***Canadian Environmental Protection Act, 1999* (CEPA 1999):**
Ecological Screening Assessment Report on
Perfluorooctane Sulfonate, Its Salts and Its Precursors that Contain the $C_8F_{17}SO_2$ or $C_8F_{17}SO_3$, or $C_8F_{17}SO_2N$ Moiety

June 2006

Environment Canada

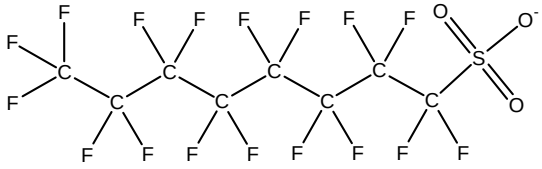

Figure 1: PFOS anion structure

SYNOPSIS

Perfluorooctane sulfonate (PFOS) is of anthropogenic origin with no known natural sources. In Canada, there is no known manufacture of perfluoroalkyl (PFA) compounds, including PFOS. Approximately 600 tonnes of PFA compounds were imported into Canada between 1997 and 2000. While PFOS represents a very small proportion of this total (< 2 %), PFOS and its precursors accounted for about 43 %. The principal applications for PFOS and its precursors are water, oil, soil and grease repellents for use on surface and paper-based applications, such as rugs and carpets, fabric and upholstery, and food packaging. PFOS and its precursors also have specialized chemical applications, such as fire-fighting foams, hydraulic fluids, carpet spot removers, mining and oil well surfactants and other specialized chemical formulations. Exposure in the Canadian environment would likely result from the release, transformation and movement of PFOS and its precursors in effluents, fugitive emissions from manufacturing sites elsewhere in the world, and releases from industrial and municipal wastewater effluents.

PFOS is resistant to hydrolysis, photolysis, microbial degradation, and metabolism by vertebrates. PFOS has been detected in fish, in wildlife worldwide and in the northern hemisphere. This includes Canadian wildlife located far from known sources or manufacturing

facilities indicating that PFOS and/or its precursors may undergo long-range transport. Maximum concentrations in liver of biota in remote areas of the Canadian Arctic include: mink (20 $\mu g.kg^{-1}$), common loon (26 $\mu g.kg^{-1}$), ringed seal (37 $\mu g.kg^{-1}$), brook trout (50 $\mu g.kg^{-1}$), Arctic fox (1400 $\mu g.kg^{-1}$) and polar bear (>4000 $\mu g.kg^{-1}$).

Unlike many other persistent organic pollutants, certain perfluorinated substances, such as PFOS, are present as ions in environmental media and partition preferentially to proteins in liver and blood rather than to lipids. Therefore, the bioaccumulation potential of PFOS may not be related to the typical mechanisms associated with bioaccumulation in lipid-rich tissues. Discretion is required when applying numeric criteria for bioaccumulation such as those outlined in the Government of Canada's Toxic Substances Management Policy (TSMP) and in the *Persistence and Bioaccumulation Regulations* under CEPA 1999 when determining whether substances such as PFOS is bioaccumulative. These numeric criteria were derived from bioaccumulation data for aquatic species and for substances which preferentially partition to lipids.

Estimated steady state PFOS bioconcentration factors (BCF) of 1100 (carcass), 5400 (liver) and 4300 (blood) have been reported for juvenile rainbow trout. The corresponding 12-day accumulation ratios were 690 (carcass), 3100 (blood), and 2900 (liver) in juvenile rainbow trout. In fish livers collected from 23 different species in Japan, bioaccumulation factors (BAFs) were calculated to range from 274 – 41 600. Following an accidental release of fire fighting foam, BAFs were calculated in the range of 6300-125 000. Estimated BCFs for the precursors n-EtFOSEA and n-MeFOSEA were 5543 and 26 000, respectively. Species differences for the elimination half-life of PFOS in biota have been determined to vary significantly: 15 days (fish); 100 days (rats), 200 days (monkeys) and years (humans). Elimination through the gills is an important route for fish which is not available to birds, terrestrial mammals (e.g., mink, polar bear, Arctic foxes) and marine mammals (e.g., seals and whales). There are three studies suggesting that PFOS biomagnifies in the Great Lakes and Arctic food webs. In the study by Kannan *et al*., (2005a), for the water-algae-zebra mussel-round goby-smallmouth bass-bald eagle or mink food chain, a biomagnification factor (BMF) of 10 to 20 was calculated in mink or bald eagles. In the study by Martin *et al*., (2004b), a benthic invertebrate/pelagic invertebrate-three forage fish -top predator fish food chain resulted in a multi-trophic level BMF of 5.88. Tomy *et al*., (2004) suggested that PFOS biomagnifies through the Arctic marine food web. The trophic level BMFs for PFOS included walrus–clam (4.6); narwhal–cod (7.2); beluga-cod (8.4);

beluga–redfish (4.0); black-legged kittiwake–cod (5.1); glaucous gull–cod (9.0); and cod-zooplankton (0.4). Whole body aquatic BCFs or BAFs are below 5000. However, the weight of evidence from both laboratory and field-based BCFs and BAFs in conjunction with the field-based BMFs (avian and aquatic) indicates that PFOS is a bioaccumulative substance.

Based on available toxicity tests, estimated no effect levels were determined for fish, birds (liver), bird (serum), and wildlife (0.491µg.L^{-1}, 0.609 µg.g^{-1}, 0.873 µg.mL^{-1} and 0.408 µg.g^{-1}, respectively). The resulting risk quotients for fish, a range of bird species (liver and serum), and wildlife were 0.25, 0.002 to 2.92, 0.43 to 2.54 and 9.2, respectively. Therefore, current levels show some wildlife organisms (e.g. polar bear, bird species) could be near or at effect levels and could be harmed by current exposures to PFOS.

The assessment is based on a weight of evidence approach regarding persistence, bioaccumulation, the widespread occurrence of and concentrations of PFOS in the environment and in biota (including remote areas of Canada), and risk quotient analyses. Based on available data, it is 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. In addition, based on available data, it is concluded that PFOS and its salts is persistent. The weight of evidence is also sufficient to conclude that PFOS and its salts are bioaccumulative.

INTRODUCTION

An ecological screening assessment was undertaken on perfluorooctane sulfonate (PFOS), its salts and its precursors containing the perfluorooctylsulfonyl ($C_8F_{17}SO_2$, $C_8F_{17}SO_3$, or $C_8F_{17}SO_2N$) moiety. The assessment was undertaken on the basis that some of these compounds were identified as part of a Domestic Substances List (DSL) pilot for screening as they met the criteria for persistence, bioaccumulation and/or inherent toxicity, pursuant to Paragraph 73(1)(b) of the *Canadian Environmental Protection Act,*1999 (CEPA 1999) and in response to a request to the Minister of the Environment to add these compounds to the Priority Substance List (PSL) for assessment of ecological and human health.

PFOS, its salts and its precursors form part of a larger chemical class of fluorochemicals referred to as perfluorinated alkyl (PFA) compounds. The term PFOS may refer to any of its anionic,

acid or salt forms. The perfluorooctylsulfonyl ($C_8F_{17}SO_2$, $C_8F_{17}SO_3$, or $C_8F_{17}SO_2N$) moiety is incorporated in a variety of compounds which have the potential to transform or degrade back to PFOS in the environment. For the purpose of this assessment, the term "precursor" refers to compounds that contain the $C_8F_{17}SO_2$ or $C_8F_{17}SO_3$, or $C_8F_{17}SO_2N$ moiety and, therefore, have the potential to transform or degrade to PFOS. The term "precursor" applies to, but is not limited to, some 50 substances identified in the ecological assessment. This assessment addresses PFOS and also considers its precursors given their similar use applications and given that PFOS is the final degradation product of PFOS precursors. While the assessment did not consider the additive effects of PFOS and its precursors, it is recognized that the precursors to PFOS contribute to the ultimate environmental loading of PFOS. Precursors may also play a key role in the long-range transport and subsequent degradation to PFOS in remote areas.

The approach taken in this ecological screening assessment was to examine the available 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, persistence, bioaccumulation and presence in the Canadian Arctic environment and wildlife. Other concerns that affect current or potential risk, such as chemical transformation and precursors, were also examined. This ecological screening assessment report does not present an exhaustive review of all available data. Rather, this report presents the most critical information in a weight of evidence approach to support the conclusions.

Data relevant to the ecological screening assessment of PFOS and its precursors was identified in original literature, review documents, international assessments (e.g., European Commission 2005, OECD 2002a, and Swedish Chemicals Inspectorate *et al.* 2004) and industry research reports. A supporting document was prepared using degradation modeling (CATABOL[1] software) to predict PFOS precursors. On-line literature database searches were conducted for select perfluoroalkyl compounds. Direct contacts were made with researchers, academics, industry and other government agencies to obtain relevant information on PFOS, its salts and its precursors. Ongoing scans were conducted of the open literature, conference proceedings and

[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 Internet. Data up to November 2005 were considered. In addition, a survey on certain perfluoroalkyl and fluoroalkyl substances, their derivatives and polymers was conducted through a *Canada Gazette* Notice under the authority of Section 71 of CEPA 1999 (Environment Canada, 2001). This survey required industry to provide data on the Canadian manufacture, import and export of certain perfluorinated alkyl compounds from 1997-2000. Existing toxicological studies submitted by industry under Section 70 of CEPA 1999 were also examined.

Following internal and external science reviews, a draft ecological screening assessment of PFOS, its salts, and its precursors was made available for a 60-day public comment period (October 2 to December 2, 2004). Following consideration of comments received, the ecological screening assessment and its associated, unpublished Supporting Working Document were subsequently revised, as appropriate, by Environment Canada. A summary of the comments and responses is available on the Internet at: http://www.ec.gc.ca/substances/ese/eng/dsl/slra.cfm The external science peer review was conducted by Canadian and international experts from government, industry and academia. These peer reviewers included 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). The conclusion of this screening assessment report does not necessarily reflect the opinion of the peer reviewers. All peer reviewer comments were considered carefully and, where appropriate, used by Environment Canada.

The associated, unpublished Supporting Working document is available upon request by e-mail from ESB.DSE@ec.gc.ca. Information on ecological screening assessments under CEPA 1999 is available at http://www.ec.gc.ca/substances/ese/eng/dsl/slra.cfm Information on the human health screening assessment is available at http://www.hc-sc.gc.ca/ewh-semt/contaminants/existsub/screen-eval-prealable/index_e.html.

SUMMARY OF CRITICAL INFORMATION FOR THE ECOLOGICAL SCREENING ASSESSMENT REPORT FOR PFOS, ITS SALTS, AND ITS PRECURSORS

1.0 IDENTITY, USES, AND SOURCES OF RELEASE

Identity

The PFOS anion (see 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. 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 physical-chemical properties of these perfluorochemicals.

This assessment defines PFOS precursors as substances containing the perfluorooctylsulfonyl ($C_8F_{17}SO_2$, $C_8F_{17}SO_3$, or $C_8F_{17}SO_2N$) moiety that have the potential to transform or degrade to PFOS. Appendix 1 lists some compounds considered as PFOS and its precursors (e.g., 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) and some other 40 precursors. However, 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).

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 un-reacted 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 to 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. 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 ($g.mol^{-1}$)	Solubility ($g.L^{-1}$)	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 to 6.80 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)

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 Organization 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 (Appendix 1) will degrade to PFOS (Dimitrov *et al*. 2004). This degradation has been further supported by expert judgment. It is, therefore, expected that once those substances listed in Appendix 1 are subjected to a biotic or abiotic degradation

[2] Administrative Records are 3M submissions to the US Environmental Protection Agency's (US EPA) Office of Pollution Prevention and Toxics.

mechanism, the perfluorinated moiety that remains will be PFOS. The rate of degradation to PFOS is not considered significant, as, over time, these substances are all expected to degrade in the environment to PFOS.

Natural Sources

There are no known natural sources of PFOS (Key *et al*. 1997). Its presence in the environment is due solely to anthropogenic activity.

Uses, Manufacturing and Imports

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 600 tonnes of perfluorinated alkyl compounds were imported into Canada during 1997–2000, with PFOS and its precursors accounting for about 43% of imported perfluorinated alkyl compounds. PFOS alone accounted for <2% of imported perfluorinated alkyl compounds (Environment Canada 2001). The most significant 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 3M phase-out plan for PFOS production was completed in 2002 (http://www.solutions.3m.com).

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.

Sources of Release

Significant PFOS releases to the Canadian environment may result from 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 that may contain PFOS or its precursors.

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 land filled 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). A laboratory-scale incineration study of PFOS and C8 perfluorosulfonamides determined that a properly operating full-scale (high temperature) incineration system can adequately dispose of PFOS and C8 perfluorosulfonamides (US EPA OPPT AR226-136). The study also indicated that incineration of these substances is not likely to be a significant source of PFOS into the environment. The C-S bond was completely destroyed indicating that transformation of any combustion products to form PFOS was also highly unlikely. Any potential formation and release of PFOS through incomplete incineration is not considered a significant source in Canada, where incineration accounts for only about 5% of waste disposal (Compass Environmental Inc. 1999).

2.0 ENVIRONMENTAL FATE, EXPOSURE AND EFFECTS

Environmental Fate of PFOS Precursors

PFOS precursors may be subject to atmospheric transport from their sources to remote areas in Canada. 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). All of the above precursors have been predicted by CATABOL modeling and/or expert judgement to degrade to PFOS (Appendix 1). 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 indicated by its relatively high Henry's law constant (1.9×10^3 $Pa \cdot m^3.mol^{-1}$) (Hekster *et al*. 2002). It has been 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 suggested that this may be partially responsible for the ubiquitous presence of PFOS measured at a distance from significant sources.

It is predicted that the precursors identified in Appendix 1 will undergo degradation once released to the environment though transformation rates may vary widely. Precursors that reach a remote region through the atmosphere or other media may be subject to both abiotic and biotic degradation routes to PFOS (Giesy and Kannan 2002; Hekster *et al*. 2002). The mechanisms of this degradation are not well understood. 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. Precursors could be entering food chains by partitioning into biota and then undergoing degradation to PFOS somewhere along the food chain. Most available experimental environmental degradation rates of PFOS precursors are for N-MeFOSE alcohol, N-EtFOSE alcohol, N-MeFOSEA and N-

EtFOSEA and are summarized in Table 2.

