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Canadian Council of Ministers of the Environment | Le Conseil canadien des ministres de l'environnement

SCIENTIFIC CRITERIA DOCUMENT FOR THE DEVELOPMENT OF THE CANADIAN SOIL AND GROUNDWATER QUALITY GUIDELINES FOR THE PROTECTION OF ENVIRONMENTAL AND HUMAN HEALTH

Perfluorooctane Sulfonate (PFOS)

PN 1625

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NOTE TO READERS

The Canadian Council of Ministers of the Environment (CCME) is the primary minister-led intergovernmental forum for collective action on environmental issues of national and international concern.

This scientific criteria document provides the background information and rationale for the development of Canadian Environmental Soil and Groundwater Quality Guidelines for perfluorooctane sulfonate (PFOS). The information in this document is current as of 2017, when the document was last revised and updated. For further technical information regarding these guidelines, please contact:

Environment and Climate Change Canada
Place Vincent Massey
351 Saint-Joseph Boulevard, 6th floor Annex
Gatineau, QC K1A 0H3
Phone: 800-668-6767 (in Canada only) or 819-997-2800 (National Capital Region)
Email: ec.rqe-eqg.ec@canada.ca

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LIST OF ABBREVIATIONS

95% CI	confidence interval at 95%
ADI	average daily intake
AFFF	aqueous film-forming foam
AK_{UF}	toxicokinetic portion of the interspecies uncertainty factor
ATSDR	Agency for Toxic Substances and Disease Registry (United States)
BAF	bioaccumulation factor
BCF	bioconcentration factor
BF	relative bioavailability factor
BMD	benchmark dose
BMDL	upper limit of the confidence interval at 95% of the BMD
BMF	biomagnification factor
BTF	biotransfer factor
bw	body weight
CCME	Canadian Council of Ministers of the Environment
CEPA	Canadian Environmental Protection Act
CEQG	Canadian Environmental Quality Guideline
CHMS	Canadian Health Measures Survey
CI	confidence interval
CGWQG	Canadian Groundwater Quality Guideline
CSoQG	Canadian Soil Quality Guideline
CWQG	Canadian Water Quality Guideline
DEA	diethanolamine
DMIR	dry matter intake rate
DTED	daily threshold effects dose
DW	drinking water
dw	dry weight
EC	Environment Canada
ECx	effective concentration (where x is the % of individuals exhibiting a specified level of impairment)
EC_{50}	effective concentration for 50% of individuals
ECF	electrochemical fluorination
ECL	effects concentration low
ED_{1C}	effect dose for primary consumer
EDI	estimated daily intake
EFSA	European Food Safety Authority
ESSD	estimated species sensitivity distribution
FCSAP	Federal Contaminated Sites Action Plan
FIR	food ingestion rate
FOSA	perfluorooctane sulfonamide
FOSE	perfluorooctane sulfonamidoethanol
FWQG	Federal Water Quality Guideline
GC-MS	gas chromatography–mass spectrometry
GD	gestation day
GM	geometric mean
GWQG	groundwater quality guideline
$GWQG_E$	groundwater guideline for the protection of ecological receptors

$GWQG_F$	groundwater quality guideline final
$GWQG_{FL}$	groundwater quality guideline for the protection of freshwater aquatic life
$GWQG_{GC}$	groundwater quality guideline for groundwater contact by soil-dependent organisms
$GWQG_{PW}$	groundwater quality guideline for the protection of potable water
HC	Health Canada
HDL	high-density lipoprotein
HPLC	high-performance liquid chromatography
HSDB	Hazardous Substances Data Bank (United States)
ICx	inhibitory concentration x%, where x is the degree of effect (e.g., reduced performance) compared to the control
IQR	interquartile range
K_d	soil-water partition coefficient
K_{oc}	organic carbon-water partition coefficient
K_{ow}	octanol-water partition coefficient
K_{pw}	protein-water partition coefficient
LC	liquid chromatography
LC_{50}	lethal concentration 50%: concentration inducing 50% mortality
LC-ESI-MS/MS	liquid chromatography–electrospray ionization–tandem mass spectrometry
LC-MS	liquid chromatography–mass spectrometry
LC-MS/MS	liquid chromatography–tandem mass spectrometry
LD_{50}	lethal dose 50%: dose inducing 50% mortality
LDL	low-density lipoprotein
LOAEL	lowest-observed-adverse-effect level
LOD	limit of detection
LOEC	lowest-observed-effect concentration
LOEL	lowest-observed-effect level
LOQ	limit of quantification
L-PFOS	linear PFOS
LWT	livestock water threshold
MAC	maximum allowable concentration
MDL	method detection limit
MOA	mode of action
MPC	maximum permissible concentration
MS	mass spectrometry
MS/MS	triple quadrupole mass spectrometry or tandem mass spectrometry
N-EtFOSA	N-ethyl perfluorooctane sulfonamide
N-EtFOSE	N-ethyl perfluorooctane sulfonamidoethanol
NHANES	National Health and Nutrition Examination Survey (United States)
N-MeFOSE	N-methyl perfluorooctane sulfonamidoethanol
NOAEL	no-observed-adverse-effect level
NOEC	no-observed effect concentration
NOEL	no-observed-effect level
OECD	Organisation for Economic Co-operation and Development
PBPK	physiologically based pharmacokinetic model
PFA	perfluorinated acid
PFAA	perfluorinated alkyl acid
PFAS	per- and polyfluoroalkyl substances

PFCA	perfluorocarboxylic acid
PFHxS	perfluorohexane sulfonic acid
PFOA	perfluorooctanoic acid
PFOS	perfluorooctane sulfonate
PFSA	perfluoroalkyl sulfonic acid
PM	particulate matter
pKa	acid dissociation constant
POSF	perfluorooctane sulfonyl fluoride
PPAR	peroxisome proliferator-activated receptor
PXR	pregnane X receptor
RIVM	National Institute for Public Health and the Environment (Netherlands)
RTDI	residual tolerable daily intake
SD	standard deviation
SFT	Norwegian Pollution Control Authority
SIR	soil ingestion rate
SPE	solid-phase extraction
SoQG	soil quality guideline
$SoQG_{1C}$	soil quality guideline for soil ingestion, primary consumers
$SoQG_{2C}$	soil quality guideline for soil ingestion, secondary consumers
$SoQG_{3C}$	soil quality guideline for soil ingestion, tertiary consumers
$SoQG_{DH}$	soil quality guideline for direct human health
$SoQG_{E}$	soil quality guideline for the protection of ecological receptors
$SoQG_{FL}$	soil quality guideline for the protection of freshwater life
$SoQG_{HH}$	soil quality guideline for the protection of human health
$SoQG_{I}$	soil quality guideline for ingestion of soil and food
$SoQG_{IR}$	soil quality guideline for the protection of irrigation water
$SoQG_{LW}$	soil quality guideline for the protection of livestock watering
$SoQG_{OM-E}$	soil quality guideline for the protection of nearby land from contamination due to off-site migration of soil via wind erosion
$SoQG_{OM-HH}$	human health soil quality guideline for off-site migration
$SoQG_{PW}$	soil quality guideline for the protection of potable groundwater
$SoQG_{SC}$	soil quality guideline for soil contact
SSV	soil screening value
TEC	threshold effects concentration
TDI	tolerable daily intake
TDS	Total Diet Study
TMF	trophic magnification factor
US EPA	United States Environmental Protection Agency
UF	uncertainty factor
UPLC-MS/MS	ultra-high-pressure liquid chromatography tandem mass spectrometry
ww	wet weight
WWTP	wastewater treatment plant
Xevo-TQ-S-MS/MS	triple quadropole mass spectrometer

GLOSSARY

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

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

Bioaccumulation: the process by which chemical compounds are taken up by terrestrial or aquatic organisms directly from the exposure medium and through the consumption of contaminated food at a faster rate than the compounds are lost through excretion or metabolism.

Biomagnification: the process of bioaccumulation by which tissue concentrations of accumulated chemical compounds are passed up through two or more trophic levels such that tissue residue concentrations increase systematically as trophic level increases.

Biomagnification factor (BMF): a measure of bioaccumulation by which tissue concentrations of accumulated chemical compounds are determined relative to tissue concentrations in two or more trophic levels.

UV-B: ultraviolet radiation in the spectrum of ≈280–320 nm in wavelength.

EXECUTIVE SUMMARY

Canadian Environmental Quality Guidelines (CEQGs) are numerical concentrations or narrative statements recommended to provide a healthy, functioning ecosystem capable of sustaining the existing and likely future uses of a site by ecological receptors and humans. Canadian Soil Quality Guidelines (CSoQGs) and Canadian Groundwater Quality Guidelines (CGWQGs) can be used as the basis for consistent assessment and remediation of soils and groundwater at contaminated sites in Canada.

The soil quality guidelines (SoQGs) were derived according to procedures described in *A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines* (CCME 2006). According to this protocol, both environmental and human health SoQGs are developed for four land uses: agricultural, residential/parkland, commercial and industrial. CCME recommends the lowest value generated by the two approaches for each of the four land uses as the CSoQG.

The groundwater quality guidelines (GWQGs) were derived according to procedures described in *A Protocol for the Derivation of Groundwater Quality Guidelines for Use at Contaminated Sites* (CCME 2015). GWQGs are developed for environmental and human health pathways, independent of land use. The lowest calculated value of each of the pathways become the CGWQGs.

This scientific criteria document provides the background information and rationale for deriving SoQGs and GWQGs for the protection of environmental and human health for perfluorooctane sulfonate (PFOS). It reviews the chemical and physical properties of PFOS, PFOS sources and emissions in Canada, the distribution and behaviour of PFOS in the environment, and the toxicological effects of PFOS in environmental species, humans and laboratory animals.

Based on CCME (2006), this document evaluates three types of exposure pathways: required pathways (direct contact and soil ingestion), applicable pathways (soil ingestion by secondary and tertiary environmental receptors, indoor air, groundwater, and produce, meat and milk ingestion), and check mechanisms (off-site migration of substances). Table 1 and Table 2 below list the SoQGs and GWQGs for each of the pathways calculated, respectively.

Table 1. Soil quality guidelines and check values for PFOS (mg/kg dw)

	Land use			
	Agricultural	**Residential/ Parkland**	**Commercial**	**Industrial**
Guideline	0.01	0.01	0.01	0.01
Human health guidelines/check values				
$SoQG_{HH}$[a]	0.01	0.01	0.01	0.01
Direct contact guideline $SoQG_{DH}$[b]	2	2	3	40
Inhalation of indoor air check $SoQG_{IAQ}$[c]	NC	NC	NC	NC
Off-site migration check $SoQG_{OM-HH}$	—	—	0.1	0.1
Soil quality guideline for the protection of potable groundwater $SoQG_{PW}$[d]	0.01	0.01	0.01	0.01
Produce, meat and milk check $SoQG_{FI}$	NC	NC	—	—
Environmental health guidelines/check values				
$SoQG_{E}$[e]	0.01	0.01	0.2 (coarse soil) 0.1 (fine soil)	0.2 (coarse soil) 0.1 (fine soil)
Soil contact guideline $SoQG_{SC}$	10	10	60	60
Soil and food ingestion guideline $SoQG_{I}$	0.01	0.01	—	—
Nutrient and energy cycling check $SoQG_{NEC}$	NC	NC	NC	NC
Off-site migration check $SoQG_{OM-E}$	—	—	0.1	0.1
Soil quality guideline for the protection of groundwater: livestock watering and irrigation $SoQG_{LW}$ and $SoQG_{Ir}$[f,g]	7 (coarse soil) 5 (fine soil)	—	—	—
Soil quality guideline for the protection of groundwater: freshwater life $SoQG_{FL}$[h]	0.2 (coarse soil) 0.1 (fine soil)	0.2 (coarse soil) 0.1 (fine soil)	0.2 (coarse soil) 0.1 (fine soil)	0.2 (coarse soil) 0.1 (fine soil)

NC = not calculated; ND = not determined; $SoQG_{E}$ = soil quality guideline for environmental health; $SoQG_{HH}$ = soil quality guideline for human health. The dash indicates a guideline or check value that is not part of the exposure scenario for this land use and therefore is not calculated.

[a] $SoQG_{HH}$ is the lowest of the human health guidelines and check values.

[b] $SoQG_{DH}$ is based on direct exposure to soil via ingestion, dermal contact and particulate inhalation.

[c] $SoQG_{IAQ}$ applies to volatile organic compounds. PFOS is essentially non-volatile.

[d] For pH between 5 and 7. Based on a K_{oc} of 1445 L/kg; PFOS K_{oc} is highly variably (229 to 6,310 L/kg; Franz Environmental Inc. 2012, 2014), therefore the level of protection afforded by the $SoQG_{PW}$ may not be appropriate for all sites. Where groundwater is used as a potable water source, groundwater concentrations should be compared directly to the $GWQG_{PW}$ value. Where groundwater is used for other purposes (e.g., irrigation of produce), this should be evaluated on a site-specific basis.

[e] $SoQG_{E}$ is the lowest of the environmental health guidelines and check values.

[f] Coarse-grained soil is soil in which more than 50% of particles (by mass) are larger than 75 µm mean diameter (D_{50} > 75 µm).

[g] Fine-grained soil is soil in which more than 50% of particles (by mass) are smaller than 75 µm mean diameter (D_{50} < 75 µm).

[h] $SoQG_{FL}$ is the concentration in *soil* that is expected to protect against potential impacts on aquatic systems from PFOS originating in soil that may enter the groundwater and subsequently discharge to a surface water body. This pathway may be applicable under any land use category where a surface water body sustaining aquatic life is present (i.e., within 10 km of the site). Where the distance to the nearest surface water body is greater than 10 km, application of the pathway should be evaluated on a case-by-case basis by considering the site-specific conditions. If surface

water bodies are located closer to the remediated soils than 10 metres, then this generic guideline may not be appropriate and a site-specific evaluation may be necessary on a case-by-base basis since the saturated zone transport model is not considered to be appropriate for use at distances less than 10 metres.

Table 2. Groundwater quality guidelines for PFOS (mg/L) considering ecological and human receptors

	Soil type[a]	
	Coarse	Fine
Final groundwater quality guideline ($GWQG_F$)[b]	0.0006	0.0006
Groundwater guideline for the protection of ecological receptors ($GWQG_E$)[c]	0.007	0.007
Groundwater contact ($GWQG_{GC}$) by soil-dependent organisms	1	1
Protection of freshwater life ($GWQG_{FL}$)[d]	0.007	0.007
Protection of marine life ($GWQG_{ML}$)	NC	NC
Protection of livestock watering ($GWQG_{LW}$)	0.3	0.3
Protection of irrigation water ($GWQG_{IR}$)	NC	NC
Management considerations ($GWQG_M$) – solubility	200	200
Groundwater guideline for the protection of human health ($GWQG_{PW}$)[e]	0.0006	0.0006

NC = not calculated

[a] Coarse-grained soil contains more than 50%, by mass, particles larger than 75 µm mean diameter (D_{50} >75 µm). Fine-grained soil contains more than 50%, by mass, particles smaller than 75 µm mean diameter (D_{50} <75 µm).

[b] $GWQG_F$ is the lowest of the pathway-specific guidelines for ecological and human receptors and considers other management factors such as substance solubility, analytical detection limits and background concentrations.

[c] $GWQG_E$ is the lowest of the pathway-specific guidelines for ecological receptors and considers other management factors such as substance solubility, analytical detection limits and background concentrations

[d] $GWQG_{FL}$ is the concentration in *groundwater* that is expected to protect against potential impacts on freshwater life from PFOS originating in soil that may enter groundwater and subsequently discharge to a surface water body. This pathway may be applicable under any land use category where a surface water body sustaining aquatic life is present (i.e., within 10 km of the site). Where the distance to the nearest surface water body is greater than 10 km, application of the pathway should be evaluated on a case-by-case basis by considering the site-specific conditions.

[e] $GWQG_{PW}$ are adopted directly from the Guidelines for Canadian Drinking Water Quality. Therefore, the $GWQG_{PW}$ is equivalent to the Maximum Acceptable Concentration (MAC) of 0.0006 mg/L (0.6 µg/L) developed by HC (2018*a*).

1. INTRODUCTION

CSoQGs[1] and CGWQGs are numerical concentrations or narrative statements that specify levels of toxic substances or other parameters in soil or groundwater that are recommended to maintain, improve or protect environmental quality and human health. They are developed using formal protocols to ensure scientifically defensible values that are consistent throughout Canada.

CSoQGs are developed according to procedures described in *A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines* (CCME 1996, revised in 2006). According to this protocol, both environmental and human health SoQGs are developed for four land uses: agricultural, residential/parkland, commercial and industrial. CCME recommends the lowest value generated by the two approaches for each of the four land uses as the CSoQG.

CGWQGs are developed according to procedures described in *A Protocol for the Derivation of Groundwater Quality Guidelines for Use at Contaminated Sites* (CCME 2015). GWQGs are developed for environmental and human health pathways, independent of land use. The lowest calculated value of each of the pathways becomes the CGWQG.

In addition, various check mechanisms considering indirect pathways of exposure (i.e., off-site migration of substances via wind and water erosion) provide protection for resources and receptors not otherwise considered in the calculation of soil guidelines.

This scientific criteria document reviews the sources and emissions of perfluorooctane sulfonate (PFOS), its distribution and behaviour in the environment, and its toxicological effects on terrestrial plants and invertebrates, birds, humans, and experimental animals.

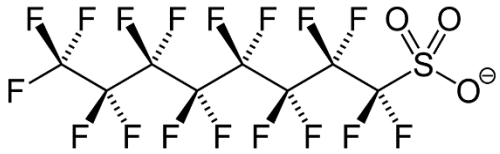
The CSoQGs and CGWQGs presented in this document are intended as general guidance. Site-specific conditions should be considered when applying these values (see CCME [1996] for guidance on developing site-specific soil objectives). CCME (2006) provides further generic implementation guidance pertaining to the guidelines. CSoQGs and CGWQGs are calculated to approximate a "no- to low-effect" level (or threshold level) based only on the toxicological information and other scientific data (fate, behaviour, etc.) available for the substance of concern. The guidelines do not consider socio-economic or technological factors. Site managers should consider these non-scientific factors at the site-specific level as part of the risk management process. Since the guidelines may be applied differently in various jurisdictions, the reader should consult appropriate authorities for the laws and regulations of the jurisdiction in which they are working for applicable implementation procedures.

[1] Soil guidelines and the data used to calculate them are, by convention, always expressed on a dry weight basis to allow the data to be standardized. In case of doubt and if the scientific criteria document does not specify whether wet or dry weight is used, readers are advised to check the references provided.

2. BACKGROUND INFORMATION

2.1. Physical and Chemical Properties

Per- and polyfluoroalkyl substances (PFAS) are a class of anthropogenic compounds that were produced and widely used in North America from the 1950s until the primary international manufacturer, phased them out of North American production in 2002 (3M 2003). Perfluorooctane sulfonate (PFOS) and its related compounds are classified as perfluorinated alkyl acids (PFAAs), a subgroup of PFAS. PFAAs consist of a fully fluorinated carbon chain, typically four to 14 carbons in length, and of a charged functional group, typically carboxylate, sulfonate or phosphonate. They are extremely stable due to the presence of carbon-fluorine bonds that are resistant to breaking (Boulanger *et al.* 2005*a*; Butt *et al*. 2010; Clarke *et al.* 2010; Lin *et al*. 2010; Mak *et al*. 2009; Yeung *et al*. 2006). PFOS is an eight-carbon perfluorinated alkane with a sulfonate group at one end ($C_8HF_{17}SO_3$). Its structure is illustrated below.



PFOS can exist as an anion, an acid, or various salts and polymers. Commercially important PFOS salts include potassium, diethanolamine (DEA), ammonium and lithium (Organisation for Economic Co-operation and Development [OECD] 2002). The main synonyms of PFOS are 1-perfluorooctanesulfonic acid, heptadecafluoro-1-octanesulfonic acid, heptadecafluorooctan-1-sulphonic acid, perfluorooctylsulfonic acid and 1-octanesulfonic acid (Agency for Toxic Substances and Disease Registry [ATSDR] 2015). PFOS is not found naturally in the environment.

PFOS precursors are substances with the perfluorooctylsulfonyl ($C_8F_{17}SO_2$, $C_8F_{17}SO_3$ or $C_8F_{17}SO_2N$) functional group (Environment Canada [EC] 2013*a*), such as perfluorooctane sulfonyl fluoride (POSF).

Table 3 lists key physical and chemical attributes of PFOS-related substances. Much of these data are for potassium salt. With a low acid dissociation constant (pKa) calculated at −3.3 (reviewed in Brooke *et al*. 2004), PFOS exists most commonly as an anion at pH values typically encountered in the environment and in the human body (European Food Safety Authority [EFSA] 2008).

PFOS is considered to be moderately soluble in water (Beach *et al*. 2006). Increased salt content in a solution decreases the solubility of PFOS (OECD 2002). PFOS is considered to be non-volatile, based on its low vapour pressure and predicted Henry's law constant (Beach *et al*. 2006; Giesy and Kannan 2001; OECD 2002). The OECD (2002) classifies PFOS as a "type 2 involatile chemical."

Like other PFAS, PFOS has both hydrophobic and hydrophilic functionalities, since it consists of a hydrophobic perfluorinated carbon tail and a hydrophilic ionic head. As such, it acts as a surfactant (Ahrens 2011; Jia *et al.* 2010). This characteristic prevents the measurement of its octanol:water partition coefficient (K_{ow}), because multiple layers are formed in octanol and water.

As a result, the calculation of a K_{ow} value from PFOS's solubility in octanol and water is also invalid. Consequently, the parameters (e.g., organic carbon-water partition coefficient [K_{oc}], soil:water partition coefficient [K_d], bioconcentration factor [BCF]) usually estimated from the K_{ow} cannot be calculated using conventional quantitative structure-activity relationship models (Beach *et al.* 2006).

Sorption coefficients for PFOS are not easily predicted. They are also not easily defined as single values, because they are affected by solution chemistry (e.g., pH), PFOS properties and adsorbent properties (e.g., particle size) (Beach *et al.* 2006; Du *et al*. 2014; Higgins and Luthy 2006). However, the adsorption of PFOS to soil, sediment and sludge appears to be strong. Published papers presenting original data for K_{oc} values were reviewed for Health Canada and Environment and Climate Change Canada (Franz Environmental Inc. 2012, 2014). The selected studies presented data from experiments conducted at relatively low aqueous concentrations (i.e., at or below approximately 1 mg/L) or from field studies, and included both marine and freshwater sediments. Franz Environmental Inc. (2012, 2014) reported a median K_{oc} of 1445 based on the literature review. Zareitalabad *et al*. (2013) compared the distribution between the concentrations of PFAS in surface water and sediments, and between the concentrations of PFAS in wastewater and sewage sludge, to the sorption coefficients obtained from laboratory experiments. Zareitalabad *et al*. (2013) suggested that the sorption of PFOS can be described as a partitioning-like process with an average log K_{oc} of 3.0. These values are within the range of log K_{oc} values (2.4-3.7) identified by the Interstate Technology Regulatory Council in its review of the literature (ITRC 2018*a*).

Zareitalabad *et al.* (2013) derived a "global average" K_d value of 178 L/kg (log K_d = 2.3; f_{OC} = 1.7%) for the distribution of PFOS between water and sediments after dividing the median PFOS sediment concentration by the median PFOS surface water concentration. The authors mentioned that sorption of PFOS under field conditions might be stronger than would be estimated from laboratory experiments because of the non-linear nature of sorption isotherms and the much smaller concentrations normally found in the field. Franz Environmental Inc. (2014) reported a median K_d value of 16 L/kg (range: 0.08–251 L/kg) based on a literature review. Most of the literature K_d values were based on laboratory experiments with sediments. Franz Environmental Inc. (2014) calculated a site-specific median K_d value of 1.6 L/kg based on maximum concentrations found in soil and groundwater samples collected at firefighter training areas at four civilian and military airports in Canada, and at one landfill site at a military base (f_{oc} range: 0.1–1.9%). Other sources have reported K_d values of 8.7 L/kg for river sediments and 12.8–35.1 L/kg for soils (3M 2003; Brooke *et al.* 2004).

Table 3. Physical and chemical properties of PFOS and some related compounds

Property	Compound				
	PFOS	**PFOS potassium salt**	**PFOS ammonium salt**	**PFOS DEA salt**	**PFOS lithium salt**
Chemical formula	$C_8HF_{17}SO_3$	$C_8F_{17}SO_3K$	$C_8F_{17}SO_3NH_4$	$C_8F_{17}SO_3NH(CH_2CH_2OH)_2$	$C_8F_{17}SO_3Li$
Chemical Abstract Services Registry Number	1763-23-1	2795-39-3	29081-56-9	70225-39-5 / 70225-14-8	29457-72-5
Molecular weight (g/mol)	500.125	538.215	517.156	605.261	506.058
Physical state	liquid[a]	white powder	no data found	no data found	off-white (yellow) powder
Melting point	no data found	≥400°C[b]	no data found	no data found	decomposes at 308°C[d]
Boiling point	133°C at 0.8 kPa[a]	not calculable	no data found	no data found	no data found
Vapour pressure at 20°C		3.31×10^{-4} Pa (measured*[b,e,f])	no data found	3.1×10^{-11} Pa[b]	no data found
Specific gravity	no data found	~0.6 at pH 7–8[e]	~1.1 at pH ~7[e]	~1.1 at pH ~7[e]	~1.1 at pH 6–8[e]
Henry's law constant ($atm \cdot m^3/mol$)	no data found	• 3.15×10^{-9} (3.19×10^{-4} $Pa.m^3/mol$)[b] • 3.05×10^{-9} pure water[e †] • 4.7×10^{-9} fresh water[e †] • 1.4×10^{-7} unfiltered sea water[e †] • 2.4×10^{-8} filtered sea water[e †] • 3.4×10^{-9} (3.45×10^{-4} $Pa.m^3/mol$)[f]	no data found	no data found	no data found
Water solubility	~550 mg/L 24–25°C[e]	• 519 mg/L (20°C) pure water[e,f] • 680 mg/L (24–25°C) fresh water[e,f] • 370 mg/L fresh water[e] • 25 mg/L filtered sea water[e] • 20.0 mg/L (22–24°C) (3.5% NaCl)[e] • 12.4 mg/L at 22–23°C natural sea water[e]	no data found	no data found	no data found
Log K_{ow}	cannot be measured[g, **]	cannot be measured [h,**]	cannot be measured [h,**]	cannot be measured [h,**]	cannot be measured [h,**]
Log K_{oc}	3.32[i] 3.16[h] 2.57[k]	no data found	no data found	no data found	no data found
pKa	<1.0[b]	−3.3[c]	-	-	-
Half-life	no data found	• atmospheric: 114 days[b] • water: 41 yrs (25°C)[h] • photolytic: >3.7 yrs[e,g]	no data found	no data found	no data found

[a] Ashford (1994) cited in Hazardous Substances Data Bank (HSDB) (2011)

[f] EC (2006*a*)

[g] Beach *et al*. (2006)

[b] Brooke *et al*. (2004)
[c] Cheng 2009
[d] US EPA (2017)
[e] OCDE (2002)
[h] ATSDR (2015)
[i] Franz Environmental Inc. (2012, 2014)
[j] Zareitalabad *et al*. (2013)
[k] US EPA (2014)
*Possibly overestimated due to volatile impurities (Brooke et al. 2004)
** Because PFOS is expected to form multiple layers in octanol-water mixtures
† Henry's law constant calculated using a vapour pressure of 3.27 × 10-9 atm

2.2. Analytical Methods

High-performance liquid chromatography–tandem mass spectrometry (HPLC-MS/MS) or liquid chromatography–mass spectrometry (LC-MS) methods are commonly used to detect perfluoroalkyls (Lindstrom *et al.* 2011b; Weremiuk *et al*. 2006; Wilson *et al.* 2007; Zhao *et al.* 2007). Gas chromatography–mass spectrometry (GC-MS) combined with derivatization and LC-MS or with tandem MS (LC-MS/MS, also known as triple quadrupole MS) can also be used to quantify fluorinated surfactants in environmental media such as water, waste water, sewage sludge and biota (CCME 2016; Meesters and Schröder 2004; Ontario Ministry of the Environment 2010; United States Environmental Protection Agency [US EPA] 2009; Weremiuk *et al.* 2006).

Strynar *et al*. (2012) describe a method for analyzing PFAS in surface soils using methanol extraction followed by ultra-high-pressure liquid chromatography tandem mass spectrometry (UPLC-MS/MS), using six-point calibration curves for each PFAS analyte. The authors noted the need to develop PFAS standard reference material to compare analytical methods (Strynar *et al*. 2012).

Various methods for extracting PFOS from biological media have been reported, including solvent extraction, solid-phase extraction (SPE), column-switching extraction, ion pair extraction and protein precipitation (reviewed in ATSDR 2015). PFOS and PFAS recovery can be significantly impacted by the choice of sampling and extraction techniques, which are areas of current research. Ahrens *et al*. (2012) showed that measuring total (neutral + ionized species) vs. neutral species of PFOS and other PFAS resulted in large differences in recovery and gas-particle distribution, especially under high-humidity conditions.

Several issues associated with measuring PFOS in environment and biological matrices have been identified. For instance, matrix components and the concentration of analytes may suppress the final yield during liquid chromatography-electrospray ionization-tandem mass spectrometry (LC-ESI-MS/MS) (Weremiuk *et al*. 2006). Background contamination of analytical blanks (Yamashita *et al*. 2004) and instrumental contamination (Longnecker *et al.* 2008; Yamashita *et al.* 2004) are also a concern.

Interference may also occur in biological matrices. For instance, taurodeoxycholic acid, a bile acid, can coelute with PFOS and interfere with its identification and quantification. An appropriate ion exchange column capable of effectively separating PFOS and taurodeoxycholic acid should thus be considered in food-monitoring studies (Ostertag *et al*. 2009*a*). Adsorption of PFOS onto nylon filters was also found to reduce PFOS recovery, so glass filters are now preferred (Axys Analytical Services, personal communication, 2013; Environmental Sciences Group 2015). Additionally,

whole-sample analysis of water samples is preferred in order to limit effects of sample stratification in sample containers (Environmental Sciences Group 2015).

Sampling for PFAS should follow recent guidance (e.g., ITRC 2020) to ensure that samples are representative and reliable.

2.3. Production, Uses and Importation in Canada

PFOS has been manufactured globally for more than 50 years, but never in Canada. PFOS was produced using the Simons electrochemical fluorination (ECF) method (Barber *et al.* 2007; Butt *et al.* 2010; EC 2006*a*; HSDB 2011). ECF produces linear as well as branched isomers (Section 3.1) (Château-Degat *et al*. 2010; Clarke *et al*. 2010; Dallaire *et al*. 2009*a*; Houde *et al*. 2008).

3M was the major global producer of PFOS and POSF-based chemicals (Section 2.4) from the 1950s to 2001. Paul *et al*. (2009) estimated worldwide PFOS production by 3M at 470 tonnes from 1970 to 2002. 3M began a voluntary phase-out of its POSF-based chemicals in 2001 and completed the phase-out of US PFOS production in 2002 (ATSDR 2015). However, China began large-scale PFOS production in 2003 (Butt *et al*. 2010). In 2006, China produced more than 203 tonnes (200 tons) of POSF (Ministry of Environmental Protection of China 2008).

In the past PFOS and PFOS-related compounds have been used for three broad purposes: surface treatment of apparel and home furnishings, paper protection, and performance chemicals. From 1997 to 2000, approximately 600 tonnes of perfluorinated alkyl compounds were imported into Canada, primarily from the United States. PFOS and its precursors constituted approximately 43% of that amount, but PFOS alone constituted <2% (reviewed in EC 2006*a*). Most perfluorinated alkyl compounds imported into Canada were used in the following items: water, oil, soil and grease repellents for fabric, packaging, rugs and carpets; surfactants and detergents; emulsifiers; wetting agents; dispersants; and aqueous film-forming foams (AFFFs) (Château-Degat *et al.* 2010; Clarke *et al.* 2010; Dallaire *et al.* 2009*a*). Specifically, the potassium salt of PFOS, used in the manufacture of AFFFs, was the most significant perfluorinated alkyl compound imported into Canada (EC 2006*a*).

PFOS, its salts and its precursors have been declared toxic and were added to Schedule 1 of the *Canadian Environmental Protection Act, 1999* (CEPA) (Government of Canada 2006). In addition, PFOS and its salts are considered to be persistent according to the *Persistence and Bioaccumulation Regulations* (Government of Canada 2000) and are considered to be bioaccumulative based on their preferential partitioning to lipid, blood and kidney in terrestrial and marine mammals as well as on evidence of their biomagnification. The manufacture, use, and importation of PFOS and PFOS-related compounds in Canada is regulated under the *Prohibition of Certain Toxic Substances Regulations, 2012* (Government of Canada 2012). This regulation prohibits the manufacture, import, sale, offer for sale and use of PFOS or of products containing PFOS, unless incidentally present, with certain exemptions (i.e., AFFF at specified concentrations and for certain purposes, aviation hydraulic fluids under certain conditions, and some products used in photographic or photolithographic process) (Government of Canada 2012). Moreover, PFOS and its salts were added to the Virtual Elimination List under subsection 65(2) of CEPA

with the promulgation of the *Perfluorooctane Sulfonate Virtual Elimination Act* (Government of Canada 2009).

2.4. Sources and Concentrations in the Canadian Environment

PFOS does not occur naturally (Butt *et al*. 2010). PFOS can be released directly into the environment as a result of its production, use (in consumer, commercial and industrial products) and disposal, or it may result indirectly from the biodegradation, photooxidation, photolysis and hydrolysis of precursor PFAS.

Industrial emissions are a direct source of perfluoroalkyls. As noted, PFOS is not produced in Canada, and US emissions of perfluoroalkyls have declined since 3M ended its production of PFOS and PFOS-related compounds in 2002 (ATSDR 2015; EC 2006*a*). Additionally, the US EPA established a Significant New Use Rule to limit the manufacture and import of several perfluoroalkyl sulfonates, including PFOS (US EPA 2013).

Prior to a ban in 2008, AFFFs containing PFOS potassium salt were imported into Canada and constitute a significant source of PFOS in the Canadian environment (EC 2006*a*). AFFFs can be released to the environment under various scenarios, namely in fire training exercises, emergency response, equipment calibration or accidental release from storage tanks, railcars and piping during delivery or transfer (ITRC 2018*a*). Releases of AFFFs from firefighting occur mainly as sewer runoff, but once released to the environment, AFFFs can also contaminate soil, surface water, and groundwater (ATSDR 2015; EC 2006*a,* ITRC 2018*a*).

PFOS and its precursors are found in sludge from wastewater treatment plants (WWTPs) (Tang *et al.* 2006). Several studies have documented significant amounts of perfluorinated compounds in wastewater effluents or sewage sludge, and higher concentrations of perfluorinated compounds downstream of WWTPs (Boulanger *et al*. 2005*b*; Furdui *et al.* 2008*a*; Sinclair and Kannan; Stock *et al*. 2007). Due to its use in many manufacturing processes and consumer products, PFOS is found in many landfills (Lang *et al*. 2017).

Incomplete combustion during incineration of PFOS-containing products can release the acidic form of PFOS into the environment. However, only 5% of waste disposal in Canada is incinerated, and incineration is not considered a significant source of PFOS in Canada (EC 2006*a*).

Several PFOS precursors have been identified, namely POSF, perfluorooctane sulfonamides (FOSAs), and perfluorooctane sulfonamidoethanols (FOSEs) such as N-methyl perfluorooctane sulfonamidoethanol (N-MeFOSE), N-ethyl perfluorooctane sulfonamidoethanol (N-EtFOSE), N-methyl perfluorooctane sulfonamide (N-MeFOSA) and N-ethyl perfluorooctane sulfonamide (N-EtFOSA) (Barber *et al*. 2007; Becker *et al.* 2008*a*; Martin *et al*. 2002). PFOS precursors are generally more volatile than PFOS, and their atmospheric transport has been considered the source of PFOS in remote regions (Egeghy and Lorber 2011; Martin *et al.* 2002). For example, alcohol derivatives (e.g., N-EtFOSE and N-MeFOSE) have been measured in the air in urban Toronto, ON, as well as in rural Long Point, ON, (Martin *et al*. 2002). As Martin *et al.* (2002) note, further investigation is needed, as PFOS yield from abiotic degradation of precursors to PFOS is thought

to be minor. Based on a literature review, Martin *et al*. (2010) suggest that *in vivo* biotransformation of precursors produces PFOS with yields greater than 32%.

2.4.1. Ambient Air

PFOS occurs in its anionic form in most environmental media. Due to its low volatility, it is not expected to partition to the atmosphere to any great extent and atmospheric degradation is likely insignificant (Brooke *et al*. 2004). PFOS releases are not reported under Canada's National Pollutant Release Inventory, and Canada's National Air Pollution Surveillance program does not measure PFOS concentrations. However, there are potential emission sources in Canada, such as fire fighting training areas, WWTPs, and landfills. Some published studies have reported PFOS concentrations in ambient air.

In 2004, both gaseous and particulate phase PFAS were measured on Cornwallis Island, NU, with a reported mean concentration of 5.9 pg/m^3 (Butt *et al*. 2010; Fromme *et al*. 2009). Boulanger *et al.* (2005*a*) measured PFOS concentrations in the air over Lake Erie and Lake Ontario between Canada and the United States. The authors did not detect PFOS in the gaseous phase, but they did detect particulate phase PFOS in four of the eight air samples, with a mean concentration of 6.4 pg/m^3 (range = 2.5–8.1 pg/m^3) for both lakes (Boulanger *et al.* 2005*a*). Conversely, in a 2007 study, PFOS concentrations were below the detection limit (<0.02 pg/m^3) in all samples (n = 6) collected from backyards in Vancouver, BC (Shoeib *et al*. 2011). No studies identified data on the concentration of PFOS in particulate matter <10 µm (PM_{10}) or <2.5 µm ($PM_{2.5}$).

Appendix A presents a summary of air monitoring data in Canada and in other parts of the world. To be conservative, the highest reported Canadian mean concentration, 6.4 pg/m^3 (Boulanger *et al*. 2005*a*), was selected to represent the background concentration of PFOS in ambient air in Canada for SoQG derivation.

2.4.2. Indoor Air

Since PFOS is essentially non-volatile, total PFOS concentrations in indoor air are mainly dependent upon PFOS concentrations in suspended particulates, which may originate from outdoor air (as a result of infiltration) and potential indoor sources such as carpeting, furniture and paint (Fraser *et al*. 2012). This could result in higher concentrations of PFOS in indoor air than in ambient air.

To date, very few studies have investigated PFAS concentrations in indoor environments. Indeed, only one Canadian study was identified. Shoeib *et al*. (2011) sampled indoor air between March and December 2007 from a subset of homes in Vancouver, BC, from which outdoor air and indoor dust were also sampled. PFOS concentrations in indoor (and outdoor) air were below the method detection limit (MDL) of 0.02 pg/m^3 in all samples (n = 39 homes) (Shoeib *et al*. 2011).

A Norwegian study provides a mean concentration of <47.4 pg/m^3 PFOS as measured in four indoor air samples (May–June 2005) from Tromsø (Barber *et al*. 2007).

Appendix A presents a summary of indoor air monitoring data in Canada and Norway.

Data published to date do not provide indoor air PFOS concentrations, as PFOS was below detection limits in all studies. To be conservative, the background outdoor air concentration, 6.4 pg/m^3 (see Section 2.4.1), was selected by default to represent the background PFOS concentration in indoor air in Canada for the SoQG derivation.

2.4.3. Indoor Settled Dust

PFOS concentrations in household dust have been documented in two Canadian studies and in several studies conducted in other countries. All studies indicate that PFOS and perfluorooctanoic acid (PFOA) are the dominant PFAS in household dust.

Kubwabo *et al*. (2005) collected house dust samples in Ottawa, ON, in 2002 and 2003. PFOS concentrations were below the MDL (<4.56 ng/g) in 33% of the samples (n = 67) with a reported mean of 443.68 ng/g (median = 37.8 ng/g, range = 2.28–5,065 ng/g). The authors also reported lower concentrations in older homes, which had significantly less carpeting than newer homes. They suggested that the higher concentrations in new homes may be associated with carpeting and the possible use of PFOS-based chemicals for the surface treatment of carpets. PFOS was detected in all the dust samples (n = 132) from homes sampled in Vancouver, BC, in 2007 and 2008, with a reported mean of 280 ng/g (median = 71 ng/g) and a range of 1.5–4,661 ng/g (Shoeib *et al*. 2011).

Knobeloch *et al*. (2012) reported a median PFOS concentration of 47 ng/g (range = 8.7–1,100 ng/g) from 39 homes in Wisconsin sampled during the spring of 2008. Fraser *et al.* (2012) measured PFAS in indoor dust from offices, homes and vehicles in 2009 in Boston, MA, and reported geometric mean (GM) PFOS concentrations in indoor dust of 14.6 ng/g (offices), 15.8 ng/g (vehicles) and 26.9 ng/g (homes).

In their analyses of house dust collected from Europe, Australia and the United States, Kato *et al.* (2009) determined that PFOA and PFOS may share similar exposure routes or sources and that PFAS in house dust showed similar trends regardless of location. However, Goosey and Harrad (2011) reported that indoor dust collected from Australian and Canadian homes had a high relative abundance of N-EtFOSA. Indoor dust samples collected from Europe and the United States showed a higher proportion of perfluorohexane sulfonic acid (PFHxS), N-MeFOSE and N-EtFOSE. Based on these findings, Goosey and Harrad (2011) concluded that there were significant differences in PFAS usage patterns from different parts of the world and even between regions. Therefore, extrapolating exposure assessments from one country or region to another should be conducted with caution.

Appendix A presents a summary of indoor dust monitoring data in Canada and in other parts of the world.

The median (50^{th} percentile) PFOS concentration, 71 ng/g, reported for indoor settled dust in Canadian homes (Shoeib *et al*. 2011) was selected as the average Canadian background indoor

dust concentration for the SoQG derivation. This study was selected because it is the Canadian study with the largest sample size and lowest MDL.

2.4.4. Surface Water

PFOS concentration data are available for surface water from rivers, the Great Lakes and tributaries, marine water, and water bodies in the vicinity of airports in Canada.

Many of the rivers and streams sampled by Gewurtz *et al*. (2013) across Canada are in cities and/or near wastewater treatment plants, and thus are influenced by urban and industrial activities. PFOS levels (GM = 10 ng/L) were highest in water collected at an urbanized site influenced by urban stormwater (Mill Creek, Kelowna, BC). In addition, concentrations in Wascana Creek, Regina, SK (GM = 7.8 ng/L; downstream of a major WWTP) were relatively high compared to the other sampling sites less impacted by urban sources. Detectable values (>2 ng/L) were also observed in water samples from southern Ontario (Hamilton Harbour, Fort Erie, Wolfe Island, Grand River [downstream of Waterloo] and the Thames River [downstream of London]), Québec City, QC, Vancouver, BC (Still Creek and Serpentine River), Abbotsford, BC (Fishtrap Creek), and three Atlantic region sites (Napan River, NB, Sackville River, NS, and Waterford River, NL). PFOS concentrations were not detected in most samples from water bodies in non-urban areas (e.g., background sites on the Fraser River, Mill Creek and the Okanagan River in BC, Kusawa Lake, YT, Lake Superior [three open water sites and one site located near Thunder Bay, ON], Grand River (upstream of Waterloo, ON), and Thames River [upstream of London, ON]).

Scott *et al.* (2009) investigated perfluorinated acid (PFA) concentrations in 38 rivers upstream and downstream of populated areas and some remote locations across Canada from 2001 to 2008. The overall mean PFOS concentration (n = 65) was 2.15 ng/L (range = 0.01–34.6 ng/L). Most sites downstream of urban centres had higher PFA concentrations than the upstream sites, but measurable concentrations were also detected in remote sites, most likely from atmospheric inputs. In a survey of PFA concentrations in rivers, Scott *et al*. (2009) found the maximum concentrations of PFOS in tributaries of Lake Erie and in the downstream St. Lawrence River and the lowest concentrations of PFAs in remote regions such as rivers located near the Garibaldi Glacier, BC. The authors also noted geographic differences in PFOS concentrations between eastern Canada (up to the Manitoba-Ontario border) and western Canada (from the Manitoba-Ontario border to British Columbia), with reported average PFOS concentrations of 4.09 ng/L for eastern Canada and 0.91 ng/L for western Canada (Scott *et al*. 2009).

