Environment and Climate Change Canada | Environnement et Changement climatique Canada

Canadian Environmental Protection Act, 1999

Federal Environmental Quality Guidelines

Perfluorooctane Sulfonate (PFOS)

Environment and Climate Change Canada

Publication Date: June 2018

Table of Contents

Introduction

Federal Environmental Quality Guidelines (FEQGs) provide benchmarks for the quality of the ambient environment. They are based solely on the toxicological effects or hazard of specific substances or groups of substances. FEQGs serve three functions: first, they can be an aid to prevent pollution by providing targets for acceptable environmental quality; second, they can assist in evaluating the significance of concentrations of chemical substances currently found in the environment (e.g., monitoring of water, sediment, and biological tissue); and third, they can serve as performance measures of the success of risk management activities. The use of FEQGs is voluntary unless prescribed in permits or other regulatory tools. Thus, FEQGs apply to the ambient environment. They are not effluent limits or "never-to-be-exceeded" values but may be used to derive them. The development of FEQGs is the responsibility of the federal Minister of the Environment under the Canadian Environmental Protection Act, 1999 (CEPA). The intent is to develop FEQGs as an adjunct to the risk assessment/risk management of priority chemicals identified in the Chemicals Management Plan (CMP) or other federal initiatives. This factsheet provides the Federal Water Quality Guideline (FWQG) (Table 1 and Figure 1), the Federal Fish Tissue Guideline (FFTG) for the protection of aquatic life, the Federal Wildlife Diet Guidelines (FWiDGs) for the protection of mammalian and avian consumers of aquatic biota, and the Federal Tissue Guideline describing the acceptable contaminant levels in bird eggs (FTG-BE) for perfluorooctane sulfonate (PFOS) (Table 1).

FEQGs for water, fish tissue, wildlife diet and bird egg are similar to Canadian Council of Ministers of the Environment (CCME) guidelines in that they are benchmarks for the quality of the ambient environment and are based solely on toxicological effects data. Where data permit, FEQGs are derived following CCME methods. FEQGs differ from Canadian Environmental Quality Guidelines (CEQG) in that FEQGs are developed where there is a federal need for a guideline (e.g. to support federal risk assessment, federal risk management or monitoring activities) but where the CCME guideline(s) for the substance has not yet been developed or is not reasonably expected to be updated in the near future. CEQGs are preferred since they address a substance of national interest. CEQGs for PFOS in soil and groundwater are currently being developed by the CCME.

Substance identity

Perfluorooctane sulfonate (PFOS) belongs to a larger group of fluorochemicals called perfluorinated alkyl compounds (Kissa 1994). This classification indicates that the main carbon chain of the compound is completely saturated with fluorine, involving highly stable C-F bonds. While PFOS can exist in its anionic form ($C_8F_{17}SO_3^-$), it also exists as an acid (CAS No. 1763-23-1), potassium salt (CAS No. 2795-39-3), ammonium salt (CAS No. 29081-56-9), diethanolamine salt (CAS No. 70225-14-8) and lithium salt (CAS No. 29457-72-5). PFOS is not found naturally in the environment, however, it has been manufactured since the 1950s (Lehmler 2005). Based on the Screening Assessment Report (SAR), Environment Canada (EC) (EC 2006) concluded that PFOS, its salts and its precursors (compounds containing the following groups: $C_8F1_7SO_2$, $C_8F1_7SO_3$ or $C_8F1_7SO_2N$) were entering the environment in a quantity that has, or may have, an immediate or long-term harmful effect on the environment and biological diversity. PFOS and its salts and its precursors meet the definition of toxic and PFOS and its salts (but not precursors) are also persistent according to the Persistence and Bioaccumulation Regulations

(SOR/2000-107) under CEPA and were added to the Stockholm Convention on Persistent Organic Pollutants Annex B (restricted) in 2009. PFOS is also considered bioaccumulative based on its preferential partitioning to lipid, blood and kidney in terrestrial and marine mammals. Moreover, PFOS and its salts were added to the Virtual Elimination List under subsection 65(2) of CEPA with the promulgation of the Perfluorooctane Sulfonate Virtual Elimination Act, SOR/2009-15 (Government of Canada 2009).

Table 1. Federal Environmental Quality Guidelines for Perfluorooctane Sulfonate (PFOS) for Surface Water, Fish Tissue, Wildlife Diet, and Bird Egg.

Water (µg/L)	Fish Tissue (mg/kg ww)*	Wildlife Diet (µg/kg ww food)**		Bird Egg (µg/g ww)
		Mammalian	Avian	
6.8	9.4	4.6	8.2	1.9

*ww = wet weight

**The wildlife diet guidelines are intended to protect either mammalian or avian species that consume aquatic biota. It is the concentration of PFOS in the aquatic biota food item, expressed on whole body, wet weight basis that could be eaten by terrestrial or semi-aquatic mammalian or avian wildlife.

Uses

Between 1997 and 2000, Canada imported approximately 600 tonnes of perfluorinated alkyl compounds. PFOS and its precursors, (the precursors contribute to overall loading in the environment), accounted for 43 % of these compounds, while PFOS alone accounted for < 2 % (EC 2001). PFOS and PFOS-related compounds are used as water, oil, soil and grease repellents. Their use can be categorized into three main categories: surface treatment of apparel and home furnishings, paper protection and performance chemicals. In the past, PFOS surface treatments were used in industrial manufacturing, in such settings as textile mills, leather tanneries, fibre production lines and carpet manufacturing plants (OECD 2002). Food and non-food industries used PFOS and PFOS-related chemicals in paper applications including food containers, food wrappers, folding cartons and masking papers (OECD 2002, Dallaire et al. 2009; Château-Degat et al. 2010; Clarke et al. 2010). Specifically, the potassium salt of PFOS, used in the manufacture of aqueous film forming foams (AFFFs), was the most significant perfluorinated alkyl compound imported into Canada (EC 2013a). As performance chemicals, PFOS-related chemicals were used in a variety of ways, for example, mining and oil well surfactants, photographic film, hydraulic fuel additives, electronics chemicals, denture cleaners and shampoos. Salts of PFOS were also used specifically as acid mist suppressants for metal plating and electronic etching baths, floor polishes, alkaline cleaners, insecticide in bait stations and as fire-fighting foams (3M Company 2000). By 2002, the primary producer in the United States completed phase out of the

production of PFOS chemicals and products containing PFOS. However, China began large-scale PFOS production in 2003 (Butt et al., 2010); in 2006 China produced more than 200 tons of the precursor, perfluorooctanesulfonyl fluoride (POSF) (Ministry of Environmental Protection of China 2008).

The manufacture, importation and use of PFOS and PFOS related compounds in Canada is regulated under the Perfluorooctane Sulfonate and its Salts and Certain Other Compounds Regulation, SOR/2008-178 (Government of Canada 2008) pursuant to the Canadian Environmental Protection Act (CEPA). This regulation prohibits the manufacture, import, sale, offer for sale and use of PFOS or of products containing PFOS, unless incidentally present, with certain exemptions (e.g., AFFF, aviation hydraulic fluids under certain conditions, and some products used in photographic or photolithographic processes) (Government of Canada 2008).

Measured concentrations

Concentrations of PFOS have been measured in various environmental media including water, fish, wildlife, sediment, air and soil. Early studies on PFOS detected concentrations in the environment ranging from a few pg/m^3 in air (Kim and Kannan 2007) to high µg/kg levels in wildlife (Giesy and Kannan 2001, 2002; Kannan et al. 2001a,b, 2002a,b, 2005; Tao et al. 2006). PFOS is the most commonly found perfluorinated compound (PFC) in the tissues of wildlife, accumulating primarily in the blood and liver (Giesy and Kannan 2001). Kannan et al. (2006) reported that PFC concentrations in polar bears were the highest in any species to date. Maximum levels of PFOS in liver of Canadian Arctic biota have been reported for mink (20 µg/kg), seal (37 µg/kg), brook trout (50 µg/kg), fox (1400 µg/kg) (Martin et al. 2004) and polar bear (3770 µg/kg) (Smithwick et al. 2005).

