参考資料８

UNITED NATIONS

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UNEP/POPS/POPRC.12/11/Add.2

Distr.: General
13 October 2016

Original: English

Stockholm Convention on Persistent Organic Pollutants

Persistent Organic Pollutants Review Committee
Twelfth meeting
Rome, 19–23 September 2016

Report of the Persistent Organic Pollutants Review Committee on the work of its twelfth meeting

Addendum

Risk profile on pentadecafluorooctanoic acid (CAS No: 335-67-1, PFOA, perfluorooctanoic acid), its salts and PFOA-related compounds

At its twelfth meeting, by its decision POPRC-12/2, the Persistent Organic Pollutants Review Committee adopted a risk profile for pentadecafluorooctanoic acid (CAS No: 335-67-1, PFOA, perfluorooctanoic acid), its salts and PFOA-related compounds on the basis of the draft contained in the note by the Secretariat (UNEP/POPS/POPRC.12/3), as revised during the meeting. The text of the risk profile as adopted is set out in the annex to the present addendum. It has not been formally edited.

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Annex

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

RISK PROFILE

Persistent Organic Pollutants Review Committee

September 2016

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Executive summary

1. The POPs Review Committee concluded that PFOA fulfilled the screening criteria in Annex D and that issues related to the inclusion of PFOA-related compounds that potentially degrade to PFOA and the inclusion of PFOA salts should be addressed in the draft risk profile (see Decision POPRC-11/4). The substances covered by this risk profile include pentadecafluorooctanoic acid (CAS No: 335-67-1, EC No: 206-397-9, PFOA, perfluorooctanoic acid) including its isomers, its salts and PFOA-related compounds.

2. PFOA and its salts are most widely used as processing aids in the production of fluoroelastomers and fluoropolymers, with polytetrafluoroethylene (PTFE) being an important fluoropolymer. PFOA-related compounds are used as surfactants and surface treatment agents (e.g. in textiles, paper and paints, fire-fighting foams) and for the manufacture of side-chain fluorinated polymers. Due to their physico-chemical properties, PFOA, its salts and PFOA-related compounds are used in a wide variety of applications and consumer products across many sectors.

3. PFOA, its salts and PFOA-related compounds are subject to a number of national regulations. Norway has banned its use in consumer products (currently the substance is being phased out); while in the U.S. there is a voluntary initiative to phase out its use. Elsewhere in Canada and the EU, steps are underway towards the setting of legally binding conditions to restrict or ban the substance.

4. From 1951 to 2004, the estimated total global production of PFOA and APFO (ammonium perfluorooctanoic acid) was 3600 – 5700 t. Current production of PFOA is predominantly carried out in China where the production of PFOA and its salts has tripled from around 30 t in 2004 to around 90 t in 2012. The most recent data that is publically available concerning global production of APFO indicates that an average 200-300 t of PFOA were produced annually (1995-2002). It is expected that current production is significantly lower due to the voluntary phasing out of the substance by U.S., EU and Japanese companies. However, its current use at global level in the production of fluoroelastomers and fluoropolymers demonstrates that there is an ongoing production and use.

5. Direct releases to the environment occur from the production of the raw substance (including PFOA as impurity in the manufacturing of PFOA-related compounds and some alternatives), during the processing, use and disposal of the chemical, from treated articles and from products contaminated with PFOA. Main emission vectors of PFOA and its salts are water, wastewater and dust particles. Historic releases to the environment from PFOA manufacturing are available from a plant in the U.S. into air and water between 1951 and 2003. Some estimates of releases during the disposal of the chemical are available, particularly from sewage treatment plants, wastewater treatment plants and landfill sites. Indirect releases occur from the degradation or transformation of precursors. PFOA-related compounds are released to air and (waste) water and will degrade to PFOA in the environment and in organisms. An assessment of sources of PFOA to the Baltic Sea estimated that 30% of the releases were due to transformation of fluorotelomers. Thus releases of PFOA from degradation contribute a major share to the releases of PFOA to the environment.

6. Degradation results show that PFOA is persistent and does not undergo any abiotic or biotic degradation under relevant environmental conditions. The monitoring data show that PFOA in soil leaches over time and can be a long term source to underlying groundwater. On this basis 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.

7. PFOA has a low to moderate potential to accumulate in aquatic (i.e. water breathing) species, but there is evidence that PFOA and its salts accumulate and biomagnify in air breathing terrestrial and marine mammals (BMFs, TMFs > 1).

8. Worldwide monitoring of water, air, sediment and biota at remote locations all detect the presence of PFOA and related compounds. Equally environmental modeling data suggest that the capacity for long range transport does exist, while others have identified key mechanisms which would make long range transport plausible. On this basis it can be concluded that PFOA directly or through precursors is subject to long range transport.

9. PFOA exposure of the general public typically takes place “human via environment” by consumption of drinking water and food including breast feeding, via uptake of contaminated indoor air and dust or from consumer products containing PFOA, its salts and related compounds. PFOA has been detected in humans in blood and breast milk from various countries. Humans are very slow eliminators of PFOA compared with other species with an estimated half-life of PFOA elimination ranging from 2 to 4 years. PFOA accumulates in humans with increasing levels with age.

10. PFOA exhibits low acute toxicity in aquatic organisms. In fish, PFOA inhibited expression of genes involved in thyroid hormone biosynthesis, induced vitellogenin gene expression, developed oocytes in the testes of males and caused ovary degeneration in females. PFOA has the potential to affect endocrine function where visible effects may not be apparent until the organisms reach adulthood.

11. Effects of repeated oral PFOA exposure in animals such as alterations to the liver, reproductive/developmental toxicity and endocrine disruption have been identified. Developmental effects that are of relevance for the oral route are perinatal mortalities, reduced pup body weight gain and development, cyanosis and necrosis, delayed ossification, changes in mammary gland development and delayed maturation.

12. In the European Union, PFOA has a legally-binding harmonized classification as Carc. 2, Repr. 1B and STOT RE 1 (liver). PFOA is quickly absorbed, not metabolised and distributed in the body, transferred to foetus through placenta and infants via breast milk. A considerable number of adverse health effects associated with PFOA exposure in humans have been reported. The C8 Science Panel (a large-scale epidemiological study conducted in the US on PFOA exposure and health effects) concluded that there was a probable link to PFOA exposure for diagnosed high cholesterol, ulcerative colitis, thyroid disease, testicular cancer, kidney cancer and pregnancy-induced hypertension. Scientific data have demonstrated PFOA-mediated immunotoxicity, primarily suppression of antibody response, in humans. There have also been other reported adverse health effects (e.g. altered reproductive or developmental effects, endocrine disruption, impaired neurodevelopment, immunotoxicity, etc.) associated with PFOA exposure in humans.

13. PFOA is persistent, bioaccumulative and toxic to animals including humans. There is widespread occurrence of PFOA and a number of PFOA-related compounds in environmental compartments and in biota and humans. Therefore, it is concluded that PFOA, its salts and related compounds that degrade to PFOA are likely, as a result of their long-range environmental transport, to lead to significant adverse human health and/or environmental effects such that global action is warranted.

1. Introduction

14. In June 2015 the European Union and its Member States submitted a proposal to list pentadecafluorooctanoic acid (CAS No: 335-67-1, PFOA, perfluorooctanoic acid), its salts and PFOA-related compounds in Annex A, B, and/or C of the Stockholm Convention (UNEP/POPS/POPRC.11/5). This proposal was considered by the Persistent Organic Pollutants Review Committee (POPRC) at its eleventh meeting held in October 2015.

15. PFOA, its salts and PFOA-related compounds fall within a family of perfluoroalkyl and polyfluoroalkyl substances (PFASs). PFASs consist of carbon chains of different chain length, where the hydrogen atoms are completely (perfluorinated) or partly (polyfluorinated) substituted by fluorine atoms (Buck et al., 2011; OECD, 2013; ECHA, 2015a). The very stable bond between carbon and fluorine is only breakable with high energy input. Therefore, perfluorinated acids, like PFOA, are not degradable in the environment. Certain polyfluorinated substances can be degraded to persistent perfluorinated substances like PFOA under environmental conditions and are therefore precursors. Those PFASs, which can be degraded to PFOA in the environment, are referred to as PFOA-related compounds. PFOA and its salts are most widely used as processing aids in the production of fluoroelastomers and fluoropolymers, with PTFE being an important fluoropolymer. PFOA-related compounds are used as surfactant and for the manufacture of side-chain fluorinated polymers (ECHA, 2015a). Due to the surfactant properties of both PFOA and its related non-polymeric surfactants, applications exist for the use of these substances, e.g. fire-fighting foams, wetting agents and cleaners. Side-chain fluorinated polymers provide durable water, oil and stain repellency and find use as surface finishes for textiles and apparel, leather, paper and cardboard, paints, lacquers and other uses (non-woven medical garments, floor waxes, and stone/wood sealants, thread sealant tapes and pastes, adhesives, products for apparel) (UNEP/POPS/POPRC.11.5; FluoroCouncil, 2016).

16. PFOA has been manufactured since 1947 when 3M developed the production process via electrochemical fluorination (ACS, 2015). PFOA-related compounds (such as fluorotelomer alcohols) have also been in use, notably with the development of fluorotelomer technologies in the 1960s and their subsequent commercialization in the 1970s and thereafter. The perfluorooctyl iodide fraction of fluorotelomers was oxidized to PFOA and subsequently used as a fluoropolymerization aide by several fluoropolymer producers globally (DuPont, 2010; FluoroCouncil, 2016). However growing concerns regarding health and environmental effects of PFOA have meant stricter controls and phase-out plans under legislation such as the regulation on registration, evaluation, authorization and restriction of

chemicals (REACH EC 1907/2006) in the EU and the *Canadian Environmental Protection Act 1999* (CEPA) as well as voluntary efforts such as the US EPA PFOA Stewardship Program (US EPA, 2015) and work by industry (OECD, 2015). With a voluntary move by the Telomer Research Program (TRP) and Fluoropolymer Manufacturing Group (FMG) members in 2006, eight of the main manufacturers of C8-substances (fluorochemicals related to eight fully fluorinated C8-atoms) in the US, Europe and Japan agreed on a 2-stage stepwise global phase out of PFOA and related long-chain substances by the end of 2015 (ACC, 2015; FluoroCouncil, 2015/2016). A similar program existed in Canada[1]. All Stewardship Program participants were successful at virtually eliminating those chemicals globally from facility emissions and product content. Beyond possible impurities, Stewardship Program participants no longer manufacture, use, or sell PFOA and related long-chain substances. The voluntary phase out does not include manufacturers using PFOA in countries like China, India or Russia (ECHA, 2015a). After the Stewardship participants announced they were phasing out PFOA and related long-chain substances, companies who were not part of the Stewardship Program increased capacities (FluoroCouncil, 2016).

17. In 2011, PFOA-relevant technology and products were added to the Catalogue for the Guidance of Industrial Structure Adjustment in China (NDRC, 2013), including that new installation of PFOA production facilities shall be restricted and that PFOA-containing paints and fluoropolymers that use PFOA in the polymerization shall be eliminated. In 2013, fluoropolymers that use PFOA in the polymerisation were recognized as products with high pollution and high environmental risk ("dual-high" products) in the Comprehensive Catalog for Environmental Protection (China MEP, 2015).

18. The proposal to list PFOA, its salts and PFOA-related compounds in Annex A, B and/or C of the Stockholm Convention (UNEP/POPS/POPRC.11.5) highlighted concerns that the presence of PFOA in the environment is also influenced by the degradation of PFOA-related compounds, including side-chain fluorinated polymers. Hence the addition of PFOA alone to the Stockholm Convention would not be sufficient to protect human health and the environment. For some substances, e.g. for the fluorotelomer alcohol 8:2 FTOH[2] degradation to PFOA has been proven by experiment. Furthermore, potential PFOA-related compounds can be substances containing a perfluorinated alkyl chain with the formula $F(CF_2)_n$- (n=7 or 8) and that is directly bonded to any chemical moiety other than a fluorine, chlorine or bromine atom or a phosphonic, phosphinic or sulfonic group. Such substances can undergo abiotic degradation resulting in release of PFOA (Nielsen, 2013, 2014; Wang et al., 2014a; Ellis et al., 2004a). C8-substances (e.g. PFOA) may occur as impurities in the C6 alternatives. Thus, the C6 alternatives also contain C8 (and longer chain) residual substances, which can be released into the environment. (ECHA, 2015a).

