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Supporting Document:

Ecological State of the Science Report on

Short-chain (C4–C7) Perfluorocarboxylic Acids (SC-PFCAs)
Short-chain (C4–C7) Perfluorosulfonic Acids (SC-PFSAs)
Long-chain (C9–C20) Perfluorosulfonic Acids (LC-PFSAs)

Information in Support of the Draft State of Per- and Polyfluoroalkyl Substances (PFAS) Report

Environment and Climate Change Canada

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Canada

Table of Contents

List of Tables and Figures

Abbreviation List[1]

AFFF	*Aqueous Film-Forming Foam*
BAF	*Bioaccumulation Factor*
BCF	*Bioconcentration Factor*
BMF	*Biomagnification Factor*
CEPA	*Canadian Environmental Protection Act, 1999*
DSL	*Domestic Substances List*
ECCC	*Environment and Climate Change Canada*
EU	*European Union*
FTO	*Fluorotelomer olefin*
FTOH	*Fluorotelomer alcohol*
Kow	*Octanol-water partitioning coefficient*
Kpw	*Protein-water partitioning coefficient*
Kmw	*Membrane-water partitioning coefficient*
LC-PFCA	*Long-chain perfluorocarboxylic acids*
LC-PFSA	*Long-chain perfluorosulfonic acids*
NDSL	*Non-Domestic Substances List*
PFAA	*Perfluoroalkyl acids*
PFAS	*Per- and poly-fluoroalkyl substances*
PFCA	*Perfluorocarboxylic acids*
PFPiA	*Perfluorophosphinic acids*
PFSiA	*Perfluorosulfinic acids*
PFSA	*Perfluorosulfonic acids*
PFSE	*N-alkyl perfluoroalkylsulfonamidoethanol*
SC-PFCA	*Short-chain perfluorocarboxylic acids*
SC-PFSA	*Short-chain perfluorosulfonic acids*
TMF	*Trophic Magnification Factor*
PFBA	*Perfluorobutanoic acid*
PFPeA	*Perfluoropentanoic acid*
PFHxA	*Perfluorohexanoic acid*
PFHpA	*Perfluoroheptanoic acid*
PFOA	*Perfluorooctanoic acid*
PFNA	*Perfluorononanoic acid*
PFDA	*Perfluorodecanoic acid*
PFUnDA	*Perfluoroundecanoic acid*
PFDoDA	*Perfluorododecanoic acid*
PFTrDA	*Perfluorotridecanoic acid*
PFTeDA	*Perfluorotetradecanoic acid*
PFPeDA	*Perfluoropentadecanoic acid*
PFHxDA	*Perfluorohexadecanoic acid*
PFHpDA	*Perfluoroheptadecanoic acid*
PFODA	*Perfluorooctadecanoic acid*
PFNDA	*Perfluorononadecanoic acid*
PFICOA	*Perfluoroeicosanoic acid*
PFHICOA	*Perfluoroheneicosanoic acid*
PFBS	*Perfluorobutane sulfonic acid*
PFPeS	*Perfluoropentane sulfonic acid*
PFHxS	*Perfluorohexane sulfonic acid*
PFHpS	*Perfluoroheptane sulfonic acid*
PFOS	*Perfluorooctane sulfonic acid*
PFNS	*Perfluorononane sulfonic acid*
PFDS	*Perfluorodecane sulfonic acid*
PFUnDS	*Perfluoroundecane sulfonic acid*
PFDoDS	*Perfluorododecane sulfonic acid*
PFTrDS	*Perfluorotridecane sulfonic acid*
PFTeDS	*Perfluorotetradecane sulfonic acid*
PFPeDS	*Perfluoropentadecane sulfonic acid*

[1] The abbreviations for PFCA and PFSA could represent either the acid or anionic forms of the chemicals.

PFHxDS	*Perfluorohexadecane sulfonic acid*
PFHpDS	*Perfluoroheptadecane sulfonic acid*
PFODS	*Perfluorooctadecane sulfonic acid*
PFNDS	*Perfluorononadecane sulfonic acid*
PFICOS	*Perfluoroicosane sulfonic acid*

Preface

This document contains additional information that is summarized or referenced in the Draft State of Per-and Polyfluoroalkyl Substances (PFAS) Report. Relevant data were identified up to March 2022.

In the Draft State of PFAS Report, this document is referenced as:

[ECCC] Environment and Climate Change Canada. 2023. Supporting Document: Ecological State of the Science Report on Short-chain PFCAs, Short-chain PFSAs, and Long-chain PFSAs. Gatineau (QC): Government of Canada.

In-text reference: (ECCC 2023)

The supporting working documentation for the figures in this document is available upon request by email from substances@ec.gc.ca.

1.0 Introduction

Since the 1950s, per- and polyfluoroalkyl substances (PFAS) have been widely used in industrial and consumer applications such as aqueous film-forming foams (AFFF) and the surface treatment of textiles, carpets, and papers where there is a need for extremely low surface energy, surface tension, and/or durable water- and oil-repellency. Their presence in the environment is due to anthropogenic activities and no natural sources exist.

For over a decade, PFOS and PFOA have attracted significant attention as contaminants of global concern. PFOS and PFOA are persistent, bioaccumulative, widespread in the global environment, found in remote areas (due to long-range transport of PFOA and PFOS, and their precursors), and can cause various adverse effects on wildlife at relevant environmental concentrations. Both substances have undergone various regulatory actions in many countries. In Canada, ecological risk assessments for perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA), and long-chain perfluorocarboxylic acids (LC-PFCAs; C9–C21) conducted under the *Canadian Environmental Protection Act, 1999* (CEPA) have concluded that these substances are harmful to the environment (EC, HC 2012; Environment Canada 2006; Environment Canada 2012). Consequently, PFOS, PFOA, and the LC-PFCAs, their salts, and their precursors were placed on the List of Toxic Substances (Schedule 1) under CEPA. Since 2008, Canada's regulations prohibit the manufacture, use, sale, offer for sale, and import of PFOS and products containing PFOS, with limited exemptions. Since 2016, Canada's regulations also prohibit the manufacture, use, sale, offer for sale, and import of PFOA and LC-PFCAs and products containing PFOA and LC-PFCAs, with limited exemptions. On May 14, 2022, Canada proposed regulations that would further restrict PFOS, PFOA, and LC-PFCAs by removing or providing time limits for most of the remaining exemptions.

Due in part to various regulatory actions worldwide (including the listing of PFOS and PFOA as Persistent Organic Pollutants under the Stockholm Convention), other perfluorinated substances (for example, SC-PFCAs, SC-PFSAs, and potentially LC-PFSAs) have been used as replacements for PFOA, PFOS, and LC-PFCAs. Initially, the shorter-chain replacement substances were thought to be alternatives with an overall lower bioaccumulation and toxicity potential on the basis of standard toxicity test results for freshwater aquatic test species such as fish, daphnia, and algae. However, it has been more recently recognized—for example, in statements from a range of experts—that these shorter chain PFAS may have impacts similar to those of PFOS and PFOA (Helsingør Statement, Scheringer et al. 2014; Madrid Statement, Blum et al. 2015; Zurich Statement 2018, Ritscher et al. 2018).

Many SC-PFCAs, SC-PFSAs, and LC-PFSAs, along with their precursors, have been detected in the Canadian environment and biota, including in the Canadian Arctic and the Great Lakes. The presence of these substances in Canada may be the result of releases from imported products or manufactured items containing these substances,

which make their way into the Canadian environment. In remote environments in Canada, their presence may be due to the long-range transport (LRT) of precursors from within Canada or from international sources that may be transported and subsequently transformed to the acids.

SC-PFCAs, SC-PFSAs, and LC-PFSAs are considered to be as persistent as PFOS, PFOA, and the LC-PFCAs due to the carbon-fluorine bond, which is one of the strongest covalent bonds (about 108–120 kcal/mole). This bond results in these substances being extremely stable and generally resistant to degradation by acids, bases, oxidants, reductants, photolytic processes, microbes, and metabolic processes. Some SC-PFCAs/PFSAs and LC-PFSAs have also been shown to biomagnify in upper trophic level wildlife to a degree comparable to the substances they are meant to replace (that is, PFOS, PFOA, and LC-PFCAs). The time frame over which these substances remain in the environment is expected to be extremely long and has not been meaningfully quantified. It is often difficult to quantify the ecological risks associated with persistent and bioaccumulative substances in the environment, but they are generally acknowledged to have the potential to cause serious, irreversible impacts on wildlife populations in the long term (Environment Canada 2006; MacLeod et al. 2014). In addition, SC-PFCAs/PFSAs and LC-PFSAs may have the capacity to cause various adverse effects in wildlife similar to those of the PFAS that they replaced.

This report provides a summary of data available from the ecological-related science literature (up to March 2022) for PFAS in the following three subgroups:

1. Short-chain (C4–C7) perfluorocarboxylic acids (SC-PFCAs), their salts, and precursors
2. Short-chain (C4–C7) perfluorosulfonic acids (SC-PFSAs), their salts, and precursors
3. Long-chain (C9–C20) perfluorosulfonic acids (LC-PFSAs), their salts, and precursors

This includes data and information on environmental persistence, bioaccumulation and trophic magnification potential, mobility, Canadian environmental monitoring data, and potential for adverse effects in the environment. Most of the content of this report focuses on SC-PFCAs/PFSAs, some of which have been used as replacements for PFAS that have been subject to restrictions in Canada and/or internationally. LC-PFSAs are believed to have more limited use as substitutes for these restricted substances, and are covered to a lesser extent in this document largely due to the lack of information on them available in the literature (except for limited information on PFDS and PFNS).

This report refers to long-chain and short-chain PFAS, where long-chain substances have a carbon chain length of 8 (C8) or higher and short-chain substances have a carbon chain length of 7 (C7) or lower. PFOS and PFOA, which are both C8, are sometimes discussed separately from other long-chain PFAS in this report. Since PFOS and PFOA are well studied, data for these substances are sometimes presented for purposes of comparison. PFOS and PFOA are also discussed separately from long-

chain PFAS with regard to past regulatory activities. Reports by other authors (for example, the OECD) may refer to perfluorinated alkyl sulfonates that have 6 (C6) or more fully fluorinated carbons (for example, PFHxS) as long-chain PFAS; however, the definitions of short-chain and long-chain PFAS used in this report are consistent with other Government of Canada publications.

For the purpose of this report, PFAAs refer to the perfluoroalkyl acids (for example, PFCAs and PFSAs). It is these stable forms that are referred to as the moieties of interest in this document. The abbreviations for individual PFCAs and PFSAs could represent either the acid or anionic forms of the chemicals; however, under environmental conditions, PFAAs exist predominantly in their anionic form.

2.0 Substance identity

By definition, PFAS contain at least one fully fluorinated methyl or methylene carbon (without any H/Cl/Br/I atom attached to it), that is, with a few noted exceptions, any chemical with at least a perfluorinated methyl group (–CF3) or a perfluorinated methylene group (–CF2–) is a PFAS (OECD 2021). The length of the fluorinated carbon chain of most of the perfluorinated substances that are monitored or detected in the environment ranges from 3 to 20 fluorinated carbons. These alkyl chains are attached to various functional groups. For example, perfluorocarboxylic acids (PFCAs) and perfluorosulfonic acids (PFSAs) are organic acids comprised of a fluorinated carbon chain terminated by a carboxylate or sulfonate functional group, respectively. The conjugate anion chemical structures for perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS) are provided as examples in Figure 1. A number of different configurations are possible for each perfluorinated acid, including the simple linear configuration and branched isomers.

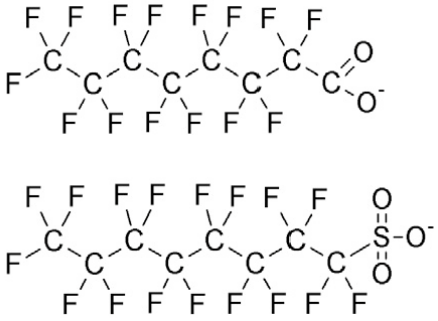

Figure 1. Conjugate anion structures for PFOA (top) and PFOS (bottom)

In terms of composition and nomenclature, the term “perfluorinated” indicates that all hydrogen atoms directly attached to the carbon chain have been replaced by fluorine atoms. The term “poly-fluorinated” indicates that only some of the hydrogen atoms have been replaced with fluorine atoms. The expression “C#” is generally used to define the total number of fluorinated carbons present in a PFSA molecule or the total number of carbons present in a PFCA molecule (which includes both fluorinated carbons and the

carbonyl group). For example, C9 PFSA has 9 carbons, all of which are fluorinated. However, C9 PFCA has 9 carbons, of which 8 are fluorinated and one is part of the carbonyl functional group.

In 2006, Canada's Domestic Substance List (DSL, circa 1984 to 1986)[2] was reviewed to identify PFAS substances (including salts and precursors) included on it at the time. This review included consideration of their potential to transform to moieties of interest on the basis of expert judgement, chemical structures, and the biodegradation estimation model, CATABOL (c2004 to 2008)[3] (Jaworska et al. 2002; Dimitrov et al. 2004, 2007, 2011). CATABOL predicted metabolites by simulating the OECD 302C 28-day biodegradation test and was trained on the basis of MITI (Japan's Ministry of International Trade and Industry) biodegradation test results. Although this model was designed to accommodate perfluorinated substances, some degradation products predicted by CATABOL may be of lower reliability or relevance in the environment, given the very limited perfluorinated degradation data in its training set. Appendices A, B, and C list the salts and precursors to SC-PFCAs/PFSAs and LC-PFSAs identified on the DSL on the basis of CATABOL modelling, expert judgement, and chemical structure as of 2006. Appendix D provides a list of precursors where empirical evidence is available, corroborating the modelling regarding their degradation potential. These lists are not considered exhaustive. It is also noted that other jurisdictions (for example, Organisation of Economic Co-operation and Development [OECD]; United States Environmental Protection Agency [US EPA]) have developed lists that may differ from those included in this report (OECD 2018).

Where suitable, the PFAS on the DSL were assigned to the following subgroups (denoted by boxes highlighted in grey in Figure 2):

1. PFOS (C8)
2. PFOA (C8)
3. LC (C9–C21) PFCAs
4. SC (C4–C7) PFCAs
5. SC (C4–C7) PFSAs
6. LC (C9–C20) PFSAs

[2] The DSL (circa 1984–1986) was a list of approximately 23 000 substances manufactured in, imported into, or used in Canada on a commercial scale. The DSL was based on substances present in Canada, under certain conditions, between January 1, 1984, and December 31, 1986. The DSL is periodically updated and now contains more than 28 000 substances.

[3] It should be noted that CATABOL has now been upgraded to the CATALOGIC 301C model (Dimitrova et al. 2017).

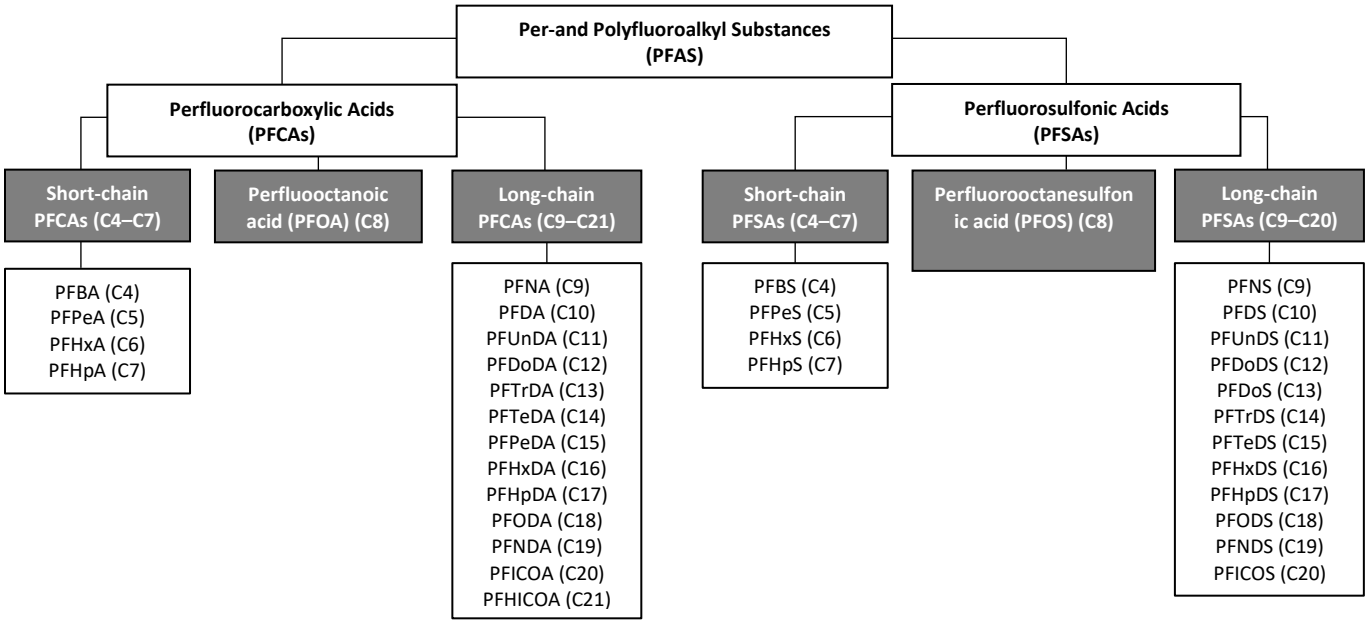

Figure 2. Substance subgroups for perfluorinated substances on Canada's Domestic Substances List.

Canada undertook an assessment of and identified the ecological risk for three groups of PFAS: PFOS, PFOA, and LC-PFCAs, and their salts and precursors (Environment Canada 2006, 2012; EC, HC 2012). As PFOS, PFOA, and the LC-PFCAs are subject to restrictions in Canada, this report is focused on the following three chemical sub-groupings (with data for the more heavily studied PFOS and PFOA substances included for comparative purposes):

1. SC-PFCAs (C4–C7), their salts and precursors
2. SC-PFSAs (C4–C7), their salts and precursors
3. LC-PFSAs (C9–C20), their salts and precursors

Consistent with Canada's definitions for precursors from past ecological risk assessments for PFOS, PFOA, and LC-PFCAs (Environment Canada 2006, 2012; EC, HC 2012), the precursors for the SC-PFCAs/PFSAs and LC-PFSAs are defined as follows:

1. Precursors to the SC-PFSAs: Compounds that contain the $C_nF_{2n+1}SO_2$ moiety where $4 \leq n \leq 7$
2. Precursors to the LC-PFSAs: Compounds that contain the $C_nF_{2n+1}SO_2$ moiety where $9 \leq n \leq 20$
3. Precursors to the SC-PFCAs: Compounds containing the perfluoroalkyl moiety (C_nF_{2n+1}) where $3 \leq n \leq 6$ that is directly bonded to any chemical moiety other than a fluorine, chlorine, or bromine atom

The approach to identifying precursors taken in this report is consistent with that used in past reports (EC, HC 2012; Environment Canada 2006; Environment Canada 2012), where precursors can be any substance that contains the moiety of interest and that can ultimately transform through reactions such as oxidation (for example, of precursors such as volatile alcohols in the atmosphere, or of precursors in wastewater treatment systems), metabolism, or hydrolysis to the final transformation product, that is, the moiety of interest. Additionally, to be consistent with ECCC's past reports for PFOS, PFOA, and LC-PFCAs (EC, HC 2012; Environment Canada 2006; Environment Canada 2012), this report does not directly consider the salts or precursors on the basis of their unique identities or properties. Rather, the contribution of precursors and salts to the environmental presence of SC-PFCAs/PFSAs and LC-PFSAs is recognized as salts and precursors contribute to the total presence of SC-PFCAs/PFSAs and LC-PFSAs in the Canadian environment, including biota, following transformation or dissolution.

3.0 Fate and behaviour in the aquatic and terrestrial environment

3.1 General characteristics

In general, most PFAS share a combination of properties such as thermal and chemical inertness, persistence, low solubility in polar and non-polar organic solvents, high density, fluidity, compressibility, and high dielectric constants (Lehmler et al. 2001). Appendices E and F show the available empirical physical-chemical data for the SC-PFCAs/PFSAs. No empirical data were found for the LC-PFSAs, and modelling was not applicable.

Chemical structure, functional groups, pKa, partitioning coefficients, and surfactant or surface-active properties impact the fate and behaviour of perfluorinated substances in the environment and have impacts on the traditional approaches to evaluating bioaccumulation and toxicity. Some key points on general characteristics for the SC-PFCAs/PFSAs and LC-PFSAs are identified in the box below. Specific information on the physical-chemical properties for precursors are not part of the scope of this report as precursors are considered only on the basis of their contribution to the total loading of moieties of interest (SC-PFCAs/PFSAs and LC-PFSAs) to the environment.

KEY POINTS ON GENERAL CHARACTERISTICS

- The presence of fluorine, instead of hydrogen, on the carbon chain makes SC-PFCAs/PFSAs and the LC-PFSAs persistent due to the strength of the carbon-fluorine bond
- The presence of fluorine contributes to a high ionization potential and low polarizability for SC-PFCAs/PFSAs and LC-PFSAs
- Empirical pKa data for SC-PFCAs indicate that the anionic form is likely the dominant form in the environment—values range from 0.43 to 0.7
- No empirical pKa data were found for the SC-PFSAs and LC-PFSAs
- Available empirical physical-chemical property data (that is, pKa, vapour pressure) indicate that water is the environmental compartment to which SC-PFCAs/PFSAs and LC-PFSAs generally partition
- Empirical water solubility data were not found for SC-PFCAs, SC-PFSAs (except for PFBS), or LC-PFSAs
- PFAAs tend to partition to water, where they are present at higher concentrations at the water surface (for example, the air-water interface) due to their surfactant properties

3.1.1 Influence of length of carbon chain and functional groups

The length of the fluorinated carbon chain, conformations, and the functional group attached to the perfluorinated chain (for example, a charged moiety such as carboxylate or sulfonate) can result in different physicochemical properties that can influence the substance's behaviour in the environment and in organisms. As a result, PFAS generally have combined properties of lipophobicity, hydrophobicity, and hydrophilicity over different portions of the molecule. In general, the functional group attached to the perfluorinated chain (for example, a charged moiety such as a carboxylate or sulfonate anion) imparts hydrophilicity to that end of the molecule (Key et al. 1997). The hydrophilic portion of the molecule can be neutral, positively charged, or negatively charged. For example, both the carboxylate and sulfonate anionic part of the molecule are considered hydrophilic, while the perfluoroalkyl portions of the molecule can be hydrophobic as well as lipophobic. The anionic functional group and the dipolar nature of the carbon-fluorine bonds of the fluorinated carbon backbone contribute to the surfactant properties, creating a hydrophobic and lipophobic molecular surface that imparts the desired properties of water/oil repellency and stain resistance (Conder et al. 2008). The resulting substances can be non-ionic, cationic, or anionic surface-active as a consequence of their amphiphilic character. Examples of neutral functional groups include fluorotelomer alcohols ($–CH_2CH_2OH$) and sulfonamides ($–SO_3NH_2$). Examples of anionic functional groups include carboxylates ($–COO^-$), sulfonates ($–SO_3^-$), and phosphates ($–OPO_3^-$). In cationic PFAS, the functional group can be a quaternary ammonium group (Parsons et al. 2008). Linear isomers appear to be predominant for PFAS detected in biota as they may have significantly slower elimination rates and/or may be present at higher exposure concentrations than branched isomers (Conder et al. 2008).

