PFAS Health, Toxicology Regulatory Subgroup

Virginia Department of Health (VDH) Office of Drinking Water
August 25, 2021
1:30 pm – 3:30 pm

1. Opening Remarks

VDH State Toxicologist, Dwight Flammia, Ph.D., invited members of the Toxicology Subgroup to join him through WebEx and discuss PFAS toxicological information. The discussion began at 1:30 pm and was recorded. A summary of the discussion and Dwight's presentation for Subgroup members will be posted on Town Hall.

2. Subgroup Members Present:

Kelly Ryan (Virginia American Water)
Paul Nyffeler (Chem Law)
William Mann (citizen)
Jillian Terhune (City of Norfolk)
Dwight Flammia (VDH, State Toxicologist)
Chris Leyen (VCLV)
Steve Risotto (American Chemistry Council)

Public in attendance:
Ellen Egan
Amanda Waters

ODW Staff:
Nelson Daniel

3. Discussions

The Subgroup discussed information that Dwight ascertained from speaking with a scientist in the U.S. Environmental Protection Agency's (EPA) IRIS (Integrated Risk Information System) program about reviewing federal guidelines for developing reference levels, health reference levels, and maximum contaminant levels (MCLs). Dwight's presentation (attached, following the summary) centered on use and application of uncertainty factors for special populations, lifestages, and subchronic animal studies. Members of the subgroup did not have any comments on this part of the presentation.

Dwight noted that EPA guidance suggests special deference should be given to chronic studies, and noted these may be lacking in studies used to develop PFAS MCLs. His conclusion was based in part on a 2002 EPA document on risk assessment (Document for a Review of the Reference Dose and Reference Concentration Processes, https://www.epa.gov/risk/review-reference-dose-and-reference-concentration-processes-document).

Dwight presented an alternative approach to developing a drinking water MCL for two specific PFAS – perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS) – that was meant to follow the Centers for Disease Control and Prevention's (CDC) approach to setting a reference level for lead in children's blood. Using PFOA or PFOS levels found in the 95th percentile serum levels in the United States, clearance factors, relative source contribution, and drinking water consumption rates, one could calculate an MCL for PFOA and PFOS. One member agreed that the approach was unique and paralleled what CDC did for lead. Further discussion was limited as MCLs are derived from health based studies.

Next, Dwight facilitated a discussion about developing a 2-3 page summary of the Subgroup's recommendations for the larger PFAS Workgroup to consider at its upcoming meeting in September. Most of the discussion centered on questions specific to PFOA and PFOS:

1. Based on the scientific literature and what other states have done to establish MCLs for PFAS, particularly PFOA and PFOS, can the Subgroup recommend a safe level of PFOA and/or PFOS in drinking water that the other subgroups and PFAS Workgroup can use to recommend MCLs? What have we learned from states with MCLs for these compounds and is it necessary to propose a safe level?

> The group discussed the specifying (or recommending) a "MCL" versus a "maximum contaminant level goal" (MCLG). Over several meetings, the Subgroup considered and evaluated how other states established MCLs – looking at the toxicological research and studies they used as the basis for each MCL. Members discussed whether or not the other states considered technical feasibility and cost in the process, and whether the Subgroup also needed to consider those issues – or if other subgroups and the larger PFAS Workgroup would use a "safe level" or MCLG, recommended by the Toxicology Subgroup, and apply factors such as cost and feasibility (required considerations under the Safe Drinking Water Act – see presentation) in the process to develop and recommend a MCL. Members discussed whether there was sufficient information about PFOA and/or PFOS to recommend a safe level, or if the MCLs other states develop were, by themselves, sufficient. One member felt that the approach New Jersey followed to develop MCLs for PFOA and PFOS was the better than other states' because it was based on more scientific research (the member also though the work Drexel University did for Pennsylvania was sound). Another member pointed out that many states had different peer-review processes that were questionable. Other members did not have an opinion.
>
> Members were concerned about Virginia adopting MCLs that may be lower than EPA – which is in the process of adopting MCLs for PFOA and PFOS. Discussion also considered whether Virginia should propose or set an MCL for a PFAS that had not been found in drinking water in Virginia (PFNA (perfluorononanoic acid) was not detected above the practical quantitation level in the sample study conducted for HB586).
>
> Subgroup members did agree that the existing scientific data is not sufficient to quantify a safe level between 8 ppt and 14 ppt (the range of other states MCLs for PFOA and

PFOS); and 70 ppt, EPA's lifetime health advisory for PFOA and PFOS (individually or combined), is too high.

