

December 30, 2021

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Washington, DC 20460

**RE: Federal Register #FRL-9222-01-OA – Notification of a Public Meeting of the Science Advisory Board
Per- and Polyfluoroalkyl Substances (PFAS) Review Panel**

Dear Dr. Shallal,

The Minnesota Department of Health (MDH) commends EPA for taking substantive action towards the development of federal drinking water standards for two per- and polyfluoroalkyl substances (PFAS). For nearly twenty years, drinking water in Minnesota has been known to be contaminated with PFOS and PFOA.

MDH appreciates the extensive work performed by EPA in the preparation of these documents, totaling nearly 1800 pages. However, the duration of the Public Comment period – roughly 6 weeks with multiple intervening major holidays – is not practical for the Science Advisory Board (SAB), stakeholders, and all other interested parties to adequately review and fully respond to the volume of information in a timely manner.

Despite the length of the documents, MDH noted critical deficiencies and is deeply concerned that the SAB and other reviewers do not have access to necessary data and rationale to properly evaluate key decision points. The many errors and inconsistencies as well as overall lack of crucial information in the PFOS/PFOA proposed approaches documents hinder the SAB's and others' abilities to critically review and make informed recommendations. Our key comments and feedback include:

- The amount of detail and rationale for key decision points is grossly inadequate to the point of not allowing for critical review.
- The benchmark dose modeling serving as the basis for the draft reference doses (RfDs) is not transparent. EPA's lack of detail in reporting does not follow published EPA guidance nor standard risk assessment practice.
- Modifications to and application of the selected toxicokinetic (TK) model (Verner 2016) [HERO: 3299692] are inadequately described to the point of not allowing for critical review.
- EPA has mischaracterized some of the steps of the EPA Exposure Decision Tree process for determining the relative source contribution (RSC).

Specific comments on these points are provided below.

Proposed Approaches for PFOA and PFOS MCLGs

General Comments

Throughout the documents, there was a general misplaced emphasis on where detail should be provided. Hundreds of pages were given to describe EPA's systematic review and the large PFOA/PFOS databases, while comparatively little space was given to decisions EPA made using those databases.

For example, it is not clear why some studies labeled as "medium confidence" were excluded from Table 21 ("POD_{HEDS} Considered for Derivation of Candidate RfD Values") while others were included. Likewise, it is not clear why some POD_{HEDS} identified in Table 21 were not carried through to candidate RfD calculation. A specific example in Table 21 of the PFOA document is the cholesterol epidemiological study (Dong, Wang et al. 2019) [HERO: 5080195]. This study was labeled "medium confidence" (the same category as the eventual critical study) and the point of departure (POD) was in the same range as many other studies used to calculate candidate RfDs. Despite this, a candidate RfD was not calculated. The same issue is noted within the PFOS document. The exclusion of cholesterol from candidate RfD derivation is especially puzzling given that the only cost/benefit document released by EPA is based on cardiovascular disease. Without a more detailed explanation for why these studies were excluded, it is impossible to review EPA's decision. This is merely one instance where vital rationale was not included; this phenomenon pervades the entire document, and a non-exhaustive list is contained in the comments below.

Benchmark Dose Modeling Evaluation

Overall, the information about the selected critical study and subsequent benchmark dose analysis provided in EPA's draft approaches documents lack many key scientific details, contrary to EPA's published Benchmark Dose Technical Guidance (USEPA 2012) [HERO: 1239433]. Specifically, it is unclear:

- what the actual values in the datasets are so that modeling can be replicated
- if EPA performed their own modeling of the datasets
- if EPA adopted the BMD/Ls directly from the critical study (EFSA Panel on Contaminants in the Food Chain, Schrenk et al. 2020)
- why EPA was comfortable with a BMD/BMDL at least an order of magnitude below the observable data when the dataset contained a no observable adverse effect concentration (NOAEC), according to other authoritative bodies

As laid out in EPA's BMD guidance, there are many fundamental elements to report with any computation of a BMD/L to aid with reviewing the proposed model. The draft approaches documents are missing several key elements, including but not limited to:

- A list of the dose-response data used
- Estimation procedure
- Estimates of model parameters
- Goodness-of-fit, log-likelihood, and AIC
- Plots of a fitted dose-response curve with data points and error bars
- Plots with confidence limits for the fitted curve, if applicable

- A comparison of the BMD/Ls from the selected BMR (5% relative deviation) to standard BMRs (0.5 and 1 standard deviation)

