



CLEAN AIR COUNCIL

**Environmental Quality Board
25 Pa. Code Chapter 250**

**Safe Drinking Water PFAS MCL Rule
Proposed Rulemaking
52 Pa.B. 1245 (February 26, 2022)**

Written Comments of Clean Air Council

April 27, 2022

Via email: RegComments@pa.gov

The Council appreciates the opportunity to provide these written comments on the Proposed Rulemaking of the Environmental Quality Board (“the Board”) for the Department of Environmental Protection (“the Department”). The comments relate to a proposal to set maximum contaminant level goals (MCLG) and maximum contaminant levels (MCL) for drinking water for two per- and polyfluoroalkyl substances (PFAS)—perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS).

The Council is a non-profit environmental health organization headquartered at 135 South 19th Street, Suite 300, Philadelphia, Pennsylvania, 19103. The Council also maintains an office in Pittsburgh. The Council has been working to protect everyone’s right to a clean environment for over 50 years. The Council has members throughout the Commonwealth who support its mission.

In February, the Board published a notice of Proposed Rulemaking. *See* 52 Pa.B. 1245 (February 26, 2022), <http://pacodeandbulletin.gov/secure/pabulletin/data/vol52/52-9/52-9.pdf>. The deadline for comments is April 27, 2022.

These comments include sections for a Table of Comments, Table of Attachments, Summary of Comments, and Comments.

Table of Comments

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 - a. Under regulations of the Agency and guidance from the Department, the Board should propose MCLs as close to the MCLGs as feasible.
 - b. The Department's sampling data for drinking water in Pennsylvania demonstrate a significant number of exceedances of the recommended MCLGs for PFOA and PFOS.
 - c. Exposure to PFOA and PFOS at levels above the recommended MCLGs is dangerous for humans and leads to adverse health effects, especially in sensitive populations such as children.
 - d. Analytic methods for detection of PFOA and PFOS are available and they can identify levels well below the recommended MCLGs.
 - e. Proposed MCLs for PFOA and PFOS equal to the recommended MCLGs would be technically feasible.
 - f. In proposing to deviate from the MCLGs, the Board relies on false comparisons with other Department practices involving the number 90%.
 - i. The use of the number 90% in the regulation of Giardia cysts does not provide a reasonable basis for proposing MCLs that are higher than the recommended MCLGs.
 - ii. The use of the 90th percentile in the lead and copper rule does not provide a reasonable basis for proposing MCLs that are higher than the recommended MCLGs.
 - iii. The use of the number 95% for turbidity does not provide a reasonable basis for proposing MCLs that are higher than the recommended MCLGs.
 - g. The benefits of setting MCLs at levels equal to the recommended MCLGs would vastly exceed costs.
 - h. The Board unreasonably relies on a flawed analysis of cost-effectiveness.

2. The Board should propose MCLs for other PFAS chemicals (PFBS, PFHpA, PFHxS, PFNA) at values that are not higher than the MCLGs recommended by the Drexel PFAS Advisory Group.
 - a. The Drexel PFAS Advisory Group identified these compounds as harmful to health and determined MCLG values that are feasible.
 - b. Setting MCLs for PFOA and PFOS would not be sufficient to protect against harm from these other compounds.
 - c. The Board erroneously excluded these compounds under the flawed rationale that cost/benefit data and analysis applied are incomplete.
 - d. The Board should propose an MCL for PFHpA because there is evidence of toxicity.
 - e. The Board should propose MCLs for these compounds because they do not necessarily co-occur with PFOA and PFOS.
 - f. Together with PFOA and PFOS, these compounds fall under EPA's definition of "dose additivity," which means that their presence at unregulated levels would magnify the adverse health effects of PFOA and PFOS.
 - g. MCLs based on the recommended MCLGs are feasible, cost effective, and can be implemented using methodologies endorsed by the Department.

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Attachment 8	Francisca Pérez, Martí Nadal, Navarro-Ortega, et al Accumulation of perfluoroalkyl substances in human tissues, <i>Environment International</i> , 59,2013, https://doi.org/10.1016/j.envint.2013.06.004 (open access)
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Attachment 16	Liu, et al, Rejection of per- and polyfluoroalkyl substances (PFASs) in aqueous film-forming foam by high-pressure membranes, Water Research 188, 2021, 116546 https://doi.org/10.1016/j.watres.2020.116546 (abstract online)
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Attachment 29	Kim M, Park MS, Son J, Park I, Lee H, Kim C, Min B, Ryoo J, Choi KS, Lee D, Lee D, et al: Perfluoroheptanoic acid affects amphibian embryogenesis by inducing the phosphorylation of ERK and JNK. Int J Mol Med 36: 1693-1700, 2015, https://pubmed.ncbi.nlm.nih.gov/26459765/ , available at https://www.spandidos-publications.com/ijmm/36/6/1693 (full article online).
Attachment 30	Zengqiang Li, Changchang Li, Zina Wen, Haoni Yan, Cheng Zou, Yang Li, Lili Tian, Zhen Lei, Huitao Li, Yiyan Wang, Ying Zhong, Ren-shan Ge, Perfluoroheptanoic acid induces Leydig cell hyperplasia but inhibits spermatogenesis in rats after pubertal exposure, Toxicology, Volume 448, 2021, 152633, ISSN 0300-483X, https://doi.org/10.1016/j.tox.2020.152633 (abstract online)
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Summary of Comments

The Council supports the notion of setting MCLs for PFAS compounds – but the Board should propose them at the appropriate levels and MCLs should apply to additional compounds presenting harm to public health, as well as perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS). The proposed rulemaking is flawed in at least two ways.

First, in the case of PFOA and PFOS, the Board unreasonably has proposed to set MCLs that are higher than the MCLGs recommended by the Drexel PFAS Advisory Group. In the case of PFOA, the Board proposes an MCL of 14 ppt even though the recommended MCLG is 8 ppt. In the case of PFOS, it proposes an MCL of 18 ppt even though the recommended MCLG is 14 ppt.

The Department's sampling data for drinking water in Pennsylvania demonstrate a significant number of exceedances of these MCLGs. Exposure above these levels is dangerous for humans and leads to adverse health effects, especially in sensitive populations such as children.

The Board is required to set MCLs as close to the MCLGs as feasible. MCLs equal to these MCLGs would be technically feasible. Analytic methods for detection of PFOA and PFOS are available and they can identify levels well below the recommended MCLGs.

To justify deviating from the MCLGs (which the Board defines as a 100% improvement from the Environmental Protection Agency's Health Advisory Level of 70 ppt), the Board relies on false comparisons with other Department practices involving the number 90%.

The benefits of setting MCLs at levels equal to the recommended MCLGs would vastly exceed costs. The Board ignores this and instead relies on a flawed analysis of cost-effectiveness.

Second, the Board should propose MCLs for other PFAS chemicals for which the Drexel PFAS Advisory Group recommended MCLGs (PFBS, PFHpA, PFHxS, PFNA). In doing so, the Drexel PFAS Advisory Group identified these compounds as harmful to health. Setting MCLs for PFOA and PFOS would not be sufficient to protect against harm from these other compounds, which do not necessarily co-occur with PFOA and PFOS.

The Board erroneously excluded these compounds under the flawed rationale that cost/benefit data and analysis applied are incomplete.

The Board should propose an MCL for PFHpA because its chemical structure is similar to those of PFOA and PFOS, and the medical studies have provided evidence of toxicity of PFHpA.

Health study data demonstrate that these additional compounds fall under the Agency's definition of "dose additivity" when present along with PFOA and PFOS. Their presence at unregulated levels would magnify the adverse health effects of PFOA and PFOS even when the latter two compounds are below the MCLGs.

The MCLGs recommended by the Drexel PFAS Advisory Group are feasible, cost effective, and can be implemented as MCLs using methodologies endorsed by the Department.

Comments

1. The Board should set Maximum Contaminant Levels (MCLs) at levels that are not higher than Maximum Contaminant Level Goals (MCLGs) for PFOA and PFOS recommended by the Drexel PFAS Advisory Group.

The Department guidelines for setting MCLs quote the Agency process as follows:

REGULATORY LEVELS

The 1996 Amendments to the Safe Drinking Water Act (SDWA) require EPA to go through several steps to determine, first, whether setting a standard is appropriate for a particular contaminant, and if so, what the standard should be. Peer-reviewed science and data support an intensive technological evaluation that includes many factors, such as:

1. Occurrence in the environment.
2. Human exposure and risks of adverse health effects in the general population and sensitive subpopulations.
3. Analytical methods of detection.
4. Technical feasibility.
5. Impacts of regulation on water systems, the economy and public health.

See Attachment 1 – Pennsylvania Department of Environmental Protection, [Health Effects and Risk Management Guidance](#) (Document 383-0400-104), (October 4, 2003), page 4.

The proposed rule sets MCLs for PFOA and PFOS at values higher than the MCLGs, apparently based on items (4) and (5):

PFOA—proposed MCL of 14 ng/L

The Board is proposing an MCL of 14 ng/L for PFOA. The proposed MCL is based on the health effects and proposed MCLG, occurrence data, technical feasibility, and costs and benefits.

...

PFOS—proposed MCL of 18 ng/L

The Board is proposing an MCL of 18 ng/L for PFOS. The proposed MCL is based on the health effects and proposed MCLG, occurrence data, technical feasibility, and costs and benefits.

See Proposed Rulemaking, pages 1251, 1254 (highlighting added for emphasis).

In light of the Department’s own guidance document and Environmental Protection Agency (“Agency”) procedures for MCLs under the federal Safe Drinking

Water Act, the Board should propose MCLs at levels that are not higher than MCLGs for PFOA and PFOS. The Board’s proposal to set MCLs at levels that are higher than MCLGs for PFOA and PFOS is unreasonable as a matter of law.

- a. Under regulations of the Agency and guidance from the Department, the Board should propose MCLs as close to the MCLGs as feasible.

The Drexel PFAS Advisory Group was “engaged by the Commonwealth of PA to provide ... recommendations for Maximum Allowable Contaminant Level Goals MCLGs to the Commonwealth of Pennsylvania for Per- and polyfluoroalkyl substances (PFAS) in drinking water.” See Attachment 2 – Drexel PFAS Advisory Group Report, page 5. It recommended MCLGs of 8 ppt and 14 ppt for PFOA and PFOS, respectively:

Table 1.

PFAS	Reference Dose	MCLG proposed
perfluorooctanoic acid (PFOA)	3.9 ng/kg/day	8 PPT
perfluorooctanesulfonic acid (PFOS)	3.1 ng/kg/day	14 PPT
perfluorononanoic acid (PFNA)	2.2 ng/kg/day	6 PPT
perfluorohexanesulfonic acid (PFHxS)	4.0 ng/kg/day	20 PPT
perfluoroheptanoic acid (PFHpA)	None derived	8 PPT
perfluorobutanesulfonic acid (PFBS)	39 ng/kg/day	55 PPT
ammonium salt of hexafluoropropylene oxide dimer (GenX)	75 ng/kg/day	108 PPT

Table 1: Summary of Reference Dose and proposed Chronic Non-Cancer MCLG for perfluorononanoic acid (PFNA), perfluorooctanesulfonic acid (PFOS), perfluorooctanoic acid (PFOA), perfluorohexanesulfonic acid (PFHxS), perfluoroheptanoic acid (PFHpA), perfluorobutanesulfonic acid (PFBS), and the ammonium salt of hexafluoropropylene oxide dimer (GenX)

Attachment 2 – Drexel PFAS Advisory Group, Maximum Contaminant Level Goal Drinking Water Recommendations for Per and Polyfluoroalkyl Substances (PFAS) in the Commonwealth of Pennsylvania (January 2021) (“Drexel PFAS Advisory Group Report”), page 7 (highlighting added for emphasis).

The Board has agreed with the MCLGs proposed by the Drexel PFAS Advisory Group. See Proposed Rulemaking, page 1251 (“The Board is proposing to set the MCLG for PFOA at the DPAG recommended level of 8 ng/L”), 1254 (“The Board is proposing to set the MCLG for PFOS at the DPAG recommended level of 14 ng/L”).

The federal Safe Drinking Water Act requires that MCLs be set “as close to the maximum contaminant level goal as is feasible”:

(4) GOALS AND STANDARDS.-

(A) MAXIMUM CONTAMINANT LEVEL GOALS.-Each maximum contaminant level goal established under this subsection shall be set at the level at which no known or anticipated adverse effects on the health of persons occur and which allows an adequate margin of safety.

(B) MAXIMUM CONTAMINANT LEVELS.-Except as provided in paragraphs (5) and (6), each national primary drinking water regulation for a contaminant for which a maximum contaminant level goal is established under this subsection shall specify a maximum contaminant level for such contaminant which is as close to the maximum contaminant level goal as is feasible.

See 42 U.S.C. §300g-1(b)(4)(A)-(B).

This is repeated in the Department’s guideline document for setting drinking water MCLs:

Determine Maximum Contaminant Level (MCL) or Treatment Technique (TT)

Once the MCLG is determined, EPA develops an enforceable standard. In most cases, the standard is a **Maximum Contaminant Level (MCL)**, the maximum permissible level of a contaminant in water, which is delivered to any user of a public water system. The SDWA, as amended in 1996, requires EPA to set the MCL as close to the MCLG as feasible, which the SDWA defines as the level that may be achieved with the use of the best available technology. Factors considered while setting MCLs include analytical and treatment feasibility, costs to large metropolitan and regional water systems, and national economic impact. **For noncarcinogens and equivocal-evidence carcinogens, the MCL is usually set at the MCLG.** For group A and B carcinogens, the target range for setting the MCL is between the 10^{-4} and 10^{-6} excess cancer risk level. If it is economically or technically unfeasible to determine the concentration level of a contaminant in water, a treatment technique can be set for the contaminant in place of an MCL.

Attachment 1 – Pennsylvania Department of Environmental Protection, [Health Effects and Risk Management Guidance](#) (October 4, 2003), page 6 (highlighting added for emphasis).

The Department repeated this principle in its response to the rulemaking petition of Delaware Riverkeeper Network:

Once the MCLG is determined, EPA sets an enforceable standard. In most cases, the standard is an MCL. **The MCL is set as close to the MCLG as feasible.** Taking cost into consideration, **EPA must determine the feasible MCL.** This is defined by the Federal SDWA as the level that may be achieved with:

- use of the best available technology or treatment approaches
- other means which EPA finds are available (after examination for efficiency under field conditions, not solely under laboratory conditions)

See Attachment 3 – Pennsylvania Department of Environmental Protection, Evaluation Report on the Delaware Riverkeeper Network Petition for Rulemaking to Set an MCL for PFOA (April 16, 2021), page 20.

In the proposed rule, the Board also stated that the MCL is set “as close to the MCLG as feasible”:

Once the MCLG is determined, the EPA sets an enforceable standard. **In most cases, the standard is an MCL. The MCL is set as close to the MCLG as feasible.** Taking cost into consideration, **the EPA must determine the feasible MCL.**

See Proposed Rulemaking, pages 1249-1250 (highlighting added for emphasis).

But the Board did not do this when it proposed MCLs for PFOA and PFOS at values higher than the MCLGs.

- b. The Department's sampling data for drinking water in Pennsylvania demonstrate a significant number of exceedances of the recommended MCLGs for PFOA and PFOS.

Based on sampling undertaken between summer 2019 and March 2021, the Department found that PFOA was detected in 27% of the samples and PFOS was detected in 25% of the samples. *See* Proposed Rulemaking, page 1247, Table 1 ("Summary of PFAS Sampling Plan results"). *See also* Attachment 4 – Pennsylvania Department of Environmental Protection, [Summary of Results for SDW Sampling Project Using EPA Method 537.1](#) (2020-2021). Extending these values to the entire drinking water system would mean that at least one-quarter of drinking water in Pennsylvania contains PFOA or PFOS.

In addition, the Department estimates that 400 of 3,785 Entry Points (EPs) in Pennsylvania (that is, more than 10% of them) exceed the MCLG for PFOA. *See* Proposed Rulemaking, page 1252 (Table 8, PFOA Comparison of Annual Costs and Benefits). It estimates that 200 of these Entry Points (more than 5% of them) exceed the MCLG for PFOA. *See* Proposed Rulemaking, page 1255 (Table 12, PFOS Comparison of Annual Costs and Benefits).

Clearly, the incidence of PFOA and PFOS above MCLGs in Pennsylvania drinking water affects a substantial fraction of the population.

- c. Exposure to PFOA and PFOS at levels above the recommended MCLG is dangerous for humans and leads to adverse health effects, especially in sensitive populations such as children.

The adverse health effects of PFOA and PFOS in drinking water have been demonstrated in numerous research articles and Federal Government documents. *See* Attachment 5 – Agency for Toxic Substances and Disease Registry, [Toxicological Profile for Perfluoroalkyls](#) (Released May 2021, Last Updated March 2020), chapters 1-3; *See* Attachment 6 – U.S. Environmental Protection Agency, [Drinking Water Health Advisories for PFOA and PFOS](#).

As noted by the Drexel PFAS Advisory Group (engaged by the Department to determine MCLGs for PFAS in drinking water), the purpose of an MCL is to protect human health:

Maximum Contaminant Level Goals (MCLGs) are maximum drinking water concentrations *designed to protect human health*. MCLGs are non-enforceable as they are chosen solely based on protection of human health

See Attachment 2 – Drexel PFAS Advisory Group Report, page 13. It reiterated this point in its report:

DPAG purposely sought to maintain an independent mindset with developing these MCLGs *and to focus on identifying concentrations that would protect human health.*

See *id.*, page 6. That report proceeds to outline the different health risks associated with PFOA and PFOS forming the basis for the recommended MCLGs. See *id.*, pages 22-31 (Section 4, PFOA), pages 32-39 (Section 5, PFOS).

The harm that would result from exposure to MCLs that are higher than the MCLG would disproportionately affect the most vulnerable population of newborns and children, since they will remain exposed to the harmful compounds for their entire lifetime.

The effects of PFAS on health are cumulative and long-lasting. Studies show that the half-life of PFOA and PFOS is of order 2.5 years or more after end of exposure. See Attachment 7 – Li, Ying et al. “Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water.” *Occupational and environmental medicine* vol. 75, 1 (2018): 46-51. doi:10.1136/oemed-2017-104651, available at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5749314/> (open access).

In fact, PFAS compounds have been shown to be prevalent in autopsy tissues including those in the brain, liver, lung, bone, and kidney:

In this study, the concentrations of 21 PFASs were analyzed in 99 samples of autopsy tissues (brain, liver, lung, bone, and kidney) from subjects who had been living in Tarragona (Catalonia, Spain). The samples were analyzed by solvent extraction and online purification by turbulent flow and liquid chromatography coupled to tandem mass spectrometry. The occurrence of PFASs was confirmed in all human tissues.

See Attachment 8 – Francisca Pérez, Martí Nadal, Navarro-Ortega, et al Accumulation of perfluoroalkyl substances in human tissues, *Environment International*, 59,2013, <https://doi.org/10.1016/j.envint.2013.06.004> (open access).

Analysis of the health impacts of various PFAS compounds shows a range of adverse outcomes to fetuses, infants, and children. See Attachment 5 – Agency for Toxic Substances and Disease Registry, [Toxicological Profile for Perfluoroalkyls](#) (Released May 2021, Last Updated March 2020), chapters 1-3, and in particular pages 629-632.

The effects of PFAS exposure during pregnancy and in infancy were found to impact health later in life in adolescents and adults. See Attachment 9 – Blake et al,

“Early life exposure to per- and polyfluoroalkyl substances (PFAS) and latent health outcomes: A review including the placenta as a target tissue and possible driver of peri- and postnatal effects.” *Toxicology* vol. 443 (2020): 152565. doi:10.1016/j.tox.2020.152565, available at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7530144/>

In determining MCLGs for PFOA and PFOS (as well as other PFAS compounds), the Drexel PFAS Advisory Group relied on the Goeden Model, which accounts for the fact that the relative source contribution of water is higher early in life:

This approach considers water consumption from conception to adulthood and adjusts for the fact that relative source contribution of water is higher early in life. It assumes that a child will have a certain level of exposure in-utero because of the PFA in the mother’s body and further exposure during breastfeeding or bottle feeding.

See Attachment 2 – Drexel PFAS Advisory Group Report, page 18 (bold italics added for emphasis). The report states that “[t]he model had sufficient data for application to MCLG recommendations for PFOA, PFOS, PFNA, and PFHxS.” *Id.*

It is also significant that infants and children consume more water per body weight. See Attachment 10 – Faizan U, Rouster AS. Nutrition and Hydration Requirements In Children and Adults. [Updated 2021 Sep 2]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK562207/> (CC BY 4.0). As a result, their effective intake is higher. This increases their vulnerability to the harmful effects of exposure.

For all these reasons (developmental exposure in utero, long half-life of PFAS in the body, higher effective intake for children, and cumulative impacts over a lifetime), the Board’s proposal to set MCLs that are higher than the recommended MCLGs would especially harm this vulnerable population.

- d. Analytic methods for detection of PFOA and PFOS are available and they can identify levels well below the recommended MCLGs.

The Department endorses two laboratory methods for the monitoring of PFOA and PFOS. These (Method 533 and Method 537.1) come from the Agency. See Attachment 11 – U.S. Environmental Protection Agency, [EPA Method 533](#), page 533-1; see also Attachment 12 – U.S. Environmental Protection Agency, [EPA Method 537.1](#), page 537.1-2.

There is no technical difficulty in detecting PFOA and PFOS at levels below the recommended MCLGs. As reflected in the proposed rule, the Minimum Reporting

Level (MRL) set by the Department for these two methods (5 ppt) is well below the MCLG for PFOA (8 ppt) and the MCLG for PFOS (14 ppt):

(1) Sampling and analysis shall be according to the following approved methods and MRLs:

<i>Contaminant</i>	<i>Methods</i>	<i>MRL (ng/L)</i>
<u>(i) PFOA</u>	<u>EPA 533, EPA 537.1, EPA 537 Version 1.1</u>	<u>5</u>
<u>(ii) PFOS</u>	<u>EPA 533, EPA 537.1, EPA 537 Version 1.1</u>	<u>5</u>

See Proposed Rulemaking, page 1271 (highlighting added for emphasis).

Therefore, the level of the MRL does not present a problem of feasibility for a proposed MCL equal to the recommended MCLGs.

- e. Proposed MCLs for PFOA and PFOS equal to the recommended MCLGs would be technically feasible.

The treatment methods endorsed by the Agency and the Department for the removal of PFOA and PFOS from drinking water are capable of reducing concentrations to levels below the MCLGs recommended by the Drexel PFAS Advisory Group.

The Agency states that the treatment technologies are up to 99% effective:

The following processes were found to be effective for the removal of perfluorooctanoic acid: GAC (up to > 99 percent removal), membrane separation &ndash high pressure membranes such as nanofiltration and reverse osmosis (up to > 99 percent removal), anion exchange (up to 99 percent removal), and powdered activated carbon (up to 95 percent removal).

Attachment 13 – U.S. Environmental Protection Agency, [Perfluorooctanoic Acid: Treatment Processes](#) (PFOA) (click “Treatment Processes” tab).

The following processes were found to be effective for the removal of PFOS: GAC (up to > 99 percent removal), membrane separation - high pressure membranes such as nanofiltration and reverse osmosis (up to > 99 percent removal), anion exchange (up to > 99 percent removal), and powdered activated carbon (up to 99 percent removal).

Attachment 14 – U.S. Environmental Protection Agency, [Perfluorooctane Sulfonate: Treatment Processes](#) (PFOS) (click “Treatment Processes” tab).

Experiments and pilot processes show that it is possible to remove PFOA and PFOS down to non-detectable levels. A full-scale drinking water treatment plant in Uppsala, Sweden over a period of two years (2015–2017) found that PFOA and PFOS removal was 100% by six different GAC systems for long periods of time (bed volumes of at least 10,000- see Fig 3). See Attachment 15 – Belkouteb, et al, Removal of per- and polyfluoroalkyl substances (PFASs) in a full-scale drinking water treatment plant: Long-term performance of granular activated carbon (GAC) and influence of flow-rate, Water Research, 182, 2020, available at <https://www.sciencedirect.com/science/article/pii/S0043135420304504> (open access).

Similarly effective results can also be achieved using membranes. For example, a study observed rejection rates for PFOA and PFOS higher than 99% for reverse osmosis and nanofiltration membranes, and “***Rejection of the full suite of PFAAs*** was consistently >98% by NF and >99% by RO indicating operating conditions did not have a significant impact on rejection.” See Attachment 16 – Liu, et al, Rejection of per- and polyfluoroalkyl substances (PFASs) in aqueous film-forming foam by high-pressure membranes, Water Research 188, 2021, 116546 <https://doi.org/10.1016/j.watres.2020.116546> (abstract online) (bold italics added for emphasis).

For the notice of proposed rulemaking, the sampling data show that the maximum value of PFOA detected was 59.6 ppt, and the maximum value of PFOS detected was 187.1 ppt:

Table 1. Summary of PFAS Sampling Plan results. Full results available at www.dep.pa.gov/pfas

Summary of PFAS Sampling Plan Results							
	PFOA	PFOS	PFNA	PFHxS	PFHpA	PFBS	Units
Total No. Samples	412	412	412	412	412	412	—
Average	2.0	2.5	0.4	1.4	0.7	1.1	ng/L
Median	0 (ND)	0 (ND)	0 (ND)	0 (ND)	0 (ND)	0 (ND)	ng/L
Minimum	0 (ND)	0 (ND)	0 (ND)	0 (ND)	0 (ND)	0 (ND)	ng/L
Maximum	59.6	187.1	18.1	140.0	32.6	64.0	ng/L
No. and % of Detects	112 (27%)	103 (25%)	23 (6%)	52 (13%)	49 (12%)	66 (16%)	—
Avg Detect Value	7.5	9.9	7.2	10.9	6.1	7.0	ng/L
Med Detect Value	5.3	6.5	5.6	4.5	4.5	4.2	ng/L
Min Detect Value	1.7	1.8	1.8	1.9	1.8	1.7	ng/L
Max Detect Value	59.6	187.1	18.1	140.0	32.6	64.0	ng/L

See Proposed Rulemaking, page 1247. A 99% effective treatment method would reduce such levels to well below the MRL of 5 ppt. (187.1 ppt x 1% = 1.871 ppt).

Indeed, the Department’s own analysis demonstrates that the MCLGs can be achieved through treatment – it just costs a little more:

Table 8. PFOA Comparison of Annual Costs and Benefits

PFOA Annual Costs and Benefits Analysis								
Value (ng/L)	Estimated # of EPs (of 3,785) > Value	Compliance Monitoring Costs (Millions)	Treatment O&M Costs		Treatment Capital Costs (Millions) per MGD* annualized over 20 years	Total Costs (Millions)	% Increase in Cost Compared to HAL	% Improvement in Health Protection Compared to HAL
			Treatment O&M Costs (Millions) per MGD*	Performance Monitoring Costs (Millions)				
HAL = 70	58	\$2.46	\$9.50	\$1.29	\$14.39	\$27.63	0%	0%
35	78	\$2.56	\$12.78	\$1.73	\$19.35	\$36.41	32%	56%
20	200	\$2.73	\$32.76	\$4.44	\$49.60	\$89.53	224%	80%
MCL = 14	218	\$2.89	\$35.71	\$4.83	\$54.07	\$97.51	253%	90%
12	270	\$2.97	\$44.23	\$5.99	\$66.97	\$120.15	335%	93%
10	313	\$3.07	\$51.28	\$6.94	\$77.63	\$138.92	403%	96%
MCLG = 8	400	\$3.39	\$65.53	\$8.87	\$99.21	\$177.00	541%	100%

*For purposes of totaling annual costs, the costs that vary with design capacity (treatment O&M and treatment capital costs) were multiplied by a benchmark design capacity of 1 MGD.

See Proposed Rulemaking, page 1252 (PFOA).

Table 12. PFOS Comparison of Annual Costs and Benefits

PFOA Annual Costs and Benefits Analysis								
Value (ng/L)	Estimated # of EPs (of 3,785) > Value	Compliance Monitoring Costs (Millions)	Treatment O&M Costs		Treatment Capital Costs (Millions) per MGD* annualized over 20 years	Total Costs (Millions)	% Increase in Cost Compared to HAL	% Improvement in Health Protection Compared to HAL
			Treatment O&M Costs (Millions) per MGD*	Performance Monitoring Costs (Millions)				
HAL = 70	96	\$2.57	\$15.73	\$2.13	\$23.81	\$44.24	—	—
35	148	\$2.64	\$24.25	\$3.28	\$36.71	\$66.87	51%	63%
20	183	\$2.70	\$29.98	\$4.06	\$45.39	\$82.13	86%	89%
MCL = 18	191	\$2.70	\$31.29	\$4.24	\$47.37	\$85.60	94%	93%
16	200	\$2.73	\$32.76	\$4.44	\$49.60	\$89.53	102%	96%
15	200	\$2.81	\$32.76	\$4.44	\$49.60	\$89.61	103%	98%
MCLG = 14	200	\$2.88	\$32.76	\$4.44	\$49.60	\$89.68	103%	100%

*For purposes of totaling annual costs, the costs that vary with design capacity (treatment O&M and treatment capital costs) were multiplied by a benchmark design capacity of 1 MGD.

See Proposed Rulemaking, page 1255 (PFOS).¹

Therefore, setting MCLs for PFOA and PFOS at levels equal to their recommended MCLGs would be technically feasible.

- f. In proposing to deviate from the recommended MCLGs, the Board relies on false comparisons with other Department practices involving the number 90%.

In the notice, the Board unreasonably states that setting the MCLs for PFOA and PFOS at values that are 90% of the difference between the Agency’s Health Advisory Level and the MCLGs would be consistent with other practices where the Department has used the number 90%:

- the requirement to achieve at least a 90% inactivation of *Giardia* cysts using disinfection processes within a filtration plant (§ 109.202(c)(1)(ii) (relating to State MCLs, MRDLs and treatment

¹ The reference to “PFOA” in the title of this Table appears to be a typographical error. It should read “PFOS.”

technique requirements) regarding treatment technique requirements for pathogenic bacteria, viruses and protozoan cysts);

- the use of the 90th percentile lead and copper levels when determining compliance with the lead and copper action levels of 0.015 mg/L and 1.3 mg/L, respectively (§ 109.1102(a) (relating to action levels and treatment technique requirements) regarding action levels for lead and copper), and
- the requirement to meet the filtered water turbidity standards in 95% of measurements taken each month (§ 109.202(c)(1)(i)).

See Proposed Rulemaking, pages 1252-1253, 1255-1256. The Board is relying on numbers taken out of context, which are inapplicable to this proposed rulemaking for PFOA and PFOS.

- i. The use of the number 90% in the regulation of *Giardia* cysts does not provide a reasonable basis for proposing MCLs that are higher than the recommended MCLGs.

The Board unreasonably relies on a false comparison with the regulation of *Giardia* cysts, which are the subject of different regulations for protozoan cysts. See [25 Pa. Code §109.202\(c\)](#) (“Treatment technique requirements for pathogenic bacteria, viruses and protozoan cysts”). This is a false comparison because the mechanism by which such pathogens affect human health is different from the mechanism for toxicants such as PFAS, both as to their prevalence in drinking water and how they affect the human body.

Moreover, within the very regulatory subsection cited by the Department (§ 109.202(c)(1)(ii)), the Department cherry-picks the regulatory percentages as a basis for comparison:

(ii) The combined total effect of disinfection processes utilized in a filtration plant shall:

(A) Achieve at least 1.0-log inactivation of *Giardia* cysts and 3.0-log inactivation of viruses as demonstrated by measurements taken under § 109.301(1). Failure to maintain the minimum log inactivation for more than 4 hours of operation constitutes a breakdown in treatment.

(B) Provide a minimum residual disinfectant concentration of 0.20 mg/L at the entry point as demonstrated by measurements taken under § 109.301(1). Failure to maintain the minimum entry point residual disinfectant concentration for more than 4 hours of operation is a treatment technique violation.

See *id.*, 25 Pa. Code §109.202(c)(1)(ii). It relies on regulatory language requiring a 1.0-log inactivation of *Giardia* cysts. This is equivalent to the 90% inactivation that was cited by the Department:

Common log-inactivation values and corresponding percent inactivation values include:

<i>Log Inactivation</i>	<i>Percent Inactivation</i>
0.5-log	68.4%
1.0-log	90.0%
1.5-log	96.8%
2.0-log	99.0%
2.5-log	99.7%
3.0-log	99.9%
4.0-log	99.99%

See *id.*, 25 Pa. Code Section 109.1 (definition of “Log inactivation”). But the very same sentence in §109.202(c)(1)(ii) also requires “3.0-log inactivation of viruses.” According to the table above, this requires a 99.9% inactivation of viruses. The Department does not explain why it should not borrow the number 99.9% as a basis for comparison, rather than 90%.

Moreover, the Department ignores the fact that these regulations also require at least 99.9% removal and inactivation of *Giardia lamblia* cysts and at least 99% removal of *Cryptosporidium* oocysts:

(c) *Treatment technique requirements for pathogenic bacteria, viruses and protozoan cysts.* A public water system shall provide adequate treatment to reliably protect users from the adverse health effects of microbiological contaminants, including pathogenic bacteria, viruses and protozoan cysts. The number and type of treatment barriers and the efficacy of treatment provided shall be commensurate with the type, degree and likelihood of contamination in the source water.

(1) A public water supplier shall provide, as a minimum, continuous filtration and disinfection for surface water and GUDI sources. The treatment technique must provide at least 99.9% removal and inactivation of *Giardia lamblia* cysts, and at least 99.99% removal and inactivation of enteric viruses. Beginning January 1, 2002, public water suppliers serving 10,000 or more people shall provide at least 99% removal of *Cryptosporidium* oocysts. Beginning January 1, 2005, public water suppliers serving fewer than 10,000 people shall provide at least 99% removal of *Cryptosporidium* oocysts. The Department, depending on source water quality conditions, may require additional treatment as necessary to meet the requirements of this chapter and to protect the public health.

See *id.*, 25 Pa. Code §109.202(c)(1). Again, the Department does not explain why it should not borrow the number 99.9% as a basis for comparison, rather than 90%.

Of course, there is no reasonable basis for the Department choosing one or the other percentage to make standards for PFOA and PFOS less stringent. The entire premise is flawed. The Board should not rely on an unrelated regulation to justify proposing MCLs that are higher than MCLGs.

- ii. The use of the 90th percentile in the lead and copper rule does not provide a reasonable basis for proposing MCLs that are higher than the recommended MCLGs.

Similarly, the comparison between the lead and copper rule and PFAS is false. The Drexel PFAS Advisory Group has recommended MCLGs for PFOA and PFOS for

the source water, as a basis for setting MCLs. In contrast, there is no MCL for lead, which is regulated in a different manner because of the nature of lead contamination in drinking water. A major concern for lead contamination in drinking water lies in the piping to the tap of the consumer, rather than in the source water itself:

For most contaminants, EPA sets an enforceable regulation called a maximum contaminant level (MCL) based on the MCLG. MCLs are set as close to the MCLGs as possible, considering cost, benefits and the ability of public water systems to detect and remove contaminants using suitable treatment technologies.

However, because lead contamination of drinking water often results from corrosion of the plumbing materials belonging to water system customers, EPA established a treatment technique rather than an MCL for lead. A treatment technique is an enforceable procedure or level of technological performance which water systems must follow to ensure control of a contaminant.

See Attachment 17 – U.S. Environmental Protection Agency, [Basic Information about Lead in Drinking Water: Drinking Water Requirements for Lead](#) (bold italics added for emphasis).

Accordingly, the regulation of lead in drinking water involves the setting of action levels and detailed requirements for monitoring the tap water:

§ 109.1102. Action levels and treatment technique requirements.

(a) *Action levels for lead and copper.*

(1) The lead action level is 0.015 mg/L.

(2) The copper action level is 1.3 mg/L.

(3) **An action level is exceeded when the concentration of a contaminant in more than 10% of tap water samples collected during a monitoring period conducted in accordance with § 109.1103 (relating to monitoring requirements) is greater than the action level.**

(4) The 90th percentile lead and copper levels shall be computed as follows:

(i) **The results of all lead or copper samples taken during a monitoring period shall be placed in ascending order from the sample with the lowest concentration to the sample with the highest concentration.** Each sampling result shall be assigned a number, ascending by single integers beginning with the number 1 for the sample with the lowest contaminant level. The number assigned to the sample with the highest contaminant level shall be equal to the total number of samples taken.

(ii) The number of samples taken during the monitoring period shall be multiplied by 0.9.

(iii) The contaminant concentration in the numbered sample yielded by the calculation in subparagraph (ii) is the 90th percentile contaminant level.

(iv) For water systems that collect five samples per monitoring period, the 90th percentile is computed by taking the average of the highest and second highest concentrations.

(v) Interpolation shall be used to compute the 90th percentile when the numbered sample indicated in subparagraph (iii) is not a whole number.

See 25 Pa. Code §109.1102(a) (highlighting added for emphasis).

The Board should not deviate from the practice of setting the MCLs “as close to the MCLGs as possible” based on a different regulatory approach for a different contaminant with different chemical and physical properties.

- iii. The use of the number 95% for turbidity does not provide a reasonable basis for proposing MCLs that are higher than the recommended MCLGs.

Finally, the Department makes a false comparison between turbidity (which is not a chemical) and PFAS compounds (which are toxicants). The Department compounds the problem of cherry-picking Section 109.202(c)(1)(ii) relating to Giardia cysts (see subcomment above) by citing the number 95% in Section 109.202(c)(1)(i) – which provides requirements for turbidity in that same regulatory section:

(i) The filtration process shall meet the following performance requirements:

(A) *Conventional or direct filtration.*

(I) The filtered water turbidity shall be less than or equal to .5 NTU in 95% of the measurements taken each month under § 109.301(1) (relating to general monitoring requirements).

(II) The filtered water turbidity shall be less than or equal to 2.0 NTU at all times, measured under § 109.301(1).

(III) Beginning January 1, 2002, for public water systems serving 10,000 or more persons, the filtered water turbidity shall meet the following criteria:

(-a-) Be less than or equal to 0.3 NTU in at least 95% of the measurements taken each month under § 109.301(1).

(-b-) Be less than or equal to 1 NTU at all times, measured under § 109.301(1).

(IV) Beginning January 1, 2005, for public water systems serving fewer than 10,000 persons, the filtered water turbidity shall meet the following criteria:

(-a-) Be less than or equal to 0.3 NTU in at least 95% of the measurements taken each month under § 109.301(1).

(-b-) Be less than or equal to 1 NTU at all times, measured under § 109.301(1).

(B) *Slow sand or diatomaceous earth filtration.*

(I) The filtered water turbidity shall be less than or equal to 1.0 NTU in 95% of the measurements taken each month under § 109.301(1).

(II) The filtered water turbidity shall be less than or equal to 2.0 NTU at all times, measured under § 109.301(1).

(C) *Membrane filtration.*

(I) Beginning August 20, 2019, for all public water systems, the filtered water turbidity must be less than or equal to 0.15 NTU in at least 95% of the measurements taken each month under § 109.301(1).

(II) Beginning August 20, 2019, for all public water systems, the filtered water turbidity must be less than or equal to 1 NTU at all times, measured under § 109.301(1).

(D) *Other filtration technologies.* The same performance criteria as those given for conventional filtration and direct filtration in clause (A) shall be achieved unless the Department specifies more stringent performance criteria based upon onsite studies, including pilot plant studies, where appropriate.

See 25 Pa. Code §109.202(c)(1)(i). The Department's argument is circular and the addition of turbidity to it does not make it more persuasive.

Turbidity is not a chemical compound, but a physical property used as an indicator for the clarity of water:

The cloudy appearance of water caused by the presence of suspended and colloidal matter. ***Technically, turbidity is an optical property of the water based on the amount of light reflected by suspended particles.*** In the waterworks field, a turbidity measurement is used to indicate the clarity of water.

See Attachment 18 – U.S. Environmental Protection Agency, [Vocabulary Catalog: Drinking Water Technical & Legal Terms](#) (bold italics added for emphasis). The direct effect of turbidity on drinking water implicates non-health properties such as color or taste.

Because turbidity is not a chemical compound, it is not appropriate to say that exposure to turbidity is associated with a particular harm to human health by way of toxicity. But that is not the case with PFOA and PFOS. The Drexel PFAS Advisory Group proposed MCLGs for PFOA and PFOS based on its determination that higher concentrations cause harm to human health. Appealing to the use of the number 95% for turbidity in the context of Giardia cysts is beside the point.

In summary, the Department relies on different regulatory approaches for different contaminants to attempt to justify setting MCLs that are higher than the MCLGs. This is unreasonable as a matter of law.

- g. The benefits of setting MCLs at levels equal to the recommended MCLGs would vastly exceed costs.

Under an economic analysis endorsed by the Agency for drinking water rulemakings, the benefits of setting MCLs equal to the MCLGs for PFOA and PFOAs would vastly outweigh the costs.

The Agency has identified economic methods to calculate the benefits of a given MCL. *See* Attachment 19 – U.S. Environmental Protection Agency, [Assessing the Benefits of Drinking Water Regulations: A Primer for Stakeholders](#), Chapter 3. Cost-of-illness (COI) is one method endorsed by the Agency. *See id.*, page 3-13 (“The cost-of-illness method has several advantages, including: (1) it is well-developed, widely applied, and easily explained; (2) many of the types of costs it includes are easily measured; and (3) existing studies provide estimates for a large number of illnesses.” Another methodology is an evaluation of mortality risk reductions. *See id.*, page 3-19).

The monetary costs of adverse health impacts from PFAS chemicals can and have been quantified. The European Union states that for PFAS compounds, “the annual health-related costs [are] estimated to be EUR 52-84 billion across Europe.” *See* Attachment 20 – European Environmental Agency, [Emerging chemical risks in Europe — ‘PFAS’](#) (Published 12 Dec 2019, Last modified 02 Mar 2022).

To relate these monetary impacts to Pennsylvania, a report by the Nordic Council of Ministers in 2019 is comparable because the relevant population is slightly smaller than that of Pennsylvania -- 10.3 million. Attachment 21 – Nordic Council of Ministers, [The Cost of Inaction: A socioeconomic analysis of environmental and health impacts linked to exposure to PFAS](#) (2019). That report calculates 12,655-41,417 cases of hypertension due to background levels/low exposure to PFAS, translating to roughly 150-500 deaths. *See id.*, page 106.

As for the value of a statistical life, “EPA recommends that the central estimate of \$7.4 million (\$2006), updated to the year of the analysis, be used in all benefits analyses that seek to quantify mortality risk reduction benefits.” Attachment 22 – U.S. Environmental Protection Agency, Mortality Risk Valuation: [Mortality Risk Valuation: What value of statistical life does EPA use?](#) Accounting for inflation, this is equal to \$10.4 million in 2022. *See* U.S. Bureau of Labor Statistics, [CPI Inflation Calculator](#).

To estimate the number of deaths associated with PFAS exposure to the Agency’s Health Advisory Levels for PFAS compounds, the Council used the death rate identified in the Nordic Council report. Note that the background levels in Europe contributing to deaths there are much lower than the Agency’s Health Advisory Levels, which the Department uses as the baseline for its cost-benefit analysis. *See* Attachment

23 – ARCADIS, [Environmental fate and effects of poly and perfluoroalkyl substances \(PFAS\)](#) (Prepared for the Concawe Soil and Groundwater Taskforce), pages 53-58.

