***Canadian Environmental Protection Act, 1999* (CEPA 1999):**
Environmental Screening Assessment Report on
Perfluorooctane Sulfonate, Its Salts and Its Precursors that Contain the $C_8F_{17}SO_2$ or $C_8F_{17}SO_3$ Moiety

April 2004

Environment Canada

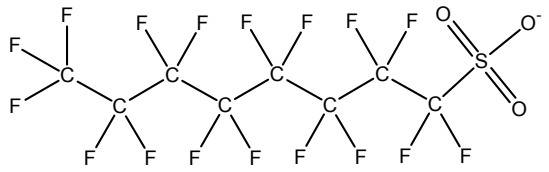

Figure 1. PFOS anion structure

Introduction

The *Canadian Environmental Protection Act, 1999* (CEPA 1999) requires the Minister of the Environment and the Minister of Health to conduct screening assessments to determine whether substances present or may present a risk to the environment or to human health. Based on the results of a screening assessment, the Ministers can propose taking no further action with respect to the substance, can suggest adding the substance to the Priority Substances List (PSL) for further assessment or can recommend the addition of the substance to the List of Toxic Substances in Schedule 1 and, where applicable, the implementation of virtual elimination.

An environmental screening assessment involves an analysis of a substance using conservative assumptions to determine whether the substance is "toxic" or capable of becoming "toxic" as defined in CEPA 1999. The approach taken in this environmental screening assessment is to examine various supporting information and develop conclusions based on a weight of evidence approach as required under Section 76.1 of CEPA 1999. The environmental screening assessment does not present an exhaustive review of all available data; rather, it presents the most critical studies and lines of evidence supporting the conclusions. One line of evidence includes consideration of risk quotients to identify potential for ecological effects. However, other concerns that affect current or potential risk, such as persistence, bioaccumulation, chemical transformation and precursors, are also examined in this report.

An environmental screening assessment was undertaken on perfluorooctane sulfonate (PFOS), its salts and compounds that containing the perfluorooctylsulfonyl ($C_8F_{17}SO_2$) or $C_8F_{17}SO_3$ groups (the latter hereafter referred to as precursors) on the basis that some of these compounds were included in the Domestic Substances List (DSL) pilot list for screening based on their meeting the criteria for persistence and/or bioaccumulation and inherent toxicity, pursuant to Paragraph 73(1)(b) of CEPA 1999, and in response to a

request to the Minister of the Environment to add these compounds to the PSL. The term PFOS may refer to any of its anionic, acid or salt forms. The perfluorooctylsulfonyl ($C_8F_{17}SO_2$) or $C_8F_{17}SO_3$ group is incorporated in a variety of compounds, and these compounds have the potential to transform or degrade back to PFOS in the environment. Due to similar use applications and the fact that PFOS is the persistent final degradation product of PFOS precursors, this environmental screening assessment addresses PFOS and its precursors together. Once PFOS is released to the environment, it is not known to undergo any further chemical, microbial or photolytic degradation and is considered persistent. Therefore, it is expected that the precursors contribute to the total amount of PFOS in the environment.

Data relevant to the environmental screening assessment of PFOS and its precursors were identified in original literature, review documents and industry research reports. A supporting document was prepared on selected perfluoroalkyl compounds, and degradation modelling (using CATABOL[1] software) to predict PFOS precursors was performed. On-line literature database searches were conducted for select perfluoroalkyl compounds. As well, direct contacts were made with researchers, academics, industry and other government agencies to obtain relevant information on PFOS and its precursors.

Ongoing scans were conducted of the open literature, conference proceedings and the Internet for relevant information. Data obtained up to February 2004 were considered in this document. In addition, an industry survey on certain perfluoroalkyl and fluoroalkyl substances, their derivatives and polymers was conducted in 2000 through a *Canada Gazette* Notice issued under authority of Section 71 of CEPA 1999. This survey collected data on the Canadian manufacture, import and export of certain perfluorinated alkyl compounds (Environment Canada 2000). Existing toxicological studies were also submitted by industry under Section 70 of CEPA 1999.

The environmental screening assessment report and associated unpublished supporting working documentation were written by a team of Environment Canada evaluators at the Existing Substances Branch, Gatineau, Quebec. The information in this report has undergone external peer review by Canadian and international experts from government, industry and academia, including S. Beach (3M), W. De Coen (University of Antwerp, Belgium), P. de Voogt (University of Amsterdam), W. de Wolf (DuPont, Germany), S. Dimitrov (Prof. As Zlatarov University, Bourgas, Bulgaria), J. Giesy (Michigan State University), O. Hernandez (US Environmental Protection Agency), S. Mabury (University of Toronto), R. Medsker (private consultant), O. Mekenyan (Prof. As Zlatarov University, Bourgas, Bulgaria), D. Muir (Environment Canada, National Water Research Institute), R. Purdy (private consultant), E. Reiner (3M), M. Santoro (3M) and B. Scott (Environment Canada, National Water Research Institute).

[1] CATABOL is a computer system for predicting biodegradability metabolic pathways and toxicity of stable biodegradation products. It is a product of the Laboratory of Mathematical Chemistry, University "Prof. As. Zlatarov," Bourgas, Bulgaria.

The environmental and human health screening assessment reports were approved by the joint Environment Canada/Health Canada CEPA Management Committee. The supporting technical report is available upon request by e-mail from PSL.LSIP@ec.gc.ca. Information on environmental screening assessments under CEPA 1999 is available at http://www.ec.gc.ca/substances/ese. Information on the screening health assessment is available at http://www.hc-sc.gc.ca/exsd-dse.

The critical information and considerations upon which the assessment is based are summarized below.

Identity, Uses, Sources and Release

Substance Identity

The PFOS anion, shown in Figure 1, has the molecular formula $C_8F_{17}SO_3^-$; the structural formula is $CF_3(CF_2)_7SO_3^-$. While PFOS can exist in anionic, acid and salt forms, the PFOS anion is the most common form at pH values in the environment and in the human body.

PFOS and its precursors all belong to the larger class of fluorochemicals referred to as perfluorinated alkyl compounds. This assessment defines PFOS precursors as substances containing the perfluorooctylsulfonyl ($C_8F_{17}SO_2$) or $C_8F_{17}SO_3$ moiety that have the potential to transform or degrade to PFOS. Appendix 1 lists some compounds considered as PFOS and its precursors — namely, the PFOS anion; PFOS acid (PFOSH); four PFOS salts; perfluorooctanesulfonyl fluoride (POSF) and four common intermediates for producing PFOS-related chemicals (N-MeFOSA, N-EtFOSA, N-MeFOSE alcohol and N-EtFOSE alcohol; for complete chemical names, see Appendix 1), all of which are considered PFOS precursors; and other precursors not in the above-named categories. The list is not considered exhaustive, as there may be other perfluorinated alkyl compounds that are also PFOS precursors. Appendix 1 was compiled based on information obtained through the Section 71 survey to industry, expert judgement and CATABOL modelling, in which 256 perfluorinated alkyl compounds were examined to determine whether non-fluorinated components of each substance were expected to degrade chemically and/or biochemically and whether the final perfluorinated degradation product was predicted to be PFOS (Mekenyan et al. 2002; Purdy 2002a).

Perfluorinated chemicals such as PFOS contain carbons that are completely saturated by fluorine. It is the strength of the C–F bonds that contributes to the extreme stability and unique properties of these perfluorochemicals.

The chemistry and identity of fluorochemical products can be complex. For example, compounds produced during the electrochemical fluorination process (e.g., POSF) are not pure chemicals, but mixtures of isomers and homologues. Similarly, POSF-derived fluorochemicals and products do not necessarily produce pure products (US EPA OPPT

AR226-0550).[2] Varying amounts of unreacted or partially reacted starting materials or intermediates, including PFOS, N-MeFOSA, N-EtFOSA, N-MeFOSE alcohol and N-EtFOSE alcohol, can be carried forward to final products at typical concentrations of 1–2% or less (US EPA OPPT AR226-0550). These residuals in final products have the potential to degrade or metabolize to PFOS (US EPA OPPT AR226-0550). Once PFOS is released to the environment, it is not known to undergo any further chemical, microbial or photolytic degradation and is therefore persistent. Therefore, as well as being commercially produced, PFOS is the final degradation product from POSF-derived fluorochemicals.

Key physical/chemical properties of PFOS and some precursors that are useful in predicting its environmental fate are listed in Table 1.

Table 1. Selected physical and chemical properties of PFOS potassium salt and common intermediates

Substance	CAS No.	Molecular weight (g/mol)	Solubility (g/L)	Vapour pressure (Pa)	Henry's law constant ($Pa \cdot m^3/mol$)[a]	Log K_{ow}	Melting point (°C)	Boiling point (°C)
PFOS (K^+)	2795-39-3	538.23	5.19 E-1	3.31 E-4	3.45 E-4	Not calculable	>400	Not calculable
N-EtFOSE alcohol	1691-99-2	571.26	1.51 E-4	5.04 E-1	1.93 E+3	4.4	55–60	N/A[b]
N-EtFOSEA	423-82-5	625.30	8.9 E-4	N/A	N/A	N/A	27–42	150 at 133.3 Pa
N-MeFOSE alcohol	24448-09-7	557.23	N/A	N/A	N/A	N/A	N/A	N/A
N-MeFOSEA	25268-77-3	611.28	N/A	N/A	N/A	5.6	N/A	N/A

[a] 1 atm = 101.3 kPa.
[b] N/A = not available.
Source: Hekster et al. (2002)

Natural Sources

Notably, all perfluorinated compounds are of anthropogenic origin, and there are no known natural sources of PFOS (Key et al. 1997). Its presence in the environment is due solely to human activity.

Manufacture and Import

Results from the Section 71 Notice indicated that PFOS and its precursors are not manufactured in Canada but rather are imported as chemicals or products from the United States for Canadian uses. They may also be components in imported manufactured articles. Approximately 587 tonnes of perfluorinated alkyl compounds were imported into Canada during 1997–2000, with PFOS and its precursors accounting for 43% of imported perfluorinated alkyl compounds. PFOS alone accounted for <2% of imported perfluorinated alkyl compounds (Environment Canada 2001). The most significant

[2] These Administrative Records are all 3M submissions to the US Environmental Protection Agency's (US EPA) Office of Pollution Prevention and Toxics. Additional information may be found in the reference list.

Canadian imports of PFOS itself were in the form of the potassium salt, used for fire-fighting foams.

As PFOS production has also been identified in Italy, Japan, Belgium, Germany and Asia, PFOS-containing consumer products could also be imported into Canada from non-US sources. It is not known whether foreign companies are phasing out of PFOS manufacturing. Therefore, the potential remains for PFOS-containing products/materials manufactured elsewhere to continue being imported into Canada; however, these quantities are unknown.

Since 2000, 3M has been phasing out its use of the perfluorooctanyl chemicals and products containing PFOS. Survey data indicated an overall decline in imports from 1997 to 2000. The US 3M phase-out plan for POSF-based products issued to the US EPA states that 3M anticipated that the manufacture and import of affected products would have ceased by the end of 2002, although 3M will continue to distribute small volumes of certain previously manufactured products to specific customers (US EPA OPPT AR226-0588).

Use

It is estimated that the majority of all perfluorinated alkyl compounds imported into Canada were used in applications involving water, oil, soil and grease repellents for fabric, packaging and rugs and carpets; and surfactants/detergents, emulsifiers, wetting agents, dispersants and fire-fighting foams. It is expected that PFOS and its precursors are present in many of these use applications.

Release

3M has compiled a preliminary review of fluorochemical use, distribution and release (US EPA OPPT AR226-0550).

Significant PFOS releases to the Canadian environment would be expected from the major use applications involving water, oil, soil and grease repellents for packaging (Environment Canada 2001).

Currently, there are no data available to reflect potential Canadian releases from the use and final disposal of a vast variety of imported finished consumer products (e.g., home furnishings and electronic goods) that may contain PFOS or PFOS precursors as part of fabric finishes or coatings. However, PFOS could eventually be released to the Canadian environment from these sources.

