UNITED NATIONS

Food and Agriculture Organization of the United Nations

RC

UNEP/FAO/RC/CRC.14/8

Distr.: General
22 May 2018

English only

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

Chemical Review Committee
Fourteenth meeting
Rome, 11–14 September 2018
Item 4 (c) (iii) of the provisional agenda*

Technical work: review of notifications of final regulatory action: perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds

Perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds: notifications of final regulatory action

Note by the Secretariat

I. Introduction

1. In accordance with paragraph 5 of Article 5 of the Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade, the Secretariat has received two notifications of final regulatory action for perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds that meet the requirements of Annex I to the Convention from Parties in the following two prior informed consent regions:

(a) Europe: Norway (industrial);[1]

(b) North America: Canada (industrial).[2]

2. The notifications received from the notifying Parties are set out in the annex to the present note. The supporting documentation provided by Norway and Canada is set out in documents UNEP/FAO/RC/CRC.14/INF/13 and UNEP/FAO/RC/CRC.14/INF/14, respectively.

II. Proposed action

3. The Committee may wish:

(a) To review the information provided in the notifications and supporting documentation from Norway and Canada related to perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds in accordance with the criteria set out in Annex II to the Convention;

(b) If it concludes that the notifications meet the criteria set out in Annex II to the Convention, to recommend to the Conference of the Parties that the chemicals in question be made subject to the prior informed consent procedure and, accordingly, be listed in Annex III to the Convention, and to agree on a workplan for the preparation of a draft decision guidance document on perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds.

* UNEP/FAO/RC/CRC.14/1.

[1] See PIC Circular XLI of June 2015.

[2] See PIC Circular XLVII of June 2018.

K1801246 280518

Annex

Notifications of final regulatory action for perfluorooctanoic acid (PFOA), its salts and PFOA-related compounds

A. Notification of final regulatory action for perfluorooctanoic acid and its salts and esters in the industrial category submitted by Norway

B. Notification of final regulatory action for perfluorooctanoic acid and its salts and precursors in the industrial category submitted by Canada

FORM FOR NOTIFICATION
OF FINAL REGULATORY ACTION TO BAN OR SEVERELY RESTRICT A CHEMICAL

Country: Norway

SECTION 1 IDENTITY OF CHEMICAL SUBJECT TO THE FINAL REGULATORY ACTION

1.1	**Common name**	**PFOA and its salts and esters;** **C8; perfluorooctanoate;** **pentadecafluoro octanoic acid; perfluoroheptanecarboxylic acid; perfluoro-n-octanoic acid; Fluorad FC-26; perfluorocaprylic acid**
1.2	**Chemical name according to an internationally recognized nomenclature (e.g. IUPAC), where such nomenclature exists**	**Perfluorooctanoic acid,** **1-Octanoic acid, 2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-pentadecafluoro-PFOA, and its salts and esters** Free Acid (X = OM+; M = H) [CAS no. 335-67-1] Ammonium Salt (X = OM+; M = NH4) [CAS no. 3825-26-1] Sodium Salt (X = OM+; M = Na) [CAS no. 335-95-5] Potassium Salt (X = OM+; M = K) [CAS no. 2395-00-8] Silver Salt (X = OM+; M = Ag) [CAS no. 335-93-3] Acid Fluoride (X = F) [CAS no. 335-66-0] Methyl Ester (X = CH3) [CAS no. 376-27-2] Ethyl Ester (X = CH2-CH3) [CAS no. 3108-24-5]
1.3	**Trade names and names of preparations**	

1.4 Code numbers

1.4.1	CAS number	335-67-1, 3825-26-1, 335-95-5, 2395-00-8, 335-93-3, 335-66-0, 376-27-2, 3108-24-5
1.4.2	Harmonized System customs code	
1.4.3	Other numbers (specify the numbering system)	INDEX no: 607-704-00-2 EC no: 206-397-9

1.5 Indication regarding previous notification on this chemical, if any

1.5.1 ☒ This is a first time notification of final regulatory action on this chemical.

1.5.2 ☐ This notification replaces all previously submitted notifications on this chemical.
Date of issue of the previous notification: _____

SECTION 2 FINAL REGULATORY ACTION

2.1 The chemical is: ☐ **banned** **OR** ☒ **severely restricted**

2.2 Information specific to the final regulatory action

2.2.1 Summary of the final regulatory action

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

2.2.2 Reference to the regulatory document, e.g. where decision is recorded or published

PFOA and its salts and esters is regulated by Chapter 2-32 of the Regulation related to restrictions of the manufacture, import and placing on the market of chemicals and other products hazardous to the human health and the environment (Product Regulation). Act no. 922 of June 2004.

2.2.3 Date of entry into force of the final regulatory action

01.06.2014

2.3 Category or categories where the final regulatory action has been taken

2.3.1 All use or uses of the chemical in your country prior to the final regulatory action

PFOA has in Norway been used in several applications because of its surfactants properties i.e. coating agent for carpets, textiles, furniture, shoes, paper, food wraps, printing plates but also in paint, floor wax, glue and photograpic film. However, more often PFOA is present in products as a chemical impurity or as trace amounts of remaining starting materials from the production of other perfluorinated compounds, e.g. side-chain fluorinated polymers. PFOA has been found in imported products like textiles treated with perfluorinated compounds in order to make them water and stain repellent. PFOA may also be found in food contact materials with non-stick properties. PFOA was previously often present in ski wax in small amounts as a chemical impurity of the perfluorinated constituents in the wax.

References:

PFOA in Norway; Survey on national sources, TA-2354/2007. Available online at: http://www.miljodirektoratet.no/old/klif/publikasjoner/2354/ta2354.pdf

Per- and polyfluorinated substances in the Nordic Countries Use, occurrence and toxicology TemaNord 2013:542

Perfluorinated alkylated substances (PFAS) in the Nordic environment. TemaNord 2004:552

2.3.2 Final regulatory action has been taken for the category ☒ Industrial

Use or uses prohibited by the final regulatory action

From 1 June 2014, it is prohibited to manufacture, import, export, and sell consumer products that contain perfluorooctanoic acid (PFOA) and individual salts and esters of PFOA (CAS nr. 335-67-1, 3825-26-1, 335-95-5, 2395-00-8, 335-93-3, 335-66-0, 376-27-2, 3108-24-5), as pure substance or in a mixture when the mixture contains 0.001 weight percent or more of the substance.

From 1 June 2014, it is prohibited to manufacture, import, export and sell textiles, carpets and other coated consumer products that contain perfluorooctanoic acid (PFOA) and individual salts and esters of PFOA (CAS nr. 335-67-1, 3825-26-1, 335-95-5, 2395-00-8, 335-93-3, 335-66-0, 376-27-2, 3108-

24-5), when the content of the substance in the product's individual parts is greater than or equal to 1 µg/m2. Individual parts comprise the materials of which the product is manufactured, and the product's individual components.

From 1 June 2014, it is prohibited to manufacture, import, export and sell consumer products that contain perfluorooctanoic acid (PFOA) and individual salts and esters of PFOA (CAS nr. 335-67-1, 3825-26-1, 335-95-5, 2395-00-8, 335-93-3, 335-66-0, 376-27-2, 3108-24-5), when the content of the substance in the product's individual parts is greater than or equal to 0.1 weight percent.

The prohibitions on manufacture and export will not apply until 1 January 2016 to:

a) adhesives, foil or tape in semiconductors,

b) photographic coatings for film, paper or printing plate.

The prohibitions on import and sale will not apply until 1 January 2018 to products for which it can be documented that the manufacture took place prior to the prohibitions in paragraphs 1 to 3, cf. paragraph 4 came into force.

The above prohibitions do not apply to food packaging, food contact materials and medical devices. The prohibitions shall not apply to spare parts for consumer products made available for sale prior to 1 June 2014.

For consumer products, this section shall prevail over other provisions of this regulation.

Use or uses that remain allowed (only in case of a severe restriction)

The above prohibitions do not apply to food packaging, food contact materials and medical devices. The prohibitions shall not apply to spare parts for consumer products made available for sale prior to 1 June 2014.

For consumer products, this section shall prevail over other provisions of this regulation.

2.3.3 Final regulatory action has been taken for the category ☐ Pesticide

Formulation(s) and use or uses prohibited by the final regulatory action

n.a.

Formulation(s) and use or uses that remain allowed (only in case of a severe restriction)

n.a.

2.4 Was the final regulatory action based on a risk or hazard evaluation?

☒ **Yes**

☐ **No** (If no, you may also complete section 2.5.3.3)

2.4.1 If yes, reference to the relevant documentation, which describes the hazard or risk evaluation

Impact assessment of regulating perfluorooctanoic acid (PFOA) and individual PFOA salts and esters in consumer products. The Norwegian version is available online: «Vurdering av konsekvenser av regulering av PFOA og enkelte salter og estere av PFOA i forbrukerprodukter".

http://www.miljodirektoratet.no/Global/dokumenter/horinger/horing2010-1463_PFOA.pdf

Perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA) and their salts. Scientific Opinion of the Panel on Contaminants in the Food chain. The EFSA Journal 2008, 653, 16-131

ECHA: Pentadecafluorooctanoic acid (PFOA) as a substance of very high concern because of its CMR and PBT properties. 14 June 2013.