Following the Department’s reasoning, an MCL that would reduce the number of deaths by 90% would still lead to 15-50 deaths/year (150-500 deaths x 10% = 15-50 deaths). Multiplied by the value of a statistical life (\$10.4 million in 2022), this results in a benefit of regulation in the range of \$156-520 million/ year. Setting an MCL equal to the MCLG (rather than at a level that is 90% of the distance between the Health Advisory Level and the MCLG) would yield an even higher benefit.

Of course, these mortality-related benefits do not encompass the entire range of health benefits from regulating PFAS compounds.

A recent paper by Cordner, et al calculates that the overall PFAS-related health-care costs in the United States are \$37–59 billion annually. *See* Attachment 24 – Alissa Cordner, Gretta Goldenman, Linda S. Birnbaum, Phil Brown, Mark F. Miller, Rosie Mueller, Sharyle Patton, Derrick H. Salvatore, and Leonardo Trasande, *The True Cost of PFAS and the Benefits of Acting Now*, *Environmental Science & Technology* 2021 55 (14), 9630-9633, DOI: 10.1021/acs.est.1c03565), available at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8296683/> (CC BY-NC-ND 4.0).

Taking the population of the Commonwealth of Pennsylvania to be 13,002,700 based on the 2020 census, when compared to the USA population of 331,449,281 (*See* Attachment 25 – U.S. Census Bureau, [QuickFacts](#)), the estimated Pennsylvania health expenditure due to PFAS-related illness would be approximately \$1.45 billion/year. Following the Department’s reasoning again, an MCL that would reduce these illnesses by 90% would generate a benefit of **\$145 million/year**. Again, setting an MCL equal to the MCLG (rather than at a level that is 90% of the distance between the Health Advisory Level and the MCLG) would yield an even higher benefit.

In the notice of proposed rulemaking, the Department skews the cost-benefit analysis by focusing on marginal costs and ignoring marginal benefits. According to the Department, the marginal cost of water treatment between the MCL values proposed by the Department and the MCLG is approximately \$80 million/year for PFOA and \$4 million/year for PFOS. *See* Proposed Rulemaking, Table 8 (PFOA), Table 12 (PFOS). But the Department does not compare this to the marginal healthcare savings within these intervals. Therefore, the Department offers a one-sided cost-benefit analysis.

To explain this in another way, the Council has prepared the following tables analyzing costs and benefits of setting MCLs for PFOA and PFOS, following the approach used by the Agency for arsenic in drinking water. *See* Attachment 26 – U.S. Environmental Protection Agency, [Arsenic in Drinking Water Rule Economic Analysis](#) (2000), Exhibit 1-1. It is noted that the economic analysis in the Nordic Council report and the Cordner, et al paper do not distinguish between the different PFAS compounds. But these are a family of compounds with similar physical and chemical structures, and they present concerns for “dose additivity,” which Clean Air Council discusses in a

subcomment below in favor of subjecting additional PFAS chemicals to drinking water regulation.

Also, the tables presented here (which follow the Agency's methodology) are based on a percentage (%) benefit as defined by the Department in the proposed rulemaking. For example, a 50% health benefit is defined as when the PFOA or PFOS MCL is set at the midpoint between the Health Advisory Level (70 ppt) and the MCLG for the particular compound.

Total Annual Benefits

(prepared by Clean Air Council)

(Estimated Monetized Total Health Benefits and Non-Quantifiable Health Benefits from Reducing PFOA and PFOS in Drinking Water)

% Benefit	PFOA (ppt)	PFOS (ppt)	Healthcare cost (\$ millions/year)	Healthcare benefit compared to HAL (70 ppt) (\$ millions/year)
0 (EPA HAL value)	70	70	1450-5200	0
50%	39	42	725-2600	725-2600
80%	20	25	290-1040	1160-4160
90%	14	20	145-520	1305-4680
100% (MCLG)	8	14	0	1450-5200

Notes:

1. Approach follows Exhibit 1-2 of the Agency's Arsenic in Drinking Water Rule Economic Analysis (2000)).

Total Annual (Compliance) Costs

(prepared by Clean Air Council)

% Benefit	PFOA (ppt)	PFOS (ppt)	Compliance cost (\$ millions/year)	Compliance cost relative to HAL (\$ millions/year)
0 (EPA HAL value)	70	70	27.63	0
50%	39	42	35+70=105	77.4
80%	20.4	25.2	89.5+75=164.5	136.9
90%	14.2	19.6	97.5+82.1=179.6	153
100% (MCLG)	8	14	177+89.7=266.7	239.1

Notes:

1. Approach follows Exhibit 1-2 of the Agency's Arsenic in Drinking Water Rule Economic Analysis (2000)).
2. Compliance cost numbers for PFOA and PFOS are approximated from Table 8 (PFOA) and Table 12 (PFOS) and the related Figure 1 (PFOA) and Figure 2 (PFOS) in the notice of proposed rulemaking.

Note that this calculation tends to overestimate the compliance cost, since it assumes that there is no overlap between the Entry Points (EPs) exceeding an MCL for PFOA and those exceeding an MCL for PFOS. The Department's data show that often there is an overlap, where the same EP has exceedances for both.

**Net Benefits and Benefit-Cost Ratios for Each Regulatory Option
(\$ millions) from Reducing PFOA and PFOS in Drinking Water**

(prepared by Clean Air Council)

% Benefit	PFOA (ppt)	PFOS (ppt)	Net benefit = healthcare benefit minus compliance cost (\$ millions/year)	Benefit/cost
0 (EPA HAL value)	70	70	(-27.6)	0
50%	39	42	620-2495	6.9-24.8
80%	20.4	25.2	995.5-3995.5	7.0-25.3
90%	14.2	19.6	1125.4-4500.4	7.3-26.1
100% (MCLG)	8	14	1183.3-4933.3	5.4-19.5

Notes:

1. Approach follows Exhibit 1-3 in EPA's "Arsenic in Drinking Water Rule Economic Analysis" (2000).

In conclusion, even at the MCLG, where the costs of treatment are the highest in the Department's range between the Health Advisory Level and the MCLG, the economic health benefits outweigh the costs by at least a factor of 5. Even if the PFAS-related healthcare expenses are only a fraction of those calculated for the European Union, they are still much higher than the compliance costs for implementing the MCL at the MCLG value.

Applying the Agency's guidance for cost-benefit analysis, the benefits from setting MCLs for PFOA and PFOS equal to the MCLGs would outweigh compliance costs.

- h. The Board unreasonably relies on a flawed analysis of cost-effectiveness.

According to the Department's guidance document for drinking water standards, a cost-benefit analysis for a proposed action involves an evaluation of the costs versus benefits to society:

Cost/benefit analysis. *A quantitative evaluation of the costs which would be incurred versus the overall benefits to society* of a proposed action such as the establishment of an acceptable dose of a toxic chemical.

See Attachment 1 – Pennsylvania Department of Environmental Protection, [Health Effects and Risk Management Guidance](#) (Document 383-0400-104), page 18 (bold italics added for emphasis). This is similar to the Department's summary of how the Agency implements the federal 1996 Safe Drinking Water Act Amendments:

After first defining an MCL or TT standard based on affordable technology, as previously, *EPA must determine whether* the costs of that standard would be justified by the benefits.

See *id.*, page 6 (bold italics added for emphasis).

According to the Agency's guidance document, the benefits of a rule involve an analysis of the effects of contamination – chiefly, reductions in human health risks:

For regulations that establish MCLs, a variety of benefits may be associated with reducing the effects of contamination on users of public water supplies (including households, commercial establishments, and industry) as well as on the water system itself. *Chief among these effects are reductions in human health risks.*

See Attachment 19 – U.S. Environmental Protection Agency, [Assessing the Benefits of Drinking Water Regulations: A Primer for Stakeholders](#), page 3 (Overview) (bold italics added for emphasis).

As discussed in a subcomment above, the Agency uses cost-of-illness (COI) and mortality risk reductions to evaluate the benefits of a drinking water rule. But in the proposed rulemaking, the Board does not do this. Rather, it defines the benefits in a circular manner that unfairly puts them on the same scale as the costs they eclipse in amount. The Department defines the benefits as improvements in public health relative to the Agency's Health Advisory Level (HAL) value:

Percent Improvement = $((\text{EPA HAL} - \text{MCLG})^{-1} \times 100) \times (\text{EPA HAL} - \text{Level "X"})$

Notice of proposed rulemaking, page 1260. In plain English, this simply divides the decrease from Agency’s Health Advisory Level to a particular level “X” by the total decrease from the Agency’s Health Advisory Level to the MCLG, and converts the result into a percentage.

To illustrate, according to the Department, there would be a 0% improvement in benefit if setting an MCL equal to the Health Advisory Level of the Agency (70 ppt). In contrast, there would be a 100% improvement in benefit in setting an MCL equal to the MCLG.

This definition of “benefit” is not consistent with the Agency’s guidelines for calculating the benefits of an MCL in drinking water:

... benefit analysts usually begin by listing the possible effects reduced by the regulations, then focus on valuing each specific effect (such as the changes in the risks of contracting a particular disease). *Values are derived for each effect, then aggregated (taking care to avoid double-counting) to determine the total benefits of the regulations.* For example, rather than directly estimating the value of a specific reduction in the concentrations of a chemical (such as arsenic or benzene), analysts generally estimate the value of the risks averted (such as the risks of incurring certain nervous system disorders or kidney cancer) and other benefits (such as improved taste or odor), then aggregate the values of these effects to determine the total benefits of the rule.

See Attachment 19 – [Assessing the Benefits of Drinking Water Regulations: A Primer for Stakeholders](#), page 3-2 (bold italics added for emphasis).

Then, the Department makes a flawed cost-effectiveness argument by balancing this numerical % with the % marginal cost of implementing the MCL when compared to the HAL:

Table 8. PFOA Comparison of Annual Costs and Benefits

PFOA Annual Costs and Benefits Analysis								
Value (ng/L)	Estimated # of EPs (of 3,785) > Value	Compliance Monitoring Costs (Millions)	Treatment O&M Costs		Treatment Capital Costs (Millions) per MGD* annualized over 20 years	Total Costs (Millions)	% Increase in Cost Compared to HAL	% Improvement in Health Protection Compared to HAL
			Treatment O&M Costs (Millions) per MGD*	Performance Monitoring Costs (Millions)				
HAL = 70	58	\$2.46	\$9.50	\$1.29	\$14.39	\$27.63	0%	0%
35	78	\$2.56	\$12.78	\$1.73	\$19.35	\$36.41	32%	56%
20	200	\$2.73	\$32.76	\$4.44	\$49.60	\$89.53	224%	80%
MCL = 14	218	\$2.89	\$35.71	\$4.83	\$54.07	\$97.51	253%	90%
12	270	\$2.97	\$44.23	\$5.99	\$66.97	\$120.15	335%	93%
10	313	\$3.07	\$51.28	\$6.94	\$77.63	\$138.92	403%	96%
MCLG = 8	400	\$3.39	\$65.53	\$8.87	\$99.21	\$177.00	541%	100%

*For purposes of totaling annual costs, the costs that vary with design capacity (treatment O&M and treatment capital costs) were multiplied by a benchmark design capacity of 1 MGD.

Notice of proposed rulemaking, page 1252.

As shown in Table 8 and Figure 1, additional improvement in public health benefits at PFOA values lower than the proposed MCL of 14 ng/L would require increasingly steep costs. For example, compared with the proposed MCL of 14 ng/L, an MCL value of 10 ng/L is estimated to achieve an additional 6% increase at an additional annual cost of approximately \$41.4 million (Table 8, Figure 1), which is a rate of approximately \$7 million in additional annual costs for every additional 1% of benefits. Compared with the HAL, the proposed MCL of 14 ng/L is estimated to achieve a 90% improvement in public health benefits at an additional annual cost of roughly \$70 million, which is a rate of approximately \$0.8 million in additional annual costs for every additional 1% of benefits.

See *id.*, page 1253. This creates a false comparison between percentages of increased costs and percentages of increased benefits. This flaw is also reflected in the ultimate conclusion:

For the aforementioned reasons, the Department believes that the proposed MCL for PFOA of 14 ng/L strikes an appropriate balance between the benefits (90% improvement in public health) and costs (253% increase in costs) when compared to the benefits and costs associated with meeting the HAL of 70 ng/L.

See *id.*

But the Board cannot reasonably compare the % benefit in health with the % increase in cost. Strictly speaking, this is not a cost-benefit analysis, but a flawed cost-effectiveness rationale that considers the numerical % of benefit without regard to specific health benefits.

The Board repeats the same erroneous approach for PFOS:

Table 12. PFOS Comparison of Annual Costs and Benefits

PFOA Annual Costs and Benefits Analysis								
Value (ng/L)	Estimated # of EPs (of 3,785) > Value	Compliance Monitoring Costs (Millions)	Treatment O&M Costs		Treatment Capital Costs (Millions) per MGD* annualized over 20 years	Total Costs (Millions)	% Increase in Cost Compared to HAL	% Improvement in Health Protection Compared to HAL
			Treatment O&M Costs (Millions) per MGD*	Performance Monitoring Costs (Millions)				
HAL = 70	96	\$2.57	\$15.73	\$2.13	\$23.81	\$44.24	—	—
35	148	\$2.64	\$24.25	\$3.28	\$36.71	\$66.87	51%	63%
20	183	\$2.70	\$29.98	\$4.06	\$45.39	\$82.13	86%	89%
MCL = 18	191	\$2.70	\$31.29	\$4.24	\$47.37	\$85.60	94%	93%
16	200	\$2.73	\$32.76	\$4.44	\$49.60	\$89.53	102%	96%
15	200	\$2.81	\$32.76	\$4.44	\$49.60	\$89.61	103%	98%
MCLG = 14	200	\$2.88	\$32.76	\$4.44	\$49.60	\$89.68	103%	100%

*For purposes of totaling annual costs, the costs that vary with design capacity (treatment O&M and treatment capital costs) were multiplied by a benchmark design capacity of 1 MGD.

See *id.*, page 1255.

As shown in Table 12 and Figure 2, additional improvement in public health benefits at PFOS values lower than the proposed MCL of 18 ng/L would require increasingly steep costs. For example, compared with the proposed MCL of 18 ng/L, an MCL value of 16 ng/L is estimated to achieve an additional 3% increase at an additional annual cost of approximately \$3.9 million (Table 12, Figure 2), which is a rate of approximately \$1.3 million in additional annual costs for every additional 1% of benefits. Compared with the HAL, the proposed MCL of 18 ng/L is estimated to achieve a 93% improvement in public health benefits at an additional annual cost of roughly \$41.4 million, which is a rate of approximately \$0.4 million in additional annual costs for every additional 1% of benefits.

See *id.*, page 1256.

For the aforementioned reasons, the Department believes that the proposed MCL for PFOS of 18 ng/L strikes a balance between the benefits (93% improvement in public health) and costs (94% increase in costs) when compared to the benefits and costs associated with meeting the HAL of 70 ng/L.

See id.

However, in the case of PFOS, the Department's analysis is even more unreasonable. The difference in cost between implementing the chosen MCL of 18 ppt when compared to the proposed MCLG is only 4.5%, or \$4 million/year, a relatively small difference.

The Department offers a flawed cost-effectiveness argument to circumvent the fact that the benefits of setting MCLs equal to the MCLGs would far exceed costs. The Board should propose MCLs that are not higher than the MCLGs recommended by the Drexel PFAS Advisory Group.

2. The Board should propose MCLs for other PFAS chemicals (PFBS, PFHpA, PFHxS, PFNA) at values that are not higher than the MCLGs recommended by the Drexel PFAS Advisory Group.

The Drexel PFAS Advisory Group recommended MCLGs for PFBS, PFHpA, PFHxS, PFNA. But the Board has proposed MCLs for only two of these compounds: PFOA and PFOS. The proposed rulemaking lists several reasons for the exclusion of other PFAS compounds:

Table 4. Reasons for not moving forward with MCLs for other PFAS.

	PFNA	PFHxS	PFHpA	PFBS	HFPO-DA
Lack of occurrence data > MCLG	x	x		x	x
Incomplete cost/benefit data and analysis	x	x	x	x	x
Reference dose was not derived due to lack of evidence on its toxicity			x		
Lack of treatability data					x

See Proposed Rulemaking, page 1250. In addition, the Board elaborated on its consideration of occurrence data:

The decision to not move forward with MCLs for additional PFAS at this time is further supported by a review of co-occurrence data... the PFOA and PFOS proposed MCLs appear to be protective of other PFAS at least 96.3% of the time.

See id.

For the following reasons, the Board’s rationale is unreasonable. To protect public health, the Board should propose MCLs for these comments at values that are not higher than the recommended MCLGs.

- a. The Drexel PFAS Advisory Group identified these compounds as harmful to health and determined MCLG values that are feasible.

The Drexel PFAS Advisory Group was “engaged by the Commonwealth of PA to provide ... recommendations for Maximum Allowable Contaminant Level Goals MCLGs to the Commonwealth of Pennsylvania for Per-and polyfluoroalkyl substances (PFAS) in drinking water.” See Attachment 2 – Drexel PFAS Advisory Group Report, page 5.

Based on current scientific and medical information regarding the effects of PFAS, it recommended MCLGs for PFBS, PFHpA, PFHxS, PFNA – in addition to PFOA and PFOS:

Table 1.

PFAS	Reference Dose	MCLG proposed
perfluorooctanoic acid (PFOA)	3.9 ng/kg/day	8 PPT
perfluorooctanesulfonic acid (PFOS)	3.1 ng/kg/day	14 PPT
perfluorononanoic acid (PFNA)	2.2 ng/kg/day	6 PPT
perfluorohexanesulfonic acid (PFHxS)	4.0 ng/kg/day	20 PPT
perfluoroheptanoic acid (PFHpA)	None derived	8 PPT
perfluorobutanesulfonic acid (PFBS)	39 ng/kg/day	55 PPT
ammonium salt of hexafluoropropylene oxide dimer (GenX)	75 ng/kg/day	108 PPT

Table 1: Summary of Reference Dose and proposed Chronic Non-Cancer MCLG for perfluorononanoic acid (PFNA), perfluorooctanesulfonic acid (PFOS), perfluorooctanoic acid (PFOA), perfluorohexanesulfonic acid (PFHxS), perfluoroheptanoic acid (PFHpA), perfluorobutanesulfonic acid (PFBS), and the ammonium salt of hexafluoropropylene oxide dimer (GenX)

See id., page 7 (Section 1, Executive Summary) (highlighting added for emphasis). These recommendations were based on the following premises:

1. These proposed Non-Cancer MCLGs are suggested with the health of the most vulnerable populations in mind
2. Individual MCLGs are advisable and the most scientifically rigorous approach
3. Non-Cancer MCLGs are low enough to protect against Cancer endpoints

See id., page 74 (Section 11, Summary).

- b. Setting MCLs for PFOA and PFOS would not be sufficient to protect against harm from these other compounds.

Table 4 of the notice of proposed rulemaking would suggest that only one compound (PFHpA) was found in water samples collected by the Department at concentrations that exceed the MCLG. This would be incorrect. Sampling data from the Department shows exceedances of the recommended MCLGs for at least PFHpA, PFHxS, and PFNA:

Department of Environmental Protection
Summary of Results for SDW Sampling Project Using EPA Method 537.1
(2020-2021)

Category	PWSID	EPID	BOL Sample # /	PWS Name	County	Date Collected	PFBS	PFHpA	PFHxS	PFNA	PFOS	PFOA
							MCLG=5 5	MCLG=8	MCLG=2 0	MCLG=6	MCL=1 8	MCL=1 4
BW	7010007	101	410- 20534-1	Paramount Senior Living (Village of Laurel Run)	Adams	11/12/20	ND	ND	140	0	11	2.6
TW	1090082	105	477250	Quakertown Borough	Bucks	9/9/20	ND	ND	ND	18.1	7	4.4
TW	1150015	101	477382	Taylor's Mobile Home Park	Chester	11/10/20	ND	10.6	4.7	ND	3.9	4.7
TI	1460073	138	477079	Aqua PA Main	Delaware	2/19/20	ND	ND	ND	8.5	ND	7.3
TW	7360976	100	477258	Conestoga Valley School Admin	Lancaster	9/9/20	ND	5.3	ND	9.3	5	4.7
TW	1460034	171	410- 19523-1	North Penn Water Authority	Montgomery	11/4/20	11	8.9	2.7	2.1	13	10
TW	1460056	100	477047	St. Gabriels Hall	Montgomery	2/13/20	ND	ND	ND	9.6	6.6	8.1

See Attachment 4 – Pennsylvania Department of Environmental Protection, [Summary of Results for SDW Sampling Project Using EPA Method 537.1](#) (2020-2021) (highlighting added for emphasis). The exceedance for PFHxS (140 ppt) was seven times the recommended MCLG (20 ppt) – and it was a sample from drinking water at an elder care facility.

The Department claims that “only 3.7% of all sites (or 16 out of 435 sites) had detections of at least 1 other PFAS at a level greater than its recommended MCLG when PFOA or PFOS levels did not exceed the proposed MCLs.” *See* Proposed Rulemaking, page 1250. That is not a very comforting explanation to people who are consumers of drinking water at these facilities.

It is significant that these samples did not show exceedances of MCLGs for PFOA and PFOS, despite showing exceedances of the recommended MCLGs for other PFAS compounds. The implication is that setting standards for only PFOA and PFOS would not protect against harmful levels of these other compounds.

- c. The Board erroneously excluded these compounds under the flawed rationale that cost/benefit data and analysis applied are incomplete.

In the notice of proposed rulemaking, the Board asserts “incomplete cost/benefit data and analysis” as a reason to not regulate PFAS compounds other than PFOA and PFOS. *See* Proposed Rulemaking, page 1250 (Table 4. Reasons for not moving forward with MCLs for other PFAS”). In the notice, the Board provides no further clarification on what this means.

For all the reasons discussed in Comment 1 regarding how the benefits would outweigh the costs of regulating PFAS compounds, it is appropriate to set MCLs for PFAS compounds for which the Drexel PFAS Advisory Group recommended MCLGs for the protection of public health.

In fact, the Department’s guidance document prioritizes the protection of public health by maximizing health benefits:

EPA must determine whether the costs of that standard would be justified by the benefits...

EPA can proceed with a standard based on the affordable technology approach, or may adjust an affordable technology-based MCL to a level that is “justified.” In the latter case, the new law’s further requirement that ***the MCL must also maximize health benefits ensures that health protection remains the paramount consideration in standard setting.***

See Attachment 1 – Pennsylvania Department of Environmental Protection, [Health Effects and Risk Management Guidance](#) (Document 383-0400-104), pages 6-7 (emphasis added by Council).

It is not appropriate for the Department to conclude that benefits can be calculated only for PFOA and PFOS and not for other PFAS compounds. The Department appears to have done this because only those two compounds are the subject

of the Agency's Health Advisory Levels. *See* Attachment 27 – U.S. Environmental Protection Agency, [Notice of availability, Lifetime Health Advisories and Health Effects Support Documents for Perfluorooctanoic Acid and Perfluorooctane Sulfonate](#), 81 Fed. Reg. 33,250 (May 25, 2016) (“EPA’s HAs, which identify the concentration of PFOA and PFOS in drinking water at or below which adverse health effects are not anticipated to occur over a lifetime of exposure, are: 0.07 parts per billion (70 parts per trillion) for PFOA and PFOS.”). The Department appears to reason that because there is a Health Advisory Level for only PFOA and PFOS, and the Department frames the benefit of the regulation as a simple percentage reduction from the Health Advisory Level, there are no data on the benefits of regulating the other PFAS compounds.

This argument is circular and makes non-regulation of other PFAS chemicals a self-fulfilling prophecy. It undermines the purpose of this regulatory initiative. The reason the Department has followed the example of other states in proposing MCLs is because the Agency has only established non-regulatory Health Advisory Levels, and only for two PFAS compounds. It is unreasonable for the Department to then identify the category of compounds subject to the Agency's Health Advisory Levels as the narrow universe of compounds that should be subject to MCLs for the protection of public health.

- d. The Board should propose an MCL for PFHpA because there is evidence of toxicity.

In the notice of proposed rulemaking, the Board states that for PFHpA, a “[r]eference dose was not derived due to lack of evidence on its toxicity.” *See* Proposed Rulemaking, page 1250. But the Drexel PFAS Advisory Group was still able to recommend an MCLG as a basis for an MCL, for the protection of public health. It is unreasonable for the Board to not propose an MCL for PFHpA, following this recommendation.

It is not correct that there is a lack of evidence of toxicity for this compound. The toxic effects of PFHpA have been demonstrated in a number of studies on animals that show adverse effects on heart, liver and other organs. These studies include the following:

1. Attachment 28 – Han, JS., Jang, S., Son, HY. et al, Subacute dermal toxicity of perfluoroalkyl carboxylic acids: comparison with different carbon-chain lengths in human skin equivalents and systemic effects of perfluoroheptanoic acid in Sprague Dawley rats. *Arch Toxicol* 94, 523–539 (2020), available at <https://doi.org/10.1007/s00204-019-02634-z> (“To evaluate systemic effects, Sprague Dawley (SD) rats were dermally treated with 250 and 1000 mg/kg PFHpA for 2 weeks and clinical and anatomic pathology were assessed. At 1000 mg/kg, 83% of the rats died, with severe ulcerative dermatitis at the application site. Adverse PFHpA-treated systemic changes were observed in the kidney, liver and testes, and histopathologic lesions such as renal tubular necrosis, hepatocellular

necrosis, and germ cell degeneration were seen at 250 and 1000 mg/kg.”) (abstract online).

2. Attachment 29 – Kim M, Park MS, Son J, Park I, Lee H, Kim C, Min B, Ryoo J, Choi KS, Lee D, Lee D, et al: Perfluoroheptanoic acid affects amphibian embryogenesis by inducing the phosphorylation of ERK and JNK. *Int J Mol Med* 36: 1693-1700, 2015, <https://pubmed.ncbi.nlm.nih.gov/26459765/>, available at <https://www.spandidos-publications.com/ijmm/36/6/1693> (“Whole-mount in situ hybridization, reverse transcriptase-polymerase chain reaction (RT-PCR), and histologic analyses detected severe defects in the liver and heart following exposure to PFHxA or PFHpA. In addition, immunoblotting revealed that PFHpA significantly increased the phosphorylation of extracellular signal-regulated kinase (ERK) and c-Jun N-terminal kinase (JNK), while PFHxA slightly increased these, as compared with the control. These results suggest that PFHxA and PFHpA are developmental toxicants and teratogens, with PFHpA producing more severe effects on liver and heart development through the induction of ERK and JNK phosphorylation.”) (full article online).
3. Attachment 30 – Zengqiang Li, Changchang Li, Zina Wen, Haoni Yan, Cheng Zou, Yang Li, Lili Tian, Zhen Lei, Huitao Li, Yiyan Wang, Ying Zhong, Ren-shan Ge, Perfluoroheptanoic acid induces Leydig cell hyperplasia but inhibits spermatogenesis in rats after pubertal exposure, *Toxicology*, Volume 448, 2021, 152633, ISSN 0300-483X, <https://doi.org/10.1016/j.tox.2020.152633> (“In conclusion, PFHpA induces Leydig cell hyperplasia due to the increase in the secretion of luteinizing hormone through negative feedback after down-regulating the expression of steroidogenic enzymes and inhibiting testosterone production in individual Leydig cells”) (abstract online).

Engaged by the Department, Drexel PFAS Advisory Group examined the available data and concluded that there is sufficient evidence to derive an MCLG for PFHpA. *See* Attachment 2 – Drexel PFAS Advisory Group Report, page 7 (Section 1, Executive Summary). It did this even though it stated there is a “paucity of evidence on its toxicity”:

8. PFHpA

PFHpA is a difficult compound to develop advisories for because there is a paucity of evidence on its toxicity. The DPAG decided to base recommendations on its chemical structure. MDHHS (2019) has made similar recommendations for other PFAS that lack sufficient scientific evidence to form conclusions about health advisory levels. Like PFOA, PFHpA is a carboxylic acid. PFHpA is a 7-carbon molecule and PFOA is an 8 carbon molecule. The DPAG concludes that the MCLG for PFHpA should be conservatively set at the same threshold for PFOA – 8 PPT.

See id., page 58 (highlighting added for emphasis). According to the report, it “decided to base recommendations on its chemical structure.” *See id.*

This was a sound recommendation that is consistent with the Agency’s approach to regulating toxic chemicals. The premise is that chemicals with a similar structure may affect their properties:

Organic compounds are often grouped according to structural similarities. Different “classes” of organic compounds refer to groups based on specific structural characteristics, including chemical bonds and functional groups. ***Similarities in chemical structure affect the properties of organic compounds***, and thus their uses and human exposure considerations.

See Attachment 31 – U.S. Environmental Protection Agency, [Exposure Assessment Tools by Chemical Classes - Other Organics](#) (last visited April 24, 2022) (bold italics added for emphasis).

The Commonwealth of Massachusetts acknowledges the toxicity of PFHpA by including it in the six regulated compounds subject to an MCL of 20 ppt. *See* Attachment 32 – Massachusetts Department of Environmental Protection, [Per- and Polyfluoroalkyl Substances \(PFAS\) Drinking Water Regulations Quick Reference Guide](#).

The Drexel PFAS Advisory Group proposed an MCLG for PFHpA based on its chemical structure (its similarity to other PFAS compounds). In addition, we have evidence of toxicity of PFHpA in lab animals. The Department has offered no reasonable rationale for rejecting this recommendation. The Board should propose an MCL for PFHpA that is not higher than the MCLG recommended by the Drexel PFAS Advisory Group.

- e. The Board should propose MCLs for these compounds because they do not necessarily co-occur with PFOA and PFOS.

Expanding upon the first reason listed in Table 4, the Board makes this statement:

The decision to not move forward with MCLs for additional PFAS at this time is further supported by a review of co-occurrence data. This review considers the frequency with which individual PFAS detections co-occurred with other PFAS detections in the occurrence data set used for this proposed rulemaking. Based on an analysis of co-occurrence data, only 3.7% of all sites (or 16 out of 435 sites) had detections of at least 1 other PFAS at a level greater than its recommended MCLG when PFOA or PFOS levels

did not exceed the proposed MCLs. In other words, the PFOA and PFOS proposed MCLs appear to be protective of other PFAS at least 96.3% of the time.

See Proposed Rulemaking, page 1250.

But as noted above, the Department's sampling data demonstrate several entry points (EPs) where other compounds are present at values above their MCLGs, even though PFOA and PFOS were not detected or were present at levels below the recommended MCLGs. The Board unreasonably assumes that proposing standards for only PFOA and PFOS will be sufficiently protective against harm from these other compounds.

The assumption is flawed because there is no uniform pattern and ratio of diverse PFAS compounds in drinking water. Over time, different PFAS compounds were manufactured and used in different applications. The actual number of water sources contaminated by PFAS compounds cannot be directly estimated based on PFOA and PFOS alone.

To illustrate, PFNA was used as the primary component (74%) of the emulsifier Surfion S-111 (CAS # 72968-3-88), which contains less than 1% of PFOA and no PFOS. *See* Attachment 33 – New Jersey Drinking Water Quality Institute Health Effects Subcommittee, [Health-Based Maximum Contaminant Level Support Document: Perfluorononanoic PERFLUORONONANOIC Acid \(PFNA\)](#) (June 22, 2015), page 3. One of the largest facilities in the world that used Surfion S-111 was located in West Deptford, New Jersey (less than 20 miles from Philadelphia). *See id.*, page 4. There is evidence of contamination from this operation:

Data provided to NJDEP about PFC use at the PVDF manufacturing facility located in Thorofare (West Deptford), NJ indicate that 86.6% of the 125,069 kg of the Surfion S-111 PFC mixture (*primarily PFNA*) used between 1991-2010 was released to the environment (air and water) (Roux Associates Inc., 2013).

See id. (bold italics added for emphasis).

Not surprisingly, the annual drinking water report for West Deptford, New Jersey identifies high levels of PFNA (up to 57 ppt), even though the highest levels of PFOA (10.8 ppt) and PFOS (4.55 ppt) were well below the MCLs proposed by the Board:

Individual Contaminants	MCLG	MCL	Level Found	Violation	Likely Source
Perfluorononanoic Acid (PFNA) Test Results Year 2020	n/a	13 ppt	Range: 0.0-57.0 Highest: 57	Y ⁵	Discharge from industrial chemical factories
Perfluorooctanoic Acid (PFOA) Test Results Year 2020	n/a	14 ppt	Range: ND-10.8 ppt Highest: 10.8 ppt	N	Discharge from industrial chemical factories
Perfluorooctane Sulfonic Acid (PFOS) Test Results Year 2020	n/a	13 ppt	Range: ND-4.55 ppt Highest: 4.55 ppt	N	Discharge from industrial chemical factories
1,4-Dioxane ⁶ Test Results Year 2020	n/a	n/a	Range: ND-0.79 ppb Highest: 0.79 ppb	N	Discharge from industrial chemical factories

⁵ PFNA: The detected levels reported here reflect all available sources during 2020, not the levels delivered to our customers. All water delivered to your tap was below the Maximum Contaminant Level (MCL). Refer to the "Information on Perfluoroalkyl Substances (PFAS)" section for more details about the Township's actions toward PFAS in your drinking water.

See Attachment 34 – West Deptford Township, [Annual Drinking Water Quality Report 2021 \(2020 Data\)](#), page 4 of 5.

The Board’s proposal to set MCLs for only PFOA and PFOS would not provide protection against situations like this – where water sources have been contaminated primarily by PFNA or other PFAS compounds.

The Board should propose MCLs for these four other PFAS compounds at levels not higher than the MCLGs recommended by the Drexel PFAS Advisory Group.

- f. Together with PFOA and PFOS, these compounds fall under EPA’s definition of “dose additivity,” which means that their presence at unregulated levels would magnify the adverse health effects of PFOA and PFOS.

According to the Agency, “the manner in which co-occurring chemicals induce toxicity in a coordinated or independent way is the basis for the concept of “additivity.” See Attachment 35 – U.S. Environmental Protection Agency, [PDF for Draft Framework for Estimating Noncancer Health Risks Associated with Mixtures of Per- and Polyfluoroalkyl Substances \(PFAS\)](#) (November 2021), page 13 (“2.0 Background on EPA Mixtures Additivity Guidance). That is the premise for the Agency’s guidance documents for chemical mixtures:

EPA developed the 1986 Chemical Mixtures Guidelines and subsequently the 2000 Supplementary Chemical Mixtures Guidance (EPA, 1986, 2000). In those guidance documents, EPA proposed a tiered hierarchy of mixtures approaches where the preferred approach is to *evaluate toxicity using hazard and dose-response data for a specific whole mixture of concern, or alternatively a sufficiently similar mixture.*

See *id.*, page 13 (emphasis added by the Council).

This recent Agency analysis of dose additivity for PFAS compounds clearly concludes that the following compounds fall under the dose additivity category:

PFOA and PFOS, *as well as other PFAS with linear or branched alkyl or alkyl ether chains and sulfonic or carboxylic acid functional groups*, share common

toxicological impacts of exposure on multiple cellular receptors, tissues, life stages, and species (ATSDR, 2021; EFSA et al., 2018, 2020).

See id., page 23 (emphasis added). For example, the Agency observed that PFOA and PFOS, along with other PFAS compounds were shown to have a collective effect on rat livers:

Recently, PFOA and PFOS, along with PFHxA, PFNA, PFDA, PFBS, and PFHxS, were shown to upregulate the PPAR α -inducible Acox1 and Cyp4a1 and the CAR-inducible Cyp2b1 and Cyp2b2 in adult male and female rat livers in 28-day repeat dose guideline studies (NTP, 2019a,b). From a molecular mechanism perspective, PFOA and PFOS both activate similar nuclear receptors and gene transcription pathways, along with several other studied PFAS including those listed above.

See id., page 24 (emphasis added). It should be noted that three of these four other PFAS compounds (PFBS, PFHxS, and PFNA) are compounds for which the Drexel PFAS Advisory Group recommended MCLGs but which the Board has not proposed MCLs.

The problem of dose additivity compounds the flaws in not proposing standards for PFNA, PFHxS, PFHpA and PFBS. Even if PFOA and PFOS are present below MCLs, their combined concentration and interaction with other PFAS chemicals without MCLs would present harm to public health.

The Board should include these compounds, either individually (no greater than their respective MCLGs), or as a sum total in the manner used by other states (Massachusetts and Maine).

- g. MCLs based on the recommended MCLGs are feasible, cost effective, and can be implemented using methodologies endorsed by the Department.

In a guidance document, the Department has stated that the MCL is “usually set at the MCLG” depending on feasibility and cost:

Once the MCLG is determined, EPA develops an enforceable standard. In most cases, the standard is a Maximum Contaminant Level (MCL), the maximum permissible level of a contaminant in water, which is delivered to any user of a public water system. The SDWA, as amended in 1996, requires EPA to set the MCL as close to the MCLG as feasible, which the SDWA defines as the level that may be achieved with the use of the best available technology. Factors considered while setting MCLs include ***analytical and***

treatment feasibility, costs to large metropolitan and regional water systems, and national economic impact. For noncarcinogens and equivocal-evidence carcinogens, *the MCL is usually set at the MCLG*.

See Attachment 1 – Pennsylvania Department of Environmental Protection, [Health Effects and Risk Management Guidance](#) (October 4, 2003), page 6 (bold italics added for emphasis).

Setting MCLs that are not higher than the recommended MCLGs is feasible. The detection limit (DL) and lowest concentration MRL (LCMRL) of the EPA-approved testing methodologies endorsed by the Department (Method 537.1 and Method 533) are well below the MCLG values:

Table 5. DLs and LCMRLs in Reagent Water

Analyte	Fortified Conc. (ng/L) ^a	DL ^b (ng/L)	LCMRL ^c (ng/L)
PFBS	4.0	1.8	6.3
PFHxA	4.0	1.0	1.7
HFPO-DA	4.0	1.9	4.3
PFHpA	4.0	0.71	0.63
PFHxS	4.0	1.4	2.4
ADONA	4.0	0.88	0.55
PFOA	4.0	0.53	0.82
PFOS	4.0	1.1	2.7
PFNA	4.0	0.70	0.83
9Cl-PF3ONS	4.0	1.4	1.8
PFDA	4.0	1.6	3.3
NMeFOSAA	4.0	2.4	4.3
PFUnA	4.0	1.6	5.2
NEtFOSAA	4.0	2.8	4.8
11Cl-PF3OUdS	4.0	1.5	1.5
PFDoA	4.0	1.2	1.3
PFTTrDA	4.0	0.72	0.53
PFTA	4.0	1.1	1.2

^a Spiking concentration used to determine DL.

^b Detection limits were determined by analyzing seven replicates over three days according to Section 9.2.8.

^c LCMRLs were calculated according to the procedure in reference 1.

See Attachment 12 – [Method 537.1](#), page 537.1-40 (highlighting added for emphasis),

Table 7. LCMRL Results

Analyte	LCMRL Fortification Levels (ng/L)	Calculated LCMRL (ng/L)
PFBA	1.0, 2.0, 4.0, 6.0, 10, 14, 20	13
PFMPA	1.0, 2.0, 4.0, 6.0, 10, 14, 20	3.8
PFPeA	1.0, 2.0, 4.0, 6.0, 10, 14, 20	3.9
PFBS	1.0, 2.0, 4.0, 6.0, 10, 14, 20	3.5
PFMBA	1.0, 2.0, 4.0, 6.0, 10, 14, 20	3.7
PFEESA	1.0, 2.0, 4.0, 6.0, 10, 14, 20	2.6
NFDHA	4.0, 6.0, 10, 14, 20, 41, 82	16
4:2FTS	1.0, 2.0, 4.0, 6.0, 10, 14, 20	4.7
PFHxA	1.0, 2.0, 4.0, 6.0, 10, 14, 20	5.3
PFPeS	1.0, 2.0, 4.0, 6.0, 10, 14, 20	6.3
HFPO-DA	1.0, 2.0, 4.0, 6.0, 10, 14, 20	3.7
PFHpA	1.0, 2.0, 4.0, 6.0, 10, 14, 20	2.6
PFHxS	1.0, 2.0, 4.0, 6.0, 10, 14, 20	3.7
ADONA	1.0, 2.0, 4.0, 6.0, 10, 14, 20	3.4
6:2FTS	1.0, 2.0, 4.0, 6.0, 10, 14, 20	14
PFOA	1.0, 2.0, 4.0, 6.0, 10, 14, 20	3.4
PFHpS	1.0, 2.0, 4.0, 6.0, 10, 14, 20	5.1
PFNA	1.0, 2.0, 4.0, 6.0, 10, 14, 20	4.8
PFOS	1.0, 2.0, 4.0, 6.0, 10, 14, 20	4.4
9Cl-PF3ONS	1.0, 2.0, 4.0, 6.0, 10, 14, 20	1.4
8:2FTS	1.0, 2.0, 4.0, 6.0, 10, 14, 20	9.1
PFDA	1.0, 2.0, 4.0, 6.0, 10, 14, 20	2.3
PFUnA	1.0, 2.0, 4.0, 6.0, 10, 14, 20	2.7
11Cl-PF3OUdS	1.0, 2.0, 4.0, 6.0, 10, 14, 20	1.6
PFDoA	1.0, 2.0, 4.0, 6.0, 10, 14, 20	2.2

See Attachment 11 – [EPA Method 533](#), page 533-37 (highlighting added for emphasis).

In addition, the treatment methods approved by the Department for the removal of PFOA and PFOS apply to these compounds:

The following processes were found to be effective for the removal of PFASs: granular activated carbon (GAC) (*up to > 99 percent*), membrane separation (*up to > 99 percent*), and ion exchange (*up to > 99 percent*). Various types of novel adsorptive media have also been found to effectively remove PFASs (up to > 99 percent removal), but results for these media published to date have been limited to batch tests at bench scale. These results cover the removal of specific PFASs including PFTriA, PFDoA, PFUnA, PFDA, **PFNA**, **PFHpA**, PFHxA, PFPeA, PFBA, PFDS, PFNS, PFHpS, **PFHxS**, PFPeS, **PFBS**, PFPrS, PFOSA, PFHxSA, PFBSA, PFMOBA, PFMOPrA, PFMOAA, PFO4DA, PFO3OA, PFO2HxA, FtS 8:2, FtS 6:2, FtS 4:2, N-EtFOSAA, N-MeFOSAA, ADONA, PFECHS, F35-B, Nafion BP2, and GenX.”

See Attachment 36 – U.S. Environmental Protection Agency, [Per- and Polyfluoroalkyl Substances, Treatment Processes](#) (click on tab for “Treatment Processes”) (bold italics added for emphasis).

Recognizing the concept of chemical additivity as it relates to PFAS compounds, the Commonwealth of Massachusetts has imposed an MCL that effectively limits PFAS compounds to standards that are more stringent than MCLGs recommended by the Drexel PFAS Advisory Group:

The amended Massachusetts Drinking Water Regulations establish a Maximum Contaminant Level (MCL) of 0.000020 milligrams per liter (mg/l) or 20 ng/l (also called parts per trillion or ppt) for the sum of six PFAS compounds (PFOS, PFOA, PFHxS, PFNA, PFHpA and PFDA).

