Rotterdam Convention

Operation of the prior informed consent procedure for banned or severely restricted chemicals

Decision Guidance Document

Perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds

Secretariat of the Rotterdam Convention
on the Prior Informed Consent Procedure
for Certain Hazardous Chemicals and Pesticides
in International Trade

Food and Agriculture Organization of the United Nations

UNEP

K2210590 141022

Introduction

The objective of the Rotterdam Convention is to promote shared responsibility and cooperative efforts among Parties in the international trade of certain hazardous chemicals in order to protect human health and the environment from potential harm and to contribute to their environmentally sound use, by facilitating information exchange about their characteristics, by providing for a national decision-making process on their import and export and by disseminating these decisions to Parties.

Candidate chemicals[1] for inclusion in the prior informed consent (PIC) procedure under the Convention are those that have been banned or severely restricted by national regulatory actions in two or more Parties[2] in two different regions. Inclusion of a chemical in the PIC procedure is based on regulatory actions taken by Parties that have addressed the risks associated with the chemical by banning or severely restricting it. Other ways might be available to control or reduce such risks. Inclusion does not, however, imply that all Parties to the Convention have banned or severely restricted the chemical. For each chemical included in Annex III to the Convention and subject to the PIC procedure, Parties are requested to make an informed decision whether they consent or not to the future import of the chemical.

At the face-to-face segment of its tenth meeting, held in Geneva from 6 to 17 June 2022, the Conference of the Parties agreed to list perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds in Annex III to the Convention and adopted the decision guidance document with the effect that these chemicals became subject to the PIC procedure.

The present decision guidance document was communicated to designated national authorities on 21 October 2022, in accordance with Articles 7 and 10 of the Convention.

Purpose of the decision guidance document

For each chemical included in Annex III to the Rotterdam Convention, a decision guidance document has been approved by the Conference of the Parties. Decision guidance documents are sent to all Parties with a request that they make a decision regarding future import of the chemical listed in the relevant category(ies) in Annex III to the Convention. Further information on import response can be found on the website of the Rotterdam Convention.[3]

Decision guidance documents are prepared by the Chemical Review Committee. The Committee is a group of government-designated experts established in line with Article 18 of the Convention, which evaluates candidate chemicals for possible inclusion in Annex III to the Convention. Decision guidance documents reflect the information provided by two or more Parties in support of their national regulatory actions to ban or severely restrict the chemical. They are not intended as the only source of information on a chemical nor are they updated or revised following their adoption by the Conference of the Parties.

There may be additional Parties that have taken regulatory actions to ban or severely restrict the chemical and others that have not banned or severely restricted it. Risk evaluations or information on alternative risk mitigation measures submitted by such Parties may be found on the Convention website (www.pic.int).

Under Article 14 of the Convention, Parties can exchange scientific, technical, economic and legal information concerning the chemicals under the scope of the Convention including toxicological, ecotoxicological and safety information. This information may be provided directly to other Parties or through the Secretariat. Information provided to the Secretariat will be posted on the Convention website.

Information on the chemical may also be available from other sources.

Disclaimer

The use of trade names in the present document is primarily intended to facilitate the correct identification of the chemical. It is not intended to imply any approval or disapproval of any particular company. As it is not possible to include all trade names currently in use, only a certain number of commonly used and published trade names have been included in the document.

While the information provided is believed to be accurate according to data available at the time of preparation of the present document, the Food and Agriculture Organization of the United Nations (FAO) and the United Nations Environment Programme (UNEP) disclaim any responsibility for omissions or any consequences that may arise

[1] According to the Convention, the term “chemical” means a substance, whether by itself or in a mixture or preparation and whether manufactured or obtained from nature but does not include any living organism. It consists of the following categories: pesticide (including severely hazardous pesticide formulations) and industrial.

[2] According to the Convention, the term “Party” means a State or regional economic integration organization that has consented to be bound by the Convention and for which the Convention is in force.

[3] http://www.pic.int/Procedures/ImportResponses/tabid/1162/language/en-US/Default.aspx.

therefrom. Neither FAO nor UNEP shall be liable for any injury, loss, damage or prejudice of any kind that may be suffered as a result of importing or prohibiting the import of this chemical.

The designations employed and the presentation of material in this publication do not imply the expression of any opinion whatsoever on the part of FAO or UNEP concerning the legal status of any country, territory, city or area or of its authorities or concerning the delimitation of its frontiers or boundaries.

STANDARD CORE SET OF ABBREVIATIONS	
<	less than
≤	less than or equal to
>	greater than
≥	greater than or equal to
µg	microgram
µm	micrometre
AFFF	Aqueous Film Forming Foam
BAF	bioaccumulation factor
BCF	bioconcentration factor
bw	body weight
BMF	biomagnification factor
°C	degree Celsius (centigrade)
CAS	Chemical Abstracts Service
cm	centimetre
DNA	deoxyribose nucleic acid
EC	European Community
EC_{50}	median effective concentration
ECHA	European Chemicals Agency
EU	European Union
FAO	Food and Agriculture Organization of the United Nations
g	gram
h	hour
IARC	International Agency for Research on Cancer
IPCS	International Programme on Chemical Safety
k	kilo- (x 1,000)
kg	lilogram
L	litre
LC_{50}	median lethal concentration
m	metre
mg	milligram
ml	millilitre
MAK	maximum workplace concentration (Germany)
NOEC	no-observed-effect concentration
OECD	Organisation for Economic Co-operation and Development
PFAS	Per- and Polyfluoroalkyl Substances
PFCA	perfluoroalkyl carboxylic acids
POPRC	Persistent Organic Pollutants Review Committee of the Stockholm Convention
PTFE	polytetrafluoroethylene
TMF	trophic magnification factor
UNEP	United Nations Environment Programme
U.S. EPA	United States Environmental Protection Agency
w/w	weight for weight
WHO	World Health Organization
wt	weight
wwt	wet weight

Decision guidance document for a banned or severely restricted chemical

PERFLUOROOCTANOIC ACID (PFOA), ITS SALTS AND PFOA-RELATED COMPOUNDS

1. Identification and uses (see Annex 1 for further details)

Common name

Perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds

Chemical name and other names or synonyms

- Perfluorooctanoic acid (PFOA) and its salts
- Any related substance (including its salts and polymers) having a linear or branched perfluoroheptyl group with the formula C_7F_{15}- directly attached to another carbon atom, as one of the structural elements
- Any related substance (including its salts and polymers) having a linear or branched perfluorooctyl group with the formula C_8F_{17}- as one of the structural elements

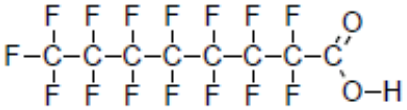
The following substances are excluded from this designation:

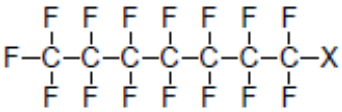
- C_8F_{17}-X, where X = F, Cl, Br
- C_8F_{17}-C(=O)OH, C_8F_{17}-C(=O)O-X′ or C_8F_{17}-CF_2-X′ (where X′ = any group, including salts)
- Perfluorooctane sulfonic acid (PFOS) and its derivatives ($C_8F_{17}SO_2X$ (X = OH, metal salt (O-M+), halide, amide, and other derivatives including polymers)).

Molecular formula

$C_8HF_{15}O_2$ (PFOA)

Chemical structure





CAS No.(s)

A comprehensive list of CAS numbers of PFOA, its salts and PFOA related chemicals is not available. Commercial mixtures containing PFOA are often not well characterized.

The following CAS numbers of PFOA, its salts and PFOA-related compounds have been specified in the notifications from Canada and Norway:

Free Acid (X = $COOM^+$; M = H) (CAS No. 335-67-1)

Ammonium Salt (X = $COOM^+$; M = NH_4) (CAS No. 3825-26-1)
Sodium Salt (X = $COOM^+$; M = Na) (CAS No. 335-95-5)

Potassium Salt (X = $COOM^+$; M = K) (CAS No. 2395-00-8)

Silver Salt (X = $COOM^+$; M = Ag) (CAS No. 335-93-3)

Acid Fluoride (X = COF) (CAS No. 335-66-0)

Methyl Ester (X = $COOCH_3$) (CAS No. 376-27-2)

Ethyl Ester (X = $COOCH_2$-CH_3) (CAS No. 3108-24-5)

The most common commercially used salt form of PFOA is the ammonium salt, referred to as APFO (CAS No. 3825-26-1).

Examples of chemicals with CAS numbers that meet the definition of PFOA, its salts and PFOA-related compounds listed in Annex A to the Stockholm Convention can be found in the “Supporting information related to the draft risk management evaluation on

	pentadecafluorooctanoic acid (CAS No. 335-67-1, PFOA), its salts and PFOA-related compounds: Non-exhaustive list of substances covered and not covered" (UNEP/POPS/POPRC.13/INF/6/Add.1).[4] Please note that some chemicals listed in this document may fall outside the scope of the chemicals covered in Annex III to the Rotterdam Convention.
Harmonized System Customs Code	29159070
Other numbers	INDEX No. 607-704-00-2 EC No. 206-397-9, EC No. 223-320-4 (ammonium salt); EC No. 206-404-5 (sodium salt) RTECS No. RH0781000 EINECS# 206-397-9
Category	Industrial
Regulated category	Industrial chemical
Use(s) in regulated category	***Canada:*** Primarily used as water, oil and grease repellants; as surfactants; and as spreading and wetting agents. AFFFs may also contain APFO (PFOA ammonium salt) as a component or a contaminant. ***Norway:*** Used in several applications, i.e. coating agent for carpets, textiles, furniture, shoes, paper, food wraps, printing plates but also in paint, floor wax, glue and photographic film. Often PFOA is present in products as a chemical impurity or as trace amounts of remaining starting materials from the production of other perfluorinated compounds, e.g. side-chain fluorinated polymers. PFOA has been found in imported products like textiles treated with perfluorinated compounds in order to make them water and stain repellent. PFOA may also be found in food contact materials with non-stick properties. PFOA was previously often present in ski wax in small amounts as a chemical impurity of the perfluorinated constituents in the wax.
Trade names	Not available.
Formulation types	Not relevant.
Uses in other categories	***Canada and Norway:*** There was historical use of PFOA as a formulant in some pesticides in Canada which has recently ceased. Currently, no reported use as a pesticide.
Basic manufacturers	3M, USA (until 2002) Arkema, Asahi, BASF, Clariant, Daikin, 3M/Dyneon, DuPont and Solvay Solexis Source: U.S. EPA (2015) *This is an indicative list of known current and former manufacturers. It is not intended to be exhaustive.*

2. Reasons for inclusion in the PIC procedure

Perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds (subsequently referred to as PFOA) are included in the PIC procedure in the industrial chemical category. PFOA is listed on the basis of final regulatory actions notified by Canada and Norway that severely restrict its use as an industrial chemical.

2.1 Final regulatory action (see Annex 2 for further details)

Canada:

Regulations that prohibit the manufacture, use, sale, offer for sale or import of PFOA and products containing PFOA, with a limited number of exemptions. (UNEP/FAO/RC/CRC.14/8 Sections 2 and 2.2.1 of the Canadian notification).

Reason: Environment

[4] In line with paragraph 9 of decision SC-9/3, the list is updated periodically by the Secretariat and made available on the website of the Stockholm Convention.

Norway:

Regulations to restrict the production, import, export or sale of consumer products that contain PFOA, its salts and esters in consumer products if they exceed certain limit values.

(UNEP/FAO/RC/CRC.16/4 Sections 2 and 2.2.1 of the Norwegian notification).

Reason: Human health and the environment

2.2 Risk evaluation (see Annex 1 for further details)

Canada:

An ecological screening assessment was undertaken on perfluorooctanoic acid (PFOA), its salts and its precursors containing the perfluorinated alkyl moiety (C_7F_{15}, C_8F_{17}) and is directly bound to any chemical moiety other than a fluorine, chlorine or bromine atom.

Once in the environment, PFOA is extremely persistent and not known to undergo significant abiotic or biotic degradation under relevant environmental conditions. PFOA is highly soluble in water and typically present as an anion (conjugate base) in solution. It has low vapour pressure; therefore, the aquatic environment is expected to be its primary sink, with some additional partitioning to sediment. The presence of PFOA in the Canadian Arctic is likely attributable to the long-range transport of PFOA (e.g., via ocean currents) and/or volatile precursors to PFOA (e.g., via atmospheric transport).

