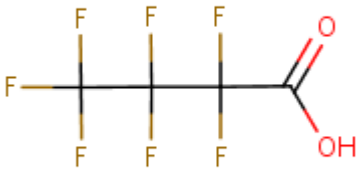
CAS # 375-22-4  	**Perfluorobutyric acid (PFBA)** Synonym[1]s: 4-02-00-00810 (Beilstein Handbook Reference); BRN 1426882; EINECS 206-786-3; Heptafluorobutanoic acid; Heptafluoro-1-butanoic acid; Heptafluorobutyric acid; Kyselina heptafluormaselna [Czech]; NSC 820; Perfluorobutanoic acid; Perfluorobutyric acid; Perfluoropropanecarboxylic acid; Kyselina heptafluormaselna **RTECS #[2]:** ET4025000 **EINECS #[3]:** 206-786-3 **Molecular Weight[4]**: 214.0359 **Molecular Formula[5]:** C4-H-F7-O2 **Common Salts: Ammonium perfluorobutyrate**
PHYSICAL CHARACTERISTICS	
Primary Use	"PFBA (CAS No. 375-22-4) and its anhydride (CAS No. 336-59-4) are used in the laboratory for acylation of alcohols, amino acids and other compounds. Derivatives are highly volatile and are used in gas chromatography separations."[6] Note that 6:2 FTOH is a volatile precursor of PFBA (Wang, et al, 2014).[7] Other precursors include 6:2 fluorotelomer sulfonamide alkylamine (FTAA) used in AFFFs. (D'agostino and Mabury 2017)[8]
Physical state, odor at room temperature & pressure	Liquid[9] Colorless liquid with an unpleasant odor[10]
Melting point; Boiling point	-17.5°C (exp.)[11]; 121°C (exp.)[12]
Solubility	447 mg/L in water[13]
Specific Gravity	Not found
SAFETY/PHYSICAL HAZARDS	
Vapor Pressure	849 Pa (exp.)[14]
Flammability	Not found
Flashpoint	Not found
Flammability Rating	Not found
Auto Ignition Point	Not found
Combustion products	Not found
Explosivity (UEL, LEL, shock sensitive)	Not found
Oxidizer	Not found
Corrosivity	Not found
pH	Not found
Reactivity	Not found
Viscosity	Not found
Odor Threshold	Not found

Particle size, shape, respirable fraction	Not found
Other physical hazards associated with process: Heat, gases under pressure, noise, vibration, ergonomic hazard	Not found
HEALTH HAZARDS	
Acute Toxicity	
Oral LD_{50}	Not found
Dermal LD_{50}	Not found
Inhalation LC_{50}	Not found
Intraperitoneal LD_{50}	Mouse, 68 uL/kg[15]
Other LD	IV, Rabbit, lethal dose:> 10uL/kg[16]
Chronic or Sub-chronic Toxicity	
IARC rating	Not found on IARC website
Carcinogenicity	Not found on Prop 65 list (as of 11/13/17);
Neurotoxicity	Not listed as neurotoxic in HAZMAP; "No available neurotoxicity studies. Secondary observations reported in the 28 and 90-day studies include delayed bilateral pupillary reflex for males exposed to a dose > 10-fold higher than the BMDL used as the basis of the short-term, subchronic and chronic HRLs. Histopathological assessment of neuronal tissues (including the optic nerve) and motor activity evaluations did not reveal any treatment-related abnormalities."[17] "Administration of up to 184 mg/kg/day PFBA by gavage for 5 consecutive days to rats had no significant effect on the gross or microscopic morphology of the brain and spinal cord (3M 2007a). In a 28-day gavage study, male rats dosed with 150 mg/kg/day, but not 30 mg/kg/day, showed a delay in bilateral pupillary reflex at the end of the treatment period (Butenhoff et al. 2012a; van Otterdijk 2007a). Results from other tests including hearing ability, static righting reflex, grip strength, and motor activity were comparable between groups and histological examination of the brain (including the optic nerve), spinal cord, and sciatic nerve was unremarkable. In a 90-day study, pupillary reflex tests conducted in weeks 8 and 12 showed delayed dilation under dark conditions in rats dosed with 30 mg/kg/day (2/40 in controls vs. 7/39 in high-dose rats; p=0.071 according to the Fisher Exact Test) (Butenhoff et al. 2012a; van Otterdijk 2007b). Since no abnormalities were recorded during a 3-week recovery period, and there were no histopathological alterations in the eyes, the effect was not considered biologically significant by the investigator. Tests for hearing ability, static righting reflex, grip strength, and motor activity