See Attachment 37 – Massachusetts Department of Environmental Protection, [Per- and Polyfluoroalkyl Substances \(PFAS\) Drinking Water Regulations Quick Reference Guide](#).

To illustrate, even if the Board adopts MCLs equal to the MCLGs recommended by the Drexel PFAS Advisory Board (including PFOA and PFOS), this would allow as much as a total of 111 ppt in PFAS chemicals (the sum of these MCLGs). This is a far less stringent standard than the MCL of 20 ppt for multiple PFAS chemicals in Massachusetts.

Even excluding PFOA and PFOS, the sum of the recommended MCLGs for PFBS, PFHpA, PFHxS, PFNA is 89 ppt, which is much higher than the Massachusetts MCL of 20 ppt. But the Department did not include those other PFAS compounds in this proposed rulemaking.

The fact that Massachusetts has set a more stringent standard indicates that more stringent MCLs are feasible.

The additional costs of regulating these compounds by setting MCLs equal to the MCLGs (as compared to regulation of PFOA and PFOS only) are not expected to be significant. The standard practice for laboratories conducting PFAS testing is to test for a number of PFAS that include other compounds of interest, in addition to PFOA and PFOS. See Attachment 37 – Anatek Labs, [UCMR5 & PFAS Testing - PFCs/PFOS/PFOA](#) (“EPA 533 - 25 perfluorinated compounds, including PFOS and PFOA: \$375/sample”); See Attachment 38 – Wisconsin State Laboratory of Hygiene, University of Wisconsin-Madison, [PFAS in Drinking Water Testing](#) (“EPA 537.1 – 18 compounds – Total Cost: \$420”). Therefore, including proposed standards for other PFAS chemicals would not impose a significant burden by way of monitoring costs.

The proposed rule cites that “[a]pproximately half of the responding laboratories noted that they offer a cost reduction for reporting fewer analytes than included in the

method, which would provide a cost savings for systems since monitoring is required for only two analytes—PFOA and PFOS.” See Proposed Rulemaking, pages 1260-1261. But total monitoring costs for PFOA and PFOS are evaluated at less than \$4 million/year. See Proposed Rulemaking, page 1252 (Table 8, PFOA Comparison of Annual Costs and Benefits), page 1255 (Table 12, PFOS Comparison of Annual Costs and Benefits). Any savings from monitoring fewer compounds would be negligible compared to treatment costs that would be expected anyway.

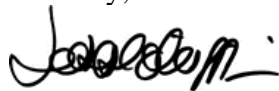
As for treatment costs, the treatment methods endorsed by the Department and the Agency for PFOA and PFOS apply to PFBS, PFHpA, PFHxS, and PFNA. Therefore, treatment for exceedances for PFOA and PFOS would also address exceedances for these compounds.

The development of MCLs for PFBS, PFHpA, PFHxS, and PFNA would increase costs only if these are detected at high values in EPs where PFOA and PFOS are not present at values above their MCLs. The Department claims that “only 3.7% of all sites (or 16 out of 435 sites) had detections of at least 1 other PFAS at a level greater than its recommended MCLG when PFOA or PFOS levels did not exceed the proposed MCLs.” See Proposed Rulemaking, page 1250. If this is indeed the case, then treatment costs would increase by only 4%, which is within the errors of estimation of the costs outlined in the proposed rule. On the other hand, if the Department’s estimate is low and more EPs contain these compounds at values that can harm human health, addressing this risk is paramount and should prevail.

Proposing MCLs for PFBS, PFHpA, PFHxS, and PFNA that are not higher than the recommended MCLGs is feasible and should not increase costs significantly, based on the Department’s own calculations.

Thank you for your consideration of the Council’s comments.

Sincerely,



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Attachment 1

DEPARTMENT OF ENVIRONMENTAL PROTECTION
Bureau of Water Supply and Wastewater Management

DOCUMENT NUMBER: 383-0400-104

TITLE: Health Effects and Risk Management Guidance

EFFECTIVE DATE: October 4, 2003

AUTHORITY: Pennsylvania's Safe Drinking Water Act (35 P.S. §721.1 *et seq.*) and regulations at 25 Pa. Code Chapter 109.

POLICY: Department staff will follow the guidance presented in this document to respond to the occurrence of regulated and unregulated contaminants found in public drinking water systems.

PURPOSE: The Health Effects and Risk Management Guidance was developed as part of the Department of Environmental Protection's (DEP) continuing effort to provide basic information and guidance to staff personnel on responding to contamination incidents.

APPLICABILITY: This guidance will apply to all public water systems.

DISCLAIMER: The policies and procedures outlined in this guidance are intended to supplement existing requirements. Nothing in the policies or procedures shall affect regulatory requirements.

The policies and procedures herein are not an adjudication or a regulation. There is no intent on the part of DEP to give the rules in these policies that weight or deference. This document establishes the framework within which DEP will exercise its administrative discretion in the future. DEP reserves the discretion to deviate from this policy statement if circumstances warrant.

PAGE LENGTH: 36 pages

LOCATION: Volume 18, Tab 1

DEFINITIONS: See 25 Pa. Code Chapter 109

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RISK MANAGEMENT OF CONTAMINANTS IN DRINKING WATER

INTRODUCTION

The Health Effects and Risk Management Guidance was developed as part of the Department of Environmental Protection's (DEP) continuing effort to provide basic information and guidance to staff personnel on regulated and unregulated contaminants, which have been identified in public or individual water systems. The guidance provides DEP's Unregulated Contaminants Guidance, Glossary of Terms, and a List of Acronyms and Abbreviations. The Health Effects and Risk Management Guidance also includes important sources of risk management information such as links to the U.S. Environmental Protection Agency (EPA) Drinking Water Standards and Health Advisories tables that summarize regulatory and guidance levels for contaminants in drinking water. Links are also provided to Contaminant Fact Sheets prepared by the EPA.

The Health Effects and Risk Management Guidance
may be found in the online document warehouse at <http://www.dep.state.pa.us> .

HEALTH ADVISORIES

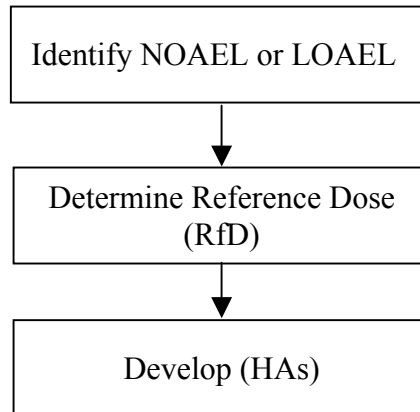
Health Advisories (HAs) provide information on contaminants that can cause adverse human health effects and are known or anticipated to occur in drinking water. HAs are guidance values prepared by the EPA based on non-cancer health effects for different durations of exposure (e.g., one-day, ten-day, and lifetime). HAs are not enforceable standards. Their purpose is to provide technical guidance to EPA regional offices, state governments, and other public health officials on health effects, analytical methodologies, and treatment technologies associated with drinking water contamination.

EPA's Office of Water periodically publishes an updated summary table that compiles the current drinking water standards and health advisories. This compilation of Drinking Water Standards and Health Advisories is available on the Internet.

Find the Drinking Water Standards and Health Advisories links at
<http://www.epa.gov/ost/drinking/standards> and
<http://www.epa.gov/ost/drinking/standards/dwstandards.pdf> .

The following is a brief illustration intended to show key steps that EPA uses to develop their HAs.

Key Steps in the Development of Health Advisories (HAs)



The first step in developing a HA is to identify a no-observable-adverse-effect level (NOAEL) or lowest-observable-adverse-effect level (LOAEL). The NOAEL and LOAEL levels are derived from experimental, usually animal, studies of appropriate duration and are expressed as milligrams contaminant per kilograms body weight per day (mg/kg/day). In these studies, toxicologists evaluate potential risk for significant increases in frequency or severity of adverse effects in an exposed human or animal population compared to its appropriate control. Then the toxicologists weigh the merits of all the tests, include margins of safety, and choose, in their best professional judgment, the NOAEL, or if the NOAEL cannot be determined, the LOAEL.

A NOAEL is the highest experimental dose of a chemical at which there are *no* statistically or biologically significant increases in frequency or severity of adverse effects in the subject population. Effects may be evident at or below a NOAEL, but they are not considered to be adverse. A LOAEL is the *lowest* dose of a chemical in a study or group of studies that produces statistically or biologically significant increases in the frequency or severity of adverse effects in the subject population.

In the next step, a reference dose (RfD) is determined by dividing the NOAEL or LOAEL by an uncertainty factor. The uncertainty factor consists of multiples of ten that reflects the degree of uncertainty inherent in the available data. Uncertainty factors may range from 1 to 10,000 but typically seen values range from 100 to 1,000. Uncertainty factors take into account extrapolations from laboratory animals to humans (10), variations in subpopulation sensitivities (10), the extrapolation from short-term to chronic studies (10), and use of a LOAEL rather than a NOAEL (10). In addition, a modifying factor is sometimes used to account for deficiencies in the entire toxicological database of the chemical.

HAs for less-than-lifetime (e.g. 1-day and 10-day) exposure are calculated by multiplying an RfD of an appropriate duration by the assumed body weight of the protected individual and dividing by the assumed water consumption for the protected individual. For example, when calculating 1-day and 10-day HAs for a child, it is assumed that the protected individual is a 10 kg. child who consumes 1 liter of water per day.

Calculation of lifetime HAs assumes the protected individual is a 70 kg. adult who consumes 2 liters of water per day. Using the RfD derived from a chronic animal or human study, a drinking water equivalent level (DWEL) is developed. The DWEL is calculated by multiplying the RfD by 70 kg.

(weight of an adult) and dividing by 2 liters (consumption of water per day by an adult). The lifetime HA is determined by multiplying the DWEL by a relative source contribution (RSC). The RSC is an additional protective measure used in the calculation of lifetime HAs to take into consideration the exposure to the contaminant from other sources, such as food or air. In the absence of quantitative data, the RSC from drinking water is conservatively assumed to be 20 percent (80 percent allowed from non-water sources). When quantitative data is available -- more likely for inorganic contaminants than for organic compounds -- proportionate exposure from drinking water can be assessed to be greater than the default 20 percent, up to a maximum of 80 percent. For example, EPA selected 70 percent for the chromium RSC because available data indicated that drinking water provides about 70 percent of the total daily chromium intake when chromium concentrations in water are at the lifetime health advisory level of 0.1 mg/L.

One-day and ten-day HAs incorporate the assumption that 100 percent of an individual's exposure to a contaminant comes from drinking water.

One-day HA: The concentration of a chemical in drinking water that is not expected to cause any adverse noncarcinogenic effects for up to one day of exposure.

Ten-day HA: The concentration of a chemical in drinking water that is not expected to cause any adverse noncarcinogenic effects for up to ten days of exposure.

Lifetime HA: The concentration of a chemical in drinking water that is not expected to cause any adverse noncarcinogenic effects for a lifetime of exposure.

CARCINOGENICITY RISK ASSESSMENT

In addition to the determination of noncarcinogenic endpoints of toxicity, contaminants are also evaluated for carcinogenic potential. Applying the criteria described in EPA's "Guidelines for Carcinogen Risk Assessment" (51 *Federal Register* 33992, 9/24/86), EPA places a contaminant into one of the following weight-of-evidence groups:

Group A: Human Carcinogen

Sufficient evidence in epidemiological studies to support causal association between exposure and cancer.

Group B: Probable Human Carcinogen

B1 - Almost sufficient to inadequate evidence in epidemiological studies.

B2 - Sufficient evidence from animal studies.

Group C: Possible Human Carcinogen

Absence of data in humans; limited evidence from animal studies.

Group D: Not Classified

Inadequate animal evidence.

Group E: No Evidence of Carcinogenicity for Humans

No evidence in multiple studies.

For chemicals classified as human or probable human carcinogens (group A or B), EPA evaluates available laboratory animal studies and human epidemiological studies. Through this evaluation EPA produces a quantitative estimate of the probability of an increased risk of cancer, given that an individual is exposed to the chemical by drinking 2 liters of drinking water for a lifetime of 70 years.

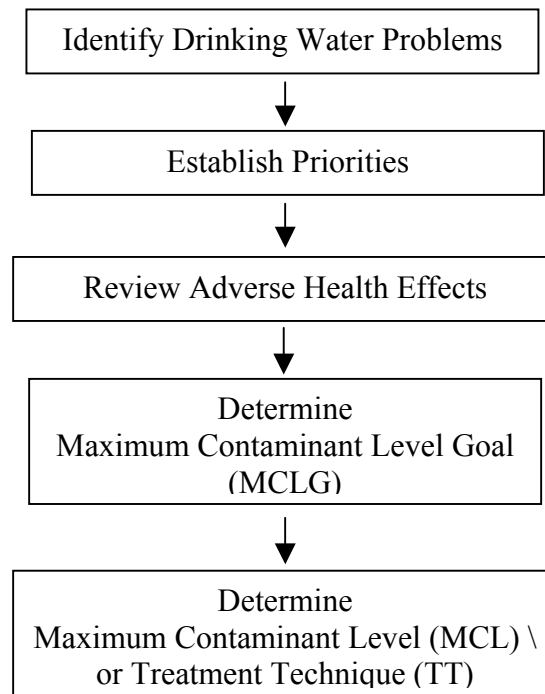
REGULATORY LEVELS

The 1996 Amendments to the Safe Drinking Water Act (SDWA) require EPA to go through several steps to determine, first, whether setting a standard is appropriate for a particular contaminant, and if so, what the standard should be. Peer-reviewed science and data support an intensive technological evaluation that includes many factors, such as:

1. Occurrence in the environment.
2. Human exposure and risks of adverse health effects in the general population and sensitive subpopulations.
3. Analytical methods of detection.
4. Technical feasibility.
5. Impacts of regulation on water systems, the economy and public health.

The following is a brief illustration intended to show key steps that EPA uses to develop standards.

Key Steps to Establish Regulatory Level Standards for Contaminants in Drinking Water



Identify Drinking Water Problems

EPA must first make determinations about which contaminants to regulate. These determinations are based on health risks and the likelihood that the contaminant occurs in public water systems at levels of concern.

Establish Priorities

The SDWA requires EPA to establish a list of contaminants to aid in priority setting for EPA's drinking water program. The Contaminant Candidate List (CCL) is a list of contaminants which, at the time of publication, are not subject to any proposed or promulgated national primary drinking water regulation (NPDWR), are known or anticipated to occur in public water systems, and may require regulations under SDWA. In establishing the list, EPA has divided the contaminants among those which are priorities for additional research, those which need additional occurrence data, and those which are priorities for consideration for rulemaking.

The CCL will be the primary source of priority contaminants for EPA's drinking water program. Contaminants for priority drinking water research, occurrence monitoring, and guidance development, including health advisories, will be drawn from the CCL. Certain contaminants on the CCL have also been designated as those from which EPA will determine whether to regulate specific contaminants. The CCL, developed with considerable input from the scientific community and other interested parties, may be found at <http://www.epa.gov/safewater/ccl/cclfs.html#table2>.

Review Adverse Health Effects, and Determine Maximum Contaminant Level Goal (MCLG)

After it reviews the health effects studies, EPA establishes regulatory levels for contaminants in drinking water. The SDWA requires EPA to simultaneously promulgate (1) a maximum contaminant level goal (MCLG), and (2) either a maximum contaminant level (MCL) or treatment technique. Conceptually, the MCLG/MCL development process consists of two stages. First, EPA sets a MCLG based on health effects alone. Then, based on the health effects data, EPA determines what is a feasible, enforceable level.

The MCLG is based solely on toxicological data and is not an enforceable concentration level. By policy, the MCLGs of human and probable human carcinogens (groups A and B) are set at zero. For contaminants in which evidence of carcinogenicity is inadequate or lacking (groups D and E), the MCLGs are set at a number derived by the same process as the lifetime health advisory described earlier. When there is equivocal evidence of carcinogenicity (group C), the MCLG preferentially is set at a number equal to the lifetime health advisory level divided by an additional uncertainty factor ranging from one to ten, to account for possible carcinogenicity. In the absence of reliable non-carcinogenic data, EPA may set the MCLG for a group C chemical at the 10^{-5} or 10^{-6} excess cancer risk level. (56 Federal Register 3533, January 30, 1991).

Determine Maximum Contaminant Level (MCL) or Treatment Technique (TT)

Once the MCLG is determined, EPA develops an enforceable standard. In most cases, the standard is a **Maximum Contaminant Level (MCL)**, the maximum permissible level of a contaminant in water, which is delivered to any user of a public water system. The SDWA, as amended in 1996, requires EPA to set the MCL as close to the MCLG as feasible, which the SDWA defines as the level that may be achieved with the use of the best available technology. Factors considered while setting MCLs include analytical and treatment feasibility, costs to large metropolitan and regional water systems, and national economic impact. For noncarcinogens and equivocal-evidence carcinogens, the MCL is usually set at the MCLG. For group A and B carcinogens, the target range for setting the MCL is between the 10^{-4} and 10^{-6} excess cancer risk level. If it is economically or technically unfeasible to determine the concentration level of a contaminant in water, a treatment technique can be set for the contaminant in place of an MCL.

When there is no reliable method that is economically and technically feasible to measure a contaminant at particularly low concentrations, a **Treatment Technique (TT)** is set rather than an MCL. A TT is an enforceable procedure or level of technological performance, which public water systems must follow to ensure control of a contaminant. Examples of TT rules are the Surface Water Treatment Rule (disinfection and filtration) and the Lead and Copper Rule (optimized corrosion control).

More detailed information on EPA's standard-setting protocol may be found at http://www.epa.gov/safewater/standard/setting.html

1996 SDWA Amendments

Future drinking water standard setting has new flexibility compared to the previous law. As a new requirement, EPA must publish a cost-benefit analysis along with MCL proposals. After first defining an MCL or TT standard based on affordable technology, as previously, EPA must determine whether the costs of that standard would be justified by the benefits. If not, then EPA may adjust an MCL to a level that "maximizes health risk reduction benefits at a cost that is justified by the benefits." Flexibility to

“minimize the overall risk of adverse health effects” is also authorized where certain means of controlling one contaminant may increase the risk from another contaminant.

The cost-benefit provision was included mainly to address the concern that the health protection benefits of certain future standards might not be “worth” their costs, even if large systems could afford to meet such standards through their economies of scale -- i.e. spreading the cost of water treatment over a large number of customers. The new standard setting retains the previous law’s approach to defining an affordable technology standard, but subjects that standard to the “justified” test. EPA can proceed with a standard based on the affordable technology approach, or may adjust an affordable technology-based MCL to a level that is “justified.” In the latter case, the new law’s further requirement that the MCL must also maximize health benefits ensures that health protection remains the paramount consideration in standard setting.

While EPA will continue to use feasibility for large systems in setting drinking water regulations, the 1996 amendments to the SDWA specifically require EPA to make small system technology assessments for both existing and future regulations. The new requirements will provide small systems with options designed specifically for their use. This should aid in the implementation of the regulations because smaller systems may be able to successfully install and operate treatment technologies to achieve compliance.

UNREGULATED CONTAMINANTS GUIDANCE

BACKGROUND

The purpose of this guidance is to address contamination of drinking water sources by contaminants without enforceable regulatory levels (unregulated contaminants). The Bureau of Water Supply and Wastewater Management (BWSWM) developed a guidance which recommends a prioritization protocol for determining a guidance level in place of a federally promulgated MCL. This guidance level in place of an MCL is referred to as the **maximum unregulated contaminant concentration**. This guidance also recommends appropriate responses when the contaminant is detected in a public water system.

If an unregulated contaminant is detected by a water supply, the first guideline for the **maximum unregulated contaminant concentration** is to search for an MCL proposed by EPA. In the absence of a proposed MCL, the maximum unregulated contaminant concentration is set as close as feasible to an alternate health criterion. This maximum unregulated contaminant concentration takes into consideration analytical and treatment technologies. For chemicals in the A or B carcinogen groups, the criterion is the 10^{-6} excess lifetime cancer risk concentration. For noncarcinogens and equivocal-evidence contaminants (in carcinogen groups C, D, and E), the appropriate health criterion is the lifetime health advisory concentration.

Calculations of maximum unregulated contaminant concentrations are recommended to be consistent with the EPA drinking water standards and health advisory data. The values developed by EPA for HAs, MCLGs, and MCLs are usually rounded to one significant figure. This rounding procedure is appropriate because using two or more significant figures implies a degree of precision that is unwarranted. As described above, the large uncertainty factors (up to 1,000), included as margins of safety by toxicologists, would affect the degree of precision. To maintain consistency, when comparing laboratory results to a maximum unregulated contaminant concentration, the laboratory result should be rounded to the same number of significant figures as that of the maximum unregulated contaminant concentration. This is the same process used to determine compliance with existing MCLs. For

example, if comparing a contaminant's laboratory result of 0.0447 mg/L to its maximum unregulated contaminant concentration of 0.04 mg/L, the laboratory result would be rounded to 0.04 mg/L. The rounded laboratory result does not exceed the maximum unregulated contaminant concentration and thus does not justify action associated with exceeding the maximum unregulated contaminant concentration.

Appropriate determinations may be made about the length of time a consumer should drink water containing a contaminant exceeding the maximum unregulated contaminant concentration. The following factors need to be considered about consumption of water containing these contaminants:

1. Concentration of the contaminant and how close that concentration is to exceeding its HA.
2. Additional sources of consumer exposure to the same contaminant (occupational, environmental, etc.).
3. Severity of anticipated adverse health effect.
4. Uncertainty factor(s) used to develop the HA (10, 100, 1000, etc.).
5. Other contaminants (i.e. chemical mixtures) contained in the water which may affect the same body organ or body function.
6. All possible routes of entry to the body (ingestion, inhalation, dermal) which may compound exposure.

In addition, tap water is most often used for activities other than drinking (ingestion through the digestive tract). A large share of the total volume of water used by residential customers may involve agitation or heating, such as bathing, showering, laundering, cleaning, dish washing, or toilet flushing. It is generally agreed that for **most** chemicals in drinking water, the risk from non-ingestion pathways is less than the risk from direct ingestion. However, volatile organic compounds may escape from the water in large enough concentrations during these activities to present an additional risk to the inhabitants. The activity of most concern is showering or bathing which may potentially expose an individual to the contaminant by inhalation or by absorption through the skin.

EPA recognizes that dermal absorption and inhalation of chemicals in the home are factors in the overall exposure from certain chemicals. However, due to its concern about the limited amount of data available and the uncertainty of proposed calculation methodologies, EPA decided in 1985 not to include exposure from showering, bathing, or swimming as part of its quantitative standard-setting protocol (50 *Federal Register* 46895, 11/13/85).

Chemicals that may penetrate the skin (skin penetrants) of most concern during showering, bathing, or swimming activities are ones that are low molecular weight, non-ionized, and soluble in both lipids (fat) and water. Permeability coefficients and pathway exposure factors are useful tools in evaluating the contribution of inhalation and dermal exposure to the total body burden. Unfortunately, these permeability coefficients and pathway exposure factors have been determined for only a few environmental contaminants. Based on their permeability coefficients, three chemicals have been experimentally identified as potential water supply contaminants. Ethylbenzene, styrene, and toluene, which are normally of concern via ingestion, may also pose a significant dermal absorption hazard at low concentrations.

Measurements of physical-chemical parameters such as vapor pressure, solubility and molecular weight may be used to evaluate a contaminant's tendency to volatilize. These parameters relating to the behavior of gases are included in Henry's Law, which was first proposed by J. W. Henry in 1800. Henry's Law Constants have been measured and are available in table form. A Henry's Law Constant for a contaminant above 0.001 atm m³/mole suggests volatilization and subsequent inhalation as a potentially significant route of exposure.

DEP recommends that an alternate source of water be used for bathing and showering activities when the concentration of a chemical is high enough to potentially pose a significant risk from ingestion. In these situations, simply providing bottled water for drinking may not adequately protect consumers from contamination of their water supply.

The Unregulated Contaminants Guidance

The "Unregulated Contaminants Guidance" pertains to all unregulated chemical contaminants **except radon and the four unregulated contaminants which comprise the total trihalomethane MCL (bromoform, bromodichloromethane, chloroform, and chlorodibromomethane)**. This guidance replaces all previous guidance for the unregulated contaminants.

This guidance is divided into three parts. Part I defines the terminology used in the guidance. Part II describes the procedure used to determine the maximum unregulated contaminant concentrations. Part III recommends actions to follow when an unregulated contaminant is detected in a public water system.

PART I - DEFINITIONS

1. Carcinogenic contaminant – a cancer-producing contaminant which has been classified by EPA as a known (Group A) or probable (Group B) human carcinogen.
2. DEP – the Department of Environmental Protection.
3. Health Advisories – guidance values prepared by the EPA based on non-cancer health effects for different durations of exposure (e.g., 1-day, 10-day, and lifetime).
4. Lifetime exposure – the total amount of exposure to a substance that a human would receive in a lifetime (usually assumed to be 70 years). The true risk is not likely to be higher and may be lower. For example, a lifetime cancer risk of 10⁻⁴ indicates an increased probability of contracting cancer for 1 person out of 10,000 people exposed to the carcinogen at a specified concentration during their entire lifetime of 70 years.
5. Maximum unregulated contaminant concentration – The maximum allowable concentration of an unregulated contaminant in finished water, as determined from health risk data by DEP.
6. Method detection limit (MDL) – The minimum concentration of a substance that can be measured and reported with 99 percent confidence that the true value is greater than zero, as determined by EPA.

7. Practical quantitation level (PQL) – The lowest level of a substance in water that can be reliably measured within specified limits of precision and accuracy during routine laboratory operating conditions, as determined by EPA (or available as needed on a case by case basis through EPA).
8. Unregulated contaminant – A contaminant for which no maximum contaminant level or treatment technique has been established under **§109.202** of the Pennsylvania Safe Drinking Water Regulations (relating to state maximum contaminant levels and treatment technique requirements).

PART II - MAXIMUM UNREGULATED CONTAMINANT CONCENTRATIONS

The following protocol should be used to establish a maximum unregulated contaminant concentration:

1. If available, the maximum unregulated contaminant concentration should be set equal to the concentration that EPA has proposed or is considering to propose as a primary maximum contaminant level for the contaminant.
2. If EPA has not proposed or is not considering to propose a primary maximum contaminant level as noted in paragraph 1, the maximum unregulated contaminant concentration should be set equal to:
 - the concentration associated with a lifetime cancer risk of 10^{-6} for carcinogenic contaminants.
 - the concentration equal to the lifetime health advisory for noncarcinogenic contaminants.
3. If the concentration specified in paragraph 2 is not equal to or greater than the practical quantitation level or is not achievable through the use of available treatment technology, the maximum unregulated contaminant concentration should be set at the lowest concentration these limiting factors will allow.

PART III - RECOMMENDED ACTION

Public water systems should supply finished water that fulfills the maximum unregulated contaminant concentrations determined according to Part II above. Compliance with the maximum unregulated contaminant concentration should be based on the running annual average concentration of quarterly results when monitoring is conducted quarterly or more frequently. If monitoring frequency is annual or less frequent, compliance should be based on the average of the initial sample and a check sample.

When a single monitoring sample demonstrates that an unregulated contaminant is present in a concentration equal to or greater than the EPA method detection limit, the water supplier should take a check sample from the same sampling point within 24 hours of receipt of the sample results indicating detection of the unregulated contaminant. (This recommendation does not apply to new source sampling conducted under **§109.503(a)(1)(iii)(B)** of the Safe Drinking Water Regulations.) If detection of an unregulated contaminant is verified as described above, the water supplier should do the following:

1. Where the average concentration of the original and a check sample is equal to or greater than the method detection limit (MDL) but less than or equal to the maximum unregulated contaminant concentration, the water supplier (community and noncommunity) should monitor

at least quarterly at the entry point(s) for the detected contaminants. After the analyses of four consecutive quarterly samples demonstrates that the concentration of the contaminant in each quarterly sample does not exceed the maximum unregulated contaminant concentration, DEP may reduce the recommended monitoring to one sample per entry point per year, or less frequently, as appropriate, to protect public health.

DEP may recommend more appropriate sampling points if the source of contamination is within the distribution system.

2. Where the average concentration of the original and a check sample is determined to exceed the maximum unregulated contaminant concentration, but is less than a concentration which poses an imminent hazard to public health, the following should be provided:
 - a. Public notification:
 - (1) The water supplier, except a bottled water or retail water supplier, shall provide Tier 2 public notification as follows:
 - (a) Report the circumstances to DEP within 1 hour of discovery of the situation.
 - (b) Provide the public notice as soon as possible, but no later than 30 days after the system learns of the situation.
 - (c) Repeat the notice every 3 months as long as the situation persists.
 - (2) The water supplier shall provide the initial public notice and any repeat notices in a form and manner that is reasonably designed to reach all persons served in the required time period. The form and manner of the public notice may vary based on the specific situation and type of water system, but the public water supplier shall at a minimum meet the following requirements:
 - (a) Community water systems shall provide notice using the following forms of delivery:
 - (i) Mail or other direct delivery to each customer receiving a bill and to other service connections to which water is delivered.
 - (ii) Any other method reasonably designed to reach other persons regularly served by the system, if they would not normally be reached by the notice required above.
 - (b) Noncommunity water systems shall provide notice using the following forms of delivery:
 - (i) Posting the notice in conspicuous locations throughout the distribution system frequented by persons served by the system, or by mail or direct delivery to each customer and service connection, when known.

- (ii) Any other method reasonably designed to reach other persons served by the system if they would not normally be reached by the notice required above.

- b. **Monitoring:** The water supplier (community and noncommunity) should be required to monitor at least quarterly at the entry point(s) for the detected contaminants. After the analyses of four consecutive quarterly samples demonstrates that the concentration of the contaminant in each quarterly sample does not exceed the maximum unregulated contaminant concentration, DEP may reduce the recommended monitoring to one sample per entry point per year.

DEP may recommend more appropriate sampling points if the source of contamination is within the distribution system.

- c. For all bottled water and retail water for which the average of the original and a check sample exceeds the maximum unregulated contaminant concentration, the water supplier should recall the contaminated water and cease distribution until the contaminant concentration is equal to or less than the maximum unregulated contaminant concentration.

- 3. Where the average concentration of the original and a check sample is determined by DEP to pose an imminent hazard to public health, the public water supplier should provide the following:

- a. **Public Notification:**

- (1) The public water supplier, except a bottled water or retail water supplier, shall provide Tier 1 public notification as follows:
 - (a) Provide a public notice as soon as possible, but no later than 24 hours after the water supplier learns of the situation.
 - (b) Report the circumstances to DEP within 1 hour of discovery of the situation.
 - (c) Initiate consultation with DEP as soon as possible, but no later than 24 hours after the water supplier learns of the situation, to determine initial and any additional public notice requirements.
 - (d) Comply with initial and any additional public notification requirements that are established as a result of the consultation with DEP. The repeat notice frequency, if applicable, for a Tier 1 public notice shall be established as a result of the consultation, but may be no less often than once every 30 days as long as the situation persists.
- (2) The form and manner used by a public water supplier shall fit the specific situation and shall be reasonably designed to reach residential, transient and

nontransient users of the water system. To reach all persons served, a water supplier shall use, at a minimum, one or more of the following forms of delivery:

- (a) Appropriate broadcast media, such as radio or television.
 - (b) Posting of the notice in conspicuous locations throughout the area served by the water system.
 - (c) Hand delivery of the notice to persons served by the water system.
 - (d) Another method approved in writing by DEP.
- b. Monitoring - The water supplier should be required to monitor at least daily at the entry point(s) for the detected contaminant until the weekly average concentration of the daily samples indicates the contaminant no longer poses an imminent threat. Monitoring should then continue as in Part III-2 above.
- c. For all bottled water and retail water for which the average of the original and a check sample exceeds the maximum unregulated contaminant concentration, the water supplier should recall the contaminated water and cease distribution until the contaminant concentration is equal to or less than the maximum unregulated contaminant concentration.
4. For systems, which have installed treatment to remove an unregulated contaminant, monitoring for the unregulated contaminant for which treatment has been installed should be conducted at least quarterly.
5. Every effort will be made to keep the Contaminant Summary information on the intranet current with revised information from EPA, but verification from BWSWM may be necessary before actions are taken.

Should the health risk be determined to be a serious one and the water supplier is unable to issue a notice to its water customers, DEP will issue a notice on behalf of the water supplier.

Find Public Notification requirements in
25 Pa. Code Chapter 109
Subchapter D. PUBLIC NOTIFICATION

In addition, community water systems are required to prepare and provide to their customers annual Consumer Confidence Reports (CCRs) on the quality of water delivered by the systems. CCRs summarize information that a community water system collects, such as the source(s) of water provided, levels of detected contaminants, violations of any state regulations, health information concerning drinking water violations and the potential risks from detected contaminants. If a system has performed voluntary monitoring that indicates the presence of non-regulated contaminants in the finished water, DEP encourages the system to report any results that may indicate a health concern. DEP considers any detection above a proposed MCL or health advisory level to indicate concern. For these contaminants, DEP recommends that the report contain: (1) the results of monitoring, and (2) an explanation of the significance of the results, noting the existence of the health advisory or proposed MCL.

DRINKING WATER STANDARDS AND HEALTH ADVISORIES TABLE

As an overview, Pennsylvania Drinking Water Standards, adopted from standards developed by EPA, are currently in effect in Pennsylvania. Generally, the Pennsylvania MCLs include both federal EPA primary MCLs as State MCLs and the current EPA secondary maximum contaminant levels (SMCLs) as State MCLs. Exceptions to the federal standards include, but are not necessarily limited to, the State primary MCL for fluoride of 2 mg/L and the secondary MCL for aluminum of 0.2 mg/L. Specific details of Pennsylvania standards and treatment techniques are provided in 25 Pa. Code Chapter 109, available on the Internet at <http://www.pacode.com/secure/data/025/chapter109/s109.202.html> . §109.202. State MCLs, MRDLs and treatment technique requirements.

Pennsylvania Drinking Water Standards: applicable to public drinking water systems Maximum Contaminant Levels: (22KB PDF file) listed by contaminant at http://www.dep.state.pa.us/dep/deputate/watermgmt/wsm/WSM_DWM/PA-MCLs.pdf and Treatment Technique Requirements: (15KB PDF file) in lieu of an MCL at http://www.dep.state.pa.us/dep/deputate/watermgmt/wsm/WSM_DWM/PA-TrtTech.pdf .

The risk management source for HAs is the federal EPA publication, *Drinking Water Standards and Health Advisories* that is a compendium of regulatory standards and guidance levels for contaminants in drinking water. It is comprised of:

1. Acute, subchronic, and lifetime health advisories developed by EPA's Office of Water, and
2. Qualitative and quantitative carcinogenic potential assessments developed by EPA.

The *Drinking Water Standards and Health Advisories* publication is accessible on the Internet at <http://www.epa.gov/waterscience/drinking/standards/> . Its HAs provide additional information on certain contaminants and are guidance values based on health effects other than cancer. In its *Drinking Water Standards and Health Advisories* table, EPA publishes the cancer risk at 10^{-4} or one in 10,000 risk level. DEP, for the calculation of its maximum unregulated contaminant concentration, uses a cancer risk at 10^{-6} or one in one million.

The cancer risk and associated contaminant concentration are assumed to be a linear (straight-line) relationship. To convert a cancer risk with its associated contaminant concentration from 10^{-4} to 10^{-6} risk level, move the decimal point of the contaminant concentration two places to the left. For example, if 0.5 milligrams per liter of contaminant would pose a lifetime risk of 10^{-4} , then 0.005 milligrams per liter would pose a risk of 10^{-6} . As a service, we are providing the associated Chemical Abstract Service registry numbers (CASN) for two contaminants listed in the **Drinking Water Advisory Table** of EPA's *Drinking Water Standards and Health Advisories*.

CHEMICAL	(CAS Number)
Methyl tertiary-butyl ether (MtBE)	1634-04-4
Sodium	7440-23-5

While the health advisory information is meant primarily to be a technical resource for staff in the drinking water program responding to contamination incidents involving *public* water supplies (water provided by municipal authorities, investor-owned utilities, schools, factories, mobile home parks, etc.), this information may also be useful to technical staff in other program areas of DEP who need to deal with contamination incidents involving drinking water supplies owned by *private* home-owners. As a follow-up to reviewing the health advisory data, users from field operations of other programs should also utilize the knowledge and expertise of the drinking water staff in their respective locations for additional advice.

CONTAMINANT FACT SHEET RESOURCES

Drinking water, including bottled water, may reasonably be expected to contain at least small amounts of some contaminants. The presence of contaminants does not necessarily indicate that water poses a health risk.

More detailed information on specific contaminants is available at <http://www.epa.gov/safewater/hfacts.html> . Consumer and technical fact sheet links are provided at <http://www.epa.gov/safewater/Pubs/standards.html#chem1> .

GLOSSARY OF TERMS

Absorbed dose. The amount of a chemical that enters the body of an exposed organism.

Absorption. The uptake of water or dissolved chemicals by a cell or an organism.

Absorption factor. The fraction of a chemical making contact with an organism that is absorbed by the organism.

Acceptable daily intake (ADI). Estimate of the largest amount of chemical to which a person can be exposed on a daily basis that is not anticipated to result in adverse effects (usually expressed in mg/kg/day). (Synonymous with RfD.)

Activated Carbon. A highly adsorbent form of carbon used to remove odors and toxic substances from water.

Active transport. An energy-expending mechanism by which a cell moves a chemical across the cell membrane from a point of lower concentration to a point of higher concentration, against the diffusion gradient.

Acute. Occurring over a short period of time; used to describe brief exposures and effects which appear promptly after exposure.

Additive Effect. Combined effect of two or more chemicals equal to the sum of their individual effects.

Administrative Order on Consent. A legal agreement signed by EPA and an individual, business, or other entity through which the violator agrees to pay for correction of violations, take the required corrective or clean-up actions, or refrain from the activity. The agreement describes actions to be taken at a site and may be subject to a public comment period.

Adsorption. The process by which chemicals are held on the surface of a mineral or soil particle. Compare with absorption.

Advisory. A non-regulatory document that communicates risk information to persons who may have to make risk management decisions.

Aerobic. Life or processes that require, or are not destroyed by, the presence of oxygen.

Ambient. Environmental or surrounding conditions.

Anaerobic. A life or process that occurs in, or is not destroyed by, the absence of oxygen.

Animal studies. Investigations using animals as surrogates for humans, on the expectation that results in animals are pertinent to humans.

Antagonism. Interference or inhibition of the effect of one chemical by the action of another chemical.

Aquifer. An underground geological formation, or group of formations, containing usable amounts of groundwater that can supply wells and springs.

Assay. A test for a particular chemical or effect.

Background level. In toxic chemical monitoring, the average presence in the environment, originally referring to naturally occurring phenomena.

Beta Particle. An elementary particle emitted by radioactive decay; may cause skin burns; is stopped by a thin sheet of paper.

Bias. An inadequacy in experimental design that leads to results or conclusions not representative of the population under study.

Bioaccumulation. Accumulation of substance in a plant or animal as a result of repeated exposure to a substance not easily expelled from the body.

Bioassay. Test which determines the effect of a chemical on a living organism.

Bioconcentration. The accumulation of a chemical in tissues of an organism (such as fish) to levels that are greater than the level in the medium (such as water) in which the organism resides. (See bioaccumulation.)

Biodegradation. Decomposition of a substance into more elementary compounds by the action of microorganisms such as bacteria.

Biotransformation. Conversion of a substance into other compounds by organisms; includes biodegradation.

BW. Body weight.

CAG. Carcinogen Assessment Group of the federal EPA.

Cancer. A disease characterized by the rapid and uncontrolled growth of aberrant cells into malignant tumors.

Carcinogen. A chemical which causes or induces cancer.

CAS registration number. A number assigned by the Chemical Abstracts Service to identify a chemical.

Central nervous system. Portion of the nervous system which consists of the brain and spinal cord; CNS.

Characteristic (Solid Waste). Any one of the four categories used in defining hazardous waste: ignitability, corrosivity, reactivity, and toxicity.

Chronic. Occurring over a long period of time, either continuously or intermittently; used to describe ongoing exposures and effects that develop only after a long exposure.

Chronic exposure. Long-term, low level exposure to a toxic chemical.

Cleanup. Actions taken to deal with a release or threat of release of a hazardous substance that could affect humans and/or the environment.

Clinical studies. Studies of humans suffering from symptoms induced by chemical exposure.

Comment period. Time given the public to review and comment on a proposed EPA action or rulemaking after it is published in the Federal Register.

Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA). Commonly known as Superfund; federal law authorizing investigation and remediation of abandoned or uncontrolled hazardous waste sites. Funded by a special tax that goes into a Trust Fund; EPA can either pay for site cleanup when responsible parties are unavailable, unwilling or unable to perform the work or take legal action to force the responsible parties to clean up the site or pay back the government for the cost of cleanup.

Confounding factors. Variables other than chemical exposure level which can affect the incidence or degree of a parameter being measured.

Consent Decree (CD). A legal document, approved and issued by a judge, that formalizes an agreement reached between EPA and potentially responsible parties (PRPs) where PRPs will perform all or part of a Superfund site cleanup.

Corrosion. The dissolving and wearing away of metal caused by a chemical reaction such as between acid water and water pipes.

Cost/benefit analysis. A quantitative evaluation of the costs which would be incurred versus the overall benefits to society of a proposed action such as the establishment of an acceptable dose of a toxic chemical.

Cost-effective alternative. The cleanup alternative selected for a site on the National Priorities List based on technical feasibility, permanence, reliability, and cost.

Cumulative exposure. The summation of exposures of an organism to a chemical over a period of time.

Curie. A quantitative measure of radioactivity equal to 3.7×10^{10} disintegrations per second.

Degradation. Chemical or biological breakdown of a complex compound into simpler compounds.

Dermal exposure. Contact between a chemical and the skin.

Diffusion. The movement of suspended or dissolved particles from a more concentrated to a less concentrated region as a result of the random movement of individual particles; the process tends to distribute them uniformly throughout the available volume.

DNA. Deoxyribonucleic acid; the molecule in which the genetic information for most living cells is encoded.

Dosage. The quantity of a chemical administered to an organism.

Dose. The actual quantity of a chemical to which an organism is exposed. (See absorbed dose.)

Dose-response. A quantitative relationship between the dose of a chemical and an effect caused by the chemical.

Dose-response evaluation. A component of risk assessment that describes the quantitative relationship between the amount of exposure to a substance and the extent of toxic injury or disease.

Dose-response relationship. The quantitative relationship between the amount of exposure to a substance and the extent of toxic injury produced.

Drinking Water Advisory. A nonregulatory concentration of a contaminant in water that is likely to be without adverse effects on both health and aesthetics.

DWEL. Drinking Water Equivalent Level - estimated exposure (in mg/L) which is interpreted to be protective for noncarcinogenic endpoints of toxicity over a lifetime of exposure, assuming that this exposure would be limited exclusively to drinking water that contained the contaminant.