2.4.2 Summary description of the risk or hazard evaluation upon which the ban or severe restriction was based.

2.4.2.1 Is the reason for the final regulatory action relevant to human health?

☒ Yes

☐ No

If yes, give summary of the hazard or risk evaluation related to human health, including the health of consumers and workers

PFOA and other perfluorinated organic compounds have been widely used and are present in various consumer products that are produced and used worldwide. A number of different perfluorinated compounds have been widely found in the environment. Extensive data in humans and animals demonstrate ready absorption of PFOA and distribution of the chemical throughout the body by non-covalent binding to plasma proteins. The liver is an important binding site, and increased liver weight in laboratory animals is one of the early, low-dose manifestations of exposure. PFOA is not readily eliminated from humans as evidenced by the half-life of 2.3 years. In contrast, half-life values for the monkey, rat, and mouse are 20.8 days, 11.5 days, and 15.6 days, respectively

Human exposure to PFAS, including PFOA and PFOS, is likely to occur via a number of vectors and routes e.g. ingestion of non-food materials, dermal contact and inhalation. PFOA has been analyzed in a limited number of European environment and food samples, and has been detected in fish and eggs. Cereals were found to be the main source in a food-basket study (Haug et al., 2010a,b). Drinking water is estimated to contribute less than 16% to the indicative exposure. PFOA was also observed to leak from non-stick coatings on cookware and from food packaging of paper treated with oil- and moisture resistant chemicals. Based on the limited data available, the EFSA CONTAM Panel identified the indicative average and high level dietary exposures of 2 and 6 ng/kg b.w. per day, respectively. However, a higher estimate was found for dietary intake of PFOA (31 ng/day) in Norway by using consumption data given by Norkost 1997 (Haug et al., 2010a).

The importance of possible pathways of non-food human exposure to PFOA is of higher importance in childhood compared to adulthood. Dust has been identified as an important source of exposure, which put toddlers at risk due to their hand-to mouth behavior. For PFOA, the total contribution from the non-food sources, mainly indoor exposure, could be as high as 50% compared to the estimated average dietary exposure to PFOA.

PFOA has also been shown to be transferred from mother to the fetus, and the relatively high plasma concentration detected in blood samples from small children is of concern. Two studies show that PFOA levels in maternal blood decreased to 54% after six months and to 7% after 12 months of breast-feeding compared to their blood levels at birth, whereas PFOA levels in the serum of six-month-old infants were 4.6 times higher than maternal blood levels at birth (Thomsen et al., 2010, Fromme et al., 2010). Another Norwegian study estimated that breast-fed infants at around 6 months of age take up 4.1 ng PFOA per kg body weight, which is 15 times higher than the uptake in adults (Haug et al., 2011).

In a study from the Norwegian Mother and Child Cohort Study, Granum et al., (2013) found a positive correlation between the maternal concentrations of PFOA and PFNA and the number of episodes of common cold for the children, and between PFOA and PFHxS and the number of episodes of gastroenteritis. The results indicate that pre-natal exposure to PFAS may be associated with immunosuppression in early childhood.

In Norway the occupational exposure of professional ski-waxers to PFOA were shown to be higher than for non-occupational exposed; blood serum values were 25 fold higher (rang 15-175 ng/ ml) than previously measured among people with a high consumption of fish (Daae et al., 2009).

Epidemiology studies have examined occupational and residential populations at or near large-scale PFOA production plants in the United States in an attempt to determine the relationship between serum PFOA concentration and various health outcomes suggested by the standard animal toxicological studies. These studies have found a positive association between serum PFOA concentration and increased cholesterol levels in the general population and in worker populations but no consistent trends for the low- and high-density protein lipids. A positive association has been found between serum PFOA concentrations and increased liver enzymes and/or decreased bilirubin in both worker and general populations, chronic kidney disease in the general population, and the odds of experiencing early menopause. Epidemiology studies demonstrate an association of serum PFOA with kidney and testicular tumors among highly exposed members of the general population. Maternal or child plasma levels of PFOA were positively associated with decreased antibody titers in children after vaccination, obesogenic effects in female children at 20 years of age, and parent reported Attention Deficit Hyperactivity Disorders.

Based on a general concern for the high levels of PFOA found in environmental samples, a national action plan was initiated by the Norwegian authorities in 2002 (later updated in 2009). Furthermore, PFOA was in 2003 added to a Norwegian national target to substantially reduce the emission of certain hazardous substances by 2020, as described in a white paper to the parliament (ministry of the Environment, Norway, 2003).

In the Norwegian "Evaluation of consequences of regulating PFOA and selected salts and esters of PFOA in consumer products", the following concerns were put forward for the proposed regulation: PFOA is present in the blood of the general population, breast milk and in umbilical cord blood. PFOA is eliminated from the body very slowly. Humans are exposed to PFOA by consuming contaminated foods or water, by breathing air that is polluted as well as by ingesting dust. Fish is an important source of exposure via food. The foetus is exposed to PFOA via umbilical cord blood and newborns are exposed via breast milk. The intake for infants via breast milk can be greater than the intake via food for adults. Infants can also come into direct contact through carpeting, and swallowing dust can be an important contributor to exposure.

PFOA is a substance of very high concern with respect to its health and environmental properties. PFOA is harmful to the reproductive system, carcinogenic, toxic and harmful to human health through repeated exposure and is also an irritant. PFOA does not degrade in the environment. PFOA is a substance similar to persistent, bio-accumulating and toxic (PBT) substances or a substance of equal concern. It is impossible to establish an acceptable level for substances with such properties in the environment, and emissions and

exposure should be limited to the greatest extent possible.

References:

Fromme, H.; Mosch, C.; Morovitz, M.; Alba-Alejandre, I.; Boehmer, S.; Kiranoglu, M.; Faber, F.; Hannibal, I.; Genzel-Boroviczény, O.; Koletzko, B.; Völkel, W. Pre- and postnatal exposure to perfluorinated compounds (PFCs). Environ Sci Technol 2010, 44, 7123–7129.

Thomsen, C.; Haug, L. S.; Stigum, H.; Frøshaug, M.; Broadwell, S. L.; Becher, G. Changes in concentrations of perfluorinated compounds, polybrominated diphenyl ethers, and polychlorinated biphenyls in Norwegian breast-milk during twelve months of lactation. Environ Sci Technol 2010, 44, 9550–9556.

Haug, L.S., Salihovic, S., Jogsten, I.E., Thomsen, C., van Bavel, B., Lindström, G., Becher,G. 2010a. Levels in food and beverages and daily intake of fluorinated compounds in Norway. Chemosphere, 80, 1137–1143.

Haug, L.S., Thomsen, C., Brantsæter, A.L., Kvalem, H.E., Haugen, M., Becker, G., Alexander, J., Meltzer, H.M., Knutsen, H.K. 2010b. Diet and particulary seafood are major sources of perfluorinated compounds in humans. Environ. Int., 36, 772–778.

Haug, L.S., Huber, S., Becher, G., Thomsen, C. 2011. Characterisation of human exposure pathways to perfluorinated compounds – comparing exposure estimates with biomarkers of exposure. Environ Int 37, 687–693.

Granum B, Haug LS, Namork E, Stølevik SB, Thomsen C, Aaberge IS, van Loveren H, Løvik M, Nygaard UC. Pre-natal exposure to perfluoroalkyl substances may be associated with altered vaccine antibody levels and immune-related healthoutcomes in early childhood. J Immunotoxicol. 2013,10:373-9.

Daae et al 2009: Kjemisk eksponering og effekter på luftveiene blant profesjonelle skismørere, ISSN nr. 1502-0932

Expected effect of the final regulatory action

Reduced risk to the human health.

2.4.2.2 Is the reason for the final regulatory action relevant to the environment?

☒ Yes

☐ No

If yes, give summary of the hazard or risk evaluation related to the environment

PFOA is an anthropogenic compound widely found in the environment including the Arctic. The long-range air and ocean transport of PFOA to the Arctic give detectable levels in sea birds, seal and polar bear. The levels in polar bears have significantly increased the last 20-30 years (Smithwick et al., 2006). Furthermore, it has been shown that other more volatile perfluorated compounds can be degraded to form PFOA and thus contribute to the increased levels observed (ECHA 2013). Calculation-models has indicated that PFOA levels in the Arctic will continue to increase up to 2030 despite the voluntary actions taken to phase-out production and use of this compound (Dietz et al., 2008).

The monitoring data show that PFOA in soil leaches can be a long term source to contaminating underlying groundwater (ECHA, 2013). Sewer and leachate are significant, human-made primary sources for emissions and dispersion of PFOA into the Norwegian environment (TA-2354). In a Nordic study of perfluorinated compounds in the environment, PFOS and PFOA dominated in the sewer samples from all six Nordic countries (ref. TemaNord 2004). PFOA was dominating in leachate samples from waste deposit sites in Norway and Finland. The presence of PFOS and PFOA was also detected in sludge from processing plants (Tom Erik Økland and Kristina Skoog; TA-2450/2008). A new study has established that PFOA is only bound to sludge to a small degree and that it mainly follows the water phase through the Nordic water treatment plants (Aquateam, 2010).

Evenset et al. (2005) established PFOS and PFOA as the most common perflourinated compounds in sediments from Isfjorden on Svalbard, Norway. A study of sediments from the Barent's Sea from 2007 shows the presence of PFOA in a number of samples with a general prevalence of PFOS and perfluorocarboxylic acids with long chain lengths over PFOA. (Bakke et al., 2007).

Measurements of PFOA in air started in the autumn of 2006 at Birkenes in Southern Norway and Zeppelin on Svalbard (Manø et al., TA-2408/2008). The values at Birkenes was on average 1.04 pg/m3, Zeppelin 0.44 pg/m3, which were lower than, for example, the west coast of Ireland and in the English Channel. PFOA is also transported long distances to the Artic via sea currents. PFOA has been detected in sea water; this confirms that long-range transboundary transport via sea currents can occur (AMAP 2009).

