Health-Based Guidance for Water
Health Risk Assessment Unit, Environmental Health Division
651-201-4899

Adopted as Rule: November 2023

Toxicological Summary for: Perfluorobutane sulfonate

CAS: **45187-15-3 [anion]**
375-73-5 [free acid]
29420-49-3 [potassium salt]
68259-10-9 [ammonium salt]
60453-92-1 [sodium salt]

Synonyms: PFBS ion; Perfluorobutanesulfonate; 1,1,2,2,3,3,4,4,4-nonafluorobutane-1-sulfonate (IUPAC name); Perfluorobutyl sulfonate

Acute Non-Cancer Health Risk Limit ($nHRL_{Acute}$) = Not Derived (Insufficient Data)

Short-term Non-Cancer Health Risk Limit ($nHRL_{Short\text{-}term}$) = 0.1 µg/L

$$\frac{\text{(Reference Dose, mg/kg-d) x (Relative Source Contribution) x (Conversion Factor)}}{\text{(Short-term Intake Rate, L/kg-d)}}$$

$$= \frac{\text{(0.000084 mg/kg-d) x (0.5)}^{*} \text{ x (1000 µg/mg)}}{\text{(0.290 L/kg-d)}^{**}}$$

= 0.14 rounded to **0.1 µg/L**

*Relative Source Contribution: MDH 2008, Section IV.E.1.
**Intake Rate: MDH 2008, Section IV.E.1. and US EPA 2019, Exposure Factors Handbook, Tables 3-1, 3-3 and 3-5.

Reference Dose/Concentration:	HED/Total UF = 0.0084/100 = 0.000084 mg/kg-d (Hsd:Sprague Dawley Rats)
Source of toxicity value:	Determined by MDH in 2022
Point of Departure (POD):	6.97 mg/kg-d (administered dose $BMDL_{1SD}$, (National Toxicology Program 2019))
Dose Adjustment Factor (DAF):	Chemical- and Study-Specific Toxicokinetic Adjustment $Half\text{-}life_{FemaleRat}/Half\text{-}life_{Human}$ = 1.3 hr/1050 hr = 0.0012, based on MDH analysis of (Huang, Dzierlenga et al. 2019) for female rats and (Xu, Fletcher et al. 2020) for humans.
Human Equivalent Dose (HED):	POD x DAF = 6.97 mg/kg-d x 0.0012 = 0.0084 mg/kg-d
Total uncertainty factor (UF):	100
Uncertainty factor allocation:	3 for interspecies differences (for toxicodynamics), 10 for intraspecies variability, and 3 for database uncertainty due to a lack of available immunotoxicity and developmental neurotoxicity studies (known sensitive effects of other

	PFAS) as well as lack of a 2-generation study in a more appropriate species
Critical effect(s):	Decreased total T4
Co-critical effect(s):	None
Additivity endpoint(s):	Thyroid (E)

Subchronic Non-Cancer Health Risk Limit ($nHRL_{Subchronic}$) = 0.1 µg/L

$$\frac{\text{(Reference Dose, mg/kg-d) x (Relative Source Contribution) x (Conversion Factor)}}{\text{(Subchronic Intake Rate, L/kg-d)}}$$

$$= \frac{(0.000084 \text{ mg/kg-d})^{\#} \times (0.2)^{*} \times (1000 \text{ µg/mg})}{(0.074 \text{ L/kg-d})^{**}}$$

= 0.23 rounded to 0.2 µg/L

[#]The calculated Subchronic RfD (0.00054 mg/kg-d) is higher than the Short-Term RfD (0.000084 mg/kg-d), which is based on thyroid effects. The Subchronic RfD must be protective of all types of adverse effects that could occur as a result of subchronic exposure, including short-term effects (MDH 2008, page 34). Therefore, the Short-Term RfD is used in place of the calculated Subchronic RfD when deriving subchronic water guidance.

[*]Relative Source Contribution: MDH 2008, Section IV.E.1.
[**]Intake Rate: MDH 2008, Section IV.E.1. and US EPA 2019, Exposure Factors Handbook, Tables 3-1, 3-3 and 3-5.

The Subchronic nHRL must be protective of shorter duration exposures that occur within the subchronic period and therefore, the Subchronic nHRL is set equal to the Short-term nHRL of 0.1 µg/L. Additivity endpoints: Thyroid (E)

Chronic Non-Cancer Health Risk Limit ($nHRL_{Chronic}$) = 0.1 µg/L

$$\frac{\text{(Reference Dose, mg/kg-d) x (Relative Source Contribution) x (Conversion Factor)}}{\text{(Chronic Intake Rate, L/kg-d)}}$$

$$= \frac{(0.000084 \text{ mg/kg-d})^{\#} \times (0.2)^{*} \times (1000 \text{ µg/mg})}{(0.045 \text{ L/kg-d})^{**}}$$

= 0.37 rounded to 0.4 µg/L

[#]The calculated Chronic RfD (0.00018 mg/kg-d) is higher than the Short-Term RfD (0.000084 mg/kg-d), which is based on thyroid effects. The Chronic RfD must be protective of all types of adverse effects that could occur as a result of shorter exposures, including short-term effects (MDH 2008, page 34). Therefore, the Short-Term RfD is used in place of the calculated Chronic RfD when deriving chronic water guidance.

[*]Relative Source Contribution: MDH 2008, Section IV.E.1.
[**]Intake Rate: MDH 2008, Section IV.E.1. and US EPA 2019, Exposure Factors Handbook, Tables 3-1, 3-3 and 3-5.