Endangerment assessment. A site-specific risk assessment of the actual or potential danger to human health or welfare and the environment from the release of hazardous substances or waste. The endangerment assessment document is prepared in support of enforcement actions under CERCLA or RCRA.

Endpoint. A biological effect used as an index of the effect of a chemical on an organism.

Environmental Response Team. EPA experts in Edison, New Jersey, and Cincinnati, Ohio, who can provide around-the-clock technical assistance to EPA regional offices and states during all types of emergencies involving hazardous waste sites and spills of hazardous substances.

Epidemiologic study. Study of human populations to identify causes of disease. Such studies often compare the health status of a group of persons who have been exposed to a suspect agent with that of a comparable non-exposed group.

Estimated Exposure Dose (EED). The measured or calculated dose to which humans are likely to be exposed considering exposure by all sources and routes.

Exposure. Contact with a chemical or physical agent which represents a potential health threat to the living organisms in that environment.

Exposure assessment. The determination or estimation (qualitative or quantitative) of the magnitude, frequency, duration, route, and extent (number of people) of exposure to a chemical.

Exposure coefficient. Term which combines information on the frequency, mode, and magnitude of contact with contaminated medium to yield a quantitative value of the amount of contaminated medium contacted per day.

Exposure level, chemical. The amount (concentration) of a chemical at the absorptive surfaces of an organism.

Exposure scenario. A set of conditions or assumptions about sources, exposure pathways, concentrations of toxic chemicals and populations (numbers, characteristics and habits) which aid the investigator in evaluating and quantifying exposure in a given situation.

Extrapolation. Estimation of unknown values by extending or projecting from known values.

Extremely hazardous substances. Any of 406 chemicals identified by EPA on the basis of toxicity; listed under SARA Title III.

Fecal coliform bacteria. A group of bacteria which are commonly found in the intestinal tracts of mammals. Their presence in water is an indication of pollution and possible contamination by pathogens.

First Draw. The water that immediately comes out when a tap is first opened. This water is likely to have the highest level of lead contamination from plumbing materials.

Fluorosis. An abnormal condition caused by excessive intake of fluorine, characterized chiefly by mottling of the teeth.

Formulation. The substance or mixture of substances which is comprised of all active and inert ingredients in a pesticide.

Fresh water. Water that generally contains less than 1,000 milligrams per liter of dissolved solids.

Gamma radiation. Gamma rays are true rays of energy in contrast to alpha and beta radiation. The properties are similar to x-rays and other electromagnetic waves. They are the most penetrating waves of radiant nuclear energy but can be blocked by dense materials such as lead.

Gavage. Type of exposure in which a substance is administered to an animal through a stomach tube.

Gene. A length of DNA that directs the synthesis of a protein.

Generator. A facility or mobile source that emits pollutants into the air or releases hazardous wastes into water or soil.

Gram. 1/454 of a pound.

Gross alpha particle activity. Total activity due to emission of alpha particles. Used as the screening measurement for radioactivity generally due to naturally occurring radionuclides. Activity is commonly measured in picocuries (pCi).

Gross beta particle activity. Total activity due to emission of beta particles. Since the decay products of fission are beta particles and gamma ray emitters, used as the screening measurement for radioactivity from man-made radionuclides. Activity is commonly measured in picocuries (pCi).

Half-life. The length of time required for the mass, concentration, or activity of a chemical or physical agent to be reduced by one-half.

Halogen. Any of a group of five chemically-related non-metallic elements (bromine, fluorine, chlorine, iodine, astatine) that form Group VIIA of the periodic table.

Hazard evaluation. A component of risk assessment that involves gathering and evaluating data on the types of health injury or disease (e.g., cancer) that may be produced by a chemical and on the conditions of exposure under which injury or disease is produced.

Hazardous Ranking System (HRS). The principle screening tool used by EPA to evaluate risks to public health and the environment associated with abandoned or uncontrolled hazardous waste sites. The HRS calculates a score based on the potential of hazardous substances spreading from the site through the air, surface water, or groundwater and on other factors such as nearby population. This score, from 0 to 100, is the primary factor in deciding if the site should be placed on the National Priorities List, and, if so, what ranking it should have compared to other sites on the list.

Hazardous substance. (1) Any material that poses a threat to human health and/or the environment. Typical hazardous substances are toxic, corrosive, ignitable, explosive, or chemically reactive. (2) Any substance named by EPA to be reported if a designated quantity of the substance is spilled in the waters of the United States or if otherwise emitted into the environment.

Heavy Metals. Metallic elements with high atomic weights which characteristically can damage living things at low concentrations and tend to accumulate in the food chain. They include mercury, chromium, cadmium, arsenic, and lead.

Hematopoiesis. The production of blood and blood cells.

Hepatic. Pertaining to the liver.

Hepatoma. A malignant tumor occurring in the liver.

Heterotrophic bacteria. Bacteria which are dependent on organic matter for food, absolutely requiring at least one organic compound for its source of carbon.

High-to-low dose extrapolation. The process of prediction of low exposure risks to rodents from the measured high exposure-high risk data.

Histology. The study of the structure of cells and tissues; usually involves microscopic examination of tissue slices.

Human equivalent dose. A dose which, when administered to humans, produces an effect equal to that produced by a dose in animals.

Human exposure evaluation. A component of risk assessment that involves describing the nature and size of the population exposed to a substance and the magnitude and duration of their exposure. The evaluation could concern past exposures, current exposures, or anticipated exposures.

Human health risk. The likelihood (or probability) that a given exposure or series of exposures may have or will damage the health of individuals experiencing the exposures.

Hydrocarbons. Organic compounds containing only carbon and hydrogen; petroleum is a complex mixture of hydrocarbons with a small amount of other substances.

Hydrogeology. The geology of groundwater, with particular emphasis on the chemistry and movement of water.

Hydrology. The science dealing with the properties, distribution, and circulation of water.

Incidence of tumors. Percentage of animals with tumors.

Incineration. (1) Burning of certain types of solid, liquid or gaseous materials. (2) A treatment technology involving destruction of waste by controlled burning at high temperatures.

Indicator organisms. Organisms whose survival and presence in an environment indicate that environment's physical conditions.

Information file (Superfund). A file that contains accurate up-to-date documents on a Superfund site. The file is usually located in a public building (repository) such as a school, library, or city hall that is convenient for local residents.

Ingestion. Type of exposure through the mouth.

Inhalation. Type of exposure through the lungs.

Injection well. A well into which fluids are injected for purposes such as waste disposal, improving the recovery of crude oil, or solution mining.

Inorganic chemicals. Chemicals not basically of carbon structure.

Integrated exposure assessment. A summation over time, in all media, of the magnitude of exposure to a toxic chemical.

Interspecies extrapolation model. Model used to extrapolate from results observed in laboratory animals to humans.

In vitro studies. Studies of chemical effects conducted in tissues, cells or subcellular extracts from an organism (i.e., not in the living organism).

In vivo studies. Studies of chemical effects conducted in intact living organisms; in vivo tests are those laboratory experiments carried out on whole animals or human volunteers.

Irreversible effect. Effect characterized by the inability of the body to partially or fully repair injury caused by a toxic agent.

Latency. Time from the first exposure to a chemical until the appearance of a toxic effect.

Leachate. A liquid that results from water collecting contaminants as it percolates through wastes, agricultural pesticides, or fertilizers. The leaching process may result in hazardous substances entering surface water, groundwater, or soil.

Lesion. A pathological or traumatic discontinuity of tissue or loss of function of a part.

Lethal. Deadly; fatal.

Lethal Concentration 50 (LC50). The concentration of a chemical in air or water which is expected to cause death in 50 percent of test animals living in that air or water.

Lethal Dose 50 (LD50). The dose of a chemical taken by mouth or absorbed by the skin which is expected to cause death in 50 percent of the test animals so treated.

Lifetime exposure. Total amount of exposure to a substance that a human would receive in a lifetime (usually assumed to be 70 years).

Linearized multistage model. Derivation of the multistage model, where the data are assumed to be linear at low doses.

LOAEL. Lowest-Observed-Adverse-Effect Level; the lowest dose in an experiment which produced an observable adverse effect.

Malignant. Very dangerous or virulent, causing or likely to cause death.

Margin of Exposure (MOE). The ratio of the no-observed-adverse-effect-level (NOAEL) to the estimated exposure dose (EED).

Margin of Safety (MOS). The older term used to describe the margin of exposure (MOE).

Mathematical Model. Model used during risk assessment to perform extrapolations.

Metabolism. The sum of the chemical reactions occurring within a cell or a whole organism; includes the energy-releasing breakdown of molecules (catabolism) and the synthesis of new molecules (anabolism).

Metabolite. Any product of metabolism, especially a transformed chemical.

Metastatic. Pertaining to the transfer of disease from one organ or part to another not directly connected with it.

Microgram (ug). One-millionth of a gram (3.5×10^{-8} oz. = 0.000000035 oz.).

Milligram (mg). One-thousandth of a gram (3.5×10^{-5} oz. = 0.000035 oz.).

Mitigation. Measures taken to reduce adverse impacts on the environment.

Modeling. Use of mathematical equations to simulate and predict real events and processes.

Modifying Factor. Uncertainty factor that is greater than zero and less than or equal to 10; the magnitude of the modifying factor depends upon the professional assessment of scientific uncertainties of the study and database not explicitly treated with the standard uncertainty factors (e.g., the

completeness of the overall database and the number of species tested); the default value for the modifying factor is 1.

Monitoring. Periodic or continuous surveillance or testing to determine the level of compliance with statutory requirements and/or pollutant levels in various media or in humans, animals, and other living things.

Monitoring Wells. Wells drilled at a hazardous waste management facility or Superfund site to collect groundwater samples for the purpose of physical, chemical, or biological analysis to determine such things as the direction in which groundwater flows and the types and amounts of contaminants present.

Mortality. The number of deaths in a given time or place.

MTD. Maximum tolerated dose, the dose that an animal species can tolerate for a major portion of its lifetime without significant impairment or toxic effect other than carcinogenicity.

Multistage model. Mathematical model based on the multistage theory of the carcinogenic process, which yields risk estimates either equal to or less than the one-hit model.

Mutagen. An agent that causes a permanent genetic change in a cell other than that which occurs during normal genetic recombination.

National Oil and Hazardous Substances Contingency Plan (NOHSCP/NCP). The federal regulation that guides the determination of the sites to be corrected under the Superfund program and the program to prevent or control spills into surface waters or other portions of the environment.

National Priorities List (NPL). EPA's list of the serious uncontrolled or abandoned hazardous waste sites identified for possible long-term remedial action under Superfund. The list is based on the Hazard Ranking System (HRS). EPA is required to update the NPL at least once a year.

National Response Center (NRC). The federal center operated by the U.S. Coast Guard that receives and evaluates reports of oil and hazardous substance releases into the environment and notifies the appropriate agency; open 24 hours a day.

National Response Team (NRT). Representative of 13 federal agencies that, as a team, coordinate federal responses to nationally significant incidents of pollution and provide advice and technical assistance to the responding agency(ies) before and during a response action.

Necrosis. Death of cells or tissue.

Neoplasm. An abnormal growth or tissue, as a tumor.

Neurotoxicity. Exerting a destructive or poisonous effect on nerve tissue.

NOAEL. No-Observed-Adverse-Effect Level; the highest dose in an experiment which did not produce an observable adverse effect.

NOEL. No-Observed-Effect Level; dose level at which no effects are noted.

Non-point source. Pollution sources which are diffuse and do not have a single point of origin or are not introduced into a receiving stream from a specific outlet.

NTP. National Toxicology Program.

Oncogenic. A substance that causes tumors, whether benign or malignant.

Oncology. Study of tumors.

One-hit model. Mathematical model based on the biological theory that a single “hit” of some minimum critical amount of a carcinogen at a cellular target -- namely DNA -- can initiate an irreversible series of events, eventually leading to a tumor.

On-Scene Coordinator (OSC). The predesignated EPA, Coast Guard, or Department of Defense official who coordinates and directs Superfund removal actions or Clean Water Act oil- or hazardous-spill corrective actions.

Operable Unit. Term for each of a number of separate activities undertaken as part of a Superfund site cleanup.

Operation and Maintenance, O & M, (Superfund). Activities conducted at a site after a Superfund site action is completed to ensure that the action is effective and operating properly.

Oral. Of the mouth; through or by the mouth.

Organic chemicals. Naturally occurring (animal- or plant-produced) or synthetic substances containing mainly carbon, hydrogen, nitrogen and oxygen. Other atoms found in organic chemicals may include chlorine, bromine, iodine, sulfur, phosphorus, and many others.

Pathogen. Any disease-causing agent, usually applied to living agents.

Pathology. The study of disease.

Pathway exposure. The route by which a contaminant travels from the source area to reach a receptor (humans, birds, etc.).

Permeability coefficient. The rate(s) that chemicals cross through the layers of dermal or respiratory cells. Constant for a given substance (moving through a given membrane).

Permissible dose. The dose of a chemical that may be received by an individual without the expectation of a significantly harmful result.

Pharmacokinetics. The dynamic behavior of chemicals inside biological systems; it includes the processes of uptake, distribution, metabolism, and excretion.

Point source. A stationary location or fixed facility from which pollutants are discharged or emitted.

Population at risk. A population subgroup that is more likely to be exposed to a chemical, or is more sensitive to a chemical, than is the general population.

Potency. Amount of material necessary to produce a given level of a deleterious effect.

Potentially Responsible Party (PRP). An individual or company (such as owners, operators, transporters, or generators) potentially responsible for, or contributing to, the contamination problems at a Superfund site. Whenever possible, EPA requires PRPs, through administrative and legal actions, to clean up hazardous waste sites PRP's have contaminated.

Potentiation. The effect of one chemical to increase the effect of another chemical.

ppb. Parts per billion.

ppm. Parts per million.

Preliminary Assessment. The process of collecting and reviewing available information about a known or suspected waste site or release.

Prevalence study. An epidemiological study which examines the relationships between diseases and exposures as they exist in a defined population at a particular point in time.

Prospective study. An epidemiological study which examines the development of disease in a group of persons determined to be presently free of the disease.

Qualitative. Descriptive of kind, type or direction, as opposed to size, magnitude, or degree.

Quality Assurance/Quality Control (QA/QC). A system of procedures, checks, audits, and corrective actions used to ensure that field sampling and laboratory analysis are of the highest achievable quality.

Quantitative. Descriptive of size, magnitude, or degree.

Receptor. (1) In biochemistry, a specialized molecule in a cell that binds a specific chemical with high specificity and high affinity. (2) In exposure assessment, an organism that receives, may receive, or has received environmental exposure to a chemical.

Record of Decision (ROD). A public document that explains which cleanup alternative(s) will be used at National Priorities List sites where, under Superfund, Trust Funds pay for the cleanup.

Red Border. An EPA document that is undergoing final review before being submitted for final management decision.

Regional Response Team (RRT). Representatives of federal, local, and state agencies who may assist in coordination before and during a Superfund response action.

Remedial Action (RA). The actual construction or implementation phase of a Superfund site cleanup that follows remedial design.

Remedial design (RD). An engineering phase that follows the Record of Decision where technical drawings and specifications are developed for the subsequent remedial action at a site on the National Priorities List.

Remedial Investigation/Feasibility Study (RI/FS). An EPA investigation at a Superfund site to gather the data necessary to determine the type and extent of contamination, and to identify and analyze cleanup alternatives. These two distinct but related studies are usually performed at the same time.

Remedial Project Manager (RPM). The EPA official responsible for overseeing remedial action at a site.

Remedial response. A long-term action that stops or substantially reduces a release or threat of a release of hazardous substances that is serious but not an immediate threat to public health.

Removal action. Short-term immediate actions taken to address release of hazardous substances that require expedited response.

Renal. Pertaining to the kidney.

Reservoir. A tissue in an organism or a place in the environment where a chemical accumulates, from which it may be released at a later time.

Resource Conservation and Recovery Act (RCRA). Federal law that regulates management and disposal of hazardous substances currently being generated, treated, transported, stored, and disposed.

Response Action. A Superfund authorized action involving either a short-term removal action or a long-term removal response that may include, but is not limited to: treatment, containment, or destruction of hazardous waste on-site or off-site; or identification and halting further movement of the contaminants.

Responsiveness Summary. A summary of oral and/or written public comments received by EPA during a comment period on key EPA documents, and EPA's responses to those comments.

Retrospective study. An epidemiological study which compares diseased persons with non-diseased persons and works back in time to determine exposures.

Reversible effect. An effect which is not permanent, especially adverse effects which diminish when exposure to a toxic chemical is ceased.

RfD. Reference dose; the daily exposure level which, during an entire lifetime of a human, appears to be without appreciable risk on the basis of all facts known at the time. (Synonymous with ADI.)

Risk. The potential for realization of unwanted adverse consequences or events.

Risk assessment. A qualitative or quantitative evaluation of the environmental and/or health risk resulting from exposure to a chemical or physical agent (pollutant); combines exposure assessment results with toxicity assessment results to estimate risk.

Risk characterization. Final component of risk assessment that involves integration of the data and analysis involved in hazard evaluation, dose-response evaluation, and human exposure evaluation to determine the likelihood that humans will experience any of the various forms of toxicity associated with a substance.

Risk estimate. A description of the probability that organisms exposed to a specified dose of chemical will develop an adverse response (e.g., cancer).

Risk factor. Characteristic (e.g., race, sex, age, obesity) or variable (e.g., smoking, occupational exposure level) associated with increased probability of a toxic effect.

Risk management. Decisions about whether an assessed risk is sufficiently high to present a public health concern and about the appropriate means for control of a risk judged to be significant.

Risk specific dose. The dose associated with a specified risk level.

Route of exposure. The avenue by which a chemical comes into contact with an organism (e.g., inhalation, ingestion, dermal contact, injection).

Safe. Condition of exposure under which there is a “practical certainty” that no harm will result in exposed individuals.

Sink. A place in the environment where a compound or material collects. (See reservoir.)

Site Inspection. The collection of information from a Superfund site necessary to score the site, using the Hazard Ranking System, and to determine if the site presents an immediate threat that requires prompt removal action.

Solder. A metallic compound used to seal the joints between pipes. Until recently, most solder contained 50 percent lead.

Sorption. A surface phenomenon which may be either absorption or adsorption, or a combination of the two; often used when the specific mechanism is not known.

Stochastic. Based on the assumption that the actions of a chemical substance result from probabilistic events.

Stratification. (1) The division of a population into subpopulations for sampling purposes. (2) The separation of environmental media into layers, as in lakes.

Subchronic. Of intermediate duration, usually used to describe studies or levels of exposure between 5 and 90 days.

Superfund. The common name used for the Comprehensive Environmental Response, Compensation, and Liability Act.

Superfund Amendments and Reauthorization Act (SARA). Modifications to CERCLA enacted on October 17, 1986.

Synergism. An interaction of two or more chemicals that result in an effect that is greater than the sum of their effects taken independently.

Systemic. Relating to the whole body, rather than its individual parts.

Systemic effects. Effects observed at sites distant from the entry point of a chemical due to its absorption and distribution into the body.

Teratogen. Substance that causes malformation or serious deviation from normal development of embryos and fetuses.

Teratogenesis. The induction of structural or functional development abnormalities by external factors acting during gestation; interference with normal embryonic development.

Therapeutic Index. The ratio of the dose required to produce toxic or lethal effects to the dose required to produce non-adverse or therapeutic response.

Threshold. The lowest dose of a chemical at which a specified measurable effect is observed and below which it is not observed.

Time-Weighted Average. The average value of a parameter (e.g., concentration of a chemical in air) that varies over time.

Tissue. A group of similar cells.

Toxicant. A harmful substance or agent that may injure an exposed organism.

Toxicity. The quality or degree of being poisonous or harmful to plant, animal or human life.

Toxicity assessment. Characterization of the toxicological properties and effects of a chemical, including all aspects of its absorption, metabolism, excretion and mechanism of action, with special emphasis on establishment of dose-response characteristics.

Transformation. Acquisition by a cell of the property of uncontrolled growth.

Treatment, storage, and disposal facility (TSD). Site where a hazardous substance is treated, stored, or disposed. TSD facilities are regulated by EPA and states under RCRA.

Trust Fund (CERCLA). A fund set up under CERCLA to help pay for cleanup of hazardous waste sites and for legal action to force those responsible for sites to clean them up.

Tumor incidence. Fraction of animals having a tumor of a certain type.

Uncertainty factor. A number (equal to or greater than one) used to divide NOAEL or LOAEL values derived from measurements in animals or small groups of humans, in order to estimate a NOAEL value for the whole human population.

Unit cancer risk. Estimate of the lifetime risk caused by each unit of exposure in the low exposure region.

Upper bound estimate. Estimate not likely to be lower than the true risk.

Volatile. Readily vaporizable at a relatively low temperature.

Working Level (WL). A unit of measure for documenting exposure to radon decay products. One working level is equal to approximately 200 picocuries per liter (pCi/L).

ACRONYMS AND ABBREVIATIONS

ARAR	applicable or relevant and appropriate requirement
ATSDR	Agency for Toxic Substances and Disease Registry
AWWA	American Water Works Association
BAT	best available technology
BEIR	National Academy of Sciences Advisory Committee on the Biological Effects of Ionizing Radiation
BTEX	benzene, toluene, ethylbenzene, and xylene(s)
BW	body weight
CAA	Clean Air Act
CAG	Carcinogen Assessment Group, U.S. EPA
CAS	Chemical Abstracts Service
CERCLA	Comprehensive Environmental Responsibility, Compensation and Liability Act (1976); Superfund
CFC	chlorofluorocarbon
CFR	Code of Federal Regulations
CFU	colony-forming units (bacteriological analysis)
CHD	county health department
CNS	central nervous system
CRAVE	Carcinogen Risk Assessment Verification Endeavor
CRL	cancer risk level
CWA	Clean Water Act
DBCP	1,2 dibromo-3-chloropropane
D/DBP	disinfectants and disinfection by-products
DNA	deoxyribonucleic acid
DW	drinking water
DWEL	Drinking Water Equivalent Level
E	exponent (e.g. 1.0 E-6 = 1.0 x 10 to the power of -6)
EDB	ethylene dibromide; 1,2-dibromoethane
EP	extraction procedure (solid waste)
EPA	U.S. Environmental Protection Agency
EQB	Environmental Quality Board, Pennsylvania
FFDCA	Federal Food, Drug, and Cosmetics Act
FIFRA	Federal Insecticide, Fungicide and Rodenticide Act
FOIA	Freedom of Information Act
FR	Federal Register
FRDS	Federal Reporting Data System
GAC	granular activated carbon
GC	gas chromatograph
GC/MS	gas chromatograph/mass spectrometer
GI	gastrointestinal
GIS	geographic information system

GRAS	generally recognized as safe
HA	health advisory
HAA	haloacetic acid
HAN	haloacetonitrile
HPLC	high-performance liquid chromatograph
HRS	hazard ranking system
HSCA	Hazard Sites Cleanup Act, Pennsylvania
HSWA	Hazardous and Solid Waste Amendments (1984)
IARC	International Agency for Research on Cancer
ICP-MS	inductively coupled plasma - mass spectrometry
insol.	insoluble
IOC	inorganic chemical
IUPAC	International Union of Pure and Applied Chemistry
kg	kilogram
Koc	organic carbon partition coefficient
Kow	n-octanol/water partition coefficient
L	liter
LC _{LO}	lethal concentration low (inhalation)
LC ₅₀	lethal concentration 50 percent (inhalation)
LD _{LO}	lethal dose low
LD ₅₀	lethal dose 50 percent
LOAEL	lowest-observed-adverse-effect level
LUST	leaking underground storage tank
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MDL	method detection limit
MED	minimum effective dose
MEK	methyl ethyl ketone
MF	modifying factor
mg/kg/dy	milligrams per kilograms of body weight per day
mg/L	milligram per liter
MOE	margin of exposure
MOS	margin of safety
MOU	memorandum of understanding
mrem	millirem
MtBE	methyl tertiary butyl ether (MTBE)
MTD	maximum tolerated dose
NAAQS	National Ambient Air Quality Standards
NAS	National Academy of Sciences
ng	nanogram
NIH	National Institutes of Health
NIOSH	National Institute of Occupational Safety and Health

NLM	National Library of Medicine
NOAEL	no-observed-adverse-effect level
NORM	naturally occurring radioactive materials
NPDES	National Pollutant Discharge Elimination System
NPDWR	National Primary Drinking Water Regulations
NPL	National Priorities List (Superfund)
NRC	National Research Council
NRC	National Response Center
NTIS	National Technical Information Service
NTP	National Toxicology Program
OGWDW	Office of Ground Water and Drinking Water, U.S. EPA
ORD	Office of Research and Development, U.S. EPA
ORP	oxidation-reduction potential
ORSANCO	Ohio River Valley Water Sanitation Commission
OSC	on-scene coordinator
OSHA	U.S. Occupational Safety and Health Administration
OSWER	Office of Solid Waste and Emergency Response, U.S. EPA
PAC	powdered activated carbon
PADWIS	Pennsylvania Drinking Water Information System
PAH	polycyclic aromatic hydrocarbon
PCB	polychlorinated biphenyl
pCi	picocurie
PEL	permissible exposure level
PENNVEST	Pennsylvania Infrastructure Investment Authority Act
POTW	publicly owned (sewage) treatment works
ppb	parts per billion
ppm	parts per million
ppt	parts per trillion
PQL	practical quantitation level
PRP	potentially responsible party
PTA	packed tower aeration
QA/QC	quality assurance/quality control
RAD	radiation absorbed dose
RBC	red blood cells
RCRA	Resource Conservation and Recovery Act, U.S. EPA
REM	roentgen equivalent man
RfD	reference dose
RfDi	inhalation reference dose
RfDo	oral reference dose
RI/FS	remedial investigation/feasibility study
ROD	Record of Decision (Superfund)
RP	responsible party
RQ	reportable quantity
RRT	regional response team
RSC	relative source contribution

RTECS	registry of toxic effects of chemical substances
SARA	Superfund Amendments and Reauthorization Act of 1986
s.c.	subcutaneous
SDWA	Safe Drinking Water Act
SI	site investigation (Superfund)
SI	International System of Units
SMCL	secondary maximum contaminant level
SOC	synthetic organic chemicals
sol.	soluble
SPC	standard plate count (bacteriological analysis)
STEL	short-term exposure limit
STORET	Storage and Retrieval of Water-Related Data
TAD	total absorbed dose
TBC	to be considered (Superfund)
TCDD	dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxin)
TCDF	tetrachlorodibenzofurans
TCE	trichloroethylene
TD	toxic dose
TDS	total dissolved solids
THM	trihalomethane
TIC	tentatively identified compounds
TLV	threshold limit value
TOC	total organic carbon
TOX	total organic halide
TPH	total petroleum hydrocarbon
TRI	Toxic Release Inventory, U.S. EPA
TSCA	Toxic Substances Control Act, U.S. EPA
TT	treatment technique
TTHM	total trihalomethanes
TWA	time-weighted average
UF	uncertainty factor
USGS	United States Geological Survey
UST	underground storage tank
ug/L	microgram per liter
VOA	volatile organic analysis
VOC	volatile organic chemical
v/v	volume per volume
WBC	white blood cells
WHO	World Health Organization
WL	working level
WQC	water quality criteria
ZRL	zero risk level

Attachment 2

**Maximum Contaminant Level Goal Drinking
Water Recommendations for Per-
and Polyfluoroalkyl Substances (PFAS)
in the Commonwealth of Pennsylvania**

**By
The Drexel PFAS Advisory Group**

January 2021

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1. Executive Summary

The Drexel PFS Advisory Group (DPAG) is a unique multidisciplinary team engaged by the Commonwealth of PA to provide recommendations for Maximum Allowable Contaminant Level Goals MCLGs to the Commonwealth of Pennsylvania for Per- and polyfluoroalkyl substances (PFAS) in drinking water. Observational epidemiology supports the need for drinking water values below the current recommendations of the United States Environmental Protection Agency (US EPA) lifetime health advisory LHA level of 70 ppt for PFOS and PFOA individually or in combination. Furthermore, the identification of other PFAS in drinking water requires a broader consensus consideration of all these substances. As of this report, the US EPA has not initiated its process for establishing MCLs or MCLGs under the Safe Drinking Water Act. Therefore, specific guidelines for the Commonwealth of Pennsylvania were deemed necessary to protect the safety and well-being of Pennsylvanians.

The DAPG consist of experts in the fields of medical toxicology, epidemiology, environmental toxicology, water drinking standards, and risk assessment. The biographies of the members of the DPAG are included as Appendix A.

The Pennsylvania Department of Environmental Protection (PADEP) tasked the DPAG to review the existing and proposed PFA standards from across the country and independently develop MCLGs to inform the initial phase of the rulemaking process for establishing state drinking water standards. (Appendix B and C) The effort commenced in January 2020 and continued to the delivery of this report. Because of restrictions on

face-to-face interactions due to the Covid19 pandemic, much of the advisory groups work was done through virtual conferences between DPAG and PA DEP during 2020.

The DPAG methodically evaluated existing and proposed standards from across the country for PFAs considered under US EPA method 537.1. PADEP asked DPAG to provide specific recommendations on perfluorononanoic acid (PFNA), perfluorooctanesulfonic acid (PFOS), perfluorooctanoic acid (PFOA), perfluorohexanesulfonic acid (PFHxS), perfluoroheptanoic acid (PFHpA), and perfluorobutanesulfonic acid (PFBS). DPAG added the ammonium salt of hexafluoropropylene oxide dimer (GenX) to the list of reviewed PFAS. This latter addition was approved by the PA DEP.

PA DEP charged the advisory group with producing MCLGs within a year. Hence, the initial effort was to review the existing national and state derive PFA assessments, review the pertinent literature in a focused manner, and generally benefit from prior efforts to develop PFAS health-based values. Once complete, the DPAG independently reconsidered all of the PFAS in question and formed draft recommendations for the PA DEP in the summer of 2020.

The PA DEP placed no expectations on the DPAG other than a scientifically defensible approach in developing these values.

Furthermore, by charging a group with developing MCLGs, the commonwealth asked that we focus on developing values that were not as much influenced by technical difficulties necessary to achieve them – e.g. measurement, remediation, or other mitigation. DPAG purposely sought to maintain an independent mindset with developing these MCLGs and to focus on identifying concentrations that would protect

human health. Each consideration and the evidence behind the evaluation as well as methodical calculation are included in the individual summaries. The Reference Dose and recommended Chronic Non-Cancer MCLGs for the seven PFAS considered are

Table 1.

PFAS	Reference Dose	MCLG proposed
perfluorooctanoic acid (PFOA)	3.9 ng/kg/day	8 PPT
perfluorooctanesulfonic acid (PFOS)	3.1 ng/kg/day	14 PPT
perfluorononanoic acid (PFNA)	2.2 ng/kg/day	6 PPT
perfluorohexanesulfonic acid (PFHxS)	4.0 ng/kg/day	20 PPT
perfluoroheptanoic acid (PFHpA)	None derived	8 PPT
perfluorobutanesulfonic acid (PFBS)	39 ng/kg/day	55 PPT
ammonium salt of hexafluoropropylene oxide dimer (GenX)	75 ng/kg/day	108 PPT

Table 1: Summary of Reference Dose and proposed Chronic Non-Cancer MCLG for perfluorononanoic acid (PFNA), perfluorooctanesulfonic acid (PFOS), perfluorooctanoic acid (PFOA), perfluorohexanesulfonic acid (PFHxS), perfluoroheptanoic acid (PFHpA), perfluorobutanesulfonic acid (PFBS), and the ammonium salt of hexafluoropropylene oxide dimer (GenX)

2. Background

Per- and polyfluoroalkyl substances (PFAS), and the polymers and surfactants made from them, are a large family of greater than 4000 man-made chemicals that contain carbon, fluorine, and other elements and have been used widely in many industrial and consumer applications since the 1950's. Perfluoroalkyl substances are aliphatic substances where all of the carbons are attached to fluorine with the exception of the last one. Polyfluoroalkyl substances are aliphatic substances where at least one, but not all of the carbons are attached to fluorine and contain the perfluoroalkyl moiety (C_nF_{2n+1}).

The carbon-fluorine bond is stable and strong. The perfluoroalkyl moiety's chemical and thermal stability as well as its lipophobic and hydrophobic properties allow it to be very useful in a variety of industries world-wide. They are used to help make products more resistant to oils, grease, stains, and water, and they are used in many industries because they help reduce friction, through their surfactant applications by lowering their surface tension properties i.e. automotive, construction, aerospace. These properties also contribute to their bioaccumulation and environmental persistence. The length of the fluorinated carbon chain distinguishes the short from the long chain PFAS. Long chain PFAS are perfluoroalkyl carboxylic acids with 8 or more carbon chains and perfluoroalkane sulfonic acids with 6 carbon chains and greater. While not specifically stated, perfluoroalkyl chains with 7 or greater carbon atoms are generally considered long chain. The fluorinated carbon chain length determines properties that influence the substance behavior in the environment, organisms, and bioaccumulation. Long chain

compounds include PFNA (9 carbon carboxylic acid), PFOA (8 carbon carboxylic acid), PFHpA (7 carbon carboxylic acid), PFOS (8 carbon sulfonic acid), and PFHxS (7 carbon sulfonic acid). Short chain PFAS include GenX chemicals (6 carbon oxide dimer acid), and PFBS (4 carbon sulfonic acid).

PFASs are present in the environment as a result of their use in a wide array of industrial, commercial, and residential products and applications, including newspaper printing, textile and paper production, metal plating, surfactants in fluoropolymer production, and aqueous film-forming foams (AFFFs), and include consumer products such as outdoor apparel, dental floss, and car wax (Prevedouros 2006, Paul 2008, Konwick 2008). PFASs are emitted to the environment both directly throughout their product and use cycle and indirectly from transformations of their precursors. The majority of emissions are released directly into aquatic environments (Prevedouros 2006, Paul 2008); however, accurate quantification of emissions and resulting environmental exposure are largely lacking (Guo 2009).

2.a. PFAS in Wastewater

PFAS have been found in wastewater treatment plant influents from both municipal and industrial sources, with treated wastewater effluents and sewage sludges (including biosolids) now being viewed as major sources of PFAS to the aquatic environment (Ahrens 2011), which may substantially impact rural water sources. A range of poly- and perfluoroalkyl acids (PFAA) have been routinely detected in wastewater effluents in various countries, including the United States (US) (see review by Hamid 2016). In addition to treated wastewater, various PFAS compounds have been detected in sewage sludges (Venkatesan 2013). In fact, a review by Clarke (2011) ranked PFAS as

the highest priority group of emerging contaminants in biosolids. Taken together, due to the unmitigated use of PFAS in consumer products and the long-term persistence of these compounds, reuse of treated wastewater or land application of biosolids may present a source of PFAS that impact rural communities and agricultural operations.

2.b. PFAS from Landfill Leachate

Due to the widespread use of PFAS in commercial products, various congeners and concentrations of PFAS are likely to be present in all landfills. Landfills receiving waste from industrial facilities (e.g., paints, textiles used in furniture, carpet, upholstery) are expected to have higher concentration of PFAS (Guerra 2014, ITRC 2020). However, low concentrations of PFAS have been detected in the range of ppt to ppb levels at municipal landfills likely due to the use of PFAS on some paper products (Arvaniti 2012, Renou 2008, ITRC 2020). It is important to note that some landfills transferred their leachate to WWTPs for treatment. Perfluoroalkyl sulfonic acids (PFSA) and Perfluoroalkyl carboxylic acids (PFCA) are the most common PFASs in landfills, which are known as PFAAs. PFCAs and PFSA have the carbon chain length C4-C18 as well as C4-C10, respectively. Additionally, PFAAs precursors (e.g., FTOH, n:2 FTCA, and n:2 FTUCAs) existing in the consumer products (Ye 2015; Kotthoff 2015) can degrade to PFAAs throughout disposal in the landfill and product use (Lang 2016, Allred 2015).

2.c. PFAS from the use of AFFF

The U.S. Department of Defense (DoD) has used aqueous film forming foam (AFFF) to suppress fires since the 1970s. PFASs are known to contaminate over 500 DoD sites (Thompson 2012), and repeated historic use at firefighter training areas has

resulted in groundwater and porous media contamination, with groundwater concentrations of select PFASs reaching low mg/L levels (Moody 1999, 2000, 2003, Anderson 2016, Murray 2010, Backe 2013, McGuire 2014, Filipovic 2015, Schultz 2004). While PFAAs are often not the dominant PFASs in AFFF formulations at impacted sites, PFAAs and 6:2 FtS are often the dominant PFASs found in contaminated groundwater (Backe 2013, Houtz 2013, McGuire 2014, Schultz 2004). The predominance of PFAAs in groundwaters is hypothesized to be a result of abiotic and biotic reactions in the subsurface that transform the parent PFAS compounds in AFFF formulation (e.g., fluorotelomer thioamido sulfonates, FtTAoS) into FtSs and PFAAs (Harding-Marjanovic 2015).

2.d. PFAS Fate and Transport in the Environment

While there are many aspects that make PFASs chemistry unique, of particular note are their biological and chemical stability, promoting their persistence in the environment), and the comparatively high solubility limits and adsorptive nature of some PFASs, especially of shorter chain length, making them relatively mobile in aqueous systems (Zareitalabad 2013). Perfluoroalkyl acids (PFAAs), which have a negatively charged head group, low volatility, and high water solubility, are considered to be highly mobile in aqueous phases (Ahrens 2011, Ahrens and Bundschuh 2014), and PFAA transport has often been observed or inferred in the environment (Moody 1999, Lindstrom 2011, McGuire 2014, Baduel 2015, Filipovic 2015). As a consequence of such mobility and concerns of their human health effects, drinking water wells at several downstream localities of DoD sites have been temporarily abandoned. The sorption behavior of PFASs is influenced by their physicochemical properties which vary

depending on their functional head group and chain length (Ahrens 2009, 2011, Ahrens and Ebinghaus 2010). PFAA sorption generally increases with increasing chain length. Longer chain length PFAAs have been demonstrated to bioaccumulate and possibly biomagnify. (Prevedouros 2006, Conder 2008) In addition to the ecological effects, bioaccumulation within a food web may lead to human exposure through dietary consumption (e.g., fish). As a consequence, sediments and biota are considered to act as a sink for longer chains PFAAs in aquatic ecosystems.

3. Approach

The DPAG reviewed a number of recommendations made by EPA and State agencies that chose to create a summative approach to PFAS, combining multiple minimal risk levels or advisory levels into one cumulative drinking water value. No clear consensus exists on this approach and the use of a summative approach was clearly designed to be a shortcut based on a presumption that the agents all have similar health effects and endpoints. While this approach may work for other toxins such as dioxins, furans, and coplanar polychlorinated biphenyls, it does not appear to be based on evidence available for PFAS. The DPAG therefore committed early in the process to developing an individual MCLG for each of the requested PFAS. DPAG further recommends that all PFAS be reviewed individually as they arise for analysis, even if the individual MCLG ultimately needs to be based on chemical similarities to other PFAS only (e.g. see PFHpA in our recommendations).

For each of the PFAS studied, the DPAG identified points of departure and rationale for selection from risk assessments published by other states, the EPA, and a TSTR. DPAG then assessed the underlying critical studies driving the selection of the POD. Every effort was made to use the experience and published findings from other agencies and build and refine on these as much as possible into a best practice approach. USEPA (2000), Beck (2016)

3.a. Maximum Contaminant Level Goals

Maximum Contaminant Level Goals (MCLGs) are maximum drinking water concentrations designed to protect human health. MCLGs are non-enforceable as they are chosen solely based on protection of human health and do not take into account whether analytical testing is available to detect the contaminant at the MCLG level or whether adequate technology exists to remediate or remove the contaminant at the MCLG level. Conversely, Maximum Contaminant Levels (MCLs), are derived from MCLGs but also take into account the availability of analytical testing, adequate technology for contaminant remediation, efficacy under field conditions, and cost. MCLGs include a margin of safety incorporated into the level via the use of uncertainty factors that ensures no adverse human health effects would result from lifetime exposure to the contaminant in drinking water at the MCLG level. MCLGs are derived separately for and non-cancer endpoints and cancer endpoints.

3.b. Non-Cancer Endpoints

The derivation of an MCLG is based on the assumption that for non-cancer endpoints, a dose threshold exists. Doses above that threshold potentially place a

person at risk for an adverse human health effect, whereas below that threshold the person is not at risk. To ensure that exposure at the MCLG and below does not place any person, including vulnerable populations, at risk, an adequate margin of safety is built into the derivation.

Available animal model studies are reviewed to determine the point of departure (POD), which is the first step in the MCLG derivation. The point of departure (POD) may be an administered dose, a modeled dose, or a serum level. If the POD is a serum level, a dose adjustment factor may be applied to derive a dose. In considering animal model studies as candidates for the POD, a number of factors should be considered, study duration (acute, subacute, chronic), route of exposure, intensity of exposure, study quality, relevance of the animal model adverse health effect to human health, and interspecies differences in absorption, distribution, metabolism and excretion of the substance. Animal model studies may be considered irrelevant for the derivation of an MCLG based on the above considerations and therefore not be used for the POD.

If an animal model study meets the criteria discussed above and is considered relevant to human health, then it serves as a candidate along with other such studies for the POD. Several PODs are available. The most commonly used POD is the no-observed-adverse-effect level (NOAEL), the highest dose administered in the animal model study that did not result in toxicity where toxicity is defined by alteration of biomarkers, change in body weight or body weight gain, lesions, or anatomical abnormalities at necropsy. In some circumstances, such as the absence of a NOAEL in an animal model study, the lowest-observed adverse-effect level (LOAEL) may be used as the POD. (USEPA 2002)

An alternative POD that may be used with robust datasets is the lower confidence limit of the benchmark dose (BMDL). Calculating the BMDL requires sufficient datapoints from the animal model study/studies that a dose-response curve can be modeled. The benchmark response (BMR) is the acceptable level of change in the animal model adverse health effect. A BMR of 10% is typically considered the acceptable level of change as it is at or near the limit of sensitivity of many bioassays. For continuous variables (e.g. body weight), a BMR of 10% corresponds to a 10% deviation in the outcome of interest, whereas for quantal data (e.g. organ toxicity) a BMR of 10% corresponds to a 10% increase in the incidence of the adverse effect. Statistical modelling of the dose response curve is used to calculate the dose that corresponds to the chosen BMR, known as the benchmark dose (BMD), and the lower 95% one-sided (or two-sided) confidence limit of the BMD is the BMDL. The DPAG, in discussion with the PA DEP, determined that the BMDL that corresponded to a BMD with a BMR of 10% (referred to as the BMDL₁₀) would be the default POD when the BMD method was employed. (USEPA 2012)

The EPA recommends a number of approaches to derive human equivalent oral exposures (HED) from a laboratory animal species derived POD. (USEPA 2002) The preferred approach is physiologically-based toxicokinetic modeling applying a dose adjustment factor. The DAF is multiplied by the animal exposure (in mg/kg/d) to achieve the human equivalent exposure (in mg/kg/d). In lieu of data to support either of these types of approaches, body weight scaling to the 3/4 power (i.e., BW^{3/4}) is endorsed as a general default procedure to extrapolate toxicologically equivalent doses of orally administered agents from all laboratory animals to humans for the purposes of deriving

an oral Reference Dose (RfD). Use of these methods is generally combined with a default interspecies uncertainty factor, UFA, reduced from 10 to $10^{0.5}$.

