Perfluorooctane sulfonate (PFOS) and its Direct Precursors: Human health tier II assessment

13 February 2015

Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
1-Octanesulfonyl fluoride, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-	307-35-7
1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, potassium salt	2795-39-3
1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, ammonium salt	29081-56-9
1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, lithium salt	29457-72-5
Ethanaminium, N,N,N-triethyl-, salt with 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-1-octanesulfonic acid (1:1)	56773-42-3
1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, compound with 2,2'-iminobis[ethanol] (1:1)	70225-14-8

Preface

This assessment was carried out by staff of the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) using the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework.

The IMAP framework addresses the human health and environmental impacts of previously unassessed industrial chemicals listed on the Australian Inventory of Chemical Substances (the Inventory).

The framework was developed with significant input from stakeholders and provides a more rapid, flexible and transparent approach for the assessment of chemicals listed on the Inventory.

Stage One of the implementation of this framework, which lasted four years from 1 July 2012, examined 3000 chemicals meeting characteristics identified by stakeholders as needing priority assessment. This included chemicals for which NICNAS already held exposure information, chemicals identified as a concern or for which regulatory action had been taken overseas, and chemicals detected in international studies analysing chemicals present in babies' umbilical cord blood.

Stage Two of IMAP began in July 2016. We are continuing to assess chemicals on the Inventory, including chemicals identified as a concern for which action has been taken overseas and chemicals that can be rapidly identified and assessed by using Stage One information. We are also continuing to publish information for chemicals on the Inventory that pose a low risk to human health or the environment or both. This work provides efficiencies and enables us to identify higher risk chemicals requiring assessment.

The IMAP framework is a science and risk-based model designed to align the assessment effort with the human health and environmental impacts of chemicals. It has three tiers of assessment, with the assessment effort increasing with each tier. The Tier I assessment is a high throughput approach using tabulated electronic data. The Tier II assessment is an evaluation of risk on a substance-by-substance or chemical category-by-category basis. Tier III assessments are conducted to address specific concerns that could not be resolved during the Tier II assessment.

These assessments are carried out by staff employed by the Australian Government Department of Health and the Australian Government Department of the Environment and Energy. The human health and environment risk assessments are conducted and published separately, using information available at the time, and may be undertaken at different tiers.

This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

For more detail on this program please visit:**www.nicnas.gov.au**

Disclaimer

ACRONYMS & ABBREVIATIONS

Grouping Rationale

Perfluorooctanesulfonate (PFOS) is a fully fluorinated eight-carbon anion, which is commonly used as a salt or incorporated into larger polymers. The majority of PFOS-related substances are polymers of high molecular weights in which PFOS is only a fraction of the polymer (OECD, 2002). Microbial degradation or metabolic breakdown of larger molecules containing the perfluorooctylsulfonyl ($C_8F_{17}SO_2$, $C_8F_{17}SO_3$, or $C_8F_{17}SO_2N$) moiety can give rise to PFOS molecules. These larger molecules are termed indirect precursors of PFOS. PFOS, its salts and its precursors form part of a larger chemical class of fluorochemicals referred to as perfluoroalkylsulfonate compounds (PFSA).

This assessment addresses PFOS and its direct precursors (PFOS salts and perfluorooctanesulfonyl fluoride (PFOSF)) as one group, given their similar uses and the fact that the PFOS anion is the degradation product of all PFOS salts and precursors in the short to medium term.

The critical health concern for the chemicals in this group is the danger of serious damage to health by prolonged exposure, harm to the unborn child, and the potential for carcinogenic effects following exposure to the final breakdown product, the PFOS anion.

The assessment of these chemicals as a group also provides additional relevant information for the risk assessment of indirect precursors of PFOS which may degrade to this perfluorinated anion in the environment. These precursors are assessed separately as the Indirect Precursors of Perfluorooctanesulfonate group.

Import, Manufacture and Use

Australian

PFOS derivatives are, or have been, used in a wide variety of applications such as textiles and leather products, metal plating, food packaging, fire fighting foams, floor polishes, denture cleansers, shampoos, coatings and coating additives, in the photographic and photolithographic industry, medical devices and in hydraulic fluids in the aviation industry.

Most PFOS direct precursors used in Australia are for mist suppression in metal plating, particularly hexavalent chromium plating. Long-term use of PFOS for this purpose is allowed under the Stockholm Convention on Persistent Organic Pollutants (the Stockholm Convention) (see **Restrictions**), but only for closed-loop systems. Open system metal plating is only permitted for a limited time—a five-year specific exemption, with the possibility of another five-year extension (Australian Government Department of Environment, 2014).

It is noted that some of the chemicals in this group may be present in the environment due to historic use, or due to release from articles or the use of chemicals not covered by this assessment.

International

As a result of listing PFOS and its salts in the annexes to the Stockholm Convention, the use of PFOS and its direct precursors is restricted to the following applications and conditions (Stockholm Convention, 2014):

Acceptable purposes (not time limited)

- photo-imaging;
- photo-resist and anti-reflective coatings for semi-conductors;
- etching agent for compound semi-conductors and ceramic filters;
- aviation hydraulic fluids;
- metal plating (hard metal plating) only in closed-loop systems;
- certain medical devices (such as ethylene tetrafluoroethylene copolymer (ETFE) layers and radio-opaque ETFE production, in-vitro diagnostic medical devices, and CCD colour filters);
- fire-fighting foam; and
- insect baits to control leaf-cutting ants from *Atta* spp. and *Acromyrmex* spp.

Specific exemptions (five years initially, renewal possible)

- photo masks in the semiconductor and liquid crystal display (LCD) industries;

- metal plating (hard metal);
- metal plating (decorative);
- electric and electronic parts for some colour printers and colour copy machines;
- insecticides to control termites and red imported fire ants;
- chemically-driven oil production;
- carpet treatments;
- leather and apparel tratments;
- textiles and upholstery treatments;
- paper and packaging;
- coatings and coating additives; and
- rubber and plastics.