Table 2: Summary of select 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% [c]$t_{½}$= estimated 40days at 25°C (indirect photolysis)	$t_{½}$ = 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); [c] US EPA AR226-1030a080

Some studies on photolysis show that this transformation mechanism will be of no importance in the breakdown of certain perfluorinated chemicals. Certain 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, and AR226-1030a106). A photolysis study on N-EtFOSE alcohol found that the primary products of indirect photolyis of this substance included PFOA, N-ethylperfluorooctane sulfonamide and perfluorooctane sulfonamide, with trace levels of additional substances including PFOS (US EPA OPPT AR226-1030a080). The study estimated an indirect photolysis half-life for N-EtFOSE alcohol of 40 days at 25°C, but noted environmental factors could lead to variation.

Persistence

PFOS is resistant to hydrolysis, photolysis, aerobic and anaerobic biodegradation and metabolism by vertebrates. 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^{-1}) (US EPA OPPT AR226-0547). The perfluorinated chain provides exceptional resistance to thermal and chemical attack (US EPA OPPT AR 226-0547). Several biodegradation studies were reviewed by the OECD which indicated no biodegradation had taken place (OECD 2002a).

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 indicated in numerous studies (Key *et al*. 1997; Giesy and Kannan 2002; Hekster *et al.* 2002; OECD 2002a). In water, PFOS was observed to persist for more than 285 days in microcosms under natural conditions (Boudreau *et al*. 2003b). 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). PFOS and some of its precursors are considered to be persistent in the Canadian environment with the environmental half-life for PFOS exceeding 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 known sources or manufacturing facilities (Martin *et al.* 2004a). This suggests that either PFOS or PFOS precursors may undergo long-range transport.

Predicting the environmental fate of PFOS can be difficult, given its physical and chemical characteristics. Due to the surface-active properties of PFOS, a meaningful log K_{ow} value cannot be determined (OECD 2002a). Unlike the situation with most 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 adsorbs via chemisorption (Hekster *et al*. 2002). A soil adsorption/desorption study using various soil, sediment and sludge matrices found that PFOS adsorbed to all matrices tested (US EPA OPPT AR226-1107). River sediments displayed the most desorption, at 39% after 48 hours, whereas sludge samples did not desorb detectable amounts of PFOS. If PFOS does bind to particulate matter in the water column, then it may settle and reside in sediment. However, as noted, desorption may also occur.

While the vapour pressure of PFOS is similar to those of other globally distributed compounds (e.g., polychlorinated biphenyls [PCBs], dichlorodiphenyltrichloroethane [DDT]), its greater 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 to 680 $mg.L^{-1}$. This has been found to decrease significantly with increasing salt content (12.4 $mg.L^{-1}$ in natural seawater at 22-23°C, and 20.0 $mg.L^{-1}$ in a 3.5% NaCl solution at 22-24°C) (US EPA OPPT AR226-0620; Hekster *et al*. 2002; OECD 2002a). 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 2002a).

Bioaccumulation

The use of log K_{ow} and physical-chemical properties to predict the potential for bioaccumulation, in general, is based on the assumption that the hydrophobic and lipophilic interactions between compound and substrate are the main mechanisms governing partitioning. This assumption has been shown to hold for non-polar and slightly polar organic chemicals. However, this assumption may not be applicable for perfluorinated substances. Due to the perfluorination as described by Key *et al*. (1997), the hydrocarbon chains are oleophilic and hydrophobic and the perfluorinated chains are both oleophobic and hydrophobic. In addition, functional groups attached to the perfluorinated chain (e.g., a charged moiety such as sulfonic acid) can impart hydrophilicity to part of the molecule. Hydrophobicity is unlikely to be the sole driving force for the partitioning of perfluorinated substances to tissues because the oleophobic repellency opposes this partitioning process (Kannan *et al*., 2001). Perfluorinated substances are also intrinsically polar chemicals because fluorine, a highly electronegative element, imparts polarity. Thus, perfluorinated substances have combined properties of oleophobicity, hydrophobicity, and hydrophilicity over portions of a particular molecule. Based on the current scientific understanding, lipid normalizing of concentrations in organisms for perfluorinated substances may not be appropriate since these substances appear to preferentially bind to proteins in liver and blood rather than accumulating in lipids.

Measures of bioaccumulation (bioconcentration factors (BCFs), bioaccumulation factors (BAFs), and biomagnification factors (BMFs)) may be used as indicators of either direct toxicity to organisms that have accumulated PFOS or indirect toxicity to organisms that consume prey containing PFOS (via food chain transfer). Concerning the potential to cause direct toxicity, the critical body burden is the minimum concentration of a substance in an organism that causes an

adverse effect. From a physiological perspective, it is the concentration of a substance at the site of toxic action within the organism that determines whether a response is observed, regardless of the external concentration. In the case of PFOS, the site of toxic action is often considered to be the liver.

Concerning the potential for toxicity to consumer organisms , it is the concentration in the whole body of a prey that is of interest since the prey is often completely consumed by the predator - including individual tissues and organs, such as the liver and blood. However, given the partitioning into liver and blood, most field measurements for perfluorinated substances have been performed for those individual organs and tissues especially for higher trophic level organisms (e.g. polar bear) where whole body analysis is not feasible due to either sampling or laboratory processing constraints. While it is feasible to measure whole body BAFs[3] on smaller, lower trophic level species, the lower trophic status of the organism would mean that, for perfluorinated substances, the estimated overall BAFs may be underestimated due to their trophic status.

Thus, from a toxicological perspective, BCFs, BAFs and BMFs based on concentrations in individual organs, such as the liver, may be more relevant when predicting potential for direct organ-specific toxicity (i.e., liver toxicity). However, BCFs and particularly BMFs based on concentrations in whole organisms may provide a useful measure of overall potential for food chain transfer. The ranges for whole body and tissue- and organ-specific BCFs/BAFs/BMFs are summarized as follows:

Table 3: Range of BCF/BAF/BMF data for PFOS in whole body, specific tissues and organs in wildlife

	Whole Body	Tissue Specific (blood or liver)
BCF	690 - 2796	2900 – 5400
BAF	None available	274 – 125 000
BMF	0.4 – 5.88	4.0 – 20

[3]**Bioconcentration** is the process by which a chemical enters an organism and/or is adsorbed on to it as a result of exposure to the chemical in water – it often refers to a condition usually achieved under laboratory and steady state conditions. **Bioaccumulation** is the process by which a chemical enters an organism as a result of uptake through all possible routes of exposure (dietary, dermal, and respiratory). **Biomagnification** is the process by which chemical concentrations increase with trophic level in a food chain and results from the trophic level transfer of a chemical through the diet from a lower to a higher trophic level.

Estimated PFOS BCFs (assumed steady-state conditions) of 1100 (carcass), 5400 (liver) and 4300 (blood) have been reported for juvenile rainbow trout; the 12-day accumulation ratio was 690 (carcass), 3100 (blood), and 2900 (liver) (Martin *et al*., 2003a). A laboratory study with bluegill sunfish gave a whole body BCF of 2796 (US EPA OPPT AR226-1030a042). 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). In the study by Kannan *et al*., (2005a), a BCF of 1000 (whole-body) was calculated in benthic invertebrates. Species differences for the elimination half-life of PFOS in biota have been determined to vary significantly: 15 days (fish); 100 days (rats), 200 days (monkeys) and years (humans) (OECD 2002a; Martin *et al.,* 2003b).

In fish livers collected from 23 different species in Japan, PFOS BAFs[4] were calculated to range from 274 - 41 600 (mean 5500) (Taniyasu *et al.* 2003). Following an accidental release of fire fighting foam into Etobicoke Creek, Moody *et al.* (2002) calculated a BAF range of 6300-125 000 for PFOS, based on measured concentrations in common shiner liver and surface water (Moody *et al.*, 2002).

Available data indicate certain fish species at specific life-stages (e.g. juvenile rainbow trout) with dietary exposure to PFAs would have BMFs lower than one and that biomagnification would not occur. Martin *et al*. (2003b) showed that PFOS did not biomagnify from food in juvenile trout[5]. However, the authors suggest caution in extrapolating these results to larger fish, e.g. mature trout, as the half lives of other substances have been shown to increase by up to a factor of 10 times in mature fish compared to juveniles. A possible driving factor for this phenomenon is that the gill-surface-area to volume ratio and relative gill ventilation rate may decrease as fish mature, i.e., elimination via the gills may become less efficient and less significant. Another contributing factor could be growth dilution which is much more significant in relatively fast growing juveniles. Nevertheless, elimination through the gills is an important

[4] The authors referred to their results as "BCFs". However, samples were field collected where sources of uptake may include dietary.

[5] The authors referred to their results as "BAFs". However, the "BAFs" reported in their study represented an estimate of the steady state ratio between the organism and its food (i.e., the BMF), which was determined using the kinetic method.

route for fish which is not available to birds, terrestrial mammals (e.g., mink, polar bear, Arctic foxes) and marine mammals (e.g., seals and whales). Furthermore, elimination from the lungs is expected to be much lower in view of the low vapour pressure and negative charge. 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).

Moody *et al*., (2002) suggested that the BAFs in their study may be overestimated due to the metabolism of accumulated precursors to PFOS. The biotransformation of PFOS precursors (e.g. PFOS precursors in fire-fighting foam) is currently not well studied. However, it is possible that the transformation of precursors to PFOS within the organism could cause the total body burden of PFOS to exceed that which would be achieved by accumulation from water and diet alone. Since the water concentration used for the BAF calculation does not account for the proportion of precursors which could be transformed to PFOS within the organism, the calculated BAF may be artificially high. In the absence of established methods to account for precursor bioaccumulation and transformation, it can be argued that the results of the Moody *et al*. (2002) study provide a relevant expression of bioaccumulative potential and conservative BAF estimates especially given that the metabolic transformation of precursors to PFOS is an additional cause for concern.

There are three studies suggesting that PFOS biomagnifies in the Great Lakes and Arctic food webs. In the study by Kannan *et al*., (2005a), for the water-algae-zebra mussel (whole body)-round goby (whole body) -smallmouth bass (muscle tissue) -bald eagle (liver, muscle, or kidney tissue) food chain, a BMF of 10 to 20 was calculated in mink (liver) or bald eagles. It should be noted that comparison of PFOS concentrations between certain species was not always direct (i.e., whole body to whole body). Eggs of fish contained notable concentrations of PFOS, suggesting oviparous transfer of PFOS. In the study by Martin *et al*., (2004b), a benthic invertebrate/pelagic invertebrate-three forage fish (whole body analyses of alewife, slimy sculpin, rainbow smelt)-top predator fish (lake trout) food chain resulted in a multi-trophic level BMF of 5.88. Martin *et al.* (2004b) noted that the benthic invertebrate and its predator fish (sculpin) had higher concentrations of PFOS than the lake trout. Martin *et al.* (2004b) also suggested that bioaccumulation was occurring at the top of the food web for PFOS and all perfluoroalkyl substances (except for PFOA). Tomy *et al*., (2004) suggested that PFOS biomagnifies through the Arctic marine food web. Again, it is noted that comparison of PFOS concentrations between certain species was not always direct (i.e., whole body to whole body).

The trophic level BMF for PFOS included walrus (liver) –clam (whole body) (4.6); narwhal (liver) –cod (whole body) (7.2); beluga (liver)-cod (whole body) (8.4); beluga (liver) –redfish (liver) (4.0); black-legged kittiwake (liver) –cod (whole body) (5.1); glaucous gull (liver) - cod (whole body) (9.0); and cod (whole body)-zooplankton (whole body) (0.4). Smithwick *et al*. (2005a) stated that polar bears as apex predators had high PFOS concentrations in their liver tissue suggesting food-chain accumulation. Concentrations of PFOS found in samples for East Greenland (mean = 2,470 $ng.g^{-1}$ ww) were similar to Hudson Bay (mean = 2730 $ng.g^{-1}$ as reported by Smithwick *et al*., (2005a); 3,100 $ng.g^{-1}$ as reported by Martin *et al*., (2004a) and both populations had significantly greater concentrations than those reported for Alaska (350 $ng.g^{-1}$ from Giesy *et al*., 2002).

The possibility of PFOS bioaccumulation in migratory birds is also a concern because migratory species, such as loons, ospreys, and cormorants, could be exposed to higher concentrations of PFOS while wintering in the US before migrating to Canada where they could experience reproductive and other effects during the breeding season. It is assumed that the main route of exposure to PFOS for birds is through the diet. The dietary exposure route is particularly relevant because biomagnification of PFOS in bird tissues can occur this way. BMFs above one are reported for several bird species (eider duck, red-throated loon, razorbill, long-tailed duck) collected in the Gulf of Gdansk (Gulkowska *et al.* 2005). In the water-algae-zebra mussel-round goby-smallmouth bass-bald eagle food chain Kannan *et al*., (2005a) suggested a PFOS BMF of 10 to 20 in bald eagles (relative to prey items). Tomy *et al*., (2004) suggested that PFOS biomagnifies through the Arctic marine food web: the trophic level BMF for PFOS included black-legged kittiwake-cod (5.1) and glaucous gull-cod (9.0). There is information indicating that PFOS has relatively shorter half-lives in blood and liver tissue in birds compared to mammals (Newsted *et al*., 2005). For example, the estimated elimination half-life for PFOS from serum is 13.6 days in male mallards whereas in male rats it is greater than 90 days. In addition, a recent study suggests that PFOS is excreted relatively rapidly from birds (Kannan *et al*., 2005). However, if birds are chronically exposed to PFOS in their diet, then biomagnification can still occur because, as pointed out in the Kannan study, the binding of perfluorinated compounds to proteins and retention by enterohepatic circulation are the major factors that determine accumulation and retention in biota.

Whole body aquatic BCFs are below 5000. However, the weight of evidence from laboratory and field-based whole body and tissue-specific BCFs and BAFs in conjunction with the field-

based BMFs (avian and aquatic) indicates that PFOS is a bioaccumulative substance.

3.0 ENVIRONMENTAL CONCENTRATIONS

Air

Martin *et al*. (2002) measured the air in Toronto and Long Point, Ontario for some precursors of PFOS. They found an average N-MeFOSE alcohol concentration of 101 pg.m^3 in Toronto and 35 pg.m^3 at Long Point. The average concentrations of N-EtFOSE alcohol were 205 in Toronto and 76 pg.m^3 in Long Point. 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.