Once the HED is identified, the reference dose (RfD) is calculated by dividing the HED by uncertainty factors (UF) to create an adequate margin of safety. UFs have a value between 10^0 (i.e. 1), $10^{0.5}$ (i.e. 3), or 10^1 (i.e. 10). A default UFH of 10 is applied for the potential variability in sensitivity to the exposure in the human population. An UFA of 10 each is applied for the uncertainty of extrapolation from an animal model to humans unless some dose adjustment factor can be accurately applied. A default UFL of 10 is applied when the LOAEL is used rather than the NOAEL or BMD. A UFS is applied when extrapolating from sub-chronic animal model studies to chronic human exposure. An additional UFD, referred to as a modifying factor, may be applied to account for uncertainty about the quality of the study or data set. All the UFS are multiplied to develop a UFT, or total uncertainty factor. Figure 1 provides an illustration but does not represent an actual PFA or the order of endpoints.

Point of Departure Determination

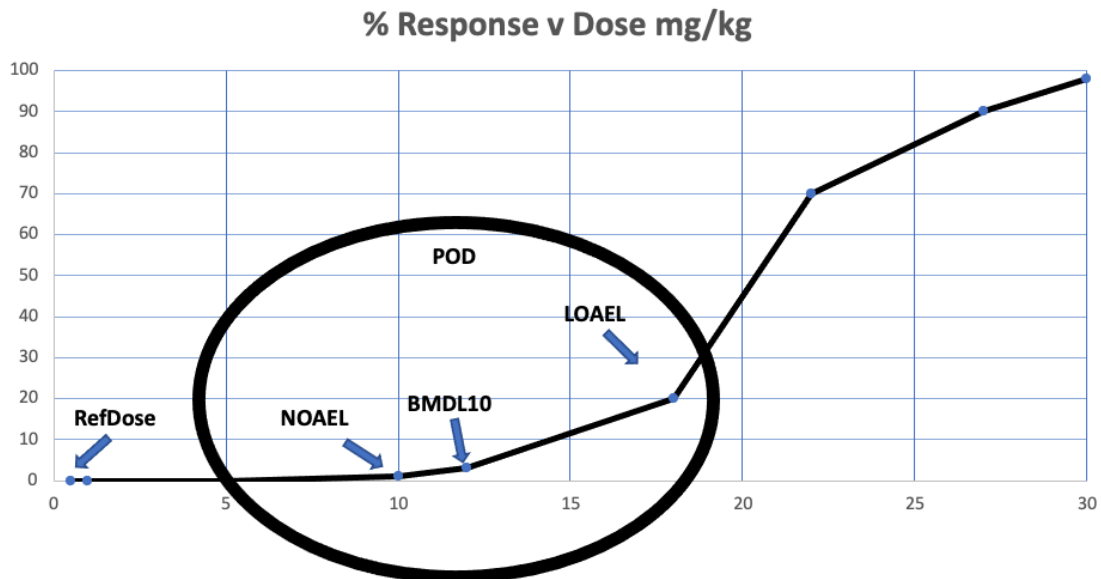


Figure 1: POD sought amongst various endpoints (LOAEL, NOAEL, BMDL₁₀) and then a Reference Dose derived.

The RfD is typically expressed in mg/kg/d and is the daily ingested dose of a substance that is considered to be without an increased risk of an adverse human health effect. The RfD can be converted into a Drinking Water Equivalent Level (DWEL), the concentration of the substance in water that would yield the RfD for the target population based on established drinking water rates. If the POD suggests that the target population is adults, then standard assumptions about weight (e.g. 70-kg adult) and consumption (2-L of water per day) are used. Different weight and consumption standards are applied if the POD suggest the target population is, for example, infants.

The MCLG is subsequently derived from the DWEL by accounting for the relative source contribution (RSC) of drinking water to total daily dose of the substance so that the total daily dose does not exceed the RfD. For substances where the relative source contribution is unknown, a default RSC of 0.2 is used. When the relative contribution of various sources to daily dose has been determined, the RSC of drinking water may be used instead of the default RSC but may be no greater than 0.8 to account for potential unknown exposure sources. (USEPA 2000)

3.c. Goeden Model discussion

An alternative method to convert RfD to MCLG is the transgenerational toxicokinetic model. This approach considers water consumption from conception to adulthood and adjusts for the fact that relative source contribution of water is higher early in life. It assumes that a child will have a certain level of exposure in-utero because of the PFA in the mother's body and further exposure during breastfeeding or bottle feeding. This model requires specific toxicokinetic information about the substance in question and cannot be applied to every substance. The model for this report was provided to the DPAG by Minnesota Department of Health (MDH) as an excel spreadsheet. Parameters for this model are listed in Appendix C. Although RfD was always calculated, the POD serum level was divided by UFT to determine a corresponding internal target human serum level (THSV). Working backward from the target human serum level, reduced by 50% to account for the RSC of an infant, an MCLG was derived from the model so that the highest serum level ever achieved from birth to adulthood never exceeded the reference dose. The model had sufficient data for application to MCLG recommendations for PFOA, PFOS, PFNA, and PFHxS. Table 2 lists some of the key

model parameters and the preferred tendency (central or upper) of the parameter. Please note: The THSV is useful for informing public health policy and interpreting population-based exposure potential. This value is based on population-based parameters and should not be used for clinical assessment or for interpreting serum levels in individuals.

Model Parameter	Tendency of Parameter	PFOA	PFOS	PFHxS	PFNA
Half-Life, days	Central	840 ^a	1241 ^b	1935	1417 ^c
Placental Transfer Ratio	Central	0.87 ^d	40 ^d	0.70 ^d	0.69 ^d
Breastmilk Transfer Ratio	Central	0.052 ^d	0.017 ^d	0.014 ^d	0.032 ^d
Volume of Distribution (V _d), L/kg	Central	0.170 ^e	0.230 ^e	0.25 ^f	0.200 ^{d,g}
Relative Source Contribution (RSC), %	Central	50	50	50	50
Duration of Exclusive Breastfeeding, months	Upper	12	12	12	12

a) Bartell 2010; b) Li 2018; c) Zhang 2013; d) MDH 2020, 2019; e) Thompson 2010; f) Sundstrom 2012; Ali 2019 g) ATSDR 2018

Table 2: Exposure Model Parameters used in transgenerational model (Goeden 2019) for derivation of proposed MCLG.

3.d. Cancer Endpoints

MCLGs for cancer endpoints are historically set at zero although there may be scenarios under which a non-zero MCLG is appropriate for a cancer endpoint. The rationale behind a zero MCLG for cancer endpoints is that historically extrapolation of cancer risk from high dose animal studies to low dose human exposures was performed using the linear no-threshold model. The absence of a threshold in this extrapolation

model results in some cancer risk being associated with any dose. Therefore, the only level goal that can be considered protective of human health is zero. (USEPA 2005)

Current carcinogen risk assessment allows for the consideration of threshold effects in extrapolation of cancer risk. A threshold effect may be present if cancer is only observed when an exposure meets a certain intensity or duration. However, the absence of cancer at low level exposures should not be assumed to constitute a threshold as low level exposures may be associated with cancer risk that is undetected due to studies that are underpowered to detect cancer at that exposure intensity. The mechanism by which the carcinogen increases cancer risk may inform whether a threshold effect is present. If the carcinogen induces cancer secondary to a toxic effect then the threshold is the dose at which the toxic effect occurs and doses below that threshold, after applying uncertainty factors, should be considered non-carcinogenic. MCLGs for carcinogens that act by a mutagenic mode of action are still set at zero as the linear-no threshold model is most appropriate for that mechanism.

Substances that are only carcinogenic above a certain exposure intensity or duration may have non-zero MCLGs utilizing the same derivation process as for non-cancer endpoints, discussed above. For such substances, the MCLG for the cancer endpoint and the MCLG for the non-cancer endpoint are both derived and the lower value of the two serves as the overall MCLG for the substance.

Numerous epidemiological studies of PFAS, especially PFOA and PFOS, have examined occupational and environmental exposures but have failed to detect consistent findings across studies. (Bonefeld-Jorgensen 2011, Chang ET 2014, Eriksen 2009, Hardell 2014, Innes 2014, Klaunig 2015, Yeung 2013). The International Agency

for Research on Cancer (IARC) has classified PFOA as “possibly carcinogenic to humans” (Group 2B), based on limited evidence in humans that it can cause testicular and kidney cancer, and limited evidence in lab animals. The EPA has not officially classified PFOA as to its carcinogenicity. EPA’s Scientific Advisory Board, based mainly from studies in lab animals, stated that PFOA shows “suggestive evidence of carcinogenicity, but not sufficient to assess human carcinogenic potential.”

PFOA and PFOS show positive associations with cancers of the prostate, kidney, testis, and thyroid but with a) only small elevations in relative risk intervals (0.5 and 2.0 (with 95% confidence intervals including 1.0), b) evidence of negative associations as well, and c) inconsistencies across the studies. Furthermore, exposure response relationships do not follow the monotonic pattern of increasing dose causing increasing response. The strongest example is that associations found at lower environmental community studies are not supported by those found in the workplace where exposures are higher by one or two orders of magnitude. Furthermore, although animal studies support target organ as the liver, testis (Leydig cells), and pancreas (acinar cells), these are not the types of cancers identified by human studies. Some drinking water recommendations rely on an effect produced by expression of peroxisome proliferator-activated receptor-alpha (PPARalpha) which is specific to rodents. For example, CEPA (2019) and NJDEP (2017, 2018) have cancer minimal risk levels for PFOA and PFOS derived heavily from animal studies. After careful review, the DPAG concluded that cancer endpoints for PFAS that rely heavily on animal studies are not supported by the totality of human and animal evidence. Furthermore, there is insufficient evidence to argue that Non-Cancer MCLGs would not be protective of cancer risk.

4. PFOA

After a literature search and a review of the available evidence and recommendations from various agencies, the DPAG developed an MCLG recommendation for PFOA based on Non-Cancer endpoints. The agencies with the most relevant inputs were the US EPA, the ATSDR (ATSDR 2018), the MDH (MDH 2020 PFOA), NJDEP (NJDEP 2017), and MDHHS (MDHHS 2019). The US EPA selected Lau (2006) because it met their criteria for chronic exposure, multiple dose groups, use of a concurrent control, and with serum data amenable for modeling. (US EPA 2016) MDH used Lau (2006) as well and used the serum level estimated by US EPA. The ATSDR selected identical LOAELs from Onishchenko (2011) and Koskela (2016). Both studies had the same populations of laboratory animals and evaluated a single dosing group. These studies identified developmental effects (neurobehavioral and skeletal) as critical. The DPAG selected Koskela (2016) and Onishchenko (2011) as the critical studies. (ATSDR 2018, Appendix A, Table A8)

The serum concentration at the LOAEL of 0.3 mg/kg/d from Onishchenko (2011) and Koskela (2016) was below the modeled serum concentrations from two immunotoxicity studies evaluated by ATSDR (a sensitive effect seen in other PFAS). (Lau 2006) MDHHS also selected the critical studies by ATSDR as also being protective for immunotoxicity. (MDHHS 2019) The DPAG rejected the BMDL from Loveless (2006) used by NJDEP. Loveless (2006) was a 14-day exposure study in rats and mice, with liver weight changes being the critical effect identified. NJDEP (2017) Liver weight changes, in and of themselves, translate questionably as an adverse effect in humans

and the POD identified was higher than those when considering immunotoxicity. From Onishchenko and Koskela, the ATSDR estimated the POD average serum concentration in the mice (8.29 mg/L) using a three-compartment pharmacokinetic model (Wambaugh 2013) using animal species-, strain-, sex-specific parameters. This was adopted by the DPAG as the POD for PFOA.

4.a. Review of Critical Studies

Koskela (2016) investigated the administration of PFOA at a dose of 0.3 mg/kg/d administered orally mixed with food to pregnant C57BL/6/Bkl mice starting on GD1 to investigate developmental outcomes on long bone morphology and bone cell differentiation. Female offspring were sacrificed at the age of 13 or 17 months for examination.

Body weights of PFOA exposed offspring were higher than controls throughout the lifetime of the animals, reaching statistical significance at 13 and 17 months. Significant increases in the femur and tibial periosteal area and medullary area were seen at 17 months but not at 13 months in PFOA exposed offspring. Tibial mineral density was decreased in PFOA exposed offspring at both 13 and 17 months. Femur and tibial cortical area, trabecular parameters, and femur mineral density were unaffected by PFOA exposure. There was no significant effect of PFOA exposure on biomechanical properties of the femur or tibia. Concentration of PFOA in pooled tibias and femurs was significantly greater in exposed offspring at both 13 and 17 months.

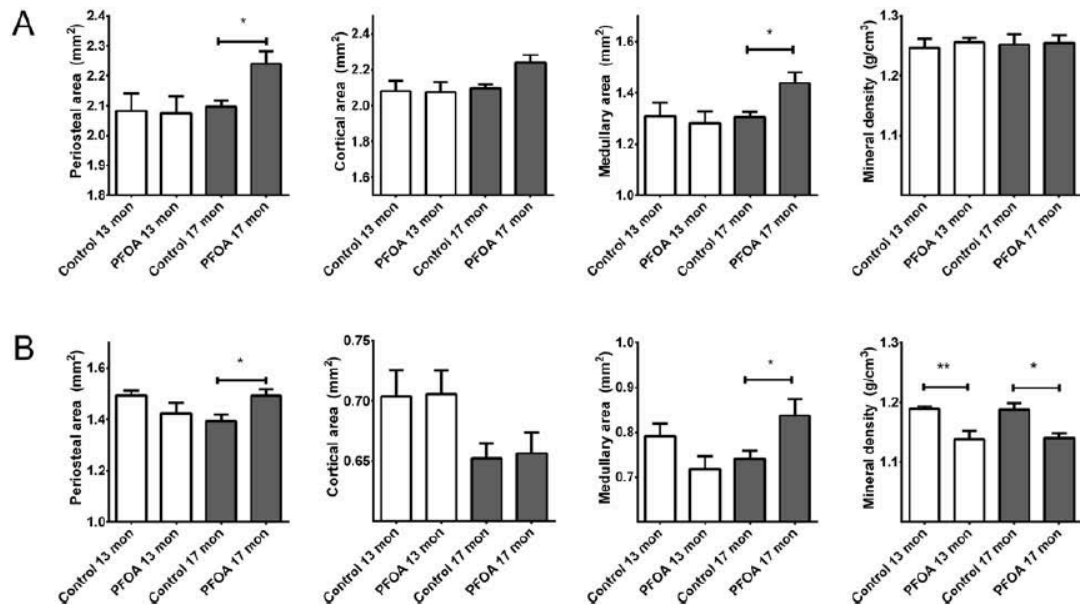


Fig. 2. Effects of PFOA on morphometrical parameters of femurs (A) and tibias (B) as analyzed by microCT. The cortical VOI reference point was set to the point where complete fusion of the growth plate was observed, offset being 250 and height 500 cross-sections proximally. Group mean \pm SE, $n = 5$. * $p < 0.05$, ** $p < 0.01$.

Figure 2: Effects of PFOA reproduced from (Koskela 2016). This represents the selected PFOA critical effect of morphometric parameters of femurs and tibias at 13 and 17 months - dosing is 0.3 mg/kg/d (LOAEL). The average serum concentration was estimated in the mice (8.29 mg/L) using a three-compartment pharmacokinetic model (Wambaugh 2013) using animal species, strain, sex-specific parameters. (ATSDR 2018)

In an *in vitro* study, the effect of PFOA on the viability of MC3T3 osteoblast precursor cells were assessed using an MTT-test on days 1, 7, and 10. A significant decrease in cell viability was seen on days 7 and 10 at a PFOA concentration of 100 mcM and above but not at a concentration of 10 mcM. A significant decrease in the alkaline phosphatase activity of osteoblasts was seen at day 7 at a PFOA concentration of 100 mcM and above but not at a concentration of 10 mcM. An increase in calcium and in OCN mRNA was seen at PFOA concentrations of 1 and 10 mcM but not at higher concentrations.

In a second in vitro study investigating the effect of PFOA on osteoclasts, the number of TRACP+ cells containing three or more nuclei was increased at PFOA concentration of 10 mcM and above with evidence for a dose response relationship. Osteoclasts were not significantly affected at 1 mcM. Resorption pit area was significantly increased at a PFOA concentration of 1 mcM, but with no evidence of a dose response relationship and a decrease in pit area with increasing PFOA concentration.

Onishchenko (2011) investigated the administration of PFOA or PFOS at a dose of 0.3 mg/kg/d administered orally via food to pregnant C57BL/6/Bkl mice starting on GD1 to investigate Motor function, circadian activity, and emotion-related behavior in exposed offspring. One pup per litter was sacrificed at birth for brain and liver tissue samples of PFOS and PFOA levels. Offspring were weaned on postnatal day 21 and injected subcutaneously with microtransponders. Test for locomotor and circadian activity were performed at age of 5 to 8 weeks. Animals were tested for emotion-related behavior in elevated plus maze and forced swim test. Test for motor strength and motor coordination were performed in animals at 3 to 4 months old.

Administration of PFOS or PFOA did not affect dam weight gain, litter size, or sex ratio. There were no differences in offspring body or brain weight between groups at birth. Absolute liver weight was increased in PFOA-exposed offspring as compared to controls, but not in PFOS-exposed offspring. Among exposed pups, PFOS concentrations at birth or greater than PFOA concentrations in the brain, but lower in the liver.

PFOS-exposed males walked significantly less than male controls when exploring a new environment, while PFOS-exposed females do not differ from controls. PFOA exposure did not have a significant effect on locomotor activity in either sex.

Circadian activity was measured using the TrafficCage system. During adaptation to the new cage, PFOS-exposed males displayed decreased activity during the first two hours of the test, while PFOS-exposed females displayed decreased activity during the first hour only. PFOA-exposed males were more active during the first hour of the test, while PFOA-exposed females demonstrated decreased activity as compared to controls. After habituation to the cage, PFOS exposure After habituation to the cage, PFOS exposure did not significantly affect activity counts over light or dark periods, either in males or females. PFOA exposed males demonstrated greater activity as compared to controls, especially during the dark phase, while PFOA exposure in females had no effect on activity level. PFOS exposure was associated with a greater number of inactive periods during both light and dark phase in both males and females, although only the difference in females reached statistical significance. PFOA demonstrated an opposite effect, decreasing the number of inactive periods in both light and dark phase which met significance in both phases for males but only in the light phase for females. (see Figure 3)

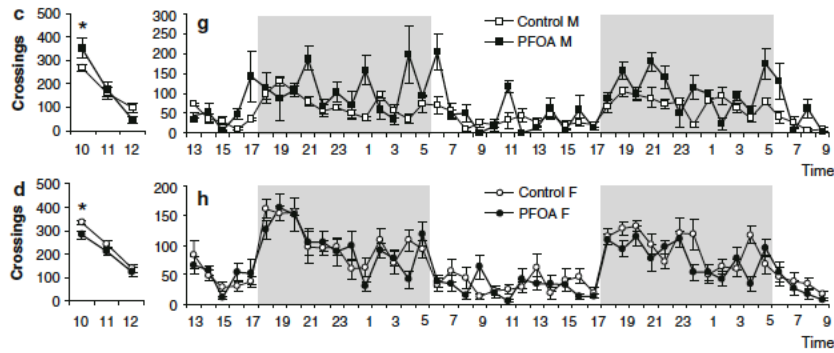


Fig. 2 Novelty-induced (a-d) and circadian activity (e-h) over 48 h in the home cage and social group in male and female mice prenatally exposed to PFOS or PFOA. Activity counts presented as number of antenna crossings in the TrafficCage (see “Materials and Methods” section for details). Gray areas correspond to a dark phase of the light-dark cycle. * $P < 0.05$, $n = 6-10$

Figure 3: Figure reproduced from Onishchenko (2011). This was selected as a PFOA critical effect for change in inactive periods seen at 0.3 mg/kg/d (LOAEL). (Onishchenko 2011) The average serum concentration was estimated in the mice (8.29 mg/L) using a three-compartment pharmacokinetic model (Wambaugh 2013) using animal species, strain, sex-specific parameters. (ATSDR 2018). Note: because the POD dose and pharmacokinetic model are the same as Koskela (2016), the derived POD serum concentrations are the same.

Evaluation for anxiety-related behavior in the elevated plus maze demonstrated that PFOS-exposed male mice walked less total distance than did controls, which was consistent with previous findings of decreased locomotor activity in this group, but which based on time spent in open and closed arms did not seem to reflect changes in anxiety-related behavior. No significant differences in anxiety-related behavior were noted in PFOS-exposed females or in PFO- exposed males or females.

No effect of PFOA or PFOS was demonstrated in either sex in depression-like behavior in the forced swimming test.

Muscle strength in the hanging wire test was less in PFOS-exposed males who had significantly shorter fall latency than controls. No effect was seen in PFOS-exposed female mice or in PFOA exposure in either sex.

Inconsistent findings were demonstrated between PFOS and PFOA exposure and motor coordination in the accelerating rotarod test. PFOA-exposed females had shorter

fall latency in every trial, but it only met statistical significance in 1 of 4 trials, while PFOA exposed males had similar fall latencies as compared to controls. PFOS-exposed females had shorter fall latency in 2 of 4 trials while PFOS-exposed males had shorter fall latency that was significant in only one of four trials.

4.b. Development of MCLG

Following the approach used by MDHHS and MDH to identify a species-specific DAF, DPAG selected the PFOA serum half-life of 840 days (2.3 years). (Bartell 2010) This was considered more relevant for exposure to the general population than occupational exposure studies used by ATSDR. (ATSDR 2018, Bartell 2010). studied 200 individuals (100 men, 100 women) exposed by drinking PFOA-contaminated water. DPAG used the volume of distribution ($V_d = 0.17 \text{ L/kg}$) selected by MDHHS and MDH that was based on human data. (Thompson 2010). These were the references used by EPA in 2016 when they derived a PFOA clearance of $1.4 \times 10^{-4} \text{ l/k/d}$ and developed their health advisory level.

DPAG accepted the UFs selected by ATSDR for a UFT of 300. (ATSDR 2018) This resulted in a THSV of 0.028 mg/L for the Goeden Model. Setting the target for the breast fed infant as 0.014 (50%RSC), the MCLG for drinking water is recommended to be 8 ng/L (8PPT) to protect breastfed infants and throughout life. (Figure 4, Table 3)

PFOA

Dose Response Modeling Method	LOAEL
POD	The average serum concentration was estimated in the mice (8.29 mg/L) using a three-compartment pharmacokinetic model (Wambaugh 2013) using animal species, strain, sex-specific parameters. (ATSDR 2018)
HED = POD x DAF (mg/kg/d)	$DAF = Ke \times Vd$ $Ke = 0.000825175 (8.2 \times 10^{-4})$ based on a human serum half-life of 840 days (Bartell 2010) $Vd = 0.17 \text{ L/kg}$ (Thompson 2010) $HED_{LOAEL} = POD_{LOAEL} \times DAF$ $HED_{LOAEL} = POD_{LOAEL} \times Ke \times Vd$ $HED_{LOAEL} = 8.29 \text{ mg/L} \times 0.000825175 \times 0.17 \text{ L/kg}$ $HED_{LOAEL} = 0.001163 \text{ mg/kg/d}$ or $1.163 \times 10^{-3} \text{ mg/kg/d}$
Uncertainty Extrapolation	
Human Variability (UFH)	10 (standard)
Animal to Human (UFA)	3 (DAF applied)
Subchronic to Chronic (UFS)	1 (Chronic effect studied)
LOAEL to NOAEL (UFL)	10 (standard)
Database (UFD)	1
Total Composite (UFT)	300
RfD = HED/UFT (mg/kg/d)	$RfD = 0.001163 \text{ mg/kg/d} / 300$ $RfD = 3.9 \text{ ng/kg/day}$ ($3.9 \times 10^{-6} \text{ mg/kg/d}$)
THSV = POD / UFT	$THSV = 8.29 \text{ mg/L} / 300$ $THSV = 0.028 \text{ mg/L}$
Receptor	Infant exposure via breastmilk for 1 year, from mother chronically exposed via water, followed by lifetime of exposure via drinking water. Protective for short-term, subchronic and chronic. (also protective of formula fed infant). Goeden Model Parameters: Placental transfer of 87% and breastmilk transfer of 5.2% (MDH (2020 PFOA)). The Human Serum half-life is set at 840 days (Bartell 2010).

	<p>The Volume of distribution of 0.17 L/kg (Thompson 2010) Other factors include, 95th percentile drinking water intake, consumers only, from birth to more than 21 years old. Upper percentile (mean plus two standard deviations) breast milk intake rate. Time-weighted average water ingestion rate from birth to 30-35 years of age is used to calculate maternal serum concentration at delivery. (Goeden 2019) A Relative Source Contribution of 50% (0.5) is applied and based on studies which showed that infants RSC is similar to NHANES 95th percentiles for 3-11 (2013-2014) and over 12 years old (2015-2016) participants. (CDC 2019)</p>
Chronic Non-Cancer MCLG	<p>The model produces a Chronic Non-Cancer MCLG of 8 ng/L (ppt). This protects health during the growth and development of a breast fed infant. (Figure 4)</p>

Table 3: Development of Non-Cancer MCLG for PFOA

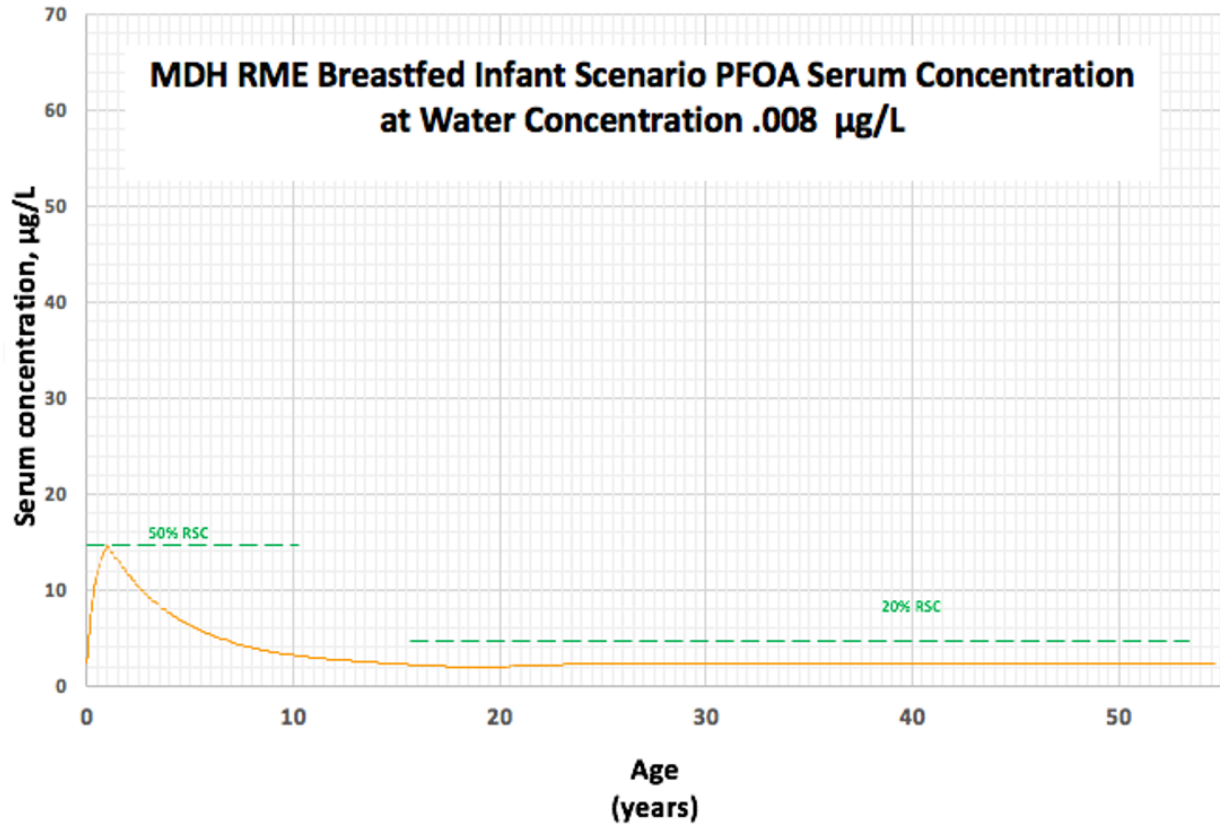


Figure 4. Using the Goeden Model, the POD and its parameters for PFOA were converted to an THSV of 0.028 mg/L. An RSC set at 50% means that half of this (0.014 mg/L) will be from ingested drinking water. The MCLG of PFOA in drinking water should then be set at 0.008 ug/L or 8 PPT to protect from adverse health events.

5. PFOS

After a literature search and a review of the available evidence and recommendations from various agencies, the DPAG developed an MCLG recommendation for PFOS based on Non-Cancer endpoints. DPAG reviewed a number of candidate MRL levels developed by US EPA and ATSDR. (ATSDR 2018, Dong I 2011, Pachkowski 2019, Peden-Adams 2008, Vassiliadou 2010, Butenhoff 2009) Although immune function has not been examined following chronic-duration oral exposure in laboratory animal studies, the lowest LOAEL doses were for immunological effects in intermediate-duration animal studies. These were seen at doses lower than hepatotoxicity or developmental effects. ATSDR did not select an immunotoxicity study as a critical study but did develop a “candidate MRL” using the immunotoxicity study by Dong (2011). The NOAEL endpoint was suppression of natural killer cell activity and anti-Sheep Red Blood Cell Antibody response in mice. Laboratory animal studies, particularly studies in mice, provide supporting evidence of the immunotoxicity of PFOS. Human epidemiological studies are consistent with this evidence as well. After the calculation of HEDs and application of UFs to all of these studies, the resultant MRLs were nearly identical to those using other studies by agencies such as MDHHS. Thus, DPAG concluded the study by Dong I (2011) and the POD of 2.36 mg/L were appropriate. This study was selected over the other immunotoxicity studies because it identified the highest NOAEL for immunotoxicity and the longest exposure duration.

5.a. Review of Critical Study

Dong I (2011) administered PFOS to adult male C57DL6 mice to investigate immunotoxicity outcomes. PFOS with 2% Tween 80 was administered by oral gavage daily for 60 days to a targeted total administered dose over that period of 0, 0.5, 1, 5, 25, and 50 mg/kg body weight with controls being administered deionized water with solubilizer only. 12 mice were included in each group. Mice were immunized on the 54th day of PFOS dosing by intravenous injection of sheep red blood cells (SRBC). Six of the 12 mice from each treatment group were sacrificed seven days later and blood was obtained by cardiac puncture. The remaining six mice were administered a booster immunization of SRBC to the right rear foot pad on the final day of PFOS dosing to investigate delayed type hypersensitivity response (DTH) and other immunoglobulin assays.

Mice exposed at the highest dose of 50 mg/kg had significantly lower body weight as compared to controls; however, body weight change was insignificant at other dose levels. Similarly, food intake on the final day of dosing was significantly less at the highest 50 mg/kg dosing group as compared to controls but there was no significant difference at other dose levels. Relative spleen and thymus weights were decreased at the highest 50 mg/kg dose, but not significantly different than other dose levels. Relative liver weight was increased at both the 25 mg/kg dose and 50 mg/kg dose as compared to controls.

Serum PFOS concentration increased in a dose response fashion with increasing absolute dose administered. There was no significant effect of treatment dose on serum corticosterone level.

IFN γ level was significantly decreased at the 50 mg/kg dose, without significant changes at other dose levels. IL-4 levels were significantly increased at the 5 mg/kg dose and above. For both IFN γ and IL-4, changes in levels were largely dose-dependent except at the lowest 0.5 mg/kg dose. The number of cells secreting IL-2 and IL-10 were decreased and increased, respectively, in the 50 mg/kg dose group, but no significant differences were seen at lower dose regimens. As with other cytokines, changes in levels were largely dose dependent at the higher dose regimens only.

With respect to immunoglobulin synthesis, IgM levels declined with a dose-response relationship at the 5 mg/kg dose and above. IgG, IgG1, and IgE production were all increased only at the 50 mg/kg dose with other lower dose regimens not affecting serum levels. IgG2a levels and delayed-type hypersensitivity response were unaffected by PFOS administration.

5.b. Development of MCLG

Dong (2011) identified immune suppression, specifically increased IL-4 and decreased Sheep RBC specific IgM levels in the mouse model. Doses administered over 60 days were converted to mg/kg/d by dividing by 60 days. Thus, doses were 0, 0.00833, 0.0167, 0.0833, 0.4167, and 0.8333 mg/kg/d. The NOAEL of 0.0167 mg/kg/day (total dose over 60 days of 1 mg/kg) was selected because it was the highest dose without a statistically significant effect. (Figure 5 is reproduced from Dong (2011; Figure 1)

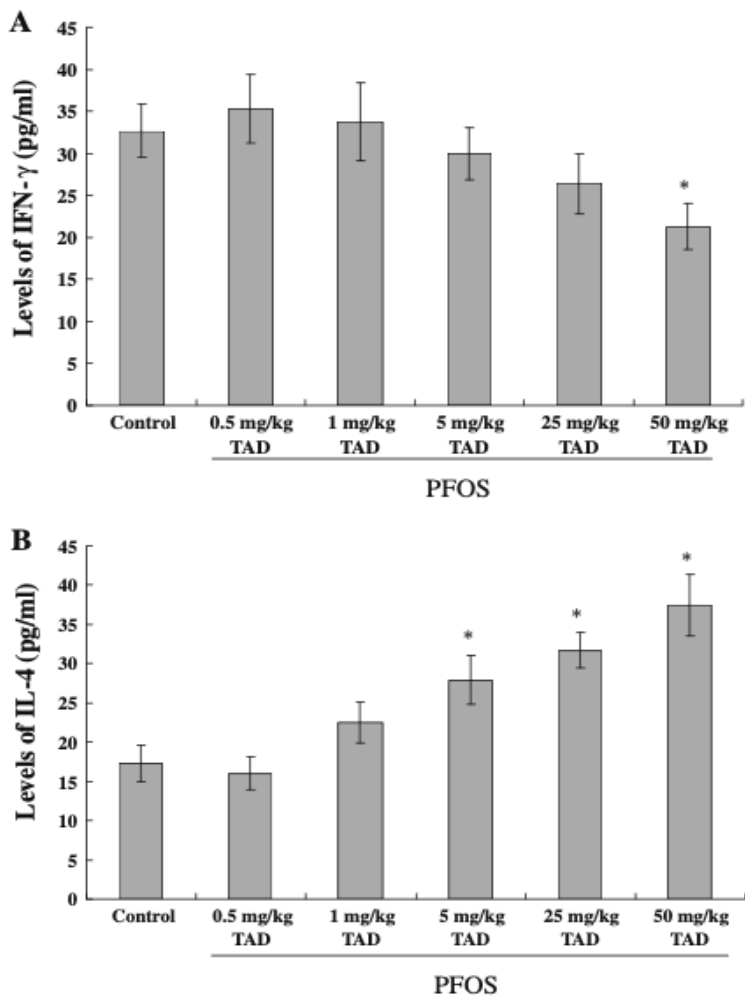


Fig. 1 IFN- γ and IL-4 levels in the splenocyte culture supernatant of splenocytes harvested from mice 24 h after the last of their 60 days of treatment, i.e., daily oral exposures to PFOS. Data are presented as mean (\pm SE) of results obtained using ELISA kits. *Significantly different from respective control ($P \leq 0.05$). The data were log transformed as required for statistical analysis. TAD Total Administered Dose over the course of 60 days. $n = 6$ in each group

Figure 5: NOAEL critical effect of increased IL-4 levels determined by Dong 2011. The dose administered is over 60 days and is thus converted to the daily dose of 0.0167 mg/kg/day (total dose of 1 mg/kg over 60 days).

Dong provided the serum PFOS level at each dose and thus the 1 mg/kg dose results in a serum PFOS level of 2.36 mg/L (\pm 0.47). This is found in Figure 6.

Table 1 PFOS concentrations in serum (mg/L), body weight, and organ indices in adult male C57BL/6 mice treated with PFOS orally for 60 days

PFOS (mg/kg TAD)	<i>n</i>	Serum PFOS (mg/L)	Serum corticosterone (ng/L)	Body weight change ^a	Food intake from day 60 to day 61 (g)	Spleen index ^b	Thymus index ^b	Liver index ^b	Kidney index ^b
Control	6	0.05 ± 0.01	443.28 ± 31.69	4.57 ± 0.42	5.06 ± 0.35	0.45 ± 0.03	0.30 ± 0.03	5.23 ± 0.16	1.48 ± 0.05
0.5	6	1.07 ± 0.11	434.62 ± 28.93	4.81 ± 0.36	5.42 ± 0.42	0.47 ± 0.02	0.27 ± 0.02	5.16 ± 0.14	1.53 ± 0.05
1	6	2.36 ± 0.47	387.14 ± 35.08	5.14 ± 0.45	5.11 ± 0.28	0.46 ± 0.02	0.33 ± 0.02	5.29 ± 0.21	1.57 ± 0.06
5	6	10.75 ± 0.82*	369.87 ± 27.51	4.83 ± 0.34	4.87 ± 0.33	0.46 ± 0.03	0.31 ± 0.02	5.75 ± 0.17	1.54 ± 0.03
25	6	22.64 ± 2.29*	453.76 ± 42.12	3.92 ± 0.47	4.42 ± 0.27	0.42 ± 0.03	0.24 ± 0.02	6.33 ± 0.16*	1.44 ± 0.06
50	6	51.71 ± 3.81*	528.39 ± 33.94	2.16 ± 0.29*	3.39 ± 0.35*	0.35 ± 0.02*	0.21 ± 0.01*	8.04 ± 0.20*	1.41 ± 0.04

PFOS concentrations, body weight (change), and organ weight data did not require transformation for statistical analysis

TAD Total Administered Dose over the course of 60 days

* Indicates that value is significantly different from respective control ($P \leq 0.05$). Data are reported as mean ± SE

^a Body weight (BW) change denotes change in weight from regimen start to finish: [Postexposure BW (g) – Pre-exposure BW (g)]

^b Calculated as: [organ weight (g)/body weight (g)] × 100

Figure 6: Serum PFOS level reported by Dong (2011) Table 1.

DPAG followed the approach adopted by MDH and MDHHS and applied the PFOS specific clearance rate of 1241 days (Li 2018) and the EPA reported Vd of 0.23 L/kg to develop the DAF. DPAG agreed with MDHHS application of a UFT of 100. This produced a THSV of 0.024 mg/mL. Setting the target to protect the breast fed infant as 0.012 mg/mL (50%RSC), the MCLG for drinking water is recommended to be 8 ng/L (8PPT) to protect breast fed infants and throughout life. (Figure 7, Table 4)

PFOS	
Dose Response Modeling Method	NOAEL
POD	2.36 µg/mL(or 2.36 mg/L)
HED = POD x DAF (mg/kg/d)	Toxicokinetic Adjustment based on Chemical- Specific Clearance Rate (Li 2018, MDH 2020 PFOS) DAF = Vd (L/kg) x (Ln2/Half-life, days) DAF = 0.23 L/kg x (0.693/1241 days) = DAF = 0.00013 L/kg/d HED = POD x DAF (mg/kg/d) HED = 2.36 mg/L x 0.00013 L/kg/d HED = 0.000307 mg/kg/d

Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3 (DAF applied)
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	3
Total Composite (UFT)	100
RfD = HED/UFT (mg/kg/d)	RfD = HED/UFT (mg/kg/d) RfD = 0.000307 mg/kg-d/100 RfD = 3.1 ng/kg/d or 3.1×10^{-6} mg/kg-d
THSV = POD/UFT	TSHV = 2.36 mg/L/100 TSHV = 0.024 mg/mL
Receptor	Infant exposure via breastmilk for 1 year, from mother chronically exposed via water, followed by lifetime of exposure via drinking water. Protective for short-term, subchronic and chronic. The 95th percentile water intake rates (Table 3-1 and 3-3, USEPA 2019) or upper percentile breastmilk intake rates (Table 15-1, USEPA 2019) were used. Breast-fed infant, which is also protective of a formula-fed infant using Minnesota Department of Health Model based on Goeden (2019). Placental transfer of 40% (MDH 2020 PFOS). Breastmilk transfer of 1.7% (MDH 2020 PFOS). Human Serum half-life of 1241 days (Li 2018) Volume of distribution of 0.23 L/kg (USA EPA 2016c) 95th percentile drinking water intake, consumers only, from birth to more than 21 years old (Goeden [2019]) Upper percentile (mean plus two

	standard deviations) breast milk intake rate (Goeden 2019) Time-weighted average water ingestion rate from birth to 30-35 years of age (to calculate maternal serum concentration at delivery) (Goeden 2019)
Chronic Non-Cancer MCLG	The model produces a Chronic Non-Cancer MCLG of 14 ng/L (ppt). This protects health during the growth and development of a breast fed infant. Figure 7

Table 4: Development of Non-Cancer MCLG for PFOS

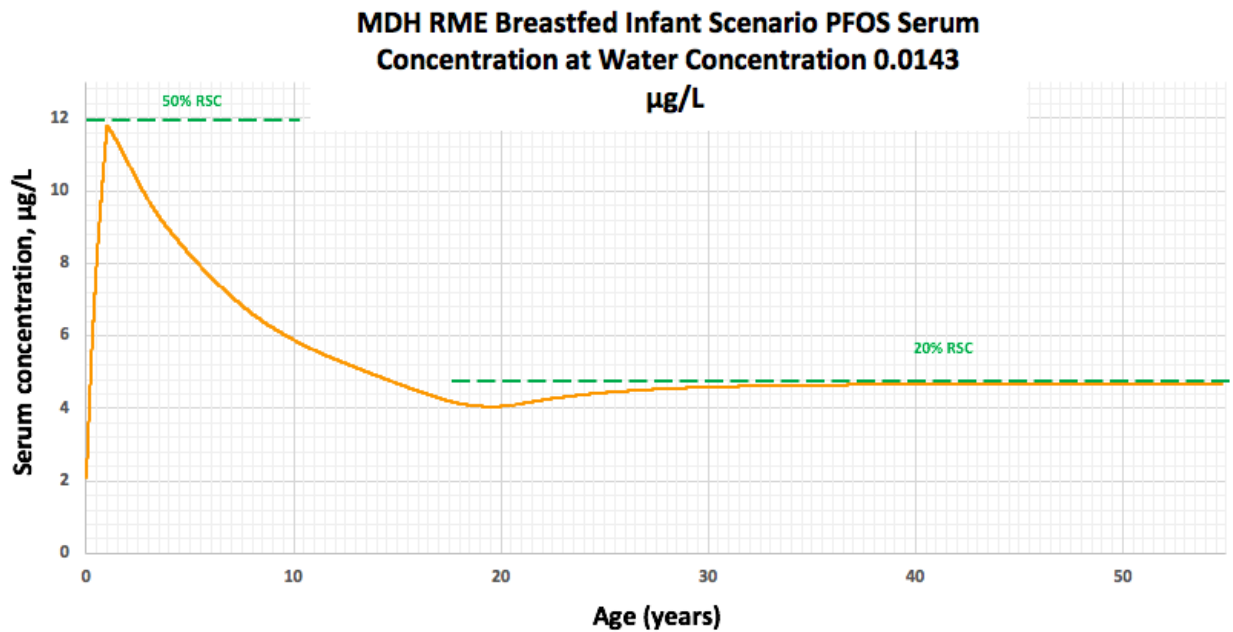


Figure 7. Using the Goeden Model, the reference dose and its parameters for PFOS were converted to an THSV of 0.024 mg/L. An RSC set at 50% means that half of this (0.012 mg/L) will be from ingested drinking water. The MCLG of PFOS in drinking water should then be set at 0.014 $\mu\text{g/L}$ or 14 PPT to protect the breast fed infant from adverse health events.