Restrictions

Australian

PFOS, its salts and perfulorooctane sulfonyl fluoride have been identified as Persistent Organic Pollutants (POPs) under Annex B of the *Stockholm Convention on Persistent Organic Pollutants* (Stockholm Convention, 2014). These substances are also listed on Annex III of the *Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade* (Rotterdam Convention, 2014).

Amendments made to regulation 11C(1) under the *Industrial Chemicals (Notification and Assessment) Regulations 1990* (Cwlth) in 2014 have resulted in controls being placed on the chemicals in this group (Australian Government Department of Health, 2014). The introduction or export of any chemical in this group is now prohibited unless approval is obtained from the NICNAS Director(Australian Government Department of Health, 2014).

The Australian Government Department of the Environment is currently undertaking a domestic treaty making process to consider ratification of the 2009 amendment to the Stockholm Convention, which includes the listing of perfluorooctanesulfonic acid, its salts and perfluorooctanesulfonyl fluoride. A National Interest Analysis (NIA) and a Regulation Impact Statement (RIS) have been prepared by Australia. The technical implications of the restrictions are now being explored and resolved in greater detail. Stakeholder consultations are in progress to refine the issues and quantify impacts of these restrictions (Australian Government Department of the Environment, 2014).

A 2008 survey conducted by the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) indicated that PFOS and related substances continue to be imported for use in (NICNAS, 2013):

- aviation hydraulic fluid;
- mist suppressant in metal plating;
- photolithography in semi-conductor manufacture; and
- photography.

These are uses that have been recognised by the POP Review Committee (POPRC) where technically feasible and less hazardous alternatives are presently not available. Other applications that were also listed by the committee are not practised in Australia.

NICNAS has noted that there have not been imports of PFOS-based fire fighting foams in recent years, and many new alternatives such as non-PFOS fluorosurfactants, silicone- and hydrocarbon-based surfactants, and fluorine-free foams are replacing the old foams for Class B fires.

International

Following the inclusion of PFOS and its direct precursors in the POPs list, all countries that are party to the Stockholm Convention are developing and implementing regulations that restrict the use of PFOS and its direct percursors to only those applications that are consistent with the Stockholm Convention's decision.

Based on the information obtained from Galleria Chemica, PFOS and its salts are listed on the pollutant inventories of the UK (UK Pollution Inventory), Japan (Pollutant Release and Transfer Register), the USA (Washington Persistent Bioaccumulative Toxins), Europe Pollutant Emission Register (EPER), and the Oslo and Paris Conventions (OSPAR) List of Chemicals for Priority Action. Perfluorooctanesulfonate and its salts are listed on the Virtual Elimination List under section 65 of the *Canadian Environmental Protection Act 1999* (Government of Canada, 2009). These chemicals must not be released to the environment at quantifiable levels.

Existing Worker Health and Safety Controls

Hazard Classification

The chemicals identified as CAS No. 2795-39-3; CAS No. 29081-56-9; CAS No. 29457-72-5 and CAS No. 70225-14-8 are classified as hazardous, with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia):

- Carc. Cat. 3; R40 (carcinogenicity);
- Repr. Cat. 2; R61 (reproductive toxicity);
- T; R48/25 (repeated dose toxicity);
- Xn; R20/22 (acute toxicity); and
- Xn; R64 (developmental effects).

The remaining chemicals in this group (CAS No. 307-35-7 and CAS No. 56773-42-3) are not listed on the HSIS (Safe Work Australia).

Exposure Standards

Australian

No specific exposure standards are available.

International

No specific exposure standards are available.

Health Hazard Information

Toxicokinetics

The majority of the toxicology studies of PFOS have been conducted with its potassium salt, a white crystalline powder at room temperature.

PFOS is well absorbed by the digestive tract following oral administration. Following a single oral dose of 4.2 mg/kg ^{14}C-PFOS in solution to male rats, at least 95 % of the total ^{14}C-carbon was systemically absorbed 24 hours after application (Johnson, Gibson & Ober, 1979a). The digestive tract and its contents contained, on average, 3.5 % of the dose. After 24 hours, the mean sum of total ^{14}carbon in the faeces, and digestive tract plus contents was 5 % of the dose, indicating that at least 95 % of the ^{14}C-PFOS dose was absorbed from the solution after administration to non-fasted rats.

Data on dermal and inhalation absorption are not available.

PFOS is mainly accumulated in the liver. In a rat study, 89 days after intravenous injection of 4.2 mg/kg ^{14}C-PFOS, the highest levels of radioactivity were found in the liver (25 %), followed by plasma, kidneys and lungs. Other tissues such as muscle, skin, bone marrow and spleen had only detectable levels of the substance (Johnson, Gibson & Ober, 1979b). No detectable ^{14}C was found in the brain. The presence of PFOS in human breast milk has been demonstrated in many studies (So et al., 2006; Karrman et al., 2007).

Urinary excretion is the primary route of elimination for PFOS in the rat. In the rat study reported above, 89 days after a single intravenous injection of ^{14}C-PFOS, over 30 % was eliminated via the urine (Johnson, Gibson & Ober, 1979b). The mean cumulative faecal excretion was only 12 %.

The mean elimination half-life of PFOS in humans was estimated to be 8.7 years (2.3–21 years) based on the serum PFOS levels deteremined in retirees who had been exposed to PFOS during their working life (Burris et al., 2002).

Acute Toxicity

Oral

PFOS and its salts have moderate acute toxicity when administered orally. The oral median lethal dose (LD50) for potassium PFOS was 233 mg/kg bw for male rats and 271 mg/kg bw for female rats (Dean et al., 1978). Signs of toxicity in the treated rats included hypoactivity, decreased limb tone and ataxia. At necropsy, yellow-stained urogenital region, stomach distension and signs of irritation of the glandular mucosa were observed.