The critical study selected by EPA for derivation of both PFOA and PFOS RfDs was (Budtz-Jørgensen and Grandjean 2018). This study was an analysis of tetanus and diphtheria antibody titers in two cohorts of Faroe Island children environmentally exposed to various PFAS, and the study authors calculated BMDs and BMDLs for five PFAS (PFOS, PFOA, PFHxS, PFNA, PFDA) with a benchmark response (BMR) of a 5% reduction in antibody titers. The justification provided in the documents for a 5% BMR is confusing; PFOA Section 4.1.6 and Appendix B.1.1 describe the BMR selection as a “reasonable and appropriate choice as anti-tetanus antibody concentrations prevent against tetanus, which is a rare, but severe and sometimes fatal infection...” This characterization implies that a 5% drop in baseline anti-tetanus antibody titer is sufficient to cause tetanus infection and potential mortality. While a decrease in anti-tetanus antibodies may be a *risk factor* for a tetanus infection, the endpoint measured (anti-tetanus antibodies) and the effect used as justification for the BMR (severe infection/death) are not equivalent; indeed, EPA states “...decreases in anti-tetanus antibody concentrations are not in themselves an adverse effect...” Tetanus is a reportable disease in the United States, with 264 cases reported to the US Centers of Disease Control between 2009-2017; any association between serum PFAS, anti-tetanus antibodies, and tetanus infection can and should be investigated directly. Similar justifications are made for the anti-diphtheria antibody results from this study; only 13 cases were reported in the US between 1996-2016. Accordingly, a more thorough explanation of the appropriateness of the selected BMR, as well as potential alternatives and comparisons to standard BMRs (e.g., 1 SD and 0.5 SD), should be provided.

Due to the lack of reporting of minimum required data for these BMD/Ls in the original publication as well as in the draft EPA documents, MDH referred to authoritative secondary reviews from other governmental bodies to better understand the analysis that appears to be directly taken from the publication of (Budtz-Jørgensen and Grandjean 2018).

Both the European Food Safety Authority (EFSA) and the California Environmental Protection Agency (CalEPA) have recently released evaluations of PFOS and PFOA. It is significant that both EFSA and CalEPA declined to use modeling results from (Budtz-Jørgensen and Grandjean 2018). When EFSA performed their own modeling of the dataset in multiple software packages (PROAST and BMDS) using an extrapolated reference value of 0, the BMDL-BMDU intervals were extremely wide and BMDLs were orders of magnitude below any observed serum levels. EFSA determined that the BMDLs produced were not suitable for risk assessment; rather, EFSA opted for a NOAEC approach with this study and ultimately selected a different study based on the same critical effect as the basis for their point of departure (EFSA Panel on Contaminants in the Food Chain, Schrenk et al. 2020). Similarly, CalEPA selected a NOAEC approach when their modeling of this dataset resulted in unacceptably large BMD:BMDL ratios and BMDLs far outside the observed serum levels (CalEPA Office of Environmental Health Hazard Assessment 2021).

The issues raised by these two authoritative bodies as well as standard BMD modeling practice raises the question whether this dataset was appropriate for modeling and, if so, what decisions were made that ensured a proper model fit. Particularly noteworthy is that within both the PFOA and PFOS documents, EPA states that for the (Budtz-Jørgensen and Grandjean 2018) dataset “no information was available to judge the fit of the model in the range of the BMDLs”; without this information, it is impossible to evaluate the acceptability of these models for the purposes of risk assessment. Additionally, EPA’s BMD guidance cautions against extrapolating to lower doses/responses than observed and suggests considering a NOAEC/LOAEC approach (USEPA 2012) [HERO: 1239433] like EFSA and CalEPA did; the BMDLs used by EPA in these evaluations are an order of magnitude or more below the observed first decile serum concentrations in (Budtz-Jørgensen and Grandjean 2018), as reported in (EFSA Panel on Contaminants in the Food Chain, Schrenk et al. 2020).

During the December 16, 2021 meeting of the SAB, EPA staff indicated that the study authors provided supplemental documents with additional details about the modeling to EPA staff to aid in their evaluation (<https://youtu.be/0lbtykd7ZsM?t=4145>, approximately 90 second clip); shockingly, this supplemental information was provided to neither SAB members nor the public for review. MDH strongly encourages EPA to transparently explain the rationale and details behind the decision to use BMD modeling from (Budtz-Jørgensen and Grandjean 2018) for RfD derivation. We urge EPA to provide all available information to stakeholders so a thorough review of the modeling can be done.

MDH notes that modeling information reported for other studies in the database, especially animal toxicology studies, were more thorough and in line with EPA guidelines.