6. PFNA

After a literature search and a review of the available evidence and recommendations from various agencies, the DPAG developed an MCLG recommendation for PFNA based on Non-Cancer endpoints. The critical study identified was Das (2015). ATSDR released a provisional minimal risk level for intermediate exposure based on an analysis of Das (Das 2015, Rogers 2014, Wolf 2010). The HED of the NOAEL of 1 mg/kg/d identified in the Das (2015) developmental toxicity study was selected as the POD for the ATSDR MRL. At this dose, there was no statistical difference from controls for developmental landmarks of eye opening, preputial separation in males, and vaginal opening in females. A TWA serum PFNA concentration was estimated for dams using the serum concentration in the control group (0.015 µg/mL) as the baseline concentrations and the terminal concentration for the 1 mg/kg/d group (13.67 µg/mL) resulting in an estimated TWA serum concentration of 6.8 µg/mL. Das (2015) provided the serum concentrations directly to the ATSDR. NJDEP (2015) used the same study and the same dose of 1 mg/kg/d, but as a LOAEL for increased liver weight in pregnant mice. DPAG studied the controversy surrounding liver weight and similar effects produced by expression of peroxisome proliferator-activated receptor-alpha (PPARalpha) which is specific to rodents. DPAG agreed with ATSDR's selected POD and further agreed with Michigan's application of the Goeden transgenerational toxicokinetic model to this POD. Interestingly, the resulting MCLG is lower than the MCL determined by NJDEP (2015).

6.a. Summary of Critical Study

This study administered PFNA to pregnant CD-1 mice by oral gavage daily on gestational day 1 - 17 to assess for developmental toxicity outcomes. Treatment groups included 1 mg/kg/d, 3 mg/kg/d, 5 mg/kg/d, and 10 mg/kg/d while controls received deionized water. Mice were allocated to two groups: one group was sacrificed on GD 17 for analysis of gravity uterus, live fetuses, and maternal and fetal liver analysis. The second group was allowed to give birth and pregnancy outcomes and postnatal survival, growth, and development of the pups were monitored.

Mice in the highest 10 mg/kg/d dose group demonstrated overt toxicity beginning on GD 8. Therefore, the highest dose utilized for the remainder of the study was 5 mg/kg/d. The 3 mg/kg/d and 5 mg/kg/d groups demonstrated increased maternal weight gain as compared to controls for GD 11 to GD 17 which of the authors opined was likely due to dose-related enlargement of maternal liver. Increases in absolute and relative liver weight were seen at necropsy on GD 17 at the 1 mg/kg/d, 3 mg/kg/d, and 5 mg/kg/d doses. These changes demonstrated a dose response relationship in pregnant mice but not in non-pregnant mice. The authors noted that liver enlargement is common to PFAA exposure and it's probably mediated by activation of the PPARalpha signaling pathway.

With respect to pregnancy outcomes, there was no effect of treatment group on number of implants, number of live fetuses, or fetal weights. Absolute and relative liver weight was increased in PFNA exposed fetuses as compared to controls; however, there was no dose-response relationship. There was no effect of treatment group on

skeletal or visceral examination of fetuses. Full litter resorption occurred at the 10 mg/kg dose; however, this was associated with overt maternal toxicity, as noted above.

Postnatal survival of pups was decreased at the 5 mg/kg/d dose with deaths starting on PND 2 and only 20% of pups surviving to weaning. Treatment at the two lower dose levels did not affect pup survival. Exposure at the 3 mg/kg/d and 5 mg/kg/d was associated with decreased weight gain in pups with a dose response relationship. Decreased body weight was more persistent in male pups without any evidence of catch up growth in the post weaning period, whereas females typically recovered to control levels by 7 weeks of age. Relative liver weight was increased in pups at all treatment levels as compared to controls. This effect became less strong in the post weaning period and at PND 70 no significant effects remained. There were dose-dependent delays in postnatal development in the 3 mg/kg/d and 5 mg/kg/d groups with respect to eye opening, preputial separation, and vaginal opening.

Analysis of liver mRNA transcripts demonstrated PPARalpha-dependent gene expression in both fetal and neonatal mouse liver with activation of other transcripts regulated by other pathways. PPARalpha activation persisted to young adulthood and then declined, which the authors attributed to body burden of PFNA.

6.b. Development of MCLG

The HED of the NOAEL of 1 mg/kg/d identified in the Das (2015) developmental toxicity study was selected as the POD for the MRL. At this dose, there was no statistical difference from controls for developmental landmarks of eye opening, preputial separation in males, and vaginal opening in females. (Figure 8)

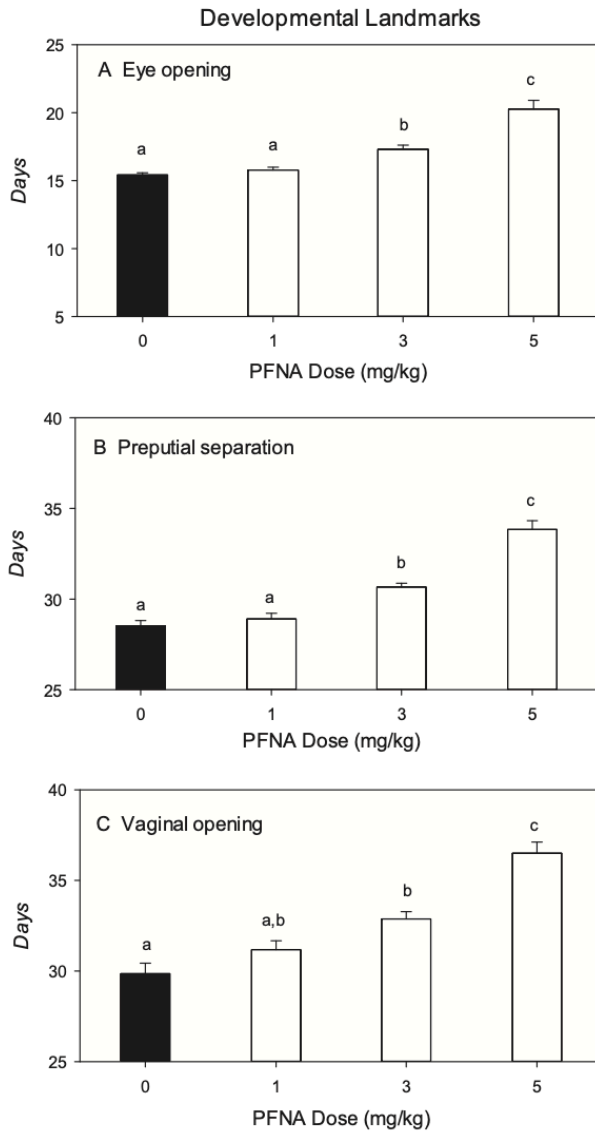


Fig. 9. Developmental landmarks of mouse offspring exposed to PFNA. Panel (A) illustrates eye opening, panel (B) preputial separation in males and panel C vaginal opening in females. ANOVA indicated a significant treatment effect. Points represent means \pm S.E. of 6–13 litters. Different letters denote significant differences ($p < 0.05$) among exposure groups determined by Tukey–Kramer test.

Figure 8: PFNA NOAEL of 1 mg/kg identified by Das (2015)

A TWA serum PFNA concentration was estimated for dams using the serum concentration in the control group (0.015 $\mu\text{g/mL}$) as the baseline concentrations and the terminal concentration for the 1 mg/kg/d group (13.67 $\mu\text{g/mL}$) resulting in an estimated

TWA serum concentration of 6.8 µg/mL. Das provided the serum concentrations directly to ATSDR. (ATSDR 2018) DPAG agreed with ATSDR's selected POD and UFTs and further agreed with MDH DAF calculations and the use of Goeden transgenerational toxicokinetic model to this POD. Setting the target to protect the breast fed infant as 0.0115 mg/mL (50%RSC), the MCLG for drinking water is recommended to be 6 ng/L (6 PPT) to protect breast fed infants and throughout life. (Figure 8, Table 5)

PFNA

Dose Response Modeling Method	NOAEL
POD	A NOAEL of 1 mg/kg/d was identified for developmental effects. Das (2015) The average serum concentration for NOAEL (1 mg/kg/d) was estimated (6.8 mg/L) in dams using an empirical clearance model (Wambaugh 2013).
$HED_{NOAEL} = POD \times DAF$ (mg/kg/d)	$DAF = Ke \times Vd$ $Ke = 0.000489165$ (4.8×10^{-4}) based on a human serum half-life of 1417 days. The human serum half-lives were an arithmetic mean of 2.5 years (913 days) for 50 year old or younger females and 4.3 years (1570 days) for females older than 50 years old and all males. An average of 3.9 years (1417 days) was calculated based on those averages. (calculated from Zhang 2013) $Vd = 0.2$ L/kg (ATSDR 2018; Ohmori 2003) $HED_{NOAEL} = POD \times DAF$ (mg/kg/d) $HED_{NOAEL} = POD \times Ke \times Vd$ $HED_{NOAEL} = 6.8$ mg/L $\times 0.000489165 \times 0.2$ L/kg $HED_{NOAEL} = 0.000665$ mg/kg/d
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	10
Total Composite (UFT)	300 (as per ATSDR 2018)
$RfD = HED/UFT$ (mg/kg/d)	$RfD = HED/UFT$ (mg/kg/d) $RfD = 0.000665$ mg/kg/d / 300 $RfD = 2.2$ ng/kg/day (2.2×10^{-6} mg/kg/d)
$THSV = POD/UFT$	$THSV = POD/UFT$ $THSV = 6.8$ mg/L / 300

	THSV = 0.023 mg/L
Receptor	Breast-fed infant, which is also protective of a formula-fed infant Placental transfer of 69%. Breastmilk transfer of 3.2% (MDH 2020) Half-life = 1417 days (3.9 years). (Zhang 2013, MDDHS 2019, ATSDR 2018) Volume of distribution = 0.2 L/kg (ATSDR 2018, Ohmori 2003). Applied to the Goeden Model. 95th percentile drinking water intake, consumers only, from birth to more than 21 years old (Goeden 2019) Upper percentile (mean plus two standard deviations) breast milk intake rate (Goeden 2019) Time-weighted average water ingestion rate from birth to 30-35 years of age (to calculate maternal serum concentration at delivery) (Goeden 2019) Relative Source Contribution of 50% (0.5) Based on NHANES 95th percentiles for 3-11 (2013-2014) and over 12 years old (2015-2016) participants (CDC 2019)
Chronic Non-Cancer MCLG	The model produces a Chronic Non-Cancer MCLG of 6 ppt. This protects health during the growth and development of a breast fed infant. Figure 8

Table 5: Development of Non-Cancer MCLG for PFNA

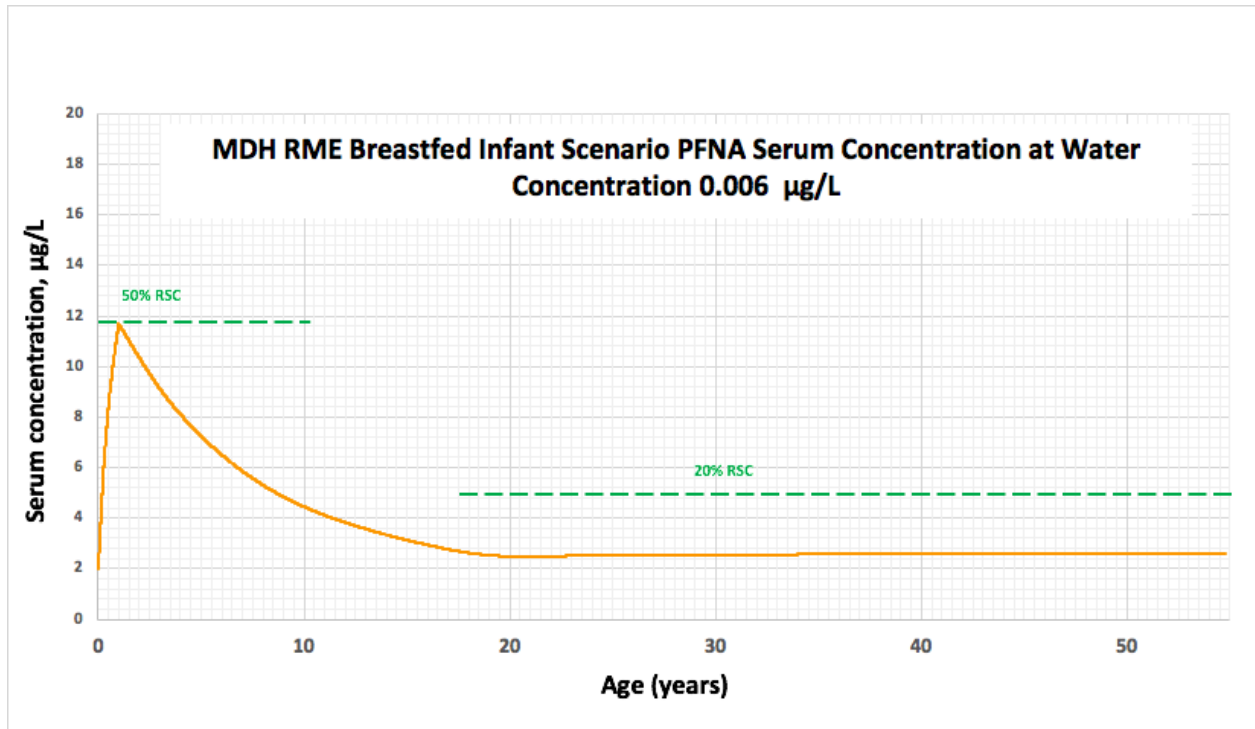


Figure 9. Using the Goeden Model, the reference dose and its parameters for PFNA were converted to an THSV of 0.023 mg/L. An RSC set at 50% means that half of this (0.0115 mg/L) will be from ingested drinking water. The MCLG of PFNA in drinking water should then be set at 0.006 ug/L or 6 PPT to protect from adverse health events.

7. PFHxS

After a literature search and a review of the available evidence and recommendations from various agencies, the DPAG developed an MCLG recommendation for PFHxS based on Non-Cancer endpoints. The critical study selected was Chang S (2018). This study identified reduced litter size following a 14 day prior to pregnancy oral exposure in Adult CD-1 female mice. Serum levels were measured at 14 days. MDHHS (MDHHS (2020 PFHXS) and NTP (2018) identified a POD of 32.4 mg/L serum concentration for male rats based on BMDL₂₀ analysis of this study. DPAG had selected a BMR of 10% (hence BMDL₁₀) as the preferred method for using BMD to select a POD and therefore rejected the use of BMDL₂₀. NHDES and Ali (2019) provided rigorous and more recent analysis and used a BMR of 50% of the Standard Deviation (BMDL_{0.5SD}). This was in keeping with EPA guidance on the selection criteria for BMRs and so was acceptable to the DPAG. The BMDL_{0.5SD} derived by Ali (2019) using data from the critical study was 13.9 mg/mL and provided the basis for the MCLG.

7.a. Summary of Critical Study

This study administered potassium perfluorohexanesulfonate (PFHxS) to CD-1 mice to assess for reproductive and developmental toxicity. Both male and female mice were assigned to one of four treatment groups: control, 0.3 mg/kg/d, 1 mg/kg/d, and 3 mg/kg/d with 30 mice of each sex assigned to each treatment group. Following an acclimation period that included observation of female mice for estrous cyclicity, male

and female mice were administered vehicle control or aqueous solution of PFHxS by oral gavage daily beginning 14 days prior to cohabitation. Males were administered vehicle or treatment for a total of at least 42 days with scheduled sacrifice one day post-last dose. F₀ females were administered vehicle or treatment until lactation day 21 with scheduled sacrifice one day later. After weaning on postnatal day 21, F₁ offspring were directly dosed with PFHxS for an additional 14 days at the same respective maternal dose.

F₀ mice were observed daily for clinical signs of toxicity before and 2 hours after oral gavage dosing. No signs of clinical toxicity were noted at any of the treatment levels. Body weights and food consumption were recorded weekly. There was a significant body-weight gain in male mice at the 0.3 mg/kg/d and 1 mg/kg/d dose levels but not at the 3 mg/kg/d dose; therefore, this was not considered to be treatment-related. There were no significant differences in body-weight gain in female mice across all treatment groups. There was no significant difference in food consumption across all treatment groups in either sex.

Functional observational battery and motor activity assessment was performed on 10 mice/sex/treatment group prior to scheduled sacrifice and no significant differences were noted across the treatment groups in any of the measured outcomes or in trend of motor activity over time.

Among F₀ mice, there was no significant difference among treatment groups with respect to any of the reproductive function outcomes investigated. In males, PFHxS did not affect sperm motility, count, density, and morphology. In females, PFHxS did not affect mating index, fertility index, or precoital interval.

With respect to pregnancy outcomes in F₀ mice, there was no significant difference between treatment groups in number of implantations, mean gestation length, number of dams with viable pups, pups born to implant ratio, and sex ratio. The number of pups born per litter and mean live litter size was significantly reduced in the 1 mg/kg/d and 3 mg/kg/d as compared to controls. The authors opined that the toxicological significance of that finding was unclear due to 1) the lack of a dose response relationship; 2) no significant difference in pup to implant ratio among treatment groups; and 3) the lack of other negative effects on developmental or reproductive outcomes.

At F₀ mice necropsy, there was no significant findings on macroscopic examinations across treatment groups. With the exception of liver weight, there was no difference across treatment groups on absolute or relative organ weights as compared to controls. PFHxS was associated with a significant, dose-dependent increase in both absolute and relative liver weight at the 1 mg/kg/d and 3 mg/kg/d in both male and female mice. This was considered to be an adaptive response.

With the exception of liver tissue, there was no difference across treatment groups in tissue histology. Liver tissue demonstrated primarily centrilobular hepatocellular hypertrophy among treatment groups with a dose-response relationship. In male mice only at the highest 3 mg/kg/d dose, mild microvesicular fatty change and minimal single-cell necrosis was noted in 6 of 10 and 4 of 10 mice, respectively. In female mice only at the highest 3 mg/kg/d dose, a low incidence of cytoplasmic vacuolation was seen in 3 out of 10 mice. Liver tissue findings were considered by the authors to be consistent with an adaptive response.

There was no difference between F₀ treatment groups with any hematology parameters or with serum TSH levels. And male mice only at the highest 3 mg/kg/d dose, there was a significant decrease in serum total cholesterol and bilirubin and a significant increase in alkaline phosphatase. This was considered to be an adaptive change related to increased metabolism of the parasites and unlikely to be of toxicological significance. There were no other significant differences in male mice in clinical chemistry parameters or in female mice in any clinical chemistry parameters.

Among F₁ mice, there was no significant difference between treatment groups on pup survival, body weight at birth or anytime thereafter, balanopreputial separation in males, vaginal patency in females, or areolae/nipple analgen retention in males. In male pups, a significantly increased anogenital distance was seen at all treatment levels as compared to controls; when adjusted to cube root body weight, a significantly increased anogenital distance was seen at the 0.3 mg/kg/d and 3 mg/kg/d treatment levels but not the 1 mg/kg/d treatment level. Among female pups, a decreased anogenital distance relative to cube root body weight was seen at the 1 mg/kg/d treatment level but no other treatment groups. The authors opined that these findings should not be considered toxicologically relevant in that no dose-response relationship was seen and that shortening of the anogenital distance rather than lengthening is indicative of anti-androgenic activity.

At F₁ mice necropsy, with the exception of liver and thyroid weight, there was no difference across treatment groups on absolute or relative organ weight as compared to controls. Absolute liver weight was significantly increased in males at the highest 3 mg/kg/d dose on PND 36 and relative liver weight was increased at the highest 3 mg/kg/d dose in males and females on PND 21 and 36. This was considered an

adaptive response. And female mice only at the highest 3 mg/kg/d dose, there was a significant increase in relative thyroid weight at PND 36 only but not on absolute thyroid weight. However, there were no thyroid histological abnormalities including hypertrophy in that group and no corresponding change in serum TSH levels.

With the exception of liver tissue, there was no difference across treatment groups in tissue histology. Liver tissue demonstrated mild centrilobular hepatocellular hypertrophy in both male and female pups with no evidence of necrosis. This was considered an adaptive response.

Analysis of liver mRNA transcript levels in F₀ and F₁ mice demonstrated increased transcripts that are sensitive to PPAR-alpha activation and CAR activation in the high-dose treatment group as compared to controls across both sexes in F₀ and F₁ mice. Cyp3a11, which is associated with PXR activation, was increased in the high-dose treatment group in F₀ males and F₁ pups of both sexes. Transcripts associated with fatty acid metabolism were increased in the high-dose treatment group across both sexes in F₀ and F₁ mice. However, transcripts associated with cellular stress were not increased.

A second toxicokinetic study was performed by the authors to determine serum and liver PFHxS concentrations at the same daily doses as the main study. The toxicokinetic study was divided into two subsets: 5 mice/sex/dose were administered PFHxS at 0.3 mg/kg/d, 1 mg/kg/d, and 3 mg/kg/d or vehicle control for 14 days prior to scheduled sacrifice. 7 mice/sex/dose were administered PFHxS at 0.3 mg/kg/d, 1 mg/kg/d, and 3 mg/kg/d or vehicle control for 14 days prior to cohabitation. Male mice were dosed for an additional 14 days with scheduled sacrifice one day post-last dose. Female mice were dosed through mating and gestation with scheduled sacrifice on gestation day 18.

Serum and liver sample collections were obtained at necropsy for male and female mice. For fetal serum and liver concentrations, pooled fetal blood and liver sample by litter were obtained at necropsy. The toxicokinetic study found that steady state observations for PFHxS were similar to that seen for PFOS as previously reported in rodent and monkey studies.

The authors concluded that at all doses studied, there was no effect of PFHxS on body weight, food consumption, estrus cyclicity, mating, fertility, gestation length, spermatogenesis, or macro and microscopic evaluation of reproductive organs in F₀ mice. A slight decrease in live litter size what is considered equivocal due to no dose response relationship and no change in the pup to implant ratio. Among F₁ mice, there was no effect of PFHxS on survival, birthweight, or reproductive development. Changes in liver weight, liver tissue microscopy, and clinical chemistry findings were all considered to be adaptive in nature.

7.b. Development of MCLG

The BMDL_{0.5SD} derived by (Ali 2019) using data from the critical study of Chang (2018) was 13.9 mg/mL and provided the basis for the MCLG. (Figure 9)

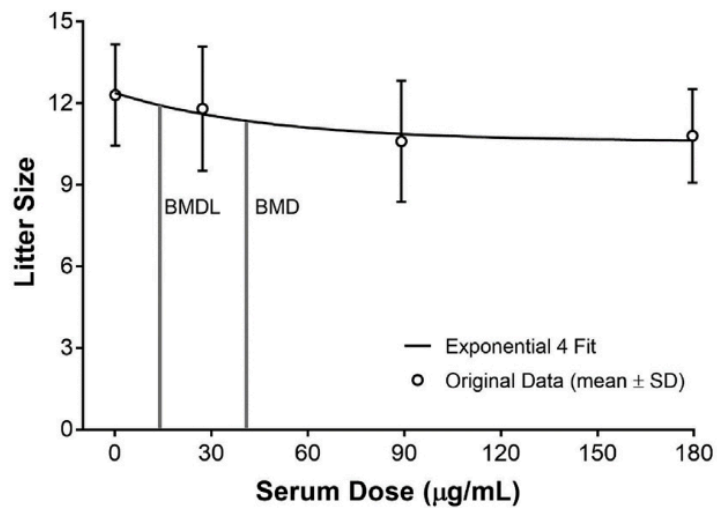


Fig. 2. Reduced litter size in female mice after 14-day oral exposure to K^+ PFHxS based on summarized data from Chang et al. (2018). The curve is calculated using the normal, constant variance exponential model. In this model, the BMD is the concentration that elicits a response 0.5 times the standard deviation below the mean of the tested population. The BMDL is the concentration corresponding to the lower 95% confidence interval.

Figure 10: $BMDL_{0.5SD}$ derived by Ali (2019) of 13.9 mg/mL using data from the critical study of Chang (2018).

DPAG agreed with the DAF, UFTs, and application of the Goeden Model by MDH and MDHHS. Setting the target to protect the breast fed infant as 0.023 mg/mL (50%RSC), the MCLG for drinking water is recommended to be 20 ng/L (20 PPT) to protect breast fed infants and throughout life. (Figure 10, Table 6)

PFHxS

Dose Response Modeling Method	lower confidence limit on the BMD on 50% of the SD (BMDL _{0.5SD})
POD	13.9 mg/mL
HED = POD x DAF	DAF based on Chemical-Specific Clearance Rate $DAF = V_d \text{ (L/kg)} \times (\ln 2 / \text{Half-life, days})$ $DAF = 0.25 \text{ L/kg} \times (\ln 2 / 1935 \text{ days})$ $DAF = 9.0 \times 10^{-2} \text{ mL/kg/d}$ $HED = POD \times DAF$ $HED = 13.9 \text{ mg/mL} \times 8.61 \times 10^{-2} \text{ mL/kg/d}$ $HED = 1.196 \times 10^{-3} \text{ mg/kg/d}$
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3 based on application of DAF
Subchronic to Chronic (UFS)	3 based on extrapolation from Chang S (2018)
LOAEL to NOAEL (UFL)	1
Database (UFD)	3 based on small number of studies
Total Composite (UFT)	300
RfD = HED/UFT (mg/kg/d)	$\text{Reference Dose} = HED / UFT$ $\text{Reference Dose} = 1.196 \times 10^{-3} \text{ mg/kg/d} / 300$ $\text{Reference Dose} = 3.98 \text{ ng/kg/d}$ (rounded to 4.0 ng/kg/d)
ITHSL = POD / UFT	$ITHSL = 13.9 \text{ mg/mL} / 300$ $ITHSL = 0.0463 \text{ mg/mL}$
Receptor	Breast-fed infant, which is also protective of a formula-fed infant. Placental transfer of 70% (MDH 2020 PFHXS). Breastmilk transfer of 1.4% (Li 2019). Half-life = 1935 days. $V_d = 0.25 \text{ L/kg}$ (USEPA 2016, Han 2012). 95th percentile drinking water intake, consumers only, from birth to more than 21 years old (Goeden [2019]) Upper percentile (mean plus two standard deviations) breast milk intake rate (Goeden 2019) Time-

	weighted average water ingestion rate from birth to 30-35 years of age (to calculate maternal serum concentration at delivery) (Goeden 2019) Relative Source Contribution of 50% (0.5). Based on NHANES 95th percentiles for 3-11 (2013-2014) and over 12 years old (2015-2016) participants (CDC 2019)
Chronic Non-Cancer MCLG	The model produces a Chronic Non-Cancer MCLG of 20 ppt. This protects health during the growth and development of a breast fed infant.

Table 6: Development of Non-Cancer MCLG for PFHxS

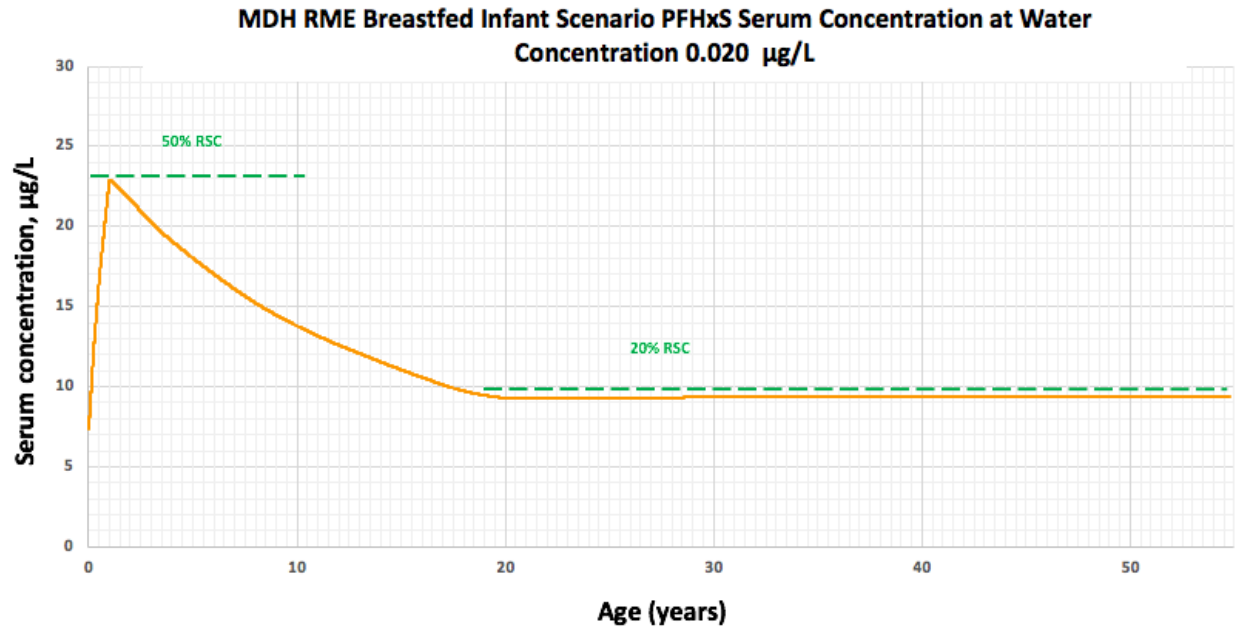


Figure 11. Using the Goeden Model, the reference dose and its parameters for PFHxS were converted to an THSV of 0.046 mg/L. An RSC set at 50% means that half of this (0.023 mg/L) will be from ingested drinking water. The MCLG of PFHXS in drinking water should then be set at 0.020 ug/L or 20 PPT to protect from adverse health events.

8. PFHpA

PFHpA is a difficult compound to develop advisories for because there is a paucity of evidence on its toxicity. The DPAG decided to base recommendations on its chemical structure. MDHHS (2019) has made similar recommendations for other PFAS that lack sufficient scientific evidence to form conclusions about health advisory levels. Like PFOA, PFHpA is a carboxylic acid. PFHpA is a 7-carbon molecule and PFOA is an 8 carbon molecule. The DPAG concludes that the MCLG for PFHpA should be conservatively set at the same threshold for PFOA – 8 PPT.

9. PFBS

After a literature search and a review of the available evidence and recommendations from various agencies, the DPAG developed an MCLG recommendation for PFBS based on Non-Cancer endpoints. The DPAG identified Feng 2017 as the critical study. The ATSDR 2018 considered the available data inadequate for identifying a critical endpoint and evaluating dose-response relationships but did not review Feng 2017. USEPA (2018 PFBS) selected Lieder (2009) and the critical effect of papillary tubular ductal epithelium hyperplasia in P0 females. They applied BMD with a BMR of 10%. The derived BMDL₁₀ (HED) of 11.5 mg/kg/d was modified with a UFT of 1000 to achieve a reference dose of 1x10⁻² (mg/kg/d). Interestingly, USEPA (2018 PFBS) identified the decreased serum total T4 in newborn (PND 1) mice from Feng 2017 as a critical effect and performed a BMD modeling, but selected a BMR of 20%

over control response rate. The modeled BMDL₂₀ and applied a UFT of 300 achieved the same reference dose of 1×10^{-2} (mg/kg/d) as the kidney critical effect from Lieder 2009. MDHHS identified the kidney effects as a potentially compensatory response and thought the thyroid effects had greater functional significance. However, they removed the allometric scaling used in the draft USEPA (2018 PFBS) and applied the PFBS specific DAF developed by MDH. Thus, MDHHS was able to develop a chemical specific HED. However, MDH did use the BMDL₂₀ identified by the US EPA to calculate their HED. DPAG chose to continue with use of the BMDL₁₀ as the standard approach where the model fit was valid and used the USEPA (2018 PFBS) BMD modeling which, in addition to the BMDL₂₀, included a calculated BMDL₁₀ of 1.84 mg/kg/d. This BMDL₁₀ POD HED of 1.84 mg/kg/d was divided by 0.149 to remove the DAF employed by USEPA (2018 PFBS) prior to subjecting the data to BMD analysis (USEPA 2018 PFBS). This results in a POD of 12.35 mg/kg/d. DPAG agreed with the application of half-life ratios by MDH of the new chemical specific DAF of 316 (human serum half-life/female mouse serum half-life = 665 hours/2.1 hours = 316). (MDH 2020 PFBS) Dividing by the new chemical specific DAF of 316 (human serum half-life/female mouse serum half-life = 665 hours/2.1 hours = 316) results in a HED of 0.039 mg/kg/d.

9.a. Review of Critical Study

This study investigated the effects of prenatal perfluorobutanesulfonate (PFBS) exposure on perinatal growth and development, people on site, and reproductive and thyroid endocrine system function in female ICR mice. PFBS potassium salt was administered orally to pregnant mice at doses of 50, 200, and 500 mg/kg/d from GD1 to

GD20. Administration of the test substance did not affect weight gain, fetal loss, or behavior of the dams at the doses studied. 30 dams were assigned to one of three experimental groups: 1) sequential examination of perinatal survival and growth, pubertal onset, and ovarian and uterine development; 2) hypothalamic-pituitary-gonadal hormone and hypothalamic pituitary thyroid hormone measurements at postnatal days 1, 30, and 60; 3) measurement of serum levels of PFBS.

Postnatal day 1 body weights of female offspring at the 200 mg/kg/d dose and above were decreased relative to controls. These dose groups remained underweight throughout weaning, pubertal, and adult periods. Delays in eye-opening, vaginal opening, and first estrous period were seen in female offspring at the 200 mg/kg/d dose and above with a dose response relationship.

Absolute and relative ovary weight were decreased at the 200 mg/kg/d dose and above, although no dose response relationship was seen. Number of primordial follicles, primary follicles, secondary follicles, early antral follicles, antral follicles, pre-ovulatory follicles, and corpora lutea were decreased at the 200 mg/kg/d dose and above, although no dose response relationship was seen.

Absolute and relative uterine weight were decreased at the 200 mg/kg/d dose and above, although no dose response relationship was seen. Total uterine diameter, endometrial thickness, and myometrial thickness were decreased at the 200 mg/kg/d dose and above, with a minimal dose response relationship.

Number of days spent in diestrus stage were significantly increased in female offspring at the 200 mg/kg/d dose and above as compared to controls, although no dose response relationship was seen. Levels of serum E2 were decreased at the 200

mg/kg/d dose and above on postnatal day 30 and 60 but not on postnatal day 1 and with no dose response relationship. Levels of luteinizing hormone (LH) were decreased at the 200 mg/kg/d dose and above on postnatal day 30 but not on postnatal day 1 or 60 with no discernible dose response relationship. Levels of P4 were decreased at the 200 mg/kg/d dose and above on postnatal day 60 but not on postnatal day 1 or 30 with no discernible dose response relationship. Levels of gonadotropin-releasing hormone (GnRH) were not affected at any of the doses studied.

Total T3 and total T4 was significantly decreased in female offspring at the 200 mg/kg/d dose and above on postnatal day 1, 30 and 60, although no clear dose response relationship was seen. TSH and hypothalamic *Trh* mRNA were both increased at the 200 mg/kg/d dose and above on postnatal day 30, but not on postnatal day 1 or 60. In dams, total T4, total T3, free T4 were decreased and TSH was increased at the 200 mg/kg/d dose and above without an obvious dose response relationship.

9.b. Development of MCLG

DPAG agreed with USEPA selection of a decreased serum total T4 in newborn (PND 1) mice from Feng 2017 but used the USEPA reported BMDL₁₀ of 1.84 mg/kg/d.

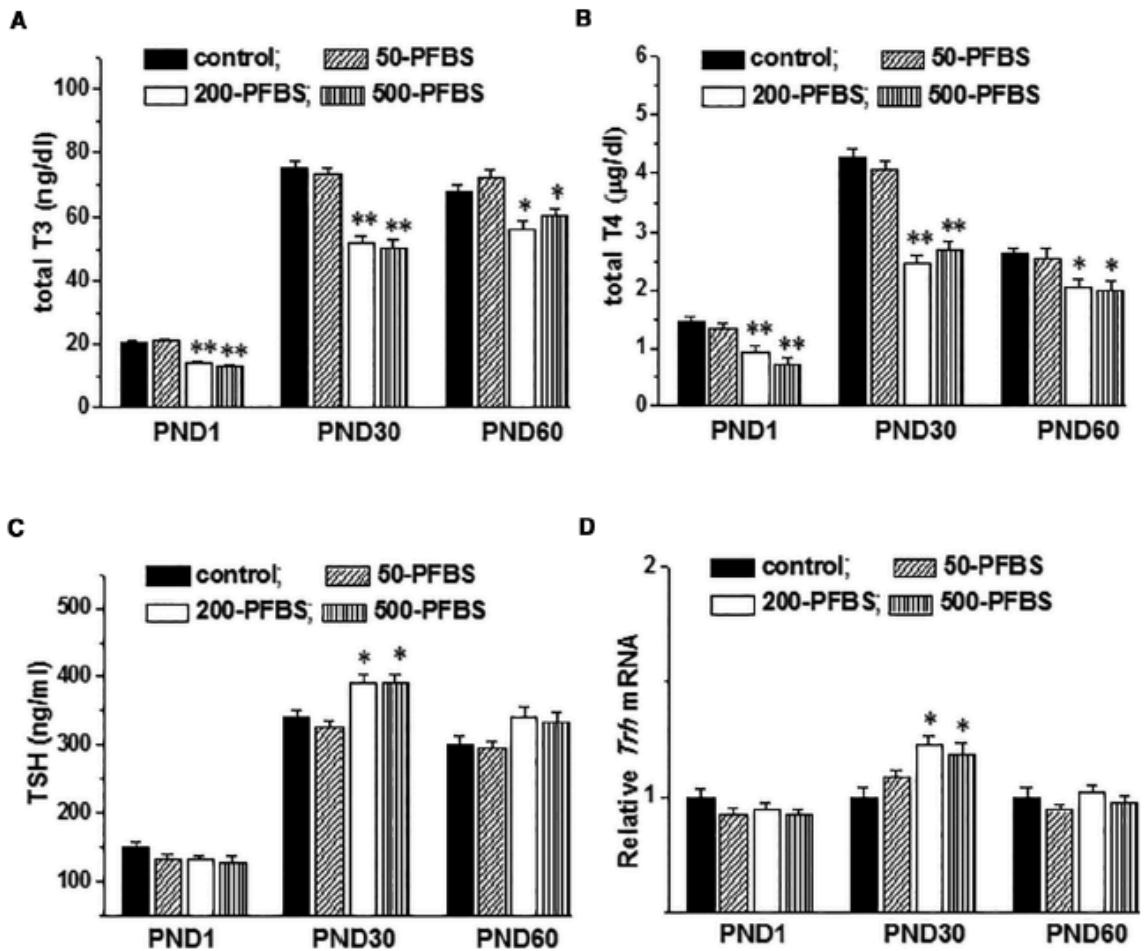


FIG. 4. Influence of prenatal perfluorobutanesulfonate (PFBS) exposure on hypothalamic-pituitary-thyroid hormone levels. Bar graphs show the levels of serum total 3,3',5-triiodothyronine (T3) (A), total thyroxine (T4) (B), thyroid-stimulating hormone (TSH) (C) and hypothalamic *Trh* mRNA (D) in postnatal day (PND) 1, PND30, and PND60 control offspring and PFBS-offspring. *P < 0.05 and **P < 0.01 versus control offspring (1-way ANOVA).

Figure 12: Critical effect of PFBS on total thyroxine (T4) levels identified by Feng 2017 used to develop BMDL₁₀ POD.

This BMDL₁₀ POD HED of 1.84 mg/kg/d was divided by 0.149 (USEPA 2018 PFBS) page F-10 to F-13) to remove the DAF employed prior to subjecting the data to BMD analysis (USEPA 2018 PFBS). This results in a POD of 12.35 mg/kg/d. Dividing by the chemical specific DAF of 316 (human serum half-life/female mouse serum half-life = 665 hours/2.1 hours = 316) (MDH 2020 PFBS) results in a HED of 0.039 mg/kg/d. DPAG agreed with the UFT applied by USEPA. Applying the USEPA ingestion rate for

birth to < 1 year old and a conservative 20% RSC, the MCLG for drinking water is recommended to be 55 ng/L (55 PPT) to protect infants and throughout life. (Table 7)

PFBS

Dose Response Modeling Method	BMDL ₁₀
POD HED Units	US EPA reported BMDL ₁₀ of 1.84 mg/kg/d. This was divided by 0.149 (USEPA 2018 PFBS) to derive a POD of 12.35 mg/kg/d.
POD x DAF = HED	DAF = (human serum half-life/female mouse serum half-life) DAF = 665 hours/2.1 hours DAF = 317 (MDH 2020 PFBS). HED = POD (BMDL ₁₀) / DAF HED = 12.35 mg/kg/d / 317day. HED = 0.0390 mg/kg/d
Uncertainty Extrapolation (USEPA 2018)	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	3 A UFS of 3 is applied because the POD comes from a developmental study of mice. Although this is a susceptible life stage, additional concern over potential hazards following longer-term (chronic) cannot be completely accounted for with this study.
LOAEL to NOAEL (UFL)	1 (BMDL)
Database (UFD)	10 The database lacks studies of chronic duration, neurodevelopment, and immunotoxicity.
Total Composite (UFT)	1000
HED/UFT= Reference Dose (mg/kg-day)	39.0 ng/kg/day (0.000039 mg/kg/d)
Receptor	infant
Ingestion Rate (L/day)	Based on National Health and Nutrition Examination Survey (NHANES) 2005–2010, 95 th percentile of water intake for consumers only (direct and indirect consumption) for infants (birth to <1 year old) of 1.106 L/day, per Table 3-17, USEPA Exposure Factors Handbook, 2019.

Body Weight (Kg)	An infant body weight of 7.8 kilograms was used and represents a time-weighted average for birth to 1 year old (Table 8-1, USEPA 2019).
Normalized Drinking Water Intake (L/kg-day)	0.142
Relative Source Contribution	20%
Chronic Non-Cancer MCLG	Chronic Non-Cancer MCLG = RfD x RSC / DWI Chronic Non-Cancer MCLG = 0.055 ug/L or 55 PPT

Table 7: Development of Non-Cancer MCLG for PFBS

10. GenX (HFPO dimer acid and its ammonium salt)

After a literature search and a review of the available evidence and recommendations from various agencies, the DPAG developed an MCLG recommendation for GenX based on Non-Cancer endpoints. US EPA 2018 selected the DuPont oral reproductive/developmental toxicity study in mice as the critical study. (DuPont-18405-1037, 2010). DPAG reviewed this and found it sufficiently robust to provide quality data.