Dermal

No data are available for any of the chemicals in the group.

Inhalation

PFOS has low acute inhalation toxicity. In a study to determine the median lethal concentration (LC50), Sprague Dawley (SD) rats were exposed to 1.9, 2.9, 4.9, 6.5, 7.0, 14, 24 or 46 mg/L PFOS dust in air for one hour. The rats were observed for abnormal signs at 15-minute intervals during the one-hour exposure, hourly for four hours after exposure, and daily thereafter for 14 days. All animals at and above 24.09 mg/l died by day six. Rats in all treated groups showed signs of toxicity including emaciation, red material around the nose or other nasal discharge, dry rales or other breathing disturbances, and general poor condition. The LC50 of 5.2 mg/L was calculated using Litchfield and Wilcoxon's method (Rusch et al., (1979).

Corrosion / Irritation

Respiratory Irritation

No data are available.

Skin Irritation

PFOS potassium salt is not irritating to skin. In a skin irritation study, not conducted according to the Organisation for Economic Co-operation and Development (OECD) guidelines, 0.5 g potassium PFOS (FC-95) was applied to the abraded skin of six albino rabbits and covered with gauze patches (Biesemeier & Harris, 1974). After 24 and 72 hours, the coverings were removed and the degree of erythema and oedema was recorded according to a standardised scale (details of scoring of results were not provided). The primary skin irritation scores were reported to be zero in all animals.

PFOS free acid is reportedly corrosive to the skin (Health Canada, 2006; HAZ-MAP, 2014). Corrosivity to skin is likely to be due to low pH of the acid.

Eye Irritation

PFOS potassium salt was a slight eye irritant in rabbits. In an eye irritation study, not conducted according to OECD guidelines, 0.1 g of potassium PFOS was instilled in one eye of New Zealand White rabbits. The other eye was left untreated and served as a control. The reaction to the test material was read against a scale of damage to the cornea, iris, and the bulbar and palpebral conjunctivae at 1, 24, 48, and 72 hours after treatment (scaling criteria were not provided or referenced). The eye irritation scores were maximal at 1 and 24 hours after treatment, then decreased over the rest of the study (Biesemeier & Harris, 1974).

PFOS free acid is reportedly corrosive to the eyes (HAZ-MAP, 2014). Corrosivity to the eyes is likely to be due to low pH of the acid.

Sensitisation

Skin Sensitisation

No data are available.

Repeated Dose Toxicity

Oral

PFOS has adverse health effects following repeated oral exposure. In general, oral exposure to PFOS and its salts results in hepatotoxicity and mortality; the dose-response curve for mortality was found to be very steep for rats and primates.

In a 90-day oral study, doses of 0, 30, 100, 300, 1000 or 3000 ppm potassium PFOS (equivalent to 0, 2, 6, 18, 60 and 200 mg/kg/day) were administered to CD rats (5/sex/group) in the diet for 90 days (Goldenthal et al., 1978a). All rats at dose levels of 300 ppm and above died. Rats in all groups showed signs of toxicity including emaciation, convulsions following handling, hunched back, red material around the eyes, increased sensitivity to external stimuli, reduced activity and moist red material around the mouth or nose. Surviving rats from 100 ppm group had significantly reduced erythrocyte, haemoglobin, haematocrit and leukocyte counts. Relative liver and kidney weights were significantly higher in these rats compared with control rats. At necropsy, treatment-related gross lesions were present in all treated groups and included varying degrees of discolouration

and/or enlargement of the liver and discolouration of the glandular mucosa of the stomach. Centrilobular to mid zonal cytoplasmic hypertrophy of hepatocytes and focal necrosis were observed in the liver; the incidence and relative severity were greater in the males than in females. In addition, especially among rats in the 300, 1000 and 3000 ppm groups, treatment-related histologic lesions were noted in the primary (thymus, bone marrow) and secondary (spleen, mesenteric lymph nodes) lymphoid organs, the stomach, intestines, muscle and skin.

In a 90-day repeated dose study in monkeys, 0, 0.5, 1.5 or 4.5 mg/kg/day PFOS (FC-95) in distilled water was administered to rhesus monkeys (2/sex/group) by gavage for 90 days (Goldenthal et al., 1978b). All monkeys in the 4.5 mg/kg/day group died or were sacrificed *in extremis* during weeks 5–7 of the study. Adverse effects in these monkeys were gastrointestinal tract toxicity including anorexia, emesis, black stools and dehydration. All monkeys in this group had decreased activity and, just before death, showed marked to severe rigidity, convulsions, generalised body trembling and prostration.

All monkeys in the 1.5 mg/kg/day group survived until the end of the study. Signs of gastrointestinal tract toxicity were noted occasionally during the study and included black stools, diarrhoea, mucus in the stool and bloody stools, anorexia and dehydration. In monkeys in the 0.5 mg/kg/day group, signs of gastrointestinal tract toxicity were noted occasionally during the study and included diarrhoea, soft stools, anorexia and emesis. Decreased activity was also noted in some of the monkeys in this group. A lowest observed adverse effect level (LOAEL) of 0.5 mg/kg bw/day was established in this study.

In a chronic carcinogenicity study, groups of 40–70 male and female Crl:CD (SD)IGS BR rats were given PFOS potassium salt in their diets at concentrations of 0.5, 2, 5, or 20 ppm (equivalent dose in mg/kg bw/day not provided) for 104 weeks (3M, 2002). Five animals/sex in the treatment groups were euthanised during weeks 4, 14 and 53 for histopathological analysis.