Toxicokinetic Models for PFOS and PFOA

On page 36 of PFOS EPA states: *“This development, including growth, effectively decreases chemical concentrations in the body and results in lower levels than would be seen in adults of similar body size.”*

It should be noted that this is only true if exposures are the same in infants and adults. However, exposures are typically higher in early life than in adulthood when expressed on a per body weight basis.

Comments on EPA’s Discussion of the (Goeden, Greene et al. 2019) [HERO: 5080506] model:

MDH would like to clarify an incorrect point made in the PFOA document about the toxicokinetic (TK) model developed by MDH staff (Goeden, Greene et al. 2019) [HERO: 5080506]. On page 40, EPA writes *“One oversight of this model is that the rate equations are in terms of concentration, but the equations do not account for the decrease in concentration due to increasing body weight (growth dilution). This is a significant factor for infants who grow quickly.”*

The implication that the MDH model does not account for growth dilution at all is inaccurate, and we request EPA correct this in the final document. Growth dilution can be accounted for in two ways: dilution of the incoming dose into the present value of the body volume, and dilution of the body burden (accumulated from past doses) into the present value of the body volume. The current MDH

concentration-based model accounts for the former but not the latter. The (Goeden, Greene et al. 2019) [HERO: 5080506] model also includes body weight increases and changes in volume of distribution (V_d). One potential limitation of the (Goeden, Greene et al. 2019) [HERO: 5080506] model is the use of PFAS serum concentration instead of PFAS mass, which estimates slightly higher serum concentration as body weight increases.

Recently, after consultation with EPA staff, MDH created a mass-based model to compare to the concentration-based model published in 2019. Our evaluation revealed several things:

- At steady state, there is no appreciable difference between the two models; all impact is during infancy.
- Mass-based peak concentrations are lower than concentration-based (approximately 20% lower for PFOS and 27% lower for PFOA) indicating that the difference between the mass-based and concentration-based models is slight, not significant.
- When results from the concentration-based model and mass-based model were compared to infant serum PFOS/PFOA concentrations reported in (Fromme 2010) [HERO: 1291085], the concentration-based model more closely matched the data reported by Fromme and colleagues. In contrast, EPA does not provide any information regarding how well the modified Verner model matches the available empirical data.

Accordingly, in MDH's assessment, the concentration-based model is not overly conservative; that is, it does not significantly overpredict serum concentrations in growing individuals, it appears to represent the empirical data more accurately, and the difference from a mass-based model is less than 30 percent. We request that EPA correct the mischaracterization in their documents.

Comments on EPA's Selection and Modifications to the (Verner 2016) [HERO: 3299692] model:

EPA selected the (Verner 2016) [HERO: 3299692] model for human dosimetry. EPA states that several parameters of the model were adjusted, including revised curves and linear interpolation for body weight and updates to umbilical cord:maternal serum and breast milk:maternal serum PFOS and PFOA ratios. Such parameters are vital in any TK model, and thorough reporting is essential for evaluation. However, there is a general lack of detail about adjustments made to and inputs used for the Verner model.

For example, the breast milk:maternal serum ratios provided in Table 20 of the PFOA document list only mean/median ratios without standard deviations/errors; there is no information provided that conveys that range of variability within those studies. For cord:maternal serum ratios, the reader is directed to Appendix D, which is an overview of the literature but does not clearly state which ratios were used in the model. EPA states that breastfeeding is assumed to end at one year of age and exposure at background levels begins. However, there is no value provided for presumed background levels of PFOA/PFOS exposure. The POD_{HED} or candidate RfD_{HED} could presumably be used as background in the calculations, but no specific explanation is provided. In addition, exposures higher in early life can be greater than in adulthood when expressed on a per body weight basis. There is no explanation as to why it is appropriate to assume a static background exposure across lifestages. A comprehensive table of the

values and range of parameters used in the model and descriptions of why those values were selected are essential.

Additionally, a figure showing simulated serum levels across a lifespan for the model is missing; this figure is necessary for understanding overall TK patterns. Given the critical BMDL is based on serum levels at age 5, it is unlikely those individuals have reached steady-state by the developmental period of interest. This timepoint (5 years of age) would also be several years after a peak serum concentration is likely to have occurred. A visualization of the model outputs would aid greatly in understanding what serum level and timepoint EPA considered to be relevant and whether there were time periods that serum concentration exceeded the POD/candidate RfD internal dose.