For the last 10 minutes of the discussion, Subgroup members considered questions about the other PFAS specified in HB586:

2. Do we have a consensus it is necessary for us to propose an MCL for PFBA, PFHpA, PFHxS, and PFNA, and if so which state process do we support and if none, what are the limitations?

The subgroup acknowledged that there is limited toxicity information on these compounds and other states rationales for selecting MCLs for some of these were inconsistent.

3. There are a few other PFAS found in the state sampling data – do Subgroup members want to make a recommendation about either the health risk or toxicity of any of these?

Nelson Daniel showed results from the sample study for all PFAS samples with results above the laboratory's practical quantitation level (PQL). He indicated that ODW staff are preparing a data summary and will release the results to the public soon.

Among the 6 PFAS listed in HB586, PFNA was not detected in any of the 63 samples in a quantity above the PQL; perfluorohexane sulfonate (PFHxS) was measured above the 3.5 ppt PQL at one sample location. The concentration was 4.9 ppt.

One PFAS that the lab found in several samples that was not included among the 6 specified in HB586 was PFPeA (perfluoropentanoic acid). One member of the Subgroup said that PFPeA may be a breakdown product of longer-chain PFAS (i.e., PFOA, PFOS), explaining its presence in samples with other PFAS.

The group agreed that a qualitative summary of the Subgroup's recommendations should be written and that providing a quantitative safe level presents too many challenges at this time. The group suggested Dwight begin writing the qualitative summary and the group will make comments/suggestion/edits to his draft.

4. Public Comment

Dwight invited members of the public to comment. No one offered comments, concerns, or questions.

5. Closing

The next Toxicology Subgroup meeting will be held in September. Dwight concluded the discussion at 3:40 pm.

Establishing Regulatory Limits for PFAS in Virginia Drinking Water

PFAS Toxicology Regulatory Subgroup

Dwight Flammia, Ph.D.
State Public Health Toxicologist
Virginia Department of Health

August 25, 2021

PFAS Toxicology Subgroup

Overview

- Opening Remarks
- Workgroup Members Introductions
- Presentations/Discussion
- Public Comment
- Next Meeting

Determining Whether or Not to Regulate

- a contaminant may have an adverse health effect;
- it is known to occur or there is a substantial likelihood that it will occur in public water systems with a frequency and at levels of public health concern; and
- in the sole judgment of the Administrator, regulation of the contaminant presents a meaningful opportunity for health risk reduction for persons served by water systems

- (Safe Drinking Water Act requirements)

Determining Whether or Not to Regulate

Drinking water regulations specify a nonenforceable MCLG, which is based solely on health effects data. Unlike an MCL, the MCLG does not reflect cost or technical feasibility considerations.

For contaminants with noncarcinogenic effects, **EPA derives an MCLG based on a reference dose, which is an estimate of the amount of a contaminant that a person can be exposed to on a daily basis that is not anticipated to cause adverse health effects for sensitive life stages and meaningful populations** (e.g., infants, children, pregnant women, the elderly, individuals with a history of serious illness, or other sensitive subpopulations) over a lifetime.

Determining Whether or Not to Regulate

For contaminants with noncarcinogenic effects, **EPA derives an MCLG based on a reference dose**, which is an estimate of the amount of a contaminant that a person can be exposed to on a daily basis that is not anticipated to cause adverse health effects for sensitive life stages and meaningful populations (e.g., infants, children, pregnant women, the elderly, individuals with a history of serious illness, or other sensitive subpopulations) over a lifetime.

This amount incorporates uncertainty factors to provide a margin of protection for sensitive subpopulations and to account for uncertainties in the data

Feasibility and Maximum Contaminant Levels

SDWA generally requires EPA to set the MCL as close to the MCLG as "feasible." The act defines "feasible" to mean feasible with the use of the best available (and field demonstrated) treatment technologies, taking cost into consideration. The level at which EPA is able to set the MCL is determined by the ability of a treatment technology to reduce a contaminant to a certain level. EPA's ability to set the MCL at the MCLG also depends on the availability of a test method that is sensitive enough to detect the contaminant at the MCLG. For contaminants regulated for noncarcinogenic effects, EPA generally has set the enforceable standard at the same level as the MCLG. If it is not technologically or economically feasible to ascertain the level of a contaminant in drinking water, EPA may establish a treatment technique in lieu of an MCL. For example, EPA's Lead and Copper Rule includes a treatment technique—primarily relying on corrosion control, among other actions—because lead and/or copper generally enters the water after it leaves the plant.