Rats given 20 ppm potassium PFOS had significantly lower mean body weights compared with control animals. At weeks 14 and 53, absolute and relative liver weights were significantly increased in this group of rats. Treatment-related histomorphologic changes were seen in livers of the rats given 5 or 20 ppm PFOS potassium salt. Animals given 5 or 20 ppm exhibited hepatotoxicity, characterised by significant increases ($P<0.05$) in centrilobular hypertrophy, centrilobular eosinophilic hepatocytic granules, centrilobular hepatocytic pigment, or centrilobular hepatocytic vacuolation. A significant increase ($P<0.05$) in hepatocellular centrilobular hypertrophy was also observed in mid-dose (2 ppm) male rats. Based on the pathological findings in the liver, the no observed adverse effect level (NOAEL) for PFOS is considered to be 0.5 ppm in male rats and 2 ppm in female rats; the LOAEL is 2 ppm in male rats and 5 ppm in female rats.

Dermal

No data are available.

Inhalation

No data are available.

Observation in humans

Several occupational studies on the health effects associated with PFOS exposure have been conducted at the 3M Decatur, Alabama plant and the Antwerp, Belgium plant. Mean serum PFOS concentrations for 263 Decatur employees were 1.32 mg/kg (Olsen et al., 2003). Many endpoints have been examined in medical surveillance programs including haematology, clinical chemistry, urine analysis, thyroid hormones and reproductive hormones. Also parameters related to health outcomes such as retrospective mortality studies, cancer incidence and the need for medical care episodes have been considered.

Data from workers at both the Decatur and Antwerp plants showed that mean serum values for triglycerides, alkaline phosphatase, total bilirubin, and alanine aminotransferase (ALT) were significantly higher in male workers (n = 421), with PFOS levels in the highest quartile (upper quartile Q4 with a mean PFOS level 2.69 versus mean PFOS level of 0.27 mg/kg in Q1). Thyroid results for male production employees indicated that serum triiodothyronine (T3) was significantly higher and thyroid hormone binding ratio (THBR) (T3 uptake) was significantly lower ($p <0.05$) in Q4 than in Q1. After multiple regressions with adjustment for potential confounders, PFOS exposure remained positively associated with serum T3 levels, and with triglycerides, but not with cholesterol. However, the dataset is limited and many confounders, including exposure to different compounds, make it difficult to interpret the data (EFSA, 2008).

Apelberg et al., (2007) investigated the association between PFOS concentrations in cord serum and gestational age, and birth weight and size, of 293 singleton births delivered in November 2004 to March 2005 in Baltimore, USA. PFOS was detected in >99% of the cord blood samples, with a median concentration of 5 ng/mL (range <0.2–34.8 ng/mL). PFOS was significantly associated with small decreases in birth weight and size, but not newborn length or gestational age. The concentrations of PFOS in cord serum were highly correlated with those of PFOA and it was suggested that the association might be related to PFOA rather than PFOS (Fei et al., 2007).

Genotoxicity

PFOS and its diethanolammonium salt tested negative in all reported in vitro and in vivo genotoxicity and mutagenicity tests. They did not induce reverse mutation at the histidine locus of *Salmonella typhimurium* or at the tryptophan locus of *Escherichia coli* when tested with or without metabolic activation. They did not induce chromosomal aberrations in human lymphocytes with or without metabolic activation and did not induce unscheduled DNA synthesis (UDS) in primary cultures of rat hepatocytes. PFOS did not induce micronuclei in the bone marrow of Crl:CD-1 BR mice and was negative in the mouse bone marrow micronucleus assay (Simmon, 1978; Murli, 1996).

Carcinogenicity

Carcinogenicity potential for PFOS potassium salt was assessed in the chronic toxicity study described above (**Repeat dose toxicity: Oral**) (3M, 2002). The results of the study indicated evidence for benign tumours of the liver, thyroid and mammary glands in rats

In this study, Crl:CD (SD)IGS BR rats were given PFOS potassium salt in the diet at concentrations of 0.5, 2, 5, or 20 ppm for 104 weeks. A significant positive trend was noted in the incidences of hepatocellular adenoma in both male and female rats. Significantly increased incidences of thyroid follicular cell adenoma in male rats ($P<0.05$) and combined thyroid follicular cell adenoma and carcinoma in female rats in the high-dose recovery group were also observed. The relevance of rodent hepatocellular adenomas to humans has been questioned, given that this has been recognised as rodent-specific effect.

In the females, except for the high-dose group (which showed a slight decrease in incidences of mammary fibro-adenoma/adenoma and combined mammary fibro adenoma and carcinoma), slight increases in benign mammary tumours were observed in all treatment groups when compared with the controls. Significant increases ($P<0.05$) in mammary fibro-adenoma/adenoma (30/50) and combined mammary fibro-adenoma, adenoma and carcinoma (36/50) were observed in the low-dose (0.5 ppm) group when compared with the controls (23/60 and 29/60, respectively). The mid-dose (2.0 ppm) group also exhibited a statistically significant ($P<0.05$) increase (31/48) in the incidence of combined mammary fibro adenoma/adenoma and carcinoma over the control group (29/60). Increases in mammary tumours in the mid–high (5 ppm) dose group did not reach statistical significance relative to the control. However the authors of the study argue that the biological significance of thyroid follicular adenomas and mammary fibroadenomas and relevance to humans is questionable, given that there was no dose-response.

