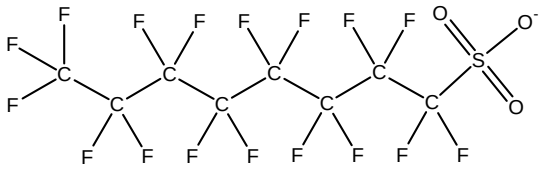
Perfluorooctane Sulfonate, Its Salts and Its Precursors that Contain the $C_8F_{17}SO_2$ or $C_8F_{17}SO_3$ Moiety



Introduction

The *Canadian Environmental Protection Act, 1999* (CEPA 1999) 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.

Screening assessments of risks to human health focus on conservative effect levels for critical endpoints and upper-bounding estimates of exposure, after consideration of all relevant identified information. Decisions based on the nature of the critical effects and margins between conservative effect levels and estimates of exposure take into account confidence in the completeness of the identified databases on both exposure and effects, within a screening context. Additional background information on screening health assessments conducted under this program is available at http://www.hc-sc.gc.ca/exsd-dse.

A screening health assessment was undertaken on perfluorooctane sulfonate (PFOS), its salts and its precursors containing the $C_8F_{17}SO_2$ or $C_8F_{17}SO_3$ moiety on the basis that some of these compounds were included in the Domestic Substances List pilot phase for screening based on their meeting the criteria for persistence and/or bioaccumulation and inherent toxicity to non-human organisms, pursuant to Section 73(1)(b) of CEPA 1999, and in response to a request to the Minister of the Environment to add these compounds to the Priority Substances List.

This screening assessment report on human health aspects and associated unpublished supporting working documentation were prepared by evaluators within the Existing Substances Division of Health Canada, and their content was reviewed at several meetings of senior

Divisional staff. The assessment report was subsequently externally reviewed for adequacy of data coverage and defensibility of the conclusions. The assessment reports on health and environmental aspects were approved by the joint Environment Canada/Health Canada CEPA Management Committee. The supporting working documentation is available upon request by e-mail from ExSD@hc-sc.gc.ca. Information on the screening environmental assessment is available at http://www.ec.gc.ca/substances/ese.

Information identified as of September 2003 was considered for inclusion in this screening health assessment. The critical information and considerations upon which the assessment is based are summarized below.

Identity, Uses and Sources of Exposure

PFOS, its salts and its precursors form part of a larger chemical class of fluorochemicals typically referred to as perfluorinated alkyl compounds (PFAs). Depending upon the intended use, the various precursors of PFOS are formed via derivatization of perfluorooctanesulfonylfluoride (POSF: $C_8F_{17}SO_2F$) (OECD, 2002), yielding molecules with the general chemical formula of $CF_3(CF_2)_7SO_2$-R. The screening health assessment of PFOS, its salts and its precursors containing the $C_8F_{17}SO_2$ or $C_8F_{17}SO_3$ moiety covers some 50 substances, many of which are on the Domestic Substances List (see Appendix 1).

The results of a survey of Canadian industry conducted in 2000 under Section 71 of CEPA 1999 to determine the manufacture, import, export and uses of specific PFAs, their derivatives and polymers indicated that there was no known manufacture of PFAs, including PFOS, in Canada. Almost 600 000 kg of PFAs were imported into Canada between 1997 and 2000, PFOS representing only a very small proportion of this total (Environment Canada, 2001). PFOS and its precursors were imported into Canada as chemicals or in various products. As noted elsewhere (OECD, 2002), the principal applications for PFOS and its precursors are for water, oil, soil and grease repellents for use on surface and paper-based applications, such as rugs and carpets, fabric and upholstery and food packaging, as well as use in specialized chemical applications, such as fire-fighting foams, hydraulic fluids, carpet spot removers, mining and oil well surfactants and other specialized chemical formulations. Owing to these use patterns, the exposure of humans to such substances would likely result from contact with, and/or the use of, certain consumer products (3M, 1999a).

The screening health assessment on PFOS, its salts and its precursors containing the $C_8F_{17}SO_2$ or $C_8F_{17}SO_3$ moiety is based upon a comparison of the margin between the levels of PFOS in the blood and liver of laboratory animals[1] that are associated with the development of toxicological effects and the levels in the blood and liver of humans. The following

[1] Available data from identified epidemiological studies were considered inadequate for such analysis.

considerations were taken into account in developing the approach for the screening health assessment of PFOS, its salts and precursors listed herein:

- Chemical, environmental and metabolic processes can lead to the removal of the perfluorinated moiety, ultimately yielding PFOS (3M, 1999a,b).
- PFOS is persistent and is not further degraded or metabolically converted to other compounds (3M, 1999a,b).
- On the basis of CATABOL modelling (Mekenyan and Dimitrov, 2002), the substances listed in Appendix 1 of this assessment were considered to have the potential to biodegrade to PFOS.
- Since PFOS is likely the ultimate perfluorinated degradation or metabolic product of the group of substances listed in Appendix 1, the level of this compound in human tissue provides a useful indicator of exposure to this group of substances from all potential sources.
- Biomonitoring in humans (and animal species) has focused on PFOS, which has been detected in the blood of non-occupationally exposed humans in North America and Europe.
- The toxicity profile of those PFOS precursors examined here (see table below) appears to be generally similar to that of PFOS itself. Available data indicate that effects associated with the PFOS precursors occur at exposures that are similar to or slightly higher than those for PFOS.