Water

In June 2000, PFOS was detected in surface water as a result of a spill of fire-fighting foam from the Toronto International airport into nearby Etobicoke Creek. Concentrations of PFOS ranging from <0.017 to 2210 µg.L^{-1} 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). Boulanger *et al.* (2004, 2005) examined concentrations of PFOS in the Great Lakes. Boulanger *et al.* (2004) analyzed PFOS in 16 water samples taken at 4 meters depth from 4 sampling sites in each of Lake Erie and Lake Ontario. They found measured arithmetic mean concentrations of 31 (sd = 6.9) ng.L^{-1} for Lake Erie and 54 (sd = 18) ng.L^{-1} for Lake Ontario. The highest value measured was 121 ng.L^{-1}. A comparison to worldwide surface water concentrations by Boulanger *et al*. (2004) showed the data to be in a similar range. In a follow up study, Boulanger *et al.* (2005) calculated steady state concentrations of PFOS in Lake Ontario using a mass balance approach of 32 ng.L^{-1} (sd = 14). It was noted in the mass balance study that inflow from Lake Erie and waste water discharges were the primary sources of PFOS to Lake Ontario with particle and gas phase deposition being a negligible portion of the annual inputs. It should be noted that the relative standard deviation on the annual mass flux from waste water discharge is greater than 100%. Therefore, the exact contribution of waste water discharge is inconclusive. Also the amount of PFOS formed from degradation of PFOS precursors is unclear. While it is expected that PFOS precursors are globally distributed within the atmosphere and will primarily enter ecosystems through wet and dry deposition, the work of Boulanger *et al*. (2005) suggests that there is the potential for point sources of PFOS to outweigh atmospheric deposition at specific sites. However, global distribution of PFOS precursors and degradation to PFOS in the

water column is still considered to be the primary route of entry of PFOS into non-industrially impacted freshwaters in Canada.

US data for PFOS are available from one study of six cities. PFOS was detected in quiet water (i.e., a pond) (2.93 $\mu g.L^{-1}$) and sewage treatment effluent (0.048–0.45 $\mu g.L^{-1}$) and sludge (60.2–130 $\mu g.kg^{-1}$ dry sludge) at cities (Port St. Lucie, Florida, and Cleveland, Tennessee) with no significant fluorochemical activities (US EPA OPPT AR226-1030a111). The Port St. Lucie surface water data for PFOS shows a decreasing trend in concentration (from 51.1 $\mu g.L^{-1}$ in 1999 to 1.54 $\mu g.L^{-1}$ in 2001). Therefore, the data may represent a single contamination event to that water system and the decreasing trend may be a result of natural removal processes. PFOS was also detected in drinking water (0.042–0.062 $\mu g.L^{-1}$), surface water (not detected [n.d.] to 0.08 $\mu g.L^{-1}$), sediments (n.d to 0.78 $\mu g.kg^{-1}$ dry sediment), sewage treatment effluents (0.04–5.29 $\mu g.L^{-1}$) and sludge (57.7–3120 $\mu g.kg^{-1}$) and landfill leachate (n.d. to 53.1 $\mu g.L^{-1}$) of four cities that have manufacturing or industrial use of fluorochemicals. Detection limits were 0.0025 $\mu g.L^{-1}$ for water and 0.08 $\mu g.kg^{-1}$ wet weight (ww) 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.

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 $\mu g.L^{-1}$) and sediment (5930 $\mu g.kg^{-1}$ ww; 12 600 $\mu g.kg^{-1}$ dry weight (dw)) 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 $\mu g.L^{-1}$ and 0.18 $\mu g.kg^{-1}$, 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 $\mu g.L^{-1}$. This may indicate an unidentified source of PFOS entering upstream.

PFOS was also detected in oceanic waters from the Pacific and Atlantic Oceans and in several coastal seawaters from Asian countries (Japan, Hong Kong, China, and Korea) (Yamashita *et*

al., 2005). PFOS was detected at concentrations ranging from 1.1 - 57 700 pg.L^{-1}. PFOS was also observed in the North Sea (estuary of the river Elbe, German Bight, southern and eastern North Sea) (Caliebe *et al*., 2004). The detection of PFOS in oceanic waters suggests another potential long-range transport mechanism to remote locations such as the Canadian Arctic.

Sediment

Suspended sediment samples were collected annually at Niagara-on-the-Lake in the Niagara River over a 22 year period (1980-2002). PFOS concentrations ranged from 5 to 1100 pg.g^{-1} (Furdui *et al.,* 2005, unpublished data). Preliminary findings suggest that PFOS concentrations increased during the study period from < 400 pg.g^{-1} in the early 1980s to > 1000 pg.g^{-1} in 2002. It was suggested that the presence of PFOS could be due to the fact that the Great Lakes region is heavily industrialized and hazardous waste disposal sites among other sources could potentially contribute to the contamination of Niagara River suspended sediments.

Biota

Appendix 2 presents the levels of PFOS measured in North American and circumpolar wildlife between 1982 and 2005. Recent Canadian Arctic and circumpolar wildlife surveys have detected PFOS and other perfluorinated acids in mammals, birds and fish, including: polar bear, ringed seals, mink, arctic fox, common loons, northern fulmars, black guillemots and fish from various locations in the Canadian Arctic (Martin *et al.* 2004a; Smithwick *et al*. 2005a,b). Data are also available for a variety of other species worldwide, including dolphin, turtles, mink, seals, fish-eating birds and oysters (Geisy and Kannan 2002; Kannan *et al.* 2002a,b).

In Canada, PFOS has been detected in mid- and higher trophic level biota such as fish, piscivorous birds, and Arctic biota far from known sources or manufacturing facilities. Maximum levels of PFOS in liver of Canadian Arctic biota have been reported for mink (20 µg.kg^{-1}), brook trout (50 µg.kg^{-1}), seal (37 µg.kg^{-1}), fox (1400 µg.kg^{-1}) and polar bear (>4000 µg.kg^{-1}) (Martin *et al*. 2004a).

The highest North American or circumpolar concentration of PFOS in mammal tissue reported in the published literature is 59 500 µg.kg^{-1} ww in mink liver from USA (Kannan *et al*., 2005a). The widespread occurrence of PFOS in wildlife worldwide and, in particular, the high concentrations detected in higher trophic level wildlife and the apex predator species, the polar bear, are important findings. Smithwick *et al*. (2005b) reported concentrations of PFOS in a

study of polar bears from 7 circumpolar locations (5 North American and 2 European locations). The highest PFOS concentration in Canadian polar bears was 3770 $\mu g.kg^{-1}$ liver (range 2000-3770 $\mu g.kg^{-1}$ liver; mean 2730 $\mu g.kg^{-1}$ liver) found in polar bear from South Hudson Bay (Smithwick *et al*., 2005b). This data was a re-analysis of polar bear samples from South Hudson Bay conducted by Martin *et al*. (2004a) which reported concentration in polar bear liver ranging from 1700->4000 $\mu g.kg^{-1}$ liver (mean = 3100 $\mu g.kg^{-1}$ liver, n = 7). The concentrations of PFOS in polar bear liver from the 3 other Canadian locations were: High Arctic 263-2410 $\mu g.kg^{-1}$ liver, mean = 1170; Northwest Territories 982-2160 $\mu g.kg^{-1}$ liver, mean = 1320 and South Baffin Island 977-2100 $\mu g.kg^{-1}$ liver, mean = 1390, respectively (Smithwick *et al*., 2005b). Polar bears have a very large home range due to their dependence on sea ice for hunting, long-range movements and breeding (Stirling and Derocher 1993). The home range may be 103 000 to 206 000 km^2 (Ferguson *et al*., 1999) and young bears may travel up to 1000 km from their mother to establish their home range. Given the very large size of the home range of polar bears, the concentrations in polar bear may reflect integration of exposure over a large geographic area.

PFOS is found in birds worldwide, including birds in Canada and North America (see Appendix 4). PFOS has been found in eagles in the Great Lakes, mallards in the Niagara River, loons in northern Quebec, gulls in the Arctic and in Canadian migratory species from the United States (e.g., common loon in North Carolina). In Canadian or Canada-US migratory species, concentrations have been measured in liver ranging from not detectable to 1780 ppb mean liver PFOS concentration (loon (northern Quebec) and bald eagle, (Michigan)), in blood plasma ranging from <1- 2220 ppb blood plasma in bald eagles and in eggs and egg yolk ranging from 21-220 ppb in double-crested cormorant in Manitoba. In several monitoring studies, PFOS residues in piscivorous water birds were found to have some of the highest liver and serum PFOS concentrations compared to other species (see Newsted *et al.,* 2005). In a study of birds in the Niagara River Region, piscivorous birds (common merganser, bufflehead) contained significantly greater PFOS concentrations than non-piscivorous birds (Sinclair *et al*., 2005). Preliminary data on temporal trends show an increase in bird PFOS concentrations, in two Canadian Arctic species (thick-billed murres and northern fulmars) from 1993 to 2004 (Butt *et al*., 2005, unpublished). It is noted that concentrations of PFOS in plasma have been reported in eagle, gulls and cormorants around the Great Lakes and in the Norwegian Arctic ranging from <1 ppb to 2220 ppb. There are no available studies reporting on blood-serum-plasma relationships in wildlife, therefore, it is unclear how concentrations in bird plasma compare to concentrations of PFOS in bird serum. It is also noted that while effect levels are not available

on egg or egg yolk basis, the measured concentrations of PFOS in egg or egg yolk of birds in Canada and North America have been reported to range from 21 to 220 ppb.

Worldwide, concentrations of PFOS in plaice (*Pleuronectes platessa*) liver (7760 $\mu g.kg^{-1}$) from the Western Scheldt estuary (southwestern Netherlands) and ornate jobfish (*Pristipomoides argyrogrammicus*) liver (7900 $\mu g.kg^{-1}$) from Kin Bay (Japan) are among the highest PFOS concentrations ever reported in wildlife (fish) (Hoff *et al*. 2003; Taniyasu *et al*. 2003). These high concentrations may be due to the proximity of a PFOS manufacturing plant (upstream of estuary) and an army base (Kin Bay, Japan) that may use PFOS in fire-fighting operations.

4.0 KEY TOXICOLOGICAL STUDIES

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. Effects in laboratory mammals include: histopathological effects, increased tumor incidence, hepatocellular adenomas, hepatocellular hypertrophy, increased liver, kidney, brain and testes weight, reduced body weight, change in estrous cycling, changes in levels of neurotransmitters, decreased serum cholesterol, decreased bilirubin, and decreased triiodothynine. In mammalian reproduction studies, effects include: decreased body weight of dams, reduced gestation time, delivery time and live litter size, transfer of PFOS to fetus and neonate via placenta and ingestion of maternal milk, and reduced survival, body weight gain and development of lactation in offspring of exposed females. These effects are more fully reported in Health Canada (2004). Previous studies have shown that perfluorinated compounds are peroxisome proliferators (Berthiaume and Wallace 2002) and tumor promoters and may inhibit gap junction intercellular communication at environmentally relevant concentrations (Hu *et al*., 2002).

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 2002a). Additional studies by Boudreau *et al*. (2003a,b) and Sanderson *et al*. (2002) not available in OECD (2002a) are also summarized.

Aquatic

A flow-through bioconcentration study with bluegill (*Lepomis macrochirus*) using PFOS potassium salt saw no significant mortality at an exposure concentration of 0.086 $mg.L^{-1}$ over a 62-day uptake phase; however, significant mortality was observed after a 35-day exposure to 0.87 $mg.L^{-1}$. The study was stopped because all the fish either had died or had been sampled (US EPA OPPT AR226-1030a042).

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*. 2003a). NOEC values were generated from the most sensitive endpoints for all organisms. The most sensitive of the organisms in this study was *D. magna*, with a 48-hour immobility NOEC of 0.8 $mg.L^{-1}$; the accompanying LC_{50} was 112 $mg.L^{-1}$, and the 48-hour IC_{50} for growth inhibition was 130 $mg.L^{-1}$. The 21-day NOEC for lethality for *D. magna* was 5.3 $mg.L^{-1}$. Autotroph inhibition of growth NOEC values were 5.3 $mg.L^{-1}$, 6.6 $mg.L^{-1}$ and 8.2 $mg.L^{-1}$ for *S. capricornutum*, *L. gibba* and *C. vulgaris*, respectively.

In an aquatic microcosm study (Boudreau *et al*. 2003a), 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 $mg.L^{-1}$ was determined for the 35-day study. The most sensitive taxonomic groups, Cladocera and Copepoda, were virtually eliminated in the 30 $mg.L^{-1}$ 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 $mg.L^{-1}$ over 14 days; several species were significantly reduced or eliminated (Sanderson *et al*. 2002). In comparison with controls, exposures of 10 $mg.L^{-1}$ and 30 $mg.L^{-1}$ 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 $mg.L^{-1}$.

A fathead minnow (*Pimephales promelas*) embryo-juvenile flow-through chronic study determined a NOEC of 0.3 $mg.L^{-1}$ over a 42-day exposure period. This value was for both

survival and growth (US EPA OPPT AR226-0097). In acute tests, the lowest 96-hour LC_{50} for freshwater fish species was 4.7 $mg.L^{-1}$ for the fathead minnow (*P. promelas)*. In salt water, a 96-hour LC_{50} of 13.7 $mg.L^{-1}$ was reported for rainbow trout (*O. mykiss*) (OECD 2002a). In a 96-hour static acute study using the freshwater mussel (*Unio complamatus*), the NOEC for mortality was 20 $mg.L^{-1}$ and the LC_{50} was 59 $mg.L^{-1}$ (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 $mg.L^{-1}$ (US EPA OPPT AR226-0101). In acute toxicity testing, a 96-hour LC_{50} of 3.6 $mg.L^{-1}$ was reported for mysid shrimp (OECD 2002a). 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 $mg.L^{-1}$. The LC_{50} for mortality was 13.8 $mg.L^{-1}$, the EC_{50} for malformed embryos was 12.1 $mg.L^{-1}$ and the NOEC for embryo malformation was 5.2 $mg.L^{-1}$. 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.