Concentrations of perfluorinated compounds in water from the Great Lakes region were documented in several studies (e.g., Boulanger *et al*. 2004; Furdui *et al.* 2008*a*; Scott *et al*. 2009). PFOS concentrations in Great Lakes water samples collected from 2002 to 2005 ranged from 0.1 to 0.996 ng/L in Lake Superior, 1.2 to 3.2 ng/L in Lake Huron, 4.0 to 39 ng/L in Lake Erie and 3.6 to 121 ng/L in Lake Ontario (Boulanger *et al*. 2004; Furdui *et al*. 2008*a*; Scott *et al*. 2010). PFOS concentrations were found to increase as water moved from one basin to another and with increasing inputs of PFAS from various point sources (Furdui *et al.*, 2008*a*). PFOS concentrations were lower in water samples from off-shore sites as compared to near-shore samples (Furdui *et al.* 2008*a*). PFOS concentrations in Lake Superior (<0.147–0.996 ng/L) were similar in water

collected from the surface and from depths (Scott *et al*. 2010). PFOS concentrations in Lake Superior tributaries were found to range from <0.041 to 0.827 ng/L (Scott *et al.* 2010). The highest PFOS concentration was found in the Nipigon River, downstream of a WWTP. PFOS measured in the upstream site was below the detection limit (i.e., <0.041 ng/L) (Scott *et al.* 2010).

Some studies have documented elevated concentrations of PFOS in surface water at remote northern sites. In 2003 and 2005, Stock *et al*. (2007) measured PFAS concentrations in surface water from four lakes on Cornwallis Island, NU: Amituk (2003 only), Char, Resolute and Meretta. The mean concentrations of PFOS for Amituk Lake and Char Lake ranged from 1.2 to 1.8 ng/L, while the mean PFOS concentrations in Resolute Lake and Meretta Lake were up to 60-fold higher (23–69 ng/L, respectively). The higher PFOS concentration measured in Resolute Lake was thought to be due to a "non-atmospheric source," possibly from historic sewage wastewater discharge from the Meretta Lake outflow or the use of AFFFs in the vicinity of Meretta Lake and Resolute Lake (the latter is located close to the Resolute Bay Airport, where AFFFs may have been used). The authors noted that PFAS concentrations from Resolute and Meretta Lakes were consistent with levels of perfluorohexanesulfonate, PFOS, perfluoroheptanoic acid and PFOA measured in other AFFF-contaminated areas (Stock *et al.* 2007).

Loewen *et al*. (2008) investigated atmospheric deposition of volatile PFAs and water concentrations of perfluorocarboxylic acids (PFCAs) and/or PFOS in alpine lakes in the Rocky Mountains (Cedar Lake near Golden, BC, and three lakes in Alberta: Emerald Lake in Yoho National Park, Bow Lake and an unnamed lake in Banff National Park). They found that airborne PFOS precursors N-MeFOSE and N-EtFOSE increased at higher altitudes, but corresponding increases in PFOS concentrations in surface water were not detected. PFOS concentrations were very low in the four lakes sampled, ranging from 0.04 to 0.1 ng/L. The highest PFOS concentration was found in Cedar Lake (821 m altitude). Loewen *et al*. (2008) suggested that local sources may have contributed to the PFOS found in lake waters at lower elevations, whereas the PFOS concentrations in lakes at higher elevations were dominated by atmospheric input of PFOS and PFOS precursors.

PFOS concentrations in marine water are also documented. Dinglasan-Panlilio *et al.* (2014) evaluated the occurrence of PFAS in the inland marine systems off the west coast of Vancouver Island, BC, and near Seattle, WA, from 2009 to 2011. PFOS was detected at almost all sampled locations (except the Juan de Fuca Strait and Tofino) at concentrations ranging from 0.2 ng/L to 5.8 ng/L.

Awad *et al*. (2011) investigated spatial and temporal trends of PFAS in water, sediment and fish tissue in creeks up- and downstream of discharge points near Toronto's Pearson International Airport, ON, nine years after a large AFFF spill (in 2000, followed by a minor spill in 2002). The highest PFOS concentration was in a stormwater management pond <100 m from the stormwater outfall where runoff from the spill was diverted. Concentration at the stormwater pond was 690 ng/L in 2003 and 290 ng/L in 2009. PFOS concentrations measured in 2003 and 2009 at sites further downstream from the stormwater outfall were in the range of values measured in Lake Ontario (15–121 ng/L) in 2003, indicating that the PFOS contaminant migration from the spill was limited at that time (Awad *et al.* 2011).

Similarly, Fowler (2011) investigated PFOS contamination at a pond on the property of and a ditch adjacent to the Hamilton International Airport, ON. The study found PFOS concentrations exceeding the Ontario Ministry of Environment and Climate Change's lowest-observed-effect concentration (LOEC) of 5,000 ng/L. PFOS concentrations were highest in the pond, but decreased with distance from the airport fire training pad (Fowler 2011). De Solla *et al*. (2012) also investigated the likelihood that PFAS contamination originated from the Hamilton International Airport after high PFOS concentrations were found in a nearby lake downstream of the airport. Similar to the previous study (Fowler 2011), de Solla *et al.* found that PFOS concentrations in water initially decreased rapidly with increasing distance for approximately 10 km but remained relatively stable between 10 and 53 km from the airport (de Solla *et al.* 2012). In contrast, Bhavsar *et al*. (2016) noticed an increase in PFOS concentrations at Lake Niapenco, downstream from the Hamilton International Airport. These authors also measured PFOS tissue concentrations in biota sampled downstream of the airport (Section 2.4.9).

PFOS has also been detected in surface water samples from rivers, lakes and streams in the United States and other parts of the world. Appendix A presents a summary of PFOS concentrations in surface water. Section 2.4.6 addresses surface water used as a source of drinking water.

2.4.5. Groundwater

PFOS has been detected in groundwater collected from commercial and industrial sites where AFFFs have been used in firefighting training exercises, or where spills have resulted in either contamination or suspected contamination of soil, surface water or groundwater. PFOS concentrations in groundwater at London International Airport, ON, were found to range from <5 to 130 µg/L at a former firefighting training area (Lebel 2012). In an investigation of a firefighting training site at Hamilton International Airport, PFOS concentrations in groundwater ranged from <0.02 to 560 µg/L (exp. Services Inc 2011).

Section 2.4.6 discusses groundwater used as a source of drinking water.

2.4.6. Drinking Water

Although PFOS is not regularly monitored at water treatment plants in Canada, the analysis has been performed for a few locations. When detected in drinking water, it is usually below 1 ng/L. Only two studies specific to Canada were identified.

Mak *et al.* (2009) evaluated tap water from Niagara-on-the-Lake, ON, for PFAS in 2006–2008. PFOS was among the most predominant PFAS, with a mean concentration of 3.3 ng/L (Mak *et al*. 2009). PFOS concentrations were determined in raw and finished drinking water samples obtained in 2009 from 31 treatment plants in six Canadian provinces. PFOS was detected above the MDL (0.077 ng/L) in only one raw water sample (0.082 ng/L), and levels in finished water were all below the MDL (Richard Carrier, Chemical Assessment Section Water and Air Quality Bureau, Health Canada [HC], personal communication, 2013). Most drinking water treatment processes do not completely remove residual PFAS contaminants from source water (Kubwabo and Lalonde

2010). Removal was only demonstrated in treatment facilities using microfiltration and reverse osmosis (Quinones and Snyder 2009). However, Rumsby *et al.* (2009) reported that treatment systems using granulated active carbon with long empty bed contact time (with appropriate regeneration, at least once per year, according to Skutlarek *et al.* [2006]; Takagi *et al*. [2011]) can also successfully remove PFAS from water.

Appendix A provides a summary of PFOS concentrations in drinking water.

A mean concentration of 3.3 ng/L (Mak *et al.* 2009) was used to represent background levels in drinking water for the SoQG derivation. This mean concentration is considered to be conservative, since it is the highest published average drinking water concentration in Canada.

2.4.7. Sediment

PFOS concentrations in sediments from areas far from point sources or PFAS industries are considered negligible.

Concentration of PFOS in sediments in Canadian rivers and lakes ranged from <0.00006 to 1,272 ng/g dry weight (dw) in several studies (Awad *et al*. 2011; Burniston *et al*. 2006; 2012; EC 2013; Gewurtz *et al*. 2013; Helm *et al.* 2011; Myers *et al.* 2012; Stock *et al*. 2007; Veillette *et al*. 2012; Yeung *et al*. 2013). As with surface water concentrations, sediment concentrations were generally highest in Lake Ontario and lowest in Lake Superior and lakes in the Canadian Arctic. Other sites across Canada representative of different drainage basins were sampled, but PFOS concentrations at these sites were below the detection level (Gewurtz *et al.* 2013). Codling *et al.* (2014*b*) measured PFAS in dated cores and surface sediments from Lake Michigan in 2010. The mean PFOS concentration was 0.44 ng/g in surface sediment and 2.70 ng/g in sediment core samples. The authors noted that PFOS concentrations reached a maximum in the late 1990s and early 2000s (Codling *et al*. 2014*b*).

Stock *et al*. (2007) collected sediment cores from three arctic lakes (Amituk, Char and Resolute) on Cornwallis Island, NU, in 2003. PFOS concentrations were generally found to decrease with sediment age and depth. Concentrations in sediments were 1.1 ng/g (0–1 cm depth) and <0.35 ng/g (1–2 cm and 2–3 cm depth) for Char Lake; 0.062 ng/g (0–1.5 cm depth), 0.027 ng/g (1.5–2.5 cm depth) and 0.022 ng/g (2.5–3.5 cm depth) for Amituk Lake; and 85 ng/g (0–1 cm depth), 33 ng/g (1–2 cm depth) and 24 ng/g (2–3 cm depth) for Resolute Lake. The elevated concentrations in Resolute Lake sediments might have been related to specific sources (e.g., historic sewage wastewater discharge, use of AFFFs in the vicinity) (Stock *et al*. 2007).

Awad *et al*. (2011) found that sediment concentrations were still elevated nine years after the first of two AFFF spill events at Pearson International Airport in Toronto, ON. The highest concentrations (13 ng/g dw) were reported in sediments in the stormwater management pond located <100 m from where AFFFs were discharged.

Appendix A summarizes concentrations of PFOS in sediments.

2.4.8. Soil

Once released to the environment, PFOS is mobile and can move through soil and contaminate groundwater (ITRC 2018*a*). PFOS can be found in soil at great distances from any known source; however direct discharges (such as AFFF) and the application of biosolids or leaching from landfills are the principal sources of PFOS-contaminated soil. A mass balance study by Filipovic *et al*. (2015) indicates that a significant portion of PFAAs from atmospheric deposition is stored in soil, where it can be a source of groundwater contamination. Strynar *et al*. (2012) estimated that approximately 6% of total PFOS production between 1970 and 2002 is distributed globally in surface soils (estimate based on a median PFOS surface soil concentration of 0.472 ng/g).

Cabrierizo et al (2018) measured PFAS concentrations in the Canadian arctic. PFOS was detected in 25 of 26 samples. The overall median concentration was 0.0068 ng/g dw (mean: 0.32 ng/g dw, 95th percentile: 0.34 ng/g dw, maximum: 7.5 ng/g dw).

Strynar *et al.* (2012) assessed PFAS concentrations in fresh surface soils and in archived soil samples (no date stated) collected in the United States (n = 10) and in five other countries (n = 50, i.e., 10 per country). Soils from areas with known PFAS contamination and those in the vicinity of industries known to use PFAS were excluded. The authors did not intend for these soils to be representative of the nation of origin. Rather, the samples were indicators of background concentrations in different soil types and parts of the world and the results provided in Strynar *et al.* (2012) refer to the whole set of samples (no country or soil type data were indicated). PFOS was detected in 48% of samples (limit of quantification [LOQ] = 0.51 ng/g dw). The overall median concentration (six countries) was 0.472 ng/g (mean: 1.17 ng/g dw, 95th percentile: 5.16 ng/g dw, maximum: 10.1 ng/g dw). Seven of the 10 soils with the highest PFAS concentrations were from the United States (one with PFOS <LOQ, the others ranging from 0.6 to 2.55 ng PFOS/g).

In another global study of 62 soil samples, Rankin *et al*. (2016) detected PFOS in all samples (n=33) from non-impacted areas on all continents (majority of samples from North America). The authors showed that PFAS concentrations were higher in the northern hemisphere than the southern hemisphere, with only relatively low concentrations found in the southern hemisphere (geometric mean ΣPFSA =30 ng/g; range = 7.0-300 ng/g; n=9), but a wide range of concentrations in the northern hemisphere (geometric mean ΣPFSA =170 ng/g; range = <LOD-3270 pg/g; n=53). From this same data set, PFOS was detected in all North American samples at concentrations ranging from 18.09-1956.34 pg/g (n=33) with a mean of 392.47 pg/g and a geomean of 220.34 pg/g.

PFOS concentrations in soils from suspected AFFF-impacted areas or from water-bearing zones have been investigated at the former firefighter training facility at Hamilton International Airport. PFOS concentrations in these soils ranged from <0.025 to 26 mg/kg (exp. Services Inc 2011).

As indicated by the above data, background concentrations of PFOS in soils are reported to be in the ng/g range. Considering that PFOS is not naturally occurring, its background concentration in Canadian soil was set to 0.

2.4.9. Biota

PFOS has been detected in aquatic and terrestrial species. Giesy and Kannan (2001) first demonstrated the global distribution of PFOS in wildlife when they detected PFOS in aquatic mammals, birds, fish, turtles and frogs from various locations, including Canada. Animals in the highest trophic levels, such as mink, bald eagles and polar bears, were found to have the highest concentrations, suggesting that PFOS is widespread and has the potential to bioaccumulate (Houde *et al*. 2008; Tang *et al*. 2006). PFOS is generally the dominant PFAS measured in biota (Houde *et al*. 2011).

Plasma PFOS concentrations in seals from the Canadian Arctic ranged from <3 to 12 ng/mL in ringed seals (*Pusa hispida*) and 11 to 49 ng/mL (mean: 28 ng/mL) in grey seals (*Halichoerus grypus*). PFOS concentrations ranging from 130 to 320 ng/g wet weight (ww) were measured in egg yolk from double-crested cormorants (*Phalacrocorax auritus*) in Lake Winnipeg, MB (Giesy and Kannan 2001). Martin *et al*. (2004*a*) detected PFOS in liver samples from Canadian Arctic wildlife collected by subsistence hunters and trappers, including birds (1992–1993), mammals (1998–2002) and fish (2001–2002). Higher-trophic-level mammals had higher PFOS concentrations than lower-trophic-level mammals. PFOS was considered non-detectable in black guillemot (*Cepphus grylle*) (<0.5 ng/g). The lowest detectable concentrations were found in northern fulmars (*Fulmarus glacialis*), with a mean of 1.3 ng/g ww (range: 1.0–1.5 ng/g ww). The highest concentrations were found in polar bears (*Ursus maritimus*), with a mean of 3,100 ng/g ww (range: 1,700 - >4,000 ng/g ww) (Martin *et al*. 2004*a*). In general, species living in the Canadian Arctic had lower concentrations of PFOS than the same species found at mid-latitude regions of the United States (e.g., Kannan *et al.* 2002*a*).

In a study comparing benthic and pelagic species (fish and invertebrates), the highest PFOS and FOSA concentrations were found in benthic organisms, such as the amphipod *Diporeia* (mean: 280 ng/g ww) and sculpins (mean: 450 ng/g ww), which feed on *Diporeia*. Pelagic species (*Mysis*, alewife [*Alosa pseudoharengus*], smelt [*Osmeridae* sp.] and lake trout [*Salvelinus namaycush*]) had lower mean concentrations (≤70 ng/g ww; Appendix A) than benthic species (Martin *et al*. 2004*b*). Houde *et al.* (2008) reported that linear PFOS (L-PFOS) was the dominant isomer detected in alewife, smelt, sculpin and lake trout sampled near Niagara-on-the-Lake, ON.

Monitoring results for lake trout and walleye (*Sander vitreus*), as top predator fish, at sites across Canada show variable PFOS levels, with the highest concentrations observed in lake trout from Lake Erie (GM = 92 ng/g ww) and Lake Ontario (GM = 51 ng/g ww) (Gewurtz *et al.* 2013). Concentrations were also found in walleye from the St. Lawrence River (30 ng/g ww), the Codette Reservoir, SK (24 ng/g ww), and Lake Diefenbaker, SK (23 ng/g ww), and in lake trout from Peninsula Harbour, ON (24 ng/g ww) and Lake Champlain, QC (17 ng/g ww) (Gewurtz *et al.* 2013). Concentrations in northern Canada, Lake Superior, and the Pacific and Atlantic regions were mostly low (<3 ng/g ww) (Gewurtz *et al*. 2013). Gewurtz *et al*. (2014) reported PFOS levels in common carp (*Cyprinus carpio*) and smallmouth bass (*Micropterus dolomieu*) from waterways downstream of the Hamilton International Airport, ON. Mean concentrations in upper Lake Niapenco were 550 ng/g ww (standard deviation [SD] = 178) in common carp and 195 ng/g ww (SD = 15) in smallmouth bass. In lower Lake Niapenco, mean concentrations were 655 ng/g ww (SD = 202) in common carp and 298 ng/g ww (SD = 29) in smallmouth bass (Gewurtz *et al*. 2014).

Gewurtz *et al*. (2013) also reported monitoring results for gull and starling eggs. For gull eggs measured individually, relatively elevated PFOS concentrations were found at urbanized areas of the Great Lakes and the St. Lawrence River (>260 ng/g ww). Pooled gull egg samples were similar, with the highest levels observed in Lake Erie (676 ng/g ww). Concentrations were lower (7–115 ng/g ww) in non-urban areas and both Atlantic and Pacific coasts. For European starlings (*Sturnus vulgaris*), elevated PFOS concentrations were found in eggs collected at a Brantford, ON, landfill (703 ng/g ww) and a Calgary, AB, landfill (148 ng/g ww). However, excluding these two landfill sites, PFOS concentrations were not higher at waste sites compared to non-waste sites. For example, PFOS concentrations (GM) were higher in the starlings collected from urbanized communities of Indus, AB (199 ng/g ww), Delta, BC (75 ng/g ww) and Hamilton, ON (41 ng/g ww) compared with landfill sites located in Langley, BC (5.6 ng/g ww), Halton, ON (29 ng/g ww), Stoney Creek, ON (28 ng/g ww) and Otter Lake, NS (18 ng/g ww) (Gewurtz *et al*. 2013).

There is some evidence that tissue PFOS concentrations have diminished in some biota, such as ringed seals inhabiting the Canadian Arctic and sea otters (*Enhydra lutris*) living along the Alaskan coast, following the phase-out of POSF-based products, including PFOS and its precursors (Armitage *et al.* 2009). However, reductions in PFOS concentrations in lake trout have yet to be observed in Canada. Lake trout is considered a sentinel indicator species and is typically sampled in monitoring programs for persistent organic pollutants in the Great Lakes (Furdui *et al*. 2008*b*). Archived lake trout samples collected between 1979 and 2004 from Lake Ontario, as part of the Department of Fisheries and Oceans' long-term monitoring program, showed that PFOS concentrations (range = 6–96 ng/g ww) were the highest of all PFAS tested. PFOS concentrations were significantly higher in samples collected in 1988 and 1993 compared to 1979. A weak decline occurred from 1993 to 1998, and 2004 concentrations were not statistically different from those from the 1980s and 1990s (Furdui *et al*. 2008*b*). Gewurtz *et al*. (2012) indicated that PFOS concentrations in lake trout may have stabilized following the voluntary phase-out of PFOS production in North America. The authors noted that this may be due to continued inputs from PFOS-containing products or the degradation of PFOS precursor compounds (Gewurtz *et al*. 2012). One study showed that PFAAs could potentially accumulate to unsafe levels in sport fish species, despite surface water and sediment levels being below guidelines (Bhavsar *et al*. 2016; Gewurtz *et al*. 2014) (Section 3.6).

Awad *et al*. (2011) found that fish sampled downstream and very close to the AFFF spill outfall that occurred at Toronto's Pearson International Airport had PFAS profiles dominated by PFOS (62–80% of total PFAS) nine years after the spill event. The fish tissue concentrations were greater than those reported at upstream locations, suggesting long-term impacts of the spill on the local environment (ongoing uptake into fish from contaminated media and food sources), persistence of PFOS in fish tissue (slow elimination) or impact of urbanization (creating ongoing inputs of PFAS).

Appendix A summarizes concentrations of PFOS in biota.

2.4.10. Commercial Food

Three Canadian studies related to PFOS concentrations in commercially available food were identified, while two studies document traditional food in northern Canada. Some studies have shown that red meat and fish contain high levels of PFOS (Ostertag *et al*. 2009*b*; Tittlemier *et al*. 2007; Tomy *et al.* 2004). Based on these studies, red meat may be a significant source of human exposure to PFOS from general dietary intake, and fish may be the predominant source of human exposure from contaminated waters.

Tittlemier *et al.* (2007) analyzed composite samples of meat or animal-derived food items from the Canadian Total Diet Study (TDS) (1992–2004). Half of the samples were collected in 2004, and others were archived samples collected from 1992 to 2001. Of the perfluorinated substances analyzed, PFOS was detected in seven out of 54 composite samples at concentrations ranging from 2.0 ng/g to 2.7 ng/g ww for marine and freshwater fish (2004), ground beef, and beef steak. Concentrations lower than the LOQ (but above the limit of detection [LOD]) were reported for microwave popcorn (0.98 ng/g), luncheon meats and cold cuts (0.5 ng/g), and freshwater fish (1998) (1.3–1.5 ng/g).

Ostertag *et al*. (2009*a*) measured PFAS in 65 composite food samples (e.g., drinks, meat and fish, plant-based foods, milk products) collected from Whitehorse, YK, as part of the 1998 TDS and estimated a dietary exposure of 0.1–0.2 ng/kg body weight [bw]/day for PFOS. In general, PFAS were not normally detected in unprocessed foods, but were found in some foods that had undergone some form of processing, suggesting that contamination occurs after the initial production or cultivation of various foods. For example, PFOS was detected in processed cheese at 1.14 ng/g ww, compared to 0.71 ng/g (below the LOQ of 0.95 ng/g) in unprocessed cheese. Concentrations were below the MDLs in other food items: (ng/g ww: cold cuts <0.68, cookies <0.15, peppers <0.15, canned lunch meats <0.37, pizza <0.20 and frozen beef dinner <0.17). Ostertag *et al.* (2009*a*) estimated exposure at an order of magnitude higher than in 1998 using data from the 2004 TDS (Tittlemier *et al*. 2007) or a combination of data from the 1998 and 2004 TDS.

Del Gobbo *et al.* (2008) detected PFOS in 15 of 21 composite fish samples they collected from Ontario supermarkets in 2006. Concentrations in raw samples ranged from non-detected to 1.68 ng/g ww, with the highest level found in yellow croaker (*Larimichthys polyactis*). PFOS levels in cooked samples ranged from non-detected to 1.14 ng/g ww, with the highest concentration found in grey mullet. Generally, Del Gobbo *et al.* (2008) found that all cooking methods reduced PFA concentrations and that baking reduced all PFA concentrations below the LOD (0.03–10 ng/g ww) in all species analyzed. However, Bhavsar *et al*. (2014) observed either no reduction or an increase in PFAS in sport fish from Ontario after cooking.

Ostertag *et al.* (2009*b*) measured the concentrations of PFAS in traditional foods in northern Canada. They detected PFOS in 39% of the 68 samples collected from Chesterfield Inlet, Igloolik, Pond Inlet and Qikiqtarjuaq, NU, between 1997 and 1999 in both aquatic foods and terrestrial foods. For aquatic foods, they found concentrations of 0.1–7.6 ng/g in ringed seal, polar bear (meat), beluga (*Delphinapterus leucas*), narwhal (*Monodon monoceros*), bearded seal (*Erignathus barbatus*), walrus (*Odobenus rosmarus*), eider duck (*Somateria mollissima*), black duck (*Anas rubripes*) and lake trout. For terrestrial foods, they found concentrations of 5.0 ng/g in baked

caribou liver and 0.1–0.2 ng/g in caribou bone marrow, heart, blood, kidney, stomach, tongue and meat (weight basis not reported). PFOS concentrations in the other samples, Arctic char (*Salvelinus alpinus*), seaweed, clams, ptarmigan (*Lagopus muta*), Arctic hare (*Lepus arcticus*), snow goose (*Chen caerulescens*) and berries, were below the detection limit (ranging from <0.1 to <0.5 ng/g depending on the sample) (Ostertag *et al*. 2009*b*).

PFOS levels in Eastern Canadian Arctic marine foods ranged from 0.28 to 1.8 ng/g ww in zooplankton and invertebrates (shrimp and clams) collected in 2001 and 2002, from 1.3 to 1.4 ng/g ww in fish collected in 2000 and 2001, and from 2.4 to 122 ng/g ww in marine mammals (whales and pinnipeds) collected between 1996 and 2000 (Butt *et al*. 2010).

Tao *et al*. (2008*a*) measured PFOS concentrations in infant formula (five brands) and cow's milk (11 brands) samples from New York, NY, and Washington, DC. PFOS was detected in only one infant formula sample and was below the LOQ of 11.0 ng/L in all the cow's milk samples (Tao *et al.* 2008*a*).

Appendix A summarizes PFOS concentrations in commercially available food.

2.4.11. Human Breast Milk

Kubwabo *et al*. (2013) did not detect PFOS in the 13 breast milk samples they collected in the Kingston, ON, region in 2003–2004. In Massachusetts, Tao *et al*. (2008*b*) reported PFOS concentrations ranging from <0.032 to 0.617 ng/mL (mean = 0.131 ng/mL) in human breast milk collected in 2004.

Appendix A summarizes PFOS concentrations in human milk.

2.4.12. Consumer Products

The PFOS anion, as well as its 4-, 5-, 6- and 7-carbon homologues, were used in Scotchgard Protector and other repellant products, as well as in the Light Water brand of AFFFs (Hebert *et al*. 2002). Other reported uses of PFOS and its derivatives include coatings on paper and food packaging, photographs, packaging and textiles (due to their ability to repel oil and water), hydraulic fluids, and metal-plating solutions (due to their stability at high temperatures) (Tang *et al*. 2006).

2.4.13. Rainwater and Snow

Loewen *et al*. (2005) measured a mean PFOS concentration of 0.59 ng/L in rainwater collected in 2004 in Winnipeg, MB. An Ontario study collected precipitation samples in 2005 at two locations (Algoma and Sibley) near Lake Superior (Scott *et al*. 2010). The average PFOS flux was 0.32 ng/m^2 per event at the Algoma site and 5.8 ng/m^2 at the Sibley site. The values for Algoma are biased low, as the leading edge of a precipitation event usually has higher concentrations of

chemicals than the bulk of the precipitation, and only events that had more than 250 mL were analyzed (smaller precipitation events were not considered).

PFOS concentrations in surface ice cap samples from the Canadian Arctic ranged from 2.6 to 86 pg/L (Young *et al*. 2007).

Appendix A provides a summary of PFOS concentrations in precipitation.

2.5. Existing Soil and Water Quality Criteria and Guidelines

Soil and water quality guidelines for PFOS and other PFAS substances have been developed by several jurisdictions. Given the high level of ongoing research into PFOS and PFAS in general, these values may change as the science evolves. ITRC (2018*b*) provides a list of existing regulations, guidelines and advisories for PFAS from many jurisdictions around the world. Analysis of the basis of variation in guidelines from different jurisdictions is beyond the scope of this document. Cordner *et al*. (2018) provide a review of several sources of variation in guideline levels for PFOS and PFOA from various American jurisdictions. Some Canadian values are provided in Table 4; for other values please consult ITRC (2018*b*) and other sources.

Table 4. Existing soil and water criteria and guidelines for PFOS in Canadian jurisdictions

Jurisdiction	Criterion/guideline	Concentration	Reference
Soil			
Government of B.C	Human Health Protection: • Intake of soil	2.5 mg/kg (wildlands, urban park, res. high density) 1 mg/kg (agr., res. low density) 7.5 mg/kg (commercial) 200 mg/kg (industrial)	Government of B.C. (1996)
	• Groundwater used for drinking water	0.35 mg/kg	
	Environmental Protection: • Toxicity to soil invertebrates and plants	40 mg/kg (wildlands natural) 70 mg/kg (wildlands reverted, agr., urban park, res. low density) 150 mg/kg (res. high density, commercial, industrial)	
	• Groundwater flow to surface water used by aquatic life	9 mg/kg	
Canada site-specific: London, ON, airport	Site-specific soil criteria (plants)	27 mg/kg dry soil	SNC Lavalin (2013)
	Site-specific soil criteria (invertebrates)	0.77 mg/kg	
	Site-specific soil criteria (mammals)	0.86 mg/kg (herbivorous mammal); 0.14 mg/kg (omnivorous mammal)	
	Site-specific soil criteria (birds)	0.006 mg/kg (omnivorous bird); 0.013 mg/kg (insectivorous bird); 0.092 mg/kg (herbivorous bird)	

Jurisdiction	Criterion/guideline	Concentration	Reference
	Final selected site-specific soil criteria	0.006 mg/kg	
Canada site-specific: Williams Lake, BC	Final site-specific soil remedial target	1.3 mg/kg	Kennedy (2010)
	Site-specific soil remedial target (plants)	9.2 mg/kg	
	Site-specific soil remedial target (wildlife)	13 mg/kg	
	Average daily intake (ADI) for Level 4 avian predators (birds)	21 µg/kg-d (0.021 mg/kg-d)	
	Screening benchmark (invertebrates)	39 mg/kg	
	Residential soil screening level	6 mg/kg	US EPA (2009)
Water			
Canada	Drinking water guideline	0.6 µg/L	HC (2018*a*)
	Federal Environmental Quality Guideline – water (for protection of freshwater aquatic life)	6.8 µg/L	ECCC (2018)
Other media			
Canada	Federal Environmental Quality Guideline – wildlife diet (to protect mammals that consume aquatic biota)	4.6 µg/kg ww food	ECCC (2018)
	Federal Environmental Quality Guideline – wildlife diet (to protect avian species that consume aquatic biota)	8.2 µg/kg ww food	
	Federal Environmental Quality Guideline- fish tissue (to protect fish from bioaccumulated contaminants)	9.4 mg/kg ww	
	Federal Environmental Quality Guideline- bird egg (to protect avian species)	1.9 µg/g ww	

3. ENVIRONMENTAL FATE AND BEHAVIOUR

PFAS and their precursors, including PFOS, are present in considerable quantities in many environmental media, and researchers assume that long-distance transport is occurring due to weather and ocean currents (Armitage *et al*. 2006; Ellis *et al*. 2004; Martin *et al.* 2004*a*). The hydrophobic and hydrophilic properties of PFAS affect their fate and behaviour in complex ways. While their transport is not well understood, PFAS, including PFOS tend to accumulate at interfaces between environmental media and this tendency may cause them to act differently depending on their concentration (ITRC 2018*a*).

PFOS is generally emitted to the environment from chemical mixtures that contain several PFAS and their precursors. Therefore, due to the degradation of more volatile precursors, PFOS and other PFAS may be found in areas far from any source, despite their relatively low volatility (Ellis *et al*. 2004). PFOS is not expected to degrade, hydrolyse or photolyse under normal environmental conditions, so it is expected to be very persistent in the environment (ITRC 2018*a*; ATSDR 2015; OECD 2002), however precursors may transform to PFOS in different environmental media (ITRC 2018*a*).

This section provides a summary of PFOS environmental fate and behaviour in various environmental media. ITRC (2018*a*) provides fate and behaviour information for PFAS relative to their principal sources, including AFFF use.

3.1. Speciation

Theoretically, a total of 199 PFOS isomers are possible (Letcher and Chu 2009), but a maximum of 11 isomers have been reported in manufactured products (Arsenault *et al.* 2008; Houde *et al.* 2008). L-PFOS makes up 65–79% of the PFOS manufactured via ECF (Arsenault *et al*. 2008; Benskin *et al*. 2007; Chu and Letcher 2009; De Silva *et al*. 2009; Greaves and Letcher 2013). Branched isomers consist primarily of monomethyl and dimethyl isomers. The major monomethyl isomers include 6-perfluoromethyl, 5-perfluoromethyl, 4-perfluoromethyl, 3-perfluoromethyl, 2-perfluoromethyl and 1-perfluoromethyl PFOS. The major dimethyl isomers include 3,3-dimethyl, 4,5-dimethyl, 3,5-dimethyl and 5,5-dimethyl PFOS (Houde *et al*. 2008).

The environmental fate, toxicity and bioaccumulation of individual PFOS isomers are not well characterized, but studies have shown the importance of considering both environmental and isomer-specific toxicological properties when assessing exposure effects to wildlife. The bioaccumulation characteristics of the PFOS perfluorodimethyl isomers differ from those of the LPFOS and PFOS monomethyl isomers. Houde *et al*. (2008) studied the transfer of isomers through the food web in Lake Ontario. L-PFOS was the dominant isomer detected in sediment and water samples as well as in wildlife tissues, especially in higher-trophic-level mammals and fish-eating birds, compared to manufactured technical grade (total) PFOS (Chu and Letcher 2009; De Silva *et al.* 2009; Gebbink and Letcher 2010). Branched isomers have also been identified and quantified in human serum (Riddell *et al.* 2009).

3.2. Atmosphere

Due to its low pKa, the anionic form of PFOS is expected to predominate at normal environmental conditions (i.e., near neutral pH) (Barber *et al*. 2007). Similarly, PFOS vapour pressure and solubility (and Henry's law constant) indicate that it is more likely to partition to water than air (Giesy and Kannan 2002; Weremiuk *et al.* 2006) and is essentially non-volatile. PFOS is generally associated with larger particulate matter diameters (Ge *et al*. 2017; Dreyer *et al*. 2015).

Precursors of PFOS and other PFAS are more volatile and can be found at long distances from their sources (Ellis *et al*. 2004; Prevedouros *et al*. 2006; Benskin *et al*. 2012). Martin *et al.* (2004*a*) demonstrated the potential for PFOS formation in the atmosphere via the transformation of precursors (notably perfluorooctanesulfonamides) in the presence of chlorine or hydroxide, the former being largely absent in the troposphere. Elevated levels of nitrogen oxides (NO_x) can alter PFCA composition via a gas-phase unzipping mechanism that preferentially generates (n-1) PFCAs over n PFCAs (Ellis *et al*. 2004). PFAS are removed from the atmosphere as wet and dry deposition after scavenging of particle-bound PFOS or partitioning of gaseous PFOS to water droplets (Dreyer *et al* 2010; Barton *et al*. 2007; Hurley *et al*. 2004). This explains, in part, the presence of non-volatile PFOS in distant environments such as the Arctic.

3.3. Water and Sediments

Due to its presence as an organic ion at environmental pH levels, low volatility, moderate water solubility and persistence, PFOS in solution is mobile in water until it is adsorbed onto particulate matter or taken up by organisms (EC 2006*a*; Cheng *et al*. 2008; Suja *et al*. 2009). In groundwater, advection will often be the principal influencing factor in PFOS mobility (ITRC 2018*a*).

The solubility of PFOS (as potassium salt) has been reported as 519–670 mg/L in water (Jing *et al*. 2009), 570 mg/L in purified water, 370 mg/L in fresh water and 25 mg/L in filtered seawater (OECD 2002).

A hydrolysis half-life of 41 years for PFOS in water was calculated at pH values of 5, 7 and 9 (as reviewed by ATSDR 2015). Although PFOS was thought to be resistant to photolysis, a study including both field and laboratory experiments demonstrated that this process may be significant (Taniyasu *et al.* 2013*a*). PFOS concentrations in solution decreased by 15% after a short period (20.5 hrs) of exposure to strong solar radiation (test conducted on snow at high altitude in Japan). After a long (1,232 hrs) period of exposure, PFOS concentrations in solution decreased by 29% (test conducted in Hawaii). A laboratory experiment on PFAS in serum showed that PFOS photolysis is higher when the solution is exposed in quartz glass, which allows full transmittance of UVB radiation (Taniyasu *et al*. 2013*a*).

PFOS in solution tends to accumulate at the air-water interface. Brusseau (2018) calculated that this increased the aqueous-phase transport retardation factor using a five stage multi-process model.

Several authors have suggested that ocean currents are responsible for the long-range transport of PFAS, including PFOS, from direct emissions and atmospheric deposition and as the result of degradation (Ahrens *et al*. 2010; 2012; Armitage *et al*. 2006; Wania 2007). In marine waters, PFAA solubility decreases and sorption increases, which will likely result in more accumulation in marine sediments (Hong *et al*. 2013). The oceans are likely the main sink for PFAS in the environment (Armitage *et al*. 2006).

PFOS in groundwater adsorbs to organic carbon in sediments, soil and sludge (3M 2003; Beach *et al*. 2006; Guelfo and Higgins 2013; Hekster *et al*. 2002; Higgins and Luthy 2006), and sorption is influenced by several factors, such as hydrophobicity, sorbent surface charge, organic carbon content and pH (Chen *et al*. 2009). Sorption increases in the presence of polyvalent cations, associated with decreasing pH (Higgins and Luthy 2006; McKenzie *et al*. 2015). Several authors have suggested that sediments, humic acid or soil are sinks for PFOS (Becker *et al*. 2008*b*; Jia *et al*. 2010; Martin *et al*. 2004*b*; Strynar *et al*. 2012), while others reported that water or biota are the sinks for PFOS (Nakata *et al*. 2006; Sanderson *et al*. 2002).

3.4. Indoor Settled Dust

PFOS has been detected in indoor dust. Emissions from and degradation of consumer products containing PFOS are likely major sources. Dust on floors and household objects may be ingested

to various degrees (Mitro *et al*. 2016). Of particular concern are children, who crawl and explore by putting items into their mouths. They represent a susceptible population and, as such, they are more susceptible to PFOS exposure through dust.

3.5. Soil

Application of AFFF, sewage sludge, accidental spills, wet and dry deposition of PFAS present in ambient air (Strynar *et al.* 2012), etc. can all lead to the presence of PFOS in soils. The sorption of PFOS to soil likely plays an important role in its attenuation and accumulation in soil and can contribute to potential PFOS exposure in soil-dwelling organisms (Higgins and Luthy 2006). At the same time, the degree of sorption affects PFOS bioavailability to biota, so sorption has implications for the general fate and toxicity of PFAS in the environment. In a review study of published data, Li *et al*. (2018) concluded that PFAS sorption behaviour could not be explained by a single soil property, or by soil organic carbon (OC) and pH combined, but that OC, pH and clay content significantly influenced sorption. Additional parameters (e.g., water temperature, electrostatics) may also influence sorption in soil and sediments (Li *et al*. 2018). In a study of six soil types, Milinovic *et al*. (2015) observed that the PFOS solid-liquid distribution coefficient (K_d) was positively correlated with OC content and that desorption yields were lower than 13% for PFOS, indicating that sorption is highly irreversible and that hydrophobicity controlled its sorption behaviour in soils. From the slope of the K_d/F_{OC}, curve, the authors calculated a K_{oc} of 2.9, which was similar to values reported (2.8-3.2) by Chen *et al*. (2012a) from similar experiments and lower than values reported (3.1-3.6) by Chen *et al*. (2009). Wei *et al*. (2017) studied PFOS sorption in six Chinese soil types and determined that sorption equilibrium was reached after 48 hours. These authors obtained much higher K_d values (14-66 L/g) than Milinovic *et al*. (2015) (0.009-0.444 L/g) and suggest that the difference is due to the dependence of PFOS sorption on soil chemical and structural features, such as Fe_2O_3 and Al_2O_3 content, as shown by a positive correlation between these compounds and soil sorption capacity.

Leaching of adsorbed PFOS, under the influence of irrigation and precipitation may drive transport of PFOS to groundwater (Lindstrom *et al*. 2011*a*; Filipovic *et al*. 2015; Hellsing *et al*. 2016; Braunig *et al*. 2017), however this process is a function of several soil and PFOS structural parameters and has not been observed in all studies (Sepulvado *et al*. 2011; Stahl *et al*. 2013; Anderson *et al*. 2016). A study of catchment areas in northern Sweden indicated that a considerable portion of PFAS stored in soil may be released to surface water over time (Filipovic *et al*. 2015).

The application of biosolids is another source of PFAS, including PFOS, to soils that would otherwise have lower inputs of these substances.

3.6. Biota

Monitoring studies suggest that PFOS is highly bioaccumulative (HSDB 2011). It is resistant to aerobic and anaerobic degradation, and to metabolism by vertebrates. According to biodegradation studies reviewed by the OECD, there is no evidence of PFOS biodegradation in any organism (reviewed in EC 2006*a*). Since concentrations in biota are dependent on uptake and elimination

rates as well as internal metabolism and transformation of precursors, concentrations can vary in biota from diverse locations and environmental media (ITRC 2018*a*).

PFOS does not appear to follow the classic pattern of distribution to adipose tissue, and K_{ow} is not generally an appropriate measure of bioaccumulation or biomagnification for PFOS. Rather than partitioning into lipid tissue, PFOS has been shown to bind to protein in plasma albumin and beta-lipoproteins (Kerstner-Wood *et al*. 2003) and to liver and liver fatty acid binding protein in higher organisms (Kerstner-Wood *et al*. 2003; Luebker *et al.* 2002). To address this unusual type of binding, a novel coefficient, the protein-water partition coefficient (log K_{pw}) has been developed, and a log K_{pw} of 4.1 has been reported for PFOS (Bischel *et al*. 2011), although there are currently few values available for comparison of this estimate.

Whole-body bioconcentration factor (BCF) values are generally lower than values based on blood or liver concentrations. Data for different marine and terrestrial species indicate that PFOA has a low to moderate bioaccumulation potential in aquatic species (on a whole-body basis), while accumulation may be higher in certain organs and tissues (e.g., liver, blood). Bhavsar *et al*. (2016) indicate that bioaccumulation factors (BAFs) and biota-sediment accumulation factors can vary widely within a species, even within a narrow size range, although accumulation tends to be greater in larger fish than in smaller fish. Further, several studies support higher bioaccumulation from water at lower trophic levels, with relatively low bioaccumulation at higher trophic levels (Bhavsar *et al*. 2016; de Solla *et al.* 2012; Gewurtz *et al*. 2014; Martin *et al*. 2004*b*; Stevens and Coryell 2007). PFOS data from one Hamilton, ON, site showed very high concentrations of contaminant in the flesh of several fish species (above consumption advisory levels) despite surface water concentrations below applicable guidelines (Bhavsar *et al*. 2016). The RIVM (2010) report experimental BCFs for various fish species, ranging from 1,100 to 5,400 L/kg ww for rainbow trout (*Oncorhynchus mykiss*), 1,124 to 2,796 L/kg ww for bluegill (*Lepomis macrochirus*), 167 to 1,750 L/kg ww for fathead minnow (*Pimephales promelas*) and 720 to 4,700 L/kg ww for carp (*Cyprinus carpio*) (RIVM 2010). Bhavsar *et al*. (2016) determined an overall log BAF of 3.4 and biota-sediment accumulation factor of 1.7 for 10 species of sport fish from Ontario. Based on BAFs calculated from study data, the authors determined that PFOS has the potential to accumulate to levels unsafe for human consumption under these conditions (Bhavsar *et al*. 2016; Gewurtz *et al*. 2014).

Xiao *et al*. (2012) investigated PFOS sorption from water to food in nine commonly consumed vegetables and three types of meat. PFOS food-water distribution coefficients ranged from 7 to 19 L/kg for vegetables and from 19 to 38 L/kg for meat. PFOS may bind to proteins and organic matter contained in vegetables and meat (Xiao *et al*. 2012). Del Gobbo *et al.* (2008) demonstrated that cooking fish reduced PFOS concentrations. However, Bhavsar *et al*. (2016) noted either no reduction or an increase in sport fish tissue concentrations after cooking using the same methods. Additionally, PFAA data from one Hamilton, ON, site showed very high concentrations of PFOS in the flesh of several fish species (above consumption advisory levels) despite surface water concentrations below applicable guidelines (Bhavsar *et al*. 2016).

Under natural conditions, PFOS can persist for over 285 days in microcosms (Boudreau *et al*. 2003). Because the half-life of PFOS exceeds the half-life criteria defined by the *Persistence and*

Bioaccumulation Regulations of CEPA, PFOS, its salts and its precursors are considered to be persistent in the Canadian environment (EC 2006*a*).

The metric for bioaccumulation in the Canadian *Persistence and Bioaccumulation Regulations* (Government of Canada 2000) is based on lipid binding and, as noted above, this metric is not appropriate for PFOS. However, given the tendency of PFOS to bioaccumulate through protein binding, EC (2006*a*, 2006*b*) concluded that the weight of evidence indicated PFOS is bioaccumulative.