3.1.2 pKa (acid dissociation constant)

Using the COSMOtherm model, Wang et al. (2011a) derived pKa estimates for the neutral form of PFCAs, PFSAs, PFSiAs and PFPIAs, some branched isomers for C4 to C8 PFCAs, and FTOHs. However, Wang et al. (2011a), cautioned that these values have high and unquantifiable uncertainties due to the estimates being highly dependent

on the chosen conformations of the neutral and anionic forms. For example, Wang et al. (2011a) had two predicted pKa values (2.897 and 0.897) for two different conformers of the PFOA anion. Available modelled pKa values for SC-PFSAs range from -0.58 to 0.33 (Wang et al. 2011a; Stockholm Convention 2018). Recent studies point to a pKa of between 0 and 1 for PFCAs (Wang et al. 2011a; Inoue et al. 2012) and even lower pKa values for PFSAs. Empirical pKa values for SC-PFCAs range from 0.43 to 0.7 (Appendix E). Empirical pKa values for SC-PFSAs and LC-PFSAs were not found.

3.1.3 Lipid and protein partitioning coefficients

Classical halogenated organic substances, such as polychlorinated biphenyls (PCBs), that are persistent and hydrophobic (as measured by the octanol-water partition coefficient for neutral species (K_{ow}) and/or octanol-water distribution coefficients at a specified pH (D_{ow}) partition to the lipids of organisms due to the high fugacity capacity of lipids for most hydrophobic substances. However, meaningful log K_{ow} or log D_{ow} values cannot be reliably measured for surface-active and ionizing substances, such as PFAS, as these substances tend to migrate to the interface of the organic and aqueous phases rather than partition between the two phases (Houde et al. 2006a). For example, PFOS forms three layers in octanol/water, and hence, a K_{ow} coefficient cannot be determined (OECD 2002). Consequently, the various physicochemical properties (for example, bioconcentration factor, soil adsorption coefficient), which can usually be estimated for conventional organic substances utilizing K_{ow} equations, cannot be estimated, and a calculated (estimated) log K_{ow} cannot be considered reliable (OECD 2002). Even if the log K_{ow} were known, it would not be suitable to be used for predictive purposes (for example, bioconcentration; OECD 2002).

Unlike the case for most organic substances, the preferential repository for PFAS in organisms is proteins, rather than lipids. Studies have shown that, at the organismal level, protein-rich tissues (for example, liver and blood) are the primary repositories, with concentrations that are orders of magnitude higher than concentrations in lipids (Martin et al. 2003b; Houde et al. 2006b). PFAS can bind to fatty acid-binding proteins, lipoproteins, and albumin, and can be sequestered in protein-rich tissues, such as yolk, liver, and blood (Cassone et al. 2012b). However, PFAS do not appear to accumulate in tissues rich in structural proteins (for example, muscle; personal communication, email from University of Pittsburgh to the Ecological Assessment Division, Environment and Climate Change Canada, dated February 13, 2020; unreferenced). Armitage et al. (2012) also suggested that the bioaccumulation potential and internal tissue distribution of perfluorinated alkyl acids may be influenced by phospholipids in the cell membranes. Jing et al. (2009) suggested that perfluorocarboxylates and sulfonates have high lipophilicity, which is not due to the perfluoroalkyl group but due to its electron-withdrawing effect on the adjacent oxoanion group. Nouhi et al. (2018) observed that PFBS and PFHxA can disturb and penetrate the phospholipid bilayer at high concentrations (that is, 50 mmol/L to 88 mmol/L).

PFAS transport into cells is likely controlled by a combination of passive diffusion and active facilitation by transporter proteins such as organic anion transporter proteins (Ng

and Hungerbühler 2013). Many of these proteins, or other proteins with similar function, have been identified in both rats and fish (De Smet et al. 1998; Manera and Britti 2006), indicating that fatty acid transporter proteins may be conserved between mammals and fish and can result in similar interactions (Jones et al. 2003). However, PFAS may also display substantial interspecies variability in tissue distribution and clearance rates, as well as gender-specific differences in elimination rates (Lee and Schultz 2010; Zhang et al. 2012b; Ng and Hungerbühler 2013). For example, PFHxS has serum elimination half-lives that vary considerably between species (Sundström et al. 2012; Numata et al. 2014) and between genders within species (Hundley et al. 2006; Sundström et al. 2012). In the study by Sundström et al. (2012), the species-specific and sex-specific elimination of PFHxS was highly expressed where male and female rats were investigated in terms of serum elimination. Results showed that females eliminated PFHxS more efficiently than did male rats. Furthermore, rats and mice appeared to be more effective at eliminating PFHxS than did monkeys (Sundström et al. 2012). Dassuncao et al. (2019) demonstrated that partitioning to phospholipids and binding to proteins are both mechanisms for the bioaccumulation of LC-PFCAs in the North Atlantic pilot whale (*Globicephala melas*). It is therefore worthwhile to consider both protein- and lipid-partitioning for marine and terrestrial mammals and birds. Protein-partitioning (characterized by coefficients such as K_{pw}) can be an additional representative mechanism governing the partitioning of PFAS along with phospholipid partitioning. However, there are no wildlife protein-partitioning coefficients available for the SC-PFCAs/PFSAs and LC-PFSAs.

Available empirical protein- or membrane-partitioning coefficients are found in Table 1. . Bischel et al. (2011) observed that SC-PFAS and LC-PFCAs bind at different locations on bovine serum albumin and that affinity for bovine serum albumin decreased from C8 PFCA to C12 PFCA, which is likely due to steric hindrances associated with longer, more rigid perfluoroalkyl chains. PFBS exhibited increased affinity relative to PFBA. Association constants determined for PFBS and PFPeA with bovine serum albumin are similar to those for LC-PFCAs, suggesting that the physiological implications of strong binding to albumin may be important for shorter-chain PFAS.

Table 1. Measured protein or membrane partitioning coefficients values for some PFAS

Chemical name	Log K_{pw} [a] (bovine serum albumin)	Log K_{mw} [b] (dipalmitoyl phospatidylcholine model bilayer membranes)	Log K_{mw} [c] (artificial phospholipid bilayer)	Log K_{mw} [d] (planar lipid bilayer membranes)
PFBS	3.9	NA	2.63	2.80–2.92
PFHxS	4.3	NA	3.82	4.08–4.18
PFOS	4.1	4.6–4.9	4.88	NA
PFBA	NA	NA	1.0	<1.7
PFPeA	3.4	NA	1.73	NA
PFHxA	4.1	NA	2.31	2.24–2.4
PFHpA	4.2	NA	2.87	2.85–2.97

Chemical name	Log K_{pw} [a] (bovine serum albumin)	Log K_{mw} [b] (dipalmitoyl phospatidylcholine model bilayer membranes)	Log K_{mw} [c] (artificial phospholipid bilayer)	Log K_{mw} [d] (planar lipid bilayer membranes)
PFOA	4.1	4.0–4.3	3.51	NA
PFNA	4.1	NA	4.04	NA
PFDA	3.9	NA	4.63	NA
PFUnA	3.7	NA	NA	NA
PFDoA	3.3	NA	NA	NA

Abbreviations: NA, not available; Log K_{mw}, distribution of a chemical between membrane and water phospholipids; Log K_{pw}, distribution of a chemical between protein and water

[a] Bischel et al. 2011

[b] Xie et al. 2010; Lehmler et al. 2006

[c] Droge 2019

[d] Ebert et al. 2020

3.2 Persistence

The presence of fluorine instead of hydrogen on the carbon chain alters the thermal, chemical, and biological characteristics of the PFAS molecule. The carbon-fluorine bond is one of the strongest in nature (3M Company 1999), making the bond extremely stable and generally resistant to degradation by acids, bases, oxidants, reductants, photolytic processes, microbes, and metabolic processes. The strong C-F bond and dense coating of electron-rich fluorine atoms protects the carbon backbone and results in an inertness to heat and chemical reagents (Hakli et al. 2008; Colomban et al. 2014). This contributes to a high ionization potential, low polarizability, low inter- and intra-molecular interactions, and low surface tension.

A number of studies show that degradation of C4 to C7 PFCAs and C4 and C6 PFSAs does not occur under environmentally relevant conditions (Hurley et al. 2004; Hori et al. 2005; Dillert et al. 2007; Hori et al. 2008; Saez et al. 2008; Park et al. 2009; Quinete et al. 2010). As a result, SC-PFCAs/PFSAs are expected to accumulate in the environment over time. Taniyasu et al. (2013) showed that some photodegradation of PFDS occurs under high altitude conditions and prolonged exposure; however, photodegradation did not occur for PFBS, PFHxS, and PFBA. Conditions achievable in an incinerator (that is, high temperatures at 900 or 1200 K) are considered capable of destroying substances that contain a carbon-fluorine bond (Tsang et al. 1998).

3.3 Bioaccumulation

3.3.1 Summary of the available bioaccumulation metrics (BCF, BAF, BMF, and TMF) for SC-PFCAs/PFSAs and LC-PFSAs

Canada's past ecological risk assessments for PFOS, PFOA, and most LC-PFCAs have shown that bioconcentration factors (BCFs) and bioaccumulation factors (BAFs), and even food web biomagnification factors (BMF) and trophic magnification factors (TMF), specifically in water-breathing aquatic organisms (that is, fish, daphnia) and algae, were generally indicative of a lower bioaccumulation potential (with the exception of some

PFOS BCF/BAFs in certain aquatic species—see Figure 3). However, food web BMF and TMF values in air-breathing marine mammals (for example, polar bears, dolphins), terrestrial mammals (for example, wolves), and birds (for example, Arctic birds) suggest a much higher bioaccumulation potential (EC, HC 2012; Environment Canada 2006; Environment Canada 2012). Thus, in the characterization of the bioaccumulation potential of SC-PFCAs/PFSAs and LC-PFSAs in this report, multiple metrics of bioaccumulation that include BCF, BAF, TMF, and BMF in aquatic, terrestrial, and avian species are considered, when available. In addition, there may be a particular relevance to tissue-specific accumulation (for example, liver, blood, kidney) as these are often the sites of significant toxicological action for PFAS.

Aquatic organisms, marine mammals and aquatic birds

In a laboratory study, Martin et al. (2003a) showed that C5 to C7 PFCAs did not bioaccumulate in any rainbow trout tissues (BAFs <0.1). PFHxA and PFHpA could not be detected in most rainbow trout tissues despite higher exposure concentrations, whereas PFBS was detectable at the last three uptake sampling intervals and at the first sampling time of the depuration phase, which allowed an estimation of depuration but not assimilation (that is, BAF <1). The Martin et al. (2003b) laboratory study showed that PFHxS had a carcass[4] BCF of 9.6 in rainbow trout, whereas the Goeritz et al. (2013) laboratory study calculated a BAF of less than 0.02 and 0.18 for PFBS and PFHxS, respectively, in rainbow trout.

Available bioconcentration and bioaccumulation data were taken from literature and graphed according to chain length, functional group, and organism class (
and Figure 4). PFOS and PFOA bioaccumulation data are included for comparative purposes. Figure 3 shows that bioconcentration or bioaccumulation levels of PFHxS in saltwater crabs, gastropods, saltwater fish, and freshwater fish are approaching Canada's BCF or BAF regulatory numeric criteria for bioaccumulation as set out in the *Persistence and Bioaccumulation Regulations of CEPA* (Canada 2000). Figure 4 shows that levels of bioconcentration or bioaccumulation of PFHxA in crab, gastropods, and saltwater fish are also approaching Canada's BCF or BAF regulatory numeric criteria for bioaccumulation (Canada 2000). These data show that read-across between chain lengths can be variable depending on the species and chain lengths chosen. For example, PFHxS has higher rates of uptake than PFOS in bivalves, and PFHxA has higher rates of uptake than either PFOA or PFHpA in freshwater fish, saltwater crab, and gastropods.

[4] Carcass: An incision was made along the ventral surface from the anus to the gills, and the entire liver was removed. The gut, consisting of esophagus, stomach, pyloric ceca, spleen, and intestines, was removed to avoid contamination of the carcass sample by feces and unabsorbed food (Martin et al. 2003a).

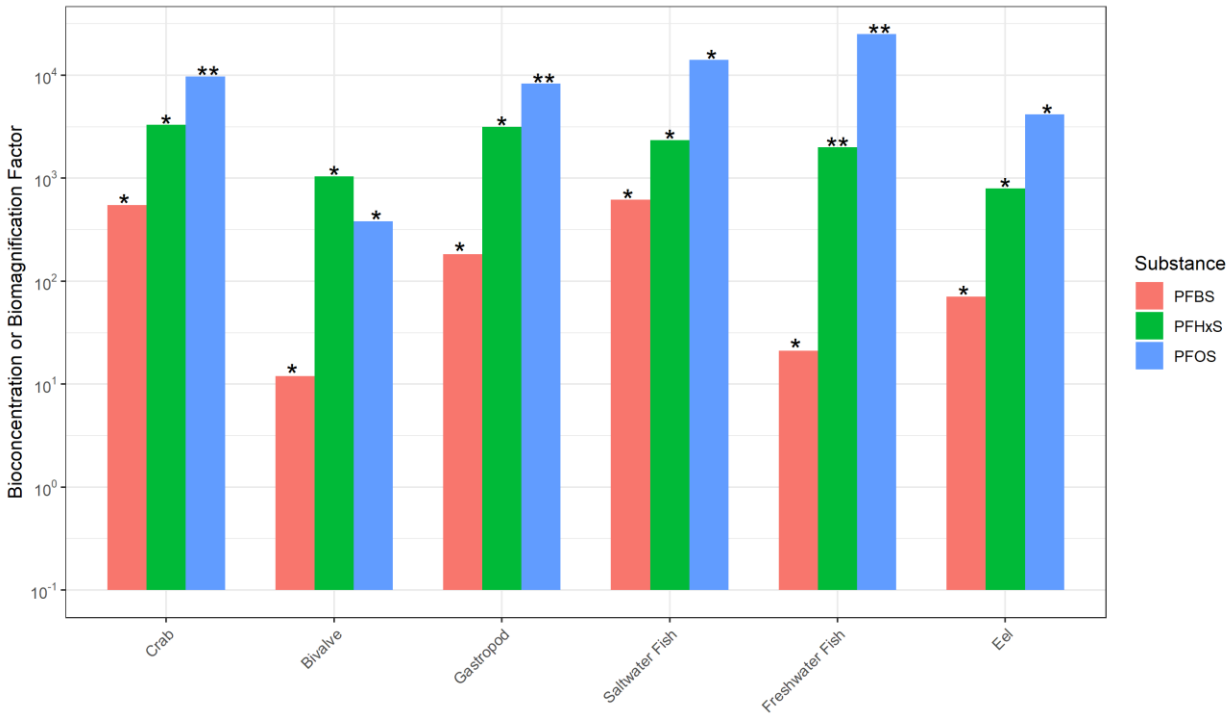

Figure 3. Available BCF/BAF (*maximum, **unknown) for PFOS and SC-PFSAs in crab (various species[a]), gastropods (various species[b]), bivalves (various species[c]), fish (various saltwater species[d] and freshwater species[e]), and eel[5]

** Unclear if the study referred to mean, average, or maximum values
[a] *Hemigrapsus sanguineus, Sesarma pictum, Hemigrapsus penicillatus, Helice tridens tridens, Philyra pisum*, and *Eriocheir sinensis*
[b] *Littorina brevicula, Monodonta labio, Umbonium thomasi, and Glossaulax didyma*
[c] *Mytilus edulis, Mactra veneriformis, Nuttallia olivacea*, and *Sinonovacula constricta*
[d] *Acanthogobius flavimanus, Sebastes schlegeli, Tridentiger obscurus, Hexagrammos otakii*, and *Mugil cephalus*
[e] *Oncorhynchus mykiss, Cyprinus carpio, Misgurnus anguillicaudatus,* and *Salvelinus namaycush*

[5] Martin et al. 2003b; Furdui et al. 2007; Kwadijk et al. 2010; Loi et al. 2011; Zhou et al. 2012; Naile et al. 2013; Gebbink et al. 2016; Menger et al. 2020

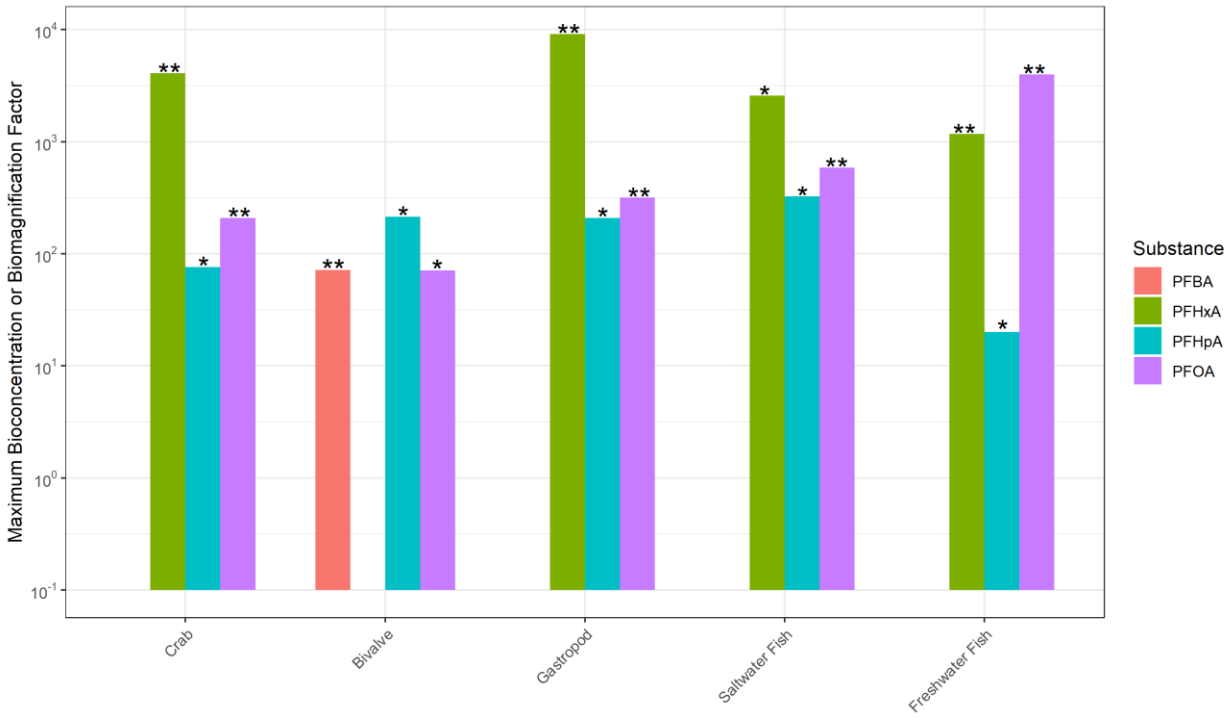

Figure 4. Available BCF/BAF (*maximum, **unknown) for PFOA and SC-PFCAs in crab (various species[a]), bivalves (various species[b]), gastropods (various species[c]), and fish (various saltwater species[d] and freshwater species[e]).[6]

** Unclear if the study referred to mean, average, or maximum values
[a] *Hemigrapsus sanguineus, Sesarma pictum, Hemigrapsus penicillatus, Helice tridens tridens, Philyra pisum,* and *Eriocheir sinensis*
[b] *Mytilus edulis, Mactra veneriformis, Nuttallia olivacea,* and *Sinonovacula constricta*
[c] *Littorina brevicula, Monodonta labio, Umbonium thomasi,* and *Glossaulax didyma*
[d] *Acanthogobius flavimanus, Sebastes schlegeli, Tridentiger obscurus, Hexagrammos otakii*, and *Mugil cephalus*
[e] *Oncorhynchus mykiss, Cyprinus carpio, Misgurnus anguillicaudatus,* and *Salvelinus namaycush*

[6] Martin et al. 2003b; Fuirdui et al. 2007; Kwadijk et al. 2010; Loi et al. 2011; Zhou et al. 2012; Naile et al. 2013; Gebbink et al. 2016; Menger et al. 2020; Rijnders et al. 2021

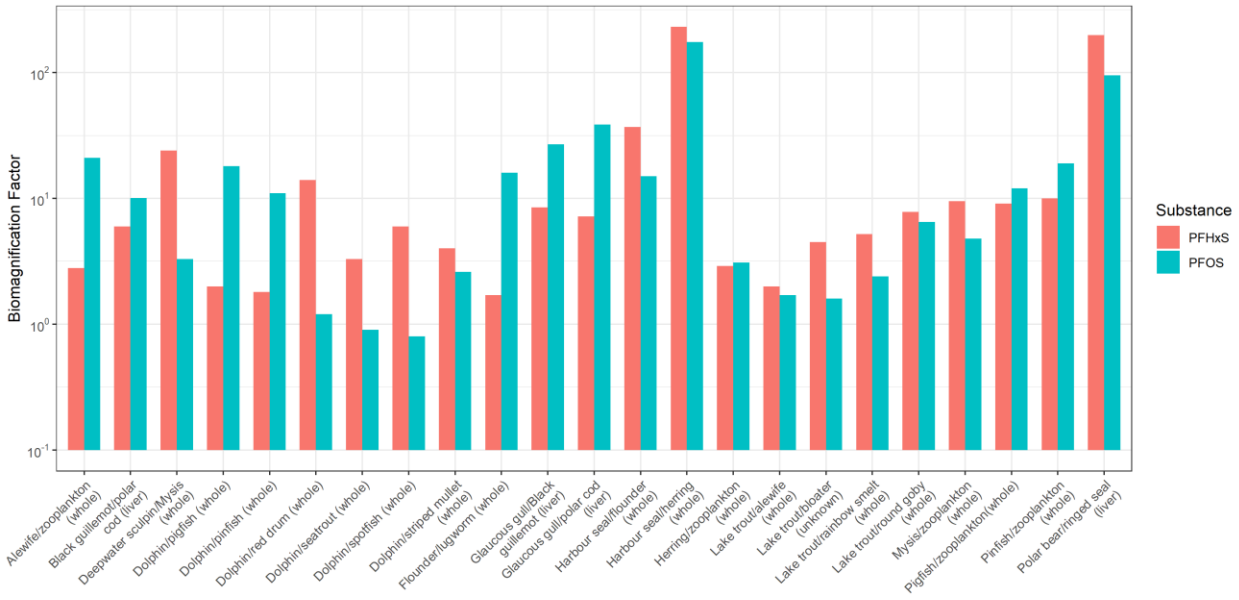

Figure 5. Comparison of BMFs for PFHxS and PFOS in various trophic levels of various food webs.[7]

Figure 5. **Comparison of BMFs for PFHxS and PFOS in various trophic levels of various food webs.** shows that food web BMF values for PFHxS and PFOS are greater than 1 (10^0) in air-breathing organisms such as dolphins, harbour seals, polar bears, and birds despite moderate BCF and BAF values (<3500) in water-breathing organisms for PFHxS. This figure also shows that BMF values for PFHxS are comparable with those for PFOS. Species differences in uptake rates can also be seen. For example, harbour seals have greater uptake rates of PFHxS than any other marine mammal, with BMFs of up to 231. Boisvert et al. (2019) reported a comparison of the arithmetic means of ratios of PFAS concentrations found in polar bear liver with those in ringed seal liver; similarly, concentrations of PFAS in polar bear liver were compared with those in ringed seal blubber from East Greenland (Scores by Sound). PFHxS, PFDS, PFBA, and PFHxA showed bear to seal ratios of >1, reflecting an increase from dietary exposure.

TMF values for PFHxS were 2.2 to 5.4 for lake trout food webs in Lake Ontario and Lake Huron, Canada, and 1.8 for a harbour seal food web in the Westerschelde, The Netherlands (Van den Heuvel-Greve et al. 2009; Ren et al. 2021, 2022). In the freshwater food web of the Yadkin-Pee Dee River (North and South Carolina, United States), TMF values were 1.08 for PFBS and below 1 for PFHpA (Penland et al. 2020). PFHxS had a TMF value of 2.09 in an Antarctic food web (Gao et al. 2020a). However, in a marine food web (Qinzhou Bay, South China Sea), TMF values were below 1 for PFHxA and PFBS (Du et al. 2021). In a temperate macrotidal estuary (Gironde, France), TMF values were below 1 for PFHpA, PFHxS, and PFHpS (Munoz et al.