Toxicological data relevant to this screening health assessment report were identified for the following substances on the Domestic Substances List (see following table), and these data were used as the basis for assessing the PFOS precursors listed in Appendix 1:

Substance	**Designation**	**CAS No.**
1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro- (perfluorooctane sulfonate)	PFOS	2795-39-3 (potassium salt) 29081-56-9 (ammonium salt) 70225-14-8 (diethanolamine salt)
1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)- (N-ethylperfluorooctane sulfonamidoethanol)	N-EtFOSE	1691-99-2
1-Octanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-N-methyl- (N-methylperfluorooctane sulfonamidoethanol)	N-MeFOSE	24448-09-7
1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro- (N-ethyl perfluorooctane sulfonamide)	N-EtFOSA	4151-50-2
1-Octanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-methyl- (N-methyl perfluorooctane sulfonamide)	N-MeFOSA	31506-32-8
Glycine, N-ethyl-N-[(heptadecafluorooctyl)-sulfonyl]-, potassium salt (potassium-N-ethyl-N((heptadecafluorooctyl)-sulfonyl)-glycinate)	PFOSAA	2991-51-7

Substance	Designation	CAS No.
Ethanaminium, N,N,N-trimethyl-2-[(2-methyl-1-oxo-2-propenyl)oxy]-, chloride, polymer with 2-ethoxyethyl 2-propenoate, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate and oxiranylmethyl 2-methyl-2-propenoate		92265-81-1

Exposure Assessment and Hazard Characterization

Although the identified data for some of these substances (see table above) were variable and limited, the available information indicates that toxicological effects of these precursors of PFOS are similar to those of PFOS itself (Table 1). Moreover, based upon the data identified, health-related effects associated with exposure to these substances would appear to be somewhat less severe and/or are observed at higher exposures (doses) than those associated with exposure to PFOS itself.

Studies considered critical to the screening health assessment of PFOS, its salts and its precursors (i.e., those with lowest effect levels) are long-term repeated-dose investigations conducted with rodents and primates. In rats receiving PFOS in the diet for 2 years, histopathological effects in the liver were observed in males and females at intakes as low as 0.06–0.23 mg/kg-bw per day and 0.07–0.21 mg/kg-bw per day, respectively (Covance Laboratories, Inc., 2002a). The mean levels of PFOS in the serum and liver of males and females with these intakes were 7.6 and 20.2 µg/ml and 26.4 and 55.1 µg/g, respectively, after 2 years of exposure (3M Environmental Laboratory, 2001). Evidence of increased thymic atrophy (in females) and reduced serum high-density lipoprotein, cholesterol, bilirubin and triiodothyronine levels (in males) were observed in monkeys administered 0.03 mg PFOS/kg-bw per day for 26 weeks (Covance Laboratories, Inc., 2002b).[2] The mean levels of PFOS in the serum of the males and females after 26 weeks on study were 15.8 and 13.2 µg/ml, respectively (3M Environmental Laboratory, 2000). The mean levels of PFOS in the liver of males and females after 27 weeks on study were 17.3 and 22.2 µg/g, respectively.

Based upon data from chronic exposure studies, there is evidence for the carcinogenicity of PFOS (increased incidence of hepatocellular adenoma in males and females) and N-EtFOSE (increased incidence of hepatocellular adenoma in females; thyroid follicular cell adenoma in males) in rats. In both of these studies, statistically significant increases in tumour incidence were observed only at the highest doses tested — that is, at doses that were above those associated with the development of non-neoplastic effects in these animals. PFOS and its related substances evaluated here are not genotoxic based upon the results of a wide range of identified *in vitro* and *in vivo* assays.

Identified data included investigations on health effects in workers occupationally exposed to PFOS. Although a significantly elevated risk of bladder cancer has been observed for

[2] Also reported in Seacat et al. (2002).

one group of workers exposed to PFOS, the identified epidemiological studies of workers occupationally exposed to PFOS are considered inadequate to assess the potential of this substance (and its precursors) to induce cancer in humans. Workers were exposed to other substances, and the observed number of cause-specific and overall deaths was relatively small. Moreover, the database is inadequate as a basis to assess aspects of weight of evidence of causality, such as consistency.

In other surveys of occupationally exposed workers in which potential relationships between exposure to PFOS (assessed by monitoring serum PFOS levels in workers, which ranged as high as 10 µg/ml) and effects upon clinical chemistries and haematological and hormonal parameters were assessed, consistent associations have not been observed. The sensitivity of these investigations is limited by low participation rates and employee turnover during study periods at certain facilities.

In a recent study providing preliminary analytical data on the occurrence and distribution of selected organic perfluorinated compounds in the blood of 56 volunteer non-occupationally exposed adult Canadians, PFOS was detected in 100% of the samples (Kubwabo et al., 2002). Measured concentrations of PFOS in the serum ranged from 0.0037 to 0.065 µg/ml; the overall mean and 95th-percentile concentrations were 0.0288 and 0.0631 µg/ml, respectively. These levels are similar to those measured in other contemporary biomonitoring studies conducted in the United States and Europe (OECD, 2002), with the overall range of serum PFOS levels not appearing to have increased markedly over the past few years.

In one study of 599 children (aged 2–12 years) in the United States conducted between 1994 and 1995, the geometric mean concentration of PFOS in the serum was 37.5 ppb (i.e., 0.0375 µg/ml); the 95th percentile was 97 ppb (0.097 µg/ml), with a small number of samples (<20) exceeding this value. Individual values ranging widely from 7 to 515 ppb (i.e., 0.0067–0.515 µg/ml) (3M Medical Department, 2002). Although an analysis of historical blood samples for PFOS conducted in the late 1990s revealed a significant increase in PFOS levels about 20 years after these substances were first produced commercially (i.e., the late 1940s), there appears to have been no substantial increasing trend in levels between the early 1970s and the late 1990s, although data are limited (3M, 1999a).

In view of the physical/chemical properties of PFOS, the substance is not expected to accumulate in breast milk. Although biomonitoring data in very young children have not been identified, PFOS levels in the sera of fetuses and very young offspring do not exceed those of exposed dams in experimental studies (Argus Research Laboratories, 1999e,f).

In analyses of liver samples collected from 30 cadavers in the United States, levels of PFOS ranged from <0.0045 (limit of quantitation) to 0.057 µg/g (Olsen et al., 2003). The mean and geometric mean concentrations were 0.0188 and 0.0152 µg/g, respectively.

Confidence in the effects assessment for PFOS and N-EtFOSE is high, owing to the available database, which covers a wide range of toxicological endpoints. Although confidence in the effects assessment for the remaining PFOS-related compounds is low, due to the lack of identified data, this is mitigated to some degree by the consideration that these compounds are likely converted to PFOS in environmental and biological media. Confidence in the measure of the "internal dose" of PFOS (i.e., levels of PFOS in serum and liver) to assess exposure of the general population to this group of compounds is high. Although the size of the sampled population of individuals in Canada is relatively small, the mean measured concentration is similar to that reported elsewhere for samples collected from other non-occupationally exposed populations in the United States and Europe. The levels of serum PFOS do not appear to have increased markedly over the past 20 years (3M, 1999a). Although there may be somewhat less confidence in the available data on the levels of PFOS in human liver, owing to the small sample size in the single report, use of this exposure metric takes into account aspects related to both the toxicokinetics and metabolism of such substances.