1.1 Chemical identity

19. The proposed substances defined within the screening dossier (UNEP/POPS/POPRC.11/5) include pentadecafluorooctanoic acid (CAS No: 335-67-1, EC No: 206-397-9, PFOA, perfluorooctanoic acid) including its salts and PFOA-related compounds.

20. Taking into account the background document to the restriction proposals for PFOA, PFOA salts and PFOA-related compounds (ECHA 2015a; ECHA 2015c), the risk profile covers:

(a) PFOA (including its isomers);

(b) Its salts; and

(c) PFOA-related compounds which, for the purposes of this risk profile, are any substances that degrade to PFOA, including any substances (including salts and polymers) having a linear or branched perfluoroheptyl group with the moiety (C7F15)C as one of the structural elements, for example:

(i) Polymers with C8 to C16 based fluorinated side chains;[3]

(ii) 8:2 fluorotelomer compounds;

[1] Environmental Performance Agreements and Results: Perfluorinated Carboxylic Acids (PFCAs) and their Precursors (2010-2015), from http://www.ec.gc.ca/epe-epa/default.asp?lang=En&n=0D8C879E-1#X-2013092511492112

[2] FTOHs are polyfluorinated compounds typically characterized by even numbered perfluorinated carbons and two nonfluorinated carbons adjacent to a hydroxyl group (Dinglasan et al., 2004). For FTOHs, the prefix 'C8:2' denotes that for "X:Y", X = the number of perfluorinated carbons within the alkyl chain, and Y = the number of non-fluorinated carbons within the alkyl chain.

[3] DuPont, 1998. Technical information: Zonyl fluorochemical intermediates.

(iii) 10:2 fluorotelomer compounds.

PFOA-related compounds do not include:

(i) C8F17-X, where X= F, Cl, Br;

(ii) Fluoropolymers[4] that are covered by CF3[CF2]n-R', where R'=any group, n>16; or

(iii) PFOS, its salts and PFOSF as listed in Annex B to the Stockholm Convention.

21. Data on PFOA are presented in Table 1 and Table 2. The screening dossier (UNEP/POPS/POPRC.11/5) also further includes information on salts of PFOA and PFOA-related compounds based on the study carried out by the OECD (2007, 2011) as well as information from an assessment conducted by Environment Canada and Health Canada (2012). To maintain a concise document tables of data for PFOA salts and related PFOA compounds are provided in a background document to this risk profile (see section 1.1 of document UNEP/POPS/POPRC.12/INF/5).

Table 1: Identity of PFOA

CAS number:	335-67-1
CAS name:	Octanoic acid, 2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-pentadecafluoro-
IUPAC name:	Pentadecafluorooctanoic acid
EC number:	206-397-9
EC name:	Pentadecafluorooctanoic acid
Molecular formula:	$C_8HF_{15}O_2$
Molecular weight:	414.07 g/mol
Synonyms:	Perfluorooctanoic acid; PFOA; Pentadecafluoro-1-octanoic acid; Perfluorocaprylic acid; Perfluoro-n-octanoic acid; Pentadecafluoro-n-octanoic acid; Pentadecafluorooctanoic acid; n-Perfluorooctanoic acid; 1-Octanoic acid, 2,2,3,3,4,4,5,5,6,6, 7,7,8,8,8-pentadecafluoro

Table 2: Overview of relevant physicochemical properties of PFOA

Property	Value	Reference/Remark
Physical state at 20°C and 101.3 kPa	Solid	Kirk, 1995
Melting/freezing point	54.3 °C 44 — 56.5 °C	Lide, 2003 Beilstein, 2005 cited in ECHA, 2013a
Boiling point	188 °C (1013.25 hPa) 189 °C (981 hPa)	Lide, 2003 Kauck and Diesslin, 1951
Vapour pressure	4.2 Pa (25° C) for PFO; extrapolated from measured data 2.3 Pa (20° C) for PFO; extrapolated from measured data 128 Pa (59.3° C) for PFO; measured	Kaiser et al., 2005; Washburn et al., 2005 Washburn et al., 2005 Washburn et al., 2005
Water solubility	9.5 g/L (25° C) 4.14 g/L (22°C)	Kauck and Diesslin, 1951 Prokop et al., 1989
Dissociation constant	<1.6, e.g. 0.5 1.5 — 2.8	Vierke et al., 2013 Kissa, 2001
pH-value	2.6 (1 g/L at 20 °C)	ECHA 2015a (reliability not assignable)

[4] Fluoropolymers are carbon-only polymer backbone with F directly attached to backbone C atoms.

22. There are two manufacturing processes to produce PFOA, its salts and PFOA-related compounds: electrochemical fluorination (ECF) and telomerization. From 1947 until 2002, the ECF process was mainly used to manufacture ammonium perfluorooctanoate (APFO) worldwide (80-90% in 2000) which results in a mixture of branched and linear isomers (78% linear and 22% branched isomers). The current extent of global ECF manufacturing is unknown; however, most of the manufacturers are using the telomerisation process nowadays, which mainly results in linear compounds (Wang et al., 2014a). Results from Jiang et al. (2015) suggest that ECF is still used by some manufacturers in China. ECHA (2013b) details the physico-chemical properties of APFO in the environment. The free PFOA stays in equilibrium with PFO, the conjugate base, in aqueous media in the environment as well as in the laboratory. The physico-chemical properties of PFOA and PFO are different. Therefore, the expected environmental fate will depend on the environmental conditions, which influence the equilibrium between base and acid (pH and pKa). APFO, which is often used in animal experiments, is very soluble in water. In aqueous solution it is present as anion PFO and the ammonium cation. Goss (2008) suggested that PFOA is expected to have a low pKa, such that >99% of the compound will occur in its anionic form (i.e., PFO) under most environmental conditions suggesting that the environmental partitioning of PFOA will be dominated by the anionic form. The dissolved anion PFO will stay in equilibrium with the corresponding acid in aqueous media. With currently available analytical methods it is not possible to distinguish between PFO and PFOA in samples. In the literature reporting human and environmental monitoring studies the concentrations are referred to as PFOA or APFO, but always both species (PFO and PFOA) are included in the given concentration (ECHA, 2013b).

23. A standardized method for the analysis of presence of PFOA in different matrices is currently not available. The unique chemical and physical properties of PFOA prevent it from being measured using conventional analysis. More complex methodology of liquid chromatography and tandem mass spectrometry (LC/MS-MS) has been proven most reliable for analyzing PFOA in biological and environmental samples and therefore the analytical method of choice (Xu et al., 2013; EFSA, 2008; Loos et al., 2007). This type of analysis has allowed for more sensitive determination of many perfluorinated chemicals (PFCs), including PFOA, in air, water, and soil (ATSDR, 2015).

1.2 Conclusion of the Review Committee regarding Annex D information

24. The POPs Review Committee evaluated the proposal regarding pentadecafluorooctanoic acid (CAS No: 335-67-1, PFOA, perfluorooctanoic acid), its salts and PFOA-related compounds (UNEP/POPS/POPRC.11/5) according to the requirements in Annex D of the Stockholm Convention at its eleventh meeting in Rome. The Committee concluded that PFOA fulfilled the screening criteria in Annex D. The Committee also decided to establish an ad-hoc working group to review the proposal further and prepare a draft risk profile in accordance with Annex E of the Convention and further that issues related to the inclusion of PFOA-related compounds that potentially degrade to PFOA and the inclusion of PFOA salts should be dealt with in developing the draft risk profile (Decision POPRC-11/4).

1.3 Data sources

25. The draft risk profile is based on the following data sources:

(a) Proposal submitted by the European Community and its member states that are Parties to the Convention (UNEP/POPS/POPRC.11/5), 2015;

(b) Information submitted by Parties and observers according to Annex E of the Convention: Albania, Austria, Canada, China, Germany, Hungary, Japan, Monaco, Norway, Romania Estonian Environmental Research Centre, FluoroCouncil, IPEN, Semiconductor Industry Associations;

(c) The Screening Assessment Report on PFOA, its salts and precursors prepared by Environment Canada and Health Canada, 2012;

(d) Screening Information Datasets (SIDS) on PFOA and the ammonium salt of PFOA prepared by the OECD, 2006, 2007, 2011, 2013;

(e) Support documents for the identification of PFOA and the ammonium salt of PFOA as a Substance of Very High Concern (SVHC) included under the EU Regulation on Registration, Evaluation, Authorization and restriction of Chemicals (REACH), (ECHA, 2013a, 2013b);

(f) Committee for Risk Assessment (RAC) and Committee for Socio-economic Analysis (SEAC) background document to the opinion on the Annex XV dossier proposing restrictions on PFOA, PFOA salts and PFOA-related compounds (ECHA, 2015a);

(g) Data from the United States Environmental Protection Agency (US EPA) PFOA Stewardship Programme;

(h) The Australian National Industrial Chemicals Notification and Assessment Scheme (NICNAS) Environment tier II assessment for both direct and indirect precursors of PFOA (NICNAS, 2015a and 2015b).

1.4 Status of the chemical under international conventions

26. PFOA is subject to one international convention. Under the Oslo/Paris Commission for the Protection of Marine Environment of the North-East Atlantic (OSPAR), a review of both PFOS and PFOA was conducted to assess the potential impact upon the environment. This led to the inclusion of PFOS on the list of chemicals for priority action in 2003. PFOA was not added to the list at that time pending additional research and review with the option for inclusion at a later date (OSPAR, 2006).

27. OECD provided a recent overview on risk reduction approaches for PFASs across countries (OECD, 2015). PFOA is subject to a number of national and/or regional regulations:

(a) In 2013, both PFOA and APFO were identified as Substances of Very High Concern (SVHC) upon their persistent, bioaccumulative and toxic properties and were included into the REACH-Candidate List (ECHA, 2013a, 2013b). Inclusion upon this list means the substances can be subject to further review and ultimately phased out under the Authorization process. Moreover, on request industry is obliged to inform consumers on the occurrence to the listed substances in consumer articles;

(b) In 2014 Germany and Norway lodged a joint proposal for the inclusion of PFOA within Annex XVII (restriction) of the REACH regulation within the EU (ECHA 2014a). The aim of the proposal was a total ban on manufacture, placing on the market and use (including import) of PFOA and its salts also including substances that may degrade to PFOA (PFOA-related compounds) in concentrations equal to or greater than 2 ppb. The proposed restriction also covers articles containing these substances. After submission of the proposal to the European Chemicals Agency (ECHA) stakeholders commented this proposal during the public consultation period and delivered new information. Subsequently, the proposal was updated by the Dossier Submitters together with ECHA's scientific committees RAC and SEAC and it has been transmitted to the European Commission for developing the final legislative proposal.

(c) PFOA was included under 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). PFOA has been classified as Carc. 2 H351, Repr 1B H360D, Lact H362, STOT RE 1 (liver) H372, Acute tox 4 H332, Acute tox 4 H302 and Eye dam 1 H318.