	showed no associations with treatment with PFBA. In addition, there were no significant gross or microscopic alterations in the brain, spinal cord, or sciatic nerve"[18]
Developmental/Reproductive Toxicity	Not found on Prop 65 list (as of 4/27/17); "The study of PFBA in CD-1 mice administered oral doses from GD1-17 at 0, 35, 175, or 350 mg/kg/day (Das et al. 2008). The highest dose resulted in significant increases in full-litter resorption and increased maternal liver weights, but neonatal survival and postnatal growth were unaffected. Eye opening was delayed in all PFBA dose groups and onset of puberty was delayed in the two highest dose groups. The general lack of developmental toxicity except at the highest doses was attributed to the rapid elimination of the chemical in the dams."[19] "Developmental delays were observed in offspring of mice exposed during pregnancy. This effect was observed at a human equivalent dose greater than 2-fold higher than the human equivalent dose upon which the short-term RfD is based. Developmental effects are identified as secondary effects."[20] "Administering PFBA to rats by gavage at doses of up to 184 mg/kg bw/day for five days, 150 mg/kg bw/day for 28 days or 30 mg/kg bw/day for 90 days did not cause significant gross or microscopic alterations in primary and secondary reproductive organs (van Otterdijk, 2007b)."[21]
Genotoxicity/Mutagenicity	"A single intraperitoneal injection (i.p.) administration of PFBA to male Fischer 344 rats had no effect on either the liver or kidney DNA (ATSDR, 2009)."[22] Not found in CCRIS, GENE-TOX (as of 4/27/17)
Endocrine Disruption/Thyroid	Found on TEDX List of Potential Endocrine Disruptors[23] "PFBA, on the other hand, stimulated *Acox/Ctel Acot1*, and *Cyp4a1/11* gene expression in primary rat hepatocytes only at concentrations of 100uM and above (Bjork and Wallace 2009)."[24] "Secondary observations, including decreased T4 levels, altered hyperplasia/hypertrophy of the follicular epithelium of the thyroid, and increased thyroid weight were noted in the 28 and 90 day studies."[25] [TURI Note: Butenhoff et al 2012a indicates decreased FT4 levels for male rats] "In a repeat dose study (Butenhoff et al., 2012(a)), male and female rats were treated with ammonium perfluorobutyrate at doses up to