Apparent assumptions of this model include that breastfeeding was limited to one year, that there was a constant breast milk:maternal serum ratio, and that weaning was an immediate process (the infant transitioning from a fully breast milk diet to background exposure at one year). In addition, it appears that central tendency values were used for the transfer ratios, half-life, and possibly breastmilk intake (the intakes in Verner's model for infants up to 12 months of age are based on (Arcus-Arth, Krowech et al. 2005) [HERO: 1005802] and appear to approximate the mean values listed in the EPA Exposure Factors Handbook (USEPA 2011) [HERO: 7485096]). MDH questions the basis of these assumptions and has concerns that the model is not adequately protective for infants with higher-than-average exposure rates.

In total, the generalized descriptions provided for the (Verner 2016) [HERO: 3299692] model precludes replication by reviewers. Many more specific details are required for a critical review of EPA's methodology.

Relative Source Contribution

At the start of the RSC discussion (Section 5.1.1 (PFOA), 5.1 (PFOS), the RSC is defined as "the portion of an exposure...equal to the RfD that is attributed to drinking water." Later, RSC estimates are described that are based on an evaluation of intake from water relative to *total intake*, not relative to the *RfD*. These estimates of water intake represent consumption of contaminated water at a level whose significance relative to the proposed MCLG is not known. It is problematic to determine an RSC based on the relation of water and non-water intakes to each other, without the context of the RfD.

In the same section, the documents state that a higher RSC value may be used for populations with higher levels of exposure from water or other sources. This is not an appropriate function for an RSC value, as it would tend to make health-based guidance values higher (less stringent) for exposed populations than for non-exposed populations.

The RSC discussion does not include a full exposure assessment. It seems incomplete, as estimates of exposure are not presented for many of the media considered. Estimates of concentrations in various media are provided (Section 5.1.3 (PFOA), 5.3 (PFOS)), but what is the exposure level in mass of chemical per kilogram of body weight per day? That is required to relate the data to the question at

hand, i.e., the RSC. Exposure is described for the EFSA report on dietary exposure, but not for other media.

Estimates of current daily intakes may not be an appropriate metric of exposure for these chemicals because of their long elimination half-lives. Serum concentrations, which represent an integral of intake over time, may be a better indicator of current body burden. EPA utilizes the serum concentration as a dose metric yet does not discuss the existence of multiple biomonitoring datasets. NHANES has reported serum levels of PFOA and PFOS in the general population since 1999. The most recent data available is from 2017-18. EPA does not discuss the possibility of using this dataset to inform exposure in the general US population. The draft RfD for PFOA, expressed as a serum concentration, is 0.017 ng/mL [$RfD_{InternalDose} = POD_{InternalDose} / UF-TOT = (0.17 \text{ ng/mL}) / (10)$]. For comparison, the 2017-18 NHANES reports geometric mean and 95th percentile PFOA serum concentrations of 1.42 and 3.77 ng/mL, respectively. For PFOS, the draft RfD expressed as a serum concentration is 0.054 ng/mL and the 2017-18 NHANES reports geometric mean and 95th percentile PFOS serum concentrations of 4.25 and 14.6 ng/mL, respectively. This would suggest that toxicologically significant levels of PFOA and PFOS are commonplace in the U.S. population.

The NHANES data are available at: https://www.cdc.gov/exposurereport/pfas_early_release.html (CDC 2021)

Infant exposures should be considered in this evaluation because they are a sensitive (and potentially highly exposed) sub-population. Elimination of PFOA and PFOS through lactation is described in Section 3.2.1.4, “Excretion,” in both documents. Nursing infants are therefore exposed to PFOA and PFOS at the start of life, and the long elimination half-life suggests the effects on PFOA levels in the body would persist. Early life exposure is also elevated by placental transfer from mother to infant during gestation, as described in Section 3.2.1.2.4 of the draft PFOA and PFOS Draft MCLG documents.

EPA has mischaracterized the steps of the EPA Exposure Decision Tree process as outlined in the (USEPA 2000) guidance for developing ambient water quality criteria. At the end of Section 5 (5.1.4 (PFOA), 5.4 (PFOS)), Box 8B is selected because “information is not available to quantitatively characterize exposure from these different sources.” However, in Figure 4-1 of the EPA (2000) guidance document, the question that leads to Box 8B says nothing about *quantitatively* characterizing exposure; it merely asks if there is *some information* available to characterize exposure. In our view, the available data (including the data presented in the draft documents as well as biomonitoring data sets as described above) constitute “some information,” and the correct outcome is Box 8C. Alternatively, the available data may meet the criteria outlined in Box 3 (are adequate data available to describe central tendencies and high-ends for relevant exposure pathways?). If answered in the affirmative, this would lead to a similar result as Box 8C, i.e., use the subtraction or percentage methods in Box 12 or Box 13, as applicable. Even if the outcome from Box 3 of the Decision Tree is in question, the question in Box 9 is critical to the PFOS and PFOA draft MCLG documents: “Are exposures from multiple sources...potentially at levels near (over 80%), at or in excess of the RfD?” Given the low RfD values discussed in these draft documents, this is a question worth addressing through comparison to the available exposure data.

