



Note: Water guidance for this chemical was updated in May 2017. Please see

<http://www.health.state.mn.us/divs/eh/risk/guidance/gw/table.html#pfoa>

Web Publication Date: 5/4/2009

Chemical Name: Perfluorooctanoic Acid

Synonyms: PFOA

CAS: 335-67-1(free acid)

335-66-0 (acid fluoride)

3825-26-1 (ammonium salt, APFO)

2395-00-8 (potassium salt)

335-93-3(silver salt)

335-95-5 (sodium salt)

The perfluorooctanoate anion does not have a specific CAS number.

Serum concentrations appear to be the best dose-metric for extrapolating to humans. At the present time the information necessary to estimate less than chronic doses (i.e., acute, short-term or subchronic) that would result in a given serum concentration is not available. Additional uncertainty exists regarding toxicokinetics in early life. Therefore, acute, short-term and subchronic HRLs will not be derived at this time.

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

Short-term Non-Cancer Health Risk Limit (nHRL_{short-term}) = Not Derived (Insufficient Data)

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

Chronic Non-Cancer Health Risk Limit (nHRL_{chronic}) = 0.3 ug/L

$$= \frac{(\text{Reference Dose, mg/kg/d}) \times (\text{Relative Source Contribution}) \times (\text{Conversion Factor})}{(\text{Chronic intake rate, L/kg/d})}$$

$$= \frac{(0.000077 \text{ mg/kg/d}) \times (0.2) \times (1000 \text{ ug/mg})}{(0.053^* \text{ L/kg-d})}$$

$$= 0.29 \text{ rounded to } \mathbf{0.3 \text{ ug/L}}$$

* Intake rate used corresponds to the time-weighted average 95th intake rate over first 19 years of life. Nineteen years represents the estimated duration to achieve steady-state serum concentration, based on a half-life of 3.8 years.

Reference Dose: 0.000077 mg/kg-d (Cynomolgus monkeys)

Source of toxicity value: MDH

Point of Departure:	23 mg/L serum concentration (serum BMDL ₁₀) (Thomford et al 2001 and Butenhoff et al 2002)
Human Equivalent Dose Adjustment:	0.0023 mg/kg-d [Dose mg/kg-d = (Ln2/1387 day half-life _{human}) x 23 mg/L x 0.2 L/kg (Vd)]
Total uncertainty factor:	30
UF allocation:	3 interspecies extrapolation for potential differences in toxicodynamics and 10 intraspecies variability
Critical effect(s):	increased relative liver weight
Co-critical effect(s):	increased liver weight with histopathological changes, decreased total serum cholesterol and triglycerides, developmental delays (e.g., altered body weight gain, delayed physical development, hepatocellular hypertrophy) in offspring, altered immune function
Additivity endpoint(s):	Development (body weight, delayed development), Hepatic (liver) system, Immune system
Secondary effect(s):	Increased incidence of full litter resorption, additional developmental delays (e.g., sexual maturation), increased pup mortality, altered mammary gland development, additional immune system effects, increased kidney weight, hematological effects, decreased thyroid hormone (TT4, T3) serum levels, increased serum estradiol levels, increased incidence of benign hepatocellular adenomas, testicular Leydig-cell tumors and pancreatic acinar-cell adenoma/carcinomas

Cancer Health Risk Limit (cHRL) = Not Applicable

Volatile: No

Summary of changes since 1993/1994 HRL promulgation:

No 1993/94 HRL value exists for PFOA. The chronic HRL (0.3 ug/L) is ~1.7-fold lower than the Good-cause exception HRL (0.5 ug/L) adopted August 1, 2007 as the result of using serum levels as the dose metric rather than administered dose.

Summary of toxicity testing for health effects identified in the Health Standards Statute:

	Endocrine	Immunotoxicity	Development	Reproductive	Neurotoxicity
Tested?	Sec. Observations ¹	Yes	Yes	Yes	Yes
Effects?	Yes	Yes ²	Yes ³	Unclear ⁴	Yes ⁵

Note: Even if testing for a specific health effect was not conducted for this chemical, information about that effect may be available from studies conducted for other purposes. Most chemicals have been subject to multiple studies in which researchers identify a dose where no effects were observed, and the lowest dose that caused one or more effects. A toxicity value based on the effect observed at the lowest dose across all available studies is considered protective of all other effects that occur at higher doses.

Comments on extent of testing or effects:

Note – comparisons based on HED LOAEL or HED BMDLs are associated with higher uncertainty than comparisons based on serum levels.

¹ Changes in serum thyroid hormone (e.g., decreased thyroxine, T4 and triiodothyronine, T3) and estradiol levels have been observed in some animal studies but not in others. These changes were observed at estimated human equivalent dose (HED) levels higher but within 3-fold of the critical study HED LOAEL and are therefore identified as secondary effects.

² Short-term immunotoxicity studies have shown that PFOA exposure suppresses humoral immunity and may adversely affect cell mediated immunity at HED doses similar to the critical study HED LOAEL. These effects have been identified as co-critical effects.

³ Developmental delays and body weight/weight gain changes in offspring have been observed at serum and HED dose levels similar to the serum and HED LOAEL of the critical study. These effects have been identified as co-critical effects. At HED doses 3- fold higher than the critical study HED LOAEL additional developmental effects (decreased pup viability, delays in eye opening, increased incidence of full-litter resorption, and alterations in mammary gland development) are observed. Effects occurring at doses approximately 3 fold higher have been identified as secondary effects.

⁴ The results of the 2-generational study indicate that fertility is not affected by treatment. Full-litter resorption was observed at HED dose levels 3-fold higher than the critical study HED LOAEL, however, it is unclear whether this resulted from maternal toxicity or a direct effect on the developing organism. Altered mammary gland development during the lactational period was observed in pregnant/lactating mice exposed to dose levels slightly higher than the critical study LOAEL during pregnancy. Increased incidence of full-litter resorption and alterations in mammary gland development have been identified as a secondary effects.

⁵ Hypoactive response to nicotine has been observed in neonatal mice given a single dose at 10 days of age. No serum level information was reported in this study and it is not possible to extrapolate from a single dose to a HED dose. The additional neurological testing has been recommended by the EPA PFOA draft Risk Assessment Science Advisory Review Board.

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