[7] Kannan et al. 2005; Houde et al. 2006a; Haukås et al. 2007; Butt et al. 2008; Van den Heuvel-Greve et al. 2009; Ren et al. 2021, 2022

2017a). Simonnet-Laprade et al. (2019a,b) showed TMF values ranging from 0.65 to 8.3 for PFHxS in a freshwater riverine food web (France). The TMF values for PFHpS ranged from 0.36 to 3.7, and TMF values for PFDS ranged from 0.73 to 17.9. Simonnet-Laprade et al. (2019a) suggested that the high variability of measured TMFs may be related to different metabolic capacities between species as well as to specific exposures in certain regions of the world, and the occurrence of unidentified precursors and their enhanced biotransformation in fish compared to invertebrates. Alternative explanations could include site differences in rates of biotransformation/growth, sediment-water concentration ratios, extent of food web omnivores, and spatial concentration gradients (Mackay et al. 2016).

The studies presented in this section show that the BMF values for the SC-PFCAs and SC-PFSAs can be comparable to those for PFOA and PFOS. The studies also demonstrate that there is a challenge in using empirical or modelled BCF and BAF data for water-breathing organisms (for example, fish) as surrogate data for BMFs/TMFs in air-breathing organisms (for example, polar bears) for PFAS. As a result, when available, empirical data for food web BMFs and TMFs for air-breathing wildlife may be the best indicators for overall bioaccumulation potential in these organisms as data for water-breathing organisms may tend to underestimate overall bioaccumulation potential.

There are several explanations to account for the discrepancy in bioaccumulation between water-breathing organisms and air-breathing organisms. Traditionally, equilibrium partitioning has assumed that if uptake occurs by the same mechanism in both water-breathing organisms (for example, fish) and air-breathing organisms (for example, polar bears), then similar uptake rates would be seen (Mackay and Fraser 2000; Kelly et al. 2004). For example, Kelly et al. (2004) stated that classical organic pollutants (that is, non-polar/non-volatile substances such as PCBs) had low elimination rates to both water and air, resulting in similar bioaccumulation rates for air-breathing and water-breathing organisms. This allowed fish BCF/BAFs to be extrapolated to characterize bioaccumulation in marine mammals and birds for these classical organic pollutants. However, extrapolation of fish-derived bioaccumulation parameters to air-breathing organisms for substances such as PFAS is highly uncertain and not recommended. Gray (2002) states that lower-trophic level organisms may take up contaminants through their body surface or through their respiratory organs by diffusion. For most small organisms (for example, plankton, polychaetes, bivalves, crustaceans), the major route of intake is by respiratory surfaces. Randall et al. (1998) demonstrated that rainbow trout (*Oncorhynchus mykiss*) had the largest proportion of tetrachlorobenzene taken up via the gills. Randall et al. (1998) also determined that the uptake of toxicants in food plays a minor role in water-breathing animals. Gray et al. (2002) stated that at higher trophic levels, marine birds and mammals do not take up contaminants from their respiratory surfaces as they are air-breathing and the concentrations of contaminants in air are low; thus, the only route for their contaminant uptake is through food.

Many PFAAs will likely be less hydrophobic, with increased water solubility, as their chain length decreases. For water-breathing organisms, this may result in a more rapid elimination of PFAAs to the water phase (via gill exchange) and a reduction in bioaccumulation. For fish, the lamellar blood-water interface of the gills is the major route of clearance (and uptake) of non-metabolizing waterborne substances such as PFAS. However, bioaccumulation in air-breathing organisms is driven primarily by volatility (of the neutral form) rather than polarity (Ankley et al. 2021). Therefore, the non-volatile nature of PFAAs may result in relatively slow elimination to air, resulting in higher bioaccumulation in air-breathing organisms (Kelly et al. 2004). The high water solubility of PFAAs causes their escaping tendency to be relatively high from gills into water, whereas the escaping tendency of PFAA to air, across the alveolar membrane of the lung, would be relatively low because of the low vapor pressure and negative charge of PFAAs. Thus, fish gills provide an additional mode of elimination for PFAAs (that is, “gill exchange”) that birds and terrestrial and marine mammals do not possess. Furthermore, the variability in bioaccumulation potential between different species may be partially related to body size, with larger air-breathing organisms having slower rates of depuration (Ankley et al. 2021).

Additionally, the simultaneous occurrence of different PFAS in wildlife adds further complexity to their bioaccumulation. Wen et al. (2017) demonstrated the inhibitory effect of LC-PFCAs on the bioconcentration of SC-PFCAs/PFSAs. The uptake and elimination rate constants of PFBS, PFBA, PFPeA, PFHxA, and PFHpA declined in all tissues, and their BCF values decreased by between 24% and 89% in the presence of LC-PFCAs (that is, PFNA, PFDA, PFUnA, and PFDoA), PFOS, and PFOA. The inhibitory effect may be attributed to their competition with LC-PFCAs, PFOS, and PFOA for transporters and protein binding sites in zebrafish.

Terrestrial invertebrates, mammals and birds (eagle)

Zhao et al. (2013a) exposed earthworms (*Eisenia fetida*) to soils artificially contaminated with C6 to C12 PFCAs and C4, C6, and C8 PFSAs. Biota-to-soil accumulation factors (BSAFs) increased with perfluorinated carbon chain length and were greater for PFSAs than for PFCAs of equal perfluoroalkyl chain length. BSAFs were 0.087 g_{oc}/g_{dw} (PFHxA), 0.122 g_{oc}/g_{dw} (PFHpA), 0.048 g_{oc}/g_{dw} (PFBS), and 0.0473 g_{oc}/g_{dw} (PFHxS). Higher soil concentrations resulted in lower BSAFs. Grønnestad et al. (2019) determined that whole-body BMF values were below 1 for C4 to C7 PFCAs for earthworm (*E. fetida*) and bank vole (*Myodes glareolus*). Rich et al. (2014) exposed earthworms (*E. fetida*) to field-collected unspiked soils with varying levels of PFAS, including a control soil, an industrially impacted biosolids-amended soil, a municipal biosolids-amended soil, and AFFF-impacted soils. With the exception of the control soil, BAFs were above 1, and PFHxS had the highest BSAF (0.23 g_{oc}/g_{ww}) in the municipal soil. Lasier et al. (2011) determined BSAFs for *Lumbriculus variegatus* in sediments from the Coosa River watershed in Georgia, United States. The mean $BSAF_{ww}$ values for PFBS and PFHpS were 0.3 and 2.6, respectively. The mean $BSAF_{ww}$ values for PFHpA and PFHxA were <0.2 and 0.06, respectively. Lasier et al. (2011) indicated that PFHxA had little potential to bioaccumulate or biomagnify in oligochaetes

but that PFHpS and PFHxS may be as bioaccumulative as PFOS, which has a mean $BSAF_{ww}$ value of 0.49.

Huang et al. (2022) suggested that short-chain PFASs (for example, PFBA, PFBS, PFHxS) also possessed high biomagnification potentials in a terrestrial food chain from the Tibetan Plateau involving plants to pika to eagle accumulation. Relatively high TMFs of 5.96, 2.43 and 5.75 were measured for PFBS, PFHxS, and PFOS in the Tibetan Plateau plant−pika−eagle food chain, while eagle (muscle)/pika (whole) BMFs for PFBA, PFBS, and PFHxS were 1.42, 1.34, and 2.29, respectively (Huang et al. 2022).

3.3.2 Special considerations on the mechanism of accumulation for PFAS

The equilibrium partitioning approach (typically used in bioaccumulation models) is usually applied to understand the bioaccumulation of classical organo-halogen pollutants (for example, PCBs) that are neutral, hydrophobic, non-volatile, and slowly metabolized. However, although SC-PFCAs/PFSAs and LC-PFSAs are non-volatile, they can have combined properties of ionization, lipophobicity, hydrophobicity, and hydrophilicity over different portions of the molecule. Therefore, the bioaccumulation of SC-PFCAs/PFSAs and LC-PFSAs in the environment can be quite difficult to predict compared with classical organic pollutants.

Chemical concentrations of neutral organic chemicals are usually lipid-normalized prior to reporting environmental levels of calculation of bioaccumulation metrics. A normalization method for ionic substances that associate with protein/plasma may be more relevant but is not yet routine. Total protein normalization may also lead to confounding issues as different proteins can have varying affinities for PFCAs and PFSAs, and the expression of these proteins may display differences between species and sexes. From a physiological perspective, it is the concentration of a substance at the site of toxic action within the organism that determines whether a response is observed, regardless of the external concentration. In the case of SC-PFCAs/PFSAs and LC-PFSAs, the site of toxic action is often considered to be the liver cells or hepatocytes. Measures of bioaccumulation metrics may be used as indicators of either direct toxicity to organisms that have accumulated PFAS or indirect toxicity to organisms that consume prey containing PFAS (via food chain transfer). Thus, from a toxicological perspective, bioaccumulation metrics that are based on concentrations in individual organs, such as the liver, may be more relevant when predicting the potential for direct organ-specific toxicity (that is, liver toxicity) of PFAS. However, certain metrics (that is, BCFs and, in particular, BMFs/TMFs) that are based on concentrations in whole organisms may provide a useful measure of overall potential for food chain transfer.

An additional consideration associated with the determination of bioaccumulation of PFAS is the exclusion of the un-metabolized precursors, which can lead to an underestimation of the overall bioaccumulation potential. Precursors to PFOS or PFOA have been shown to metabolize in rodents, resulting in the formation of PFOS or PFOA. For example, Nabb et al. (2007) showed that 8:2 fluorotelomer alcohol (8:2 FTOH) can

be metabolized to PFOA. Letcher et al. (2014) showed that polar bears can rapidly de-alkylate FOSA (a precursor to PFOS). Therefore, the presence and metabolic transformation of precursors in wildlife can add to the critical body burden of some perfluorinated substances in wildlife.

Traditionally, equilibrium partitioning assumes that the metabolic transformation of a substance in an organism would enable rapid elimination of the substance, thus reducing its levels in the organism (Kelly et al. 2004). However, metabolic transformation for SC-PFCAs/PFSAs and LC-PFSAs is not likely to occur in wildlife. The observed rate of depuration is generally slower than for any previously investigated surfactant in fish, which may be partially attributable to the lack of metabolism or biotransformation (Martin et al. 2003b). The relatively slow depuration half-lives, combined with observations of high blood, liver, and gall bladder concentrations, support the theory that perfluorinated substances can enter into enterohepatic recirculation in fish—the process whereby substances are continuously recycled between blood, liver, gall bladder, and intestines, and where resorption occurs via the portal vein (Martin et al. 2003b).

4.0 Environmental occurrence

This section demonstrates that SC-PFCAs/PFSAs and LC-PFSAs are found in the Canadian environment despite the fact that these substances are not known to be manufactured, imported, or used in Canada as pure substances. There is currently a moderate amount of data on the presence of SC-PFCAs/PFSAs in the Canadian environment but few data on LC-PFSAs. Current concentrations of SC-PFCAs/PFSAs and LC-PFSAs may reflect the contribution of precursors and salts that have already transformed to the moiety of interest. Because the salts typically dissociate at environmentally relevant pH, the salts or acids will be in their anionic form in the environment. This report does not directly consider the potential mixture effects amongst the individual moieties of interest and their salts and precursors.

4.1 Sources of exposure

One mechanism that accounts for the presence of SC-PFCAs/PFSAs and LC-PFSAs is the release of consumer, commercial, or industrial products containing SC-PFCAs/SC-PFSAs and LC-PFSAs or their precursors in wastewater treatment systems and landfills, as well as indirectly through the land application of wastewater biosolids. Another mechanism is the long-range transport of precursors that are deposited across Canada, including in remote areas such as the Canadian Arctic. A third mechanism is the potential transformation or biotransformation (that is, metabolism) of precursors in biota (Nabb et al. 2007; Butt et al. 2010a, 2010b; Kim et al. 2012, 2014). Appendix D provides empirical data identifying substances as precursors for SC-PFCAs/PFSAs. No equivalent data were found in publicly available literature for the LC-PFSAs. It is expected that, in the absence of shifts in use patterns or regulatory actions,

concentrations of SC-PFCAs/PFSAs in the environment will increase over time due to their resistance to degradation under normal environmental conditions.

4.1.1 Releases from products

The release and degradation of consumer, industrial, or commercial products that contain SC-PFCAs/SC-PFSAs/LC-PFSAs or their precursors is another mechanism that accounts for the presence of these substances in Canada's populated areas. For example, the presence of residual unbound fluorotelomer alcohols (that is, 4:2 FTOH, 6:2 FTOH, 8:2 FTOH, and 10:2 FTOH) was identified in several commercial consumer products such as stain repellents, paints, polishes, and other coatings (Dinglasan-Panlilio and Mabury 2006). PFCAs can be formed from the atmospheric oxidation of fluorotelomer alcohols.

Another example is the use of aqueous film-forming foams (AFFF), which can be a source of SC-PFAS in the Canadian environment. AFFF products may be used at airports (for example, Moody et al. 2002), during railway incidents (for example, Munoz et al. 2017b) and at military bases (for example, Lescord et al. 2015). Between 2000 and 2015, countries including the United States, Canada, the United Kingdom, Australia, Norway, the Netherlands, Germany, and Sweden introduced regulations and guidelines to phase out and limit the use of PFOS, PFOA, and their precursors in AFFF. While manufacturers have since modified their formulations to eliminate PFOS, AFFF formulations continue to include SC-PFAS. Early alternatives to PFOS-based AFFF that contained longer chain (C8-based) fluorotelomers are being phased out by producers, which has created a shift towards shorter chain (that is, C6-, C4-, and C3-based) perfluoroalkylated chemicals (National Academies of Sciences, Engineering, and Medicine 2017). The most common and most widely used are C6-based fluorotelomer AFFF (National Academies of Sciences, Engineering, and Medicine 2017; Hatton et al. 2018).

A third example of a PFAS source is fluorinated ski waxes. Ski waxes usually contain semi-fluorinated n-alkanes and PFCAs (Chropeňová et al. 2016; Plassmann and Berger 2013). According to Nordic Ecolabelling (2018), product development of ski wax with fluorine is transitioning to the use of SC-PFAS, such as C6 PFCAs. For example, Plassmann and Berger (2013) detected C6 to C22 PFCAs in fluorinated ski waxes. In addition, ski wax was assumed to be a source of PFAS near ski resorts in Slovakia and Norway, where some SC-PFCAs/PFSAs and PFDS were found in pine needles (Chropeňová et al. 2016). Snow meltwater sampled from ski tracks in Norway showed the presence of C6 and C7 PFCAs (Langford et al. 2010 as cited in Plassmann and Berger 2013). Lastly, snow samples taken from a ski area in Sweden after a skiing competition also showed the presence of C6 to C22 PFCAs. No equivalent studies in Canada were identified.

4.1.2 Long-range transport

Long-range atmospheric transport of SC-PFCAs/PFSAs and LC-PFSAs is unlikely given their low volatility. However, the long-range transport of volatile precursors is believed to be a pathway that accounts for the presence of fluorinated acids in the Canadian Arctic. In remote areas including the Canadian Arctic, precursors are presumed to be slowly oxidized by atmospheric radical species to give fluorinated acids that can be deposited by precipitation (Waterland and Dobbs 2007). Fluorotelomer alcohols (FTOHs) ($C_nF_{2n+1}CH_2CH_2OH$) were the first molecular class to be proposed as precursors for PFCAs and subsequently detected in low concentrations in the northern hemispheric troposphere (Waterland and Dobbs 2007). Other potential precursors include fluorotelomer olefins (FTOs) and N-alkyl perfluoroalkylsulfonamidoethanols (PFSEs).

The estimated atmospheric lifetimes of FTOHs (12 to 20 days) and FTOs (8 days) permit their transport to remote regions (Waterland and Dobbs 2007). Photoreactor studies have indicated that PFSEs may contribute to the observed environmental burden of both PFSA and PFCA substances (Waterland and Dobbs 2007). N-methylperfluorobutanesulfonamidoethyl alcohol (NMeFBSE) has the potential for atmospheric transformation to PFBS through oxidation by hydroxyl radicals (D'eon et al. 2006) (see Appendix D). In addition, Pickard et al. (2018) collected and sampled a 15-m ice core representing 38 years of deposition (1977 to 2015) from the Devon Ice Cap (Nunavut, Canada). By modelling air mass transport densities and comparing temporal trends in deposition with production changes of possible sources, Pickard et al. (2018) found that continental Asia was the largest PFAS contributor impacting the Devon Ice Cap and that the deposition of PFAS was dominated by atmospheric formation from volatile precursors. PFCAs from C2 to C13 were detected on the Devon Ice Cap with concentrations ranging from 0.00321 ng/L to 0.751 ng/L. PFSAs from C4 to C8 were detected with concentrations ranging from 0.00018 ng/L to 0.391 ng/L. Pickard et al. (2020) also detected PFBA in the Mt. Oxford icefield cores at concentrations ranging from <0.04 ng/L to 1.34 ng/L and 0.003 ng/L to 1.90 ng/L. Pickard et al. (2018) detected neither PFHxS nor PFDS in the Devon Ice Cap. However, MacInnis et al. (2017) detected PFDS but not PFHxS in a snow pit on the Devon Ice Cap representing deposition for the years 1993 to 2007.

Long-range oceanic transport of the acids and their precursors has also been proposed as a potential pathway to account for the presence of fluorinated acids in the Canadian Arctic, since perfluorinated alkyl acids, their salts, and conjugate bases are water soluble and have no appreciable vapour pressure. One hypothesis for the origin of perfluorinated alkyl acids, their salts, and conjugate bases in the atmosphere (and subsequent deposition on land) is transfer from the surface ocean by sea spray, given that the surface active properties of perfluorinated alkyl acids result in their enrichment on the “surface of bursting bubbles” (Reth et al. 2011). More specifically, the water-to-air transfer of C6 to C14 PFCAs and C6, C8, and C10 PFSAs in a laboratory-scale sea spray simulator was studied by Reth et al. (2011). This study found that the sequestration of the perfluorinated alkyl acids, their salts, and conjugate bases out of bulk water to the air-water surface increased exponentially with the length of the

perfluorinated alkyl chain. Thus, it is likely that oceanic transport of primary acid emissions plays a role in their transport to the Canadian Arctic. In field tests at two Norwegian coastal sites, C6 and C7 PFCAs and PFSAs were measured in air samples and were positively correlated with Na^+ ion concentrations. This also suggests that sea spray aerosols are a source of atmospheric PFAAs in coastal areas (Sha et al. 2022).

4.2 Abiotic concentrations in Canada

4.2.1 Surface water

Short-chain PFCAs/PFSAs and LC-PFSAs have been detected in surface freshwater, rain, and snow across Canada, including the Canadian Arctic. Overall, the average concentrations were higher in surface water (which ranged from less than the limit of detection [LOD] to 277 ng/L) than in snow (0.006 ng/L to 8.9 ng/L; Meyer et al. 2011; Bhavsar et al. 2016; MacInnis et al. 2019a). Average concentrations in rain were not reported. PFHxS had the highest measured maximum concentration in surface fresh water at 49 600 ng/L, which was reported in Etobicoke Creek, Ontario, following a spill of AFFF at the Lester B. Pearson Airport (Moody et al. 2001). PFBA had the highest maximum concentrations in both rain and snow at 14 ng/L and 52 ng/L, respectively (Gewurtz et al. 2019; MacInnis et al. 2019a).

D'Agostino and Mabury (2017) measured surface freshwater concentrations from Nunavut, with PFPeA having the highest measured mean concentration at 76 ng/L. Maximum measured concentrations in rain from Nova Scotia, Ontario, British Columbia, and Quebec for SC-PFCAs ranged from 0.9 ng/L to 14 ng/L (Scott et al. 2006a,b; Gewurtz et al. 2019). In the Canadian High Arctic Circle, snowpacks from Lake Hazen had measurable concentrations of PFBA, PFPeA, PFHxA, PFHpA, and PFBS between 2013 and 2014, with the highest concentration measured for PFBA at 52 ng/L (MacInnis et al. 2019a). The snowpacks also had PFBS measurements of up to 0.4 ng/L and PFHxS measurements of up to 0.44 ng/L (MacInnis et al. 2019a). Measurements of PFAS in Lake Hazen also suggest that snowmelt contributed to surface water concentrations of PFCAs.

Concentrations of SC-PFSA/PFCA and LC-PFSAs measured in Canada between 2000 and 2020 are represented using Tukey box plots in Figure 6. Tukey box plots are interpreted as follows: the lower and upper hinges (edges) of the box represent the first and third quantiles (Q1 and Q3), which are the 25th and 75th percentiles, respectively, while the black horizontal line within the box represents the second quantile, also known as the 50th percentile (median). The distance between the 25th and 75th percentile is called the interquartile range (IQR). The lower whisker represents the lowest data that are within the Q1 − 1.5 x IQR threshold, and the upper whisker represents the highest data that are within the Q3 + 1.5 x IQR threshold. Data exceeding these thresholds appear as individual points (for example, circles, triangles, squares). However, if the minimum and maximum are within these thresholds, they represent the lower and upper whiskers, and no outliers are present.

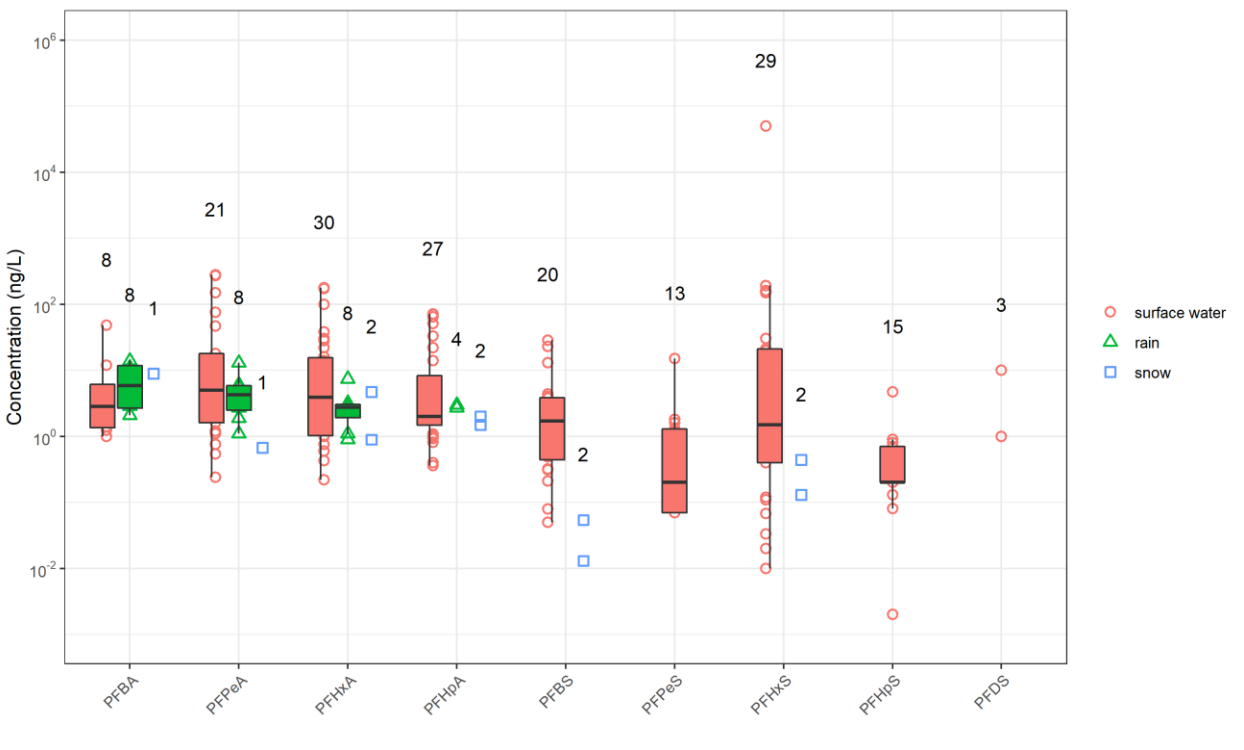

Figure 6. Concentrations of SC-PFSAs, SC-PFCAs, and LC-PFSAs in surface water, rain and, snow from 2000 to 2020 (ng/L).[8] The numbers above each box represent the number of data points included.

