Perfluorobutanesulfonate (PFBS) and its direct precursors: Human health tier II assessment

12 December 2019

Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
1-Butanesulfonyl fluoride, 1,1,2,2,3,3,4,4,4-nonafluoro-	375-72-4
1-Butanesulfonic acid, 1,1,2,2,3,3,4,4,4-nonafluoro-	375-73-5
1-Butanesulfonic acid, 1,1,2,2,3,3,4,4,4-nonafluoro-, potassium salt	29420-49-3
1-Butanesulfonic acid, 1,1,2,2,3,3,4,4,4-nonafluoro-, anhydride	36913-91-4
1-Butanesulfonic acid, 1,1,2,2,3,3,4,4,4-nonafluoro-, ammonium salt	68259-10-9

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

The chemicals in this group are short chain perfluorinated chemicals containing four perfluorinated carbons terminating with a sulfonate group. The chemicals have the potential to hydrolyse and/or dissociate into the environmentally persistent perfluorobutanesulfonate (PFBS) anion.

These PFBS based chemicals were introduced as alternatives to the longer chain perfluoroalkyl sulfonates (PFAS) (containing carbon chain lengths of 6 or higher) including substances which may be a source of the hazardous perfluorooctanesulfonate (PFOS) anion in the environment (UNEP, 2013). As the critical health concerns for the longer chain PFAS chemicals are bioaccumulation potential and systemic long term effects (NICNASa), the focus of this assessment for the shorter chain PFBS chemicals will be on their potential for similar long term effects. Data for acute and local effects have been included where available.

The assessment of these chemicals as a group also provides additional relevant information for the risk assessment of more complex derivatives of PFBS which may degrade to this perfluorinated anion in the environment. These more complex derivatives of PFBS have been assessed separately as 'Indirect precursors of perfluorobutanesulfonate PFBS' (NICNASb).

In this assessment, the abbreviation "PFBS" is used to denote the conjugate base anion of perfluorobutanesulfonic acid (i.e. the perfluorobutanesulfonate anion). However, it is noted that this abbreviation is commonly used to refer to a range of substances which may form the anion in the environment, and also for the parent acid.

The chemicals are being re-assessed at the Tier II level under the IMAP framework following the availability of new data. Conclusions based on the new data supersede the decisions made in the previous Tier II IMAP assessment (published February 2015).

Import, Manufacture and Use

Australian

The 3M company is the primary producer of substances based on PFBS technology internationally (Poulsen, et al., 2005; UNEP, 2013). 3M have recently confirmed that they are not directly introducing the chemicals in this group to Australia.

Information collected by NICNAS in 2005 indicated that potassium PFBS was not manufactured or used in Australia. Additionally, it was reported that no PFBS derivatives were manufactured in Australia (NICNAS, 2005).

It is noted that 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

The potassium salt of PFBS (CAS No. 29420-49-3) is marketed as a flame retardant for polycarbonate resins (OECD, 2013).

The use of PFBS derivatives as an alternative to the use of PFOS has been identified for the following uses (Poulsen et al. 2005; UNEP, 2013):

- impregnation of textiles, leather and carpets;
- industrial and commercial cleaning products
- surface coatings, paint and varnish;
- oil production and mining;
- semiconductor industry; and
- electroplating.

Some of these uses are likely to be for chemicals based on PFBS 9direct and indirect precursors of PFBS) rather than the chemicals being assessed in this report.

All chemicals in this group are pre-registered under the Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) legislation. Only PFBSF (perfluorobutyl sulfonyl fluoride) has been registered under the REACH legislation as an intermediate used in the manufacture of other substances (REACH).

No specific industrial uses have been identified for PFBS anhydride.

No evidence of the presence of these chemicals in consumer products was found in available North American databases, indicating that the chemicals are not likely to be widely available for domestic uses.

Restrictions

Australian

No known restrictions have been identified.

International

No specific restrictions have been identified.

Existing Worker Health and Safety Controls

Hazard Classification

The chemicals are classified as hazardous, with the following hazard categories and hazard statements for human health in the Hazardous Chemicals Information System (HCIS) (Safe Work Australia):

Causes serious eye irritation - Cat. 2A (H319)

This classification is based on the recommended amendment to the hazard classification in the Hazardous Substances Information System (HSIS) (the Safe Work Australia online classification database at the time) from the IMAP assessment published in Tranche 12 (February 2015).

Exposure Standards

Australian

No specific exposure standards are available.

International

No specific exposure standards are available.

Health Hazard Information

The critical concern for this group of chemicals and the focus of this assessment relates to potential long-term effects following chronic low level exposure; acute effects are a secondary concern, as high level acute exposure is not expected to occur in Australia.

Toxicokinetics

In an intravenous and oral uptake study in rats, potassium PFBS (KPFBS) was absorbed rapidly when given by the oral route. Serum peak concentrations of the chemical occurred within 20 minutes after oral dosing (Olsen et al., 2009). Tissue distribution of PFBS was determined in male mice after a 1–5-day dietary exposure to environmentally relevant levels of 35S-labelled PFBS (Bogdanska et al., 2014). Following 1, 3, and 5 days of exposure, total estimated recovery of PFBS from all tissues evaluated was 10 %, 5 % and 3.4 % of the ingested dose, respectively. The declining recovery with time reflects the lack of accumulation in tissues after the first few days, with continued elimination in the urine. PFBS was detected in all tissues examined; however, the tissue levels and tissue to blood ratios of PFBS were in all cases significantly lower than those of PFOS under similar circumstances. In most cases, tissue levels plateaued after 3 days. PFBS did not accumulate in either liver or kidney. The chemical was shown to actively bind to human albumin protein in serum (94 % binding at 100 % physiological concentrations of albumin) with negligible binding to the other liver-manufactured proteins, gamma globulin, alpha globulin, fibrinogen, alpha-2-macroglobulin, transferrin and beta lipoproteins. Apparent saturation of the binding above 100 ppm in rat, human and monkey serum was observed in this study (NICNAS, 2005).