	150 and 30 mg/kg bw/day for 28 and 90 days, respectively. Reduced serum thyroxine with no change in serum thyrotropin was reported in female rats. In males, the effects were generally mild, reversible on cessation of treatment, and included: hepatic hypertrophy with minimal to slight hepatocellular hypertrophy, hypothyroxinaemia without evidence of a thyroid follicular response; reduced serum total cholesterol; mild reductions in red blood cell parameters without evidence of an effect on red blood cell turnover; and delayed bilateral pupillary light reflex. According to the study authors, the hypothyroxinaemia likely resulted from a combination of competitive displacement of thyroxine as well as increased metabolism and elimination of thyroxine. Hypothyroxinaemia was not accompanied by an elevation of thyroxine stimulating hormone (TSH); nor was dosing with ammonium perfluorobutyrate accompanied by evidence of a hypertrophic or hyperplastic response of the thyroid follicles, based on morphometric endpoints."[26] "The homeobox genes, including *Hex*, encode a family of transcription factors that plays a vital role in cell differentiation during development (Gehring, 1987), including early thyroid development (Thomas et al., 1998).The mRNA abundance of Hex was significantly different between cells exposed to PFOS and PFBS, and similar differences were also seen when comparing between cells exposed to PFOA versus PFBA; but in the case of the sulphonates the magnitudes of the response in cells exposed to the replacement chemicals was statistically significantly less than those in cells exposed to PFOS, whereas PFBA caused a significantly greater effect than PFOA. This suggests that for thyroid-related processes PFOS may be a conservative predictor for the effects of replacement chemicals, whereas PFOA may under predict the effects (Naile 2012)."[27] "Gender-related differences were found; PFOS, PFHxS, PFBS, and PFOA levels were higher in males ($p<0.05$), and the mean concentration of $\sum_8$PFASs was 1.5 times greater in males (6.02 ng/mL) than in females (4.15 ng/mL). PFOS and $\sum_8$PFASs were significantly negatively correlated with FT3 and FT4 and positively correlated with TSH while PFPeA and PFHxA were significantly positively correlated with TGAb and TMAb in all the samples."… PFBA was detected in 75.2% of the serum of samples. (Li et al 2017c)[28]

	See aquatic toxicity below. PFBA had no effect in a radio-ligand-binding assay (Weiss 2009).[29]
Immunotoxicity	Not found
Other organ toxicity	The liver toxicity and peroxisome proliferation potency in rats of PFAS increase with the carbon chain length until C_9, and the activity was higher in response to carboxylates compared to sulfonates (Wolf et al. 2008).[30] PFBA can cause peroxisome proliferation, induction of peroxisomal fatty acid oxidation and hepatomegaly, suggesting that PFBA activates the nuclear receptor, peroxisome proliferator–activated-receptor-α (PPAR-α) in mice and humans (Foreman et al. 2009). PFBA has a slighter effect on indicators of peroxisome proliferation than PFOA (Ikeda et al. 1985). PFBA had also a slighter effect than PFHxA but had a higher PPARα activity in the liver than PFBS, PFHxS and PFOS (Wolf et al. 2008).[31] "All PFCAs led to increased PPARα and PPARγ activity from exposure concentrations of 30 uM or 100uM, except for PFBA, which did not cause any change in PPARγ activity" (Rosenmai 2016).[32] "Effects observed in the liver in studies with PFHxA and PFBA were generally mild and reversible."[33] "PFBA administered to rats by gavage at doses of up to 184 mg/kg/day for five days, or up to 150 mg/kg bw/day for 28 days, did not cause any morphological alterations in the respiratory tract, gastrointestinal tract or skeletal muscle. There were no significant gross or microscopic alterations in the spleen, thymus or mesenteric lymph nodes, or in haematological parameters (van Otterdijk, 2007a; van Otterdijk, 2007b)."[34] Doses of 30 mg/kg/day, but not 6 mg/kg/day, for 90 days resulted in significant reductions in red blood cell counts, hemoglobin, and hematocrit, and an increase in red cell distribution width in male rats (Butenhoff et al. 2012a; van Otterdijk 2007b). This dose level also caused a reduction in mean corpuscular hemoglobin and reduced mean corpuscular hemoglobin concentration in male rats. The lower hemoglobin and hematocrit observed in males were still detected at the end of a 3-week recovery period. These hematological effects were considered minor and not evidence of an adverse effect on red blood cell turnover by the investigator based on lack of alterations in bone marrow or the spleen.[35]
Skin, Eye and Respiratory Effects	
*Irritant – **S**kin, **E**ye, or **R**espiratory*	Toxic Pneumonitis[36]