(d) The Norwegian Environment Agency published an amendment to the consumer products regulation in 2014 banning the use of PFOA in consumer products and textiles. This has a transitional period allowing the import and sale of products before phase out;

(e) Within Canada following the screening assessment conducted in 2012, PFOA, its salts and precursors were found to meet the requirements of Section 64a of CEPA and were added to Schedule 1, the List of Toxic Substances. In June 2006, the Government of Canada published a Notice of Action Plan for the assessment and management of perfluorocarboxylic acids and their precursors. The Action Plan included measures to prevent the introduction of new substances into Canada that would contribute to the level of PFCAs (perfluorocarboxylic acids) in the environment, and to seek action from industry to address sources of PFCAs already in Canadian commerce. To this end, a voluntary Environmental Performance Agreement was signed on March 30, 2010. Signatories to the Performance Agreement agreed to reduce the amount of PFOA and long-chain perfluorocarboxylic acids in perfluorinated chemicals in Canadian commerce by 95% by December 31, 2010, and to virtually eliminate them by December 31, 2015. Furthermore in April 2015, the proposed *Regulations Amending the Prohibition of Certain Toxic Substances Regulations, 2012,* were published in Canada. These amendments propose to prohibit PFOA, its salts and precursors and products containing them, unless present in manufactured items. Furthermore, the amendments propose time-limited exemptions and ongoing permitted uses for certain applications where the development of alternatives is underway or where there are currently no known alternatives;

(f) In the United States of America, the US EPA established the PFOA Stewardship Programme in 2006. This is a programme that includes eight major manufacturers of PFOA, its salts and PFOA-related compounds (Arkema, Asahi, BASF, Clariant, Daikin, 3M/Dyneon, DuPont, Solvay

Solexis). The programme is a voluntary initiative to the phase-out the manufacture and use of PFOA, PFOA precursors and related higher homologue substances (US EPA, 2015).

(g) Russia regulates APFO in occupational air. A number of short- and middle chain PFASs are regulated in occupational air and water (OECD, 2013).

2. Summary information relevant to the risk profile

2.1 Sources

2.1.1 Production, trade, stockpiles

28. The following table summarizes information related to the production of PFOA, its salts, APFO and FTOH according to several literature sources. Further information is contained in section 2.1.1 of document UNEP/POPS/POPRC.12/INF/5.

Table 3: Production of PFOA, its salts, APFO and FTOH

Year/Period	Production	Value/Range [in t or t/y]	Reference
1992-2002	PFOA production 3M [in US]	113 t/y	3M communication to US EPA, 2003
2009	Estimated worldwide production of FTOH	11,000-14,000 t/y	Umweltbundesamt, 2009
2014	Production of PFOA related substances in the EU (actual volume is likely to be greater)	100 – 1,000 t/y	ECHA, 2015a
2003	Production of PFOA and its salts in China	30 t/y	Li et al., 2015
2012	Production of PFOA and its salts in China	90 t/y	Li et al., 2015

29. In 2005, producers were located in Italy, U.S., South America, Japan and China. Up until 2010, production was also carried out in Italy. All production of PFOA and its salts in the EU has now ceased, and manufacturing in Japan and the U.S. should now have stopped with the intention to phase out production by the end of 2015 (ECHA, 2015a). However, in China, the production of PFOA and its salts has tripled from around 30 t in 2004 to around 90 t in 2012 (Li et al., 2015).

30. In 2014, production of PFOA-related compounds in the EU was reported to be between 100-1,000 tonnes per annum (ECHA, 2015a). However, this volume is derived from a search in the ECHA-database of four PFOA-related compounds registered under REACH, and in its report ECHA concludes that the actual volume is likely to be greater. No other information concerning the production of PFOA-related compounds was identified in the submissions by parties, or in the literature reviewed.

31. Data concerning imports and exports of PFOA is limited and no information concerning global volume of trade flows has been identified owing to industry confidentiality claims (ECHA, 2015a).

32. PFOA ammonium salt was imported into Canada in quantities ranging between 0.1 to 100 t (2004 survey) (Environment Canada and Health Canada, 2012).

33. ECHA 2015a provides estimates on import figures for the EU. The main information related to imports is summarized in the following (detailed information see section 2.1.1 of document UNEP/POPS/POPRC.12/INF/5).

Table 4: Import of PFOA, its salts and PFOA related substances to the EU

Year	Imports to the EU	Value [in t]
2014	PFOA and its salts (decreasing trend since 2008)	~20
2014	PFOA and its salts (mixtures i.e. in fluoropolymer dispersions)	~10
2014	PFOA and its salts in articles (high uncertainty)	<10
2015	PFOA and its salts forecast to fall after 2015	<0.15
2014	PTFE	3 – 16
2014	PFOA related substances	100 – 1,000
2014	PFOA related substances in textiles (the total volume of PFOA related substances in imported articles is unknown)	1,000 – 10,000

Reference: ECHA (2015a)

34. According to the information gathered in a survey conducted by the OECD in 2009 (OECD, 2011), the reported concentration of PFOA in products containing the substance varies between <1-160 ppm. Concentrations of other PFOA-related compounds in products vary between 1-4,200 ppm, and concentrations of C8:2 fluorotelomers in products range between 5-35,000 ppm. Also ECHA (2015a) provides in its appendix data on concentrations of PFOA and related compounds in products such as outdoor clothing, workers protection clothing, membranes for apparel, treated home textile and upholstery, treated non-woven medical garments, leather finishing, carpets, impregnating sprays/ waterproofing agents, firefighting foams, treated paper, paints and inks, cleaning agents, floor waxes/wood sealants, lubricants and sealant tapes (refer to ECHA, 2015a, Appendix B, Tables A.B.2-4 and A.B.2-9 for more information).

35. Information on imports of products containing PFOA and PFOA-related compounds was only identified for Australia, Albania and the EU, as follows:

(a) Australia reported the import of an antifoam product containing less than 10% of a PFOA-related chemical (constituting ~10 kg of PFOA) in 2005 for use in a dyeing process with sulfur dyes (NICNAS, 2015b). Australia assesses individual chemical entities in consumer products and chemicals released from articles. Articles are not within the scope of the *Industrial Chemicals Notification and Assessment Act* (Australia 2016);

(b) In 2014, the EU reported the import of between 1,000-10,000 tonnes of PFOA-related compounds contained in textile articles (specifically within outdoor jackets). Of note, this volume is expected to decline after 2015 due to the availability of alternatives (estimated imports thereafter are between 300-3,000 tonnes per annum) (ECHA, 2015a);

(c) Albania provided information within the Annex E survey (Albania, 2015) for the quantity of goods imported that may contain PFOA for the years 2012–2015. This ranged between 3–20 tonnes for total net weight of all goods.

36. Stockpiles of firefighting foams containing PFAS including PFOA are likely to be present at military bases, airports, oil production facilities and rigs and other facilities (Baduel et al., 2015; Anderson et al., 2016; FluoroCouncil 2016). For example, the US military did possess approximately 11 million liters of aqueous fire-fighting foam containing PFCs including PFOA (Place and Field, 2012). Two studies report on estimated quantities of AFFF in the U.S. (Darwin, 2004, 2011).

2.1.2 Uses

37. Due to their physico-chemical properties, PFOA, its salts and its related compounds are used in a wide variety of applications and consumer products across many sectors (primarily automotive, electronics, construction and aerospace industries) (ECHA, 2015a; OECD, 2013). ECHA (2015a) provides estimation on the amounts of PFOA and its salts as well as on PFOA related substances used in the EU in different sectors/industries. The main information is summarized in the following.

Table 5: Use of PFOA, its salts and PFOA-related compounds in different sectors in the EU

Use in the EU	Sector/Industry	Value/Range [in t]
PFOA-related compounds	Textile and leather treatment	~1,000
	Paper treatment	>150 – 200
	Firefighting agents	>50 – 100
	Paints and inks	>50 – 100
	Others uses	>0.1 – 0.5
PFOA and its salts	Manufacture of fluoropolymers	<20
	Photo industry	1.0
	Semiconductor industry	<0.05
	Other uses 0.5-1.5 t	0.5-1.5

Reference: ECHA (2015a)

38. PFOA is predominantly used in the form of APFO in aqueous solution as an emulsifier and processing aid in the manufacture of many fluoropolymers, such as PTFE, FEP (fluorinated ethylene propylene), PFA (perfluoroalkoxy alkane) or PVDF (polyvinylidene fluoride) (Emmett et al., 2006; OECD, 2006; ECHA, 2015a). PFOA is a contaminant in fluorochemicals and telomer products (Emmett et al., 2006). Fluoropolymers are used across many sectors for different purposes, including: in the manufacture of hoses, cable and gaskets; non-stick coatings on cookware; and personal care products (Begley et al., 2005; Environment Canada and Health Canada, 2012; U.S.EPA, 2009; van der Putte et al., 2010; ECHA 2015a). APFO is also used in the processing of paint, photographic film additives and in the textile coating industry (OECD, 2006), and can be found in certain aqueous

fire-fighting foams (OECD, 2006; Prevedouros et al. 2006; Environment Canada and Health Canada, 2012). PFOA is also used, as a surfactant and processing aid in the manufacture of semi-conductors used in the photolithographic process (van der Putte et al., 2010; ESIA, 2015) and as a replacement for perfluorooctane sulfonate (PFOS) (ECHA, 2015a).

39. PFOA-related compounds are used as surfactants and/or fluorinated polymers for a variety of products including ski waxes (Freberg et al., 2010; Nilsson et al., 2010a, 2010b) and treatments for leather, other textiles (such as outdoor textiles and carpets) (Washburn et al., 2005; Begley et al., 2005), and paper packaging of microwave popcorn bags (Sinclair et al., 2007). High PFOA levels were identified in ski waxes (up to about 2000 µg/kg PFOA), outdoor textiles (up to 19 µg/m^2 PFOA) and some baking papers (up to 15 µg/m^2 PFOA) (Kotthoff et al., 2015). PFOA-related compounds are used as a surface treatment agent in both the stone and paper industries (China, 2015), in medical devices (Austria, 2015), as an antifoam products for use in the dyeing process using sulfur dyes (NICNAS, 2015b), and as a leveling agent in paints and inks. PFOA-related compounds are used as (1) non-polymeric substances for firefighting foams, wetting agents or cleaners (OECD, 2013; van der Putte et al., 2010) or (2) as part of side-chain fluorinated polymers, such as fluoroacrylate polymers (US EPA, 2009; van der Putte et al., 2010). Side-chain fluorinated polymers are used to provide a water, grease and soil protection, for example in applications such as textiles, products for apparel, leather, paper and cardboard (e.g. in food packaging), paints and lacquers (e.g. exterior and interior architectural paints), non-woven medical garments, floor waxes and stone/wood sealants, thread sealant tapes and pastes, adhesives or products for apparel. Fluorotelomers is a term often used in the literature, referring to substances produced with the telomerization process. Fluorotelomers might be PFOA-related compounds if they contain the respective chain length. For fluorotelomers it was reported that 80% are used in polymers and 20% in non-polymeric applications. Identified major uses of PFOA-related compounds in Europe are surface treated textiles, fire-fighting foam, surface treated paper and paints and inks (ECHA, 2015a).

2.1.3 Releases to the environment

40. Numerous direct and indirect sources of PFOA, its salts and PFOA-related compounds contribute to the overall release of PFOA to the environment. Direct releases to the environment occur from the production of the raw substance, during the processing, use and disposal of the chemical and of products containing the chemical. Main emission vectors are water, wastewater and dust particles. Indirect releases to the environment occur due to the formation of PFOA from PFOA-related compounds. They are released to air and waste water during manufacture of the substances themselves, from side-chain fluorinated polymers and during use and disposal of consumer articles treated with PFOA-related compounds. When emitted to the atmosphere, they can be degraded to PFOA, and deposited on soil or surface waters (see also para 2.1.3 of document UNEP/POPS/POPRC.12/INF/5). They are also washed out from the atmosphere via precipitation (ECHA, 2015a). The estimated global cumulative emission of PFOA ranged from 2078 to 18366 t over the period of 1951 to 2030, thus presenting the compound with the highest cumulative emission of the C_4-C_{14} PFCAs (Wang et al., 2014a). The study also indicates a decrease in estimated emissions in North America, Western Europe and Japan, concurrently with increases in India, Russia and China.