Proposed Conclusion for Human Health

The two laboratory studies considered critical for the margin of exposure analysis are the chronic toxicity study in rats, with a large number of animals per dose group exposed for nearly their lifetime, and the study in which small groups of monkeys (considered as better surrogates for humans) received PFOS for 26 weeks. The levels of PFOS in the serum and liver of these animals at the critical effect level are less than those associated with the critical effect in the F_0 and F_1 rats in the two-generation reproduction/developmental toxicity study.

Most often in the screening health assessment of Existing Substances under CEPA 1999, margins of exposure are based upon a comparison of the doses (intakes) administered to laboratory animals at which substance-induced effects were observed with the upper-bound estimate of human intake. While such estimates of intake for PFOS are included in the supporting working document, they are less reliable as a basis for development of margins than those based on serum levels. In this case, therefore, comparisons have been based upon information on the levels of PFOS in the serum and liver from animals administered PFOS and data from human biomonitoring studies. This obviates the need to take into account the significant uncertainty associated with a determination of the upper-bound estimate of human intake of PFOS, owing to the limited available data on levels of PFOS and precursors in air, foodstuffs, drinking water and breast milk and resulting from contact with household materials treated with such perfluorinated substances. Moreover, the levels of PFOS in human tissues provide a useful indicator of the combined exposure from all sources.

Comparisons of the levels of PFOS in the serum and liver of animals at the critical effect level with levels in serum and/or liver from human adult and children biomonitoring studies are presented in the following table:

Critical study and effect	PFOS dose metric at critical effect	Metric(s) of human exposure to PFOS	Margin of exposure (critical effect/human exposure)
Microscopic changes in the liver of rats (m + f) receiving PFOS in the diet for 2 years[1]	**Serum PFOS level:** 13.9 µg/ml[2]	**Mean serum PFOS level in adults in Canada[3]:** 0.028 µg/ml	496
		95th percentile of human serum PFOS level in adults in Canada[3]: 0.0631 µg/ml	220
		Mean serum PFOS level in children in the United States[4]: 0.0375 µg/ml	371
		95th percentile of serum PFOS level in children in the United States[4]: 0.097 µg/ml	143
	Liver PFOS level: 40.8 µg/g[5]	**Mean[6] liver PFOS level:** 0.0188 µg/g	2170[7]
Thymic atrophy (f), reduced serum high-density lipoprotein (m), cholesterol (m), triiodothyronine (m) and total bilirubin (m) in monkeys administered PFOS for 26 weeks[1]	**Serum PFOS level:** 14.5 µg/ml[8]	**Mean serum PFOS level in adults in Canada[3]:** 0.028 µg/ml	518
		95th percentile of human serum PFOS level in adults in Canada[3]: 0.0631 µg/ml	230
		Mean serum PFOS level in children in the United States[4]: 0.0375 µg/ml	387
		95th percentile of serum PFOS level in children in the United States[4]: 0.097 µg/ml	149
	Liver PFOS level: 19.8 µg/g[9]	**Mean[10] liver PFOS level:** 0.0188 µg/g	1053[11]

1 Covance Laboratories, Inc. (2002a).
2 Average of mean levels in males (7.6 µg/ml) and females (20.2 µg/ml).
3 Kubwabo et al. (2002).
4 3M Medical Department (2002).
5 Average of mean levels in males 26.4 (µg/g) and females (55.1 µg/g).
6 Mean level of PFOS in livers from 30 cadavers (Olsen et al., 2003).
7 Published data on 95th percentile not available. Margin of exposure based upon highest level of PFOS in human liver from this study (0.057 µg/g) is 716.
8 Average of mean levels in males (15.8 µg/ml) and females (13.2 µg/ml) (week 26).
9 Average of mean levels in males 17.3 (µg/g) and females (22.2 µg/g) (week 27).
10 Mean level of PFOS in livers from 30 cadavers (Olsen et al., 2003).
11 Published data on 95th percentile not available. Margin of exposure based upon highest level of PFOS in human liver from this study (0.057 µg/g) is 347.

These margins are considered adequate to address elements of uncertainty, including

intraspecies variation, interspecies variation and biological adversity or severity of the effects considered critical here. These margins will also be protective for the increased incidence of tumours observed in the chronic study of PFOS in rats, since the tumours were observed only at doses of PFOS that were higher than those that induced non-neoplastic effects and since the weight of evidence indicates that PFOS (and its precursors) are not genotoxic. While the margins for blood levels in children are somewhat less (approximately 145 for the 95th-percentile values), more appropriate margins for comparison with the effect level from long-term studies are those for adults (approximately 225 for the 95th-percentile values), since they are exposed for a greater portion of their life span. In addition, the critical lowest-observed-effect levels selected for development of these margins of exposure are very conservative, being about an order of magnitude less than values in other studies (i.e., for effects observed in reproductive studies with rats). The margins are also based on more relevant metrics of exposure to PFOS than dose in experimental studies and deterministic estimates of daily intake in children and adults and, as a result, account for a significant portion of the uncertainties associated with interspecies and intraspecies differences in pharmacokinetics (usually accounted for by 4-fold and 3.2-fold default uncertainty factors, respectively). The higher margins for values in liver, although based on limited data, take into account even a greater proportion of uncertainty in toxicokinetics. The margins also take into account limitations of the database for human exposure. Use of the 95th percentiles for the serum levels is also more conservative than deterministic estimates of exposure, which are based on mean intakes of environmental media.

Based upon these margins of exposure, it is proposed that PFOS, its salts and its precursors not be considered “toxic” as defined in Paragraph 64(c) of CEPA 1999.

Proposed Recommendations to the Ministers of Environment and Health

On the basis of environmental considerations, it is proposed that PFOS, its salts and its precursors, as a group, be considered “toxic” as defined in Section 64 of CEPA 1999 (see http://www.ec.gc.ca/substances/ese).

