher, et al. 1985; Lau, et al. 2007), including their presence and persistence in:
  - Water.
  - Soil and sediment.
  - Ambient air.
  - Humans and laboratory animals.
  - Aquatic, benthic, and upland wildlife.
- The toxicokinetics and toxicological response for these chemicals appears closely related depending on species observed (Lau, et al. 2007; Kelly, et al. 2009; Lindstrom, et al. 2011; White, et al. 2011).

3.0 Persistence and Bioaccumulation within the Organism

Fluorine atoms are substituted for the hydrogen atoms that compose part of the hydrocarbon backbone in PFAS compounds. The fluorine-carbon bonds are stronger than the hydrogen-carbon bonds they replace, conferring extremely high chemical and thermal stability on these substances, and is manifested in high environmental persistence. In addition, some long-chain

PFAS bioaccumulate in the environment and can also undergo biomagnification (Li 2009; Stahl, et al. 2009).

The anions of PFOA, PFNA, and PFDA have been detected in a variety of wildlife across the globe. Detection of chemicals in wildlife does not necessarily imply high bioaccumulation potential for any specific chemical, but does comprise a standard element of many environmental monitoring programs. The large number of biota samples collected that contain quantifiable amounts of PFCAs, the ongoing scientific discourse regarding the high persistence and long-range fate and transport of PFCAs, and perceived similarities with perfluorinated sulfonates (including PFOS) have prompted concerns regarding the bioaccumulation potential of PFCAs (Conder, et al. 2008). PFOS is reported to have a very low Henry's law constant[4], indicating aquatic environments may be a significant sink for PFOS with a potential for bioaccumulation in fish (Boudreau, et al. 2003).

PFOS and longer chain PFCAs (> C8) bioaccumulate and persist in protein-rich compartments of fish and birds, and in marine mammal tissues, such as carcass, blood, and liver. In Washington, PFOS and other long-chain PFAAs have been detected in freshwater fish fillet and liver samples, as well as in osprey eggs (Ecology 2017). PFOS has been the most frequently detected PFAA in zooplankton and other invertebrate studies. However, most studies showed concentrations of PFOS and other PFAAs very close to the limits of detection (Reiner and Place 2015). Available evidence shows the likely potential for bioaccumulation or biomagnification in marine or terrestrial species (USEPA 2009). Concentrations of PFOA/PFOS in plants vary greatly, depending on the concentrations applied to the soil, as well and soil-to-plant uptake factors. The uptake and storage of these substances in the vegetative parts of the plants appear to be greater than the transfer to the storage organs within the plants (Stahl, et al. 2009). PFAA studies of birds have benefited from having species derived from many regions of the planet, including both aquatic and terrestrial ecosystems, representing a broad range of PFAA sources. The majority of these studies focus on birds coming from the Arctic, North America, and Europe. However, there does appear to be limited studies from the Southern Hemisphere (Antarctica and the Southern Ocean). Initial studies focused on PFOS and PFOA, but the number of PFAAs examined has recently expanded to precursor compounds, PFCAs, and PFSAs (Reiner and Place 2015).

In an earthworm study focused on bioaccumulation of PFAAs, the highest BAF (139 g soil dry wt. / g worm dry wt.) was observed for PFHxS in a soil contaminated with firefighting foam (Rich, et al. 2015). BAFs increased with chain length for PFCAs but decreased with chain length for PFSAs (Rich, et al. 2015). The unexpected finding for PFSAs may relate to decreased bioavailability. Overall, results from this study indicated that PFAA bioaccumulation into earthworms depends on soil concentrations, soil characteristics, analyte, and duration of exposure, and that accumulation into earthworms may be a potential route of entry of PFAAs into terrestrial foodwebs (Rich, et al. 2015).

[4] Henry's law constant refers to the ratio of a chemical concentration in the air to its concentration in water. Henry's law constant can vary significantly with temperature for some hazardous substances (Ecology 2007).

Bioaccumulation is generally apparent for a variety of long-chain PFAS compounds in both terrestrial and aquatic wildlife. However, as shown in Table 2, BAF values in aquatic biota vary by specific compound, species, and tissue.

Table 2: BAF Values for Aquatic Biota.[5]

PFAS	Species	Tissue	BAF (L/Kg)	Reference
PFOS	Bluegill	Fillet	2700	MPCA 2013
PFOS	Carp	Fillet	1237	MPCA 2013
PFOS	Freshwater Drum	Fillet	3077	MPCA 2013
PFOS	Smallmouth Bass	Fillet	2845	MPCA 2013
PFOS	White Bass	Fillet	4618	MPCA 2013
PFOS	Common Shiner	Liver	6300 – 125000	Moody, et al. 2002
PFOS	Rainbow Trout	Carcass	690	ECCC 2017
PFOS	Rainbow Trout	Blood	3100	ECCC 2017
PFOS	Rainbow Trout	Liver	2900	ECCC 2017
PFOS	Phytoplankton	Whole unit	169	Loi, et al. 2011
PFOS	Lake Trout	Whole unit	31623	De Silva, et al. 2011
PFOA	Phytoplankton	Whole unit	292	Loi, et al. 2011
PFOA	Lake Trout	Whole unit	126	De Silva, et al. 2011
PFOA	Rainbow Trout	Blood	27	OECD 2008
PFOA	Rainbow Trout	Liver	8	OECD 2008
PFOA	Rainbow Trout	Whole unit	4	OECD 2008
PFHxS	Phytoplankton	Whole unit	58	Loi, et al. 2011
PFNA	Phytoplankton	Whole unit	1650	Loi, et al. 2011
PFDA	Phytoplankton	Whole unit	765	Loi, et al. 2011
PFECHS	Lake Trout	Whole unit	631	De Silva, et al. 2011
PFUnDA	Phytoplankton	Whole unit	4510	Loi, et al. 2011