The fathead minnow early life stages study has one of the lowest NOEC values (0.3 $mg.L^{-1}$, Klimisch ranking of 1) (OECD, 2002a). However, a recent study by Macdonald *et al.* (2004), although ranked 2 on the Klimisch scale, calculated NOEC values that are lower. MacDonald *et al*. (2004) reported a 10 day NOEC of 0.0491 $mg.L^{-1}$ for the growth and survival of the aquatic midge (*Chironomus tentans*). The Klimisch ranking of 2 was determined for this study for two main reasons: (i) the use of static renewal exposures every 48 hrs and, (ii) the measurement of concentrations at the end of the study period (as opposed to after each 48 hour renewal). However, there was good agreement between the nominal and measured concentrations for the 10-day study. Also since PFOS is not a volatile substance, losses due to volatilization are considered negligible. Therefore, there is high confidence in the 10-day exposure values while the 60-day exposures should be treated with caution. As such, the 10-day NOEC from the MacDonald *et al* (2004) study was chosen as the most appropriate CTV for aquatic organisms.

Terrestrial Invertebrates

The OECD (2002a) 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 $mg.kg^{-1}$ body weight (bw), with a 95% confidence interval of 316–440 $mg.kg^{-1}$ bw. The 14-day No Observed Effect Concentration (NOEC) for burrowing behaviour, body weight and clinical signs of toxicity was 77 $mg.kg^{-1}$ bw, and the 14-day LOEC for the same endpoints was 141 $mg.kg^{-1}$ bw.

Wildlife

Avian

Studies on the effect of PFOS on birds include chronic studies on mallard (*Anas platyrhynchos)* and bobwhite quail (*Colinus virginianus*) (US EPA OPPT AR 226-1738 and AR226-1831) and acute studies on mallard, bobwhite quail and Japanese quail (*Coturnix coturnix japonica*) (US EPA OPPT AR226-0103 and 104, McNabb *et al*. 2005). Given the persistent nature of PFOS, effects from chronic exposure are of particular interest in this assessment and are detailed in Appendix 3.

Mallard and bobwhite quail were exposed to PFOS in feed for 21 weeks and a variety of endpoints examined including changes in: adult body and organ weights, feed consumption rate, fertility, hatchability, and offspring survival. Mallards were exposed to PFOS at dietary concentrations of 0, 10, 50 and 150 ppm for up to 21 weeks (US EPA OPPT AR 226-1735). Due to signs of overt toxicity, adult mallards in the 50 and 150 ppm treatments were euthanized at the end of 7 and 5 weeks, respectively. At 10 ppm, there was an increase in the incidence of small testes size and decreased spermatogenesis in adult males. At 10 ppm, there were no statistically significant treatment-related effects on adult body weight, feed consumption, fertility, hatchability or offspring health or survival compared to controls. Concentrations in serum and liver at the 10 ppm treatment group were 87.3 $\mu g.mL^{-1}$ and 60.9 $\mu g.g^{-1}$ wet weight livers, respectively. No effects were observed for female mallards and offspring for the 10 ppm treatment group. For the 10 ppm treatment group, concentrations in serum and liver of adult females were 76.9 $\mu g.mL^{-1}$ serum (at 5 weeks), 16.6 $\mu g.mL^{-1}$ (at 21 weeks) and 10.8 $\mu g.g^{-1}$ wet weight liver, respectively. The difference in concentration of PFOS in serum of females between

5 weeks and 21 weeks may likely reflect maternal transfer of PFOS to egg or hatchling. Concentrations in liver and serum in males are appropriate basis for developing liver-based ENEVs.

Reduction in testes size is commonly mediated by reduced circulating testosterone which is also responsible for the development and maintenance of the testicular ultrastructure, seminiferous tubule differentiation, the excurrent ducts and numerous secondary sexual characteristics including reproductive behaviour, territorial defense, and courtship singing (Mineau and Shutt, 2005). None of these potential effects were included in the AR226-1738 study. Also in this study, male mallards were housed in groups with multiple females greatly simplifying the process of attaining a female and copulating compared to birds in wild condition. As a result, effects of reduced testosterone production in exposed birds may be masked in this experimental design. While post-reproductive testicular regression does occur in male birds, there is some uncertainty about the ecological significance of this effect. From the study summary, it is difficult to ascertain how long after reproduction was completed that the birds were sacrificed, which would have an effect on the level of testicular regression. Therefore, changes in testes size and testicular regression are considered endpoints of interest, albeit with some uncertainty as to the impact on bird population.

Northern bobwhite quails were also exposed to PFOS at dietary concentrations of 0, 10, 50 and 150 ppm for up to 21 weeks (US EPA OPPT 226-1831). As in mallard, signs of overt toxicity were observed at the 50 and 150 ppm treatments and those tests were terminated at the end of 7 and 5 weeks, respectively. At 10 ppm in diet, minor overt signs of toxicity were observed in adults, there was a statistically significant increase in liver weight (females) an increase in the incidence of small testes size (males), and a statistically significant reduction in survivability in quail chicks as a percentage of eggs set ($p < 0.05$). The increase in the number of adult males in the 10 ppm treatment group with reduced testes size was not accompanied by any morphological change in spermatogenesis. Additionally, there were slight, but not statistically significant treatment-related reductions in fertility, and hatchability. At 10 ppm in diet, there were no PFOS-related effects on adult body weight or feed consumption. Based on these effects, reproductive effects in bobwhite quail was determined to be 10 ppm PFOS in feed, based on 21 weeks of exposure. Concentrations in serum and liver of adult females was 84 $\mu g.mL^{-1}$ serum (at 5 weeks) 8.7 $\mu g.mL^{-1}$ (at 21 weeks) and 4.9 $\mu g.g^{-1}$ wet weight liver, respectively and in adult males 141 $\mu g.mL^{-1}$ and 88.5 $\mu g.g^{-1}$, respectively. As in mallard, the difference in concentration

of PFOS in serum of females between 5 weeks and 21 weeks may likely reflect maternal transfer of PFOS to eggs.

The effect of 10 ppm in diet of birds includes effects of smaller testes size and decreased spermatogenesis in mallard and on survivability of hatchlings, increased liver weight in females and decreased testes size in males in quails. The CTV of 10 ppm is associated with concentration in serum and liver of mallard males at the end of the test of 87.3 $\mu g.mL^{-1}$ and 60.9 $\mu g.g^{-1}$, respectively. Concentrations in male quail serum and liver at the end of the test are comparable.

McNabb *et al*. (2005) studied the acute effect of PFOS on thyroid function in bobwhite and Japanese quail. Adult quail were dosed orally with 5 $mg.kg^{-1}$ body weight and sampled at 7 days (bobwhite quail) or at 7 and 14 days (Japanese quail). At these sampling times in both species, plasma thyroid hormones (both T4 and T3) were decreased indicating organismal level hypothyroidism. Body weights tended to be decreased and relative thyroid weights tended to be increased, the latter effect suggesting some hypothalamic- pituitary-thyroid axis response to decreased circulating thyroid hormones. The authors commented that thyroid gland-thyroid hormone content was much less affected by PFOS treatment in bobwhite quail than would have been expected based on the degree of circulating thyroid hormone depression that was observed. In Japanese quail, thyroid gland-thyroid hormone content was decreased at 7 days of exposure but showed some recovery by 14 days of exposure compared to controls for these same times.

Acute effects in dietary studies of PFOS in juvenile mallard (*Anas platyrhynchos*) and bobwhite quail (*Colinus virginianus*) are available (US EPA OPPT AR 226-0103, AR 226-0953) and examined mortality, growth, behaviour, and feed consumption. For mallards, the 8 day dietary LC_{50} was 603 $mg.kg^{-1}$ feed. The No-Observed-Adverse-Effect-Level (NOAEL) based on lethality was 141 $mg.kg^{-1}$ feed. The NOAEL based on reductions in body weight and feed consumption was 35 $mg.kg^{-1}$ feed. For bobwhite quail, the 8 day dietary LC_{50} was 212 $mg.kg^{-1}$ feed. The NOAEL based on lethality was 70.3 $mg.kg^{-1}$ feed.

Mammalian

Given the lack of ecotoxicological studies using wild species, studies on laboratory mammals were used in this assessment as surrogates for wildlife mammals. Key mammalian toxicity

studies are described by Health Canada (2004). A set of CTVs for mammal (liver) and bird (serum and liver) have been selected, as summarized in Appendices 2 and 3. A 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 Lowest-Observed-Effect Levels (LOELs) of 40.8 mg/kg in liver and 13.9 mg/L in serum.

Further supporting evidence for a No-Observed-Effect-Concentration (NOEC) in the low $mg.kg^{-1}$ or $mg.L^{-1}$ range in liver and sera includes results from a two-generation rat study (US EPA OPPT AR226-0569). A 2 generation rat study with PFOS administered by oral gavage reported a NOEC at the dose 0.1 $mg.kg^{-1}$ bw/day at which concentration in liver and sera were 14.4 $mg.kg^{-1}$ and 5.3 $mg.L^{-1}$, respectively (US EPA OPPT AR226-0569). The Lowest-Observed Effect-Concentration (LOEC) at dose 0.4 $mg.kg^{-1}$ bw/day was associated with reduced dam body mass. At the LOEC the concentration in liver and sera were 58 $mg.kg^{-1}$ and 19 $mg.L^{-1}$, respectively.

Cynomolgus monkeys administered PFOS for 26 weeks at 3 dose levels were observed to have thymic atrophy (females), and reduced high density lipoprotein, cholesterol, triiodothyronine, total bilirubin levels (males) (Covance Labs 2002a). The LOEL dose was 0.03 $mg.kg^{-1}$ bw/day at which average mean female and male concentrations in sera and liver were 19.8 $\mu g.g^{-1}$ and 14.5 $\mu g.mL^{-1}$, respectively.

Mode of Action

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.

5.0 RISK QUOTIENT ANALYSES

Risk quotient analyses, integrating known or potential exposures with known or potential adverse environmental effects, were performed for PFOS. An analysis of exposure pathways and subsequent identification of sensitive receptors were used to select environmental assessment endpoints (e.g., reduced body weight gain, increased offspring mortality, reductions in development, adverse histopathological effects in mammals etc.). For each endpoint, an EEV was selected based on empirical data from monitoring studies. Monitoring data from the Canadian environment were used preferentially for EEVs, but data from US or other countries were considered to supplement available Canadian data and as an indication of potential exposure to this persistent and bioaccumulative substance. EEVs usually represented protective 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. 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. Given the physical-chemical nature of PFOS and its salts, 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. Application factors were derived using a multiplicative approach, which uses 10-fold factors (unless case-specific factors can be estimated) 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, extrapolation from single-species tests to ecosystems and extrapolation from low effect level to chronic no effect level.

Although risk quotients may be used to indicate potential to cause environmental harm for persistent and bioaccumulative substances, risks are likely to be underestimated using traditional quotient approaches. For example, if releases of a persistent substance have continued or increased in recent years, but maximum steady state concentrations have not yet been achieved in the environment, measured EEVs may underestimate possible exposure. In addition, ENEVs may underestimate potential for long term impacts of persistent and bioaccumulative substances

since maximum concentrations are often not reached in the tissues of laboratory organisms, because toxicity test durations are insufficient to achieve steady state. Risk quotients derived for PFOS and its precursors are summarized in Appendices 3, 4, and 5

Mammalian Wildlife

In Canada, the highest mean PFOS concentrations in wildlife were reported in a study of polar bears from 7 circumpolar locations. The highest Canadian concentrations were found in polar bear from South Hudson Bay (range 2000-3770 $\mu g.kg^{-1}$ ww liver, mean 2730 $\mu g.kg^{-1}$ ww liver) (Smithwick *et al*. 2005b). Concentrations in Canadian Arctic polar bear are among the highest in polar bears worldwide but the exposure concentrations are not considered an anomaly given similar concentrations in polar bears in other North America and European Arctic locations and higher concentrations in other wildlife globally (e.g., fish in Japan and the Netherlands). Given the relatively small sample size, which suggests further sampling could identify higher concentrations, and the fact that the species is a top level predator, the maximum exposure concentration in Canadian polar bear liver was considered appropriate for use in the risk quotient calculation.

In the assessment of risk to Canadian wildlife, the exposure concentration of 3770 $\mu g.kg^{-1}$ ww liver from the Canadian South Hudson Bay polar bear was used as the EEV for wildlife. The CTV for mammalian wildlife 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 $mg.kg^{-1}$ bw per day and 0.07–0.21 $mg.kg^{-1}$ bw per day, respectively (Covance Laboratories Inc. 2002). Average values were determined for males and females, to establish corresponding LOELs of 40.8 $\mu g.g^{-1}$ in liver with an application factor of 100 to give an ENEV of 0.408 $ug.g^{-1}$ liver. A risk quotient of 9.2 was, therefore, calculated using the maximum exposure concentration of 3770 $\mu g.kg^{-1}$ ww liver from the South Hudson polar bear (Appendix 5). However, other risk quotients were also calculated given the range of toxicological endpoints but with the same maximum exposure concentration of 3770 $\mu g.kg^{-1}$ ww liver from South Hudson Bay polar bear. These risk quotients were consistently above 1 ranging from 2.1 to 19 (Appendix 4). While the maximum concentration of PFOS in South Hudson polar bears is 3770 $ug.kg^{-1}$ ww liver and risk quotients are calculated using this value, the mean concentration from this population is 2730 $ug.kg^{-1}$ ww liver and risk quotients would, therefore, be approximately 27 % lower but within the same order of magnitude as risk quotients calculated using the maximum concentration. It is also noted that mean PFOS concentration in polar bear liver from 3 other Canadian locations (High Arctic, Northwest Territories, and South Baffin Island) ranged

from 1170 – 1390 μg.kg^{-1}. Risk quotients derived using the 2 year rat CTV results and mean concentrations resulted in quotients ranging from 2.9 to 3.4.

It is noted that the maximum concentration in East Greenland polar bear was higher (6340 μg.kg^{-1}) than the South Hudson Bay bears and a risk quotient calculated from this value using the same application factors would yield quotient of 15.4. Using the highest tissue concentration (4870 μg.kg^{-1} liver) found in mink in the Midwestern United States would yield a risk quotient (11.9) of the same order of magnitude, which could also be considered relevant to Canadian wildlife in mid-latitudes. The risk quotient analysis indicates that the greatest potential risk from PFOS in the environment occurs in higher trophic level mammals.