To study the effect in humans, a retrospective study was conducted to evaluate the mortality experience of a cohort of employees of a perfluorooctanesulphonyl fluoride (POSF) based fluorochemical production facility in Decatur, Alabama (Alexander et al., 2003). The study followed all workers with at least one year of cumulative employment at the facility and assigned to one of three exposure subgroups; high exposed, low exposed, and non-exposed, based on biological monitoring data for perfluorooctane sulphonate (PFOS). The overall mortality rates for the cohort and the exposure subcohorts were lower than expected in the general population. Three cases of death from bladder cancer (out of a total of 2083 workers followed) were reported indicating a higher risk of bladder cancer from PFOS exposure. However, it was not clear whether these three cases could be attributed to fluorochemical exposure, as no employee specific exposure data for PFOS and other fluorochemicals were available. Information on potentially confounding factors, for example, smoking, were also not available for this cohort and influence of other workplace exposures was not assessed. Current toxicological evidence does not indicate that the bladder is a target of PFOS. The study authors concluded that “with only three cases, the possibility that the finding is due to chance cannot entirely be ruled out (Alexander et al., 2003).

In a follow-up study (Alexander & Olsen, 2007), the incidence of bladder cancer was ascertained by postal questionnaire to all living current and former employees of the facility (N = 1400) and death certificates for deceased workers (N = 185). Exposure to PFOS was estimated with work history records and weighted with biological monitoring data. Bladder cancer risk within the cohort was evaluated using Poisson regression by cumulative PFOS exposure. Eleven cases of primary bladder cancer were

identified from the surveys. Compared with employees in the lowest cumulative exposure category, the relative risk of bladder cancer was 0.83. No statistically significant association between PFOS exposure and an increased risk of bladder cancer was found in this study.

Detailed medical records (episodes of care) of workers at 3M's Decatur plant were used as an estimate of worker morbidity. The risk ratio for episodes of care for overall cancers was greatest in the group of employees with the highest and longest exposures to fluorochemicals. Increased risk of episodes of medical care was also reported for male reproductive cancers in long-time, high-exposure employees (EFSA, 2008).

Eriksen et al. (2009) investigated the association between plasma levels of PFOA and PFOS and cancer risk within a prospective Danish cohort of participants with no previous cancer diagnosis at enrolment. Between December 1, 1993 and May 31, 1997, and through July 1,2006, the authors identified 713 participants with prostate cancer, 332 with bladder cancer,128 with pancreatic cancer, and 67 with liver cancer in the entire cohort. Plasma concentrations of PFOA and PFOS were measured in each participant (1240 cancer patients and 772 healthy individuals) by use of high-pressure liquid chromatography coupled to tandem mass spectrometry. No clear differences were found in the incidence rate ratios for these cancers in relation to plasma concentrations of PFOA or PFOS. The authors concluded that PFOA and PFOS plasma concentrations in the general Danish population are not associated with increased risk of prostate, bladder, pancreatic, or liver cancer.

Reproductive and Developmental Toxicity

In animal studies, PFOS and its salts did not have an adverse effect on the reproductive parameters, but were toxic to development.

In a two-generation reproduction study in rats (Christian et al., 1999a), PFOS was administered to male and female rats by gavage at doses of 0, 0.1, 0.4, 1.6 and 3.2 mg/kg bw/day for 42 days before mating. The dosing continued in female rats during pregnancy and lactation. In the F0 generation female rats, there did not appear to be any effects on oestrous cycling, mating and fertility parameters, the numbers of corpora lutea and implantations, or in the number of viable or non-viable embryos. However, gestation length in F0 females was significantly reduced in the high-dose group and there was also a significant reduction in the number of implantation sites followed by a concomitant reduction in litter size. Reduced survival was observed in F1 offspring at the highest doses of 1.6 and 3.2 mg/kg bw/day. Pup body weights were significantly reduced in the two highest dose groups. In the F1 generation male rats, there was no effect on mating or fertility parameters; necroscopic examinations; absolute or relative weights of testes, seminal vesicles, right epididymis, or prostate; and terminal body weights. Clinical observations for the F1 generation females were likewise unremarkable. There were no statistically significant differences reported for mating and fertility parameters; gestation index; pregnancy rates; and necroscopic examinations.

In the F2 generation of the group treated with 0.4 mg/kg bw/day, birth weight was reduced (LOAEL). No other toxicological signs were reported in the F2 rats. The NOAEL was 0.1 mg/kg bw/day.

A cross-fostering study was conducted to determine whether the low pup survival rates of pups from PFOS-treated rats observed in the above study were a result of in utero exposure to PFOS, or as a result of exposure during lactation; thus the potential for a distinction to be made between prenatal and postnatal effects following continuous maternal treatment (Christian et al., 1999b). In this study, two groups of 25 female SD rats were administered 0 and 1.6 mg/kg/day PFOS in 0.5% Tween-80 by gavage, beginning 42 days before mating with untreated males, and continuing throughout gestation and into day 21 of lactation. PFOS concentrations in the serum of treated dams were 59.2–157 ug/ml.

Following completion of parturition, litters were immediately removed from their respective dams and placed with either a control or PFOS-treated dam for rearing. This cross-fostering procedure resulted in four groups of 12–13 dams or pups as follows:

A) control dams with litters from PFOS-treated dams, i.e. in utero exposure only;

B) control dams with litters from control dams, i.e. negative control;

C) PFOS treated dams with litters from PFOS-treated dams, i.e., both in utero and post-natal exposure; and

D) PFOS-treated dams with litters from control dams, i.e., post-natal exposure only.

Reductions in gestation length, average number of implantation sites and live litter size were observed in treated animals. Pup mortality was observed in two of the cross-fostered groups, A (10%) and C (19%). In addition, on day 4 of lactation, the number of live pups, numbers of surviving pups per litter, and live litter sizes were also reduced in these two groups. Pup mortality in groups B and D during lactation days 2-4 were at 1.6% and 1.1%, respectively.

Serum PFOS concentrations in the pups from treated dams, fostered with untreated dams (A), were 47.6–59.2 ug/ml. Serum PFOS concentrations in the pups from treated dams, fostered with treated dams (C), were 79.5–96.9 ug/ml. Serum PFOS concentrations in the pups from untreated dams, fostered with untreated dams (B), were below the limit of detection. Serum PFOS concentrations in the pups from untreated dams, fostered with treated dams (D), ranged from below the limit of detection to 35.7 ug/ml. These data indicate that exposure to PFOS can occur both in utero and via milk from treated dams.