In summary, persistence and bioaccumulation within the organism appear to be dependent on chain length. PFAS that contain six or more perfluorinated carbons have the potential to bioaccumulate within ecological receptors. It is apparent that wildlife from around the world are exposed to PFAAs. There is a tendency for animals living closer to industrialized regions to have higher concentrations of PFAAs compared to those living in more remote locations. The main compound found in most wildlife species is PFOS. However, especially in the more recent studies, the long chain PFCAs are frequently being detected and measured (Reiner and Place 2015).

4.0 Toxicokinetics and Toxicological Response

The toxicokinetic properties and toxicological responses of PFOS and PFOA have been studied in some detail. While there appears to be more literature available for aquatic than upland receptors, enough information is available for both to summarize the fate, as well as possible

[5] Typically, a BAF is calculated as: Chemical concentration in the organism / Chemical concentration in the matrix. A higher BAF indicates more contaminant accumulates within the organism, relative to the environmental matrix.

adverse effects, of these contaminants. In particular, animal studies with both PFOS and PFOA have shown that they are well-absorbed orally, but poorly eliminated; they are not metabolized, and undergo extensive re-uptake from enterohepatic circulation[6] (Lau, et al. 2007).

Conder, et al. (2008) have noted that the principal repository of bioaccumulated PFCA and PFSA in organisms is not lipid but protein. Although a portion of these chemicals is hydrophobic and may interact with lipids, the presence of the carboxylate or sulfonate functional group imparts high hydrophilicity, thereby making the molecule partly lipophilic and partly hydrophilic. Several studies have suggested that PFAAs are proteinophilic. For example, PFO in both rats and humans was strongly associated with serum albumin and other cytosolic proteins, and the proteinophilic nature of this class of chemicals has been hypothesized for the longer-chain PFAS (seven to eight fluorinated carbons). In support of this hypothesis, PFD (nine fluorinated carbons) has been shown to be more potent than PFO (seven fluorinated carbons) in binding to avian and carp serum proteins (Conder, et al. 2008).

In general, studies indicate that PFAAs are proteinophilic. For example, the tissue distribution of PFOA is undoubtedly dictated to some extent by its ability to bind avidly to plasma and other proteins (Kennedy, et al. 2004). In contrast to the protein binding ability of those chemicals with longer fluorinated carbon chains, the shorter perfluorinated compounds (PFSA and PFCA with four and three fluorinated carbons, respectively) were found to be 1-2 orders of magnitude less proteinophilic (Conder, et al. 2008).

Comparing adverse effects among studies can be confounded by differences in genetics (e.g. species, gender), as well as differences in dose regimen (e.g. spacing, magnitude, duration, and route of administration). However, if the toxic mechanism is conserved, and some measure of the tissue concentration (i.e., dosimetry) at the biological target can be determined, then it is expected that this dosimetric anchor would be conserved across studies. Careful consideration of toxicokinetics is therefore required in order to link chemical exposure to toxicity (Wambaugh 2015). As a result, the tables presented later in this chapter illustrate effects associated with chemical concentrations in water (aquatic species) or chemical dose (upland species), rather than use for regulatory purposes.

4.1 Ecological Receptors in the Aquatic Environment

Aquatic plant studies have detected toxicological effects in microalgae (Latala, et al. 2009) and green algae (Ding, et al. 2012) for a variety of endpoints, including physiology, membrane potential, and growth rate (Rodea-Palomares, et al. 2015; Mhadhbi, et al. 2012). PFOS has been shown to be moderately toxic to aquatic invertebrates with acute toxicity values (48 and 96 hr. LC50) in the range of 10-300 mg/L, while PFOA has been shown to be only slightly toxic to aquatic invertebrates, with toxicity values in the range of 100-1000 mg/L (Li 2009). Boudreau, et al. (2003) indicates that PFOS is acutely toxic to freshwater organisms at or near 100 mg/L. Chronic toxicity would be expected to occur at lower concentrations.

[6] Enterohepatic circulation refers to the circulation of the chemical from the liver to the bile, followed by entry into the small intestine, absorption by the enterocyte (intestinal absorptive cells) and then transport back to the liver.

Several toxicological effects have been tabulated for PFOA and PFOS in aquatic biota (Table 3). A variety of endpoints and effect concentrations are listed. As expected, gene expression effects occur at low concentrations (e.g., Cheng, et al. 2011; Spachmo and Arukwe 2012), relative to concentrations linked with deficits in apical endpoints (e.g., growth, survival, reproduction).

Table 3: Toxicological effects of PFOA and PFOS in aquatic species.