Pelagic Organisms

A recent study by Macdonald *et al.* (2004) reported a 10 day NOEC of 0.0491 mg.L^{-1} for the growth and survival of the aquatic midge (*Chironomus tentans*). As such, the 10-day NOEC from the MacDonald *et al* (2004) study was chosen as the most appropriate CTV. An application factor of 10 was applied to account for lab to field variations and an application of 10 was applied to convert an acute endpoint to a chronic endpoint resulting in an ENEV of 0.491 μg.L^{-1}. The EEV chosen for Canadian waters is the highest value measured in the Boulanger *et al.* (2004) study (121 ng.L^{-1} measured in Lake Ontario). The risk quotient is calculated as follows: 0.121 / 0.491 = 0.25 (Appendix 5).

Avian

For avian species, the CTV is based on the effects observed for male mallards in the 10 ppm (feed concentration) treatment group based on 21 weeks of exposure. At this dose, the level of PFOS in serum and liver were 87.3 μg.mL^{-1} and 60.9 μg.g^{-1} ww liver, respectively. Given the uncertainty in using this value (in the absence of a NOAEL) and that the effects in males (reduced testis size and effects on spermatogenesis) may have occurred before the end of study when the PFOS liver concentration was measured, an application factor of 10 is used to account for laboratory to field extrapolation and interspecies variability and an additional application factor of 10 is used to extrapolate from the observed effect level to a NOAEL. Therefore, the estimated no-effect value (ENEV) for PFOS in birds is 0.87 μg.mL^{-1} serum and 0.609 μg.g^{-1} liver. Risk quotients using these ENEVs are compared to EEV for a number of avian species that are native to Canada, including many piscivorous birds and migratory species (see Appendix 3). The range of risk quotients are either above or approaching one which indicate

potential for harm at concentrations observed in native species, including migratory species.

6.0 DISCUSSION

There are special concerns about highly persistent and bioaccumulative substances. Although current science is unable to accurately predict the ecological effects of these substances, they are generally acknowledged to have the potential to cause serious, irreversible impacts. Assessments of such substances must therefore be performed using a protective, preventative and precautionary approach to ensure that such harm does not occur.

Evidence that a substance is persistent and bioaccumulative may itself be a significant indication of its potential to cause environmental harm. Persistent substances remain in the environment for long periods of time, increasing the probability and the duration of exposure. Persistent substances that are subject to long-range transport are of particular concern because they can result in low-level, regional or global contamination. Releases of small amounts of persistent and bioaccumulative substances may lead to relatively high concentrations in organisms over wide areas. Bioaccumulative and persistent substances may also biomagnify through the food chain, resulting in internal exposures for top predators. Since they are widespread, several different persistent and bioaccumulative substances may be present simultaneously in the tissues of organisms, increasing the likelihood and potential severity of harm.

Other information can increase concerns regarding the potential for persistent and bioaccumulative substances to cause environmental harm. For example, there is a particular concern for substances that, based on laboratory toxicity tests, have the potential to harm organisms at low concentrations, and/or have modes of toxic action beyond narcosis. A substance which does not naturally occur in the environment may also have an elevated potential to cause harm as organisms may not have evolved specific strategies for mitigating exposures and effects. Monitoring studies indicating that a substance is widespread in the environment and/or that concentrations have been increasing over time may be an indicator of elevated exposure potential. A substance that is used in Canada in moderate to large quantities (e.g., greater than 1,000 kg/yr) in a variety of locations, and/or if use quantities are increasing, may also be taken as an indicator of elevated exposure potential.

Uncertainties

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 throughout the Canadian Arctic, far from known sources. Environmental pathways of PFOS to biota are not well understood because information on degradation is lacking, and there are relatively few monitoring data on concentrations of various precursors in air, water, effluents and sediment in Canada. While mechanisms of toxic action of PFOS are not well understood, a range of toxicological effects have been reported in a variety of species. Currently, there is limited information on the toxicology of PFOS precursors and the potential for combined or synergistic effects with PFOS.

Persistence

The weight of evidence on the persistence of PFOS, the degradation of precursors to PFOS, and the volatilization and atmospheric transport of the precursors to PFOS, indicate that PFOS has the potential to move in the environment.

PFOS is resistant to hydrolysis, photolysis, microbial degradation and metabolism by vertebrates and is persistent. 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 known sources or manufacturing facilities of PFOS and its precursors. This indicates that PFOS and/or its precursors may undergo long-range transport. 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*. 2003b). 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). Therefore, precursors to PFOS, in addition to contributing to the ultimate loading of this persistent and bioaccumulative substance, also contribute to its widespread occurrence.

Concentrations in Biota

The worldwide and widespread occurrence of PFOS in wildlife and in the Canadian polar bear where high PFOS concentrations have been detected (Martin *et al*. 2004a, Smithwick *et al*. 2005a,b,c) have significant bearing on the conclusions of this assessment. Indications of high concentrations in top predators are of concern. While the sample sizes for the Canadian polar bears are small, the PFOS levels in polar bear liver are corroborated by samples from 6 other circumpolar locations. Eastern Greenland polar bear livers were in the same order of magnitude but had higher concentrations. Since PFOS is known to partition to liver, the availability of field measured concentrations of PFOS in liver tissue which can be compared to toxicological effects in liver at certain liver concentrations are particularly relevant to this assessment and reduces some of the uncertainties that are typical for persistent and bioaccumulative substances. However, risks could still be underestimated if steady state conditions were not achieved in exposed wildlife or in laboratory toxicity tests.

There are no known local sources of PFOS at the sampling site of the South Hudson Bay polar bear and there are no PFOS manufacturing sites in the area. While accidental release from sources such as fire fighting foams cannot be entirely ruled out, it is noted that mean liver PFOS concentrations in polar bears from 7 circumpolar locations were within an order of magnitude of each other, varying only by a factor of 3-4. Most importantly, it is expected that, given the very large home range of polar bear, concentrations in these mammals may reflect integration of exposure over large geographic areas. It is also noted that the concentrations of PFOS in polar bear are 5-10 times higher than the concentration of all other perfluoroalkyl substances. The PFOS concentrations in polar bear liver were also higher than any other previously reported concentrations of persistent organochlorine chemicals (e.g., PCBs, chlordane or hexachlorocyclohexane) in polar bear fat (Martin *et al.,* 2004a).

Bioaccumulation

In vertebrates, PFOS preferentially partitions to proteins in liver and blood. The bioaccumulation potential of PFOS may not be related to the typical mechanisms associated with bioaccumulation in lipid-rich tissues. The weight of evidence considered for bioaccumulation includes both laboratory and field-based BAFs, BCFs, BMFs (avian and aquatic), and data on elimination half-lives in a range of species. Whole-body laboratory BCFs in fish ranged from 690 to 2796 and are below 5000. Tissue-based field BAFs in Canadian biota ranged from 6300 to 125 000. The bioaccumulative tendencies of PFOS, suggested by the BCF/

BAF values, are confirmed by tissue-based field BMF studies (BMF values ranged from 0.4 – 20) indicating the potential for biomagnification. In addition to information on PFOS, estimated BCFs for the precursors n-EtFOSEA and n-MeFOSEA were 5543 and 26 000, respectively.

In Canada, PFOS has been detected in higher trophic level biota and predators such as fish, piscivorous birds (double-crested cormorant), mink, and Arctic biota (polar bear) far from known sources or manufacturing facilities. In Canadian Arctic biota, PFOS concentrations in liver ranged from 20 $\mu g.kg^{-1}$ (mink) to > 4000 $\mu g.kg^{-1}$ (polar bear). Also, predator species such as eagles have been shown to accumulate higher PFOS concentrations than birds from lower trophic levels. Chronic and acute effects of PFOS have been observed in laboratory studies with mallard, Japanese quail, and northern bobwhite quail. Effects noted in the chronic reproductive studies for mallards and bobwhite quail include reduced testicular size in quails and mallards (including altered spermatogenesis), increased liver weight in female quails and reduced 14 day survivability in quail chicks as a percentage of eggs set. The testicular regression is accompanied by a histologically-visible effect on spermatogenesis in the mallard. Effects of PFOS on thyroid function have also been reported. Even with reductions in manufacturing of PFOS by some North American manufacturers, wildlife such as birds can continue to be exposed to persistent and bioaccumulative substances such as PFOS by virtue of their persistence and long-term accumulation. Therefore, the weight of evidence is sufficient to conclude that PFOS and its salts are bioaccumulative.

7.0 CONCLUSION

The presence of PFOS, its salts and its precursors results primarily from anthropogenic activity. PFOS and its salts are extremely persistent in all media and can bioaccumulate and biomagnify in mammals and piscivorous birds. 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 piscivorous birds and mammals; and 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 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.

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

CAS No.	Common name	Chemical name	Molecular formula	PFOS Precursor (Catabol)[b]	PFOS Precursor (expert judgment)
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-octanesulfonic 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 diethanolamine (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-heptadecafluoro-N-methyl-	$C_9H_4F_{17}NO_2S$	Y	Y

CAS No.	Common name	Chemical name	Molecular formula	PFOS Precursor (Catabol)[b]	PFOS Precursor (expert judgment)
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	$C_{12}H_{11}F_{17}NO_6PS_2{\cdot}H_3$	Y	Y

CAS No.	Common name	Chemical name	Molecular formula	PFOS Precursor (Catabol)[b]	PFOS Precursor (expert judgment)
			N		
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
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,	$(C_{23}H_{44}O_2 \cdot C_{21}H_{40}O_2 \cdot C_{19}H_{36}O_2 \cdot C_{14}H_{10}F_{17}N$	could not be modelled	Y

CAS No.	Common name	Chemical name	Molecular formula	PFOS Precursor (Catabol)[b]	PFOS Precursor (expert judgment)
		hexadecyl 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 octadecyl 2-propenoate	$O_4S{\cdot}C_{13}H_{10}F_{15}NO_4S{\cdot}C_{12}H_{10}F_{13}NO_4S{\cdot}C_{11}H_{10}F_{11}NO_4S{\cdot}C_{10}H_{10}F_9NO_4S)_x$		
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-	$(C_{14}H_{10}F_{17}NO_4S{\cdot}C_{13}H_{10}F_{15}NO_4S{\cdot}C_{12}H_{10}F_{13}NO_4S{\cdot}C_{11}H_{10}F_{11}NO_4S{\cdot}C_{10}H_{10}F_9NO_4S{\cdot}(C_2H_4O)_nC_8H_{10}O_3{\cdot}(C_2H_4$	could not be modelled	Y

CAS No.	Common name	Chemical name	Molecular formula	PFOS Precursor (Catabol)[b]	PFOS Precursor (expert judgment)
		oxo-2-propenyl)oxy]poly(oxy-1,2-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	$O)_nC_4H_6O_2)_x \cdot C_8H_{18}S$		
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-hydroxybis(2-methylpropanol)di-	$C_{18}H_{28}Cl_4Cr_2F_{17}NO_9S$	Y	Y

CAS No.	Common name	Chemical name	Molecular formula	PFOS Precursor (Catabol)[b]	PFOS Precursor (expert judgment)
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
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

CAS No.	Common name	Chemical name	Molecular formula	PFOS Precursor (Catabol)[b]	PFOS Precursor (expert judgment)
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	$(C_{23}H_{44}O_2 \cdot C_{21}H_{40}O_2 \cdot C_{14}H_{10}F_{17}NO_4S \cdot C_{13}H_{10}F_{15}NO_4S \cdot C_{12}H_{10}F_{13}NO_4S \cdot C_{11}H_{10}F_{11}NO_4S \cdot C_{10}H_{10}F_9NO_4S \cdot (C_2H_4O)_nC_4H_6O_2 \cdot \text{Unspecified})_x$	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]	$C_{15}H_{10}N_2O_2 \cdot \text{Unspecified}$	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, lauryl methacrylate and 2-[methyl[(perfluoro-C_{4-8}-alkyl)sulfonyl]amino]ethyl methacrylate	$(C_{16}H_{30}O_2 \cdot C_8H_{14}O_2 \cdot C_4H_6O_2)_x$	Y	Y
179005-06-2		Sulfonamides, C_{4-8}-alkane, perfluoro, N-[3-(dimethyloxidoamino)propyl], potassium salts	N/A	could not be modelled	Y

CAS No.	Common name	Chemical name	Molecular formula	PFOS Precursor (Catabol)[b]	PFOS Precursor (expert judgment)
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).
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).

