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

Web Publication Date: August 2020

Toxicological Summary for: Perfluorobutane sulfonate

CAS: **45187-15-3 (anion)**
375-73-5 (acid)
Synonyms: PFBS, Perfluorobutane sulfonic acid, Nonafluorobutanesulphonic acid

Acute Non-Cancer Health Based Value ($nHBV_{Acute}$) = Not Derived (Insufficient Data)

Short-term Non-Cancer Health Based Value ($nHBV_{Short\text{-}term}$) = 3 µg/L

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

$$= \frac{\text{(0.0016 mg/kg-d) x (0.5)}^{*} \text{ x (1000 µg/mg)}}{\text{(0.290 L/kg-d)}^{**}}$$

= 2.75 rounded to **3 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 = 0.158/100 = 0.0016 mg/kg-d (ICR mouse)
Source of toxicity value:	Determined by MDH in 2017
Point of Departure (POD):	50 mg/kg-d (administered dose NOAEL, developmental study by Feng et al 2017)
Dose Adjustment Factor (DAF):	$t_{½Human}$/ $t_{½FemaleMouse}$ = 665 hr/2.1 hr = 317, Chemical-Specific Toxicokinetic Adjustment ($t_{½Human}$ based on Olsen et al 2009, $t_{½FemaleMouse}$ based on Lau 2017 and Rumpler et al 2016)
Human Equivalent Dose (HED):	POD/DAF = 50 mg/kg-d/317 = 0.158 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 (concerns regarding neurological effects and persistent effects observed following *in utero* only exposure)
Critical effect(s):	Decreased offspring body weight, delayed eye opening, delayed vaginal opening and first estrus, as well as reproductive hormone changes, decreased ovarian follicle number and uterine weight in young adult female

	offspring exposed *in utero*; decreased total and free thyroxine (T4) and triiodothyronine (T3), as well as increased thyroid stimulating hormone (TSH) in both pregnant animals and in offspring
Co-critical effect(s):	None
Additivity endpoint(s):	Developmental, Female Reproductive System (E), Thyroid (E)

Subchronic Non-Cancer Health Based Value ($nHBV_{Subchronic}$) = $nHBV_{Short\text{-}term}$ = 3 µg/L

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

$$= \frac{\text{(0.0013 mg/kg-d) x (0.2)}^{*} \text{ x (1000 µg/mg)}}{\text{(0.074 L/kg-d)}^{**}}$$

= 3.51 rounded to 4 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 = 0.129/100 = 0.0013 mg/kg-d (Sprague Dawley rats)
Source of toxicity value:	Determined by MDH in 2017
Point of Departure (POD):	45 mg/kg-d (administered dose $BMDL_{10}$, based on epithelial hyperplasia in kidneys of F0 females, 2-generation study by Lieder et al 2009b and York 2003b)
Dose Adjustment Factor (DAF):	$t_{½Human}$/ $t_{½FemaleRat}$ = 665 hr/1.9 hr = 350, Chemical-Specific Toxicokinetic Adjustment ($t_{½Human}$ based on Olsen et al 2009, $t_{½FemaleRat}$ based on MDH evaluation of data presented in Olsen et al 2009)
Human Equivalent Dose (HED):	POD/DAF = 45 mg/kg-d/350 = 0.129 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 (concerns regarding neurological effects and persistent effects observed following *in utero* only exposure)
Critical effect(s):	Kidney epithelial and tubular/ductal hyperplasia
Co-critical effect(s):	Focal papillary edema and necrosis in the kidney
Additivity endpoint(s):	Renal (kidney) system

The Subchronic nHBV must be protective of short-term exposures that occur within the subchronic period and therefore, the Subchronic nHBV is set equal to the Short-term nHBV of 3 µg/L. Additivity endpoints: Developmental, Female Reproductive System (E), Thyroid (E).

Chronic Non-Cancer Health Based Value ($nHBV_{Chronic}$) = 2 µg/L

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

$$= \frac{\text{(0.00043 mg/kg-d) x (0.2)}^{*} \text{ x (1000 µg/mg)}}{\text{(0.045 L/kg-d)}^{**}}$$

= 1.9 rounded to **2 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 = 0.129/300 = 0.00043 mg/kg-d (Sprague Dawley rats)
Source of toxicity value:	Determined by MDH in 2017
Point of Departure (POD):	45 mg/kg-d (administered dose $BMDL_{10}$, based on epithelial hyperplasia in kidneys of F0 females, 2-generation study by Lieder et al 2009b and York 2003b)
Dose Adjustment Factor (DAF):	$t_{½Human}$/ $t_{½FemaleRat}$ = 665 hr/1.9 hr = 350, Chemical-Specific Toxicokinetic Adjustment ($t_{½Human}$ based on Olsen et al 2009, $t_{½FemaleRat}$ based on MDH evaluation of data presented in Olsen et al 2009)
Human Equivalent Dose (HED):	POD x DAF = 45 mg/kg-d/350 = 0.129 mg/kg-d
Total uncertainty factor (UF):	300
Uncertainty factor allocation:	3 for interspecies differences (for toxicodynamics), 10 for intraspecies variability, 3 for database uncertainty (concerns regarding neurological effects and persistent effects observed following *in utero* only exposure), and 3 for use of a subchronic study for the chronic duration.
Critical effect(s):	Kidney epithelial and tubular/ductal hyperplasia
Co-critical effect(s):	Focal papillary edema and necrosis in the kidney
Additivity endpoint(s):	Renal (kidney) system