Chemical	Species	Matrix	Concentration (ug/L)	Effect	Reference
PFOA	Blue-Green Algae	freshwater	5000 (LOEC)	Physiology/Membrane Potential	Rodea-Palomares, et al. 2015
PFOA	Atlantic salmon	freshwater	100 (LOEC)	Genetics/Bone Development	Spachmo and Arukwe 2012
PFOA	Sea urchin	salt water	20000 (LOEC)	Growth/Length	Mhadhbi, et al. 2012
PFOA	Mysid	salt water	7800 (EC10)	Mortality	Mhadhbi, et al. 2012
PFOS	Mysid	salt water	530 (LOEC)	Survival	Drottar and Krueger 2000
PFOS	African Clawed frog	freshwater	0.1 (LOEC)	Genetics/Up-regulation of thyroid hormone regulated genes	Cheng, et al. 2011
PFOS	Water flea	freshwater	312.5 (LOEC)	Reproduction	Ji, et al. 2008
PFOS	Fathead minnow	freshwater	3300 (NOEC)	Survival	Drottar and Krueger 2000
PFOS	Algae	salt water	12200 (EC10)	Population/Growth rate	Mhadhbi, et al. 2012
PFOS	Mysid	salt water	3200 (EC10)	Mortality	Mhadhbi, et al. 2012
PFOS	Sea Urchin	salt water	2000 (EC10)	Growth/Length (EC10)	Mhadhbi, et al. 2012

Freshwater biota: PFOA concentrations were observed in the following order in the tissues of rainbow trout (*Oncorhynchus mykiss*): blood > kidney > liver > gall bladder > gonads > adipose > muscle tissue, at average water exposure concentrations between 0.014 and 1.7 µg/L (Martin, et al. 2003). PFAAs also were detectable in the gills, suggesting that this was the site of uptake, depuration, or both, as has been determined for other xenobiotics[7] (Martin, et al. 2003). In addition, it was found that PFAS inhibited growth and had detrimental effects on photosynthesis on green algae (*P. subcapitata*, *S. capricornutum* and *C. vulgaris*) (Ding, et al. 2012; Boudreau, et al. 2003), as well as the floating macrophyte, *L. gibba* (Boudreau, et al. 2003).

Marine Environment: PFCs in arctic marine areas were found in tissue/fluids of fish, seaducks, and beluga whales (Kelly, et al. 2009). PFOA, PFNA, PFDA, and Perfluoroundecanoic acid (PFUnA) were commonly detected in sediments and macroalgae. PFOS and C_7 – C_{14} PFCAs were routinely detected in fish, seaduck, and beluga whale samples (Kelly, et al. 2009). High PFS concentrations have been detected in dolphin plasma and tissue samples in which PFOS, C_8 and C_{10} – PFCAs predominated in most matrices (Houde, et al. 2006). In addition, a preliminary screening of PFOS and related compounds has been performed in liver samples of fish, birds, and marine mammals from Greenland and the Faroe Islands (Bossi, et al. 2005). PFOS was the predominant fluorochemical in the biota analyzed, followed by PFOSA. Biomagnification of

[7] Xenobiotics are chemical substances found within an organism that are neither naturally produced by the organism nor expected to be present within the organism.

PFOS along the marine food chain were in the order of: shorthorn sculpin < ringed seal < polar bear. The greatest concentration of PFOS was found in the liver of polar bears (mean: 1285 ng/g wet weight, n = 2) (Bossi, et al. 2005).

4.2 Ecological Receptors in the Upland Environment

Upland Plants: Standard terrestrial plant test species are often used to evaluate the effect of contaminants on native wild plant species (USEPA 2012). For example, effects of PFAS on growth and reproduction have been studied in lettuce, pak choi, and cucumber (Li, et al. 2009; Ding, et al. 2012). There were no obvious effects on seed germination for the species. However, based on EC_{10}, EC_{50}, and NOECs, the five day root elongation sensitivity of test plants to both PFOS and PFOA were in the order of: lettuce > pak choi > cucumber (Li, et al. 2009). In addition, another study evaluated the toxicity effects of seven PFCs in a five day test on root elongation of lettuce (*L. sativa*) (PFBA; 2,2,3,3,4,4,5,5 Octafluoro-1-pentanol; PFOA; PFNA; PFDA; PFUnA; PFDoA) (Ding, et al. 2012). This study indicated that the toxic effects of the seven PFCs increased with increasing fluorinated carbon chain length. It should be noted that extrapolating effects of PFAS on these test species to upland plants introduces additional uncertainty into an assessment of wild native plant species.

Upland Wildlife (and surrogate species): Limited information is available on the toxicokinetics and toxicological properties of PFOS and PFOA on upland wildlife receptors. Because few studies have determined safe exposure levels (NOAELs) for situations in which wildlife have been exposed over an entire lifespan or several generations, chronic exposures to a particular chemical are often estimated from toxicity studies conducted on a surrogate species. In many cases, the only available information is from studies on a laboratory species (primarily rats and mice) (Sample, et al. 1996). While not ideal, these surrogate species do provide valuable information.