3.6.1. Bioconcentration Factors in Plants

The concentration of PFOS in plants relative to soil is of particular importance when considering the potential exposure of terrestrial wildlife to PFOS. Several studies have examined the transfer of PFOS from soil to plants (Brignole *et al*. 2003; Lechner and Knapp 2011; Stahl *et al*. 2009; Yoo *et al*. 2011) using a variety of terms, including transfer factor (Lechner and Knapp 2011; Stahl *et al*. 2009), grass-soil accumulation factor (Yoo *et al*. 2011) and BAF (Beach *et al*. 2006; Brignole *et al*. 2003). In general, bioconcentration of PFOS in plants seems to occur when soil concentrations are in the range of 0 to 50 mg/kg soil, above which they appear to decrease. Table 5 summarizes the data used to calculate the overall BCF, and Appendix D presents background information.

Lechner and Knapp (2011) reported average BCFs for cucumber (*Cucumis sativus*), potato (*Solanum tuberosum*) and carrot (*Daucus carota* subsp. *sativus*) (0.59, 0.82 and 1.24, respectively) grown in soil at 0.01 and 0.6 mg/kg dry soil (cucumber), 0.02 and 0.3 mg/kg dry soil (potato) and 0.01 and 0.5 mg/kg dry soil (carrot) (Appendix D). At these low concentrations, transfer from soil to plant increased with increasing PFOS soil concentrations. BCFs were higher in shoots or vegetative portions than in the peeled or edible parts (Appendix D). BCFs for PFOS ranged from 0.034 to 0.129 for Bermuda grass (*Cynodon dactylon*), tall fescue (*Festuca arundinacea*) and Kentucky bluegrass (*Poa pratensis*), with a GM of 0.057 across the three plant species (Yoo *et al*. 2011).

Stahl *et al*. (2009) showed that PFOS uptake varied across species (perennial ryegrass [*Lolium perenne*], spring wheat [*Triticum aestivu*], oat [*Avena sativa*], maize [*Zea mays*] and potato) sown in loess soil diluted with quartz sand and spiked with 0.25, 1, 10, 25 or 50 mg PFOS/kg delivered by aqueous solution and exposed for three to four months (except ryegrass, which was harvested after 1, 2.5, 3.5 and 5 months). In general, the concentration of PFOS in plant parts increased with increased soil concentrations. BCFs calculated by Environment Canada, from data provided by Stahl *et al*. (2009), ranged from negligible (wheat and maize grain) to 4.2 times the concentration in soil (ryegrass after fourth cutting) at 10 mg PFOS/kg in soil. The average BCF across test concentrations and plant species was 0.29. As in Lechner and Knapp (2011), concentrations in the vegetative compartment (straw or peels) can be considerably higher than in the storage portion of the plant (grain or tubers). For example, concentrations in the straw portion of maize, oats and wheat were approximately 34, 60 and 3,600 times higher, respectively, than concentrations in the grain portion of the same plant. The study authors offered no explanation for this phenomenon.

Beach *et al*. (2006) calculated BCFs from the Brignole *et al*. (2003) data for seven plant species: onion, ryegrass, alfalfa (*Medicago sativa*), flax (*Linum usitatissimum*), lettuce (*Lactuca sativa*), soybean (*Glycine max*) and tomato (*Lycopersicon esculentum*) (see Appendix D). Except onion, BCFs were higher in the vegetative portion than in the grain or fruit. As noted above, BCFs appear to be highest when soil PFOS was 0–50 mg/kg and then decreased when PFOS in soil exceeded 50 mg/kg.

Since whole-plant BCFs were considered the most relevant to estimate wildlife exposure, the harvest index (the proportion of a given plant compartment relative to total above-ground plant biomass), was used to provide a weighted BCF (Appendix D).
BCFs based on wet weight (Lechner and Knapp 2011; Stahl *et al*. 2009) were converted to dry weight basis using a dry matter fraction of 0.15 (US EPA 1993) for dicot and monocots such that:

$$\text{Conversion factor for BCF} = \text{plant wet mass}/(\text{plant wet mass} - \text{plant water fraction})$$

where:

- plant wet mass = total wet mass of plant = 1 g
- plant water fraction = mass of water in total plant = 0.85 g
- wet weight to dry weight conversion factor = $1/(1 - 0.85) = 1/0.15 = 6.67$ (following US EPA 1993). (Note that soil was already reported on a dry weight basis).

BCF data were available for 16 plant species. Given that this is a small proportion of all possible plant species that wildlife might consume, and given that no BCF data exist for some plants that wildlife are known to consume (e.g., berries), all available plant species were included in deriving a grand mean weighted BCF of 0.35, which is considered representative for plants (Table 5).

Table 5. Summary of soil-plant BCFs used to calculate grand mean of plant BCFs for PFOS

Name (component)	Soil-plant BCF (plant/soil basis specified)[a]	Reference[b]	Soil-plant BCF (dw plant/dw soil)	Harvest index (range)	Harvest index (average)	Reference for harvest index	Weighted BCF[c]	BCF value used
Bermuda grass (*Cynodon dactylon*)	0.035 dry/dry	Yoo *et al*. (2011)	0.035	-	-	-	-	0.035
Tall fescue (*Festuca arundinacea*)	0.066 dry/dry	Yoo *et al*. (2011)	0.066	-	-	-	-	0.066
Kentucky bluegrass (*Poa pratensis*)	0.083 dry/dry	Yoo *et al*. (2011)	0.083	-	-	-	-	0.083
Potato (tuber) (*Solanum tuberosum*)	0.0004 wet/dry	Stahl *et al*. (2009)	0.003	-	-	-	-	0.003
Maize (grain) (*Zea mays*)	0.005 dry/dry	Stahl *et al*. (2009)	0.005	-	0.5	Pennington (2013)	0.081	0.081
Maize (straw) (*Zea mays*)	0.157 dry/dry		0.157	-				
Oat (grain) (*Avena sativa*)	0.007 dry/dry	Stahl *et al*. (2009)	0.007	0.11–0.48	0.3	Unkovich *et al.* (2010)	0.28	0.28
Oat (straw) (*Avena sativa*)	0.4 dry/dry		0.4					
Spring wheat (grain) (*Triticum aestivum*)	0.0002 dry/dry	Stahl *et al*. (2009)	0.0002	0.38–0.41	0.40	Hay (1995)	0.45	0.45
Spring wheat (straw) (*Triticum aestivum*)	0.753 dry/dry		0.753					
Perennial ryegrass (*Lolium perenne*)	0.538 dry/dry	Stahl *et al*. (2009)	0.41	0.33–0.49	0.41	Hay (1995)	0.54	0.54
Alfalfa (*Medicago sativa*)	0.59 dry/dry	Brignole *et al.* (2003)	0.59	0.127	0.127	Bolaños-Aguilar *et al*. (2002)	0.59	0.59
Flax (grain) (*Linum usitatissimum*)	0.077 dry/dry	Brignole *et al.* (2003)	0.077	0.24	0.24	Gailans (2010)	1.08	1.08
Flax (straw) (*Linum usitatissimum*)	1.4 dry/dry		1.4					
Tomato (fruit) (Lycopersicon esculentum)	0.065 dry/dry	Brignole *et al*. (2003)	0.065	0.54	0.54	Gianfagna *et al.* (1998)	0.96	0.96

Name (component)	Soil-plant BCF (plant/soil basis specified)[a]	Reference[b]	Soil-plant BCF (dw plant/dw soil)	Harvest index (range)	Harvest index (average)	Reference for harvest index	Weighted BCF[c]	BCF value used
Tomato (vegetative) (*Lycopersicon esculentum*)	2.02 dry/dry		2.02					
Onion (fruit) (*Allium cepa*)	1.4 dry/dry	Brignole *et al*. (2003)	1.4	0.82–0.85	0.835	Abdissa *et al.* (2011)	1.33	1.33
Onion (vegetative) (*Allium cepa*)	0.95 dry/dry		0.95					
Soybean (grain) (*Glycine max*)	0.13 dry/dry	Brignole *et al*. (2003)	0.13	0.35–0.53	0.44	Hay (1995)	1.33	1.33
Soybean (vegetative) (*Glycine max*)	2.28 dry/dry		2.28					
Lettuce (*Lactuca sativa*)	1.39 dry/dry	Brignole *et al*. (2003)	1.39	–	–	–	–	1.4
Ryegrass (*Lolium perenne*)	1.58 dry/dry	Brignole *et al.* (2003)	1.58	–	–	–	–	1.6
Potato (peeled edible) (*Solanum tuberosum*)	0.004 wet/dry	Lechner and Knapp (2011)	0.03	-	0.66	Lechner and Knapp (2011)	0.964	0.96
Potato (peelings) (*Solanum tuberosum*)	0.03 wet/dry		0.20	-				
Potato (vegetative) (*Solanum tuberosum*)	0.359 wet/dry		2.39	-				
Carrot (peeled edible) (*Daucus carota* subsp. *sativus*)	0.045 wet/dry	Lechner and Knapp (2011)	0.30	-	0.565	Lechner and Knapp (2011)	0.1.38	1.4
Carrot (peelings) (*Daucus carota* subsp. *Sativus*)	0.033 wet/dry		0.22	-				
Carrot (vegetative) (*Daucus carota* subsp. *Sativus*)	0.373 wet/dry		2.49	-				

Name (component)	Soil-plant BCF (plant/soil basis specified)[a]	Reference[b]	Soil-plant BCF (dw plant/dw soil)	Harvest index (range)	Harvest index (average)	Reference for harvest index	Weighted BCF[c]	BCF value used
Cucumber (fruit) (*Cucumis sativus*)	0.002 wet/dry	Lechner and Knapp (2011)	0.01	-	0.47	Lechner and Knapp (2011)	0.763	0.76
Cucumber (vegetative) (*Cucumis sativus*)	0.214 wet/dry		1.43	-				
GM of weighted BCF from all plant data								0.35

[a] BCF was reported as either ww plant/dw soil or dw plant/dw soil.
Where required, conversion to dw basis = (ww plant/dw soil) × 6.67 (following US EPA 1993).
[b] See Appendix D
[c] BCFs were weighted according to harvest index as follows: weighted $BCF = \sum(BCF_{component} \times HI_{component})$.
For example:
$WeightedBCF_{oat} = \sum(BCF_{oat\ grain} \times 0.3) + (BCF_{oat\ straw} \times (1 - 0.3)) = (0.007 \times 0.3) + (0.4 \times 0.7) = 0.28.$

3.6.2. Bioconcentration in Invertebrates

The accumulation and potential toxicity of PFOS in invertebrates is important both with respect to the vital roles invertebrates play in soil function and as a food source for birds and mammals. Stubberud (2006) examined the toxicity and accumulation of PFOS in the earthworm *Eisenia fetida* and determined BCFs at three test concentrations (10, 20 and 40 mg/kg dry soil) such that BCF = [earthworm ww]/[soil ww] = 2.8, 2.2, and 1.8 (ww basis), respectively, or 14.6, 11.8, and 9.6 on dw basis (Table 6). For the purpose of comparison, these PFOS BCF values for *E. fetida* are of the same order of magnitude as BCFs determined in the worm *E. andrei* for polyaromatic hydrocarbons (2.4–8.2 as adjusted for lipid and organic matter) (Jager *et al*. 2000), and for ortho-PCBs (1–34) (Matscheko *et al*. 2002).

Stubberud (2006) noted that BCFs were not adjusted for fat content in the worm or soil organic matter content, as is often done for other organic compounds, since PFOS does not appear to follow the classic pattern of accumulation in adipose tissue.

The Norwegian Pollution Control Authority (SFT; now the Climate and Pollution Agency) measured the soil-to-earthworm BCF in earthworms in PFAS-contaminated soil from four fire-training facilities (Table 6) (SFT 2008). Wet weight values were converted to dry weight using invertebrate wet fraction of 0.84 and soil moisture fraction of 0.15 (US EPA 1993).

Based on these two studies, the GMs of the available soil-worm BCFs for PFOS are 2.0 (ww basis) and 10.9 (dw basis).

Table 6. BCFs of PFOS in the earthworm *Eisenia fetida*

Site/soil type; distance from fire training area; sampling depth where reported	Concentration in worm (ng/g ww)	Concentration in worm (ng/g dw)[a]	Concentration in soil (ng/g ww soil)	Concentration in soil (ng/g dw soil)[b]	BCF $[worm]_{wet}/[soil]_{wet}$	BCF $[worm]_{dry}/[soil]_{dry}$	Reference
Artificial OECD soil (70% sand, 20% clay, 10% peat)	19,300	120,625	7,000	8,260	2.8	14.6	Stubberud (2006)
Artificial OECD soil (70% sand, 20% clay, 10% peat)	33,300	208,125	15,000	17,700	2.2	11.8	Stubberud (2006)
Artificial OECD soil (70% sand, 20% clay, 10% peat)	46,300	289,375	25,500	30,090	1.8	9.6	Stubberud (2006)
Mongstad oil refinery; distance: 25 m	265	1,656	377	445	0.7	3.7	SFT (2008)
Mongstad oil refinery; distance: 40 m	4,882	30,512	4,359	5,144	1.1	5.9	SFT (2008)
Mongstad oil refinery; distance: 200 m	64	400	10	12	6.5	34.2	SFT (2008)
Mongstad oil refinery; distance: 25 m	1,838	11,488	603	712	3.0	16.1	SFT (2008)
Mongstad oil refinery; distance: 52 m	16,814	105,088	6,880	8,118	2.4	12.9	SFT (2008)
Solberg Scandinavian AS fire training area; distance: 25 m	116	725	236	278	0.5	2.6	SFT (2008)
Solberg Scandinavian fire training area; distance: 38 m	75	469	55	65	1.4	7.2	SFT (2008)
Solberg Scandinavian fire training area; distance: 155 m	22	138	32	37	0.7	3.7	SFT (2008)
Gardermoen airport; distance: 1 m; depth: 0–5 cm	5,938	37,112	959	1,132	6.2	32.8	SFT (2008)
Gardermoen airport; distance: 1 m; depth: 10–30 cm	6,317	39,481	1,721	2,031	3.7	19.4	SFT (2008)
Gardermoen airport; distance: 1 m; depth: 65–80 cm	2,086	13,038	845	997	2.5	13.1	SFT (2008)
Rygge Air Station; distance: 26 m	649	4,056	227	268	2.9	15.1	SFT (2008)
Rygge Air Station; distance: 38 m	209	1,306	136	160	1.5	8.2	SFT (2008)
Rygge Air Station; distance: 174 m	117	731	29	34	4.1	21.5	SFT (2008)
Geometric mean					**2.0**	**10.9**	

[a] Wet weight conversion factor (worm) = earthworm wet mass/(earthworm wet mass − earthworm water fraction). Assuming a 1 g worm = 1/(1 − 0.84) = 1/0.156 = 6.25 (US EPA 1993).

[b] Wet weight conversion factor (soil) = soil wet mass/(soil wet mass − soil water fraction). Assuming 1 g wet soil = 1/(1 − 0.15) = 1/0.85 = 1.18 (US EPA 1993).

3.6.3. Bioaccumulation and Biomagnification in Terrestrial Mammals

Studies on the bioaccumulation and biomagnification of PFOS in terrestrial mammals have been conducted on caribou and wolf (Müller *et al*. 2011), sheep (Kowalczyk *et al*. 2012), and dairy cows (Vestegren *et al*. 2013).

Müller *et al*. (2011) reported the biomagnification factors (BMFs) and trophic magnification factors (TMFs) of perfluorinated carboxylates and perfluorinated sulfonates in terrestrial food webs consisting of lichen and other vegetation, caribou (*Rangifer tarandus groenlandicus*), and wolf (*Canis lupus*) associated with two caribou herds in remote northern areas in Canada (the Porcupine herd in northern Yukon and the Bathurst herd in Northwest Territories/western Nunavut). BMFs for PFOS were highly tissue specific, ranging from a low of 0.8 for $wolf_{liver}/caribou_{liver}$ to a high of 9.1 for $caribou_{whole}$/vegetation (see Table 7).

Table 7. BMFs and TMFs for plant-caribou-wolf food chain

Tissue type	Name of herd (geographic region)	Reported magnification factor
Biomagnification factor		
$Caribou_{muscle}$/lichen	Porcupine	2.0 ± 1.8
	Bathurst	3.6 ± 1.0
$Caribou_{muscle}$/vegetation	Porcupine	4.0 ± 3.7
	Bathurst	3.1 ± 0.9
$Caribou_{liver}$/lichen	Porcupine	49 ± 19
	Bathurst	102 ± 18
$Caribou_{liver}$/vegetation	Porcupine	97 ± 46
	Bathurst	88 ± 18
$Wolf_{muscle}/caribou_{muscle}$	Porcupine	4.5 ± 3.8
	Bathurst	1.8 ± 0.5
$Wolf_{liver}/caribou_{muscle}$	Porcupine	51 ± 43
	Bathurst	22 ± 5.8
$Wolf_{liver}/caribou_{liver}$	Porcupine	2.1 ± 0.6
	Bathurst	0.8 ± 0.1
$Caribou_{whole\ body}$/lichen	Porcupine	4.8 ± 2.3
	Bathurst	9.1 ± 1.6
$Caribou_{whole\ body}$/vegetation	Porcupine	9.1 ± 4.9
	Bathurst	7.9 ± 1.6
$Wolf_{whole\ body}/caribou_{whole\ body}$	Porcupine	3.3 ± 1.1
	Bathurst	1.2 ± 0.2
Trophic magnification factor		
$Wolf_{liver}/caribou_{liver}$/lichen	Porcupine	6.7 ± 0.3
	Bathurst	5.2 ± 0.4
$Wolf_{whole\ body}/caribou_{whole\ body}$/lichen	Porcupine	2.6 ± 0.1
	Bathurst	2.4 ± 0.1
$Wolf_{liver}/caribou_{liver}$/vegetation	Porcupine	5.1 ± 0.4
	Bathurst	4.3 ±0.4
$Wolf_{whole\ body}/caribou_{whole\ body}$/vegetation	Porcupine	2.2 ± 0.1
	Bathurst	2.3 ± 0.1

Source: Müller *et al*. (2011) supporting information.
BMF = carnivore/herbivore
TMF = see text for detail

Although no BAF from soil → carnivore was identified in the literature, data do exist for both the (soil→ plant) and (plant → grazing herbivore → carnivore) portions of the food chain. These can be combined to determine the soil → carnivore BAF using two methods. There is good agreement in the values derived using the two methods, so the simpler, more intuitive method is preferred for its simplicity and reproducibility.[2] This method involves combining the BMFs over three trophic levels such that:

$$BMF = \frac{Carnivore}{Soil} = \frac{Plant}{Soil} \times \frac{Herbivore}{Plant} \times \frac{Carnivore}{Herbivore}$$

$$= BCF_{soil \rightarrow plant} \times BAF_{plant \rightarrow herbivore} \times BAF_{herbivore \rightarrow carnivore}$$

where BCF soil → plant (0.35) is the GM of weighted BCFs shown in Table 7.

For the Porcupine herd: $BMF_{soil \rightarrow carnivore} = 0.35 \times 9.1 \times 3.3 = 10.5$

For the Bathurst herd: $BMF_{soil \rightarrow carnivore} = 0.35 \times 7.9 \times 1.2 = 3.3$

Therefore, the GM for the two herds = $\sqrt{}(10.5 \times 3.3) = 5.9$. This substantiates that PFOS is bioaccumulative in higher-level terrestrial ecosystems.

In a pilot study, Kowaleczyk *et al*. (2012) fed sheep PFOS-contaminated corn silage (90 µg/kg dry matter). Although the sample size was extremely small (n = 2), transfer of PFOS from feed to milk and meat was identified. Sheep 1 was fed 1.16 µg PFOS/kg bw/day for 21 days and allowed a further 21 days of non-PFOS diet. Sheep 2 was fed 1.45 µg PFOS/kg bw/day for 21 days and then slaughtered (no clearing period). Both sheep showed marked PFOS accumulation. Levels in sheep 1 and sheep 2 tissues were: plasma (168 and 240 µg/L), liver (885 and 1,172 µg/kg ww), kidney (172 and 286 µg/kg) and muscle (24 and 35 µg/kg), respectively. Elimination through milk (2%), and urine and feces (4%) was slow and indicated that the 21-day PFOS-free feeding period was not sufficient for a marked decrease in PFOS levels in organs and tissue (94% of dose was not excreted). For sheep 1, the BAFs (concentration in tissue/feed) were 9.8 in liver, 1.9 in kidney and 0.3 in muscle. For sheep 2, the BAFs were 13.0 in liver, 3.2 in kidney and 0.4 in muscle. Unfortunately, the concentration of PFOS in soil was not reported and therefore the soil-silage-sheep transfer values could not be calculated.

In a study of dairy cows in Sweden (Vestegren et al. 2013), silage, barley, supplements and water contributed 86, 10, 3 and 1%, respectively of the PFOS intake through diet. The authors noted that

[2] In the second, more complex method, BMF = $BAF_{plant \rightarrow carnivore}$ is based on the TMF, where TMF = e^b and b is the slope of ln C_{ww} = a + (b × TL), Cww = concentration of PFOS in an individual organism, and TL = trophic level.
Then BMF=[Consumer][Diet]=e(a+bTLConsumer)e(a+bTLDiet)=e(a+bTLConsumer-(a+bTLConsumer)=e(b(∆TL), and ∆TL is approximated as 2.25 for vegetation-to-whole wolf (Müller *et al*. (2011); Table 7 geometric mean of TMFs for Porcupine and Bathurst herds).e2.25b=eb2.25=TMF2.25
Average TMFplant→ carnivore= $2.25^{2.25}$ = 6.2. This value is reassuringly close to the value of 5.9 derived using the first method.

the PFOS concentration in the rural groundwater was very low (0.073 ng/L) and that if Stockholm tap water had been the source of the cows' drinking water instead, it would have contributed 46% of the total intake of PFOS. Accumulation of PFOS in cow tissue was greatest in liver (130 ng/kg), followed by blood (110 ng/kg) and muscle (21 ng/kg). Elimination was primarily via feces (45%) and milk (40%) and, to a lesser degree, urine (15%). Bioaccumulation was reported as a biotransfer factor (BTF) and calculated as concentration of chemical in tissue (ng/kg) divided by total daily intake rate of chemical (ng/day). Authors expressed these as log BTF, but the values have been converted here for ease of comparison with other studies. The BTFs were 0.07 for muscle and 0.02 for milk (reported as log BTF muscle: −1.15 and log BTF milk: −1.67).

The above studies with wildlife and livestock indicate that PFOS can biomagnify to a significant degree in the environment. Therefore, the soil and food ingestion pathways for primary, secondary and tertiary consumers should be considered in the development of CSOQG for the environment.

3.7. Snow

Some authors have investigated the fate of PFAS in snow. MacInnis *et al.* (2017), Codling *et al*. (2014*a*) and Meyer *et al*. (2011) observed that snow in Sweden and Canada generally contained higher concentrations of short-chain PFAS. They also observed that PFAS migrated to deeper snow during snowmelt and that concentrations of the shorter-chain perfluoroalkyl carboxylic acids (PFCAs; including PFOA) tended to diminish as snow melted, but that longer-chain PFCAs and perfluoroalkyl sulfonic acids (PFSAs; including PFOS) concentrations increased (Codling *et al.* 2014*a*). Meyer *et al*. (2011) observed an early drop in short-chain PFAS in runoff water during early snowmelt in association with the influx of snowmelt water, which has a relatively low concentration of PFAS when compared to the surface water being measured. However, the authors also observed a peak of long-chain PFAS in stream water, presumably due to mobilization of particles from impervious surfaces in the urban environment.

Taniyasu *et al*. (2013*b*) also noted an increase in PFAS in freshly fallen snow over several days in Japan, which Codling *et al*. (2014*a*) indicate could be due to ice surface–mediated photochemical formation of PFAS from precursors.

These trends may have implications for temporal sampling and impacts at northern sites where PFAS accumulation could occur where the snowpack does not thaw annually.

4. BEHAVIOUR AND EFFECTS IN BIOTA

The toxicological data available for PFOS for plants, invertebrates, mammals and birds identified using the search strategy described in Appendix B are presented in:

- Appendix E: Toxicity Data of PFOS to Terrestrial Plants and Invertebrates Acceptable/Selected for Use for Soil Quality Guideline Derivation
- Appendix F: Toxicity Data of PFOS to Terrestrial Plants and Invertebrates Consulted but Not Used for Soil Quality Guideline Derivation
- Appendix G: Acceptable/Selected Mammalian and Avian Toxicity Data for PFOS

- Appendix H: EC_{25}, IC_{25} and LC_{20} Data Used for Species Sensitivity Distribution Used to Derive Soil Contact Value for Agricultural, Residential/Parkland and Commercial and Industrial Land Uses for PFOS.

4.1. Plants and Invertebrates

Two studies (Brignole *et al*. 2003; Zhao *et al*. 2011) investigated the toxicity of PFOS to plant growth, seedling emergence and plant mortality in eight plant species and were acceptable for guideline derivation. Species tested were: alfalfa, ryegrass, soybean, lettuce, flax, tomato, onion and pak choi (*Brassica chinensis*). Five studies of acceptable quality for guideline derivation (EC 2015; Joung *et al*. 2010; Sindermann *et al*. 2002; Stubberud 2006; Xu *et al*. 2011) reported the toxicity of PFOS to three invertebrate species: the earthworm *Eisenia fetida*, (endpoints: number of cocoons, number of juveniles, average and total weight of juveniles and mortality), the springtail, *Folsomia candida*, and oribatid mite, *Oppia nitens* (endpoints for both were number of juveniles produced and mortality). Appendix E presents data from the acceptable studies.

Additional endpoints were also available in some of these studies or in other studies (Brignole *et al.* 2003; Joung *et al*. 2010; Sindermann *et al*. 2003; Stubberud 2006) but were considered unacceptable for guideline derivation, either because endpoints were reported at an unknown concentration, were extrapolated beyond the measurement range or were related to behaviour. A further six studies on terrestrial biota (Li 2008; Mommaerts *et al*. 2011; OECD 2002; Qu *et al*. 2010; Van Gossum *et al*. 2010; Xu *et al*. 2011) were reviewed but not used, since the test medium was filter paper, agar or sugar solution rather than soil (see Appendix F).

From the available information, plants and invertebrates have overlapping sensitivity to PFOS, with plants appearing to be slightly more sensitive than invertebrates. The most sensitive species was lettuce, where plant height was reduced by 23% and weight by 35%, after 21-day exposure to PFOS at 3.91 mg/kg (Brignole *et al*. 2003). For invertebrates, the most sensitive effect was a 25% reduction in average weight per juvenile observed after 56-day exposure at 12 mg/kg (Stubberud 2006).

LOEC values ranged widely both in concentration and effect level, from 3.91 mg/kg for adverse effect on lettuce height (23% decrease) and weight (35% decrease) to 1,000 mg/kg for decreased emergence of alfalfa (64%), lettuce (86%), tomato (89%) and flax (100%) after 21 days of exposure (Brignole *et al*. 2003). This wide variation in effect level is one reason why the LOEC method is not the preferred method for deriving SoQGs. For earthworm, LOECs ranged from 20 mg/kg for average weight of juveniles to 80 mg/kg for number of cocoons and juveniles (Stubberud 2006).

Acceptable IC_{25} (inhibitory concentration of 25%), EC_{25} (effective concentration for 25% of individuals) and LOECs (where adverse effect levels were close to the 25% level, i.e., ranged from 20 to 30% and were not redundant with the EC_{25} or IC_{25}) were available for seven plant species and three invertebrate species. IC_{25}/EC_{25} ranged from 3.91 mg/kg for reduced height in lettuce to 393 mg/kg for reduced emergence of lettuce, both following 21-day exposure (Brignole *et al*.

2003). For invertebrates, IC_{25} ranged from 12 to 256 mg/kg for effects on average weight of juvenile earthworm and earthworm survival (Joung *et al*. 2010; Stubberud 2006).

In plants, median effect levels ranged from an IC_{50} (inhibitory concentration 50%) of 20.1 mg/kg for decreased shoot weight for lettuce (Brignole *et al*. 2003) to an EC_{50} (effective concentration for 50% of individuals) of 745 mg/kg for seedling emergence for alfalfa (Brignole *et al*. 2003). Soybean was considerably less sensitive, with no adverse effect on survival at 1,000 mg/kg (Brignole *et al*. 2003). In the invertebrates, median effect levels ranged from 23 mg/kg for the IC_{50} for number of juveniles (EC 2015) to 955 mg/kg for LC_{50} (lethal concentration 50%) (Xu *et al*. 2011).

4.2. Vertebrates, Birds and Other Wildlife

Toxicity data in non-human vertebrates (rat, mice, rabbit and monkey), and avian species (bobwhite quail [*Colinus virginianus*], Japanese quail [*Coturnix japonica*] and mallard duck [*Anas platyrhynchos*]) were reviewed during the derivation of federal water quality guidelines to protect wildlife (EC 2013*b*), and key mammalian and avian toxicity endpoints were identified. Since the exposure route in the key studies was by ingestion in diet, and since no other toxicological data using wild species were available, the same key mammalian and avian toxicity endpoints were considered appropriate for use in deriving soil guidelines to protect wildlife exposed to PFOS in diet. A summary of the data is provided below and in Section 5.4 and Section 5.5. Species that are relevant (i.e., commonly occurring) and have feeding habits or body characteristics that make them conservative representative model species (e.g., high food intake to body weight ratio) were used in the food chain calculations.

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

Nine studies were evaluated for four mammal species: cynomolgus monkeys, rabbits (*Oryctolagus cuniculus*), mice and rats. Since no toxicity data were available for wildlife, the lowest-observed-adverse-effect level (LOAEL) from these toxicity data was used.

The lowest adverse effects dose for primary consumer (ED_{1C}) was based on the LOAEL in a two-year chronic toxicity diet study in rat (Covance Laboratories Inc. 2002), which reported hepatocellular degeneration at concentration in diet of 2 mg PFOS/kg food. This value is the same

critical value used in the EC (2006*a*) Screening Assessment Report to assess risk to mammalian wildlife. This concentration corresponded to an intake of 0.06–0.23 mg/kg bw/day (males) or 0.07–0.21 mg/kg bw/day (females), or an average intake of 0.1086 mg/kg bw/day given the average weekly food consumption rates for males and females over the 104-week test period.

Toxicity data for three avian species exposed to PFOS via diet were available: northern bobwhite quail, Japanese quail, and mallard. The ED_{1C} LOAEL dose rate in northern bobwhite was 772 µg/kg bw/day (0.772 mg/kg bw/day), which resulted in reduced chick survival 14-day post exposure (Newsted *et al*. 2007).

5. BEHAVIOUR AND EFFECTS IN HUMANS AND NON-HUMAN MAMMALIAN SPECIES

Several international health agencies have reviewed the behaviour and effects of PFOS in humans and mammalian species (e.g., ATSDR 2015; EFSA 2008; HC 2006). The present document focusses on the studies most relevant to the PFOS toxicity reference values that will be used to develop SoQGs for the protection of human health. This document is based on the review conducted in 2013 (Sanexen 2013) and on the subsequent dose-response analysis (HC 2018*a*).

PFOS has not been reviewed for carcinogenicity by the International Agency for Research on Cancer, the US EPA Integrated Risk Information System or the US National Toxicology Program. PFOS has been identified as non-genotoxic in many assays, so a threshold approach should be used to assess the risk of cancer.

5.1. Toxicokinetics

PFOS is readily absorbed after oral exposure. Metabolic elimination seems to play no relevant role in primates, and the elimination half-lives in primates are significantly longer than in rodents. Nevertheless, when repeatedly administered, PFOS shows a tendency to accumulate in rats.

5.2. Absorption

5.2.1. Oral Route

Studies conducted in laboratory animals indicate that PFOS is readily absorbed through the gastrointestinal tract of rats, but no controlled studies in humans were available. Greater than 95% of an administered dose of [^{14}C]PFOS 4.2 mg/kg-bw was absorbed after 24 hours in non-fasted rats (Johnson and Ober 1979; 1999). Another study conducted in male Sprague Dawley rats reported similar absorption rates (97.2% and 97.4%) within 24 hours of receiving 5 and 20 mg/kg, respectively by gavage (Cui *et al.* 2010).

Blood uptake of PFOS varied depending on the isomer in male Sprague Dawley rats administered a single oral dose of 400 mg/kg-bw PFOS (70% L-PFOS [n-PFOS]). The relative blood uptake of the various isomers was 0.24–17.5 times the blood uptake of the n-PFOS (Benskin *et al*. 2009).

5.2.1.1. Inhalation and Dermal Routes

No studies on the absorption of PFOS following inhalation exposure in animals or humans were located. However, higher serum concentrations observed in fluorochemical production industry workers compared to the general US population suggest that absorption could possibly occur through inhalation exposure (ATSDR 2015).

Limited information for animals suggests that dermal absorption of PFOS may occur. Albino rabbits (one per sex) were administered 5,000 mg PFOS/kg-bw to clipped, intact skin under occluded conditions for 24 hours (O'Malley and Ebbens 1981, as cited in 3M 1999). Blood fluoride levels from day 1 (before exposure) were 10.3 ppm (males) and 0.9 ppm (in females), and from day 28 were 130 ppm (in males) and 128 ppm (in females).

5.2.2. Distribution

The experimental data available in rats, mice and monkeys (volume of distribution) are consistent with an extracellular distribution of PFOS (Chang *et al*. 2012).

5.2.2.1. Distribution into Blood, Organs and Tissues

In rats and mice, PFOS is found mainly in the liver, kidneys, lungs and blood, with lower levels in most other organs, including the central nervous system (Austin *et al*. 2003; Benskin *et al*. 2009; Bogdanska et al. 2011; Chang *et al*. 2012; De Silva *et al*. 2009). A similar pattern is observed based on primate (Seacat *et al*. 2002) and human data (Kärrman *et al*. 2010; Maestri *et al*. 2006; Olsen *et al*. 2003*a*), with serum/plasma, liver and lungs having relatively high concentrations of PFOS. Data in both rodents (Chang *et al*. 2012; Johnson and Ober 1979) and humans (Ehresman *et al*. 2007) indicate that PFOS is not selectively retained in red blood cells and is preferentially bound to albumin in serum and, to a lesser extent, plasma γ-globulin, α-globulin, α-2-macroglobulin, transferrin and β-lipoproteins (ATSDR 2015; Butenhoff *et al.* 2012*b*). Lungs are target organs in neonate rats (Grasty *et al*. 2005*a*), where PFOS has been associated with cyanosis (Borg *et al*. 2010). PFOS has also been found in amniotic fluid in humans (Jensen *et al*. 2012). Cord blood concentrations correlate to maternal serum concentrations in humans (Fei *et al*. 2007; Gützkow *et al*. 2012; Inoue *et al*. 2004; Liu *et al.* 2011; Midasch *et al*. 2007; Needham *et al*. 2011). PFOS has been shown to cross the placenta in humans (EFSA 2008) and rodents (Borg *et al.* 2010; Chang *et al*. 2009; HC 2006; Kim *et al.* 2011*a*; Lau *et al*. 2003; Loccisano *et al*. 2012*a*, 2012*b*; Luebker *et al*. 2005*a*; Thibodeaux *et al*. 2003; Wang *et al*. 2010; 2011; Zeng *et al*. 2011). PFOS was also shown to competitively bind to transthyretin, which is the main thyroxin carrier in the cerebrospinal fluid. Present at very high concentrations during the prenatal period and early life, transthyretin plays an important role in central nervous system development (Weiss *et al*. 2009).

While PFOS has been shown to cross the blood-brain barrier in neonates, it does not seem to pass as freely in adults, or it is actively extruded from cerebral spinal fluid in adults (Harada *et al*. 2007). The authors suggested that this active transport might be saturated at high doses and the immaturity of the blood-brain barrier might induce toxicological effects in the developing central nervous system (Harada *et al.* 2007; Lau *et al*. 2006). See HC (2018*a*) for more details about the distribution of PFOS.

5.2.2.2. Age, Gender and Species-specific Differences in PFOS Distribution

Human PFOS blood levels were shown to be influenced by age and gender. In the general US population (National Health and Nutrition Examination Survey [NHANES] 1999–2008 data) and in other reference populations (not exposed to contaminated drinking water), serum PFOS levels were reported to be significantly higher in males than in females (Frisbee *et al*. 2009; Harada *et al.* 2004, Ingelido *et al*. 2010; Kato *et al.* 2011; Mondal *et al*. 2012), regardless of age (Kato *et al.* 2011). However, as age increased, PFOS concentrations decreased in males and increased in females (Kato *et al.* 2011). Mondal *et al*. (2012) showed that children had 42% higher average PFOS levels than their mothers throughout childhood. No sex-related differences were observed in one study in cynomolgus monkeys (Chang *et al*. 2012). Studies in rodents have found gender-related differences in tissue concentrations where serum PFOS levels were higher in female than male rats (Chang *et al*. 2012; Seacat *et al*. 2003) but not in liver (Seacat *et al*. 2003). Studies have also found age-related variations in mice (Liu *et al*. 2009*b*).

5.2.2.3. Lactational Transfer

PFOS can be transferred from the mother to the infant through lactation. PFOS levels in human milk were correlated with levels in maternal serum (as reviewed by Liu *et al*. 2011). Some studies showed that PFOS milk concentrations decreased as the number of infants breastfed by the mother increased (Kadar *et al*. 2011; Tao *et al*. 2008*b*) and decreased significantly through the lactation period (decrease of milk concentration of 37% over one year of lactation) (Thomsen *et al.* 2010). Maternal serum levels were shown to decrease in a similar manner with lactation (Monroy *et al*. 2008; von Ehrenstein *et al*. 2009). However, another study reported an upward trend for PFOS levels in milk through six months of lactation (Tao *et al*. 2008*b*).

5.2.3. Metabolism

The available data indicate that PFOS is not metabolized. Studies conducted in rodents and non-human primates did not reveal quantitatively significant metabolism of PFOS (as reviewed by ATSDR 2015). There are no reports of PFOS metabolites formed *in vivo* (EFSA 2008).

5.2.4. Elimination

Data available regarding the elimination of PFOS indicate marked inter-species variations, with a dramatically longer elimination half-life from serum in humans (years) than in non-human primates (months) and rats (weeks). These large differences represent a significant limitation for inter-species extrapolations, as substantially different steady-state internal doses will result from similar external PFOS dosages (i.e., mg/kg/day); steady-state serum concentrations achieved in humans may be several orders of magnitude greater than in monkeys or rats. Further, longer exposure durations would be required to achieve steady-state concentrations in humans than in monkeys or rats. However, such species-specific differences can be accounted for in physiologically-based pharmacokinetic (PBPK) models (see Section 5.2.6).

In rodents, PFOS is excreted mainly in urine, bile and feces. In humans, excretion in bile has been shown to be more important than in urine; however, both renal and biliary/fecal excretion rates were much lower than in rodents and non-human primates (Genuis *et al*. 2010; Harada *et al*. 2007). Menstrual bleeding and lactation may be significant routes of elimination in human females (Harada and Koizumi 2009; Kim *et al*. 2011*a*; von Ehrenstein 2009). The above findings are supported by PBPK modelling with data from Luebker *et al*. (2005*a*), which indicated that the dam may eliminate PFOS via the fetus and lactation to pups, which are exposed in turn. Overall, the modelling predicted the internal exposure to be greater in fetal and infant plasma and brain than in the corresponding maternal tissues, the latter effect likely being a function of immaturity of the fetal blood-brain barrier (Loccisano *et al*. 2012*a*, 2012b). See HC (2018*a*) for more information on elimination.

5.2.5. Concentrations in Human Tissues and Body Fluids

PFOS has been measured in the serum of occupationally exposed workers (during the manufacture or processing of PFAS) as well as in the serum and cord blood of the general population. Serum concentrations reflect cumulative exposure over several years (ATSDR 2015). Plasma concentrations in males were higher than in females from the general Canadian population, as measured in the Canadian Health Measures Survey (CHMS), cycles 1 and 2 (HC 2010*a*; HC 2013). Both the CHMS and US NHANES study data showed a downward temporal trend in PFOS plasma and serum concentrations (HC 2010*a*; HC 2013; Kato *et al*. 2011). See HC (2018*a*) for more details.

5.2.6. Pharmacokinetic Models

Several pharmacokinetic and PBPK models have been developed for humans and experimental animals (Andersen *et al*. 2006; Loccisano *et al*. 2011; 2012*a*, 2010*b*; 2013; Tan *et al*. 2008). These models reproduced controlled dosing data in rats and monkeys (Andersen *et al*. 2006; Loccisano *et al*. 2012*a*, 2012*b*; 2013; Tan *et al*. 2008) and biomonitoring data in humans (Loccisano *et al*. 2011). No controlled dosing data was available for humans. However, there is only moderate confidence in the models because different model codes were used for distinct species, model fit to some data sets was not optimal, and there were weaknesses in addressing observed (but not

understood) sex differences in the models (HC 2018*a*). These factors, along with the lack of controlled dosing data for model validation in humans, led HC (2018*a*) to assess PFOS using ratios of PBPK model–predicted dose metrics to calculate AK_{UF} (toxicokinetic portion of the interspecies uncertainty factor). This approach can account for the non-linear kinetics of PFOS and facilitate better comparison of inter-study data. Using the Loccisano models and physical and chemical-specific parameters (instead of default uncertainty or allometric scaling factors) from Campbell and Clewell (2013), steady-state plasma and liver concentrations were calculated (see HC 2018*a*). Plasma values were considered appropriate proxies for adverse hepatic effects and allowed for better consistency across a wide variety of endpoints (HC 2018*a*) to determine the points of departure. These values were based on ingestion via drinking water only. See HC (2018*a*) for more details of this approach and pharmokinetic and PBPK models developed for PFOS.

5.3. Acute Toxicity

Acute exposure in humans or animal models refers to exposures of <14 days, but often involves a single administered dose (HC 2010*b*). This section summarizes the effects of PFOS in acute animal studies. No studies documenting the acute toxicity of PFOS in epidemiological studies through the oral, inhalation or dermal route were located.

5.3.1. Oral Exposure

5.3.1.1. Mortality

A mean oral LD_{50} (lethal dose 50%) of 251 (199–318) mg/kg bw was calculated for CD rats based on a single gavage administration of PFOS (100–1,000 mg/kg-bw; five/sex/group) (Dean and Jessup 1978).

5.3.1.2. Neurotoxicity

Neurological effects were observed in rodents after single oral doses of PFOS (Johansson et al. 2008; 2009; Kawamoto *et al*. 2011; Sato *et al*. 2009). Johansson *et al*. (2008) determined a LOAEL of 0.75 mg/kg bw/day (no no-observed-adverse-effect level [NOAEL]) for time-dependent and dose-related neurodevelopmental effects in male NMRI mice administered a single gavage dose of PFOS (0.75 or 11.3 mg) at the age of 10 days. Neonatal exposure under these conditions significantly increased the level of proteins that are important for normal brain development (Johansson *et al*. 2009).

5.3.1.3. Thyroid Hormones

Yu *et al*. (2011) identified a LOAEL of 1.0 mg/kg bw/day (NOAEL = 0.2 mg/kg bw/day) for altered serum thyroid hormones in female Sprague Dawley rats exposed via gavage over five days.

5.3.1.4. Liver Toxicity

Elcombe *et al.* (2012*a*) identified a LOAEL of 1.9 mg/kg bw/day for liver toxicity after dietary exposure (1.9 or 9.6 mg/kg bw/day; seven days) of male Sprague Dawley rats to K^+PFOS (potassium perfluorooctanesulfonate). Other observed effects include decreased body weight, increased liver weight, hepatocellular hypertrophy, decreased cholesterolemia, decreased serum alanine aminotransferase or aspartate transaminase, and increased activity of key hepatic marker enzymes (peroxisome proliferator-activated receptor alpha [PPARα], constituted activated receptor and PXR (pregnane X receptor).

5.3.2. Inhalation

An inhalation LC_{50} of 5,200 mg/m^3 (95% confidence interval [CI]: 4,400–6,400) was determined in Sprague Dawley rats (five/sex/group) exposed to 1,890–45,970 mg/m^3 PFOS dust in air for one hour (Rusch 1979; Rusch *et al*. 1979).

5.3.3. Dermal

Skin irritation was not observed in a study with dermally exposed New Zealand white rabbits (Biesemeier and Harris 1974, as cited in HC [2018*a*]). However, eye irritation was reported in rabbits after 0.1 mL ocular application, with washout after 5 or 30 seconds (Biesemeier and Harris 1974; Corning Hazleton Inc. 1997; Hazleton Laboratories America Inc. 1987; Hazleton Wisconsin Inc. 1994; Riker Laboratories Inc. 1981, as cited in HC 2018*a*; Warf Institute Inc. 1975).

5.4. Subchronic Exposure

Subchronic exposure in humans and animal models is generally considered to be greater than 14 days and <90 days (HC 2010*b*). This section summarizes the effects of oral exposure to PFOS in subchronic animal studies. No epidemiological studies documenting the subchronic toxicity of PFOS were located, and the relevance of extrapolating results from experimental animals to humans must be assessed based on the data describing inter-species differences.