Most recently, the CMP monitoring program reported PFOS concentrations from locations across Canada over the period from 2006-2011 in various media (Environment and Climate Change Canada (ECCC) 2016, Government of Canada 2016, EC 2013b). Between 2007 and 2010, Environment Canada collected water samples (n = 569) from 11 drainage regions across Canada (Pacific Coast, Fraser-Lower Mainland, Okanagan-Similkameen, Yukon, Assiniboine-Red, Great Lakes, Ottawa, St. Lawrence, St. John-St. Croix, Maritime Coastal and Newfoundland-Labrador). All surface water samples had PFOS concentrations at least 200-fold lower than the FEQG for water (6.8 µg/L). The maximum surface water concentration reported was 10 ng/L (0.01 µg/L).

In the 2011-2014 CMP monitoring period, PFOS concentrations were below the FEQG for fish health in all 11 drainage regions sampled (Government of Canada 2016). Importantly however, in some instances, PFOS levels in fish exceeded the FEQG for the protection of mammals and birds that eat the fish, suggesting that this compound could represent a potential risk to wildlife predators in seven of 11 drainage regions (Columbia, Yukon, Assiniboine-Red, Winnipeg, Great Lakes, St. Lawrence and Maritime Coastal). In the analysis of concentrations of PFOS in lake trout from Lake Ontario from 1979-2014, geometric mean lake trout tissue concentrations rose from 1979-2002, peaking at approximately 80 to 110 µg/kg wet weight in 2002 and then appear to be decreasing to approximately 40-60 µg/kg by 2013-2014 (ECCC 2016).

Similarly, from 2006 to 2010, Environment Canada collected top predator fish (lake trout and walleye) (n = 441) from 21 sites in 13 drainage regions and analyzed PFOS in their tissue (Government of Canada 2016, EC 2013b). PFOS levels varied considerably with the highest concentrations in urban areas compared with more remote lakes. The highest concentrations in lake trout were from Lake Erie (geometric mean = 90 µg/kg ww) and Lake Ontario (geometric mean = 62 µg/kg ww) and were mostly low (< 3 µg/kg ww) in fish from water bodies located in northern Canada, Pacific and Atlantic regions and Lake Superior. Notably, the analysis found that the concentration of PFOS was below the FEQG for fish health (i.e. below 8.3 mg/kg ww = 8300 µg/kg ww) in all sampled drainage regions (Pacific Coast, Okanagan-Similkameen, Columbia, Yukon, Peace-Athabasca, Lower Mackenzie, Assiniboine-Red, Winnipeg, Lower Saskatchewan-Nelson, Churchill, Great Lakes, St. Lawrence and Maritime Coastal). PFOS levels in fish exceeded the FEQG for the protection on mammals and birds that eat fish, suggesting that this compound could represent a potential risk to wildlife predators. Over this period, eight of the 13 sampled drainage regions (Okanagan-Similkameen, Columbia, Assiniboine-Red, Winnipeg, Lower Saskatchewan, Great Lakes, St. Lawrence and Maritime Coastal) had some concentrations of PFOS that exceeded the FEQG for wildlife (i.e. 4.6 µg/kg ww food for mammals, and 8.2 µg/kg ww food for birds).

CMP also monitored PFOS in gull and starling eggs from 2008-2011 to characterize PFOS in aquatic and terrestrial birds, respectively (EC 2013b). In individual gull eggs, PFOS concentrations were relatively elevated in the Great Lakes and St. Lawrence River with levels > 0.260 µg/g ww; concentrations were lower (0.007 to 0.115 µg/g ww) in non-urban areas as well as in marine colonies on both the Atlantic and Pacific coasts. Pooled samples collected between 2009 and 2011 similarly showed the highest concentration in gull eggs were from Lake Erie (0.676 µg/g ww). Starlings are terrestrial birds and feed lower in the food web than gulls; and while the highest concentrations of PFOS in starling eggs were those located at the Brantford, Ontario landfill (0.702 µg/g ww) which is located in a highly urbanized region in southern Ontario, concentrations in urban sites and landfill sites generally overlapped. Concentrations of PFOS in starling eggs at urban sites were: Indus (AB) (0.199 µg/g ww), Delta (BC) (0.075 µg/g ww), Hamilton (ON) (0.041 µg/g ww) compared with starlings eggs at landfill sites located in Halton (ON) (0.029 µg/g ww), Stoney Creek(ON) (0.028 µg/g ww), Otter Lake (NS) (0.018 µg/g ww), and Langley (BC) (0.0056 µg/g ww). In all cases the levels in eggs of terrestrial and aquatic-feeding birds were below the FEQG for bird egg (1.9 µg/g ww).

FEQGs for PFOS do not exist for sediment. In 2008, 65 surface sediment samples were collected at 18 sites across Canada (EC 2013b). The highest PFOS concentration in sediment was found in Lake Ontario (0.010 µg/g dry-weight). Values were also reported to range from 0.00016 to 0.0024 µg/g dry weight for sediments in Lake Erie (ON), Lake Huron (ON), Lake Superior (ON), Hamilton harbour (ON), Toronto harbour (ON), near Thunder Bay (ON), Lake Saint Pierre (QC), Nappan River (NB), Kejimikujik Lake (NS), Little Sackville (NS) and Osoyoos Lake (BC). PFOS was non-detectable at the other sites monitored. Average PFOS concentrations in suspended sediments from the Niagara River at Niagara-on-the-Lake (ON), collected annually over a 22 year period (1980-2002) increased from < 0.0004 µg/g (<400 pg/g) in the early 1980s to more than 0.001 µg/g (1000 pg/g) in 2002 (Lucaciu et al. 2005).

FEQGs do not exist for PFOS in air. However, monitoring PFOS in air across Canada provides information on PFOS levels within the country as well as quantities entering Canada from international sources (EC 2013b). Air measurements have been obtained using two methods: high volume air sampling and passive air sampling. High volume air samplers measure a larger volume of air and are better for detecting the low PFOS concentrations often found in the environment. However, passive air samplers can be advantageous under many circumstances because of their simplicity, ease of transport to remote sites, and because they do not require a power source. Sampling using high-volume samples was conducted at three locations in Canada in 2009 (EC 2013b), and it was observed that PFOS concentrations in air (geometric mean) were more than three times higher in Toronto (1.5 pg/m^3) compared with Lake Superior (0.43 pg/m^3). PFOS was below the detection limit of 0.2 pg/m^3 at the Canadian High Arctic station of Alert, NU; however its precursors were detected up to several pg/m^3.

Sampling using passive samplers was conducted at eight locations across Canada over a three-month period in 2009 (EC 2013b). PFOS concentrations were detected in Toronto, ON (8 pg/m^3), an agricultural site in Saskatchewan (5 pg/m^3), Whistler, BC (4 pg/m^3), and Alert, NU (2 pg/m^3). One site in northern Ontario had elevated PFOS concentrations of 18 pg/m^3; however these data points were only based on one sample. PFOS was not detected at the other Canadian sites. The PFOS levels measured in Canada using passive samplers were substantially lower than in Paris, France (150 pg/m^3), but comparable to Sydney, Florida (3.4 pg/m^3), Tudor Hill, Bermuda (6.1 pg/m^3), Malin Head, Ireland (3.3 pg/m^3), and Hilo, Hawaii (6.6 pg/m^3).

In general, the monitoring results showed that PFOS air concentrations in urban locations (e.g., Toronto) were on the same order of magnitude as more remote sites (e.g., Lake Superior), demonstrating the widespread distribution of PFOS in the Canadian atmosphere. PFOS precursors measured in the air of Toronto identified average concentrations of N-MeFOSE alcohol of 101 pg/m^3 and N-EtFOSE alcohol (see list of abbreviations below) of 205 pg/m^3 (Martin et al. 2002). Boulanger et al. (2004) reported mean surface water (4 m depth) concentrations of 31 (sd = 6.9) ng/L for Lake Erie and 54 (sd = 18) ng/L for Lake Ontario.