US EPA selected liver effects (single-cell necrosis in male mice) as the critical effect for deriving the subchronic and chronic RfDs for GenX (HFPO dimer acid and its ammonium salt). USEPA (2018) evaluated the relevance of this endpoint in humans and noted that, per Hall, (Hall 2012) liver effects accompanied by effects such as necrosis or inflammation, among others, are indicative of liver tissue damage (USEPA, 2018). This effect is distinct from PPAR α -mediated rodent hepatocarcinogenesis. US EPA performed BMD modeling with a BMR of 10%. They reported a BMDL₁₀ of 0.15 mg/kg/d based on BMD Multistage 2 model. DAF of 0.15 was developed using allometric scaling, per USEPA (2018 GenX) guidance, since no chemical-specific data on human serum half-life was available that would allow this conversion. Conversely, NCDEQ (NCDDHS 2017) decided against BMD modeling, stating it was statistically unreliable due to poor model fit and large confidence interval. They chose a NOAEL POD and applied a UFT of 1000 to achieve a subsequent RfD at 100 ng/kg/day.

Ultimately, DPAG adopted the approach used by the EPA to develop a HED_{BMDL10} , applied a UFT 300 and produced an RfD of 76.7 ng/kg/day. The ingestion modeling used by NCDEQ to target bottle fed infants was in keeping with the DPAG approach of targeting the most vulnerable populations for protective MCLG. The final MCLG is 108 PPT.

10.a. Review of Critical Study

This study investigated subchronic toxicity of H-28548 (HFPO dimer acid ammonium salt) in Crl:CD1(ICR) mice. Adult male and female mice were administered H-28548 at a dose of 0, 0.1, 0.5, or 5 mg/kg/d by oral gavage with a total of 10 mice per sex per dose for 96 (males) or 97 (females) days. Mice were observed daily for signs of acute toxicity. Body weight, food consumption, and detail the clinical observations were performed weekly. Ophthalmology examination, functional observational battery, and motor activity were evaluated at outset and at the conclusion of the study. Hematology and clinical chemistry studies were performed at study conclusion. Surviving mice were sacrificed and gross and microscopic pathological examinations were performed.

Body weight and body weight gain were increased in the male 5 mg/kg/d dose group relative to control, which was attributed to increased liver weight and not considered an adverse effect. No statistically significant change in body weight or body weight gain were seen any other dose groups. Food consumption and food efficiency were increased in the male 5 mg/kg/d dose group relative to control, which was attributed to increased liver weight and body weight, respectively, and not considered an adverse

effect. No statistically significant change in food consumption or food efficiency were seen any other dose groups.

No acute toxicity or test substance related deaths were seen at any of the doses studied. The test substance had no effect on functional observational battery outcomes at any of the doses studied.

Mean corpuscular hemoglobin (MCHC) was decreased in the male 5 mg/kg/d group relative to controls; because the decrease was minimal (97% of control) and there were no other statistically significant changes in red cell parameters, this outcome was considered to be spurious. Platelet count was increased in males at 0.5 and 5 mg/kg/d, but this did not demonstrate a dose-response relationship, was not associated with clinical signs or pathological changes, and was not seen in a previous 28-day gavage study and was considered to be unrelated to the test substance and not adverse. Absolute monocyte count was decreased in females at 0.1 mg/kg/d. However, similar changes were not demonstrated in the higher dose groups and this effect was considered to be not test substance related or adverse.

AST, ALT, sorbitol dehydrogenase, alkaline phosphatase and total bile acids were increased in the male 5 mg/kg/d group as compared to controls. ALT, sorbitol dehydrogenase, and alkaline phosphatase were increased in the female 5 mg/kg/d group as compared to controls. Changes in these parameters correlated with hepatocellular damage and/or cholestasis and were considered to be adverse effects related to the test substance. Significant differences in liver function parameters were not seen at the lower test doses. Total protein and albumin were increased, and total cholesterol was decreased in male mice at the 5 mg/kg/d dose, however the magnitude

of change was small, was considered to be related to the test substance but non-adverse in nature. Albumin was increased and bilirubin was decreased in the female 5 mg/kg/d group, however the magnitude of change was small and was considered to be non-adverse. Decreased Bilirubin was also seen in male mice at the 0.5 mg/kg/d dose, but this finding was not replicated at higher doses and was considered to be spurious.

Serum potassium was decreased in male and female mice at the 5 mg/kg/d dose. The changes were not associated with any clinical signs of hypokalemia and this finding was considered to be non-adverse. Chloride was higher in male mice at the 5 mg/kg/d dose, which was considered to be unrelated to the test substance and non-adverse.

Absolute and relative liver weight were increased in male mice at the 0.5 and 5 mg/kg/d those groups relative to control, with a dose response relationship. Absolute and relative liver weight were increased in female mice at the 5 mg/kg/d dose group only. These changes were associated with gross and microscopic pathology findings and were considered to be treatment related.

Relative kidney weight as compared to brain was increased in males at the 5 mg/kg/d dose group; however, absolute and relative kidney weight as compared to body were unchanged and this finding therefore was considered to be of uncertain significance. Relative brain and epididymis weight were lower and relative heart weight as compared to brain was higher in males at the 5 mg/kg/d dose; however, absolute changes in the organ weights were not significant and these findings were not associated with any microscopic pathology findings and were considered to be not related to the test substance. Relative spleen weight was decreased in females at the

0.5 and 5 mg/kg/d dose groups; however, there was no dose response relationship or findings on microscopic pathology examination and these findings were therefore considered spurious and unrelated to the test substance. Absolute and relative ovary weight were increased in females at the 0.5 mg/kg/d dose; however, there was no dose response relationship, the increased ovary weight was attributed to ovarian cysts present in three female mice in that dose group, and this finding was therefore considered spurious and unrelated to the test substance.

There was a significant increase in enlarged and discolored livers in males at the 0.5 and 5 mg/kg/d dose group and in females at the 5 mg/kg/d dose group as compared to controls. These findings were considered to be related to the test substance. There were no other findings on gross pathology examination that were considered to be related to the test substance.

On microscopic examination, hepatocellular hypertrophy without liver cell injury was seen in male mice at the 0.5 mg/kg/d dose, which was considered to be treatment related but not adverse. Hepatocellular hypertrophy, hepatocellular single cell necrosis, and increased pigment concentration in Kupffer cells were seen in both male and female mice at the 5 mg/kg/d dose. An increased number of mitotic figures were seen in male but not female mice at the same dose. Incidences and severity of liver changes were greater in males as compared to females. These changes correlated with clinical chemistry effects and were considered to be both treatment related and adverse effects. Minimal renal tubular epithelial hypertrophy was seen in male mice at the 5 mg/kg/d dose, but this was not associated with renal tubular cell degeneration or necrosis or any

change in clinical chemistry parameters and was therefore considered to be non-adverse. No other microscopic observations were considered to be treatment related.

An additional pharmacokinetic study was performed in which male and female adult mice were administered the same H-28548 doses at 5 mice per sex dose per timepoint and evaluated for plasma concentration of the test substance approximately two hours after dosing on test days 0, 28, and 95. These mice were also evaluated for bodyweight, food consumption, and clinical signs of overt toxicity but did not have the ophthalmology (postexposure), neurobehavioral, hematology, clinical chemistry, or pathology examinations. Test substance concentration in blood was similar on days 0, 28, and 95 and female mice indicating rapid clearance of the substance from the blood and steady state concentrations achieved on the first day of dosing. In male mice, steady state concentration was achieved by day 28.

10.b. Development of MCLG

DAPG adopted the USEPA performed BMD modeling with a BMR of 10% and a reported $BMDL_{10}$ of 0.15 mg/kg/d based on BMD Multistage 2 model. A DAF of 0.15 was developed using allometric scaling, per USEPA (2018 GenX) guidance, since no chemical-specific data on human serum half-life was available that would allow this conversion. DPAG adopted the approach used by the EPA to develop a $HED_{BMDL_{10}}$, applied a UFT 300 and produced an RfD of 76.7 ng/kg/day. The ingestion modeling used by NCDHHS (2017) to target bottle fed infants was in keeping the DPAG approach of targeting the most vulnerable populations for protective MCLG (Table 8). The final MCLG is 108 PPT.

GenX	
Method of Administered Dose conversion to Internal Serum Level	BMR 10% BMDL ₁₀ of 0.15 mg/kg/d based on BMD Multistage 2 model developed by USEPA (2018 GenX)
Method to Derive Human Equivalent Dose	Allometric DAF = $(BWA^{1/4}/BWH^{1/4})$
Dose Response Modeling Method	BMDL ₁₀ from USEPA (2018 GenX)
HED _{BMDL10} = POD x DAF	DAF = $(BWA^{1/4}/BWH^{1/4})$ DAF = $(0.0372 \text{ kg})^{1/4}/(80 \text{ kg})^{1/4}$ DAF = 0.15 HED _{BMDL10} = POD (BMDL ₁₀) x DAF HED _{BMDL10} = 0.15mg/kg/d x 0.15 HED _{BMDL10} = 0.0225 mg/kg/d
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	3
LOAEL to NOAEL (UFL)	1 (BMDL)
Database (UFD)	3 (insufficient number of studies)
Total Composite (UFT)	300
RfD = HED/UFT (mg/kg/d)	76.7 ng/kg/day (76.7 x10 ⁻⁶ mg/kg/d)
Receptor	Bottle fed infant
Ingestion Rate (L/day)	Based on National Health and Nutrition Examination Survey (NHANES) 2005–2010, 95 th percentile of water intake for consumers only (direct and indirect consumption) for infants (birth to <1 year old) of 1.106 L/day, per Table 3-17, USEPA Exposure Factors Handbook, 2019.
Body Weight BW (Kg)	An infant body weight of 7.8 kilograms was used and represents a time-weighted average for birth to 1 year old (Table 8-1, USEPA 2019).

Normalized Drinking Water Intake (NDWI) (L/kg-day)	0.142
Relative Source Contribution (RSC)	20%
MCLG	$MCLG = RfD \times RSC / NDWI$ MCLG = 0.108 ug/L or 108 PPT

Table 8: Development of Non-Cancer MCLG for GenX

11. Summary

The DPAG had the opportunity to build on the diligent work of a great number of US and State agencies who preceded us. We strove to find the best practices wherever possible and apply them in a scientifically valid and data driven manner. As new information becomes available, we would welcome the opportunity to review these MCLG recommendations and modify when appropriate. The summary of recommendations are as follows:

1. These proposed Non-Cancer MCLGs are suggested with the health of the most vulnerable populations in mind
2. Individual MCLGs are advisable and the most scientifically rigorous approach
3. Non-Cancer MCLGs are low enough to protect against Cancer endpoints

PFAS	Reference Dose	MCLG proposed
perfluorooctanoic acid (PFOA)	3.9 ng/kg/day	8 PPT
perfluorooctanesulfonic acid (PFOS)	3.1 ng/kg/day	14 PPT
perfluorononanoic acid (PFNA)	2.2 ng/kg/day	6 PPT
perfluorohexanesulfonic acid (PFHxS)	4.0 ng/kg/day	20 PPT
perfluoroheptanoic acid (PFHpA)	None derived	8 PPT
perfluorobutanesulfonic acid (PFBS)	39 ng/kg/day	55 PPT
ammonium salt of hexafluoropropylene oxide dimer (GenX)	75 ng/kg/day	108 PPT

We would like to thank the Commonwealth of Pennsylvania and the Pennsylvania Department of Environmental Protection for the opportunity to participate in this important work and protect the health and safety of Pennsylvanians.

References

Ahrens L (2011). "Polyfluoroalkyl compounds in the aquatic environment: a review of their occurrence and fate." *Journal of Environmental Monitoring* 13 (1):20-31. doi: 10.1039/c0em00373e.

Ahrens L, Bundschuh M (2014). "Fate and effects of poly- and perfluoroalkyl substances in the aquatic environment: A review." *Environmental toxicology and chemistry* 33 (9):1921-1929.

Ahrens L, Ebinghaus R (2010). "Spatial distribution of polyfluoroalkyl compounds in dab (*Limanda limanda*) bile fluids from Iceland and the North Sea." *Marine Pollution Bulletin* 60 (1):145-148.

Ahrens L, Siebert U, Ebinghaus R (2009). "Total body burden and tissue distribution of polyfluorinated compounds in harbor seals (*Phoca vitulina*) from the German Bight." *Marine Pollution Bulletin* 58 (4):520-525.

Ali JM, Roberts SM, Gordon DS, Stuchal LD (2019). "Derivation of a chronic reference dose for perfluorohexane sulfonate (PFHxS) for reproductive toxicity in mice." 2019. *Regul Toxicol Pharmacol*. 2019 Nov;108:104452. doi: 10.1016/j.yrtph.2019.104452. Epub 2019 Sep 2. PMID: 31487490

Anderson RH, Long GC, Porter RC, and Anderson JK (2016). "Occurrence of select perfluoroalkyl substances at US Air Force aqueous film-forming foam release sites other than fire-training areas: Field-validation of critical fate and transport properties." *Chemosphere* 150:678-685. doi: 10.1016/j.chemosphere.2016.01.014.

ATSDR (2018). Agency for Toxic Substances and Disease Registry. Toxicological Profile for Perfluoroalkyls. Draft for Public Comment. June 2018. <https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf> Accessed online December 9, 2020

Backe WJ, Day TC, Field JA (2013). "Zwitterionic, cationic, and anionic fluorinated chemicals in aqueous film forming foam formulations and groundwater from US military bases by nonaqueous large-volume injection HPLC-MS/MS." *Environmental science & technology* 47 (10):5226-5234.

Baduel C, Paxman CJ, Mueller JF (2015). "Perfluoroalkyl substances in a firefighting training ground (FTG), distribution and potential future release." *Journal of hazardous materials* 296:46-53.

Bartell SM, Calafat AM, Lyu C, Kato K, Rayn P, Steenland K (2010). "Rate of decline in serum PFOA concentrations after granular activated carbon filtration at two public water systems in Ohio and West Virginia." *Environ Health Perspect* 118(2):222-228.

Beck NB, Becker RA, Erraguntla N, Farland WH, Grant RL, Gray G, Kirman C, LaKind, JS, Lewis RJ, Nance P, Pottenger LH, Santos SL, Shirley S, Simon T, and Dourson ML (2016). "Approaches for describing and communicating overall uncertainty in toxicity characterizations: U.S. Environmental Protection Agency's Integrated Risk Information System (IRIS) as a case study." *Environment International* 89-90, 110-128.

Bonfeld-Jorgensen EC, Long M, Bossi R, Ayotte P, Asmund G, Kruger T, (2011) "Perfluorinated compounds are related to breast cancer risk in Greenlandic Inuit: a case control study." *Environ Health* 2011:10;88.

Butenhoff J, Chang SC, Ehresman DJ, York RG 2009. "Evaluation of potential reproductive and developmental toxicity of potassium perfluorohexanesulfonate in Sprague Dawley rats." *Reprod. Toxicol.* 2009:27;331–341.

California Environmental Protection Agency (CEPA) (2019). Notification Level Recommendations for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS) in Drinking Water. Prepared by Pesticide and Environmental Toxicology Branch Office of Environmental Health Hazard Assessment. Accessed online Dec 2020. <https://oehha.ca.gov/water/notification-level/notification-level-recommendations-perfluorooctanoic-acid-pfoa>

Center for Disease Control (CDC) (2019). Fourth National Report on Human Exposure to Environmental Chemicals, Updated Tables, January 2019, Volume One. https://www.cdc.gov/exposurereport/pdf/FourthReport_UpdatedTables_Volume1_Jan2019-508.pdf

Chang ET, Adami H, Boffetta P, Cole P, Starr TB, Mandel JS (2014). "A critical review of perfluorooctanoate and perfluorooctanesulfonate exposure and cancer risk in humans." *Critical Reviews in Toxicology* 2014:44:sup1, 1-81, DOI: 10.3109/10408444.2014.905767

Chang S, Butenhoff JL, Parker GA, Coder PS, Zitzow JD, Krisko RM, Bjork JA, Wallace, KB, Seed JG (2018). "Reproductive and developmental toxicity of potassium perfluorohexanesulfonate in CD-1 mice." *Reprod Toxicol* 2018:78;150–168.

Clarke BO, Smith SR (2011). "Review of 'emerging' organic contaminants in biosolids and assessment of international research priorities for the agricultural use of biosolids." *Environment international* 37(1):226-247.

Conder JM, Hoke RA, de Wolf W, Russell MH, Buck RC (2008). "Are PFCAs bioaccumulative? A critical review and comparison with regulatory criteria and persistent lipophilic compounds." *Environmental science & technology* 42 (4):995-1003.

Das KP, Grey BE, Rosen MB, (2015). "Developmental toxicity of perfluorononanoic acid in mice." *Reproductive Toxicology* 51:133-144.

Dong GH, Liu MM, Wang D, Zheng L, Liang ZF, Jin YH (2011). "Sub-chronic effect of perfluorooctanesulfonate (PFOS) on the balance of type 1 and type 2 cytokine in adult C57BL6 mice." *Arch Toxicol.* Oct;85(10):1235-44. doi: 10.1007/s00204-011-0661-x. Epub 2011 Feb 16. PMID: 21327619.

DuPont-18405-1037: E.I. du Pont de Nemours and Company (2010). An Oral (Gavage) Reproduction/Developmental Toxicity Screening Study of H-28548 in Mice. USEPA OPPTS 870.3550; OECD Test Guideline 421. Study conducted by WIL Research Laboratories, LLC (Study Completion Date: December 29, 2010), Ashland, OH. (as cited in USEPA (2018)).

Eriksen KT, Sørensen M, McLaughlin JK, Lipworth L, Tjønneland A, Overvad K, Raaschou-Nielsen O (2009). "Perfluorooctanoate and perfluorooctanesulfonate plasma levels and risk of cancer in the general Danish population." *J Natl Cancer Inst.* 2009 Apr 15;101(8):605-9. doi: 10.1093/jnci/djp041. Epub 2009 Apr 7. PMID: 19351918.

Feng X, Cao X, Zhao S, Wang X, Hua X, Chen L, Chen L (2017). "Exposure of pregnant mice to perfluorobutanesulfonate causes hypothyroxinemia and developmental abnormalities in female offspring." *Toxicol Sci* 155: 409-419.

Filipovic MA, Woldegiorgis K, Norström MB (2015). "Historical usage of aqueous fillm forming foam: A case study of the widespread distribution of perfluoroalkyl acids from a military airport to groundwater, lakes, soils and fish." *Chemosphere* 189:39-45.

Goeden HM, Greene CW, Jacobus, JA (2019) "A transgenerational toxicokinetic model and its use in derivation of Minnesota PFOA water guidance." *J. Exposure Sci. Env. Epidemiol.* 29:183-195.

Guo Z, Liu X, Krebs KA, Roache NF. 2009. "Perfluorocarboxylic acid content in 116 articles of commerce." Research Triangle Park, NC: US Environmental Protection Agency.

Hall AP, Elcombe CR, Foster JR, (2012) "Liver Hypertrophy: A Review of Adaptive (Adverse and Non-adverse) Changes—Conclusions from the 3rd International ESTP Expert Workshop." *Toxicologic Pathol.* 40:971-94.

Hamid H, Li L (2016). "Role of wastewater treatment plant (WWTP) in environmental cycling of poly-and perfluoroalkyl (PFAS) compounds." *Ecocycles* 2 (2):43-53.

Han X, Nabb DL, Russell MH, Kennedy GL, Rickard RW (2012). "Renal elimination of perfluorocarboxylates (PFCAs)." *Chem Res Toxicol.* 25:35–46

Hardell E, Karrman A, van Bavel, Bao J, Carlberg M, Hardell L (2014). "Case-control study on perfluorinated alkyl acids (PFAAs) and the risk of prostate cancer." *Environ Int* 2014,63;35–9.

Harding-Marjanovic, Katie C, Erika F Houtz, Shan Yi, Jennifer A Field, David L Sedlak, and Lisa Alvarez-Cohen (2015). "Aerobic biotransformation of fluorotelomer thioether amido sulfonate (Lodyne) in AFFF-amended microcosms." *Environmental science & technology* 49(13):7666-7674.

Houtz EF, Higgins CP, Field JA (2013). "Persistence of perfluoroalkyl acid precursors in AFFF-impacted groundwater and soil." *Environmental Science & Technology* 47 (15):8187-8195. doi: 10.1021/es4018877.

Innes KE, Wimsatt JH, Frisbee S, Ducatman AM (2014). "Inverse association of colorectal cancer prevalence to serum levels of perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA) in a large Appalachian population." *BMC Cancer* 2014,14,45.

Institute of Medicine (2011). *Committee on Standards for Developing Trustworthy Clinical Practice Guidelines*; Editors: Robin Graham, Michelle Mancher, Dianne Miller Wolman, Sheldon Greenfield, and Earl Steinberg. *Clinical Practice Guidelines We Can Trust*. Washington (DC): National Academies Press (US); 2011.

Klaunig JE, Shinohara M, Iwai H, Chengelis CP, Kirkpatrick JB, Wang Z, Bruner RH (2015). "Evaluation of the chronic toxicity and carcinogenicity of perfluorohexanoic acid (PFHxA) in Sprague-Dawley rats." *Toxicol. Pathol.* 43 (2), 209–220.

Konwick BJ, Tomy GT, Ismail N, Peterson JT, Fauver RF, Higginbotham D, Fisk AT (2008). "Concentrations and patterns of perfluoroalkyl acids in Georgia, USA surface waters near and distant to a major use source." *Environmental toxicology and chemistry* 27 (10):2011-2018.

Koskela A, Finnilä MA, Korkalainen M, Spulber S, Koponen J, Håkansson H, Tuukkanen J, Viluksela M (2016) "Effects of developmental exposure to perfluorooctanoic acid (PFOA) on long bone morphology and bone cell differentiation." *Toxicol Appl Pharmacol.* 301:14-21.

Li Y, Fletcher T, Mucs D, Scott K, Lindh CH, Tallving P, Jakobsson K (2018). "Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water." *Occup Environ Med.* 2018 Jan;75(1):46-51. doi: 10.1136/oemed-2017-104651. Epub 2017 Nov 13. PMID: 29133598; PMCID: PMC5749314.

Lindstrom AB, Strynar MJ, Libelo EL (2011). "Polyfluorinated Compounds: Past, Present, and Future." *Environmental Science & Technology* 45 (19):7954-7961. doi:

Loveless SE, Finlay C, Everds NE, Frame SR, Gillies PJ, O'Connor JC, Powley CR, Kennedy GL (2006). "Comparative responses of rats and mice exposed to linear/branched, linear, or branched ammonium perfluorooctanoate (APFO)." *Toxicology*. 2006 Mar 15;220(2-3):203-17. doi: 10.1016/j.tox.2006.01.003. Epub 2006 Jan 31. PMID: 16448737.

McGuire MEC, Schaefer T, Richards WJ, Backe JA, Field EF, Houtz E, Sedlak DL, Guelfo JL, Wunsch A, Higgins CP (2014). "Evidence of remediation-induced alteration of subsurface poly-and perfluoroalkyl substance distribution at a former firefighter training area." *Environmental Science & Technology* 2014;48 (12):6644-6652. doi: 10.1021/es5006187.

MDHHS (2019). Michigan Department of Health and Human Services. Michigan Public health drinking water screening levels for PFAS Michigan Department of Health and Human Services, Division of Environmental Health Michigan PFAS Action Response Team Human Health Workgroup February 22, 2019
https://www.michigan.gov/documents/pfasresponse/MDHHS_Public_Health_Drinking_Water_Screening_Levels_for_PFAS_651683_7.pdf

MDH (2020 PFOA). Minnesota Department of Health. Toxicological Summary for: Perfluorooctanoate (PFOA). Accessed online December 2020.
<https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfoa.pdf>

MDH (2020 PFOS). Minnesota Department of Health. Toxicological Summary for Perfluorooctane sulfonate (PFOS). Accessed online December 2020.
<https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfos.pdf>

MDH (2020 PFHXS). Minnesota Department of Health. Toxicological Summary for: Perfluorohexane sulfonate (PFHxS). Accessed online December 2020.
<https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfhxs.pdf>

MDH (2020 PFBS). Minnesota Department of Health. Toxicological Summary for: Perfluorobutane Sulfonate (PFBS). Accessed online December 2020.
<https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfbssummary.pdf>

Moody CA, Field JA (1999). "Determination of perfluorocarboxylates in groundwater impacted by fire-fighting activity." *Environmental Science & Technology* 1999;33 (16):2800-2806. doi: 10.1021/es981355+.

Moody CA, Field JA (2000). "Perfluorinated surfactants and the environmental implications of their use in fire-fighting foams." *Environmental Science & Technology* 2000;34(18):3864-3870. doi: 10.1021/es991359u.

Moody CA, Hebert GN, Strauss SH, Field JA (2003). Occurrence and persistence of perfluorooctanesulfonate and other perfluorinated surfactants in groundwater at a fire-training area at Wurtsmith Air Force Base, Michigan, USA. *Journal of Environmental Monitoring*.

Murray KE, Thomas SM, Bodour AA (2010). "Prioritizing research for trace pollutants and emerging contaminants in the freshwater environment." *Environmental Pollution* 158 (12):3462-3471. doi: 10.1016/j.envpol.2010.08.009.

NRC (2009). National Research Council. *Science and Decisions: Advancing Risk Assessment*. Washington D.C.: National Academies Press.

NJDEP (2015). New Jersey DEP DWQI. Maximum contaminant level recommendation for perfluorononanoic acid in drinking water. Trenton, NJ: New Jersey Drinking Water Quality Institute. <https://www.nj.gov/dep/watersupply/pdf/pfoa-recommend.pdf>

NJDEP (2017). New Jersey DEP DWQI. Appendix A - Health-based Maximum Contaminant Level Support Document: Perfluorooctanoic Acid (PFOA). Trenton, NJ: New Jersey Drinking Water Quality Institute. <https://www.nj.gov/dep/watersupply/pdf/pfoa-appendixa.pdf>

NJDEP (2018). New Jersey DEP DWQI. Appendix A - Health-based Maximum Contaminant Level Support Document: Perfluorooctane sulfonate (PFOS). Trenton, NJ: New Jersey Drinking Water Quality Institute. <https://www.state.nj.us/dep/watersupply/pdf/pfos-recommendation-appendix-a.pdf>

NCDHHS (2017). North Carolina Department of Health and Human Services. Questions and Answers Regarding North Carolina Department of Health and Human Services Updated Risk Assessment for GenX (Perfluoro-2-propoxypropanoic acid). July 14, 2017. Accessed online Dec 2020. <https://files.nc.gov/ncdeq/GenX/DEQ-GenX%20FAQ%2008212017%201.pdf>

National Toxicology Program (NTP) (2018). TOX-96: Toxicity Report Tables and Curves for Short-term Studies: Perfluorinated Compounds: Sulfonates. Retrieved from https://tools.niehs.nih.gov/cebs3/views/?action=main.dataReview&bin_id=3874 .

Ohmori K, Kudo N, Katayama K, (2003). Comparison of the toxicokinetics between perfluorocarboxylic acids with different carbon chain length. *Toxicology* 2003;184:135-140.

Onishchenko N, Fischer C, Wan Ibrahim WN, Negri S, Spulber S, Cottica D, Ceccatelli S (2011) Prenatal exposure to PFOS or PFOA alters motor function in mice in a sex-related manner. *Neurotox Res*. 2011;19(3):452-61.

Pachkowski B, Post GB, Stern AH (2019). The derivation of a Reference Dose (RfD) for perfluorooctane sulfonate (PFOS) based on immune suppression. *Environ Res.* 2019;171:452- 469.

Paul AG, Jones KC, Sweetman AJ (2008). A first global production, emission, and environmental inventory for perfluorooctane sulfonate. *Environmental science & technology* 2008;43 (2):386-392.

Peden-Adams MM, Keller JM, EuDaly JM, Berger J, Gilkeson GS, Keil DE (2008). Suppression of Humoral Immunity in Mice following Exposure to Perfluorooctane Sulfonate. *Toxicol. Sci.* 2008:104(1); 144-154.

Prevedouros K, Cousins IT, Buck RC, Korzeniowski SH (2006). Sources, fate and transport of perfluorocarboxylates. *Environmental Science & Technology* 2006;40 (1):32-44. doi: 10.1021/es0512475.

Schultz M, Barofsky DF, Field JA (2004). Quantitative determination of fluorotelomer sulfonates in groundwater by LC MS/MS. *Environ Sci Technol.* 2004;38:1828-35. doi: 10.1021/es035031j.

Sundstrom M, Chang SC, Noker PE, Gorman GS, Hart JA, Ehresman DJ, Bergman A, Butenhoff JL. (2012). Comparative pharmacokinetics of perfluorohexanesulfonate (PFHxS) in rats, mice, and monkeys. *Reproductive Toxicology*, 33, 441-451.

Thompson J, Lorber M, Toms LML, Kato K, Calafat AM, Mueller JF (2010). Use of simple pharmacokinetic modeling to characterize exposure of Australians to perfluorooctanoic acid and perfluorooctane sulfonic acid. *Environment International* 36:390–397.

Thompson T, Ells S, Greenberg M, Leeson A, Magar V, Menzie C, Patton C, Pound M, Reible D, Rider D (2012). SERDP and ESTCP Workshop on Research and Development Needs for Long-Term Management of Contaminated Sediments. DTIC Document.

United States Environmental Protection Agency (2000). Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health. EPA-822-B-00-004. USEPA Office of Water, Washington, DC. Accessed online Dec 2020. <https://www.epa.gov/wqc/methodology-deriving-ambient-water-quality-criteria-protection-human-health-2000-documents>

United States Environmental Protection Agency (USEPA) (2002). *A Review of the Reference Dose and Reference Concentration Processes*. *Risk Assessment Forum*. EPA/630/P-02/002F, December 2002.

United States Environmental Protection Agency (USEPA) (2005). Guidelines for Carcinogen Risk Assessment. Report #EPA/630/P-03/001F.

United States Environmental Protection Agency (USEPA) (2011). Recommended Use of Body Weight^{3/4} as the Default Method in Derivation of the Oral Reference Dose. EPA/100/R11/0001. USEPA, Office of the Science Advisor, Risk Assessment Forum, Washington, DC. Accessed online Dec 2020.

<https://www.epa.gov/sites/production/files/2013-09/documents/recommended-use-of-bw34.pdf>

United States Environmental Protection Agency (USEPA) (2012). Benchmark Dose Technical Guidance. Report #EPA/100/R-12/001.

https://www.epa.gov/sites/production/files/2015-01/documents/benchmark_dose_guidance.pdf

United States Environmental Protection Agency (USEPA) (2018 PFBS). Human Health Toxicity Values for Perfluorobutane Sulfonic Acid (CASRN 375-73-5) and Related Compound Potassium Perfluorobutane Sulfonate (CASRN 29420-49-3). Public Comment Draft. EPA-823- R-18-307. USEPA, Office of Research and Development, Washington, DC. Accessed online Dec 2020.

https://www.epa.gov/sites/production/files/2018-11/documents/pfbs_public_comment_draft_toxicity_assessment_nov2018-508.pdf

United States Environmental Protection Agency (USEPA) (2018 GenX). Human Health Toxicity Values for Hexafluoropropylene Oxide (HFPO) Dimer Acid and Its Ammonium Salt (CASRN 13252-13-6 and CASRN 62037-80-3), Also Known as “GenX Chemicals”. Public Comment Draft. EPA-823- P-18-001. USEPA Office of Water, Washington, DC. Accessed online Dec 2020.

https://www.epa.gov/sites/production/files/2018-11/documents/genx_public_comment_draft_toxicity_assessment_nov2018-508.pdf

United States Environmental Protection Agency (USEPA) (2019) Exposure Factors Handbook Chapter 3 (Update): Ingestion of Water and Other Select Liquids.

EPA/600/R-18/259F. USEPA Office of Research and Development, Washington, DC. Accessed online Dec 2020. <https://www.epa.gov/expobox/about-exposure-factors-handbook>

Vassiliadou I, Costopoulou D, Ferderigou A, Leondiadis L (2010). Levels of perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA) in blood samples from different groups of adults living in Greece. *Chemosphere* 2010,80,1199–206.

Venkatesan AK, Halden RU (2013). National inventory of perfluoroalkyl substances in archived US biosolids from the 2001 EPA National Sewage Sludge Survey. *Journal of hazardous materials* 252:413-418.

Wambaugh JF, Setzer RW, Pitruzzello AM, (2013). “Dosimetric anchoring of in vivo and in vitro studies for perfluorooctanoate and perfluorooctanesulfonate.” *Toxicol Sci* 136(2):308-327.

Yeung LW, Guruge KS, Taniyasu S, Yamashita N, Angus PW, Herath CB (2013). "Profiles of perfluoroalkyl substances in the liver and serum of patients with liver cancer and cirrhosis in Australia." *Ecotoxicol Environ Saf* 2013 , 96 , 139 – 46

Zareitalabad P, Siemens J, Hamer M, Amelung W (2013). "Perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS) in surface waters, sediments, soils and wastewater - A review on concentrations and distribution coefficients." *Chemosphere* 91 (6):725-732. doi: 10.1016/j.chemosphere.2013.02.024.

Zhang Y, Beesoon S, Zhu L, Martin JW (2013). "Biomonitoring of perfluoroalkyl acids in human urine and estimates of biological half-life." *Environ Sci Technol* 47(18):10619-10627.

Appendix A: Drexel PFAS Advisory Group (DPAG)

Drexel PFAS Advisory Group (DPAG) adhered an evidence-based approach in developing its proposal. (Institute of Medicine (2011), NRC (2009)) The process was transparent and reviewed by PADEP at regular intervals. No member disclosed a conflict of interest. The panel was multidisciplinary and included a wide array of expertise. Literature and scientific evidence were reviewed with a systematic approach that rated the quality of the evidence, grade the strength of recommendations, incorporate values and preferences, and acknowledge differences in opinion. Recommendations were articulated in a structured framework repeatable across each PFA examined. They are now submitted for external review by DEP.

Project Leader and Medical Toxicologist:

- Richard J Hamilton MD FAAEM, FACEP, FACMT. Professor and Chair, Emergency Medicine, Drexel University College of Medicine. Board Certified in Medical Toxicology by the American Board of Emergency Medicine and is a Fellow of the American College of Medical Toxicology.

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- David Vearrier MD FAAEM, FACMT, FAACT Professor of Emergency Medicine, Drexel University College of Medicine. Board Certified in Medical Toxicology by the American Board of Emergency Medicine and is a Fellow of the American College of Medical Toxicology and a Fellow of the American Academy of Clinical Toxicology.
- Rita McKeever MD FAAEM, FACMT, Associate Professor of Emergency Medicine, Drexel University College of Medicine. Board Certified in Medical

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Expert Panel:

- Charles N Haas Ph.D - LD Betz Professor of Environmental Engineering & Head, Dept. of Civil, Architectural & Environmental Engineering, Drexel University
- Christopher Sales Ph.D. Assistant Professor, Architectural & Environmental Engineering, Drexel University
- Marie Kurtz PhD, Senior Scientist; Assistant Research Professor, Academy of Natural Sciences, Drexel University
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- Tom Hipper, MSPH, MA Adjunct Professor, Program Manager of the Center for Public Health Readiness and Communication Dornsife School of Public Health, Drexel University

Appendix B: Acronyms and Abbreviations List

<p>ATSDR: Agency for Toxic Substances and Disease Registry BMD: benchmark dose BMDL: lower confidence limit on the benchmark dose BMR: benchmark response BW: body weight Bwa: body weight animal BWh: body weight human CDC: Centers for Disease Control and Prevention CEPA: California Environmental Protection Agency DPAG: Drexel PFAS Advisory Group DAF: dosimetric adjustment factor GD: gestational day GenX: ammonium salt of hexafluoropropylene oxide dimer HBV: health-based value HED: human equivalent dose HED_{LOAEL}: HED determined by LOAEL HED_{BMDL10}: HED determined by a BMR of 10% HED_{BMDL0.5SD}: HED determined by a BMR of 50% of SD HFPO: hexafluoropropylene oxide HRA: health risk assessment THSV = Internal Target Human Serum Value kg: kilogram L: liter LD: lactation day LHA: lifetime health advisory LOAEL: lowest observed adverse effect level MCL: Maximum Contaminant Level MDH: Minnesota Department of Health MDHHS: Michigan Department of Health and Human Services mg: milligram mg/kg/d: milligrams per kilogram per day MI: Michigan ml: milliliter MPART: Michigan PFAS Action Response Team</p>	<p>NCDHHS: North Carolina Department of Health and Human Services NHDES: New Hampshire Department of Environmental Services NHANES: National Health and Nutrition Examination Survey NJDEP: New Jersey Department of Environmental Protection ng: nanogram NOAEL: no observed adverse effect level OECD: Organization for Economic Co-operation and Development PA DEP: Pennsylvania Department of Environmental Protection PFAS: per- and polyfluoroalkyl substances PFBS: perfluorobutane sulfonic acid PFHpA : perfluoroheptanoic acid PFHxA: perfluorohexanoic acid PFHxS: perfluorohexane sulfonic acid PFNA: perfluorononanoic acid PFOA: perfluorooctanoic acid PFOS: perfluorooctane sulfonic acid PND: postnatal day POD: point of departure PODHED: point of departure human equivalent dose PPAR: peroxisome proliferator-activated receptor ppt: parts per trillion RfD: reference dose RSC: relative source contribution TWA: time weighted average UF: uncertainty factor µg: microgram USEPA: United States Environmental Protection Agency</p>
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Attachment 3

PENNSYLVANIA DEPARTMENT OF ENVIRONMENTAL PROTECTION
EVALUATION REPORT
ON THE
DELAWARE RIVERKEEPER NETWORK PETITION FOR
RULEMAKING
TO SET AN MCL FOR PFOA

April 16, 2021

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A. DESCRIPTION OF THE PETITION FOR RULEMAKING PROCEDURE

Any person may petition the Environmental Quality Board (“EQB”) to initiate a rulemaking proceeding for the issuance, amendment, or repeal of a regulation administered and enforced by the Pennsylvania Department of Environmental Protection (“Department”). 71 P.S. § 510-20(h). The EQB has developed a policy for processing petitions for rulemaking. *See* 25 Pa. Code Chapter 23 (relating to Policy for Processing Petitions – Statement of Policy). Among other things, a petition for rulemaking must contain the following information: (1) the petitioner’s name, address, and telephone number; (2) a description of the action requested including suggested regulatory language if the petition requests the EQB to adopt or amend regulations; (3) the reason the petitioner is requesting the action from the EQB; and (4) the types of persons, businesses, and organizations likely to be impacted by the proposal. 25 Pa. Code § 23.1 (relating to Petitions).

When a petition for rulemaking is submitted, the Department examines the petition before it is submitted to the EQB to determine if it meets the following conditions: (1) the petition is complete as required by § 23.1; (2) the petition requests an action that can be taken by the EQB; and (3) the requested action does not conflict with Federal law. 25 Pa. Code § 23.2 (relating to Departmental review).

The Department then notifies the EQB and the petitioner of its determination. If the Department determines that the petition is not appropriate, the notification will state why and give the petitioner 30 days to modify the request. 25 Pa. Code § 23.3 (relating to Notification).

Where the Department determines that a petition is appropriate, the petitioner may make a five-minute presentation to the EQB and the Department will also make a recommendation as to whether to accept the petition. 25 Pa. Code § 23.4 (relating to Oral presentation).

The EQB may refuse to accept a petition if: (1) the EQB has within the past two years considered the issue addressed in the petition; (2) the action requested by the petitioner is currently under litigation; (3) the requested action is inappropriate for policy or regulatory considerations; or (4) the petition involves an issue previously considered by the EQB, and it does not contain information that is new or sufficiently different to warrant reconsideration of that issue. 25 Pa. Code § 23.5 (relating to Board determination).

If the EQB accepts the petition, a notice of acceptance will be published in the *Pennsylvania Bulletin* and a report will be prepared. 25 Pa. Code § 23.6 (relating to Notice of acceptance and Department report).

Once the report is completed, the Department will send a copy of it to the petitioner who may then submit to the Department a written response to the report within 30 days of the mailing of the report. 25 Pa. Code § 23.7 (relating to Response to report).

The Department will prepare a recommendation to the EQB based on the report and comments received from the petitioner. If regulatory amendments are recommended, the Department will develop a proposed rulemaking for EQB consideration within 6 months after the Department mailed its report to the petitioner. If regulatory amendments are not recommended, the Department will present its recommendation and basis to the EQB at the first meeting occurring at least 45 days after the Department mailed its report to the petitioner. 25 Pa. Code § 23.8 (relating to Board consideration).

B. DESCRIPTION OF THE DELAWARE RIVERKEEPER NETWORK PETITION

1. Procedural Description

On May 8, 2017, the EQB received a petition to promulgate a rule to set a drinking water maximum contaminant level (MCL) for perfluorooctanoic acid (PFOA) not to exceed 6 parts per trillion (ppt or nanograms per liter (ng/L)).

The petition was submitted by Tracy Carluccio, Deputy Director on behalf of the Delaware Riverkeeper Network (DRN), 925 Canal Street, Suite 3701, Bristol, PA 19007.

On June 22, 2017, the Department sent a letter to Ms. Carluccio that notified DRN that the petition met the established criteria in Section 23.2 of the EQB's petition policy. The letter also set August 15, 2017 as the date the EQB would consider the petition.

At the August 15, 2017 EQB meeting, Ms. Carluccio, on behalf of DRN, made a brief presentation as to why the EQB should accept the petition for further study. The Department recommended that the EQB accept the petition for further study. The EQB voted unanimously to accept the petition for further study.

On August 26, 2017, the Department published a notice of acceptance of the petition in the *Pennsylvania Bulletin*. See 47 Pa.B. 4986 (August 26, 2017).

2. Petition Description

The petition asserts that the EQB should promulgate a rule “to set an MCL for PFOA not to exceed 6 ppt.” In support of this petition, Ms. Carluccio, on behalf of DRN, cites PFOA monitoring data from the U.S. Environmental Protection Agency's (EPA) Unregulated Contaminant Monitoring Rule 3 (UCMR 3), 77 FR 26072 (May 2, 2012), information and data from several contamination sites in Bucks and Montgomery counties and other sites across the

state, and scientific studies and reports to support the conclusions that PFOA is in many public water systems in Pennsylvania, that the EPA's Health Advisory Level (HAL) of 70 ppt is ineffective at protecting public health, and that a more protective standard not to exceed 6 ppt should be set for PFOA to protect Pennsylvania citizens. *See* Petition, p. 15. *Please Note: No suggested regulatory language was provided by DRN.*

C. DEPARTMENT RESPONSE TO THE PETITION

1. PFOA

PFOA is a man-made chemical in a large family of chemicals called per- and poly-fluoroalkyl substances (PFAS), which are used to make products more resistant to stains, grease, and water. Major U.S. manufacturers voluntarily agreed to phase out production of PFOA by the end of 2015. However, exposure remains possible due to its widespread use and legacy in the environment from former manufacturing sites and sites where PFOA was used. PFOA has been found in both groundwater and surface water in Pennsylvania and across the country. PFOA is a concern because it readily dissolves in water, bioaccumulates, and is persistent in the environment.