In comparison to PFOS, PFBS is eliminated by various species within shorter periods of time (OECD, 2013). Based on the available data for KPFBS, the chemicals in this group appear to be rapidly excreted by kidneys; with up to 85 % excreted in the

urine within 24 hours post dosing. Using urine elimination data, the mean half-life values of PFBS for male and female rats were determined to be 3.1 and 2.4 hours, respectively (Chengelis et al., 2009). In monkeys, the half-lives, following intravenous administration at 10 mg/kg body weight, were determined to be 13 hours for males and 11 hours for females (Olsen et al., 2009).

Humans have a longer serum elimination half-life than both rodents and monkeys. Olsen et al. (2009) evaluated the elimination of human serum KPFBS in a group of workers with occupational exposure, with serum concentrations measured up to 180 days after cessation of further KPFBS work-related activity. Among the six subjects (five male, one female), the geometric mean serum elimination half-life for KPFBS was 25.8 days (95 % confidence interval = 16.6–40.2 days). Urine appeared to be a major route of elimination based on observed levels of PFBS in urine in the human study.

Study data from the United States suggest that PFBS was below the detection limits (0.01 ng/mL) in human blood samples from participants from the National Health and Nutrition Examination Survey (NHANES) conducted by the Centers for Disease Control and Prevention's (CDC's) National Center for Health Statistics collected between 2003-2010 (CDC, 2014). PFBS has been detected at low levels in studies targeting subpopulations in some countries (Glynn et al., 2012; Bao et al., 2014). A trend in increased levels of PFBS in blood samples from primiparous women in Sweden taken between 1996 and 2010 has been reported. However, levels in 2010 were still <0.1 ng/mL. In a study in the blood serum of Taiwanese children between 2009-2010, PFBS was reported in the range 0.07 to 0.86 ng/mL with a median concentration of 0.48 ng/mL (Bao et al., 2014). No data are available on the levels of PFBS in the Australian population.

Acute Toxicity

Oral

Limited data are available for the chemicals. Based on the data available for KPFBS, the chemicals in this group are not considered to be acutely toxic following oral exposure.

KPFBS has low acute toxicity via the oral route with an oral median lethal dose (LD50) >2000 mg/kg bw in rats. A single oral dose was administered by gavage to fasted animals. No treatment-related adverse clinical observations, mortality, changes in body weight or gross pathology were found (NICNAS, 2005).

Dermal

Based on the data available for potassium PFBS and PFBSF, the chemicals in this group are not considered to be acutely toxic following dermal exposure.

Potassium PFBS and PFBSF have low acute toxicity via the dermal route, with dermal LD50 values >2000 mg/kg bw. A single dose of 2000 mg/kg bw of each chemical was administered to Sprague Dawley rats under semi-occlusive conditions for 24 hours. There were no clinical signs of reaction to treatment, nor dermal response to treatment observed in any animal throughout the studies (NICNAS, 2005; REACH).

Inhalation

Based on the available data, inhalation effects were observed at relatively high doses and classification is; therefore, not considered warranted.

Sprague Dawley (SD) rats were exposed (nose only) to vapours of PFBSF for 4 h at 1000, 5000 or 15000 ppm. Exposure of animals in the 15000 ppm group was ceased after a short period of time for humane reasons, due to the nature and severity of the clinical signs (restless behaviour, vocalisation, convulsions and pronounced jumping, resulting in protrusion of snout/limbs and entrapment of snouts through the bars of the confinement cage). No mortality was observed at lower dose levels. Clinical signs of toxicity seen during exposure at 1000 and 5000 ppm concentrations included exaggerated breathing and decreased breathing rate, restless behaviour followed by reduced motor activity, and sudden movements characterised by pronounced jumping. A median lethal concentration (LC50) was not determined but is considered to be >5000 ppm (62 mg/L) (REACH).

No information on acute inhalation toxicity is available for KPFBS.

Corrosion / Irritation

Skin Irritation

Potassium PFBS was not irritating to rabbit skin when applied as a single dose of 500 mg for four hours under occlusive conditions. No signs of erythema or oedema were observed at 60 minutes or at 24, 48 and 72 hours (NICNAS, 2005).

Irritation studies for PFBSF were not available; however, no signs of erythema or oedema were observed in the acute dermal toxicity study when PFBSF was administered under semi-occlusive conditions for 24 hours (see **Dermal Acute Toxicity** section).

Potassium PFBS is expected to be less irritating than PFBS (which is strongly acidic) and PFBSF and PFBS anhydride (which are reactive and could hydrolyse to PFBS); therefore, skin irritant/corrosive effects cannot be ruled out for these members of the group.

Eye Irritation

The chemicals are classified as hazardous with the risk phrase 'Causes serious eye irritation' - Cat. 2A (H319) in HSIS (Safe work Australia). The available data support this classification.

Potassium PFBS was found to be an irritant to rabbit eyes treated with 80 mg of the chemical for 24 hours. Corneal opacity, iritis and redness and oedema of the conjunctivae were observed at 1, 24, 48 and 72 hours post treatment. By day 21, the scores had reversed in all but one animal (NICNAS, 2005).

Eye irritant or corrosive effects cannot be ruled out for PFBS (which is strongly acidic) and PFBSF and PFBS anhydride (which are reactive and could hydrolyse to PFBS), while the ammonium salt is expected to have similar effects to the potassium salt. Therefore, in the absence of data, the above classification is deemed appropriate for the entire group. If data on the irritation potential become available for the individual group members, these should be used to determine individual classifications.

Sensitisation

Skin Sensitisation

Based on the negative results observed for potassium PFBS in a guinea pig maximisation test (GPMT) and for PFBSF in a local lymph node assay (LLNA), the chemicals in this group are not considered skin sensitisers.

Potassium PFBS showed no evidence of skin sensitisation in a GPMT with an intradermal induction of 12.5 mg and topical induction of 50 mg KPFBS. The rats were challenged with a topical application of 166 mg KPFBS (NICNAS, 2005).

PFBSF was non sensitising in the LLNA when tested at concentrations of 10, 25 or 50 % (weight/volume). It was also negative in a GPMT (REACH).