PFOA has been detected at trace levels in the northern hemisphere. In North America, higher levels were measured in surface waters in the vicinity of US fluoropolymer manufacturing facilities (<0.025–1900 µg/L) and in groundwater near US military bases (not detected, ND, to 6570 µg/L). PFOA was detected in effluent from Canadian wastewater treatment facilities at concentrations ranging from 0.007 to 0.055 µg/L. PFOA was also detected in the influent at US wastewater treatment facilities at concentrations ranging from 0.0074–0.089 µg/L.

Trace levels of PFOA have been measured in Canadian freshwater (ND–11.3 µg/L) and freshwater sediments (0.3–7.5 µg/kg). PFOA has also been detected in a variety of Canadian biota (ND–90 µg/kg wet weight (kg-wwt) tissue) in southern Ontario and the Canadian Arctic. The highest concentration of PFOA in Canadian organisms was found in the benthic invertebrate *Diporeia hoyi* at 90 µg/kg-wwt, followed by turbot liver at 26.5 µg/kg-wwt, polar bear liver at 13 µg/kg-wwt, caribou liver at 12.2 µg/kg-wwt, ringed seal liver at 8.7 µg/kg-wwt and walrus liver at 5.8 µg/kg-wwt. Following an accidental release of fire-fighting foam in Etobicoke Creek (Ontario), PFOA was measured in common shiner liver at a maximum concentration of 91 µg/kg-wwt. However, at the time of the assessment in 2012, PFOA concentrations in Canadian biota (tissue specific and whole body) were below the highest concentration found in US biota (up to 1934.5 µg/kg-wwt in gar liver).

Temporal or spatial trends in PFOA concentrations in guillemot eggs, lake trout, thick-billed murres, northern fulmars or ringed seals could not be determined at the time of assessment. However, temporal trends were found for PFOA concentrations in polar bears (1972 – 2002 and 1984 – 2006) and sea otters (1992 – 2002). PFOA doubling time in liver tissue was calculated to be 7.3 ± 2.8 years for Baffin Island polar bears and 13.9 ± 14.2 years for Barrow, Alaska, polar bears; central East Greenland polar bears showed an annual increase of 2.3% in PFOA concentrations. Concentrations of PFOA also increased significantly over a 10-year period for adult female sea otters.

Due to the perfluorination, the perfluorinated chains are both oleophobic and hydrophobic. PFOA primarily binds to albumin proteins in the blood of biota and, as a result, is present in blood and highly perfused tissues such as liver and kidney, rather than lipid tissue. There is experimental evidence indicating that PFOA is not highly bioaccumulative in fish. However, these results should not be extrapolated to non-aquatic species, since gills provide an additional mode of elimination for PFOA that air-breathing organisms, such as terrestrial and marine mammals, do not possess. Field studies indicating biomagnification factors greater than 1 for the Arctic and other mammals (such as narwhal, beluga, polar bear, walrus, bottlenose dolphins, and harbor seals) suggest that PFOA may bioaccumulate and biomagnify in terrestrial and marine mammals. Reported field biomagnification factors for terrestrial and marine mammals ranged from 0.03–31. Polar bears, as the apex predator in the Arctic marine food web, have been shown to be the most contaminated with PFOA relative to other Arctic terrestrial organisms.

At the time of the assessment, the risk quotients for pelagic organisms indicated a low likelihood of risk from exposures at current concentrations in the aquatic environment. The risk quotient for Canadian mammalian wildlife (i.e., polar bears) is less than 1. However, due to the persistent nature of the substance, its tendency to accumulate and biomagnify in a variety of terrestrial and marine mammals, its hepatotoxicity, and the upward temporal trend of PFOA concentrations in polar bears and other species, it was concluded at the time of the assessment that PFOA concentrations in polar bears may approach exposures resulting in harm.

The assessment was based on a weight of evidence approach regarding persistence, bioaccumulation, temporal trends in some species (i.e. the polar bear), long-range transport and the widespread occurrence and concentrations

of PFOA in the environment and in biota (including remote areas of Canada). Based on the information presented in the screening assessment in 2012, it was concluded that PFOA, its salts and its precursors are entering or may be entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity.

Norway:

The notification and its supporting material provide a large amount of data relating to human exposure, as well as information from European Food and Safety Agency document "Perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA) and their salts: scientific opinion of the Panel on Contaminants in the Food Chain" and ECHA document "Pentadecafluorooctanoic acid (PFOA) as a substance of very high concern because of its carcinogenic, mutagenic or toxic for reproduction (CMR) and persistent, bio-accumulating and toxic (PBT) properties". The Norwegian studies show that PFOA is transferred from the mother to the fetus, and that relatively high plasma concentrations are detected in blood samples from small children. Information on occupational exposure of professional Norwegian ski-waxers, leading to higher PFOA concentrations in blood serum, is also provided. Information in the risk evaluation points to widespread occurrence and concentrations of PFOA in the Norwegian environment (air, water and sediment). Persistence, bioaccumulation, temporal trends in some Arctic species (e.g., the polar bear) and evidence of long-range transport warrant concern.

PFOA is a substance of very high concern with respect to its health and environmental properties. PFOA is harmful to the reproductive system, is carcinogenic, toxic and harmful to human health through repeated exposure, and is also an irritant. PFOA does not degrade in the environment. PFOA is a PBT substance.

The notification concludes it is impossible to establish an acceptable level for substances with such properties in the environment, and that emissions and exposure should be limited to the greatest extent possible.

3. Protective measures that have been applied concerning the chemical

3.1 Regulatory measures to reduce exposure

Canada: Perfluorooctanoic acid, which has the molecular formula $C_7F_{15}CO_2H$, its salts, and its precursors (collectively referred to as PFOA) and products containing them are subject to the Prohibition of Certain Toxic Substances Regulations, 2012 (the Regulations), under the Canadian Environmental Protection Act, 1999 (CEPA). The Regulations prohibit the import, manufacture, use, sale and offer for sale of PFOA, and products containing PFOA, with a limited number of exemptions.

The risk management objective for PFOA is to achieve the lowest level of releases into the Canadian environment which is technically or economically feasible. The prohibitions do not apply to any toxic substance that is:

(a) contained in a hazardous waste, hazardous recyclable material or non-hazardous waste to which Division 8 of Part 7 of CEPA applies;

(b) contained in a pest control product as defined in subsection 2(1) of the *Pest Control Products Act*;

(c) present as a contaminant in a chemical feedstock that is used in a process from which there are no releases of the toxic substance and on the condition that the toxic substance is destroyed or completely converted in that process to a substance that is not a toxic substance set out in either Schedule 1 or 2 of the regulations; or

(d) to be used in a laboratory for analysis, in scientific research or as a laboratory analytical standard.

The Regulations do not prohibit:

(a) The import, manufacture, use, sale and offer for sale of PFOA or a product containing it, if PFOA is incidentally present (subsection 6(1) of the Regulations);

(b) The import, manufacture, use, sale and offer for sale of PFOA or a product containing them, before January 1, 2017, if it is designed for use in water-based inks or in photo media coatings, (paragraph 6(2)(b) of the Regulations);

(c) The import, use, sale and offer for sale of aqueous film forming foam for fire-fighting operations that contain PFOA (subsection 6(2.2) of the Regulations);

(d) The import, use, sale or offer for sale of manufactured items containing PFOA (subsection 6(2.4) of the Regulations);

(e) The use or import of products containing PFOA, if the product is for personal use (subsection 6(4) of the Regulations);

(f) The use, sale or offer for sale of:

(i) Products containing PFOA if manufactured or imported before the Regulations come into force (paragraph 7(2)(a) of the Regulations);

(ii) Water-based inks and photo media coatings containing PFOA that were manufactured or imported before January 1, 2017 (subsection 7(1) of the Regulations);

(iii) PFOA or products containing it if they were manufactured or imported in accordance with a permit (section 8 of the Regulations).

(UNEP/FAO/RC/CRC.14/8 Section 2.3.2 of the Canadian notification).

Norway: Since 4 July 2020, PFOA, its salts and PFOA-related compounds have been restricted in Norway as follows:

(1) Shall not be manufactured, or placed on the market as substances on their own from 4 July 2020.

(2) Shall not, from 4 July 2020, be used in the production of, or placed on the market in:

(a) Another substance, as a constituent;

(b) A mixture;

(c) An article, in a concentration equal to or above 25 ppb of PFOA including its salts or 1 000 ppb of one or a combination of PFOA-related substances.

(3) Points 1 and 2 shall apply from:

(a) 4 July 2022 to:

(i) Equipment used to manufacture semi-conductors;

(ii) Latex printing inks.

(b) 4 July 2023 to:

(i) Textiles for the protection of workers from risks to their health and safety;

(ii) Membranes intended for use in medical textiles, filtration in water treatment, production processes and effluent treatment;

(iii) Plasma nano-coatings.

(c) 4 July 2032 to medical devices other than implantable medical devices within the scope of Directive 93/42/EEC.

(4) Points 1 and 2 shall not apply to any of the following:

(a) Perfluorooctane sulfonic acid and its derivatives, which are listed in Part A of Annex I to Regulation (EC) No 850/2004[5];

(b) The manufacture of a substance where this occurs as an unavoidable by-product of the manufacture of fluorochemicals with a carbon chain equal to or shorter than 6 atoms;

(c) A substance that is to be used, or is used as a transported isolated intermediate, provided that the conditions in points (a) to (f) of Article 18(4) of this Regulation are met;

(d) A substance, constituent of another substance or mixture that is to be used, or is used:

(i) In the production of implantable medical devices within the scope of Directive 93/42/EEC;

[5] Regulation (EC) No 850/2004 was replaced with Regulation (EU) No 2019/1021 in 2019.

(ii) In photographic coatings applied to films, papers or printing plates;

(iii) In photo-lithography processes for semiconductors or in etching processes for compound semiconductors;

(e) Concentrated fire-fighting foam mixtures that were placed on the market before 4 July 2020 and are to be used, or are used in the production of other fire-fighting foam mixtures.

(5) Point 2(b) shall not apply to fire-fighting foam mixtures which were:

(a) Placed on the market before 4 July 2020; or

(b) Produced in accordance with point 4(e), provided that, where they are used for training purposes, emissions to the environment are minimised and effluents collected are safely disposed of.

(6) Point 2(c) shall not apply to:

(a) Articles placed on the market before 4 July 2020;

(b) Implantable medical devices produced in accordance with point 4(d)(i);

(c) Articles coated with the photographic coatings referred to in point 4(d)(ii);

(d) Semiconductors or compound semiconductors referred to in point 4(d)(iii).

Use or uses that remain allowed:

(4) Points 1 and 2 shall not apply to any of the following:

(a) Perfluorooctane sulfonic acid and its derivatives, which are listed in Part A of Annex I to Regulation (EC) No 850/2004[6];

(b) The manufacture of a substance where this occurs as an unavoidable by-product of the manufacture of fluorochemicals with a carbon chain equal to or shorter than 6 atoms;

(c) A substance that is to be used, or is used as a transported isolated intermediate, provided that the conditions in points (a) to (f) of Article 18(4) of this Regulation are met;

(d) A substance, constituent of another substance or mixture that is to be used, or is used:

(i) In the production of implantable medical devices within the scope of Directive 93/42/EEC;

(ii) In photographic coatings applied to films, papers or printing plates;

(iii) In photo-lithography processes for semiconductors or in etching processes for compound semiconductors;

(e) Concentrated fire-fighting foam mixtures that were placed on the market before 4 July 2020 and are to be used, or are used in the production of other fire-fighting foam mixtures.

(5) Point 2(b) shall not apply to fire-fighting foam mixtures which were:

(a) Placed on the market before 4 July 2020; or

(b) Produced in accordance with point 4(e), provided that, where they are used for training purposes, emissions to the environment are minimised and effluents collected are safely disposed of.

(6) Point 2(c) shall not apply to:

(a) Articles placed on the market before 4 July 2020;

(b) Implantable medical devices produced in accordance with point 4(d)(i);

(c) Articles coated with the photographic coatings referred to in point 4(d)(ii);

[6] Regulation (EC) No 850/2004 was replaced with Regulation (EU) No 2019/1021 in 2019.

(d) Semiconductors or compound semiconductors referred to in point 4(d)(iii).