5.4.1. Oral Exposure

Most available experimental data were obtained in mice, rats and monkeys exposed through the oral route (diet, gavage or drinking water). The database includes general toxicity studies (including studies focussing on thyroid hormones) and specialized studies for neurotoxicity and immunotoxicity. The most sensitive endpoint categories identified include the following:

- Modulation of the immune response, which appears to be the most sensitive target (DeWitt *et al*. 2009; Dong *et al*. 2009; 2011; Peden-Adams *et al*. 2008; Zheng *et al*. 2009; 2011). A LOAEL of 0.00166 mg/kg bw/day (serum PFOS: 91.5 ng/mL) and a NOAEL of

0.000166 mg/kg bw/day (serum PFOS: 17.8 ng/mL) was determined in mice (Peden-Adams *et al*. 2008).

- Liver toxicity (Butenhoff *et al*. 2012*a*), with a LOAEL of 0.024 mg/kg bw/day, and alteration of serum lipids (Elcombe *et al*. 2012*b*; Seacat *et al*. 2002; 2003), with a LOAEL of 0.03 mg/kg bw/day.
- Altered thyroid hormones in pregnant/non-pregnant animals and in offspring after gestational and/or lactational exposure (Lau *et al*. 2003; Seacat *et al*. 2002; Thibodeaux *et al*. 2003; Yu *et al*. 2009*b*), with a LOAEL of 0.15 mg/kg bw/day (Seacat *et al*. 2002).

Effects observed at higher doses in mice, rats and/or monkeys include other developmental outcomes (delayed eye opening, skeletal/visceral abnormalities, heart and lung injuries), decreased body weight or reduced weight gain, hepatotoxicity, increased organ weight (liver, kidney, spleen), and altered glucose levels.

5.4.1.1. Immunotoxicity

The magnitude of immunotoxic effects across species (including non-laboratory animals) varies considerably, as do strain and route of administration. Enough evidence exists for several immunologic effects for them to be considered in the derivation of toxicological reference values. Current evidence suggests that the male B6C3F1 mouse is the most sensitive experimental model. The lowest immune effects LOAEL from this study is 0.00166 mg/kg bw/day (NOAEL = 0.000166 mg/kg bw/day), for a dose-related suppression of sheep red blood cell–specific immunoglobulin M (IgM) T cell–dependent antibody response following gavage exposure for 28 days (Peden-Adams *et al*. 2008). In contrast, Qazi *et al*. (2010*a*) did not observe any changes in male C57BL/6 mice for the same endpoint. Male mice were more sensitive than female mice for many immune effects after PFOS exposure. Other immune effects were also observed at higher doses in this and other studies (DeWitt *et al.* 2009; Dong *et al.* 2009; 2011; Guruge *et al*. 2009; Keil *et al*. 2008; Zheng *et al*. 2009; 2011). More details on these and other studies can be found in HC (2018*a*). More details on immunologic effects identified by the IPCS (2012) continuum of strength of evidence are also described in HC (2018*a*).

5.4.1.2. Hepatic Effects

Increased liver weight was the hepatic effect occurring at the lowest exposure levels in various studies. Dong *et al*. (2009) determined a LOAEL of 0.0833 mg/kg bw/day for C57Bl/6 mice exposed via gavage for 60 days (Dong *et al*. 2009). This effect was observed in many other mouse studies (Dong *et al*. 2012; 2011; Era *et al*. 2009; Qazi *et al*. 2009; 2010*a*, 2010*b*; Thibodeaux *et al*. 2003; Wan *et al*. 2011; Wang *et al*. 2011; Yahia *et al*. 2008; Zhang *et al*. 2013; Zheng *et al*. 2009; 2011). However, the effect was not observed at some higher doses for shorter durations (Abbott *et al*. 2009; Fair *et al*. 2011; Wan *et al*. 2011). In female rats, the LOAEL was determined at 0.15 mg/kg bw/day (Lefebvre *et al*. 2008), and several others also observed the effect: Goldenthal *et al*. (1978*a*), NOTOX (1999), Seacat *et al*. (2003), Thibodeaux *et al*. (2003), Cui *et al*. (2009), Yu *et al*. (2009*a*) and Elcombe *et al*. (2012*a*). In monkeys, the LOAEL was 0.75 mg/kg bw/day (Seacat *et al*. 2002). Other histological changes in the liver and increases in serum enzymes (indicators of adverse hepatic effects) were observed at higher levels in several rat studies

(Butenhoff *et al*. 2012*a*; Cui *et al*. 2009; Elcombe *et al*. 2012*a*; Goldenthal *et al*. 1978*a*; NOTOX 1999; Seacat *et al*. 2003) and in monkeys (Seacat *et al*. 2002).

Serum enzyme levels were also altered in several studies (Goldenthal *et al*. 1978*a*, 1978*b*; Qazi *et al*. 2010*a*; Seacat *et al*. 2002; 2003).

5.4.2. Serum Lipids and Other Systemic Effects

Based on data from Seacat *et al*. (2002), HC (2013*a*) determined a LOAEL of 0.03 mg/kg bw/day for reduced HDL in male cynomolgus monkeys and 0.15 mg/kg bw/day for decreased total cholesterol in females. Other reported liver effects observed in monkeys receiving higher doses were decreased low-density lipoprotein (LDL), triglycerides, serum bilirubin and estradiol; increased serum bile acids; decreased body weight; increased liver weight and centrilobular vacuolation; hypertrophy and mild bile stasis; increased glycogen content (Seacat *et al*. 2002); lipid depletion in the adrenals; atrophy of pancreatic acinar and serous alveolar cells; signs of gastrointestinal toxicity; dehydration; general body trembling; and weight loss (Goldenthal *et al*. 1978*b*). Mortality was also observed at >0.75 mg/kg bw/day (Goldenthal *et al.* 1978*b*; Seacat *et al*. 2002). Clinical effects were reversed after 211 days of recovery amongst surviving animals (Seacat *et al*. 2002). More information on these studies and studies in rodents can be found in HC (2018*a*).

5.4.2.1. Neurotoxicity

Zeng *et al*. (2011) identified the lowest LOAEL for neurotoxic effects (0.1 mg/kg bw/day) for structural modification of the synapses of the hippocampus in Sprague Dawley rat pups following prenatal exposure. This was also accompanied by pro-inflammatory effects and reduced mRNA levels of synapsin 1, synapsin 2 and synaptophysin, which might be responsible for altered brain development. LOAELs were also identified for apoptosis of hippocampal cells and alterations of proteins involved in apoptosis in mice (2.15 mg/kg bw/day) (Long *et al*. 2013) and motor effects in rats (1.0 mg/kg bw/day) (Butenhoff *et al*. 2009). Other neurotoxic effects included behavioural and neuromotor effects in mice and rats (Fuentes *et al*. 2007*a*, 2007*b,* 2007*c*; Kawamoto *et al*. 2011; Ribes *et al*. 2010; Wang *et al*. 2010; 2012) and changes in expression of calcium-related signalling molecules in rats (Johansson *et al*. 2009; Liu *et al*. 2010*a*, 2010*b*).

5.4.2.2. Thyroid Hormones

Altered thyroid hormones have been reported in Sprague Dawley rats (Yu *et al*. 2009*a*) and cynomolgus monkeys (Seacat *et al*. 2002). In humans, EFSA (2008) and HC (2013*a*) independently reanalyzed data from Seacat *et al*. (2002) and both arrived at a LOAEL of 0.15 mg/kg bw/day (NOAEL = 0.03 mg/kg bw/day) for decreased TT3 (both sexes) and TT4 (females). Changes in thyroid hormones in rats were similar to those in monkeys, with dose-dependent decreases in thyroxin and reduced triiodothyronine at higher doses (Luebker *et al*. 2005*b*; Thibodeaux *et al*. 2003; Yu *et al*. 2009*a*; Wang *et al*. 2011) and changes in free thyroxin and

triiodothyronine were also observed. A serum PFOS LOAEL of 5,000 mg/ml was identified in rats for decreased total thyroxin levels (Yu *et al*. 2009*a*).

5.5. Chronic Exposure

Chronic exposure is generally assumed to be greater than 90 days (HC 2010*b*). This section summarizes effects of PFOS in chronic animal and human epidemiological studies. More information can be found in HC (2018*a*).

5.5.1. Experimental Studies in Rodents

5.5.1.1. Oral

Only one oral chronic study was identified, which determined a LOAEL of 0.024 mg/kg bw/day for cystic degeneration in the liver (Butenhoff *et al*. 2012*a*) following two years of oral exposure of Sprague Dawley rats via feed. At higher doses, several other hepatic effects were noted in males and females, as well as reduced spleen and thyroid/parathyroid weights, decreased serum total cholesterol and mortality in males and increased spleen, liver, kidney and brain weights, and reduced adrenal gland weight in females. See HC (2018*a*) for more details.

5.5.1.2. Reproductive and Developmental Toxicity

The reproductive and developmental database for PFOS is robust. A two-generation study was carried out in rats (Christian *et al*. 1999; Luebker *et al*. 2005*a*), and reproductive and developmental parameters have been investigated in many one-generation studies in rats, mice and rabbits.

Several studies (Johansson *et al*. 2009; Liu *et al*. 2010*b*; Wang *et al*. 2010; 2012; Zheng *et al*. 2011) identified changes in various neurotransmitters and proteins in the brain at ≥0.1 mg/kg bw/day. The most common neurobehavioral changes occurred at ≥0.3 mg/kg bw/day (HC 2018*a*). Neuromotor effects (decreased exploratory activity, increased number of resting periods and decreased muscle strength) were observed in adult mice exposed *in utero*, with more pronounced effects in males, at a LOAEL of 0.3 mg/kg bw/day (Onishchenko *et al*. 2011). Liu *et al*. (2009*a*) observed decreased success in the water maze test for rats exposed to 7.2 ppm in the diet (pre- and postnatally). Motor, learning and memory effects were less pronounced and at higher doses in rats (Butenhoff *et al*. 2009; Lau *et al*. 2003; Thibodeaux *et al.* 2003).

The lowest developmental LOAEL (0.1 mg/kg bw/day) for reduced post-weaning food consumption was identified in the parental F1 males of a two-generation reproductive study in rats (Christian *et al*. 1999). Other effects include reduced body weight gain and body weight, and signs of liver toxicity in the F0 generation. Christian *et al*. (1999) observed similar effects, as well as reduced number/survival of fetuses/pups and behavioural/development delays (F1) and reduced

body weight (F2). Similar fetal/pup weight effects were also observed in other studies in rats (Thibodeaux *et al*. 2003; Wang *et al*. 2011; Wetzel 1983) and mice (Era *et al*. 2009).

Changes in thyroid hormones and thyroid-related gene and protein expression were noted in rats (Chang *et al*. 2009; Thibodeaux *et al*. 2003; Wang *et al*. 2011; Yu *et al*. 2009*b*) and mice (Thibodeaux *et al*. 2003). Some of these effects may not reflect altered thyroid function, as the liver may be involved binding hormones (thyroxin) (Yu *et al*. 2009*b*), and effects on total thyroxin were shown to be transient or reversible by time of weaning in some studies (Lau *et al*. 2003; Thibodeaux *et al*. 2003). In contrast, no effects on some thyroid hormone levels were observed in mice (Fair *et al*. 2011) and rats (Lau *et al*. 2003).

Luebker *et al*. (2005*a*, 2005*b*) determined a LOAEL of 0.8 mg/kg bw/day for decreased pup survival and gestation length, and similar effects were observed in other studies in rats (Lau *et al*. 2003; Thibodeaux *et al*. 2003) and mice (Abbott *et al*. 2009; Yahia *et al*. 2008). Lau *et al*. (2003) determined a BMD_5 and $BMDL_5$ of 1.07 and 0.58 mg/kg bw/day, respectively. Grasty *et al*. (2003) suggest that late gestation (gestation day [GD] 17–20) is the critical window for neonatal mortality.

Prenatal PFOS exposure caused developmental landmark delays (see HC 2018*a*) and structural anomalies. Both heart injury (mitochondrial) (Xia *et al*. 2011) and lung injury (hemorrhage, thickened interalveolar septum, focal lung consolidation, inflammatory cell infiltration) (Chen *et al*. 2012*b*) were observed in rats exposed *in utero* through pregnancy (GD1 or 2–21), with LOAELs of 2 mg/kg bw/day. These and additional lung effects were observed at higher doses in rats and mice (Borg *et al*. 2010; Chen *et al*. 2012*b*; Grasty *et al*. 2003; 2005*b*). Grasty *et al*. (2003, 2005*b*) propose that PFOS-induced effects on lung maturation are possibly linked to early mortality observed in dams, fetuses and pups. However, Ye *et al*. (2012) did not observe any mortality in a study on rats exposed later in gestation (GD 12–18), but they did observe altered lung gene expression.

A LOAEL of 1.0 mg/kg bw/day (BMD_5 and $BMDL_5$ = 0.055 and 0.016 mg/kg bw/day, respectively) was determined for sternal effects in CD-1 mice (Lau *et al*. 2003; Thibodeaux *et al*. 2003) and ICR mice (Yahia *et al*. 2008). These authors and Wetzel (1983) also noted other skeletal anomalies at higher doses. Higher BMD_5 and $BMDL_5$ (0.313 and 0.122 mg/kg bw/day, respectively) were identified for sternal effects in rats (Lau *et al*. 2003; Thibodeaux *et al*. 2003). Era *et al*. (2009) identified a LOAEL of 13.0 mg/kg bw/day for cleft palate in mice. This was the same LOAEL as determined by Keil *et al*. (2008) for immune function changes in adult male mice exposed to PFOS *in utero*. In rabbits, fetal effects (reduced body weight and delayed ossification) occurred only at maternally toxic doses (Case *et al*. 2001).

Several studies showed decreased serum lipids in rats following exposure to PFOS prior to mating and during gestation and/or lactation (Elcombe *et al.* 2012*a*; Luebker *et al*. 2005*b*; Seacat *et al.* 2003; Thibodeaux *et al.* 2003). Luebker *et al*. (2005*b*) determined a LOAEL of 0.4 mg/kg bw/day for decreased total serum cholesterol. The same study identified a transitory increase in serum cholesterol and LDL at a higher dose.

Decreased reproductive organ weights were observed at lower levels in female rats (≥0.166 mg/kg bw/day) (Fair *et al*. 2011; Wetzel 1983) than in male rats (Christian *et al*. 1999; Cui *et al*. 2009).

Other reproductive effects observed in rodents include decreased gestation length (Christian *et al*. 1999; Luebker *et al*. 2005*a*, 2005*b*), fewer implantation sites and reduced lactation in rats (Luebker *et al*. 2005*a*), increased fetal resorptions, dead fetuses and still births in rats, mice and rabbits (Lau *et al*. 2003; Luebker *et al*. 2005*a*; Wetzel 1983; Yahia *et al*. 2008), and reduced litter size in rats (Christian *et al*. 1999; Xia *et al*. 2011) and rabbits (Case *et al*. 2001).

5.5.2. Epidemiologic Studies

Studies have followed large cohorts of workers and environmentally exposed populations. These studies have observed significant relationships between PFOS exposure and lipid levels, liver and thyroid function, and reproductive (fecundity, age of puberty and sperm quality), immunological, and developmental (birth weight) outcomes. Although each of these studies presents some limitations, including study design, bias and confounders, the human weight of evidence strongly supports the argument that PFOS has detrimental health effects. These effects include increased serum lipids (cholesterol, LDL), increased serum levels of hepatic markers (enzymes), altered thyroid hormones (no clear pattern), delayed puberty, modulation of immune response, reduced birth weight, reduced fecundity and neurological effects. Deriving a safe exposure dose based on studies in humans remains a challenge because of the difficulty in characterizing a dose-response pattern with current studies. However, results from these studies inform the relevance of animal to human extrapolation. More details can be found in HC (2018*a*).

Most occupational studies available were conducted in workers from manufacturing plants in the United States (Decatur, AL, and Cottage Grove, MN) and in Belgium (Antwerp). The environmental studies mostly refer to cross-sectional studies conducted within populations exposed through the consumption of contaminated drinking water or within the general population in the United States (based on NHANES) and in European countries.

5.5.2.1. Lipidemia

Significant associations between serum PFOS and increased total cholesterol and/or alteration of other lipid parameters (LDL, HDL, triglycerides) were reported in studies on occupationally exposed workers (Olsen *et al*. 2003*a*; 2012), on exposed community residents (Fitz-Simon *et al*. 2013; Frisbee *et al*. 2010; Kerger *et al*. 2011; Steenland *et al*. 2009), in the general population of the United States (Nelson *et al*. 2010) and in an Inuit population of Nunavik (northern Québec) (Château-Degat *et al*. 2010). However, data for HDL, LDL and triglycerides were not consistent across studies. Increased PFOS exposure also resulted in increased uric acid in two occupational studies and one general population study (as reviewed by Steenland *et al*. 2010). See HC (2018*a*) for more information.

5.5.2.2. Liver

An association between PFOS exposure and alteration in liver enzymes has been observed, but no definitive conclusion on liver toxicity can be drawn, due to study limitations and low magnitude

of enzymatic changes (HC 2018*a*). A cross-sectional study found no association between PFOS serum levels (range = 20–2,110 ng/mL) and hepatic parameters in employees of the 3M Cottage Grove plant (Olsen *et al*. 2003*b*). A small linear association between levels of PFOS and alanine aminotransaminase was reported in participants of the C8 Health Project (Gallo *et al*. 2012), but the clinical significance is unknown. An occupational study comparing hepatic enzymes before and after the demolition of manufacturing facilities found a significant association between PFOS and decreased aminotransaminase among workers with baseline PFOS levels similar to those of the general population. The study found no association between PFOS and total bilirubin, alkaline phosphatase or aspartate transaminase (Olsen *et al*. 2012).

5.5.2.3. Kidney

Shankar *et al*. (2011) reported an increased risk of chronic kidney disease (reduced estimated glomerular filtration rate) in a cross-sectional study of the general US population. Causality is difficult to establish for adverse kidney effects, as altered kidney function could cause an increase in serum PFOS levels (HC 2018*a*).

5.5.2.4. Thyroid System

Inconsistent effects on thyroid hormone levels were observed in PFOS-exposed populations. Studies observed associations between serum PFOS and total thyroxin, free thyroxin, triiodothyronine and thyroid-stimulating hormone levels. However, no clear trend can be established because of various weaknesses in the data: results were equivocal; it was not possible to calculate cumulative exposure; individuals with thyroid diseases were excluded, possibly biasing the results; and temporality cannot be established with the cross-sectional study design (HC 2018*a*). See HC (2018*a*) for more details.

5.5.2.5. Immunological Outcomes

Studies in environmentally exposed populations have identified associations between PFOS levels and decreased antibodies against various illnesses. However, the influence of PFOS exposure on clinical immunosuppression (i.e., incidence of illness) appears to be more tenuous, as conflicting results were common, the data set is small and there is low consistency across studies. Variations between genders and other factors make interpretation more uncertain (HC 2018*a*). See HC (2018*a*) for more information.

5.5.2.6. Reproductive and Developmental Toxicity

Epidemiological studies have observed effects on birth weight, developmental milestones, thyroid hormones, immune system, fecundity and age of puberty, which indicate that humans may be vulnerable to early-life PFOS exposure (Andersen *et al*. 2010; Apelberg *et al*. 2007*b*; Gump *et al*.

2011; Hoffman *et al*. 2010; Maisonet *et al*. 2012; Stein *et al*. 2009; Stein and Savitz 2011; Washino *et al*. 2009).

The main findings suggest a possible link between PFOS exposure and reduced fecundity (*Fei et al.* 2009; Whitworth *et al*. 2012) and delayed puberty (Lopez-Espinosa *et al*. 2011). However, the evidence is not sufficient to establish a causal relationship for reproductive effects. More information on these and other studies are found in HC (2018*a*).

The effects of prenatal and early-life PFOS exposure in humans have been examined with respect to a number of endpoints, including birth weight, developmental milestones, neurological function and immune system. The sometimes conflicting, equivocal results are difficult to interpret with any certainty. However, the results indicate that fetuses, neonates and young children may be potentially vulnerable populations. The most compelling evidence is for an inverse association between early pregnancy exposure and birthweight, as observed in several studies (Apelberg *et al*. 2007*b*; Maisonet *et al*. 2012; Stein *et al*. 2009; Washino *et al*. 2009) but not in others (Fei *et al*. 2007; 2008; Hamm *et al*. 2010; Monroy *et al*. 2008). Other effects were altered thyroid hormones (Kim *et al*. 2011*b*) and neurobehavioural effects (Fei and Olsen 2011; Hoffman *et al*. 2010; Stein and Savitz 2011). More information is available in HC (2018*a*).

5.6. Carcinogenicity and Genotoxicity

One two-year carcinogenicity study in rats (Butenhoff *et al*. 2012*a*; Thomford 2002) indicates that PFOS is tumorigenic in rat liver (hepatocellular and follicular cell adenomas in males and females, and follicular carcinoma in females). However, evidence was not sufficient to draw conclusions about other tumours (thyroid and mammary gland) (EFSA 2008; HC 2006).

Although elevated incidence of some cancers has been observed in a few epidemiological studies (Alexander and Olsen 2007; Bonefeld-Jorgensen *et al*. 2011; Olsen *et al*. 2001), definitive conclusions could not be drawn, due to the low number of cases, equivocal results, confounding factors and participant selection bias. Another prospective cohort study found no increased incidence of several cancers (Eriksen *et al*. 2009).

Considering the negative results of a large series of *in vitro* and *in vivo* short-term tests at gene-, chromosome- or DNA-repair levels, EFSA and Health Canada concluded that PFOS and its salts are not genotoxic (EFSA, 2008; HC 2006). Data published more recently (see the following subsections) agree with this statement. See HC (2018*a*) for more details.

5.7. Mode of Action

Mode of action (MOA) analysis was considered for effects occurring at the lowest PFOS levels (immune effects in mice, lipid effects in monkeys and mice, liver weight increase in rats and mice, liver histological changes in rats, hepatocellular tumours in rats, and thyroid hormone changes in monkeys, rats and mice). A MOA analysis using recent guidance (Meek *et al*. 2014) could be performed only for peroxisome proliferation effects on liver endpoints. Evaluation of all other

endpoints was preliminary. Based on the analysis, no endpoints were considered irrelevant to humans, and the results suggest that the tolerable daily intake (TDI) approach is the most appropriate method for cancer risk assessment. Results of the MOA evaluations are summarized below, with further details in HC (2018*a*).

5.7.1. Direct-acting Mutagenicity

Results from both *in vivo*, *in vitro*, and rat studies do not support the direct-acting mutagenicity MOA. Therefore, low-dose linear extrapolation is not appropriate for PFOS-induced tumours (HC 2018*a*).

5.7.2. Peroxisome Proliferation

As data were insufficient to apply the evolved Bradford Hill criteria to evaluate the MOA, the weight of evidence analysis was limited to the evaluation of the dose-response of key events for peroxisome proliferation in rat liver (HC 2018*a*). Three main key events in the peroxisome proliferation MOA lead to liver histological effects and hepatocellular tumours: 1) the activation of hepatic PPARα receptors, which leads to 2) altered cell growth pathways that inhibit apoptosis and/or promote cell replication, eventually leading to 3) hepatocyte proliferation (Corton *et al.* 2014).

5.7.2.1. Comparison of Dose–response of Key Events and Outcomes

For modes of action to be deemed relevant to adverse outcomes, dose-response concordance—i.e., the observation of early key events at doses that are lower than or equal to later key events and the adverse outcome—is required. Because it appears that liver proliferation, hepatocellular adenomas and cystic degeneration precede PPARα activation, adverse hepatic effects observed in rats exposed for two years to PFOS do not appear to be driven by a peroxisome proliferation MOA. For this reason, human relevance of PFOS-induced hepatic effects cannot be discarded (HC 2018*a*). Moreover, hepatic effects do not appear to be specific to rodents; the LOAEL for hepatocellular hypertrophy accompanied by cytoplasmic vacuolation in monkeys (0.75 mg/kg bw/day; Seacat *et al*. 2002) is on the same order of magnitude as in rats (0.242 mg/kg bw/day; Butenhoff *et al*. 2012*a*).

Although insufficient data exist to examine the impact of PPAR activation on changes in serum lipid, thyroid and immune parameters, peroxisome proliferation is plausible for all endpoints (HC 2018*a*). However, the peroxisome proliferation MOA for these endpoints cannot be fully examined until further data are produced. For more details see HC (2018*a*).

5.7.3. Sex Hormone Disruption

PFOS appeared to have some impact on sex hormone disruption in a variety of *in vitro* assays of estrogenicity, and sex differences were observed in immune response, with males more sensitive than females. However, no studies have been developed to identify whether this effect is associated with sex hormones; therefore, there are insufficient data to evaluate the MOA. See HC (2018*a*) for more details.

5.7.4. Immune Suppression

Immune suppression in rats (decrease in immunoglobin M and natural killer cell levels) has been observed at lower doses than those that were tumorigenic. Although natural killer cells are involved in eliminating cancer cells, no studies have investigated the role of PFOS-induced immunosuppression in tumour development, so a detailed analysis of this MOA cannot be performed.

5.7.5. Other Modes of Action

Insufficient data exist for an assessment of other potential MOAs, particularly in regard to PPAR activation/peroxisome proliferation for other endpoints that were not included in the above MOA analysis.

5.8. Toxicological Limits

Several agencies have derived toxicological limits for PFOS. Health Canada and the Federal-Provincial-Territorial Committee on Drinking Water have developed oral TDI values for carcinogenic and non-carcinogenic effects (HC 2018*a*).

A value of 0.0011 mg/kg bw/day, based on a $BMDL_{10}$ of 0.318 mg/kg bw/day for hepatocellular adenomas in male rats, was derived from Butenhoff *et al*. (2012*a*) data. This value was adjusted to consider the purity of the test material (86.9%) to give 0.276 mg/kg bw/day and the adjusted $BMDL_{10}$, which was divided by an uncertainty factor (UF) of 25 to account for interspecies and intraspecies variability (HC 2018*a*).

A value of 0.00006 mg/kg bw/day, based on a NOAEL of 0.024 mg/kg bw/day for hepatocellular hypertrophy in rats, was derived from Butenhoff *et al*. (2012*a*) data. This value was adjusted using the AK_{UF} dose- and species-specific adjustment factor of 14 for rats (Section 5.2.6) to give a human equivalent dose of 0.021 mg/kg bw/day (HC 2018*a*). A composite UF 25 was applied to account for inter- and intraspecies uncertainty (HC 2018*a*).

A value of 0.0001 mg/kg bw/day, based on a NOAEL of 0.03 mg/kg bw/day for thyroid hormone changes in monkeys, was derived from Seacat *et al*. (2002) data. This value was adjusted using the AK_{UF} dose- and species-specific adjustment factor of 4 for monkeys (Section 5.2.6) to

determine a human equivalent dose (HC 2018*a*). A composite UF 75 was applied to account for inter- and intra-species uncertainty and the shorter exposure relative to life span in monkeys (HC 2018*a*).

The lowest TDI, being protective of the three critical effects, was selected for the determination of SoQGs for PFOS for the protection of human health.

See HC (2018*a*) for more details.

6. DERIVATION OF ENVIRONMENTAL QUALITY GUIDELINES

CSoQGs are derived for the protection of receptors under four different land uses: agricultural, residential/parkland, commercial and industrial. *A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines* (CCME 2006) was used to establish guidelines for agricultural, residential/parkland, commercial and industrial land uses for PFOS presented hereafter.

Given the physical and chemical properties of PFOS, various soil exposure pathways were evaluated for different land uses (Table 8). This chapter describes the derivation of the SoQGs for soil contact ($SoQG_{SC}$), soil and food ingestion ($SoQG_{I}$), protection of freshwater life ($SoQG_{FL}$), protection of livestock watering ($SoQG_{LW}$) and irrigation water ($SoQG_{IR}$), and the off-site migration check ($SoQG_{OM\text{-}E}$). The nutrient and energy cycling check was not derived, because of lack of data.

Table 8 shows a summary of the environmental quality guidelines for PFOS for ecological receptors for agricultural, residential/parkland, commercial and industrial land uses.

Table 8. Required exposure pathways for development of Canadian Soil Quality Guidelines for ecological receptors

Pathway	Agricultural	Residential/ Parkland	Commercial	Industrial
Soil contact ($SoQG_{SC}$)[a]	Required	Required	required	required
Soil ingestion, primary consumers ($SoQG_{1C}$)	required for all substances	required for biomagnifying substances[b]	NR	NR
Soil ingestion: secondary and tertiary consumers ($SoQG_{2C}$, $SoQG_{3C}$)	required for biomagnifying substances[b]	required for biomagnifying substances[b]	NR	NR
Nutrient and energy cycling	check mechanism used if data are sufficient			
Groundwater: freshwater life ($SoQG_{FL}$)	for soluble substances only[c]			
Groundwater: agricultural (irrigation, $SoQG_{IR}$, and livestock watering, $SoQG_{LW}$)	for soluble substances only[c]	NR	NR	NR
Off-site migration ($SoQG_{OM-E}$)	NR	NR	for non-volatile substances only[d]	for non-volatile substances only[d]

Source: CCME (2006).
NR = not required
[a] SC = soil contact, 1C = primary consumer, 2C = secondary consumer, 3C = tertiary consumer; FL = freshwater life, LW = livestock watering, IR = irrigation water, OM-E = off-site migration (environment).
[b] PFOS is considered to biomagnify in aquatic systems (Moermond *et al*. 2010; Swedish KEMI 2004).
[c] While the chemistry of fluorochemicals is complex and PFOS consists of both hydrophilic and hydrophobic components, the solubility of PFOS (as potassium salt) has been reported as 370 mg/L (0.37 g/L) in fresh water (OECD 2002) and is considered soluble.
[d] PFOS is considered non-volatile (Hekster *et al*. 2002).

6.1. Agricultural and Residential/Parkland Land Uses

6.1.1. Soil Quality Guidelines for Soil Contact

The derivation of the SoQGs for soil contact ($SoQG_{SC}$) is based on toxicological data for vascular plants and soil invertebrates. Data were evaluated and placed in two categories: "acceptable" or "selected" for guideline derivation (Appendix E) and "unacceptable" for guideline derivation (or "consulted" but not used for guideline derivation) (Appendix F). Data used to derive the species sensitivity distribution are noted in bold text in Appendix E. Common reasons for classifying a study as "consulted" include test soil properties that may result in excessively high (e.g., pH <4) or low bioavailability (e.g., high organic matter), test media other than soil (e.g., filter paper, sugar solution or agar), lacking study information, and improper or lacking statistics, controls or replication.

Seven acceptable studies reporting 155 acceptable soil ecotoxicity endpoints were identified for terrestrial biota in direct contact with soil. Two studies (Brignole *et al*. 2003, Zhao *et al*. 2011) investigated the toxicity of PFOS covering eight plant species and 115 endpoints. Five studies (EC 2015; Joung *et al*. 2010; Sindermann *et al*. 2002; Stubberud 2006; Xu *et al*. 2011) covered three soil invertebrate species and 40 endpoints. All acceptable endpoints were screened to ensure that only the most appropriate endpoints and derivation methods were retained in guideline derivation. Briefly, the screening criteria were (see also section 7.5.5.1 of CCME 2006):

- If a single study had multiple endpoints, only discrete endpoints were used. For example, if a study reported an EC_{25} and EC_{50} from the same experiment, only one endpoint was used. EC_{25} and/or IC_{25} endpoints were preferred (or EC_X or IC_X where X is close to 25).
- Biologically relevant effects were preferred (e.g., growth, reproduction or survival over physiological or behavioural).
- Studies with longer test durations were preferred.

There were enough toxicity studies and endpoints to meet the minimum data requirements for the preferred CCME weight of evidence method using the EC_{25}/IC_{25} data distribution method (Appendix H, Appendix K), which requires data from a minimum of two crop/plant species and two invertebrate species. The PFOS data set contained a total of 32 acceptable IC_{20}, EC_{25}, and LC_{20} data points (23 data points from eight plant species and nine data points from three invertebrate species). These data points were combined into an estimated species sensitivity distribution (ESSD), where the rank percentile was plotted against soil PFOS concentration on a log scale (Figure 1). The 25th percentile of the ESSD was used as the starting point for the soil contact guideline for agricultural and residential/parkland land uses (CCME 2006). The 25th percentile of the ESSD corresponds to the eighth rank position and is equal to 22.1 mg PFOS/kg soil. Given the roughly even balance of three to four endpoints per test species (i.e., not biased towards one species) an uncertainty factor (UF) of 2 was used.

The threshold effects concentration (TEC) was calculated as follows:

$$TEC = \frac{ESSD_{25}}{UF}$$

where:

- TEC = threshold effects concentration (mg/kg), i.e., guideline value
- $ESSD_{25}$ = estimated species sensitivity distribution, 25th percentile of the distribution (mg/kg) (= 22.1 mg PFOS/kg soil)
- UF = uncertainty factor (= 2)

$$TEC = 22.1 \div 2 \ = 11.05\ mg/kg$$

The soil contact guideline for agricultural and residential/parkland land uses is the TEC (rounded to one significant figure), or 10 mg/kg soil.

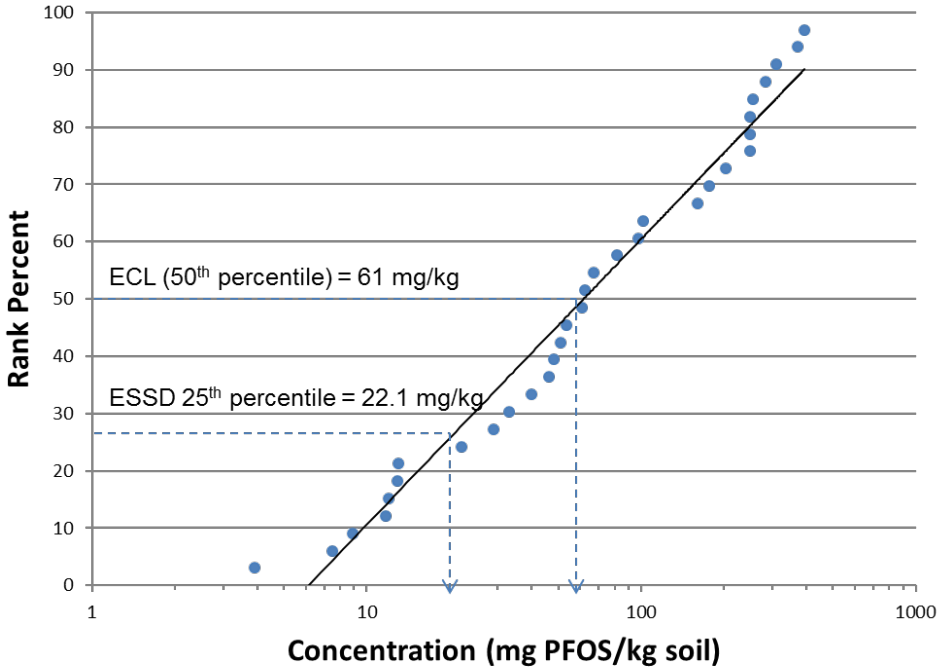


Figure 1. ESSD (rank percent of EC_{25}/IC_{25} data) for PFOS for terrestrial plants and invertebrates showing $ESSD_{25}$ and effects concentration low (ECL) at $ESSD_{50}$ used to derive guidelines to protect ecological receptors (see Appendix E for data)

6.1.2. Soil Quality Guidelines for Ingestion of Soil and Food

PFOS is a bioaccumulative substance, so the SoQG for agricultural and residential/parkland land uses considers the exposure of organisms to PFOS through ingestion of soil and food ($SoQG_I$). This section describes the derivation of SoQGs to protect primary consumers (1C), secondary consumers (2C) and tertiary consumers (3C) from adverse effects due to ingestion of contaminated soil and food. The lowest of $SoQG_{1C}$, $SoQG_{2C}$ and $SoQG_{3C}$ is taken as the $SoQG_I$ of soil and food for agricultural and residential/parkland land uses. $SoQG_I$ is not required for commercial or industrial land uses.

6.1.2.1. Calculating the Daily Threshold Effects Dose

Calculation of the $SoQG_I$ is based on the lowest-observed-adverse-effects dose (ED_{1C}) taken from the mammalian and avian toxicological data in Appendix G. The lowest ED_{1C} for mammals was 0.1086 mg/kg bw/day (from Covance Laboratories Inc 2002) based on anatomic pathology in rat liver after 104-week exposure to PFOS in diet. The ED_{1C} for avian species was 0.772 mg/kg bw/day (from Newsted *et al*. 2007) based on reduced survivability of 14-d old Northern bobwhite quail chicks.

The lowest ED_{1C} is used to calculate the daily threshold effects dose (DTED) according to the equation:

$$DTED_{1C} = \frac{lowest\ ED_{1C}}{UF}$$

where:

- $DTED_{1C}$ = daily threshold effects dose of the primary consumer (mg/kg bw_{1C}/day)
- lowest ED_{1C} = lowest effects dose (mg/kg bw_{1C}/day)
- UF = uncertainty factor (if needed).

The ED_{1C} values from the available mammalian and avian data are considered biologically significant, as both studies were of long duration and provided more than the minimum data requirements. The uncertainties relate primarily to lack of knowledge of inter-species sensitivity, given the paucity of wildlife species in the data set. Therefore, a safety factor of 2 was selected (CCME 2006) for both the mammalian and avian effects dose.

Thus:

$$DTED_{1C\ mammals} = lowest\ ED_{1C} \div UF$$

$$= \frac{0.1086 \frac{mg}{kg\ bw\ d}}{2}$$

$$= 0.0543 \frac{mg}{kg\ bw\ d}$$

and

$$DTED_{1C\ avian} = \frac{0.772 \frac{mg}{kg\ bw\ d}}{2}$$

$$= 0.386 \frac{mg}{kg\ bw\ d}$$

$SoQG_{1C}$, $SoQG_{2C}$ and $SoQG_{3C}$ were derived by scaling the available dietary toxicity data for laboratory mammals and birds to the body weight, food ingestion and soil ingestion rates for mammalian and avian wildlife species. The calculations also account for the portion of diet that is plant, invertebrate or prey on a dry weight basis. Characteristics of receptors used to calculate $SoQG_{1C}$, $SoQG_{2C}$ and $SoQG_{3C}$ are provided in Appendix L. DTEDs calculated for the other receptors and trophic levels, $SoQG_{1C}$, $SoQG_{2C}$ and $SoQG_{3C}$, are summarized in Appendix L.

6.1.2.2. Soil Quality Guideline for Soil and Food Ingestion

An animal may be exposed to a contaminant by more than one exposure route. Total exposure comes from a combination of contaminated food, direct soil ingestion, dermal contact, contaminated drinking water, and inhalation of air and dust. Exposure from all of these routes should not exceed the DTED. Assuming that drinking water, dermal contact and inhalation account for 25% of the total exposure (CCME 2006), then exposure from soil and food ingestion should not exceed 75% of the DTED.

Under this scenario, the primary consumer (1C) may be exposed to a contaminant via soil ingestion and by ingesting contaminants taken up by plants growing in contaminated soil. This pathway was assessed for an herbivorous mammal (meadow vole) and herbivorous bird (rock dove).

a) soil → plant → herbivorous mammal (meadow vole)

b) soil → plant→ herbivorous bird (rock dove)

Based on their ratios of food intake to body weight, the meadow vole was selected to represent herbivorous mammals and the rock dove was selected to represent herbivorous birds (FCSAP 2012). Transfer of contaminant from soil to plant is estimated using bioconcentration factors (BCF).

The secondary (2C) food chain is more complex and involves up to three trophic levels. It can be represented by the following pathways:

a) soil → prey (earthworms)→ predator (secondary consumer; mammal or bird)

b) soil→ plant → prey (primary consumer) → predator (secondary consumer)

The model developed to represent this food chain and to derive the $SoQG_{2C}$ for secondary consumers is similar to the one used in deriving the $SoQG_{1C}$. However, to account for biomagnification from contaminated food and soil to the prey (through the soil → plant → prey or soil → prey pathways), an appropriate bioaccumulation factor (BAF) from soil to primary or secondary prey, or more complex pathways, is used instead of BCF. Biomagnification factors between adjacent trophic levels are not used.

The pathways for tertiary consumers (3C) consider predators consuming prey items which themselves have fed on contaminated plants. The following exposure pathways were considered for tertiary consumers.

a) soil → plant → caribou → carnivorous mammal (wolf)

b) soil → (plant + invertebrates + mammals + birds) → omnivorous mammal (red fox)

Although other omnivorous wildlife could be considered (e.g., bear or lynx), values for food ingestion, diet composition, SIR and so on are available for the carnivorous species wolf and the omnivorous red fox (FCSAP 2012) and therefore these species were considered.

Calculations for deriving the $SoQG_{1C}$ for herbivorous mammal (meadow vole) and herbivorous bird (rock dove) are provided in CCME (2006). Input data for the calculations is provided in Appendix L.

6.1.2.3. Final $SoQG_I$

As described in CCME (2006), the lowest of $SoQG_{1C}$, $SoQG_{2C}$ and $SoQG_{3C}$ is taken as the SoQG for ingestion of soil and food, or $SoQG_I$. In the case of PFOS, $SoQG_{2C}$ was the lowest, and therefore $SoQG_I$ is 0.01 mg/kg dry soil.

6.1.3. Soil Quality Guidelines for the Protection of Livestock Watering and Irrigation Water

Contamination that migrates to groundwater may affect the water quality in dugouts or in water wells used for livestock watering or crop irrigation. These pathways apply only for agricultural land use.

Determining the $SoQG_{LW}$ and $SoQG_{IR}$ involves applying the same groundwater model as for the $SoQG_{FL}$, but transport through the saturated zone is not considered. That is, it assumes that dugouts or wells could be installed within the contaminated area. The guidelines are calculated by setting the allowable receptor groundwater concentration in the model equal to the livestock water (for the $SoQG_{LW}$) and irrigation water (for the $SoQG_{IR}$) from the Canadian Water Quality Guidelines (CWQGs).

Since a CWQG for livestock water is not available, the livestock water threshold (LWT) value can be developed using the following equation:

$$LWT = \frac{DTED \times BW}{WIR}$$

where:

- DTED = 0.0543 mg/kg bw/day as noted previously
- BW = 550 kg for cattle (CCME 2000)
- WIR = livestock water ingestion rate = 100 L/day for cattle (CCME 2000).

Therefore:

$$LWT = 0.299\ \frac{mg}{L}$$

Since the calculated LWT value is lower than the pure phase solubility of PFOS, the $SoQG_{LW}$ calculation is required.

Using the same groundwater model as for the $SoQG_{FL}$ (Section 6.3), but with transport through the saturated zone not considered, the resulting $SoQG_{LW}$ is 7 mg/kg for coarse soil and 5 mg/kg for fine soil.

Since an irrigation water guideline is not available, the $SoQG_{IR}$ calculation is not required (CCME 2006).

6.2. Commercial and Industrial Land Uses

6.2.1. Soil Quality Guidelines for Soil Contact

The derivation of the soil quality guideline for soil contact ($SoQG_{SC}$) is based on toxicological data for vascular plants and invertebrates. The $SoQG_{SC}$ for commercial and industrial land uses was derived using the same data and weight of evidence approach for guideline derivation as described in Section 6.1.

The soil contact guideline for commercial and industrial land uses is the effects concentration low (ECL), which is calculated as follows:

$$ECL = ESSD_{50}$$

where:

- ECL = effects concentration low (mg/kg), i.e., guideline value
- $ESSD_{50}$ = estimated species sensitivity distribution, 50^{th} percentile of the distribution (mg/kg) (= 61 mg PFOS/kg soil).

A total of 32 acceptable EC_{20} and EC_{25} were ranked, and the 50th percentile is used as the basis for the soil contact guideline for commercial and industrial land uses (CCME 2006). The $ESSD_{50}$ corresponds to a rank of 16, which had a value of 61 mg/kg soil (see Figure 1), so the ECL was determined to be 61 mg/kg soil. Therefore, the soil contact guideline for commercial and industrial land uses is 60 mg/kg soil (rounded to one significant figure).

6.2.2. Soil Quality Guidelines for Off-site Migration

Exposure scenarios used to derive SoQGs for commercial and industrial sites only consider on-site exposure. However, transfers of contaminated soil from one property to another are possible by environmental routes such as wind and water erosion (CCME 2006).

The purpose of the environmental soil quality guideline check for off-site migration ($SoQG_{OM-E}$) is to check whether the SoQG for commercial or industrial land use would result in unacceptable

adverse effects (i.e., not to exceed agricultural guideline) to more sensitive adjacent land uses due to contaminant migration from wind or water erosion over a specified time period. The $SoQG_{OM-E}$ check was derived using CCME (2006).

The $SoQG_{OM-E}$ check is 0.1 mg/kg soil. (Note: Value corrected February 2, 2022).