	"Skin irritant/corrosive effects cannot be ruled out for PFBA anhydride (which is reactive and could hydrolyse to PFBA)."[37] "Ammonium PFHx was considered to be a severe eye irritant in rabbits. As a salt, it is considered to have lower irritation potential than the acids and anhydrides in the group; therefore, in the absence of additional information, classification is considered warranted for all chemicals in this group."[38]
*Corrosive – **S**, **E**, or **R***	Skin Burns[39]; Corrosive to skin and eyes[40] Corrosive substance that can cause injury to the skin, eyes, and respiratory tract[41]
*Permanent Damage – **S**, **E**, or **R***	Not found
*Sensitizer– **S** & **R***	Not found in AOEC database (as of 4/27/17); "No data are available for the chemicals in this group. Based on data for the analogues, PFOA and its ammonium salt (NICNAS), the chemicals in this group are not considered skin sensitisers."[42]
Asthmagen – Initiator or Exacerbator	Not found in AOEC database (as of 4/27/17)
Skin Absorption, Kp	Not found
LOAEL	Not found
NOAEL	In another study sequential 28-day and 90-day oral toxicity studies have been performed in male and female rats with ammonium perfluorobutanoate/perfluorobutyrate (PFBA) at doses up to 150 mg/kg/day in males and 30 mg/kg/day in females, and ammonium perfluorooctanoate (PFOA) was used as a comparator at a dose of 30 mg/kg/day in the 28-days study (Butenhoff et al. 2012(a)). Female rats were unaffected by PFBA with the no-observable-adverse-effect -levels (NOAELs) >150 mg PFBA/kg/day in the 28-day study and >30 mg PFBA/kg/day in the 90 days study. Effects in males included: increased liver weight, slight to minimal hepatocellular hypertrophy; decreased serum total cholesterol; and reduced serum thyroxin. The NOAEL for males was 6 mg PFBA/kg/day in both the short-and long-term study. A comparative dosing with 30 mg/kg/day PFOA resulted in increased incidence of clinical signs of toxicity (e.g. hunched posture), increased liver weight in females as well as males, and a major (75%) reduction in body weight of males. Thus, the relative response of rats to dosing with PFBA as compared to PFOA was considered by the authors to be the result of both the more rapid toxicokinetic clearance in rodents and lesser toxicodynamic potency of PFBA.[43] "In a 90-day repeated dose study, Crl:CD rats were administered PFBA at 6 or 30 mg/kg/day for 90 days (van Otterdijk, 2007b). Alterations in haematological parameters were noted in the high dose group rats, but these were not considered adverse effects by the study's authors, based on a lack of alteration in the bone marrow or spleen. At the

	highest dose tested, 30 mg/kg/day, increased absolute liver weight (23 %), increased serum ALP activity and reduced total serum protein were noted. PFBA also caused diffused panlobular hepatocyte hypertrophy. These effects were reversible during a 21-day recovery period; the NOAEL for the study was established as 6 mg/kg/day."[44] "6.9 mg/kg-d (NOAEL, NOTOX 2007b 90-day study)"[45]
Benchmark Dose Response (BMD)	$BMDL_{10}$ = 3.01 mg/kg-d (calculated by Butenhoff, 2007 based on NOTOX 2007a 28-day study)[46]
Toxicokinetics	PFBA is predominantly excreted in the urine. In a study with male and female rats, 51-90 % and 101-112 % of PFBA was excreted in urine within 24 hours, respectively, but only 0-3 % was excreted in the feces. In mice, 65-68 % was excreted in urine by female mice after 24 hours compared with approximately 35 % in male mice. 4-11 % was excreted in feces by both sexes. In monkeys, 41 and 46 % of the administered dose of PFBA was excreted in urine by male and females, respectively (Chang *et al.* 2008). The serum elimination half-lives of PFBA in **rats** given 30 mg/kg b. w. in drinking water were 9 hours for males and 1.76 hours for females (Chang *et al.* 2008). If PFBA was administered intravenously the half-lives were a little shorter (6 and 1 hours). For **mice** given oral doses of PFBA as the ammonium salt the half-lives were 5-16 hours for males and about 3 hours for females. For **monkeys** given 10 mg PFBA/kg b. w. intravenous the half-lives were 40 hours for males and 41 hours for females. For **humans** the half-lives were about 72 and 87 hours for males and females, respectively. The last values were determined in workers and after a PFBA drinking water pollution incident in Minnesota, where levels were 1-2 µg PFBA/L. The relatively short residence time in the blood doesn't mean that PFBA is quickly excreted in humans. Analysis of Spanish autopsy tissues revealed that the highest concentrations of most PFAS were found in lung tissues, and that the short-chain PFBA surprisingly had the highest concentration, which was 100 times higher than for e.g. PFOS. Also in the kidneys PFBA had the highest concentration of all, and that was six times higher than the concentration of PFOS. PFBA was also measured in the liver and brain (Perez *et al.* 2013). Thus, PFBA seems to behave differently in humans compared to experimental animals.[47] "In comparison to PFOA and PFOS, … PFBA…, are likely eliminated by various species within shorter periods of time, whereas PFHxS has even longer elimination half-lives."[48]
Synergistic or Antagonistic Effects	Additivity endpoints: Developmental; Hematologic (blood) system; Hepatic (liver) system; Thyroid (E)"[49]