(UNEP/FAO/RC/CRC.16/4 Sections 2 and 2.2.1 of the Norwegian notification).

3.2 Other measures to reduce exposure

An overview related to risk reduction approaches for PFASs was provided by OECD. The document includes information on existing risk reduction approaches in countries including voluntary risk reduction measures taken by corporations (see pp. 61 to 64 in OECD, 2015). National and/or regional regulations related to PFOA comprise the following:

(a) In 2013, the EU identified both PFOA and its ammonium salt (APFO) as Substances of Very High Concern (SVHC) due to their persistent, bioaccumulative and toxic properties, and PFOA and APFO were included into the REACH-Candidate List. On request industry is obliged to inform consumers on the occurrence to the listed substances in consumer articles if the SVHC in those articles is present in a concentration of more than 0.1 % (w/w). PFOA/APFO is restricted as a substance or in a mixture for the supply to consumers according to regulation (EU) 317/2014;

(b) PFOA has been included in Annex XVII (restriction) of the REACH regulation within the EU (Commission regulation (EU) 2017/1000 of 13 June 2017). PFOA shall not be manufactured or placed on the market as substances on their own from 4 July 2020, or be used in the production of, or placed on the market in another substance, as a constituent, a mixture, or an article, in a concentration equal to or above 25 ppb of PFOA including its salts or 1 000 ppb of one or a combination of PFOA-related compounds. The restriction includes several exemptions;

(c) In the EU, PFOA was included in Annex VI of the Classification, Labelling and Packaging (CLP) Regulation (Regulation (EC) No 1272/2008), by the Commission Regulation (EU) No 944/2013 of 2 October 2013 (index number: 607-704-00-2);

(d) In the U.S., the United States Environment Protection Agency (U.S. EPA) established the 2010/2015 PFOA Stewardship Programme in 2006. This is a programme that included eight major OECD based manufacturers of PFOA, its salts and PFOA-related compounds (Arkema, Asahi, BASF, Clariant, Daikin, 3M/Dyneon, DuPont and Solvay Solexis). The programme was a voluntary initiative to the substantial phase-out the manufacture and use of PFOA, PFOA precursors and related higher homologue substances (U.S. EPA, 2015). It was successfully completed at the end of 2015. On 21 January 2015, the U.S. EPA proposed a Significant New Use Rule under the Toxic Substances Control Act (TSCA) to require manufacturers of PFOA and PFOA-related chemicals, including as part of articles, and processors of these chemicals to notify U.S. EPA at least 90 days before starting or resuming new uses of these chemicals in any products. This notification would allow U.S. EPA the opportunity to evaluate the new use and, if necessary, take action to prohibit or limit the activity.[7] While in general, eligible polymers are exempted from the full U.S. EPA new chemical premanufacture notice and review process, effective 26 January 2010 the U.S. EPA rescinded the exemption for polymers containing as an integral part of their composition, except as impurities, certain perfluoroalkyl moieties consisting of a CF_3- or longer chain length. This exclusion included polymers that contain any one or more of the following: perfluoroalkyl sulfonates (PFAS), perfluoroalkyl carboxylates (PFAC), fluorotelomers, or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule;

(e) In China, several national actions were taken in 2011 to restrict new installations of PFOA production facilities, to eliminate PFOA-containing paints and fluoropolymers that use PFOA in the polymerization and to encourage the development of alternatives to PFOA. In 2013, fluoropolymer coatings for non-stick pans, kitchenware and food processing equipment that use PFOA in the polymerization were recognized as products with high pollution and high environmental risk in the Comprehensive Catalogue for Environmental Protection. In January 2017, new technical requirements for textile products came into force, in particular establishing limits of PFOA levels to 0.05 mg/kg in coated infant textile products and to 0.1 mg/kg in all other coated textile products, respectively.

(UNEP/POPS/POPRC.12/11/Add.2 section 2.3.2 of the POPRC Risk Profile)

(UNEP/POPS/POPRC.13/7/Add.2, Chapter 1.4 and 1.5 of the POPRC Risk Management Evaluation)

[7] https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/and-polyfluoroalkyl-substances-pfass-under-tsca.

Stockholm Convention on Persistent Organic Pollutants

At its twelfth meeting, by its decision POPRC-12/2, the Persistent Organic Pollutants Review Committee of the Stockholm Convention adopted a risk profile for perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds (UNEP/POPS/POPRC.12/11/Add.2) and decided, in accordance with paragraph 7 (a) of Article 8 of the Stockholm Convention on Persistent Organic Pollutants, that PFOA, its salts and PFOA-related compounds were likely, as a result of their long-range environmental transport, to lead to significant adverse human health and environmental effects such that global action was warranted. POPs Review Committee recommended to the Conference of the Parties that it consider listing perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds in Annex A to the Convention with specific exemptions (see UNEP/POPS/POPRC-14/2, UNEP/POPS/POPRC.14/6/Add.2, UNEP/POPS/COP.9/14).

In May, 2019, the Conference of the Parties of the Stockholm Convention decided to list perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds in Annex A to the Stockholm Convention (SC-9/12) with the following definition:

> "Perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds" means the following:
>
> (i) Perfluorooctanoic acid (PFOA; CAS No. 335-67-1), including any of its branched isomers;
>
> (ii) Its salts;
>
> (iii) PFOA-related compounds which, for the purposes of the Convention, are any substances that degrade to PFOA, including any substances (including salts and polymers) having a linear or branched perfluoroheptyl group with the moiety (C_7F_{15})C as one of the structural elements;
>
> The following compounds are not included as PFOA-related compounds:
>
> (i) C_8F_{17}-X, where X= F, Cl, Br;
>
> (ii) Fluoropolymers that are covered by $CF_3[CF_2]n$-R', where R'=any group, n>16;
>
> (iii) Perfluoroalkyl carboxylic and phosphonic acids (including their salts, esters, halides and anhydrides) with ≥8 perfluorinated carbons;
>
> (iv) Perfluoroalkane sulfonic acids (including their salts, esters, halides and anhydrides) with ≥9 perfluorinated carbons;
>
> (v) Perfluorooctane sulfonic acid (PFOS), its salts and perfluorooctane sulfonyl fluoride (PFOSF), as listed in Annex B to the Convention.

3.3 Alternatives

It is essential that before a country considers substituting alternatives, it ensures that the use is relevant to its national needs, and the anticipated local conditions of use. The hazards of the substitute materials and the controls needed for safe use should also be evaluated.

Canada:

In Canada, manufacturing has been the main industrial sector using PFOA, specifically, paper and chemical manufacturing (note PFOA is not manufactured or imported in Canada; however, its salts and precursors have been reported to be imported). Elsewhere, PFOA has been used in the production of fluoropolymers and fluorotelomers and as additive and component in consumer and industrial products.

In January 2006, the U.S. EPA introduced a voluntary Stewardship Program to reduce facility emissions and product content of PFOA and related chemicals on a global basis and to work toward eliminating emissions and product content of these chemicals by 2015. This Stewardship Program has been a major driver for companies to reduce residuals in products and to switch from PFOA products to safer alternatives.

The U.S. EPA is also reviewing substitutes for PFOA, PFOS, and other long-chain perfluorinated substances as part of its review process for new chemicals under U.S. EPA's New Chemicals Program. Over 150 alternatives of various types have been received and reviewed by U.S. EPA. Under the U.S. EPA's New Chemical Review of Alternatives for PFOA and related chemicals, shorter chain-length perfluorinated telomeric substances have been notified as alternatives for a variety of uses including, for example, textile, carpet and paper additive uses, and tile surface treatments. The major industry users in the global community have replaced uses of C-8 and higher homologues with alternatives.

Substances which are new to Canada, including new substitutes for PFOA, are subject to the New Substances provisions of CEPA and the New Substances Notification Regulations. Any company intending to import or manufacture such a substance must submit a notification, with the substance undergoing an assessment by

Environment Canada and Health Canada to determine whether it meets the definition of "toxic" set out in section 64 of CEPA. Many substitutes to PFOA have been notified to Environment Canada's New Substances Program.

(UNEP/FAO/RC/CRC.14/8 section 2.5.3.2 of the Canadian notification)

Norway:

U.S. EPA's review of alternatives to perfluorinated chemical substances has been ongoing since 2000 and is consistent with the approaches to alternatives encouraged under the 2010/15 PFOA Stewardship Program. Through June 2008, over 100 alternatives of various types have been received and reviewed by U.S. EPA. https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/risk-management-and-polyfluoroalkyl-substances-pfass#tab-3

Additional information on alternatives could also be found in these two publications:

(a) OECD/UNEP Global PFC Group, 2013;

(b) Wang et al., 2013.

(UNEP/FAO/RC/CRC.16/4 section 2.5.3.2 of the Norwegian notification)

General:

Several potential alternatives for use in textiles such as short-chain fluorinated alternatives, non-fluorine containing alternatives and non-chemical alternatives have been identified in the Risk Management Evaluation of perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds under the Stockholm Convention, including those that meet regulatory requirements and are in current use. Alternatives to PFOA for manufacture of polytetrafluoroethylene (PTFE) exist and have been commercialised. Non-fluorinated alternatives and the move to digital imaging have successfully replaced PFOA in the imaging and printing industry. Alternatives to all uses of PFOA in fire-fighting foams exist and include fluorine-free solutions as well as fluorosurfactants with C6-fluorotelomers. Fluorine-free foams are comparable to fluorine-based AFFFs and fire-fighting foams with PFOA in their performance and in meeting relevant certifications for almost all uses. Based on current data, prices of fluorine-free and fluorine containing AFFFs are comparable. Some concerns were expressed about the importance of effective fire-fighting foams for liquid fuel fires, the potential unavailability of suitable alternatives and the cost of their use and implementation, considering that some time to move to alternatives without PFASs may be needed. In the USA, non-fluorinated AFFF have been considered to meet the Federal performance standards and acceptable for use in airports in 2018 (FAA Reauthorization Act of 2018 (HR 302)).

For more information, see chapter 4.2.3 of the POPRC Addendum to the Risk Management Evaluation UNEP/POPS/POPRC.14/6/Add.2.

3.4 Socio-economic effects

Canada:

The scientific evidence has demonstrated that the substance PFOA and its salts are persistent and that they accumulate and biomagnify in terrestrial and marine animals. The ongoing release of PFOA may result in harm to the Canadian environment.

The Regulations protect the Canadian environment by preventing the reintroduction of PFOA as industry is already working towards phasing out these substances.

The Regulations were expected to have a low-cost impact on the industry. The substances were never manufactured in Canada and are only known to be imported within products or manufactured items. Furthermore, industry sectors have already completed the transition to alternatives, or were expected to do so prior to the coming into force of the Regulations. Development of alternatives to PFOA in water-based inks and photo media coatings were underway, and companies were expected to eliminate their use of these substances by the end of 2016, when the temporary exemption expired. For aqueous film-forming foams containing PFOA, which are allowed under the Regulations, the development of alternatives has begun and will be monitored.

(UNEP/FAO/RC/CRC.14/8 section 2.5.3.1 of the Canadian notification)

Norway:

The regulation proposal may result in some increased costs but will result in significant reductions of the amount of PFOA introduced into the environment and it will reduce the risk of health and environmental damages. The benefits are therefore expected to outweigh the costs on the basis of the proposal’s anticipated positive effects for health and the environment.

(UNEP/FAO/RC/CRC.16/4 section 2.5.3.1 of the Norwegian notification)

4. Hazards and Risks to human health and the environment	
4.1 Hazard Classification	
WHO / IPCS	Not available.
IARC	Possibly carcinogenic to humans (Group 2B).
European Union	Harmonised classification - Annex VI of Regulation (EC) No 1272/2008 (CLP Regulation) for PFOA (CAS 335-67-1) (https://echa.europa.eu/information-on-chemicals/cl-inventory-database/-/discli/details/67229): Acute Tox. 4 -H302 (Harmful if swallowed) -H332 (Harmful if inhaled) Eye Dam. 1 -H318 (Causes serious eye damage) Carc. 2 -H351 (Suspected of causing cancer) Lact. -H362 (May cause harm to breast-fed children) STOT RE 1 -H372 (liver) (Causes damage to organs (liver) through prolonged or repeated exposure) Repr. 1B -H360D (May damage the unborn child) Due to its PBT and CMR properties, PFOA and its ammonium salt (APFO) has been identified as substances of very high concern (SVHC) under REACH Regulation No 1907/2006 by unanimous agreement between EU Member States in July 2013. (Section 3.1 of the Canadian (UNEP/FAO/RC/CRC.14/8) and Norwegian (UNEP/FAO/RC/CRC.16/4) notifications)
U.S. EPA	Not available.