4.2.2 Sediments

Measured concentrations of SC-PFCAs/PFSAs and LC-PFSAs in core and surface sediments were taken from literature and graphed according to chain length and functional group (

[8] Moody et al. 2001; Scott et al. 2006a,b, 2009; Furdui et al. 2008a; Awad et al. 2011; Meyer et al. 2011; De Solla et al. 2012; Houde et al. 2014; Lescord et al. 2015; Bhavsar et al. 2016; D'Agostino and Mabury 2017; Gewurtz et al. 2019; MacInnis et al. 2019a; Picard et al. 2021; Kaboré et al. 2022; MacInnis et al. 2022

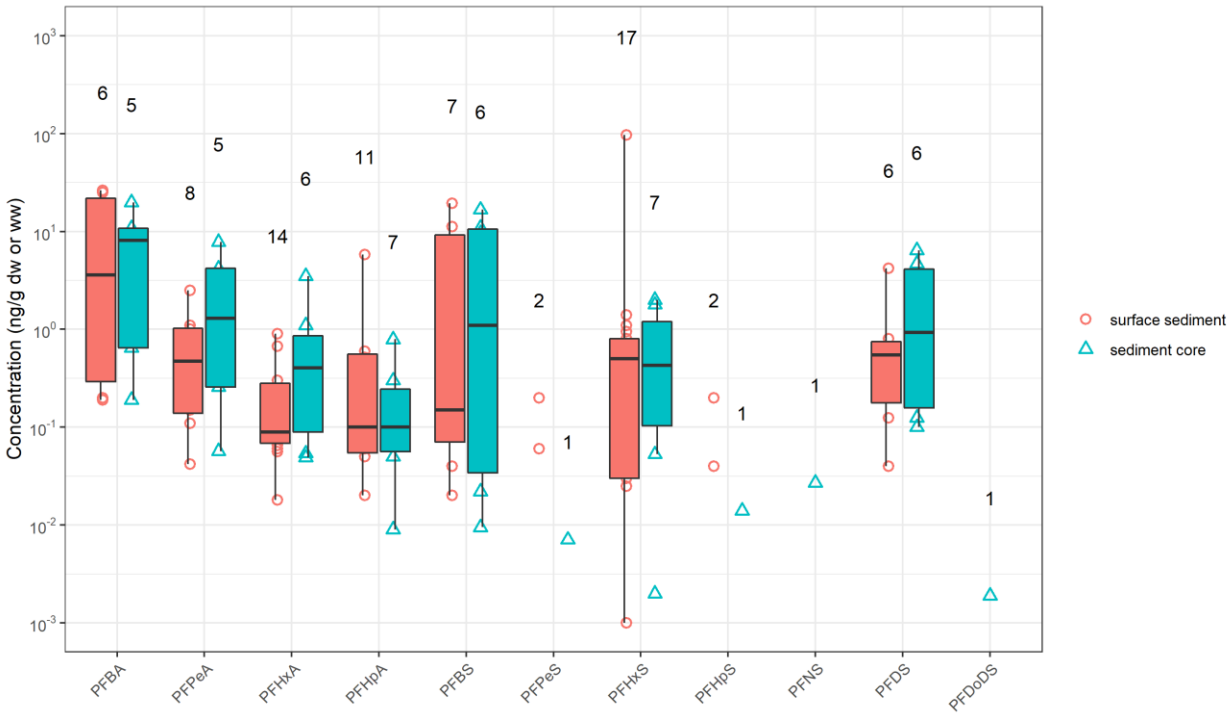

Figure **7**). Only sediment core samples were reported for PFNS and PFDoDS, which were detected in Lake Ontario at a mean concentration of 0.02 ng/g and 0.0019 ng/g, respectively (personal communication, email from the Aquatic Contaminants Research Division, Environment and Climate Change Canada, to the Ecological Assessment Division, Environment and Climate Change Canada, dated March 24, 2020; unreferenced). PFHxS had the highest surface sediment concentration at 96.5 ng/g dw in Lake Niapenco, Ontario (Bhavsar et al. 2016), and PFBA had the highest sediment core concentration at 19.8 ng/g dw in Lake Ontario (Codling et al. 2018).

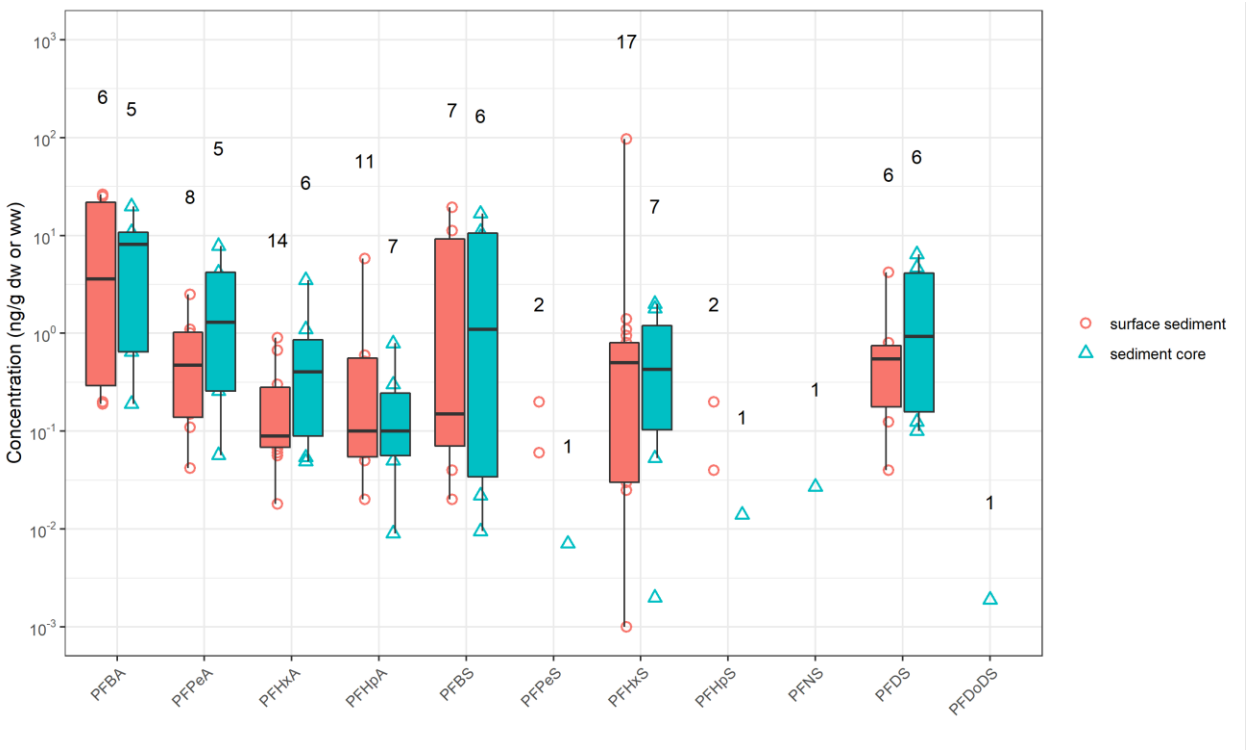

Figure 7. Concentrations of SC-PFSAs, SC-PFCAs, and LC-PFSAs in surface sediment and sediment core (ng/g dw or ww).[9] The numbers above each box represent the number of data points included.

4.2.3 Soil

Measured soil concentrations of SC-PFCAs/PFSAs and LC-PFSAs were available from Cabrerizo et al. (2018) and Liu et al. (2022). Soil concentrations were graphed according to chain length and functional group (

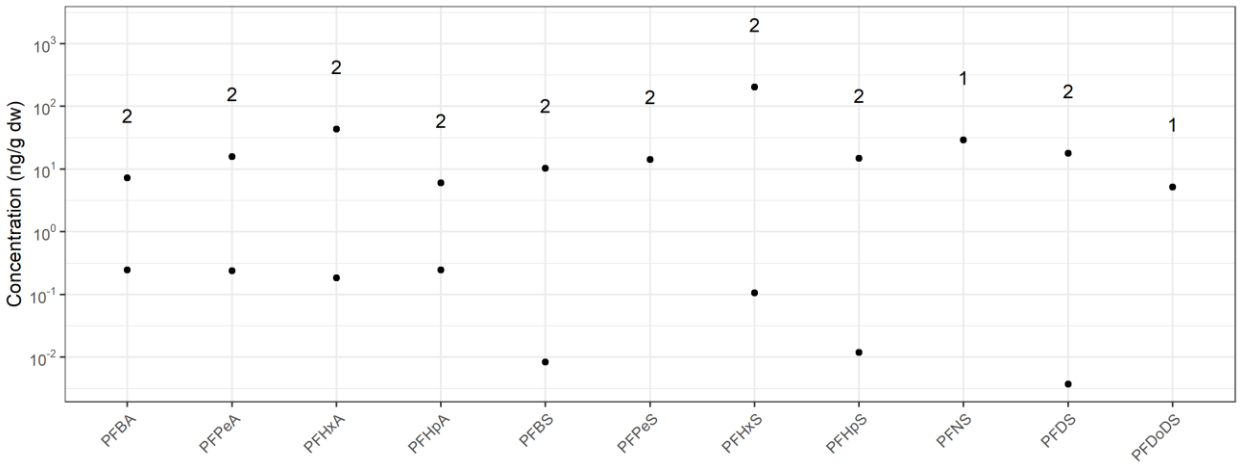

Figure **8**). Soil samples were taken from Cornwallis Island and Melville Island, both

[9] Kelly et al. 2009; Awad et al. 2011; Yeung et al. 2013; Lescord et al. 2015; Bhavsar et al. 2016; D'Agostino and Mabury 2017; Munoz et al. 2017b; Codling et al. 2018; MacInnis et al. 2019b

located in Nunavut, as well as from firefighting training areas in airports in central and eastern Canada. PFHxS had the highest measured concentration of 203.1 ng/g dw, followed by PFHxA at 43 ng/g and PFNS at 28.9 ng/g (Liu et al. 2022). Cabrerizo et al. (2018) noted that PFBA was correlated with a few other PFCAs, suggesting that the presence of PFBA in Arctic soils may come from sources other than atmospheric transport and the transformation of PFCA precursors. Cabrerizo et al. (2018) indicated that a more likely source of PFBA was chlorofluorocarbon replacement chemicals (including hydrofluoroethers and hydrofluorocarbons that contain the C_4F_9 moiety), which are known to yield PFBA.

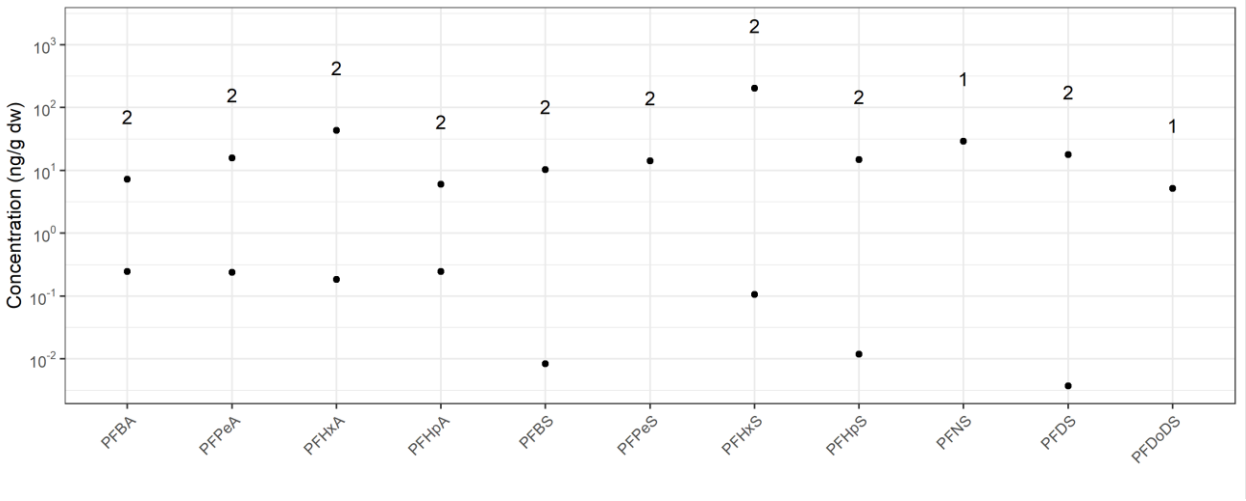

Figure 8. Concentrations of SC-PFSAs, SC-PFCAs, and LC-PFSAs in soil (ng/g dw).[10] The numbers above each box represent the number of data points included.

4.2.4 Wastewater treatment plants and landfills

SC-PFCAs/PFSAs were detected in landfill leachate, urban effluent, and Arctic effluent (that is, lagoons) in Canada. Measurements of LC-PFSAs have not yet been reported. Maximum concentrations of SC-PFCAs (C4 to C7) and SC-PFSAs (C4 and C6) in landfill leachate in Ontario ranged from 0.21 µg/L to 1.3 µg/L, with PFHxS having the highest maximum concentration, followed by PFBS (Propp et al. 2021). Higher concentrations of SC-PFCAs/PFSAs have been detected in Arctic lagoon effluent in comparison with urban effluent from temperate wastewater treatment plants (WWTPs). Mean concentrations of SC-PFCAs (C4 to C7) in effluent from a WWTP in Toronto, Ontario, ranged from 2.2 ng/L to 5.3 ng/L (Scott et al. 2006a), and PFHxS had the highest maximum concentration of 7.5 ng/L. However, SC-PFCAs (C4 to C7) and SC-PFSAs (C4 and C6) were also measured in Arctic lagoon influent (below detection up to 20 ng/L) and effluent (below detection up to 18 ng/L), and PFHxA had the highest maximum concentrations (Gewurtz et al. 2020). Gewurtz et al. (2020) hypothesized that low potable water consumption, dry and hostile weather conditions, poor ventilation, small apartment size, and long use times of household items contribute to elevated

[10] Cabrerizo et al. 2018 and Liu et al. 2022

indoor PFAA concentrations, which would contribute to elevated concentrations found in both influent and effluent concentrations at Arctic lagoons in comparison with temperate WWTPs.

Guerra et al. (2014) sampled influent and effluent from 13 WWTPs in Canada. Mean concentrations of SC-PFSAs/PFCAs (C4 to C7) in influent and effluent ranged from <1.0 ng/L to 453 ng/L and <1.0 ng/L to 419 ng/L, respectively. The highest measured mean concentrations were for PFHxS and were measured at a WWTP servicing 80% industrial airport waste water, as well as a small population of 3000 inhabitants. PFHxS was followed by PFHxA, and then PFPeA (Guerra et al. 2014).

4.2.5 Temporal trends

In addition to the measurement of SC-PFCAs/PFSAs and LC-PFSAs in the Canadian environment, certain temporal trends have been identified. Between 2006 and 2018, Gewurtz et al. (2019) monitored PFAS in Great Lakes precipitation. They determined that PFOA, PFNA, PFDA, and PFOS significantly decreased over the monitoring period likely due to phase-outs and regulatory actions aimed at PFOS, PFOA, and other LC-PFCAs and their precursors. Conversely, concentrations of PFBA and PFHxA appeared to increase in the later years of the study period (Gewurtz et al. 2019). Similar trends were seen in studies of beluga whales (*Delphinapterus leucas*) in the St. Lawrence River. Barrett et al. (2021) measured PFAS concentrations in beluga whale liver and found that PFOS concentrations were lower in samples taken during the period of 2010 to 2017 compared with samples from 2000 to 2009. SC-PFCAs (C4 to C7) were detected at higher concentrations between 2013 and 2017, whereas they were rarely detected in earlier samples (Barrett et al. 2021).

A review by Muir and Miaz (2021) found that there were significant declining annual median concentrations in the Great Lakes for total PFCAs (C7 to C12), PFOA, total PFSAs, and PFOS between 2004 and 2017, with a significant drop-off in total PFSAs noted beginning around 2010 to 2011. Conversely, there was a dramatic increase in total SC-PFCA (C4 to C6) concentrations between the periods of 2000 to 2009 and 2015 to 2019. In comparisons between these two time periods, median concentrations of PFHxA, PFHpA, and PFOA increased by 3.0-, 16-, and 1.4-fold, respectively, and a large change was also observed for PFBA (870-fold) due to non-detect levels reported from 2000 to 2009. Median PFOS was also higher from 2015 to 2019 (2.6-fold), while PFBS and PFHxS were lower (1.7- and 7.8-fold). Higher concentrations of PFOS, PFPeA, PFHxA, PFHpA, and PFOA were observed in the combined urban and nonurban-influenced sites compared with open lake sites, although the differences were less than 2-fold (Muir and Miaz 2021).

4.3 Concentrations in Canadian biota

4.3.1 Urban and industrial areas

For urban (St. Lawrence River and Great Lakes) and industrial areas (for example, landfills and industrial sites), available measured biota concentrations for SC-PFCAs/PFSAs and LC-PFSAs were obtained from literature and graphed according to chain length and functional group in Figure 9 and

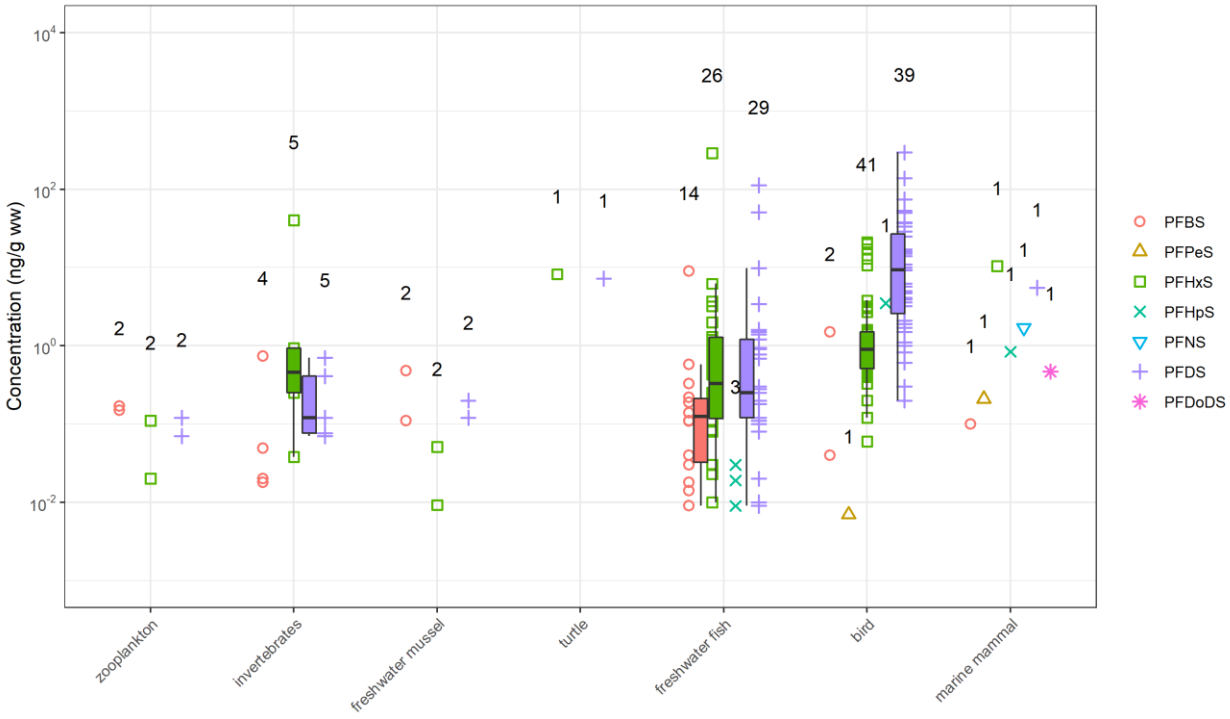

Figure **10**.

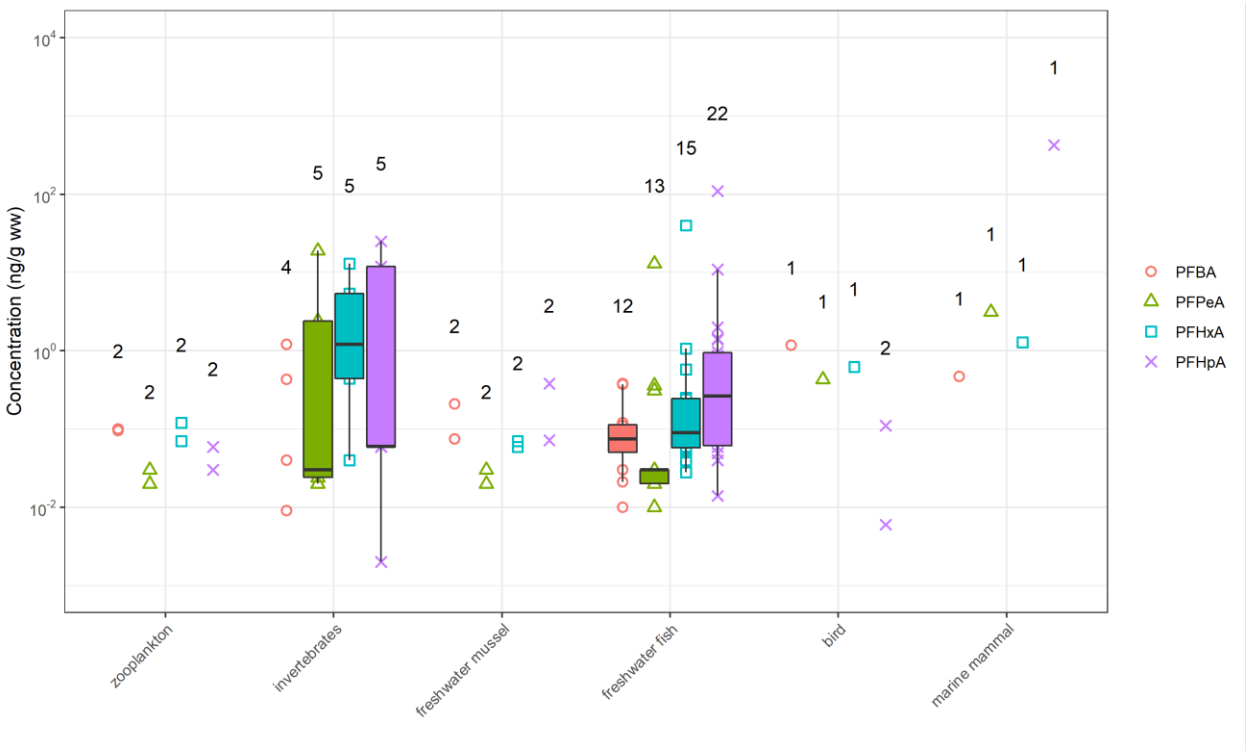

Figure 9. Concentrations of SC-PFCAs in biota in urban areas (ng/g ww).[11] These include zooplankton, invertebrates, freshwater mussel, freshwater fish, birds, and marine mammals. The numbers above each box represent the number of data points included.