A study of samples from polar bears in Greenland collected during the period 1984-2006 showed a significant annual increase in the levels of PFOS and some perfluorocarboxylic acids. For PFOA there was an average annual increase of 2.3%. The sum of the concentrations of perflourinated compounds was higher than the concentration of known chloro-organic priority substances. It is assumed that if the most marked increase continues, the level for harmful effect could be exceeded in 2014-2024 (Dietz et al. 2008).

The Norwegian Government has established national goals for discharge and emission reductions and cessation for 2010 and 2020, (Prop. 1 S (2009-2010) from the Norwegian Ministry of the Environment, Proposition to the Storting (Storting bill) for the 2010 budget year

for the priority substances hazardous to health and the environment (the Priority List). Perfluorooctanoic acid (PFOA) is one of the substances included in those national goals.

In the Norwegian "Evaluation of consequences of regulating PFOA and selected salts and esters of PFOA in consumer products", the following concerns were put forward for the proposed regulation: PFOA is a man-made substance that does not occur in nature. PFOA is currently widely dispersed in the environment, including in the Arctic. PFOA is transported long distances with air and sea currents, and its presence has been detected in the Arctic in (among other things) sea birds, seals and polar bears. In polar bear a significant increase in the levels of PFOA has been detected over the past 20-30 years. Other more volatile, perflourinated compounds have also been detected, which can slowly degrade to produce PFOA. Model calculations show that concentrations of PFOA in the Arctic will continue to increase until 2030 in spite of the voluntary measures that have been taken.

References:

Smithwick M, Norstrom R.J., Mabury S.A., Solomon K., Evans T.J., Stirling I., Taylor M.K., Muir D.C.G. 2006. Temporal trends of perfluoroalkyl contaminants in polar bears (Ursus maritimus) from two locations in the North American arctic, 1972-2002. Environ. Sci. Technol. 40(4):1139-1143.

ECHA: Pentadecafluorooctanoic acid (PFOA) as a substance of very high concern because of its CMR and PBT properties. 14 June 2013.

Dietz et al. 2008; "Increasing Perfluoroalkyl Contaminants in East Greenland Polar Bears (Ursus maritimus): A New Toxic Threat to the Arctic Bears", Environ. Sci. Technol. 2008, 42, 2701–2707

TemaNord 2004:552.Perfluorinated alkylated substances (PFAS) in the Nordic environment.

Tom Erik Økland and Kristina Skoog; TA-2450/2008. Available online at: http://www.miljodirektoratet.no/old/klif/publikasjoner/2450/ta2450.pdf

Aquateam, 2010. Undersøkelse av miljøgifter ved fire norske renseanlegg: PFOA, Bisfenol A, Triklosan, Siloksan (D5), Dodecylfenol og 2,4,6-Tri-tert.betylfenol. [Study of priority substances in four Norwegian processing plants: PFOA, Bisphenol A, Triclosan, Siloksan (D5), Dodecylfenol og 2,4,6-Tri-tert.betylphenol] Klif TA 2636/2010. Available online at: http://www.miljodirektoratet.no/old/klif/publikasjoner/2636/ta2636.pdf

TA-2354. PFOA in Norway; Survey on national sources, TA-2354/2007. Available online at: http://www.miljodirektoratet.no/old/klif/publikasjoner/2354/ta2354.pdf

Evenset et al. 2005: Miljøgifter i marine sedimenter, Isfjorden, Svalbard. [Priority substances in marine sediments, Isfjordenm Svalbard.] Akvaplan-NIVA-rapport APN-414. 3341.

Bakke, T., Fjeld, E., Skaare, B., Berge. J.A., Green, N., Ruus, A., Schlabach, M., and Botnen, H., 2007. Kartlegging av metaller og utvalgte nye organiske miljøgifter 2006. Krom, arsen, perfluoralkylstoffer, dikloretan, klorbenzener, pentaklorfenol, HCBD og DEHP. [Mapping out of metals and select new organic environmental toxins 2006; Chromium, arsenic, perfluoroalkyl substances, dichloroethane, chlorobenzenes, pentachlorophenol, HCBD and DEHP] SFT TA2284/2007. NIVA report 5464-2007. 105s.

Manø S, Herzke D, Schlabach M, Nye miljøgifter i luft [New priority substances in air] (TA-2408/2008). . Available online at: http://www.miljodirektoratet.no/old/klif/publikasjoner/2408/ta2408.pdf

AMAP, 2009. Arctic Pollution 2009 (POPs, Human Health, Radioactivity). Arctic Monitoring and Assessment Programme (AMAP), Oslo, Norway. xi+83 pp

Impact assessment of regulating perfluorooctanoic acid (PFOA) and individual PFOA salts and esters in consumer products. The Norwegian version is available online: «Vurdering av konsekvenser av regulering av PFOA og enkelte salter og estere av PFOA i forbrukerprodukter".
http://www.miljodirektoratet.no/Global/dokumenter/horinger/horing2010-1463_PFOA.pdf

Expected effect of the final regulatory action

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

2.5 Other relevant information regarding the final regulatory action

2.5.1 Estimated quantity of the chemical produced, imported, exported and used

	Quantity per year (MT)	Year
produced	n.a	
imported	n.a	
exported	n.a	
used	n.a	

2.5.2 Indication, to the extent possible, of the likely relevance of the final regulatory action to other states and regions

Similar concerns to those identified are likely to be encountered in other countries where the substance is used. PFOA is present in various products that are distributed globally. Adaptation of the methods of manufacture to meet the Norwegian requirements may lead to reduced levels of PFOA in products in other countries as well. Several textile brands have phased out the use of perfluorinated compounds as water repellence treatment because of the negative attention that has been given to such compounds from various stakeholders.

2.5.3 Other relevant information that may cover:

2.5.3.1 Assessment of socio-economic effects of the final regulatory action

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

2.5.3.2 Information on alternatives and their relative risks, e.g. IPM, chemical and non-chemical alternatives

EPA's review of alternatives to perfluorinated chemical substances has been ongoing since 2000 and is consistent with the approaches to alternatives encouraged under the 2010/15 PFOA Stewardship Program. Through June 2008, over 100 alternatives of various types have been received and reviewed by EPA. http://www.epa.gov/oppt/pfoa/pubs/altnewchems.html#overview

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

OECD/UNEP Global PFC Group, Synthesis paper on per- and polyfluorinated chemicals (PFCs), 2013.

Wang, Z., Cousins, I.T., Scheringer, M., Hungerbühler, K., 2013. Fluorinated alternatives to long-chain perfluoroalkyl carboxylic acids (PFCAs), perfluoroalkane sulfonic acids (PFSAs) and their potential precursors. Environ Int 60, 242-248

2.5.3.3 Basis for the final regulatory action if other than hazard or risk evaluation

n.a.

2.5.3.4 Additional information related to the chemical or the final regulatory action, if any

n.a.

SECTION 3 PROPERTIES

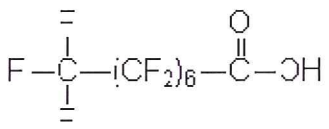
3.1 Information on hazard classification where the chemical is subject to classification requirements

International classification systems e.g. WHO, IARC, etc.	Hazard class

Other classification systems e.g. EU, USEPA	Hazard class
GHS classification according to regulation (EC) 1272/2008	Carcinogenicity, Category 2; H351 Reproductive toxicity, Category 1B, H360D Reproductive toxicity, Additional category for effects on or via lactation; H362 Acute toxicity, Category 4, oral; H302 Acute toxicity, Category 4, inhalation; H332 Specific Target Organ toxicity (repeated exposure), Category 1; H372 Serious eye damage, Category 1; H318
REACH	Due to its PBT and CMR properties, PFOA and its ammonium salt (APFO) has been identified as substances of very high concern (SVHC) under REACH by unanimous agreement between EU Member States in July 2013.

3.2 Further information on the properties of the chemical

3.2.1 Description of physico-chemical properties of the chemical



Chemical Name: Perfluorooctanoic Acid

Molecular formula: C8 H F15 O2

Structural formula: F-CF2-CF2-CF2-CF2-CF2-CF2-CF2-C(=O)-X,

The free acid and some common derivatives have the following CAS numbers:

The perfluorooctanoate anion does not have a specific CAS number.

Free Acid (X = OM+; M = H) [CAS no: 335-67-1]

Ammonium Salt (X = OM+; M = NH4) [CAS no: 3825-26-1]

Sodium Salt (X = OM+; M = Na) [CAS no: 335-95-5]

Potassium Salt (X = OM+; M = K) [CAS no: 2395-00-8]

Silver Salt (X = OM+; M = Ag) [CAS no: 335-93-3]

Acid Fluoride (X = F) [CAS no: 335-66-0]

Methyl Ester (X = CH3) [CAS no: 376-27-2]

Ethyl Ester (X = CH2-CH3) [CAS no: 3108-24-5]

Synonyms: 1-Octanoic acid, 2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-pentadecafluoro-PFOA

Molecular weight: 414.07 g/mol

Melting point: 54.3°C

Boiling point: 192.4°C

Vapor pressure: 2,3-128 Pa, 20°C

Water solubility: 3.4 g/L, 20°C; 4.14g/L, 22 °C; 9.5 g/L, 25°C

pH (1g/L, 20°C): 2.6 (MSDS Merck)

Log Kow: 6.3

Log Koc: 2.06 (Higgins and Luthy, 2006)

Reference

IFA, Institut fur Arbeitsscultz der Deutschen Gesetzlichen Unfallversicherung (http://gestis-en.itrust.de/nxt/gateway.dll/gestis_en/493012.xml?f=templates$fn=default.htm$3.0)

http://echa.europa.eu/documents/10162/e9cddee6-3164-473d-b590-8fcf9caa50e7

Perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA) and their salts. Scientific Opinion of the Panel on Contaminants in the Food chain. The EFSA Journal 2008, 653, 16-131.