The Chronic nHRL must be protective of shorter duration exposures that occur within the chronic period and therefore, the Chronic nHRL is set equal to the Short-Term nHRL of 0.1 µg/L. Additivity endpoints: Thyroid (E)

Cancer Health Risk Limit (cHRL) = Not Applicable

Chemical Mixtures: Exposure to chemicals in combination may cause adverse effects that would not be predicted based on separate exposures to individual chemicals. When multiple contaminants occur as a mixture in water, the cumulative risk should be assessed (MDH 2008, Section IV.E.3). To download the calculator, see MDH's Water Guidance and Additivity Calculator https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/guidance.xlsx

Volatile: No

Summary of Guidance Value History:
In 2009, Health-Based Values (HBVs) for PFBS were first derived: 9 µg/L for Subchronic durations and 7 µg/L for Chronic durations. These HBVs were adopted as HRLs in 2011.

In 2017, MDH re-evaluated the 2011 guidance and derived new HBVs of 3 µg/L for Short-Term and Subchronic durations and 2 µg/L for Chronic durations based on new toxicokinetic information in mice, a reassessment of toxicokinetic information in rats, and a new developmental toxicity study in mice.

In 2020, MDH updated the intake rates used in the calculation of water guidance values based on the most recent EPA Exposure Factors Handbook. This update did not change the PFBS 2017 guidance values.

In 2022, MDH re-evaluated the 2020 guidance and derived new HBVs of 0.1 µg/L for Short-Term, Subchronic, and Chronic durations. The 2022 values are lower than the previous values as a result of: 1) new toxicokinetic information in humans and rats, and 2) a new toxicity study in rats evaluating sensitive thyroid endpoints.

In November 2023, the guidance values were adopted into Minnesota Rules, 4717.7860, as Health Risk Limits (HRLs).

Summary of toxicity testing for health effects identified in the Health Standards Statute (144.0751):
Even if testing for a specific health effect was not conducted for this chemical, information about that effect might be available from studies conducted for other purposes. MDH has considered the following information in developing health protective guidance.

	Endocrine	Immunotoxicity	Development	Reproductive	Neurotoxicity
Tested for specific effect?	Yes	No	Yes	Yes	Yes

	Endocrine	Immunotoxicity	Development	Reproductive	Neurotoxicity
Effects observed?	Yes[1]	-[2]	Yes[3]	Yes[4]	Yes[5]

Comments on extent of testing or effects:

[1] Male and female rats exposed to PFBS orally had large decreases in various thyroid hormones at a dose 900-fold higher than the Short-Term RfD; the effect on one thyroid hormone (tT4) served as the basis for the Short-Term RfD. A decrease in serum thyroid hormones is an effect consistently observed in other PFAS compounds.

An oral developmental study evaluated female mice exposed in utero to PFBS. Delays in vaginal opening and changes in estrus cycling as well as changes in uterine and ovarian size were reported. Pubertal and adult female offspring exhibited decreases in serum estrogen and progesterone levels with elevation of luteinizing hormone levels. Decreases in serum tT4 and T3 were observed in conjunction with slight increases in TSH in female offspring as well as their mothers. These effects all occurred at doses at least 1400-fold higher than the Short-Term RfD.

[2]An study evaluated the association between 11 PFAS chemicals and immunological markers in children from Taiwan. Associations of several PFAS chemicals, including PFBS, with asthma and asthma related biomarkers were found. Associations for PFBS were fewer and weaker than those for several other PFAS chemicals. Concentrations of individual PFAS were positively correlated, and therefore it is not possible to determine whether associations apply to multiple PFASs or to only a subset of individual PFAS. A more recent study following a cohort of several hundred children in Shanghai, China found an association between PFBS concentration in maternal cord blood with increased frequency of respiratory tract infections and decreased IgG concentration in 5-year-old children, suggesting that pre/perinatal exposures to PFBS impacts future immune function in children.

No PFBS immunotoxicity studies have been conducted in laboratory animals. Immunotoxicity has been identified as a sensitive endpoint for several other PFAS. A database uncertainty factor of 3 was incorporated, in part, to address the need for immunotoxicity testing.

[3] Two oral developmental studies (one in rats and one in mice) and a 2-generation study in rats have been conducted. The developmental effects reported in the mouse study included decreased pup body weight, decreased serum thyroid hormones, delayed eye opening, delayed vaginal opening and first estrus as well as smaller ovarian and uterine size in adult offspring. These effects were observed at doses 1400-fold higher than the Short-Term RfD. The developmental study in rats reported decreased fetal body weight at doses >14000-fold higher than the Short-term RfD. In the 2-generation study in rats, no developmental effects were identified at the highest dose tested (14000-fold higher than the Short-Term RfD). However, female rats excrete PFBS much more quickly than humans, which may limit the applicability of this 2-generation study. A database uncertainty factor of 3 was incorporated, in part, to address the lack of a 2-generation study in a more appropriate species.

[4]Researchers examined the association between PFAS chemicals and endometriosis-related infertility among Chinese reproductive-age women in a case-control study. Women with endometriosis-related

infertility had significantly higher median levels of PFBS compared with those without the disease. PFBS was the only PFAS identified with a significant positive association, while several other PFAS chemicals exhibited an inverse association. Limitations of this study include no identification of the time course, disease survey reported levels may not reflect actual exposure, and no physical exam data was measured for controls.

An oral 2-generation study in rats has been conducted. No treatment related effects on female reproductive parameters were noted. Decreased number of spermatids per gram testes (P0) and increased incidence of abnormal sperm (F1) were noted at HED dose levels 37000-fold higher than the Short-term RfD.

[5]Neurological alterations were reported in the 28-day but not the 90-day oral study in adult rats. The results of the study are difficult to interpret. The longer study did not report any treatment related effects. The effects in the 28-day study occurred at HED dose levels 1400-fold higher than the Short-term RfD.

A database UF was incorporated, in part, to address the need for additional neurological testing, particularly in developmental life stages.

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