Table 1: Summary of health effects information for PFOS and related compounds

Endpoint	PFOS	N-EtFOSE	N-EtFOSA	N-MeFOSE	N-MeFOSA	PFOSAA	Ethanaminium...
Acute toxicity: oral	**Oral LD_{50}** rat (m/f) **LD_{50} = 251 mg/kg-bw** (International Research and Development Corporation, 1978a) [Additional studies: Hazleton Laboratories America, Inc., 1987a; Hazleton Wisconsin, Inc., 1994a; Corning Hazleton, Inc., 1997a]	**Oral LD_{50}** rat (m/f) **LD_{50} = 1467 mg/kg-bw** (International Research and Development Corporation, 1978b)	**Oral LD_{50}** rat (m/f) **LD_{50} = >500 and <5000 mg/kg-bw** (Riker Laboratories, Inc., 1981b) [Additional study: Riker Laboratories, Inc., 1987]	**Oral LD_{50}** rat (m/f) **LD_{50} = >1000 and <5000 mg/kg-bw** (Riker Laboratories, Inc., 1979)	**Oral LD_{50}** rat (m/f) **LD_{50} = 350 mg/kg-bw** (Hazleton Laboratories America, Inc., 1985a) [Additional studies: Riker Laboratories, Inc., 1981c; Hazleton Laboratories America, Inc., 1985b]	**Oral LD_{50}** rat (m/f) **LD_{50} = >0.5 and <5 ml/kg-bw** (Biosearch, Inc., 1978c) [Additional studies: Biosearch, Inc., 1978a; Hazleton Laboratories America, Inc., 1988a]	**Oral LD_{50}** rat (m/f) **LD_{50} = >5 g/kg-bw** (Hazleton Wisconsin, Inc., 1991a)
Acute toxicity: dermal						**Dermal LD_{50}** rabbit (m/f) 24-hour covered **LD_{50} = >2000 mg/kg-bw** (Hazleton Laboratories America, Inc., 1988b)	
Acute toxicity: inhalation	**Inhalation LC_{50}** rat (m/f) **LC_{50} = 5200 mg/m^3** (Bio/Dynamics, Inc., 1979a)	**Inhalation LC_{50}** rat (m/f) **LC_{50} = >6.5 g/m^3** (Hazleton Laboratories America, Inc., 1981)				**Inhalation LC_{50}** rat (m/f) **LC_{50} = >22 g/m^3 and <66 g/m^3** (Bio/Dynamics, Inc., 1979b)	

Endpoint	PFOS	N-EtFOSE	N-EtFOSA	N-MeFOSE	N-MeFOSA	PFOSAA	Ethanaminium...
Irritation: ocular	**Severe irritation:** rabbit 0.1 ml ocular application, washout after 5 or 30 seconds (Riker Laboratories, Inc., 1981a) [Additional studies: **mild to moderate irritation:** Warf Institute, Inc., 1974, 1975; Hazleton Laboratories America, Inc., 1987b; Hazleton Wisconsin, Inc., 1994b; Corning Hazleton, Inc., 1997b]		**Minimal irritation:** rabbit (f) 0.1 g ocular application (Riker Laboratories, Inc., 1984)	**No irritation:** rabbit 0.1 g ocular application (Biosearch, Inc., 1978b)	**Minimal irritation:** rabbit (f) 0.09 g ocular application (Hazleton Laboratories America, Inc., 1985c) [Additional study: Hazleton Laboratories America, Inc., 1985d]	**Mild irritation:** rabbit 0.1 ml ocular application (unwashed) (Hazleton Laboratories America, Inc., 1988c) [Additional study: Biosearch, Inc., 1978d]	**Moderate irritation:** rabbit 0.1 ml ocular application (unwashed) (Hazleton Wisconsin, Inc., 1991b)
Short-term repeated-dose toxicity	**Oral gavage LOAEL** rat (m/f), 28 days **LOAEL = 3 mg/kg-bw per day** hepatocellular hypertrophy (m/f); increased relative liver weight (m/f); increased relative kidney weight (f); reduced body weight (f) (NOTOX, 1999) [Additional study: Austin et al., 2003]			**Dietary LOEL** rat (m/f), at least 4 weeks **LOEL (m/f) = 2.4–4.1 mg/kg-bw per day** increased relative liver weight (f), hepatocellular hypertrophy (m) (investigators give LOAEL of 35–63 mg/kg-bw per day, ignoring liver effects at lower doses) (Covance Laboratories, Inc., 2000a)			

Endpoint	PFOS	N-EtFOSE	N-EtFOSA	N-MeFOSE	N-MeFOSA	PFOSAA	Ethanaminium...
Subchronic toxicity	**Oral gavage LOEL** rhesus monkey (m/f), 90 days **LOEL = 0.5 mg/kg-bw per day** clinical signs of toxicity and increased leukocytes (International Research and Development Corporation, 1978e) [Additional study: International Research and Development Corporation, 1978c]	**Oral diet LOEL** rat (m/f), 90 days **LOEL (m) = 2 mg/kg-bw per day** slight hepatocellular vacuolization; decreased hemoglobin and hematocrit (International Research and Development Corporation, 1978d) [Additional study: International Research and Development Corporation, 1979]		**Oral diet LOEL** rat (m/f), 13 weeks **LOEL (m) = 2 mg/kg-bw per day** increased relative brain, kidney, liver and testis weights; histopathological changes in the liver, reductions in serum cholesterol and triglycerides (Covance Laboratories, Inc., 1999d)			