Repeated Dose Toxicity

Oral

Based on the data available, the chemicals in this group are not considered to cause serious systemic effects from repeated oral exposure.

In a 28-day repeat dose study (NTP, 2019) SD rats (10/sex/dose group) were administered KPFBS in deionised water with 2 % Tween 80 by gavage, for 28 days at doses of 0, 62.6, 125, 250, 500, or 1000 mg/kg bw/day. Control animals received the vehicle only. Nine male and eight female rats died from day 15 to day 25 in the 1000 mg/kg bw/day group. These deaths were considered treatment-related, but the cause of death was unknown. Hepatocellular necrosis was observed in the early death animals administered 1000 mg/kg bw/day, but not in lower dose rats. There was no clinical pathology interpretation for the 1000 mg/kg/day male and female rats due to the high mortality in these groups.

In rats that survived till the end of study, mean body weights were slightly reduced at the highest dose. Significant dose-related increases were observed in relative liver weight in 62.5 mg/kg bw/day males and absolute and relative liver weights in the 125, 250, and 500 mg/kg bw/day males and females. The liver weight changes appeared to correlate with the observed histopathological changes. The incidence of hepatocyte hypertrophy were significantly increased in males at 125 mg/kg bw/day and higher doses and in females only at high doses. Hepatocyte cytoplasmic alterations were observed in both sexes at high doses (500 and 1000 mg/kg bw/day). The severity of these changes was dose-dependent. Slight increases in hepatocellular injury biomarker enzymes, alkaline phosphatase (ALP), alanine aminotransferase (ALT), and aspartate aminotransferase (AST) activities were observed in the 500 mg/kg bw/day groups. Bilirubin concentrations were significantly increased in male and female rats treated with 500 mg/kg bw/day. There were consistent decreases in cholesterol concentrations in males and to a lesser extent in females (high dose only). Decreases in triglyceride concentration were observed in treated males. These changes are consistent with the known effects of PPARa activation on lipid metabolism. Increased expression of Acox1, Cyp4a1, Cyp2b1, and Cyp2b2 in treated rats, compared to the controls, was observed indicating increased peroxisome proliferator-activated receptor alpha (PPARa) and CAR activity. Significant decreases in free T4, total T4, and total T3 levels occurred in a dose-response manner in all male and female dose groups. In males, at 0, 62.5, 125, 250 and 500 PFBS mg/kg bw /day, the reported total T3 was 117.7, 87.8, 64.5, 60.2 and 50.5 ng/100 mL, respectively; reported free T4 was 2.1, 0.64, 0.32, 0.3 and 0.3 ng/100 mL, respectively; and total T4 was 3.34, 0.90, 0.22, 0.1 and 0.29 ng/100 mL, respectively. In females, at 0, 62.5, 125, 250 and 500 PFBS mg/kg bw /day, the reported total T3 was 89.3, 61.8, 61.5, 52.4 and 51.3 ng/100 mL, respectively; the reported free T4 1.54, 0.72, 0.55, 0.48 and 0.36 ng/100 mL, respectively; and reported total T4 was 3.1, 1.48, 1.12, 0.90 and 0.97 ng/100 mL, respectively. TSH levels were unchanged.

Male kidney weights (absolute and relative to body weight) were increased in the 500 mg/kg/day group. In females, dose-related and significant increases in the relative right kidney weights occurred in all the dose groups. Absolute and relative heart and thymus weights were significantly decreased in males and females dosed with 500 mg/kg bw/day PFBS. The incidence of bone marrow hypocellularity, although of mild to marked severity, was significantly higher in rats (both sexes) treated with 1000 mg/kg bw/day PBFS.

Slight but significant increases in blood urea nitrogen concentrations were observed in the 250 and 500 mg/kg bw/day male rats. Globulin concentrations were decreased in all male dose groups, resulting in increased albumin/globulin ratio. Slight decreases in the male rat erythrocytes were observed, most consistently in those administered 125 mg/kg bw/day and higher doses. This was characterised by significantly decreased haematocrit values, haemoglobin concentrations, and erythrocyte and reticulocyte counts compared to those of the control group. In female rats, the reticulocyte count was significantly decreased in 125 mg/kg bw/day and higher dose groups.

Olfactory epithelium degeneration increased in a dose dependent manner in both sexes at 250, 500, and 1000 mg/kg bw/day PFBS, but necrosis of the epithelium was observed only at highest dose. Incidences of thymus atrophy and minimal to mild epithelium hyperplasia of the forestomach were significantly higher in the 1000 mg/kg bw/day group males. The incidence of papilla necrosis (minimal to mild severity) in kidneys was significantly increased in 1000 mg/kg bw/day females.

Females administered with 250 or 500 mg/kg bw/day PFBS displayed alteration in the oestrous cycle (extended diestrus in the 250 mg/kg bw/day group and irregular or not cycling in the 500 mg/kg bw/day females).

A NOAEL was not established in this study, as effects on thyroid hormones and liver and kidney weights were observed at all dose levels.

In a 28-day study with potassium PFBS in SD rats (0, 100, 300 and 900 mg/kg bw/day), a NOAEL was established as 300 mg/kg bw/day in females and males based on significant increase in kidney and liver weights, respectively, in animals that received 900 mg/kg bw/day. No histopathological findings were reported (NICNAS, 2005). At this dose, changes in serum phosphorous and potassium were not considered adverse.

In a 90-day oral repeat dose study (York, 2003a), Crl:CD®(SD)IGS BR V AF/Plus® rats (10 rats/sex/group) were administered 0, 60, 200 or 600 mg/kg bw/day PFBS, or aqueous 0.1 % carboxymethylcellulose (vehicle), once daily by gavage for at least 90 days. Observations for clinical signs were made daily and detailed clinical observations were conducted for all male and female rats. A functional observational battery and motor activity assessment were conducted on five male and five female rats per group.

Histological examination was performed on all tissues from the 0 and 600 mg/kg bw/day groups and also on the nasal cavities and turbinates, stomachs and kidneys of the male and female rats in the 60 and 200 mg/kg bw/day dosage groups.

