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

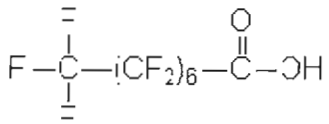
3.1 Information on hazard classification where the chemical is subject to classification requirements

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.

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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

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PLEASE RETURN THE COMPLETED FORM TO:

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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.