ENVIRONMENTAL PROTECTION AGENCY 40 CFR Part 141 [EPA–HQ–OW–2019–0583; FRL–10005–88– OW] Announcement of Preliminary Regulatory Determinations for Contaminants on the Fourth Drinking Water Contaminant Candidate List

Federal Register / Vol. 85, No. 47 / Tuesday, March 10, 2020 / Proposed Rules

- If appropriate and available, the Agency quantitatively takes into account exposure data applicable to **sensitive populations or lifestages** when deriving HRLs for regulatory determinations. When data are not available on sensitive populations, the derivation of the RfD typically includes an uncertainty factor to account for the weakness in the database. Additionally, the EPA will use exposure factors relevant to the sensitive population in deriving the HRL.

ENVIRONMENTAL PROTECTION AGENCY 40 CFR Part 141 [EPA–HQ–OW–2019–0583; FRL–10005–88– OW] Announcement of Preliminary Regulatory Determinations for Contaminants on the Fourth Drinking Water Contaminant Candidate List

Federal Register / Vol. 85, No. 47 / Tuesday, March 10, 2020 / Proposed Rules

HRLs for contaminants with a threshold dose-response (typically nc cancer endpoints) are calculated as follows:

$$HRL = RfD * \frac{BW}{DWI} * RSC$$

HRL – health reference level

ENVIRONMENTAL PROTECTION AGENCY 40 CFR Part 141 [EPA–HQ–OW–2019–0583; FRL–10005–88– OW] Announcement of Preliminary Regulatory Determinations for Contaminants on the Fourth Drinking Water Contaminant Candidate List

Federal Register / Vol. 85, No. 47 / Tuesday, March 10, 2020 / Proposed Rules

- In prioritizing the contaminants of greatest public health concern for regulatory determination, Section 1412(b)(1)(C) of SDWA requires the Agency to consider ‘‘among other factors of public health concern, the effect of such contaminants upon subgroups that comprise a meaningful portion of the general population (such as infants, children, pregnant women, the elderly, individuals with a history of serious illness, or other subpopulations) that are identifiable as being at greater risk of adverse health effects due to exposure to contaminants in drinking water compared to the general population.’’ If appropriate and if adequate data are available, the Agency will use data from sensitive populations and lifestages quantitatively when deriving HRLs for regulatory determinations in the following manner

ENVIRONMENTAL PROTECTION AGENCY 40 CFR Part 141 [EPA–HQ–OW–2019–0583; FRL–10005–88– OW] Announcement of Preliminary Regulatory Determinations for Contaminants on the Fourth Drinking Water Contaminant Candidate List

Federal Register / Vol. 85, No. 47 / Tuesday, March 10, 2020 / Proposed Rules

- For non-carcinogens, an HRL can be developed for a sensitive population if data are available to associate exposure with the critical health endpoint in a specific group or during a specific period of sensitivity. Age specific drinking water intake (DWI) to body weight (BW) ratio values from the Exposure Factors Handbook (USEPA, 2011b) can be used to reflect the period of exposure more accurately. The Agency can also apply specific uncertainty factors (UFs) when deriving the RfD if toxicological data are lacking for a sensitive population. Two common justifications for UFs that can be applied to account for sensitive populations are: (1) Variation in sensitivity among the members of the human population (i.e., intraspecies variability) and (2) uncertainty associated with an incomplete database.