All animals survived until scheduled sacrifice and appeared normal at scheduled sacrifice. Discharge of a pigmented secretion from the nose occurred in two and three male rats in the 200 and 600 mg/kg/day dosage groups, respectively. All other clinical observations were considered unrelated to the test substance. There were no significant or biologically important differences between treated and control rats in the measures of motor activity or functional observational battery (FOB).

Absolute and relative spleen weights were significantly reduced in the male rats in the 60, 200 and 600 mg/kg/day dose groups. No organ weights were affected in females at any dose level. Average values for red blood cells, haemoglobin concentration and haematocrit were significantly reduced in male rats in the 200 and 600 mg/kg/day dosage groups. Total protein and albumin values were significantly reduced in females in the 600 mg/kg/day dosage group. Serum chloride was significantly higher in male rats in the 600 mg/kg bw/day group, but all other clinical chemistry parameters were unaffected in either sex at doses of PFBS as high as 600 mg/kg/day.

Increased incidences of hyperplasia of the epithelial cells of the medullary and papillary tubules and ducts in the inner medullary region were observed in kidneys of male and female rats dosed 600 mg/kg bw/day group. Necrosis of individual squamous epithelial cells in the limiting ridge of the forestomach was also observed in these rats.

A NOAEL of less than 60 mg/kg bw/day was established in male rats, based on significant reductions in absolute and relative spleen weights at all dose levels, and reductions in red blood cells, haemoglobin concentration and haematocrit at 200 and 600 mg/kg bw/day. The NOAEL for female rats was 600 mg/kg/day based on significant reductions in average total protein and albumin values.

In a 90-day oral repeat dose study (Lieder et al., 2009a), SD rats were dosed by gavage with KPFBS) at doses of 60, 200 and 600 mg/kg bw/day. No treatment-related mortality, bodyweight changes or neurological effects were reported. All rats appeared normal at sacrifice. Chromorhinorrhea (perioral) and urine-stained abdominal fur were observed in males at 600 mg/kg bw/day. All other clinical observations were considered unrelated to the test substance, because the incidences were not dose dependent and/or the observations only occurred in one rat in a group. Red blood cell counts, haemoglobin and haematocrit values were reduced in males receiving 200 and 600 mg/kg bw/day; however, there were no adverse histopathological findings in bone marrow. Total protein and albumin were lower in females at 600 mg/kg bw/day. There were no significant changes in clinical chemistry in either sex.

Microscopic changes were observed in the kidneys and stomach of the male and female rats of the 600 mg/kg bw/day dose group. In stomachs, increased incidence of necrosis of individual squamous epithelial cells in the limiting ridge of the forestomach was noted. This change was characterised by individual squamous epithelial cells with dark pyknotic nuclei surrounded by a clear cytoplasmic halo. These effects were likely due to accumulative direct irritation resulting from oral dosing of KPFBS. In kidneys, minimal to mild hyperplasia of the epithelial cells of the medullary and papillary tubules and of the ducts in the inner medullary region were observed. These results are likely due to a response to high concentration of KPFBS in tubules and ducts and represent a minimal-to-mild effect. There were no corresponding changes in kidney weights, and clinical chemistry parameters related to kidney function were unchanged. Microscopic changes of an equivocal and uncertain nature were observed in the nasal mucosa and were likely attributable to the route of dosing (oral gavage). The NOAEL from the study was 200 mg/kg bw/day based on the increased incidence of renal hyperplasia in males and females.

Dermal

No data are available for the chemicals in this group.

Inhalation

Based on the limited data available, the chemicals are not expected to cause serious damage to health from repeated inhalation exposure.

In a sub-chronic study, Crl:CD SD rats were exposed (whole body) to vapours of PFBSF for 6 hours/day, 5 days/week for 4 weeks. The mean analysed concentrations of the chemical were 47, 162 and 459 ppm. Clinical signs observed immediately post exposure included vocalising and agitation when handled, walking on toes (abnormal gait) and hyperactivity, consistent with a neurotoxic effect. These signs were transient and generally resolved the following day with no evidence of sustained neurotoxicity. There were

no effects on haematological or blood chemistry parameters. Histopathological examination of the respiratory tract revealed no treatment-related findings (REACH).

Genotoxicity

Based on the results from negative in vitro genotoxicity studies, the chemicals in this group are not considered to be genotoxic.

Potassium PFBS was not mutagenic (up to 5000 µg/plate) to *Salmonella typhimurium* (TA98, TA100, TA1535 and TA1537 strains) or *Escherichia coli* (WP2 uvrA strain) with or without metabolic activation. The chemical was negative (up to 5000 µg/mL) in a chromosomal aberration test in Chinese hamster ovary W-B1 cells with or without metabolic activation (NICNAS, 2005).

In a genotoxicity study reported by the US National Toxicity Program (NTP 2019), PFBS (concentration range of 50 to 5,000 µg/plate) was judged to be equivocal in *S. typhimurium* strain TA98 due to a mix of negative and positive results that were observed both in the presence and absence of S9. No mutagenic activity was seen in *S. typhimurium* strain TA100 using a similar concentration range or in *E. coli* strain WP2 uvrA/pKM101 (concentration range of 50 to 1,000 µg/plate).

No increases in the frequencies of micronucleated erythrocytes were observed in peripheral blood samples from male or female rats administered PFBS (concentration range of 62.6 to 500 mg/kg bw/day) for 28 days. However, PFBS caused significant, dose-dependent decreases in the percentage of PCEs in the peripheral blood of both sexes, as did PFHxSK in males, suggesting that the bone marrow was a target for cytotoxicity induced by these chemicals (NTP 2018).

PFBSF was negative (with and without metabolic activation) in Ames test (up to 5000 µg) in both S. *typhimurium* (TA135, TA 1537, TA98 and TA100 strains) and E. coli (WP2uvrA/pKM101 strain), and in a chromosomal aberration test using human lymphocytes (REACH).

In a genotoxicity study (NTP 2019), equivocal results were obtained with PFBS (concentration range of 50 to 5,000 µg/plate) in *S. typhimurium* strain TA98 in the presence and absence of S9. No mutagenic activity was seen in *S. typhimurium* strain TA100 using a similar concentration range or in *E. coli* strain WP2 uvrA/pKM101 (concentration range of 50 to 1,000 µg/plate).