Cancer Health Based Value (cHBV) = Not Applicable

Cancer classification:	Not Classified
Slope factor (SF):	Not Applicable
Source of cancer slope factor (SF):	Not Applicable
Tumor site(s):	Not Applicable

Volatile: No

Summary of Guidance Value History:

MDH promulgated subchronic and chronic Non-Cancer Health Risk Limits (nHRL) of 9 and 7 µg/L, respectively, in 2011. In 2017, MDH re-evaluated the noncancer HRLs, resulting in new or revised nHBVs for the short-term (3 µg/L), subchronic (3 µg/L), and chronic (2 µg/L) durations. The 2017 values changed from the 2011 HRLs as a result of: 1) using new or revised half-life values to derive chemical-specific human equivalent doses (HEDs), 2) incorporating recently published toxicological studies for short-term guidance derivation, 3) using MDH's most recent risk assessment methodology, and 4) rounding to one significant digit. In 2020 MDH incorporated updated intake rates (US EPA 2019) into the calculations, which did not result in changes to the 2017 values.

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
Effects observed?	Yes[1]	--[2]	Yes[3]	Yes[4]	Yes[5]

Comments on extent of testing or effects:

[1]The oral developmental study in mice included evaluation of serum thyroid hormone levels in mothers and female offspring as well as reproductive hormone levels in offspring. Following *in utero* exposure, pubertal and adult female offspring exhibited decreases in serum estrogen and progesterone levels with elevation of luteinizing hormone levels. In addition, 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 form the basis of the Short-term RfD and were observed at HEDs 400 to 1500-fold higher than the Short-term, Subchronic, and Chronic RfDs. Several other per- and polyfluoroalkyl substances (PFAS) have been reported to alter serum thyroid hormone levels.

An *in vitro* thyroid hormone binding study has also been conducted. PFBS exhibited low potency for binding to the human thyroid hormone transport protein transthyretin.

[2]An epidemiological study, conducted in Taiwanese children, included evaluation of associations between 11 PFAS and immunological markers. Associations were found for several PFAS, including PFBS, with asthma and asthma-related biomarkers. Associations for PFBS were fewer and weaker than those for other PFAS, and in this study, limitations in the data precluded identification of specific PFAS driving the associated effects.

No *in vivo* immunotoxicity studies have been conducted in laboratory animals. *In vitro* studies have demonstrated that while less potent than PFOS, PFBS decreased cytokine release in monocytes.

[3]Two oral developmental studies (one in rats and one in mice) and a 2-generational study in rats have been conducted. The developmental study in mice reported developmental effects in female

offspring exposed *in utero*, including decreased pup body weight, developmental delays (e.g. eye opening, vaginal opening) and latent effects on various female reproductive system parameters (e.g, decreased uterine size and number of ovarian follicles). (The authors also conducted assessments on male offspring but those data have not yet been published.) The effects reported in female offspring observed in the mouse developmental study form the basis of the Short-term RfD and were observed at HEDs 400 to 1500-fold higher than the Subchronic and Chronic RfDs, respectively.

[4]A case-control epidemiology study examined the association between PFAS and endometriosis-related infertility among Chinese women of reproductive age. According to the authors, women with endometriosis-related infertility had significantly higher median levels of PFBS compared to those without the disease. However, due to the nature of the data including lack of physical exam in controls and temporality determination, interpretation of this study is limited in scope.

An oral 2-generation study in rats has been conducted. The average numbers of estrous stages were not significantly different in F1 females, however the number with > 6 consecutive days of diestrus was statistically-significantly increased in a mid-dose group and significantly decreased in the highest dose group. Decreased number of spermatids per gram testes and increased incidence of abnormal sperm were noted at HEDs >3000-fold higher than the Short-term, Subchronic or Chronic RfDs. Mating and fertility parameters were unaffected. Also see footnote 3 above for developmental effects.

[5]Neurological alterations were reported in the 28-day but not the 90-day oral study in rats. The results from the 28-day study are difficult to interpret. Treated males did differ from control males, however, the decreases in tail flick, rotorod latency and foot splay did not exhibit a dose-response at the doses tested. In contrast, treated females exhibited an increase in rotorod latency. The 90-day study, which included some neurotoxicity assessments but not a peripheral neuropathy assessment per se, did not report any treatment-related effects. The effects in the 28-day study occurred at HED levels 300 to 1100-fold higher than the Short-term, Subchronic, or Chronic RfDs.

An *in vitro* neurotoxicity study on undifferentiating and differentiating PC12 cells has been conducted. PFBS exhibited little or no effect on undifferentiating cells but did alter the expression phenotype in differentiating cells.

A database UF was incorporated into the derivation of the Short-term, Subchronic and Chronic RfDs, in part, to address the need for additional neurological testing.

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