41. The manufacturing of PFOA has been identified as a major direct source of PFOA in the environment (Armitage et al., 2009; Prevedouros et al., 2006). During the manufacturing of PFOA the substance can be emitted into the environment either via waste water or into the air. Prevedouros and co-workers (2006) estimated global PFOA manufacturing emissions: 45 t in 1999, 15 t in 2004, 7 t in 2006. PFOA can also be directly released from manufacturing and use of PFOA-related compounds. (Pistocchi and Loos, 2009; Loos et al., 2008; Dauchy et al., 2012). As detailed by Wang et al. (2014a), releases related to the manufacturing and use of PFOA have likely changed along with changes in practices at manufacturing sites (i.e. with vs. without control measures of waste streams). A recent publication documents the first source-specific inventory for environmental releases of PFOA/PFO in China from 2004 to 2012 and estimates cumulative environmental releases reaching 250 tonnes (t) over a period of nine years (Li et al., 2015) and several studies confirm the relevance of PFOA in China. Chinese samples from river/estuary systems were highly polluted by an industrial point source discharging mainly PFOA (Heydebreck et al., 2015) and an individual plant producing PTFE and other fluoropolymers releases extraordinary high quantities of PFOA into a river (Wang et al., 2016). There is a positive correlation between the proximity to a fluoropolymer production facility and PFOA concentrations in rivers (Shi et al., 2015) and substantial levels of per- and polyfluoroalkyl substances were detected in outdoor dust in China with PFOA being a predominant substance (Yao et al., 2016). Historic releases to the environment from production are available for a U.S. plants in West Virginia (Emmett et al., 2006; Paustenbach et al., 2007; Lerner, 2005) and from other large scale production until 2002 (Minnesota State Dep, 2016; Oliaei et al., 2013; Minnesota Pollution Control Agency, 2016).

42. The manufacture of fluoropolymers is considered the main direct emission source of PFOA, where APFO is used as processing aid (Armitage et al., 2009; Prevedouros et al., 2006). From fluoropolymer production sites, PFOA is emitted to air (mainly particle bound) and water. Fluoropolymer dispersions are often used to coat metal and fabric surfaces. During the dispersion processing and subsequent use and disposal of consumer articles are released. Environmental releases from the direct use of PFOA are also possible from the photo industry and the semiconductor industry (van der Putte et al., 2010; ESIA, 2015; SIA, 2015).

43. Releases to the indoor domestic environment arise from the use of products containing PFOA and its related compounds for several products purchased from retail in the US, with the largest releases from use reported for professional carpet-care liquids, pre-treated carpeting, floor waxes and stone/tile/wood sealers and household textiles and upholstery (US EPA, 2009a). Emissions from outdoor jackets occur mainly during washing and impregnation procedures (Umweltbundesamt, 2014).

44. The use of PFOA-related compounds results in direct and indirect emissions of PFOA These result from impurities in fluorotelomer based products and from degradation of fluorotelomer based products as well as from the manufacture, use and disposal of side-chain fluorinated polymers (ECHA, 2015a; Environment Canada and Health Canada, 2012). The manufacture of side-chain fluorinated polymers represents one major industrial use of PFOA-related compounds (Russell et al., 2008; ECHA 2015a; Danish Ministry of Environment, 2013).

45. Releases to the environment occur from management of waste water and of solid waste consisting of or contaminated with PFOA, its salts or PFOA-related compounds. Industrial wastewater from fluoropolymer manufacturing is considered the most important point source of PFOA to surface water. Waste water treatment plants do not remove PFOA efficiently. Thus, a large share remains in the water phase and enters surface water bodies and degradation of PFOA-related compounds during the treatment can even lead to higher PFOA emissions (ECHA 2015a; Schultz et al., 2006; Bayerisches Landesamt für Umwelt, 2010; Houtz et al., 2016; UNEP/POPS/POPRC.12/INF/5). During solid waste management PFOA releases may result from incineration, landfilling and recycling (Yamada et al., 2005; Poulsen et al., 2005) and it is assumed that recycling of contaminated wastes contributes to environmental releases (ECHA, 2015a). There are a number of studies available related to waste water, sewage sludge or landfill leachate and waste treatment that include measurements of direct releases to the environment from the disposal of PFOA and its related compounds (Muir and Scott, 2003; Boulanger et al., 2005; Yan et al., 2015; Ikonomou, 2006; Busch et al., 2010; Guo, 2008; US EPA, 2009b; OECD 2011).

46. PFOA-related compounds will degrade to PFOA in sludge, soil, water and air (Wang et al., 2005a, 2005b; Moody and Field, 1999; IPEN, 2015; Dasu and Lee, 2016; Bizkarguenaga et al., 2016), and such degradation has been noted across many products containing PFOA-related compounds (Dasu et al., 2013; Ellis et al., 2003; Ellis et al., 2004a; Frömel and Knepper, 2010; Gauthier and Mabury, 2005; Hilal et al., 2004; Jackson and Mabury, 2013; Jackson et al. 2013; Rayne and Forest, 2010; Renner, 2008; Wang et al., 2005a; Wang et al., 2005b; Washington et al., 2009; Young and Mabury, 2010; Young et al., 2008; Zhang et al., 2013; Butt et al., 2014; Rankin et al., 2014; Washington et al., 2015). Release of PFOA to the environment results from its use in fire fighting foams. Aqueous film forming foams (AFFF) were developed in the early 1960s with PFASs as key components and constitute a remaining source of PFOA e.g. in firefighting training pads or around military aviation bases (Baduel et al., 2015; Australia, 2016).

47. There are no known natural sources of PFOA or their related compounds (Kissa, 1994; Environment Canada and Health Canada, 2012; IPEN 2015).

48. These above paragraphs provide overview information on releases to the environment. For more detailed information, please see section 2.1.3 of document UNEP/POPS/POPRC.12/INF/5.

Contribution from salts of PFOA and PFOA-related compounds to the environmental occurrence of PFOA

49. The nomination proposal for PFOA, its salts and PFOA-related compounds (UNEP/POPS/POPRC.11/5) highlights concerns that PFOA-related compounds could be contributing to environmental concentrations of PFOA through degradation. This point was raised by the Danish Ministry of Environment (2013) who suggests that: *"An assessment of PFOA to the Baltic Sea estimated with high uncertainty that 30% of the releases were due to the transformation of fluorotelomers"*.

50. Studies by Prevedouros et al. (2006) and Kim et al. (2012 and 2013) looked at the fate and behaviour of FTOHs (6:2 and 8:2 FTOHs) within terrestrial environments, suggesting that degradation products from 8:2 FTOHs will include PFOA. Prevedouros et al. (2006) estimate that around 1% of

PFCA emissions originate from fluorotelomer raw material degradation which would include PFOA; while Kim et al. (2012) highlights a biotransformation process within soils, whereby soil bacteria degraded FTOHs (both long and short chain) into a range of degradation products which include PFOA. Washington et al (2009) incubated an acrylate-linked fluorotelomer polymer in soil microcosms and monitored the microcosms for possible fluorotelomer and PFC degradation products. These and other results suggest that fluorotelomer-polymer degradation is a source of PFOA to the environment (Washington et al., 2009; Washington et al., 2014; refer to Koch et al., 2006, 2009; Russell et al., 2008, 2010 for a broader view of degradation and half-lives).

51. Thermolysis of PTFE also yields hexafluoropropylene and other compounds including PFOA (Ellis et al., 2001; Schlummer et al., 2015) while monomers used to make long-chain fluorotelomers and degraded from them can be biotransformed to PFOA (Butt et al., 2010a). Taylor (2009) and Taylor et al. (2014) in collaboration with DuPont could not detect PFOA during laboratory-scale combustion testing. Krusik and Roe (2004) and Krusik et al. (2005) studied the thermal decomposition of the ammonium salt of perfluorooctanoic acid (APFO) and PFOA. APFO and PFOA are thermally unstable at waste incineration temperatures (~1000°C). For example, the half-life for APFO is estimated to be less than 0.2 s at 350 °C. In light of the historically large production of fluorotelomer-based polymers (FTPs) and the poor efficacy of conventional treatments for recovery of PFCAs from waste streams, degradation of FTP based materials have the potential to increase environmental levels of PFCAs.

52. FTPs manufactured to date potentially could increase PFCAs 4- to 8-fold over current oceanic loads, largely depending on the integrity of disposal units to contain PFCAs upon hydrolytic generation from FTPs (Washington and Jenkins, 2015; refer to Koch et al., 2006, 2009; Russell et al., 2008, 2010 for alternative assessments indicating lower global loading potential).

53. PFOA is also found in packaging materials. For example, PFOA and related substances in fluoropolymer resin products vary up to 150 mg/kg and 5–3000 mg/kg in fluropolymer dispersion products in non-US based production locations. PFOA can also be extracted at low concentrations from textiles and packaging treated with side-chain-fluorinated polymers. The releases of substances from the different parts of the life cycle and the significance of the exposure of humans and the environment is still not fully understood and more information is needed (Danish Ministry of Environment, 2013).

54. Fluorotelomer carboxylic acids (FTCAs) and the corresponding unsaturated acids (FTUCAs) are intermediates in the biodegradation of industrially produced fluorotelomer alcohols (FTOHs) to environmentally persistent perfluorinated carboxylic acids (PFCAs). The fate of these compounds was investigated in a simple sediment–water microcosm system. Microcosms were spiked with 8:2 FTCA, 10:2 FTCA and 8:2 FTUCA, or 10:2 FTUCA. The FTCAs and FTUCAs investigated degrade rapidly. Identifiable degradation products of FTCAs and FTUCAs were observed and include PFOA for both, 8:2 and 10:2 compounds. (Myers and Mabury 2010; Butt et al., 2014**).**

55. Environment Canada and Health Canada (2012) and ECHA (2015a) compile scientific evidence on the degradation/transformation of PFOA related substances based on different sources (the information from both sources is compiled in section 2.1.3 of document UNEP/POPS/POPRC.12/INF/5).

56. ECHA (2015a) provides further details on the role of PFOA-related compounds to degrade and contribute to environmental concentrations of PFOA: *"The yields of PFOA are in most studies in the range 1.7 – 20%. The duration of the studies varies from 28 days to 90 days. One study was performed with a longer time scale of 7 months. The yield of PFOA in this study was in the range of 10 — 40% (Wang et al., 2009).This indicates that some of the degradation steps may take some time although the estimated half-life of the PFOA-related compounds is in the range of days. Thus, it can be hypothesized that in the environment PFOA yields from PFOA-related compounds are much higher than measured in the short time degradation experiments. We further hypothesize that over a long time frame of 5 — 10 years PFOA yields from PFOA-related compounds degradation are around 80%".*

57. In conclusion, PFOA-related compounds have the capacity to degrade to PFOA in the natural environment contributing to the environmental concentrations of PFOA. The quantities of PFOA generated via degradation from PFOA-related compounds are more debatable with values of between 1.7% - 40% quoted. However, as noted by ECHA (2015a), these results are based on shorter term tests and over longer periods of time the quantity of PFOA may be much higher (around 80%).

2.2 Environmental fate

2.2.1 Persistence

58. PFOA is extremely stable within the natural environment due to its chemical properties and does not degrade under environmentally relevant conditions. This fact is corroborated by the US EPA (2014), Australian NICNAS (2015a and 2015b), Environment Canada and Health Canada (2012) and OECD (2006). In particular, both assessments conducted by Environment Canada and Health Canada and OECD noted that there was no clear degradation of PFOA under abiotic or biotic conditions.

59. 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 (Todd, 1979; Hatfield, 2001; 3M, 2001). In aquatic environments where PFOA undergoes indirect photolysis, the half-life was estimated to be longer than 349 days (Hatfield, 2001; ECHA, 2013a, 2013b). A review of half-life data and mechanisms of decay is presented in Table 6.

Table 6: Reported findings on the persistence of PFOA (adapted from Environment Canada and Health Canada, 2012)

Medium	Mechanism	Degradation Half-life	References
Water	Photolysis	No photodegradation	Todd 1979; Hatfield, 2001
Water	Indirect photolysis	> 349 days	Hatfield, 2001
Water	Hydrolysis	~ 235 years	3M, 2001
Air	Hydroxyl reaction	~ 130 days	Hurley et al., 2004
Sludge	Biodegradation	> 2.5 months	Pace Analytical , 2001
Soil/sludge	Biodegradation	> 259 days	Liou et al., 2010

60. 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; see OECD, 2006; Hurley et al., 2004).