Chromosomal damage in erythrocyte precursor cells in the peripheral blood was determined in male and female rats from the 28-day oral repeat dose study (NTP, 2018). No increases in the frequencies of micronucleated erythrocytes (either immature or mature) were observed in peripheral blood samples from male or female rats administered PFBS (concentration range of 62.6 to 500 mg/kg/day), PFBS caused significant, dose-dependent decreases in the percentage of PCEs in the peripheral blood of both sexes, indicating that the chemical interacted with the bone marrow.

Carcinogenicity

No data are available for the chemicals in this group. Whilst PFOS is classified as hazardous with the risk phrase Carc. Cat 3 - Limited evidence of a carcinogenic effect (Xn; R40) (NICNASa), histopathological changes (both neoplastic and non-neoplastic) observed in 90-day studies with PFOS at the sites of tumour formation were not observed in studies using PFBS (see **Oral Repeat Dose Toxicity** section).

Reproductive and Developmental Toxicity

Based on the available data on potassium PFBS, the chemicals are not considered to cause reproductive or developmental toxicity.

In a two-generation reproduction study, conducted according to OECD TG 416, Sprague Dawley rats (30 rats/sex/dose group) were orally administered daily dosages of 0, 30,100, 300 or 1000 mg/kg bw KPFBS in 0.1 % carboxymethyl cellulose (vehicle) (Lieder et al., 2009b). The dosing was started at least 70 days prior to and through mating (males and females), as well as during gestation and lactation (females only). First generation (F1) pups were dosed similarly, beginning at weaning. Second generation (F2) pups were not directly dosed but potentially exposed to PFBS through placental transfer and nursing. The study was terminated 3 weeks after the birth of F2 pups. The endpoints evaluated included body weight, food consumption, clinical signs, oestrus cycling, sperm quality, fertility, gestation indices, litter outcomes, and developmental landmarks.

In male parental generation rats, liver weights (absolute and relative to body weight and brain weight) were significantly increased in the 300 and 1000 mg/kg/day dose groups and mild hepatocellular hypertrophy was observed in these rats. No liver hypertrophy was observed in P-generation female rats. In kidneys of male and female rats of the 300 and 1000 mg/kg bw/day groups, minimal to moderate hyperplasia of the tubular and ductular epithelium of the inner medulla/papilla was observed. One incident of moderate focal papillary necrosis in males at 1000 mg/kg bw/day and three incidents of minimal-to-moderate focal papillary necrosis in females were observed in the 300 mg/kg/day dose group; although none were observed in the 1000 mg/kg/day dose group. There were no significant changes in other organ weights. No changes in male or female sex organs or reproductive parameters were noted.

In F1-generation males, terminal body weights and body weight change from weaning to termination were significantly reduced for the 1000 mg/kg bw/day males. The changes in the liver consisted of 3 and 14 incidences of mild hepatocellular hypertrophy in the 300 and 1000 mg/kg/day dose groups, respectively. Liver cells were enlarged due to an increased amount of finely granular eosinophilic cytoplasm. In kidneys, increased incidence and severity of hyperplasia of the tubular and ductular epithelium of the inner medulla/papilla area were observed in the 1000 mg/kg/day dose group.

In F1-generation females, treatment-related microscopic changes in kidneys in the 300 and 1000 mg/kg/day groups were similar to those observed in the respective P-generation females. The changes consisted of 13 and 15 incidences (at 300 and 1000 mg/kg/day, respectively) of minimal to moderate severity of hyperplasia of the tubular and ductular epithelium of the inner medulla/papilla. There were also seven and four incidences of minimal to mild focal papillary oedema in the 300 and 1000 mg/kg/day dose groups, respectively. All other organ weights and ratios of organ-to-body weight and organ-to-brain weight were similar to control values.

Pups (F2) had normal body weights. There were no abnormal clinical or necropsy observations for F2-generation pups from F1 maternal rats given KPFBS. Increases in some organ weights were observed, but they were not considered treatment-related.

An NOAEL of 100 mg/kg bw/day for both parental generation and F1 offspring was established based on hyperplasia of the tubular and ductular epithelium of the inner medulla/papilla in rats at doses of 300 mg/kg bw/day and above.

In a two-generation reproductive toxicity study, reported in NICNAS (2005), male and female rats were administered 0, 30, 100, 300 or 1000 mg/kg bw/day, at least 70 days prior to and through mating (males and females), as well as during gestation and lactation (females only). There was no evidence of adverse effects on reproduction, fertility or lactation. The NOAEL for reproductive toxicity in parental and first generation (F1) rats was 1000 mg/kg bw/day. The NOAEL for systemic toxicity in the parental and F1 rats was 100 mg/kg bw/day based on treatment-related microscopic changes in the kidney and liver at 300 mg/kg bw/day. No adverse effects were observed in second generation (F2) pups in doses as high as 1000 mg/kg bw/day.

In a developmental study, pregnant Crl:CD®(SD)IGS BR V AF/Plus® rats (8/group), (York, 2003b) were administered 0 (vehicle), 100, 300, 1000 and 2000 mg/kg bw/day PFBS in suspension orally (gavage) on gestation days 6 through 20 (GDs 6 through 20). All rats were sacrificed on GD 21 and examined for the number and distribution of corpora lutea, implantation sites and uterine contents. The gravid uterus was excised and weighed. Gross necropsy of the thoracic, abdominal and pelvic viscera was performed. Foetuses were weighed and examined for gross external alterations and sex. All rats survived to scheduled sacrifice. Clinical observations were limited to urine stained abdominal fur in four rats in the 2000 mg/kg bw/day dosage group. No additional gross lesions were identified. Rats in the 2000 mg/kg/day dosage group lost body weight on GDs 6 to 9. Gravid uterine weights were reduced (15.4 %) for the rats in the 2000 mg/kg bw/day dosage group, compared to the control group.