Environmental releases from surface treatments for rugs and carpets are expected during use and may involve discharges to process wastewater and air during initial applications (e.g., to uncut carpets) (US EPA OPPT AR226-0550). Additional wastes occur from cutting, shearing or packaging operations and are generally landfilled or recycled. As well, end use of consumer articles will create losses (e.g., it is estimated that vacuuming

and cleaning of carpets create releases; final disposal of treated carpets is generally to landfills) (US EPA OPPT AR226-0550). Industry Canada (2002) statistics indicate that approximately 22 active carpet and rug mills were operating in Canada in 1999. This number does not account for those establishments classified as "non-employers" or where carpet manufacturing is not the primary activity.

In the case of fire-fighting foams, final disposal would primarily be to sewers (wastewater treatment), although uncontrolled releases to surface waters or land may occur (US EPA OPPT AR226-0550).

It has been suggested that PFOSH (PFOS acid) may be released to the environment from incomplete combustion during incineration of PFOS-containing products (US EPA 2002). While work continues in the United States to validate this release scenario, any potential formation and release of PFOS through incineration would likely be a minor source in Canada, where incineration accounts for only about 5% of waste disposal (Compass Environmental Inc. 1999).

Fate, Exposure and Effects

Environmental Fate of PFOS Precursors

PFOS precursors may be subject to atmospheric transport from their sources to remote areas. While exact transport mechanisms and pathways are currently unknown, the vapour pressures of PFOS precursors, such as N-EtFOSEA and N-MeFOSEA, may exceed 0.5 Pa (1000 times greater than that of PFOS) (Giesy and Kannan 2002). Several PFOS precursors are considered volatile, including N-EtFOSE alcohol, N-MeFOSE alcohol, N-MeFOSA and N-EtFOSA (US EPA OPPT AR226-0620). Two PFOS precursors, N-EtFOSE alcohol and N-MeFOSE alcohol, have been measured in air in Toronto and Long Point, Canada (Martin et al. 2002). For precursors released to the water compartment, the vapour pressure may be significant enough to allow the substance to enter into the atmosphere. For N-EtFOSE alcohol, the tendency to leave the water phase is supported by its relatively high Henry's law constant (1.9×10^3 $Pa \cdot m^3/mol$) (Hekster et al. 2002). 3M has reported that when these PFOS precursors are present as residuals in products, they could evaporate into the atmosphere when the products containing them are sprayed and dried (US EPA OPPT AR226-0620). The volatility of certain PFOS precursors may lead to their long-range atmospheric transport (Martin et al. 2002). Although evidence of long-range transport of precursors is limited, it is expected that this is at least partially responsible for the ubiquitous presence of PFOS measured at a distance from significant sources.

It is expected that the precursors identified in Appendix 1 will undergo degradation once released to the environment. The perfluorinated moiety is known to be very resistant to degradation, a property attributed to the C–F bond, one of the strongest chemical bonds in nature (~110 kcal/mol) (US EPA OPPT AR226-0547). The perfluorinated chain provides exceptional resistance to thermal and chemical attack (US EPA OPPT AR 226-0547).

Precursors that reach a remote region through the atmosphere or other media are expected to undergo abiotic or biotic degradation to PFOS (Giesy and Kannan 2002; Hekster et al. 2002). The mechanism of this degradation is not well understood; however, it is expected to involve both abiotic and biotic degradation routes. The available experimental environmental degradation rates of PFOS precursors are limited to N-MeFOSE alcohol, N-EtFOSE alchohol, N-MeFOSEA and N-EtFOSEA and are summarized in Table 2.

Table 2. Summary of available data on transformation of PFOS and its precursors

Substance	Biodegradation	Biotransformation	Photolysis	Hydrolysis
PFOS (K^+)	0%	N/A [b]	0%	$t_{½}$ > 41 years
N-MeFOSE alcohol	N/A	N/A	N/A	$t_{½}$ = 6.3 years
N-EtFOSE alcohol	To PFOS/PFOA[a]	N/A	0%	$t_{½}$ = 7.3 years 92% after 24 hours to PFOS (alkaline)
N-MeFOSEA	N/A	N/A	N/A	$t_{½}$ = 99 days at pH 7, 25°C (extrapolated)
N-EtFOSEA	N/A	N/A	N/A	$t_{½}$ = 35 days at pH 7, 25°C

[a] PFOA = perfluorooctanoic acid.
[b] N/A = not available.
Source: Hekster et al. (2002)

The two most common intermediate substances used for producing PFOS, N-EtFOSE alcohol and N-MeFOSE alcohol, were tested at several pH concentrations for hydrolysis potential (US EPA OPPT AR226-1030a076, AR226-1030a079). While some of the alcohols disappeared during the test, no PFOS was generated. No hydrolysis studies on N-EtFOSE esters or N-MeFOSE esters were found in a search of US EPA OPPT AR226, which deals entirely with perfluorinated alkyl compounds (US EPA OPPT AR226-0001 through AR226-1040).

The available studies on photolysis show that this transformation mechanism will be of no importance in the breakdown of perfluorinated chemicals. The tests with PFOS, perfluorooctanoic acid (PFOA), POSF and N-EtFOSE alcohol show no photodegradation at all (Hekster et al. 2002; US EPA OPPT AR226-0184, AR226-1030a041). Aqueous photolytic screening studies carried out with N-EtFOSE alcohol, N-MeFOSE alcohol, N-EtFOSA and N-MeFOSA as well as on a surfactant and foamer product showed no direct photolysis, although some underwent indirect photolysis. The primary products were PFOA, perfluorooctane sulfonic acid (PFOSA) and N-EtFOSA (US EPA OPPT AR226-1030a073, AR226-1030a074, AR226-1030a080, AR226-1030a106).

Although experimental evidence on the degradation of PFOS precursors to PFOS is very limited, the precursors are expected to degrade through bacterial-mediated degradation pathways. The biodegradation software, CATABOL, which simulates Organisation for Economic Co-operation and Development (OECD) 302C 28-day biodegradation tests and which has been designed to accommodate perfluorinated compounds, predicts that the

majority of those substances identified as precursors (see Appendix 1) will degrade to PFOS (Dimitrov et al. 2004). This degradation has been further supported by expert judgement. It is therefore expected that once those substances listed in Appendix 1 are subjected to a biotic or abiotic degradation mechanism, the perfluorinated moiety that remains will be PFOS. The rate of degradation to PFOS is not considered significant to this assessment, as, over time, these substances are all expected to degrade in the Canadian environment to PFOS.

Environmental Fate of PFOS

Once the precursors degrade to PFOS, the substance will remain indefinitely, as there are no known degradation mechanisms for PFOS in the environment.

Due to the high energy of the C–F bond, PFOS is resistant to hydrolysis, photolysis, aerobic and anaerobic biodegradation and metabolism by vertebrates. The estimated half-life for PFOS is reported as >41 years (Hekster et al. 2002), but may be significantly longer than 41 years. The persistent nature of PFOS is supported by numerous studies (Key et al. 1997; Giesy and Kannan 2002; Hekster et al. 2002; OECD 2002). PFOS is considered to be persistent in the Canadian environment, as the environmental half-life for PFOS is considered to exceed the half-life criteria for persistence as defined by the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

Once PFOS is in the environment, it may enter the food chain or be further distributed at a distance from its source. PFOS has been detected in wildlife at remote sites far from sources or manufacturing facilities (Martin et al. 2004), providing evidence of the bioaccumulation potential and giving support to the persistent nature of PFOS. This also suggests that either PFOS or PFOS precursors may undergo long-range transport. POSF, a precursor and analogue to PFOS, is resistant to atmospheric hydroxyl radical attack and is considered persistent in air, with an atmospheric half-life of 3.7 years (US EPA OPPT AR226-1030a104). In water, PFOS was observed to persist for more than 285 days in microcosms under natural conditions (Boudreau et al. 2003). The OECD PFOS hazard document reviewed several biodegradation studies that indicated that no biodegradation had taken place (OECD 2002).

Examining the physical and chemical properties of PFOS to provide an indication of the environmental fate can be difficult, given the unique physical and chemical characteristics of PFOS. Due to the surface-active properties of PFOS, a log K_{ow} value cannot be determined (OECD 2002). Unlike the situation with other hydrocarbons, hydrophobic and hydrophilic interactions are not the primary partitioning mechanisms, but electrostatic interactions may be more important. It has been suggested that PFOS absorbs via chemisorption (Hekster et al. 2002). A soil adsorption/desorption study using various soil, sediment and sludge matrices found that PFOS appeared to adsorb to all matrices tested (3M Environmental Laboratory 2002). River sediment displayed the most desorption, at 39% after 48 hours, whereas sludge samples did not desorb detectable

amounts of test substance. If PFOS does bind to particulate matter in the water column, it can be expected to ultimately settle and reside in sediment.

While the vapour pressure of PFOS is similar to those of other globally distributed compounds (e.g., polychlorinated biphenyls [PCBs], dichlorodiphenyltrichloroethane [DDT]), its water solubility indicates that PFOS is less likely to partition to and be transported in air (Giesy and Kannan 2002). PFOS potassium salt has a water solubility value of 519 mg/L, which has been found to decrease significantly with increasing salt content (US EPA OPPT AR226-0620; Hekster et al. 2002; OECD 2002). Preliminary results of equilibrium partitioning modelling have predicted that PFOS partitions predominantly to the water compartment (80%), with only moderate partitioning to soils and sediments (20%) (US EPA OPPT AR226-0060; CEMC 2001). The OECD review of PFOS data suggested that any PFOS released to a water body would tend to remain in that medium, unless otherwise adsorbed onto particulate matter or taken up by organisms (OECD 2002).

Bioaccumulation

For many organic compounds, the bioaccumulation factor (BAF) may be derived from the octanol/water partition coefficient, because most organic compounds accumulate in lipids. Since perfluorinated surfactants likely elicit a different partitioning behaviour, the K_{ow} is not a suitable predictor for bioaccumulation.

The available, reliable studies on bioaccumulation show that PFOS bioaccumulates and is excreted to a very small extent. Evidence includes calculated BAFs and bioconcentration factors (BCFs), as well as the presence of PFOS in tissue and blood of wildlife in remote areas, including the Canadian Arctic, where manufacturing does not occur.

In an *in situ* bioaccumulation study following an accidental release of fire-fighting foam into Etobicoke Creek, tissue measurements were taken 6 months after the spill, and water concentrations were taken on day 153 in running water (Moody et al. 2002). Calculated BAFs in fish ranged from 6300 to 125 000 for PFOS, based on measured concentrations in common shiner (*Notropus cornutus*) liver and surface water. This BAF is high in comparison with BCF values available. Moody et al. (2002) suggested that accumulated perfluorinated derivatives are metabolized to PFOS, thus overestimating the BAF of PFOS. Nevertheless, it is evidence of the bioaccumulative nature of PFOS, and available BAFs remain substantially higher than the bioaccumulation criterion of 5000 under the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

Giesy and Kannan (2002) determined the water concentration and whole fish burdens of PFOS at Guntersville Dam in Alabama from data supplied in US EPA OPPT AR226-1030a161. From these values, BAFs ranging from 830 to 26 000 were calculated for PFOS for channel catfish (*Ictalurus punctatus*) and largemouth bass (*Micropterus salmoides*), respectively (Purdy 2002b). The difference could be due to largemouth bass

being higher in the food chain and receiving more PFOS via food (US EPA OPPT AR226-1030a161).

PFOS has been found to bioconcentrate in fish (OECD 2002). Estimated BCFs of 1100 (carcass), 5400 (liver) and 4300 (blood) have been reported for juvenile rainbow trout (*Oncorhynchus mykiss*) (Martin et al. 2003a). In fish livers collected from 23 different species in Japan, BCFs were calculated to range from 274 to 41 600 (mean 5500) (Taniyasu et al. 2003). While fish may be able to eliminate PFOS via their gills, this mode of elimination is not available to higher trophic level predators (Martin et al. 2003b), and high concentrations of PFOS have been found in the liver and blood of higher trophic level predators that consume fish (e.g., polar bears, mink and birds). Indeed, maximum levels of PFOS in liver of Canadian Arctic biota have been reported for mink (20 µg/kg), trout (50 µg/kg), seal (37 µg/kg), fox (1400 µg/kg) and polar bear (>4000 µg/kg) (Martin et al. 2003b) (see Table 3). Species differences for the elimination half-life of PFOS in biota have been shown to vary significantly: 15 days (fish), 100 days (rats) and 200 days (monkeys) (OECD 2002; Martin et al. 2003a). In addition to information on PFOS, the US Interagency Testing Committee estimated BCFs for N-EtFOSEA and N-MeFOSEA using structure–activity models to be 5543 and 26 000, respectively (Giesy and Kannan 2002).