3.2.2 Description of toxicological properties of the chemical

The EU Technical Committee on Classification and Labelling of Dangerous Substances has agreed that PFOA should be classified as follows (Summary Record of the Technical Committee Meeting of 3rd October 2006):

Carc. Cat. 3; R40, Repr. Cat 2; R61, NC Repr. Cat. 3; R62, T; R48/23, Xn; R20/22, Xn; R48/22, Xi; R36.

In brief as described for mammals in ECHA 2011, 2013:

Uptake,metabolism and elimination

PFOA is efficiently taken-up by mammals from all exposure routs, and is not readily eliminated. In humans, half-life is estimated to 2.3 years (Bartell et al., 2010), but even longer half-life has been estimated for retired workers from the 3M plant with high plasma PFOA levels (Burris et al., 2000, 2002, Olsen et al., 2005). In contrast, half-life values for the monkey, rat, and mouse are 20.8 days, 11.5 days, and 15.6 days, respectively. PFOA is transferred to the foetus where it accumulates in the liver, it is also transferred to the child via breast milk.

PFCs are amphiphilic and bind to serum proteins and proteins in cell membranes, and accumulate in blood and internal organ such as liver, kidneys, testes and brain. Metabolic transformation seems to be less important for elimination. Urine is the primary route of excretion and there are large sex and species differences in the excretion of PFOA. The reason for the differences in elimination is likely that PFOA is a substrate for renal organic anion transporters, regulating active renal reabsorption, and these transporters are differentially expressed between species and sex (Han et al. 2012, as referenced in ECHA 2013).

Acute toxicity

PFOA exhibits moderate acute, oral and inhalation toxicity (ECHA 2013).

Repeated dose toxicity

In sub-acute and chronic studies, PFOA affected primarily the liver and can cause developmental and reproductive toxic effects at relatively low dose levels in experimental animals. Twenty-eight day oral toxicity studies in rats and mice showed mortality and dose-related reduced weight gain and increased liver weight at PFOA dietary concentrations of 30 mg/kg and higher (Christopher and Martin, 1977; Metrick and Marias, 1977 as referenced in EFSA 2008,) or drinking water concentrations of 50 mg/L and above (So et al., 2007 as referenced in EFSA 2008).

Carcinogenicity

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

hyperplasia in male rats (ECHA 2011).

Mutagenicity

PFOA has not been shown to be mutagen. The negative outcome in a comprehensive series of in vitro and in vivo short-term tests at gene and/or chromosome level indicates that APFO is devoid of significant genotoxic activity (ECHA 2011).

Developmental and reproductive toxicity

PFOA has been shown to cause developmental and reproductive toxic effects at relatively low dose levels in experimental animal. Several studies observed complete litter loss at doses of 5 mg/kg bw/day. Increased postnatal pup mortality, decreased pup body weight and delayed sexual maturation were observed in several mice studies, A two generation reproductive toxicity study in rats has shown post-weaning mortality, reduced growth, and delayed sexual maturation. Follow-up developmental toxicity studies in mice have shown a pattern of neonatal mortality similar to that observed in mice; this consists of a dose-related increase in mortality during the first several days after birth. Cross-fostering studies have shown that the critical period of exposure is during the prenatal period. Further studies have shown delayed development of the mammary glands in both the dams and female offspring with systemic toxicity in rodents and monkeys following long-term exposure by the oral route (ECHA 2011).

Reference

(ECHA), E.C.A., 2008. Summary Of Classification and Labelling: Harmonised classification – Annex VI of Regulation (EC) No 1272/2008 (CLP Regulation) – PFOS. vol. 2012.

(ECHA), E.C.A., 2011. Opinions of the Committee for Risk Assessment on proposals for harmonised classification and labelling – PFOA. vol. 2012.

ECHA/RAC/CLH report 2011. Annex 1, Background document to the Opinion proposing harmonised classification and labelling at Community level of Perfluorooctanoic acid (PFOA).

Perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA) and their salts. Scientific Opinion of the Panel on Contaminants in the Food chain. The EFSA Journal 2008, 653, 16-131

ECHA: Pentadecafluorooctanoic acid (PFOA) as a substance of very high concern because of its CMR and PBT properties. 14 June 2013.

3.2.3 Description of ecotoxicological properties of the chemical

Persistence

PFOA is persistent and do not undergo any abiotic or biotic degradation under relevant environmental condition and meets the criteria for being very Persistent (vP) according to ECHA Annex XIII, 2013.

Bioaccumulation

PFOA does not seem to bio-concentrate in water-breathing animals. The high water solubility of PFOA enables fish to quickly excrete this substance via gill permeation, facilitated by the high water throughput.

In air-breathing animals, PFOA has been found in terrestrial species as well as in endangered species as polar bear and in animals that may become endangered in near future (such as narwhale and beluga whale). Once taken up in the body, PFOA tend to partition to liver and blood.

BMFs range from 1.3 – 125 for selected predator prey relationship

TMFs rang from 1.1 – 13 for selected food chains.

Trend analysis of two herring gull colonies (Røst and Hørnøya) in northern Norway found that PFOA concentrations increased significantly between 1983 and 1993 for the Røst colony but not for Hørnøya colony. There was also an increase post-1993 in both colonies. The eggs from the Røst colony had significantly higher PFOA concentrations compared with the Hørnøya colony in 1993 and 2003 (Verreault et al. 2007). The levels in polar bears have also significantly increased the last 20-30 years (Smithwick et al., 2006).

Ecotoxicology

The acute and chronic toxicity of APFO and PFOA to environmental species is considered to be low (OECD, 2006).

There are studies showing the potential for PFOA to affect endocrine function where visible effects may not be apparent until the organisms reach adulthood. In female and male rare minnows, 3–30 mg/L PFOA elicited inhibition of the thyroid hormone biosynthesis genes, induced vitellogenin expression in males, developed oocytes in the testes of male fish and caused ovary degeneration in females (Environment Canada 2012).

However, the persistency, due to low degree of degradation both abiotic and biotic, combined with bioaccumulation in air-breathing animals is of concern. The accumulation of PFOA in foetus and breast-feed pups is of concern especially for endangered species such as the polar bear (Byttingsvik et al., 2008). Polar bears, as the apex predator in the Arctic marine food web, have been shown to be the

most contaminated with PFOA relative to other Arctic terrestrial organisms (Dietz et al., 2008). Furthermore, the great species and gender differences in elimination time for PFOA combined with the developmental and reproductive toxicity and carcinogenic properties seen in controlled animal experiments indicate that emission should be limited to the greatest extent possible. PFOA, its salts and its precursors are entering or may be entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity.

Reference

ECHA: Pentadecafluorooctanoic acid (PFOA) as a substance of very high concern because of its CMR and PBT properties. 14 June 2013.

Smithwick M, Norstrom R.J., Mabury S.A., Solomon K., Evans T.J., Stirling I., Taylor M.K., Muir D.C.G. 2006. Temporal trends of perfluoroalkyl contaminants in polar bears (Ursus maritimus) from two locations in the North American arctic, 1972-2002. Environ. Sci. Technol. 40(4):1139-1143.

OECD, 2006, SIDS Initial Assessment Report PFOA.

Environment Canada (2012): Screening Assessment Report; Perfluorooctanic Acid, its Salts, and its Precursors.

Verreault J, Berger U, Gabrielsen GW. 2007. Trends of perfluorinated alkyl substances in herring gull eggs from two coastal colonies in northern Norway: 1983–2003. Environ Sci Technol 41(19): 6671–6677.

Bytingsvik J, van Leeuwen SP, Hamers T, Swart K, Aars J, Lie E, Nilsen EM, Wiig O, Derocher AE, Jenssen BM. Perfluoroalkyl substances in polar bear mother-cub pairs: a comparative study based on plasma levels from 1998 and 2008. Environ Int. 2012, 49:92-9.

Dietz et al. 2008; "Increasing Perfluoroalkyl Contaminants in East Greenland Polar Bears (Ursus maritimus): A New Toxic Threat to the Arctic Bears", Environ. Sci. Technol. 2008, 42, 2701–2707.