There were no dead foetuses or late resorptions, and no dams had whole litter resorptions. All placentae appeared normal. No foetal gross alterations were identified. Male and female foetal body weights were reduced (12.2 % and 13.0 %, respectively), in the 2000 mg/kg bw/day dose group, as compared with the control group. There were no other test substance-related effects on the following parameters evaluated at Caesarean-sectioning: litter averages for corpora lutea, implantations, litter sizes, live foetuses, early resorptions, percent resorbed conceptuses and percent live male foetuses. A NOAEL was not established in this study.

A pre-natal developmental toxicity study in rats, administered 0, 100, 300 or 1000 mg/kg bw/day PFBS by gavage on gestation days 6–20, had a NOAEL of 300 mg/kg bw/day for maternal toxicity based on reduced body weight gains and food consumption at 1000 mg/kg bw/day and a NOAEL of 1000 mg/kg bw/day for developmental toxicity (NICNAS, 2005).

Effects of feeding PFBS to pregnant dams on the growth and development of female offspring was studied in ICR mice (Feng et al., 2017). Pregnant mice (30 dams/dose group) were administered PFBS by gavage at doses of 50, 200 or 500 mg/kg bw/day from gestation day 1 (GD1) to GD20. Controls received an equivalent volume of vehicle (0.1% carboxymethyl cellulose).

Administration of PFBS led to a dose-dependent increase in serum levels of PFBS (1.75, 74, 332 and 721 ng/ml at 0, 50, 200 and 500 mg/kg bw/day PFBS respectively), measured at GD20. Reduced total T4, total T3 and free T4 levels, and increased TSH

levels were noted in dams in the 200 and 500 mg/kg bw/day groups at GD20, indicating that exposure to PFBS during gestation reduces maternal thyroid hormone levels, and possibly leads to hypothyroxinemia in neonates (see below).

All offspring from treated dams (PFBS-offspring) were born alive and the numbers of offspring from parental dams were not significantly different from those in the control group. Body weights of postnatal day 1 (PND1) female offspring in the 200 and 500 mg/kg bw/day groups were slightly lower than those in the control group, and these remained underweight throughout the neonatal (PND1), pubertal (PND30) and adult (PND60) stages.

Offspring from dams at 200 and 500 mg/kg bw/day had significantly reduced serum total T3 and T4 levels at postnatal day (PND) 1, 30 and 60 (PND1, PND30 and PND60 stages), compared to offspring from control dams. However, serum TSH and hypothalamic Trh mRNA levels in these two dose groups were elevated only at PND30 (but not at PND1 or PND60) when compared with control offspring. The percentage decrease in T4 levels at PND60 (23 %) was less than that at PND30 (42%).

Slight but significant delays (approximately 1.5–2 days) in eye opening were observed in the 200 and 500 mg/kg bw/day PFBS-offspring compared with those from control mice. These offspring also exhibited delayed vaginal opening, and their first oestrus was delayed for up to 5 days.

The size of ovaries in the female offspring of treated dams were smaller at PND60, and the relative weights were lower than those of control offspring. These changes were; however, not dose dependent. The offspring from treated dams showed fewer primordial follicles, primary follicles, secondary follicles, early antral follicles, antral follicles and pre-ovulatory follicles, as well as fewer corpora lutea than control offspring at dioestrus. Decreases in uterine size and weight, compared with controls were noted, and total uterine section diameter and endometrial and myometrial thickness were also significantly reduced. These changes were not dose dependent, with the 500 mg/kg bw/day offspring showing similar changes to the offspring from 200 mg/kg bw/day group.

To explore the endocrine pathways underlying PFBS-induced impairments in growth and development and pubertal onset, the hypothalamic–pituitary–gonadal hormone levels in neonatal, pubertal and adult dioestrus PFBS-offspring were examined. Compared with control offspring, the 200 and 500 mg/kg bw/day exhibited reduced serum oestrogen (E2) levels at postnatal days 30 and 60. These changes were not dose dependent as there were no differences in E2 levels between the two dose groups. A slight increase in the level of serum LH was observed in the 200 and 500 mg/kg bw/day offspring only at PND30 but not at PND60. The levels of hypothalamic GnRH in 200 and 500 mg/kg bw/day PFBS-offspring did not differ significantly from those in control offspring. At PND60, the 200/500 mg/kg bw/day PFBS-offspring exhibited lower serum progesteron levels at dioestrus than the control offspring.

An NOAEL of 50 mg/kg bw/day was established based on decreased T3, free T4, and delayed eyes opening, vaginal opening, and first oestrous in pups from treated dams.

Zhou et al., (2016) investigated the relationship between serum PFASs and reproductive hormone concentrations in Taiwanese adolescents. In the cross-sectional design study, a sample of 225 healthy adolescents (102 boys and 123 girls, aged 13–15 years), was selected from seven public schools in the Taipei City of Northern Taiwan. Information regarding demographic characteristics such as age, sex, parental education, environmental tobacco smoke (ETS) exposure and exercise was collected via a self-reported questionnaire.

Serum samples were collected for each child after 8 h of fasting. Reproductive hormones, testosterone and oestradiol, were measured in 50 µL serum by immunoluminometric assay and ten PFASs (PFBS, perfluorohexanesulfonate (PFHxS), perfluorooctanesulfonate (PFOS), perfluorohexanoic acid (PFHxA), perfluoroheptanoic acid (PFHpA), perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDA), perfluorododecanoic acid (PFDoA), and perfluorotetradecanoic acid (PFTA) were measured in 0.5 mL of serum using high-performance liquid chromatography (HPLC).

Analysis of data indicated that long chain PFASs, especially PFOS and PFTA, represented the highest concentrations of PFASs in serum; with median levels of 29.9 ng/mL and 6.0 ng/mL, respectively in boys, and median levels of 28.8 ng/mL and 4.5 ng/mL, respectively in girls. There were no significant differences in PFAS concentrations between male and female adolescents.