In conclusion, pups from control dams that were cross-fostered with PFOS-treated dams (post-natal exposure only) had the same low mortality rate (1.1 %) as pups from control dams cross-fostered with control dams (1.6 %; negative control). Mortality rates in the remaining two groups, however, (i.e. control dams with litters from PFOS-treated dams, i.e., in utero exposure only; and PFOS-treated dams with litters from PFOS-treated dams, i.e., both in utero and post-natal exposure), were much higher at 10 % and 19 %, respectively. Under the limited conditions of the study, the data appear to indicate that reduced pup survival is mainly a result of in utero exposure to PFOS and that post-natal exposure via milk in conjunction with in utero exposure could also contribute to reduced pup survival. Additionally, PFOS was observed in the serum of pups from rats exposed to the test substance as well as in the serum of pups exposed via lactation only (not exposed in utero).

There is a large database on the developmental toxicity of PFOS in rats and rabbits. Foetal toxicity and neonatal effects have been observed at doses similar to or below those resulting in maternal toxicity. Observed developmental effects include decreases in gestation length, pup viability, reduction of foetal weight, cleft palate, anasarca (oedema), delayed ossification of bones (sternebrae and phalanges) and cardiac abnormalities (ventricular septal defects and enlargement of the right atrium). Dose–response curves are generally steep, with high mortality observed early after birth. The late gestational stage seems to be a very vulnerable period (Case et al., 2001; Luebker et al., 2005). PFOS-exposed neonates showed thyroxine (T4) reductions at all dose groups, but not T3 or TSH. Changes in thyroid hormones, observed after pregnant rats were exposed to PFOS, could influence brain development and hence affect behaviour in the offspring. The ontogeny of neurochemical and neurobehavioral markers was evaluated after prenatal PFOS exposure (Lau et al., 2003).

Grasty et al. (2003) investigated the critical window for prenatal exposure to PFOS, by administering PFOS potassium salt to pregnant rats by gavage at 25 mg/kg bw on GD 2–5, 6–9, 10–13, 14–17 or 17–20, or at 25 or 50 mg/kg bw on GD 19–20. Neonatal rat mortality occurred after dosing in all time periods, but the incidence of neonatal death increased as the exposure occurred later during gestation, reaching 100 % in the treatment group of GD17–20. Considering that PFOS-induced organ toxicity is incompatible with postnatal survival, the authors suggested that maturation of the lung and pulmonary function is a plausible target for PFOS.

Administration of PFOS by gavage to groups of 22 pregnant rats during GD 6–15 at doses of 0, 1, 5 and 10 mg/kg bw/day (Gortner, 1980) resulted in maternal toxicity (decreased body weight) with a NOAEL of 5 mg/kg bw/day and a LOAEL of 10 mg/kg bw/day. However, in all dose groups the most notable signs of developmental toxicity were abnormalities of the lens of the eye, the incidence of which was significantly greater than control only in the top dose group (10 mg/kg bw/day). In a similar study, gavage administration of PFOS to pregnant rats between GD 6 and 15 resulted in maternal weight loss and developmental toxicity in the 5 and 10 mg/kg bw/day dose groups. Reduced birth weight as well as visceral anomalies, delayed ossification and skeletal variations were observed. A NOAEL of 1 mg/kg bw/day and a LOAEL of 5 mg/kg bw/day for maternal and developmental toxicity were indicated (Wetzel, 1983).

Risk Characterisation

Critical Health Effects

PFOS has moderate acute toxicity from oral exposure and low to moderate toxicity from inhalation exposure. It was found to be mildly irritating to the eyes and non-irritating to the skin of rabbits.

Repeated exposure to PFOS resulted in hepatotoxicity and mortality; the dose–response curve for mortality is very steep for rats and primates. Adverse signs of toxicity include hepatic vacuolisation and hepatocellular centrilobular hypertrophy, gastrointestinal effects, haematological abnormalities, convulsions and death.

Long-term exposure of rodents to PFOS caused significant increases in the incidence of benign tumours of the liver; thyroid and mammary glands. Workers occupationally exposed to PFOS were followed up. The results showed that workers in jobs involving high exposure to PFOS-based materials had a 13-times increased risk for bladder cancer mortality compared with the general population of Alabama.

Postnatal deaths and other developmental effects were reported at low doses in offspring in rats exposed to PFOS. The available data indicate that reduced pup survival is mainly a result of in utero exposure to PFOS, although post-natal exposure via milk in conjunction with in utero exposure could also contribute to reduced pup survival.

Public Risk Characterisation

Given the uses identified for these chemicals, it is unlikely that the public will be exposed. Hence, the public risk from these chemicals is not considered to be unreasonable. While there are epidemiological findings in workers exposed to these chemicals, they do not provide clear evidence of effects in humans, and public exposure to similar levels is not expected.

Secondary exposure to PFOS via the environment

Public exposure to PFOS could occur through secondary exposure via the environment. In Australia, PFOS has been found in drinking water at concentrations up to 0.02 micrograms per litre (NICNAS, 2015). While long-term studies in animals showed adverse effects from exposure to PFOS, epidemiological studies in workers exposed to PFOS did not provide clear evidence of effects in humans; and exposure of the general public to similar levels is not expected. Serum concentrations of PFOS in the Australian population have decreased from 2002 through to 2011, with the concentrations in serum ranging from 4.4-17.4 ng/mL (Toms et al., 2014). These levels are similar to median serum PFOS levels ranging between 1.93–44.7 µg/L reported in a recent study in subjects from different geographic locations (China) (Zeng et al., 2015). These serum levels are several orders of magnitude lower than the maternal serum levels in experimental studies (82 µg/mL) that showed developmental toxicity in the offspring (Luebker et al., 2005). In addition, PFOS was not detected in a survey of 65 foods and beverages packaged in glass, paper, plastic or cans, conducted by Food Standards Australia New Zealand (FSANZ, 2010). Nevertheless, since these chemicals will ultimately degrade to PFOS, which is extremely persistent in all media and can bioaccumulate, it is recommended that the chemicals in this group be restricted to only essential uses for which no suitable or less hazardous alternatives are available.