APPENDIX 2 PFOS Concentrations in Selected Wildlife in North America and Circumpolar Regions, 1982-2005

Type	Tissue	Species	Sampling locations	Sampling Date	Reference	PFOS (ppb)[a]				n[b]
						Min	Max	Mean	SD	
Mammal	Liver	Polar bear (*Ursus maritimus*)	East Greenland	January, 1999 – September, 2001	Smithwick *et al*., 2005	911	6340			29[e]
Mammal	Liver	Mink (*Mustela vison*)	Midwestern United States	1999-2000	AR 226-1030a157	93	4870			30
Mammal	Liver	Mink (*Mustela vison*)	Massachusetts	1999-2000	AR 226-1030a157	87	4300			31
Mammal	Liver	Mink (Male)	Kalamazoo River watershed, Michigan, USA	2000-2001	Kannan *et al*., 2005	1280	59500	18000		7
Mammal	Liver	Mink (Female)	Kalamazoo River watershed, Michigan, USA	2000-2001	Kannan *et al*., 2005		41			1
Mammal	Liver	Polar bear (*Ursus maritimus*)	Canadian Arctic	February 2002	Martin *et al*, 2004a	1700	>4000			7
Mammal	Liver	Polar bear (*Ursus maritimus*)	South Hudson Bay (Sanikiluaq)[c]	2002	Smithwick *et al*., 2005	2000	3770[c]			NR
Mammal	Liver	Mink (*Mustela vison*)	South Carolina	1999-2000	AR 226-1030a157	65	3110			9
Mammal	Liver	Polar bear (*Ursus maritimus*)	High Arctic (Resolute, Grise Fjord, and Pond Inlet, NWT)	February – May, 2002	Smithwick *et al*., 2005	263	2410	1170		26[e]
Mammal	Liver	Polar bear (*Ursus maritimus*)	North West Territories	2001	Smithwick *et al*., 2005	982	2160	1320		7
Mammal	Liver	Polar bear (*Ursus maritimus*)	South Baffin Island (Pangnirtung, Qikiqtarjuaq, Iqualuit, and Kimmirut, NWT)[c]	February - May, 2002	Smithwick *et al*., 2005	977	2100[c]	1390		26[e]
Mammal	Liver	Polar bear (*Ursus maritimus*)	Svalbard, Norway		Smithwick *et al*., 2005	756	1990	1290		

Type	Tissue	Species	Sampling locations	Sampling Date	Reference	PFOS (ppb)[a]				n[b]
						Min	Max	Mean	SD	
Mammal	Liver	Bottlenose dolphin (*Tursiops truncates*)	Florida Coastal Waters	September, 1991-March, 2000	Kannan *et al*., 2001a	48.2	1520	489	356	20
Mammal	Liver	Polar bear (*Ursus maritimus*)	Chukchi Sea (Chukchi and Bearing Seas, Alaska)	2001	Smithwick *et al*., 2005	435	1480			7
Mammal	Liver	Arctic fox (*Alopex lagopus*)	Canadian Arctic	March, 2001	Martin *et al*., 2004a	6.1	1400			10
Mammal	Liver	Polar bear (*Ursus maritimus*)	Beaufort Sea, Alaska	1993-2002	Kannan *et al*., 2005	502	1130	793	195	8
Mammal	Liver	Polar bear (*Ursus maritimus*)	Chukchi Sea, Alaska	1994-2002	Kannan *et al*., 2005	137	1020	537	204	27
Mammal	Liver	River otter (*Lutra canadensis*)	Washington and Oregon, USA	1999-2000	AR 226-1030a157	34	994[f]			5
Mammal	Liver	River otter (*Lutra canadensis*)	West Coast, USA	1996-1997	Kannan *et al*., 2001a	33.6	994[f]	329		5
Mammal	Liver	Polar bear (*Ursus maritimus*)	Barrow and other sites in Alaska	1990-2000	AR 226-1030a160	175	678[f]			17
Mammal	Liver	Polar bear (*Ursus maritimus*)	Alaska, USA	December, 1997-June, 1999	Kannan *et al*., 2001a	175	678[f]	350		17
Mammal	Liver	Striped dolphin (*Stenella coeruleoalba*)	Florida Coastal Waters	September, 1994 – July, 1997	Kannan *et al*., 2001a	36.6	388	212		2
Mammal	Liver	Mink (*Mustela vison*)	Louisiana	1999-2000	AR 226-1030a157	40	318			7
Mammal	Liver	Short-snouted spinner dolphin (*Stenella clymene*)	Florida Coastal Waters	June, 1995	Kannan *et al*., 2001a	78.7	168	123	36.3	3
Mammal	Liver	Ringed seal (*Phoca hispida*)	East Greenland (Ittoqqortoormiit)	1999	Bossi *et al*., 2005	13.7	130.5			10

Type	Tissue	Species	Sampling locations	Sampling Date	Reference	PFOS (ppb)[a]				n[b]
						Min	Max	Mean	SD	
Mammal	Liver	Ringed seal (*Phoca hispida*)	East Greenland (Ittoqqortoormiit)	2003	Bossi *et al*., 2005	61.0	130.0			10
Mammal	Liver	Northern fur seal (*Callorhinus ursinus*)	Pribilof Islands, Alaska	1990-2000	AR 226-1030a160	<10	122[f]			13
Mammal	Liver	Northern fur seal (*Callorhinus ursinus*)	Alaska, USA	1995 - 1998	Kannan *et al*., 2001a	<10	122[f]			13
Mammal	Liver	Ringed seal (*Phoca hispida*)	West Greenland (Qeqertarsuaq)	1994	Bossi *et al*., 2005	18.9	77.3			9
Mammal	Liver	Ringed seal (*Phoca hispida*)	East Greenland (Ittoqqortoormiit)	1986	Bossi *et al*., 2005	10.1	71.2			8
Mammal	Liver	Rough-toothed dolphin (*Steno bredanensis*)	Florida Coastal Waters	November, 1995	Kannan *et al*., 2001a	42.8	65.6	54.2		2
Mammal	Liver	Harbor seal (*Phoca vitulina*)	West Coast, USA	1991-October, 1997	Kannan *et al*., 2001a	10.3	57.1	27.1		3
Mammal	Liver	Ringed seal (*Phoca hispida*)	East Greenland (Ittoqqortoormiit)	1994	Bossi *et al*., 2005	14.6	53.2			8
Mammal	Liver	California sea lion (*Zalophis californianus*)	West Coast, USA	August, 1993-November, 1997	Kannan *et al*., 2001a	4.6	49.4	26.6		6
Mammal	Liver	Ringed seal (*Phoca hispida*)	West Greenland (Qeqertarsuaq)	2003	Bossi *et al*., 2005	14.0	49.0			10
Mammal	Liver	Ringed seal (*Phoca hispida*)	Canadian Arctic	Spring 1998	Martin *et al*., 2004a	10	37			10
Mammal	Liver	Ringed seal (*Phoca hispida*)	West Greenland (Qeqertarsuaq)	1999	Bossi *et al*., 2005	14.6	36.7			10
Mammal	Liver	Ringed seal (*Phoca hispida*)	West Greenland (Qeqertarsuaq)	1982	Bossi *et al*., 2005	5.8	23.3			10
Mammal	Liver	Ringed seal (*Phoca hispida*)	Canadian Arctic	2001	Martin *et al*., 2004a	8.6	23			9

Type	Tissue	Species	Sampling locations	Sampling Date	Reference	PFOS (ppb)[a]				n[b]
						Min	Max	Mean	SD	
Mammal	Liver	Pygmy sperm whale (*Kogia breviceps*)	Florida Coastal Waters	August, 1994-February, 2000	Kannan *et al*., 2001a	6.6	23.0	14.8		2
Mammal	Liver	Mink (*Mustela vison*)	Canadian Arctic	Winter 2001; Winter 2002	Martin *et al*., 2004a	1.3	20			10
Mammal	Liver	Narwhale (*Monodon monoceros)*	Cape Dorset	2000	Tomy *et al*., 2004	5.4	17.7	10.9	2.3	5
Mammal	Liver	Beluga whale (*Delphinapterus leucas*)	Grise Fjord	1996	Tomy *et al*., 2004	9.8	15.8	12.6	1.1	5
Mammal	Liver	Southern sea otter (*Enhydra lutris nereis*)	West Coast, USA	February, 1993-October, 1994	Kannan *et al*., 2001a	<5	14.3	8.9		8
Mammal	Liver	Elephant seal (*Mirounga augustirostris*)	West Coast, USA	January, 1991-May, 1997	Kannan *et al*., 2001a	<5	9.8	9.3		5
Mammal	Liver	Walrus (*Odobenus rosmarus*)	Frobisher Bay, Iqualuit	1998	Tomy *et al*., 2004	1.4	3.6	2.4	0.4	5
Mammal	Liver	Northern fur seal (*Callorhinus ursinus*)	West Coast, USA	October, 1997	Kannan *et al*., 2001a			133		NR
Mammal	Kidney	Southern sea otter (*Enhydra lutris nereis*)	West Coast, USA	March, 1993-August, 1994	Kannan *et al*., 2001a	<35				3
Mammal	Brain	Southern sea otter (*Enhydra lutris nereis*)	West Coast, USA	March, 1993-February, 1994	Kannan *et al*., 2001a	<35				2
Mammal	Blood	Bottlenose dolphins (Tursiops truncates)	Charleston, SC	August, 2003	Houde *et al*., 2005	472	3073	1171	93	47
Mammal	Blood	Bottlenose dolphins (*Tursiops truncates*)	Indian River Lagoon, FL	July, 2003	Houde *et al*., 2005	69	2010	462	82	42
Mammal	Blood	Bottlenose dolphins (*Tursiops truncates*)	Sarasota Bay, FL	June, 2003	Houde *et al*., 2005	194	1715	658	131	13
Mammal	Blood	Bottlenose dolphins (*Tursiops truncates*)	Delaware Bay, NJ	September, 2003	Houde *et al*., 2005	232	1240	646	174	5

Type	Tissue	Species	Sampling locations	Sampling Date	Reference	PFOS (ppb)[a]				n[b]
						Min	Max	Mean	SD	
Mammal	Blood	Grey seal (*Halichoerus grypus*)	Baltic Sea	1990-2000	AR 226-1030a160	14	76			16
Mammal	Blood	Polar bear (*Ursus maritimus*)	Alaska, USA	1999	Kannan *et al*., 2001a	26	52[f]	34		14
Mammal	Blood	Polar bear (*Ursus maritimus*)	Barrow and other sites in Alaska	1990-2000	AR 226-1030a160	26	52[f]			14
Mammal	Blood	Grey seal (*Halichoerus grypus*)	Sable Island, Canada	1990-2000	AR 226-1030a160	<13	49			12
Mammal	Blood	Ringed seal (*Phoca hispida*)	Baffin Island, Canada	1990-2000	AR 226-1030a160	<3.13	12			16
Mammal	Blood	Northern fur seal pup (*Callorhinus ursinus*)	Alaska, USA	1995	Kannan *et al*., 2001a	<6	12			19
Mammal	Blood	Ringed seal (*Phoca hispida*)	Northern Baltic Sea (Bothnian Bay)	1998	Kannan *et al*., 2001a			242	142	10
Mammal	Blood	Ringed seal (*Phoca hispida*)	Northern Baltic Sea (Bothnian Bay)	1996	Kannan *et al*., 2001a			133	47	10
Mammal	Blood	Ringed seal (*Phoca hispida*)	Northern Baltic Sea (Bothnian Bay)	1997	Kannan *et al*., 2001a			92	81	9
Mammal	Blood	Gray seal (*Halichoerus grypus*)	Northern Baltic Sea (Bothnian Bay)	1997	Kannan *et al*., 2001a			43.9	19	10
Mammal	Blood	Gray seal (*Halichoerus grypus*)	Northern Baltic Sea (Bothnian Bay)	1996	Kannan *et al*., 2001a			42	21	9
Mammal	Blood	Gray seal (*Halichoerus grypus*)	Sable Island (Canada)	1998	Kannan *et al*., 2001a			27.7	11	12
Mammal	Blood	Gray seal (*Halichoerus grypus*)	Northern Baltic Sea (Bothnian Bay)	1998	Kannan *et al*., 2001a			25.5	9.6	7
Mammal	Blood	Ringed seal (*Phoca hispida*)	Arctic (Spitsbergen)	1998	Kannan *et al*., 2001a			10.1	2.7	8

Type	Tissue	Species	Sampling locations	Sampling Date	Reference	PFOS (ppb)[a]				n[b]
						Min	Max	Mean	SD	
Mammal	Blood	Ringed seal (*Phoca hispida*)	Arctic (Spitsbergen)	1996	Kannan *et al*., 2001a			8.1	2.5	10
Mammal	Blood	Northern fur seal adult (*Callorhinus ursinus*)	Alaska, USA	1995	Kannan *et al*., 2001a	<6				10
Mammal	Blood	Northern fur seal subadult (*Callorhinus ursinus*)	Alaska, USA	1995	Kannan *et al*., 2001a	<6				7
Mammal	Blood	Northern fur seal (*Callorhinus ursinus*)	Alaska, USA	1995	Kannan *et al*., 2001a	<6				8
Mammal	Blood	Steller sea lion	Alaska, USA	1999	Kannan *et al*., 2001a	<6				12
Mammal	Plasma	Loggerhead sea turtle (*Caretta caretta*)	Core Sound (North Carolina), South Carolina. Georgia, and Florida	June-July, 2003	Keller *et al*., 2005	1.4	96.8	11	17.2	73
Mammal	Plasma	Snapping Turtle (*Chelydra serpentina*)	Kalamazoo River watershed, Michigan, USA	1999	Kannan *et al*., 2005	105	169	137		2
Mammal	Plasma	Snapping Turtle (*Chelydra serpentina*)	Kalamazoo River watershed, Michigan, USA	1999	Kannan *et al*., 2005	<1	8.8	6.13		3
Mammal	Plasma	Kemp's Ridley sea turtle (*Lepidochelys kempii*)	Core Sound (North Carolina), South Carolina. Georgia, and Florida	June, 2003	Keller *et al*., 2005	13.8	60.2	39.4	17.1	6
Bird	Liver	Brandt's cormorant	California, USA	June, 1997	Kannan *et al*., 2001b	46	1780			2
Bird	Liver	Red-throated loon	Various Locations, USA	February, 1998-May, 1998	Kannan *et al*., 2001b	34	1120			3
Bird	Liver	Bald Eagle (*Haliaeetus leucocephalus*)	Upper Peninsula of Michigan, USA	2000	Kannan *et al*., 2005	26.5	1740			6
Bird	Liver	White pelican	Various Locations, USA	November, 1996-August, 1997	Kannan *et al*., 2001b	30	1120			6

Type	Tissue	Species	Sampling locations	Sampling Date	Reference	PFOS (ppb)[a]				n[b]
						Min	Max	Mean	SD	
Bird	Liver	Great egret	Various Locations, USA	May, 1996-February, 1998	Kannan *et al*., 2001b	27	1030			7
Bird	Liver	Osprey	Various Locations, USA	September, 1996-October, 1997	Kannan *et al*., 2001b	42	959			4
Bird	Liver	Great blue heron	St. Martinville, Los Angeles, USA	June, 1996	Kannan *et al*., 2001b	162	916			2
Bird	Liver	Great black-backed gull	Carteret County, NC, USA	January, 1998-March, 1998	Kannan *et al*., 2001b	187	841			2
Bird	Liver	Black-crowned night heron	California, USA	June, 1997-May, 1998	Kannan *et al*., 2001b	32	648	393		5
Bird	Liver	Common loon	Various Locations, USA	November, 1997-December, 1998	Kannan *et al*., 2001b	<12	595			19
Bird	Liver	Brown pelican	Various Locations, USA	1997	Kannan *et al*., 2001b	118	533			3
Bird	Liver	Bald Eagle	Various Locations, USA	February, 1995-March, 1997	Kannan *et al*., 2001b	24	467			4
Bird	Liver	Snowy egret	Florida, USA	July, 1997-November, 1997	Kannan *et al*., 2001b	43	413			3
Bird	Liver	Herring gull	Various Locations, USA	October, 1996-February, 1998	Kannan *et al*., 2001b	16	353			5
Bird	Liver	Double crested cormorant	St. Martinville, Los Angeles, USA	June, 1996	Kannan *et al*., 2001b	51	288			2
Bird	Liver	Franklin's gull	Red Rocks Lakes, Beaverhead County, MT, USA	July, 1997-August, 1997	Kannan *et al*., 2001b	<12	61	40		4
Bird	Liver	Glaucous gulls (*Larus hyperboreus*)	Northwater Polynya, Arctic (open water between Canada	April-July, 1998	Tomy *et al*., 2004	9.9	33.2	20.2	3.9	5