Impact assessment of regulating perfluorooctanoic acid (PFOA) and individual PFOA salts and esters in consumer products. The Norwegian version is available online: «Vurdering av konsekvenser av regulering av PFOA og enkelte salter og estere av PFOA i forbrukerprodukter" (Norwegian risk assement),
http ://www.miljodirektoratet.no/Global/dokumenter/horinger/horing2010-1463_PFOA.pdf

SECTION 4 DESIGNATED NATIONAL AUTHORITY

Institution	Norwegian Environment Agency

Address	P.O.Box 5672 Sluppen, 7485 Trondheim
Name of person in charge	Trine Celius
Position of person in charge	Senior advisor
Telephone	
Telefax	
E-mail address	Trine.Celius@miljodir.no

Date, signature of DNA and official seal: 28/4-15 Trine Celius

NORWEGIAN ENVIRONMENT AGENCY
P.O. box 5672 Sluppen
7485 Trondheim
Tel.: 03400 / 73 58 05 00
Fax: 73 58 05 01

PLEASE RETURN THE COMPLETED FORM TO:

Secretariat for the Rotterdam Convention
Food and Agriculture Organization
of the United Nations (FAO)
Viale delle Terme di Caracalla
00153 Rome, Italy
Tel: (+39 06) 5705 2188
Fax: (+39 06) 5705 3224
E-mail: pic@fao.org

OR

Secretariat for the Rotterdam Convention
United Nations Environment
Programme (UNEP)
11-13, Chemin des Anémones
CH – 1219 Châtelaine, Geneva, Switzerland
Tel: (+41 22) 917 8296
Fax: (+41 22) 917 8082
E-mail: pic@pic.int

Definitions for the purposes of the Rotterdam Convention according to Article 2:

(a) 'Chemical' means a substance whether by itself or in a mixture or preparation and whether manufactured or obtained from nature, but does not include any living organism. It consists of the following categories: pesticide (including severely hazardous pesticide formulations) and industrial;

(b) 'Banned chemical' means a chemical all uses of which within one or more categories have been prohibited by final regulatory action, in order to protect human health or the environment. It includes a chemical that has been refused approval for first-time use or has been withdrawn by industry either from the domestic market or from further consideration in the domestic approval process and where there is clear

evidence that such action has been taken in order to protect human health or the environment;

(c) 'Severely restricted chemical' means a chemical virtually all use of which within one or more categories has been prohibited by final regulatory action in order to protect human health or the environment, but for which certain specific uses remain allowed. It includes a chemical that has, for virtually all use, been refused for approval or been withdrawn by industry either from the domestic market or from further consideration in the domestic approval process, and where there is clear evidence that such action has been taken in order to protect human health or the environment;

(d) 'Final regulatory action' means an action taken by a Party, that does not require subsequent regulatory action by that Party, the purpose of which is to ban or severely restrict a chemical.

FORM FOR NOTIFICATION
OF FINAL REGULATORY ACTION TO BAN OR SEVERELY RESTRICT A CHEMICAL

Country: CANADA

SECTION 1 IDENTITY OF CHEMICAL SUBJECT TO THE FINAL REGULATORY ACTION

1.1	**Common name**	• Perfluorooctanoic acid, which has the molecular formula $C_7F_{15}CO_2H$, and its salts. • Compounds that consist of a perfluorinated alkyl group that has the molecular formula C_nF_{2n+1} in which n = 7 or 8 and that is directly bonded to any chemical moiety other than a fluorine, chlorine or bromine atom Collectively referred to as PFOA.
1.2	**Chemical name according to an internationally recognized nomenclature (e.g. IUPAC), where such nomenclature exists**	PFOA (list is not exhaustive): Octanoic acid, 2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-pentadecafluoro- Perfluorooctanoate (PFO,conjugate base of the free acid) Branched perfluorooctanoic acid PFOA Salts and precursors: See Table A-1 in Annex A.
1.3	**Trade names and names of preparations**	

1.4 Code numbers

1.4.1 CAS number	PFOA: PFOA: 335-67-1 PFO: 45285-51-6 PFOA salts: PFOA (NH4+): 3825-26-1 PFOA linear/branched (NH4+): 90480-56-1 PFOA (Na+): 335-95-5 PFOA (K+): 2395-00-8 PFOA (Ag+): 335-93-3 PFOA potential Precursors: See Table A-1 in Annex A
1.4.2 Harmonized System customs code	PFOA and related: 29159070
1.4.3 Other numbers (specify the numbering system)	PFOA (list is not exhaustive): RTECS# RH0781000; EINECS# 206-397-9 PFOA salts: PFOA (NH4+): EINECS# 223-320-4 PFOA (Na+): EINECS# 206-404-5

1.5 Indication regarding previous notification on this chemical, if any

1.5.1 ☒ This is a first time notification of final regulatory action on this chemical.

1.5.2 ☐ This notification replaces all previously submitted notifications on this chemical.
Date of issue of the previous notification: _____

SECTION 2 FINAL REGULATORY ACTION

2.1 The chemical is: ☐ **banned** **OR** ☒ **severely restricted**

2.2 Information specific to the final regulatory action

2.2.1 Summary of the final regulatory action

Perfluorooctanoic acid, which has the molecular formula $C_7F_{15}CO_2H$, its salts, and its precursors (collectively referred to as PFOA) and products containing them are subject to the *Prohibition of Certain Toxic Substances Regulations, 2012* (the Regulations) as amended in 2016, under the *Canadian Environmental Protection Act, 1999* (CEPA).

The *Prohibition of Certain Toxic Substances Regulations, 2012* prohibit the import, manufacture, use, sale and offer for sale of PFOA, and products containing PFOA, with a limited number of exemptions.

2.2.2 Reference to the regulatory document, e.g. where decision is recorded or published

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

2.2.3 Date of entry into force of the final regulatory action

December 23, 2016.

2.3 Category or categories where the final regulatory action has been taken

2.3.1 All use or uses of the chemical in your country prior to the final regulatory action

PFOA and long-chain perfluorocarboxylic acids (LC-PFCAs) are primarily used as water, oil and grease repellants; as surfactants; and as spreading and wetting agents.

While PFOA and LC-PFCAs are not manufactured in Canada, they were historically imported and may have been imported for use in the following manufacturing sectors: textile mills, paper and packaging, paints and coatings, inks and photo media, chemical manufacturing, electrical and electronics, cleaning products, plastic and rubber products. A study conducted for the Department estimated that approximately 308 tonnes of PFOA and LC-PFCAs were imported into Canada in 2010.

Reference:
Regulatory Impact Analysis Statement, Regulations Amending the Prohibition of Certain Toxic Substances Regulations, 2012. Environment Canada and Health Canada. October 2016.
http://www.gazette.gc.ca/rp-pr/p2/2016/2016-10-05/html/sor-dors252-eng.html

2.3.2 Final regulatory action has been taken for the category

☒ Industrial

Use or uses prohibited by the final regulatory action

The Regulations prohibit the manufacture, use, sale, offer for sale or import of PFOA and products containing PFOA, unless the substance is incidentally present. A limited number of exemptions are listed below.

Use or uses that remain allowed (only in case of a severe restriction)

The Regulations do not apply to any toxic substance that is:

a) contained in a hazardous waste, hazardous recyclable material or non-hazardous waste to which Division 8 of Part 7 of CEPA applies;
b) contained in a pest control product as defined in subsection 2(1) of the *Pest Control Products Act*;
c) present as a contaminant in a chemical feedstock that is used in a process from which there are no releases of the toxic substance and on the condition that the toxic substance is destroyed or completely converted in that process to a substance that is not a toxic substance set out in either Schedule 1 or 2 of the regulations; or
d) to be used in a laboratory for analysis, in scientific research or as a laboratory analytical standard.

The Regulations do not prohibit:

- The import, manufacture, use, sale and offer for sale of PFOA or a product containing them, if PFOA are incidentally present [subsection 6(1) of the Regulations]
- The import, manufacture, use, sale and offer for sale of PFOA or a product containing them, before January 1, 2017, if it is designed for use in water-based inks or in photo media coatings, [paragraph 6(2)(b) of the Regulations]
- The import, use, sale and offer for sale of aqueous film forming foam for fire-fighting operations that contain PFOA [subsection 6(2.2) of the Regulations]
- The import, use, sale or offer for sale of manufactured items containing PFOA [subsection 6(2.4) of the Regulations]
- The use or import of products containing PFOA, if the product is for personal use [subsection 6(4) of the Regulations].
- The use, sale or offer for sale of:
    - Products containing PFOA if manufactured or imported before the Regulations come into force [paragraph 7(2)(a) of the Regulations]
    - Water-based inks and photo media coatings containing PFOA that were manufactured or imported before January 1, 2017 [subsection 7(1) of the Regulations]
    - PFOA or products containing them if they were manufactured or imported in accordance with a permit (section 8 of the Regulations).

The Regulations allow manufacturers and importers of PFOA and products

containing PFOA to apply for a permit to continue their activities after the coming into force of the amendments or after expiry of a temporary exemption. Permits are valid for one year and can potentially be renewed twice allowing manufacturers and importers to continue their activities for an additional three years.

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

Substance Prohibition Summary for Perfluorooctanoic acid, its salts, and its precursors and Long-Chain Perfluorocarboxylic acids, their salts, and their precursors. Environment and Climate Change Canada. July 2017. http://ec.gc.ca/lcpe-cepa/default.asp?lang=En&xml=3E603995-6012-4D22-993B-0ADEA222C2C4

2.3.3 Final regulatory action has been taken for the category ☐ Pesticide

Formulation(s) and use or uses prohibited by the final regulatory action

N/A

Formulation(s) and use or uses that remain allowed

(only in case of a severe restriction)

N/A

2.4 Was the final regulatory action based on a risk or hazard evaluation? ☒ **Yes**

☐ **No** (If no, you may also complete section 2.5.3.3)

2.4.1 If yes, reference to the relevant documentation, which describes the hazard or risk evaluation

The *Canadian Environmental Protection Act, 1999* (CEPA) requires the federal Ministers of Health and the Environment to conduct screening assessments to determine, in an expeditious manner, whether a substance poses a risk to human health or the environment. On the basis of a screening assessment, the Ministers can propose to take no further action in respect of the substance, to add the substance to the Priority Substances List for a more in-depth

assessment or to recommend that the substance be added to the List of Toxic Substances in Schedule 1 of the Act.

Reference:

Government of Canada. August 2012. Screening Assessment Report on Perfluorooctanoic Acid, Its Salts and Its Precursors.

http://www.ec.gc.ca/ese-ees/default.asp?lang=En&n=370AB133-1

2.4.2 Summary description of the risk or hazard evaluation upon which the ban or severe restriction was based.

2.4.2.1 Is the reason for the final regulatory action relevant to human health?

☐ Yes

☒ No

If yes, give summary of the hazard or risk evaluation related to human health, including the health of consumers and workers

N/A

Expected effect of the final regulatory action

N/A

2.4.2.2 Is the reason for the final regulatory action relevant to the environment?

☒ Yes

☐ No

If yes, give summary of the hazard or risk evaluation related to the environment

An ecological screening assessment was undertaken on perfluorooctanoic acid (PFOA), its salts and its precursors containing the perfluorinated alkyl moiety (C_7H_{15}, C_8H_{17}) and is directly bound to any chemical moiety other than a fluorine, chlorine or bromine atom.