Environmental and Human Health Exposure and Risk Values	
RfC/RfD	Not found in the IRIS database (as of 4/27/17) Reference dose for MNDPH health risk limit calculations = 0.0038 mg/kg-d (laboratory animal); Human Equivalent dose = 3.01/8 = 0.38 mg/kg-d (factor of 8 adjusts for half-life duration of 3 days in humans vs. 9.22 hours in male rats)[50] "ITV = 0.024 mg/kg/day; critical impact/effect – hepatic effects, Butenhoff et al., 2012, NOAEL = 6 mg/kg/day"[51]
ATSDR-MRL	Not found on the ATSDR June 2017 List (as of 11/14/17)
Adverse Effect Levels: DNEL, PNEC, PNEL	Not found
Health Based Exposure Limits	
NIOSH-REL/IDLH/Ceiling Limits	Not found in the NIOSH Pocket Guide
OSHA-PEL	Not found in the Z Tables
ACGIH TLV-TWA	Not found in RTECS (as of 11/14/17)
TLV-STEL	Not found in RTECS (as of 11/14/17)
Biomonitoring Action Limits	Not found
Drinking Water Standards	Not found
Other	Short-term Non-Cancer Health Risk Limit = 7 ug/L[52] Subchronic Noncancer Health Risk Limit = 7 ug/L[53] Chronic Noncancer Health Risk Limit = 7 ug/L[54] **Non-harmonized** classifications from CLP Database: Skin Corr. 1A H314; Eye Dam. 1 H318; Met. Corr. 1 H290; Skin Corr. 1B H314; STOT SE 3 H335 (not specified); H370
ENVIRONMENTAL & ECO-SYSTEM HAZARDS	
Persistence	Perfluorinated carboxylic acids, including PFBA, are not transformed/degraded by hydrolysis or photolysis to any appreciable extent, nor are they biodegradable under aerobic or anaerobic environmental conditions in water or soil.[55] Therefore, PFBA is extremely persistent and is not expected to transform in biota or in the environment. "Short-chain PFAAs are generally more mobile than their long-chain homologues; they are not retarded in soil and have already been detected in groundwater".[56] <u>Presence in the environment</u>: PFAS contamination in groundwater near 3M plant -"As of July 2007, 455 private and non-community public wells have been sampled for the expanded list of seven PFCs. PFBA has been detected in 363 wells; it is the most commonly detected and widely distributed PFC in the Oakdale-Lake Elmo area, followed by PFOA and then PFOS."[57]