4.2 Exposure limits

Dietary intake including water is considered as the most important route of human exposure to PFOA based on studies from various countries. Taking this into consideration, among others, the U.S. EPA has issued a lifetime drinking water Health Advisory for combined PFOA/PFOS of 0.07 micrograms per liter (µg/L) based on a reference dose (RfD) derived from a developmental toxicity study in mice.

(UNEP/POPS/POPRC.12/11/Add.2 section 2.3.2 of the POPRC Risk Profile)

The European Food and Safety Agency, in their scientific evaluation on the risks to human health related to the presence of perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA) in food, derived a health-based guidance value that was based on human epidemiological studies. After benchmark modelling of serum levels of PFOS and PFOA, and estimating the corresponding daily intakes, the CONTAM Panel established a tolerable weekly intake (TWI) of 6 ng/kg bw per week for PFOA. Exposure of a considerable proportion of the population exceeds the proposed TWIs. http://www.efsa.europa.eu/en/efsajournal/pub/5194
https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2018.5194

Occupational exposure limits: MAK: (inhalable fraction): 0.005 mg/m^3; peak limitation category: II(8); skin absorption (H); carcinogen category: 4; pregnancy risk group: B Source: List of MAK and BAT values DFG 2016 https://onlinelibrary.wiley.com/doi/pdf/10.1002/9783527805983.ch2

German Biological Exposure Indices (BEI) 5 mg/l in serum http://gestis-en.itrust.de

4.3 Packaging and labelling	
The United Nations Committee of Experts on the Transportation of Dangerous Goods classifies the chemical (PFOA (CAS 335-67-1)) in:	
Hazard Class and Packing Group:	UN #3261 CORROSIVE SOLID, ACIDIC, ORGANIC, N.O.S. UN Hazard Class: 8; UN Pack Group: III (http://www.inchem.org/documents/icsc/icsc/eics1613.htm)
International Maritime Dangerous Goods (IMDG) Code	UN #3261 CORROSIVE SOLID, ACIDIC, ORGANIC, N.O.S.
Transport Emergency Card	Transport Emergency Card: TEC (R)-80GC4-II+III

4.4 First aid

NOTE: The following advice is based on information available from the World Health Organisation and the notifying countries and was correct at the time of publication. This advice is provided for information only and is not intended to supersede any national first aid protocols.

The information below refers to PFOA (CAS 335-67-1).

	Prevention	First Aid
Inhalation	Use local exhaust or breathing protection.	Fresh air, rest. Artificial respiration may be needed. Refer for medical attention.
Skin	Protective gloves. Protective clothing.	Wear protective gloves when administering first aid. Remove contaminated clothes. Rinse and then wash skin with water and soap.
Eyes	Wear safety goggles or eye protection in combination with breathing protection if powder.	Rinse with plenty of water for several minutes (remove contact lenses if easily possible). Refer immediately for medical attention.
Ingestion	Do not eat, drink, or smoke during work.	Rinse mouth. Give one or two glasses of water to drink. Refer for medical attention

http://www.inchem.org/documents/icsc/icsc/eics1613.htm International Chemical Safety Card (ICSC) 1613

4.5 Waste management

DISPOSAL: Dispose of as hazardous waste in compliance with local, regional and national regulations. Dispose of wastes in an approved waste disposal facility.

Annexes

Annex 1 **Further information on the substance**

Annex 2 **Details on final regulatory action reported**

Annex 3 **Address of designated national authorities**

Annex 4 **References**

Annex 1 Further information on the substance

The information presented in the present annex reflects the conclusions of the two notifying Parties, namely Canada and Norway. Where possible, information provided by these two Parties on hazards has been presented together, while the risk assessments, which are specific to the conditions prevailing in the Parties, are presented separately. This information is taken from the documents referenced in the notifications in support of the final regulatory actions relating to PFOA.

The notifications from Norway and Canada were first reported in PIC Circular LI (51) in June 2020 and PIC Circular XLVII (47) in June 2018, respectively.

The notification from Canada contained a non-exhaustive list of PFOA, its salts and precursors in Annex A. The notification from Norway also specified eight CAS numbers as an example, noting however that more chemicals are covered by the definition. The information below refers to perfluorooctanoic acid.

Further information on perfluorooctanoic acid

1	**Identity and Physico-Chemical properties**	
1.1	**Identity**	Octanoic acid, 2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-pentadecafluoro- (UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)
1.2	**Formula**	$C_8HF_{15}O_2$ (UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)
1.3	**Colour and Texture**	Solid (UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)
1.4	**Decomposition temperature**	Decomposes on heating above 300°C (ICSC 1613)
1.5	**Density (g/cm^3)**	Not available.
1.6	**Resistance to acids**	Not available.
1.7	**Resistance to alkalis**	Not available.
1.8	**Tensile strength (10^3 kg/cm^2)**	Not available.
2	**Toxicological properties**	
2.1	**General**	
2.1.1	**Mode of Action**	Data on the mode of action are insufficient (UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)
2.1.2	**Symptoms of poisoning**	Not available.
2.1.3	**Absorption, distribution, excretion and metabolism in mammals**	In humans, PFOA is well absorbed by all routes of exposure; it has not been demonstrated to be metabolized and has a relatively long half-life. Salts of PFOA are expected to dissociate in biological media to produce the perfluorooctanoate (PFO) anion, and are therefore considered toxicologically equivalent to PFOA. Low concentrations of PFOA have been identified in blood samples from non-occupationally exposed Canadians, including newborns, indicating environmental exposure to PFOA and/or compounds that can degrade to PFOA. Canadians are also potentially exposed to PFOA in utero and through lactational transfer. The relative contributions of PFOA and its salts and precursors to total PFOA exposure were not characterized; rather the focus was on aggregate exposure to the moiety of toxicological concern, PFOA. (UNEP/FAO/RC/CRC.14/8 Canadian notification) PFOA is efficiently taken-up by mammals from all exposure routes, and is not readily eliminated. In humans, half-life is estimated to 2.3 years, but even longer half-life has been estimated for retired workers from a perfluoroalkyl manufacturing plant with high plasma PFOA levels. In contrast, half-life values for the monkey, rat, and mouse

are 20.8 days, 11.5 days, and 15.6 days, respectively. PFOA is transferred to the fetus where it accumulates in the liver, it is also transferred to the child via breast milk.

Perfluorinated chemicals (PFCs) are amphiphilic. They bind to serum proteins and proteins in cell membranes, and accumulate in the blood and internal organs such as liver, kidneys, testes and brain. Metabolic transformation seems to be less important for elimination. Urine is the primary route of excretion and there are large sex and species differences in the excretion of PFOA. The reason for the differences in elimination is likely that PFOA is a substrate for renal organic anion transporters, regulating active renal reabsorption, and these transporters are differentially expressed between species and sex.

(UNEP/FAO/RC/CRC.16/4 Norwegian notification)

PFOA is readily absorbed after exposure (ingestion) and accumulates in serum and highly perfused organs, mainly in the liver and kidney, due to PFOA primarily binding to albumin proteins in the blood. There is evidence that PFOA levels in humans accumulate and increase with age. PFOA does not undergo metabolism or biotransformation in the body. As mentioned earlier, the half-life of PFOA elimination in humans is long, ranging between 2 and 4 years.

PFOA is known to be transmitted to the fetus in cord blood and to the newborn in breast milk. Developing fetuses and newborns are particularly sensitive to PFOA-induced toxicity. Positive correlation in PFOA level between maternal and cord blood samples has been reported in several birth cohort studies in Spain and Norway. As PFOA can be transferred to infants through breast-feeding, the ECHA's Risk Assessment Committee (RAC) agreed on an additional classification of PFOA on lactation effects (CLP: Lact. H362: May cause harm to breast-fed children).

(UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)

2.2 Toxicology studies

The toxicity of PFOA has been evaluated by ECHA, U.S. EPA, the Canadian ministries and EFSA. In the European Union, PFOA (CAS 335-67-1, index number 607-704-00-2) has a legally-binding harmonized classification. This substance was included under the Classification, Labelling and Packaging (CLP) Regulation (Regulation (EC) No 1272/2008), by the Commission Regulation (EU) No 944/2013. PFOA has been classified as Carc. 2 H351, Repr 1B H360D, Lact. H362, STOT RE 1 H372 (liver), Acute tox. 4 H332, Acute tox. 4 H302 and Eye dam. 1 H318.

(UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile; https://echa.europa.eu/information-on-chemicals/cl-inventory-database/-/discli/details/67229)

2.2.1 Acute toxicity

Toxicity studies in laboratory animals were used to determine the critical effects and associated serum levels of PFOA. Following oral dosing of PFOA ammonium salt (APFO), increased liver weight in mice and altered lipid parameters in rats were observed in short-term (14-day) toxicity studies; increased liver weight was noted in a 26-week toxicity study in monkeys; and increased liver weight in dams, alterations in fetal ossification and early puberty in male pups were found in a developmental toxicity study in mice.

(UNEP/FAO/RC/CRC.14/8 Canadian notification)

PFOA exhibits moderate acute, oral and inhalation toxicity

(UNEP/FAO/RC/CRC.16/4 Norwegian notification)

2.2.2 Short term toxicity

In sub-acute and chronic studies, PFOA affected primarily the liver and can cause developmental and reproductive toxic effects at relatively low dose levels in experimental animals. Twenty-eight-day oral toxicity studies in rats and mice showed mortality and dose-related reduced weight gain and increased liver weight at PFOA dietary concentrations of 30 mg/kg and higher or drinking water concentrations of 50 mg/L and above.

(UNEP/FAO/RC/CRC.16/4 Norwegian notification)

2.2.3 Genotoxicity (including mutagenicity)

PFOA has not been shown to be mutagen. The negative outcome in a comprehensive series of *in vitro* and *in vivo* short-term tests at gene and/or chromosome level indicates that PFOA is devoid of significant genotoxic activity.

(UNEP/FAO/RC/CRC.16/4 Norwegian notification)

2.2.4 Long term toxicity and carcinogenicity

PFOA increased the tumour incidence in rats, mainly in the liver. Based on the weight of evidence at present, the carcinogenic effects in rats appear to be due to indirect/non-genotoxic modes of action. PFOA has been shown to induce hepatocellular adenomas, Leydig cell adenomas and pancreatic acinar cell hyperplasia in male rats.

(UNEP/FAO/RC/CRC.16/4 Norwegian notification)

In 2-year carcinogenicity bioassays in rats, males administered a high dose of APFO in the diet had significantly higher incidences of adenomas of the liver hepatocytes, Leydig cells in the testes and pancreatic acinar cells. No evidence of carcinogenic activity was seen in the female rats. Liver tumours in male rats may be induced via liver toxicity resulting from PFOA-induced peroxisome proliferation, and additional pathways secondary to peroxisome proliferation may be involved in the generation of tumours at other sites. As primates are much less susceptible than rodents to peroxisome proliferation, the PFOA-induced tumours in male rats are considered to have little or no relevance for humans. Although blood levels of PFOA were not determined in the chronic studies, the oral dose of APFO was several times higher than those in the critical short-term and subchronic studies. Although there is some evidence to suggest that PFOA may be capable of causing indirect oxidative DNA damage, the genotoxicity database indicates that PFOA is not mutagenic. Thus, as the tumours observed in male rats are not considered to have resulted from direct interaction with genetic material, a threshold approach is used to assess risk to human health.

(UNEP/FAO/RC/CRC.14/8 Canadian notification)

Animal studies have demonstrated the induction of tumours mediated by PFOA or APFO, and hepatic activation of the peroxisome proliferator-activated receptor α (PPARα) has been proposed as a mechanism of induction of hepatic tumours. However, the PPAR-agonist mode of action proposed for rat liver, testes and pancreatic tumours may not be relevant for humans. However, human relevance had not been definitively determined according to established frameworks a decade ago, and PFOA compounds have also not been tested for carcinogenic potential in any laboratory animal species other than rats. Therefore, the RAC came to the conclusion that data on the mode of action are insufficient to conclude that APFO-induced tumours in animals are not relevant for humans, and therefore, PFOA is classified as Carc 2. Based on limited evidence in humans that PFOA causes testicular and renal cancer as well as limited evidence in experimental animals, IARC has classified PFOA as a Group 2B substance (possibly carcinogenic to humans).

(UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)

Findings from studies seem to indicate a PFOA-mediated effect on the endocrine system. Prenatal exposure of PFOA may alter testosterone concentrations in females, and an inverse correlation between parathyroid hormone 2 receptor (PTH2R) and PFOA exposure was also reported in a study of 189 women. As for men, a study reported an inverse relationship between PFOA serum level in men and expression of nuclear receptors such as estrogen and androgen receptors. Also, early menopause in women with high PFOA levels has been observed in the C8 Health Project.

PFOA has been implicated to act as a so-called obesogene similar to other endocrine disruptive compounds that can act directly on ligands for nuclear hormone receptors or affect components in metabolic signaling pathways. A human prospective cohort study showed a correlation between low dose PFOA exposure of 655 Danish pregnant women and obesogenic effects in their offspring at 20 years of age. Maternal PFOA concentrations were positively associated with serum insulin and leptin levels and inversely associated with adiponectin levels in female offspring. On the other hand, the C8 Health Project concluded that PFOA exposure in early life was not associated with overweight and obesity risk in adulthood.

Evidence from several epidemiological studies seems to suggest an association between exposure to PFOA and changes in different thyroid hormones leading to altered thyroid function inducing thyroid disease such as hypothyroidism or hyperthyroidism. However, there have also been studies that reported inconsistent findings between PFOA exposure and thyroid diseases (i.e. inverse relation between subclinical hyperthyroidism and PFOA or no association between hypothyroidism and PFOA).

The potential of PFOA to affect estrogen receptor (ER) and androgen receptor (AR) transactivity as well as aromatase enzyme activity was analysed in an *in vitro* study, and it was shown that PFOA significantly induced ER transactivity yet antagonized AR activity in a concentration-dependent manner. In addition, when PFOA was mixed with 6 other PFCs, a mixture effect more than additive was observed on AR function, emphasizing the importance of considering the combined action of PFCs in assessing related health risks.

(UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)

2.2.5 Effects on reproduction

PFOA has been shown to cause developmental and reproductive toxic effects at relatively low dose levels in experimental animals. Several studies observed complete litter loss at doses of 5 mg/kg bw/day. Increased postnatal pup mortality, decreased pup body weight and delayed sexual maturation were observed in several mice studies. A two-generation reproductive toxicity study in rats has shown post-weaning mortality, reduced growth, and delayed sexual maturation. Follow-up developmental toxicity studies in mice have shown a pattern of neonatal mortality similar to that observed in rats; this consists of a dose-related increase in mortality during the first several days after birth. Cross-fostering studies have shown that the critical period of exposure is during the prenatal period. Further studies have shown delayed development of the mammary glands in both the dams and female offspring with systemic toxicity in rodents and monkeys following long-term exposure by the oral route.

(UNEP/FAO/RC/CRC.16/4 Norwegian notification)

2.2.6 Neurotoxicity/ delayed neurotoxicity, Special studies where available

Impaired neurodevelopment has been associated with PFOA. An inverse relationship between prenatal PFOA concentrations in mothers and neurodevelopment as determined with the mental development index (MDI) in female (not male) offspring at 6 months of age was observed in a Japanese birth cohort (Hokkaido) study. However, this relationship was not observed with offspring at 18 months of age. Also, no correlation between PFOA levels and birth weight was observed in the same cohort study. Statistically significant inverse associations between PFOA and memory impairment has been reported. On the other hand, there are studies that reported no association between PFOA exposure and impaired neurodevelopment or behavior.

(UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)

2.2.7 Summary of mammalian toxicity and overall evaluation

PFOA is classified among others
as Carc. 2, Repr. 1B and STOT RE 1 (liver) according to Regulation (EU) No 944/2013. IARC also categorised PFOA as a Group 2B substance (possibly carcinogenic to humans). There have been reported adverse health effects such as elevated cholesterol levels, altered reproductive/developmental effects, endocrine disruption, impaired neurodevelopment, as well as increased risk of cancer associated with PFOA exposure in humans. Scientific data have demonstrated PFOA-mediated immunotoxicity, primarily suppression of antibody response, in humans. Although the findings are limited, the reported adverse health effects suggest additional public health concerns.

(UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)

3	Human exposure/Risk evaluation	
3.1	**Food**	Human exposure typically takes place “human via environment” by consumption of drinking water and food, via uptake of contaminated indoor dust or from consumer products containing PFOA and its related compounds. PFOA has been detected in humans in blood and breast milk from various countries. Babies are susceptible to PFOA exposure via breastfeeding or trans-placental passage, and people who live near fluoropolymer manufacturing facilities have been shown to have higher levels of serum PFOA than those from the general population. (UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile) The available data indicate that Canadians are exposed to PFOA and its precursors in the environment, including via air, drinking water and food; and from the use of consumer products, such as new non-stick cookware and perfluorinated compound (PFC)-treated apparel and household materials such as carpets and upholstery. (UNEP/FAO/RC/CRC.14/8, Canadian notification)
3.2	**Air**	PFOA has been detected in the air of remote areas (UNEP/POPS/POPRC.12/11/Add.2), as well as household dust (Shoeib et al., 2011) and airborne particulate matter (Yu et al., 2018).
3.3	**Water**	Human PFOA exposure occurs via dietary intake of food and drinking water, exposure to contaminated indoor dust or consumer products containing PFOA and its related compounds. Studies have demonstrated the presence of PFOA within humans, mainly in blood and breast milk samples. Fetuses and newborns are susceptible to PFOA exposure via breastfeeding or trans-placental passage. Occupational exposure or exposure near sites of production resulted in higher serum PFOA levels than exposure of general population. (UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)
3.4	**Occupational exposure**	There are a few reports of detected PFOA serum levels in the occupationally exposed workers. Indoor dust and total suspended particles seem to be important occupational exposure routes in fluorochemical manufacturing and is also considered relevant in domestic settings. In some cases, elevated serum PFOA levels can be largely attributed to exposure to PFOA-related compounds such as 8:2 FTOH. Persons living in the vicinity of fluorochemical manufacturing plants have higher PFOA levels than the general population. (UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)
3.5	**Medical data contributing to regulatory decision**	Not available.
3.6	**Public exposure**	Analysis of serum samples demonstrate that PFOA is detectable across the general population in various countries. (UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)
3.7	**Summary-overall risk evaluation**	The Norwegian notification and its supporting material provide a large amount of data relating to human exposure, as well as information from European Food and Safety Agency document “Perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA) and their salts: scientific opinion of the Panel on Contaminants in the Food Chain” and European Chemicals Agency document “Pentadecafluorooctanoic acid (PFOA) as a substance of very high concern because of its CMR and PBT properties”. The Norwegian studies show that PFOA is transferred from the mother to the fetus, and that relatively high plasma concentrations are detected in blood samples from small children. Information on occupational exposure of professional Norwegian ski-waxers, leading to higher PFOA concentrations in blood serum, is also provided. Information in the risk evaluation points to widespread occurrence and concentrations of PFOA in the Norwegian environment (air, water and sediment). Persistence, bioaccumulation, temporal trends in some Arctic species (e.g., the polar bear) and evidence of long-range transport warrant concern.

PFOA is a substance of very high concern with respect to its health and environmental properties. PFOA is harmful to the reproductive system, is a possible carcinogen, toxic and harmful to human health through repeated exposure, and may cause serious eye damage. PFOA does not degrade in the environment. PFOA is a persistent, bio-accumulating and toxic (PBT) substance.

The notification concludes it is impossible to establish an acceptable level for substances with such properties in the environment, and that emissions and exposure should be limited to the greatest extent possible.

(UNEP/FAO/RC/CRC.16/4 Norwegian notification)

4	**Environmental fate and effects**	
4.1	**Fate**	Once in the environment, PFOA is extremely persistent and not known to undergo significant abiotic or biotic degradation under relevant environmental conditions. (UNEP/FAO/RC/CRC.14/8, Canadian notification)
4.1.1	**Soil**	Based on high persistence, it was not possible to calculate half-lives in soil or sediment. (UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)
4.1.2	**Water**	PFOA is highly soluble in water and typically present as an anion (conjugate base) in solution. It has low vapour pressure; therefore, the aquatic environment is expected to be its primary sink, with some additional partitioning to sediment. (UNEP/FAO/RC/CRC.14/8, Canadian notification)
4.1.3	**Air**	On the basis of the available data, abiotic degradation of PFOA in the atmosphere is expected to be slow. The atmospheric lifetime of PFOA has been predicted to be 130 days (conclusion by analogy from short-chain perfluorinated acids). (UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)
4.1.4	**Bioconcentration**	PFOA does not seem to bio-concentrate in water-breathing animals. The high water solubility of PFOA enables fish to quickly excrete this substance via gill permeation, facilitated by the high water throughput. In air-breathing animals, PFOA has been found in terrestrial species as well as in endangered species as polar bear and in animals that may become endangered in near future (such as narwhal and beluga whale). Once taken up in the body, PFOA tends to partition to liver and blood. BMFs range from 1.3 – 125 for selected predator prey relationship TMFs range from 1.1 – 13 for selected food chains. (UNEP/FAO/RC/CRC.16/4 Norwegian notification) The assessment of bioaccumulation for PFOA is complicated by its physical properties, which make assessment of log K_{ow}, BCF and BAF approaches challenging. PFOA does not accumulate in water-breathing animals according to the criteria of the Stockholm Convention. This can be explained by the way that fish process and excrete PFOA through their gills. PFOA biomagnifies in air breathing mammals. PFOA has been detected within the body tissues of air-breathing aquatic species. For terrestrial species, the presence of PFOA is readily detected, with a number of studies indicating BMF and TMF scores of greater than 1. There is evidence that PFOA bioaccumulates in air-breathing mammals and other terrestrial species, including humans. (UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)
4.1.5	**Persistence**	PFOA is extremely stable within the natural environment due to its chemical properties and does not degrade under environmentally relevant conditions. Based on the available experimental evidence it is concluded that PFOA is highly persistent in all environmental compartments, with a strong resistance to all conventional mechanisms of degradation under relevant environmental conditions. Within the aquatic compartment under natural environmental conditions, PFOA has a half-life of greater than 92 years with the most likely value of 235 years and shows no

obvious decay from photodegradation. In aquatic environments where PFOA undergoes indirect photolysis, the half-life was estimated to be longer than 349 days.

(UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)

4.2 Effects on non-target organisms

4.2.1 Terrestrial vertebrates

The potential impact of exposure to perfluorinated compounds on liver lesions was investigated in East Greenland polar bears. Liver parameters examined included mononuclear cell infiltrations, lipid granulomas, steatosis, Ito cells and bile duct hyperplasia/portal fibrosis. The population consisted of 28 females and 29 males harvested by local hunters between 1999 and 2002. Liver samples were analyzed for PFOS, perfluorononanoic acid, perfluoroundecanoic acid, perfluorodecanoic acid, perfluorotetradecanoic acid, PFOA, perfluorooctanesulfonamide, perfluorodecanoate and perfluorohexanesulfonate. In 23 cases, the concentration of PFOA was below the detection limit (0.0012 µg/g-wwt). Liver samples were also analyzed for several perfluorinated compounds including C9, C10, C11, C12 and C13 PFCAs. Sixty-five percent of the polar bears had total PFAS concentrations above 1 µg/g-wwt. In female bears, the total PFAS concentration ranged from 0.256 to 2.77 µg/g-wwt; in male bears, the total PFAS concentration ranged from 0.114 to 3.052 µg/g-wwt. All PFAS compounds in the analysis were summed, so a direct cause–effect correlation with a particular perfluorinated compound, such as PFOA, cannot be determined. East Greenland polar bears are also contaminated with other substances, such as organochlorines (polychlorinated biphenyls or PCB, dichlorodiphenyltrichloroethane or DDT) and mercury, which may function as confounding synergistic co-factors in the development of the lesions. The authors concluded that the statistical analysis did not answer the question of whether chronic exposure to perfluorinated compounds is associated with liver lesions in polar bears; however, these lesions were similar to those produced by perfluorinated compounds under laboratory conditions.