6.3. Soil Quality Guidelines and Groundwater Quality Guidelines for the Protection of Freshwater Aquatic Life ($SoQG_{FL}$, $GWQG_{FL}$)

Contaminants present in soil can migrate to groundwater given certain hydrologic and hydrogeological conditions and the characteristics of the contaminant. CCME (2015) is a companion document to CCME (2006) and provides a method for deriving GWQGs based on various exposure scenarios of human and ecological receptors to contaminated groundwater. For ecological receptors, groundwater guidelines are developed to either maintain specific uses of groundwater (e.g., irrigation or livestock watering where water quality guidelines for these uses exist) or to protect receptors in environments that may come in contact with contaminated groundwater directly or indirectly, due to contaminant migration (e.g., plants and invertebrates living in soil or surface water bodies). The GWQGs are not intended to protect organisms living in aquifers, but rather to protect the uses of groundwater or downgradient receptors.

The general conceptual model in CCME (2006; 2015) describes the fate and transport of a contaminant through soil and groundwater to a discharge point to surface water in four steps that account for:

1. Partitioning of the substance between soil, soil vapour and soil pore water (leachate)
2. Leaching of the contaminant through the unsaturated zone to the groundwater table
3. Mixing and dilution of the leachate into groundwater
4. Saturated-zone transport of the contaminant to a downgradient receptor (i.e., horizontal transport and attenuation of the substance in groundwater from edge of contamination to receptor (the surface water).

Because of the interrelationship between soil and groundwater, and the partitioning of contaminants between the solid, liquid and gas phases, the same conceptual model is used to derive the Canadian groundwater quality guideline for groundwater contact by soil-dependent organisms ($GWQG_{GC}$), the $SoQG_{FL}$, the groundwater quality guideline for the protection of freshwater life ($GWQG_{FL}$) and the soil quality guideline for the protection of potable water ($SoQG_{PW}$).

Not all four of the above steps will apply at all sites. Specifically, unsaturated zone transport (component 2) applies only if the contamination is not in contact with groundwater, and is therefore not applied in generic guideline development. Also, saturated zone transport (component 4) applies only if there is a lateral separation between the remediated site and the groundwater receptor. For generic guidelines, it is assumed that a well or livestock dugout (or potable water well) could be installed at the edge of (or even within) the boundaries of the remediated area.

The $SoQG_{FL}$ is a concentration in soil calculated to protect surface water aquatic life. The $GWQG_{FL}$ is the concentration in groundwater that is protective of surface freshwater aquatic life where there is a minimum 10 m lateral separation between the point of measurement and the surface water body. Both the $SoQG_{FL}$ and $GWQG_{FL}$ guidelines were developed in accordance with the fate and transport model in CCME (2006, 2015). The $SoQG_{FL}$ is independent of land use classifications and may be excluded on a site-specific basis if there are no surface water bodies in the vicinity of the site.

As input value to the fate and transport models in CCME (2015), the surface water quality guideline was set equal to the Federal Water Quality Guideline (FWQG) for protection of aquatic life (6.8 µg/L) (ECCC 2018) (Appendix L). Since PFOS is not biodegradable (EC 2013*b*; OECD 2002), the allowable concentration in groundwater ($GWQG_{FL}$) is also 6.8 µg/L (0.0068 mg/L, rounded to 0.007 mg/L) in both fine and coarse soil. The models and default parameters in CCME (2006), with the exception of a travel time of 100 years, are used to develop the soil concentration ($SoQG_{FL}$) that is expected to prevent PFOS moving through soil and groundwater from exceeding the surface water quality guideline. A travel time of 500 years was used to be consistent with the travel used for the development of the $GWQG_{FL}$. $SoQG_{FL}$ was determined to be 0.1 mg/kg for fine soil and 0.2 mg/kg for coarse soil (Table 9). Inputs for the calculation are shown in Appendix L.

6.4. Groundwater Quality Guidelines for Direct Groundwater Contact and Livestock Watering

The groundwater value to protect soil organisms (such as plants) from adverse effects via direct contact with groundwater ($GWQG_{GC}$) is calculated for both fine and coarse soil according to CCME (2015).

The $GWQG_{GC}$ is 1 mg/L for both coarse and fine soils.

The groundwater value to protect livestock from adverse effects via livestock watering (GWQGLW) is numerically equal to the Canadian Water Quality Guidelines for the Protection of Agricultural Water Uses (Irrigation and Livestock Water) (CCME 1999). No CWQGLW value exists, however in the interim, a LWT value can be calculated as 0.3 mg/L, as noted in Section 6.1.3. Since no minimum groundwater attenuation can be assumed before the receptor (i.e., livestock) may be exposed to the groundwater, the GWQGLW is numerically equivalent to the LWT of 0.3 mg/L.

6.5 Groundwater Quality Guideline Management Considerations

The $GWQG_{E}$ is set at the lowest guideline calculated for the various ecological receptors, which for PFOS is the $GWQG_{FL}$ of 0.007 mg/L.

The candidate final groundwater guideline for ecological receptors ($GWQG_{E}$) is checked against various management considerations. For PFOS these are:

1. The candidate final guideline should not exceed 50% of the chemical's aqueous solubility, due to the potential for chemical concentrations approaching maximum solubility to result in non-aqueous phase liquids, which may act as an ongoing contaminant source. In the case of PFOS, $GWQG_E$ of 0.007 mg/L (or 7 µg/L) is well below the aqueous solubility of PFOS (370 mg/L), and therefore the formation of non-aqueous phase liquids at the guideline level is highly unlikely.
2. The candidate final guideline should be reasonable, workable and usable, and therefore checked against the practical quantitation limit of the available analytical methods achievable in Canada. The $GWQG_E$ is above the maximum laboratory reporting limit for PFOS in water of 0.02 µg/L recommended in CCME (2016) and is therefore reasonable, workable and useable.

The candidate final guideline should not be below naturally occurring background levels of the substance. Since PFOS is not a naturally occurring substance, background levels of the substance in the environment should be essentially zero. The candidate final guideline for ecological receptors is above this level.

Based on CCME (2006), SoQGs and GWQGs for ecological receptors were calculated. The final SoQGs and GWQGs are the lowest of the values calculated for all applicable exposure pathways for PFOS for each land use (land use is applicable for SoQGs only). A summary of the calculated environmental SoQGs and GWQGs is provided in Table 9. While CCME recognizes that a large number of perfluorinated compounds can co-exist at a site, environmental guidelines are only provided for PFOS at this time. Guidelines for other perfluorinated compounds are outside the scope of this document.

Table 9. Summary of Canadian Environmental SoQGs and GWQGs for PFOS for ecological receptors

Pathway	Agricultural	Residential/ Parkland	Commercial	Industrial
Soil				
Final $SoQG_E$	0.01 mg/kg	0.01 mg/kg	0.2 mg/kg (coarse soil)[a] 0.1 mg/kg (fine soil)[b]	0.2 mg/kg (coarse soil) 0.1 mg/kg (fine soil)
Soil contact ($SoQG_{SC}$)	10 mg/kg	10 mg/kg	60 mg/kg	60 mg/kg
Soil ingestion ($SoQG_{1C}$)	2 mg/kg	2 mg/kg	NR	NR
Soil ingestion ($SoQG_{2C}$)	0.01 mg/kg	0.01 mg/kg	NR	NR
Soil ingestion ($SoQG_{3C}$)	0.6 mg/kg	0.6 mg/kg	NR	NR
SoQG to protect groundwater used for agriculture (irrigation: $SoQG_{IR}$, and livestock watering: $SoQG_{LW}$)	7 mg/kg (coarse soil) 5 mg/kg (fine soil)	NR	NR	NR
SoQG to protect freshwater life ($SoQG_{FL}$)[c]	0.2 mg/kg (coarse soil) 0.1 mg/kg (fine soil)			

Pathway	Agricultural	Residential/ Parkland	Commercial	Industrial
Nutrient and energy cycling check	NC	NC	NC	NC
Off-site migration check ($SoQG_{OM-E}$)[d]	NR	NR	0.1 mg/kg*	0.1 mg/kg*
Groundwater				
Final $GWQG_E$ [e]	0.007 mg/L			
Groundwater contact ($GWQG_{GC}$) by soil-dependent organisms	1 mg/L			
Protection of freshwater life ($GWQG_{FL}$)[f]	0.007 mg/L			
Protection of marine life ($GWQG_{ML}$)	NC			
Protection of livestock watering ($GWQG_{LW}$)	0.3 mg/L	NR	NR	NR
Protection of irrigation water ($GWQG_{IR}$)	NC	NR	NR	NR
Management considerations ($GWQG_M$) – solubility	200 mg/L			

NC = not calculated due to lack of data
NR = not required
1C = primary consumer, 2C = secondary consumer, 3C = tertiary consumer
FL = freshwater life
LW = livestock watering
OM-E = off-site migration – environmental

[a] Coarse-grained soil is soil in which more than 50% of particles (by mass) are larger than 75 μm mean diameter (D_{50} > 75 μm).

[b] Fine-grained soil is soil in which more than 50% of particles (by mass) are smaller than 75 μm mean diameter (D_{50} < 75 μm).

[c] $SoQG_{FL}$ is the concentration in *soil* that is expected to protect against potential impacts on aquatic systems from PFOS originating in soil that may enter the groundwater and subsequently discharge to a surface water body. This pathway may be applicable under any land use category where a surface water body sustaining aquatic life is present (i.e., within 10 km of the site). Where the distance to the nearest surface water body is greater than 10 km, application of the pathway should be evaluated on a case-by-case basis by considering the site-specific conditions. Also note, if surface water bodies are located closer to the remediated soils than 10 metres, then this generic guideline may not be appropriate and a site-specific evaluation may be necessary on a case-by-base basis since the saturated zone transport model is not considered to be appropriate for use at distances less than 10 metres.

[d] For commercial and industrial sites, receptors exposed to on-site soil are considered. However, contaminated soil can move from one site to another via wind and water erosion. The $SoQG_{OM-E}$ addresses the movement of soil from a commercial or industrial site to adjacent more sensitive land (e.g., agricultural property). Given the uncertainties surrounding the model used to generate the $SoQG_{OM-E}$, it is considered a check mechanism, and professional judgment should be used to determine whether this pathway should modify the SoQG (see CCME 2006).

[e] $GWQG_E$ is the lowest of the pathway-specific guidelines for ecological receptors and considers other management factors such as substance solubility, analytical detection limits and background concentrations

[f] $GWQG_{FL}$ is the concentration in *groundwater* that is expected to protect against potential impacts on freshwater life from PFOS originating in soil that may enter groundwater and subsequently discharge to a surface water body. This pathway may be applicable under any land use category where a surface water body sustaining aquatic life is present (i.e., within 10 km of the site). Where the distance to the nearest surface water body is greater than 10 km, application of the pathway should be evaluated on a case-by-case basis by considering the site-specific conditions.

* Value corrected February 2, 2022.

7. DERIVATION OF HUMAN HEALTH SOIL QUALITY GUIDELINES

CCME (2006) was used to establish guidelines for agricultural, residential/parkland, commercial and industrial land uses for PFOS presented hereafter.

7.1. Protocol

As indicated in Section 5.6, PFOS is considered non-genotoxic. For substances of this type, it is believed that there is a threshold for the critical effect (i.e., below a certain point of exposure, no adverse effects are anticipated). For threshold substances, two key factors are considered in deriving SoQGs for the protection of human health ($SoQG_{HH}$).

First, it is recognized that, exclusive of hazardous waste sites and any other point source of pollution, everyone is exposed to a "background" level of substances that cannot be avoided. For PFOS, this background exposure arises primarily from food. In setting soil guidelines, the background estimated daily intake (EDI) is subtracted from the tolerable daily intake (TDI) as part of the guideline derivation process.

Second, a multimedia approach to the development of guidelines has evolved whereby guidelines for one medium are established, recognizing that guidelines for other media may also be required. Guidelines must be established in a manner where total simultaneous exposure at the guideline levels for all media will not result in exposure exceeding the TDI. Thus, in order to set soil guidelines for threshold contaminants, a portion of the residual tolerable daily intake (RTDI, i.e., the TDI minus the EDI) must be attributed to each of the five primary media to which people are exposed (i.e., air, water, soil, food and consumer products). Therefore, 20% of the RTDI intake for threshold substances is apportioned to each of these media.

In cases for which the mechanism of toxicity varies by exposure route, it is possible to calculate SoQGs using TDIs for each exposure route (i.e., soil ingestion only, dermal contact only or particulate inhalation only). The final direct contact SoQG is then the lowest of the calculated values for each direct exposure pathway.

In order to calculate a quantitative guideline, it is necessary to define one or more scenarios by which exposure will occur. As over 80% of Canadians live in cities (Statistics Canada 2005), an urban exposure scenario is the most common situation expected.

7.2. Estimated Daily Intakes

The EDI is an estimate of the typical total concurrent background multimedia exposure from all known or suspected sources for the average Canadian (Section 2.4). It does not include exposures that may occur from a contaminated site or activities that may result in increased exposure to substances that are not considered background exposure (e.g., hobbies). The principal daily sources of PFOS for the general population are through food ingestion and the associated use of

coated consumer products such as pans and grease-resistant food packaging, followed by transfer from stain-treated household items.

For the purpose of SoQG derivation, EDIs are developed for the following age classes: infants (birth to six months), toddlers (seven months to four years), children (five to 11 years), teenagers (12 to 19 years) and adults (20 years and older) using the equations found in CCME (2006). Appendix I presents the exposure parameters.

The media considered for calculating the EDI were ambient air, indoor air, indoor settled dust, soil, drinking water, human breast milk and food. Data pertaining to consumer products is limited and is thus not typically included in the EDI estimate. However, the potential migration of PFOA from consumer products into food or into the indoor environment (air and dust) is indirectly accounted for in the derivation. This migration was not taken into consideration for other routes of exposure such as dermal contact with impregnated textiles. This could lead to underestimation of the EDIs.

The general equation for the derivation of EDIs is as follows:

$$EDI = \sum_{i=1}^{n} ED_i$$

and

$$ED_i = \frac{C \times CR \times BF \times EF}{BW}$$

where:

- EDI = estimated daily intake (ng/kg bw/day)
- ED_i = exposure dose from pathway i (ng/kg bw/day)
- C = contaminant concentration in medium (e.g., ng/L)
- CR = media-specific contact rate (e.g., L/day)
- BF = bioavailability factor (1 by default, unitless)
- EF = exposure factor, which is the product of the exposure frequency (events/year) and exposure duration (years/lifetime) (unitless)
- BW = body weight (kg).

The estimated total average EDIs for PFOS for the general Canadian population, measured in ng/kg bw/day, are 1.7 for infants, 3.8 for toddlers, 3.8 for children, 2.8 for teens, and 2.3 for adults. Details are available in Appendix J.

7.3. Exposure Limits for Human Receptors

As presented in Section 5.8, an oral TDI of 6×10^{-5} mg/kg bw/day (60 ng/kg bw/day) was adopted for PFOS by Health Canada and the Federal-Provincial-Territorial Committee on Drinking Water

(HC 2018*a*). As no toxicity reference values were identified for dermal and inhalation exposure, the oral TDI was applied to these routes.

7.4. Relative Absorption Factors

Relative absorption factors may be applied when the critical toxicological study has used a different medium than that under investigation, in order to account for the difference in absorption of the substance via different exposure routes or in different media.

The TDI for PFOS was based on a diet study (Butenhoff *et al*. 2012*a*). The available data regarding oral absorption indicate PFOS is readily absorbed from the gastrointestinal tract (see Section 5.2.1). The oral bioavailability of PFOS in soil may be lower due to various factors (e.g., sorption onto soil particles, prevailing gastrointestinal conditions). However, the information available to date is insufficient to determine the bioavailability of PFOS in soils. As a result, a relative absorption factor of 100% was selected for exposure via ingestion. Similarly, due to the lack of available information, a conservative factor of 100% was selected by default for the relative bioavailability via inhalation.

Few data are available regarding PFOS dermal absorption. However, available data for PFOA (another perfluorinated compound presenting similar physicochemical properties as PFOS) indicate limited dermal absorption. On this basis and because the two compounds share similar physicochemical properties, the absorption factor (10%) identified for PFOA was applied for PFOS.

7.5. Ingestion, Inhalation and Dermal Pathways

Direct exposure pathways are the most likely routes of exposure to non-volatile contaminants in soil. No SoQG for indoor air quality ($SoQG_{IAQ}$) was derived, as PFOS is not volatile.

Appendix L contains the input parameters used to derive the $SoQG_{HH}$.

7.5.1. Agricultural and Residential/Parkland Land Uses

Agricultural lands are characterized by the presence of a farm with a residence where humans may be on-site 24 hours a day. The most sensitive receptor for this land use is the toddler, as this age category has the largest exposure-to-body weight ratio.

Using the above assumptions and the input parameters presented in Appendix L, a soil quality guideline for direct exposure to soils that applies to agricultural and residential/parkland land uses may be determined by using the equation below (from CCME 2006):

$$SQG_{DH} = \frac{(TDI - EDI) \times SF \times BW}{[(AF_G \times SIR) + (AF_S \times SR) + (AF_L \times IR_S)ET_2] \times ET_1} + BSC$$

The resulting soil quality guideline for direct human health ($SoQG_{DH}$) for agricultural and residential/parkland land uses is 2 mg/kg.

7.5.2. Commercial Land Use

Commercial sites include places with unrestricted access, such as shopping malls that may include daycare facilities, but do not include any manufacturing or residential areas. Access to commercial sites is assumed to be <24 hours (10 hours/day; 5 days/week; 48 weeks/year). Discretion should be used in employing the commercial land use classification; in scenarios where 24-hour access by children or toddlers or residential occupancy by any individual is possible, the residential/parkland classification may be more appropriate. Toddlers are assumed to be the most sensitive receptors at commercial sites.

Using the above assumptions and the input parameters presented in Appendix L, the PFOS $SoQG_{DH}$ for commercial land use is 3 mg/kg.

7.5.3. Industrial Land Use

Typically, industrial lands have limited or restricted access to the public so that adult, occupational exposure will predominate. An example of industrial land use is a manufacturing plant. The most common exposure scenario is expected to be unintentional soil ingestion by an adult. The potential for off-site migration of substances (i.e., via soils and dust) is evaluated for industrial land use scenarios (Section 7.8).

Using the above assumptions and the input parameters presented above in Appendix L, the PFOS $SoQG_{DH}$ for industrial land use is 40 mg/kg.

7.6. Protection of Groundwater Used as a Source of Raw Drinking Water

For PFOS, sorption is the only chemical-specific attenuation mechanism in soil and groundwater, since PFOS does not volatilize or biodegrade (EC 2013*b*; OECD 2002). Other attenuation mechanisms are based on purely hydrogeological and hydrological conditions. On this basis, K_{oc} is a key parameter to derive an SoQG for potable water ($SoQG_{PW}$) for PFOS. Franz Environmental Inc. (2012) identified a median K_{oc} for PFOS for pH between 5 and 7, of 1445 L/kg.

This K_{oc} was used to derive a $SoQG_{PW}$ of 0.01 mg/kg for both coarse and fine soils, using the same input parameters as those used to derive $SoQG_{FL}$ (described in Section 6.3). However, the level of protection afforded by this $SoQG_{PW}$ may not be appropriate for all sites, because PFOS sorption

is highly variable, with reported K_{oc} values varying from 229 to 6,310 L/kg (Franz Environmental Inc. 2012).

PFOS concentrations in groundwater that could be used as potable water can be compared directly to the $GWQG_{PW}$. To protect human health, the allowable concentration in potable water is the $GWQG_{PW}$ (0.0006 mg/L), as described below.

According to CCME (2015), $GWQG_{PW}$ (Table 2) is adopted directly from the Guidelines for Canadian Drinking Water Quality, developed by Health Canada. Therefore, the $GWQG_{PW}$ is equivalent to the Maximum Acceptable Concentration (MAC) of 0.0006 mg/L developed by HC (2018*a*). CCME (2015) recommends that this value be used to directly screen samples from groundwater that may be used as a drinking water source. Where groundwater is used for other purposes (e.g., irrigation of produce), this should be evaluated on a site-specific basis.

7.7. Guideline for Consumption of Produce, Meat and Milk

Exposure through local produce, meat and dairy is possible. A literature study conducted in 2018 found that accumulation patterns of PFAS differ between fish, mammals, and plants and many factors can influence both uptake and bioaccumulation (Intrinsik 2018). Protein-rich foods (fish, meats, dairy, etc.) were found to be the primary dietary source of human exposure to PFOS (Intrinsik 2018; EFSA 2012).

There is limited available transfer factor information for fish, shellfish and mammals due to variability and uncertainty inherent in the data, which is attributable to several different factors, including kinetics, ecology, region, presence of precursors, tissues, and species differences. Based on the 2018 literature review, the available information does not support the derivation of generic transfer factors for animal-based foods for use in the derivation of SoQGs to protect human health (Intrinsik 2018). PFOS has also been widely detected in plant-based foods. Intrinsik (2018) found very limited data for plant-based food. The food concentration data evaluated suggest that fruits, vegetables and cereals contribute less to human exposure than protein-rich foods (Intrinsik 2018).

Should consumption of produce, meat and milk be relevant at a site, site-specific conditions and parameters would need to be considered to develop a site-specific guideline, as outlined in CCME (2006). Transfer factors should be site-specific and specific to the tissues relevant to consumption (e.g., root, shoot, leaves, fruit, organ meat, muscle, skin, etc.). Consideration should also be made for potential differences between exposure concentrations, plant species, and adjustments for soil organic carbon and other soil properties, such as pH and redox potential.

7.8. Guideline for Off-site Migration ($SoQG_{OM-HH}$) for Commercial and Industrial Land Uses

As described in Section 6.4, an off-site migration check is necessary to protect sensitive land uses. If the guidelines for commercial or industrial sites are found to be above the human health soil quality guideline for off-site migration ($SoQG_{OM-HH}$), then the adjacent property could potentially

become contaminated from off-site deposition (CCME 2006). The $SoQG_{OM-HH}$ calculated for commercial and for industrial land uses is 0.1 mg/kg (rounded to one significant figure).

7.9. Final Human Health Soil and Groundwater Quality Guidelines

Based on CCME (2006), three types of exposure pathways are evaluated: required pathways (direct contact), applicable pathways (indoor air, groundwater, and produce, meat and milk ingestion) and check mechanisms (off-site migration of chemicals). Table 10 lists the SoQGs for each of the evaluated pathways for human health receptors. The $GWQG_{PW}$ for PFOS was derived according to CCME (2015).

PFOS is one substance of a suite of PFAS. Currently, guidance concerning PFOS and PFOA is available for different media and from several jurisdictions (ITRC 2018*b*). The health effects of PFOS and PFOA are similar and well documented (Section 5). Based on science current to 2016, PFOS and PFOA impact the liver in similar ways, so additivity of PFOA and PFOS needs to be considered at contaminated sites (HC 2019*a*, 2019*b*). Thus, when PFOS and PFOA are found together in soil or groundwater, to protect human health, CCME recommends that both chemicals be considered together. This is done by adding the ratio of the measured concentration for PFOS to its relevant guideline ($SoQG_{HH}$ or $GWQG_{PW}$) with the ratio of the measured concentration for PFOA to its relevant guideline[3]; if the result is less than or equal to one (≤1.0), then the soil or groundwater is considered acceptable for its expected use. Current science does not justify the use of this approach for other PFAS.

Recommended additivity approach:

$$\frac{[PFOS]}{SoQG_{HH-PFOS}\ or\ GWQG_{PW-PFOS}} + \frac{[PFOA]}{SoQG_{HH-PFOA}\ or\ GWQG_{PW-PFOA}} \leq 1$$

where:

- [PFOS] and [PFOA] are the measured soil or groundwater concentrations
- $SoQG_{HH\text{-}PFOS}$ and $SoQG_{HH\text{-}PFOA}$ are the SoQGs for the protection of human health, for PFOS and PFOA respectively
- $GWQG_{PW\text{-}PFOS}$ and $GWQG_{PW\text{-}PFOA}$ are the GWQGs for the protection of human health, for PFOS and PFOA respectively.

[3] At the time of publication, $SoQG_{HH}$ and $GWQG_{PW}$ have not been produced for PFOA. Consult the local jurisdiction to determine whether other reference values can be used in the additivity equation, for example the MAC for PFOA (HC 2018*b*) or soil screening value for PFOA (HC 2019*a*, 2019*b*).

Table 10. Summary of Canadian Environmental SoQGs for PFOS (mg/kg dw) for human receptors

	Land use			
	Agricultural	**Residential/ Parkland**	**Commercial**	**Industrial**
Guideline	0.01	0.01	0.01	0.01
Human health guidelines/check values				
$SoQG_{HH}$[a]	0.01	0.01	0.01	0.01
Direct contact guideline $SoQG_{DH}$[b]	2	2	3	40
Inhalation of indoor air guideline $SoQG_{IAQ}$[c]	NC	NC	NC	NC
Soil quality guideline for the protection of potable groundwater ($SoQG_{PW}$)[d]	0.01	0.01	0.01	0.01
Check Mechanisms				
Produce, meat and milk check $SoQG_{FI}$	NC	NC	—	—
Off-site migration check $SoQG_{OM-HH}$	—	—	0.1	0.1

NC = not calculated; $SoQG_{HH}$ = SoQG

[a] The $SoQG_{HH}$ is the lowest of the human health guidelines and check values.

[b] The $SoQG_{DH}$ is based on direct exposure to soil via ingestion, dermal contact and particulate inhalation.

[c] The inhalation of indoor air guideline applies to volatile organic compounds. PFOS is essentially non-volatile.

[d] For pH between 5 and 7. Based on a K_{oc} of 1445 L/kg; PFOS K_{oc} is highly variably (229 to 6,310 L/kg; Franz Environmental Inc. 2014), therefore the level of protection afforded by this $SoQG_{PW}$ may not be appropriate for all sites. Where groundwater is used as a potable water source, groundwater concentrations should be compared directly to the $GWQG_{PW}$ value.

8. RECOMMENDED CANADIAN SOIL QUALITY AND CANADIAN GROUNDWATER QUALITY GUIDELINES

According to CCME (2006), both environmental and human health SoQGs are developed for four land uses: agricultural, residential/parkland, commercial and industrial. The lowest value generated by the two approaches for each of the four land uses are recommended as the CSoQGs and are presented in Table 1.
The recommended CGWQGs are presented in Table 2.

The human health effects of PFOS and PFOA are similar and well documented. Therefore, additivity of PFOA and PFOS needs to be considered at contaminated sites. When PFOS and PFOA are found together in soil, the best approach to protect human health is to consider both chemicals together when comparing to the screening values. This can be done using the approach described in Section 7.9.

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APPENDIX A. SUMMARY OF PFOS CONCENTRATIONS IN ENVIRONMENTAL MEDIA, FOOD AND HUMAN FLUIDS/TISSUES

Ambient/outdoor air (pg/m^3)

Location	n	Range	Mean	Median	Geometric mean	Year	Analytical method	Comments	Source
Vancouver, BC	6	<0.02	<0.02	<0.02	<0.02	2007–2008	HPLC-MS/MS	DL=0.40 ng/g	Shoeib *et al*. (2011)
Cornwallis Island, NU	10		5.90			2004	LC-MS/MS	sample DL=0.88 pg/m^3; particulate phase	Stock *et al*. (2007)
Toronto, semi-urban	14	0.33-2.62	1.06(0.63)		0.89	2010	LC-ESI-MS/MS; LOD=0.001	Annular diffusion denuder sampler – gas phase; % recovery=70	Ahrens *et al*. (2012)
	14	n.d.-2.29	0.5(0.64)		0.37		LC-ESI-MS/MS; LOD=3.232	Annular diffusion denuder sampler – particle phase; % recovery=69	
	14	n.d.-0.62	0.23(0.18)		0.21		LC-ESI-MS/MS; LOD=1.092	High volume air sampler- gas phase; % recovery=53	
	14	n.d.-4.08	1.82(1.3)		1.85		LC-ESI-MS/MS; LOD=1.866	High volume air sampler- gas phase; % recovery=69	
Toronto, ON	?				1.5		High volume		Gewurtz *et al*. (2013)
Toronto, ON					8		Passive		
Saskatchewan, agricultural					5		Passive		
Whistler, BC					4		Passive		
Alert, NU					2		Passive		
Lake Erie & Lake Ontario, ON	8 (4<DL)	ND <DL–8.10	6.4 ± 3.3			Aug 2003 Aug 2003	LC/MS	Gas phase Particulate phase	Boulanger *et al*. (2005*a*)
Lake Superior, ON					0.43		High volume sampler		Gewurtz *et al*. (2013)
Albany, NY	8	0.94–3.0	1.70			May & Jul 2006	SPE; HPLC-MS/MS	Gas phase; LOQ=0.07 pg/m^3 Particulate phase	Kim & Kannan (2007)

Location	n	Range	Mean	Median	Geometric mean	Year	Analytical method	Comments	Source
	8	0.35–1.16	0.64			May & Jul 2006			
Hilo, HI					6.6		Passive		Gewurtz *et al.* (2013)
Sydney, FL					3.4		Passive		
Tudor Hill, Bermuda					6.1		Passive sampler		Gewurtz *et al.* (2013)
Marine air,northwest Europe	3	0.40–2.50	1.36			Oct 2005			Jahnke *et al*. (2007)
Paris, France					150		Passive sampler		Gewurtz *et al*. (2013)
Kjeller, Norway	2		1			Nov–Dec 2005	LC-TOF-MS	DL not reported; Particulate phase	Barber *et al.* (2007)
Hazelrigg, United Kingdom	10		1.60			Nov 2005–Feb 2006	LC-TOF-MS	DL not reported; Particulate phase	Barber *et al.* (2007)
Manchester, United Kingdom	2		46.00			Feb–Mar 2005			
Manchester, United Kingdom	1		7.10			Nov–Dec 2005			
Mace Head, Ireland	4		<1.8			Mar 2006			
Malin Head, Ireland					3.3		Passive sampler		Gewurtz *et al.* (2013)
Marine air, east coast of Africa	5	0.05–1.90	0.54			Oct–Nov 2005			Jahnke *et al.* (2007)
Morioka	8	0.46–1.19	0.70			July 2003	HPLC-LC/MS	LOD=0.091 ng/g; LOQ=0.302 ng/g	Harada *et al*. (2005)
Oyamazaki	12	2.51–9.80			5.2 ± 1.43	Apr 2001–Mar 2002			
Oyamazaki	12	2.32–21.80			5.31 ± 1.20	Apr 2001–Mar 2002	HPLC-LC/MS	LOD=0.091 ng/g; LOQ=0.302 ng/g	Sasaki *et al.* (2003)
Fukuchiyama	12	0.00–2.12			0.61 ± 1.30	Apr 2001–Mar 2002			

Note: Due to differences in analytical methods used in the various studies, reported concentrations should not be compared directly.

ND = not detected
DL = detection limit
LC = liquid chromatography
LOD = limit of detection
LOQ = limit of quantification
HPLC = high-performance liquid chromatography
MS = mass spectrometry
SPE = solid-phase extraction
TOF = time of flight

Indoor air (pg/m^3)

Location	Type of sample	n	Range	Mean	Median	Geometric mean	Year sampled	Analytical method	Comments	Source
Canada Vancouver, BC	Indoor air	39	<0.02	<0.02	<0 .02	<0.02	2007–2008	HPLC-MS/MS	DL=0.40 ng/g	Shoeib *et al.* (2011)
Norway Tromso	Indoor air (particulate phase)	4		<47.4			May–Jun 2005	LC-TOF-MS	DL not reported	Barber *et al.* (2007)

Note: Due to differences in analytical methods used in the various studies, reported concentrations should not be compared directly.
DL = detection limit
LC = liquid chromatography
HPLC = high-performance liquid chromatography
MS = mass spectrometry
TOF = time of flight

Dust (ng/g)

Location	Type of sample	n	Range	Mean	Median	Geometric mean	Year sampled	Analytical method	Comments	Source
Canada										
Ottawa, ON	Indoor dust	67	2.28*–5,065	443.68*	37.80*		2002–2003	LC-MS/MS	MDL=4.56 ng/g; 33% <MDL; (*ND replaced by ½ MDL)	Kubwabo *et al.* (2005)
Toronto, ON	Indoor dust	19	42–1,300	290	140		2007–2009	triple quadrupole MS, ES negative ionization MS/MS	not reported	Goosey & Harrad (2011)
Vancouver, BC	Indoor dust	132	1.50–4,661	280	71	73	2007–2008	HPLC-MS/MS	DL=0.40 ng/g	Shoeib *et al.* (2011)
United States										
North Carolina & Ohio	Indoor dust	112	<8.9–12,100	761	201		2000–2001	LC-MS/MS	LOQ=8.93; 94.6 % >LOQ; mean & median <LOQ replaced by LOQ/√2	Strynar & Lindstrom (2008)

Location	Type of sample	n	Range	Mean	Median	Geometric mean	Year sampled	Analytical method	Comments	Source
Wisconsin	Indoor dust	39	8.7–1,100	168	47		2008	HPLC-MS/MS	residential vacuum cleaner dust	Knobeloch *et al*. 2012
Boston, MA	Indoor dust: offices	31	6.80-98.2			14.6	2009	UPLC/MS/MS	LOQ=7 ng/g; GM & SD <LOQ replaced by LOQ/√2	Fraser *et al*. (2012)
	Indoor dust: houses	30	14.1-280			26.9			LOQ=7 ng/g; GM & SD <LOQ replaced by LOQ/√2	
	Indoor dust: cars	13	10.1-280			15.8			LOQ=7 ng/g; GM & SD; <LOQ replaced by LOQ/√2	
Sweden										
Stockholm	Indoor dust: apartments	38	8–1,100		85		2006–2007	HPLC-MS/MS	LOQ=12 ng/g	Björklund *et al.* (2009)
	Indoor dust: cars	5	8–33		12					
	Indoor dust: daycares	10	23–65		31					
	Indoor dust: houses	10	15–120		39					
	Indoor dust: offices	10	29–490		110					
Japan										
Japan	Indoor dust	16	11–2,500	200	24.50		–	LC-ESI-MS/MS	vacuum cleaner dust	Moriwaki *et al*. (2003)
Fukuchiyama	Outdoor dust	12	37.99–427.41			97.4 ± 1.20	Apr 2001–Mar 2002	HPCL-LC/MS	LOD=0.091 ng/g; LOQ=0.302 ng/g	Sasaki *et al.* (2003)
Oyamazaki		12	37.99–427.41			97.4 ± 1.20				
Oyamazaki	Outdoor dust	11	19.7–168.0			72.2 ± 1.77	Apr 2001–Mar 2002	HPCL-LC/MS	LOD=0.091 ng/g; LOQ=0.302 ng/g	Harada *et al*. (2005)

Location	Type of sample	n	Range	Mean	Median	Geometric mean	Year sampled	Analytical method	Comments	Source
Worldwide Europe, Australia, United States	Indoor dust	39	<2.6–18,071		479.60		2004	on-line SPE-HPLC-MS/MS	LOQ=2 .6 ng/g	Kato *et al*. (2009)

Note: Due to differences in analytical methods used in the various studies, reported concentrations should not be compared directly.
DL = detection limit
ES = electrospray
ESI = electrospray ionization
HPLC = high-performance liquid chromatography
LC = liquid chromatography
LOD = limit of detection
LOQ = limit of quantification
MDL = method detection limit
MS = mass spectrometry
ND = not detected
SPE = solid-phase extraction
UPLC = ultra-performance liquid chromatography

Surface water (ng/L)

Location	n	Range	Mean	Median	Year sampled	Analytical method	Comments	Source
Canada								
Canada-wide (38 rivers & tributaries)	65	0.010–34.64	2.15	0.91	2001–2008	GC/MS	38 rivers & tributaries; MDL=0.004 ng/L	Scott *et al.* (2009)
Canada: eastern	27		4.09		2001–2006		Eastern Canada: Atlantic Provinces to MB/ON border	
Canada: western	38		0.91		2005–2008		Western Canada: MB/ON border to BC	
Prince Edward Island	3	0.634–1.26			2006			
Québec	6	0.356–7.819			2006			
Ontario	18	<0.004–34.60			2001–2005			
Manitoba	10	0.335–1.60			2005–2008			
Saskatchewan	8	0.086–5.08			2005–2007			
Alberta	7	<0.004–2.97			2005–2007			
British Columbia	13	<0.004–1.51			2005			
British Columbia (marine water)		0.2–5.8			2009–2011	LC-MS/MS	LOQ=0.5 ng/L. Detected in Puget Sound, WA, & Clayoquot & Barkley Sounds, BC; not detected in Strait of Juan de Fuca & Tofino, BC.	Dinglasan-Panlilio *et al*. (2014)

Location	n	Range	Mean	Median	Year sampled	Analytical method	Comments	Source
Cornwallis Island, NU	24	0.9–90			Aug 2003–Aug 2005	LC-MS/MS	LOQ=0.5 ng/L	Stock *et al.* (2007)
Amituk Lake, NU	3	0.9–1.50	1.2		Aug 2003			
Char Lake, NU	3	1.1–2.30	1.8		Jul 2005			
	3	0.9–2.50	1.8		Aug 2003			
Meretta Lake, NU	3	55–57	56		Jul 2005			
Resolute Lake, NU	3	49–90	69		Aug 2003			
	3	23–24	23		Jul 2005			
	3	41–43	42		Aug 2005			
	3	44–46	46		Aug 2005			
Hamilton, ON, near/downstream of airport	11	38.4–392			Oct 2010	LC-MS/MS	impacted sites 1.61–52.36km downstream	de Solla *et al*. (2012)
	11	30.2–458						
Hamilton, ON:	1		60.8		Oct 2010		14.77 km downstream	
Welland River, downstream of airport	1		46					
	1		78.2					
Hamilton airport, ON, reference sites	4		6.1 ± 1.5		Oct 2010			
	4		7.2 ± 3.2					
Hamilton airport, ON, adjacent sites	2	9.7–29.40	19.55		Oct 2010			
	2	7.0–22.20	14.6					
Golden, BC: Cedar Lake, Emerald Lake, Bow Lake, Lake #4	4	0.04–0.10			Sep 2004	LC-MS	18L samples/site, split into duplicate	Loewen *et al*. (2008)
Kelowna, BC: Mill Creek			10					Gewurtz *et al*. (2013)
Regina, SK: Wascana Creek			7.8					
Great Lakes (Canada or United States)								
Lake Erie	8	11–39			2003	SPE; LC/MS	LOQ=0.7 ng/L	Boulanger *et al*. (2004)
Lake Erie	3	4.0–5.30			2004	HPLC-MS/MS	LOQ=0.5ng/L	Furdui *et al*. (2008*a*)

Location	n	Range	Mean	Median	Year sampled	Analytical method	Comments	Source
Lake Ontario	8	15–121			2003			Boulanger *et al*. (2004)
Lake Ontario	1		6.6		2002	HPLC-MS/MS	LOQ=0.5 ng/L	Furdui *et al.* (2008*a*)
	7	3.6–8.4			2004			
	3	3.6–37.6			2005			
Lake Huron	3	2.0–3.2			2004	HPLC-MS/MS	LOQ=0.5 ng/L	Furdui *et al*. (2008*a*)
	3	1.2–1.8			2005			
Lake Michigan	4	0.93–3.1			–	LC/MS	LOQ=0.28ng/L	Simcik & Dorweiler (2005)
Lake Superior	3	0.1–0.3			2005	HPLC-MS/MS	LOQ=0.5ng/L; concentrations below LOQ by signal to noise >3	Furdui *et al.* (2008*a*)
Lake Superior		<0.147–0.996	0.29		2005		site 80, all depths	Scott *et al*. (2010)
		<0.147–0.701	0.349		2005		site 170, all depths	
			0.135		May 2005			
			0.234		Aug 2005			
			0.301		Sep–Oct 2005		surface water, 10 sites	
Lake Superior: Lake Siskiwit			0.277		Sep 2005		5 m depth	Scott *et al.* (2010)
			0.289				15 m depth	
Lake Superior tributaries		<0.041–0.827						Scott *et al.* (2010)
United States								
Raisin River, MI	1		3.5		Mar 2001	SPE;HPLCES/MS/MS		Kannan *et al.* (2005*a*)
St. Clair River, MI	3	1.9–3.90	2.6		Apr 2001			
Detroit, MI	10	<0.08–6.13	3.48		2001	HPLC-MS/MS		Sinclair *et al*. (2004)
Flint, MI	4	1.50–12.31	4.90					
Lansing, MI	3	1.04–4.96	2.68					
Northeastern MI	2	0.87–6.34	3.60					
Northwestern MI	2	<0.8–4.48	4.48					
Saginaw Bay, MI	5	3.10–12.69	7.52					
Southwestern MI	5	7.22–29.26	16.10					
Upper Peninsula, MI	7	<0.8–3.09	1.84					
Western MI	6	<0.8–5.32	1.79					

Location	n	Range	Mean	Median	Year sampled	Analytical method	Comments	Source
Mississippi & Missouri river basins	173	<1.0–245		3.01	Mar & Aug 2008	SPE; UPLC-MS/MS	(95% CI: 2.03–3.78) LOD=0.02 ng/L	Nakayama *et al*. (2010)
New York State waters	53	0.8–1,090			Jul 2004	SPE; HPLC-MS/MS	LOQ=0.8 ng/L	Sinclair *et al*. (2006)
Erie Canal, NY	3	5.7–13		6.4				
Finger Lakes, NY	13	1.3–2.6		1.6				
Hudson River, NY	8	1.5–3.4		1.7				
Lake Champlain, NY	4	0.8–7.7		2.7				
Lake Erie, NY	3	2.8–5.5		3				
Lake Ontario, NY	13	2.9–30		4.9				
Niagara River, NY	3	3.3–6.7		5.5				
Oneida Lake, NY	1			3.5				
Lake Onondaga, NY	3	198–1,090		756				
Albany, NY	11	<0.25–9.30	4.14	2.88	Feb–Nov 2006	SPE; HPLC-MS/MS	LOQ=2.5 ng/L; lake water, urban area	Kim & Kannan (2007)
	14	<0.25–14.60	2.21	0.81	Jan–Mar 2007		LOQ=2.5 ng/L; surface water runoff	
Minneapolis, MN: Lake of the Isles, Lake Calhoun, Lake Harriet & Minnesota River	4	2.4–47	19.9		Undated	single-quadrupole LC/MS	urban surface waters; LOQ=0.28ng/L	Simcik & Dorweiler (2005)
Minneapolis, MN: Lake Calhoun	1		47				LOQ=0.28ng/L	
Lake Harriet, MN	1		21				LOQ=0.28ng/L	
Lake of the Isles, MN	1		2.4				LOQ=0.28ng/L	
Minnesota River, MN	1		9				LOQ=0.28ng/L	
Tettegouche & Nipisiquit, Tettegouche State Park, Loiten, Little Trout, Voyageurs National Park, MN	4	<0.28–1.20	0.42		Undated		remote lakes; LOQ=0.28ng/L	
Little Trout, MN	1		1.2				LOQ=0.28ng/L	

Location	n	Range	Mean	Median	Year sampled	Analytical method	Comments	Source
Loiten, MN	1		ND				LOQ=0.28ng/L	
Nipisiquit, MN	1		ND				LOQ=0.28ng/L	
Tettegouche, MN	1		0.2				LOQ=0.28ng/L	
Cape Fear Basin, NC	100	30.0–132		28.9	Spring 2006	SPE; HPLC-MS/MS	LOQ=0.2ng/L	Nakayama *et al*. (2007)
Tennessee River, AL	20	16.8–54.10	32		Nov 2000	SPE;HPLC-MS/MS	upstream of fluorochemical plant; LOQ=10–25ng/L; LOD=5 ng/L	Hansen *et al*. (2002)
	20	30.3–144	114				downstream of fluorochemical plant; LOQ=10–25ng/L; LOD=5 ng/L	
Clayton County, GA	5		18		2008		wetland in urbanized area (drinking water study)	Quinones & Snyder (2009)
Iowa City, IA	3		23 ± 1.5		Triplicate samples; river water samples; potentially high background concentrations	SPE; LC-MS/MS	LOQ=3	Boulanger *et al.* (2005*b*)
San José, CA,	4	9.3–20			May 2006	LC-MS/MS	DL=10	Plumlee *et al*. (2008)
Coyote Creek	4	4.8–25			Jun 2007		DL=2	
San Jose, CA, Upper	4	27–38			May 2006		DL=10	
Silver Creek	4	41–56			Jun 2007		DL=2	
Pacific Ocean								
Central to eastern surface water	14	0.0011–0.020			2002–2004	SPE-HPLC-ES/MS/MS		Yamashita *et al*. (2005)
Central to eastern deep water: 400–4,400 m	2	0.0032–0.0034						
Western	2	0.054–0.078						
Coastal China	14	0.023–9.68						
Coastal Hong Kong	12	0.07–2.60						
Coastal Korea	10	0.039–2.53						

Location	n	Range	Mean	Median	Year sampled	Analytical method	Comments	Source
Offshore Japan	4	0.04–0.075						
Sulu Sea ocean waters: surface water	5	<0.017–0.109						
Sulu Sea deep water: 1,000–3,000m	2	<0.017–0.024						
South China Sea	2	0.0080–0.113						
Tokyo Bay	8	0.338–57.7						
Atlantic Ocean								
Mid Atlantic Ocean waters	7	0.037–0.073			2002–2004	SPE-HPLC-ES/MS/MS		Yamashita *et al.* (2005)
North Atlantic Ocean waters	9	0.0086–0.036						
Sweden								
2 pristine catchment area streams	19	<7-102 pg/L			2011-12	UPLC-MS/MS	2 analytical batches	Filipovic *et al*. 2015
Austria								
Danube River system	9	<4.0–35				LLE-LC-MS/MS	river water	Clara *et al.* (2009)
Danube River	3	<4.0–<4.5						
Liesing River	3	13–22					tributary of the Danube River	
Schwechat	3	<5.1–35					tributary of the Danube River	
Germany								
Rhine River & tributaries	38	<2–26			Mar 2006	SPE; LC-ESI/MS/MS		Skutlarek *et al.* (2006)
Ruhr River	11	<2–22			May 2006			
Moehne River & Lake	4	<2–193			May 2006			
Elpe River	1	<2			May 2006			
Lenne River	3	<2–11			May 2006			
Rhine River & tributaries	2	5–9			Mar 2006			
Volme River	1	<2			May 2006			
Moehne River & selected tributaries	12	<2–3,160			May 2006		contaminated soils in agricultural area	

Location	n	Range	Mean	Median	Year sampled	Analytical method	Comments	Source
Italy								
Lake Maggiore	8	7.2–8.60	7.8 ± 0.6		Feb–Apr 2006	SPE; RP-LC-MS/MS	MDL=0.1ng/L; lake water	Loos *et al*. (2007)
	9	<0.1–38.5					MDL=0.1ng/L; affected rivers	
	3	<0.1–0.30					MDL=0.1ng/L; mountain rivers	
	2	<0.1	<0.1				MDL=0.1ng/L; mountain springs	
Netherlands								
Amsterdam (Rhine River)	6	0.3–28	5.2 ± 9.2		Spring 2008	SPE; HPLC-MS/MS	infiltrated river water	Eschauzier *et al*. (2010)
Poland								
Southern	11	1.10–153			Oct–Dec 2004	HPLC-MS/MS	inland sites	Rostkowski *et al.* (2009)
Northern	14	0.24–19			Oct–Dec 2004		inland sites	
Gulf of Gdańsk	9	0.28–0.96			May 2005		brackish water	
Spain								
Tarragona, Catalonia	4	<0.24–5.88			Feb 2007	HPLC-MS/MS	river water	Ericson *et al*. (2008)

Note: Due to differences in analytical methods used in the various studies, reported concentrations should not be compared directly.