	Seasonal accumulation of PFAS measured in 10 m shallow firn core from high altitude glacier at Mt. Ortles, Italy. PFBA, along with PFOA and PFNA, were the most abundant PFAS measured. PFBA: 0.3 to 1.7 ng /L[58] Comparable results to another study: lake water from Lake Macun : 0.8 ng/L; remote lake in Swiss Alps, (Greenpeace, 2015).[59] Chinese study downstream of Beijing Airport: surface water concentrations were observed for PFBA (10.6–41.5 ng/L)[60] Other concentrations in the environment:[61] Rhine River 1.33 ng/L Scheldt River up to 335 ng/L (downstream of fluorochemical plant) Upper Mississippi: 2.73 ng/L Llobregat River- Spain 19.5 ng/L "The prevalence of PFBA compared to other perfluorinated compounds has been noted in all of these studies. In one study PFBA was found to be present at concentrations at least four times higher than those of PFOS, PFOA and PFHxA" The multi-matrices samples from snow, lake water, surface runoff water and coastal seawater were collected in Fildes Peninsula, King George Island, Antarctica in 2011. High concentration and mostly frequency of PFBA occurred in snow (up to 1,112 pg/L), lake water (up to 2,670 pg/L) and SRW (1,431 pg/L) while detected in the range of method detection limited (MDL) in the coastal seawaters indicate that PFBA is mainly originated from atmospheric dust contamination and also affected by the degradation of their precursors[62]
Bioaccumulation	Perfluorinated chemicals have been observed to bioaccumulate by binding to proteins in plasma and liver, rather than the more conventional partitioning to fatty tissue...[63] PFBA has been observed to be eliminated more rapidly than PFOA, which may account for the decrease in mammalian bioaccumulation potential observed with shorter chain PFAS. Plant uptake: Study using reclaimed water to irrigate lettuce and strawberry crops using C3-C8 PFCAs and C4, C6 and C8 PFASs. Trends showed decreasing concentrations in shoots and fruit with increasing chain length. PFBA and PFPeA accumulated the most in edible portions compared with other PFAAs. At typical WWTP effluent concentrations of PFAAs (0.02-4 µg/L), PFBA values reached 3 µg/g in lettuces and 2 µg/g in strawberry

	fruit. At higher concentrations representative of contaminated waters (10-40 µg/L), PFBA accumulated to 25 µg/g in lettuce and up to 11 µg/g in strawberry fruit. For reference, accumulated PFOA levels in this study could well exceed the provisional health advisory levels for drinking water. "Bioaccumulation factors for lettuce were correlated to carbon chain length of PFAAs, showing approximately a 0.4 to 0.6 log decrease per CF2 group. This study confirms that PFAAs can enter and bioaccumulate in food crops irrigated with reclaimed water. Bioaccumulation potential depends on analyte functional group and chain length, concentration in the reclaimed water, and organic carbon content of the soil."[64] <u>Presence in biota</u> Chinese Fluorochemical industrial park fenceline community - Home produced eggs. PFBA concentrations in eggs:[65] • 2 km from fenceline: 110 ng/g yolk, 22.5 ng/g whole egg • 20 km from fenceline: 1.75 ng/g yolk, 0.54 ng/g whole egg (20 km contamination is roughly same range as commercially produced eggs) In a Spanish study of breast milk and baby formula and food, PFBA was found in 100% of all samples:[66] • breast milk, avg concentration 50 ng/L, max 155 ng/L • infant formula powder, avg 165 ng/L, max 496 ng/L • dry cereal baby food, avg 276 ng/L, max 968 ng/L • baby food pots, avg 519 ng/L, max 5,013 ng/L Ski wax technicians, exposed to high levels of 8:2 FTOH, had whole blood samples analyzed. PFBA 0.51 µg/L (considerably less than PFOA 110 µg/L)[67]
Ecological Toxicity	"It is noted that long term intergenerational studies for two C8 perfluorinated acids (PFOA and perfluorooctanesulfonic acid (PFOS)) have indicated potential for chronic toxicity that may not be identified in standard ecotoxicity tests. These studies suggest effects may manifest in offspring when the parent generation is exposed to the chemical (NICNAS, 2015a; 2015c). No intergenerational studies were identified for any of the chemicals in this group *[short chain PFAS including PFBA]*. Nevertheless, it is noted that the measured developmental toxicity of PFBA in fish is over 100 times lower than that of PFOS (Hagenaars, et al., 2011; NICNAS, 2015c)."[68]
Aquatic Toxicity: LC_{50}, EC_{50}, ErC_{50}, NOAEC/NOEC	48 hr EC_{50} 5,251 mg/L[69]