A study was performed exploring the induction of liver tumors in Wistar rats for several chemicals, including PFOA (Abdellatif, et al. 1990). In comparison to controls, this study indicated that PFOA caused a 24-fold increase in the peroxisomal β-oxidation of fatty acids, but only about a 2-fold increase in catalase activity. These results suggest that PFOA has a promoting action on liver carcinogenesis.

In other laboratory studies, exposure to PFOA significantly increased offspring relative liver weights in all treatment groups in a full gestation study, and offspring of PFOA-treated dams exhibited significantly stunted mammary epithelial growth, as assessed by developmental scoring (Macon, et al. 2011). Evaluation of internal dosimetry in offspring revealed that PFOA concentrations remained elevated in liver and serum for up to 6 weeks and that brain concentrations were low and undetectable after 4 weeks. Additionally, in wild-type mice, concentrations of PFOA measured in the serum and liver were directly correlated with increasing dose to the animal, while the livers had ultrastructural changes induced by PFOA (Wolf, et al. 2008).

Reproductive and developmental effects are presented for several PFAS in terrestrial species, as shown in Table 4. Again, a variety of endpoints and dose levels are listed. Most of these data are for surrogate test animals, which imperfectly represent wildlife species.

Table 4: Reproductive and developmental effects of selected PFAS compounds in upland and surrogate species (data reported in Stahl, et al. 2011) [NR = Not Reported].

Chemical	Species	Dose (mg/kg – BW/day)	Gestation Day (day)	Effect	Reference
PFOS	Rats	1 – 10	6 – 15	Decrease body mass and lens abnormalities	Gortner 1980
PFOS	Quail	10 – 150 mg/kg feed	NR	Decrease viability of the 14 day old progeny; slight increase in incidences of small testes, however spermatogenesis and fertility were not affected	Newsted, et al. 2007
PFOS	Rabbits	0.1 – 3.75	6 – 20	Decrease in weight gain of the maternal animal; decreased birth weight and delayed ossification	Case, et al. 2001
PFOS	Leghorn chickens	1 – 5 mg/kg egg	Before incubation	No effect on hatching rate; increase spleen mass; right wings shorter; frequent occurrence of brain asymmetry; decrease immunoglobulin; increase plasma lysozyme activity; increase liver mass; increase body weight	Peden-Adams, et al. 2009
PFOA	Rats	1 – 30	NR	Decrease body weight; increase liver and kidney mass; decrease birth weight; delayed puberty; increase mortality rate after weaning	Butenhoff, et al. 2004a, 2004b
PFOA	Mice	1 – 40	During gestation	Liver enlargement; decrease in full term gestation, viable fetuses, fetus weight and postnatal viability; growth deficit; delayed opening of eyes; accelerated sexual maturity of male progeny	Lau, et al. 2006
PFOA	Chickens	5 – 40 mg/kg egg	Before incubation	Impaired hatching rate; high prevalence of splayed legs; chicks with partial or complete loss of yellow pigment in the down	Yanai, et al. 2008
PFBA	Mice	35 – 350	1 – 17	No adverse effects on survival rate of progeny or their postnatal growth; delayed opening of eyes; delayed onset of puberty; at the highest dosage: loss of complete litter	Das, et al. 2008
PFDA	Mice	0.25 to 32	10 – 13	Decrease in weight gain of maternal animal at high doses, fetal body weight reduced a low doses, no malformations observed	Harris and Birnbaum 1989
PFDA	Mice	0.03 – 12.8	6 – 15	Decrease in weight gain of maternal animal at high doses, fetal body weight reduced a low doses, no malformations observed	Harris and Birnbaum 1989

5.0 Summary

A distinction between effects of short-chain PFAS vs. long-chain PFAS in aquatic and terrestrial receptors is described in reviews by the Australian National Industrial Chemicals Notification and Assessment Scheme (IMAP 2017a, 2017b, and 2017c vs. IMAP 2017d, 2017e, 2017f, and 2017g, respectively), the Environmental Protection Agency (USEPA 2017), and the Organisation for Economic Cooperation and Development (OECD 2015). Both short and long-chain PFAS are environmentally persistent. However, in contrast to short-chain PFAS, long-chain PFAS tends to bioaccumulate within ecological receptors, often eliciting adverse toxicological effects. On the other hand, short-chain PFAS are more water soluble and show greater mobility in the environment, relative to long-chain PFAS. Furthermore, it is also clear that the estimation of potential risks to receptors is dose dependent. As a result, the amount of contaminant in environmental matrices (e.g., soil, sediment, surface water), as well as those concentrations in dietary prey items, will ultimately contribute to ecological risk.

6.0 References

Abdellatif, A.G., Preat, V., Vamecq, J., Nilsson, R., and Roberfroid, M. (1990). *Peroxisome proliferation and modulation of rat liver carcinogenesis by 2,4-dichlorophenoxyacetic acid, 2,4,5-trichlorophenoxyacetic acid, perfluorooctanoic acid and nafenopin.* Carcinogenesis. 11(11), 1899-1902.

Bossi, R., Riget, F.F., Dietz, R., Sonne, P.F., Dam, M., and Vorkamp, K. (2005). *Preliminary screening of perfluorooctane sulfonate (PFOS) and other fluorochemicals in fish, birds and marine mammals from Greenland and the Faroe Islands.* Environmental Pollution. 136, 323-329.