Endpoint	PFOS	N-EtFOSE	N-EtFOSA	N-MeFOSE	N-MeFOSA	PFOSAA	Ethanaminium...
Carcinogenicity/ chronic	**Oral diet LOAEL** rat (m/f), 104 weeks **LOAEL = 0.06–0.23 mg/kg-bw per day** increased incidence of non-neoplastic changes in the liver (statistically significant increased incidence of hepatocellular adenoma in males and females at intakes of 0.64–2.21 mg/kg-bw per day) (Covance Laboratories, Inc., 2002a) **Oral LOEL** cynomolgus monkey (m/f), 26 weeks **LOEL (m/f) = 0.03 mg/kg-bw per day** *m*: reduced high-density lipoprotein and triiodothyronine levels, thymic atrophy *f*: thymic atrophy (Covance Laboratories, Inc., 2002b)	**Oral diet LOEL** rat (m/f) 104-week cancer bioassay with N-EtFOSE narrow range (98.1%) **LOEL (m) = 0.86–2.618 mg/kg-bw per day LOEL (f) = 4.213–10.166 mg/kg-bw per day** *m/f*: increased incidence of histopathological effects in liver *f*: significant ($p < 0.05$) reduced serum triglycerides after 104 weeks (statistically significant increased incidences of thyroid follicular adenoma in males at intakes of 3.1–8.72 mg/kg-bw per day and of hepatocellular adenoma in females at intakes of 4.213–10.166 mg/kg-bw per day) (Covance Laboratories, Inc., 2001) [Additional study: Riker Laboratories, Inc., 1983]					
Genotoxicity and related endpoints: *in vivo*	**Negative:** mouse (m/f) bone marrow micronucleus 950 mg/kg-bw; acute oral gavage (Corning Hazleton, Inc., 1996b)	**Negative:** mouse (m/f) bone marrow micronucleus 2200 mg/kg-bw; acute oral gavage (Corning Hazleton, Inc., 1996a) [Additional studies: Corning Hazleton, Inc., 1993; Hazleton Washington, Inc., 1993a]	**Negative:** mouse (m/f) bone marrow micronucleus, 4000 mg/kg-bw, acute oral gavage (Corning Hazleton, Inc., 1996c)	**Negative:** rat (m/f) bone marrow micronucleus, 5000 mg/kg-bw, acute oral gavage, and rat hepatic unscheduled DNA synthesis *in vivo/in vitro* (Hazleton Washington, Inc., 1993b,c)			
Genotoxicity and related endpoints: *in vitro*	**Negative:** with/without metabolic activation: Ames *Salmonella/E. coli*	**Negative (-S9), questionable (+S9):** mouse lymphoma L5178Y; *in vitro*	**Negative:** Ames *Salmonella* mutagenicity and	**Negative:** with/without metabolic	**Negative:** with/without metabolic activation: Ames *Salmonella* mutagenicity	**Negative:** with/without metabolic	

Endpoint	PFOS	N-EtFOSE	N-EtFOSA	N-MeFOSE	N-MeFOSA	PFOSAA	Ethanaminium...
	mutagenicity, *S. cerevisiae* mitotic recombinogenicity, rat hepatocyte unscheduled DNA synthesis and human lymphocyte chromosomal aberration *in vitro* assays (Litton Bionetics, Inc., 1978; SRI International, 1978, 1980, 1981; Covance Laboratories, Inc., 1999a,b,c)	mutation -S9/+S9 (NOTOX, 1998) [Additional **negative** *in vitro* study: Covance Laboratories, Inc., 2000b]	Chinese hamster ovary sister chromatid exchange *in vitro* assays (U.S. EPA, 1989)	activation: Ames *Salmonella* mutagenicity, mouse L5178Y lymphoma mutation and human lymphocyte chromosomal aberration *in vitro* assays (NOTOX, 1994a,b,c)	and yeast recombination *in vitro* assays (SRI International, 1985)	activation: Ames *Salmonella* mutagenicity and yeast recombination *in vitro* assays (SRI International, 1982)	
Reproductive/ developmental toxicity, rat	**LOEL maternal/LOEL fetal** rat (f) oral gavage, days 6–15 of gestation **LOEL maternal = 5 mg/kg-bw per day** **LOEL fetal = 1 mg/kg-bw per day** *maternal*: decreased weight gain; decreased body weight minus gravid uterine weight; clinical effects *fetal*: incomplete skull closure twice that of controls (Hazleton Laboratories America, Inc., 1983b) [Additional studies: Riker Laboratories, Inc., 1980; Argus Research Laboratories, Inc., 1999e,f]	**LOEL maternal/LOEL fetal** rat (f) oral gavage, days 6–17 of gestation **LOEL maternal = 10 mg/kg-bw per day** **LOEL fetal = 10 mg/kg-bw per day** *maternal*: reduced body weight gain *fetal*: reduced live fetal body weight and increased skeletal alterations, ossification alterations (Argus Research Laboratories, Inc., 1998) [Additional studies: Riker Laboratories, Inc., 1981d; Hazleton Laboratories America, Inc., 1983a, 1984]					

Endpoint	PFOS	N-EtFOSE	N-EtFOSA	N-MeFOSE	N-MeFOSA	PFOSAA	Ethanaminium...
Reproductive/ developmental toxicity, rat two-generation	**LOEL F_0/LOEL F_1/LOEL F_2:** rat (m/f) oral gavage, F_0 males: from 6 weeks before to the end of mating, F_0 females: from 6 weeks before mating through to the 21st day of lactation (DL 21), F_1 males: from 22 days after birth to the end of mating (started 90 days after birth), F_1 females: from 22 days after birth to DL 21 (for F_2) **LOEL F_0 (m) = 0.4 mg/kg-bw per day** **LOEL F_0 (f) = 1.6 mg/kg-bw per day** **LOEL F_1 (m/f) = 1.6 mg/kg-bw per day** **LOEL F_2 (m/f) = >0.4 mg/kg-bw per day** *F_0 (m)* reduced body weight gains, *F_0 (f)* reduced body weight gains during precohabitation, *F_1 (m/f)* significantly reduced litter sizes and both viability and lactation indices; reductions in development, including delayed eye opening, surface righting, pinna unfolding and air righting reflex (Argus Research Laboratories, Inc., 1999a) [Additional study: Argus Research Laboratories, Inc., 2000]	**LOEL F_0/LOEL F_1/LOEL F_2:** rat (m/f) oral gavage, F_0 males: from 28 days before to end of mating, females: from 28 days before through to the 21st day of lactation (DL 21), F_1 males from 22 days after birth to end of mating (started 90 days after birth), F_1 females: from 22 days after birth through to DL 21 (for F_2) **LOEL F_0 (m/f) = 5 mg/kg-bw per day** **LOEL F_1 (m/f) = 1 mg/kg-bw per day** **LOEL F_2 (m/f) = 5 mg/kg-bw per day** *F_0* reduced body weight gains (m/f); increased relative left testis weight; reduced duration of gestation, *F_1* reduced body weight gains (m/f), *F_2* reduced viability and lactation indices; reduced mean litter weight (Argus Research Laboratories, Inc., 1999b)					