Type	Tissue	Species	Sampling locations	Sampling Date	Reference	PFOS (ppb)[a]				n[b]
						Min	Max	Mean	SD	
			and Greenland)							
Bird	Liver	Common loon (*Gavia immer*)	Canadian Arctic	1992	Martin *et al*., 2004a	11	26			5
Bird	Liver	Black-legged kittiwake (*Rissa tridactyla*)	Northwater Polynya, Arctic (open water between Canada and Greenland)	April-July, 1998	Tomy *et al*., 2004	1.2	20	10.0	4.6	4
Bird	Liver	Northern fulmar (*Fulmarus glacialis*)	Canadian Arctic	1993	Martin *et al*., 2004a	1	1.5			5
Bird	Liver	Bufflehead (*Bucephala albeola*)	Niagara River Region, New York, USA	1994-1996; 1999-2000	Sinclair *et al*., 2005			635	281	3
Bird	Liver	Common merganser (*Mergus merganser*)	Niagara River Region, New York, USA	1994-1996; 1999-2000	Sinclair *et al*., 2005			441	154	20
Bird	Liver	Black duck (*Anas rubripes*)	Niagara River Region, New York, USA	1994-1996	Sinclair *et al*., 2005			204	0	1
Bird	Liver	Common goldeneye (*Bucephala clangula*)	Niagara River Region, New York, USA	1994-1996; 1999-2000	Sinclair *et al*., 2005			204	119	20
Bird	Liver	Mallard (*Anas platyrhynchos*)	Niagara River Region, New York, USA	1994-1996	Sinclair *et al*., 2005			172	124	31
Bird	Liver	Lesser scaup (*Aythya affinis*)	Niagara River Region, New York, USA	1999-2000	Sinclair *et al*., 2005			148	65	6
Bird	Liver	Greater scaup (*Aythya marila*)	Niagara River Region, New York, USA	1995-1996	Sinclair *et al*., 2005			82	24	2
Bird	Liver	Hooded merganser (*Lophodytes cucullatus*)	Niagara River Region, New York, USA	1994-1996	Sinclair *et al*., 2005			35	24	2
Bird	Liver	Surf scoter (*Melanitta perspicillata*)	Niagara River Region, New York, USA	1994-1996	Sinclair *et al*., 2005			28	0	1
Bird	Liver	Ring-neck (*Aytha collaris*)	Niagara River Region, New York, USA	1994-1996	Sinclair *et al*., 2005			16	0	1

Type	Tissue	Species	Sampling locations	Sampling Date	Reference	PFOS (ppb)[a]				n[b]
						Min	Max	Mean	SD	
Bird	Liver	Brown pelican (*Pelecanus occidentalis*)	Mississippi	NR	Giesy (2003)	460				NR
Bird	Liver	Common loon (*Gavia immer*)	North Carolina	NR	Giesy (2003)	290				NR
Bird	Liver	Wood stork	Various Locations, USA	September, 1996	Kannan *et al*., 2001b	158				1
Bird	Liver	Northern gannet	Carteret County, NC, USA	March, 1998	Kannan *et al*., 2001b	85				1
Bird	Liver	Laysan albatross (*Diomedea immutabilis*)	Midway Atoll	NR	Giesy (2003)	<35				NR
Bird	Liver	White-faced ibis	Sacramento Valley, CA, USA	January, 1995	Kannan *et al*., 2001b	17				1
Bird	Liver	Black guillemot (*Cepphus grylle*)	Canadian Arctic	1993	Martin *et al*., 2004a	n.d.				5
Bird	Eggs	Double-crested cormorant (*Phalacrocorax auritus*)	Great Lakes	1990-1998	AR 226-1030a159	21	220[f]			4
Bird	Egg Yolk	Double crested cormorant	Manitoba, Canada	June, 1995	Kannan *et al*., 2001b	21	220[f]			4
Bird	Egg	Glaucous gulls (*Larus hyperboreus*)	Bear Island (Norwegian Arctic)	2004	Verreault *et al*., 2005	51.7	196	104	13.2	10
Bird	Egg Yolk	Ring-billed gull	Great Lakes Region (Michigan)	June, 1995	Kannan *et al*., 2001b	30	126			3
Bird	Blood	Double-crested cormorant (*Phalacrocora x auritus*)	Great Lakes Region (Michigan)	July, 1991	Kannan *et al*., 2001b	34	243			8
Bird	Blood	Herring gull	Great Lakes Region (Michigan)	July, 1991	Kannan *et al*., 2001b	57	68			2
Bird	Plasma	Bald eagle (*Haliaeetus*	Michigan, Wisconsin and	1990-1998	AR 226-	<1	2220			33

Type	Tissue	Species	Sampling locations	Sampling Date	Reference	PFOS (ppb)[a]				n[b]
						Min	Max	Mean	SD	
		leucocephalus)	Minnesota		1030a159					
Bird	Plasma	Bald Eagle	Midwestern United States	June, 1990-October, 1993	Kannan *et al*., 2001b	<1	2220			33
Bird	Plasma	Herring gull	Great Lakes Region (Michigan)	July, 1991	Kannan *et al*., 2001b	239	391			2
Bird	Plasma	Double crested cormorant	Great Lakes Region (Michigan)	July, 1991	Kannan *et al*., 2001b	63	372			4
Bird	Plasma	Glaucous gulls (*Larus hyperboreus*)	Svalbard (ice edge) and Bear Island (Norwegian Arctic)	2004	Verreault *et al.*, 2005	48.1	349	134	16.6	20
Bird	Gall Bladder	Bald Eagle (*Haliaeetus leucocephalus*)	Upper Peninsula of Michigan, USA	2000	Kannan *et al*., 2005		1490			1
Bird	Kidney	Bald Eagle (*Haliaeetus leucocephalus*)	Upper Peninsula of Michigan, USA	2000	Kannan *et al*., 2005	35	1480			4
Bird	Muscle	Bald Eagle (*Haliaeetus leucocephalus*)	Upper Peninsula of Michigan, USA	2000	Kannan *et al*., 2005	<7.5	96.2			6
Bird	Ovary	Bald Eagle (*Haliaeetus leucocephalus*)	Upper Peninsula of Michigan, USA	2000	Kannan *et al*., 2005		68.0			1
Bird	Testes	Bald Eagle (*Haliaeetus leucocephalus*)	Upper Peninsula of Michigan, USA	2000	Kannan *et al*., 2005		183			1
Fish	Liver	Striped bass (*Morone saxatilis*)	Tennessee River, Guntersville Dam	June 21-22, 2000	AR 226-1030a161	385	2430			9
Fish	Liver	Chinook salmon (*Oncorhynchus tshawytscha*)	Great Lakes/inland Michigan lakes	Prior to June 2001	AR 226-1030a156	32	173[f]			6
Fish	Liver	Chinook salmon	Webber Dam, Grand River, Michigan	1999-2000	Sinclair *et al*., 2004	32	173[f]			6
Fish	Liver	Various species[d]	Inland Lakes, Michigan	1999-2000	Sinclair *et al*.,	<7.7	120			35

Type	Tissue	Species	Sampling locations	Sampling Date	Reference	PFOS (ppb)[a]				n[b]
						Min	Max	Mean	SD	
					2004					
Fish	Liver	Lake whitefish (*Coregonus clupeaformis*)	Great Lakes/inland Michigan lakes	Prior to June 2001	AR 226-1030a156	33	81[f]			5
Fish	Liver	Lake whitefish	Great Lakes / Thunder Bay, Lake Huron	2003-2004	Sinclair *et al*., 2004	33	81[f]			5
Fish	Liver	Brook trout (*Salvelinus fontinalis*)	Canadian Arctic	July, 2002	Martin *et al*., 2004a	29	50			2
Fish	Liver	Brown trout (*Salmo trutta*)	Great Lakes/inland Michigan lakes	Prior to June 2001	AR 226-1030a156	<17	26[f]			10
Fish	Liver	Brown trout	Great Lakes / Lake Superior / Marquette	2003-2004	Sinclair *et al*., 2004	<17	26[f]			10
Fish	Liver	White sucker (*Catostomus commersoni*)	Canadian Arctic	July, 2002	Martin *et al*., 2004a	6.5	8.6			3
Fish	Liver	Redfish (*Sebastes mentella*)	Davis Strait	October 2000, 2001	Tomy *et al*., 2004	nd	6.3	1.4	0.9	7
Fish	Liver	Lake whitefish (*Coregonus clupeaformis*)	Canadian Arctic	July, 2002	Martin *et al*., 2004a	12				2
Fish	Liver	Lake trout (*Salvelinus namaycush*)	Canadian Arctic	July, 2002	Martin *et al*., 2004a	31				1
Fish	Liver	Northern pike (*Esox lucius*)	Canadian Arctic	July, 2002	Martin *et al*., 2004a	5.7				1
Fish	Liver	Arctic sculpin (*Myoxocephalus scorpioides*)	Canadian Arctic	July, 2002	Martin *et al*., 2004a	12				1
Fish	Muscle	Carp (*Cyprinus carpio*)	Saginaw Bay, Michigan	Prior to June 2001	AR 226-1030a156	59	287			10
Fish	Muscle	Chinook salmon (*Oncorhynchus*	Great Lakes/inland Michigan lakes	Prior to June 2001	AR 226-1030a156	<7	189[f]			6

Type	Tissue	Species	Sampling locations	Sampling Date	Reference	PFOS (ppb)[a]				n[b]
						Min	Max	Mean	SD	
		tshawytscha)								
Fish	Muscle	Lake whitefish (*Coregonus clupeaformis*)	Great Lakes/inland Michigan lakes	Prior to June 2001	AR 226-1030a156	97	168[f]			5
Fish	Muscle	Carp	Saginaw Bay, Michigan	1999-2000	Sinclair *et al*., 2004	59	297			10
Fish	Muscle	Chinook salmon	Webber Dam, Grand River, Michigan	1999-2000	Sinclair *et al*., 2004	<7	189[f]			6
Fish	Muscle	Lake whitefish	Great Lakes / Thunder Bay, Lake Huron	2003-2004	Sinclair *et al*., 2004	97	168[f]			5
Fish	Muscle	Brown trout	Great Lakes / Lake Superior / Marquette	2003-2004	Sinclair *et al*., 2004	<7	46			10
Fish	Whole Body	Arctic cod (*Boreogadus saida*)	Davis Strait	October 2000, 2001	Tomy *et al*., 2004	0.3	4.7	1.3	0.7	6
Fish	Whole Body	Slimy sculpin (*Cottus cognatus*)	Lake Ontario	2001	Martin *et al*., 2004b			450	98	5
Fish	Whole Body	Round Gobies (*Neogobius melanostomus*)	Raisin River, Michigan, USA	October, 1998-September, 1999	Kannan *et al*., 2005	6.6	11.2			3
Fish	Whole Body	Round Gobies (*Neogobius melanostomus*)	St. Clair River, Michigan, USA	September, 1998-July, 1999	Kannan *et al*., 2005	7.7	21.5			8
Fish	Whole Body	Round Gobies (*Neogobius melanostomus*)	Calumet River, Michigan, USA	July, 1999	Kannan *et al*., 2005		4.1			1
Fish	Whole Body	Lake Trout (*Salvelinus namaycush*)	Lake Ontario	2001	Martin *et al*., 2004b			170	64	7
Fish	Whole Body	Rainbow smelt (*Osmerus mordax*)	Lake Ontario	2001	Martin *et al*., 2004b			110	55	6
Fish	Whole Body	Alewife (*Alosa pseudoharengus*)	Lake Ontario	2001	Martin *et al*., 2004b			46	15	6
Fish	Eggs	Lake whitefish	Great Lakes/inland Michigan	Prior to June	AR 226-	145	381[f]			2

Type	Tissue	Species	Sampling locations	Sampling Date	Reference	PFOS (ppb)[a]				n[b]
						Min	Max	Mean	SD	
		(*Coregonus clupeaformis*)	lakes	2001	1030a156					
Fish	Eggs	Lake whitefish	Great Lakes / Thunder Bay, Lake Huron	2003-2004	Sinclair *et al*., 2004	145	381[f]			2
Fish	Eggs	Various species[d]	Inland Lakes, Michigan	1999-2000	Sinclair *et al*., 2004	<7.7	222			19
Fish	Eggs	Brown trout (*Salmo trutta*)	Great Lakes/inland Michigan lakes	Prior to June 2001	AR 226-1030a156	49	75[f]			3
Fish	Eggs	Brown trout	Great Lakes / Lake Superior / Marquette	2003-2004	Sinclair *et al*., 2004	49	75[f]			3
Fish	Skinless Fillets	Smallmouth Bass (*Micropterus dolomieui*)	Raisin River, Michigan, USA	September, 1998-September, 1999	Kannan *et al*., 2005	2.0	41.3			8
Fish	Skinless Fillets	Smallmouth Bass (*Micropterus dolomieui*)	Calumet River, Michigan, USA	May, 1998-August, 1999	Kannan *et al*., 2005	2.5	7.6			4
Fish	Skinless Fillets	Smallmouth Bass (*Micropterus dolomieui*)	St. Clair River, Michigan, USA	October, 1998; August, 1999	Kannan *et al*., 2005	<2	2.7			2
Crustacean	Whole Body	Clams (*Mya truncate; Serripes groenlandica*)	Frobisher Bay	May, 2002	Tomy *et al*., 2004	0.08	0.6	0.28	0.09	5
Invertebrate	Whole Body	Zooplankton (mixed)	Frobisher Bay	May, 2002	Tomy *et al*., 2004	1.1	2.6	1.8	0.3	5
Invertebrate	Whole Body	Shrimp (*Pandalus borealis; Hymenodora glacialis*)	Davis Strait	October 2000, 2001	Tomy *et al*., 2004	nd	0.9	0.35	0.15	7
Invertebrate	Whole Body	Diporeia (*Diporeia hoyi*)	Lake Ontario	2001	Martin *et al*., 2004b			280	33	NR
Invertebrate	Whole Body	Amphipods	Raisin River, Michigan, USA	September, 1999	Kannan *et al*., 2005		2.9			1
Invertebrate	Whole	Amphipods	St. Clair River, Michigan,	September, 1999	Kannan *et al*.,		<2			1