Boudreau, T.M., Sibley, P.K., Mabury, S.A., Muir, D.G.C., and Solomon, K.R. (2003). *Laboratory evaluation of the toxicity of perfluorooctane sulfonate (PFOS) on Selenastrum capricornutum, Chlorella vulgaris, Lemna gibba, Daphnia magna, and Daphnia pulicaria.* Archives of Environmental Contamination and Toxicology. 44, 307-313.

Buck, R.C., Franklin, J., Berger, U., Conder, J.M., Cousins, I.T., de Voogt, P., Jensen, A.A., Kannan, K., Mabury, S.A., and van Leeuwen, S.P.J. (2011). *Perfluoroalkyl and polyfluoroalkyl substances in the environment: Terminology, classification, and origins.* Integr. Environ. Assess. Manag. 7(4), 513-541.

Butenhoff, J.L., Kennedy, G.L., Frame, S.R., O'Connor, J.C., and York, R.G. (2004a). *The reproductive toxicology of ammonium perfluorooctanoate (APFO) in the rat.* Toxicology. 196, 95-116.

Butenhoff, J.L., Kennedy, G.L., Hinderliter, P.M., Lieder, P.H., Jung, R., Hansen, K.J., Gorman, G.S., Noker, P.E., and Thomford, P.J. (2004b). *Pharmacokinetics of perfluorooctanoate in cynomolgus monkeys.* Toxicol. Sci. 82, 394-406.

Case, M.T., York, R.G., and Christian, M.S. (2001). *Rat and rabbit oral developmental toxicology studies with two perfluorinated compounds.* In. J. Toxicol. 20, 101-109.

Cheng, Y., Cui, Y., Chen, H.M., and Xie, W.P. (2011). *Thyroid disruption effects of environmental level perfluorooctane sulfonates (PFOS) in Xenopus laevis.* Ecotoxicology. 20(8), 2069-2078.

Conder, J.M., Hoke, R.A., De Wolf, W., Russell, M.H., and Buck, R.C. (2008). *Are PFCAs bioaccumulative? A critical review and comparison with regulatory criteria and persistent lipophilic compounds*. Environ. Sci. Technol. 42, 995-1003.

Das, K.P., Grey, B.E., Zehr, R.D., Wood, C.R., Butenhoff, J.L., Chang, S.C., Ehresman, D.J., Tan, Y.M., and Lau, C. (2008). *Effects of perfluorobutyrate exposure during pregnancy in the mouse.* Toxicol. Sci. 105, 173-181.

De Silva, A.O., Spencer, C., Scott, B.F., Backus, S. and Muir, D.C.G. (2011). *Detection of a cyclic perfluorinated acid, perfluoroethylcyclohexane sulfonate, in the great lakes of North America.* Environ. Sci. Technol. 45, 8060-8066.

Ding, G., Wouterse, M., Baerselman, R., and Peijnenburg, W.J.G.M. (2012). *Toxicity of Polyfluorinated and perfluorinated compounds to lettuce (Lactuca sativa) and green algae (Pseudokirchneriella subcapitata).* Arch. Environ. Contam. Toxicol. 62(1), 49-55.

Drottar K.R., Krueger H.O. (2000). *PFOS: A 96-hr static acute toxicity test with the fathead minnow (Pimephales promelas).* Wildlife International, Ltd., Project No. 454-102, EPA Docket AR226-0083.

Ecology. (2007). *Model Toxics Control Act statute and regulation, Chapter 173-340 WAC.* (Ecology Publication No. 94-06). Lacey, WA: Washington State Department of Ecology, Toxics Cleanup Program. Document can be found at: http://apps.leg.wa.gov/WAC/default.aspx?cite=173-340

Ecology. (2017). *Survey of Per- and Poly-fluoroalkyl Substances (PFAS) in Rivers and Lakes, 2016*. (Ecology Publication No. 17-03-021). Lacey, WA: Washington State Department of Ecology, Environmental Assessment Program. Document can be found at: https://fortress.wa.gov/ecy/publications/documents/1703021.pdf

ECCC - Environment and Climate Change Canada. (2017). *Ecological screening assessment report on perfluorooctane sulfonate, its salts and its precursors that contain the C8F17SO2 or C8F17SO3, or C8F17SO2N moiety*. Document can be found at: https://www.ec.gc.ca/lcpe-cepa/default.asp?lang=En&n=98B1954A-1&offset=1&toc=hide

Gortner, E.G. (1980). *Oral teratology study of FC-95 in rats.* Riker Laboratories, Inc. Experiment Number: 0680TR0008, December, 1980. AR-226 226-0016 US Environmental Protection Agency.

Guelfo, J.L., and Higgins, C.P. (2013). Subsurface transport of perfluoroalkyl acids at aqueous film-forming foam (AFFF)-impacted sites. Enivron. Sci. Technol. 47, 4164-4171.

Harris, M.W., and Birnbaum, L.S. (1989). *Developmental toxicity of Perfluorodecanoic acid in C57BL/6N mice*. Fundam. Appl. Toxicol. 12, 442-448.