95% CI = 95% confidence interval
ES = electrospray
ESI = electrospray ionization
GC = gas chromatography
HPLC = high-performance liquid chromatography
LC = liquid chromatography
LLE = liquid liquid extraction
LOD = limit of detection
LOQ = limit of quantification
MDL = method detection limit
MS = mass spectrometry
RP = reverse phase
SPE = solid-phase extraction
UPLC = ultra-performance liquid chromatography

Drinking water (ng/L)

Location	n	Range	Mean	Median	Year sampled	Analytical method	Comments	Source
Canada								
Canada-wide: raw water	12	0.001*–0.082			2009–2010	HPLC-MS/MS	one sample above MDL of 0.077 ng/L	Kubwabo & Lalonde (2010); C. Kubwabo (personal communication, 2011)
Canada-wide: finished water	12	0.001*–0.047*					all samples <MDL; 20 water distribution centres	
Niagara-on-the-Lake, ON	5		3.3		2006–2008	LC-MS/MS	tap water	Mak *et al*. (2009)
United States								
Lake Havasu City, AZ	6		9.4 ± 1.7		2008	SPE; HPLC-MS/MS	finished DW; urban areas	Quinones & Snyder (2009)
Orange County, CA	5		<1.0				indirect potable use	
Los Angeles, CA	5		57 ± 7.7				indirect potable use	
Aurora, CO	3		<1.0				finished DW; urban areas	
Clayton County, GA	7		22 ± 5.4				finished DW; urban areas	
Minneapolis, MN	7		<1.0				finished DW; urban areas	
Las Vegas, NV	33		1.2 ± 0.9				finished DW; urban areas	
Albany, NY	5		1.4		2006–2008	LC-MS/MS	TW	Mak *et al*. (2009)
Germany								
Ruhr area	21	<2–22			May 2006	SPE; LC-ESI/MS/MS		Skutlarek *et al*. (2006)
Sites outside Ruhr area	16	<2–6						
Ruhr River	692	<10–100		15	2008–2009	SPE; HPLC-MS/MS	DW. 26 waterworks; 37% <LOD; LOQ=10 ng/L	Wilhelm *et al*. (2010)
Italy								
Lake Maggiore	6	6.2–9.7	8.1 ± 1.2		Feb & Apr 2006	SPE; RP-LC-MS/MS	TW	Loos *et al*. (2007)
Spain								
Tarragona, Catalonia	4	0.39–0.87			Feb 2007	HPLC-MS/MS	municipal DW (TW)	Ericson *et al*. (2008)

Location	n	Range	Mean	Median	Year sampled	Analytical method	Comments	Source
	4	<0.24	<0.24				bottled water	
	4 0	<0.12–58.12	3.72 ± 10.73	0.51	Feb 2008	UPLC-MS/MS	municipal DW; 5 ND samples	
United Kingdom		<11–45					20 sites, DW; LOD=10ng/L	Atkinson *et al*. (2008)
China		0.042–11			2006–2008	LC-MS/MS	means range; TW for 10 cities	Mak *et al*. (2009)
India		0.033–8.4			2006–2008	LC-MS/MS	means range; TW for 4 cities	Mak *et al*. (2009)
Japan								
Japan, 6 cities		0.066–4.9			2006–2008	LC-MS/MS	means range; TW for 6 cities	Mak *et al*. (2009)
Osaka	2 6	0.26–22	3.6 ± 4.5	2.7	2006–2007 (winter & summer)	SPE; HPLC-MS/MS	raw water from lakes, rivers, groundwater; 14 treatment plants LOQ=0.1 ng/L	Takagi *et al.* (2008)
	2 6	0.16–22	2.7 ± 4.0	2.1			potable tap water	

DW = drinking water
ESI = electrospray ionization
HPLC = high-performance liquid chromatography
LC = liquid chromatography
LOD = limit of detection
LOQ = limit of quantification
MDL = method detection limit
MS = mass spectrometry
RP = reverse phase
SPE = solid-phase extraction
TW = tap water
UPLC = ultra-performance liquid chromatography

Precipitation

Location	n	Range	Mean	Year sampled	Analytical method	Comments	Source
Canada							
Algoma, ON	9	0.025–0.825 ng/m^2 per event	0.32 ng/m^2 per event	Jan–Jun 2005	HPLC-MS/MS	monthly samples	Scott *et al*. (2010)
Sibley, ON	8	<DL–13.4 ng/m^2 per event	5.8 ng/m^2 per event	May–Dec 2005		monthly samples; DL not stated.	
Lake Superior, ON			7,151 g/year				
Winnipeg, MB	3		0.59 ± 0.04 ng/L	Jul 2004	HPLC-MS/MS	one rainfall event	Loewen *et al*. (2005)
Sweden: 2 pristine catchment basins				2011			Filipovic *et al*. 2015
Rain	6-10	7-110 pg/L				% <DL=0	

Location	n	Range	Mean	Year sampled	Analytical method	Comments	Source
Snow	9	<3-63 pg/L				% <DL=12	
Italy Lake Maggiore	4	3.3–16.7 ng/L		Feb-Apr 2006	LC-MS-MS	4 sites; 4 grab samples	Loos *et al*. (2007)
Netherlands Amsterdam: Rhine River	6	<0.1–14 ng/L	1.8 ± 4.0 ng/L	Spring 2008	SPE; HPLC-MS/MS	infiltrated rain water	Eschauzier *et al*. (2010)

DL = detection limit
HPLC = high-performance liquid chromatography
LC = liquid chromatography
SPE = solid-phase extraction
MS = mass spectrometry

Sediment (ng/g)

Location	n	Min	Max	Mean	Year sampled	Analytical method	Comments	Source
Canada								
Arctic: Nunavut	9	0.022	85		Summer	LC-MS/MS	sediment core samples	Stock *et al*. (2007)
Cornwallis Island–Resolute Lake, NU	3	24	85	47.3	2003		(0–1, 1–2, 2–3 cm)	
Cornwallis Island–Char Lake, NU	3	<0.35	1.1				(0–1, 1–2, 2–3 cm)	
Cornwallis Island–Amituk Lake, NU	3	0.022	0.062				(0–1.5, 1.5–2.5, 2.5–3.5 cm)	
Ellesmere Island, NU		<0.066					(0–2 cm)	Veillette *et al.* (2012)
Toronto, ON, near Pearson Int'l Airport:							AFFF releases in 2000, 2002, 2005	Awad *et al*. (2011)
Spring Creek Pond				13.0	2003	LC-MS/MS	stormwater management pond,	
				13.0	2009		<100 m from AFFF discharge	
Spring Creek		<0.1	1.3		2003		2 sampling sites, 2 km upstream;	
				<0.1	2006		100 m downstream of Spring Creek	
		0.3	0.8		2009		Pond, which received AFFF discharge	
Etobicoke Creek		<0.1	2.7		2003		7 locations: 800 m; 5 km upstream,	
		<0.1	2.2		2006		1.7–16.5 km downstream of outfall	
		<0.2	1.4		2009			

Location	n	Min	Max	Mean	Year sampled	Analytical method	Comments	Source
Lake Ontario	25	0.684	51.8			HPLC/MS/MS	Niagara Basin, Mississauga Basin, Rochester Basin	Yeung *et al.* (2013)
		1.2	49			HPLC/MS/MS	Niagara Basin, Mississauga Basin, Rochester Basin, Marsh Creek, Central Hamilton Harbour	Myers *et al*. (2012)
		<0.1	>0.999			HPLC/MS/MS	tributaries draining to lake	Burniston *et al*. (2012)
		<1.2			2000	LC/MS/MS		Helm *et al*. (2007)
	22			10		LC/MS/MS		Gewurtz *et al*. (2013)
			28			LC/MS/MS		Stock *et al.* (2007)
	0.64	1.9				LC/MS/MS	Hamilton Harbour & Toronto Harbour	EC (2013*b*)
Lake Erie	<0..47				2004	LC/MS/MS		Helm *et al*. (2007)
	<0.1	>0.999				LC/MS/MS		Burniston *et al*. (2012)
	1	0.89				LC/MS/MS		EC (2013*b*)
Lake Huron		<1.7				LC/MS/MS	Georgian Bay	Helm *et al*. (2007)
		<0.1	0.999			LC/MS/MS	tributaries draining to lake	Burniston *et al.* (2012)
	1	2.2				LC/MS/MS		EC (2013*b*)
Lake Superior		<0.00006	<0.1			LC/MS/MS		Burniston *et al.* (2012)
				0.54			Thunder Bay	
		<0.44				LC/MS/MS	PFOS detected in 38% of samples	Helm *et al*. (2007)
	2	0.54	1.4			LC/MS/MS	open lake & Thunder Bay	EC (2013*b*)
Great Lakes (Ontario, Erie, Huron, Superior)	103	<0.6	1,272			HPLC/MS/MS	Great Lakes tributaries	Burniston *et al*. (2006)
Lake Simcoe	22	0.21	2.0			LC/MS/MS		Helm *et al.* (2011)
	22			0.76		HPLC/MS/MS		Gewurtz *et al.* (2013)
Lac St Pierre, QC	5			0.16		LC/MS/MS		EC (2013)
Nappan River, NB	1			2		LC/MS/MS		Gewurtz *et al*. (2013)
Kejimikujik Lake, NS	1			0.28		LC/MS/MS		EC (2013*b*)
Little Sackville, NS	1			0.19		LC/MS/MS		EC (2013*b*)

Location	n	Min	Max	Mean	Year sampled	Analytical method	Comments	Source
Osoyoos Lake, BC	1			0.36		LC/MS/MS		EC (2013*b*)
United States								
Lake Michigan	27	ND	1.15	0.45	2010	LC-MS/MS	Ponar-surface samples	Codling *et al*. (2014*b*)
	48	ND	12.78	2.70			sediment core samples (1900–2010)	

LC = liquid chromatography
MS = mass spectrometry
ND = not detected

Soil (ng/g)

Location	n	Range	Mean	Median	Geometric mean	Analytical method	Comments	Source
Canadian Arctic	27	<LOQ-7.47	0.322 (±0.287)	0.00675	0.00869	UPLC-MS/MS	Cornwallis Island	Cabrerizo *et al.* 2018
North America	33	0.018–1.956	0.3925 (±0.463)	0.226	0.220	Multi-stage extraction.	North America including Puerto Rico. Remote areas	Rankin *et al*. 2016
Asia	6	0.074-0.406	0.175 (±0.1218)	0.150	0.150	Xevo-TQ-S-MS/MS.	Middle East + China and Japan. Remote areas	
Europe	10	0.0071-3.130	0.863 (±1.161)	0.0837	0.167	LOQ=t>$t_{0.001}$; LOD=t>$t_{0.05}$	Northern Europe + Germany and Ireland. Remote areas	
Australia	4	0.044-0.258	0.138 (±0.098)	0.125	0.110	compared to process blank	Including New Zealand. Remote areas	
Africa	5	0.014-0.135	0.066 (±0.051)	0.072	0.046		Central West, East and South Africa. Remote areas	
South America	3	0.0267-0.048	0.036 (±0.011)	0.036	0.035		Lower latitudes	
Antarctica	1		0.007 (±0.00089)				Scott Base/McMurdo region	

Location	n	Range	Mean	Median	Geometric mean	Analytical method	Comments	Source
Global values: United States, China, Japan, Norway, Greece, and Mexico	60	<LOQ-0.001	0.001	0.00047		UPLC-MS/MS; LOQ=0.51	60 samples (10 each country) randomly selected from fresh and archived soils, considered indicators of background concentrations in different soils and parts of the world. 52% <LOQ	Strynar *et al*. 2012

Note: Due to differences in analytical methods used in the various studies, reported concentrations should not be compared directly.
LOD = limit of detection
LOQ = limit of quantification
UPLC-MS/MS = ultra high pressure liquid chromatography with tandem mass spectrometry
Xevo-TQ-S-MS/MS = tandem quadropole mass spectrometer.

Snow (pg/L)

Location	Date	n	Min	Max	Mean or (Median)	Comments	Reference
Sweden	2009	24	2.6	253	(20.5)		Codling *et al*. (2014*a*)
Canadian Arctic	1996–2005	30	2.6	86	(5.7)		Young *et al*. (2007)
	2008	28	69	680	280		MacInnis *et al.* (2017)
Svalbard, Norway	2006	4			33.9	SD=13.1	Kwok *et al*. (2013)
Beaufort, US Arctic	2010	3	<20		<20		Cai *et al*. (2012)
Sweden	2012		<3	49		snowmelt	Filipovic *et al*. (2015)
	2009		2.6	253		snowmelt	
Tibet	2010		25	64.2		snowmelt	

Biota

Species	Location	Type of sample	Units	n	Range	Mean ± SD	Year	Analytical method	Reference
Canada & Great Lakes									
Invertebrates									
Amphipod (fw) (*Gammarus* or *Hyalella* sp.)	Reference creeks, near Hamilton Airport (Stn 12, 13, 14, 15)	whole-body; composite	ng/g ww	9		19.1 ± 8	2010	LC-MS/MS	de Solla *et al.* (2012)
Amphipod (fw)	Adjacent Welland River (St 17)	whole-body; composite	ng/g ww	1		169.7			

Species	Location	Type of sample	Units	n	Range	Mean ± SD	Year	Analytical method	Reference
Amphipod (fw)	Adjacent Welland River (St 16)	whole-body; composite	ng/g ww	2		65.7 ± 0.4			
Amphipod (fw)	Welland River downstream of airport (Stn 5)	whole-body; composite	ng/g ww	2		721.35 ± 42.8			
Damselfly (*Zygoptera* sp.)	Welland River Stn 5	whole-body; composite	ng/g ww	2		170.32			
Freshwater shrimp (*Caridea* sp.)	Welland River Stn 5	whole-body; composite	ng/g ww	3		157.46			
Freshwater shrimp	Welland River downstream of airport (other sites)	whole-body; composite	ng/g ww	3		75.51			
Amphipod (fw) *Diporeia*	Lake Ontario, Niagara-on-the-Lake, ON	whole-body; 1 composite; 3 subsamples	ng/g ww	3		280 ± 33	2001	LC-MS/MS	Martin *et al.* (2004*b*)
Mysid shrimp (fw) *Mysis*	Lake Ontario, Niagara-on-the-Lake, ON	whole-body; 1 composite; 3 subsamples	ng/g ww	3		13 ± 1.8	2001		
Amphibians & fish									
Bullhead (juvenile) (*Ameiurus* spp.)	Stn 5	1 fish	ng/g ww	1		350.83		LC-MS/MS	de Solla *et al.* 2012)
Sunfish (juvenile) (*Centrarchidae* spp.)		1 fish	ng/g ww	1		507.93			
Lake trout (*Salvelinus namaycush*)	Lake Ontario, eastern basin, ON	4–5 whole fish per sample	ng/g ww	6	6–96		1979–2004	LC-MS/MS	Furdui *et al.* (2008*b*)
Lake whitefish (*Coregonus clupeaformis*)	Lake Huron, MI	liver	ng/g ww	5	33–81	67	1999–2000	HPLC-ES/MS/MS	Kannan *et al.* (2005*a*)
Lake whitefish	Lake Huron, MI	eggs	ng/g ww	2	145–381	263			
Brown trout (*Salmo trutta*)	Marquette, Lake Superior, USA	eggs	ng/g ww	3	49–75	64			

Species	Location	Type of sample	Units	n	Range	Mean ± SD	Year	Analytical method	Reference
Arctic sculpin (*Myoxocephalus scorpioides*)	Kuujjuarapik, QC	liver	ng/g ww	1		12	2002	LC-MS/MS	Martin *et al.* (2004*a*)
Brook trout (*Salvelinus fontinalis*)	Kuujjuarapik, QC	liver	ng/g ww	2	29–50	39	2002		
Lake whitefish	Kuujjuarapik, QC	liver	ng/g ww	2	12	12	2002		
White sucker (*Catostomus commersoni*)	Kuujjuarapik, QC	liver	ng/g ww	3	6.5–8.6	7.6	2002		
Northern pike (*Esox lucius*)	Kuujjuarapik, QC	liver	ng/g ww	1	5.7		2002		
Lake trout	Lac Minto, QC	liver	ng/g ww	1	31		2002		
Alewife (*Alosa pseudoharengus*)	Niagara-on-the-Lake, ON	whole-body composites (2 fish/composite)	ng/g ww	6		46 ± 15	2001	LC-MS/MS	Martin *et al.* (2004*b*)
Lake trout (*Salvelinus namaycush*)	Niagara-on-the-Lake, ON	whole-fish homogenate (7 fish/homogenate)	ng/g ww	8		170 ± 64	2001		
Sculpin (*Cottus cognatus*)	Niagara-on-the-Lake, ON	whole-body composite (3 fish/composite)	ng/g ww	5		450 ± 98			
Smelt (*Osmerus mordax*)	Niagara-on-the-Lake, ON	whole-body composites (5 fish/composite)	ng/g ww	6		110 ± 55	2001		
Lake trout	Lake Erie, ON		ng/g ww			92			Gewurtz *et al.* (2013)
Lake trout	Lake Ontario, ON		ng/g ww			51			
Walleye (*Sander vitreus*)	St. Lawrence River, ON		ng/g ww			30			
Walleye	Codette Reservoir, SK		ng/g ww			24			

Species	Location	Type of sample	Units	n	Range	Mean ± SD	Year	Analytical method	Reference
Walleye	Lake Diefenbaker, SK		ng/g ww			23			
Lake trout	Peninsula Harbour, ON		ng/g ww			24			
Lake trout	Lake Champlain, QC		ng/g ww			17			
Birds									
Double-crested cormorant (*Phalacrocorax auritus*)	Lake Winnipeg, NB	egg yolk	ng/g ww	4	130–320	210		ES-MS/MS	Giesy & Kannan (2001)
Double-crested cormorant	Lake Huron, MI	plasma	ng/mL	6	1–270	170			
Double-crested cormorant	Lake Huron, MI	plasma	ng/mL	3	110–430	260			
Herring gull (*Larus argentatus*)	Lake Huron, MI	plasma	ng/mL	2	66–79	73			
Herring gull	Lake Huron, MI	plasma	ng/mL	2	280–450	370			
Ring-billed gull (*Larus delawarensis*)	Lake Huron, MI	egg yolk	ng/g ww	3	<35–150				
Double-crested cormorant	Scarecrow Island, Canada	blood	ng/mL	1		132	1990–1998	HPLC-MS	Kannan *et al.* (2001)
Double-crested cormorant	Lake Winnipegosis, MB	egg yolk	ng/g ww	4	21–220	157			
Double-crested cormorant	Horrn Island, Lake Superior, MI	blood	ng/mL	1		36			
Double-crested cormorant	Lake Huron, MI	plasma	ng/mL	2	209–372				
Herring gull	Lake Huron, MI	blood	ng/mL	2	57–68				
Herring gull	Lake Huron, MI	plasma	ng/mL	2	239–391				
Ring-billed gull	Lake Huron, MI	egg yolk	ng/g ww	3	30–126	67			
Double-crested cormorant	Lake Superior	plasma	ng/mL	2	63–95				
Double-crested cormorant	Little Charity Island, Lake Huron, MI	blood	ng/mL	2	164–188	176			
Double-crested cormorant	Otter Island, Lake Superior, WI	blood	ng/mL	1		34			

Species	Location	Type of sample	Units	n	Range	Mean ± SD	Year	Analytical method	Reference
Common loon (*Gavia immer*)	Kuujjuarapik, QC	liver	ng/g ww	5	11–26	20	1992	LC-MS/MS	Martin *et al*. (2004*a*)
Black guillemot (*Cepphus grylle*)	Prince Leopold Island, NU	liver	ng/g ww	5	ND		1993		
Northern fulmar (*Fulmarus glacialis*)	Prince Leopold Island, NU	liver	ng/g ww	5	1.0–1.5	1.3	1993		
European starling (*Sturnus vulgaris*)	Brantford, ON, landfill	egg	ng/g ww			703			Gewurtz *et al.* (2013)
European starling	Calgary, AB, landfill	egg	ng/g ww			148			
European starling	Graves Island Provincial Park, NS	egg	ng/g ww			11			
European starling	Abbotsford, BC	egg	ng/g ww			6.3			
European starling	Pointe-aux- Prairies, QC	egg	ng/g ww			13			
European starling	Indus, AB	egg	ng/g ww			199			
European starling	Delta, BC	egg	ng/g ww			75			
European starling	Hamilton, ON	egg	ng/g ww			41			
European starling	Langley, BC, landfill	egg	ng/g ww			5.6			
European starling	Halton, ON, landfill	egg	ng/g ww			29			
European starling	Stoney Creek, ON, landfill	egg	ng/g ww			28			
European starling	Otter Lake, ON, landfill	egg	ng/g ww			18			
Gull	Lake Erie, ON	egg (pooled)	ng/g ww			676			
Other animals									
Snapping turtle (*Chelydra serpentina*)	Cootes Paradise, Hamilton, ON	plasma from individual turtles	ng/g ww	7		53 ± 17.1		LC-MS/MS	de Solla *et al*. (2012)
Snapping turtle	Credit River, Mississauga, ON		ng/g ww	10		171.4 ± 120			
Snapping turtle	Humber River, Toronto, ON		ng/g ww	7		121.4 ± 90.1			
Snapping turtle	Island Lake, Orangeville, ON (reference site)		ng/g ww	4		15.1 ± 9			
Snapping turtle	Lake Niapenco E, Hamilton ON		ng/g ww	9		2,376.7 ± 1,460.3			

Species	Location	Type of sample	Units	n	Range	Mean ± SD	Year	Analytical method	Reference
Snapping turtle	Lake Niapenco W (downstream of airport), Hamilton, ON		ng/g ww	9		2,065.2 ± 649.6			
Snapping turtle	Upper Welland River, Hamilton, ON		ng/g ww	1		2,269.4			
Gray seal (*Halichoerus grypus*)	Canadian Arctic	plasma	ng/mL	12	11–49	28		ES-MS/MS	Giesy & Kannan (2001)
Ringed seal (*Pusa hispida*)	Canadian Arctic	plasma	ng/mL	24	<3–12				
Arctic fox (*Vulpes lagopus*)	Arviat, NU	liver	ng/g ww	10	6.1–1,400	250	2001	LC-MS/MS	Martin *et al*. (2004*a*)
Ringed seal	Grise Fiord, NU	liver	ng/g ww	10	10–37	19	2001		
Ringed seal	Holman, NT	liver	ng/g ww	9	8.6–23	16	1998		
Polar bear (*Ursus maritimus*)	Sanikiluaq, NU	liver	ng/g ww	7	1,700–>4,000	3,100	2002		
Mink (*Mustela vison*)	Watson Lake Area, YK	liver	ng/g ww	10	1.3–20	8.7	2001		
United States & Great Lakes									
Invertebrates									
Eastern oyster (*Crassostrea virginica*)	Gulf of Mexico & Chesapeake Bay	soft tissue homogenates	ng/g dw		<42–1,225		1996–1998	HPLC-MS	Kannan *et al.* (2002*b*)
Amphibians & fish									
Brown trout	Michigan waters	eggs	ng/g ww	3	49–75	64		ES/MS/MS	Giesy & Kannan (2001)
Brown trout	Michigan waters	liver	ng/g ww	10	<17–26				
Brown trout	Michigan waters	muscle	ng/g ww	10	<6–46				
Brown trout	Michigan waters	eggs	ng/g ww	3	49–75	64			
Brown trout	Michigan waters	liver	ng/g ww	10	<17–26				
Chinook salmon (*Oncorhynchus tshawytscha*)	Michigan waters	liver	ng/g ww	6	33–170	110			
Chinook salmon	Michigan waters	muscle	ng/g ww	6	7–190	110			

Species	Location	Type of sample	Units	n	Range	Mean ± SD	Year	Analytical method	Reference
Lake whitefish	Michigan waters	eggs	ng/g ww	2	150–380	260			
Lake whitefish	Michigan waters	liver	ng/g ww	5	33–81	67			
Lake whitefish	Michigan waters	muscle	ng/g ww	5	97–170	130			
Yellowfin tuna (*Thunnus albacares*)	Northern North Pacific Ocean	liver	ng/g ww	12	<7				
Carp	Saginaw Bay, MI	muscle	ng/g ww	10	60–300	120			
Green frog (*Rana clamitans*)	Southwest MI	liver	ng/g ww	4	<35–290				
Chinook salmon	Grand River, MI	liver	ng/g ww	6	32–173	100	1999–2000	HPLC-ES/MS/MS	Kannan *et al.* (2005*a*)
Green frog, adult	Kalamazoo, MI	liver	ng/g ww	2	50–285	168	1998		
Green frog, adult	Kalamazoo, MI	liver	ng/g ww	2	<35	<35	1998		
Carp	Saginaw Bay, MI	muscle	ng/g ww	10	59–297	124	1999–2000		
Smallmouth bass (*Micropterus dolomieu*) (3 males)	Canada Lake, NY	liver	ng/g ww	2	39–77	56 ± 19	2001–2003	HPLC-MS/MS	Sinclair *et al.* (2006)
Smallmouth bass ((3 males)	Canada Lake, NY	liver	ng/g ww	3	58–95	76 ± 15			
Largemouth bass (*Micropterus salmoides*) (3 females)	Canadarago Lake, NY	liver	ng/g ww	3	14–126	58 ± 49			
large mouth bass (1 male)	Cuba Lake, NY	liver	ng/g ww	1		16			
Largemouth bass (2 females, 1 male)	Dunham Reservoir, NY	liver	ng/g ww	3	16–32	21 ± 7			
Smallmouth bass (3 males)	Dunham Reservoir, NY	liver	ng/g ww	3	33–104	70 ± 29			
Smallmouth bass (3 females)	Effley Falls, NY (remote site)	liver	ng/g ww	3	42–109	66 ± 30			

Species	Location	Type of sample	Units	n	Range	Mean ± SD	Year	Analytical method	Reference
Largemouth bass (3 females)	Goodyear Lake, NY	liver	ng/g ww	3	9–71	38 ± 25			
Largemouth bass (1 female, 2 males)	Lake Huntington, NY	liver	ng/g ww	3	79–142	102 ± 28			
Largemouth bass (3 females)	Loch Sheldrake, NY	liver	ng/g ww	3	45–78	56 ± 16			
Smallmouth bass (3 females)	Meacham Lake, NY	liver	ng/g ww	3	16–47	32 ± 12			
Smallmouth bass (2 females, 2 males)	Otsego Lake, NY	liver	ng/g ww	4	10–29	22 ± 8			
Largemouth bass (2 females; 1 male)	Payne Lake, NY (remote site)	liver	ng/g ww	3	21–41	30 ± 8			
Smallmouth bass (3 females)	Polliwog Pond, NY	liver	ng/g ww	3	32–57	40 ± 12			
Smallmouth bass (3 females)	Rio Reservoir, NY	liver	ng/g ww	3	86–98	93 ± 6			
Largemouth bass (3 females)	Rock Pond, NY	liver	ng/g ww	3	32–58	46 ± 11			
Smallmouth bass (3 females)	Soft Maple Dam, NY	liver	ng/g ww	3	41–114	79 ± 30			
Smallmouth bass (2 females)	Star Lake, NY (remote site)	liver	ng/g ww	2	45–69	57 ± 12			
Largemouth bass (1 female)	Star Lake, NY (remote site)	liver	ng/g ww	1		207			
Largemouth bass (4 males)	Swan Pond, NY	liver	ng/g ww	4	224–315	282 ± 35			
Smallmouth bass (2 females, 1 male)	Sylvia Lake, NY	liver	ng/g ww	3	14–75	38 ± 27			
Smallmouth bass (3 males)	Tupper Lake, NY	liver	ng/g ww	3	58–120	98 ± 28			
Largemouth bass (1 female)	Willis Lake, NY	liver	ng/g ww	1		23			

Species	Location	Type of sample	Units	n	Range	Mean ± SD	Year	Analytical method	Reference
Birds									
Bald eagle (*Haliaeetus leucocephalus*)	Midwestern	plasma	ng/mL	26	1–2,570	360		ES/MS/MS	Giesy & Kannan (2001)
Brown pelican (*Pelecanus occidentalis*)	Mississippi	liver	ng/g ww	2	290–620	460			
Common loon	North Carolina	liver	ng/g ww	8	35–690	290			
Double-crested cormorant	Gull Island, MI	blood	ng/mL	1		78	1990–1998	HPLC-MS	Kannan *et al.* (2001)
Brandt's cormorant (*Phalacrocorax penicillatus*)	San Diego, CA	liver	ng/g ww	2	46–1,780	907			
Double-crested cormorant	Saint Martin Island	blood	ng/mL	2	124–243	184			
Bald eagle	Upper Peninsula, MI	liver	ng/g	6	26.5–1,740		2000	LC-MS/MS	Kannan *et al*. (2005*a*)
Bald eagle		kidney	ng/g	4	35–1,480				
Bald eagle		gall bladder	ng/g	1	1,490				
Bald eagle		muscle	ng/g	6	<7.5–96.2				
Bald eagle		testes	ng/g	1	183				
Bald eagle		ovary	ng/g	1	68				
Other animals									
Polar bear	Alaska	liver	ng/g ww	17	180–680	350		ES-MS/MS	Giesy & Kannan (2001)
California sea lion (*Zalophus californianus*)	Coastal California	liver	ng/g ww	6	<35–49				
Elephant seal (*Mirounga* sp.)	Coastal California	liver	ng/g ww	5	<35				
Harbour seal (*Phoca vitulina*)	Coastal California	liver	ng/g ww	3	<35–57				
Sea otter (*Enhydra lutris*)	Coastal California	liver	ng/g ww	8	<35				
Northern fur seal (*Callorhinus ursinus*)	Coastal waters of Alaska	liver	ng/g ww	14	<35–120				

Species	Location	Type of sample	Units	n	Range	Mean ± SD	Year	Analytical method	Reference
Snapping turtle	Lake St. Clair, MI	plasma	ng/mL	5	1–170	72			
Mink (*Mustela vison*)	Midwestern	liver	ng/g ww	18	970–3,680	2,630			
Yellow-blotched map turtle (*Graptemys flavimaculata*)	Mississippi	liver	ng/g ww	6	39–700	190			
River otter (*Lontra canadensis*)	Northwestern	liver	ng/g ww	5	34–990	330			
River otter (6 yr old)	Bremerton, WA	liver	ng/g ww	1		288	1997–1998	ES-MS/MS	Kannan *et al*. (2002*a*)
River otter (5 yr old)	Eglon, WA	liver	ng/g ww	2	173–422	297 ± 176	1997–1998		
River otter (2–8 yr old)	Fort Ward, WA	liver	ng/g ww	3	139–189	156	1997–1998		
Mink (adult female)	Illinois	liver	ng/g ww	11	93–5,140	1,610	1995–1996		
Mink (juvenile female)	Illinois	liver	ng/g ww	10	243–3,650	1,450	1995–1996		
Mink (male adult)	Illinois	liver	ng/g ww	21	47–1,990	680	1995–1996		
Mink (male juvenile)	Illinois	liver	ng/g ww	21	64–4,870	1,210	1995–1996		
Mink (unidentified)	Illinois	liver	ng/g ww	2	1,900–2,700	2,290	1995–1996		
Mink (male juvenile)	Louisiana	liver	ng/g ww	7	40–320	140	1995–1996		
Mink (adult male)	Massachusetts	liver	ng/g ww	12	20–1,100	450	1995–1996		
Mink (juvenile male)	Massachusetts	liver	ng/g ww	1		67	1995–1996		
Mink (male)	Massachusetts	liver	ng/g ww	1		74	1995–1996		
Mink (juvenile female)	Massachusetts	liver	ng/g ww	2	70–140	110	1995–1996		

Species	Location	Type of sample	Units	n	Range	Mean ± SD	Year	Analytical method	Reference
Mink (female)	Massachusetts	liver	ng/g ww	1		410	1995–1996		
Mink (adult female)	Massachusetts	liver	ng/g ww	14	50–620	220	1995–1996		
River otter (2 yr old)	Nehalem River, OR	liver	ng/g ww	1		82.8	1997–1998		
River otter (2–3 yr old)	Silverdale, WA	liver	ng/g ww	2	151–248	199	1997–1998		
River otter (3–4 yr old)	Soleduck River, WA	liver	ng/g ww	2	25–62	43	1997–1998		
Mink (juvenile female)	South Carolina	liver	ng/g ww	2	650–1,500	1,070	1995–1996		
Mink (juvenile male)	South Carolina	liver	ng/g ww	7	1,240–3,110	2,370	1995–1996		
River otter (1–5 yr old)	Willamette River, OR	liver	ng/g ww	7	97–994	579	1997–1998		
River otters (2–4 yr old)	Yaquina River, OR	liver	ng/g ww	2	34–45	39	1997–1998		
Mink, females	Kalamazoo, MI	liver	ng/g ww	1	41	41	2000–2001	LC-MS/MS	Kannan *et al.* (2005*a*)
Mink, males (0.5–3 yr old)		liver	ng/g ww	7	1,280–59 500	18 000	2000–2001		
Snapping turtle, adult female		liver	ng/g ww	3	<1–8.8	6.13	1999–2000		
Snapping turtle, adult male		liver	ng/g ww	2	105–169	137 ± 45.3	1999–2000		
Polar bears, male, Beaufort Sea subpopulation	Alaska	liver	ng/g ww	7	502–1,130	755 ± 179	1993–2002	HPLC-MS/MS	Kannan *et al.* (2005*b*)
Polar bears, male, Chukchi Sea subpopulation	Alaska	liver	ng/g ww	14	292–1,020	592 ± 219			

Species	Location	Type of sample	Units	n	Range	Mean ± SD	Year	Analytical method	Reference
Other regions									
Amphibians & fish									
Bluefin tuna (*Thunnus thynnus*)	Mediterranean Sea	liver	ng/g ww	8	21–87	48		ES-MS/MS	Giesy & Kannan (2001)
Herring	Landsort, Baltic Sea	whole-fish homogenates	ng/g ww	10	1.7–2.8	2.3 (median)	2005	HPLC-MS/MS	Holmstrom & Berger (2008)
Birds									
Common cormorant (*Phalacrocorax carbo*)	Italy	liver	ng/g ww	12	33–470	96		ES-MS/MS	Giesy & Kannan (2001)
Black-tailed gull (*Larus crassirostris*)	Hokkaido, Japan	plasma	ng/mL	24	2–12	6			
Black-tailed gull	Korea	liver	ng/g ww	15	70–500	170			
Laysan albatross (*Phoebastria immutabilis*) & black-footed albatross (*Phoebastria nigripes*)	Midway Atoll, North Pacific	liver	ng/g ww	9	<35				
Laysan & black-footed albatross	Midway Atoll, North Pacific	plasma	ng/mL	3	9–26	18			
Laysan & black-footed albatross	Midway Atoll, North Pacific	plasma	ng/mL	10	3–39	9			
Polar skua (*Stercorarius maccormicki*)	Terra Nova Bay, Antarctica	plasma	ng/mL	2	<1–1.4				
Common guillemot (*Uria aalge*)	Stora Karlsö, Baltic Sea	muscle	ng/g ww	8	9.8–17	14 (median)	1989	HPLC-MS/MS	Holmstrom & Berger (2008)
Common guillemot	Stora Karlsö, Baltic Sea	kidney	ng/g ww	10	92–183	127 (median)			
Common guillemot	Stora Karlsö, Baltic Sea	adult liver	ng/g ww	13	91–150	121 (median)			
Common guillemot	Stora Karlsö, Baltic Sea	chick liver	ng/g ww	10	185–322	309 (median)			
Common guillemot	Stora Karlsö, Baltic Sea	eggs	ng/g ww	8	243–432	325 (median)			

Species	Location	Type of sample	Units	n	Range	Mean ± SD	Year	Analytical method	Reference
Other animals									
Gray seal	Baltic Sea	plasma	ng/mL	26	14–76	37		ES-MS/MS	Giesy & Kannan (2001)
Ringed seal	Baltic Sea	plasma	ng/mL	18	16–230	110			
Bottlenose dolphin	Mediterranean Sea	liver	ng/g	5	170–430	270			
Striped dolphin (*Stenella coeruleoalba*)	Mediterranean Sea	liver	ng/g	4	65–160	100			
Ringed seal	Norwegian Arctic	plasma	ng/mL	18	5–14	9			
Ganges river dolphin (*Platanista gangetica*)	Ganges River, India	liver	ng/g	2	<35–81				
Weddell seal (*Leptonychotes weddellii*)	Terra Nova Bay, Antarctica	liver	ng/g	1	<35				

ES = electrospray
HPLC = high-performance liquid chromatography
LC = liquid chromatography
MS = mass spectrometry
ND = not detected
SD = standard deviation
ww = wet weight

Biota consumed as food (ng/g ww)

Organism (part & preparation)	n	Range	Mean ± SD	Year	Analytical method	Comments	Reference
Canada: Nunavut							
Aquatic traditional foods				1997–1998	HPLC-ESI-MS/MS	Archived locally harvested foods	Ostertag *et al.* (2009*b*)
Ringed seal (*Pusa hispida*)							
Liver, raw	2	3.8–7.6	5.7				
Blood, raw	3		2.9 ± 2.1				
Meat, boiled	1		0.5				
Blubber, raw	2	<0.1–0.1					
Polar bear (*Ursus maritimus*)							
Meat, frozen	1		4.0				
Beluga whale (*Delphinapterus leucas*)							

Organism (part & preparation)	n	Range	Mean ± SD	Year	Analytical method	Comments	Reference
Blubber, raw	1		1.5				
Meat, dried	2	1.6–3.6					
Muktuk, raw	1		0.4				
Narwhal (*Monodon monoceros*)							
Blubber, raw	1		0.2				
Muktuk, raw	2	<0.4	<0.4				
Muktuk, frozen	2	0.4–1.6					
Bearded seal (*Erignathus barbatus*)							
Intestine, boiled	2	0.5–0.6					
Meat, boiled	1		0.2				
Walrus (*Odobenus rosmarus*)							
Blubber, aged	1		<0.1				
Kauk, raw	1		0.2				
Meat, raw	2	<0.3	<0.3				
Meat, aged	1		<0.2				
Eider duck (*Somateria mollissima*)							
Whole, boiled	1		1.6				
Black duck (*Anas rubripes*)							
Meat, boiled	1		0.3				
Arctic char (*Salvelinus alpinus*)							
Whole, raw	3		<0.5				
Lake trout (*Salvelinus namaycush*)							
Whole, raw	2	0.1–0.4					
Seaweed							
Whole, raw	1		<0.2				
Clams							
Whole, raw	1		<0.2				
Terrestrial traditional foods							
Caribou (*Rangifer tarandus*)							
Liver, baked	1		5.0				
Liver, raw	3		2.7 ± 2.3				

Organism (part & preparation)	n	Range	Mean ± SD	Year	Analytical method	Comments	Reference
Meat, boiled	2	<0.3–0.1					
Meat, dried	2		<0.4				
Meat, raw	2		<0.2				
Meat, roasted	1		<0.2				
Bone marrow, boiled	1		0.2				
Heart, blood, raw	1		0.2				
Fat, raw	1		<0.2				
Kidneys, raw	2	0.1	0.1				
Kidneys, boiled	1		<0.2				
Stomach, raw	1		0.1				
Tongue, raw	3		0.2 ± 0.2				
Ptarmigan (*Lagopus muta*)							
Whole, raw	3		<0.2				
Arctic hare (*Lepus arcticus*)							
Meat, raw	2		<0.2				
Snow goose (*Chen caerulescens*)							
Meat, raw	1		<0.2				
Berries							
Whole, raw	3		<0.1				

ESI = electrospray ionization
HPLC = high-performance liquid chromatography
MS = mass spectrometry
SD = standard deviation
ww = wet weight

Foods (ng/g ww)

Location & food composite	n	Range	Concentration	Year sampled/collected	Analytical method	Comment	Reference
Canada							
Toronto, Mississauga, Ottawa, ON					LC-MS/MS	composite sample LODs range: 0.03–10 ng/g ww	Del Gobbo *et al*. (2008)
Catfish, fried	9		0.90				
Catfish, raw	9		1.57				
Cuttlefish, raw	9		ND				
Grey mullet, fried	9		1.14				
Grouper, fried	9		0.47				
Grouper, raw	9		ND				
Monkfish, boiled	9		0.22				
Monkfish, raw	9		1.34				
Octopus, boiled	9		0.23				
Octopus, raw	9		ND				
Red snapper, boiled	9		0.21				
Red snapper, fried	9		0.78				
Red snapper, raw	9		1.46				
Sea squirt, boiled	9		ND				
Sea squirt, raw	9		ND				
Skate, boiled	9		0.88				
Skate, raw	9		1.51				
Whiting, raw	9		ND				
Yellow croaker, boiled	9		0.89				
Yellow croaker, fried	9		0.68				
Yellow croaker, raw	9		1.68				
Winnipeg, MB, Canadian TDS							Tittlemier *et al*. (2007)
Meat, poultry & eggs							
Beef steak	unknown		2.7	2004	HPLC-MS/MS	composite samples	
Cold cuts	unknown		0.5*			composite (beef bologna & pastrami) measured concentration >LOD but <LOQ	
Ground beef	unknown		2.1			composite sample	
Roast beef	unknown		<0.6			composite sample	