Fate, behaviour and partitioning

Understanding of the environmental fate of PFOS continues to improve with advances in both collection of experimental data and predictive approaches, although the compounds' physical/chemical properties, notably its hydrophobic/oleophobic nature, continue to make this challenging (Rayne and Forest 2009a, Jing et al. 2009). Due to the high surface-active (surfactant) properties octanol/water (K_{ow}) partition coefficient cannot be measured simply (OECD 2002), although an indirect measure using ion-transfer cyclic voltammetry has determined a log P of 2.45 indicating lipophilicity (Jing et al. 2009). Also, sediment organic carbon – water partition coefficients (K_{oc}) for perfluorinated compounds (PFCs) (Rayne and Forest 2009b) indicate that although longer unbranched sulfonates and carboxylates tended to partition to organic matter, there was high variability in partitioning on a congener- and isomer-specific basis. PFOS is persistent in the environment and the strength of the carbon-fluorine bond renders it resistant to hydrolysis, photolysis and biodegradation. It is therefore considered to be an environmentally stable compound (EC 2006). PFOS appears to be the end stage metabolite or

ultimate degradation product of several fluorochemicals produced using perfluorooctane sulphonyl fluoride (Giesy and Kannan 2002). Thus, PFOS precursors contribute to the overall loading of PFOS in the environment.

PFOS is expected to behave differently than traditional hydrophobic pollutants, as it contains both hydrophobic and hydrophilic functional groups. The potassium salt of PFOS has a solubility of approximately 680 mg/L in pure water, 370 mg/L in fresh water, and 12.4 mg/L in sea water (OECD 2002). As a strong acid, PFOS will completely dissociate to ionic forms in neutral water (Jones et al. 2003). In addition, PFOS is not expected to volatilize based on its vapour pressure and predicted Henry's Law constant (OECD 2002). A number of studies report significant sorption of PFOS to sediments (Higgins and Luthy 2006, 2007; Nakata et al. 2006; Chen et al. 2012; Ahrens et al 2010, 2011; Kwadijk et al. 2010; Labadie and Chevreuil 2011) while others do not (Hansen et al. 2002; Senthilkumar et al. 2007). It has, therefore, been suggested that the sorption and desorption behaviour of PFOS may be greatly affected by different sorption conditions, such as the physiochemical characteristics of the sorbent and the environmental conditions of the aqueous system (Liu et al. 2001). You et al. (2010) inferred that PFOS would be largely removed from the water column with an increase in salinity or pH, and get trapped in the sediments with little bioavailability. In addition, these researchers found correlations between distribution coefficients (K_d) and the fraction of organic carbon, demonstrating that despite its surfactant properties hydrophobic partitioning is important to the sorption of PFOS to soil and sediments.

Bioconcentration factors (BCF – water exposures only) for PFOS ranged from 31.6 to 3614 L/kg for whole body measurements, with an average value of 779 L/kg. The highest value came from a laboratory study performed on bluegill sunfish (Lepomis macrochirus) (Drottar et al. 2002). BCFs ranged from 484 to 5400 L/kg in specific tissues, with an average value of 2660 L/kg. The maximum value of 5400 L/kg was calculated for rainbow trout (Oncorhynchus mykiss) liver (Martin et al. 2003). Bioaccumulation factors (BAF water and dietary exposure, or field measured) for whole body ranged from 113 to 11 150 L/kg and the maximum value of 11 150 L/kg was observed in brown mussel (Perna perna) (Quinete et al. 2009). Tissue-specific BAFs (liver) ranged from 460 to 275 000 L/kg; the highest value was for livers of tucuxi dolphin (Sotalia guianensis) (Quinete et al. 2009). Based on data presented in the SAR (EC 2006), a geometric mean BAF value of 1614 L/kg was derived for aquatic organisms. The value was based on data for six fish and four invertebrate species. For freshwater organisms, whole body biomagnification factors (BMF) ranged from 0.17 to 7.5 with the mean value of 2.6. The maximum BMF of 7.5 was observed by Houde et al. (2008) and represents the trophic transfer from an invertebrate (Diporeia hoyi) to the forage fish, slimy sculpin (Cottus cognatus). EC (2006) therefore concluded that PFOS is bioaccumulative based on its preferential partitioning to lipid, blood and kidney in terrestrial and marine mammals.

Mode of action

While the modes of action of PFOS are not entirely understood, they appear to be diverse. Suggested modes of action include activation of the nuclear peroxisome proliferator activated receptor-alpha (PPAR-α) (Berthiaume and Wallace 2002; Hickey et al. 2009, Rosen et al. 2010). These receptors alter gene expression related to a broad spectrum of action but include fatty acid

metabolism and transport, cholesterol transport (Feige et al. 2006), glucose metabolism, inflammation response and development. In contrast, toxic effects have also been demonstrated that do not involve PPAR mechanisms (O'Brien et al. 2009). PFOS is believed to interfere at the mitochondrial level through the uncoupling of oxidative phosphorylation. This uncoupling causes a reduction in the production of ATP, thereby reducing energy stores. Other modes of action that have been hypothesized include inflammation-independent leakage of liver cell membranes in fish, which leads to cell necrosis (Hoff et al. 2003); an interference with the homeostasis of DNA metabolism (Hoff et al. 2003); inhibition of glycogen synthesis; increased glycogen breakdown (Hagenaars et al. 2008); and, the inhibition of intercellular communication processes involving gap junctions (Hu et al. 2002). Altered neurochemistry from a single dose of PFOS to neonatal mice resulted in developmental neurotoxicity (Johansson et al. 2008). Finally, endocrine modulation effects on the estrogen receptor and thyroid receptor occurred in zebrafish (Du et al. 2013).

Aquatic toxicity

Aquatic toxicity values for chronic (long-term) exposures to PFOS (87-99% active ingredient) ranged from 10 to 53000 µg/L, with sensitivities overlapping among taxa (Table 2). At 10 µg/L there were no effects on damselfly survival during a 320-d exposure whereas medaka showed reduced growth in a 14-d exposure (Table 2). Plant data were the most diverse. The most sensitive plant species was watermilfoil (Myriophyllum sibiricum) with a 42-d EC_{10} for reduced growth of 100 µg/L. Data were found for two amphibians; there were no effects on survival of African clawed frog (Xenopus laevis) at 100 µg/L whereas the 60-d maximum acceptable toxicant concentration for development in leopard frog (Rana pipiens) was 1732 µg/L. The 21-day LC_{10} for survival of early life stage of rainbow trout (Oncorhynchus mykiss) was 470 µg/L (EC 2014).

Wildlife toxicity

PFOS is hepatotoxic and the effects include increased liver weights, observed in mallards, northern bobwhite and laboratory rats (Gallagher et al. 2003a; Luebker et al. 2005; York 1999), as well as hepatocellular adenomas (EC 2006) and peroxisome proliferation (Luebker et al. 2005). McNabb et al. (2005) studied the effects of PFOS on the thyroid function in northern bobwhite. After seven days of exposure to a dose of 5 mg/kg body weight (bw), plasma thyroid hormones decreased, indicating organism-level hypothyroidism. When cynomolgus monkeys were administered PFOS (0.03, 0.15, 0.75 mg/kg bw·day for 26 weeks), they had reduced high density lipoprotein and cholesterol (Thomford 2000). Other previously-observed toxic effects of PFOS have included a reduction in testicular size and altered spermatogenesis in both quails and mallards, reduced survival of quail chicks exposed only in ovo (Gallagher et al. 2003a,b; Newsted et al. 2007), and a reduced dam body mass in rats (York 1999). Thresholds for effects are similar in mammals and birds (Newsted et al. 2007).

Federal Environmental Quality Guidelines Derivation

Federal Water Quality Guidelines

The Federal Water Quality Guideline (FWQG) developed here identifies a benchmark for aquatic ecosystems that is intended to protect all forms of aquatic life for indefinite exposure periods. A species sensitivity distribution (SSD) curve was developed using the long-term toxicity data for two amphibian, five fish, five invertebrate and eight plant species (Figure 1 and Table 2). Each species for which appropriate toxicity data were available was ranked according to sensitivity, and its position on the SSD was determined. This guideline is only applicable to freshwater aquatic life, first, because there were no marine data, and second, because PFOS is expected to behave differently due to reduced solubility in marine water, as discussed. Fish tissue guidelines or wildlife dietary guidelines (see below) should be used in conjunction with water quality guidelines where a substance may bioaccumulate in higher trophic levels.