(UNEP/FAO/RC/CRC.14/8, Canadian notification)

There is experimental evidence in terrestrial organisms showing the potential for PFOA to induce alterations to the liver, endocrine dysfunction, developmental toxicity and tumour formation. Adverse effects include alterations in sexual maturation and pubertal timing, changes in mammary gland development as well as induction of a variety of tumours. There are some indications of PFOA-mediated immunomodulation. Because of the tendency of PFOA to bioaccumulate, PFOA concentrations in polar bears might increase over time and approach exposures resulting in harm.

(UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)

4.2.2 Aquatic species

In traditional toxicity studies, PFOA exhibits moderate to low acute toxicities in pelagic organisms, including fish (70–2470 mg/L). PFOA exhibits low chronic toxicities in benthic organisms (>100 mg/L). There is one study on the toxicity of PFOA and its salts in avian wildlife. In this study, PFOA was found to have no effect on embryonic pipping success for white leghorn chickens at concentrations up to 10 µg/g of embryos. However, PFOA accumulated in the liver of these embryos to concentrations 2.9 – 4.5 times greater than the initial whole-egg concentration.

A study examined freshwater male tilapia (*Oreochromis niloticus*) as the *in vitro* model to detect the induction of vitellogenin. Vitellogenin is an egg yolk precursor protein expressed in females of fish, amphibians, reptiles (including birds), insects and the platypus. In the presence of substances that affect endocrine function, males can also express the vitellogenin gene. Cultured male tilapia hepatocytes were exposed to PFOA, 4:2 FTOH, 6:2 FTOH and 8:2 FTOH for 48 hours. A dose-dependent induction of vitellogenin was observed in PFOA- and 6:2 FTOH-treated cells, whereas vitellogenin remained unchanged for 4:2 FTOH and 8:2 FTOH. The estimated 48-hour median effective concentration (EC50) values were 2.9×10^{-5} M (12 mg/L) for PFOA and 2.8×10^{-5} M (12.9 mg/L) for 6:2 FTOH. In the time course study, vitellogenin induction took place at 48 hours (PFOA), 72 hours (4:2 FTOH), 12 hours (6:2 FTOH) and 72 hours (8:2 FTOH) and increased further after 96 hours of exposure. Co-exposure to a mixture of individual perfluorinated compounds and 17β-estradiol for 48 hours significantly inhibited 17β-estradiol-

induced hepatocellular vitellogenin production in a dose-dependent manner, except for 4:2 FTOH. The estimated 48-hour median inhibitory concentration (IC50) values were 5.1×10^{-7} M(0.21 mg/L) for PFOA, 1.1×10^{-6} M (0.51 mg/L) for 6:2 FTOH and 7.5×10^{-7} M (0.35 mg/L) for 8:2 FTOH. In order to further investigate the estrogenic mechanism, the hepatocytes were co-exposed to a mixture of PFOA and 6:2 FTOH plus the known estrogen receptor inhibitor tamoxifen for 48 hours. The overall results demonstrated that PFOA and FTOHs have estrogenic activities and that exposure to a combination of 17β-estradiol and PFOA or FTOHs produces anti-estrogenic effects. The results of the estrogen receptor inhibition assay further suggested that the estrogenic effect of PFOA and FTOHs may be mediated by the estrogen receptor pathway in primary cultured tilapia hepatocytes. A study assessed the effects of PFOA on male and female rare minnows (*Gobiocypris rarus*) at concentrations of 3, 10 and 30 mg/L for 28 days. Exposure to PFOA at 3 mg/L elicited moderate hepatocellular hypertrophy in the livers of both male and female fish. Male rare minnows exposed to PFOA at 10 mg/L showed eosinophilic hyaline droplets in the cytoplasm of the hepatocytes; female rare minnows displayed more eosinophilic hyaline droplets in the cytoplasm of the hepatocytes, hepatocellular hypertrophy and vacuolar degeneration. Rare minnows exposed to PFOA at 30 mg/L showed severe hepatic histopathological changes and disruption of mitochondrial functions. The inhibition of the thyroid hormone biosynthesis genes and the induction of estrogen-responsive genes may indicate a role in endocrine function. Another study further identified the potential protein biomarkers for PFOA exposure in the livers of the rare minnows at 3, 10 and 30 mg/L for 28 days, finding the abundance of 34 and 48 protein spots altered in males and females, respectively. These proteins were involved in intracellular fatty acid transport, oxidative stress, macromolecule catabolism, the cell cycle, maintenance of intracellular Ca^{2+} homeostasis and mitochondrial function. In another article, the authors studied the in vivo effects of waterborne PFOA on the expression of hepatic estrogen-responsive genes, vitellogenin, and estrogen receptor and on the gonadal development in freshwater rare minnow (*Gobiocypris rarus*). The study showed mature females exposed to 3, 10, and 30 mg/L PFOA for 28 days had degenerating vitellogenic-stage oocytes (atresia) in the ovaries. In males exposed to 10 mg/L PFOA, primary growth–stage oocytes (pre-vitellogenic oocytes) developed in some testes. The number of sperm and various stages of germ cells within the spermatogenic cycle in the 10 and 30 mg/L PFOA treatments were lower than those in control males. PFOA increased hepatic vitellogenin concentration and induced testis-ova gonads in mature male rare minnows at 10 and 30 mg/L for 28 days. It was shown that PFOA can disrupt the activity of estrogen by inducing hepatic estrogen-responsive genes in males, although the mechanism of development of testes-ova in rare minnows by PFOA exposure is not known.

The toxicity of PFOA was examined with respect to the multixenobiotic resistance mechanism in the marine mussel, *Mytilus californianus*. This mechanism acts as a cellular first line of defence against broad classes of xenobiotics exporting moderately hydrophobic chemicals from cells via adenosine triphosphate (ATP)–dependent, transmembrane transport proteins. The most studied transporter is the P-glycoprotein, which is a fragile defence mechanism and can be compromised by some xenobiotics. This increased sensitivity, referred to as chemosensitization, arises from the ability of the P-glycoprotein to recognize and bind to multiple xenobiotic substrates, resulting in the saturation of the binding capacity. Non-toxic substances can also be chemosensitizers and cause adverse effects on organisms by allowing normally excluded toxic substances to accumulate in the cell. PFOA at 50 μM (20 mg/L) was found to significantly inhibit the P-glycoprotein in *Mytilus californianus* and thus is a chemosensitizer for that organism. The study also showed that this inhibition was reversible once the marine mussel was removed from contamination and placed in clean seawater.

The effect of PFOA on immune function and clinical blood parameters has been examined in bottlenose dolphins and sea turtles from Florida, Georgia and South Carolina. It should be noted that a direct cause–effect relationship cannot be clearly established, as there may be other co-occurring contaminants. The results revealed that there may be increases in indicators of inflammation and immunity in bottlenose dolphin blood parameters in relation to PFOA, suggesting that PFOA may alter biomarkers of health in marine mammals. Examples of biomarkers analyzed in

bottlenose dolphins include absolute numbers of lymphocytes, serum triglyceride, serum total protein, serum albumin, serum cortisol, C-reactive protein, lysozyme activity and B-cell proliferation. Serum triglyceride exhibited stronger relationships to PFOA in females than in males. Lipopolysaccharide-induced lymphocyte proliferation (B-cell proliferation) had positive but weak correlations with PFOA in male bottlenose dolphins, and a strong correlation was observed between PFOA and lysozyme activity (a measurement of innate immunity) in the same species. However, in another study, no correlations were found between any perfluorinated compound, including PFOA, and blood chemistry parameters (e.g. cholesterol, creatinine, albumin, total serum ion etc.) for the northern fur seal (*Callorhinus ursinus*).

Low levels of PFAs may also alter biomarkers of health in loggerhead sea turtles. Examples of biomarkers analyzed in loggerhead sea turtles include plasma total protein, plasma globulin, T-cell proliferation, plasma lysozyme activity and B-cell proliferation.

(UNEP/FAO/RC/CRC.14/8, Canadian notification)

Acute aquatic toxicity is low in standard ecotoxicity tests; moderate to low acute toxicities are seen in pelagic organisms including fish and low chronic toxicities in benthic organisms. Adverse effects include intergenerational toxicity in the first offspring generation and some PFOA-mediated toxicity in freshwater algae and other aquatic organisms.

(UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile)

4.2.3 Honeybees and other arthropods

Not available.

4.2.4 Earthworms

Not available.

4.2.5 Soil microorganisms

The soil-dwelling nematode *Caenorhabditis elegans* has been shown to be a suitable test organism, showing both lethal and sublethal effects, in the ecotoxicological assessments of liquid and soil media. Acute lethal toxicity and multigenerational sublethal toxicity (fecundity and reproduction) were examined using PFOA concentrations of 0, 0.01 mM (4.14 mg/L), 0.1 mM (41.4 mg/L), 0.5 mM (207 mg/L), 1.0 mM (414.07 mg/L) and 5.0 mM (2100 mg/L) for 48 hours. All concentrations up to 0.1 mM (41.4 mg/L) showed no acute lethality until 48 hours. Acute lethality appeared at concentrations greater than 0.5 mM (207 mg/L) and did not depend on the incubation time. EC_{50}s were calculated for 1 hour (3.85 mM or 1590 mg/L), 2 hours (2.80 mM or 1160 mg/L), 3 hours (2.70 mM or 1120 mg/L), 4 hours (2.65 mM or 1100 mg/L), 24 hours (2.75 mM or 1140 mg/L) and 48 hours (2.35 mM or 973 mg/L). In the multi-generational test, generation–response and concentration-response relationships were not observed for PFOA.

(Government of Canada, 2012a)

4.2.6 Terrestrial plants

In seed germination and 5-day root elongation toxicity tests on lettuce (*Lactuca sativa*), cucumber (*Cucumis sativus*) and pakchoi (*Brassica rapa chinensis*), PFOA had no effect on cucumber seed germination, with both LC_{50} and NOEC values greater than 2000 mg/L. The LC_{50} and NOEC values for lettuce seed germination were 1734 and 1000 mg/L, respectively. The LC_{50} and NOEC values for pakchoi seed germination were 579 and 250 mg/L, respectively. The EC_{50} for root elongation for the three species ranged from 263 to 1254 mg/L. PFOA almost completely inhibited lettuce and pakchoi root growth at or above 1000 mg/L. NOECs for root elongation for the three species ranged from <62.5 to 250 mg/L.

In a study on the soil-to-plant carryover of a mixture of PFOA/PFOS on spring wheat, oats, potatoes, maize, and perennial ryegrass. Concentrations ranged from 0.25 to 50 mg/kg of PFOA/PFOS as an aqueous solution. PFOA concentrations were higher than PFOS in all plants except for potatoes with uptake/storage more intensive in the vegetative portion than the storage organ. Visible abnormalities were noted at concentrations > 10 mg/kg. At 25 – 50 mg/kg PFOA/PFOS, necrosis was observed in both oats and potatoes, a yellowing of the ryegrass leaves, and diminished growth for spring wheat.