After adjustment for confounding factors, no significant association between serum PFASs and reproductive hormone was found, except for PFNA with ln(oestradiol) levels. When stratified by sex, serum PFOS, PFDA, PFHxA, and PFNA in males were negatively associated with ln(testosterone) levels. No significant association between serum PFBS and sex hormones was noted in male or female subjects. It was noted that the median concentrations of PFOS in adolescents in the study were much higher than average concentrations reported in Australian children in age group 5–15 years measured in 2010–11 (Toms et al., 2009).

Risk Characterisation

Critical Health Effects

The critical health effect for risk assessment is eye irritation. Potassium PFBS is expected to be less irritating than PFBS (which is strongly acidic) and PFBSF and PFBS anhydride (which are reactive and could hydrolyse to PFBS); therefore, corrosive effects cannot be ruled out for these members of the group.

Data available indicate that PFBS has a more favourable toxicological profile and bioaccumulation potential than the long-chain perfluoroalkyl substances. Chronic low level effects on human health have not been identified.

The U.S. Environmental Protection Agency (USEPA) has recently derived sub-chronic and chronic oral reference doses (RfDs) for perfluorobutane sulfonic acid (PFBS). The basis for the derivation of oral RfDs was developed following a review of 37 toxicity studies selected for their relevance to PFBS effects on human health.

Thyroid, kidney and developmental endpoints were identified as potential health effects from repeated exposures in utero and/or during adulthood. Perturbation of thyroid hormone levels and kidney histopathology (papillary epithelial tubular/ductal hyperplasia) were used as endpoints for derivation of candidate sub-chronic and chronic oral RfDs.

However, relevance of these effects to human situations is not known. The USEPA review concluded that the number of human studies was too small to draw conclusions about possible associations between PFBS exposure and potential health outcomes such as alteration of menstruation, reproductive hormones or semen parameters, kidney function, lung function and lipid profile. Of the examined outcomes, only asthma, serum cholesterol, and high-density lipoprotein levels were found to exhibit a significant positive association with PFBS exposure.

In the rodent studies PFBS affected thyroid hormone levels and kidney histopathology only at high doses. The lowest doses of PFBS at which effects on T4 levels and kidney hyperplasia were observed (LOAEL) were 200 mg/kg bw/day and 300 mg/kg bw/day, respectively. Serum PFBS measured in the general population in several studies was very low. In the American Red Cross samples collected in 2015, only 8.4 % had a quantifiable serum PFBS concentration; the majority of samples were below the lower limit of quantitation (4.2 ng/mL) (Olsen et al., 2017). The 2013–2014 National Health and Nutrition Examination Survey (NHANES) data reported the 95th percentile for PFBS at or below the level of detection (0.1 ng/mL) (Ye et al., 2018).

The chemical, PFBS, has a very short half-life in animals and humans. The mean serum elimination half-life in workers with occupational exposure to PFBS was 27.7 days compared to PFOS and PFOA whose half-lives are 8.7 years and 3.8 years, respectively.

Public Risk Characterisation

Use in consumer products

Based on the available use information, the chemicals are not likely to be available for domestic or cosmetic uses. Hence, the public risk from direct use of these chemicals is not considered to be unreasonable

Secondary exposure via the environment

Public exposure to PFBS could occur through secondary exposure via the environment. It is noted that 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. The chemicals in this group have been found to be highly persistent, and environmental levels may continue to increase over time due to release from these sources (NICNASc).

Data available indicate that PFBS has a more favourable toxicological profile and bioaccumulation potential than the long-chain perfluoroalkyl substances. Chronic low level effects on human health have not been identified. Should hazard data become available indicating adverse health effects, further assessment of the chemicals in this group may be necessary to inform the risk of secondary exposure to PFBS.

Occupational Risk Characterisation

Based on the available use information, the chemicals are not likely to used in significant quantities in Australia. Therefore, the chemicals are not considered to pose an unreasonable risk to the health of workers. Based on the available data, the hazard

classification in the HCIS (Safe Work Australia) is considered appropriate for all the chemicals in the group; however, corrosive effects for PFBS (acid), PFBSF and PFBS anhydride cannot be ruled out. If these chemicals are introduced into Australia, adequate control measures to minimise dermal and ocular exposure to the chemicals should be implemented.

NICNAS Recommendation

Current risk management measures are considered adequate to protect public and workers' health and safety, provided that all requirements are met under workplace health and safety, and poisons legislation as adopted by the relevant state or territory.

However, should hazard data become available indicating adverse health effects, further assessment of the chemicals in this group may be necessary to inform the risk of secondary exposure to PFBS.

Regulatory Control

Work Health and Safety

The chemicals are recommended for classification and labelling aligned with the Globally Harmonized System of Classification and Labelling of Chemicals (GHS) as below. This does not consider classification of physical hazards and environmental hazards.

Whilst data were not available to classify PFBS (acid), PFBSF and PPFBS anhydride for any possible corrosive effects, these cannot be ruled out. If data on the irritation potential become available for the individual group members, this should be used to determine individual classifications.

From 1 January 2017, under the model Work Health and Safety Regulations, chemicals are no longer to be classified under the Approved Criteria for Classifying Hazardous Substances system.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Irritation / Corrosivity	Not Applicable*	Causes serious eye irritation - Cat. 2A (H319)*

[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 industry

Control measures to minimise the risk from ocular and dermal 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 chemical is used. Examples of control measures which may minimise the risk include, but are not limited to:

- 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 chemical.

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 assist with meeting 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 chemical 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 the chemicals has not been undertaken as part of this assessment.