Occupational Risk Characterisation

During product formulation, dermal, ocular and inhalation exposure might occur, particularly where manual or open processes are used. These could include transfer and blending activities, quality control analysis, and cleaning and maintaining equipment. Worker exposure to the chemicals at lower concentrations could also occur while using formulated products containing the chemicals. The level and route of exposure will vary depending on the method of application and work practices employed.

NICNAS Recommendation

PFOS and its salts and PFOSF (direct precursors) have been added to the list of Persistent Organic Pollutants (POPs) under the Stockholm Convention (Stockholm Convention, 2014). POPs are chemicals that are toxic, persist in the environment, accumulate in the food chain, and pose a risk of causing adverse effects to human health and the environment, even at low concentrations. NICNAS is working in conjunction with the Department of the Environment (DoE), to introduce regulatory measures to restrict the manufacture, import and use of PFOS and related compounds.

Regulatory Control

Public Health

The need for regulatory control for public health will be determined as part of the regulatory measures for chemicals declared as POPs. These may include restrictions on the manufacture, import and use of PFOS and the related compounds.

Work Health and Safety

The need for regulatory control for workers health will be determined as part of the regulatory measures for chemicals declared as POPs. These may include restrictions on the manufacture, import and use of PFOS and the related compounds.

The chemicals are recommended for classification and labelling under the current approved criteria and adopted GHS as below. This assessment does not consider classification of physical and environmental hazards.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Harmful if swallowed (Xn; R22)* Harmful by inhalation (Xn; R20)*	Toxic if swallowed - Cat. 3 (H301) Harmful if inhaled - Cat. 4 (H332)
Repeat Dose Toxicity	Toxic: Danger of serious damage to health by prolonged exposure if swallowed (T; R48/25)*	Causes damage to organs through prolonged or repeated exposure - Cat. 1 (H372)
Carcinogenicity	Carc. Cat 3 - Limited evidence of a carcinogenic effect (Xn; R40)*	Suspected of causing cancer - Cat. 2 (H351)
Reproductive and Developmental Toxicity	May cause harm to breastfed babies (Xn; R64)* Repro. Cat 2 - May cause harm to the unborn child (T; R61)*	May cause harm to breast-fed children (H362) May damage the unborn child - Cat. 1B (H360D)

[a] Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(2004)].

[b] Globally Harmonized System of Classification and Labelling of Chemicals (GHS) United Nations, 2009. Third Edition.

[*] Existing Hazard Classification. No change recommended to this classification

Advice for consumers

Products containing the chemicals should be used according to the instructions on the label.

Advice for industry

Control measures

Control measures to minimise the risk from oral exposure to the chemicals should be implemented in accordance with the hierarchy of controls. Approaches to minimise risk include substitution, isolation and engineering controls. Measures required to eliminate, or minimise risk arising from storing, handling and using a hazardous chemical depend on the physical form and the manner in which the chemicals are used. Examples of control measures which could minimise the risk include, but are not limited to:

- using closed systems or isolating operations;
- health monitoring for any worker who is at risk of exposure to the chemical[s], if valid techniques are available to monitor the effect on the worker's health;
- minimising manual processes and work tasks through automating processes;

- work procedures that minimise splashes and spills;
- regularly cleaning equipment and work areas; and
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemicals.

Guidance on managing risks from hazardous chemicals are provided in the *Managing risks of hazardous chemicals in the workplace—Code of practice* available on the Safe Work Australia website.

Personal protective equipment should not solely be relied upon to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk. Guidance in selecting personal protective equipment can be obtained from Australian, Australian/New Zealand or other approved standards.

Obligations under workplace health and safety legislation

Information in this report should be taken into account to help meet obligations under workplace health and safety legislation as adopted by the relevant state or territory. This includes, but is not limited to:

- ensuring that hazardous chemicals are correctly classified and labelled;
- ensuring that (material) safety data sheets ((M)SDS) containing accurate information about the hazards (relating to both health hazards and physicochemical (physical) hazards) of the chemicals are prepared; and
- managing risks arising from storing, handling and using a hazardous chemical.

Your work health and safety regulator should be contacted for information on the work health and safety laws in your jurisdiction.

Information on how to prepare an (M)SDS and how to label containers of hazardous chemicals are provided in relevant codes of practice such as the *Preparation of safety data sheets for hazardous chemicals—Code of practice* and *Labelling of workplace hazardous chemicals—Code of practice*, respectively. These codes of practice are available from the Safe Work Australia website.

A review of the physical hazards of these chemicals has not been undertaken as part of this assessment.