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

PFOA has been detected at trace levels in the northern hemisphere. In North America, higher levels were measured in surface waters in the vicinity of US fluoropolymer manufacturing facilities (<0.025–1900 µg/L) and in groundwater near US military bases (not detected [ND] to 6570 µg/L). PFOA was detected in

effluent from Canadian wastewater treatment facilities at concentrations ranging from 0.007 to 0.055 µg/L. PFOA was also detected in the influent at US wastewater treatment facilities at concentrations ranging from 0.0074–0.089 µg/L.

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

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

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

The risk quotients for pelagic organisms indicate a low likelihood of risk from exposures at current concentrations in the aquatic environment. The risk quotient for Canadian mammalian wildlife (i.e., polar bears) is less than 1; however, due to the persistence of the substance, its tendency to accumulate and biomagnify in a variety of terrestrial and marine mammals, its hepatotoxicity, and the upward temporal trend of PFOA concentrations in polar bears and some other species, PFOA concentrations in polar bears may

approach exposures resulting in harm.

The assessment is based on a weight of evidence approach regarding persistence, bioaccumulation, temporal trends in some species (i.e. the polar bear), long-range transport and the widespread occurrence and concentrations of PFOA in the environment and in biota (including remote areas of Canada). Based on the information presented in the screening assessment, it is concluded that PFOA, its salts and its precursors are entering or may be entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity.

Reference:

Government of Canada. August 2012. Screening Assessment Report on Perfluorooctanoic Acid, Its Salts and Its Precursors.

http://www.ec.gc.ca/ese-ees/default.asp?lang=En&n=370AB133-1

Expected effect of the final regulatory action

The risk management objective for PFOA is to achieve the lowest level of releases into the Canadian environment which is technically or economically feasible.

The final regulatory action protects the Canadian environment from risks associated with the manufacture, use, sale, offer for sale or import of PFOA and certain products containing PFOA.

Reference:
Regulatory Impact Analysis Statement, Regulations Amending the Prohibition of Certain Toxic Substances Regulations, 2012. Environment Canada and Health Canada. October 2016.
http://www.gazette.gc.ca/rp-pr/p2/2016/2016-10-05/html/sor-dors252-eng.html

2.5 Other relevant information regarding the final regulatory action

2.5.1 Estimated quantity of the chemical produced, imported, exported and used

	Quantity per year (MT)	Year
produced	0	2010
imported	308 Tonnes (PFOA + LC-PFCA)	2010
exported	0	2010
used		

2.5.2 Indication, to the extent possible, of the likely relevance of the final regulatory action to other states and regions

A number of countries and organizations (including United States, European

Union, Norway, Stockholm Convention on Persistent Organic Pollutants and Protocol to the 1979 Convention on Long-Range Transboundary Air Pollution) have either put in place or are proposing management measures to control the manufacture, import, use and releases of Perfluorinated Chemicals (PFCs) and manufactured products containing PFCs. Therefore, the countries aforementioned and many others would not be affected by these Regulations.

PFOA has been proposed for listing under the Stockholm Convention, as a Persistent Organic Pollutant (POP). As a result of past releases to the environment due especially to human activities, POPs are now widely distributed over large regions (including those where POPs have never been used) and, in some cases, they are found around the globe. POPs can be found in people and animals living in regions such as the Arctic, thousands of kilometres from any major POPs source.

2.5.3 Other relevant information that may cover:

2.5.3.1 Assessment of socio-economic effects of the final regulatory action

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

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

The Regulations are expected to have a low cost impact on industry. The substances are not currently manufactured in Canada and are only known to be imported. Furthermore, industry sectors have already completed the transition to alternatives, or are expected to do so prior to the coming into force of the Regulations. Development of alternatives to PFOA and LC-PFCAs in water-based inks and photo media coatings were underway, and companies were expected to eliminate their use of these substances by the end of 2016, when the temporary exemption expired. For aqueous film-forming foams containing PFOA and LC-PFCAs, which are allowed under the amendments, the development of alternatives has begun and will be monitored.

Reference:
Regulatory Impact Analysis Statement, Regulations Amending the Prohibition

of Certain Toxic Substances Regulations, 2012. Environment Canada and Health Canada. October 2016.

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

2.5.3.2 Information on alternatives and their relative risks, e.g. IPM, chemical and non-chemical alternatives

In Canada, manufacturing has been the main industrial sector using PFOA and long-chain PFCAs, specifically, paper and chemical manufacturing (note PFOA and long-chain PFCAs are not manufactured or imported; however their salts and precursors have been reported to be imported). Both PFOA and long-chain PFCAs have been used in the production of fluoropolymers and fluoroteomers and as additives and components in consumer and industrial products.

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

The US EPA is also reviewing substitutes for PFOA, PFOS, and other long-chain perfluorinated substances as part of its review process for new chemicals under EPA's New Chemicals Program. Over 150 alternatives of various types have been received and reviewed by EPA. Under the US EPA's New Chemical Review of Alternatives for PFOA and Related Chemicals, shorter chain-length perfluorinated telomeric substances have been notified as alternatives for a variety of uses including, for example, textile, carpet and paper additive uses and tile surface treatments. The major industry users in the global community are moving quickly to replace uses of C-8 and higher homologues. Based on work done to date by industry, C-6 (Fluorotelomer) and C-4 (Sulfonate) chemistries meet the criteria for replacement of most current C-8 and higher homologue uses, and are preferred as drop-in replacements. Alternatives with a shorter fluorinated alkyl chain are persistent in the environment but have rapid bioelimination.

While fluoro-alternatives exist for most, although not all current uses, there will likely not be one single replacement but rather several alternatives. Non-fluorinated alternatives are available for some applications but may not work as well.

Substances which are new to Canada, including new substitutes for PFOA and Long Chain PFCAs, are subject to the New Substances provisions of CEPA and the New Substances Notification Regulations. Any company intending to import or manufacture such a substance must submit a notification, with the substance undergoing an assessment by Environment Canada and Health Canada to determine whether it meets the definition of "toxic" set out in section 64 of CEPA. Many substitutes to PFOA and long-chain PFCAs have been notified to Environment Canada's New Substances Program.

Reference:
Government of Canada. August 2012. Proposed Risk Management Approach for Perfluorooctanoic Acid (PFOA), its Salts, and its Precursors and Long-Chain (C9-C20) Perfluorocarboxylic Acids (PFCAs), their Salts, and their Precursors. http://www.ec.gc.ca/ese-ees/default.asp?lang=En&n=451C95ED-1

2.5.3.3 Basis for the final regulatory action if other than hazard or risk evaluation

N/A

2.5.3.4 Additional information related to the chemical or the final regulatory action, if any

N/A

SECTION 3 PROPERTIES

3.1 Information on hazard classification where the chemical is subject to classification requirements

International classification systems e.g. WHO, IARC, etc.	Hazard class

Other classification systems e.g. EU, USEPA	Hazard class
Harmonised classification - Annex VI of Regulation (EC) No 1272/2008 (CLP Regulation)	PFOA (CAS 335-67-1): https://echa.europa.eu/information-on-chemicals/cl-inventory-database/-/discli/details/67229 Acute Tox. 4 -H302 (Harmful if swallowed) -H332 (Harmful if inhaled) Eye Dam. 1

	-H318 (Causes serious eye damage) Carc. 2 -H351 (Suspected of causing cancer) Lact. -H362 (May cause harm to breast-fed children) STOT RE 1 -H372 (liver) (Causes damage to organs (liver) through prolonged or repeated exposure) Repr. 1B -H360D (May damage the unborn child)

3.2 Further information on the properties of the chemical

3.2.1 Description of physico-chemical properties of the chemical

The PFOA anion has the molecular formula $C_8F_{15}O_2^-$. The structural formula is $CF_3(CF_2)_6COO^-$. While PFOA can exist in anionic, acid and salt forms, the PFOA anion is the most common form at pH values in the environment and in the human body. The free acid and the ammonium salt are solid at 20°C.

PFOA and its salts and precursors belong to a class of chemicals known as perfluorocarboxylic acids (PFCAs). PFCAs, in turn, belong to the broader class of chemicals known as perfluoroalkyls (PFAs). Perfluorinated chemicals such as PFOA contain carbons that are completely saturated by fluorine. It is the strength of the C-F bonds that contributes to the extreme stability and physical-chemical properties of these perfluorochemicals.

The precursors have the potential to degrade or metabolize to PFOA. Once PFOA is released to the environment, it is not known to undergo any further chemical, microbial or photolytic degradation and is, therefore, persistent. As well as being commercially produced, PFOA can be formed in the environment through transformation or degradation from a variety of other perfluorinated . chemicals.

Key physical/chemical properties of PFOA are listed in Table 1 below.