61. Persistence within the terrestrial environment mirrors the position seen in the aquatic environment, with high persistence, slow degradation and long half-lives. Both the REACH Annex XV restriction dossier (ECHA, 2015a) and REACH proposal for identification of PFOA as an SVHC (ECHA 2013a) state that PFOA was not biodegradable and that, based on high persistence, it was not possible to calculate half-lives in soil or sediment. This was the same conclusion presented within the screening dossier (UNEP/POPS/POPRC.11.5).

62. More detailed information on persistence is provided in section 2.2.1 of document UNEP/POPS/POPRC.12/INF/5.

Summary on persistence

63. 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.

2.2.2 Bioaccumulation

Screening assessment based on physical-chemical properties

64. Due to the high surfactant capacity of PFOA and its ability to form multiple layers in octanol/water, it is not possible to analyze log K_{ow} directly (US EPA, 2002). As quoted in Annex D of the Stockholm Convention and reported in Table 2, the derived estimates vary from above to below the critical log K_{ow} value of 5. Studies for BCF and BAF also highlight values below the critical value of 5,000 quoted by Annex D. However, based on the physical properties, PFOA is known to undergo a protein bioaccumulation mechanism rather than lipid partitioning (UNEP/POPS/POPRC.3/INF/8/2003), which makes standard BCF/BAF analysis less meaningful. Thus the use of log K_{ow}, BCF and BAF have been demonstrated to be inappropriate measures of bioaccumulation of PFOA (ECHA, 2013a).

65. To assess whether PFOA meets the bioaccumulation criterion, the focus is placed on the Annex D paragraph c) ii and iii; the evidence for concern within species as a part of monitoring. This includes an approach utilising biomagnification factors (BMF) and trophic magnification factors (TMF), which has been adopted alongside monitoring of higher trophic tier species to substantiate that bioaccumulation occurs in the natural environment. BMF and TMF approaches assess predator-prey relations and the whole food chain, respectively, to evaluate the flow of material and substantiate

whether biomagnification might take place across trophic levels. A number of studies have assessed both BMF and TMF within ecosystems spanning the Arctic, Canada, USA, Netherlands and Brazil with reported values of >1 (critical value), suggesting that bioaccumulation of PFOA is taking place across trophic levels (Houde et al., 2006; Kelly et al., 2009; Martin et al., 2004; Tomy et al., 2004, 2009; Butt et al., 2008).

66. A literature review of studies reporting the BMF and TMF of PFOA indicated wide ranges of BMF (min=0.04; max=125) and TMF (min=0.58; max=13), raising the concern of the reliability of these values as indicators of bioaccumulation potential. This might be a result of several factors such as nonachievement of steady-state conditions, uncertainties in the feeding ecology and the impact of metabolism of precursor compounds. It is suggested that BMFs and TMFs to be determined in dietary studies performed under strictly controlled conditions with consideration of measured elimination half-lives (Franklin, 2016).

Bioconcentration studies in aquatic organisms

67. Because of the high surfactant capacity and solubility of PFOA, fish might excrete PFOA through the gills leading to reduce uptake and bioaccumulation. This explains the often low values seen in BMF/BAF tests using fish. Equally for BMF/TMF analysis where the higher predators within the food chain are fish species it can be seen that the critical values fall below 1. BCF values within the aquatic environment tend to be low. Studies based on water breathing animals provide evidence that the bioaccumulation criterion in the aquatic environment is not met (OECD, 2006; Kelly et al., 2009). There are several studies investigating the isomer profile of PFOA in the aquatic organisms. In a marine food web, the linear isomer of PFOA is the dominant form found in biota. In another study, branched PFOA isomers accumulated in rainbow trout tissue (Fang et al., 2014; De Silva et al., 2009).

68. Studies for air breathing aquatic species are scarce. However, the potential for bioaccumulation with marine mammals is confirmed. There is evidence that PFOA biomagnifies in air-breathing mammals because BMFs range from 1.3 – 125 for selected predator prey relationships and TMFs range from 1.1 to 13 for selected food chains. For non-fish species, particularly air-breathing terrestrial and avian species, bioaccumulation is shown to occur (Houde et al., 2006; Butt et al., 2008; van den Heuvel-Greve et al., 2009; Müller et al., 2011).

69. More detailed information on bioconcentration in aquatic organisms is provided in section 2.2.2 of document UNEP/POPS/POPRC.12/INF/5.

Bioaccumulation studies in terrestrial organisms

70. A number of studies have been completed highlighting the presence of PFOA in higher-tier terrestrial species. Studies include an overview of the presence of perfluorinated chemicals in marine, limnetic and terrestrial biota in Germany (Rüdel et al., 2011). PFOA was detected in liver samples from Canadian bears, and ecosystem studies in Canada highlighted the presence of PFOA above detectable limits in lichen, caribou and wolves. Trophic magnification factors for wolf/caribou/lichen (or vegetation) ranged from 1.1 to 2.4. (Martin et al., 2004; Müller et al., 2011). In these three studies, the potential for PFOA to bioaccumulate within terrestrial species was confirmed. A feeding study with fattening pigs showed that PFAAs accumulated in their tissues. The authors modeled related toxicokinetics and concluded that pigs exhibit longer elimination half-lives (236 days for PFOA) than reported for most organisms (Numata et al., 2014).

71. More detailed information on bioconcentration in terrestrial organisms is provided in section 2.2.2 of document UNEP/POPS/POPRC.12/INF/5.

Summary on bioaccumulation

72. 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.

73. 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 (see specific section 2.3.2 on human exposure).

2.2.3 Potential for long-range environmental transport

74. The screening dossier (UNEP/POPS/POPRC.11/5) highlights the high persistence and stability of PFOA, its salts and PFOA-related compounds, particularly within the air compartment. Hurley et al. (2004) quote that the atmospheric lifetime of PFOA with respect to hydroxyl radicals has been predicted to be 130 days (see Table 6). This would be sufficient amount of time to allow PFOA to travel considerable distances from the point of emission.

75. Franklin (2002) calculated an atmospheric lifetime of PFOA to be in the order of days if PFOA is emitted from a ground source, and therefore, PFOA is likely not subject to long-range transport. However, if PFOA is produced from an atmospheric source (i.e. via precursors) and if the major loss mechanism is wet or dry deposition, then it may have a lifetime of 20-30 days before deposition (Ellis et al, 2004b). This would be sufficient amount of time to allow PFOA transport over many thousands of kilometres.

76. Modeling studies have reported oceanic transport as the dominant pathway for distributing PFOA (primarily from direct sources) to the Arctic. It is believed that ocean circulation and its variations are factors that determine the long-range distribution and fate of PFOA (Stemmler and Lammel, 2010; Armitage et al., 2009).

Presence of PFOA, its salts and PFOA-related compounds in remote areas

77. Alongside the persistent characteristics of PFOA and environmental modeling data to suggest the capacity for long-range transport, environmental monitoring data from a number of studies also exist to help corroborate the model estimates. Table 7 provides details of monitoring for PFOA within surface water, ice, sediment and biota from remote locations far from the point of use and emission.

Table 7: Examples of presence of PFOA in remote areas

Sample	Value	Remarks	Reference
Surface Water			
Canadian Arctic lakes (Meretta, Resolute, Char, Small, North, and 9 Mile)	0.6 – 17 ng/L (range)	2010-2011	(Lescord et al., 2015)
Sea Water / Ice			
Baydaratskaya Bay (Russian Federation)	0.1307 (±0.077.2) ng/L (average ± std dev.)	2007	(Saez et al., 2008)
Greenland Sea	0.020 – 0.111 ng/L (range)		(Theobald et al., 2007; ECHA, 2015a)
Sediment			
Canadian Arctic lakes (Char Lake and Resolute Lake)	1.7 and 7.5 ng/g dw <1.1 and 2.3 ng/g dw 1.2 and <1.8 ng/g dw (range)	0-1 cm (depth) 1-2 cm 2-3 cm	(Stock et al., 2007)
Air			
High volume air samples collected from vessels on Atlantic Ocean, Southern Ocean and Baltic Sea High volume air samples from a land-based site close to Hamburg, Germany	0.7 pg/m^3 (average) 0.3 pg/m^3 (average)	2007-2008	(Dreyer et al., 2009)
Birkenes Zeppelin Andøya (Norway)	0.32 pg/m^3 0.22 pg/m^3 0.19 pg/m^3 (average)	2014	(NILU, 2015)
Cornwallis Island (Canada)	1.4 pg/m^3 (average)	2004	(Stock et al., 2007)
Biota			
Polar bear (liver) (East Greenland)	0.6 – 14 ng/g ww 6.8 – 15.8 ng/g ww 11.8 – 17.6 ng/g ww (range) 14 ng/g ww 12.3 ng/g ww 4.8 ng/g ww (annual average)	1990 (year) 1995 2006 2006 2010 2011	(Dietz et al., 2007) (Rigét et al., 2013)

Sample	Value	Remarks	Reference
Polar bear (liver) (North American Arctic, European Arctic)	2.4 – 36 ng/g ww (range of geometric means)		(Smithwick et al., 2005)
Ringed seal (liver) (Arviat – Canadian Arctic)	<0.85 – 3.6 ng/g ww (range)		(Butt et al., 2007)
Ringed seal (liver) (East Greenland)	1.6 ng/g ww 2.2 ng/g ww <Limit of detection (annual average)	2006 2008 2010	(Rigét et al., 2013)

78. There is evidence of PFOA present in the remotes regions of the Arctic ice cap (US EPA, 2014), surface and sea water in Arctic regions (Butt et al., 2010b; AMAP, 2014; Benskin et al., 2012a, 2012b; Veillette et al., 2012; Kwok et al., 2013; Cai et al., 2012; Zhao et al., 2012a) snow and ice cores from China (e.g. northern China and Tibetan Plateau) (Wang et al., 2014b; Shan et al., 2015), air, snow/ice cores, water, sediment and biota samples from Norway (Xie et al., 2015; NILU, 2015; Kwok et al., 2013), snow samples from Slovakia, Switzerland and Italy (Cobbing, 2015) as well as air, water and fauna samples from Antarctica and the Southern Hemisphere (Zhao et al., 2012a; Tao et al., 2006; Danish Ministry of Environment, 2013; Wang et al., 2015).

79. Lescord et al. (2015) report evidence of PFOA in lake food webs from the Canadian high Arctic. Evidence is also reported to show the contamination in eggs of the high-artic ivory gull (*Pagophila eburnea*) (Lucia et al., 2015) and other Arctic and North Atlantic mammals (Bytingsvik et al., 2012; Rotander et al., 2012; Rigét et al., 2013). Different PFOA isomers were detected in liver samples of polar bears from the Canadian Arctic and eastern Greenland such that Greenland polar bear samples showed a variety of branched isomers while only the linear PFOA isomer was determined in Canadian samples (de Silva and Mabury, 2004). Regarding the Arctic, PFOA was detected in liver samples of killer whales, seals, and polar bears from East Greenland (Gebbink et al., 2016).

80. Fluorotelomer alcohols such as n-$C_8F_{17}CH_2CH_2OH$ appear to be a significant global source of persistent bioaccumulative perfluorocarboxylic acid pollution, and present modeling results show that with current estimates of chemistry and fluxes the atmospheric oxidation of 8:2 FTOH can provide a quantitative explanation for the presence of PFCAs including PFOA in remote regions (Wallington et al., 2006; Young et al., 2007). Besides, biotransformation of 8:2 FTOH to PFOA has been shown in laboratory experiments (Butt et al., 2014).

Potential mechanisms for long range transport

81. Bengtson Nash et al. (2010) and Prevedouros et al. (2006) discuss two key mechanisms to aid the long range transport of PFOA to remote regions. The first mechanism involves a release to environment either as air-borne dust particles contaminated directly by PFOA or salts of PFOA, or a direct release to surface waters. These releases then undergo long range transport through a cycle of deposition and volatization from marine waters, with the transport in water via the marine environment likely to be the dominant delivery method to the Arctic regions. It has been suggested that hydrospheric transport will form a slow but primary input pathway of PFCs to the Antarctic region (Bengtson Nash et al., 2010). The second mechanism involves a delivery through air-borne precursors (such as FTOHs) again through contaminated dust particles and then the degradation of these materials to form PFOA *in situ* on arrival.