Reference Dose (RfD): Description and Use in Health Risk Assessments
Background Document 1A
March 15, 1993

- The U.S. EPA is concerned about the potential toxic effects in humans associated with all possible exposures to chemicals. The magnitude, frequency, and duration of exposure may vary considerably in different situations. Animal studies are conducted using a variety of exposure durations (e.g., acute, subchronic, and chronic) and schedules (e.g., single, intermittent, or continuous dosing). Information from all these studies is useful in the **hazard identification phase of risk assessment**. For example, overt neurological problems identified in high-dose acute studies tend to reinforce the observation of subtle neurological changes seen in low-dose chronic studies. **Special attention is given to studies involving low-dose, chronic exposures, since such exposures can elicit effects absent in higher dose, shorter exposures, through mechanisms such as accumulation of toxicants in the organisms.**

EPA 2002 Document

Table 4-2. Factors for evaluating evidence regarding identification and characterization of susceptible subpopulations[a]

Factor	Increased weight	Decreased weight
Timing (life stage) - response relationship	Effects occur at greater magnitude at one or more life stage(s)	No difference in effects at different life stage(s)
Type of effect	Different types of effects in specific subpopulations	Same effect(s) across all potential subpopulations
Dose-response relationship	Effect occurs at lower exposures in one or more subpopulation(s)	No evidence for differential dose-response across different subpopulations
Latency of effect	Latency to observed effect different in specific subpopulations	No difference between subpopulations in latency to effect
Seriousness/ reversibility of effects	Effects different in seriousness or degree of reversibility in specific subpopulations and/or differences in later consequence of an initially reversible effect	No differences between subpopulations in seriousness and/or reversibility of effects, or in later consequences of an initially reversible effect

[a] Subpopulations may be defined by gender, individuals at different life stages (fetus, child, adult, elderly), differences in genetic polymorphisms, and/or pre-existing diseases or conditions that may result in differential sensitivity to adverse effects from exposure to a specific toxic agent.

EPA 2002 Document

- the minimum dataset for low-confidence and high-confidence RfDs and RfCs has been specifically defined as follows (U.S. EPA, 1994, 2002c):
  - minimum dataset for a low confidence chronic RfD or RfC is a single subchronic study.
  - The minimum dataset for a high confidence chronic RfD or RfC is a chronic study in two species, a single two-generation reproductive toxicity study, and a developmental toxicity study in two species by the appropriate route of exposure.

EPA 2002 Document

Effects seen at the termination of a chronic study may be due to cumulative damage from a continued repeated chemical insult, but they could also be a latent response from an earlier single or short-term multiple exposure. Thus, latent effects might be revealed in chronic studies, but it would not be clear whether they were the result of acute/short-term exposure or the chronic exposure. Specific information on the latency of a response would follow only from a clearer understanding of the mechanism of the effect and from actual “stop exposure” protocols (e.g., the satellite studies depicted in Figure 3-1) or from shorter-term exposures with follow-up over a much longer period of time. It thus follows that any chemical database that does not have exposure-response studies of lifetime duration or any specific exposure-latency protocols would not cover the possibility of latent effects.

EPA 2002 Document

Derivation of a reference value based on shorter-term exposure guideline protocols would have to fully consider the aspect of reversibility in interpretation of the data. It is important to understand the difference between an endpoint that is truly reversible and one that is related to or is a precursor of other adverse effects. For example, low birth weight may be “reversible” through catch-up growth postnatally, but it also may be related to developmental delays or other health outcomes that result from prenatal growth reduction/retardation.

EPA 2002 Document

The Panel recommends that endpoint- or life stage-specific reference values such as the RfD_{DT} (reference dose for developmental toxicity), which were originally proposed in Guidelines for Developmental Toxicity Risk Assessment (U.S. EPA, 1991), not be derived. Rather, **a sample reference value should be calculated for each relevant and appropriate endpoint and these should then be considered in the derivation of various duration reference values. Reference values should be derived to be protective of all types of effects for a given duration of exposure and are intended to protect the population as a whole, including potentially susceptible subgroups. Thus, the RfD_{DT} concept of a critical window of exposure for some health effects is addressed in the adoption of the less-than-chronic reference values.** This recommendation does not preclude, however, using specific common endpoints in the assessment of cumulative risk for mixtures or chemicals that have a common mode of action or for risk management purposes.

Guidelines for Reproductive Toxicity Risk Assessment EPA 1996

- Pharmacokinetic studies in reproductive toxicology are most useful if the data are obtained with animals that are at the same reproductive status and stage of life (e.g., pregnant, nonpregnant, embryo or fetus, neonate, prepubertal, adult) at which reproductive insults are expected to occur in humans.