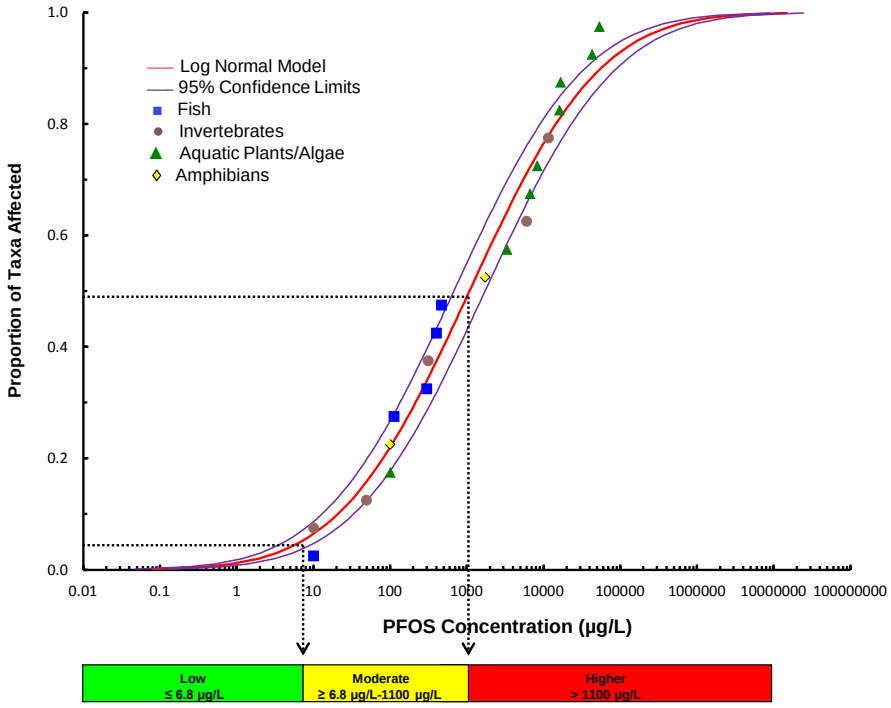

Figure 1. Species sensitivity distribution (SSD) for the chronic toxicity of PFOS and relative likelihood of adverse effects of PFOS to freshwater aquatic life.

The Canadian Water Quality Guideline protocol (CCME 2007) was followed for developing the FWQG for PFOS, with the exception that additional data on six reliable surrogate species were included. While sufficient data exist when the surrogate species are not included, the inclusion of data on surrogate species provides greater breadth and depth to understanding the toxicity of PFOS to aquatic organisms for which, in general, very few species of the total number of aquatic species are tested. Several cumulative distribution functions were fit to the data using regression methods and the best model was selected based on goodness-of-fit. The log normal model provided the best fit for the data and the 5th percentile of the SSD plot is 6.8 µg/L, with lower and upper confidence limits of 4.2 and 11 µg/L, respectively (Figure 1).

Table 2. Chronic Aquatic Toxicity Data Used for Developing the Federal Water Quality Guideline for PFOS.
(see list of abbreviations for description of endpoints).

Species	Group	Endpoint	Concentration (µg/L)	Reference
Japanese medaka (Oryzias latipes)	■	14-d LOEC (growth)	10	Ji et al. (2008)
Damselfly (Enallagma cyathigerum)	●	320-d NOEC (survival)	10	Bots (2010)
Aquatic midge (Chironomus tentans)	●	10-d NOEC (growth, survival)	49	MacDonald et al. (2004)
Watermilfoil (Myriophyllum sibiricum)	▲	42-d EC_{10} (growth)	100	Hanson et al. (2005)
African clawed frog (Xenopus laevis)	◆	67-d NOEC (survival)	100	Cheng et al. (2011)
Zebrafish (Danio rerio)	■	40-d MATC (growth)	112	Du et al. (2009)
Bluegill sunfish (Lepomis macrochirus)	■	35-d MATC (survival)	300	Drottar et al. (2002)
Water flea (Moina macrocopa)	●	7-d LOEC (reproduction)	313	Ji et al. (2008)
Fathead minnow (Pimephales promelas)	■	42-d MATC (survival)	400	Drottar and Krueger (2000a)
Rainbow trout (Oncorhynchus mykiss)	■	21-d LC_{10} (survival)	470	EC (2014)
Leopard frog (Rana pipiens)	◆	60-d MATC (development)	1732	Ankley et al. (2004)
Watermilfoil (Myriophyllum spicatum)	▲	28-d EC_{10} (dry weight)	3300	Hanson et al. (2005)
Water flea (Daphnia pulicaria)	●	21-d EC_{10} (survival)	6000	Sanderson et al. (2004)
Duckweed (Lemna gibba)	▲	7-d IC_{10} (wet weight)	6600	Boudreau et al. (2003)
Green algae (Chlorella vulgaris)	▲	96-h IC_{10} (cell density)	8200	Boudreau et al. (2003)
Water flea (Daphnia magna)	●	21-d EC_{10} (survival)[a]	12000	Boudreau et al. (2003) Sanderson et al.

				(2004)
Green algae (Pseudokirchneriella subcapitata formerly Selenastrum capricornutum)	▲	96-h IC_{10} (cell density)[a]	16000	Boudreau et al. (2003) Drottar and Krueger (2000b)
Diatom (Navicula pelliculosa)	▲	96-h MATC (growth)	16500	Sutherland and Krueger (2001)
Blue-green algae (Anabaena flos-aquae)	▲	96-h IC_{10} (cell density)	42600	Desjardins et al. (2001)
Green algae (Scenedesmus obliquus)	▲	72-h IC_{10} (growth)[a]	53000	Liu et al. (2008)

Legend: ◇ =Amphibian; ■ = Fish; ● = Invertebrate; ▲ = Plant

[a] Effect concentration is the geometric mean of comparable endpoints

The 5th percentile calculated from the SSD, 6.8 µg/L, is the Federal Water Quality Guideline for protection of freshwater organisms (Figure 1).The guideline represents the concentration at which one would expect either no, or only a very low, likelihood of adverse effects on aquatic life. In addition to this guideline, two additional concentration ranges are provided for use in risk management. At concentrations between greater than the FWQG and the 50th percentile of the SSD (i.e. > 6.8 to 1100 µg/L) there is a moderate likelihood of adverse effects to aquatic life. Concentrations greater than the 50th percentile (> 1100 µg/L) have a higher likelihood of adverse effects. The "moderate" and "higher" benchmarks may be used in setting less protective interim targets for waters that are already degraded or where there may be socio-economic considerations that preclude the ability to meet the FWQG. This value is not designed to protect against possible bioaccumulation exposures of higher trophic levels. Instead, tissue residue concentrations are developed below.

Federal Fish Tissue Guideline

The Federal Fish Tissue Guideline (FFTG) is a benchmark for aquatic ecosystems that is intended to protect fish from the direct adverse effects of bioaccumulated contaminants. FFTGs supplement water quality guidelines in that they provide a different metric with which to assess potential adverse effects. FFTGs apply to both freshwater and marine fish, and specify the concentration of PFOS found in whole body fish tissue (wet weight) not expected to result in adverse effects to the fish themselves. The FFTG may not be appropriate to evaluate the impacts of PFOS found in other aquatic biota (amphibians, invertebrates or plants).

It is preferable to develop tissue guidelines from studies that relate tissue concentrations to toxic effects. A study with bluegill, designed to measure bioaccumulation also provided information on residues related to toxic effects (Drottar et al. 2002). Bluegill exposed to 0.086 mg/L PFOS for 62 days accumulated 81 mg/kg ww without significant effects on survival. In contrast, bluegill exposed to 0.87 mg/L experienced heavy mortality at tissue residues starting at 159±16 mg/kg ww ranging to 241±29 mg/kg ww on day 28, at which point mortality was nearly complete. Dividing the no effect value by a safety factor of 10 gives a FFTG of 8.1 mg/kg whole body wet weight.