Houde, M., Bujas, T.A.D., Small, J., Wells, R.S., Fair, P.A., Bossart, G.D., Solomon, K.R., and Muir, D.C.G. (2006). *Biomagnification of Perfluoroalkyl compounds in the bottlenose dolphin (Tursiops truncates) food web.* Environ. Sci. Technol. 40, 4138-4144.

IMAP. (2017a). *Environment Tier II Assessment for perfluorobutanesulfonic acid and its direct precursors*. Accelerated Assessment of Industrial Chemicals in Australia. Document can be found at: https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-assessments/tier-ii-environment-assessments/perfluorobutanesulfonic-acid-and-its-direct-precursors#EnvironmentalEffects

IMAP. (2017b). *Environment Tier II Assessment for indirect precursors of perfluorobutanesulfonate (PFBS).* Accelerated Assessment of Industrial Chemicals in Australia. Document can be found at: https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-assessments/tier-ii-environment-assessments/indirect-precursors-of-perfluorobutanesulfonate-pfbs#EnvironmentalEffects

IMAP. (2017c). *Environment Tier II Assessment for short-chain perfluorocarboxylic acids and their direct precursors.* Accelerated Assessment of Industrial Chemicals in Australia. Document can be found at: https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-assessments/tier-ii-environment-assessments/short-chain-perfluorocarboxylic-acids-and-their-direct-precursors

IMAP. (2017d). *Environment Tier II Assessment for direct precursors to perfluoroheptanesulfonate (PFHpS), perfluorohexanesulfonate (PFHxS) and perfluoropentanesulfonate (PFPeS).* Accelerated Assessment of Industrial Chemicals in Australia. Document can be found at: https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-assessments/tier-ii-environment-assessments/direct-precursors-to-perfluoroheptanesulfonate-pfhps,-perfluorohexanesulfonate-pfhxs-and-perfluoropentanesulfonate-pfpes#EnvironmentalEffects

IMAP. (2017e). *Environment Tier II Assessment for direct precursors to perfluorooctanesulfonate (PFOS).* Accelerated Assessment of Industrial Chemicals in Australia. Document can be found at: https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-assessments/tier-ii-environment-assessments/direct-precursors-to-perfluorooctanesulfonate-pfos#EnvironmentalEffects

IMAP. (2017f). *Environment Tier II Assessment for perfluorooctanoic acid (PFOA) and its direct precursors.* Accelerated Assessment of Industrial Chemicals in Australia. Document can be found at: https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-assessments/tier-ii-environment-assessments/perfluorooctanoic-acid-and-its-direct-precursors#EnvironmentalEffects

IMAP. (2017g). *Environment Tier II Assessment for direct precursors to perfluorocyclohexane sulfonate and related perfluoroalkylcyclohexane sulfonates.* Document can be found at: https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-assessments/tier-ii-environment-assessments/direct-precursors-to-perfluorocyclohexane-sulfonate-and-related-perfluoroalkylcyclohexane-sulfonates

ITRC. (2018). *Environmental fate and transport for per- and polyfluoroalkyl susbstances.* Interstate Technology and Regulatory Council, Washington, D.C. Document can be found at: https://pfas-1.itrcweb.org/wp-content/uploads/2018/03/pfas_fact_sheet_fate_and_transport__3_16_18.pdf

Ji, K., Kim, Y., Oh, S., Ahn, B., Jo, H., and Choi, K. (2008). *Toxicity of perfluorooctane sulfonic acid and perfluorooctanoic acid on freshwater macroinvertebrates (Daphnia magna and Moina macrocopa) and fish (Oryzias latipes).* Environ. Toxicol. Chem. 27(10), 2159-2168.

Kelly, B.C., Ikonomou, M.G., Blair, J.D., Surridge, B., Hoover, D., Grace, R., and Gobas, P.C. (2009). *Perfluoroalkyl contaminants in an arctic marine food web: Trophic magnification and wildlife exposure.* Environ. Sci. Technol. 43, 4037-4043.

Kennedy, G.L., Butenhoff, J.L., Olsen, G.W., O'Connor, J.C., Seacat, A.M., Perkins, R.G., Biegel, L.B., Murphy, S.R., and Farrar, D.G. (2004). *The toxicology of perfluorooctanoate*. Critical Reviews in Toxicology. 34(4), 351-384.

Key, B.D., Howell, R.D., and Criddle, C.S. (1997). *Fluorinated organic in the biosphere*. Environ. Sci. Technol. 31, 2445-2454.

Latala, A.M., Nedzi, M., and Stepnowski, P. (2009). *Acute toxicity assessment of perfluorinated carboxyl acids towards the Baltic microalgae.* Environ. Toxicol. Pharmacol. 28(2), 167-171.

Lau, C., Thibodeaux, J.R., Hanson, R.G., Narotsky, M.G., Rogers, J.M., Lindstrom, A.B., and Strynar, M.J. (2006). *Effects of perfluorooctanoic acid exposure during pregnancy in the mouse.* Toxicol. Sci. 90, 510-518.

Lau, C., Anitole, K., Hodes, C., Lai, D., Pfahles-Hutchens, A., and Seed, J. (2007). *Perfluoroalkyl acids: A review of monitoring and toxicological findings.* Toxicological Sciences. 99(2), 366-394.