Exposed rodents have shown preferential distribution of perfluorinated alkyl compounds in blood and liver rather than in lipids (Taniyasu et al. 2002; Martin et al. 2003b). BCFs for perfluorinated alkyl compounds have also been found to be higher in fish blood and liver than in fish carcass (Martin et al. 2003a).

Some PFOS precursors have been measured in air (Martin et al. 2002). These precursors, N-MeFOSE alcohol and N-EtFOSE alcohol, are relatively volatile, especially for such large chemicals, and they have relatively high octanol/water partition coefficients. They could be entering food chains by partitioning into biota and then undergoing degradation to PFOS somewhere along the food chain. The amount of PFOS and precursors in an animal depends on what it is eating, how much the prey metabolizes intermediates and which degradation pathways occur in the predator (Purdy 2002b).

When rats metabolize N-MeFOSE-based compounds, several metabolites have been confirmed in tissue samples, including PFOS and N-MeFOSE alcohol (3M Environmental Laboratory 2001a, 2001b). PFOS appears to be the final product of rat and probably other vertebrate metabolism of POSF-based substances.

Environmental Concentrations

Martin et al. (2002) measured the air in Toronto and Long Point for some precursors of PFOS. They found an average N-MeFOSE alcohol concentration of 101 pg/m^3 in Toronto air and 35 pg/m^3 at Long Point. The average concentrations of N-EtFOSE alcohol were 205 and 76 pg/m^3, respectively. No air concentration data for PFOS or precursors from other countries were found.

In June 2000, PFOS was detected in surface water as a result of a spill of a fire-fighting foam from the Toronto International airport into nearby Etobicoke Creek. Concentrations of PFOS ranging from <0.017 to 2210 µg/L were detected in creek water samples over a 153-day sampling period. PFOS was not detected at the upstream sample site (Moody et al. 2002). No Canadian monitoring data for PFOS were found in sediment, effluent or sludge.

US data for PFOS are available from one study of six cities. PFOS was detected in quiet water (i.e., a pond) (2.93 µg/L) and sewage treatment effluent (0.048–0.45 µg/L) and sludge (60.2–130 µg/kg dry sludge) at cities (Port St. Lucie, Florida, and Cleveland, Tennessee) with no significant fluorochemical activities (US EPA OPPT AR226-1030a111). PFOS was also detected in drinking water (0.042–0.062 µg/L), surface water (not detected [n.d.] to 0.08 µg/L), sediments (n.d.–0.78 µg/kg dry sediment), sewage treatment effluents (0.04–5.29 µg/L) and sludge (57.7–3120 µg/kg) and landfill leachate (n.d.–53.1 µg/L) of four cities that have manufacturing or industrial use of fluorochemicals. Detection limits were 0.0025 µg/L for water and 0.080 µg/kg wet weight for sediment and sludge. Sediment concentrations appear to be approximately 10-fold higher than water concentrations, indicating that there is a tendency to partition from the water to sediment.

Samples of the surface microlayer of natural water were also collected but not analyzed in the six-city US study. The reason given was that an accepted method for microlayer sample handling was not available, and consequently the evaluation and interpretation of the data were not available (US EPA OPPT AR226-1030a111). It is predicted that these samples would have shown high concentrations, because PFOS is surface active and has been found, in a limited sample set, to magnify 200-fold in the surface microlayer compared with the concentration in the underlying water (Purdy 2002b).

In a recent monitoring study near the vicinity of a fluorochemical manufacturing facility located on the Tennessee River (Alabama), PFOS was detected in all surface water and sediment samples collected. The highest concentrations for surface water (151 µg/L) and sediment (5930 µg/kg wet weight; 12 600 µg/kg dry weight) were found at a location near the point of discharge of a combined industrial effluent. However, the study found that downstream concentrations were not statistically greater than those upstream and concluded that the combined industrial effluent did not significantly affect fluorochemical (including PFOS) concentrations in the main stem of the river. For the upstream reference site (Guntersville Dam), estimated average PFOS surface water and sediment concentrations were 0.009 µg/L and 0.18 µg/kg, respectively (US EPA OPPT AR226-1030a161).

In another study, low levels of PFOS were found throughout a 130-km stretch of the Tennessee River (Hansen et al. 2002). The average PFOS concentration upstream of the fluorochemical manufacturing facility was 0.032 µg/L, suggesting an unidentified source of PFOS entering the river upstream.

Table 3 presents the levels of PFOS found in wildlife worldwide. A recent Canadian survey detected PFOS and other perfluorinated acids in fish, birds and mammals from various locations in the Canadian Arctic (Martin et al. 2004). Data are also available for a variety of species, including oysters along the Gulf of Mexico and southern Atlantic coast of the United States, fish-eating birds in Asia, Europe and North America, seals in the Caspian Sea, and polar bears and mink in North America.

Table 3. PFOS concentrations in selected wildlife.

Tissue	Species	Sampling locations	Reference[a]	PFOS (ppb)[b]	n
Liver	Chinook salmon (*Oncorhynchus tshawytscha*)	Great Lakes/inland Michigan lakes	226-1030a156	32–173	6
Liver	Lake whitefish (*Coregonus clupeaformis*)	Great Lakes/inland Michigan lakes	226-1030a156	33–81	5
Liver	Brown trout (*Salmo trutta*)	Great Lakes/inland Michigan lakes	226-1030a156	<17–26	10
Eggs	Lake whitefish (*Coregonus clupeaformis*)	Great Lakes/inland Michigan lakes	226-1030a156	145–381	2
Eggs	Brown trout (*Salmo trutta*)	Great Lakes/inland Michigan lakes	226-1030a156	49–75	3
Muscle	Carp (*Cyprinus carpio*)	Saginaw Bay, Michigan	226-1030a156	59–287	10
Muscle	Chinook salmon (*Oncorhynchus tshawytscha*)	Great Lakes/inland Michigan lakes	226-1030a156	<7–189	6
Muscle	Lake whitefish (*Coregonus clupeaformis*)	Great Lakes/inland Michigan lakes	226-1030a156	97–168	5
Liver	Striped bass (*Morone saxatilis*)	Tennessee River, Guntersville Dam	226-1030a161	385–2430	9
Liver	River otter (*Lutra canadensis*)	Washington and Oregon	226-1030a157	34–994	5
Liver	Mink (*Mustela vison*)	Midwestern United States	226-1030a157	93–4870	30
Liver	Mink (*Mustela vison*)	Massachusetts	226-1030a157	87–4300	31
Liver	Mink (*Mustela vison*)	Louisiana	226-1030a157	40–318	7
Liver	Mink (*Mustela vison*)	South Carolina	226-1030a157	65–3110	9
Liver	Northern fur seal (*Callorhinus ursinus*)	Pribilof Islands	226-1030a160	<10–122	13
Liver	Ringed seal (*Phoca hispida*)	Canadian Arctic	Martin et al.*	8.6–23	9
Liver	Ringed seal (*Phoca hispida*)	Canadian Arctic	Martin et al.*	10–37	10
Liver	Mink (*Mustela vison*)	Canadian Arctic	Martin et al.*	1.3–20	10
Liver	Common loon (*Gavia immer*)	Canadian Arctic	Martin et al.*	11–26	5
Liver	Northern fulmar (*Fulmarus glacialis*)	Canadian Arctic	Martin et al.*	1–1.5	5

Tissue	Species	Sampling locations	Reference[a]	PFOS (ppb)[b]	n
Liver	Black guillemot (*Cepphus grylle*)	Canadian Arctic	Martin et al.*	n.d.	5
Liver	White sucker (*Catostomus commersoni*)	Canadian Arctic	Martin et al.*	6.5–8.6	3
Liver	Brook trout (*Salvelinus fontinalis*)	Canadian Arctic	Martin et al.*	29–50	2
Liver	Lake whitefish (*Coregonus clupeaformis*)	Canadian Arctic	Martin et al.*	12	2
Liver	Lake trout (*Salvelinus namaycush*)	Canadian Arctic	Martin et al.*	31	1
Liver	Northern pike (*Esox lucius*)	Canadian Arctic	Martin et al.*	5.7	1
Liver	Arctic sculpin (*Myoxocephalus scorpioides*)	Canadian Arctic	Martin et al.*	12	1
Liver	Arctic fox (*Alopex lagopus*)	Canadian Arctic	Martin et al.*	6.1–1400	10
Liver	Polar bear (*Ursus maritimus*)	Canadian Arctic	Martin et al.*	1700–>4000	7
Liver	Polar bear (*Ursus maritimus*)	Barrow and other sites in Alaska	226-1030a160	175–678	17
Blood	Polar bear (*Ursus maritimus*)	Barrow and other sites in Alaska	226-1030a160	26–52	14
Blood	Grey seal (*Halichoerus grypus*)	Sable Island, Canada	226-1030a160	<13–49	12
Blood	Grey seal (*Halichoerus grypus*)	Baltic Sea	226-1030a160	14–76	16
Blood	Ringed seal (*Phoca hispida*)	Baffin Island, Canada	226-1030a160	<3.13–12	16
Blood	Double-crested cormorant (*Phalacrocorax auritus*)	Great Lakes	226-1030a159	34–243	8
Eggs	Double-crested cormorant (*Phalacrocorax auritus*)	Great Lakes	226-1030a159	21–220	4
Plasma	Bald eagle (*Haliaeetus leucocephalus*)	Michigan, Wisconsin and Minnesota	226-1030a159	<1–2220	33
Liver	Laysan albatross (*Diomedea immutabilis*)	Midway Atoll	Giesy*	<35	n.r.[c]
Liver	Common loon (*Gavia immer*)	North Carolina	Giesy*	290	n.r.
Liver	Brown pelican (*Pelecanus occidentalis*)	Mississippi	Giesy*	460	n.r.
Liver	Common cormorant (*Phalacrocorax carbo*)	Italy	Giesy*	96	n.r.
Liver	Black-tailed gull (*Larus crassirostris*)	Korea	Giesy*	170	n.r.

Tissue	Species	Sampling locations	Reference[a]	PFOS (ppb)[b]	n
Liver	Black-tailed gull (*Larus crassirostris*)	Tokyo (Haneda Airport), Japan	Kannan et al.*	230	1
Liver	Black-eared kite (*Milvus lineatus*)	Tokyo (Haneda Airport), Japan	Kannan et al.*	450	1
Liver	Common cormorant (*Phalacrocorax carbo*)	Sagami River, Japan	Kannan et al.*	170–650	8

[a] References: US EPA OPPT AR226-1030a156, AR226-1030a157, AR226-1030a158, AR226-1030a159, AR226-1030a160 as summarized by Giesy and Kannan (2002); except entries denoted by *, which are from Martin et al. (2004), Kannan et al. (2002) and Giesy (2003).
[b] Units are parts per billion (ppb) = µg/kg for tissue; µg/L for fluids.
[c] n.r. = not reported.

The highest tissue concentration in Table 3 is 4870 µg/kg in mink liver from the Midwestern United States. In Canada, the highest PFOS concentration was found in polar bear liver (maximum = >4000 µg/kg, mean = 3100 µg/kg; n = 7) (Martin et al. 2004). The PFOS concentrations in polar bear liver were higher than any other previously reported concentrations of persistent organochlorine chemicals (e.g., PCBs, chlordane, hexachlorocyclohexane) in polar bear fat. A general data trend indicated that mammals feeding at higher trophic levels had higher PFOS concentrations than those feeding at lower trophic levels. Elsewhere, PFOS concentrations in plaice (*Pleuronectes platessa*) liver (7760 µg/kg) from the Western Scheldt estuary (southwestern Netherlands) and ornate jobfish (*Pristipomoides argyrogrammicus*) liver (7900 µg/kg) from Kin Bay (Japan) are among the highest PFOS concentrations ever reported in wildlife (fish) (Hoff et al. 2003; Taniyasu et al. 2003). Factors helping to explain such high concentrations may be the proximity of a PFOS manufacturing plant (upstream of estuary) and an army base (Kin Bay, Japan) that could be using PFOS in fire-fighting operations.