82. ECHA discusses the mechanism of atmospheric transport. Due to the relative vapor pressures of APFO, PFOA, and perfluorooctanoate (PFO), the chemical form potentially most subject to gas-phase atmospheric transport is PFOA (ECHA, 2015a). Franklin (2002) suggested that in the presence of water in air, gaseous PFOA condenses to aerosol particles and dissociates to corresponding PFO. This would suggest that cycling between atmosphere and ground through wet/dry deposition and resuspension is possible and would aid the long range transport of PFOA. Climate change leading to melting snow and ice has also been proposed as a potential mechanism (Zhao et al., 2012a).

83. More detailed information on long-range transport is provided in section 2.2.3 of document UNEP/POPS/POPRC.12/INF/5.

Summary on long-range environmental transport

84. PFOA is highly stable and resistant to degradation. Monitoring of water, air, sediment and biota at remote locations all detect the presence of PFOA. Equally, environmental modeling data suggest that the capacity for long range transport (e.g. oceanic transport) does exist while others have identified key mechanisms involving precursors that would make long range transport plausible. On this basis, it can be concluded that PFOA meets the Annex D criterion for long range transport.

2.3 Exposure

2.3.1 Environmental monitoring data

85. Various studies demonstrate that PFOA is ubiquitously present in the environment. ECHA (2015a) contains a selection of studies, which report detections of PFOA and related compounds in several compartments (surface water, deep-sea water, drinking water, wastewater treatment plant, sediment, groundwater, soil, atmosphere, dust, biota, and human) at worldwide sampling locations (see ECHA, 2015a, Table A.B.4-8 in Appendix B; see also section 2.3.1 of document UNEP/POPS/POPRC.12/INF/5). No large-scale monitoring program has been conducted for PFOA and only limited time trend studies are available. There is not sufficient information available to conclude on the trend of environmental concentrations. The available time trends studies indicate both decreasing and increasing trends in biota in Europe as well as in arctic regions (ECHA 2015a, Roos and Benson, 2016, Faxneld et al., 2014; Rigét et al., 2013).As PFOA is not degradable this decreasing trend is not proven by water and sediment samples suggesting that oceans and sediments are sinks of PFOA (ECHA 2015a).

86. Environmental monitoring data has been gathered for a number of sites still producing PFOA and its related compounds in China. PFOA has been found in sediment, river water, surface and groundwater as well as drinking water, primarily near PTFE and perfluorobutane sulfonate production facility or fluorine industry parks (Bao et al., 2011; Chen et al., 2015; Shi et al., 2015, Yao et al., 2014). Although it is widely accepted that telomerization is currently the predominant manufacturing method for PFOA, yielding an isomerically pure and linear product, electrochemical fluorination is still used by some manufacturers in China (Jiang et al., 2015). This is confirmed with the analysis of isomer profiles of PFOA in fresh snow samples collected in northern China, which were in agreement with the signature of the historical 3M electrochemical fluorination products, suggesting that these products were still produced and used in China (Shan et al., 2015).

87. PFOA was found in the majority of ground water samples in the EU and of public drinking water systems in the U.S. (Loos et al., 2010; Post et al., 2009). Reasons for groundwater contamination are e.g. release to surface soils, general atmospheric deposition, landfill leachate, historical use of AFFF e.g. in the vicinity of airports and fire-fighting training sites (Xiao et al., 2015; Eschauzier et al., 2013; Yan et al., 2015; Filipovic et al., 2015; KemI, 2016). Currently, there is a large-scale drinking water monitoring program funded by the U.S. EPA under the Safe Drinking Water Act called the Unregulated Contaminant Monitoring Rule (UCMR), where over 4800 public water systems are being monitored for 30 contaminants including PFOA (US EPA, 2016).

88. An assessment of the relative contribution of historic (i.e. electronically fluorinated) and contemporary (i.e. telomer) sources of PFOA in various seawater samples indicated that the dominant PFOA source(s) to the Pacific and Canadian Arctic Archipelago are either from (1) direct emissions of contemporary PFOA via manufacturing or use in Asia or (2) from atmospheric transport and oxidation of contemporary PFOA-precursors (Benskin et al., 2012c).

89. Environmental monitoring data are available for surface water and sediment in Japan (Japan, 2015), for the river Po in Italy (ECHA, 2014b), landfill leachate, water and gull eggs and sediment in Australia (Gallen et al., 2016; Thompson et al., 2011a) and for river sediments from South Africa PFOA with higher concentrations than those found in other countries (Mudumbi et al. 2014).

90. There is abundant evidence to show detected levels of PFOA in marine, limnetic and terrestrial biota worldwide (de Silva and Mabury, 2004; Martin et al., 2004; Smithwick et al., 2005; Houde et al., 2006; Butt et al., 2007; Dietz et al., 2007; Ishibashi et al., 2008; Sonne et al., 2008; de Silva et al., 2009; Müller et al., 2011; Rüdel et al., 2011; Bytingsvik et al., 2012; Rotander et al., 2012; NILU, 2013; Fang et al., 2014; Lescord et al., 2015; Lucia et al., 2015). As previously discussed in Sections 2.2.2 and 2.2.3, bioaccumulation of PFOA occurs across trophic levels, and the detection of PFOA in biota at remote locations suggest the capacity of PFOA for long range transport, respectively.

91. A previous study reported higher levels of PFOA in pine needles (Pinus mugo) near ski areas (Chropeňová et al. 2016a). PFOA application as stain repellents in modern outdoor clothes and in ski waxes is assumed to be a potential important source in ski resorts for releases to the high mountains environment and potential for human exposure (Chropeňová et al. 2016b).

92. More detailed information on environmental monitoring is provided in section 2.3.1 of document UNEP/POPS/POPRC.12/INF/5.

2.3.2 Human Exposure

93. A large number of studies have been conducted to determine human exposure to PFOA. Among other things, findings identify common routes to exposure and vulnerable groups to exposure as well as reporting concentration levels found in samples.

94. Human exposure typically takes place "human via environment"[5] by consumption of drinking water and food, via uptake of contaminated indoor dust or from consumer products containing PFOA and its related substances and, to a lesser degree, through dermal contact. (ECHA, 2013a ; Post et al., 2009 ; Shoeib et al., 2015 ; Wilhelm et al., 2010 ; Schwanz et al., 2015 ; D'Hollander et al., 2015 ; Eriksson et al., 2013 ; Carlsson et al., 2014 ; Yamada et al., 2014). Indoor air and dust are also important sources of human PFOA exposure (Haug et al., 2011; Gebbink et al., 2015). PFOA then concentrates within the blood, liver, kidney and lungs (ECHA, 2013a).

95. Several studies have suggested that PFOA levels increase with age (Haug et al., 2010, 2011; Christensen et al., 2016) and that breast feeding, diet and indoor environment are important factors for PFOA exposure that need to be addressed in the evaluation of human exposure and accumulation of PFOA (Haug et al., 2010, 2011; Brantsater et al. 2013; ECHA, 2013a).

96. Dietary intake including water is considered as the most important route of human exposure to PFOA based on studies from various countries. There is evidence that for children, the intake is higher (Vestergren et al., 2008; Haug et al., 2010, 2011; Thompson et al., 2011b; Cornelis et al., 2012, Shan et al., 2016). 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 (US EPA, 2016b).

97. The issue of human exposure via consumer products was examined by Vestergren et al. (2015), who modeled emissions from consumer products imported from China and finds that 1.5% of PFOA discharged to wastewater and 0.3% of 8:2 FTOH emissions to air in Norway can be attributed to these products imported to Norway.

98. Analysis of serum samples demonstrate that PFOA is detectable across the general population in various countries (Emmett et al. 2006; Vestergren and Cousins, 2009; Fromme et al., 2009; Health Canada, 2013; CDC, 2015; Cho et al., 2015; Góralczyk et al., 2015; Wuttke et al., 2013; Vorkamp et al., 2014).

99. Based on the existing studies of serum/plasma PFOA concentrations in the general European adult population and in children worldwide, the average median (and maximum) PFOA serum levels were calculated to be 3.5 (21) ng/mL and 6.4 (108) ng/mL for European adults and children worldwide, respectively (ECHA, 2015a; also see UNEP/POPS/POPRC.12/INF/5 for list of studies).[6]

100. It has been estimated that serum and plasma PFOA concentrations in the European populations ranged from <0.5 – 40 µg/mL (Fromme et al., 2009; Vestergren and Cousins, 2009). PFOA was also detected in the general American population and Canadian population including children in Nunavik as well as Canadians living on reserve with geometric mean concentrations ranging from 1.39 – 2.5 µg/L (Health Canada, 2013; Wuttke et al., 2013 AMAP, 2015; CDC, 2015). Levels of PFOA were also found in people throughout the circumpolar Arctic, and in some countries such as Iceland and Norway, PFOA levels within the sampled populations were among the highest compared to other POPs (AMAP, 2015). A US study of older male anglers found PFOA serum levels similar to those of the US general population, and increasing age as well as alcohol consumption were observed to be predictors of PFOA levels (Christensen et al., 2016). Several studies indicated that human PFOA concentrations in Australian adults and Danish pregnant women decreased over time (Toms et al., 2014; Bjerregaard-Olesen et al., 2016) and that concentrations of PFOA in Australian women of child-bearing age are higher compared to Germany and the USA (Toms et al., 2014).

101. There are a few reports of detected PFOA serum levels in the occupationally exposed workers, (Guruge et al., 2005; Fromme et al., 2009; Freberg et al., 2010; Nilsson et al., 2010b). Indoor dust and total suspended particles seem to be important occupational exposure routes in fluorochemical manufacturing and is also considered relevant in domestic settings (Gao et al., 2015; Eriksson and Kärrman, 2015; Tian et al., 2016). In some cases, elevated serum PFOA levels can be largely

[5] Indirect exposure of humans via the environment may occur by consumption of food (fish, crops, meat and milk) and drinking water as well as inhalation of air (ECHA, 2012).

[6] It should be mentioned that for the calculation of average median (and maximum) PFOA serum levels reported in ECHA, 2015a, one outlying value from the study by Mondal et al., (2012) was included in the calculation. By removing this outlying value, the average median (and maximum) PFOA serum levels in children is 2.52 ng/mL (15.9 ng/mL)

attributed to exposure to PFOA-related compounds such as 8:2 FTOH (Nilsson et al., 2010a, 2010b). Persons living in the vicinity of fluorochemical manufacturing plants have higher PFOA levels than the general population (Emmett et al., 2006; Oliaei et al., 2013; Hoffman et al., 2011).

102. Mothers excrete PFOA via breast milk, which is considered an important source of exposure to breast-fed infants, whose PFOA exposure level is considerably higher than adults (Haug et al., 2011). PFOA is also transferred to the fetus through the placenta, and it has been reported that total branched PFOA isomers cross the placenta more efficiently than linear isomers (Beeson et al. 2011). Time interval between pregnancies has been shown to be strongly correlated with increased concentrations of PFOA in pregnant women, possibly reflecting the re-accumulation of PFOA in maternal blood with increasing time between pregnancies (Brantsæter et al., 2013). Due to *in utero* exposure and exposure from breast milk and drinking water, children up to about 12 years of age have higher serum PFOA concentrations than their mothers (Mondal et al., 2012). Breastfeeding is an important exposure pathway to PFOA as well as trans-placental passage (Umweltbundesamt AT, 2012, 2013; Carious et al., 2015; Forns et al., 2015; Papadopoulou et al., 2015; Hanssen et al., 2013; Mogensen et al., 2015, Brantsæter et al., 2013).

103. Studies related to breastfeeding from Jordan, Spain and Italy indicate that PFOA levels are higher in older women and that there is a greater transfer of PFC during breastfeeding by primiparous and thus a higher exposure to these contaminants for the first child (Al-sheyab et al., 2015; Motas Guzmàn et al., 2016; Barbarossa et al., 2013).