The model in (Hu, Tokranov et al. 2019) [HERO: 7922429](described in 5.1.4 (PFOA), 5.4 (PFOS)) modeled *plasma* concentrations, but the text here refers to modeled/predicted *serum* concentrations. Is

there a distinction being made? Did the evaluation of this paper consider (1) the age of the samples, and (2) the question of assuming steady-state conditions when some populations of concern may be experiencing exposures that exceed the levels indicated by their current intake (due to a long elimination half-life)?

Cancer Slope Factor Development

For PFOA cancer slope factors (CSFs) based on animal toxicological data, more detail is needed in describing how animal PODs were converted to HEDs. A minor additional point: readers are directed to Appendix C for modeling details while these details are actually contained in Appendix B; this type of error occurs throughout the documents.

For PFOA CSFs based on epidemiological data, it is not entirely clear how EPA's analysis differs from CalEPA's (CalEPA Office of Environmental Health Hazard Assessment 2021). It is stated that "EPA calculated CSFs for RCC [renal cell carcinoma] from Shearer, 2021 based on the method used by CalEPA", but CalEPA's CSF was 2-fold lower than EPA's central tendency slope and 6-fold lower than EPA's 95th percentile confidence interval (CI). Additional discussion of EPA's methodology and any differences from CalEPA's is necessary.

The serum-based PFOA CSF for the 95th percentile CI is 3.52×10^{-6} per ng/mL. The value 1 ng/mL is close to the 2017-018 NHANES geometric mean (1.42 ng/mL) and nearly 4-fold lower than the 95th percentile (3.77 ng/mL). A discussion from EPA about the consistency between the proposed CSF and measured incidence of RCC in the American population would be useful context.

Draft Framework for Estimating Noncancer Risk for PFAS Mixtures

MDH supports EPA's proposal of a target organ-specific hazard index approach to PFAS mixtures and, indeed, chemical mixtures more generally. This approach has been used by MDH since the inception and promulgation of our Health Risk Limit rule in 1993. MDH and other state agencies in Minnesota have successfully used this approach to understand potential health impacts for those affected by PFAS contamination. It is a powerful tool for directing resources to Minnesotans most affected by PFAS contamination, especially when there are multiple PFAS present.

As noted by EPA, PFAS do have some special considerations for this type of additivity. The use of human equivalency doses (HEDs) in this framework will be key, given the importance of chemical-specific TK information for PFAS when deriving RfDs. A more thorough discussion of these challenges within the document would be appreciated.

Analysis of Cardiovascular Disease Risk Reduction

The decision to perform a risk/benefit analysis pertaining to cardiovascular effects is premature and puzzling, given that the proposed critical study concerns the immune system, and all other candidate RfDs are based on developmental endpoints. EPA states that cardiovascular disease was not considered for derivation of PODs since findings for an association were mixed and the study related to total

cholesterol (the only serum lipid considered for derivation of a POD) was specifically excluded from candidate RfD consideration (rationale not stated). When it is appropriate to do such an analysis in the MCL process, a focus on the critical effects (developmental/immune) would be more relevant.

Conclusions

Minnesota has eagerly awaited progress on PFAS regulation from the federal government. MDH has continued to engage in research and response actions regarding PFAS since the early 2000s, when the 3M Corporation disclosed that PFAS-containing wastes had been disposed of in several sites that contaminated groundwater across a wide area of the eastern Twin Cities metropolitan area. Staff at MDH have done extensive research on the toxicology, toxicokinetics, environmental fate and transport, laboratory analysis, and treatment of these chemicals in support of needed public health interventions to reduce exposures to PFAS through drinking water, fish consumption, and other exposure pathways.

Considering the significant deficiencies identified thus far, our position is that at least one more round of public drafts is required. MDH requests revision and release of updated drafts of the PFOA and PFOS proposed approaches for SAB/public review and comment. MDH also recommends expanding the SAB to include additional expertise in chemical risk assessment to ensure a full, transparent, and accurate evaluation of EPA's proposed approaches.

MDH recognizes the unique challenges associated with PFAS risk assessment and welcomes the opportunity to provide comments on EPA's proposed approaches. We look forward to a continued partnership. If you have any questions regarding these comments, please contact Dr. Helen Goeden at 651-201-4904, helen.goeden@state.mn.us, or James Kelly at 651-201-4910, james.kelly@state.mn.us. Sincerely,

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