(Government of Canada, 2012a)

		In plants, PFOA can cause visible abnormalities and alter root growth. (UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile).
5	**Environmental Exposure/Risk Evaluation**	
5.1	**Terrestrial vertebrates**	Temporal trends were found for PFOA concentrations in polar bears (1972 – 2002 and 1984 – 2006) and sea otters (1992 – 2002). PFOA doubling time in liver tissue was calculated to be 7.3 ± 2.8 years for Baffin Island polar bears and 13.9 ± 14.2 years for Barrow, Alaska, polar bears; central East Greenland polar bears showed an annual increase of 2.3% in PFOA concentrations. Concentrations of PFOA also increased significantly over a 10-year period for adult female sea otters. The risk quotient for Canadian mammalian wildlife (i.e., polar bears) is less than 1; however, due to the persistence of the substance, its tendency to accumulate and biomagnify in a variety of terrestrial and marine mammals, its hepatotoxicity, and the upward temporal trend of PFOA concentrations in polar bears and some other species, PFOA concentrations in polar bears may approach exposures resulting in harm. The assessment is based on a weight of evidence approach regarding persistence, bioaccumulation, temporal trends in some species (i.e. the polar bear), long-range transport and the widespread occurrence and concentrations of PFOA in the environment and in biota (including remote areas of Canada). Based on the information presented in the screening assessment, it is concluded that PFOA, its salts and its precursors are entering or may be entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity. (UNEP/FAO/RC/CRC.14/8, Canadian notification) The notification concludes it is impossible to establish an acceptable level for substances (like PFOA) with such properties in the environment, and that emissions and exposure should be limited to the greatest extent possible. (UNEP/FAO/RC/CRC.16/4 Norwegian notification)
5.2	**Aquatic species**	Trace levels of PFOA have been measured in Canadian freshwater (ND–11.3 μg/L) and freshwater sediments (0.3–7.5 μg/kg). PFOA has also been detected in a variety of Canadian biota (ND–90 μg/kg wet weight (kg-wwt) tissue) in southern Ontario and the Canadian Arctic. The risk quotients for pelagic organisms indicate a low likelihood of risk from exposures at current concentrations in the aquatic environment. (UNEP/FAO/RC/CRC.14/8, Canadian notification) PFOA has been detected within the body tissues of air-breathing aquatic species. (UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile) The notification concludes it is impossible to establish an acceptable level for substances (like PFOA) with such properties in the environment, and that emissions and exposure should be limited to the greatest extent possible. (UNEP/FAO/RC/CRC.16/4 Norwegian notification)
5.3	**Honey bees**	Not available.
5.4	**Earthworms**	Not available.
5.5	**Soil microorganisms**	Not available.
5.6	**Summary – overall risk evaluation**	Based on the available experimental evidence, it is concluded that PFOA is highly stable and persistent within the natural environment. PFOA is unlikely to degrade under conditions present in the natural environment and has been shown to have long half-lives within the environment. Monitoring data show that PFOA in soil leaches over time and can be a long-term contamination source to underlying groundwater. PFOA has been found in marine, limnetic and terrestrial biota worldwide, and bioaccumulation of PFOA occurs across trophic levels. Assessment of bioaccumulation for PFOA is complicated by its physical properties as a surfactant, which make analysis for development of log Kow values not directly possible. PFOA accumulates and biomagnifies in air breathing animals and other terrestrial species

including humans but not in water breathing animals as fish excrete PFOA through their gills.

Monitoring of water, snow, air, sediment and biota at remote locations all detect the presence of PFOA. Equally, environmental modeling data and other information enable to conclude that PFOA meets the criterion for long range transport.

PFOA exhibits adverse effects for both terrestrial and aquatic species. Ecotoxicity data indicate a low acute toxicity for aquatic organisms. There is also experimental evidence in terrestrial organisms showing the potential for PFOA to induce changes in liver function, endocrine function, development as well as immune responses, and induction of tumours has been shown in rats exposed to PFOA. The adverse effects of PFOA in biota have not yet been elucidated, but because of the tendency of PFOA to bioaccumulate, PFOA concentrations in biota, especially polar bears, might increase over time and approach exposures resulting in harm.

(UNEP/POPS/POPRC.12/11/Add.2, POPRC Risk Profile Chapter 3)

Annex 2 – Details on final regulatory actions reported

Country Name: Canada

1	**Effective date(s) of entry into force of actions**	23 December 2016.
	Reference to the regulatory document	*Prohibition of Certain Toxic Substances Regulations, 2012* (SOR/2012-285), as amended, 2016 (SOR/2016-252) under the *Canadian Environmental Protection Act, 1999* (CEPA). http://www.gazette.gc.ca/rp-pr/p2/2016/2016-10-05/html/sor-dors252-eng.html
2	**Succinct details of the final regulatory action(s)**	Perfluorooctanoic acid, which has the molecular formula $C_7F_{15}CO_2H$, its salts, and its precursors (collectively referred to as PFOA) and products containing them are subject to the *Prohibition of Certain Toxic Substances Regulations, 2012* (the Regulations) as amended in 2016, under the *Canadian Environmental Protection Act, 1999* (CEPA). The *Prohibition of Certain Toxic Substances Regulations, 2012* prohibit the import, manufacture, use, sale and offer for sale of PFOA, and products containing PFOA, with a limited number of exemptions.
3	**Reasons for action**	The regulatory action was based on concerns related to the environment.
4	**Basis for inclusion into Annex III**	The regulatory action was taken to protect environment. The regulatory action was based on a risk evaluation taking into account the prevailing conditions in Canada. The screening assessment, on which the regulatory action in based on and that was performed in Canada (http://www.ec.gc.ca/ese-ees/default.asp?lanq=En&n=370AB133-1) makes use of the extensive information on uses, releases and environmental levels of PFOA in Canada, including the Canadian Arctic.
4.1	**Risk evaluation**	The assessment is based on a weight of evidence approach regarding persistence, bioaccumulation, temporal trends in some species (i.e. the polar bear), long-range transport and the widespread occurrence and concentrations of PFOA in the environment and in biota (including remote areas of Canada). Based on the information presented in the screening assessment, it is concluded that PFOA, its salts and its precursors are entering or may be entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity. (UNEP/FAO/RC/CRC.14/8 Canadian notification)
4.2	**Criteria used**	Risk to environment.
	Relevance to other States and Region	The notification states that, once in the environment, PFOA is extremely persistent and not known to undergo significant abiotic or biotic degradation under relevant environmental conditions. PFOA is highly soluble in water and typically present as an anion (conjugate base) in solution. It has low vapour pressure; therefore, the aquatic environment is expected to be its primary sink, with some additional partitioning to sediment. The presence of PFOA in the Canadian Arctic is likely attributable to the long-range transport of PFOA (e.g., via ocean currents) and/or volatile precursors to PFOA (e.g., via atmospheric transport). PFOA has been detected at trace levels in the northern hemisphere. The notification states that a number of countries and organizations (including the European Union, Norway, the United States of America, the Stockholm Convention on Persistent Organic Pollutants and the Protocol to the United Nations Economic Commission for Europe Convention on Long-Range Transboundary Air Pollution of 1979) either have put in place or are proposing management measures to control the manufacture, import, use and releases of perfluoroalkyl substances (PFAS) and manufactured products containing PFAS.

		Given the hazards and long-range transport of this substance as described in the screening assessment on which the regulatory action is based, any state or region in which exposure or release is possible may find the regulatory action relevant.
5	**Alternatives**	In January 2006, the U.S. EPA introduced a voluntary 2010/2015 PFOA Stewardship Program to reduce facility emissions and product content of PFOA and related chemicals on a global basis and to work toward eliminating emissions and product content of these chemicals by 2015. This Stewardship Program has been a major driver for companies to reduce residuals in products and to switch from PFOA products to safer alternatives. The U.S. EPA is also reviewing substitutes for PFOA, PFOS, and other long-chain perfluorinated substances as part of its review process for new chemicals under EPA's New Chemicals Program. Over 150 alternatives of various types have been received and reviewed by EPA. Under the U.S. EPA's New Chemical Review of Alternatives for PFOA and related chemicals, shorter chain-length perfluorinated telomeric substances have been notified as alternatives for a variety of uses including, for example, textile, carpet and paper additive uses and tile surface treatments. The major industry users in the global community have replaced uses of C-8 and higher homologues with alternatives. (UNEP/FAO/RC/CRC.14/8 Canadian notification)
6	**Waste management**	The notifying Party did not provide information on waste management of PFOA or articles containing it.
7	**Other**	None.

Country Name: Norway		
1	**Effective date(s) of entry into force of actions**	4 July 2020
	Reference to the regulatory document	The Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) Act No. 516 of May 2008, as amended on 3 October 2017, implementing COMMISSION REGULATION (EU) 2017/1000 of 13 June 2017.
2	**Succinct details of the final regulatory action(s)**	PFOA, its salts and PFOA-related substances shall not be manufactured or placed on the market as substances on their own, or be used in the production of or placed on the market in another substance, as a constituent, a mixture or an article, in a concentration equal to or above 25 ppb of PFOA including its salts or 1 000 ppb of one or a combination of PFOA-related substances. The restriction identifies several exemptions, some of which are time-limited and some open-ended.
3	**Reasons for action**	The regulatory action was based on concerns related to human health and the environment.
4	**Basis for inclusion into Annex III**	The final regulatory action was taken to protect human health and the environment. The regulatory action was based on a risk evaluation taking into account the prevailing conditions in Norway.
4.1	**Risk evaluation**	In the Norwegian "Evaluation of consequences of regulating PFOA and selected salts and esters of PFOA in consumer products", the following concerns were put forward for the proposed regulation: PFOA is present in the blood of the general population, breast milk and in umbilical cord blood. PFOA is eliminated from the body very slowly. Humans are exposed to PFOA by consuming contaminated foods or water, by breathing air that is polluted as well as by ingesting dust. Fish is an important source of exposure via food. The fetus is exposed to PFOA via umbilical cord blood and newborns are exposed via breast milk. The intake for infants via breast milk can be greater than the intake via food for adults. Infants can also come into direct contact through carpeting, and swallowing dust can be an important contributor to exposure. PFOA is a substance of very high concern with respect to its health and environmental properties. PFOA is harmful to the reproductive system, carcinogenic, toxic and harmful to human health through repeated exposure and may cause serious eye damage. PFOA does not degrade in the environment. PFOA is a substance similar to persistent, bio-accumulating and toxic (PBT) substances or a substance of equal concern. It is impossible to establish an acceptable level for substances with such properties in the environment, and emissions and exposure should be limited to the greatest extent possible. (UNEP/FAO/RC/CRC.16/4 Norwegian notification)
4.2	**Criteria used**	Risk to human health and the environment.
	Relevance to other States and Region	The notification states that concerns similar to those identified in Norway are likely to be encountered in other countries where the substance is used. PFOA is present in various globally distributed products. Adaptation of manufacturing methods to meet the Norwegian requirements may lead to reduced levels of PFOA in products in other countries as well. Several textile brands have phased out the use of perfluorinated compounds for water repellence treatment because of the negative attention directed at such compounds by various stakeholders. The notification also cites Norway's "Evaluation of consequences of regulating PFOA and selected salts and esters of PFOA in consumer products", according to which PFOA is transported long distances via air and sea currents, and its presence has been detected in the Arctic in a variety of species, including sea birds, seals and polar bears. The substance has also been identified as CMR and PBT, which are relevant concerns for any state or region in which PFOA may be released.

5	**Alternatives**	U.S. EPA's review of alternatives to perfluorinated chemical substances has been ongoing since 2000 and is consistent with the approaches to alternatives encouraged under the 2010/15 PFOA Stewardship Program. Through June 2008, over 100 alternatives of various types have been received and reviewed by U.S. EPA. https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/fact-sheet-20102015-pfoa-stewardship-program Additional information on alternatives could also be found in these two publications: OECD/UNEP Global PFC Group, Synthesis paper on per- and polyfluorinated chemicals (PFCs), 2013. Wang, Z., Cousins, I.T., Scheringer, M., Hungerbühler, K., 2013. Fluorinated alternatives to long-chain perfluoroalkyl carboxylic acids (PFCAs), perfluoroalkane sulfonic acids (PFSAs) and their potential precursors. Environ Int 60, 242-248 (UNEP/FAO/RC/CRC.14/8 Norwegian notification)
6	**Waste management**	The notifying Party did not provide information on waste management of PFOA or articles containing it.
7	**Other**	None.