Effects

The toxicity of PFOS has been studied in a variety of aquatic and terrestrial species, including aquatic plants, invertebrates and vertebrates and terrestrial invertebrates, birds and mammals. Adverse effects range from growth inhibition, histopathological effects, atrophied thymus, change in species diversity in a microcosm and mortality. Toxicity data are essentially limited to PFOS. The following is a summary of the key studies used to identify the Critical Toxicity Value (CTV) for PFOS. A more complete review of effects is given in the OECD hazard review of PFOS, which discusses effects on fish, invertebrates, aquatic plants (algae and higher plants), amphibians and microorganisms (OECD 2002). Additional studies by Boudreau et al. (2003) and Sanderson et al. (2002) not available in OECD (2002) are also summarized.

The most sensitive endpoint in aquatic organisms occurred in a flow-through bioconcentration study with bluegill (*Lepomis macrochirus*) using PFOS potassium salt. No significant mortality was seen at an exposure concentration of 0.086 mg/L over a 62-day uptake phase; however, significant mortality was observed after a 35-day exposure to 0.87 mg/L. The study was stopped because all the fish either had died or had been

sampled (US EPA OPPT AR226-1030a042). The No-Observed-Effect Concentration (NOEC) of 0.086 mg/L is the lowest no-adverse-effect concentration for aquatic organisms and was therefore selected as the CTV for aquatic organisms.

Results have been published from a laboratory evaluation of the toxicity of PFOS to five aquatic organisms: green algae (*S. capricornutum* and *C. vulgaris*), duckweed (*L. gibba*) and water flea (*D. magna* and *D. pulicaria*) (Boudreau et al. 2002). NOEC values were generated from the most sensitive endpoints for all organisms. Based on effect (immobility) values, the most sensitive of the organisms in this study was *D. magna*, with a 48-hour immobility NOEC of 0.8 mg/L; the accompanying LC_{50} was 112 mg/L, and the 48-hour IC_{50} for growth inhibition was 130 mg/L. The 21-day NOEC for lethality for *D. magna* was 5.3 mg/L. Autotroph inhibition of growth NOEC values were 5.3 mg/L, 6.6 mg/L and 8.2 mg/L for *S. capricornutum*, *L. gibba* and *C. vulgaris*, respectively.

In an aquatic microcosm study (Boudreau et al. 2003), a field evaluation assessed the toxicological risk associated with PFOS across levels of biological organization. The zooplankton community was significantly affected by the treatment for all sampling times. A community-level NOEC of 3.0 mg/L was determined for the 35-day study. The most sensitive taxonomic groups, Cladocera and Copepoda, were virtually eliminated in the 30 mg/L treatments after 7 days, although specific survival rates were not quantified.

In a laboratory microcosm study that examined impacts to zooplankton following exposure to PFOS, adverse effects were observed at 10 mg/L over 14 days; several species were significantly reduced or eliminated (Sanderson et al. 2002). In comparison with controls, exposures of 10 mg/L and 30 mg/L resulted in an average 70% change in species diversity and total zooplankton. The most sensitive species in the study was *Cyclops diaptomus.* The statistically significant effect concentrations for all species endpoints (abundance) were above 1 mg/L.

A fathead minnow (*Pimephales promelas*) embryo-juvenile flow-through chronic study determined a NOEC of 0.3 mg/L over a 42-day exposure period. This value was for both survival and growth (US EPA OPPT AR226-0097). The slightly higher NOEC may be due to the shorter exposure time. In acute tests, the lowest 96-hour LC_{50} for freshwater fish species was 4.7 mg/L for the fathead minnow (*P. promelas*); in salt water, a 96-hour LC_{50} of 13.7 mg/L was reported for rainbow trout (*O. mykiss*) (OECD 2002). In a 96-hour static acute study using the freshwater mussel, *Unio complamatus*, the NOEC for mortality was 20 mg/L and the LC_{50} was 59 mg/L (US EPA OPPT AR226-0091, AR226-1030a047). The most sensitive saltwater invertebrate studied was the saltwater mysid, *Mysidopsis bahia*. Survival, growth and reproduction were assessed over an exposure period of 35 days. The NOECs determined for growth and reproduction were both 0.25 mg/L (US EPA OPPT AR226-0101). In acute toxicity testing, a 96-hour LC_{50} of 3.6 mg/L was reported for the mysid shrimp (OECD 2002). There was one study reported for embryo teratogenesis in aquatic organisms, which involved a 96-hour static renewal study on the frog, *Xenopus laevis* (US EPA OPPT AR226-1030a057). The minimum concentration that inhibited growth was 7.97 mg/L. The LC_{50} for mortality was 13.8 mg/L, the EC_{50} for malformed embryos was 12.1 mg/L and the NOEC for embryo

malformation was 5.2 mg/L. Calculated teratogenic indices ranged from 0.9 to 1.1, indicating that PFOS has a low potential to be a developmental hazard in this species.

PFOS is toxic to birds. Acute dietary studies were conducted on mallard (*Anas platyrhynchos*) and northern bobwhite (*Colinus virginianus*) (US EPA OPPT AR226-1030a049). Birds were fed PFOS in the diet for 5 days. Mortality, body weight and food consumption were monitored throughout. PFOS levels were quantified in sera and liver of mallards and bobwhites sacrificed on days 8 and 22 post-exposure, as well as from some animals that died before the scheduled sampling times. Mallard was the more sensitive of the two species tested, and the most sensitive endpoint was the 8-day Lowest-Observed-Effect Concentration (LOEC) for reduced body weight gain, at 29.7 mg PFOS/kg liver wet weight. This value is the CTV for birds (NOEC = 15.3 mg PFOS/kg liver wet weight). For northern bobwhite, the mean LOEC in the day 8 group was 70.3 mg/kg liver wet weight, and the NOEC was 45.2 mg/kg liver wet weight.

As no wild mammal studies were found, laboratory mammal studies were used as surrogates for wild mammals. The CTV for mammal (liver) and bird (serum) was selected from a 2-year dietary rat study in which histopathological effects in the liver were seen in males and females at intakes as low as 0.06–0.23 mg PFOS/kg bw per day and 0.07–0.21 mg PFOS/kg bw per day, respectively (Covance Laboratories, Inc. 2002). Average values were determined for males and females, to establish Lowest-Observed-Effect Levels (LOELs) of 40.8 mg/kg in liver and 13.9 mg/L in serum.

Supporting evidence for a NOEC in the low mg/kg or mg/L range in liver and sera includes results from a two-generation rat study (US EPA OPPT AR226-0569). In this study, the NOECs were determined to be 0.1 mg/kg bw per day dosed via gavage, 5.3 mg/L in sera and 14.4 mg/kg in liver. The LOECs were 0.4 mg/kg bw per day, 19 mg/L sera and 58 mg/kg liver. The effect was reduction in dam body mass (US EPA OPPT AR226-0569).

Additional studies in primates are summarized in the screening assessment report for PFOS and its precursors prepared by Health Canada (2004).

The OECD review summarizes data indicating moderate to high toxicity of PFOS to honey bees (*Apis mellifera*). In an acute oral test, a 72-hour LD_{50} for ingestion of PFOS was 0.40 µg/bee, and a 72-hour No-Observed-Effect Level (NOEL) was 0.21 µg/bee. A contact test found a 96-hour LD_{50} of 4.78 µg/bee and a 96-hour NOEL of 1.93 µg/bee.

Results have been reported for an acute toxicity study with the earthworm in an artificial soil substrate (US EPA OPPT AR226-1106). The PFOS potassium salt 14-day LC_{50} was determined to be 373 mg/kg bw, with a 95% confidence interval of 316–440 mg/kg bw. The 14-day NOEC for burrowing behaviour, body weight and clinical signs of toxicity was 77 mg/kg bw, and the 14-day LOEC for the same endpoints was 141 mg/kg bw.

Body mass reduction or poor food efficiency was seen in most toxicity studies and species (Haughom and Spydevold 1992; Campbell et al. 1993a, 1993b; US EPA OPPT

AR226-0137, AR226-0139, AR226-0144, AR226-0949, AR226-0953, AR226-0956, AR226-0957, AR226-0958, AR226-0967). This is consistent with the mechanism of toxicity being the uncoupling of oxidative phosphorylation (US EPA OPPT AR226-0167, AR226-0169, AR226-0240). This mode of action, however, is not known with certainty to explain PFOS toxicity. There are other mechanisms that can be hypothesized. A study with rats (Luebker et al. 2002) tested the hypothesis that PFOS, PFOA and other perfluorinated chemicals can interfere with the binding affinity and capacity of liver binding proteins for fatty acids; the results revealed that the most potent competitor is PFOS. A study with common carp (*Cyprinus carpio*) by Hoff et al. (2003) has suggested that PFOS induces inflammation-independent enzyme leakage through liver cell membranes that might be related to cell necrosis. It was also suggested that PFOS might interfere with homeostasis of DNA metabolism.

Proposed Conclusion for the Environment

The approach taken in this environmental screening assessment was to examine various supporting information and develop conclusions based on a weight of evidence approach as required under Section 76.1 of CEPA 1999. Particular consideration was given to risk quotient analyses and persistence, bioaccumulation and presence in the Canadian Arctic.

PFOS and its precursors are part of a larger chemical class of fluorochemicals typically referred to as perfluorinated alkyl compounds. This screening assessment of PFOS and its precursors defines PFOS precursors as substances containing the perfluorooctylsulfonyl ($C_8F_{17}SO_2$) or $C_8F_{17}SO_3$ moiety that have the potential to transform or degrade to PFOS. It includes, but is not limited to, PFOS and some 50 substances and precursors identified in Appendix 1. While the assessment did not consider additive effects of PFOS and all its precursors, it is recognized that precursors contribute to the ultimate loadings of PFOS. Precursors may also play a key role in the long-range transport of PFOS to remote areas.

Risk Quotient Analysis

Risk quotient analyses, integrating known or potential exposures with known or potential adverse environmental effects, were performed for PFOS (see Table 4). An analysis of exposure pathways and subsequent identification of sensitive receptors were used to select environmental assessment endpoints (e.g., adverse reproductive effects on sensitive fish species in a community). For each endpoint, a conservative Estimated Exposure Value (EEV) was selected based on empirical data from monitoring studies. Data from the Canadian and North American environment were used preferentially for EEVs. EEVs usually represented worst-case scenarios, as an indication of the potential for these substances to reach concentrations of concern and to identify areas where those concerns would be most likely.

PFOS has been detected throughout the world, including in areas distant from sources. While it is recognized that limited sampling of North American surface waters has indicated that concentrations of PFOS are low (ng/L to µg/L) and may not represent an immediate concern for acute toxicity, the use of non-Canadian quiet water data to

calculate risk quotients was considered reasonable given the concern for the possible long-term impact of this substance. PFOS has been found at high levels in certain Canadian wildlife (e.g., polar bears) and globally (e.g., fish in Japan and the Netherlands). In the environment, higher concentrations of this persistent substance have been detected in sediments near industrial effluents.

The highest measured ambient or "background" PFOS concentration in water in the United States was used as a surrogate for Canadian data, after excluding potential outliers, as there were few data available for water in Canada. Although concentrations measured near manufacturing or processing plants are known to be higher, these concentrations were not considered to be reflective of the Canadian situation and were not used in calculating risk quotients. The highest measured ambient concentration exceeded the effects threshold, indicating a potential for effects on aquatic biota, birds and mammals.

Maximum concentrations in liver of wildlife in remote areas of the Canadian Arctic include the following: mink (20 µg/kg), common loon (26 µg/kg), ringed seal (37 µg/kg), brook trout (50 µg/kg), Arctic fox (1400 µg/kg) and polar bear (>4000 µg/kg) (Martin et al. 2003b). Concentrations in liver of higher trophic level biota appear to be higher than those found in lower trophic level biota.

An Estimated No-Effects Value (ENEV) was determined by dividing a CTV by an application factor. CTVs typically represented the lowest ecotoxicity value from an available and acceptable data set. Preference was generally given for chronic toxicity data, as long-term exposure was a concern. Where these data were not available, acute toxicity data were used.