This value is corroborated by using an equilibrium partitioning approach to estimate a whole body concentration from the federal water quality guideline and the degree to which fish accumulate PFOS either directly from water (bioconcentration factors) or via both food and water (bioaccumulation factors). Although PFOS accumulates in the liver, and is hepatotoxic, monitoring efforts have been directed at measuring the concentration of PFOS in the whole body of fish. Therefore, although liver BAF values were available for PFOS, the FFTG developed here is based on the whole-body accumulation of PFOS.

Accumulation factors, summarized in EC (2006), included lab and field studies with fish, invertebrates and algae from marine and fresh waters, and were reported on a wet-weight (ww) basis. The geometric mean values selected for the calculation were BCFs for bluegill sunfish (Drottar et al. 2002) and carp (Inoue et al. 2012). BCF/BAF values for marine fish were generally higher, but were not considered.

The FFTG was developed as follows:

$$FFTG = (FWQG)\ (BAF_{geomean}) = (6.8\ \mu g/L)\ (1378\ L/kg) = 9.4\ mg/kg\ ww$$

Therefore the FFTG is 9.4 mg/kg body weight fish.

There are several uncertainties inherent in this guideline. The direct correlation between tissue residue and toxic effect was only done in one fish species, using two toxicant concentrations, but in other respects, was of high quality and long duration. Uncertainties also include those in the FWQG in the section above, plus those involved in the BCF/BAF estimation (point estimates of both the tissue and waterborne concentrations). There were few data for freshwater fish.

Federal Wildlife Dietary Guidelines

The Federal Wildlife Dietary Guidelines (FWiDGs) are intended to protect mammalian and avian consumers of aquatic biota. These are benchmarks for concentrations of toxic substances in aquatic biota (whole body, wet-weight) that are consumed by terrestrial and semi-aquatic wildlife. The FWiDGs may not be appropriate to extrapolate the impacts of PFOS to terrestrial consumers other than mammalian and avian species (e.g., reptiles).

FWiDGs for PFOS were developed using laboratory-based toxicity data and associated critical toxicity values (CTVs). The CTV of a study was the lowest treatment dose at which adverse effects were observed amongst organisms as a result of PFOS consumption. CTVs were divided by an uncertainty factor (UF) of 100 to produce a set of tolerable daily intake (TDI) values. The UF of 100 was chosen to account for extrapolation from laboratory to field conditions, and for extrapolation from the observed effects level to a no-effect level. Finally, reference concentrations were calculated for a number of species based on the minimum mammalian TDI (for mammals) and avian TDI (for birds), and the food intake to body weight ratio (FI:BW) specific to that species.

Mammalian: Nine studies were evaluated for four different species, cynomolgus monkeys (Macaca fasicularis), rabbits (Oryctolagus cuniculus), mice and rats. TDIs, calculated as the critical toxicity value divided by an uncertainty factor of 100, ranged from 1.1 to 112 µg/kg bw·d. The lowest TDI of 1.1 µg/kg bw·d was reported for rats and came from a two-year, chronic toxicity diet study (Covance Laboratories 2002). The mammalian FWiDG of 4.6 µg/kg food was calculated by dividing the minimum observed TDI of 1.1 µg/kg bw·d by the maximum mammalian FI:BW of 0.24 kg food/kg bw·d for American mink (CCME 1998).

Avian: Dietary PFOS toxicity to three avian species, mallard (Anas platyrhynchos), northern bobwhite (quail) (Colinus virginianus) and Japanese quail (Coturnix coturnix japonica) were evaluated. For developing the avian FWiDG the selected CTV is the LOAEL dose rate in northern bobwhite of 772 µg/kg bw·d that resulted in reduced chick survival post exposure. By applying an UF of 100, a TDI of 7.7 µg/kg bw·d is produced and an avian FWiDG of 8.2 µg/kg food is calculated by dividing that TDI by the maximum avian FI:BW of 0.94 kg food/kg bw·d for Wilson's storm-petrel (CCME 1998). Given the long duration of both the avian and mammalian studies, the uncertainties relate primarily to lack of knowledge of interspecies sensitivity given the paucity of wildlife species in the data set. Therefore an uncertainty factor of 100 was selected (CCME 1998) for both the avian and mammalian dietary guidelines.

Federal Tissue Quality Guideline for Bird Egg

Laboratory studies provided egg toxicity data for three avian species: northern bobwhite, mallard and white leghorn chicken. For studies performed using mallard and quail as test subjects, the contaminant was administered via maternal transfer from the diet, which is a more natural route of exposure than the chicken studies which administered PFOS via direct injection into the air cell of the egg.

The maternal transfer studies established a no observed adverse effect level (NOAEL) of 53 µg PFOS/mL egg yolk in mallard; a lowest observed adverse effect level (LOAEL) could not be determined. In quail, based on number of survivors as a percentage of eggs set, a LOAEL of 62 µg/mL egg yolk was established; the NOAEL in the pilot study with quail was 33 µg/mL yolk (Newsted et al. 2005).

Studies where PFOS was injected into the air cell of freshly-laid chicken eggs with subsequent incubation found that egg pipping (initial cracking of the egg by the chick during hatching) was reduced to about 67% at 5 µg/g PFOS whole egg compared with controls or with eggs injected with 0.1 µg/g whole egg (O'Brien et al. 2009). Peden-Adams et al. (2009) found no mortality in chicken eggs injected with 1, 2.5 or 5 µg/g egg and no effects on growth. They did however find significant tissue-level effects at all concentrations on development (brain asymmetry, significant only at the lowest concentration, no dose-response) and immune function (no dose response). The ecological significance of these effects is not known. A third study using PFOS injection into chicken eggs (Molina et al. 2006) was considered unacceptable (see O'Brien et al. 2009).

A field study compared reproductive success in tree swallows from a contaminated urban lake versus a reference lake (Custer et al. 2012). The authors concluded that PFOS concentrations above 0.15 µg/g egg were detrimental to hatching success, however, this study could not be

considered in guideline development because of large variability in hatch success between the two field seasons, large variations in egg PFOS concentrations within clutches and concurrent exposure to other perfluorinated substances. Nevertheless, the study should be borne in mind when interpreting PFOS residues in bird eggs.

The egg tissue residue guideline was developed by dividing the LOAEL for quail of 62 µg/mL yolk by a safety factor of 10 to give 6.2 µg/mL. This was subsequently converted to whole egg concentrations for easier comparison with archived whole egg tissue. Most PFOS is contained in the yolk (Newsted et al. 2007; Gebbink and Letcher 2012). Using yolk: albumin ratio of 3:7 (Gebbink and Letcher 2012), and assuming egg density of about 1, the final FTG-BE is 1.9 µg/g whole egg.

References

3M Company. 2000. Voluntary use and exposure information profile for perfluorooctane sulfonic acid and various salt forms. USEPA Docket No. AR226-043.

Ahrens, L., L.W.Y. Leung, S. Taniyasu, P.K.S. Lam, N. Yamashita. 2011. Partitioning of perfluorooctanoate (PFOA), perfluorooctane sulfonate (PFOS) and perfluorooctane sulfonamide (PFOSA) between water and sediment. Chemosphere 85(5): 731-737.

Ahrens, L., S. Taniyasu, L.W.Y. Yeung, N. Yamishita, P.K.S. Lam and R. Ebinghaus. 2010. Distribution of polyfluoroalkyl compounds in water, suspended particulate matter and sediment form Tokyo Bay, Japan. Chemosphere 79: 266-272.

Ankley, G.T., D.W. Kuehl, M.D. Kahl and K.M. Jensen. 2004. Partial life-cycle toxicity and bioconcentration modeling of perfluorooctanesulfonate in the northern leopard frog (Rana pipiens). Environ. Toxicol. Chem. 23: 2745–2755.

Berthiaume, J. and K.B. Wallace. 2002. Perfluorooctane, perfluorooctane sulfonate, and N-ethyl perfluorooctane sulfonamido ethanol; peroxisome proliferation and mitochondrial biogenesis. Toxicology Letters 129: 23-32.

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

Bots, J., L.D. Bruyn, T. Snijkers, B.V.D. Branden and H.V. Gossum. 2010. Exposure to perflurooctane sulfonic acid (PFOS) adversely affects the life-cycle of the damselfly Enallagma cyathigerum. Environmental Pollution 158: 901-905.