[11] Moody et al. 2002; Furdui et al. 2007; Oakes et al. 2010; Awad et al. 2011; De Solla et al. 2012; Letcher et al. 2015; Munoz et al. 2017b; Wu et al. 2020; Barrett et al. 2021; Ren et al. 2021, 2022; Kaboré et al. 2022

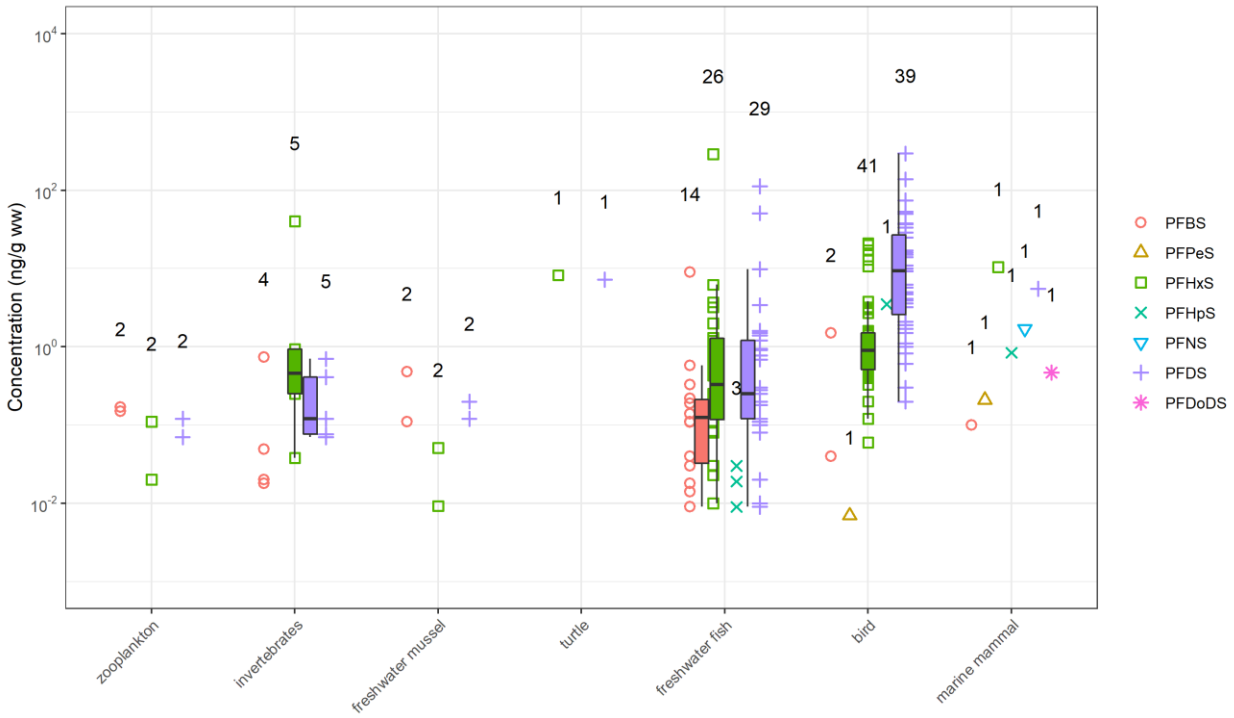

Figure 10. Concentrations of SC- and LC-PFSAs in biota in urban areas (ng/g ww).[12] These include zooplankton, invertebrates, freshwater mussel, turtle, freshwater fish, birds and marine mammals. The numbers above each box represent the number of data points included.

PFCAs and PFSAs were measured in bird eggs of European starling, black guillemot, thick-billed murre, northern fulmar, black-legged kittiwake, gulls, and bald eagle (Gebbink et al. 2011; Braune and Letcher 2013; Letcher et al. 2015; Gewurtz et al. 2016, 2018; Wu et al. 2020). PFDS had the highest maximum concentrations in European starling (*Sturnus vulgaris*) eggs at 295 ng/g ww, located near a landfill in Calgary, Alberta (Gewurtz et al. 2018). Mean concentrations of up to 50 ng/g ww were reported in bird eggs, with the highest concentration for PFDS found in gull eggs from colony sites in Hamilton Harbour in Lake Ontario (Gewurtz et al. 2016). Bald eagle eggs collected from the Great Lakes region had concentrations of PFCA and PFSA (C4 to C7) of up to 11 ng/g ww, with the highest concentration being for PFHxS, followed by PFHpS at 3.5 ng/g ww (Wu et al. 2020). PFPeS was only reported in one instance in bald eagle eggs in the Great Lakes region (Wu et al. 2020).

Freshwater amphipods (*Gammarus* or *Hyallela* sp.) and snapping turtles (*Chelydra serpentine*) were sampled from Welland River and Lake Niapenco, which are

[12] Moody et al. 2002; Furdui et al. 2007, 2008b; Oakes et al. 2010; Awad et al. 2011; Gebbink et al. 2011; De Solla et al. 2012; Houde et al. 2014; Letcher et al. 2015; Miller et al. 2015; Gewurtz et al. 2016; Munoz et al. 2017b; Gewurtz et al. 2018, Wu et al. 2020; Barrett et al. 2021; Point et al. 2021; Ren et al. 2021, 2022; Kaboré et al. 2022

downstream of the John C. Munro International Airport in Hamilton, Ontario (De Solla et al. 2012). Whole body amphipod arithmetic mean concentrations for PFHpS were highest at 40.2 ng/g ww, followed by PFHpA at 25.1 ng/g ww and then PFPeA at 19 ng/g ww. Snapping turtle plasma contained PFHxS with arithmetic mean concentrations that ranged from <0.1 ng/g ww to 8.2 ng/g ww as well as PFDS with arithmetic mean concentrations that ranged from 0.2 ng/g ww to 7.2 ng/g ww (De Solla et al. 2012).

PFCAs and PFSAs were measured in freshwater fish, including yellow perch (*Perca flavescens*) from the St. Lawrence River, Quebec, and in Lake Huron. Whole body mean concentrations for PFDS were 0.65 ng/g ww upstream of the wastewater treatment plant on the St. Lawrence River, and 0.25 ng/g ww to 0.78 ng/g ww downstream of the wastewater treatment plant. In Lake Huron, PFCAs (C4 to C7) and PFSAs (C4, C6, and C10) ranged from below the detection limit (<0.059 to <0.12) to 0.33, with PFBS having the highest mean concentration, followed by PFHxS at 0.19 ng/g ww (Ren et al. 2022). PFCAs (C4 to C7) and PFSAs (C4, C6, and C10) were measured in lake trout (*Salvelinus namaycush*) in the Great Lakes. Whole body mean concentrations ranged from below the detection limit (<0.059) to 9.8 ng/g ww, with PFDS having the highest mean concentration in Lake Erie (Furdui et al. 2007; Ren et al. 2021, 2022).

4.3.2 Canadian Arctic

Available measured biota concentrations for SC-PFCAs/PFSAs and LC-PFSAs were obtained from literature and graphed according to chain length and functional group in Figure 11 and Figure 12. PFDS was the only LC-PFSA measured in biota.

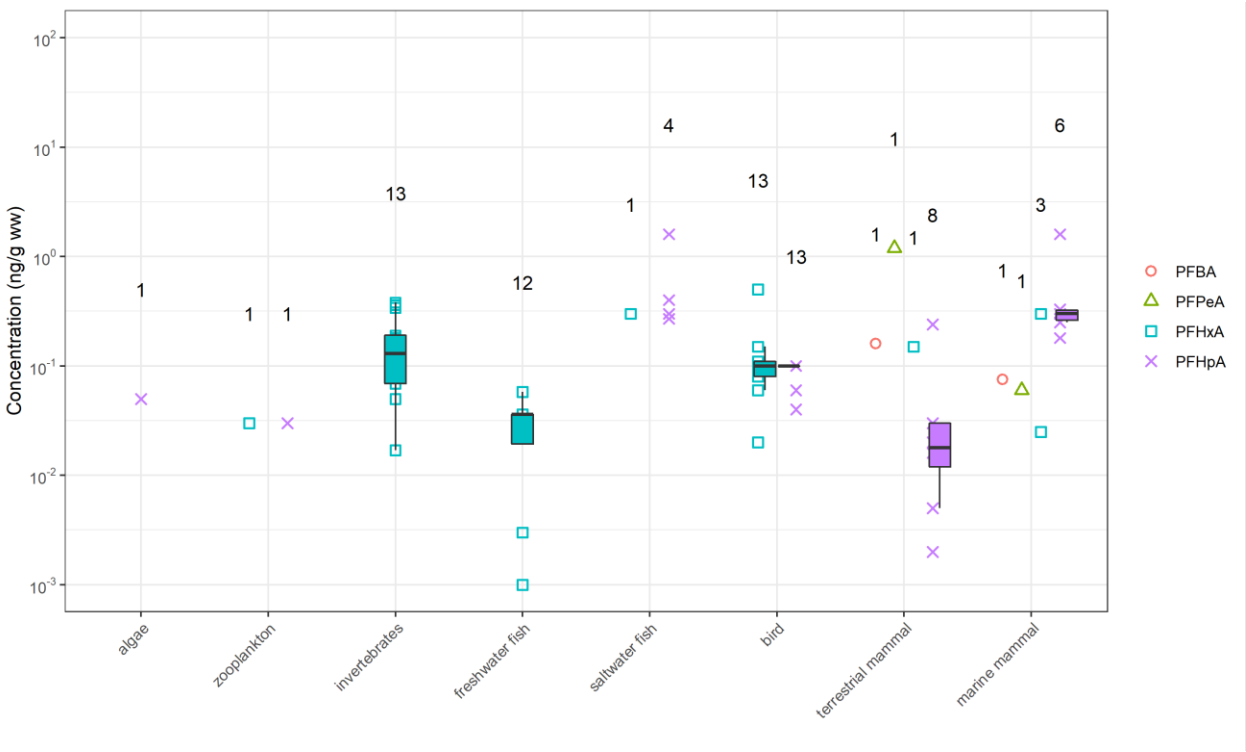

Figure 11. Concentrations of SC-PFCAs in Arctic biota (ng/g ww).[13] Arctic biota include algae, zooplankton, invertebrates, freshwater fish, saltwater fish, birds, terrestrial mammals, and marine mammals. The numbers above each box represent the number of data points included.

[13] Butt et al. 2007a,b, 2008; Powley et al. 2008; Kelly et al. 2009; Braune and Letcher 2013, Braune et al. 2014; Lescord et al. 2015; Larter et al. 2017; Letcher et al. 2018; Roos et al. 2021

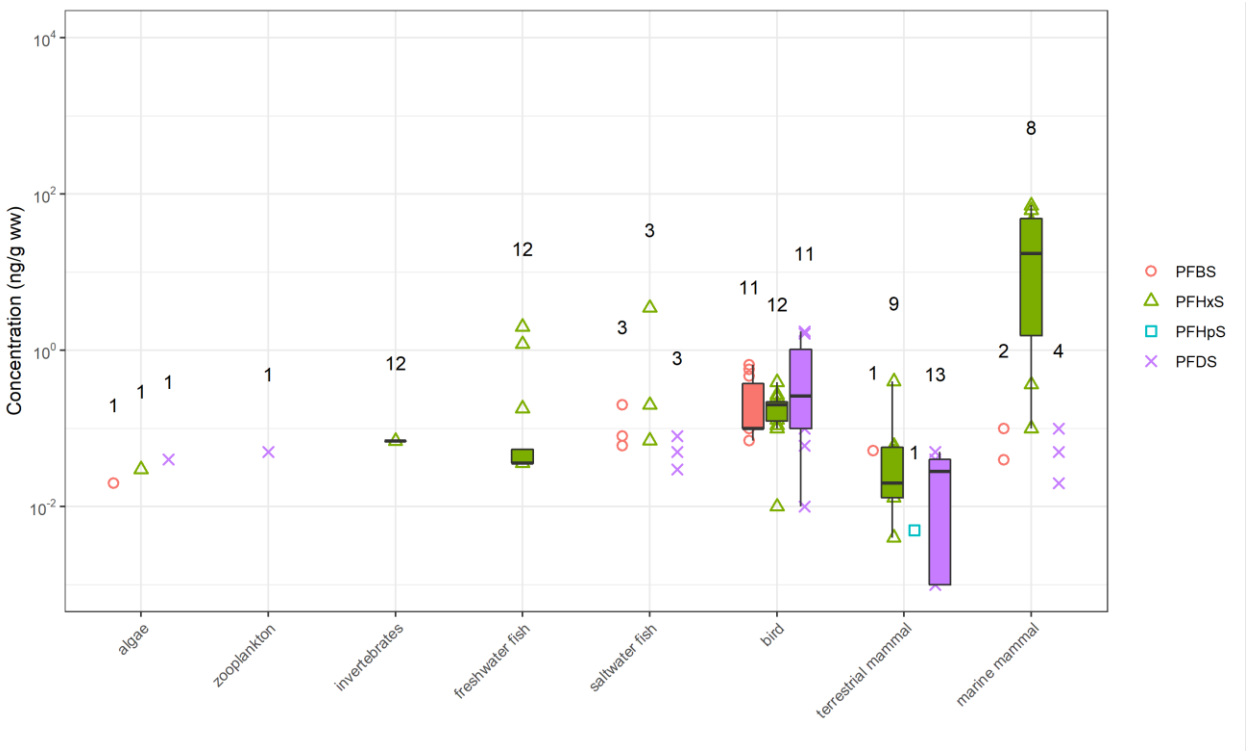

Figure 12. Concentrations of SC- and LC-PFSAs in Arctic biota (ng/g ww).[14] Arctic biota include algae, zooplankton, invertebrates, freshwater fish, saltwater fish, birds, terrestrial mammals, and marine mammals. The numbers above each box represent the number of data points included.

Braune and Letcher (2013) measured PFSAs, PFCAs, and precursor compounds in seabird eggs in the Canadian Arctic. Of the birds that were sampled (northern fulmar [*Fulmarus glacialis*], thick-billed murre [*Uria lomvia*], black-legged kittiwake [*Rissa tridactyla*], black guillemot [*Cepphus grylle*], and glaucous gulls [*Larus hyperboreus*]), glaucous gulls from Prince Leopold, Nunavut, had the highest mean egg concentration with PFDS measured at 1.75 ng/g ww. Mean measured egg concentrations were generally less than the limit of detection (<0.1 ng/g ww to <0.2 ng/g ww) for PFHxA and PFHpA (Braune and Letcher 2013). Braune et al. (2014) determined that mean measured liver concentrations of PFBS and PFHxS in thick-billed murres and northern fulmars ranged from ND to 0.65 ng/g ww (LOD: 0.1 ng/g ww), with the highest mean concentration found for PFBS in thick-billed murre. Liver concentrations of SC-PFCAs ranged from <0.1 ng/g ww to 0.15 ng/g ww, with PFHxA having the highest concentration at 0.15 ng/g ww.

SC-PFSAs measured in muscle and whole body of Arctic freshwater fish (Arctic char [*Salvelinus alpinus*]) and saltwater fish (Arctic cod [*Boreogadus saida*], capelin [*Mallotus villosus*], and *Salmo* sp.) were generally below detection limits (<0.03 whole body to

[14] Smithwick et al. 2005b; Butt et al. 2007a,b, 2008; Powley et al. 2008; Kelly et al. 2009; Reiner et al. 2011; Braune and Letcher 2013, Braune et al. 2014; Lescord et al. 2015; Gamberg et al. 2017; Larter et al. 2017; Letcher et al. 2018; Roos et al. 2021

<0.2 ng/g ww muscle; Powley et al. 2008; Kelly et al. 2009; Lescord et al. 2015). Kelly et al. (2009) measured concentrations of up to 3.5 ng/g ww and 1.6 ng/g ww PFHxA and PFHpA, respectively, in *Salmo* sp. in the Hudson Bay. Concentrations of PFHxA in muscle of Arctic char from Nunavut had measurable mean concentrations of 0.058 ng/g ww; however, levels in whole body were below the LOD (0.036 ng/g ww; Lescord et al. 2015).

Overall, SC- and LC-PFSAs had higher concentrations in marine mammals than in terrestrial mammals. Ringed seals (*Phoca hispida*) across the Canadian Arctic had mean concentrations ranging up to 2.5 ng/g ww in liver (Butt et al. 2007a, 2008). Beluga whales (*Delphinapterus leucas)* located near Newfoundland did not have measurable levels of PFHxS in their livers for the year 1986 (Reiner et al. 2011). However, beluga whales from Hudson Bay had maximum PFHxS and PFDS concentrations of up to 3.76 ng/g ww and 5.12 ng/g ww, respectively, in their livers from 1999 to 2004 (Kelly et al. 2009).

All SC-PFCAs, with the exception of PFHpA, were below the method detection limits (<0.025 ng/g ww to <0.075 ng/g ww lipid) for measurements in fat of polar bears located in the Hudson Bay during the years 2013 to 2014. PFHpA had a maximum geometric mean lipid concentration of 0.33 ng/g ww (Letcher et al. 2018). PFBS and PFHxS had geometric mean lipid concentrations of 0.04 ng/g ww and 8.28 ng/g ww, respectively (Letcher et al. 2018). The average concentration of PFHxS in the Northwest Territories and Nunavut in 2002 ranged from 35.9 ng/g ww to 71.4 ng/g ww (Smithwick et al. 2005b).

All SC-PFCAs and some SC-PFSAs and LC-PFSAs (C4, C6, C7, C10) were detected in moose (*Alces alces*) in the Northwest Territories, with PFHxS having the highest maximum concentration in liver of 0.106 ng/g ww, followed by PFBS at 0.087 ng/g ww (Larter et al. 2017). PFHxS, PFDS, and PFHpA were measured in caribou (*Rangifer tarandus*) from Nunavut between 2002 and 2016. The maximum liver concentrations were up to 0.6 ng/g ww for PFHxS, followed by PFDS at 0.3 ng/g ww (Roos et al. 2021).

Lescord et al. (2015) sampled Arctic char and benthic and pelagic invertebrates in Meretta Lake and Resolute Lake, Nunavut, which are downstream of the local airport. These lakes were likely affected by wastewater discharges (with little treatment) from both the airport and a military base during the years 1949 to 1998. Only PFHxS and PFHxA were analyzed. PFHxS was not detected in benthic invertebrates but was detected in juvenile and adult Arctic char muscle tissue with mean concentrations of up to 2.0 ng/g ww and 1.2 ng/g ww, respectively. PFHxA was detected in benthic invertebrates with mean concentrations of up to 0.38 ng/g ww and in juvenile/adult Arctic char muscle tissue with mean concentrations of up to 0.04 ng/g ww. The total PFAS concentration in Arctic char from Meretta and Resolute Lakes was 100 times higher compared to fish found in nearby reference lakes.

4.3.3 Release events

In June 2000, 22 000 L of AFFF was accidentally released at Lester B. Pearson International Airport (Toronto, Ontario) due to a fire alarm malfunction. The AFFF then entered Etobicoke Creek, a tributary to Lake Ontario (Moody et al. 2002). Fish sampling occurred 21 and 153 days post-incident at Etobicoke Creek upstream and downstream of the release site. With the exception of PFHpA, all analyzed SC-PFCAs and SC-PFSAs (C4 and C6) were detected in common shiner at higher maximum concentrations downstream than upstream of the release site. The source of the upstream concentrations is not known. PFBS was not detected upstream of the release site.

In August 2005, 48 000 L of AFFF entered Etobicoke Creek. The foam was used to douse an aircraft fuselage fire (Oakes et al. 2010). Oakes et al. (2010) analyzed blacknose dace (*Rhinichthys atratulus*) liver collected 9 and 122 days post-incident at Etobicoke Creek upstream and downstream of the release site. PFHxS, PFHxA, and PFHpA were below the limit of quantification upstream and downstream of the release site. The results by Oakes et al. (2010) suggested that the AFFF that was used likely contained telomerized polyfluorinated materials and that the use of this formulation may be attributed to the phase-out of perfluorinated acids in AFFF.

In July 2013, approximately 33 000 L of AFFF entered Lake Mégantic and the Chaudière River near the municipality of Lac-Mégantic, Quebec (Munoz et al. 2017b). Archived white sucker (*Catostomus commersonii*) muscle tissue collected two years prior to the incident from Lake Mégantic was used as a reference. White suckers from Lake Mégantic and the Chaudière River were sampled 1, 3, and 12 months post-incident. The reference tissue indicated that PFBA, PFPeA, PFHxS, and PFDS were already present in Lake Mégantic and the Chaudière River, but the sources were not identified. Concentrations of SC-PFCAs and SC-PFSAs (C4, C6, and C7) ranged from less than the limit of detection (LOD) to 0.37 ng/g ww post-incident and from <LOD to 0.36 ng/g ww 12 months post-incident. Maximum PFBA concentrations were lower post-incident. Conversely, maximum PFPeA, PFHxS, and PFDS concentrations increased post-incident. PFHxA, PFHpA, and PFHpS were not detected in reference samples. However, PFHxA and PFHpA were measured post-incident, and PFHpS was detected only 12 months post-incident. PFBS was not detected either pre- or post-incident. The authors stated that it was unlikely that the PFAAs detected near the accident site were from the AFFF formulation. However, the presence of SC-PFCAs could have been due to the environmental transformation of fluorotelomer-based PFAS.

PFAS are resistant to heat and chemical extremes, which makes most conventional treatment technologies ineffective for PFAS removal or destruction. Additionally, different treatment technologies are limited in their ability to be widely used and are thus limited to locations that are economically and logistically feasible. However, PFAS treatment and remediation technologies are rapidly evolving and advancing for potential application at contaminated sites.

4.4 Concentrations in marine wildlife, terrestrial wildlife, and birds worldwide

A review of available literature from 2002 to 2022[15] indicates that measured concentrations of SC-PFCAs/PFSAs in marine mammals, terrestrial wildlife, and birds outside of Canada are generally higher than those measured in Canada.

Antarctic gentoo penguins (*Pygoscelis papua*) and Adélie penguins (*Pygoscelis adeliae*) had mean egg concentrations of PFHpA of between 0.5 ng/g ww and 2.5 ng/g ww, whereas PFHxS was not detected for either species of penguins (Schiavone et al. 2009). Schiavone et al. (2009) indicate that Antarctic penguins feed at the top of the polar marine food chain and, due to their non-migratory and non-nomadic species breeding, tissue concentrations of PFAS are an indication of local contamination. In the dung of Papua penguins, PFBS concentrations were between 10.9 ng/g and 45.9 ng/g, between 2.17 ng/g and 3.77 ng/g for PFHxS, and between 19.9 ng/g and 237 ng/g for PFHxA. PFDS, PFBA, PFPeA, and PFHpA were below the method limit of quantification (that is, 0.8 ng/g to 6.36 ng/g for PFCAs, and 2.6 ng/g to 23.9 ng/g for PFSAs; Llorca et al. 2012).

SC-PFCAs and SC-PFSAs (C4 to C7), as well as PFDS, were detected in marine wildlife in various compartments such as dung, liver, brain, blood, lipid, muscle, plasma, serum, or kidney. Polar bears (*Ursus maritimus*) from Greenland and Svalbard, Norway, had measured concentrations of PFBS, PFHxS, PFHpS, PFDS, PFHpA, and PFHxA. The highest maximum liver concentration was measured for PFHxS at 4430 ng/g ww (Smithwick et al. 2005b). Maximum liver concentrations ranged from 0.071 ng/g to 390 ng/g ww for SC-PFCAs/PFSAs and LC-PFSAs in other marine wildlife: seals (for example, *Phoca vitulina*, *Leptonychotes weddellii*); whales (for example, *Delphinapterus leucas*, *Orcinus orca*); dolphins (for example, *Tursiops truncates*, *Pontoporia blainvillei*); porpoises (for example, *Neophocaena phocaenoides*, *Phocoena phocoena*), shark (*Sphyrnidae* sp.); saltwater fish (for example, *Salmo salar, Oreochromis* sp.); turtles (*Caretta caretta*) (that is, Tseng et al. 2006; Gebbink et al. 2016). Tilapia (*Oreochromis* sp.; sampled from a fish market in Taiwan) had the highest maximum concentration at 390 ng/g for PFPeA (Tseng et al. 2006).