Endpoint	PFOS	N-EtFOSE	N-EtFOSA	N-MeFOSE	N-MeFOSA	PFOSAA	Ethanaminium...
Reproductive/ developmental toxicity, rabbit	**LOEL maternal/ LOEL fetal** rabbit (f) oral gavage, days 7–20 of gestation **LOEL maternal = 1.0 mg/kg-bw per day LOEL fetal = 2.5 mg/kg-bw per day** *maternal*: reduced body weight gain over entire exposure period *fetal*: decreased ossification of sternal centres per fetus per litter; reduced body weight (Argus Research Laboratories, Inc., 1999d)	**LOEL maternal/LOEL fetal** rabbit (f) oral gavage, days 7–20 of gestation **LOEL maternal = 2.5 mg/kg-bw per day LOEL fetal = >3.75 mg/kg-bw per day** *maternal*: reduced body weight gain; increased late resorptions and abortions (Argus Research Laboratories, Inc., 1999c) [Additional study: Riker Laboratories, Inc., 1981e]	**LOEL offspring** rabbit (f) oral gavage; days 19–28 of gestation **LOEL offspring = 0.3 mg/kg-bw per day** increased neonatal mortality throughout pre-weaning period (Stump et al., 1997)				

LOEL = lowest-observed-effect level; LOAEL = lowest-observed-adverse-effect level; LC_{50} = median lethal concentration; LD_{50} = median lethal dose; m = male; f = female; bw = body weight.

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APPENDIX 1

PFOS AND RELATED SUBSTANCES

CAS No.	Chemical name	Molecular formula
N/A	1-Octanesulfonate, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-	$C_8F_{17}SO_3^-$
1691-99-2	1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-	$C_{12}H_{10}F_{17}NO_3S$
2250-98-8	1-Octanesulfonamide, N,N',N''-[phosphinylidynetris(oxy-2,1-ethanediyl)]tris[N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-	$C_{36}H_{27}F_{51}N_3O_{10}PS_3$
2795-39-3	1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, potassium salt	$C_8HF_{17}O_3S{\cdot}K$
2991-51-7	Glycine, N-ethyl-N-[(heptadecafluorooctyl)sulfonyl]-, potassium salt	$C_{12}H_8F_{17}NO_4S{\cdot}K$
4151-50-2	1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-	$C_{10}H_6F_{17}NO_2S$
24448-09-7	1-Octanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-N-methyl-	$C_{11}H_8F_{17}NO_3S$
29081-56-9	1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, ammonium salt	$C_8HF_{17}O_3S{\cdot}H_3N$
29117-08-6	Poly(oxy-1,2-ethanediyl), α-[2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl]-ω-hydroxy-	$(C_2H_4O)_nC_{12}H_{10}F_{17}NO_3S$
30381-98-7	1-Octanesulfonamide, N,N-[phosphinicobis(oxy-2,1-ethanediyl)]bis[N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, ammonium salt	$C_{24}H_{19}F_{34}N_2O_8PS_2{\cdot}H_3N$
31506-32-8	1-Octanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-methyl-	$C_9H_4F_{17}NO_2S$
25268-77-3	2-Propenoic acid, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester	$C_{14}H_{10}F_{17}NO_4S$
423-82-5	2-Propenoic acid, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester	$C_{15}H_{12}F_{17}NO_4S$
38006-74-5	1-Propanaminium, 3-[[(heptadecafluorooctyl)sulfonyl]amino]-N,N,N-trimethyl-, chloride	$C_{14}H_{16}F_{17}N_2O_2S{\cdot}Cl$
52550-45-5	Poly(oxy-1,2-ethanediyl), α-[2-[[(heptadecafluorooctyl)sulfonyl]propylamino]ethyl]-ω-hydroxy-	$(C_2H_4O)_nC_{13}H_{12}F_{17}NO_3S$
56773-42-3	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)	$C_8H_{20}N{\cdot}C_8F_{17}O_3S$
57589-85-2	Benzoic acid, 2,3,4,5-tetrachloro-6-[[[3-[[(heptadecafluorooctyl)sulfonyl]oxy]phenyl]amino]carbonyl]-, monopotassium salt	$C_{22}H_6Cl_4F_{17}NO_6S{\cdot}K$
67939-88-2	1-Octanesulfonamide, N-[3-(dimethylamino)propyl]-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, monohydrochloride	$C_{13}H_{13}F_{17}N_2O_2S{\cdot}ClH$
67969-69-1	1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-[2-(phosphonooxy)ethyl]-, diammonium salt	$C_{12}H_{11}F_{17}NO_6PS_2{\cdot}H_3N$
68298-11-3	1-Propanaminium, 3-[[(heptadecafluorooctyl)sulfonyl](3-sulfopropyl)amino]-N-(2-hydroxyethyl)-N,N-dimethyl-, hydroxide, inner salt	$C_{18}H_{23}F_{17}N_2O_6S_2$
68298-62-4	2-Propenoic acid, 2-[butyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester, telomer with 2-[butyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, methyloxirane polymer with oxirane di-2-propenoate, methyloxirane polymer with oxirane mono-2-propenoate and 1-octanethiol	$(C_{17}H_{16}F_{17}NO_4S{\cdot}C_{16}H_{16}F_{15}NO_4S{\cdot}W_{99}{\cdot}W_{99})_x{\cdot}C_8H_{18}S$