Guidelines for Reproductive Toxicity Risk Assessment EPA 1996

- It should be recognized that, based on the definitions used in these Guidelines, **almost any segment of the human population may be at risk for a reproductive effect**. Although the reproductive effects of exposures may be manifested while the exposure is occurring (e.g., menstrual disorder, decreased sperm count, spontaneous abortion) **some effects may not be detectable until later in life** (e.g., endocrine disruption of reproductive tract development, premature reproductive senescence due to oocyte depletion), **long after exposure has ceased.**

Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000)

- **Generally, less-than-90-day experimental studies are not used to derive an RfD**. This is based on the rationale that studies lasting for less than 90 days may be too short to detect various toxic effects. However, EPA, has in certain circumstances, derived an RfD based on a less-than- 90-day study. For example, the RfD for nonradioactive effects of uranium is based on a 30-day rabbit study (USEPA, 1989). The short-term exposure period was used, because it was adequate for determining doses that cause chronic toxicity. In other cases, it may be appropriate to use a less-than-90-day study because the critical effect is expressed in less than 90 days. For example, the RfD for nitrate was derived and verified using studies that were less than 3-months in duration (USEPA, 1991b). For nitrate, the critical effect of methemoglobinemia in infants occurs in less than 90 days. When it can be demonstrated from other data in the toxicological database that the critical adverse effect is expressed within the study period and that a longer exposure duration would not exacerbate the observed effect or cause the appearance of some other adverse effect, the Agency may choose to use less-than-90-day studies as the basis of the RfD. Such values would have to be used with care because of the uncertainty in determining if other effects might be expressed if exposure was of greater duration than 90 days.

PFOA Drinking Water Concentration and Serum Level Relationship

Serum n-Perfluorooctanoic acid (n-PFOA) (2013 - 2016)

CAS Number 335-67-1

Linear PFOA isomer

Geometric mean and selected percentiles of serum concentrations (in µg/L) for the U.S. population from the National Health and Nutrition Examination Survey.

Demographic Categories	Survey (years)	Geometric mean (95% CI)	50th Percentile (95% CI)	75th Percentile (95% CI)	90th Percentile (95% CI)	95th Percentile (95% CI)	Sample size
Total population	13-14	1.83 (1.65-2.03)	1.90 (1.80-2.10)	2.90 (2.60-3.20)	4.10 (3.50-5.10)	5.30 (4.40-6.00)	2165
Total population	15-16	1.46 (1.37-1.56)	1.50 (1.40-1.70)	2.40 (2.20-2.50)	3.30 (3.00-3.50)	4.10 (3.80-4.60)	1993
Age 12-19 years	13-14	1.56 (1.41-1.74)	1.60 (1.30-1.80)	2.10 (1.90-2.50)	2.70 (2.30-3.40)	3.40 (2.70-4.80)	401
Age 12-19 years	15-16	1.17 (1.06-1.28)	1.20 (1.10-1.40)	1.60 (1.40-1.80)	2.00 (1.70-2.50)	2.40 (2.00-2.90)	353
Age 20 + years	13-14	1.87 (1.68-2.08)	2.00 (1.80-2.20)	3.00 (2.70-3.30)	4.40 (3.60-5.10)	5.40 (4.60-6.20)	1764
Age 20 + years	15-16	1.50 (1.41-1.61)	1.60 (1.50-1.80)	2.40 (2.30-2.60)	3.40 (3.10-3.60)	4.20 (4.00-4.90)	1640
Males	13-14	2.18 (1.98-2.39)	2.30 (2.10-2.50)	3.20 (2.80-3.50)	4.60 (3.70-5.30)	5.40 (4.60-5.90)	1031
Males	15-16	1.70 (1.56-1.86)	1.80 (1.60-2.00)	2.50 (2.30-2.60)	3.30 (3.00-3.60)	4.00 (3.60-4.80)	964
Females	13-14	1.55 (1.37-1.75)	1.60 (1.40-1.80)	2.60 (2.20-2.90)	3.70 (3.30-4.70)	4.80 (3.90-6.60)	1134
Females	15-16	1.26 (1.18-1.35)	1.30 (1.20-1.40)	2.10 (1.90-2.40)	3.30 (2.80-3.60)	4.10 (3.60-4.90)	1029