[15] Giesy and Newsted 2001; Kannan et al. 2002a; Kannan et al. 2002b; Kannan et al. 2002c; Taniyasu et al. 2002; Taniyasu et al. 2003; Corsolini and Kannan 2004; Bossi et al. 2005a; Bossi et al. 2005b; Gulkowska et al. 2005; Houde et al. 2005; Keller et al. 2005; Smithwick et al. 2005a; Van de Vijver et al. 2005; Verreault et al. 2005; Falandysz et al. 2006; Gulkowska et al. 2006; Nakata et al. 2006; Olivero-Verbel et al. 2006; Shaw et al. 2006; Tseng et al. 2006; Senthilkumar et al. 2007; Verreault et al. 2007; Holmstrom and Berger 2008; Ishibashi et al. 2008a; Leonel et al. 2008; Li et al. 2008; Wang et al. 2008; Ye et al. 2008a,b; Yoo et al. 2008; Ahrens et al. 2009; Berger and Haukås 2005; Herzke et al. 2009; Meyer et al. 2009; Miljeteig et al. 2009; Quinete et al. 2009; Schiavone et al. 2009; Yeung et al. 2009; Bao et al. 2010; Bengtson Nash et al. 2010; Delinsky et al. 2010; Flanary et al. 2012; Holmstrom et al. 2010; Naile et al. 2010; O'Connell et al. 2010; Wille et al. 2010; Zhang et al. 2010; Bao et al. 2011; Murakami et al. 2011; Reiner et al. 2011; Wille et al. 2011; Zhang et al. 2011; Bytingsvik et al. 2012; Greaves et al. 2012; Llorca et al. 2012; Morales et al. 2012; Rotander et al. 2012; Shi et al. 2012; Yang et al. 2012; Zhou et al. 2012; Greaves et al. 2013; Leat et al. 2013; Naile et al. 2013; Persson et al. 2013; Riget et al. 2013; Wang et al. 2013b; Young et al. 2013; Bayat et al. 2014; Routti et al. 2015; Bost et al. 2016; Gebbink et al. 2016; Routti et al. 2016; Taylor and Johnson 2016; Dassuncao et al. 2017; Schlabach et al. 2017; Liu et al. 2018a; Tartu et al. 2018; Göckener et al. 2021; Park et al. 2021; Sun et al. 2021; Sharp et al. 2021; Teunen et al. 2021; Androulakakis et al. 2022; Hong et al. 2022; Huang et al. 2022; O'Rourke et al. 2022; Nolen et al. 2022; Szabo et al. 2022; Wilkinson et al. 2022

In birds, SC-PFCAs/PFSAs and PFDS were measured in blood, egg, egg yolk, liver, plasma, serum, whole blood, or whole body. PFPeS and other LC-PFSAs were not analyzed. The highest maximum liver concentrations were measured for PFHxS in the grey heron (*Ardea cinerea;* sampled in Belgium) at 121 ng/g ww, followed by the Eurasian sparrowhawk (*Accipiter nisus*; sampled in Belgium) at 41 ng/g ww (Meyer et al. 2009). The highest maximum egg concentrations were found for PFPeA in cormorants (*Phalacrocorax carbo*) at 17.3 ng/g (Rüdel et al. 2011).

In terrestrial wildlife, PFBA, PFPeA, PFHxA, PFHpA, PFBS, PFHxS, and PFDS were measured in liver and serum. The highest maximum concentrations were found for PFHxS in the liver of wild American mink (*Neovison vison*) sampled in Sweden at 139 ng/g ww (Persson et al. 2013), followed by liver measurements in the wild American mink sampled in the United States at 85 ng/g ww (Kannan et al. 2002a). In blood serum, the average PFHxS concentrations detected in captive African lion (*Panthera leo*) and Bengal tiger (*Panthera tigris tigris*) were 0.091 ng/ml and 0.164 ng/ml, respectively. PFBS, PFHpA, and PFHxA were below the limits of quantification (that is, 0.05 ng/ml to 0.25 ng/ml) for both the Bengal tiger and the African lion (Li et al. 2008). The Chinese alligator (*Alligator sinensis*) had maximum concentrations of PFBA, PFPeA, PFHpA, PFBS, and PFHxS that ranged from 0.03 ng/mL to 1.5 ng/mL, with PFHxS having the highest maximum concentration (Wang et al. 2013a).

5.0 Toxicity

5.1 Acute and chronic toxicity

Available toxicity data for freshwater aquatic species for SC-PFCAs/PFSAs have endpoint values ranging from 32 mg/L to 20 250 mg/L (Table 2.). No data were found for the LC-PFSAs.

Table 2. Acute/chronic toxicity data for SC-PFCAs/PFSAs

Substance(s)	Species	Endpoint	Range of values	Reference
PFBS PFHxS	*Scenedesmus obliquus; Pseudokirchneriella subcapitata*	72h EC_{50}/IC_{50} (growth)	600–>20 250 mg/L	Rosal et al. 2010; Liu et al. 2008
PFBA PFPeA PFHxA PFHpA	*S. obliquus; P. subcapitata*; *Raphidocelis subcapitata*	72h EC_{50}/IC_{50} (growth)	82–>1000 mg/L	Boudreau et al. 2002b; Hoke et al. 2012
PFBA PFPeA PFHxA	Rainbow trout (*Oncorhynchus mykiss*);	96h LC_{50} (mortality)	32–13 795 mg/L	Hoke et al. 2012; Godfrey et al. 2017; Ulhaq et al. 2013

Substance(s)	Species	Endpoint	Range of values	Reference
	zebrafish (*Danio rerio*)			
PFBS	Zebrafish (*D. rerio*)	96h EC_{50} (mortality)	450 mg/L	Ulhaq et al. 2013
PFBA PFPeA PFHxA PFHpA	*Daphnia magna*	48h EC_{50} (immobilization)	96–>1000 mg/L	Boudreau et al. 2002b; Ding et al. 2012
PFHxA	*D. magna*	21d EC_{50} (reproduction)	776 mg/L	Barmentlo et al. 2015
PFHxA	*D. magna*	21d EC_{50} (population growth)	853 mg/L	Barmentlo et al. 2015
PFBA PFPeA PFHxA	*Brachionus calyciflorus*	24h LC_{50} (immobility)	110–140 mg/L	Wang et al. 2014
PFBS	*Vibrio fischerii*	15 min EC_{50} (luminescence)	8386 and 17 520 mg/L	Rosal et al. 2010
PFHxA PFBA	Zebrafish liver cells (*D. rerio*)	96h EC_{50} (cell viability)	500 ppm (PFHxA) 563 ppm (PFBA)	Mahapatra et al. 2016
PFBA PFPeA PFHxA	Duckweed (*Lemna gibba*)	7d IC_{50}	630–>2000 mg/L PFBA: >4.7M PFPeA: >3.8M PFHpA: >2.8M	Boudreau et al. 2002a; Boudreau et al. 2002b
PFBS PFHxS	Earthworm (*Eisenia fetida*)	30d NOEC (growth/mortality)	>1000 ng/g	Zhao et al. 2013a
PFHxS	Fathead minnow (*Pimephales promelas*)	42d NOEC (reproduction and development)	1200 µg/L	Suski et al. 2021
PFHxA	*Chlorella vulgaris;* *Skeletonema marinoi;* *Geitlerinema amphibium*	72h EC_{50} (growth)	12.84 mM; 4.72 mM; 3.18 mM	Latala et al. 2009

Substance(s)	Species	Endpoint	Range of values	Reference
PFHpA	*C. vulgaris;* *S. marinoi;* *G. amphibium*	72h EC_{50} (growth)	5.21 mM; 2.40 mM; 1.42 mM	Latala et al. 2009
PFBS PFHxS PFHpA	Earthworm (*E. fetida*)	21d exposure (mortality) (sandy loam soil spiked with PFAS)	100% survival at 0.1, 1, 10, and 1000 µg/kg dw for PFHxS and PFHpA 97.5% survival at 1000 µg/kg dw for PFBS 95% survival at 100 000 µg/kg dw for PFHxS and PFHpA	Karnjanapiboonwong et al. 2018
PFHxS PFHxA	Cell line from *Xenopus tropicalis*	48h±1h IC_{50} (cytotoxicity)	499 ppm 2217 ppm	Hoover et al. 2019
PFHxA	Northern bobwhite quail (*Colinus virginianus*)	90d LOAEL oral exposure via drinking water (reproduction, growth, survival)	0.10 ng/ml (LOAEL exposure concentration CTV; growth) 0.0149 µg/kg body wt/d (LOAEL ADI CTV; growth)	Dennis et al. 2021
PFHxA PFHxS	Zebrafish larvae (*D. rerio*)	5 days post-fertilization LC_{50} (mortality)	290 µM 340 µM	Annunziato et al. 2019

Substance(s)	Species	Endpoint	Range of values	Reference
PFBA PFPeA PFHpA	*Selenastrum capricornutum; C. vulgaris*	96h IC_{50} (growth)	>2.8 M− >3.8 M	Boudreau et al. 2002b
PFBA PFPeA PFHpA	*D. magna; Daphnia pulicaria*	48h EC_{50} (mortality)	>2.8 M− >4.7 M	Boudreau et al. 2002b

Abbreviations: EC_{50}, the concentration of a substance that is estimated to cause some effect on 50% of the test organisms; IC_{50}, the concentration of a substance that is estimated to cause some inhibition on 50% of the test organisms; LC_{50}, median lethal concentration; LOEC, lowest observed effect concentration; NOEC, no observed effect concentration; LOAEL, lowest observed adverse effect level

Perfluorinated substances are persistent and bioaccumulate preferentially in air-breathing marine mammals, terrestrial mammals, and birds (see section 3.0). It is expected that these substances would have greater potential for exposure and adverse effects in air-breathing organisms due to their greater bioaccumulation potential. New approach methodology endpoints such as multi-generational effects and endocrine-related effects, along with consideration of cumulative effects if possible, could help in characterizing the potential toxicity for SC-PFCAs/PFSAs and LC-PFSAs in air-breathing organisms.

Another potential method is the use of data from standard mammalian laboratory studies (for example, rat) as surrogates for toxicity in wildlife species. However, caution is needed when extrapolating from mammalian data to certain wildlife species. For example, Letcher et al. (2014) examined the *in vitro* hepatic metabolism of a fluoroalkyl sulfonamide precursor of PFOS (that is, N-EtFOSA) for the polar bear (*Ursus maritimus*), beluga whale (*Delphinapterus leucas)*, ringed seal (*Pusa hispida*), and laboratory rat (*Rattus rattus*). The authors expected that the *in vitro* incubation parameters for the polar bear, seal, and whale would be equivalent to those for the rat, given that they are all mammalian species. On the contrary, results showed that the extent of *in vitro* depletion of N-EtFOSA was equivalent for rat and polar bear microsomes (that is, >95%); however, the extent of *in vitro* depletion was lower for ringed seals (that is, 65%), and there was no significant depletion for the beluga whale in comparison to the rat. As a result, Letcher et al. (2014) indicated that interpretive caution must be exercised when comparing the absolute quantitative differences in the degree of metabolite depletion among different species. Nabb et al. (2007) showed that the clearance rates for 8:2 FTOH (a precursor to PFOA) in liver microsomes and cytosol differed among species, with rat > mouse > human > rainbow trout. Overall, the results indicated that 8:2 FTOH is extensively metabolized in rats and mice and, to a lesser extent, in humans and rainbow trout.

5.2 Mode of action

The toxicokinetics of perfluorinated substances have been studied in mammals (for example, bovine and human serum albumin, rats), where it was observed that perfluorinated substances bind strongly to plasma albumin and that transport into cells

is likely controlled by a combination of passive diffusion and active facilitation by transporter proteins such as organic anion transporter proteins. These proteins are renal transporters that facilitate the reabsorption of organic anions from urine back to blood and are thought to be responsible for the long metabolic half-life of some perfluorinated substances (Ng and Hungerbühler 2013, 2014). It was also observed that these proteins are more highly expressed in male rats than in females, which may explain the gender differences in clearance rates for PFOA. Perfluorinated substances have also been observed to bind to cytosolic fatty acid binding proteins, which are ubiquitous in a number of cell types and serve as a sink in some tissues.

Perfluorinated substances affect liver function, including lipid and lipoprotein metabolism. These substances can alter lipid metabolism through peroxisome proliferation, alter xenobiotic metabolism by activating the cytochrome CYP-450 system, and alter serum cholesterol levels by inducing or repressing key genes (Hickey et al. 2009). A review by Sonne (2010) indicated that CYP-450 biotransformation of some PFAS in polar bears can result in highly toxic metabolites that are retained in blood plasma and various tissues. Some PFAS may also induce endocrine disruption indirectly through metabolism of endogenous hormones or vitamins, and CYP-450 activity may act as a biomarker for exposure to perfluorinated substances (Sonne 2010).

In addition, perfluorinated substances are known to activate the peroxisome proliferating receptor (PPAR-α) in wildlife (for example, Lake Baikal seals, Ishibashi et al. 2008b; whales and dolphins, Kurtz et al. 2019; polar bears, Routti et al. 2019b), which increases the abundance of hepatic peroxisomes and induces peroxisomal and mitochondrial enzymes involved with β-oxidation, cytochrome P450 (CYP-450) fatty acid ω-oxidation, and cholesterol homeostasis via ligand-dependent activation of the hepatic PPAR-α (Holden and Tugwood 1999; Bosgra et al. 2005). Prolonged exposure to peroxisome proliferators can result in hepatocarcinogenesis, although marked differences in susceptibility between species have been observed. Although these studies do not specifically analyze SC-PFCAs/PFSAs or LC-PFSAs, it is expected that the mode of action is applicable to all homologues of PFCAs and PFSAs.

5.3 Multi-generational effects

There is a concern for substances that have the potential to harm organisms at low concentrations and/or that have modes of toxic action beyond narcosis (for example, endocrine-related effects). The long-term ecological effects of highly persistent and bioaccumulative substances cannot be accurately predicted; however, these types of substances are acknowledged as having the potential to cause serious, irreversible impacts. Persistent substances, such as perfluorinated substances, remain in the environment for long periods of time, which increases the probability and duration of exposure as well as the potential for long-range transport, resulting in regional or global contamination. A substance that does not naturally occur in the environment may also have an increased potential to cause harm, as organisms may not have evolved specific strategies for mitigating exposures and effects (Macleod et al. 2014). Multi-generational

toxicity studies can be used as a tool to determine the long-term ecological effects of substances such as SC-PFCAs/PFSAs and LC-PFSAs as these substances are extremely persistent (see section 4.2). Some studies indicate that these substances can be bioaccumulative in air-breathing organisms (see section 3.3.1). Table 3. presents the studies currently available on multi-generational effects.

Table 3. Available multi-generational toxicity data

Substance	Species	Endpoint	Response	Reference
PFBS	Marine medaka (*Oryzias melastigma*)	Intestinal alterations; mortality	F1 had significantly increased mortality (up to 60%) compared to F0 (up to 40%) at 2.9 and 9.5 µg/L. F0 and F1 had intestinal inflammation.	Chen et al. 2018
PFBS	Marine medaka (*Oryzias melastigma*)	Reproduction (life cycle exposure – embryo to sexual maturity)	PFBS transferred to F1 offspring eggs but PFBS not detected in F1 adults and F2 eggs.	Chen et al. 2019
PFBS	*Chironomus riparius*	Survival; growth; development; reproduction	Larvae were exposed to 10 µg/L for 10 generations: • Reduced growth for some generations • Higher mortality in G2 offspring • Significant differences in development time in G8 and G10 • Lowered egg production in G6 and G10	Marziali et al. 2019
PFBS	*Caenorhabditis elegans*	Lethality; locomotion; reproduction; lifespan; growth; chemotactic behaviour	6 generations: • Reduced life span and brood size in parents at >0.1 mM • No effect on reproduction and lifespan at <0.01 mM • Multi-generational exposure at 0.0005 mM affected F4 and F5 progeny • 0.01–2.0 mM retarded parent locomotion behaviour	Chowdhury et al. 2021

PFBS	*Caenorhabditis elegans*	Lipid metabolism	4 generations: • Stimulated lipid content in F4 but not F1 • Lipid metabolism and pathway were disturbed differently from F1 to F4	Li et al. 2021
PFHxS	*Caenorhabditis elegans*	Lipid metabolism	4 generations: • Stimulated lipid content in F1 and F4 • Lipid metabolism and pathway were disturbed similarly in F1 and F4	Li et al. 2021

5.4 Endocrine-related effects

In some instances, substances may cause adverse effects in living organisms by interfering with the normal functioning of the endocrine system. Adverse effects may include delayed or impaired growth, altered intellectual and sexual development, increased susceptibility to certain cancers, disturbances in immune and nervous system function, and a lowered ability to reproduce or produce healthy offspring. Various studies indicate that endocrine-active substances may have the most pronounced effects during early developmental periods (such as prenatal and early postnatal development), when hormone-sensitive systems are developing (HC 2022). Available laboratory studies related to endocrine-related effects (sub-organismal and organism-level) for SC-PFCAs/PFSAs and one LC-PFSA (that is, PFDS) are provided in Table 4. and Table 5. .

Table 4. Examples of endocrine-related and other effects for SC-PFCAs/PFSAs and LC-PFSAs

Substance(s) *(tested separately unless otherwise indicated)*	**Species**	**Endpoint**	**Value**	**Response/effect**	**Reference**
PFHxS PFHpS PFPeA PFHxA PFHpA	White leghorn chicken (*Gallus domesticus*) embryo hepatocytes	Gene expression	10–50 µM	PFHxS and PFHpS resulted in upregulation of various genes related to metabolism and protein binding.	Hickey et al. 2009

Substance(s) *(tested separately unless otherwise indicated)*	**Species**	**Endpoint**	**Value**	**Response/effect**	**Reference**
				C5–C7 PFCAs resulted in upregulation of mRNA.	
PFHxS	White leghorn chicken (*Gallus domesticus*)	Gene expression	≥890 ng/g	Hepatic mRNA expression of two TH–responsive genes was upregulated in liver tissue of embryos; mRNA of phase I metabolizing enzyme, cytochrome P450 3A37 induced	Cassone et al. 2012a
PFHxS	White leghorn chicken (*Gallus domesticus*)	Pipping success	<38 000 ng/g	63% pipping success; tarsus length and embryo mass significantly decreased	Cassone et al. 2012a
PFHxA	White leghorn chicken (*Gallus domesticus*)	Pipping success	>9700 ng/g	80% pipping success; tarsus length and embryo mass not significantly affected	Cassone et al. 2012a
PFHxA	White leghorn chicken (*Gallus domesticus*)	Gene expression	>9700 ng/g	No mRNA transcripts were significantly affected	Cassone et al. 2012b
PFHxA	White leghorn chicken	24h cell cytotoxicity	30 or 50 µM	Significantly decreased cell viability	Vongphachan et al. 2011

Substance(s) *(tested separately unless otherwise indicated)*	**Species**	**Endpoint**	**Value**	**Response/effect**	**Reference**
	(*Gallus domesticus*)				
PFHxA PFHpA	White leghorn chicken (*Gallus domesticus*) embryonic neuronal cells	Gene expression (thyroid responsive genes)	3 or 10 µM	Upregulation of thyroid responsive genes	Vongphachan et al. 2011
PFHxA PFHpA	Herring gull (*Larus argentatus*)	24h cell cytotoxicity	3 or 10 µM	No effect	Vongphachan et al. 2011
PFHxA PFHpA	Herring gull (*Larus argentatus*) embryonic neuronal cells	Gene expression	3 or 10 µM	Upregulation of signal transduction and transcription factor activity	Vongphachan et al. 2011
PFPeA PFHxA PFHpA PFDS	Rainbow trout (*Oncorhynchus mykiss*)	14d plasma vitellogenin levels	250 ppm (with respect to diet ww)	Effects from C5–C7 PFCAs were not significant compared to 17β-estradiol PFDS had minor increase compared to 17β-estradiol	Benninghoff et al. 2011
PFDS	Northern pike (*Esox lucius*)	Plasma vitellogenin levels	8.1–15.4 ng/g ww	Possible correlation between vitellogenin expression in liver and vitellogenin protein activity in plasma	Houde et al. 2013
Mixture of: PFBA PFHxA	Fathead minnow (*Pimephales*	Comparison of altered transcripts	PFBA: 0.05 µg/L	**Mixture response:**	Rodriguez-Jorquera et al. 2019

Substance(s) *(tested separately unless otherwise indicated)*	**Species**	**Endpoint**	**Value**	**Response/effect**	**Reference**
PFHpA PFOA PFNA PFDA PFOS	*promelas*) (adult male)	In liver and whole blood	<u>PFHxA:</u> 0.1 µg/L <u>PFHpA:</u> 0.1 µg/L <u>PFOA:</u> 0.2 µg/L <u>PFNA:</u> 0.05 µg/L <u>PFDA:</u> 0.05 µg/L <u>PFOS:</u> 0.35 µg/L	The number of altered genes in blood was 5–10x greater than with liver induction of fatty acid transport/ metabolism, induction of xenobiotic metabolism (clearance), induction of mitochondria effects, induction of telomerase-associated genes, and induction of immune system-related genes in both tissues.	
PFBA PFPeA PFHxA PFHpA PFHxS	Lake Baikal seal (*Pusa sibirica*)	Ratio PPAR-α activation compared to PFOA (=1)	7.8–250 µM	Ratios ranged from 0.26 to 0.89 where PFHpA had the greatest induction, followed by PFPeA, PFHxA, PFBA, and PFHxS. PFHxS was activated but PFBS was not.	Ishibashi et al. 2011
PFPeA PFHxA PFHpA PFHxS	Marine mussel (*Mytilus*	P-glycoprotein	100 nM	Fluoroescence ranged from <15 to 20 where C5–C7	Stevenson et al. 2006

Substance(s) *(tested separately unless otherwise indicated)*	**Species**	**Endpoint**	**Value**	**Response/effect**	**Reference**
PFDS	*californianus*)	transporter activation		PFCAs and PFDS were not significant chemo-sensitizers. PFHxS was a significant P-glycoprotein inhibitor and chemo-sensitizer.	
PFHxS	Northern leopard frog (*Rana pipiens*)	40d growth and development	0.01–1 mg/L	Delayed metamorphosis	Hoover et al. 2017

The PPAR-α mechanism has been proposed as a mode of action for liver toxicity in mammals. However, there is only one study (Ishibashi et al. 2011) that has examined the relative potency of the PPAR-α mechanism in a marine mammal, that is, the Lake Baikal seals. Other studies examined a variety of endocrine responses such as thyroid response, vitellogenin expression, or chemosensitivity. However, it is unclear whether these sub-organismal responses can be directly linked to an actual gross effect on the whole organism or collectively at the population level. Additionally, the relative potency amongst the various pathways (for example, thyroid, estrogen, chemosensitivity, or PPAR-α) is unclear, and the relevance of these responses amongst the various pathways and various species is also unclear.

Read-across for perfluorinated substances on the basis of chain length can be difficult due to the inconsistent responses for the same endpoint amongst various species and taxonomic groups. Ishibashi et al. (2011) found that, relative to PFOA, the Lake Baikal seal PPAR-α was more strongly activated by PFHxS but not by PFBS. In addition, relative to PFOA, PFPeA had greater activation than PFHxA. No studies comparing relative activation of other SC-PFCAs and SC-PFSAs with PFOS appear to have been conducted. Additionally, a single species may not be representative within a taxonomic group. For example, there was no impact on cell cytotoxicity from PFHxA in white leghorn chicken (Cassone et al. 2012b), but there was an impact on cell cytotoxicity from PFHxA in herring gulls (Vongphachan et al. 2011). Houde et al. (2013) showed that there was no correlation between PFDS and plasma vitellogenin in rainbow trout, but PFDS may be correlated to plasma vitellogenin levels in northern pike. Other laboratory-based endocrine-related studies have shown observed effects for SC-PFCA/PFSAs mixtures and/or PFDS in various wildlife species, including top predators

(that is, Nobels et al. 2010; Gorrochategui et al. 2016; Annunziato et al. 2019; Menger et al. 2020; Omagamre et al. 2020; Wang et al. 2020; Rericha et al. 2021; Solé et al. 2021). Some illustrative examples are described in Table 4..