The toxicity of PFOS has been studied in a variety of aquatic and terrestrial species, including aquatic plants, invertebrates and vertebrates and terrestrial invertebrates, birds and mammals. Adverse effects range from growth inhibition, histopathological effects, atrophied thymus, disruption of reproductive cycle, change in species diversity in a microcosm and mortality. The most sensitive endpoint for aquatic species was the mortality of bluegill (*Lepomis macrochirus*) after a 35-day exposure to PFOS potassium salt (LOEC 0.87 mg/L; NOEC 0.086 mg/L). Adverse histopathological effects were observed in rats exposed to PFOS in a 2-year laboratory study (LOELs were established at 40.8 µg/g in liver and 13.9 mg/L serum). Reduced body weight gain was observed in mallard (*Anas platyrhynchos*) exposed to PFOS in dietary studies, with the LOEC at 29.7 mg/kg liver. Given available information that PFOS appears to distribute preferentially in liver and blood (Taniyasu et al. 2002; Martin et al. 2003b), risk quotients in these tissues were developed for birds and terrestrial mammals.

Application factors were derived using a multiplicative approach, which uses 10-fold factors to account for various sources of uncertainty associated with making extrapolations and inferences related to the following: intra- and interspecies variations; differentially sensitive biological endpoints; laboratory to field impact extrapolation, required to extrapolate from single-species tests to ecosystems; and potential effects from

concurrent presence of other substances. For substances that meet persistence and bioaccumulation criteria as outlined in the CEPA 1999 Persistence and Bioaccumulation Regulations (Government of Canada 2000), an additional application factor of 10 is applied to the CTV.

Risk quotients derived for PFOS and its precursors are summarized in Table 4. Exposure data used as EEVs are found in Table 4. Toxicity data used to determine CTVs are summarized in the section Fate, Exposure and Effects.

The risk quotient analysis indicates that the greatest potential risk from PFOS in the environment occurs in higher trophic level mammals (risk quotient >98) and fish-eating birds (risk quotient 21.9 in liver; 160 in serum); there is also some level of risk for fish (risk quotient 3.4).

While certain data gaps and uncertainties exist, there is nonetheless a substantial body of information on PFOS and its precursors. For example, while the mechanism of transport of PFOS and its precursors to the Arctic is not clear, they appear to be mobile in some form, as PFOS has been measured in biota in the Canadian Arctic far from known anthropogenic sources. Environmental pathways of PFOS to biota are not well understood because information on degradation is lacking, as are monitoring data on concentrations of various precursors in air, water, effluents and sediment in Canada. Concentrations of PFOS and its precursors in the surface water microlayer are unknown; however, given the physical properties of PFOS and its precursors, these may be considerably higher than concentrations in the water column as a whole. While mechanisms of toxic action of PFOS are not well understood, a range of toxicological effects have been reported in a variety of species. Finally, while toxicological studies have focused on the effect of PFOS itself, data on potential impacts of combined exposure to PFOS and its different precursors are limited or unknown.

Persistence

PFOS is resistant to hydrolysis, photolysis, microbial degradation and metabolism by vertebrates and is persistent as defined in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

The weight of evidence, given information on PFOS persistence, degradation of precursors to PFOS, volatilization and atmospheric transport, indicates that while PFOS has little potential to move in the environment, the precursors that will degrade to PFOS have the potential to do so, which may explain the high levels reported in the Arctic. Once the precursors degrade to PFOS, they are expected to persist indefinitely in the environment. The precursor POSF is persistent in air, with an atmospheric half-life of 3.7 years (US EPA OPPT AR226-1030a104). In water, PFOS persisted over 285 days in microcosms under natural conditions (Boudreau et al. 2003). While the vapour pressure of PFOS is similar to those of other globally distributed compounds (e.g., PCBs, DDT), its water solubility indicates that PFOS itself is less likely to partition to and be transported in air (Giesy and Kannan 2002). Although PFOS itself has low volatility,

several PFOS precursors are considered volatile, including N-EtFOSE alcohol, N-MeFOSE alcohol, N-MeFOSA and N-EtFOSA (US EPA OPPT AR226-0620). When present in residuals in products, these PFOS precursors could evaporate into the atmosphere when the products containing them are sprayed and dried (US EPA OPPT AR226-0620). There is therefore potential for atmospheric transport of PFOS precursors. However, further data are required to accurately characterize this potential.

The available information suggests that the potential contribution of PFOS to stratospheric ozone depletion and to ground-level ozone formation is negligible, and its potential contribution to global warming is not known.

PFOS is present in biota, notably in vertebrates, throughout the world, including in a range of fish, birds and mammals in remote sites, including the Canadian Arctic, far from sources or manufacturing facilities of PFOS and its precursors. This indicates that PFOS is persistent in the environment and that its precursors may undergo long-range transport.

Bioaccumulation

PFOS has high potential for bioaccumulation, and the weight of evidence for bioaccumulation includes estimates for BAFs and BCFs that exceed the bioaccumulation criteria in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000). BAFs based on measured concentrations in biota in Canada, notably the Arctic, and in the United States and Japan range from 830 to 125 000. BCF values for fish range from 274 to 41 600. While fish may be able to eliminate PFOS via their gills, this mode of elimination is not available to higher trophic level predators (e.g., polar bear, mink and eagles) that consume fish. In addition to information on PFOS, estimated BCFs for N-EtFOSEA and N-MeFOSEA using structure–activity models were 5543 and 26 000, respectively.

CEPA 1999 recognizes the particular concerns associated with persistent and bioaccumulative substances. As indicated in the federal Toxic Substances Management Policy, “persistence and bioaccumulation can be used as qualitative surrogates for long-term exposure of environmental biota.”

Given the inherent properties of PFOS and its precursors, together with demonstrated or potential environmental concentrations that may exceed the effect levels for higher trophic level biota such as fish and fish-eating birds and mammals; given the widespread occurrence of PFOS in biota, including in remote areas; and given that PFOS precursors may contribute to the overall presence of PFOS in the environment, **it is therefore concluded that PFOS, its salts and its precursors are entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity and thus satisfy the definition of “toxic” under Paragraph 64(a) of CEPA 1999. PFOS and its salts meet the criteria for persistence and bioaccumulation as defined in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000). Based on available data for PFOS, its salts and its precursors, their**

presence in the environment results primarily from human activity. PFOS, its salts and its precursors are not naturally occurring radionuclides or naturally occurring inorganic substances.

Proposed Recommendation to the Ministers of Environment and Health

It is proposed that PFOS, its salts and its precursors be considered "toxic" as defined in Section 64 of CEPA 1999.

It is proposed that consideration be given to the implementation of virtual elimination of PFOS and its salts under subsection 65(3) of CEPA 1999.

References

3M Environmental Laboratory. 2001a. Analytical laboratory report, determination of the presence and concentration of PFOS, PFOSA, PFOSAA, EtFOSE-OH, M556 and PFOSEA in serum and liver samples of Crl:CD(SD) IGS BR rats exposed to N-ethyl perfluorooctanesulfonamido ethanol. 3M Environmental Laboratory Report No. Tox-001, Laboratory Request No. U2103, 3M Reference No. T-6316.1 (on the 5th CD 3M provided to US Environmental Protection Agency for Administrative Record 226).

3M Environmental Laboratory. 2001b. Analytical laboratory report, determination of the presence and concentration of PFOS, PFOSA, PFOSAA, EtFOSE-OH, M556 and PFOSEA in serum and liver samples of Crl:CD(SD) IGS BR rats exposed to N-ethyl perfluorooctanesulfonamido ethanol. 3M Environmental Laboratory Report No. Tox-002, Laboratory Request No. U2104, 3M Reference No. T-6316.1 (on the 5th CD 3M provided to US Environmental Protection Agency for Administrative Record 226).

3M Environmental Laboratory. 2002. Soil adsorption revised. June 12, 2002, revision to 3M Technical Report "Soil Adsorption/Desorption Study of Potassium Perfluorooctanesulfonate (PFOS)." Project No. E00-1311, final report completion date June 4, 2001. Amended report completion date May 24, 2002. (submitted to US EPA AR-226-1107.)

Boudreau, T.M., P.K. Sibley, D.C.G. Muir, S.A. Mabury and K.R. Solomon. 2002. Laboratory evaluation of the toxicity of perfluorooctane sulfonate (PFOS) on *Selenastrum capricornutum*, *Chlorella vulgaris*, *Lemna gibba*, *Daphnia magna*, and *Daphnia pulicaria*. Arch. Environ. Contam. Toxicol. 44: 307–313.

Boudreau, T.M., C.J. Wilson, W.J. Cheong, P.K. Sibley, S.A. Mabury, D.C.G. Muir and K.R. Solomon. 2003. Response of the zooplankton community and environmental fate of perfluorooctane sulfonic acid in aquatic microcosms. Environ. Toxicol. Chem. 22: 2739–2745.

Campbell, S.M., S.P. Lynn and J.B. Beavers. 1993a. Lithium perfluorooctane sulfonate (6861D11). A dietary LC50 study with the northern bobwhite. Wildlife International Ltd. Project No. 319-101. Sponsored by S.C. Johnson & Son, Inc.

Campbell, S.M., S.P. Lynn and J.B. Beavers. 1993b. Lithium perfluorooctane sulfonate (6861D11). A dietary LC50 study with the mallard. Wildlife International Ltd. Project No. 319-102. Sponsored by S.C. Johnson & Son, Inc.

CEMC (Canadian Environmental Modelling Centre). 2001. Trent University Canadian Environmental Modelling Centre Newsletter, Summer 2001 Progress Report, Peterborough, Ontario (http://www.trentu.ca/cemc/news01_07.pdf).

CITI (Chemicals Inspection & Testing Institute). 1992. Biodegradation and bioaccumulation: data of existing chemicals based on the CSCL Japan. Compiled under the supervision of Chemical Products Safety Division, Basic Industries Bureau, Ministry of International Trade & Industry, Tokyo, Japan. October 1992.

Compass Environmental Inc. 1999. Municipal solid waste incineration in Canada: An update on operations 1997–1998. Prepared for Environment Canada and Federal Panel on Energy Research Development, Burlington, Ontario.

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. Study No. 6239-183, Madison, Wisconsin.

Dimitrov, S., V. Kamenska, J.D. Walker, W. Windle, R. Purdy, M. Lewis and O. Mekenyan. 2004. Predicting the biodegradation products of perfluorinated chemicals using CATABOL, SAR and QSAR. Environ. Res. 15(1): 69–82.

Environment Canada. 2000. Industry survey of perfluoroalkyl substances as conducted through a *Canada Gazette* Notice issued under the authority of Section 71 of CEPA 1999.

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.

Giesy, J. 2003. Personal communication (comments on draft PFOS assessment report), February 2003, to Existing Substances Branch, Environment Canada, Gatineau, Quebec. Department of Zoology, National Food Safety and Toxicology Centre, Institute for Environmental Toxicology, Michigan State University, East Lansing, Michigan.

Giesy, J.P. and K. Kannan. 2002. Perfluorochemical surfactants in the environment. Environ. Sci. Technol. 36(7): 147A–152A.

Government of Canada. 2000. Persistence and Bioaccumulation Regulations. *Canada Gazette*, Part II, Vol. 134, No. 7, March 29, 2000.

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. Environ. Sci. Technol. 36: 1681–1685.

Haughom, B. and O. Spydevold. 1992. The mechanism underlying the hypolipemic effect of perfluorooctanoic acid (PFOA), perfluorooctane sulphonic acid (PFOSA) and clofibric acid. Biochim. Biophys. Acta 1128: 65–72.

Health Canada. 2004. Perfluorooctane sulfonate, its salts and its precursors that contain the $C_8F_{17}SO_2$ or $C_8F_{17}SO_3$ moiety. Draft screening health assessment report. Prepared by Existing Substances Division, Health Canada, Ottawa, Ontario.

Hekster, F.M., P. de Voogt, A.M.C.M. Pijinenburg and R.W.P.M. Laane. 2002. Perfluoroalkylated substances — aquatic environmental assessment. Report RIKZ/2002.043. Prepared at the University of Amsterdam and RIKZ (The State Institute for Coast and Sea), July 1, 2002. 99 pp.