Boulanger, B., J. Vargo, J.L. Schnoor and K.C. Hornbuckle. 2004. Detection of perfluorooctane surfactants in Great Lakes water. Environmental Science and Technology 38: 4064-4070.

Butt, C.M., U. Berger, R. Bossi and G.T. Tomy. 2010. Levels and trends of poly- and perfluorinated compounds in the Arctic environment. Sci. Total Environ. 408(15): 2936-65.

[CCME] Canadian Council of Ministers of the Environment. 1998. Protocol for the Derivation of Canadian Tissue Residue Guidelines for the Protection of Wildlife that Consume Aquatic Biota. In: Canadian Environmental Quality Guidelines. Canadian Council of Ministers of the Environment, Winnipeg, Manitoba. Available from: http://www.ccme.ca/publications/list_publications.html#link4

[CCME] Canadian Council of Ministers of the Environment. 2007. A Protocol for the Derivation of Water Quality Guidelines for the Protection of Aquatic Life. In: Canadian Environmental Quality Guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg. Available from: http://ceqg-rcqe.ccme.ca/

Château-Degat, M.L., D. Pereg, R. Dallaire, P. Ayotte, S. Dery and E. Dewailly. 2010. Effects of perfluorooctanesulfonate exposure on plasma lipid levels in the Inuit population of Nunavik (Northern Quebec). Environ. Res. 110(7): 710-17.

Chen, H., C. Zhang, Y. Yu and J. Jianbo. 2012. Sorption of perfluorooctane sufonate (PFOS) on marine sediments. Marine Poll. Bull. 64(5): 902-906.

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

Clarke, D.B., V.A. Bailey, A. Routledge, A.S. Lloyd, S. Hird, D.N. Mortimer and M. Gem. 2010. Dietary intake estimate for perfluorooctanesulphonic acid (PFOS) and other perfluorocompounds (PFCs) in UK retail foods following determination using standard addition LC-MS/MS. Food Additives and Contaminants: Part A, 27(4): 530-45.

Covance Laboratories, Inc. 2002. Final report: 104-week dietary chronic toxicity and carcinogenicity study with perfluorooctane sulfonic acid potassium salt (PFOS; T-6295) in rats. #6329-183. In: Health Canada, 2010; OECD, 2002.

Custer, C.M., T.W. Custer, H.L. Schoenfuss, B.H. Poganski and L. Solem. 2012. Exposure and effects of perfluoroalkyl compounds on tree swallows nesting at Lake Johanna in east central Minnesota, USA. Reprod. Toxicol. 33:556–562.

Dallaire, R., P. Ayotte, D. Pereg, S. Déry, P. Dumas, E. Langlois and E. Dewailly. 2009. Determinants of plasma concentrations of perfluorooctanesulfonate and brominated organic compounds in Nunavik Inuit adults (Canada). Environ. Sci. Tech. 43(13): 5130-36.

Desjardins, D., C.A. Sutherland, R.L. VanHoven and H.O. Krueger. 2001. PFOS: A 96-hour toxicity test with the freshwater algae, with Anabena flos-aquae, with protocol. Wildlife International Ltd. Project number 454A-110B.

Drottar, K.R. and H.O. Krueger. 2000a. PFOS: An early life stage toxicity tests with the fathead minnow (Pimephales promelas). Wildlife International Ltd. AR226-0097.

Drottar, K.R. and H.O. Krueger. 2000b. PFOS: A 96-hour toxicity test with the freshwater algae Selenastrum capricornutum, with protocol. Wildlife International Ltd. Project number 454A-103A.

Drottar, K.R., R.L. VanHoven and H.O. Krueger. 2002. Perfluorooctane sulfonate, potassium salt (PFOS): a flow-through bioconcentration test with the bluegill (Lepomis macrochirus) - amended. Wildlife International Ltd. Project number 454A-134 USEPA docket AR226-1108.

Du, G., J. Hu, H. Huang,Y. Qin, X. Han, D. Wu, L. Song, Y. Xia, and X. Wang. 2013. Perfluorooctane sulfonate (PFOS) affects hormone receptor activity, steroidogenesis, and expression of endocrine-related genes in vitro and in vivo. Environ. Toxicol. Chem. 32: 353–360.

Du, Y., X. Shi, C. Liu, K. Yu and B. Zhou. 2009. Chronic effects of water-borne PFOS exposure on growth, survival and hepatotoxicity of zebrafish: a partial life-cycle test. Chemosphere 74: 723-729.

[EC] Environment Canada. 2001. Primary report on PFAs from Section 71 survey prepared by Use Patterns Section, Chemicals Control Division, Commercial Chemicals Evaluation Branch, Environment Canada, Hull, Canada.

[EC] Environment Canada. 2006. Screening Assessment on Perfluorooctane Sulfonate, Its Salts and Its Precursors that Contain the $C_8F_{17}SO_2$ or $C_8F_{17}SO_3$, or $C_8F_{17}SO_2N$ Moiety. Gatineau (QC). Environment Canada, Ecological Assessment Division. Available upon request.

[EC] Environment Canada. 2013a. Ecological Screening Assessment Report on Perfluorooctane Sulfonate, Its Salts and Its Precursors that Contain the C8F17SO2 or C8F17SO3, or C8F17SO2N Moiety. Accessed November 2014 at: http://www.ec.gc.ca/lcpe-cepa/default.asp?lang=En&n=98B1954A-1&offset=1&toc=show.

[EC] Environment Canada. 2013b. Perfluorooctane sulfonate in the Canadian Environment. Environmental Monitoring and Surveillance in Support of the Chemicals Management Plan. Ottawa, Canada. 22 pp. En14-96/2013E-PDF. ISBN 978-1-100-22426-8.

[EC] Environment Canada. 2014. Perfluorooctane sulfonate (PFOS) Toxicology Project Report. Prepared by Environmental Toxicology Section, Pacific and Yukon Laboratory for Environmental Testing, Environment Canada, North Vancouver, BC. 47 pp. Available at: http://www.ec.gc.ca/toxiques-toxics/default.asp?lang=En&n=7331A46C-1.

[ECCC] Environment and Climate Change Canada. 2016 (in preparation). National Assessment of FEQG exceedances of PFOS since the last update (2011-2014). Monitoring of Perfluorooctane Sulfonate in the Environment under the Chemicals Management Plan. Draft report provided to the Canadian Environmental Sustainability Indicator program by CMP Research and Monitoring, Emerging Priorities Division. June 7, 2016.

Feige, J.N., L. Gelman, L. Michalik, B. Desvergne and W. Wahli. 2006. From molecular action to physiological outputs: peroxisome proliferator-activated receptors are nuclear receptors at the crossroads of key cellular functions. Progress in Lipid Research 45: 120–159.

Gallagher, S.P., R.L. Van Hoven, J.B. Beavers and M. Jaber. 2003a. PFOS: A reproduction study with the northern bobwhite. Wildlife International Ltd. Project number 454-103.

Gallagher, S.P., R.L. Van Hoven and J.B. Beavers. 2003b. PFOS: A pilot reproduction study with the mallard. Wildlife International Ltd. Project number 454-105.

Gebbink, WA, RJ Letcher. 2012. Comparative tissue and body compartment accumulation and maternal transfer to eggs of perfluoroalkyl sulfonates and carboxylates in Great Lakes herring gulls. Environ Pollut 162:40-47.

Giesy, J.P. and K., Kannan. 2001. Global distribution of perfluorooctane sulfonate in wildlife. Environmental Science Technology 35: 1339-1342.

Giesy, J.P. and K., Kannan. 2002. Perfluorochemical surfactants in the environment. Environmental Science and Technology 36: 147A–152A.

Government of Canada. 2008. Perfluorooctane Sulfonate and its Salts and Certain Other Compounds Regulation, SOR/2008-178. Canada Gazette Part II. 2008. Available at: http://laws-lois.justice.gc.ca/PDF/SOR-2008-178.pdf. Accessed Aug. 5, 2015.

Government of Canada. 2009. Regulations adding perfluorooctane sulfonate and its salts to the Virtual Elimination List, SOR/2009-15. Canada Gazette II. 2009. 143(3): 76-79. Available at: http://laws-lois.justice.gc.ca/eng/regulations/SOR-2009-15/page-1.html#ord. Accessed Aug. 5, 2015.