5.5 Cumulative effects

While the range of different PFAS examined in many studies has historically been relatively limited, studies have increasingly noted broad occurrence and co-exposure to a range of PFAS. With respect to field-based wildlife studies, it is difficult to uniquely distinguish effects caused by exposure to SC-PFCAs/PFSAs and LC-PFSAs, given that exposures from other PFAS (for example, PFOS or PFOA) or other contaminants cannot be excluded (Knudsen et al. 2007; Letcher et al. 2010; Liu et al. 2018b; Routti et al. 2019a; Hansen et al. 2020). PFAS (including related substances) are also often summed as a group and statistically correlated with the effect observed.

For example, a mixture of PFAS (i.e, PFHxS, PFOS, PFOA, and C9 to C14 PFCAs) was associated with the disruption of thyroid hormone homeostasis in polar bears (*Ursus maritimus*) from the Barents Sea (Bourgeon et al. 2017). However, these polar bears also had concentrations of 38 organochlorine substances, including polychlorinated biphenyls (PCBs), polybrominated diphenyl ethers (PBDEs), and 10 phenolic substances as well as 8 other PFAS that may also have contributed to the effect observed. Liu et al. (2018a) analyzed pooled polar bear serum from the Hudson Bay and Beaufort Sea subpopulations in the Canadian Arctic and found 5 classes of PCB metabolites, 4 classes of perfluorinated sulfonates, and 4 classes of other polychlorinated substances (that is, chlorinated aromatics, tetrachloro aromatic sulfate, heptachlorinated hydroxylated nitroaromatics, and hexachlorinated substances). Knudsen et al. (2007) measured insecticides (for example, mirex), PFAS, hexachlorocyclohexanes, toxaphenes, dioxins, furans, PCBs, brominated substances, endosulfans, and mercury in northern fulmars (*Fulmarus glacialis*) from the Barents Sea. Gao et al. (2020b) measured 3108 substances (388 contaminants and 2720 metabolites) in wild crucian carp (*Carassius auratus*) from Taihu Lake, China. These studies highlight that field-based mixture studies can be confounding when determining whether a singular substance or group of substances is affecting the health and condition of the wildlife species under investigation. Thus, a direct cause-effect correlation is difficult as statistical correlations do not by themselves imply causal relationships.

Additionally, concentrations of PFOS, PFOA, or long-chain (C9 and longer) PFCAs are usually found at higher levels than the SC-PFCAs/PFSAs due to legacy environmental contamination, which suggests that PFOS and PFOA can have greater contributions to the effect seen than any other substance. For example, Eggers Pedersen et al. (2015) indicated that the average brain total PFSA (ΣPFSA) concentration was 29 ng/g ww where PFOS accounted for 91% of the total concentration, and that the average ΣPFCA concentration was 99 ng/g ww where 3 LC-PFCAs combined accounted for 79% of the total concentration. As a result of their comparatively lower tissue concentrations, SC-PFCAs/PFSAs may appear to contribute little to observed effects. However, field studies are only indicative of current (and not potential) effects, and it is anticipated that

SC-PFCAs/PFSAs will comprise an increasingly large proportion of PFAS in the environment due to the increased use of short-chain PFAS in place of those that have been regulated. Furthermore, it is possible that tissue concentrations may increase beyond comparable increases in environmental concentrations if observed inhibitory effects of LC-PFCAs on the uptake of SC-PFCAs/PFSAs are reduced (Wen et al. 2017; see section 3.3.1). Future field studies will likely be better positioned to assess the relative contributions of SC-PFCAs and SC-PFSAs to overall toxicity.

Read-across for perfluorinated substances is also difficult when dealing with mixtures of PFAS due to species and sex differences in effects. For example, the ΣPFAS is correlated with eggshell thinning in great tits (Groffen et al. 2019) but not in ivory gulls (Miljeteig et al. 2012).

Recognizing the associated uncertainty, several field-based wildlife studies have shown statistical correlations with observed effects for SC-PFCA/PFSAs mixtures and PFDS in various wildlife species, including top predators (that is, Grønnestad et al. 2018; Guillette et al. 2020; Hansen et al. 2020; Parolini et al. 2020; Persson and Magnusson 2015; Sun et al. 2020; Sun et al. 2021; Tartu et al. 2014). Some illustrative examples are described in Table 5. .

Table 5. Examples of cumulative effects studies for PFAS

Substance mixture	Species	Endpoint	Value	Effect/observations	Reference
ΣPFAS (includes PFHxS, PFOS, PFOA, and long-chain PFCAs)	Bottlenose dolphin (*Tursiops truncatus*) in Florida and South Carolina (US)	Immune function	Up to 0.001 mg/mL plasma	Increases in indicators of inflammatory immunity in relation to PFAS	Peden-Adams et al. 2004a
ΣPFAS (includes PFHxS, PFOS, PFOA, and long-chain PFCAs)	Bottlenose dolphin (*Tursiops truncatus*) in Florida (US)	Life cycle and reproductive parameters	58–210 ng/g ww milk Sum PFCAs: 9.5 ng/g ww (mean) Sum PFSAs:	Sexually immature calves (<10 years; mean ΣPFAS = 1410 ng/g ww) were significantly more contaminated than mothers (mean ΣPFAS = 366 ng/g ww). PFAS levels in nulliparous females (not observed with	Houde et al. 2006c

Substance mixture	Species	Endpoint	Value	Effect/observations	Reference
			125 ng/g ww (mean) milk	calves) were significantly greater than those detected in uniparous females (observed with one calf), suggesting PFAS off-loading during or after parturition.	
ΣPFAS (includes PFBA, PFPeA, PFHxA, PFHpA, PFOA, long-chain PFCAs, PFHxS, PFOS)	Loggerhead and Kemp's Ridley turtles in South Carolina (US)	Biomarkers of immune function and clinical blood parameters	Up to 1.2E-05 mg/mL plasma	Low levels of PFAS (3.43–106 ng/mL) can alter biomarkers. PFBA and PFPeA were not detected.	Peden-Adams et al. 2004b
ΣPFSA (includes PFBS, PFHxS, PFOS, PFDS) ΣPFCA (includes PFHxA, PFHpA, PFOA, and long-chain PFCAs)	Polar bear (*Ursus maritimus*) in East Greenland	Brain neurotoxicity	29 ng/g ww ΣPFSAs 99 ng/g ww ΣPFCAs	Could not determine whether there is a correlation between neurochemical transmitter systems and brain-specific bioaccumulation with respect to cognitive processes and motor function. Results were inconclusive as to whether observed alterations in neurochemical signalling are causing negative effects on the neurochemistry of East Greenland polar bears.	Eggers Pedersen et al. 2015
ΣPFSA (includes PFBS, PFHxS, PFOS, PFDS)	Polar bear (*Ursus maritimus*) in East	Alteration in brain steroid levels	26 ng/g ww ΣPFSAs	Positive associations between PFSAs/PFCAs and 17α-	Eggers Pedersen et al. 2016

Substance mixture	Species	Endpoint	Value	Effect/observations	Reference
ΣPFCA (includes PFHxA, PFHpA, PFOA, and long-chain PFCAs)	Greenland		88 ng/g ww ΣPFCAs	hydroxypregnenolone and testosterone across brain regions were found, which indicate that an increase in PFAS agrees with an increase in levels of steroid hormones. However, the study could not determine whether alterations in brain steroid levels arise from interference with *de novo* steroid synthesis or via disruption of peripheral steroidogenic tissues in gonads and feedback mechanisms.	
ΣPFAS (includes PFHxS, PFOS, PFOA, long-chain PFCAs)	Polar bear (*Ursus maritimus*) in East Greenland	Liver lesions	114–3052 ng/g ww	Could not determine whether chronic exposure to ΣPFAS is associated with the appearance of liver lesions	Sonne et al. 2008
ΣPFAS (including PFHxS, PFOS, PFDS, PFHpA, PFOA, long-chain PFCAs)	Bottlenose dolphin (*Tursiops truncatus*) in South Carolina (US)	Immune system, kidney, liver function	0.002 mg/mL ΣPFSAs 0.0002 mg/mL ΣPFCAs	Chronic exposure appears to produce immune perturbations and tissue toxicity. PFAS may alter immune, hematopoietic, renal, and hepatic function.	Fair et al. 2013

Substance mixture	Species	Endpoint	Value	Effect/observations	Reference
ΣPFAS (includes PFOS, PFDS, PFHxS, long-chain PFCAs)	Lesser black-backed gull (*Larus fuscus*) in Norway	Sex ratio	Up to 1 ng/g	No correlation with sex ratio skewing	Erikstad et al. 2009
ΣPFAS (includes PFBA, PFPeA, PFHxA, PFHpA, PFOA, long-chain PFCAs, PFBS, PFHxS, PFOS, PFDS)	Great tits (*Parus major*) in Belgium	Egg laying, clutch size, hatching success, fledgling success, total breeding success	<0.26–1489 ng/g ww	Associated with eggshell thinning, reduced hatching success, early onset of egg laying, reduction in total breeding success PFPeA, PFHxA, PFHpA, PFBS, PFHxS were not detected	Groffen et al. 2019
ΣPFSA (includes PFHxS, PFHpS, PFOS) ΣPFCA (includes PFOA, long-chain PFCAs)	Black-legged kittiwake (*Rissa tridactyla*) and northern fulmar (*Fulmarus glacialis*) in Norway	Circulating thyroid hormone concentration	8.03–104 ng/g ww ΣPFSA 3.56–35.5 ng/g ww ΣPFCA	Positive associations between total thyroxin and PFHpS, PFOS, and PFNA in both species Disruption of thyroxin homeostasis may cause developmental effects in young birds	Nøst et al. 2012
ΣPFAS (includes PFHxS, PFOS, PFDS, long-chain PFCAs)	Ivory gull (*Pagophila eburnean*) in the Norweigan and Russian Arctic	Eggshell thickness, retinol (vitamin A), α-tocopherol (vitamin E)	30.9–164 ng/g ww	No association with eggshell thickness No association with α-tocopherol	Miljeteig et al. 2012

6.0 Summary

Summaries of the report's key findings are provided below.

Persistence and bioaccumulation
Owing to the extremely high strength of the C-F bond, which imparts high stability to SC-PFCAs/PFSAs and LC-PFSAs, it is expected that these substances will remain in the environment and within certain biota for a very long time. This is supported by a number of studies that show that SC-PFCAs/PFSAs do not degrade under environmentally relevant conditions. As a result, these substances are expected to persist in the environment and wildlife.

Empirical freshwater aquatic organism BCF/BAF data cannot be used alone to reliably predict the food web bioaccumulation for SC-PFCAs/PFSAs and LC-PFSAs. Results for typically tested model organisms (for example, fish) may underestimate the food web bioaccumulation potential. For SC-PFCAs/PFSAs and LC-PFSAs, air-breathing marine mammals, terrestrial mammals, and birds may have higher food web biomagnification and trophic magnification potential in comparison with the water-breathing organisms (such as fish) that are typically considered in bioaccumulation modelling. In general, it has been observed that species differences can result in inconsistent rates of bioaccumulation, making extrapolations amongst species and between chain lengths difficult. However, despite these difficulties, evidence from multiple studies suggests that BMF values for SC-PFCAs and SC-PFSAs can be comparable to those of PFOA and PFOS. Additionally, there are sufficient food web biomagnification data available for PFHxS to provide an early indication of that substance's overall bioaccumulation potential in marine mammals (for example, polar bears and dolphins) and birds.

Evidence that a substance is persistent and bioaccumulative may itself be a significant indication of its potential to cause environmental harm. Persistent substances remain in the environment for a very long time, which increases their probability, magnitude, and duration of exposure to wildlife. Persistent substances that are subject to long-range transport can result in regional or global contamination. Consequently, releases of SC-PFCAs/PFSAs and LC-PFSAs can lead to elevated concentrations in organisms across wide areas over a long period of time. These persistent and bioaccumulative substances also can biomagnify through the food chain, resulting in increased internal concentrations for top predators (Environment Canada 2006). As a result of their broad co-occurrence in the environment, many of these persistent and bioaccumulative substances may be present simultaneously in the tissues of organisms, increasing the likelihood and potential severity of harm.

Occurrence of SC-PFCAs/PFSAs and LC-PFSA in Canada
Some measured concentration data are available for most SC-PFCAs/PFSAs in most media within Canada. There are some monitoring data for water, snow, rain, sediment, landfill leachate, WWTP influent/effluent, and biota, although there is a lack of data for soils.

As might be expected given their extreme persistence, mobility, and long-range transport potential, SC-PFCAs/PFSAs and LC-PFSAs have been detected in many media and locations throughout Canada. Overall, average concentrations were higher in surface water (ranging from less than the LOD to 277 ng/L) than in snow (0.006 ng/L to 8.9 ng/L). PFHxS had the highest measured concentration in surface fresh water (49 600 ng/L). PFBA had the highest maximum concentrations in both rain and snow at 14 ng/L and 52 ng/L, respectively. PFHxS (96.5 ng/g dw) and PFBA (19.8 ng/g) had the highest measured maximum concentrations in Canadian sediment. SC-PFCAs/PFSAs have also been detected in landfill leachate, urban WWTPs, and Arctic lagoons in Canada.

Furthermore, SC-PFCAs/PFSAs and LC-PFSAs have also been detected in various Canadian biota, including those from urban and industrial areas, the Canadian Arctic, and in close proximity to release events (that is, from AFFF).

Although there is a growing amount of data demonstrating the broad presence of SC-PFCAs/PFSAs in the Canadian environment, there are few data available on the LC-PFSAs, with the exception of PFDS. It has been noted that current concentrations of SC-PFCAs/PFSAs and LC-PFSAs can reflect the contribution of precursors and salts that have already transformed or dissociated to the moiety of interest.

Ecotoxicity

Median toxicity values for acute and chronic toxicity tests for SC-PFCAs and select SC-PFSAs (PFBS and PFHxS) in freshwater aquatic organisms range from 32 mg/L to 20 250 mg/L. There is no information on acute or chronic toxicity data for the LC-PFSAs, although some endocrine-related effects data are available for PFDS. However, the acute and chronic toxicities in freshwater aquatic organisms likely do not reflect the toxicity and exposure potential in air-breathing marine mammals, terrestrial mammals, and avian species. SC-PFCAs/PFSAs and some LC-PFSAs demonstrate greater food web bioaccumulation in air-breathing marine mammals and avian species compared to freshwater aquatic organisms. The higher potential for food web bioaccumulation coupled with the persistent nature of these substances increases the probability and the duration of exposure and, ultimately, the probability of reaching internal toxicity thresholds. A number of multi-generational studies have demonstrated lethal and sub-lethal effects at much lower concentrations than those observed for acute and chronic toxicity tests. Various endocrine-related studies have also identified effects at concentrations orders of magnitude lower than those observed with traditional toxicity tests.

Due to the large number of PFAS that may be used and the demonstration of broad environmental occurrence and co-exposure to PFAS, it is expected that cumulative toxicity as a result of exposure to various PFAS chain lengths and functional groups will increase the ecological impact of SC-PFCAs/PFSAs and LC-PFSAs in Canada. Because of legacy concentrations of PFOS, PFOA, and LC-PFCAs, current field studies are limited in their ability to assess contributions of SC-PFCAs/PFSAs and LC-PFSAs to

cumulative effects; however, future studies should be better positioned to evaluate the relative contributions of these substances as a result of changes in use patterns.

Overall conclusions/summary

Despite the fact that substance-specific information is lacking for many of these PFAS, what is known about their persistence, mobility, bioaccumulation, and toxicity profiles on the basis of the empirical information presented throughout this report suggests that these SC-PFCAs/PFSAs and LC-PFSAs may share similar ecological concerns with PFAS that have been previously assessed and regulated. Given their extreme persistence properties, it is expected that these substances will remain and accumulate in the environment once released. Consequently, it is expected that the potential for adverse effects resulting from continued exposure to SC-PFCAs, SC-PFSAs, and LC-PFSAs will increase with increasing environmental loads.

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Appendix A. Non-exhaustive list of SC-PFCAs, their salts and their precursors

Table A-1. Non-exhaustive list of SC-PFCAs, their salts and their precursors (precursors as identified via CATABOL modelling)

CAS RN[a]	Chemical name	Common name or acronym	Inventory[b]
375-22-4	Perfluorobutanoic acid	PFBA	NDSL
2706-90-3	Perfluoropentanoic acid	PFPeA	NDSL
307-24-4	Perfluorohexanoic acid	PFHxA	NDSL
375-85-9	Perfluoroheptanoic acid	PFHpA	NDSL
68259-11-0	Pentanoic acid, nonafluoro-, ammonium salt	PFPeA ammonium salt	DSL
21615-47-4	Hexanoic acid, undecafluoro-, ammonium salt	PFHxA ammonium salt	DSL
6130-43-4	Heptanoic acid, tridecafluoro-, ammonium salt	PFHpA ammonium salt	DSL
1799-84-4	2-Propenoic acid, 2-methyl-, 3,3,4,4,5,5,6,6,6-nonafluorohexyl ester	Precursor	DSL
2043-47-2	1-Hexanol, 3,3,4,4,5,5,6,6,6-nonafluoro-	Precursor	DSL
2144-53-8	2-Propenoic acid, 2-methyl-, 3,3,4,4,5,5,6,6,7,7,8,8,8-tridecafluorooctyl ester	Precursor	DSL
17527-29-6	2-Propenoic acid, 3,3,4,4,5,5,6,6,7,7,8,8,8-tridecafluorooctyl ester	Precursor	DSL
52591-27-2	2-Propenoic acid, 3,3,4,4,5,5,6,6,6-nonafluorohexyl ester	Precursor	DSL
647-42-7	1-Octanol, 3,3,4,4,5,5,6,6,7,7,8,8,8-tridecafluoro-	Precursor	DSL
678-39-7	1-Decanol, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heptadecafluoro-	Precursor	DSL
13695-31-3	2-Propenoic acid, 2-methyl-, 2,2,3,3,4,4,4-heptafluorobutyl ester	Precursor	NDSL
54950-05-9	Butanedioic acid, sulfo-, 1,4-bis(3,3,4,4,5,5,6,6,7,7,8,8,8-tridecafluorooctyl) ester, sodium salt	Precursor	NDSL
40143-76-8	Perfluorohexane phosphonic acid	Precursor	Not on DSL or NDSL
40143-77-9	Bis(perfluorohexyl)phosphinic acid	Precursor	Not on DSL or NDSL

Abbreviations: DSL, Domestic Substances List; NDSL, Non-Domestic Substances List.
[a] CAS RN idenitified via DSL search engine and/or US EPA Chemistry Dashboard.

[b] Substances not appearing on the DSL are not used commercially in Canada above trigger quantities specified in the *New Substances Notification Regulations (Chemicals and Polymers)*. Substances listed on the NDSL are subject to notification and requirements set out in the *New Substances Notification Regulations (Chemicals and Polymers)*, but with reduced information requirements. Substances on the NDSL are those that are on the *US Toxic Substances Control Act* (TSCA) inventory.

Appendix B. Non-exhaustive list of SC-PFSAs, their salts and precursors

Table B-1. Non-exhaustive list of SC-PFSAs, their salts and precursors (precursors as identified via CATABOL modelling)

CAS RN[a]	Chemical name	Common name or acronym	Inventory[b]
375-73-5	Perfluorobutane sulfonic acid	PFBS	Not on the DSL or NDSL
2706-91-4	Perfluoropentane-1-sulfonic acid	PFPeS	Not on the DSL or NDSL
355-46-4	Perfluorohexane sulfonic acid	PFHxS	Not on the DSL or NDSL
375-92-8	Perfluoroheptane sulfonic acid	PFHpS	Not on the DSL or NDSL
29420-49-3	1-Butanesulfonic acid, 1,1,2,2,3,3,4,4,4-nonafluoro-, potassium salt	PFBS potassium salt	DSL
68259-10-9	1-Butanesulfonic acid, 1,1,2,2,3,3,4,4,4-nonafluoro-, ammonium salt	PFBS ammonium salt	DSL
3872-25-1	1-Pentanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,5-undecafluoro-,potassium salt	PFPeS potassium salt	DSL
68259-09-6	1-Pentanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,5-undecafluoro-,ammonium salt	PFPeS ammonium salt	DSL
3871-99-6	1-Hexanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro-, potassium salt	PFHxS potassium salt	DSL
68259-08-5	1-Hexanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro-, ammonium salt	PFHxS ammonium salt	DSL
55120-77-9	1-Hexanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro-, lithium salt	PFHxS lithium salt	DSL
60270-55-5	1-Heptanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-, potassium salt	PFHpS potassium salt	DSL
68259-07-4	1-Heptanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-, ammonium salt	PFHpS ammonium salt	DSL
117806-54-9	1-Heptanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-, lithium salt	PFHpS lithium salt	DSL
34454-97-2	1-Butanesulfonamide, 1,1,2,2,3,3,4,4,4-nonafluoro-N-(2-hydroxyethyl)-N-methyl-	Precursor	DSL
68298-12-4	1-Butanesulfonamide, 1,1,2,2,3,3,4,4,4-nonafluoro-N-methyl-	Precursor	DSL
34449-89-3	1-Butanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,4-nonafluoro-N-(2-hydroxyethyl)-	Precursor	DSL

CAS RN[a]	Chemical name	Common name or acronym	Inventory[b]
38850-58-7	1-Propanaminium, N-(2-hydroxyethyl)-N,N-dimethyl-3-[(3-sulfopropyl)[(tridecafluorohexyl)sulfonyl]amino]-, hydroxide, inner salt	Precursor	DSL
52166-82-2	1-Propanaminium, N,N,N-trimethyl-3-[[(tridecafluorohexyl)sulfonyl]amino]-, chloride	Precursor	DSL
53518-00-6	1-Propanaminium, N,N,N-trimethyl-3-[[(nonafluorobutyl)sulfonyl]amino]-, chloride	Precursor	DSL
56372-23-7	Poly(oxy-1,2-ethanediyl), a-[2-[ethyl[(tridecafluorohexyl)sulfonyl]amino]ethyl]-w -hydroxy-	Precursor	DSL
67584-42-3	Cyclohexanesulfonic acid, decafluoro(pentafluoroethyl)-, potassium salt	Precursor	DSL
67584-51-4	Glycine, N-ethyl-N-[(nonafluorobutyl)sulfonyl]-, potassium salt	Precursor	DSL
67584-52-5	Glycine, N-ethyl-N-[(undecafluoropentyl)sulfonyl]-, potassium salt	Precursor	DSL
67584-53-6	Glycine, N-ethyl-N-[(tridecafluorohexyl)sulfonyl]-, potassium salt	Precursor	DSL
67584-58-1	1-Propanaminium, N,N,N-trimethyl-3-[[(pentadecafluoroheptyl)sulfonyl]amino]-, iodide	Precursor	DSL
67584-62-7	Glycine, N-ethyl-N-[(pentadecafluoroheptyl)sulfonyl]-, potassium salt	Precursor	DSL
67939-94-0	1-Heptanesulfoamide, N,N',N" -[phosphinylidynetris(oxy-2,1-ethanediyl)]tris[N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-	Precursor	DSL
67939-95-1	1-Propanaminium, N,N,N-trimethyl-3-[[(nonafluorobutyl)sulfonyl]amino]-, iodide	Precursor	DSL
67939-97-3	1-Heptanesulfonamide, N,N'-[phosphinicobis(oxy-2,1-ethanediyl)]bis[N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-, ammonium salt	Precursor	DSL
67939-98-4	1-Heptanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-N-[2-(phosphonooxy)ethyl]-, diammonium salt	Precursor	DSL
68156-01-4	Cyclohexanesulfonic acid, nonafluorobis(trifluoromethyl)-, potassium salt	Precursor	DSL
68156-07-0	Cyclohexanesulfonic acid, decafluoro(trifluoromethyl)-, potassium salt	Precursor	DSL
68298-79-3	Poly(oxy-1,2-ethanediyl), a-[2-[ethyl[(nonafluorobutyl)sulfonyl]amino]ethyl]-w -hydroxy-	Precursor	DSL
68298-80-6	Poly(oxy-1,2-ethanediyl), a-[2-[ethyl[(undecafluoropentyl)sulfonyl]amino]ethyl]-w -hydroxy-	Precursor	DSL