	We evaluated the toxicity of PFOA, PFHxA and PFBA on a zebrafish liver cell line and investigated the effects of exposure on cell metabolism. Gross toxicity after 96 h of exposure was highest for PFOA and PFO(-) , while PFHxA and PFBA exhibited lower toxicity (Mahapatra et al, 2017).[70] …our study revealed that PFASs with shorter carbon chains are less toxic than PFOA, and that exposure to sublethal dosage of PFOA, PFHxA or PFBA affects cell metabolism (Mahapatra et al 2017).[71] LC_{50} value (with 95% confidence interval) for zebrafish embryos exposed to PFBA = 13, 795 ppm (CI = 8,932-54,103)(Godfrey 2017).[72] At 6dpf, zebrafish exposed to all halogenated chemicals, both old use and next generation, had smaller posterior swim bladder and increased expression in the gene encoding thyroid peroxidase, tpo and the genes encoding two swim bladder surfactant proteins, sp-a and sp-c. These results mirrored the effects of thyroid hormone-exposed positive controls. … Effects on the anterior swim bladder at 28dpf, after exposure to MMI as well as both old and new halogenated chemicals, were the same, i.e., absence of SB in ~50% of fish, which were also of smaller body size. Overall, our results suggest thyroid disruption by the halogenated compounds tested via the swim bladder surfactant system. However, with the exception of TBBPA and TDCPP, the concentrations tested (~5-137ppm) are not likely to be found in the environment (Godfrey 2017a).[73]
Fate and Transport: Aquatic, Terrestrial and atmospheric	PFBA has been measured in various locations worldwide, including remote locations. "It has been hypothesized that distribution may occur through atmospheric transport of acids, transport of anions in surface water or ocean currents, and/or transport of volatile precursors (e.g. fluorotelomer alcohols) (Ellis, et al., 2004; OECD, 2013a)"[74]
Transport Issues	"There are probably no real environmental sinks of PFAAs; the long-term fate of these substances is transport to deep ocean water and/or sediment burial".[75]
Factors affecting bioavailability	Not found
Global Environmental Impacts	
Ozone Depletion Potential (ODP)	Not found
Global Climate Change	Not found
Greenhouse Gas Production	Not found
Acid Rain Formation	Not found
Special Reports	

EU	**Human Health Tier II Assessment for Short chain perfluorocarboxylic acids and their direct precursors,** NICNAS, https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-group-assessment-report?assessment_id=1686 **NICNAS 2016a Environment Tier II Assessment for Short-Chain Perfluorocarboxylic Acids and their Direct Precursors**, NICNAS, updated April 19, 2017, https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-assessments/tier-ii-environment-assessments/short-chain-perfluorocarboxylic-acids-and-their-direct-precursors **Short-chain Polyfluoroalkyl Substances (PFAS)** – A literature review of information on human health effects and environmental fate and effect aspects of short-chain PFAS, Environmental project No. 1707, 2015 http://www2.mst.dk/Udgiv/publications/2015/05/978-87-93352-15-5.pdf **Polyfluoroalkyl substances (PFASs) in textiles for children** – Survey of chemical substances in consumer products No. 136, 2015 http://www2.mst.dk/Udgiv/publications/2015/04/978-87-93352-12-4.pdf **Survey of PFOS, PFOA and other perfluoroalkyl and polyfluoroalkyl substances** – Part of the LOUS-review, Environmental project No. 1475, 2013 http://www2.mst.dk/Udgiv/publications/2013/04/978-87-93026-03-2.pdf