Government of Canada 2016. Perfluorooctane Sulfonate (PFOS) in Fish and Water. Canadian Environmental Sustainability Indicators (CESI). https://www.ec.gc.ca/indicateurs-indicators/default.asp?lang=en&n=9AA98870-1. Last modified 2016-02-17. Accessed June 7, 2016

Hagenaars, A., D. Knapen, I.J. Meyer, K. van der Ven, P. Hoff and E. De Coen. 2008. Toxicity of perfluorooctane sulfonate (PFOS) in the liver of common carp (Cyprinus carpio). Aquatic Toxicology 88: 155-163.

Hansen, K.J., H.O. Johnson, J.S. Eldridge, J.L. Butenhoff, and L.A. Dick. 2002. Quantitative characterization of trace levels of PFOS and PFOA in the Tennessee River. Environmental Science and Technology 36: 1681-1685.

Hanson, M.L., P.K. Sibley, R.A. Brain, S.A. Mabury and K.R. Solomon. 2005. Microcosm evaluation of the toxicity and risk to aquatic macrophytes from perfluorooctane sulfonic acid. Arch. Environ. Contam. Toxicol. 48: 329-337.

Hickey, N. J., D. Crump, S. P. Jones, and S. W. Kennedy. 2009. Effects of 18 perfluoroalkyl compounds on mRNA expression in chicken embryo hepatocyte cultures. Toxicological Sciences. 111:311-320.

Higgins, C.P., and R.G. Luthy. 2006. Sorption of perfluorinated surfactants on sediments. Environmental Science and Technology 40: 7251-7256.

Higgins, C.P. and R.G. Luthy. 2007. Modeling sorption of anionic surfactants onto sediment materials: an a priori approach for perfluoroalkyl surfactants and linear alkylbenzene sulfonates. Environ. Sci. Technol. 41(9): 3254-3261.

Hoff, P.T., W. Van Dongen, E.L. Esmans, R. Blust and W.M. De Coen. 2003. Evaluation of the toxicological effects of perfluorooctane sulfonic acid in the common carp (Cyprinus carpio). Aquatic Toxicology 62: 349-359.

Houde, M., C. Gertje, J.M. Small, S. Backus, X. Wang, M. Alaee and D.C.G. Muir. 2008. Fractionation and bioaccumulation of perfluorooctane sulfonate (PFOS) isomers in a Lake Ontario food web. Environmental Science and Technology 42: 9397-9403.

Hu, W.Y., P.D. Jones, B.L. Upham, J.E. Trosko, C. Lau and J.P. Giesy. 2002. Inhibition of gap-junction intercellular communication by perfluorinated compounds in rat liver and dolphin kidney epithelial cell lines in vitro and Sprague–Dawley rats in vivo. Toxicological Sciences 68: 429–436.

Inoue, Y., N. Hashizume, N. Yakata, H. Murakami, Y. Suzuki, E. Kikushima and M. Otsuka. 2012. Unique physicochemical properties of perfluorinated compounds and their bioconcentration in common carp (Cyprinus carpio L.). Arch Envrion, Contam, Toxicol. 63(2): 272-680.

Johansson, N., A Friedriksson, P. Eriksson. 2008. Neonatal exposure to perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) causes neurobehavioural defects in adult mice. Neurotoxicology 29: 160-169.

Jones, P.D., W. Hu, W. De Coen, J.L. Newsted, and J.P. Giesy. 2003. Binding of perfluorinated fatty acids to serum proteins. Environmental Toxicology and Chemistry 22: 2639-2649.

Ji, K., Y. Kim, S. Oh, B. Ahn, H. Jo and K. Choi. 2008. Toxicity of sulfonic acid and perfluorooctanoic acid on freshwater macroinvertebrates (Daphnia magna) and fish (Oryzias latipes). Environmental Toxicology and Chemistry 27: 2159-2168.

Jing, P. P.J. Rodgers and S. Amemiya. 2009. High lipophilicity of perfluroalkyl carboxylate and sulfonate: Implications for their membrane permeability. J. Am. Chem. Soc. 131: 2290-2296.

Kannan, K., J. Koistinen, K. Beckmen, T. Evans, J.F. Gorzelany, K.J. Hansen, P.D. Jones, E. Helle, M. Nyman and J.P. Giesy. 2001a. Accumulation of perfluorooctane sulfonate in marine mammals. Environmental Science Technology 35: 1593-1598.

Kannan, K., J.C. Franson, W.W. Bowerman, K.J. Hansen, P.D. Jones and J.P. Giesy. 2001b. Perfluorooctane sulfonate in fish-eating birds including bald eagles and albatrosses. Environmental Science and Technology 35: 3065-3070.

Kannan, K., J. Newstead, R.S. Halbrook, and J.P. Giesy. 2002a. Perfluorooctane sulfonate and related fluorinated hydrocarbons in mink and river otters from the United States. Environmental Science and Technology 36: 2566–2571.

Kannan, K., S. Corsolini, J. Falandysz, G. Oehme, S. Focardi, and J.P. Giesy. 2002b. Perfluorooctane sulfonate and related fluorinated hydrocarbons in marine mammals, fishes and birds from coasts of the Baltic and the Mediterranean seas. Environmental Science and Technology 36: 3210-3216.

Kannan, K., L. Tao, E. Sinclair, S.D. Pastva, D.J. Jude and J.P. Giesy. 2005. Perfluorinated compounds in aquatic organisms at various trophic levels in a Great Lakes food chain. Archives of Environmental Contamination and Toxicology 48: 559-566.

Kannan, K., E. Perrotta and N.J. Thomas. 2006. Association between perfluorinated compounds and pathological conditions in southern sea otters. Environmental Science and Technology 40: 4943-4948.

Kim, S.K. and K. Kannan. 2007. Perfluorinated acids, in air, rain, snow, surface runoff, and lakes: relative importance of pathways to contamination of urban lakes. Environmental Science and Technology 41: 8328-8334.

Kissa, E. 1994. Fluorinated Surfactants, Synthesis, Properties, Applications. Marcel Dekker. New York, NY.

Kwadijk, C.J.A.F., P.Korytar and A.A. Koelmans. 2010. Distribution of perfluorinated compounds in aquatic systems in The Netherlands. Environ. Sci. Technol. 44:3746-3751.

Labadie, P. and M. Chevreuil. 2011. Partitioning behaviour of perfluorinated alkyl contaminants between water, sediment and fish in the Orge River (nearby Paris, France). Environ. Poll. 159: 391-397.

Lehmler, H.J. 2005. Synthesis of environmentally relevant fluorinated surfactants—a review. Chemosphere 5: 1471-1496.

Liu, R., X. Liu, H. Tang, and Y. Su. 2001. Sorption behaviour of dye compounds onto natural sediment of Qinghe River. Journal of Colloid and Interface Science 239: 475–482.

Liu, W., S. Chen, X. Quan and Y.H. Jin. 2008. Toxic effect of serial perfluorosulfonic and perfluorocarboxylic acids on the membrane system of a freshwater alga measured by flow cytometry. Environmental Toxicology and Chemistry 27: 1597-1604.

Lucaciu, C., V, Furdui, P. Crozier, C. Marvin, E. Reiner, F. Wania and S. Mabury. 2005. Temporal study of perfluorinated alkyl substances in Niagara River suspended sediments. Abstract SETAC 2005, Baltimore, Maryland, November 2005. http://abstracts.co.allenpress.com/pweb/setac2005/document/57070

Luebker, D.J., M.T. Case, R.G. York, J.A. Moore, K.J. Hansen and J.L. Butenhoff. 2005. Two-generation reproduction and cross-foster studies of perfluorooctane sulfonate (PFOS) in rats. Toxicology 215: 126-148.

MacDonald, M.M., A.L. Warne, N.L. Stock, S.A. Mabury, K.R. Solomon and P.K. Sibley. 2004. Toxicity of perfluorooctane sulfonic acid and perfluorooctanoic acid to Chironomus tentans. Environmental Toxicology and Chemistry 23: 2116-2123.