CAS RN[a]	Chemical name	Common name or acronym	Inventory[b]
68298-81-7	Poly(oxy-1,2-ethanediyl), a-[2-[ethyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl]-w-hydroxy-	Precursor	DSL
68541-01-5	Benzoic acid, 2,3,4,5-tetrachloro-6-[[[3-[[(pentadecafluoroheptyl)sulfonyl]oxy]phenyl]amino]carbonyl]-, monopotassium salt	Precursor	DSL
68541-02-6	Benzoic acid, 2,3,4,5-tetrachloro-6-[[[3-[[(undecafluoropentyl)sulfonyl]oxy]phenyl]amino]carbonyl]-, monopotassium salt	Precursor	DSL
68555-74-8	1-Pentanesulfonamide, 1,1,2,2,3,3,4,4,5,5,5-undecafluoro-N-(2-hydroxyethyl)-N-methyl-	Precursor	DSL
68555-81-7	1-Propanaminium, N,N,N-trimethyl-3-[[(pentadecafluoroheptyl)sulfonyl]amino]-, chloride	Precursor	DSL
68568-54-7	Benzoic acid, 2,3,4,5-tetrachloro-6-[[[3-[[(nonafluorobutyl)sulfonyl]oxy]phenyl]amino]carbonyl]-, monopotassium salt	Precursor	DSL
68815-72-5	Benzoic acid, 2,3,4,5-tetrachloro-6-[[[3-[[(tridecafluorohexyl)sulfonyl]oxy]phenyl]amino]carbonyl]-, monopotassium salt	Precursor	DSL
68891-97-4[c]	Diaquatetrachloro[μ-[N-ethyl-N-[(pentadecafluoroheptyl)sulfonyl]glycinato-O1:O1']]-μ-hydroxybis(propan-2-ol)chromium	Precursor	DSL
68891-98-5[c]	Chromium, diaquatetrachloro[μ-[N-ethyl-N-[(tridecafluorohexyl)sulfonyl]glycinato-O1:O1']]-μ-hydroxybis(2-propanol)di-	Precursor	DSL
68891-99-6[c]	Chromium, diaquatetrachloro[μ-[N-ethyl-N-[(undecafluoropentyl)sulfonyl]glycinato-O1:O1']]-μ-hydroxybis(2-propanol)di-	Precursor	DSL
68900-97-0[c]	Chromium, diaquatetrachloro[μ-[N-ethyl-N-[(nonafluorobutyl)sulfonyl]glycinato-O1:O1']]-μ-hydroxybis(2-propanol)di-	Precursor	DSL
68957-55-1	1-Propanaminium, N,N,N-trimethyl-3-[[(undecafluoropentyl)sulfonyl]amino]-, chloride	Precursor	DSL
68957-57-3	1-Propanaminium, N,N,N-trimethyl-3-[[(undecafluoropentyl)sulfonyl]amino]-, iodide	Precursor	DSL
68957-58-4	1-Propanaminium, N,N,N-trimethyl-3-[[(tridecafluorohexyl)sulfonyl]amino]-, iodide	Precursor	DSL
68957-59-5	1-Butanesulfonamide, N-[3-(dimethylamino)propyl]-1,1,2,2,3,3,4,4,4-nonafluoro-, monohydrochloride	Precursor	DSL
68957-60-8	1-Pentanesulfonamide, N-[3-(dimethylamino)propyl]-1,1,2,2,3,3,4,4,5,5,5-undecafluoro-, monohydrochloride	Precursor	DSL

CAS RN[a]	Chemical name	Common name or acronym	Inventory[b]
68957-61-9	1-Hexanesulfonamide, N-[3-(dimethylamino)propyl]-1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro-, monohydrochloride	Precursor	DSL
68958-60-1	Poly(oxy-1,2-ethanediyl), a-[2-[ethyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl]-w -methoxy-	Precursor	DSL
70225-16-0	1-Hexanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro-, compd. with 2,2 -iminobis[ethanol] (1:1)	Precursor	DSL
70225-17-1	1-Pentanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,5-undecafluoro-,compd. with 2,2 -iminobis[ethanol] (1:1)	Precursor	DSL
70225-18-2	1-Butanesulfonic acid, 1,1,2,2,3,3,4,4,4-nonafluoro-, compd. with 2,2 -iminobis[ethanol] (1:1)	Precursor	DSL
67940-02-7	1-Heptanesulfonamide, N-[3-(dimethylamino)propyl]-1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-, monohydrochloride	Precursor	DSL
68259-14-3	1-Heptanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-N-methyl-	Precursor	DSL
68259-15-4	1-Hexanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro-N-methyl-	Precursor	DSL
68298-13-5	1-Pentanesulfonamide, 1,1,2,2,3,3,4,4,5,5,5-undecafluoro-N-methyl-	Precursor	DSL
68555-72-6	1-Pentanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,5-undecafluoro-N-(2-hydroxyethyl)-	Precursor	DSL
68555-73-7	1-Heptanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-N-(2-hydroxyethyl)-	Precursor	DSL
68555-75-9	1-Hexanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro-N-(2-hydroxyethyl)-N-methyl-	Precursor	DSL
68555-76-0	1-Heptanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-N-(2-hydroxyethyl)-N-methyl-	Precursor	DSL
68957-62-0	1-Heptanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-	Precursor	DSL
70225-15-9	1-Heptanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-, compd. with 2,2 -iminobis[ethanol] (1:1)	Precursor	DSL

CAS RN[a]	Chemical name	Common name or acronym	Inventory[b]
34455-03-3	1-Hexanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro-N-(2-hydroxyethyl)-	Precursor	DSL
67584-55-8	2-Propenoic acid, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl ester	Precursor	NDSL
No CAS Identified	Poly(Butyl methacrylate/ heptyl methacrylate/ 2-(3,3,4,4,5,5,6,6,7,7,8,8,8-tridecafluoro -n -methyl-1-octaneosulfonamido) ethyl acrylate	Precursor	NA
27619-97-2	1-Octanesulfonic acid, 3,3,4,4,5,5,6,6,7,7,8,8,8-tridecafluoro-	THPFOS (precursor)	NDSL
73772-32-4	1-Propanesulfonic acid, 3-[[3-(dimethylamino)propyl][(tridecafluorohexyl)sulfonyl]amino]-2-hydroxy-, monosodium salt	Precursor	NDSL
81190-38-7	1-Propanaminium, N-(2-hydroxyethyl)-3-[(2-hydroxy-3-sulfopropyl)[(tridecafluorohexyl)sulfonyl]amino]-N,N-dimethyl-, hydroxide, monosodium salt	Precursor	NDSL
59587-38-1	Potassium 3,3,4,4,5,5,6,6,7,7,8,8,8-tridecafluorooctanesulphonate	Precursor	TSCA
133875-90-8	1-Propanium, N-(carboxymethyl)-N,N-dimethyl-SB=3-[[(3,3,4,4,5,5...n,n,n,-polyfluoroalkyld)sulfonyl]amino}-, NM=hydroxide, inner salt	Precursor	TSCA

Abbreviations: DSL, Domestic Substances List; NDSL, Non-Domestic Substances List; TSCA, Toxic Substance Control Act (United States); NA, not available

[a] CAS RN idenitified via DSL search engine and/or US EPA Chemistry Dashboard

[b] Substances not appearing on the DSL are not used commercially in Canada above trigger quantities specified in the *New Substances Notification Regulations (Chemicals and Polymers)*. Substances listed on the NDSL are subject to notification and requirements set out in the *New Substances Notification Regulations (Chemicals and Polymers)* but with reduced information requirements. Substances on the NDSL are those found on the *US Toxic Substances Control Act* (TSCA) inventory.

[c] Also within the scope of Canada's Priority Substance List 1 assessment of "Chromium and its compounds".

Appendix C. Non-exhaustive list of LC-PFSAs and their salts

Table C-1. Non-exhaustive list of LC-PFSAs and their salts

CAS RN[a]	Chemical Name	Common Name or Acronym	Inventory[b]
68259-12-1	Perfluorononane sulfonic acid	PFNS	Not on the DSL or NDSL
335-77-3 or 2806-15-7	Perfluorodecane sulfonic acid	PFDS	Not on the DSL or NDSL
749786-16-1	Perfluoroundecane sulfonic acid	PFUnDS	Not on the DSL or NDSL
79780-39-5	Perfluorododecane sulfonic acid	PFDoDS	Not on the DSL or NDSL
No CAS identified	Perfluorotridecane sulfonic acid	PFTrS	Not on the DSL or NDSL
No CAS Identified	Pefluorotetradecane sulfonic acid	PFTeDS	Not on the DSL or NDSL
No CAS Identified	Perfluoropentadecane sulfonic acid	PFPeDS	Not on the DSL or NDSL
No CAS Identified	Perfluorohexadecane sulfonic acid	PFHxDS	Not on the DSL or NDSL
No CAS Identified	Perfluoroheptadecane sulfonic acid	PFHpDS	Not on the DSL or NDSL
No CAS Identified	Perfluorooctadecane sulfonic acid	PFODS	Not on the DSL or NDSL
No CAS Identified	Perfluorononadecane sulfonic acid	PFNDS	Not on the DSL or NDSL
No CAS identified	Perfluoroicosane sulfonic acid	PFICOS	Not on the DSL or NDSL
67906-42-7	1-Decanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heneicosafluoro-, ammonium salt	PFDS ammonium salt	DSL
17202-41-4	1-Nonanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,9,9,9-nonadecafluoro-, ammonium salt	PFNS ammonium salt	DSL

Abbreviations: DSL, Domestic Substances List; NDSL, Non-Domestic Substances List.
[a] CAS RN idenitified via DSL search engine and/or US EPA Chemistry Dashboard.
[b] Substances not appearing on the DSL are not used commercially in Canada above trigger quantities specified in the *New Substances Notification Regulations (Chemicals and Polymers)*. Substances listed on the NDSL are subject to notification and requirements set out in the *New Substances Notification Regulations (Chemicals and Polymers)* but with reduced information requirements. Substances on the NDSL are those that are on the *US Toxic Substances Control Act* (TSCA) inventory.

Appendix D. Empirical evidence of precursors for SC-PFCAs and SC-PFSAs

Note: Most available empirical data identifying precursors are limited to the short-chain PFCAs. There is one empirical study identifying a precursor for the short-chain PFSAs. There is no empirical evidence identifying precursors for the long-chain PFSAs. Empirical evidence for precursors includes that for fluorotelomer olefins (FTO) and N-alkyl perfluoroalkylsulfonamidoethanols (PFSE). The estimated "atmospheric lifetimes" of FTOHs (12 to 20 days), FTOs (8 days), and a major degradation product of PFSEs (20 to 50 days) permit their transport to remote regions. Photoreactor studies have indicated that PFSEs may contribute to the observed environmental burden of both PFSAs and PFCAs substances (Waterland and Dobbs 2007).

Table D-1. Empirical evidence of precursors for SC-PFCAs and SC-PFSAs

Precursor	Pathway and/or final degradation product	Media/organism	Reference
N-methyl perfluorobutane sulfonamidoethanol (NMeFBSE)	PFBS	Gas phase	D'Eon et al. 2006
6:2 fluorotelomer alcohol (6:2 FTOH)	PFBA PFHxA	*Pseudomonas oleovorans*	Kim et al. 2012
6:2 fluorotelomer alcohol (6:2 FTOH)	PFHxA	*Pseudomonas butanovora*	Kim et al. 2012
6:2 fluorotelomer alcohol (6:2 FTOH)	PFBA	*Pseudomonas fluorescens DSM 8341* in presence of formate	Kim et al. 2014
6:2 fluorotelomer alcohol (6:2 FTOH)	PFPeA	*Pseudomonas fluorescens DSM 8341*	Kim et al. 2014
6:2 fluorotelomer alcohol (6:2 FTOH)	PFPeA	*Pseudomonas oleovorans*	Kim et al. 2014
8:2 fluorotelomer alcohol (8:2 FTOH)	PFHxA	*Pseudomonas oleovorans; Pseudomonas butanovora*	Kim et al. 2012
8:2 fluorotelomer alcohol (8:2 FTOH)	8:2 FTOH → 7:3 FTCA→PFHpA (C7 PFCA)	Juvenile rainbow trout	Butt et al. 2010a
8:2 fluorotelomer alcohol (8:2 FTOH)	PFPeA PFHxA PFHpA	Male juvenile rainbow trout	Nabb et al. 2007
8:2 fluorotelomer acrylate (8:2 FTAc)	7:3 FTCA→PFHpA (C7 PFCA)	Juvenile rainbow trout	Butt et al. 2010b
6:2 fluorotelomer alcohol (6:2 FTOH)	PFHxA	Mixed aerobic bacterial culture from activated sludge (industrial facility)	Liu et al. 2010a
6:2 fluorotelomer alcohol (6:2 FTOH)	PFHxA	Anaerobic digester sludge from wastewater	Zhang et al. 2013

Precursor	Pathway and/or final degradation product	Media/organism	Reference
		treatment plant (Delaware, US)	
6:2 fluorotelomer sulfonate (6:2 FTS)	PFBA PFPeA PFHxA	Activated sludge from wastewater treatment plants (Pennysylvania, Maryland, Delaware, US)	Wang et al. 2011b
8:2 fluorotelomer alcohol (8:2 FTOH)	PFHxA	Wastewater treatment plant mixed bacterial culture and activated sludge (industrial facility)	Wang et al. 2005
8:2 fluorotelomer alcohol (8:2 FTOH)	PFHxA	Anaerobic digester sludge from wastewater treatment plant (Delaware, US)	Zhang et al. 2013
1H,1H,2H,2H,8H,8H-perfluorododecanol (DTFA)	PFBA PFPeA	Mixed bacterial culture from activated sludge (wastewater treatment facility for plant that produced fluorinated compounds)	Arakaki et al. 2010
7:3 polyfluorinated carboxylic acid (7:3 acid)	PFHpA	Wastewater treatment activated sludge (Pennyslvania, US)	Wang et al. 2012
5:3 polyfluorinated carboxylic acid (5:3 acid)	PFBA PFPeA	Wastewater treatment activated sludge (Pennyslyvania, US)	Wang et al. 2012
Disubstituted polyfluoroalkyl phosphate (6:2 diPAP)	→6:2 monoPAP →5:3 FTCA →PFPeA →6:2 monoPAP → 6:2 FTCA →PFHxA →6:2 monoPAP → 6:2 FTOH → 6:2 FTCA →PFHpA	Mixed liquor (mixture of raw wastewater and sewage sludge, Toronto, Canada)	Lee et al. 2010
Polyethoxylated 2-perfluoroalkylethanols (fluorotelomer ethoxylates)	PFHxA	Wastewater treatment plant effluent (Hesse, Germany)	Frömel and Knepper 2010
N-methyl perfluorobutane sulfonamidoethanol (NMeFBSE)	PFBA	Gas phase	D'Eon et al. 2006

Precursor	Pathway and/or final degradation product	Media/organism	Reference
4:2 fluorotelomer alcohol (4:2 FTOH)	PFBA PFPeA	Gas phase	Ellis et al. 2004
4:2 fluorotelomer iodide (4:2 FTI)	PFBA PFPeA	Gas phase	Young et al. 2008
6:2 fluorotelomer alcohol (6:2 FTOH)	PFHxA PFHpA	Gas phase	Ellis et al. 2004
8-carbon polyfluorinated amides (PFAMs)	PFBA	Atmospheric oxidation	Jackson et al. 2013
N-ethyl perfluorobutanesulfonamide (NEtFBSA)	PFBA PFPeA	Gas phase	Martin et al. 2006
8:2 fluorotelomer alcohol (8:2 FTOH)	PFHxA PFHpA	Soil	Liu et al. 2007
8:2 fluorotelomer alcohol (8:2 FTOH)	PFHxA	Aerobic soil (Sassafras, Manning, Chalmers)	Wang et al. 2009
8:2 fluorotelomer stearate monoester (8:2 FTS)	PFHxA PFHpA	Forest silt loam (West Lafayette, Indiana, US)	Dasu et al. 2013
6:2 fluorotelomer alcohol (6:2 FTOH)	PFBA PFPeA PFHxA	Aerobic Sassafras soil	Liu et al. 2010a Wang et al. 2010
6:2 fluorotelomer alcohol (6:2 FTOH)	PFPeA PFHxA PFHpA	Oxidation at surface of Mauritian sand and Icelandic ash	Styler et al. 2013
6:2 fluorotelomer alcohol (6:2 FTOH)	PFPeA PFHxA	Sassafras soil	Liu et al. 2010b
6:2 fluorotelomer alcohol (6:2 FTOH)	PFBA PFPeA PFHxA	Aerobic river sediment (Brandywine Creek, Pennsylvania, US)	Zhao et al. 2013b
Acrylate-linked fluorotelomer polymer	PFHxA PFHpA	Soil microcosm	Washington et al. 2009
8:2 fluorotelomer carboxylic acid (8:2 FTCA)	PFHpA	Sediment-water microcosm	Myers and Mabury 2010
10:2 fluorotelomer unsaturated carboxylic acid (10:2 FTUCA)	PFHpA	Sediment-water microcosm	Myers and Mabury 2010
Perfluorooct-1- and -2-enes and 4-trifluoromethyl-1,1,1,2,3,4,5,5,5-nonafluoropent-2-ene	PFHxA PFHpA	Ozonolysis	Odinokov et al. 1997

Appendix E. Available empirical physical-chemical properties for SC-PFCAs

Table E-1. Available empirical physical-chemical properties for SC-PFCAs

CAS RN (Chemical Name)	Acronym	Value	Reference
Log K_{ow}			
375-22-4 (Perfluorobutanoic acid)	PFBA	-0.52 (neutral form†)	Jing et al. 2009
2706-90-3 (Perfluoropentanoic acid)	PFPeA	0.09 (neutral form†)	Jing et al. 2009
307-24-4 (Perfluorohexanoic acid)	PFHxA	0.70 (neutral form†)	Jing et al. 2009
375-85-9 (Perfluoroheptanoic acid)	PFHpA	1.31 (neutral form†)	Jing et al. 2009
Log K_d			
375-22-4 (Perfluorobutanoic acid)	PFBA	1.18	Zhang et al. 2012a
2706-90-3 (Perfluoropentanoic acid)	PFPeA	1.14	Zhang et al. 2012a
307-24-4 (Perfluorohexanoic acid)	PFHxA	1.33	Zhang et al. 2012a
375-85-9 (Perfluoroheptanoic acid)	PFHpA	1.24	Zhang et al. 2012a
Log K_{oc} (L/kg)			
375-22-4 (Perfluorobutanoic acid)	PFBA	2.62	Zhang et al. 2012a
2706-90-3 (Perfluoropentanoic acid)	PFPeA	1.70–2.11	Zhao et al. 2012
2706-90-3 (Perfluoropentanoic acid)	PFPeA	2.54	Zhang et al. 2012a
307-24-4 (Perfluorohexanoic acid)	PFHxA	2.72	Zhang et al. 2012a
375-85-9 (Perfluoroheptanoic acid)	PFHpA	1.72–2.05	Zhao et al. 2012
Vapour pressure (Pa)			
375-22-4 (Perfluorobutanoic acid)	PFBA	2.93	Bhhatarai and Gramatica 2011
375-22-4 (Perfluorobutanoic acid)	PFBA	2.63	Kim et al. 2015
375-22-4 (Perfluorobutanoic acid)	PFBA	1333	MSDS 2004
375-85-9 (Perfluoroheptanoic acid)	PFHpA	1.32	Bhhatarai and Gramatica 2011
375-85-9 (Perfluoroheptanoic acid)	PFHpA	1.88	Kim et al. 2015
Boiling point (ºC)			
375-22-4 (Perfluorobutanoic acid)	PFBA	120	Kirk-Othmer 1994
375-22-4 (Perfluorobutanoic acid)	PFBA	120	SDS 2004b
2706-90-3 (Perfluoropentanoic acid)	PFPeA	139	Kirk-Othmer 1994
375-85-9 (Perfluorohepatanoic acid)	PFHpA	89	Kirk-Othmer 1994
375-85-9 (Perfluorohepatanoic acid)	PFHpA	175	SDS 2004b
Melting point (ºC)			
375-22-4 (Perfluorobutanoic acid)	PFBA	-17.5	Kirk-Othmer 1994
Density (g/mL, 20ºC)			

CAS RN (Chemical Name)	Acronym	Value	Reference
375-22-4 (Perfluorobutanoic acid)	PFBA	1.641	Kirk-Othmer 1994; SDS 2004b
2706-90-3 (Perfluoropentanoic acid)	PFPeA	1.713	Kirk-Othmer 1994
375-85-9 (Perfluoroheptanoic acid)	PFHpA	1.792	Kirk-Othmer 1994
pKa (acidity constant)			
422-64-0 (Perfluoropropanoic acid)	PFPrA (C3)	0.62	Moroi et al. 2001
375-22-4 (Perfluorobutanoic acid)	PFBA (C4)	0.43	Moroi et al. 2001
2706-90-3 (Perfluoropentanoic acid)	PFPeA (C5)	0.74	Moroi et al. 2001

Abbreviations: pK_a, acid dissociation constant; Kow, octanol-water partition coefficient; Koc, organic carbon-water partition coefficient; Kd, equilibrium dissociation constant

† As SC-PFCAs are considered to have surface-active properties, preferentially partition to proteins, and are ionizing, estimates of log K_{ow} and/or using log K_{ow} on the basis of the neutral form can result in unreliable predictions for bioaccumulation

Appendix F. Available empirical physical-chemical properties for SC-PFSAs

Table F-1. Available empirical physical-chemical properties for SC-PFSAs

CAS RN (chemical name)	Acronym	Value	Reference
Water solubility (g/L)			
375-73-5 (Perfluorobutane sulfonic acid)	PFBS	67 g/L	Kim et al. 2015
Log K_{ow}			
375-73-5 (Perfluorobutane sulfonic acid)	PFBS	-0.30 (neutral form†)	Jing et al. 2009
Log K_{oc} (L/kg)			
375-73-5 (Perfluorobutane sulfonic acid)	PFBS	1.75–2.09	Zhao et al. 2012
355-46-4 (Perfluorohexane sulfonic acid)	PFHxS	0.74–1.70	Zhao et al. 2014
355-46-4 (Perfluorohexane sulfonic acid)	PFHxS	2.05	Guelfo and Higgins 2013
355-46-4 (Perfluorohexane sulfonic acid)	PFHxS	2.40	D'Agostino and Mabury 2017
355-46-4 (Perfluorohexane sulfonic acid)	PFHxS	1.8–2.76	Chen et al. 2018
355-46-4 (Perfluorohexane sulfonic acid)	PFHxS	2.02–2.14	Zhao et al. 2012
335-77-3/2806-15-7 (Perfluorodecane sulfonic acid)	PFDS	3.53–3.66	Higgins and Luthy 2006
Melting point (ºC)			
355-46-4 (Perfluorohexane sulfonic acid)	PFHxS	41	Kim et al. 2015
Boiling point (ºC)			
355-46-4 (Perfluorohexane sulfonic acid)	PFHxS	159–160	SDS 2004a
355-46-4 (Perfluorohexane sulfonic acid)	PFHxS	238–239	Kosswig 2000
Density (g/mL, 20ºC)			
355-46-4 (Perfluorohexane sulfonic acid)	PFHxS	1.762	Kirk-Othmer 1994

Abbreviations: pK_a, acid dissociation constant; Kow, octanol-water partition coefficient; Koc, organic carbon-water partition coefficient; Kd, equilibrium dissociation constant

† As SC-PFSAs are considered to have surface-active properties, preferentially partition to proteins, and are ionizing, estimates of log K_{ow} and/or using log K_{ow} on the basis of the neutral form can result in unreliable predictions for bioaccumulation.