104. A recent study reported the detection of PFOA in 27 out of 30 human hair samples with a PFOA concentration range of 25-74 pg/g hair and an average of 46 pg/g hair (Alves et al., 2015).

105. Humans are very slow eliminators of PFOA compared with other species such as rodents, pigs and monkeys (Olsen et al., 2007; Numata et al., 2014) with a half-life ranging between 2 and 4 years (ECHA, 2014a; Olsen et al., 2007; Russell et al., 2015; Bartell et al., 2010; Han et al., 2012).

106. More detailed information on human exposure is provided in section 2.3.2 of document UNEP/POPS/POPRC.12/INF/5.

Summary of human exposure

107. 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 substances. 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. There is evidence that PFOA levels in humans accumulate and increase with age. Humans are very slow eliminators of PFOA compared with other species with an average half-life of 2-4 years.

2.4 Hazard assessment for endpoints of concern

Adverse effects on aquatic organisms

108. According to several information sources, data currently available for PFOA indicate some adverse effects on a number of aquatic organisms. Generally, acute aquatic toxicity is low in standard ecotoxicity tests (NICNAS, 2015a); moderate to low acute toxicities are seen in pelagic organisms including fish, and low chronic toxicities in benthic organisms (Environment Canada and Health Canada, 2012). Adverse effects include intergenerational toxicity in the first offspring generation (Ji et al., 2008) and some PFOA-mediated toxicity in freshwater algae (Elnabarawy et al., 1981; Ward et al., 1995a, 1995b, 1996a, 1996b, 1996c; Boudreau, 2002; Thompson et al., 2004 as cited in Environment Canada and Health Canada, 2012; Latała et al., 2009) and other aquatic organisms (3M Company 1987a, 1990a, 1996a, b, c; Beach 1995a cited in Environment and Health Canada 2012). Further, PFOA-mediated effects on fish development, particularly in reproduction, have been observed (Ulhaq et al. 2015; Wei et al., 2007; Wei et al., 2008). Studies with other aquatic organisms such as freshwater male tilapia, marine mussels and Baikal seals showed estrogenic effects, hepatotoxicity, inflammation, and chemosensitivity (Liu et al., 2007; Stevenson et al., 2006; Ishibashi et al., 2008). Field studies related to effects of PFOA on immune function and clinical blood parameters in dolphins and sea turtles revealed increases in indicators of inflammation and immunity (Peden-Adams et al., 2004a, 2004b). Increased pro-inflammatory responses in male Japanese medaka were also observed (Yang, 2010). Activation of peroxisome proliferator–activated receptor α were shown in Baikal seals (Ishibashi et al., 2008b). Evidence indicates that PFOA could exacerbate the adverse effects triggered by certain types of pesticides (Rodea-Palomares et al., 2015).

Adverse effects on terrestrial organisms

109. The effects of repeated oral exposure to PFOA have been evaluated in mice (Loveless et al., 2006; Christopher and Marisa, 1977; Griffith and Long, 1980; Lau et al., 2006; Macon et al., 2011; Abbott et al., 2007; Wolf et al., 2007), rats (Metrick and Marisa, 1977; Griffith and Long, 1980; Goldenthal, 1978; Palazzolo, 1993; Biegel et al., 2001; Perkins et al., 2004) and monkeys (Goldenthal, 1978b; Griffith and Long, 1980; Thomford, 2001; Butenhoff et al., 2002). Hepatocellular hypertrophy was observed in all species. At lower doses, reduced body weight as well as increased kidney and liver weight were noted. Also, degeneration and/or focal to multifocal necrosis were reported with increased severity at doses between 1.5 to 15 mg/kg bw/day in rats and mice. Mortality was observed at high doses (ECHA, 2015a).

110. No-observed- and lowest-observed-adverse-effect-levels (NOAELs and LOAELs respectively) were identified from critical and relevant studies for the observed effects. The NOAEL and LOAEL for increased liver weight and hepatocellular hypertrophy based on subchronic toxicity studies in rats are 0.056 mg/kg bw/day and 0.64 mg/kg bw/day, respectively (ECHA, 2015a; Perkins et al., 2004). The LOAEL of maternal toxicity was 1 mg/kg bw/day based on increased liver weight based on a developmental toxicity study in CD-1 mice (Macon et al. 2011). White et al. (2009) identified a LOAEL of 1.0 mg/kg bw/day for delayed mammary gland development in F1 (the first offspring generation). Abbott et al. (2007) calculated a NOAEL of 0.3 mg/kg bw/day for neonatal survival based on developmental exposure of mice.

111. Toxicological studies in rats have shown that PFOA reduces serum lipids while it increases hepatic triglycerides, probably through the activation of the peroxisome proliferator-activated receptor α (PPARα) (Haugom and Spydevold, 1992; Bjork et al., 2011). A study by Butenhoff et al. reported a dose-dependent increase in serum triglycerides in monkeys and only a moderate and non-significant reduction in cholesterol with increasing PFOA (Butenhoff et al., 2002).

112. The induction of tumours has been demonstrated in rats exposed to PFOA. Rats chronically exposed to PFOA resulted in increased incidences of liver adenomas, Leydig cell hyperplasia/adenomas and pancreatic acinar cell tumours (PACT) in male Sprague-Dawley rats (Biegel et al., 2001). Mammary fibroadenoma in the female rats was observed in another chronic PFOA exposure study although this observation has since been disputed after an independent group of pathologists (Pathology Working Group) re-examined the tissue and reached a consensus that incidence of mammary gland neoplasms were not affected by chronic PFOA exposure (Hardisty et al., 2010; Butenhoff et al., 2012).

113. Animal studies show that PFOA increases the incidence of complete litter loss, postnatal mortality, decreases foetal body weight, delays ossification, changes mammary gland development and delays maturation in several developmental studies in mice (and some in rat) depending on strain, dose, time and length of exposure (Lau et al., 2006; Abbott et al., 2007; Macon et al., 2011; White et al., 2007, 2009, 2011; Wolf et al., 2007; Yang et al., 2009; Zhao et al., 2012b; Dixon et al., 2012, Suh et al., 2011; Albrecht et al., 2013). The LOAEL of maternal toxicity was determined as 1 mg/kg bw/day, and the NOAEL for neonatal survival was 0.3 mg/kg bw/day (Lau et al., 2006; Abbott et al., 2007). The National Institute of Environmental Health Sciences (US) reviewed the evidence for the effects of PFOA on foetal growth in animals, and the authors concluded that there is sufficient evidence of decreased foetal growth in non-human mammalian species (Koustas et al., 2014).

114. Effects of gestational exposure to PFOA on the development of the mammary gland from lactating dams and female pups have been demonstrated (White et al., 2007, 2009). PFOA, when exposed in a critical window of susceptibility (GD 10-17), was shown to induce changes in offspring mammary gland development in CD-1 mice (Macon et al., 2011). In addition, chronic low dose exposure of PFOA reduced mammary gland development in F_1 as well as F_2 (Yang et al. 2009). Zhao et al. (2010) observed that PFOA stimulates mammary gland development in C57BL/6 mice. Some of observed PFOA-induced developmental/reproductive effects might be mediated by PPARα (Zhao et al., 2012b; Albrecht et al., 2013). However, it has been mentioned that PFOA-induced alterations in mammary gland development might be to be dependent on steroid production in ovaries and independent of PPARα (Zhao et al., 2010).

115. There are studies suggesting that PFOA may alter steroid hormone production or act indirectly via ovarian effects as a mean of endocrine disruption (Zhao et al., 2010, 2012b; York, 2002; Butenhoff et al., 2004; Suh et al., 2011; Hines et al., 2009). PFOA has been reported to alter sexual maturation and pubertal timing in female and male offspring of rats and in multiple strains of mice (York, 2002; Butenhoff et al., 2004, Yang et al., 2009; Dixon et al. 2012), indicating a disruption of the normal steroid hormone regulation. Also, inhibited expression of the placental prolactin-family hormone genes was observed in the placenta of mice exposed to PFOA during gestation (Suh et al., 2011).

116. There are indications of immune effects mediated by PFOA. Short-term dietary exposure to PFOA resulted in a reduction in thymus weight, decreased number of thymocytes and splenocytes and suppressed IgM antibody synthesis in C57BL/6 mice (DeWitt et al., 2008; DeWitt et al., 2009; Qazi et al., 2009). Adult offspring exposed to PFOA given to dams from gestation through lactation exhibited altered immune responses such as reduced splenic T cells and IL-10 production from these cells (Hu et al., 2012). PFOA was also shown to increase histamine release from mast cells as well as exacerbate the IgE-dependent local allergic reaction in mice (Singh et al., 2012). Lastly, suppressed T-cell-mediated immunity in Japanese quails was observed after exposure to PFOA via drinking water for 8 weeks (Smits and Nain, 2013).

117. The authors of the background document for the restriction of PFOA (ECHA, 2015a) emphasize the importance of assessing mice studies instead of rat studies as basis for DNEL-setting when this is based on animal studies due to the longer half-life of PFOA in mice compared to rats.

118. Studies with chicken showed no significant outcomes such as alterations in embryonic pipping success or in biochemical parameters at concentrations up to 10 µg/g ww of embryo and doses up to 1.0 mg/kg body weight for 3 weeks (O'Brien et al., 2009; Yeung et al., 2009). However, a recent study observed developmental toxicity (namely reduced embryo survival) of PFOA in cormorant (*Phalacrocorax carbo sinensis*), herring gull (*Larus argentatus*) and the domestic White Leghorn chicken (*Gallus gallus domesticus*), with chicken being the most sensitive species (Nordén et al., 2016).

119. The soil-dwelling nematode *Caenorhabditis elegans* showed lethal effects with EC_{50} concentrations of 3.85 mM after 1 hour of exposure and 2.35 mM after 48 hours of exposure (Tominaga et al., 2004).

120. Studies with terrestrial plants such as lettuce (*Lactuca sativa*), cucumber (*Cucumis sativus*) and pakchoi (*Brassica rapa chinensis*), spring wheat, oats, potatoes, maize, and perennial ryegrass showed species-dependent adverse effects (e.g. root growth and necrosis) mediated by PFOA (Li, 2008; Stahl et al., 2009).

121. PFOA is primarily detected in the livers of biota such as polar bears and seals (Martin et al., 2004; Smithwick et al., 2005; Dietz et al., 2007; Sonne et al., 2008; Butt et al., 2007; Ishibashi et al., 2008), but the adverse effects of PFOA in such biota have not yet been elucidated. Ishibashi et al., (2008) showed activation of PPARα in the livers of Baikal seals, but no PFOA-mediated adverse effect in the livers was reported. Also, Sonne et al., (2008) concluded that it is not clear whether chronic exposure to PFOA is associated with liver lesions in polar bears, but it is possible that at sufficient concentrations in polar bears, PFOA might induce hepatic alterations.

122. Environment Canada and Health Canada (2012) concluded that the risk quotients (PEC/PNEC) indicate low likelihood of risk to pelagic organisms, mammalian wildlife from exposures at current concentrations in the environment. However, according to the ECHA (2014), the properties of persistent, bioaccumulative, and toxic (PBT) chemicals create uncertainty in the risk estimation to the environment and human health when performing quantitative risk assessment. ECHA stated that using the currently available methods, a "safe" concentration in the environment cannot be established quantitatively with sufficient reliability for an acceptable risk. Furthermore, 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. Indeed, a temporal trend analysis indicated an annual increase of 2.3% in PFOA levels in East Greenland polar bears from 1984 to 2006 (Dietz et al., 2007).

123. Detailed information on adverse effects on aquatic and terrestrial organisms is presented in the background document (see section 2.4 of document UNEP/POPS/POPRC.12/INF/5).

Summary of ecotoxicological effects

124. 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.

125. 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. In plants, PFOA can cause visible abnormalities and alter root

growth. Because of the tendency of PFOA to bioaccumulate, PFOA concentrations in polar bears might increase over time and approach exposures resulting in harm.