Hoff, P.T., K. Van De Vijver, W. Van Dongen, E.L. Esmans, R. Blust and W.M. De Coen. 2003. Perfluorooctane sulfonic acid in bib (*Trisoperus luscus*) and plaice (*Pleuronectes platessa*) from the Western Scheldt and the Belgian North Sea: distribution and biochemical effects. Environ. Toxicol. Chem. 22: 608–614.

Industry Canada. 2002. Canadian industry statistics: Establishments: Carpet and Rug Mills (NAICS 31411). Information obtained on-line on December 4, 2002 (http://strategis.ic.gc.ca/canadian_industry_statistics/cis.nsf/idE/cis31411estE.html).

Kannan, K., J.-W. Choi, N. Iseki, K. Senthikumar, D.H. Kim, S. Masunaga and J.P. Giesy. 2002. Concentrations of perfluorinated acids in livers of birds from Japan and Korea. Chemosphere 49: 225–231.

Key, B.D., R.D. Howell and C.S. Criddle. 1997. Fluorinated organics in the biosphere. Environ. Sci. Technol. 31: 2445–2454.

Luebker, D.J., K.J. Hansen, M. Bass, J.L. Butenhoff and A.M. Seacat. 2002. Interactions of fluorochemicals with rat liver fatty acid-binding protein. Toxicology 176: 175–185.

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. 2003a. Bioconcentration and tissue distribution of perfluorinated acids in rainbow trout (*Oncorhynchus mykiss*). Environ. Toxicol. Chem. 22: 196–204.

Martin, J.W., S.A. Mabury, K.R. Solomon and D.C.G. Muir. 2003b. Dietary accumulation of perfluorinated acids in juvenile rainbow trout (*Oncorhynchus mykiss*). Environ. Toxicol. Chem. 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. Environ. Sci. Technol. 38(2): 373–380.

Mekenyan, O., S. Dimitrov and S. Temelkov. 2002. PFOS metabolic pathways and metabolic distributions: Generated by catabolic simulator (2001–2002). Results compiled and edited by P. Robinson, Existing Substances Branch, Environment Canada, Gatineau, Quebec.

Moody, C.A., J.W. Martin, W.C. Kwan, D.C.G. Muir and S.A. Mabury. 2002. Monitoring perfluorinated surfactants in biota and surface water samples following an accidental release of fire-fighting foam into Etobicoke Creek. Environ. Sci. Technol. 36: 545–551.

OECD (Organisation for Economic Co-operation and Development). 2002. Hazard assessment of perfluorooctane sulfonate (PFOS) and its salts. ENV/JM/RD(2002)17/FINAL, November 21, Paris. 362 pp.

Purdy, R. 2002a. Personal communication (by e-mail), October 9, 2002, to Existing Substances Branch, Environment Canada, Gatineau, Quebec.

Purdy, R. 2002b. Personal communication, May 2002, to Existing Substances Branch, Environment Canada, Gatineau, Quebec.

Robinson, P. 2002. CATABOL: Microbial catabolic pathways model. A summary of the biodegradation and metabolic pathways estimation program developed by Prof. Ovanes Mekenyan et al. Background documentation, Existing Substances Branch, Environment Canada, Gatineau, Quebec.

Sanderson, H., T.M. Boudreau, S.A. Mabury, W. Cheong and K.R. Solomon. 2002. Ecological impact and environmental fate of perfluorooctane sulfonate on the zooplankton community in indoor microcosms. Environ. Toxicol. Chem. 21: 1490–1496.

Taniyasu, S., K. Kannan, Y. Horii and N. Yamashita. 2002. Other halogenated POPs of concern. The first environmental survey of perfluorooctane sulfonate (PFOS) and related compounds in Japan. Organohalogen Compd. 59: 311–314.

Taniyasu, S., K. Kannan, Y. Horii, N. Hanari and N. Yamashita. 2003. A survey of perfluorooctane sulfonate and related perfluorinated organic compounds in water, fish, birds, and humans from Japan. Environ. Sci. Technol. 37: 2634–2639.

US EPA. 2002. Perfluoroalkyl sulfonates; Significant new use rule (SNUR); Final rule and supplemental proposed rule. Fed. Regist. 67(47). 40 CFR Part 721, March 11, 2002.

US EPA OPPT AR226-0060. 3M submission (not dated). Data summaries, completed 1999. Transport between environmental compartments (fugacity): perfluorooctanesulfonate.

US EPA OPPT AR226-0091. 3M submission dated 4/26/00. PFOS: a 96-hour static acute toxicity test with the freshwater mussel (*Unio complamatus*), with protocol.

US EPA OPPT AR226-0097. 3M submission dated 4/26/00. PFOS: an early life-stage toxicity test with the fathead minnow (*Pimephales promelas*), with protocol.

US EPA OPPT AR226-0101. 3M submission dated 4/26/00. PFOS: a flow-through life cycle toxicity test with the saltwater mysid (*Mysidopsis bahia*), with protocol.

US EPA OPPT AR226-0137. 3M submission dated 12/18/78. Ninety-day subacute rhesus monkey toxicity study, with Fluorad fluorochemical surfactant FC95.

US EPA OPPT AR226-0139. 3M submission dated 11/10/78. Ninety-day subacute rat toxicity study, with Fluorad fluorochemical surfactant FC-95.

US EPA OPPT AR226-0144. 3M submission not dated. 4-week capsule toxicity study with perfluorooctane sulfonic acid potassium salt (PFOS; T-6295) in cynomolgus monkeys (includes draft final report, cell proliferation report, protocol and memorandum from Marvin Case re histopathology review of liver tissue).

US EPA OPPT AR226-0167. 3M submission dated 2/4/98. The effect of perfluorinated arylalkylsulfonamides on bioenergetics of rat liver mitochondria.

US EPA OPPT AR226-0169. 3M submission not dated. Summary of the effects of PFC's on mitochondrial bioenergetics *in vitro*.

US EPA OPPT AR226-0184. 3M submission dated 4/6/00. Attachment to letter to C. Auer dated May 4, 2000. Ongoing studies on perfluorooctanesulfonates (sic): abiotic degradation studies of perfluorooctane sulfonate.

US EPA OPPT AR226-0240. 3M submission not dated. Mechanism of toxicity of a unique pesticide: N-ethylperfluorooctane sulfonamide (NEPFOS), and its metabolite perfluorooctane sulfonamide (PFOS) to isolated rabbit renal cortical mitochondria (RCM), abstract from 1989 Society of Toxicology meeting.

US EPA OPPT AR226-0547. 3M submission dated 5/2/99. The science of organic fluorochemistry.

US EPA OPPT AR226-0550. 3M submission dated 5/26/99. Fluorochemical use, distribution and release overview.

US EPA OPPT AR226-0569. 3M submission dated 6/10/99. Summary PFOS rat two-generation reproduction study.

US EPA OPPT AR226-0588. 3M submission dated 06/16/00. Phase-out plan for POSF-based products.

US EPA OPPT AR226-0620. 3M submission dated 3/1/00. Sulfonated perfluorochemicals in the environment: sources, dispersion, fate and effects.

US EPA OPPT AR226-0949. 3M submission dated 1/11/99. Final report — protocol 418-012: oral (stomach tube) developmental toxicity of PFOS in rabbits.

US EPA OPPT AR226-0953. 3M submission dated 4/26/00. PFOS: a dietary LC50 study with the northern bobwhite, with protocol.

US EPA OPPT AR226-0956. 3M submission not dated. Summary report: 104-week dietary chronic study and carcinogenicity study with perfluorooctane sulfonic acid potassium salt (PFOS: T-6295) in rats — week 53.

US EPA OPPT AR226-0957. 3M submission dated 4/12/00. Draft final report: 26-week capsule toxicity study with perfluorooctane sulfonic acid potassium salt (PFOS: T-6295) in cynomolgus monkeys, volume I.

US EPA OPPT AR226-0958. 3M submission dated 4/12/00. Draft final report: 26-week capsule toxicity study with perfluorooctane sulfonic acid potassium salt (PFOS: T-6295) in cynomolgus monkeys, volume II.

US EPA OPPT AR226-0967. 3M submission dated 1/11/99. Final report: protocol 418-010 — oral (stomach tube) developmental toxicity study of N-EtFOSE in rabbits.

US EPA OPPT AR226-1030a041. 3M submission dated 4/23/01. The 35-day aerobic biodegradation study of PFOS.

US EPA OPPT AR226-1030a042. 3M submission dated 6/21/01, revised 7/16/02. Perfluorooctanesulfonate, potassium salt (PFOS): a flow-through bioconcentration test with the bluegill (*Lepomis macrochirus*).

US EPA OPPT AR226-1030a047. 3M submission dated 6/9/00. Analysis of PFOS in test organisms from the 96-hour static acute toxicity test with the freshwater mussel.

US EPA OPPT AR226-1030a049. 3M submission dated 3/19/01. Laboratory report — revision 1. Analytical report of data for PFOS dietary LC50 study with mallards.

US EPA OPPT AR226-1030a057. 3M submission dated 4/6/01. PFOS: a frog embryo teratogenesis assay — *Xenopus* (PETAX).

US EPA OPPT AR226-1030a073. 3M submission dated 8/6/80. Photolysis study on FM-3925 (direct and indirect).

US EPA OPPT AR226-1030a074. 3M submission dated 11/7/80. Photolysis of FM3925 in aqueous solutions (direct).

US EPA OPPT AR226-1030a076. 3M submission dated 3/30/01. Hydrolysis reactions of 2-(N-methylperfluorooctanesulfonamido)-ethyl alcohol (N-MeFOSE alcohol).

US EPA OPPT AR226-1030a079. 3M submission dated 2/23/01. Hydrolysis reactions of 2-(N-ethylperfluorooctanesulfonamide)-ethyl alcohol (N-EtFOSE alcohol).

US EPA OPPT AR 226-1030a080. 3M submission dated 4/19/01. Screening studies on the aqueous photolytic degradation of 2-(N-ethylperfluorooctanesulfonamido)-ethyl alcohol (N-EtFOSE alcohol).

US EPA OPPT AR 226-1030a104. 3M submission dated 6/12/01. Indirect photolysis of gaseous perfluorooctane sulfonyl fluoride (POSF) by Fourier transform infrared (FTIR) spectroscopy.

US EPA OPPT AR 226-1030a106. 3M submission not dated. Executive summary of photolysis studies.

US EPA OPPT AR226-1030a111. 3M submission dated 6/25/01. Environmental monitoring — Multi-city study water, sludge, sediment, POTW effluent and landfill leachate samples.

US EPA OPPT AR226-1030a156. 3M submission dated 6/20/01. Accumulation of perfluorooctane sulfonate and related fluorochemicals in fish tissues.

US EPA OPPT AR226-1030a157. 3M submission dated 6/20/01. Accumulation of perfluorooctane sulfonate and related fluorochemicals in mink and river otters.

US EPA OPPT AR226-1030a158. 3M submission dated 6/20/01. Perfluorooctane sulfonate and related fluorochemicals in oyster, *Crassostrea virginica*, from the Gulf of Mexico and Chesapeake Bay.

US EPA OPPT AR226-1030a159. 3M submission dated 6/20/01. Perfluorooctane sulfonate and related fluorochemicals in fish-eating water birds.

US EPA OPPT AR226-1030a160. 3M submission dated 6/20/01. Accumulation of perfluorooctane sulfonate in marine mammals.

US EPA OPPT AR226-1030a161. 3M submission dated 6/01/01. Selected fluorochemicals in the Decatur, Alabama area.

US EPA OPPT AR226-1106. 3M submission dated 5/10/02. Final report and robust summary PFOS: an acute toxicity study with the earthworm in an artificial soil substrate. Wildlife International, Ltd. Project No. 454-111.