The Department became aware of PFOA detections in public water systems as a result of EPA's UCMR 3 rule. The Federal Safe Drinking Water Act (Federal SDWA) requires EPA to establish criteria for a program to monitor not more than 30 unregulated contaminants every 5 years. The purpose of the rule is to gather occurrence data and refine analytical methods in order to inform a regulatory determination. Monitoring for 28 chemicals and two viruses was conducted by select public water systems (those serving greater than 10,000 people and a random selection of smaller systems) from January 2013 through December 2015. This included 175 public water systems in Pennsylvania. The UCMR rules are direct implementation rules with EPA as the lead agency and states providing assistance. Six (6) out of 175 public water systems had detections for PFOA:

- Warminster Municipal Authority
- Warrington Township Water & Sewer Department
- Horsham Water & Sewer Authority
- United Water -- Harrisburg (now Suez)

- Doylestown Township Municipal Authority
- Aqua PA – Bristol

2. Status of an MCL for PFOA

The Department is authorized to administer and enforce environmental regulations under the Pennsylvania Safe Drinking Water Act (Pennsylvania SDWA), 35 P.S. § 721.5. The EQB is authorized to adopt such rules and regulations, governing the provision of drinking water to the public, as it deems necessary for the implementation of the Pennsylvania SDWA, 35 P.S. § 721.4. Under the SDWA, an MCL is defined as the maximum permissible level of a contaminant in water which is delivered to any user of a public water system.

The Federal SDWA authorizes EPA to set national health-based standards to protect against contaminants that may be found in drinking water, 42 U.S.C. § 300g-1. Under the Federal SDWA, EPA promulgates primary MCLs, which are enforceable standards. EPA may also publish health advisories, which are non-enforceable and non-regulatory, for contaminants not subject to any national primary drinking water regulation. The Federal SDWA grants States primary enforcement responsibility (primacy) for public water systems when EPA determines that a State meets certain requirements, including adopting drinking water regulations that are no less stringent than the national primary drinking water regulations promulgated by EPA, 42 U.S.C. § 300g-2.

The Pennsylvania SDWA was enacted in 1984. The Pennsylvania SDWA imposed a mandatory duty upon the Department to adopt a public water supply program that includes certain program elements necessary to assume primacy under the Federal SDWA, including MCLs. The Department established a public water supply program that met the criteria and was granted primacy by EPA on November 30, 1984. 50 FR 342 (January 3, 1985).

The Pennsylvania SDWA provides direction regarding how MCLs are to be developed, 35 P.S. § 721.4(a). Under the Pennsylvania SDWA, the EQB *shall* adopt MCLs no less stringent than those promulgated under the Federal SDWA for all contaminants regulated under the national primary drinking water regulations. In addition, the EQB *may* adopt MCLs for any contaminant that an MCL has not been promulgated. EPA has not promulgated an MCL for PFOA under the national primary drinking water regulations. EPA has published a health advisory for PFOA, which established a combined lifetime HAL of 70 ppt for PFOA and perfluorooctanesulfonic acid (PFOS). 81 FR 33250 (May 25, 2016).

As referenced above, the Petition for Rulemaking was presented at the August 15, 2017 EQB meeting, at which the Department recommended that the EQB accept the petition for further evaluation to help inform whether additional measures are needed to protect public health. During the meeting, the Department stated that it had never in its history set an MCL and would require toxicology expertise to evaluate the rulemaking petition and prepare the report. It was expected that this would require independent work, research, and review. The Department provided updates to the EQB on June 19, 2018 and June 18, 2019, where the Department expressed the need for more time and provided a summary of the challenges and actions taken to secure the necessary expertise to evaluate the rulemaking petition and prepare this report. These and other actions taken by the Department to address PFOA are described below in Section 3.

3. Department actions to address PFOA

a. Actions to implement EPA's HAL as an interim measure

Following EPA's publication in May 2016 of the final HAL of 70 ppt for the combined concentration of PFOA and PFOS, the Department developed its strategy in July 2016 for

addressing PFOA and PFOS levels in public water systems that exceed the HAL. The Department's strategy is based on existing authority and long-standing policies and procedures for implementing HALs. The Department's authority to address unregulated contaminants includes the following:

- Pennsylvania SDWA, Section 10. Emergencies and imminent hazards.

(b) Department may order temporary emergency actions.—The department, upon receipt of information that a contaminant which is present in or is likely to enter a public water system may present an imminent and substantial risk to the health of persons, may take or order a public water system to take such temporary emergency actions as it deems necessary in order to protect the health of such persons. The department may assess the responsible water supplier with costs of temporary actions taken by the department, except where such action is in the normal course of its duties.

(c) Department may implement emergency measures.—The department shall be authorized to implement whatever measures may be necessary and appropriate to notify the public of an emergency or imminent hazard and to assess costs of notification on the responsible water supplier.

- Title 25 Pa. Code § 109.4. General requirements.

Public water suppliers shall:

- (1) Protect the water sources under the supplier's control.*
- (2) Provide treatment adequate to assure that the public health is protected.*
- (3) Provide and effectively operate and maintain public water system facilities.*

(4) Take whatever investigative or corrective action is necessary to assure that safe and potable water is continuously supplied to the users.

- Title 25 Pa. Code § 109.302. Special monitoring requirements.

(b) The Department may require a public water supplier to conduct additional monitoring to provide information on contamination of the water supply where a potential health hazard may exist in the water supply and monitoring required under § 109.301 may not be adequate to protect the public health.

(c) The Department may require a public water supplier to conduct special monitoring for an unregulated contaminant if the Department has reason to believe the contaminant is present in the public water system and creates a health risk to the users of the public water system.

The Department's long-standing risk management strategy for unregulated contaminants can be found in the following guidance: *Health Effects and Risk Management Guidance* ([383-0400-104](#)).

As per the guidance and long-standing protocols, when levels exceed a lifetime HAL, a Tier 2 situation has occurred. Water supplier follow-up actions may include:

- One-hour reporting of sample results to the Department (25 Pa. Code § 109.701(a)(3)) to ensure the Department is immediately alerted to the situation and can provide the necessary oversight regarding investigative and corrective actions
- Collection of confirmation samples (25 Pa. Code § 109.302(c))
- Issuance of Tier 2 Public Notification (PN) within 30 days of receipt of sample results exceeding the HAL (25 Pa. Code § 109.409)
- Quarterly monitoring at each entry point (EP) to the distribution system that exceeded the HAL (25 Pa. Code § 109.302(d)) to continue to track contaminant levels

- If levels continue to exceed the HAL, additional actions may be needed to reduce levels to below the HAL (taking contaminated sources off-line, blending, installing treatment, etc.) (25 Pa. Code § 109.4)

Taken together, these actions implemented EPA's HAL prior to submission of the petition, and served as an interim measure while the Department evaluated whether the HAL is sufficiently protective.

b. Toxicology services contract

At the time of submission of the petition, neither the Department nor the Pennsylvania Department of Health (DOH) employed a full-time toxicologist. The DOH had access to a retired toxicologist on a very limited basis (90 days per year) as an annuitant. The DOH recognized the need to hire one or more full time toxicologists and initiated the hiring process in late 2017. The DOH began interviewing candidates in January of 2018, but had difficulty filling the position for various reasons. The DOH was finally able to fill the toxicologist position in July of 2019.

While the DOH was working to fill the toxicologist position, the Department moved forward in early 2019 with plans to secure additional toxicology resources to assist in evaluating the petition. The Department developed a scope of work and began soliciting interest in a toxicology services contract in May of 2019. The Department reviewed the submitted quotes for services in July of 2019 and awarded the contract to Drexel University. The contract was finalized and executed in December of 2019. The contract was for a one-year period and included: (1) a review and analysis of work by other states and federal agencies that had developed PFAS action levels and MCLs; and (2) an independent review of the data, science, and studies, and development of recommended maximum contaminant level goals (MCLG) for select PFAS. MCLGs are non-

enforceable as they are developed solely based on health effects and do not take into consideration other factors, such as limitations with analytical methods and available treatment technologies and cost. MCLGs are the starting point for determining MCLs. Please refer to Section D.2. for more information about MCLGs and the process to set MCLs.

The scope of work included the review of several PFAS in addition to PFOA to provide the Department with more complete health effects information for additional PFAS of concern, to better position the Department to address co-occurring PFAS, to align with state sampling efforts, and to create efficiencies in evaluating multiple PFAS simultaneously. The additional PFAS include PFOS, perfluorobutane sulfonic acid (PFBS), perfluorononanoic acid (PFNA), perfluorohexanesulfonic acid (PFHxS), and perfluoroheptanoic acid (PFHpA). The contract continued throughout 2020, with Drexel providing updates to Department and DOH staff every few months. The project experienced some delays due to the COVID-19 pandemic. The project deliverables were completed and submitted to the Department at the end of January 2021. The deliverables include the “Drexel PFAS Workbook”, which contains the review and analysis of work by other states and federal agencies, and the “MCLG Drinking Water Recommendations for PFAS in the Commonwealth of Pennsylvania” report. These documents are included in the Appendix to this report. Here is a brief summary of Drexel’s report.

Drexel’s MCLG Drinking Water Recommendations for PFAS Report: The report was developed by the Drexel PFAS Advisory Group (DPAG), which is a unique multidisciplinary team consisting of experts in the fields of medical toxicology, epidemiology, environmental toxicology, drinking water standards, and risk assessment. The DPAG evaluated existing and proposed standards from across the country. The DPAG was also charged with developing recommended

MCLGs. In order to do this, the DPAG reviewed the pertinent literature and work done across the country, and independently developed recommended MCLGs.

As mentioned previously and as further discussed in the report, MCLGs are non-enforceable as they are developed solely based on health effects and do not take into consideration other factors, such as limitations with analytical methods and available treatment technologies and cost. MCLGs are the starting point for determining MCLs. The DPAG's recommended MCLG for PFOA is 8 ppt. The DPAG conducted a literature search and review of the available evidence and recommendations from various agencies and developed an MCLG recommendation based on Non-Cancer endpoints. The report includes a discussion of the relevant inputs. The DPAG selected Koskela (2016) and Onishchenko (2011) as the critical studies. Table 1 below represents DPAG's development of the Non-Cancer MCLG for PFOA.

Table 1. The Drexel PFAS Advisory Group’s development of the Non-Cancer MCLG for PFOA

PFOA	
Dose Response Modeling Method	LOAEL
POD	The average serum concentration was estimated in the mice (8.29 mg/L) using a three-compartment pharmacokinetic model (Wambaugh et al. 2013) using animal species, strain, sex-specific parameters. (ATSDR 2018)
HED = POD x DAF (mg/kg/d)	DAF = Ke x Vd Ke = 0.000825175 (8.2 x 10 ⁻⁴) based on a human serum half-life of 840 days (Bartell et al. 2010) Vd = 0.17 L/kg (Thompson et al. 2010) HED _{LOAEL} = POD _{LOAEL} x DAF HED _{LOAEL} = POD _{LOAEL} x Ke x Vd HED _{LOAEL} = 8.29 mg/L x 0.000825175 x 0.17 L/kg HED _{LOAEL} = 0.001163 mg/kg/d or 1.163 x 10 ⁻³ mg/kg/d
Uncertainty Extrapolation	
Human Variability (UFH)	10 (standard)
Animal to Human (UFA)	3 (DAF applied)
Subchronic to Chronic (UFS)	1 (Chronic effect studied)
LOAEL to NOAEL (UFL)	10 (standard)
Database (UFD)	1
Total Composite (UFT)	300
RfD = HED/UFT (mg/kg/d)	RfD = 0.001163 mg/kg/d/300 RfD = 3.9 ng/kg/day (3.9 x 10 ⁻⁶ mg/kg/d)
THSV = POD / UFT	THSV = 8.29 mg/L / 300 THSV = 0.028 mg/L
Receptor	Infant exposure via breastmilk for 1 year, from mother chronically exposed via water, followed by lifetime of exposure via drinking water. Protective for short-term, subchronic and chronic. (also protective of formula fed infant). Goeden Model Parameters: Placental transfer of 87% and breastmilk transfer of 5.2% (MDH (2020 PFOA)). The Human Serum half-life is set at 840 days (Bartell et al. 2010). The Volume of distribution of 0.17 L/kg (Thompson et al. [2010]) Other factors include, 95th percentile drinking water intake, consumers only, from birth to more than 21 years old. Upper percentile (mean plus two standard deviations) breast milk intake rate. Time-weighted average water ingestion rate from birth to 30-35 years of age is used to calculate maternal serum concentration at delivery. (Goeden et al. [2019]) A Relative Source Contribution of 50% (0.5) is applied and based on studies which showed that infants RSC is similar to NHANES 95th percentiles for 3-11 (2013-2014) and over 12 years old (2015-2016) participants. (CDC 2019)
Chronic Non-Cancer MCLG	The model produces a Chronic Non-Cancer MCLG of 8 ng/L (ppt). This protects health during the growth and development of a breast fed infant. Figure 2

c. PFAS sampling plan

During this same time period, the Department announced it would begin sampling for PFAS at public water systems across the state. The PFAS Sampling Plan was developed in early 2019 and the final plan was posted to the Department's [PFAS webpage](#) in April of 2019.

The PFAS Sampling Plan is intended to prioritize sites for PFAS sampling and generate statewide occurrence data. Several factors were considered in developing the plan including:

- Location of potential sources of PFAS contamination (PSOC)
- Known locations of PFAS contamination
- Relative risk to users of nearby public water system sources of drinking water
- Selection of public water system sources to serve as a control group
- Available funds - \$500,000

The selection process involved a combination of spatial analysis and programmatic review. The spatial analysis included the creation of a Geographic Information System (GIS) project using ArcMap 10.4.1 that focused on public water system source locations and information about PSOCs. The sampling pool was prioritized based on relative risk and included community water systems and nontransient noncommunity water systems.

In order to prioritize sampling, the selection process included an assessment of the potential risk from nearby PSOCs. Several layers containing locational and other information specific to PSOCs were created or otherwise included in the GIS. These layers include the following industries and land uses:

- Military bases
- Fire training schools/sites
- Airports
- Landfills
- HSCA sites
- Superfund sites

- Manufacturing facilities:
 - Apparel and other products made from fabrics
 - Chemicals
 - Electronic and electrical equipment
 - Fabricated metal products
 - Paper products
 - Plastic products
 - Textile and leather products
 - Upholstered furniture

Based on the compilation of PSOCs, the information was used to select public water system sources that are located within ½ mile of a PSOC. The targeted sample pool included approximately 493 public water system sources. A second query was performed to identify baseline sources to serve as a control group. Baseline sources are located in a HUC-12 watershed (a watershed assigned a 12-digit [hydrologic unit code](#), or HUC, by the U.S. Geological Survey) with at least 75% forested land and at least five miles from a PSOC. Figure 1 is a map of the pool of public water system sources for sampling.

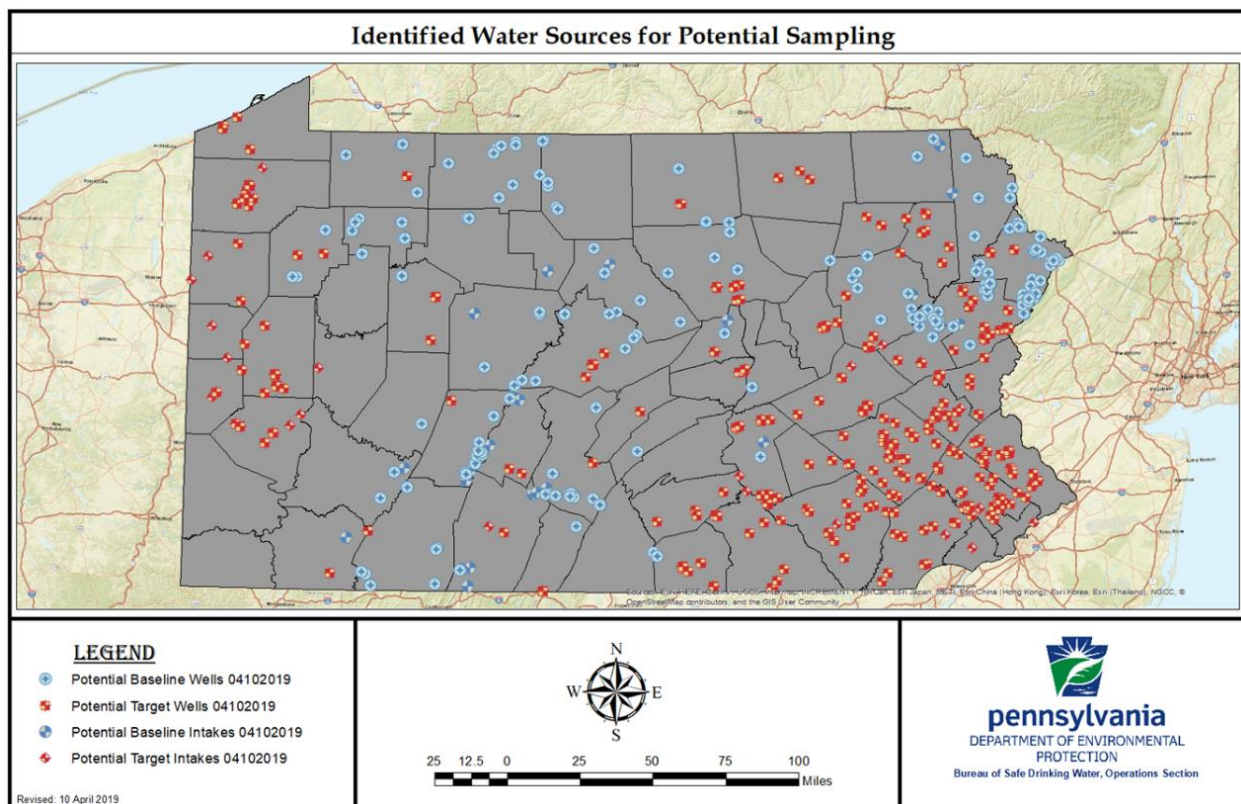


Figure 1. Public water system sources identified for sampling.

The Sampling Plan also includes maps of the various GIS data layers of PSOCs. Figure 2 is an example of the map of industrial sites.

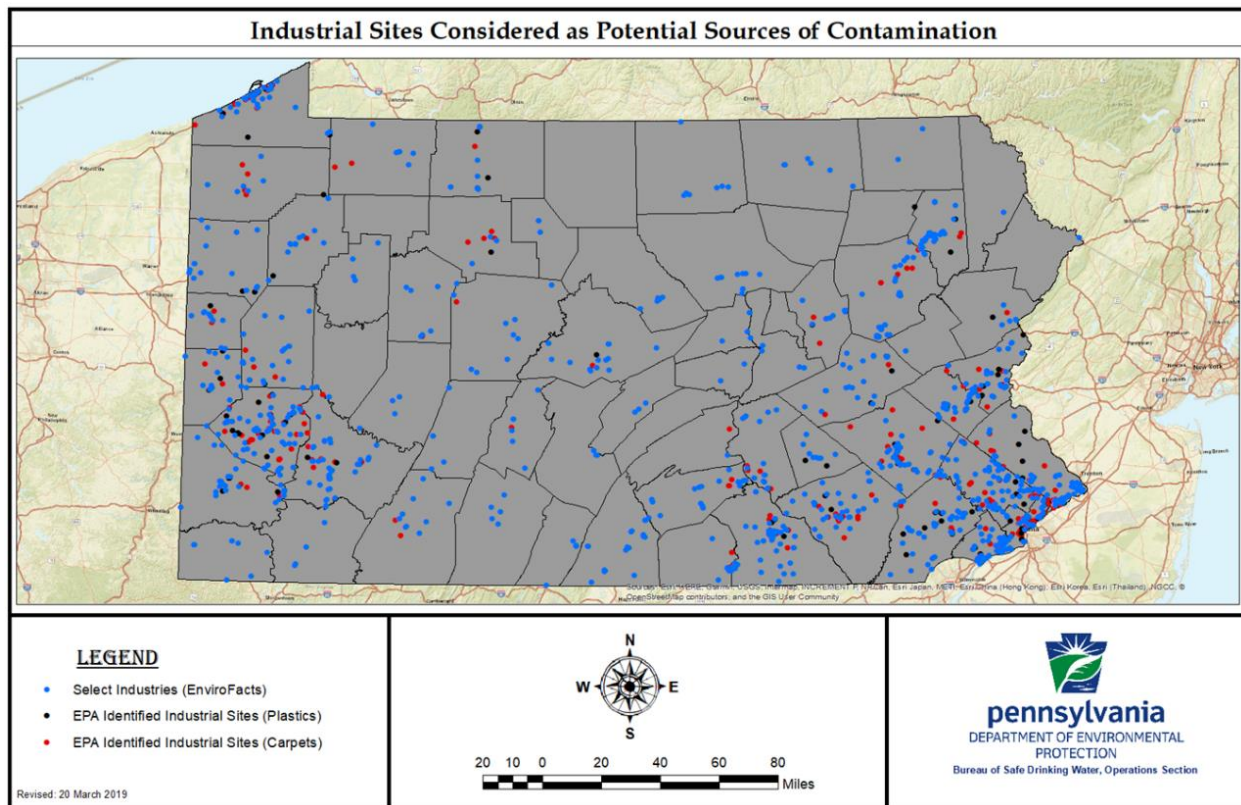


Figure 2. Potential sources of PFAS contamination (PSOC).

The final plan included the collection of samples from 360 targeted public water system sources and 40 baseline sources for a total of 400 samples. Sampling began in June of 2019 and included analysis of six (6) PFAS (PFOS, PFOA, PFNA, PFHxS, PFHpA, and PFBS) to be consistent with EPA’s UCMR 3. However, the Department had the opportunity in 2020 to expand the sampling to 18 PFAS by using EPA Method 537.1. Sampling was repeated for the public water systems that were sampled in 2019, and sampling continued for the remainder of the water systems throughout 2020. Note that sampling was halted in March of 2020 due to the pandemic and stay-at-home orders. Sampling resumed in August of 2020 under an approved return to work plan with

appropriate health and safety measures. The first release of 2020 sample results was posted to the Department’s PFAS webpage on March 12, 2021 and included 114 samples collected from February through September 2020. Here is the link: [Statewide Sampling Plan 2020 Results](#).

Sampling was completed by the end of March 2021. However, results for approximately 20 samples are still pending, and the review of quality assurance data for other recently reported results is ongoing. Table 2 presents a brief summary of the PFOA sample results to date (Note: The Department anticipates that all results will be received and confirmed in time to include a complete summary of PFOA samples in the final report presented to the EQB):

Table 2. Summary of PFOA sample results to date

	PFOA	Units
Average	3.2	ng/l
Median	ND	ng/l
Minimum	ND	ng/l
Maximum	59.6	ng/l
# Detects		
	40	
Average Detect Value	9.0	ng/l
Median Detect Value	6.5	ng/l
Min Detect Value	4.0	ng/l
Max Detect Value	59.6	ng/l

d. BOL PFAS analytical capabilities

The Department’s Bureau of Laboratories (BOL) also worked to purchase and install lab equipment to conduct PFAS testing. BOL was able to achieve proficiency for EPA Method 537.1 and received accreditation from New Jersey in December of 2019. BOL was instrumental in assisting with completing the work under the PFAS Sampling Plan.

D. DEPARTMENT ANALYSIS OF THE PETITION FOR RULEMAKING

1. The Petition Contends that an MCL should be set for PFOA not to exceed 6 ppt

DRN contends that EPA's HAL of 70 ppt has been shown to be ineffective at protecting the public health. Petition p. 2. DRN references two studies and reports to support this: the New Jersey Drinking Water Quality Institute (NJDWQI) report and the Cambridge Environmental Consulting (CEC) study. Petition p. 15.

According to DRN, the NJDWQI transmitted to the New Jersey Department of Environmental Protection its recommendation of an MCL for PFOA of 14 ppt. And while DRN referenced the NJDWQI work as supportive of its conclusion, it also stated that NJDWQI's recommendation may not be protective enough.

DRN also referenced a report prepared by CEC of an evaluation of the NJDWQI work. The CEC study disagreed with several of NJDWQI's findings and concluded that the proposed drinking water MCL for PFOA of 14 ppt is not adequately protective of all population segments. Instead, the CEC study recommended that the proposed MCL for PFOA should be lowered to 1 ppt, or alternatively, should be no higher than 6 ppt. Petition p. 19.

2. Recommendation

The Petition for Rulemaking recommends that the EQB should promulgate a rule to set an MCL for PFOA not to exceed 6 ppt. Petition p. 18. However, DRN fails to recognize the process that the Department must follow when setting an MCL. Specifically, the Department must consider other factors in addition to health effects when proposing an MCL as required by the Federal SDWA and Pennsylvania's Regulatory Review Act (RRA), 71 P.S. §§ 745.1—745.15.

Among other things, the Department must consider technical limitations such as available analytical methods and detection and reporting limits, treatability of the contaminant and available treatment technologies, and costs. 71 P.S. § 745.5b.

In addition to state requirements, the Department needs to consult the Federal SDWA and its implementing regulations. *See* 42 U.S.C. §§ 300f—300j-9; *see also* 40 CFR Parts 141, 142, and 143. For example, within the definitions in the Federal SDWA:

- “MCLG” means the maximum level of a contaminant in drinking water at which no known or anticipated adverse effect on the health of persons served would occur, and which allows an adequate margin of safety. MCLGs are non-enforceable health goals.
- “MCL” means the maximum permissible level of a contaminant in water which is delivered to any user of a public water system.

EPA further explains the difference between MCLGs and MCLs and how the agency sets standards at the following link: www.epa.gov/sdwa/how-epa-regulates-drinking-water-contaminants. In establishing an MCL, the Department would also be informed by EPA’s procedure to establish an MCL as detailed below. It is important to understand the process of setting an MCL because similar criteria are required of the Department under the RRA. In addition, in order to retain primacy, the Department’s standard setting process would need to be as stringent as the federal process.

After reviewing health effects data, EPA sets an MCLG. MCLGs are non-enforceable public health goals. MCLGs consider only public health and not the limits of detection and treatment technology effectiveness. Therefore, MCLGs sometimes are set at levels which water systems cannot meet because of technological limitations.

Once the MCLG is determined, EPA sets an enforceable standard. **In most cases, the standard is an MCL. The MCL is set as close to the MCLG as feasible.** Taking cost into consideration, EPA must determine the feasible MCL. This is defined by the Federal SDWA as the level that may be achieved with:

- use of the best available technology or treatment approaches
- other means which EPA finds are available (after examination for efficiency under field conditions, not solely under laboratory conditions)

As a part of the rule analysis, the Federal SDWA also requires EPA to prepare a health risk reduction and cost analysis in support of any standard. EPA must analyze the quantifiable and non-quantifiable benefits that are likely to occur as the result of compliance with the proposed standard. EPA must also analyze certain increased costs that will result from the proposed drinking water standard. In addition, EPA must consider:

- Incremental costs and benefits associated with the proposed and alternative MCL values
- The contaminant's adverse health effects on the general population and sensitive subpopulations
- Any increased health risk to the general population that may occur as a result of the new MCL
- Other relevant factors such as data quality and the nature of the risks

Where the benefits of a new MCL do not justify the costs, EPA may adjust the MCL for a particular class or group of systems to a level that maximizes health risk reduction benefits at a cost that is justified by the benefits.

The setting of an MCL is not as simple as just picking a number. MCL rules must include the necessary provisions to define applicability, the means to comply, and how compliance will be determined. For example, which water systems must comply with the MCL, what are the approved analytical methods, which treatment technologies are approved, how will systems monitor for the contaminant, and how will compliance be determined? All of these details are missing from the Petition for Rulemaking, so it is unclear how the recommended MCL would apply or be implemented.

In analyzing the Petition for Rulemaking, the Department has determined that DRN did not consider all of the relevant factors when recommending the MCL for PFOA not to exceed 6 ppt. As a result, it is recommended that the number advocated for in the Petition for Rulemaking not be the basis for a proposed rulemaking to establish an MCL for PFOA.

E. CONCLUSION

The Department has implemented a number of actions to address PFOA and protect public health. As a result of the work done by Drexel University on behalf of the Department and the occurrence data generated from the PFAS Sampling Plan, the Department believes that additional measures are needed to further protect the public. However, DRN did not include all of the relevant factors that the Department must consider when proposing an MCL. As a result, it is recommended that the number advocated for in the Petition for Rulemaking not be the basis for a proposed rulemaking to establish an MCL for PFOA. While the Department agrees that it should move forward with a proposed rulemaking to set an MCL for PFOA, it does not believe that DRN's proposed MCL was developed appropriately. The Department's proposed rulemaking should be based on available data, studies, and science, and should consider all factors such as health effects, technical limitations, and cost as required under the Federal SDWA and RRA. As a result, the Department recommends that the EQB move forward with a proposed rulemaking to establish an MCL for PFOA. The Department anticipates that it will have a proposed rulemaking developed by the fourth quarter of 2021.

F. APPENDIX

1. Maximum Contaminant Level Goal Drinking Water Recommendations for Per- and Polyfluoroalkyl Substances (PFAS) in the Commonwealth of Pennsylvania, The Drexel PFAS Advisory Board, January 2021.
2. Drexel PFAS Workbook, June 2020.

Attachment 4

Summary of Results for SDW Sampling Project Using EPA Method 537.1

Category	PWSID	EPID	BOL Sample # / ELLE Job #	PWS Name	County	Date Collected	11CI-PF3OUds	9CI-PF3ONS	ADONA	HFPO-DA	NETFOSAA	NMeFOSAA	PFDA	PFDoA	PFTA	PFTrDA	PFHxA	PFUnA	PFBS	PFHpA	PFHxS	PFNA	PFOS	PFOA	Sum_2 (PFOS + PFOA)	Units	
							11-chloroheptafluoro-3-oxaundecane-1-sulfonic acid	9-chlorohexadecafluoro-3-oxanone-1-fulfonic acid	4,8-dioxa-3H-perfluorononanoic acid	Hexafluoropropylene oxide dimer acid	N-ethyl perfluorooctanesulfonamidoacetic acid	N-methyl perfluorooctanesulfonamidoacetic acid	Perfluorododecanoic acid	Perfluorodecanoic acid	Perfluorotetradecanoic acid	Perfluorotridecanoic acid	Perfluoroheptanoic acid	Perfluoroheptanoic acid	Perfluorobutane sulfonic acid	Perfluorohexanoic acid	Perfluorohexane sulfonic acid	Perfluorononanoic acid	Perfluorooctane sulfonic acid	Perfluorooctanoic acid			
							763051-92-9	756426-58-1	919005-14-4	13252-13-6	2991-50-6	2355-31-9	335-76-2	307-55-1	376-06-7	72629-94-8	307-24-4	2058-94-8	375-73-5	375-85-9	355-46-4	375-95-1	1763-23-1	335-67-1			
TW	1460022	109	0477528	Collegeville Trappe Joint PWD	Montgomery	1/12/2021	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	7.4	12.8	20.2	ng/l	
TI	1460023	101	0477300	East Greenville Boro Water Dept	Montgomery	9/29/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l
TW	1460023	102	0477312	East Greenville Boro Water Dept	Montgomery	9/29/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	6.5	ND	ND	ND	ND	5.1	5.7	8.5	14.2	ng/l	
TW	1460034	113	410-19523-1	North Penn Water Authority	Montgomery	11/4/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.7	ND	4.8	2.0	2.0	ND	5.2	6.4	11.6	ng/l		
TW	1460034	125	410-19523-1	North Penn Water Authority	Montgomery	11/4/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	12.0	ND	3.9	13.0	3.1	3.4	11.0	25.0	36.0	ng/l		
TW	1460034	171	410-19523-1	North Penn Water Authority	Montgomery	11/4/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	13.0	ND	11.0	8.9	2.7	2.1	13.0	10.0	23.0	ng/l		
TW	1460036	104	410-19733-1	Upper Hanover Water Authority	Montgomery	11/5/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.0	ND	4.2	3.4	7.6	ng/l		
TW	1460039	102	410-19733-1	Red Hill Water Authority	Montgomery	11/5/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	8.1	ND	27.0	5.8	2.8	ND	6.4	18.0	24.4	ng/l		
TI	1460046	101	0477011	PA American - Norristown	Montgomery	2/5/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	4.0	ND	ND	ND	ND	ND	ND	5.4	5.4	ng/l		
TW	1460048	112	0477009	North Wales Water Auth.	Montgomery	2/5/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	1460050	105	0477017	Telford Borough Auth.	Montgomery	2/5/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	87.5	ND	ND	32.6	ND	ND	7.5	9.6	17.1	ng/l		
TW	1460055	107	0477049	Audubon Water Company	Montgomery	2/14/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	12.2	ND	3.9	8.3	4.9	ND	8.9	16.4	25.3	ng/l		
TW	1460055	111	0477057	Audubon Water Company	Montgomery	2/14/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	4.0	4.0	ng/l		
TW	1460056	100	0477047	St. Gabriels Hall	Montgomery	2/13/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	5.2	ND	ND	ND	ND	9.6	6.6	8.1	14.7	ng/l		
TW	1460062	101	0477362	Avante Apartments	Montgomery	11/4/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	8.5	ND	10.9	4.4	15.3	ng/l		
TW	1460073	105	0477041	Aqua PA Main System	Montgomery	2/13/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	4.8	4.8	9.6	ng/l		
TW	1460073	107	0477039	Aqua PA Main System	Montgomery	2/13/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.5	ND	5.4	ND	7.6	7.1	14.7	ng/l		
TI	1460073	116	0477045	Aqua PA Main System	Montgomery	2/13/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TI	1460073	117	0477043	Aqua PA Main System	Montgomery	2/13/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	4.6	4.6	ng/l		
TW	1460086	101	0477364	St. Lukes Knoll	Montgomery	11/4/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	6.4	ND	4.4	ND	9.1	8.6	17.7	ng/l		
TW	1460340	101	410-19523-1	Gilbertsville Elementary	Montgomery	11/4/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	29.0	ND	2.8	6.8	2.6	1.8	6.6	12.0	18.6	ng/l		
TW	4470012	100	0477486	Geisinger Medical Center	Montour	12/9/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3480044	105	0477029	Royal Oaks MHP	Northampton	2/6/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
BI	3480046	101	0477083	City of Bethlehem	Northampton	2/20/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3480052	102	0477021	Hellertown Boro Auth	Northampton	2/6/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3480052	103	0477019	Hellertown Boro Auth	Northampton	2/6/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3480055	103	0477178	PAW Blue Mtn Div.	Northampton	8/12/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3480066	103	0477023	Bath Municipal Water Works	Northampton	2/6/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3480066	104	0477025	Bath Municipal Water Works	Northampton	2/6/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	5.0	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	4490004	101	0477502	Bucknell View MHP	Northumberland	12/15/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
BW	4490020	101	0477658	Herndon Boro Jackson Twp JMA	Northumberland	3/31/2021	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	4490021	100	0477500	D&H Trailer Park	Northumberland	12/15/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
BI	4490023	102	410-18882-1	PA American White Deer	Northumberland	10/29/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	7500028	101	0477464	Kinkora Pythian Home	Perry	12/3/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	7500039	101	0477462	Carson Long Inst / Talmudic Properties	Perry	12/3/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	6.8	ND	ND	ND	0.0	ng/l		
TW	7500366	101	0477460	Mahanoy Centre	Perry	12/3/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	17.4	ND	13.3	ND	13.3	ng/l			
TI	1510001	101	0477089	Philadelphia Water Department	Philadelphia	2/25/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	1510800	101	0477081	Schuykill Center for Env. Ed.	Philadelphia	2/19/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	7.8	ND	ND	4.4	ND	ND	ND	9.9	9.9	ng/l		
TW	2520107	101	410-26088	Pike County Correctional Facility	Pike	1/12/2021	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	2520981	101	410-26088	Millbrook 5&7	Pike	1/12/2021	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
BW	6530007	101	410-18884-1	Roulette Twp Water Authority	Potter	10/28/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
BW	6530013	101	410-18884-1	Shinglehouse Boro Water Dept	Potter	10/28/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3540026	100	0477440	Mountain Water Authority	Schuylkill	12/1/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3540032	101	410-18604-1	PA AM Water Co - Frackville	Schuylkill	10/28/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	4.9	3.1	3.5	1.9	12.0	14.0	29.0	3.9	32.9	ng/l		
TW	3540038	101	0477452	Schuylkill Co Mun Auth	Schuylkill	12/2/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TI	3540038	102	0477454	Schuylkill Co Mun Auth	Schuylkill	12/2/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3540045	104	410-18604-1	Tower City Authority	Schuylkill	10/28/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3540054	101	0477438	SCMA Pinebrook	Schuylkill	12/1/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3540468	101	0477550	Shalmet Corp	Schuylkill	2/17/2021	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3540898	101	0477552	Omnova Solutions	Schuylkill	2/17/2021	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3540898	103	0477554	Omnova Solutions	Schuylkill	2/17/2021	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3540923	101	0477548	Eitel Presses	Schuylkill	2/17/2021	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3540957	101	0477546	Practice Management Advisors	Schuylkill	2/17/2021	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3540973	101	0477556	Fed Ex Auburn	Schuylkill	2/17/2021	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	3540977	101	0477162	Keystone Potato	Schuylkill	8/11/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	4550009	106	0477572	Aqua PA Monroe	Snyder	2/23/2021	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	4550028	101	0477504	Penn Township Municipal Auth	Snyder	12/15/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l	
TW	4550320	101	0477160																								

Summary of Results for SDW Sampling Project Using EPA Method 537.1

Category	PWSID	EPID	BOL Sample # / ELLE Job #	PWS Name	County	Date Collected	11CI-PF3OUdS	9CI-PF3ONS	ADONA	HFPO-DA	NETFOSAA	NMeFOSAA	PFDA	PFDoA	PFTA	PFTrDA	PFHxA	PFUnA	PFBS	PFHpA	PFHxS	PFNA	PFOS	PFOA	Sum_2 (PFOS + PFOA)	Units									
							11-chloroicosafafluoro-3-oxaundecane-1-sulfonic acid	9-chlorohexadecafluoro-3-oxanone-1-fulfonic acid	4,8-dioxa-3H-perfluorononanoic acid	Hexafluoropropylene oxide dimer acid	N-ethyl perfluorooctanesulfonamidoacetic acid	N-methyl perfluorooctanesulfonamidoacetic acid	Perfluorododecanoic acid	Perfluorodecanoic acid	Perfluorotetradecanoic acid	Perfluorotridecanoic acid	Perfluorohexanoic acid	Perfluoro undecanoic acid	Perfluorobutane sulfonic acid	Perfluoroheptanoic acid	Perfluorohexane sulfonic acid	Perfluorononanoic acid	Perfluorooctane sulfonic acid	Perfluorooctanoic acid			763051-92-9	756426-58-1	919005-14-4	13252-13-6	2991-50-6	2355-31-9	335-76-2	307-55-1	376-06-7
TW	2660036	102	410-23827	Aqua PA Factoryville	Wyoming	12/11/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l										
TW	2660039	101	410-23827	Eaton Sewer and Water	Wyoming	12/11/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.1	ND	ND	ND	4.0	1.8	5.8	ng/l										
TW	2660380	100	410-23827	Proctor and Gamble Paper Products	Wyoming	12/11/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l										
TW	2660385	101	0477324	Village Shopping Center	Wyoming	10/8/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.8	ND	ND	ND	9.1	ND	9.1	ng/l										
TW	7670022	101	0477512	Locust Manor MHP	York	12/22/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l										
TW	7670027	101	0477510	Laurelwood MHP	York	12/22/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l										
TW	7670071	101	410-16342-1	Dillsburg Area Authority	York	10/7/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l										
TW	7670071	102	410-24743	Dillsburg Area Authority	York	12/22/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	4.1	ND	2.7	ND	5.0	3.2	8.2	ng/l										
TW	7670073	105	410-16448-1	Dover Twp Water Sys	York	10/7/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.5	ND	3.6	1.8	1.9	ND	6.8	6.8	13.6	ng/l										
TW	7670082	101	410-15545-1	New Freedom Boro Water Auth	York	9/30/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	4.8	ND	ND	ND	ND	4.3	3.0	7.3	ng/l										
TW	7670082	103	410-15545-1	New Freedom Boro Water Auth	York	9/30/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.7	ND	3.9	ND	ND	ND	ND	2.3	2.3	ng/l										
TW	7670082	104	410-15545-1	New Freedom Boro Water Auth	York	9/30/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	6.0	ND	12.0	4.5	3.5	15.0	3.9	5.2	9.1	ng/l										
TW	7670082	107	410-24743	New Freedom Boro Water Auth	York	12/22/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l										
TW	7670088	101	410-15545-1	Shrewsbury Borough	York	9/30/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	4.6	ND	20.0	2.8	3.2	ND	13.0	6.8	19.8	ng/l										
TW	7670088	108	410-24743	Shrewsbury Borough	York	12/22/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.0	ND	2.8	1.8	ND	ND	ND	4.7	4.7	ng/l										
TW	7670310	101	410-16448-1	Garrod Hydraulics	York	10/7/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l										
TW	7670333	101	410-24743	Joseph Machine Co	York	12/22/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.6	ND	2.6	ng/l									
TW	7670356	101	410-24736	Winterstown United Methodist Church	York	12/22/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	7.2	ND	ND	3.4	ND	ND	ND	7.0	7.0	ng/l										
TW	7671084	101	0477224	Key Plastics	York	8/27/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l										
TW	7671084	102	0477226	Key Plastics	York	8/27/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l										
TW	7671087	101	0477508	Whales Snails and Puppy Dog Tails	York	12/22/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	11.0	4.4	4.4	8.8	ng/l										
TW	7671303	101	410-24736	N Hopewell Winterstown Elem	York	12/22/2020	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.0	ng/l										
KEY																																			
TW/TI = Target Well (GW)/Target Intake (SW)							Average	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.9	0.0	1.1	0.7	1.4	0.4	2.5	2.0		ng/l								
BW/BI = Baseline Well (GW)/Baseline Intake (SW)							Median	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND		ng/l							
EPID=entry point ID number; the entry point is the first finished water tap after treatment							Minimum	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND		ng/l							
ND = not detected (the result was below the laboratory's reporting limit)							Maximum	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	87.5	3.1	64.0	32.6	140.0	18.1	187.1	59.6		ng/l								
Laboratory reporting limits for all 18 PFAS range from 1.7 to 4.0 ng/L																																			
							# Detects	none	none	none	none	none	none	none	none	none	none	80	2	66	49	52	23	103	112										
							Average Detect Value											9.9	2.6	7.0	6.1	10.9	7.2	9.9	7.5		ng/l								
							Median Detect Value											6.1	2.6	4.2	4.5	4.5	5.6	6.5	5.3		ng/l								
							Min Detect Value											1.7	2.0	1.7	1.8	1.9	1.8	1.8	1.7		ng/l								
							Max Value											87.5	3.1	64.0	32.6	140.0	18.1	187.1	59.6		ng/l								