Drinking Water Conc. (µg/L) x 0.016 L/kg/day = Serum Conc. (µg/L) x Clearance (1.4 x 10-4 L/kg/day)

$$\text{Drinking Water Conc. (µg/L)} = \frac{4.1\ \mu g/L \times 1.4 \times 10\text{-}4\ L/kg/day}{0.016\ L/kg/day} = 0.0358\ \mu g/L \text{ or } 35\ ppt$$

If drinking water contributes 20% then limit in drinking water should be 7 ppt

PFOS Drinking Water Concentration and Serum Level Relationship

Serum n-Perfluorooctane sulfonic acid (n-PFOS) (2013 - 2016)

CAS Number 1763-23-1

Linear PFOS isomer

Geometric mean and selected percentiles of serum concentrations (in µg/L) for the U.S. population from the National Health and Nutrition Examination Survey.

Demographic Categories	Survey (years)	Geometric mean (95% CI)	50th Percentile (95% CI)	75th Percentile (95% CI)	90th Percentile (95% CI)	95th Percentile (95% CI)	Sample size
Total population	13-14	3.45 (3.10-3.85)	3.50 (3.20-3.90)	6.00 (5.20-6.60)	9.90 (8.50-11.3)	14.0 (11.0-16.7)	2165
Total population	15-16	3.20 (2.93-3.49)	3.20 (2.80-3.60)	5.60 (4.90-6.40)	8.90 (7.70-10.4)	12.8 (10.1-17.9)	1993
Age 12-19 years	13-14	2.59 (2.29-2.93)	2.70 (2.30-2.90)	3.90 (3.40-4.80)	6.20 (4.80-7.00)	7.10 (6.20-8.60)	401
Age 12-19 years	15-16	2.07 (1.90-2.26)	2.00 (1.80-2.40)	3.10 (2.60-3.50)	4.40 (3.80-4.90)	5.10 (4.40-6.60)	353
Age 20 + years	13-14	3.59 (3.21-4.02)	3.70 (3.40-4.10)	6.30 (5.50-7.20)	10.3 (9.10-12.3)	15.1 (11.9-17.7)	1764
Age 20 + years	15-16	3.38 (3.07-3.73)	3.50 (3.00-3.90)	5.90 (5.20-6.80)	9.20 (8.10-11.1)	13.9 (10.0-19.7)	1640
Males	13-14	4.26 (3.74-4.86)	4.20 (3.70-4.80)	6.60 (6.00-7.90)	10.8 (9.30-15.1)	15.4 (12.1-20.2)	1031
Males	15-16	4.10 (3.70-4.53)	4.00 (3.50-4.70)	6.80 (5.70-7.60)	10.4 (8.60-12.9)	14.7 (10.5-20.4)	964
Females	13-14	2.83 (2.56-3.13)	2.90 (2.60-3.20)	4.90 (4.40-5.60)	8.50 (7.30-9.90)	11.6 (9.90-13.0)	1134
Females	15-16	2.53 (2.28-2.80)	2.50 (2.30-2.70)	4.30 (3.80-5.00)	7.50 (6.00-8.50)	10.1 (8.10-14.2)	1029

Drinking Water Conc. (µg/L) x 0.016 L/kg/day = Serum Conc. (µg/L) x Clearance (8.1 x 10-5 L/kg/day)

$$\text{Drinking Water Conc. (µg/L)} = \frac{12.8\ \mu g/L \times 8.1 \times 10\text{-}5\ L/kg/day}{0.016\ L/kg/day} = 0.0648\ \mu g/L \text{ or } 65\ ppt$$

If drinking water contributes 20% then limit in drinking water should be 13 ppt

0.016 L/kg/day is the mean U.S. daily water ingestion rate; EPA 2011

Discussion for Subgroup

EPA is in the rule making process for PFOA and PFOS.

Do we have a consensus on adopting any PFOA or PFOS MCL developed by any state and if so which state and if not, what are our reasons?

Is it necessary that our subgroup propose an MCL for PFOA and PFOS?

Discussion for Subgroup

PFBA, PFHpA, PFHxS, and PFNA MCL making processes for states with MCL for each PFAS has been discussed.

Do we have a consensus it is necessary for us to propose an MCL and if so which state process do we support and if none, what are the limitations.

Discussion for Subgroup

There are a few other PFAS found in the state sampling data - How should we move on this.

Discussion

- Public Comment
- Next Meeting
  - September 8, 2021