Appendix 1. List of PFOS and its precursors identified through Section 71 CEPA 1999 industry survey, CATABOL modelling and expert judgement[a]

CAS No.	Common name	Chemical name	Molecular formula	PFOS (Catabol)[b]	PFOS (expert judgement)
N/A	PFOS anion	1-Octanesulfonate, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-	$C_8F_{17}SO_3^-$		
1763-23-1	PFOS acid (perfluoro-octane-sulfonic acid) (also called PFOSH)	1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-	$C_8F_{17}SO_3H$	Y	Y
2795-39-3	PFOS potassium (K^+) salt	1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, potassium salt	$C_8F_{17}SO_3K$	Y	Y
29081-56-9	PFOS ammonium (NH_4^+) salt	1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, ammonium salt	$C_8F_{17}SO_3NH_4$	Y	Y
29457-72-5	PFOS lithium (Li^+) salt	1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, lithium salt	$C_8F_{17}SO_3Li$	Y	Y
70225-14-8	PFOS diethanol-amine (DEA) salt	1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, compd. with 2,2-iminobis[ethanol] (1:1)	$C_8F_{17}SO_3NH(CH_2CH_2OH)_2$	Y	Y
307-35-7	POSF	1-Octanesulfonyl fluoride, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-	$C_8F_{18}O_2S$	Y	Y
1691-99-2	N-EtFOSE alcohol	1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-	$C_{12}H_{10}F_{17}NO_3S$	Y	Y
4151-50-2	N-EtFOSA	1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-	$C_{10}H_6F_{17}NO_2S$	Y	Y
24448-09-7	N-MeFOSE alcohol	1-Octanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-N-methyl-	$C_{11}H_8F_{17}NO_3S$	Y	Y
31506-32-8	N-MeFOSA	1-Octanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-	$C_9H_4F_{17}NO_2S$	Y	Y

CAS No.	Common name	Chemical name	Molecular formula	PFOS (Catabol)[b]	PFOS (expert judgement)
		heptadecafluoro-N-methyl-			
25268-77-3	N-MeFOSEA	2-Propenoic acid, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester	$C_{14}H_{10}F_{17}NO_4S$	Y	Y
423-82-5	N-EtFOSEA	2-Propenoic acid, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester	$C_{15}H_{12}F_{17}NO_4S$	Y	Y
2250-98-8		1-Octanesulfonamide, N,N',N''-[phosphinylidynetris(oxy-2,1-ethanediyl)]tris[N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-	$C_{36}H_{27}F_{51}N_3O_{10}PS_3$	Y	Y
2991-51-7		Glycine, N-ethyl-N-[(heptadecafluorooctyl)sulfonyl]-, potassium salt	$C_{12}H_8F_{17}NO_4S{\cdot}K$	Y	Y
29117-08-6		Poly(oxy-1,2-ethanediyl), α-[2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl]-ω-hydroxy-	$(C_2H_4O)_nC_{12}H_{10}F_{17}NO_3S$	could not be modelled	Y
30381-98-7		1-Octanesulfonamide, N,N-[phosphinicobis(oxy-2,1-ethanediyl)]bis[N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, ammonium salt	$C_{24}H_{19}F_{34}N_2O_8PS_2{\cdot}H_3N$	Y	Y
38006-74-5		1-Propanaminium, 3-[[(heptadecafluorooctyl)sulfonyl]amino]-N,N,N-trimethyl-, chloride	$C_{14}H_{16}F_{17}N_2O_2S{\cdot}Cl$	Y	Y
52550-45-5		Poly(oxy-1,2-ethanediyl), α-[2-[[(heptadecafluorooctyl)sulfonyl]propylamino]ethyl]-ω-hydroxy-	$(C_2H_4O)_nC_{13}H_{12}F_{17}NO_3S$	could not be modelled	Y
56773-42-3		Ethanaminium, N,N,N-triethyl-, salt with 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-1-octanesulfonic acid (1:1)	$C_8H_{20}N{\cdot}C_8F_{17}O_3S$	Y	Y
57589-85-2		Benzoic acid, 2,3,4,5-tetrachloro-6-[[[3-[[(heptadecafluorooctyl)sulfonyl]oxy]phenyl]amino]carbonyl]-, monopotassium salt	$C_{22}H_6Cl_4F_{17}NO_6S{\cdot}K$	Y	Y
67939-88-2		1-Octanesulfonamide, N-[3-(dimethylamino)propyl]-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, monohydrochloride	$C_{13}H_{13}F_{17}N_2O_2S{\cdot}ClH$	Y	Y
67969-69-1		1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-[2-(phosphonooxy)ethyl]-, diammonium salt	$C_{12}H_{11}F_{17}NO_6PS_2{\cdot}H_3N$	Y	Y
68298-11-3		1-Propanaminium, 3-[[(heptadecafluorooctyl)sulfonyl](3-sulfopropyl)amino]-N-(2-hydroxyethyl)-N,N-dimethyl-, hydroxide, inner salt	$C_{18}H_{23}F_{17}N_2O_6S_2$	Y	Y

CAS No.	Common name	Chemical name	Molecular formula	PFOS (Catabol)[b]	PFOS (expert judgement)
68298-62-4		2-Propenoic acid, 2-[butyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester, telomer with 2-[butyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, methyloxirane polymer with oxirane di-2-propenoate, methyloxirane polymer with oxirane mono-2-propenoate and 1-octanethiol	$(C_{17}H_{16}F_{17}NO_4S \cdot C_{16}H_{16}F_{15}NO_4S \cdot W_{99} \cdot W_{99})_x \cdot C_8H_{18}S$	could not be modelled	Y
68298-78-2		2-Propenoic acid, 2-methyl-, 2-[[[[5-[[[2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethoxy]carbonyl]amino]-2-methylphenyl]amino]carbonyl]oxy]propyl ester, telomer with butyl 2-propenoate, 2-[[[[5-[[[2-[ethyl[(nonafluorobutyl)sulfonyl]amino]ethoxy]carbonyl]amino]-2-methylphenyl]amino]carbonyl]oxy]propyl 2-methyl-2-propenoate, 2-[[[[5-[[[2-[ethyl[(pentadecafluoroheptyl)sulfonyl]amino]ethoxy]carbonyl]amino]-2-methylphenyl]amino]carbonyl]oxy]propyl 2-methyl-2-propenoate, 2-[[[[5-[[[2-[ethyl[(tridecafluorohexyl)sulfonyl]amino]ethoxy]carbonyl]amino]-2-methylphenyl]amino]carbonyl]oxy]propyl 2-methyl-2-propenoate, 2-[[[[5-[[[2-[ethyl[(undecafluoropentyl)sulfonyl]amino]ethoxy]carbonyl]amino]-2-methylphenyl]amino]carbonyl]oxy]propyl 2-methyl-2-propenoate, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate and 1-octanethiol	$(C_{28}H_{28}F_{17}N_3O_8S \cdot C_{27}H_{28}F_{15}N_3O_8S \cdot C_{26}H_{28}F_{13}N_3O_8S \cdot C_{25}H_{28}F_{11}N_3O_8S \cdot C_{24}H_{28}F_9N_3O_8S \cdot C_{14}H_{10}F_{17}NO_4S \cdot C_{13}H_{10}F_{15}NO_4S \cdot C_{12}H_{10}F_{13}NO_4S \cdot C_{11}H_{10}F_{11}NO_4S \cdot C_{10}H_{10}F_9NO_4S \cdot C_7H_{12}O_2)_x \cdot C_8H_{18}S$	could not be modelled	Y
68329-56-6		2-Propenoic acid, eicosyl ester, polymer with 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate, hexadecyl 2-propenoate, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-	$(C_{23}H_{44}O_2 \cdot C_{21}H_{40}O_2 \cdot C_{19}H_{36}O_2 \cdot C_{14}H_{10}F_{17}NO_4S \cdot C_{13}H_{10}F_{15}NO_4S \cdot C_{12}H_{10}F_{13}NO_4S \cdot C_{11}H_{10}F_{11}NO_4S \cdot C_{10}H_{10}F_9NO_4S)_x$	could not be modelled	Y

CAS No.	Common name	Chemical name	Molecular formula	PFOS (Catabol)[b]	PFOS (expert judgement)
		[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate and octadecyl 2-propenoate			
68555-90-8		2-Propenoic acid, butyl ester, polymer with 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate and 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate	$(C_{14}H_{10}F_{17}NO_4S\cdot C_{13}H_{10}F_{15}NO_4S\cdot C_{12}H_{10}F_{13}NO_4S\cdot C_{11}H_{10}F_{11}NO_4S\cdot C_{10}H_{10}F_9NO_4S\cdot C_7H_{12}O_2)_x$	could not be modelled	Y
68555-91-9		2-Propenoic acid, 2-methyl-, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester, polymer with 2-[ethyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate and octadecyl 2-methyl-2-propenoate	$(C_{22}H_{42}O_2\cdot C_{16}H_{14}F_{17}NO_4S\cdot C_{15}H_{14}F_{15}NO_4S\cdot C_{14}H_{14}F_{13}NO_4S\cdot C_{13}H_{14}F_{11}NO_4S\cdot C_{12}H_{14}F_9NO_4S)_x$	could not be modelled	Y
68555-92-0		2-Propenoic acid, 2-methyl-, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester, polymer with 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate and octadecyl 2-methyl-2-propenoate	$(C_{22}H_{42}O_2\cdot C_{15}H_{12}F_{17}NO_4S\cdot C_{14}H_{12}F_{15}NO_4S\cdot C_{13}H_{12}F_{13}NO_4S\cdot C_{12}H_{12}F_{11}NO_4S\cdot C_{11}H_{12}F_9NO_4S)_x$	could not be modelled	Y
68586-14-1		2-Propenoic acid, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester, telomer with 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, α-(2-methyl-1-oxo-2-propenyl)-ω-hydroxypoly(oxy-1,2-ethanediyl), α-(2-methyl-1-oxo-2-propenyl)-ω-[(2-methyl-1-oxo-2-propenyl)oxy]poly(oxy-1,2-	$(C_{14}H_{10}F_{17}NO_4S\cdot C_{13}H_{10}F_{15}NO_4S\cdot C_{12}H_{10}F_{13}NO_4S\cdot C_{11}H_{10}F_{11}NO_4S\cdot C_{10}H_{10}F_9NO_4S\cdot (C_2H_4O)_nC_8H_{10}O_3\cdot (C_2H_4O)_nC_4H_6O_2)_x\cdot C_8H_{18}S$	could not be modelled	Y

CAS No.	Common name	Chemical name	Molecular formula	PFOS (Catabol)[b]	PFOS (expert judgement)
		ethanediyl), 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate and 1-octanethiol			
68649-26-3		1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-, reaction products with N-ethyl-1,1,2,2,3,3,4,4,4-nonafluoro-N-(2-hydroxyethyl)-1-butanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-N-(2-hydroxyethyl)-1-heptanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro-N-(2-hydroxyethyl)-1-hexanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,5-undecafluoro-N-(2-hydroxyethyl)-1-pentanesulfonamide, polymethylenepolyphenylene isocyanate and stearyl alc.	$(C_{18}H_{38}O{\cdot}C_{12}H_{10}F_{17}NO_3S{\cdot}C_{11}H_{10}F_{15}NO_3S{\cdot}C_{10}H_{10}F_{13}NO_3S{\cdot}.C\text{-}H_{10}F_{11}NO_3S{\cdot}C_8H_{10}F_9NO_3S{\cdot}\text{Unspecified})_x$	could not be modelled	Y
68867-62-9		2-Propenoic acid, 2-methyl-, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester, telomer with 2-[ethyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 1-octanethiol and α-(1-oxo-2-propenyl)-ω-methoxypoly(oxy-1,2-ethanediyl)	$(C_{16}H_{14}F_{17}NO_4S{\cdot}C_{15}H_{14}F_{15}NO_4S{\cdot}C_{14}H_{14}F_{13}NO_4S{\cdot}C_{13}H_{14}F_{11}NO_4S{\cdot}C_{12}H_{14}F_9NO_4S{\cdot}(C_2H_4O)_nC_4H_6O_2)_x{\cdot}C_8H_{18}S$	could not be modelled	Y
68877-32-7		2-Propenoic acid, 2-methyl-, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester, polymer with 2-[ethyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate and 2-methyl-1,3-butadiene	$(C_{16}H_{14}F_{17}NO_4S{\cdot}C_{15}H_{14}F_{15}NO_4S{\cdot}C_{14}H_{14}F_{13}NO_4S{\cdot}C_{13}H_{14}F_{11}NO_4S{\cdot}C_{12}H_{14}F_9NO_4S{\cdot}C_5H_8)_x$	could not be modelled	Y
68891-96-3		Chromium, diaquatetrachloro[μ-[N-ethyl-N-[(heptadecafluorooctyl)sulfonyl]glycinato-O':O"]]μ-	$C_{18}H_{28}Cl_4Cr_2F_{17}NO_9S$	Y	Y