Li, Mei-Hui. (2009). *Short Communication: Toxicity of perfluorooctane sulfonate and perfluorooctanoic acid to plants and aquatic invertebrates.* Environmental Toxicology DOI. 10.1002/tox, 95-101.

Lindstrom, A.B., Strynar, M.J., and Libelo, E.L. (2011). *Polyfluorinated compounds: past, present, and future.* Environ. Sci. Technol. 45, 7954-7961.

Loi, E., I., Yeung, L.W.Y., Taniyasu, S., Lam, P.K.S., Kannan, K., and Yamashita, N. (2011). *Trophic magnification of poly and perfluorinated compounds in a subtropical food web.* Environ. Sci. Technol. 45, 5506-5513.

Macon, M.B., Villanueva, L.R., Gibbs-Tatum, K., Zehr, R.D., Strynar, M.J., Stanko, J.P., White, S.S., Helfant, L., and Fenton, S. (2011). *Prenatal perfluorooctanoic acid exposure in CD-1 mice: Low-dose developmental effects and internal dosimetry.* Toxicological Sciences. 122(1) 134-145.

Martin, J.W., Mabury, S.A., Solomon, K.R., and Muir D.C.G. (2003). *Bioconcentration and tissue distribution of Perfluorinated acids in rainbow trout (Oncorhynchus mykiss).* Environ. Sci. Technol. 22, 196-204.

Mhadhbi, L., Rial, D., Perez, S., and Beiras, R. (2012). *Ecological risk assessment of perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS) in marine environment using Isochrysis galbana, Paracentrotus lividus, Siriella armata, and Psetta maxima.* J. Environ. Monit. 14(5), 1375-1382.

Moody C.A., Martin J.W., Kwan W.C., Muir D.C.G., Mabury S.A. (2002). *Monitoring Perfluorinated Surfactants in Biota and Surface Water Samples Following an Accidental Release of Fire-Fighting Foam into Etobicoke Creek.* Environ. Sci. Technol. 36, 545-551.

MPCA – Minnesota Pollution Control Agency. (2013). *Minnesota Pollution Control Agency Aquatic Life Criteria.* Document can be found at: https://www.pca.state.mn.us/sites/default/files/pfos-mississippiriver.pdf

Newsted, J.L., Coady, K.K., Beach, S.A., Butenhoff, J.L., Gallagher, S., and Giesy, J.P. (2007). *Effects of perfluorooctane sulfonate on mallard and northern bobwhite quail exposed chronically via the diet.* Environ. Toxicol. Pharmacol. 23, 1-9.

OECD. (2008). *SIDS initial assessment report for ammonium perfluorooctanoate and perfluorooctanoic acid.* Organisation for Economic Cooperation and Development. Paris, France. Document can be found at: http://webnet.oecd.org/HPV/UI/handler.axd?id=1f391916-96ba-46f6-a7ce-c96712da3b7e

OECD. (2013). OECD/UNEP Global PFC Group: synthesis paper on per- and polyfluorinated chemicals (PFCs). Organisation for Economic Cooperation and Development. Paris, France. Document can be found at: https://www.oecd.org/env/ehs/risk-management/PFC_FINAL-Web.pdf

OECD. (2015). *Working towards a global emission inventory of PFAS: Focus on PFCAS – Status quo and the way forward.* Organisation for Economic Cooperation and Development. Paris, France. Document can be found at: https://www.oecd.org/chemicalsafety/risk-management/Working%20Towards%20a%20Global%20Emission%20Inventory%20of%20PFAS.pdf

Peden-Adams, M.M., Stuckey, J.E., Gaworecki, K.M., Berger-Ritchie, J., Bryant, K., Jodice, P.G., Scott, T.R., Ferrario, J.B., Guan, B., Vigo, C., Boone, J.S., McGuinn, W.D., DeWitt, J.C., and Keil, D.E. (2009). *Developmental toxicity in white leghorn chickens following in ovo exposure to perfluorooctane sulfonate (PFOS).* Reprod. Toxicol. 27, 307-318.

Presher, D., Gross, U., Wotzka, J., Txcheu-Schlueter, M., and Starke, W. (1985). *Environmental behavior of fluoro surfactants: Part 2: Study on biochemical degradability.* Acta Hydrochim. Hydrobiol. 13, 17-24.

Ramboll Environ. (2016). *Companion report to Ramboll Environ's January 2014 assessment for POP criteria for specific short-chain perfluorinated alkyl substances.* Ramboll Environ. Arlington, VA.

Reiner, J.L., and Place, B.J. (2015). Chapter 5: Perfluorinated alkyl acids in wildlife. In Dewitt, J.C. (Eds.), *Toxicological effects of perfluoroalkyl and polyfluoroalkyl substances* (pp. 127 – 150). Location: Springer International Publishing.

Rich, C.D., Blaine, A.C., Hundal, L., Higgins, C.P. (2015). *Bioaccumulation of perfluoroalkyl acids by earthworms (Eisenia fetida) exposed to contaminated soils.* Environ. Sci. Technol. 49(2), 881-888.

Rodea-Palomares, I., Makowski, M., Gonzalo, S., Gonzalez-Pleiter, M., Leganes, F., and Fernandez-Pinas, F. (2015). *Effect of PFOA/PFOS pre-exposure on the toxicity of the herbicides 2,4-D, Atrazine, Diuron and Paraquat to a model aquatic photosynthetic microorganism.* Chemosphere. 139, 65-72.