Martin, J.W., D.C.G. Muir, C.A. Moody, D.A. Ellis, W.C. Kwan, K.R. Solomon and S.A. Mabury. 2002. Collection of airborne fluorinated organics and analysis by gas chromatography/chemical ionization mass spectrometry. Anal. Chem. 74: 584–590.

Martin, J.W., S.A. Mabury, K.R. Solomon, and D.C.G. Muir. 2003. Dietary accumulation of perfluorinated acids in juvenile rainbow trout (Oncorhynchus mykiss). Environmental Toxicology and Chemistry 22: 189–195.

Martin, J.W., M.M. Smithwick, B. Braune, P.F. Hoekstra, D.C.G. Muir and S.A. Mabury. 2004. Identification of long-chain perfluorinated acids in biota from the Canadian Arctic. Environmental Science and Technology 38: 373–380.

McNabb, F.M.A., L. Smith and K. Clark. 2005. Effects of perfluorooctane sulfonate (PFOS) on thyroid function in quail. Presentation at 26th Annual Meeting of SETAC, Baltimore, Maryland, November 13-17, 2005.

Ministry of Environmental Protection of China. 2008. Comments on the revised draft risk profile for SCCP. 7.

Molina, E. D., R. Balander, S.D. Fitzgerald, J.P. Giesy, K. Kannan, R. Mitchell and S.J. Bursian. 2006. Effects of air cell injection of perfluorooctane sulfonate before incubation on development of the white leghorn chicken (Gallus domesticus) embryo. Environmental Toxicology and Chemistry 25 (1):227-232.

Nakata, H., K. Kannan, T. Nasu, H.S. Cho, E. Sinclair and A. Takemura. 2006. Perfluorinated contaminants in sediments and aquatic organisms collected from shallow water and tidal flat areas of the Ariake Sea, Japan: Environmental fate of perfluorooctane sulfonate in aquatic ecosystems. Environmental Science and Technology 40: 4916-4921.

Newsted, J.L., P.D. Jones, K. Coady, and J.P. Giesy. 2005. Avian toxicity reference values for perfluorooctane sulfonate. Environmental Science Technology 39: 9357-9362

Newsted, J.L., K.K. Coady, S.A. Beach, J.L. Butenhoff, S. Gallagher and J.P. Giesy. 2007. Effects of perfluorooctane sulfonate on mallard and northern bobwhite quail exposed chronically via the diet. Environmental Toxicology and Pharmacology 23: 1-9.

O'Brien, J.M., A.C. Carew, S. Chu, R.J. Letcher, and S.W. Kennedy. 2009. Perfluorooctane sulfonate (PFOS) toxicity in domestic chicken (Gallus gallus domesticus) embryos in the absence of effects on peroxisome proliferator activated receptor alpha (PPARα)-regulated genes. Comparative Biochemistry and Physiology, Part C. 149:524-530.

OECD. 2002. Hazard assessment of perfluorooctane sulfonate (PFOS) and its salts. ENV/JM/RD(2002)17/FINAL, November 21, Paris. 362 pp.

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

Quinete, N., Q. Wu, T. Zhang, S.H. Yun, I. Moreira and K. Kannan. 2009. Specific profiles of perfluorinated compounds in surface and drinking waters and accumulation in mussels, fish, and dolphins from southeastern Brazil. Chemosphere 77: 863-869.

Rayne, S. and K. Forest. 2009a. Perfluoroalkyl sulfonic and carboxylic acids: a critical review of physicochemical properties, levels and patterns in waters and wastewaters, and treatment methods. J. Environ. Sci. Health A 44:1145-1199.

Rayne, S. and K. Forest. 2009b. Congener-specific organic carbon-normalized soil and sediment-water partitioning coefficients for the C1 through C8 perfluoroalkyl carboxylic and sulfonic acids. J. Environ. Sci. Health A 44:1374-1387

Rosen, M.B., Schmid, J.R. Schmid, J.C. Corton, R.D. Zehr, K.P. Das, B.D. Abbott and C. Lau. 2010. Gene expression profiling in wild-type and PPARα-null mice exposed to perfluorooctane sulfonate reveals PPARα-independent effects. PPAR Research. 2010. 23pp.

Sanderson, H., T.M. Boudreau, S.A. Mabury and K.R. Solomon. 2004. Effects of perfluoroctanoic acid on the zooplankton community. Ecotoxicity and Environmental Safety 58: 68-76.

Senthilkumar, K., E. Ohi, K. Sajwan, T. Takasuga and K. Kannan. 2007. Perfluorinated compounds in river water, river sediment, market fish, and wildlife samples from Japan. Bulletin of Environmental Contamination and Toxicology 79: 427-431.

Smithwick, M., S.A. Mabury, K.R. Solomon, C. Sonne, J.W. Martin, E.W. Born, R. Dietz, A.E. Derocher, R.J. Letcher, T.J. Evans, G.W. Gabrielsen, J. Nagy, I Stirling, M.K. Taylor and D.C.G. Muir. 2005. Circumpolar study of perfluoroalkyl contaminants in polar bears (Ursus maritimus). Environmental Science and Technology 39: 5517-5523.

Sutherland, C.A. and H.O. Krueger. 2001. A 96-hour toxicity test with the freshwater diatom (Navicula pelliculosa). Wildlife International Ltd. Project number 454A-112.

Tao, L., K. Kannan, N. Kajiwara, M.M. Costa, G. Fillmann, S. Takahashi and S. Tanabe. 2006. Perfluorooctane sulfonate and related fluorochemicals in albatrosses, elephant seals, penguins, and polar skuas from the Southern Ocean. Environmental Science and Technology 40: 7642-7648.

Thomford, P.J. 2000. 4-Week capsule toxicity study with perfluorooctane sulfonic acid potassium salt (PFOS; T-6295) in cynomolgus monkeys. Covance Laboratories Inc. Covance 6329-223. 3M Study No. T-6295.7.

York, R. 1999. PFOS rat two-generation reproduction study. Argus Research Laboratories, Inc. US EPA OPPT AR226-0569.

You, C., J. Chengxia and P. Gang. 2010. Effect of salinity and sediment characteristics on the sorption and desorption of perfluorooctane sulfonate at sediment-water interface. Environmental Pollution 158: 1343-1347.

List of Acronyms and Abbreviations

AFFF– aqueous film forming foam

BAF – bioaccumulation factor: the ratio of the concentration of a chemical compound in an organism relative to the concentration in the exposure medium, based on uptake from the surrounding medium and food

BCF – bioconcentration factor: the ratio of the concentration of a chemical compound in an organism relative to the concentration of the compound in the exposure medium (e.g. soil or water)

BMF – biomagnification factor: a measure of bioaccumulation by which tissue concentrations of accumulated chemical compounds are determine relative to tissue concentrations in two or more trophic levels

CCME – Canadian Council of Ministers of Environment

CEPA – Canadian Environmental Protection Act

CEQG – Canadian Environmental Quality Guideline

CMP – Chemicals Management Plan

CTV – critical toxicity value

ECCC – Environment and Climate Change Canada

EC – effect concentration

FEQG – Federal Environmental Quality Guideline
FFTG – Federal Fish Tissue Guideline
Fi:BW – food intake: body weight ratio
FTG-BE – Federal Tissue Guideline for Bird Egg
FWiDG – Federal Wildlife Diet Guideline
FWQG – Federal Water Quality Guidelines
IC – inhibition concentration
K_d – distribution coefficient
K_{ow} – octanol water partition coefficient
LOEC – lowest observed effect concentration
LOAEL – lowest observed adverse effect level
MATC – maximum acceptable toxicant concentration
N-EtFOSE alcohol – 2-(N-ethylperfluoro-1-octanesulfonamido)-ethanol
N-MeFOSE alcohol – 2-(N-methylperfluoro-1-octanesulfonamido)-ethanol
NOAEL – no observed adverse effect level
NOEC – no observed effect level
OECD – Organization for Economic Co-operation and Development
PFCs – perfluorinated compounds
PFOS – perfluorooctane sulfonate
POSF – perfluorooctanesulfonyl fluoride
SAR – screening assessment report
SSD – species sensitivity distribution
TDI – tolerable daily intake
UF – uncertainty factor