Adverse effects on human health

126. The toxicity of PFOA has been evaluated by ECHA, US EPA, the Canadian ministries and EFSA (ECHA, 2011, 2015a; Environment Canada and Health Canada, 2012; US EPA, 2006, 2016b; EFSA, 2008). In the European Union, PFOA has a legally-binding harmonized classification as Carc. 2, Repr. 1B and STOT RE 1 (liver) according to Regulation (EU) No 944/2013.

127. 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. PFOA does not undergo metabolism or biotransformation in the body (Environment Canada and Health Canada, 2012; Post et al., 2012). As mentioned earlier, the half-life of PFOA elimination in humans is long, ranging between 2 and 4 years (Olsen et al., 2007; Russell et al., 2015).

128. Animal studies have demonstrated the induction of tumours mediated by PFOA or APFO, and hepatic activation of PPARα has been proposed as a mechanism of induction of hepatic tumours (Klaunig et al., 2003). 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 (Meek et al., 2003; Boobis et al., 2006), and PFOA compounds have also not been tested for carcinogenic potential in any laboratory animal species other than rats (Environment Canada and Health Canada, 2012). Therefore, the ECHA's Risk Assessment Committee (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) (Benbrahim-Tallaa et al., 2014; IARC, 2016).

129. 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 (US EPA 2016b). Positive correlation in PFOA level between maternal and cord blood samples has been reported in several birth cohort studies in Spain and Norway (Manzano-Salgado et al., 2015; Gutzkow et al., 2012). As APFO can be transferred to infants through breast-feeding, the RAC agreed on an additional classification of PFOA on lactation effects (CLP: Lact. H362: May cause harm to breast-fed children) (ECHA, 2011).

Epidemiological studies

130. Several epidemiological studies investigated exposure and related health effects in workers and different population groups. One of the most extensive epidemiological studies regarding PFOA in humans is the C8 Health Project, a population-wide health study to assess whether or not there is a probable link between C8 exposure and disease in the community. The background behind the C8 Health Project can be found online (C8 Science Panel, 2013).

131. Exposure of PFOA and elevated cholesterol levels (e.g. hypercholesterolemia) as well as other lipid parameters (e.g. uric acid, serum lipid) have been positively correlated in studies involving occupationally-exposed workers (Sakr et al., 2007), highly exposed community residents (Steenland et al., 2009; Steenland et al., 2010a; Frisbee et al., 2010; Fitz-Simon et al., 2013), the general population (Eriksen et al., 2013; Nelson et al., 2010) and children especially boys (Qin et al., 2016).

132. There is also evidence from the C8 Health Project to suggest potential association between PFOA exposure and increased risk of testicular and/or kidney cancers (Barry et al., 2013; Vieira et al., 2013; Steenland, 2012). There are a few reports where no significant association between PFOA exposure and human cancer risk was observed (Chang et al., 2014; Leonard et al., 2008). The Eriksen study acknowledges the possibility of information bias regarding the exposure related to effect (Eriksen et al., 2009). According to IARC (2016) there is limited evidence in humans for the carcinogenicity of PFOA. A positive association was observed for cancers of the testis and kidney.

133. Ulcerative colitis and rheumatoid arthritis have been positively associated with PFOA exposure among workers (Steenland et al., 2015).

134. Adverse reproductive effects have been reported in some epidemiological studies. A positive association between serum PFOA levels and the rate of menopause has been reported in several studies such that women in the highest quintile of PFOA exposure have higher chances of experiencing earlier menopause (Taylor et al., 2013; Knox et al., 2011a), but reverse causation,

i.e. earlier menopause leading to increased PFOA, has been suggested (Dhingra et al. 2016a, 2016b). This would suggest that women who experience earlier menopause might be at risk of higher body burden of PFOA. Another study in Canada involving more than 1,700 women showed possible reduction in fecundity in women with higher plasma levels of PFOA (Velez et al., 2015). Serum PFOA is positively associated with pregnancy-induced hypertension (Darrow et al., 2013), and preeclampsia is weakly associated with PFOA level (Savitz et al., 2012; Stein et al., 2009). Higher serum PFOA levels have been found in patients with polycystic ovary syndrome (Vagi et al., 2014).

135. Developmental effects in humans may be associated with PFOA exposure. On one hand, an inverse correlation between PFOA and birth weight, ponderal index and head circumference has been reported in several mother-child cohort studies (Fei et al., 2007; Apelberg et al., 2007; Maisonet et al., 2012; Chen et al., 2012, Wu et al., 2012; Whitworth et al., 2012a). A novel meta-analysis methodology was applied to review the evidence of 9 human studies, and the outcomes of this meta-analysis indicated that there was sufficient evidence to suggest that developmental exposure to PFOA reduces foetal growth (Johnson et al., 2014; Lam et al., 2014). On the other hand, mothers of low birth weight babies might have altered renal function such as less plasma volume expansion, therefore leading to reduced clearance of PFOA through glomerular filtration (Whitworth et al., 2012b; Verner et al., 2015; Sagiv et al., 2015). Furthermore, there are other studies that reported no significant association between maternal serum PFOA levels and birth weight (Washino et al., 2009; Stein et al., 2009; Monroy et al., 2008; Hamm et al., 2010).

136. Other reported adverse effects observed in children and adolescents associated with higher levels of serum PFOA include impaired neurodevelopment (see below), adiposity (Halldorsson et al., 2012 ; Braun et al., 2016), dyslipidemia (Geiger et al., 2014), altered renal function (Kataria et al., 2015), reduced humoral immune response (Grandjean et al., 2012; Grandjean and Budtz-Jørgensen, 2013), lower levels of insulin-like growth factor-1 and sex hormones (Lopez-Espinosa et al., 2016), higher odds of self-reported food allergies (Buser and Scinicariello, 2016) and later age of puberty in girls (Lopez-Espinosa et al., 2011; Holtcamp, 2012).

Neurotoxicity in humans

137. 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 (Goudarzi et al., 2016). Also, no correlation between PFOA levels and birth weight was observed in the same cohort study (Washino et al., 2009). Statistically significant inverse associations between PFOA and memory impairment has been reported (Gallo et al., 2013). On the other hand, there are studies that reported no association between PFOA exposure and impaired neurodevelopment or behavior (Chen et al., 2013; Stein et al., 2013).

Immunotoxicity

138. The U.S. National Institute of Environmental Health Sciences' National Toxicology Program (NTP) systematically reviewed and evaluated evidence on exposure to PFOS or PFOA and immune-related health effects to determine whether exposure to either chemical is associated with immunotoxicity for humans. Based on the health effects data from 33 human studies, 93 animal studies, and 27 in vitro/mechanistic studies relevant for addressing the objective, NTP published its draft findings in June of 2016 concluding that PFOA is presumed to be an immune hazard to humans. The NTP based this conclusion primarily on evidence of suppression of the antibody response and increased hypersensitivity, as well as additional evidence that is primarily from epidemiological studies that PFOA reduced infectious disease resistance and increased autoimmune disease (US NIEHS, 2016).

139. Reduced humoral immune response has been observed in a few studies (Grandjean et al., 2012; Looker et al., 2014; Kielsen et al., 2015). In particular, elevated PFOA serum levels are associated with reduced antibody titer rise, especially to A/H3N2 influenza virus and an increased risk of not attaining a protection threshold antibody concentration (Looker et al., 2014). In the Danish National Birth Cohort study, it was reported that prenatal exposure to PFOA is not associated with increased risk of infectious diseases leading to hospitalisation in early childhood; however, when the analysis was stratified by gender, girls showed a slightly higher risk of hospitalisation for infections associated with higher maternal PFOA levels (incidence rate ratio of 1.74 at the highest quartile compared with the lowest) (Fei et al., 2010).

140. Using data from a study of immunotoxicity in children (Grandjean et al., 2012), BMDLs were calculated to be approximately 0.3 ng/mL for PFOA, in terms of the serum concentration decrease of specific antibodies. Using an uncertainty factor of ten to take into account individual susceptibility, the BMDLs would result in a reference dose serum concentration of about or below 0.1 ng/mL.

Endocrine Disruption

141. Findings from studies seem to indicate a PFOA-mediated effect on the endocrine system. Prenatal exposure of PFOA may alter testosterone concentrations in females (Maisonet et al., 2015), and an inverse correlation between parathyroid hormone 2 receptor (PTH2R) and PFOA exposure was also reported in a study of 189 women (Galloway et al., 2015). As for men, a study by La Rocca et al. (2015) 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 have been observed in the C8 Health Project (Knox et al., 2011a, 2011b).

142. 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 (Halldorsson et al., 2012). 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 (Barry et al., 2014).

143. 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 (Shrestha et al., 2015; Lopez-Espinosa et al., 2012; Knox et al., 2011b; Kim et al., 2011; Melzer et al., 2010; C8 Science Panel, 2013; Yang et al., 2016). 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) (as summarised in Steenland et al., 2010b and C8 Science Panel, 2013).

144. 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 (Kjeldsen and Bonefeld-Jørgensen, 2013).

145. According to the study of Su et al., (2016), PFOA showed a potential protective effect against glucose intolerance and the risk of diabetes.

146. Detailed information on adverse effects on human health is presented in the background document (see section 2.4 of document UNEP/POPS/POPRC.12/INF/5).

Summary of adverse effects on human health

147. PFOA is classified 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.

3. Synthesis of information

148. PFOA has been manufactured since 1947 and is currently manufactured mainly as the salt APFO. The global volume of production has gradually increased over time with ongoing production, for example, in China.

149. PFOA-related compounds have the capacity to degrade to PFOA and hence to contribute to environmental concentrations of PFOA. It is unlikely that actions against PFOA and APFO alone can fully minimize and eliminate environmental concentrations. In particular, fluorotelomer alcohols (FTOHs) have been identified as a potential source of PFOA. Numerous direct and indirect sources of

PFOA, its salts and PFOA-related compounds contribute to the overall release of PFOA to the environment. Main emission vectors are water, wastewater, dust particles and air.

150. Fluoropolymers and fluorotelomer-based polymers (FTPs) are used across many sectors for different purposes, including the manufacture of stain- and water-resistant coatings on textiles and carpets; hoses, cable and gaskets; non-stick coatings on cookware; and personal care products. Traces of PFOA can also be found in certain aqueous fire-fighting foams.

151. 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.

152. 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 K_{ow} 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.

153. 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.

154. 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 substances. 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. Humans are very slow eliminators of PFOA compared with other species. The half-life of PFOA elimination in human blood is long, ranging from 2-4 years.

155. 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.

156. PFOA is classified in line with the EU GHS criteria as Carc. 2, Repr. 1B and STOT RE 1 (liver) (Regulation (EU) No 944/2013). IARC has also categorized PFOA a Class 2B carcinogen with particular regard to testicular and kidney cancers. High cholesterol, inflammatory diseases, ulcerative colitis, thyroid disease, immune effects, pregnancy-induced hypertension, endocrine disruption and impaired neuro- as well as reproductive development have been found to be associated with PFOA exposure in humans in epidemiological studies, but more evidence is needed to determine the relationship between PFOA and the observed health effects.

4. Concluding statement

157. PFOA, its isomers, salts (since the late 1940s) and PFOA-related compounds (since the 1960s) have been widely used on a global basis. However, growing evidence of health and environmental effects has meant that concerns regarding the use of PFOA have existed for a number of decades. Stricter regulatory control and voluntary action by industry to phase-out the production and use of PFOA have been in place since 2006.

158. PFOA has been demonstrated to be highly persistent with the capacity to undergo long range transport far from the point of use. This is evidenced by monitoring data of PFOA in air, water, soil/sediment and biota in both local and remote locations like the Arctic. Additionally, PFOA-related compounds exist and have the capacity within the natural environment to degrade and form PFOA, further aiding the long range transport of these substances. PFOA can bioaccumulate in air-breathing mammals and other terrestrial species including humans. Evidence also exists to highlight the potential adverse effects of PFOA upon both environmental species and humans.

159. Based on the persistence, bioaccumulation, toxicity in mammals including humans and widespread occurrence in environmental compartments, it is concluded that PFOA, its isomers, salts and related compounds that degrade to PFOA, as a result of their long-range environmental transport,

are likely to lead to significant adverse human health and environmental effects such that global action is warranted.

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