CAS No.	Chemical name	Molecular formula
68298-78-2	2-Propenoic acid, 2-methyl-, 2-[[[[5-[[[2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethoxy]carbonyl]amino]-2-methylphenyl]amino]carbonyl]oxy]propyl ester, telomer with butyl 2-propenoate, 2-[[[[5-[[[2-[ethyl[(nonafluorobutyl)sulfonyl]amino]ethoxy]carbonyl]amino]-2-methylphenyl]amino]carbonyl]oxy]propyl 2-methyl-2-propenoate, 2-[[[[5-[[[2-[ethyl[(pentadecafluoroheptyl)sulfonyl]amino]ethoxy]carbonyl]amino]-2-methylphenyl]amino]carbonyl]oxy]propyl 2-methyl-2-propenoate, 2-[[[[5-[[[2-[ethyl[(tridecafluorohexyl)sulfonyl]amino]ethoxy]carbonyl]amino]-2-methylphenyl]amino]carbonyl]oxy]propyl 2-methyl-2-propenoate, 2-[[[[5-[[[2-[ethyl[(undecafluoropentyl)sulfonyl]amino]ethoxy]carbonyl]amino]-2-methylphenyl]amino]carbonyl]oxy]propyl 2-methyl-2-propenoate, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate and 1-octanethiol	$(C_{28}H_{28}F_{17}N_3O_8S \cdot C_{27}H_{28}F_{15}N_3O_8S \cdot C_{26}H_{28}F_{13}N_3O_8S \cdot C_{25}H_{28}F_{11}N_3O_8S \cdot C_{24}H_{28}F_9N_3O_8S \cdot C_{14}H_{10}F_{17}NO_4S \cdot C_{13}H_{10}F_{15}NO_4S \cdot C_{12}H_{10}F_{13}NO_4S \cdot C_{11}H_{10}F_{11}NO_4S \cdot C_{10}H_{10}F_9NO_4S \cdot C_7H_{12}O_2)_x \cdot C_8H_{18}S$
68329-56-6	2-Propenoic acid, eicosyl ester, polymer with 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate, hexadecyl 2-propenoate, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate and octadecyl 2-propenoate	$(C_{23}H_{44}O_2 \cdot C_{21}H_{40}O_2 \cdot C_{19}H_{36}O_2 \cdot C_{14}H_{10}F_{17}NO_4S \cdot C_{13}H_{10}F_{15}NO_4S \cdot C_{12}H_{10}F_{13}NO_4S \cdot C_{11}H_{10}F_{11}NO_4S \cdot C_{10}H_{10}F_9NO_4S)_x$
68555-90-8	2-Propenoic acid, butyl ester, polymer with 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate and 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate	$(C_{14}H_{10}F_{17}NO_4S \cdot C_{13}H_{10}F_{15}NO_4S \cdot C_{12}H_{10}F_{13}NO_4S \cdot C_{11}H_{10}F_{11}NO_4S \cdot C_{10}H_{10}F_9NO_4S \cdot C_7H_{12}O_2)_x$
68555-91-9	2-Propenoic acid, 2-methyl-, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester, polymer with 2-[ethyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate and octadecyl 2-methyl-2-propenoate	$(C_{22}H_{42}O_2 \cdot C_{16}H_{14}F_{17}NO_4S \cdot C_{15}H_{14}F_{15}NO_4S \cdot C_{14}H_{14}F_{13}NO_4S \cdot C_{13}H_{14}F_{11}NO_4S \cdot C_{12}H_{14}F_9NO_4S)_x$
68555-92-0	2-Propenoic acid, 2-methyl-, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester, polymer with 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-methyl-2-	$(C_{22}H_{42}O_2 \cdot C_{15}H_{12}F_{17}NO_4S \cdot C_{14}H_{12}F_{15}NO_4S \cdot C_{13}H_{12}F_{13}NO_4S \cdot C_{12}H_{12}F_{11}NO_4S \cdot C_{11}H_{12}F_9NO_4S)_x$

CAS No.	Chemical name	Molecular formula
	propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate and octadecyl 2-methyl-2-propenoate	
68586-14-1	2-Propenoic acid, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester, telomer with 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, α-(2-methyl-1-oxo-2-propenyl)-ω-hydroxypoly(oxy-1,2-ethanediyl), α-(2-methyl-1-oxo-2-propenyl)-ω-[(2-methyl-1-oxo-2-propenyl)oxy]poly(oxy-1,2-ethanediyl), 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate and 1-octanethiol	$(C_{14}H_{10}F_{17}NO_4S·C_{13}H_{10}F_{15}NO_4S·C_{12}H_{10}F_{13}NO_4S·C_{11}H_{10}F_{11}NO_4S·C_{10}H_{10}F_9NO_4S·(C_2H_4O)_nC_8H_{10}O_3·(C_2H_4O)_nC_4H_6O_2)_x·C_8H_{18}S$
68649-26-3	1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-, reaction products with N-ethyl-1,1,2,2,3,3,4,4,4-nonafluoro-N-(2-hydroxyethyl)-1-butanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-N-(2-hydroxyethyl)-1-heptanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro-N-(2-hydroxyethyl)-1-hexanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,5-undecafluoro-N-(2-hydroxyethyl)-1-pentanesulfonamide, polymethylenepolyphenylene isocyanate and stearyl alc.	N/A
68867-62-9	2-Propenoic acid, 2-methyl-, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester, telomer with 2-[ethyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 1-octanethiol and α-(1-oxo-2-propenyl)-ω-methoxypoly(oxy-1,2-ethanediyl)	$(C_{16}H_{14}F_{17}NO_4S·C_{15}H_{14}F_{15}NO_4S·C_{14}H_{14}F_{13}NO_4S·C_{13}H_{14}F_{11}NO_4S·C_{12}H_{14}F_9NO_4S·(C_2H_4O)_nC_4H_6O_2)_x·C_8H_{18}S$
68877-32-7	2-Propenoic acid, 2-methyl-, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester, polymer with 2-[ethyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate and 2-methyl-1,3-butadiene	$(C_{16}H_{14}F_{17}NO_4S·C_{15}H_{14}F_{15}NO_4S·C_{14}H_{14}F_{13}NO_4S·C_{13}H_{14}F_{11}NO_4S·C_{12}H_{14}F_9NO_4S·C_5H_8)_x$
68891-96-3	Chromium, diaquatetrachloro[μ-[N-ethyl-N-[(heptadecafluorooctyl)sulfonyl]glycinato-O':O"]]μ-hydroxybis(2-methylpropanol)di-	$C_{18}H_{28}Cl_4Cr_2F_{17}NO_9S$
68958-61-2	Poly(oxy-1,2-ethanediyl), α-[2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl]-ω-methoxy-	$(C_2H_4O)_nC_{13}H_{12}F_{17}NO_3S$
70225-14-8	1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, compd. with 2,2-iminobis[ethanol] (1:1)	$C_8HF_{17}O_3S·C_4H_{11}NO_2$
70776-36-2	2-Propenoic acid, 2-methyl-, octadecyl ester, polymer with 1,1-dichloroethene, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate, N-(hydroxymethyl)-2-propenamide, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-	$(C_{22}H_{42}O_2·C_{14}H_{10}F_{17}NO_4S·C_{13}H_{10}F_{15}NO_4S·C_{12}H_{10}F_{13}NO_4S·C_{11}H_{10}F_{11}NO_4S·C_{10}H_{10}F_9NO_4S·C_4H_7NO_2·C_2H_2Cl_2)_x$