Location & food composite	n	Range	Concentration	Year sampled/collected	Analytical method	Comment	Reference
Luncheon meats, cold cuts	unknown		0.5*			composite sample. measured concentration >LOD but <LOQ	
Fish & seafood							
Fish, freshwater	unknown		1.3–1.5*	1998	HPLC-MS/MS	archived composite (smelt, perch) measured concentration >LOD but <LOQ	
Fish, freshwater	unknown		2	2004	HPLC-MS/MS	composite (trout, pickerel)	
Fish, marine	unknown		2.6			composite (haddock, cod, sole)	
Prepared foods							
Pizza	unknown		<1	1998	HPLC-MS/MS	archived composite	
Breads & cereals							
Microwave popcorn	unknown		0.98*	1999	HPLC-MS/MS	measured concentration >LOD but <LOQ; archived sample	
Whitehorse, YT, Canadian TDS							Ostertag *et al.* (2009*a*)
Meat, poultry & eggs							
Cold cuts	4		<0.68	1998	HPLC-MS/MS	archived composite samples; concentration blank corrected	
Lunchmeats, canned	4		<0.37			archived composite samples; concentration blank corrected	
Dairy							
Cheese	4		0.71	1998	HPLC-MS/MS	archived composite sample; measured concentration blank corrected, <LOQ of 0.95 ng/g ww	
Cheese, processed	4		1.14			archived composite sample; concentration blank corrected	
Vegetables							
Peppers	4		<0.15	1998	HPLC-MS/MS	archived composite; concentrations blank corrected	
Prepared foods							
Cookies	4		<0.15	1998	HPLC-MS/MS	archived composite; concentrations blank corrected	
Frozen dinner, beef	4		<0.17			archived composite; concentrations blank corrected	

Location & food composite	n	Range	Concentration	Year sampled/collected	Analytical method	Comment	Reference
Pizza	4		<0.20			archived composite; concentrations blank corrected	
United States							
Freshwater fish							
Great Lakes	157	max=80	15.2 (50th percentile)	2010	HPLC-MS/MS	MDL=0.13 ng/g; 157 composite samples (423 fish), 18 species; detection=100%	Stahl *et al.* (2014)
Urban rivers	162	max=127	10.7 (50th percentile)	2010		MDL=5.35 ng/g; 162 composite samples (682 fish), 25 species; detection=73%	
United Kingdom							
Meat, poultry & eggs							Clarke *et al.* (2010)
Chicken liver	2		<1	2007–2008	SPE-HPLC-MS/MS	composite samples	
Chicken liver pâté	1		<1				
Duck liver pâté	1		<1				
Lamb liver	7	<1–5	2.6				
Ox liver	5	<1–5	1.8				
Pig liver	5	<1–4	2.2				
Venison liver	4	1–10	5.0				
All livers	25	1–10	2.5				
Ox kidney	4	<1–3	1.8				
Pig kidney	4	<1–4	1.6				
All kidney	12	1–3	1.4				
Lamb heart	2		<1				
Black pudding	2		<1				
Meat (not offal)	16		<1				
Egg	12	<1–1	<1				
Fish & seafood							
Eel (fresh)	3	<1–2	1.3	2007–2008	SPE; HPLC-MS/MS	composite samples	
Eel (smoked)	3	<1–59	20				
Herring	4	<1–1	1				
Mackerel	4	<1–2	1.2				

Location & food composite	n	Range	Concentration	Year sampled/collected	Analytical method	Comment	Reference
Atlantic salmon (farmed)	5		<1				
Atlantic salmon (wild)	2		<1				
Alaskan salmon (wild)	1		<1				
Sardines	6	1–3	2.0				
Sprats	3	1–4	3.0				
Trout (farmed)	4	<1–1	1.0				
Whitebait	6	<1–40	15				
Carp	6	<1–8	5.5				
All oily fish	47	<1–59	1.1				
Cod	4	<1–2	1.5				
Haddock	4	<1–1	1.0				
Plaice	2	<1–1	1				
Sole	2	<1–1	<1				
All whitefish	12	<1–2	1.2				
Crab	6	2–13	6.3				
Crayfish	1		<1				
Langoustine	1		<1				
Pacific oysters	2	1–10	2.5				
Prawns	2	<1–1	<1				
All shellfish	12	1–13	4.4				
Dairy							
Cheese	10	<1–10	<1.9	2007–2008	SPE; HPLC-MS/MS	composite samples	
Milk	11		<1				
Breads & cereals							
Bread	4		<1	2007–2008	SPE; HPLC-MS/MS	composite samples	
Cereals	4		<1				
Popcorn	4		<1		HPLC-MS/MS		
Vegetables							
Potatoes & products	21		<1	2007–2008	SPE; HPLC-MS/MS	composite samples	
Vegetables	42		<1				
Condiments, oils							
Jams	6		<1	2007–2008		composite samples	

Location & food composite	n	Range	Concentration	Year sampled/collected	Analytical method	Comment	Reference
Fish oil supplements	4		<1		SPE; HPLC-MS/MS		
Vegetable oils	6		<1				

LC = liquid chromatography
MS = mass spectrometry
LOD = limit of detection
HPLC = high-performance liquid chromatography
LOQ = limit of quantification
MDL = method detection limit
ND = not detected
SPE = solid-phase extraction
ww = wet weight

Human body fluids (ng/mL)

Location	Body fluid	n	Range	Geometric mean	Mean ± SD	Year	Analytical method	Comments	Reference
Canada									
	plasma	2,880		8.85; 95% CI: 7.97–9.82	11.31 (95% CI: 10.02–12.60)	2007–2009	UPLC-MS/MS	CHMS participants 20–79 yr old	HC (2010*c*)
	plasma	1,017		6.9; 95% CI:6.2–7.6	6.8 (95% CI: 6.0–7.6) (median)	2009–2011	UPLC-MS/MS	CHMS participants 20–79 yr old	HC (2013*b*)
Hamilton, ON	serum (maternal)	101	10.8–22.9		18.31 ± 10.95; 16.6 (median)	2004–2005	HPLC-MS/MS	hospital-based, maternal serum: 24-28 wk gestation	Monroy *et al*. (2008)
	serum (maternal)	101	9.19–20.22		16.19 ± 10.43; 14.54 (median)	2004–2005	HPLC-MS/MS	maternal serum: at delivery	
	blood (umbilical)	105	3.92–9.11		7.19 ± 5.73; 6.08 (median)	2004–2005	LC-MS/MS		
Nunavik, QC	plasma	720		18.6; 95% CI: 17.8–19.5	25.7 (SE: 1.0)	2005	LC-MS/MS	male & female Inuit 18–74 yr old	Château-Degat *et al.* (2010)
Nunavik, QC	plasma	857	0.480–470	18.68		2005	LC-MS/MS	male & female Inuit 18–74 yr old	Dallaire *et al*. (2009*a*)
Nunavik, QC	plasma	621	0.480–470	18.28		2005	LC-MS/MS	male & female Inuit 18–74 yr old	Dallaire *et al*. (2009*b*)
Nunavik, QC	plasma	86	0.910–31	3.369; 95% CI: 3.798–4.056		2006–2008	HPLC-MS/MS	Inuit toddlers (11–54 mos old) attending childcare centres	Turgeon O'Brien *et al*. (2012)

Location	Body fluid	n	Range	Geometric mean	Mean ± SD	Year	Analytical method	Comments	Reference
Ottawa, ON; Gatineau, QC	serum	56	3.7–65.1		28.8 ± 14.3 (overall); 28.3 (m); 29.7 (f)	Jan 2002	HPLC-MS/MS	>20-yr-old volunteers (35 m; 21 f) MDL=1.1; LOQ=3.6 ng/mL	Kubwabo *et al.* (2004)
Kingston region, ON	breast milk	13			ND	2003–2004	LC-MS/MS	ND = 13 (LOD not provided)	Kubwabo *et al.* (2013)
United States									
	serum	1562		30.4; 95% CI: 27.1–33.9	30.2 (median); 95% CI: 27.8-33.9	1999–2000	SPE; HPLC-MS/MS	NHANES; All age groups, genders combined	CDC (2018)
		2094		20.7; 95% CI: 19.2-22.3	21.2 (median); 95% CI: 19.8-22.4	2003–2004			
		2120		17.1; 95% CI: 16.0–18.2	17.5 (median); 95% CI: 16.8-18.6	2005-2006			
		2100		13.2; 95% CI: 12.2–14.2	13.6 (median); 95% CI: 12.8-14.7	2007-2008			
		2233		9.32; 95% CI: 8.13–10.7	9.70 (median); 95% CI: 8.50-10.8	2009-2010			
		1904		6.31; 95% CI: 5.84–6.82	6.53 (median); 95% CI: 5.99-7.13	2011-2012			
		2165		4.99; 95% CI: 4.50–5.52	5.20 (median); 95% CI: 4.80-5.70	2013-2014			
	serum	640		19.3; 95% CI: 17.5–21.4	19.9 (median); 95% CI: 17.8–22.0	2003–2004	SPE; HPLC-MS/MS	NHANES adolescents	Calafat *et al.* (2007*b*); CDC (2012)
		2094		20.7; 95% CI: 19.2–22.3	21.2 (median); 95% CI 19.8–22.4	2003–2004		NHANES LOD=0.4 µg/L	
		640		19.3; 95% CI: 17.5–21.4	19.9 (median); 95% CI: 17.6–21.9	2003–2004		NHANES adolescents (12–19 yr old)	
	serum	23		30.0 ng/mL	31.1	2001–2002	SPE; HPLC-MS/MS	residents	Calafat *et al.* (2006)
	serum	1,562		30.4; 95% CI: 27.1–33.9	30.2 (median); 95% CI: 27.8–33.8	1999–2000	SPE; HPLC-MS/MS	NHANES participants ≥12 yr old; LOD=0.2 µg/L	Calafat *et al.* (2007*a*)
		543		29.1; 95% CI: 26.2–32.4	29.4 (median); 95% CI: 26.8–34.2			NHANES participants ≥12 yr old; LOD=0.2 µg/L.	

Location	Body fluid	n	Range	Geometric mean	Mean ± SD	Year	Analytical method	Comments	Reference
		364		27.5; 95% CI: 24.9–30.2	27.9 (median); 95% CI: 24.8–29.7			NHANES participants ≥20 yr old; LOD=0.2 µg/L	
		295		33.0; 95% CI: 28.0–38.8	33.6 (median); 95% CI: 28.0–38.7			NHANES participants ≥40 yr old; LOD=0.2 µg/L	
		360		33.3; 95% CI: 28.5–38.8	33.7 (median); 95% CI: 27.4–39.9			NHANES participants ≥60 yr old; LOD=0.2 µg/L	
		819		28.0; 95% CI: 24.6–31.8	27.7 (median); 95% CI: 24.5–30.2			NHANES female participants. LOD=0.2 µg/L	
		743		33.4; 95% CI: 29.6–37.6	34.8 (median); 95% CI: 31.1–37.9			NHANES male participants. LOD=0.2 µg/L	
		584		22.7; 95% CI: 19.8–25.9	23.7 (median); 95% CI: 20.8–27.2			NHANES Mexican-American participants LOD=0.2 µg/L	
		309		33.0; 95% CI: 26.2–41.6	32.0 (median); 95% CI: 24.3–45.7			NHANES non-Hispanic black participants LOD=0.2 µg/L	
		529		32.0; 95% CI: 29.1–35.2	32.4 (median); 95% CI: 29.3–35.5			NHANES non-Hispanic white participants LOD=0.2 µg/L	
	serum	645	<4.3–1,656	34.9; 95% CI: 33.3–36.5	35.8 (median)	2000–2001	HPLC-ES-MS/MS	blood donors	Olsen *et al*. (2003*c*)
	serum	24	<6.1–58.3	14.7; 95% CI: 11.1–19.4	17.7; 95% CI: 13.0–22.5		HPLC-ES-MS/MS	deceased donors; 1/2 LOQ used for concentrations <LOQ	Olsen *et al.* (2003*d*)
	serum	598	6.7–515	37.5; 95% CI: 36.0–39.1			HPLC-MS/MS	children (2–12 years old) with group A streptococcal infections	Olsen *et al*. (2004*a*)
	serum	616		4.30; 95% CI: 4.11-4.50		2015		Red Cross blood donors across the US.	Olsen *et al*. 2017
Atlanta, GA	serum	20	3.6–164		55.8	2003	SPE; HPLC-MS/MS	50% men; 90% Caucasian; 42.3 ± 10.2 year mean age; LOD=0.4 ng/mL	Kuklenyik *et al*. (2004)
Baltimore, MD	serum (umbilical)	299	<0.2–34.8	4.9		2004–2005	SPE; HPLC-MS/MS	Baltimore THREE study	Apelberg *et al*. (2007*a*, 2007*b*)
Washington County, MD	serum	178		30.1; 95% CI: 27.8–32.6	29.5 (median)	1974	LC-MS/MS	1,974 blood samples; LLOQ=3.9 ng/mL	Olsen *et al.* (2005)

Location	Body fluid	n	Range	Geometric mean	Mean ± SD	Year	Analytical method	Comments	Reference
		178		33.3; 95% CI: 31.1–35.6	34.7 (median) (IQR 25.0–44.0)	1989	LC-MS/MS	1,989 blood samples; LLOQ=3.9 ng/mL	
Massachusetts	breast milk	45	<0.032–0.617		0.131 (mean) 0.106 (median)	2004	HPLC-ESI-MS/MS	women 22–43 yr old	Tao *et al*. (2008*a*)
North Carolina	breast milk	64	<LOQ		<0.60	2004–2005	SPE; HPLC-MS/MS	women 18–38 yr old	von Ehrenstein *et al.* (2009)
	serum (maternal)	n=34 n=30			visit 1: 21.9 visit 2: 18.8		SPE; HPLC-MS/MS	visit 1: 2–7 weeks postpartum visit 2: 3–4 months postpartum	
Midwest	serum	16			not detected	2004–2005	GC-MS/MS	pooled samples ≥10 individuals/sample; 1 sample pooled from 1,000–1,500 males	De Silva & Mabury (2006)
Minneapolis-St. Paul, MN	plasma	40	6.6–36.9	15.1; 95% CI: 13.3–17.1	16.3; 95% CI: 14.0–18.2	2005	HPLC-MS/MS	blood donors	Olsen *et al*. (2007)
California	serum	93	max=39.4	8.51	11.02	2011–2013	SPE; HPLC-MS/MS	CA women with PFOS in drinking water supply	Hurley *et al*. (2016)
		1,240	max=99.8	6.76	8.42			CA women without PFOS in drinking water supply	
Seattle, WA	serum	238	<3.4–175	31; 95% CI GM: 28.8–33.4	30.2 (median)	2000–2001	HPLC-MS/MS	elderly	Olsen *et al.* (2004*b*)
Belgium	breast milk	22	<0.4–28.2		2.9		UPLC ES-MS/MS	women 18–30 yr old	Roosens *et al*. (2010)
Denmark	plasma (maternal)	1,399	6.4–106.7		35.3 ± 13.0	1996–2002	SPE; HPLC-MS/MS	Danish National Birth Cohort (1st-trimester women)	Fei *et al*. (2007)
	plasma (maternal)	200			29.9 ± 11.0	1996–2002	SPE; HPLC-MS/MS	Danish National Birth Cohort (2nd-trimester women)	
	plasma (umbilical)	50			11 ± 4.7	1996–2002	SPE; HPLC-MS/MS	Danish National Birth Cohort (male & female umbilical blood)	

Location	Body fluid	n	Range	Geometric mean	Mean ± SD	Year	Analytical method	Comments	Reference
Germany									
Sauerland, North Rhine-Westphalia	breast milk	183			0.09L			detected in samples; ND=84; DL not stated; means based on detects	Wilhelm *et al.* (2008)
Leipzig	breast milk	38	0.033–0.309		0.126		LC-MS/MS	from breast milk bank	Volkel *et al*. (2008)
Munich		19	0.028–0.239		0.116	2006	LC-MS/MS	fresh samples from healthy donors	
Germany, Hungary	breast milk	70	0.028–0.639		0.158		LC-MS/MS	pooled data	Volkel *et al.* (2008)
Hungary	breast milk	13	0.96–0.639		0.317	1996–1997	LC-MS/MS	hospital supply	Volkel *et al.* (2008)
Poland	blood	15	5.2–24		12 ± 5.7	2003	HPLC-ESI-MS	male dockers, 19–62 yr old	Falandysz *et al*. (2006)
Gulf of Gdańsk	(whole)	15	6.6–25		13 ± 5.2	2003	HPLC-ESI-MS	2 female/13 male farmers, 19–62 yr old	
		15	14–84		41 ± 23	2003	HPLC-ESI-MS	4 female/11 male Baltic Sea fish consumers, 19–62 yr old	
		15	6.7–84		16 ± 12	2003	HPLC-ESI-MS	5 female/10 male Gdańsk general population, 19–62 yr old	
Spain									
Catalonia	breast milk	10	0.07–0.22		0.12	2007	UPLC-ES-MS/MS	women 30–39 yr old	Kärrman *et al.* (2010)
Barcelona	breast milk	20	<LOQ–865	0.1–0.2	0.122	2008	LC-SPE-MS/MS		Llorca *et al*. (2010)
Norway									
Oslo	breast milk	68	0.028–0.36		0.11 (median)	2001–2009	LC-MS	median age: 29 yr	Thomsen *et al.* (2010)
Sweden									
Stockholm	breast milk	75			0.023	1972	HPLC-MS/MS	average maternal age: 27–28 yr	Sundström *et al.* (2011)

Location	Body fluid	n	Range	Geometric mean	Mean ± SD	Year	Analytical method	Comments	Reference
		78			0.059	1976		average maternal age: 27–28 yr	
		116			0.103	1980		average maternal age: 27–28 yr	
		102			0.172	1984–1985		average maternal age: 27–28 yr	
		20			0.211	1988		average maternal age: 30 yr	
		20			0.202	1990		average maternal age: 30 yr	
		20			0.222	1992		average maternal age: 29 yr	
		20			0.219	1994		average maternal age: 29 yr	
		20			0.214	1995		average maternal age: 30 yr	
		20			0.224	1996		average maternal age: 31 yr	
		20			0.237	1997		average maternal age: 31 yr	
		20			0.212	1998		average maternal age: 30 yr	
		20			0.234	1999		average maternal age: 31 yr	
		20			0.213	2000		average maternal age: 30 yr	
		20			0.198	2001		average maternal age: 30 yr	
		20			0.210	2002		average maternal age: 30 yr	
		15			0.179	2003		average maternal age: 31 yr	
		20			0.188	2004		average maternal age: 30 yr	
		20			0.122	2007		average maternal age: 27 yr	
		18			0.075	2008		average maternal age: 28 yr	
	breast milk	12	0.06–0.47		0.201 ± 0.117 (mean); 0.166 (median)	2004	HPLC	Swedish women 22–33 yr	Kärrman *et al*. (2007)
Uppsala	serum (maternal)	12	8.2–48		20.7 ± 10.5 (mean); 18.7 (median)	2004	HPLC-MS/MS	MDL=0.2 ng/mL	Kärrman *et al*. (2007)
Japan									
	Serum (umbilical)	15	1.6–5.3		8.9 maternal serum; 2.9 cord blood	2003	LC-MS		Inoue *et al*. 2004 cited in ATSDR 2015
	Serum (maternal)	15	4.9–17.6	8.4	8.9 ± 3.2	2003	LC-MS	maternal blood collected from gestation weeks 38–41	

Location	Body fluid	n	Range	Geometric mean	Mean ± SD	Year	Analytical method	Comments	Reference
China									
Zhoushan	breast milk	19	0.045–0.36			2004	HPLC-MS/MS	average maternal age: 25.8 yr	So *et al*. (2006) cited in ATSDR 2015

95% CI = 95% confidence interval
CHMS = Canadian Health Measure Survey
DL = detection limit
ES = electrospray
ESI = electrospray ionization
GC = gas chromatography
HPLC = high-performance liquid chromatography
IQR = interquartile range
LC = liquid chromatography
LLOQ = lower limit of quantification
LOD = limit of detection
LOQ = limit of quantification
MDL = method detection limit
MS = mass spectrometry
ND = not detected
NHANES = National Health and Nutrition Examination Survey
SD = standard deviation
SE = standard error of the mean
SPE = solid-phase extraction
UPLC = ultra-performance liquid chromatography

Human tissue

Location	Tissue	n	Geometric mean ng/g	Mean ± SD	Year	Analytical method	Comments	Reference
United States	liver	30	15.3; 95% CI: 13.0–22.5	18.8; 95% CI: 11.9–19.6	-	HPLC-ES-MS/MS	deceased donors; 15 <LOQ; 1/2 LOQ used for statistics when <LOQ	Olsen (2003*c*)
Spain	liver	12	22.3	26.6 ww ± 14.4	2008	UPLC-ES-MS/MS	6 males & 6 females, 27–79 yr old	Kärrman *et al.* (2010)

95% CI = 95% confidence interval
ES = electrospray
HPLC = high-performance liquid chromatography
LOQ = limit of quantification
MS = mass spectrometry
SD = standard deviation
UPLC = ultra-performance liquid chromatography
ww = wet weight

APPENDIX B. LITERATURE SEARCH STRATEGY FOR PERFLUOROOCTANE SULFONATE SOIL TOXICITY DATA

A search for relevant terrestrial and wildlife ecotoxicity data for perfluorooctane sulfonate (PFOS) was performed in February of 2012 following a tiered approach. First, terrestrial data from Environment Canada's final screening assessment report (EC 2006*a*) and its supporting document (EC 2006*b*), the UK Environment Agency's environmental risk evaluation report for PFOS (Brooke et al. 2004) and the UK Environment Agency report on PFOS (UK Environment Agency 2009) were collated. Further data were sought from the open literature from 2004 to 2012 using the resources described below (the starting point was before the last date of literature consulted for the screening assessment report, and was the last date of the literature search for the UK Environment Agency report).

Published literature data were also obtained from searches on the following databases: Scopus, Elton Bryson Stephens Company and US EPA EcoSSL (see US EPA n.d.).

Each database was searched with keywords related to PFOS and terrestrial ecotoxicity and secondary poisoning. Abstracts of articles were screened for potentially relevant articles. Full details of the search terms are found below.

All relevant studies were evaluated for acceptability for use by applying principles in CCME (2006). Any relevant references cited in the studies above were also obtained and examined.

Table B-1. Search terms used for literature searching

Chemical keywords	perfluorooctane sulphonate, perfluorooctane sulfonate, PFOS
Terrestrial ecotoxicity keywords	toxicity or terrestrial or soil or earthworm or worm or invertebrate or insect or arthropod or plant
Secondary poisoning keywords	Mammal or avian or bird or rat or mouse or dog or monkey or duck or mallard or bobwhite or quail or wildlife or livestock

APPENDIX C. SOIL-WATER AND SEDIMENT-WATER PARTITION COEFFICIENTS FOR PFOS

Soil type/soil source	Aqueous concentration of PFOS	Soil-water partition coefficient K_d (L/kg)	Organic carbon-water partition coefficient, K_{oc} (L/kg)	Log K_{oc}	Organic carbon fraction, f_{oc} (%)	pH	Reference
Jyndevad, Denmark: soil, agricultural topsoil, A horizon, sandy soil	0.2–1.0µg/L	15	1,500	3.18	1	6.1	Enevoldsen & Juhler (2010)
Sj. Odde, Denmark: soil, agricultural soil, A horizon, clayey soil	0.2–1.0 µg/L	17	4,048	3.61	0.425	7.6	Enevoldsen & Juhler (2010)
Minnesota aquifer material from landfill	1,000 µg/L	1.23	3,514	3.55	0.035	7.2	Ferrey *et al.* (2012)
Minnesota aquifer material from landfill at end of 740 d study	1,000 µg/L	0.08	229	2.36	0.035	8.1–8.8	Ferrey *et al*. (2012)
Clay	500 µg/L	18.3	704	2.85	2.6	7.2	3M (2001)
Clay loam	500 µg/L	9.72	374	2.57	2.6	6	3M (2001)
Sandy loam	500 µg/L	35.3	1,260	3.10	2.8	7.8	3M (2001)
Clay	1,000 µg/L approx.	18.3	610	2.8	3.0	NA	Johnson *et al*. (2007)
Clay loam	1,000 µg/L approx.	9.72	324	2.6	3.0	NA	Johnson *et al*. (2007)
Sandy loam	1,000 µg/L approx.	35.3	1,177	3.1	0.0	NA	Johnson *et al*. (2007)
Ottawa sand	1,000 µg/L approx.	2.81	NA	NA	0.0	NA	Johnson *et al*. (2007)
High-iron sand	1,000 µg/L approx.	8.9	NA	NA	3.0	NA	Johnson *et al.* (2007)
Kaolinite clay	NA	5.31	265.5	2.4	0.02–0.03	NA	Johnson *et al*. (2007)
River sediment	500 µg/L	7.42	571	2.76	1.3	7.7	3M (2001)
Marine sediment, S1, Dalian coastal area, China	10 µg/L	38.0	2,659	3.42	1.43	7.1–7.6	Chen *et al.* (2012*a*)
Marine sediment, S2, Dalian coastal area, China	10 µg/L	25.7	2,596	3.41	0.99	7.1–7.6	Chen *et al.* (2012*a*)
Marine sediment, S3, Dalian coastal area, China	10 µg/L	25.1	3,101	3.49	0.81	7.1–7.6	Chen *et al*. (2012*a*)
Marine sediment, S4, Dalian coastal area, China	10 µg/L	20.0	2,660	3.42	0.75	7.1 - 7.6	Chen *et al*. (2012*a*)

Soil type/soil source	Aqueous concentration of PFOS	Soil-water partition coefficient K_d (L/kg)	Organic carbon-water partition coefficient, K_{oc} (L/kg)	Log K_{oc}	Organic carbon fraction, f_{oc} (%)	pH	Reference
Marine sediment, S5, Dalian coastal area, China	10 µg/L	15.8	3,774	3.58	0.42	7.1–7.6	Chen *et al.* (2012*a*)
Soil from paddy field, Panjin, China	5 µg/L	12.3	1,349	3.13	0.91	NA	Chen *et al*. (2009)
5 freshwater sediments (rivers, lakes), USA	0.5–100 µg/L	16	372	2.57	4.3 average	5.7–7.5	Higgins & Luthy (2006)
Sandy river sediment, Kogaigawa, Japan	Up to 300 pmoles/L	1.5	5,012	3.7	0.03	8–8.3	Ahrens *et al.* (2011)
Muddy river sediment, Sakuragawa, Japan	Up to 300 pmoles/L	50.6	3,162	3.5	1.6	8–8.3	Ahrens *et al.* (2011)
Muddy marine sediment, Tokyo Bay, Japan	Up to 300 pmoles/L	27.6	2,512	3.4	1.1	8–8.3	Ahrens *et al.* (2011)
Marine sediment cores, Tokyo Bay, Japan	NA	126	6,310	3.8	1.5–1.7	8–8.3	Ahrens *et al.* (2010)
19 sediment samples, rivers, lakes & canals, The Netherlands	0.0047–0.32 µg/L	224	1,445	3.16	NA	NA	Kwadjik *et al*. (2010)
Sediment, Orge River, France (near Paris)	0.0174 µg/L	251	5,012	3.7	NA	NA	Labadie & Chevreuil (2011)
Lake Michigan sediment	1,000 µg/L approx.	7.52	376	2.4	0.00	NA.	Johnson *et al*. (2007)
River sediment	1,000 µg/L approx.	7.42	NA	2.8	NA	NA	Johnson *et al.* (2007)
Goethite (iron oxyhydroxide)	1,000 µg/L approx.	7.88	NA		NA	NA	Johnson *et al.* (2007)
Minimum to maximum		0.08–251	229–6,310	2.36–3.8			
Geometric mean		13.58	1,378	3.09			
Median		15.99	1,445	3.16			
Average (arithmetic mean)		34.70	2,112	3.32			
n =		30	26	27			

Source: Franz (2012, 2014).
NA = not available; cells with NA were not included in the calculation of GM, median & average K_{oc}.

APPENDIX D. BIOCONCENTRATION OF PFOS IN PLANTS

Soil-plant BCFs for PFOS in three crops (from Lechner and Knapp 2011)

Plant	Concentration of PFOS in soil (mg/kg soil)	Concentration of PFOS in plant (mg/kg)			BCF ((mg PFOS/kg plant material)/(mg PFOS/kg dw soil))			Harvest index (Proportion of plant compartment relative to whole harvest weight)[a]			Weighted BCF based on harvest index	Average weighted BCF for plant species	Average weighted BCF for plant species (dry/dry) (US EPA 1993)[b]
		peeled edible parts	peel	shoot	peeled edible parts	peel	shoot	peeled edible parts	peel	shoot			
Cucumber (*Cucumis sativus var. Pepinova*)	0.010	0.000075	NA	0.0012	0.008	NA	0.120	0.449	NA	0.551	0.07	0.088	0.59
	0.556	0.0013	NA	0.119	0.002	NA	0.214	0.482	NA	0.518	0.11		
Potato (*Solanum tuberosum*)	0.015	0.000075	0.0002	0.0041	0.005	0.013	0.273	0.589	0.091	0.32	0.09	0.12	0.82
	0.317	0.0007	0.015	0.141	0.002	0.047	0.445	0.556	0.083	0.361	0.17		
Carrot (*Daucus carota* subsp. *Sativus var. Flyaway*)	0.010	0.0005	0.0003	0.0032	0.050	0.030	0.320	0.436	0.144	0.420	0.16	0.19	1.24
	0.458	0.0184	0.0164	0.195	0.040	0.036	0.426	0.391	0.150	0.459	0.22		

NA = not analyzed

[a] Harvest indices for cucumber, potato and carrot are based on data provided by Lechner and Knapp (2011).

[b] Conversion to dw basis = (ww plant/dw soil) × 6.67, following US EPA (1993).

Soil-plant BCFs for PFOS in 3 plant species grown in sludge-amended soil (from Yoo *et al*. 2011)

Plant	Concentration in soil (ng/g dry soil)	Concentration in plant (ng/g dry plant)	Calculated soil-plant BCF (dry/dry basis)
Tall fescue (*Festuca arundinacea*)	35	1.2	0.034
Tall fescue	158	20.4	0.129
Tall fescue	203	13.1	0.065
Tall fescue (GM)			0.066
Bermuda grass (*Cynodon dactylon*)	118	4.1	0.035
Kentucky bluegrass (*Poa pratensis*)	203	16.8	0.083

Note: No harvest index was applied to these plant species, since Yoo *et al* (2011) did not report relative accumulation in grain and straw separately.

Soil to plant BCFs for PFOS in five crops (from Stahl *et al*. 2009)

Concentration of PFOS in soil (mg/kg soil)		0.25	1	10	25	50	Average BCF
		BCF (mg/kg plant dw/mg/kg soil)[a]					
Ryegrass (*Lolium perenne*)	1st cutting	0.044	0.119	0.0759	0.161	0.234	0.538[b,c]
	2nd cutting	0.188	0.200	0.653	0.827	0.601	
	3rd cutting	−0.076	0.032	0.256	0.408	0.364	
	4th cutting	−0.008	0.380	4.19	1.56	0.558	
Wheat (*Triticum spp.*)	Straw	0.120	0.250	0.993	0.863	1.54	0.75
	grain	0.000	0.000	0.002	0.002	0.007	0.0002
Oat (*Avena sativa*)	straw	0.040	0.104	0.265	0.762	0.827	0.40
	grain	0.000	0.015	0.012	0.004	0.002	0.007
Maize (*Zea mays*)	straw	0.120	0.101	0.206	0.202	0.158	0.157
	ear	0.002	0.003	0.004	0.008	0.006	0.005
Potato (*Solanum tuberosum*)	tuber	0.000	0.000	0.0006	0.0006	0.0007	0.0004
	peels	0.0180	0.012	0.0085	0.0068	0.118	0.015
Average BCF by test concentration		0.04	0.10	0.56	0.40	0.37	0.293

[a] Study provided concentration of PFOS in plants on dry weight basis for maize, oat, wheat and ryegrass and on wet weight basis for potato.

[b] GM of all the average values for each cutting.

[c] When the BCF value (4.19) for ryegrass (4th cutting at 10 mg PFOS/kg soil) is considered an outlier and removed from the calculations, then the BCF for ryegrass would range from negligible to 1.56; the average BCF for the 4th cutting of ryegrass would be 0.246, and the overall grand mean BCF for ryegrass would be 0.285. Since the BCF for ryegrass would continue to lie within the range for the other crops, the values derived from the full data set were maintained.

Soil to plant BCFs for PFOS in seven crops (from Brignole *et al*. 2003 in Beach *et al*. 2006).

Plant	Tissue	Soil PFOS concentration (mg/kg dw)				BCF (dry plant/dry soil basis)	
		3.6	**11.1**	**50.8**	**278**	**Range**	**Average BCF across available test concentrations**
Onion (*Allium cepa*) (67 days)	vegetative	NR	0.95	NR	NR	0.95	0.95
	fruit	0.87	2.0	NR	NR	0.87–2.0	1.44
	average						1.20
Ryegrass (*Lolium perenne*) (205 days)	vegetative	2.3	2.8	0.96	0.24	0.24–2.8	1.58
Alfalfa (*Medicago sativa*) (141 days)	vegetative	1.7	0.38	0.22	0.06	0.06–1.7	0.59
Flax (*Linum usitatissimum*) (94 days)	vegetative	1.4	1.69	1.1	NR	1.1–1.69	1.40
	fruit	0.06	0.12	0.05	NR	0.05–0.12	0.077
	average						0.74
Lettuce (*Lactuca sativa*) (67 days)	vegetative	2.4	0.95	0.83	NR	0.83–2.4	1.39
Soybean (*Glycine max*) (67 days)	vegetative	4.3	3.2	1.2	0.41	0.41–4.3	2.28
	fruit	0.39	0.08	0.02	0.01	0.01–0.39	0.13
	average						1.20
Tomato (*Lycopersicon esculentum*) (94 days)	vegetative	NR	3.05	0.99	NR	0.99–3.05	2.02
	fruit	NR	0.09	0.04	NR	0.04–0.09	0.065
	average						1.06

NR = not reported

APPENDIX E. TOXICITY DATA OF PFOS TO TERRESTRIAL PLANTS AND INVERTEBRATES ACCEPTABLE/SELECTED FOR USE FOR SOIL QUALITY GUIDELINE DERIVATION

Organism	Effect	Endpoint	Effect magnitude (%)	Effect concentration (mg/kg)	Soil pH	Soil OM %	Test substrate	Reference
Lettuce (*Lactuca sativa*)	**growth (plant height, 21 days)**	**LOEC**	**23**	**3.91**	**7.79**	**2.10**	artificial soil (sand 49%, silt 30%, clay 21%)	Brignole *et al.* (2003)
	growth (shoot weight)	LOEC	35	3.91				
	growth (plant height, 21 days)	IC_{25}	25	6.79				
	growth (shoot weight, 21 days)	**IC_{25}**	**25**	**8.92**				
	growth (shoot weight, 21 days)	IC_{50}	50	20.1				
	growth (plant height, 21 days)	IC_{50}	50	39.9				
	mortality (21 days)	NOEC	3	62.5				
	emergence (21 days)	NOEC	6	250				
	mortality (21 days)	**LOEC**	**23**	**250**				
		LC_{25}	25	257				
		LC_{50}	50	386				
	emergence (21 days)	**EC_{25}**	**25**	**393**				
		EC_{50}	50	564				
		LOEC	86	1,000				
Alfalfa (*Medicago sativa*)	**growth (shoot weight, 21 days)**	**IC_{25}**	**25**	**53.3**	7.79	2.10	artificial soil (sand 49%, silt 30%, clay 21%)	Brignole *et al.* (2003)
	growth (plant height, 21 days)	NOEC	6	62.5				
	growth (shoot weight, 21 days)	NOEC	11	62.5				

Organism	Effect	Endpoint	Effect magnitude (%)	Effect concentration (mg/kg)	Soil pH	Soil OM %	Test substrate	Reference
	mortality (21 days)	NOEC	9	62.5				
	growth (plant height, 21 days)	**IC_{25}**	**25**	**102**				
	growth (shoot weight, 21 days)	IC_{50}	50	146				
	growth (plant height, 21 days)	IC_{50}	50	249				
	emergence (21 days)	NOEC	19	250				
	mortality (21 days)	**LOEC**	**29**	**250**				
	growth (plant height, 21 days)	LOEC	57	250				
	growth (shoot weight, 21 days)	LOEC	78	250				
	mortality (21 days)	LC25	25	251				
	emergence (21 days)	**EC25**	**25**	**372**				
	mortality (21 days)	LC50	50	452				
	emergence (21 days)	EC50	50	745				
	emergence (21 days)	LOEC	64	1,000				
Flax (*Linum usitatissimum*)	growth (plant height, 21 days)	NOEC	8	62.5	7.79	2.10	artificial soil (sand 49%, silt 30%, clay 21%)	Brignole *et al*. (2003)
	growth (shoot weight, 21 days)	NOEC	18	62.5				
	Emergence	NOEC	0	62.5				
	mortality of emerged seedlings (21 days)	NOEC	−17	62.5				
	growth (shoot weight, 21 days)	**IC_{25}**	**25**	**81.6**				
	growth (plant height, 21 days)	**IC_{25}**	**25**	**97.6**				

Organism	Effect	Endpoint	Effect magnitude (%)	Effect concentration (mg/kg)	Soil pH	Soil OM %	Test substrate	Reference
	growth (shoot weight, 21 days)	IC_{50}	50	119				
	growth (plant height, 21 days)	IC_{50}	50	140				
	growth (plant height, 21 days)	LOEC	86	250				
	growth (shoot weight, 21 days)	LOEC	91	250				
	mortality of emerged seedlings (21 days)	LOEC	45	250				
	mortality of emerged seedlings (21 days)	LC_{25}	25	251				
	mortality of emerged seedlings (21 days)	LC_{50}	50	452				
	Emergence	LOEC	100	1,000				
Onion (*Allium cepa*)	growth (shoot weight, 21 days)	NOEC	15	3.91	7.79	2.10	artificial soil (sand 49%, silt 30%, clay 21%)	Brignole *et al*., (2003)
	shoot weight (21 days)	**IC_{25}**	**25**	**12.9**				
	growth (shoot weight, 21 days)	LOEC	31	15.6				
	growth (plant height, 21 days)	NOEC	10	15.6				
	mortality (21 days)	NOEC	6	15.6				
	growth (shoot weight, 21 days)	IC_{50}	50	28.1				
	growth (plant height, 21 days)	**IC_{25}**	**25**	**29.1**				
	growth (plant height, 21 days)	IC_{50}	50	46.5				
	mortality of emerged seedlings (21 days)	LC_{25}		47.1				

Organism	Effect	Endpoint	Effect magnitude (%)	Effect concentration (mg/kg)	Soil pH	Soil OM %	Test substrate	Reference
	emergence (21 days)	**EC_{25}**	**25**	**50.8**				
	mortality (21 days)	LC_{50}	50	57.3				
	mortality (21 days)	LOEC	62	62.5				
	growth (plant height, 21 days)	LOEC	68	62.5				
	emergence (21 days)	NOEC	19	62.5				
		EC_{50}	50	208				
		LOEC	53	250				
Ryegrass (*Lolium perenne*)	growth (plant height, 21 days)	NOEC	9	3.91	7.79	2.10	artificial soil (sand 49%, silt 30%, clay 21%)	Brignole *et al*. (2003)
	growth (shoot weight, 21 days)	NOEC	12	3.91				
	shoot weight (21 days)	**IC_{25}**	**25**	**7.51**				
	growth (plant height, 21 days)	LOEC	19	15.6				
	growth (shoot weight, 21 days)	LOEC	39	15.6				
	growth (plant height, 21 days)	**IC_{25}**	**25**	**46.3**				
	shoot weight (21 days)	IC_{50}	50	53.8				
	emergence (21 days)	NOEC	−6	62.5				
	mortality of emerged seedlings (21 days)	NOEC	−3	62.5				
	growth (plant height, 21 days)	IC_{50}	50	131				
	mortality of emerged seedlings (21 days)	LC_{25}	25	174				
	emergence (21 days)	**EC_{25}**	**25**	**203**				

Organism	Effect	Endpoint	Effect magnitude (%)	Effect concentration (mg/kg)	Soil pH	Soil OM %	Test substrate	Reference
	emergence (21 days)	LOEC	28	250				
	mortality (21 days)	**LOEC**	**34**	**250**				
	mortality of emerged seedlings (21 days)	LC_{50}	50	310				
	emergence (21 days)	EC_{50}	50	344				
Soybean (*Glycine max*)	growth (plant height, 21 days)	NOEC	−8	65.5	7.79	2.10	artificial soil (sand 49%, silt 30%, clay 21%)	Brignole *et al.* (2003)
	growth (shoot weight, 21 days)	NOEC	−10	62.5				
	growth (shoot weight, 21 days)	**IC**	**25**	**160**				
	growth (plant height, 21 days)	LOEC	21	250				
	growth (shoot weight, 21 days)	LOEC	43	250				
	growth (plant height, 21 days)	**IC_{25}**	**25**	**284**				
	growth (shoot weight, 21 days)	IC_{50}	50	326				
	growth (plant height, 21 days)	IC_{50}	50	464				
	emergence (21 days)	NOEC	0	1,000				
	mortality (21 days)	NOEC	0	1,000				
Tomato (*Lycopersicon esculentum*)	**growth (shoot weight, 21 days)**	**IC_{25}**	**25**	**11.7**	7.79	2.10	artificial soil (sand 49%, silt 30%, clay 21%)	Brignole *et al.* (2003)
	growth (plant height, 21 days)	NOEC	9	15.6				
	growth (shoot weight, 21 days)	NOEC	19	15.6				

Organism	Effect	Endpoint	Effect magnitude (%)	Effect concentration (mg/kg)	Soil pH	Soil OM %	Test substrate	Reference
	mortality (21 days)	NOEC	14	15.6				
	growth (plant height, 21 days)	**IC_{25}**	**25**	**22.1**				
	growth (shoot weight, 21 days)	IC_{50}	50	28.5				
	mortality (21 days)	**LOEC**	**27**	**62.5**				
	growth (plant height, 21 days)	LOEC	50	62.5				
	growth (shoot weight, 21 days)	LOEC	79	62.5				
	mortality (21 days)	LC_{25}	25	68.7				
	growth (plant height, 21 days)	IC_{50}	50	93.9				
	mortality (21 days)	LC_{50}	50	105				
	emergence (21 days)	NOEC	22	250				
	emergence (21 days)	**EC_{25}**	**25**	**311**				
	emergence (21 days)	EC_{50}	50	474				
	Emergence (21 days)	LOEC	89	1,000				
Pak choi (*Brassica chinensis*)	growth (root elongation, 7 days)	IC_{10}	10	40	4.73	0.50	Jiangxi soil (sand 53.85%, silt 18.04%, clay 8.12%)	Zhao *et al.* (2011)
		NOEC	NR	50				
		IC_{50}	50	95				
	growth (root elongation, 7 days)	NOEC	NR	50	5.43	0.70	Hainan soil (sand 38.2%, silt 26.9%, clay 34.9%)	
		IC_{10}	10	58				
		IC_{50}	50	107				

Organism	Effect	Endpoint	Effect magnitude (%)	Effect concentration (mg/kg)	Soil pH	Soil OM %	Test substrate	Reference
	growth (root elongation, 7 days)	NOEC	NR	50	6.60	0.97	Jiangsu soil (sand 57.4%, silt 28%, clay 14.6%)	
		IC_{10}	10	72				
		IC_{50}	50	122				
	growth (root elongation, 7 days)	IC_{10}	10	83	7.69	1.05	Sichuan soil (sand 47.6%, silt 37.4%, clay 14.9%)	
		NOEC	NR	100				
		IC_{50}	50	119				
	growth (root elongation, 7 days)	IC_{10}	10	90	7.40	2.00	Liaoning soil (sand 61.4%, silt 25.0%, clay 13.6%)	
		NOEC	NR	150				
		IC_{50}	50	178				
Earthworm (*Eisenia fetida*)	**number of cocoons (28 days)**	NOEC	NR	40	5.5–6.5	10	artificial soil (sand 70%, silt 10%, clay 20%)	Stubberud (2006)
		IC_{10}	10	43				
		IC_{25}	**25**	**67**				
		LOEC	NR	80				
		IC_{50}	50	103				
		NOEC	NR	40				
		NOEC	NR	10				
	average weight per juvenile (56 days)							
		IC_{25}	**25**	**12**				
		LOEC	NR	20				
		IC_{50}	50	131				
		IC_{25}	25	<10				

Organism	Effect	Endpoint	Effect magnitude (%)	Effect concentration (mg/kg)	Soil pH	Soil OM %	Test substrate	Reference
	total weight of juveniles (56 days)							
		NOEC	NR	20				
		IC_{50}	50	29				
		LOEC	**NR**	**40**				
		IC_{10}	10	25				
	number of juveniles (56 days)							
		NOEC	NR	40				
		IC_{25}	**25**	**48**				
		IC_{50}	50	80				
		LOEC	NR	80				
Earthworm (*Eisenia fetida*)	mortality (7 days)	NOEC	NR	160	6.26–6.41	10	artificial soil (sand 70%, silt 10%, clay 20%)	Joung *et al.* (2010)
	mortality (14 days)	**LC_{20}**	**20**	**256**				
	mortality (14 days)	LC_{50}	50	365.4				
	mortality (7 days)	LC_{50}	50	405.3				
	body weight (14 days)	NOEC	NR	410				
Earthworm (*Eisenia fetida*)	growth (14 days)	NOEC	7.5	289	6.00	NR	artificial soil (sand 70%, silt 10%, clay 20%)	Sindermann *et al.* (2002)
	mortality (14 days)	LC_{50}	7.5	373	6.00			
Earthworm (*Eisenia fetida*)	mortality (14 days)	LC_{50}	50	542.1	6.50		natural soil (sand 31.3%, silt 46.3%, clay 22.4%)	Xu *et al.* (2011)
	mortality (14 days)	LC_{50}	50	955.3			artificial soil (sand 70%, silt 10%, clay 20%)	
Earthworm (*Eisenia fetida*)	mortality (14 days)	LC_{50}	50	515.4	6-6.5	various	various	Joung *et al*. (2010), Sindermann *et al*. (2002) &