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Chemical Identities

Chemical Name in the Inventory and Synonyms	**1-Octanesulfonyl fluoride, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-** 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-Heptadecafluoro-1-octanesulfonyl fluoride 1-Octanesulfonyl fluoride, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-
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	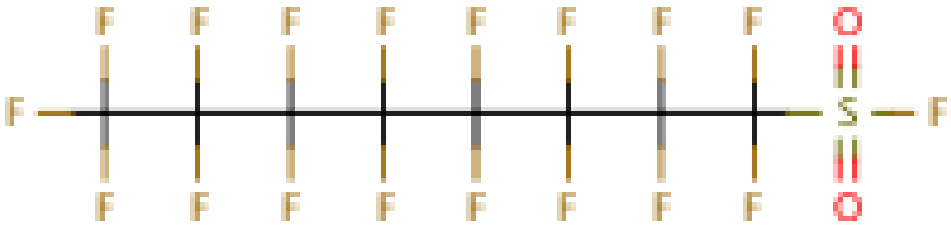
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Molecular Weight	502

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	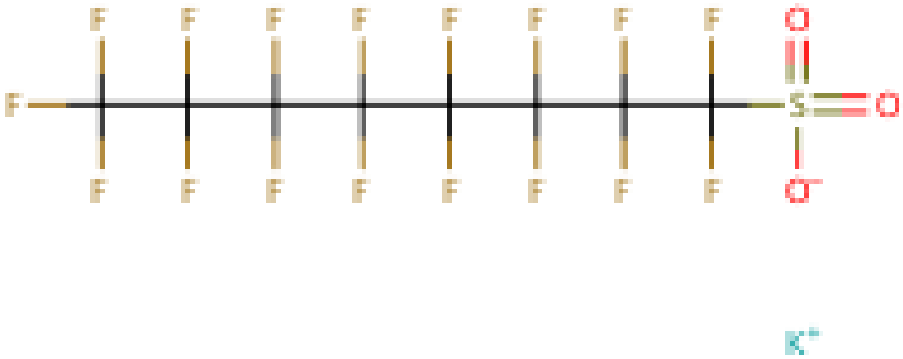
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	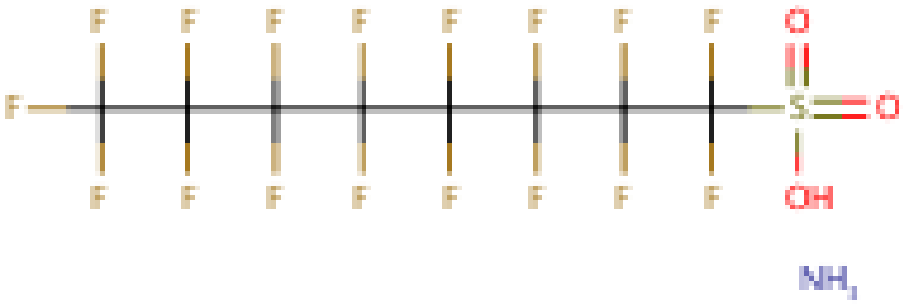
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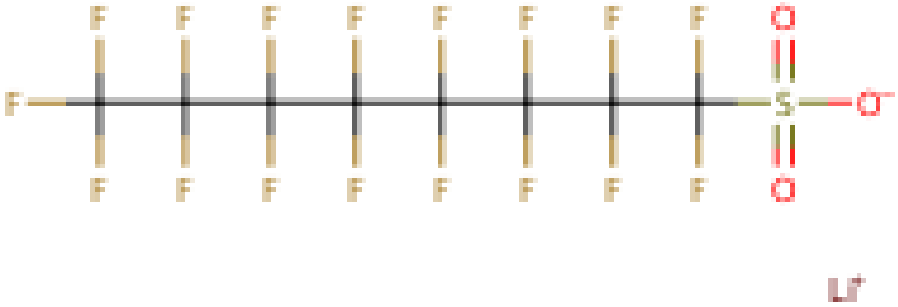

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Molecular Weight	506

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	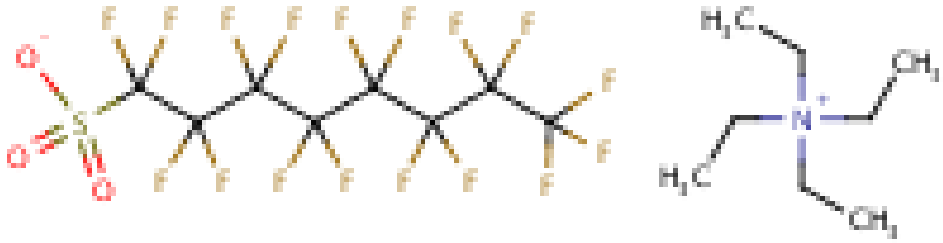
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	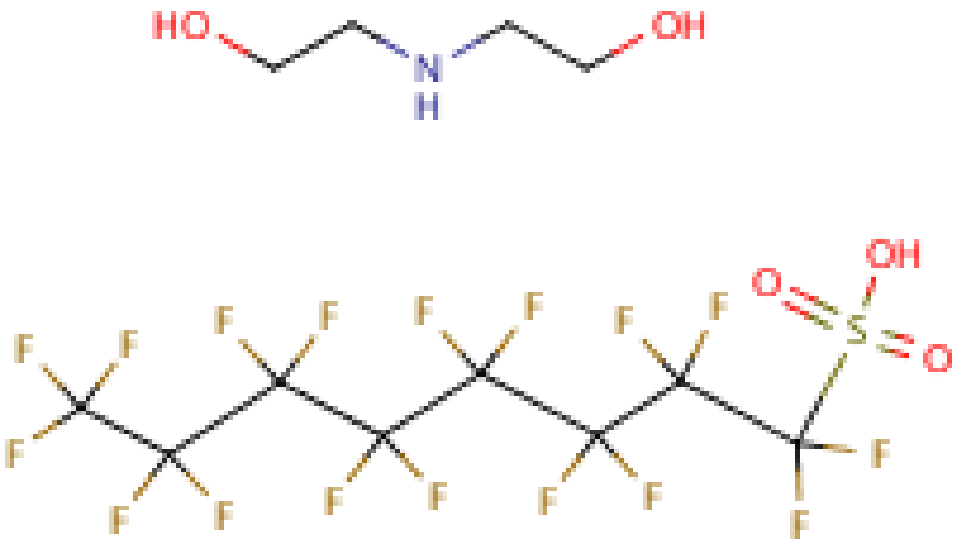
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