CAS No.	Chemical name	Molecular formula
	[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate and 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate	
71487-20-2	2-Propenoic acid, 2-methyl-, methyl ester, polymer with ethenylbenzene, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate and 2-propenoic acid	$(C_{14}H_{10}F_{17}NO_4S{\cdot}C_{13}H_{10}F_{15}NO_4S{\cdot}C_{12}H_{10}F_{13}NO_4S{\cdot}C_{11}H_{10}F_{11}NO_4S{\cdot}C_{10}H_{10}F_9NO_4S{\cdot}C_8H_8{\cdot}C_5H_8O_2{\cdot}C_3H_4O_2)_x$
92265-81-1	Ethanaminium, N,N,N-trimethyl-2-[(2-methyl-1-oxo-2-propenyl)oxy]-, chloride, polymer with 2-ethoxyethyl 2-propenoate, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate and oxiranylmethyl 2-methyl-2-propenoate	$(C_{14}H_{10}F_{17}NO_4S{\cdot}C_9H_{18}NO_2{\cdot}C_7H_{12}O_3{\cdot}C_7H_{10}O_3{\cdot}Cl)_x$
94313-84-5	Carbamic acid, [5-[[[2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethoxy]carbonyl]amino]-2-methylphenyl]-, 9-octadecenyl ester, (Z)-	$C_{38}H_{50}F_{17}N_3O_6S$
98999-57-6	Sulfonamides, $C_{7\text{-}8}$-alkane, perfluoro, N-methyl-N-[2-[(1-oxo-2-propenyl)oxy]ethyl], polymers with 2-ethoxyethyl acrylate, glycidyl methacrylate and N,N,N-trimethyl-2-[(2-methyl-1-oxo-propenyl)oxy]ethanaminium chloride	$(C_{14}H_{10}F_{17}NO_4S{\cdot}C_9H_{18}NO_2{\cdot}C_7H_{12}O_3{\cdot}C_7H_{10}O_3{\cdot}Cl)_x$
178094-69-4	1-Octanesulfonamide, N-[3-(dimethyloxidoamino)propyl]-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, potassium salt	$C_{13}H_{12}F_{17}N_2O_3S{\cdot}K$
N/A	2-(Perfluoro-N-methyl-$C_{4\text{-}8}$-1-alkanesulfonamido)ethyl esters of trimers of C_{18} unsaturated fatty acids	N/A
29457-72-5	1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, lithium salt	$C_8HF_{17}O_3S{\cdot}Li$
68909-15-9	2-Propenoic acid, eicosyl ester, polymers with branched octyl acrylate, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl acrylate, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl acrylate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl acrylate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl acrylate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl acrylate, polyethylene glycol acrylate Me ether and stearyl acrylate	$(C_{23}H_{44}O_2{\cdot}C_{21}H_{40}O_2{\cdot}C_{14}H_{10}F_{17}NO_4S{\cdot}C_{13}H_{10}F_{15}NO_4S{\cdot}C_{12}H_{10}F_{13}NO_4S{\cdot}C_{11}H_{10}F_{11}NO_4S{\cdot}C_{10}H_{10}F_9NO_4S{\cdot}(C_2H_4O)_nC_4H_6O_2{\cdot}\text{Unspecified})_x$
148684-79-1	Sulfonamides, $C_{4\text{-}8}$-alkane, perfluoro, N-(hydroxyethyl)-N-methyl, reaction products with 1,6-diisocyanatohexane homopolymer and ethylene glycol	N/A
30295-51-3	1-Octanesulfonamide, N-[3-(dimethyloxidoamino)propyl]-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-	N/A
91081-99-1	Sulfonamides, $C_{4\text{-}8}$-alkane, perfluoro, N-(hydroxyethyl)-N-methyl, reaction products with epichlorohydrin, adipates (esters)	N/A
N/A	Fatty acids, C_{18}-unsatd., dimers, 2-[methyl[(perfluoro-$C_{4\text{-}8}$-alkyl)sulfonyl]amino]ethyl esters	N/A
68081-83-4	Carbamic acid, (4-methyl-1,3-phenylene)bis-, bis[2-[ethyl[(perfluoro-$C_{4\text{-}8}$-alkyl)sulfonyl]amino]ethyl] ester	
68608-14-0	Sulfonamides, $C_{4\text{-}8}$-alkane, perfluoro, N-ethyl-N-(hydroxyethyl), reaction products with 1,1'-methylenebis[4-isocyanatobenzene]	

CAS No.	Chemical name	Molecular formula
307-35-7	1-Octanesulfonyl fluoride, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-	N/A
376-14-7	2-Propenoic acid, 2-methyl-, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester	N/A
14650-24-9	2-Propenoic acid, 2-methyl-, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester	N/A
94133-90-1	1-Propanesulfonic acid, 3-[[3-(dimethylamino)propyl][(heptadecafluorooctyl)sulfonyl]amino]-2-hydroxy-, monosodium salt	N/A
127133-66-8	2-Propenoic acid, 2-methyl-, polymers with Bu methacrylate, lauryl methacrylate and 2-[methyl[(perfluoro-C_{4-8}-alkyl)sulfonyl]amino]ethyl methacrylate	N/A
179005-06-2	Sulfonamides, C_{4-8}-alkane, perfluoro, N-[3-(dimethyloxidoamino)propyl], potassium salts	N/A
179005-07-3	Sulfonamides, C_{4-8}-alkane, perfluoro, N-[3-(dimethyloxidoamino)propyl]	N/A
ROF	Residual Organic Fluorochemicals (impurities)	N/A
1763-23-1	1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-	$C_8HF_{17}O_3S$

N/A = not available; Me = methyl; Bu = butyl