Type	Tissue	Species	Sampling locations	Sampling Date	Reference	PFOS (ppb)[a]				n[b]
						Min	Max	Mean	SD	
	Body		USA		2005					
Invertebrate	Whole Body	Amphipods	Calumet River, Michigan, USA	July, 1999	Kannan *et al*., 2005		<2			1
Invertebrate	Whole Body	Mysis (*Mysis relicta*)	Lake Ontario	2001	Martin *et al*., 2004b			13	8	NR
Crustacean		Crayfish	Raisin River, Michigan, USA	September, 1999	Kannan *et al*., 2005		4.3			1
Crustacean		Crayfish	St. Clair River, Michigan, USA	September, 1999	Kannan *et al*., 2005		2.4			1
Crustacean		Crayfish	Calumet River, Michigan, USA	July, 1999	Kannan *et al*., 2005		3.7			1
Crustacean	Soft Tissue	Zebra Mussel (*Dreissena polymorpha*)	Raisin River, Michigan, USA	October, 1998; August, 1999	Kannan *et al*., 2005	<2	3.1			2
Crustacean	Soft Tissue	Zebra Mussel (*Dreissena polymorpha*)	St. Clair River, Michigan, USA	November, 1998	Kannan *et al*., 2005		<2			1
Crustacean	Soft Tissue	Zebra Mussel (*Dreissena polymorpha*)	Calumet River, Michigan, USA	September, 1998	Kannan *et al*., 2005	<2	<2			3
Algae	Whole Body	Benthic Algae	Calumet River, Michigan, USA	July, 1999	Kannan *et al*., 2005		3.1			1
Algae	Whole Body	Benthic Algae	St. Clair River, Michigan, USA	July, 1999	Kannan *et al*., 2005		2.6			1
Algae	Whole Body	Benthic Algae	Raisin River, Michigan, USA	September, 1999	Kannan *et al*., 2005		2.4			1
Amphibian	Liver	Green Frog (*Rana clamitans*)	Kalamazoo River watershed, Michigan, USA	1998	Kannan *et al*., 2005	50	285	168		2
Amphibian	Liver	Green Frog *(Rana clamitans*)	Kalamazoo River watershed, Michigan, USA	1998	Kannan *et al*., 2005		<35			2

[a] Units are parts per billion (ppb) = $\mu g.kg^{-1}$ for tissue (wet weight unless otherwise noted); $\mu g.L^{-1}$ for liquids.
[b] n = sample size; NR = not reported.
[c] Note that the Smithwick *et al*. (2005) polar bear data from South Hudson Bay and South Baffin Island are samples re-analyzed from Martin *et al*. (2004a)
[d] various species include: Coho salmon, lake trout, white sucker, carp, redhorse sucker, and largemouth bass from several Michigan lakes and rivers (Sinclair *et al*., 2004)
[e] Number of samples analyzed for these subsets of Polar Bear samples is unclear in Smithwick *et al*. (2005)
[f] duplicate data obtained from 2 different sources, however, discrepancies were noted for the sampling dates

APPENDIX 3 RISK QUOTIENTS FOR NORTH AMERICAN MIGRATORY BIRDS [a]

Species[a]/Tissue	Sample Location	EEV (Maximum concentration of PFOS in bird liver (µg.g^{-1} ww liver) or plasma (µg.ml^{-1})	Reference	ENEV[b]	Q
Liver					
Bald eagle (*Haliaeetus leucocephalus*)	Michigan	1.74	Kannan *et al*., 2004	0.609	2.86
Bald eagle (*Haliaeetus leucocephalus*)	Illinois	0.467	Kannan *et al*., 2001	0.609	0.77
Osprey	Florida	0.959	Kannan *et al*., 2001	0.609	1.57
Common loon (*Gavia immer*)	North Carolina	0.595	Kannan *et al*., 2001	0.609	0.98
Common loon (*Gavia immer*)	Northern Quebec	0.026	Martin *et al*., 2004	0.609	0.04
Red-throated loon	North Carolina	1.12	Kannan *et al*., 2001	0.609	1.84
Double-crested cormorant (*Phalacrocorax auritus*)	Louisiana	0.288	Kannan *et al*., 2001	0.609	0.47
Mallard (*Anas platyrhynchos*)	Niagara River	0.425	Sinclair *et al*., 2005	0.609	0.70
Common merganser (*Mergus merganser*)	Niagara River	0.715	Sinclair *et al*., 2005	0.609	1.17
Bufflehead (*Bucephala abeola*)	Niagara River	0.882	Sinclair *et al*., 2005	0.609	1.45

Common goldeneye (*Bucephala clangula*)	Niagara River	0.505	Sinclair *et al*., 2005	0.609	0.83
Lesser scaup (*Aythya affinis*)	Niagara River	0.240	Sinclair *et al*., 2005	0.609	0.39
Brandt's cormorant	California	1.78	Kannan *et al*., 2001	0.609	2.92
Northern fulmar (*Fulmarus glacialis*)	Canadian arctic	0.0015	Martin *et al*., 2004	0.609	0.002
Black guillemot (*Cepphus grylle*)	Canadian arctic	Not detected	Martin *et al*., 2004	0.609	-
Black-legged kittiwake (*Rissa tridactyla*)	Canadian arctic	0.02	Tomy *et al*., 2004	0.609	0.03
Glaucous gulls (*Larus hyperboreus*)	Canadian arctic	0.0332	Tomy *et al*., 2004	0.609	0.05
Great black-backed gull	North Carolina	0.841	Kannan *et al*., 2001	0.609	1.38
Herring gull	North Carolina	0.353	Kannan *et al*., 2001	0.609	0.58
Franklin's gull	Montana	0.061	Kannan *et al*., 2001	0.609	0.10
Black-crowned night heron	California	0.648	Kannan *et al*., 2001	0.609	1.06
Great blue heron	Louisiana	0.916	Kannan *et al*., 2001	0.609	1.50
Great egret	Florida	1.03	Kannan *et*	0.609	1.69

			al., 2001		
White pelican	California	1.12	Kannan *et al*., 2001	0.609	1.84
Serum and Plasma					
Bald eagle (*Haliaeetus leucocephalus*)	Wisconsin	2.220	Kannan *et al*., 2001	0.873	2.54
Double-crested cormorant (*Phalacrocorax auritus*)	Lake Huron	0.372	Kannan *et al*., 2001	0.873	0.43
Herring gull	Lake Huron	0.391	Kannan *et al*., 2001	0.873	0.45

[a] All species presented here are Canada-US migratory species.
[b] An application factor of 100 is used to derive the ENEV from the CTV.

APPENDIX 4: Summary of Data used in Risk Quotient (Q) Analyses of PFOS

Pelagic Organism **Freshwater Midge – Lake Ontario**					**Birds (liver)**					**Birds (serum)**					**Wildlife (Arctic Polar Bear – liver)**				
EEV[a] (µg.L^{-1})	**CTV[b] (µg.L^{-1})**	**AF[c]**	**ENEV (µg.L^{-1})**	**Q (EEV/ ENEV**	**EEV[d] (µg.g^{-1} liver)**	**CTV[e] (µg.g^{-1} liver)**	**AF[f]**	**ENEV (µg.g^{-1} liver)**	**Q (EEV/ ENEV)**	**EEV[g] (µg.mL^{-1} serum)**	**CTV (µg.mL^{-1} serum)**	**AF[f]**	**ENEV (µg.mL^{-1} serum)**	**Q (EEV/ ENEV**	**EEV[h] (µg.g^{-1} liver)**	**CTV[i] (µg.g^{-1} liver)**	**AF[f]**	**ENEV (µg.g^{-1} liver)**	**Q (EEV/ ENEV)**
0.121	49.1	100	0.491	0.25	0.015 - 1.78	60.9	100	0.609	0.002 – 2.92	0.372 – 2.20	87.3	100	0.87	0.43 – 2.54	3.77	40.8	100	0.408	9.2

[a] The highest measured value for Canadian waters of 121 ng.L^{-1} (Lake Ontario) as reported in Boulanger *et al*. (2004)

[b] 10-day NOEC of 0.0491 mg.L^{-1} for the growth and survival of the aquatic midge (*Chironomus tentans*) as reported in MacDonald *et al.* (2004)

[c] An application factor of 100 was applied to account for lab to field variations and to convert an acute endpoint to a chronic endpoint

[d] A range of estimated exposure values in liver for a number of avian species were used (see Appendix 3)

[e] 21 week study (increase in the incidence of small testes size and decreased spermatogenesis) for adult male mallards determined to be 10 ppm PFOS in feed. At this dose, the level of PFOS in liver (ww) was 60.9 µg.g^{-1}

[f] An application factor of 100 applied for extrapolation from laboratory to field conditions and for intraspecies and interspecies variations in sensitivity, and extrapolation from the observed effects level to a no-effect level

[g] A range of estimated exposure values in serum for a number of avian species were used (see Appendix 3)

[h] In Canada, the highest mean PFOS concentrations in wildlife were reported in a study of polar bears from 7 locations. The highest Canadian concentrations were found in polar bear from South Hudson Bay (range 2000-3770 µg.kg^{-1} ww liver, mean 2730 µg.kg^{-1} ww liver) (Smithwick *et al*. 2005). This data was a re-analysis of polar bear samples from South Hudson Bay conducted by Martin *et al*. (2004) which reported concentration in polar bear liver of 1700->4000 µg.kg^{-1} ww liver, mean = 3100 µg.kg^{-1} ww liver.

[i] 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.kg^{-1} bw per day and 0.07–0.21 mg.kg^{-1} bw per day, respectively (Covance Laboratories, Inc. 2002). Average values were determined for males and females, to establish LOELs of 40.8 µg.g^{-1} in liver and 13.9 mg.L^{-1} in serum.

APPENDIX 5: Risk Quotients for PFOS Comparing Concentrations in South Hudson Bay Polar Bears to Effects in Laboratory Mammal Toxicity

Test organism/ study type	Reference	Effect	Critical Effect Level	Concentration in Serum (CTV) ($mg.L^{-1}$ serum)	Concentration in liver at critical effect (CTV) ($\mu g.kg^{-1}$ ww liver)	AF[a]	ENEV ($ug.kg^{-1}$ liver)	EEV[b] ($\mu g.kg^{-1}$ liver)	Q (EEV/ENEV)
Rat receiving 0, 0.5, 2, or 5 ppm PFOS-K salt in diet for 104 weeks (2 years)	Covance 2002	Microscopic changes in liver	LOEL (m/f) = 2.0 ppm diet (0.06 to 0.23 $mg.kg^{-1}$ bw/d)	13.9 $mg.L^{-1}$	40800 (40.8 $\mu g.g^{-1}$)	100	408	3770	9.24
Monkeys administered 0.03, 0.15, 0.75 $mg.kg^{-1}$ bw/d PFOS for 26 weeks	Covance Labs 2002a	Thymic atrophy, reduced serum HDLP, cholesterol, triiodotyhronine, total bilirubin	LOEL (m/f) = 0.03 $mg.kg^{-1}$ bw/d	14.5 $mg.L^{-1}$	19800 (19.8 $\mu g.g^{-1}$)	100	198	3770	19.04
2 Generation- rat- F0 dosing 0.1, 0.4, 1.6, 3.2 $mg.kg^{-1}$ bw/d; oral gavage males: from 6 weeks before, to end of mating. females: from 6 weeks before mating through to the 21st day of lactation (DL21). F1: 0.1, 0.4 $mg.kg^{-1}$ bw/d; oral gavage males: from 22 days after birth to the end of mating (started 90 days after birth). females: from 22 days after birth through to DL 21 (for F2).	Argus Research Lab 2000 #418-008 (also cited as US EPA OPPT AR-226 0569)	F0 males: reduced body weight gains	F0 male: NOEL = 0.1 $mg.kg^{-1}$ bw/d LOEL = 0.4 $mg.kg^{-1}$ bw/d	10.5 $mg.L^{-1}$ 45.4 $mg.L^{-1}$	84900 (84.9 $\mu g.g^{-1}$) 176000 (176 $\mu g.g^{-1}$)	10 40	8490 4400	3770	0.44 0.85
		F0 female: reduced body weight gains during precohabitation	F0 female: NOEL = 0.4 $mg.kg^{-1}$ bw/d LOEL = 1.6 $mg.kg^{-1}$ bw/d	18.9 $mg.L^{-1}$ 82 $mg.L^{-1}$	58000 (58 $\mu g.g^{-1}$) 184000 (184 $\mu g.g^{-1}$)	10 40	5800 18400	3770	0.65 0.2

		F1: significantly reduced litter sizes and both viability and lactation indices. Reductions in development including delayed eye opening, surface righting, pinna unfolding and air righting reflex.	F1: NOEL = 0.4 $mg.kg^{-1}$ bw/d LOEL = 1.6 $mg.kg^{-1}$ bw/d	NA	57.6 ppm 70.4 ppm	10 40	5760 (5.76 ppm) 1760 (1.75 ppm)	3770	0.65 2.14
Rat-Oral gavage 42 days precohabitation to day 21 of lactation; cross fostering study. Doses 0 and 1.6 $mg.kg^{-1}$ bw/d	Argus Research Lab 2000 #418-014	Maternal: LOEL decrease in body weight gain, reduced gestation time, delivery time and litter size Offspring: LOEL increased mortality, reduced body weight, increased hepatocyte peroxisomes, increased type II pneumocytes and lamellar bodies in the lung.	Maternal LOEL at dose 1.6 $mg.kg^{-1}$ bw/d Offspring LOEL at maternal dose of 1.6 $mg.kg^{-1}$ bw/d (level in liver of offspring at this dose was 70.4 $\mu g.g^{-1}$= 70400 $\mu g.kg^{-1}$ from study ARL 418-008)		70400 (70.4 ppm)	40	1760	3770	2.14

a AF of 10 applied for interspecies variability and lab-to-field extrapolation, multiplicative with generic factor of 10 for extrapolation from LOEC to NOEC OR study specific factor of 4 where study information was available.

b EEV is maximum concentration of PFOS in Canadian polar bears, South Hudson Bay