Notes on chemical research: Not found in NIOSH Pocket Guide; HSDB (no primary record available);

ToxPlanet folders:

ATSDR – No info in the ATSDR Tox profile for perfluoroalkyls; Australian Gov't – NICNAS CCID (NZ EPA); ChEBI – Chemical Entities of Biological Interest; CPCat – Chemical and Product Categories; CTD – Comparative Toxicogenomics Database; Danish EPA – Publications; ECOTOX; EFSA – European Food Safety Authority; EPA; **HazMAP**
ITER – International Toxicity Estimates for Risk Assessment; Minnesota Department of Health; NIST – National Institute of Standards and Technology – Chemistry WebBook PubChem; **RTECS;** SPIN – Substances in Preparations in Nordic Countries; TEDX

[1] www.expub.com; Chemical Identity Page for Perfluorobutyric acid.
[2] www.expub.com; RTECS for Butyric acid, heptafluoro-.
[3] www.expub.com; Chemical Identity Page for Perfluorobutyric acid.

[4] U.S. National Library of Medicine, ChemIDplus, a Toxnet Database, entry for "Perfluorobutyric acid.", accessed online at: https://chem.nlm.nih.gov/chemidplus/name/perfluorobutanoic%20acid

[5] U.S. National Library of Medicine, ChemIDplus, a Toxnet Database, entry for "Perfluorobutyric acid.", accessed online at: https://chem.nlm.nih.gov/chemidplus/name/perfluorobutanoic%20acid

[6] **NICNAS 2017:** Australian Government, Department of Health, National Industrial Chemicals Notification and Assessment Scheme (NICNAS). HUMAN HEALTH TIER II ASSESSMENT FOR Short chain perfluorocarboxylic acids and their direct precursors. Accessed online at: https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-group-assessment-report?assessment_id=1686.

[7] **Kirchgeorg 2016:** Kirchgeorg T, et al. Seasonal accumulation of persistent organic pollutants on a high altitude glacier in the Eastern Alps. *Environmental Pollution* **218** (2016) 804-812.

[8] **D'Agostino and Mabury 2017**: D'Agostino LA, Mabury SA. Aerobic biodegradation of 2 fluorotelomer sulfonamide-based aqueous film-forming foam components produces perfluoroalkyl carboxylates. *Environ Toxicol Chem.* 2017 Feb 1 doi: 10.1002/etc.3750. [Epub ahead of print].

[9] **NICNAS 2016a:** Australian Government, Department of Health, National Industrial Chemicals Notification and Assessment Scheme (NICNAS). Environment Tier II Assessment for Short-Chain Perfluorocarboxylic Acids and their Direct Precursors, Last update April 19, 2017. Accessed online at: https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-assessments/tier-ii-environment-assessments/short-chain-perfluorocarboxylic-acids-and-their-direct-precursors#PhysicalandChemicalProperties,

[10] HAZMAP.

[11] NICNAS 2016a.

[12] NICNAS 2016a.

[13] **Danish EPA 2015b**: Danish Environmental Protection Agency. Short-chain Polyfluoroalkyl substances (PFAS) – A literature review of information on human health effects and environmental fate and effect aspects of short-chain PFAS. Environmental project No. 1707, 2015. Page 49. Accessed online at: http://www2.mst.dk/Udgiv/publications/2015/05/978-87-93352-15-5.pdf

[14] NICNAS 2016a.

[15] www.expub.com; RTECS for Butyric acid, heptafluoro-.

[16] www.expub.com; RTECS for Butyric acid, heptafluoro-.

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