Table 1. Physical/chemical properties of PFOA

Property	Value[1]	Type	Reference
Abbreviations: K_{oa}, octanol–air partition coefficient; K_{ow}, octanol–water partition coefficient; K_{oc}, sediment organic carbon coefficient; pK_a, acid dissociation constant. [1] Value in parentheses provided in the original reference.			
Molecular mass (g/mol)	414.0639	–	–
Melting point (°C)	52-54		Barton et al 2007

Vapour pressure (Pa)	2.2 (at 20 °C)	Calculated	Barton et al 2007
Henry's law constant ($Pa \cdot m^3/mol$)	2.4	Calculated	Barton et al 2007
log K_{ow} (dimensionless)	5 ± 0.5	Modelled	Jasinski et al. 2009
	3.62–6.30	Modelled	Arp et al 2006
log K_{oa}(dimensionless)	5.73–6.80	Modelled	Arp et al 2006
log K_{oc} (dimensionless)	2.06 (sediment pH 5.7-7.6)	Experimental	Higgins and Luthy 2006
Water solubility (g/L)	3.5 (neutral to alkaline pH) 0.0007 (acidic pH)		Barton et al 2007
pK_a (dimensionless)	2.5	Experimental	Kissa (1994)
	3.8 +/- 0.1	Experimental	Burns et al. (2008)
	0 - 4	Modelled and analogues	Goss and Arp (2009)

Reference

Government of Canada. August 2012. Screening Assessment Report on Perfluorooctanoic Acid, Its Salts and Its Precursors.
http://www.ec.gc.ca/ese-ees/default.asp?lang=En&n=370AB133-1

3.2.2 Description of toxicological properties of the chemical

In humans, PFOA is well absorbed by all routes of exposure; it has not been demonstrated to be metabolized and has a relatively long half-life. Salts of PFOA are expected to dissociate in biological media to produce the perfluorooctanoate (PFO) moiety, and are therefore considered toxicologically equivalent to PFOA. Low concentrations of PFOA have been identified in blood samples from non-occupationally exposed Canadians, including newborns, indicating environmental exposure to PFOA and/or compounds that can degrade to PFOA. The available data indicate that Canadians are exposed to PFOA and its precursors in the environment, including via air, drinking water and food; and from the use of consumer products, such as new non-stick cookware and perfluorinated compound (PFC)-treated apparel and household materials such as carpets and upholstery. Canadians are also potentially exposed to PFOA *in utero* and through lactational transfer. The relative contributions of PFOA and its salts and precursors to total PFOA exposure were not characterized; rather the focus was on aggregate exposure to the moiety of toxicological concern, PFOA.

Epidemiological studies have not identified a causal relationship between PFOA exposure and adverse health effects in humans. Therefore, toxicity studies in laboratory animals were used to determine the critical effects and associated serum levels of PFOA. Following oral dosing of PFOA ammonium salt (APFO), increased liver weight in mice and altered lipid parameters in rats were observed in short-term (14-day) toxicity studies; increased liver weight was noted in a 26-

week toxicity study in monkeys; and increased liver weight in dams, alterations in fetal ossification and early puberty in male pups were found in a developmental toxicity study in mice.

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

Reference

Government of Canada. August 2012. Screening Assessment Report on Perfluorooctanoic Acid, Its Salts and Its Precursors.
http://www.ec.gc.ca/ese-ees/default.asp?lang=En&n=370AB133-1

3.2.3 Description of ecotoxicological properties of the chemical

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

A study examined freshwater male tilapia (*Oreochromis niloticus*) as the *in vitro* model to detect the induction of vitellogenin. Vitellogenin is an egg yolk precursor protein expressed in females of fish, amphibians, reptiles (including birds), insects and the platypus. In the presence of substances that affect endocrine function, males can also express the vitellogenin gene. Cultured male tilapia hepatocytes were exposed to PFOA, 4:2 FTOH, 6:2 FTOH and 8:2 FTOH for 48 hours. A dose-dependent induction of vitellogenin was observed in PFOA- and 6:2 FTOH–treated cells, whereas vitellogenin remained unchanged for 4:2 FTOH

and 8:2 FTOH. The estimated 48-hour median effective concentration (EC_{50}) values were 2.9×10^{-5} M (12 mg/L) for PFOA and 2.8×10^{-5} M (12.9 mg/L) for 6:2 FTOH. In the time course study, vitellogenin induction took place at 48 hours (PFOA), 72 hours (4:2 FTOH), 12 hours (6:2 FTOH) and 72 hours (8:2 FTOH) and increased further after 96 hours of exposure. Co-exposure to a mixture of individual perfluorinated compounds and 17β-estradiol for 48 hours significantly inhibited 17β-estradiol-induced hepatocellular vitellogenin production in a dose-dependent manner, except for 4:2 FTOH. The estimated 48-hour median inhibitory concentration (IC_{50}) values were 5.1×10^{-7} M(0.21 mg/L) for PFOA, 1.1×10^{-6} M (0.51 mg/L) for 6:2 FTOH and 7.5×10^{-7} M (0.35 mg/L) for 8:2 FTOH. In order to further investigate the estrogenic mechanism, the hepatocytes were co-exposed to a mixture of PFOA and 6:2 FTOH plus the known estrogen receptor inhibitor tamoxifen for 48 hours. The overall results demonstrated that PFOA and FTOHs have estrogenic activities and that exposure to a combination of 17β-estradiol and PFOA or FTOHs produces anti-estrogenic effects. The results of the estrogen receptor inhibition assay further suggested that the estrogenic effect of PFOA and FTOHs may be mediated by the estrogen receptor pathway in primary cultured tilapia hepatocytes.

A study assessed the effects of PFOA on male and female rare minnows (*Gobiocypris rarus*) at concentrations of 3, 10 and 30 mg/L for 28 days. Exposure to PFOA at 3 mg/L elicited moderate hepatocellular hypertrophy in the livers of both male and female fish. Male rare minnows exposed to PFOA at 10 mg/L showed eosinophilic hyaline droplets in the cytoplasm of the hepatocytes; female rare minnows displayed more eosinophilic hyaline droplets in the cytoplasm of the hepatocytes, hepatocellular hypertrophy and vacuolar degeneration. Rare minnows exposed to PFOA at 30 mg/L showed severe hepatic histopathological changes and disruption of mitochondrial functions. The inhibition of the thyroid hormone biosynthesis genes and the induction of estrogen-responsive genes may indicate a role in endocrine function. Another study further identified the potential protein biomarkers for PFOA exposure in the livers of the rare minnows at 3, 10 and 30 mg/L for 28 days, finding the abundance of 34 and 48 protein spots altered in males and females, respectively. These proteins were involved in intracellular fatty acid transport, oxidative stress, macromolecule catabolism, the cell cycle, maintenance of intracellular Ca^{2+} homeostasis and mitochondrial function. In another article, the authors studied the *in vivo* effects of waterborne PFOA on the expression of hepatic estrogen-responsive genes, vitellogenin, and estrogen receptor and on the gonadal development in freshwater rare minnow (*Gobiocypris rarus*). The study showed mature females exposed to 3, 10, and 30 mg/L PFOA for 28 d had degenerating vitellogenic-stage oocytes (atresia) in the ovaries. In males exposed to 10 mg/L PFOA, primary growth–stage oocytes (pre-vitellogenic oocytes) developed in some testes. The number of sperm and various stages of germ cells within the spermatogenic cycle in the 10 and 30

mg/L PFOA treatments were lower than those in control males. PFOA increased hepatic vitellogenin concentration and induced testis-ova gonads in mature male rare minnows at 10 and 30 mg/L for 28 days. It was showed that PFOA can disrupt the activity of estrogen by inducing hepatic estrogen-responsive genes in males, although the mechanism of development of testes-ova in rare minnows by PFOA exposure is not known.

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

Another study showed that PFOA activates the mammalian peroxisome proliferator–activated receptor α (PPARα) in the livers of Baikal seals--the first reported identification of PPARα complementary deoxyribonucleic acid (DNA) in an aquatic wildlife species. PPAR is a member of the ligand-activated nuclear hormone receptor superfamily. PPARα plays a critical physiological role as a lipid sensor and a regulator of lipid metabolism. The lowest-observed-effect concentration (LOEC) for PFOA was 62.5 μM (25.9 mg/L). Yang (2010) exposed male Japanese medaka (*Oryzias latipes*) 10, 50, and 100 mg/L PFOA for 7 days. There were no impacts on survival, relative live and gonad size, and condition factor at any concentration. However, PFOA induced inhibition of catalase activity at high doses with no changes of superoxide dismutase or glutathione peroxidase activities in the liver suggesting that PFOA may induce peroxisomal fatty acid oxidation and impose oxidative stress through the alteration of cellular oxidative homeostasis in the liver. PFOA also increased the mRNA levels of pro-inflammatory cytokines suggesting that it may be involved in inflammation and tissue injury. The effects on peroxisome proliferation and cytokine expression suggest that chronic exposure to PFOA can be carcinogenic in the Japanese medaka – peroxisome proliferation is a key carcinogenic mechanism and chronic inflammation is associated with an increased risk of cancer in rodents.

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

The effect of PFOA on immune function and clinical blood parameters has been examined in bottlenose dolphins and sea turtles from Florida, Georgia and South Carolina. It should be noted that a direct cause–effect relationship cannot be clearly established, as there may be other co-occurring contaminants. The results revealed that there may be increases in indicators of inflammation and immunity in bottlenose dolphin blood parameters in relation to PFOA, suggesting that PFOA may alter biomarkers of health in marine mammals. Examples of biomarkers analyzed in bottlenose dolphins include absolute numbers of lymphocytes, serum triglyceride, serum total protein, serum albumin, serum cortisol, C-reactive protein, lysozyme activity and B-cell proliferation. Serum triglyceride exhibited stronger relationships to PFOA in females than in males. Lipopolysaccharide-induced lymphocyte proliferation (B-cell proliferation) had positive but weak correlations with PFOA in male bottlenose dolphins, and a strong correlation was observed between PFOA and lysozyme activity (a measurement of innate immunity) in the same species. However, in another study, no correlations were found between any perfluorinated compound, including PFOA, and blood chemistry parameters (e.g. cholesterol, creatinine,

albumin, total serum ion etc.) for the northern fur seal (*Callorhinus ursinus*).