Sample, B.E., Opresko, D.M., and Suter II, G.W. (1996). *Toxicological Benchmarks for Wildlife: 1996 Revision.* Prepared by the Risk Assessment Program. Health Sciences Research Division, Oak Ridge Tennessee, for the U.S. Department of Energy, Office of Environmental Management.

Spachmo, B., and Arukwe, A. (2012). *Endocrine and developmental effects in Atlantic salmon (Salmo salar) exposed to perfluorooctane sulfonic or perfluorooctane carboxylic acids.* Aquat. Toxicol. 108, 112-124.

Stahl, T., Heyn, J., Thiele, H., Hüther, J., Failing, K., Georgii, S., and Brunn, H. (2009). *Carryover of perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) from soil to plants.* Arch. Environ. Contam. Toxicol. 57, 289-298.

Stahl, T., Mattern, D., and Brunn, H. (2011). *Toxicology of perfluorinated compounds.* Environmental Sciences Europe. 23:38.

USEPA. (2009). *Long-chain perfluorinated chemicals (PFCs) action plan.* U.S. Environmental Protection Agency.

USEPA. (2012). *Ecological Effects Test Guidelines, OCSPP 850.4100: Seedling Emergence and Seedling Growth.* Office of Chemical Safety and Pollution Prevention, EPA 712-C-012.

USEPA. (2017). *Risk management for per- and polyfluoroalkyl substances (PFAS) under TSCA.* United States Environmental Protection Agency. Document can be found at: https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/risk-management-and-polyfluoroalkyl-substances-PFAS

Wambaugh, J. (2015). Chapter 14: Dosimetric anchoring of toxicological studies. In Dewitt, J.C. (Eds.), *Toxicological effects of perfluoroalkyl and polyfluoroalkyl substances* (pp. 337 – 361). Location: Springer International Publishing.

Wang, Z., DeWitt, J.C., Higgins, C.P., and Cousins, I.T. (2017). *A never-ending story of per- and polyfluorinated substances (PFASs)?* Environ. Sci. Technol. 51(5), 2508-2518.

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

White, S.S., Fenton, S.E., and Hines, E.P. (2011). *Endocrine disrupting properties of perfluorooctanoic acid*. J Steroid Biochem. Mol. Biol. 127(1-2), 16-26.

Wolf, D.C., Moore, T., Abbott, B.D., Rosen, M.B., Das, K.P., Zehr, R.D., Lindstrom, A.B., Strynar, M.J., and Lau, C. (2008). *Comparative hepatic effects of perfluorooctanoic acid and WY 14,643 in PPAR-α knockout and wild-type mice.* Society of Toxicology Pathology. 36, 632-639.

Yanai, J., Dotan, S., Goz, R., Pinkas, A., Seidler, F.J., Slotkin, T.A., and Zimmerman, F. (2008). *Exposure of developing chicks to perfluorooctanoic acid induces defects in prehatch and early posthatch development.* J. Toxicol. Environ. Health A. 71, 131- 133.

7.0 Acronyms

General Terms:

BAF	Bioaccumulation Factor
BCF	Bioconcentration Factor
CAP	Chemical Action Plan
$EC_{(...)}$	Percent effect concentration
LOEC	Lowest observed effects concentration
NOAEL	No observed adverse effects level
NOEC	No observed effects concentration

PFAS-related Terms:

6:2 FTAC	6:2 Fluorotelomer acrylate
6:2 FTMAC	6:2 Fluorotelomer methacrylate
6:2 FTOH	6:2 Fluorotelomer alcohol
FT	Fluorotelomer
PASF	Perfluoroalkane sulfonyl fluoride
PFAA	Perfluoroalkyl acid
PFAS	Per- and Polyfluoroalkyl substances
PFBA	Perfluorobutanoic acid
PFBS	Perfluorobutane sulfonate
PFC	Perfluoroalkyl contaminants
PFCA	Perfluoroalkyl carboxylic acid
PFD	Perfluorodecanoate
PFDA	Perfluorodecanoic acid
PFDo	Perfluorododecanoate
PFDoA	Perfluorododecanoic acid
PFECHS	Perfluoroethylcyclohexanesulfonate
PFHp	Perfluoroheptanoate
PFHpS	Perfluoroheptanesulfonate
PFHx	Perfluorohexanoate
PFHxA	Perfluorohexanoic acid
PFHxS	Perfluorohexane sulfonate
PFN	Perfluorononanoate
PFNA	Perfluorononanoic acid
PFO	Perfluorooctanoate
PFOA	Perfluorooctanoic acid
PFOS	Perfluorooctane sulfonate
PFOSA	Perfluorooctane sulfonamide
PFPeS	Perfluoropetanesulfonate
PFPn	Perfluoropentanoate
PFS	Perfluoroalkyl substance
PFSA	Perfluoroalkyl sulfonic acid

PFT	Perfluorotetradecanoate
PFTri	Perfluorotridecanoate
PFU	Perfluoroundecanoate
PFUnA/PFUnDA	Perfluoroundecanoic acid