Organism	Effect	Endpoint	Effect magnitude (%)	Effect concentration (mg/kg)	Soil pH	Soil OM %	Test substrate	Reference
								Xu *et al.* (2011)
Springtail (*Folsomia candida*)	**number of live juveniles produced (28 days)**	**IC_{25}**	**25**	**61 (41–91)**	6	2.6	coarse soil (sand 75.2%, silt 16.2%, clay 8.6%)	EC (2015)
Springtail (*Folsomia candida*)	number of live juveniles produced (28 days)	IC_{50}	50	78 (59–103)	6	2.6	coarse soil (sand 75.2%, silt 16.2%, clay 8.6%)	EC (2015)
Springtail (*Folsomia candida*)	**number of live juveniles produced (28 days)**	**IC_{25}**	**25**	**177 (114–275)**	6.8	15.2	fine soil (sand 36.1%, silt 28.3%, clay 31.9%)	EC (2015)
Springtail (*Folsomia candida*)	number of live juveniles produced (28 days)	IC_{50}	50	227 (168–307)	6.8	15.2	fine soil (sand 36.1%, silt 28.3%, clay 31.9%)	EC (2015)
Springtail (*Folsomia candida*)	mortality (28 day)	LC_{50}	50	111 (87–142)	6	2.6	coarse soil (sand 75.2%, silt 16.2%, clay 8.6%)	EC (2015)
Springtail (*Folsomia candida*)	mortality (28 day)	LC_{50}	50	>350	6.8	15.2	fine soil (sand 36.1%, silt 28.3%, clay 31.9%)	EC (2015)
Mite (*Oppia nitens*)	**number of live juveniles produced (28 days)**	**IC_{25}**	**25**	**13 (8–21)**	6	2.6	coarse soil (sand 75.2%, silt 16.2%, clay 8.6%)	EC (2015)
Mite (*Oppia nitens*)	number of live juveniles produced (28 days)	IC_{50}	50	23(18–31)	6	2.6	coarse soil (sand 75.2%, silt 16.2%, clay 8.6%)	EC (2015)
Mite (*Oppia nitens*)	**number of live juveniles produced (28 days)**	**IC_{25}**	**25**	**33(19–59)**	6.8	15.2	fine soil (sand 36.1%, silt 28.3%, clay 31.9%)	EC (2015)
Mite (*Oppia nitens*)	number of live juveniles produced (28 days)	IC_{50}	50	96 (66–139)	6.8	15.2	fine soil (sand 36.1%, silt 28.3%, clay 31.9%)	EC (2015)

Organism	Effect	Endpoint	Effect magnitude (%)	Effect concentration (mg/kg)	Soil pH	Soil OM %	Test substrate	Reference
Mite (*Oppia nitens*)	mortality (28 day)	LC_{50}	50	63 (57–70)	6	2.6	coarse soil (sand 75.2%, silt 16.2%, clay 8.6%)	EC (2015)
Mite (*Oppia nitens*)	mortality (28 day)	LC_{50}	50	>180	6.8	15.2	fine soil (sand 36.1%, silt 28.3%, clay 31.9%)	EC (2015)

Note: Values highlighted in **bold** indicate EC_{25}, IC_{25} or LC_{20} values used in the species sensitivity distribution used to calculate the TEC and the ECL.
OM = organic matter

APPENDIX F. TOXICITY DATA OF PFOS TO TERRESTRIAL PLANTS AND INVERTEBRATES CONSULTED BUT NOT USED FOR SOIL QUALITY GUIDELINE DERIVATION

Organism	Effect	Endpoint	Effect concentration (mg/kg, unless otherwise stated)	Soil pH	Soil OM (%)	Test substrate	Comments	Reference
Lettuce (*Lactuca sativa*)	height (21 days)	NOEC	<3.91	7.2	2.1	Artificial soil: sand 49%, silt 30%, clay 21%	No numeric NOEC value. Protocol advises not to use <X for NOEC. LOEC was lowest concentration tested.	Brignole *et al*. (2003)
	shoot weight (21 days)	NOEC	<3.91					
Flax (*Linum usitatissimum*)	emergence (21 days)	EC_{25}	372				No or poor dose-response relationship.	
		EC_{50}	745				No or poor dose-response relationship.	
Soybean (*Glycine max*)	emergence	NOEC	>1,000				No effect at max test concentration, therefore no corresponding LOEC. Protocol guidance is to use paired NOEC/LOEC, therefore not considered acceptable for guideline derivation.	
Pak choi (*Brassica chinensis*)	root elongation (7 days)	NOEC	150			Soil (Heilongjiang)	80% organic matter in soil; not considered acceptable for generic guideline derivation.	Zhao *et al*. (2011)
		IC_{10}	115	5.5–6.5	10			
		IC_{50}	>200					
Earthworm (*Eisenia fetida*)	average weight per juvenile (56 days)	IC_{10}	0.9			Artificial soil: 70% sand, 20% clay, 10% OM	Authors recommended not using EC_{10}, as was extrapolated beyond tested range.	Stubberud (2006)
	total weight of juveniles (56 days)	IC_{10}	4					

Organism	Effect	Endpoint	Effect concentration (mg/kg, unless otherwise stated)	Soil pH	Soil OM (%)	Test substrate	Comments	Reference
Earthworm (*Eisenia fetida*)	mortality (14 days)	NOEC	77			Artificial soil	Study implied measured effect on burrowing behaviour, clinical signs of toxicity & body weight, but only reported burrowing that was not considered an acceptable endpoint.	Sindermann *et al.* (2003)
		LOEC	141	6.2–6.4			Reported lethality LOEC = 141 mg/kg (7.5% mortality), but tables show body weight LOEC = 488 mg/kg (75% mortality), which was considered too high for a LOEC.	
Earthworm (*Eisenia fetida*)	mortality (7 days) (14 days)	NOEC NOEC	160 160			artificial soil 10% OM, 10% clay, 70% sand	The NOEC from the longer exposure study (14-day) was used.	Joung *et al.* (2010)
Earthworm (*Eisenia fetida*)	effect not reported (7 days)	NOEC	289			Artificial soil	Effect was not reported.	3M (2002), cited in Brooke *et al*. (2004)
	mortality (14 days)	LC_{50}	398					
Non-soil test substrate								
Lettuce (*Lactuca sativa*)	root length (120 hours)	IC_{10}	24 mg/L			filter paper		Li (2008)
		IC_{50}	99					
		NOEC	50					
	seed germination (120 hours)	IC_{50}	>200					
		NOEC	>200					

Organism	Effect	Endpoint	Effect concentration (mg/kg, unless otherwise stated)	Soil pH	Soil OM (%)	Test substrate	Comments	Reference
Pak choi (*Brassica rapa chinensis*)	root length (120 hours)	IC_{10}	71 mg/L			filter paper		
		IC_{50}	130					
		NOEC	50					
	seed germination (120 hours)	LC_{50}	>200					
		LOEC	>200					
Cucumber (*Cucumis sativus*)	root length (120 hours)	IC_{10}	not calculated			filter paper		
		EC_{50}	>200 mg/L					
		NOEC	>200					
	seed germination (120 hours)	LC_{50}	>200					
		NOEC	>200					
Wheat (*Triticum aestivum* L.)	decreased total chlorophyll concentration (7 days)	NOEC	10 mg/L			solution		Qu *et al.* (2010)
		EC_{30}	100					
	decreased protein content in leaf (7 days)	NOEC	10					
		EC_{20}	100					
		NOEC	100					
		EC_{20}	200					
	root length (7 days)	NOEC	10					
		EC_{15}	200					

Organism	Effect	Endpoint	Effect concentration (mg/kg, unless otherwise stated)	Soil pH	Soil OM (%)	Test substrate	Comments	Reference
	dry weight root (7 days)	NOEC	10					
		EC_{15}	200					
Earthworm (not specified)	mortality (14 days)	LC_{50}	13.64 µL/cm2			filter paper		Xu *et al.* (2011)
Nematode (*Caenorhabditis elegans*)	fecundity & reproduction	EC_{50}	10 pM–100 nM range			agar nematode growth medium	No dose-response relationship.	Tominaga *et al*. (2004)
Fruit fly (*Drosophila hydei*)	number of offspring (4 days)	IC_{50}	5 ng/mL			sugar water		Van Gossum *et al.* (2010)
		IC_{100}	5,000					
Bumblebee (*Bombus terrestris* L.)	mortality (14 days)	LC_{100}	5 mg/L			sugar water		Mommaerts *et al.* (2011)
		LC_{50}	2					
	mortality (11 weeks)	LC_{50}	1.01					
	ovary length (11 weeks)	IC_{50}	1					
	egg degeneration (11 weeks)	IC_{100}	1					
	accumulation in body (5 weeks)	accumulation in worker bees	100				2,184 ng/g accumulated in worker bee (BAF = 27.9)	
	decreased mitochondrial electron transport activity (5 weeks)	IC_{25}	1					

Organism	Effect	Endpoint	Effect concentration (mg/kg, unless otherwise stated)	Soil pH	Soil OM (%)	Test substrate	Comments	Reference
Honey bee (*Apis mellifera*)	mortality (72 hours)	LD_{50}	0.4 µg/bee			oral test, not soil		OECD (2002)
	effect not reported (72 hours)	NOEC	0.21					
	mortality (96 hours)	LD_{50}	4.78			contact test		
	effect not reported (96 hours)	NOEL	1.93					

OM = organic matter

APPENDIX G. ACCEPTABLE/SELECTED MAMMALIAN AND AVIAN TOXICITY DATA FOR PFOS

Organism	Effect	Exposure duration	Exposure route	Endpoint	Concentration or dose	Ranking	Reference
Rat (Sprague Dawley)	development toxicity		gavage	NOAEL	1 mg/kg/day diet	selected	OECD (2002)
	maternal toxicity considered to be treatment-related, consisted of hunched posture, anorexia, bloody vaginal discharge, uterine stains, alopecia, rough haircoat	GD 6–15	gavage	NOAEL	1 mg/kg/day diet	selected	
	maternal toxicity-reduced body weight	GD 6–15	gavage	NOAEL	5 mg/kg/day diet	selected	
	eye abnormalities, development toxicity		gavage	LOAEL	1 mg/kg/day diet	selected	
	decreased maternal body weight, food consumption, uterine weight & increased gastrointestinal lesions, decreased fetal weight		gavage	LOAEL	5 mg/kg/day diet	selected	
	decreased fetal body weight, increased external & visceral anomalies & variations, development toxicity		gavage	LOAEL	5 mg/kg/day diet	selected	
	mean body weight reduced, maternal body weight		gavage	LOAEL	10 mg/kg/day diet	selected	
	maternal toxicity (F0)	6 weeks; prior to & during mating	gavage	NOAEL	0.1 mg/kg/day feed	selected	
	development toxicity (F1)		gavage	NOAEL	0.1 mg/kg/day feed	selected	
	F1 generation offspring		gavage	NOAEL	0.1 mg/kg/day feed	selected	
	developmental toxicity (F2)	9.5 weeks for F1	gavage	NOAEL	0.1 mg/kg/day feed	selected	
	reduced body weight & food consumption, maternal toxicity (F0)		gavage	LOAEL	0.4 or 1.6 mg/kg/day feed	selected	
	reduction in implantation sites, litter size, pup viability, growth & survival (F1); 26% of pups found dead		gavage	LOAEL	1.6 mg/kg/day feed	selected	
	reductions in pup growth &statistically significant mean pup weights, development toxicity (F2)		gavage	LOAEL	0.4 mg/kg/day feed	selected	OECD (2002)

Organism	Effect	Exposure duration	Exposure route	Endpoint	Concentration or dose	Ranking	Reference
							(Christian *et al*. 1999)
	loss of mean body weight in females at 10 mg/kg PFOS; greater relative liver weight when compared to control	28 days	gavage	NOAEL	1.25 mg/kg/day	selected	Kim *et al*. (2011*a*)
	reduced body weight in females; relative liver weight much higher for 10 mg/kg than the control	28 days	gavage	LOAEL	10 mg/kg/day	selected	
Rat (CD)	reduced liver weight	90 days	diet	LOAEL	2 mg/kg/day diet	selected	OECD (2002)
	decreased body weight	90 days	diet	EC_8	2 mg/kg/day (mean body weight reduced by 8.7 & 8%)	selected	
	mortality	90 days	diet	LC_{100}	18 mg/kg/day	selected	
Rat (male Wistar)	increased liver weight; increased relative brain weight; decreased food consumption & body weight	13 weeks; effects seen at 2 weeks	diet: *ad libitum*	LOAEL	32 mg/kg diet	selected	Kawamoto *et al.* (2011)
	reduced pup body weight (2nd-generation offspring)		gavage	NOAEL LOAEL	1 mg/kg/day 0.4 mg/kg/day	selected	Seed (2000)
Rat[a]	**anatomic pathology findings in the liver (hepatocellular degeneration)**	**104 weeks**	**diet**	**LOAEL**	**2 ppm (0.1086 mg/kg bw/day. Based on mean achieved dose level for M & F at 2 ppm diet). Used to calculate TDI for federal wildlife diet guideline to protect mammals. Range of mean achieved doses: M: 0.064–0.226; F: 0.073–0.213**	**selected**	**Covance Labs Inc. (2002); Thomford (2000)**

Organism	Effect	Exposure duration	Exposure route	Endpoint	Concentration or dose	Ranking	Reference
Mice (Adult female B6C3F1)	decreased uterus weight	28 days	oral		5 mg/kg dose	selected	Fair *et al*. (2011)
White rabbits (New Zealand; *Oryctolagus cuniculus*)	maternal toxicity	GD 7–20	gavage	NOAEL	0.1 mg/kg/day diet	selected	OECD (2002)
	development toxicity		gavage	NOAEL	1 mg/kg/day diet	selected	
	abortions & decreased body weight gains & food consumption, maternal body weight, maternal toxicity		gavage	LOAEL	1 mg/kg/day diet	selected	
	decreased fetal body weight & increased fetal alterations, development toxicity		gavage	LOAEL	2.5 mg/kg/day diet	selected	
Mallard duck (*Anas platyrhynchos*)	body weight	21 weeks	diet	NOAEL	10 mg/kg feed	selected	Newsted *et al.* (2005)
	body weight, reproduction	21 weeks	diet	LOAEL	50 mg/kg feed	selected	
	signs of toxicity (one hen), body weight (male & females), histopathology (testicular regression, adipose tissue microvesiculation for adult males)	6 weeks	diet	NOEC	6.2 mg/kg ww diet, in feed	selected	Gallagher *et al*. (2003*b*)
	mortality (adult)	20 weeks	diet	NOAEC	≥7.6 mg/kg a.i. – ww diet, in feed	selected	
	body weight (adult female)	20 weeks	diet	NOAEC	≥17.6 mg/kg a.i. – ww diet, in feed	selected	
	reproduction (adult)	20 weeks	diet	NOAEC	≥17.6 mg/kg a.i. – ww diet, in feed	selected	
	14-day survivability (offspring)	20 weeks (parent)	diet	NOAEC	≥17.6 mg/kg a.i. – ww diet, in feed	selected	
	hatchling/juvenile body weight (offspring)	20 weeks (parent)	diet	NOAEC	≥17.6 mg/kg a.i. – ww diet, in feed	selected	
	body weight	6–20 weeks	diet	NOEL LOEL	6.2 mg/kg feed 17.6 mg/kg feed	selected	
	signs of toxicity (one hen), body weight (male & females), histopathology (testicular regression, adipose tissue microvesiculation for adult males)	20 weeks	diet	LOEC	17.6 mg/kg a.i. – ww diet, in feed	selected	
	reduced body weight	8 days	diet	NOAEL LOAEL	35.1 mg/kg feed 70.3 mg/kg feed	selected (short-term)	Newsted *et al*. (2005)

Organism	Effect	Exposure duration	Exposure route	Endpoint	Concentration or dose	Ranking	Reference
	mortality based on ADI	8 days	diet	LD_{50}	150 mg/kg feed	selected (short-term)	
	mortality	8 days	diet	LC_{50}	603 mg/kg feed	selected (short-term)	
	body weight, feed consumption		diet	NOEC	36.6 mg/kg feed	selected (short-term)	OECD (2002)
	mortality	post-exposure observation 3–17 days	diet	NOEC	146 mg/kg feed	selected (short-term)	
	mortality	5 days	diet	LC_{50}	628 mg/kg feed (CI=448-958)	selected (short-term)	
Northern bobwhite quail (*Colinus virginianus*)	body weight, no treatment-related effects on reproduction or liver weight for males.	21 weeks	diet	NOAEL	10 mg/kg feed	selected	Newsted *et al.* (2005)
Northern bobwhite quail[b]	**reduced survivability of 14-day-old chicks**	**21 weeks**	**diet**	**LOAEL**	**10 mg/kg feed (0.772 mg/kg bw/day)**	**selected**	**Newsted *et al*. (2007)**
Northern bobwhite quail	body weight (adult)		diet	NOAEL	0.579 mg/kg bw/day	selected	Gallagher *et al*. (2003*a*)
	feed intake		diet	NOAEL	0.579 mg/kg bw/day	selected	
	body weight (adult male) (adult female)		diet	NOAEC NOAEC	6.2 mg/kg ww diet, in feed ≥17.6 mg/kg ww diet, in feed	selected	
	mortality (adult)	20 weeks	diet	NOAEC	17.6 mg/kg ww diet, in feed	selected	
	reproduction (adult)	20 weeks	diet	NOAEC	≥17.6 mg/kg ww diet, in feed	selected	
	14-day survivability (offspring)	20 weeks	diet	NOAEC	≥17.6 mg/kg ww diet, in feed	selected	
	body weight (adult male)	20 weeks	diet	LOAEC	17.6 mg/kg ww diet, in feed	selected	
	body weight (adult)		diet	LOAEL	2 mg/kg/ bw/day	selected	

Organism	Effect	Exposure duration	Exposure route	Endpoint	Concentration or dose	Ranking	Reference
	feed intake		diet	LOAEL	2 mg/kg bw/day	selected	
	mortality, signs of toxicity, body weight, feed consumption, reproductive phase		diet	NOEC	<10 mg/kg diet	selected	
	mortality (adult)	21 weeks	diet	NOAEC	10 mg/kg ww diet, in feed	selected	
	body weight (adult)	21 weeks	diet	NOAEC	10 mg/kg ww diet, in feed	selected	
	reproductive (adult)	21 weeks	diet	LOAEC	10 mg/kg ww diet, in feed	selected	
	14-day survivability (offspring)	21 weeks	diet	LOAEC	10 mg/kg ww diet, in feed	selected	
	mortality (adult)	7 weeks	diet	LOAEC	50 mg/kg ww diet, in feed	selected	
	body weight (adult)	7 weeks	diet	LOAEC	50 mg/kg ww diet, in feed	selected	
	mortality, body weight	post-exposure observation 3–17 days	diet	NOEC	73.2 mg/kg feed	selected (short-term)	OECD (2002)
	mortality	5 days	diet	LC_{50}	220 mg/kg feed	selected (short-term)	
	11% mortality, reduced body weight		diet	LOEC	146 mg/kg feed	selected (short-term)	
	mortality	8 days	diet	LC_{50}	212 mg/kg feed	selected (short-term)	Newsted *et al*. (2005)
	statistically significant reduction in body weight	8 days	diet	LOAEL	141 mg/kg feed	selected (short-term)	
	mortality, body weight (decrease), no apparent toxic effects	8 days	diet	NOAEL	70.3 mg/kg feed	selected (short-term)	
	mortality based on ADI	8 days	diet	LD_{50}	61 mg/kg feed	selected (short-term)	
Consulted (not considered suitable for guideline derivation)							

Organism	Effect	Exposure duration	Exposure route	Endpoint	Concentration or dose	Ranking	Reference
Cynomolgus monkey (*Macaca fascicularis*)	death, liver effects, effect on cholesterol	26 weeks + 52 weeks recovery period	oral capsule	NOEL	0.15 mg/kg/day diet	consulted	Seacat *et al.* (2002)
	death, liver effects, effect on cholesterol	26 weeks + 52 weeks recovery period	oral capsule	LOAEL	0.75 mg/kg/day	consulted	
Female Wistar rats (180–200 g bw)	liver weight	5 days	gavage	12% increase	3.0 mg/kg bw	consulted	Yu *et al.* (2011)
Rhesus monkey (*Macaca mulatta*)	gastrointestinal tract	90 days	gavage	LOAEL	0.5 mg/kg/day diet	consulted	OECD (2002)
	Mortality	90 days (all died between weeks 5 & 7)	gavage	LC_{100}	4.5 mg/kg/day	consulted	
Rat (Sprague Dawley)	development toxicity	On GD 6–15	gavage	NOAEL	<1 mg/kg/day diet	consulted	
	tumours found in liver & thyroid gland (males)	14 weeks	gavage	NOAEL LOAEL	0.5 mg/kg 2 mg/kg	consulted	Seacat *et al.* (2003)
	tumours found in liver & thyroid gland (females)	14 weeks	gavage	NOAEL LOAEL	2 mg/kg 5 mg/kg	consulted	
	(F2 generation offspring) pup mortality & decrease weight gain	42 days prior to mating to GD 20	gavage	NOAEL	0.4 mg/kg/day diet	consulted	Luebker *et al.* (2005*a*)
Rat	reduced survival & body weight gain in neonatal rats	42 days prior to mating to GD 20	gavage	LOAEL	1.6 mg/kg/day diet	consulted	
	gestation length	42 days prior to mating to GD 20	gavage		0.8 mg/kg diet	consulted	
	decreased viability	42 days prior to mating to GD20	Gavage		1.6 mg/kg diet	Consulted	
	maternal body weight at term	on GD 2–20	gavage (1 mL/kg-day)	BMD_5 $BMDL_5$	0.224 mg/kg 0.150 mg/kg	consulted	Thibodeaux *et al.* (2003)
	fetal sternal defects	on GD 2–20	gavage (1 mL/kg-day)	BMD_5 $BMDL_5$	0.313 mg/kg 0.122 mg/kg	consulted	
	fetal cleft palate	on GD 2–20	gavage (1 mL/kg-day)	BMD_5 $BMDL_5$	8.85 mg/kg 3.33 mg/kg	consulted	

Organism	Effect	Exposure duration	Exposure route	Endpoint	Concentration or dose	Ranking	Reference
Mouse	maternal body weight at term	on GD 2–20	gavage (1 mL/kg-day)	BMD_5 $BMDL_5$	15.5 mg/kg 3.14 mg/kg	consulted	
	fetal sternal defects	on GD 2–20	gavage (1 mL/kg-day)	BMD_5 $BMDL_5$	0.055 mg/kg 0.016 mg/kg	consulted	
	fetal cleft palate	on GD 2–20	gavage (1 mL/kg-day)	BMD_5 $BMDL_5$	7.03mg/kg 3.53 mg/kg	consulted	
Rat (pregnant Sprague Dawley)	postnatal survival	postnatal day 8	gavage (parent) (1 mL/kg-day)	BMD_5 $BMDL_5$	1.07 mg/kg 0.58 mg/kg	consulted	Lau *et al*. (2003)
Mouse	postnatal survival	postnatal day 7	gavage (1 mL/kg-day)	BMD_5 $BMDL_5$	7.02 mg/kg 3.88 mg/kg	consulted	
Rat (Sprague Dawley)	fatty change in liver of male rats	28 days	gavage	LOAEL	5 mg/kg/day	consulted	Kim *et al.* (2011*a*)
Cynomolgus monkey	clinical & pathological findings	26 weeks	gelatin capsule	NOAEL	0.03 mg/kg/day	consulted	Thomford (2000)
Chicken *(Gallus gallus domesticus)*	hatchability	1 injection	injection of 0.1 µL treatment	LOAEL LC_{50}	100 ng/g egg (= 0.1 µg/g) 4.9 µg/g egg	consulted	Molina *et al.* (2006)
White leghorn chicken	hatching success	Post-hatch 14 days; length of study 5 weeks	injection *in ovo*	NOEL	4.6 µg/g egg [PFOS ion]	consulted	Peden-Adams *et al*. (2009)
	egg viability	post-hatch 14 days; length of study 5 weeks	injection *in ovo*	NOEL	4.6 µg/g egg [PFOS ion]	consulted	
	gross pathology	post-hatch 14 days; length of study 5 weeks	injection *in ovo*	NOEL LOEL	<0.93 µg/g egg 0.93 µg/g egg	consulted	
	pippability	3 weeks	injection *in ovo*	NOEL LOEL	5 µg/g egg 100 µg/g egg	consulted	O'Brien *et al*. (2009)
	increased hepatic concentration	3 weeks	injection *in ovo*	NOEL LOEL	<0.1 µg/g egg 0.1 µg/g egg	consulted	

Selected = considered acceptable for use in guideline derivation
Consulted = reviewed but considered unsuitable for guideline derivation

a.i. = active ingredient
GD = gestation day

LOEL = lowest-observed-effect level NOEC = no-observed-effect concentration
NOEL = no-observed-effect level
[a] Bolded entry for rat is the lowest-observed-adverse-effects dose (ED_{1C}) for mammals and was used as the basis for the $SoQG_I$ for mammals.
[b] Bolded entry for Northern bobwhite quail is the lowest-observed-adverse-effects dose (ED_{1C}) for avian species and was used as the basis for the $SoQG_I$ for avian species.

APPENDIX H. EC_{25}, IC_{25} AND LC_{20} DATA USED FOR SPECIES SENSITIVITY DISTRIBUTION USED TO DERIVE SOIL CONTACT VALUE FOR AGRICULTURAL, RESIDENTIAL/PARKLAND AND COMMERCIAL AND INDUSTRIAL LAND USES FOR PFOS

Species name	Exposure duration (days)	Exposure concentration (mg/kg)	Endpoint	Effect	Concentration (mg/kg soil)	Magnitude of effect (%)	Reference
Lettuce (*Lactuca sativa*)	21	3.91, 15.6, 62.5, 250, 1,000	LOEC	height	3.91	23% reduction in height	Brignole *et al*. (2003)
Ryegrass (*Lolium perenne*)	21	3.91, 15.6, 62.5, 250, 1,000	IC_{25}	shoot weight	7.51	25	Brignole *et al.* (2003)
Lettuce (*Lactuca sativa*)	21	3.91, 15.6, 62.5, 250, 1,000	IC_{25}	shoot weight	8.92	25	Brignole *et al*. (2003)
Tomato (*Lycopersicon esculentum*)	21	3.91, 15.6, 62.5, 250, 1,000	IC_{25}	shoot weight	11.7	25	Brignole *et al.* (2003)
Earthworm (*Eisenia fetida*)	56	0, 10, 20, 40, 80, 150, 250, 500	IC_{25}	avg weight per juvenile	12	25	Stubberud (2006)
Onion (*Allium cepa*)	21	3.91, 15.6, 62.5, 250, 1,000	IC_{25}	shoot weight	12.9	25	Brignole *et al.* (2003)
Soil mite *(Oppia nitens)*	28	0, 21, 46, 84, 160, 340, 680	IC_{25}	number of live juveniles produced (coarse soil)	13	25	EC (2015)
Tomato (*Lycopersicon esculentum*)	21	3.91, 15.6, 62.5, 250, 1,000	IC_{25}	height	22.1	25	Brignole *et al*. (2003)
Onion *(Allium cepa)*	21	3.91, 15.6, 62.5, 250, 1,000	IC_{25}	height	29.1	25	Brignole *et al*. (2003)
Soil mite *(Oppia nitens)*	28	0, 18, 24, 41, 54, 85, 180	IC_{25}	number of live juveniles	33	25	EC (2015)

Species name	Exposure duration (days)	Exposure concentration (mg/kg)	Endpoint	Effect	Concentration (mg/kg soil)	Magnitude of effect (%)	Reference
				produced (fine soil)			
Earthworm (*Eisenia fetida*)	56	0, 10, 20, 40, 80, 150, 250, 500	LOEC	total weight of juveniles	40		Stubberud (2006)
Ryegrass (*Lolium perenne*)	21	3.91, 15.6, 62.5, 250, 1,000	IC_{25}	height	46.3	25	Brignole *et al.* (2003)
Earthworm (*Eisenia fetida*)	56	0, 10, 20, 40, 80, 150, 250, 500	IC_{25}	number of juveniles	48	25	Stubberud (2006)
Onion (*Allium cepa*)	21	3.91, 15.6, 62.5, 250, 1,000	EC_{25}	emergence	50.8	25	Brignole *et al*. (2003)
Alfalfa (*Medicago sativa*)	21	3.91, 15.6, 62.5, 250, 1,000	IC_{25}	shoot weight	53.3	25	Brignole *et al.* (2003)
Springtail (*Folsomia candida*)	28	0, 16, 27, 48, 85, 140, 230, 320	IC_{25}	number of live juveniles produced (coarse soil)	61	25	EC (2015)
Tomato (*Lycopersicon esculentum*)	21	3.91, 15.6, 62.5, 250, 1,000	LOEC	survival of emerged seedlings	62.5	27% reduction in seedling survival	Brignole *et al*. (2003)
Earthworm (*Eisenia fetida*)	28	0, 10, 20, 40, 80, 150, 250, 500	IC_{25}	number of cocoons	67	25	Stubberud (2006)
Flax (*Linum usitatissimum*)	21	3.91, 15.6, 62.5, 250, 1,000	IC_{25}	shoot weight	81.6		Brignole *et al*. (2003)
Flax (*Linum usitatissimum*)	21	3.91, 15.6, 62.5, 250, 1,000	IC_{25}	height	97.6	25	Brignole *et al*. (2003)
Alfalfa (*Medicago sativa*)	21	3.91, 15.6, 62.5, 250, 1,000	IC_{25}	height	102	25	Brignole *et al.* (2003)
Soybean (*Glycine max*)	21	3.91, 15.6, 62.5, 250, 1,000	IC_{25}	shoot weight	160	25	Brignole *et al.* (2003)

Species name	Exposure duration (days)	Exposure concentration (mg/kg)	Endpoint	Effect	Concentration (mg/kg soil)	Magnitude of effect (%)	Reference
Springtail (*Folsomia candida*)	28	0, 14, 29, 59, 82, 130, 220, 350	IC_{25}	number of live juveniles produced (fine soil)	177	25	EC (2015)
Ryegrass (*Lolium perenne*)	21	3.91, 15.6, 62.5, 250, 1,000	EC_{25}	emergence	203	25	Brignole *et al.* (2003)
Ryegrass (*Lolium perenne*)	21	3.91, 15.6, 62.5, 250, 1,000	LOEC	survival of emerged seedlings	250	34% reduction in seedling survival	Brignole *et al*. (2003)
Alfalfa (*Medicago sativa*)	21	3.91, 15.6, 62.5, 250, 1,000	LOEC	survival of emerged seedlings	250	29% reduction in survival	Brignole *et al.* (2003)
Lettuce (*Lactuca sativa*)	21	3.91, 15.6, 62.5, 250, 1,000	LOEC	survival	250	23% reduction in seedling survival	Brignole *et al.* (2003)
Earthworm (*Eisenia fetida*)	14	100, 160, 256, 410, 655, 1,050 (dry weight)	LOEC	survival	256	20% reduced survival	Joung *et al*. (2010)
Soybean (*Glycine max*)	21	3.91, 15.6, 62.5, 250, 1,000	IC_{25}	height	284	25	Brignole *et al*. (2003)
Tomato (*Lycopersicon esculentum*)	21	3.91, 15.6, 62.5, 250, 1,000	EC_{25}	emergence	311	25	Brignole *et al.* (2003)
Alfalfa (*Medicago sativa*)	21	3.91, 15.6, 62.5, 250, 1,000	EC_{25}	emergence	372	25	Brignole *et al.* (2003)
Lettuce (*Lactuca sativa*)	21	3.91, 15.6, 62.5, 250, 1,000	EC_{25}	emergence	393	25	Brignole *et al*. (2003)

APPENDIX I. TYPICAL VALUES FOR PHYSIOLOGICAL PARAMETERS AND INTAKES OF AIR, WATER, SOIL AND DUST USED IN THE CALCULATION OF THE EDIS FOR THE CANADIAN GENERAL POPULATION

Receptor characteristic	Breastfed infant (0–6 mo)	Non-breastfed infant (0–6 mo)	Toddler (7 mo–4 yr)	Child (5–11 yr)	Teen (12–19 yr)	Adult (20+ yr)
Body weight[a] (kg)	8.2	8.2	16.5	32.9	59.7	70.7
Inhalation rate[a,b] (m^3/day)	2.2	2.2	8.3	14.5	15.6	16.6
Water ingestion rate[a] (L/day)	0.3	0.3	0.6	0.8	1.0	1.5
Soil ingestion rate[a] (g/day)	0.02	0.02	0.08	0.02	0.02	0.02
Indoor dust ingestion rate[c] (g/day)	0.036	0.036	0.041	0.032	0.0022	0.0026
Skin surface area[a] (cm^2)						
Hands	320	320	430	590	800	890
Arms	550	550	890	1,480	2,230	2,510
Legs	910	910	1,690	3,070	4,970	5,720
Soil loading to exposed skin[a] (kg/cm^2/event)						
Hands	1.0×10^{-7}	1.0×10^{-7}	1.0×10^{-7}	1.0×10^{-7}	1.0×10^{-7}	1.0×10^{-7}
Surfaces other than hands (arms, legs)	1.0×10^{-8}	1.0×10^{-8}	1.0×10^{-8}	1.0×10^{-8}	1.0×10^{-8}	8.0×10^{-8}

[a] Based on Allan *et al.* (2008) inhalation rate and 7.6×10^{-10} m^3/day concentration of airborne suspended soil particles.

[b] The time spent outdoors is assumed to be 1.5 hours/day for all age groups. Time spent outdoors by infant, toddler or child is assumed to be equivalent to that of an adult if child or infant is assumed to be accompanied by an adult.

[c] Wilson *et al*. (2012).

APPENDIX J. ESTIMATED TOTAL AVERAGE DAILY INTAKES (NG/KG BW/DAY) OF PFOS BY AGE CLASS FOR THE CANADIAN GENERAL POPULATION

Medium of exposure	Typical concentration	Daily PFOS intake (ng/kg bw/day)					
		Breastfed infant (0–6 mo)	Non-breastfed infant (0–6 mo)	Toddler (7 mo–4 yr)	Child (5–11 yr)	Teen (12–19 yr)	Adult (20+ yr)
Air							
Ambient air (inhalation)	6.4 pg/m^3 [a]	1.1×10^{-4}	1.1×10^{-4}	2.0×10^{-4}	1.8×10^{-4}	1.0×10^{-4}	9.4×10^{-5}
Indoor air (inhalation)	6.4 pg/m^3 [b]	1.6×10^{-3}	1.6×10^{-3}	3.0×10^{-3}	2.6×10^{-3}	1.6×10^{-3}	1.4×10^{-3}
Drinking water	**3.3 ng/L[c]**						
Drinking water (ingestion)		NA	0.121	0.120	0.080	0.055	0.070
Indoor settled dust[d]	**71 ng/g[e]**						
Settled dust (ingestion)		0.312	0.312	0.176	0.069	0.003	0.003
Settled dust (dermal)[f]		0.40	0.40	0.30	0.23	0.18	0.17
Soil[g]							
Soil (ingestion)		NC	NC	NC	NC	NC	NC
Soil (dermal)		NC	NC	NC	NC	NC	NC
Food[h]							
Food (ingestion)		NA	0.88	3.25	3.42	2.58	2.04
Total EDI		**NC**	**1.7**	**3.8**	**3.8**	**2.8**	**2.3**

NA = not available
NC = not calculated
Intake rates are provided in Appendix I.

[a] Mean concentration (n=8) in particulate phase collected in the Great Lakes regions (Lakes Erie and Ontario) (Boulanger *et al*. 2005*a*), which is the highest mean Canadian concentration reported. See Section 2.4.1.

[b] By default, the indoor air PFOS concentration was set equal to the outdoor air concentration (Section 2.4.2).

[c] Mean concentration in tap water (n=5) collected at Niagara-on-the-Lake in 2006–2008 (Mak *et al*. 2009), which is the highest published Canadian mean drinking water concentration (Section 2.4.6).

[d] Not taken into account inCCME (2006).

[e] GM concentration for indoor dust collected in Vancouver, BC, in 2007–2008 (median = 71 ng/g) (Shoeib *et al*. 2011), which is from the Canadian study with the largest sample size (n=132) and with the lower MDL (0.40 ng/g) (Section 2.4.3).

[f] Using a frequency event of 1 per day.

[g] As PFOS does not naturally exist in soils and as no Canadian data are available, its Canadian background soil concentration was set to 0.

[h] The food EDIs were provided by Health Canada. They are based on the 2008 TDS (Tittlemeier et al. 2007); (Section 2.4.10).

APPENDIX K. DATA REQUIREMENTS TO CALCULATE THE SOIL CONTACT SoQG USING CCME PREFERRED WEIGHT OF EVIDENCE METHOD

Weight of evidence method 1a: IC_{25}/EC_{25} data distribution					
	Number of invertebrate data points	**Number of terrestrial plant data points**	**Total number of studies**	**Total number of data points**	**Soil contact guideline based on weight of evidence method**
Minimum data set required for SoQG	2	2	3	10	
Available data for PFOS	9	23	4	32	
References	Stubberud (2006); Joung *et al.* (2010); EC (2015)	Brignole *et al*. (2003)			agricultural & residential/parkland land uses=11 mg/kg (10 mg/kg, rounded) commercial & industrial land uses=61 mg/kg (60 mg/kg, rounded)

APPENDIX L. SUMMARY OF INPUT PARAMETERS FOR GUIDELINE CALCULATION

Human health input parameters

Acronym	Description	Land use[a]		
		Agr. and Res./Park.	Commercial	Industrial
TDI	tolerable daily intake (mg/kg bw/day) per oral route of exposure (HC 2018*a*)	6×10^{-5}	6×10^{-5}	6×10^{-5}
EDI	estimated daily intake (ng/kg bw/day) (Section 7.2)	3.8	3.8	2.3
SF	soil allocation factor (default – unitless; CCME 2006)	0.2	0.2	0.2
BW	body weight (kg; CCME 2006)	16.5	16.5	70.7
AF_G	relative absorption factor for PFOS across the gut (unitless)	1	1	1
SIR	soil ingestion rate (kg/day; CCME 2006)	8×10^{-5}	8×10^{-5}	2×10^{-5}
AF_S	relative absorption factor for PFOS across the skin (unitless)	0.1	0.1	0.1
SR	soil dermal contact rate (kg/day; CCME 2006)[b]	6.9×10^{-5}	6.9×10^{-5}	1.14×10^{-4}
AF_L	relative absorption factor for PFOS across the lung (unitless)	1	1	1
IR_S	soil inhalation rate (kg/day)[c]	6.3×10^{-9}	6.3×10^{-9}	1.3×10^{-8}
ET_1	exposure term 1 (default – unitless; CCME 2006)	1	0.66	0.66
ET_2	exposure term 2 (default – unitless; CCME 2006)	1	0.42	0.42
BSC	background soil concentration (mg/kg – assumed)	0	0	0

[a] Agr. = agricultural, res./park.= residential/parkland.

[b] Soil dermal contact rate = (hands surface area × soil loading to exposed skin of the hands) + (arms surface area × soil loading to exposed skin of the arms) + ([toddler only] legs surface area × soil loading to exposed skin of legs).

[c] Value derived from the daily inhalation rate (Allan *et al*. 2008) for the critical receptor, assuming the airborne concentration of suspended soil particulate above a contaminated site is 7.6×10^{-10} kg/m^3.

Soil and hydrological input parameters

Symbol	Parameter	Fine Soil	Coarse Soil
ρ_B	Soil Bulk Density (kg/L, CCME 2006)	1.4	1.7
θ_t	Soil Total Porosity (cm^3/cm^3, CCME 2006)	0.47	0.36
θ_w	Soil Moisture-Filled Porosity (cm^3/cm^3, CCME 2006)	0.168	0.119
θ_a	Soil Vapour-Filled Porosity (cm^3/cm^3, CCME 2006)	0.302	0.241
f_{oc}	Fraction of Organic Carbon (mass/mass, CCME 2006)	0.005	0.005
K	Saturated Hydraulic Conductivity (m/y, CCME 2006	32	320
i	Hydraulic Gradient (m/m, CCME 2006)	0.028	0.028
I	Recharge (Infiltration) Rate (m/y, CCME 2006)	0.2	0.28

Site input parameters

Symbol	Parameter	Value
Y	Contaminant Source Width (m, CCME 2006)	10
X	Contaminant Source Length (m, CCME 2006)	10
Z	Contaminant Source Depth (m, CCME 2006)	3
x	Distance to Surface Water (m, CCME 2006)	10
x	Distance to Potable Water User (m, CCME 2006)	0
x	Distance to Agricultural Water User (m, CCME 2006)	0
d	Depth to Groundwater (water table) (m, CCME 2006)	3
d_a	Depth of unconfined aquifer (m, CCME 2006)	5
t	Time since contaminant release (y, CCME 2006)	500

Chemical and physical properties

Symbol	Symbol	Parameter
K_{oc}	Soil Organic Carbon/Water Partition Coefficient (L/kg, Franz Environmental 2012, 2014)	1445
H_L	Henry's law coefficient ((mg/L)/(mg/L), EC 2006*a*)	1.44×10^{-7}

	Degradation	
$t_{1/2}$	Degradation half-life	Does not degrade

Water Quality Guidelines

Symbol	Water Quality Guideline	Value
C_{GW}	Canadian Drinking Water Guideline (HC 2018*a*, mg/L)	0.0006
C_W	Protection of Freshwater Aquatic Life (ECCC 2018, mg/L)	0.0068

$SoQG_l$ input parameters for primary, secondary, and tertiary consumer

Trophic level	Feeding guild	Representative species	BW[a] (kg)	FIR[b] (kg dw/day)	Diet[b]	BCF[c] or BAF[d] (unitless)	SIR[e] (kg dw/day)	Soil BF[f] (unitless)	DTED[g] (mg/kg bw/day)	SoQG (mg/kg soil)
Primary consumer (1C)	herbivorous mammal	meadow vole (*Microtus pennsylvanicus*)	0.035	0.00173	plants	0.35	0.000041	1	0.054	2.2
	herbivorous bird	rock dove (*Columba livia*)	0.31	0.039	plants	0.35	0.00078	1	0.386	6.2
Secondary consumer (2C)	insectivorous mammal	common (masked) shrew (*Sorex araneus*)	0.004	0.0013	2.5% plants	0.35	0.000032	1	0.054	0.011
					95% invertebrates	10.9				
					2.5% small mammals	2.97				
	omnivorous mammal	deer mouse (*Peromyscus maniculatus*)	0.02	0.0009	50% plants	0.35	0.000018	1	0.054	0.17
					50% invertebrates	10.9				
	omnivorous bird	American robin (*Turdus migratorius*)	0.08	0.015	60% plants	0.35	0.00059	1	0.386	0.33
					40% invertebrates	10.9				
Tertiary consumer (3C)	carnivorous mammal	wolf (*Canis lupus*)	80	0.42	mammals	2.97	0.0118	1	0.054	2.6
	omnivorous mammal	red fox (*Vulpes vulpes*)	3.8	0.05	15% plants	0.35	0.0015	1	0.054	0.63
					25% invertebrates	10.9				
					60% mammals and birds	2.97				

[a] BW = body weight, from FCSAP (2012), BC MOE (2001), and Gibbs et al (2001).

[b] FIR = food ingestion rate. Diet and FIR from FCSAP (2012), BC MOE (2001), and Gibbs et al (2001). Converted from fresh weight, except rock dove and common shrew, using moisture content of 85% for plants and small mammals, 84% for invertebrates. Rock dove diet assumed to be dry weight in Gibbs et al (2001).

[c] BCF = bioconcentration factor, see Sections 3.6.1 to 3.6.2.

[d] BAF = bioaccumulation factor, see Sections 3.6.1 to 3.6.3.

[e] SIR = soil ingestion rate, from Beyer et al (1994) and FCSAP (2012). Common shrew SIR assumed to be the same as meadow vole, and grey wolf the same as red fox.

[f] BF = bioavailability factor

[g] DTED = daily threshold effects dose, see Section 6.1.2.1.