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

Reference

Government of Canada. August 2012. Screening Assessment Report on Perfluorooctanoic Acid, Its Salts and Its Precursors.
http://www.ec.gc.ca/ese-ees/default.asp?lang=En&n=370AB133-1

SECTION 4 DESIGNATED NATIONAL AUTHORITY

Institution	Environment and Climate Change Canada Environmental Protection Branch Industrial Sectors, Chemicals, and Waste Directorate Chemical Production Division
Address	Place Vincent Massey 351 St. Joseph Blvd., 19th Floor Gatineau, Quebec, K1A 0H3 CANADA
Name of person in charge	Ms. Nathalie Morin
Position of person in charge	Director, Chemical Production Division
Telephone	819-420-8047
Telefax	819-938-4218
E-mail address	nathalie.morin4@canada.ca; ec.substancedexportationcontrolee-exportcontrolledsubstance.ec@canada.ca

Date, signature of DNA and official seal: _____

Feb 23, 2018

PLEASE RETURN THE COMPLETED FORM TO:

Secretariat for the Rotterdam Convention
Food and Agriculture Organization
of the United Nations (FAO)
Viale delle Terme di Caracalla
00153 Rome, Italy
Tel: (+39 06) 5705 2188
Fax: (+39 06) 5705 3224
E-mail: pic@fao.org

OR

Secretariat for the Rotterdam Convention
United Nations Environment
Programme (UNEP)
11-13, Chemin des Anémones
CH – 1219 Châtelaine, Geneva, Switzerland
Tel: (+41 22) 917 8296
Fax: (+41 22) 917 8082
E-mail: pic@pic.int

Definitions for the purposes of the Rotterdam Convention according to Article 2:

(a) 'Chemical' means a substance whether by itself or in a mixture or preparation and whether manufactured or obtained from nature, but does not include any living organism. It consists of the following categories: pesticide (including severely hazardous pesticide formulations) and industrial;

(b) 'Banned chemical' means a chemical all uses of which within one or more categories have been prohibited by final regulatory action, in order to protect human health or the environment. It includes a chemical that has been refused approval for first-time use or has been withdrawn by industry either from the domestic market or from further consideration in the domestic approval process and where there is clear evidence that such action has been taken in order to protect human health or the environment;

(c) 'Severely restricted chemical' means a chemical virtually all use of which within one or more categories has been prohibited by final regulatory action in order to protect human health or the environment, but for which certain specific uses remain allowed. It includes a chemical that has, for virtually all use, been refused for approval or been withdrawn by industry either from the domestic market or from further consideration in the domestic approval process, and where there is clear evidence that such action has been taken in order to protect human health or the environment;

(d) 'Final regulatory action' means an action taken by a Party, that does not require subsequent regulatory action by that Party, the purpose of which is to ban or severely restrict a chemical.

Annex A

Table A-1. List of PFOA and its Principal Salts and Precursors (This List is not Considered Exhaustive) [1]

Name	CAS RN	Molecular formula
PFOA free acid (Octanoic acid, pentadecafluoro-)	335-67-1	$C_8HF_{15}O_2$
Perfluorooctanoate (PFO, conjugate base of the free acid)	45285-51-6	$C_8F_{15}O_2^-$
Branched perfluorooctanoic acid	90480-55-0	$C_8HF_{15}O_2$
Principal salts		
PFOA ammonium salt (APFO, Octanoic acid, pentadecafluoro-, ammonium salt)	3825-26-1	$C_8F_{15}O_2^-NH_4^+$
Ammonium salt, linear/branched PFOA (Octanoic acid, pentadecafluoro-, branched, ammonium salt)	90480-56-1	$C_8F_{15}O_2^-NH_4^+$
PFOA sodium salt	335-95-5	$C_8F_{15}O_2^-Na^+$
PFOA potassium salt	2395-00-8	$C_8F_{15}O_2^-K^+$
PFOA silver salt	335-93-3	$C_8F_{15}O_2^-Ag^+$
Potential precursors[1]		
2-Propenoic acid, 2-methyl-, 2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-pentadecafluorooctyl ester, polymer with 2-propenoic acid	53515-73-4	$(C_{15}H_{11}F_{15}O_4)_x$
Propanamide, 3-[(γ-ω-perfluoro-C_{4-10}-alkyl)thio] derivatives	68187-42-8	NA
Poly(difluoromethylene), α-fluoro-ω-[2- [[2-(trimethylammonio)ethyl]thio]ethyl]-, methyl sulfate	65530-57-6	NA
Poly(difluoromethylene), α,α'-[phosphinicobis(oxy-2,1-ethanediyl)]bis[ω-fluoro-	65530-62-3	NA
Poly(difluoromethylene), α-fluoro-ω-[2-(phosphonooxy)ethyl]-	65530-61-2	NA
Thiols, C_{8-20}, γ-ω-perfluoro, telomers with acrylamide	70969-47-0	NA
Carbamic acid, [2-(sulfothio)ethyl]-, C-(3,3,4,4,5,5,6,6,7,7,8,8,8-tridecafluorooctyl) ester, monosodium salt	82199-07-3	NA
Carbamic acid, [2-(sulphothio)ethyl]-, C-(γ-ω-perfluoro-C_{6-9}-alkyl) esters, monosodium salts	95370-51-7	NA
1,3-Propanediol, 2,2-bis[[(γ-ω-perfluoro-C_{4-10}-alkyl)thio]methyl] derivatives, phosphates, ammonium salts	148240-85-1	NA
1,3-Propanediol, 2,2-bis[[(γ-ω-perfluoro-C_{6-12}-alkyl)thio]methyl] derivatives, phosphates, ammonium salts	148240-87-3	NA
Thiols, C_{4-20}, γ-ω-perfluoro, co-telomers with acrylic acid and acrylamide	NA	NA

Table A-1. List of PFOA and its Principal Salts and Precursors (This List is not Considered Exhaustive) [1]

Name	CAS RN	Molecular formula
1-Decanol, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heptadecafluoro (or 1,1,2,2-tetrahydroperfluoro-1-decanol or 8:2 fluorotelomer alcohol)	678-39-7	$C_{10}F_{17}H_5O$
Octanoyl fluoride, pentadecafluoro-	335-66-0	$C_8F_{16}O$
Octanoic acid, pentadecafluoro-, methyl ester	376-27-2	$C_9H_3F_{15}O_2$
Octanoic acid, pentadecafluoro-, ethyl ester	3108-24-5	$C_{10}H_5F_{15}O_2$
8:2 Fluorotelomer acrylate polymers[2]	NA	NA
1,1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8-heptadecafluoro-10-iododecane (C8-2 iodide)	2043-53-0	NA
2-propenoic acid, 2-methyl-, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heptadecafluorodecyl methacrylate (C8-2 methacrylate)	1996-88-9	NA
2-propenoic acid, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heptadecafluorodecyl acrylate (C8-2 acrylate)	27905-45-9	NA
3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heptadecafluorodec-1-ene (C8-2 olefin)	21652-58-4	NA
Phosphoric acid surfactants (e.g., 8:2 polyfluoroalkyl phosphoric acid diester or 8:2 diPAP)[4]	NA	*x*:2 diPAP $(F(CF_2)_xCH_2CH_2O)_2P(O)OH$
Perfluorooctylsulfonamides[3]	NA	$F[CF_2]_8SO_2NRR'$ where R and R' can be CH_2CH_2OH, CH_3, CH_2CH_3, or H
1,3-Propanediol, 2,2-bis[[(γ-ω-perfluoro-C10-20-alkyl)thio]methyl] derivs., phosphates, ammonium salts	148240-89-5	NA
Oxirane, methyl-, polymer with oxirane, mono[2-hydroxy-3-[(γ-ω-perfluoro-C8-20-alkyl)thio]propyl] ethers	183146-60-3	NA
Poly(difluoromethylene), α-fluoro-ω-(2-sulfoethyl)-	80010-37-3	NA
2-Propenoic acid, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,11,11,12,12,12-heneicosafluorododecyl ester, polymer with 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heptadecafluorodecyl 2-propenoate, alpha-(2-methyl-1-oxo-2-propenyl)-omega-[(2-methyl-1-oxo-2-propenyl)oxy]poly(oxy-1, 2-ethanediyl), 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,11,11,12,12,13,13,14,14,15,15,16,16,16-nonacosafluorohexadecyl 2-propenoate, octadecyl 2-propenoate,	116984-14-6	NA

Table A-1. List of PFOA and its Principal Salts and Precursors (This List is not Considered Exhaustive) [1]

Name	CAS RN	Molecular formula
3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,11,11,12,12,13,13,14,14,14-pentacosafluorotetradecyl 2-propenoate and 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,11,11,12,12,13,13,14,14,15,15,16,16,17,17,18,18,18-tritriacontafluorooctadecyl 2-propenoate		
Pentanoic acid, 4,4-bis[(γ-ω-perfluoro-C8-20-alkyl)thio]derivs., compds. with diethanolamine	71608-61-2	NA

CAS RN, Chemical Abstracts Service Registry Number; DSL, Domestic Substances List; NA, not available; NDSL, Non-Domestic Substances List.

[1] Precursors are as identified through CATABOL (c2004–2008), expert judgment, and literature and are non-exhaustive.

[2] Van Zelm et al. (2008)

[3] De Silva et el. (2009)

[4] D'Eon and Mabury (2007)