CAS No.	Common name	Chemical name	Molecular formula	PFOS (Catabol)[b]	PFOS (expert judgement)
		hydroxybis(2-methylpropanol)di-			
68958-61-2		Poly(oxy-1,2-ethanediyl), α-[2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl]-ω-methoxy-	$(C_2H_4O)_nC_{13}H_{12}F_{17}NO_3S$	could not be modelled	Y
70776-36-2		2-Propenoic acid, 2-methyl-, octadecyl ester, polymer with 1,1-dichloroethene, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate, N-(hydroxymethyl)-2-propenamide, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate and 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate	$(C_{22}H_{42}O_2{\cdot}C_{14}H_{10}F_{17}NO_4S{\cdot}C_{13}H_{10}F_{15}NO_4S{\cdot}C_{12}H_{10}F_{13}NO_4S{\cdot}C_{11}H_{10}F_{11}NO_4S{\cdot}C_{10}H_{10}F_9NO_4S{\cdot}C_4H_7NO_2{\cdot}C_2H_2Cl_2)_x$	could not be modelled	Y
71487-20-2		2-Propenoic acid, 2-methyl-, methyl ester, polymer with ethenylbenzene, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate and 2-propenoic acid	$(C_{14}H_{10}F_{17}NO_4S{\cdot}C_{13}H_{10}F_{15}NO_4S{\cdot}C_{12}H_{10}F_{13}NO_4S{\cdot}C_{11}H_{10}F_{11}NO_4S{\cdot}C_{10}H_{10}F_9NO_4S{\cdot}C_8H_8{\cdot}C_5H_8O_2{\cdot}C_3H_4O_2)_x$	could not be modelled	Y
92265-81-1		Ethanaminium, N,N,N-trimethyl-2-[(2-methyl-1-oxo-2-propenyl)oxy]-, chloride, polymer with 2-ethoxyethyl 2-propenoate, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate and oxiranylmethyl 2-methyl-2-propenoate	$(C_{14}H_{10}F_{17}NO_4S{\cdot}C_9H_{18}NO_2{\cdot}C_7H_{12}O_3{\cdot}C_7H_{10}O_3{\cdot}Cl)_x$	N	Y
94313-84-5		Carbamic acid, [5-[[[2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethoxy]carbonyl]amino]-2-methylphenyl]-, 9-octadecenyl ester, (Z)-	$C_{38}H_{50}F_{17}N_3O_6S$	Y	Y
98999-57-6		Sulfonamides, C_{7-8}-alkane, perfluoro, N-methyl-N-[2-[(1-oxo-2-propenyl)oxy]ethyl], polymers with 2-ethoxyethyl acrylate, glycidyl methacrylate and N,N,N-trimethyl-2-[(2-methyl-1-oxo-propenyl)oxy]ethanaminium chloride	$(C_{14}H_{10}F_{17}NO_4S{\cdot}C_9H_{18}NO_2{\cdot}C_7H_{12}O_3{\cdot}C_7H_{10}O_3{\cdot}Cl)_x$	could not be modelled	Y

CAS No.	Common name	Chemical name	Molecular formula	PFOS (Catabol)[b]	PFOS (expert judgement)
178094-69-4		1-Octanesulfonamide, N-[3-(dimethyloxidoamino)propyl]-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, potassium salt	$C_{13}H_{12}F_{17}N_2O_3S\cdot K$	Y	Y
N/A		2-(Perfluoro-N-methyl-C_{4-8}-1-alkanesulfonamido)ethyl esters of trimers of C_{18} unsaturated fatty acids	N/A	could not be modelled	Y
68909-15-9		2-Propenoic acid, eicosyl ester, polymers with branched octyl acrylate, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl acrylate, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl acrylate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl acrylate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl acrylate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl acrylate, polyethylene glycol acrylate Me ether and stearyl acrylate	$(C_{23}H_{44}O_2\cdot C_{21}H_{40}O_2\cdot C_{14}H_{10}F_{17}NO_4S\cdot C_{13}H_{10}F_{15}NO_4S\cdot C_{12}H_{10}F_{13}NO_4S\cdot C_{11}H_{10}F_{11}NO_4S\cdot C_{10}H_{10}F_9NO_4S\cdot(C_2H_4O)_nC_4H_6O_2\cdot$ Unspecified)$_x$	could not be modelled	Y
148684-79-1		Sulfonamides, C_{4-8}-alkane, perfluoro, N-(hydroxyethyl)-N-methyl, reaction products with 1,6-diisocyanatohexane homopolymer and ethylene glycol	N/A	could not be modelled	Y
30295-51-3		1-Octanesulfonamide, N-[3-(dimethyloxidoamino)propyl]-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-	N/A	Y	Y
91081-99-1		Sulfonamides, C_{4-8}-alkane, perfluoro, N-(hydroxyethyl)-N-methyl, reaction products with epichlorohydrin, adipates (esters)	N/A	could not be modelled	Y
N/A		Fatty acids, C_{18}-unsatd., dimers, 2-[methyl[(perfluoro-C_{4-8}-alkyl)sulfonyl]amino]ethyl esters	N/A	Y	Y
68081-83-4		Carbamic acid, (4-methyl-1,3-phenylene)bis-, bis[2-[ethyl[(perfluoro-C_{4-8}-alkyl)sulfonyl]amino]ethyl] ester		Y	Y
68608-14-0		Sulfonamides, C_{4-8}-alkane, perfluoro, N-ethyl-N-(hydroxyethyl), reaction products with 1,1'-methylenebis[4-isocyanatobenzene]	$C_{15}H_{10}N_2O_2\cdot$Unspecified	Y	Y
376-14-7		2-Propenoic acid, 2-methyl-, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester	$C_{16}H_{14}F_{17}NO_4S$	Y	Y
14650-24-9		2-Propenoic acid, 2-methyl-, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester	$C_{15}H_{12}F_{17}NO_4S$	Y	Y
94133-90-1		1-Propanesulfonic acid, 3-[[3-(dimethylamino)propyl][(heptadecafluorooctyl)sulfonyl]amino]-2-hydroxy-, monosodium salt	$C_{16}H_{19}F_{17}N_2O_6S_2\cdot Na$	Y	Y
127133-66-8		2-Propenoic acid, 2-methyl-, polymers with Bu methacrylate,	$(C_{16}H_{30}O_2\cdot C_8H_{14}O_2\cdot C_4H$	Y	Y

CAS No.	Common name	Chemical name	Molecular formula	PFOS (Catabol)[b]	PFOS (expert judgement)
		lauryl methacrylate and 2-[methyl[(perfluoro-C_{4-8}-alkyl)sulfonyl]amino]ethyl methacrylate	$_6O_2)_x$		
179005-06-2		Sulfonamides, C_{4-8}-alkane, perfluoro, N-[3-(dimethyloxidoamino)propyl], potassium salts	N/A	could not be modelled	Y
179005-07-3		Sulfonamides, C_{4-8}-alkane, perfluoro, N-[3-(dimethyloxidoamino)propyl]	N/A	could not be modelled	Y
ROF		Residual Organic Fluorochemicals (impurities)	N/A	Y	Y

[a] Notes:
1. References: Mekenyan et al. (2002); Purdy (2002a).
2. N/A = not available; Bu = butyl; Et = ethyl; Me = methyl.
3. This list is not necessarily an exhaustive list of all possible PFOS precursors.

[b] For each substance modelled, CATABOL generates a microbial metabolic pathway tree based upon the parent "query" structure and a prediction for biodegradability. The metabolic pathway tree module is based on a training data set primarily from the University of Minnesota Biocatalysis/Biodegradation database (UM-BBD) and expert knowledge. The metabolic tree contains the products of microbial biodegradation from the parent compound down to carbon dioxide and water or stable metabolites. Some of the chemicals could not be modelled by CATABOL due to the lack of SMILES notation.

The biodegradation simulator is based on a database of 742 substances tested by CITI (1992) using the Modified MITI Test (I), which follows the OECD 301C test methods and is one of six methods approved by the OECD for ready biodegradability. A more complete description of CATABOL modelling is provided in Robinson (2002).

Table 4. Summary of data used in risk quotient (Q) analysis of PFOS and its precursors

Substance	Pelagic organisms					Birds (liver)					Birds (serum)					Mammals (liver)				
	EEV[a] (µg/L)	CTV[b] (µg/L)	AF[c]	ENEV (µg/L)	Q (EEV/ ENEV)	EEV[d] (µg/g liver)	CTV[e] (µg/g liver)	AF[f]	ENEV (µg/g liver)	Q (EEV/ ENEV)	EEV[g] (mg/L serum)	CTV[h] (mg/L serum)	AF[f]	ENEV (mg/L serum)	Q (EEV/ ENEV)	EEV[i] (µg/g liver)	CTV[j] (µg/g liver)	AF[f]	ENEV (µg/g liver)	Q (EEV/ ENEV)
PFOS	2.93	86.0	100	0.86	3.4	0.65	29.7	1000	0.0297	21.9	2.22	13.9	1000	0.0139	160	>4.0	40.8	1000	0.0408	>98.0

[a] Due to a lack of empirical data characterizing PFOS and its precursors in Canadian water, data from the United States were used as a surrogate for Canadian data. The highest relevant water concentration found was 2.93 µg/L in quiet water (pond) close to Port St. Lucie, Florida. The site was considered a reference site, since PFOS is not manufactured there. A dilution factor was not used to calculate the EEV, since PFOS has been determined to be persistent and bioaccumulative.

[b] US EPA OPPT AR226-1030a042 on bluegill (*Lepomis macrochirus*).

[c] AF (application factors): 10 applied for extrapolation from laboratory to field conditions and for intraspecies and interspecies variations in sensitivity; 10 applied because PFOS and its precursors are bioaccumulative and persistent.

[d] The highest level of PFOS in liver of wild birds was 0.65 mg/kg liver (650 µg/kg liver) in common cormorant in Japan. This value was considered an appropriate surrogate given the limited information for PFOS in liver of Canadian birds. It is noted that risk quotients for a range of fish-eating birds worldwide ranged from <1.18 in albatross in the mid-Pacific (EEV <0.035 mg/kg liver) to 15.5 in brown pelican in Mississippi (EEV 0.46 mg/kg liver).

[e] LOEC of 29.7 mg/kg (29.7 µg/g) liver wet weight for effects on body weight gain in mallard feeding study (US EPA OPPT AR226-1030a049).

[f] AF (application factors): 10 applied for extrapolation from laboratory to field conditions and for intraspecies and interspecies variations in sensitivity; 10 applied because PFOS, its salts and its precursors are bioaccumulative and persistent; an additional 10 applied to extrapolate from LOEC to a chronic NOEC.

[g] The highest level of PFOS measured in wildlife in serum or plasma was reported in bald eagle from Michigan, Wisconsin and Minnesota at a maximum concentration of 2220 µg/L or 2.22 mg/L (US EPA OPPT AR226-1030a159).

[h] As no data exist on concentrations of PFOS in bird serum for specific toxicological endpoints, levels of PFOS in serum of rats for known toxicological effects were used as surrogates.

[i] In Canada, the highest PFOS concentration was found in polar bear liver (maximum >4.0 mg/kg liver) (Martin et al. 2004). The PFOS concentrations in polar bear liver were higher than any other previously reported concentrations of persistent organochlorine chemicals (e.g., PCBs, chlordane or hexachlorocyclohexane) in polar bear fat. It is noted that using the highest tissue concentration (4.87 mg/kg liver) found in the livers of mink in the Midwestern United States would yield a risk quotient (119) of the same order of magnitude, which could also be considered relevant to Canadian wildlife in mid-latitudes. The polar bear data were selected given that they are more recent and are Canadian.

[j] As no wild mammal studies were found, laboratory mammal studies were used as surrogates. The CTV for mammals was selected from a 2-year dietary rat study in which histopathological effects in the liver were seen in males and females at intakes as low as 0.06–0.23 mg PFOS/kg bw per day and 0.07–0.21 mg PFOS/kg bw per day, respectively (Covance Laboratories, Inc. 2002). Average values were determined for males and females, to establish LOELs of 40.8 µg/g in liver and 13.9 mg/L in serum.