Annex 3 – Addresses of designated national authorities	
Canada ***C***	
From PIC website (March 2019): Ms. Nicole Folliet Director Chemical Production Division Environment and Climate Change Canada 351 St. Joseph Boulevard K1A 0H3 Gatineau Québec Canada Phone: +1 819 420 7708 Fax: +1 819 938 4218 Email: nicole.folliet@canada.ca; ec.substancedexportationcontrolee-exportcontrolledsubstance.ec@canada.ca	
Norway ***C***	
From PIC website (September 2020): Ms. Christel Moræus Olsen Senior Advisor, Rotterdam Convention Designated National Authority for Industrial Chemicals and Biocides Section for Biocides and Declaration of Chemicals/ Chemicals and Waste Department Norwegian Environment Agency Torgarden P.O. Box 5672 7485 Trondheim Norway Phone: +47 735 80 500 Fax: +47 735 80 501 Email: christel.moraeus.olsen@miljodir.no, pic@miljodir.no	Ms. Mitsuko Komada Senior Advisor, Rotterdam Convention Designated National Authority for Industrial Chemicals and Biocides Norwegian Environment Agency Torgarden P.O. Box 5672 7485 Trondheim Norway Phone: +47 73 580 500 Fax: +47 73 580 501 Email: mitsuko.komada@miljodir.no, pic@miljodir.no

C Industrial chemicals

Annex 4 – References

Regulatory actions

Canada
Prohibition of Certain Toxic Substances Regulations, 2012 (SOR/2012-285), as amended, 2016 (SOR/2016-252) under the Canadian Environmental Protection Act, 1999 (CEPA).
http://www.gazette.gc.ca/rp-pr/p2/2016/2016-10-05/html/sor-dors252-eng.html

Norway
COMMISSION REGULATION (EU) 2017/1000 of 13 June 2017 amending Annex XVII to Regulation (EC) No 1907/2006 of the European Parliament and of the Council concerning the Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) as regards perfluorooctanoic acid (PFOA), its salts and PFOA-related substances

Supporting documentation provided by Canada

- Government of Canada. August 2012a. Screening Assessment Report on Perfluorooctanoic Acid, Its Salts and Its Precursors. http://www.ec.gc.ca/ese-ees/default.asp?lang=En&n=370AB133-1
- Government of Canada. August 2012b. Proposed Risk Management Approach for Perfluorooctanoic Acid (PFOA), its Salts, and its Precursors and Long-Chain (C9-C20) Perfluorocarboxylic Acids (PFCAs), their Salts, and their Precursors. http://www.ec.gc.ca/ese-ees/default.asp?lang=En&n=451C95ED-1
- Environment Canada and Health Canada. October 2016. Regulatory Impact Analysis Statement, Regulations Amending the Prohibition of Certain Toxic Substances Regulations, 2012.

  http://www.gazette.gc.ca/rp-pr/p2/2016/2016-10-05/html/sor-dors252-eng.html

Supporting documentation provided by Norway

- PFOA in Norway; Survey on national sources, TA-2354/2007. (See UNEP/FAO/RC/CRC.14/INF/13). https://www.researchgate.net/publication/299430492_PFOA_in_Norway_Survey_of_national_sources /Per- and polyfluorinated substances in the Nordic Countries Use, occurrence and toxicology TemaNord 2013:542
- Perfluorinated alkylated substances (PFAS) in the Nordic environment. TemaNord 2004:552
- Impact assessment of regulating perfluorooctanoic acid (PFOA) and individual PFOA salts and esters in consumer products. The Norwegian version is available online: «Vurdering av konsekvenser av regulering av PFOA og enkelte salter og estere av PFOA i forbrukerprodukter". (See UNEP/FAO/RC/CRC.14/INF/13). https://docplayer.me/6087932-Vurdering-av-konsekvenser-av-regulering-av-pfoa-og-enkelte-salter-og-estere-av-pfoa-i-forbrukerprodukter.html
- Perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA) and their salts. Scientific Opinion of the Panel on Contaminants in the Food chain. The EFSA Journal 2008, 653, 16-131
- ECHA: Pentadecafluorooctanoic acid (PFOA) as a substance of very high concern because of its CMR and PBT properties. 14 June 2013.
- Fromme, H.; Mosch, C.; Morovitz, M.; Alba-Alejandre, I.; Boehmer, S.; Kiranoglu, M.; Faber, F.; Hannibal, I.; Genzel-Boroviczény, O.; Koletzko, B.; Völkel, W. Pre- and postnatal exposure to perfluorinated compounds (PFCs). Environ Sci Technol 2010, 44, 7123–7129.
- Thomsen, C.; Haug, L. S.; Stigum, H.; Frøshaug, M.; Broadwell, S. L.; Becher, G. Changes in concentrations of perfluorinated compounds, polybrominated diphenyl ethers, and polychlorinated biphenyls in Norwegian breast-milk during twelve months of lactation. Environ Sci Technol 2010, 44, 9550–9556.
- Haug, L.S., Salihovic, S., Jogsten, I.E., Thomsen, C., van Bavel, B., Lindström, G., Becher, G. 2010a. Levels in food and beverages and daily intake of fluorinated compounds in Norway. Chemosphere, 80, 1137–1143.
- Haug, L.S., Thomsen, C., Brantsæter, A.L., Kvalem, H.E., Haugen, M., Becker, G., Alexander, J., Meltzer, H.M., Knutsen, H.K. 2010b. Diet and particulary seafood are major sources of perfluorinated compounds in humans. Environ. Int., 36, 772–778.
- Haug, L.S., Huber, S., Becher, G., Thomsen, C. 2011. Characterisation of human exposure pathways to perfluorinated compounds – comparing exposure estimates with biomarkers of exposure. Environ Int 37, 687–693.
- Granum B, Haug LS, Namork E, Stølevik SB, Thomsen C, Aaberge IS, van Loveren H, Løvik M, Nygaard UC. Pre-natal exposure to perfluoroalkyl substances may be associated with altered vaccine antibody levels and immune-related healthoutcomes in early childhood. J Immunotoxicol. 2013,10:373-9.

- Daae et al 2009: Kjemisk eksponering og effekter på luftveiene blant profesjonelle skismørere, ISSN nr. 1502-0932
- Smithwick M, Norstrom R.J., Mabury S.A., Solomon K., Evans T.J., Stirling I., Taylor M.K., Muir D.C.G. 2006. Temporal trends of perfluoroalkyl contaminants in polar bears (Ursus maritimus) from two locations in the North American arctic, 1972-2002. Environ. Sci. Technol. 40(4):1139-1143.
- Dietz et al. 2008; "Increasing Perfluoroalkyl Contaminants in East Greenland Polar Bears (Ursus maritimus): A New Toxic Threat to the Arctic Bears", Environ. Sci. Technol. 2008, 42, 2701–2707
- Tom Erik Økland and Kristina Skoog; TA-2450/2008. (See UNEP/FAO/RC/CRC.14/INF/13).
- Aquateam, 2010. Undersøkelse av miljøgifter ved fire norske renseanlegg: PFOA, Bisfenol A, Triklosan, Siloksan (D5), Dodecylfenol og 2,4,6-Tri-tert.betylfenol. (Study of priority substances in four Norwegian processing plants: PFOA, Bisphenol A, Triclosan, Siloksan (D5), Dodecylfenol og 2,4,6-Tri-tert.betylphenol) Klif TA 2636/2010. (See UNEP/FAO/RC/CRC.14/INF/13).
- Evenset et al. 2005: Miljøgifter i marine sedimenter, Isfjorden, Svalbard. (Priority substances in marine sediments, Isfjordenm Svalbard.) Akvaplan-NIVA-rapport APN-414. 3341.
- Bakke, T., Fjeld, E., Skaare, B., Berge. J.A., Green, N., Ruus, A., Schlabach, M., and Botnen, H., 2007. Kartlegging av metaller og utvalgte nye organiske miljøgifter 2006. Krom, arsen, perfluoralkylstoffer, dikloretan, klorbenzener, pentaklorfenol, HCBD og DEHP. (Mapping out of metals and select new organic environmental toxins 2006; Chromium, arsenic, perfluoroalkyl substances, dichloroethane, chlorobenzenes, pentachlorophenol, HCBD and DEHP) SFT TA2284/2007. NIVA report 5464-2007. 105s.
- Manø S, Herzke D, Schlabach M, Nye miljøgifter i luft (New priority substances in air) (TA-2408/2008). (See UNEP/FAO/RC/CRC.14/INF/13).
- AMAP, 2009. Arctic Pollution 2009 (POPs, Human Health, Radioactivity). Arctic Monitoring and Assessment Programme (AMAP), Oslo, Norway. xi+83 pp
- OECD/UNEP Global PFC Group, Synthesis paper on per- and polyfluorinated chemicals (PFCs), 2013.
- Wang, Z., Cousins, I.T., Scheringer, M., Hungerbühler, K., 2013. Fluorinated alternatives to long-chain perfluoroalkyl carboxylic acids (PFCAs), perfluoroalkane sulfonic acids (PFSAs) and their potential precursors. Environ Int 60, 242-248
- IFA, Institut fur Arbeitsscultz der Deutschen Gesetzlichen Unfallversicherung (See UNEP/FAO/RC/CRC.14/INF/13).
- ECHA, 2014. Annex XV Restriction Report: Proposal For a Restriction - Perfluorooctanoic acid (PFOA), PFOA salts and PFOA - related substances http://echa.europa.eu/documents/10162/e9cddee6-3164-473d-b590-8fcf9caa50e7
- Perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA) and their salts. Scientific Opinion of the Panel on Contaminants in the Food chain. The EFSA Journal 2008, 653, 16-131.
- ECHA, E.C.A., 2008. Summary of Classification and Labelling: Harmonised classification – Annex VI of Regulation (EC) No 1272/2008 (CLP Regulation) – PFOS. vol. 2012.
- (ECHA), E.C.A., 2011. Opinions of the Committee for Risk Assessment on proposals for harmonised classification and labelling – PFOA. vol. 2012.
- ECHA/RAC/CLH report 2011. Annex 1, Background document to the Opinion proposing harmonised classification and labelling at Community level of Perfluorooctanoic acid (PFOA).
- OECD, 2006, SIDS Initial Assessment Report PFOA.
- Environment Canada (2012): Screening Assessment Report; Perfluorooctanic Acid, its Salts, and its Precursors.
- Verreault J, Berger U, Gabrielsen GW. 2007. Trends of perfluorinated alkyl substances in herring gull eggs from two coastal colonies in northern Norway: 1983–2003. Environ Sci Technol 41(19): 6671–6677.
- Bytingsvik J, van Leeuwen SP, Hamers T, Swart K, Aars J, Lie E, Nilsen EM, Wiig O, Derocher AE, Jenssen BM. Perfluoroalkyl substances in polar bear mother-cub pairs: a comparative study based on plasma levels from 1998 and 2008. Environ Int. 2012, 49:92-9.

Other Documents

UNEP/FAO/RC/CRC.14/8. (2018). Perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds: notifications of final regulatory action.

UNEP/FAO/RC/CRC.16/4. (2020). Perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds: notifications of final regulatory action.

UNEP/POPS/POPRC.13/INF/6/Add.1. (2017). Supporting information related to the draft risk management evaluation on pentadecafluorooctanoic acid (CAS No: 335-67-1, PFOA, perfluorooctanoic acid), its salts and PFOA-related compounds: Non-exhaustive lists of substances covered and not covered by the draft risk management evaluation.

Decision SC-9/13. (2019), see UNEP/POPS/COP.9/30, annex.

UNEP/POPS/POPRC.12/11/Add.2. (2016). Risk profile on pentadecafluorooctanoic acid (CAS No: 335-67-1, PFOA, perfluorooctanoic acid), its salts and PFOA-related compounds.

UNEP/POPS/POPRC.13/7/Add.2 (2017). Risk management evaluation on pentadecafluorooctanoic acid (CAS No: 335-67-1, PFOA, perfluorooctanoic acid), its salts and PFOA-related compounds.

UNEP/POPS/POPRC.14/2. (2018). Report of the Persistent Organic Pollutants Review Committee on the work of its fourteenth meeting.

UNEP/POPS/POPRC.14/6/Add.2 (2018). Addendum to the risk management evaluation on perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds.

Schoeib, M., Harner, T., Webster, G.M., Lee, S.C., 2011. Indoor Sources of Poly- and Perfluorinated Compounds (PFCS) in Vancouver, Canada: Implications for Human Exposure. Environ. Sci. Technol. 2011, 45, 19, 7999-8005. https://pubs.acs.org/doi/pdf/10.1021/es103562v)

UU.S. EPA, 2015. United States Environmental Protection Agency, PFOA Stewardship Program. Available from: http://www.epa.gov/assessing-and-managing-chemicals-under-tsca/20102015-pfoa-stewardship-program.

Yu, N., Guo, H., Yang, J., Jin, L., Wang X., Shi, W., Zhang, X., Yu, X., Wei, S. 2018. Non-Target and Suspect Screening of Per- and Polyfluoroalkyl Substances in Airborne Particulate Matter in China. Environ. Sci. Technol. 2018, 52, 8205−8214

Relevant guidelines and reference documents

IPCS (2017): PERFLUOROOCTANOIC ACID. International Chemical Safety Card (ICSC) 1413. Available at: http://www.ilo.org/dyn/icsc/showcard.display?p_version=2&p_card_id=1613

United Nations (2013): UN Recommendations on the Transport of Dangerous Goods - Model Regulations, Eighteenth revised edition. Available at: http://www.unece.org/trans/danger/publi/unrec/rev18/18files_e.html

IMO (2016): International Maritime Dangerous Goods Code (IMDG Code). Available at: http://www.imo.org/en/Publications/IMDGCode/Pages/Default.aspx