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Last Update 12 December 2019

Chemical Identities

Chemical Name in the Inventory and Synonyms	**1-Butanesulfonyl fluoride, 1,1,2,2,3,3,4,4,4-nonafluoro-** 1,1,2,2,3,3,4,4,4-nonafluoro-1-butanesulfonyl fluoride 1-butanesulfonyl fluoride, 1,1,2,2,3,3,4,4,4-nonafluoro- perfluorobutanesulfonyl fluoride PFBSF
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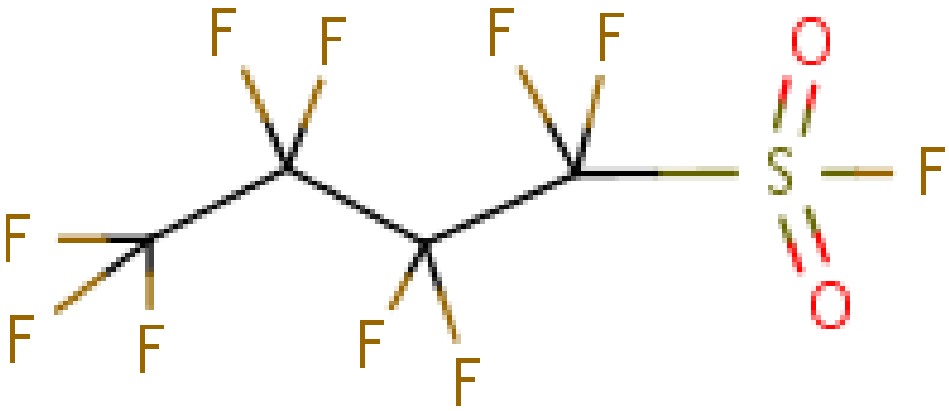
Structural Formula	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Molecular Weight	302.1

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	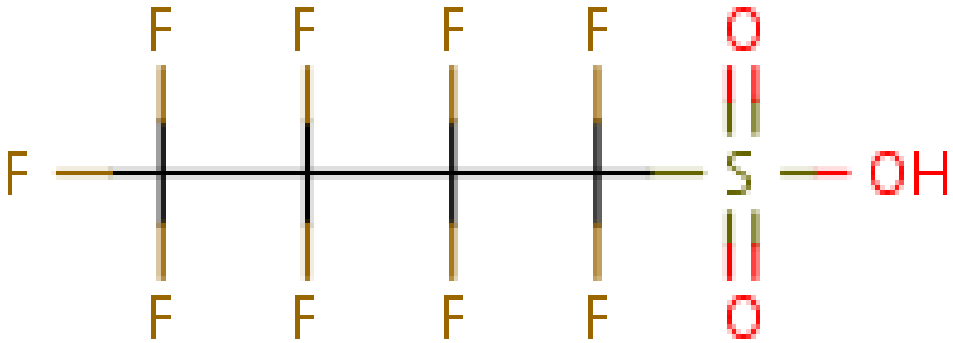
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	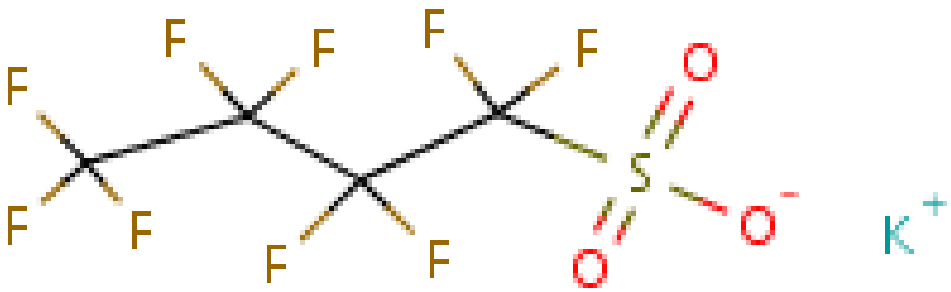
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Molecular Weight	338.2

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	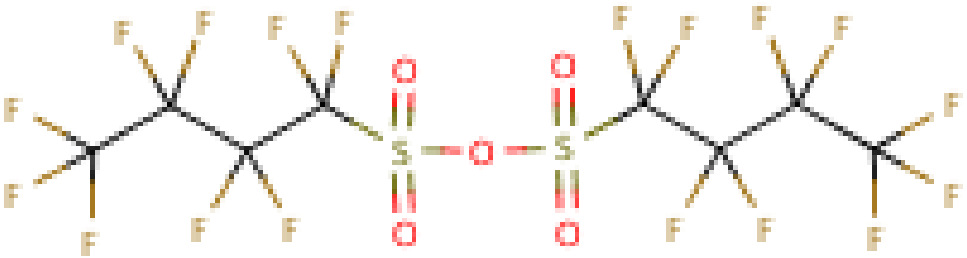
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Chemical Name in the Inventory and Synonyms	**1-Butanesulfonic acid, 1,1,2,2,3,3,4,4,4-nonafluoro-, ammonium salt** nonafluoro-1-butanesulfonic acid, ammonium salt ammonium 1,1,2,2,3,3,4,4,4-nonafluorobutane-1-sulfonate ammonium PFBS ammonium perfluorobutanesulfonic acid
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	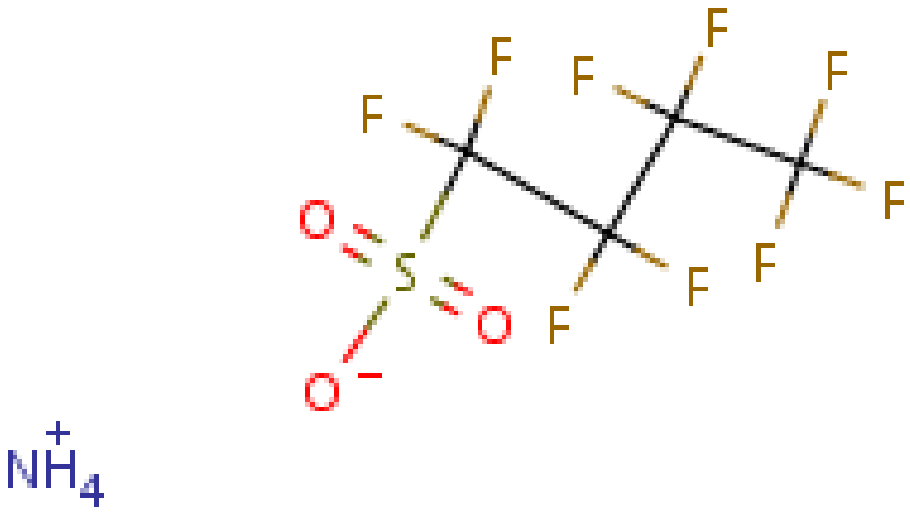
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Molecular Weight	317.1

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