July 19, 2019

Commissioner Martin Suuberg
Massachusetts Department of Environmental Protection
One Winter Street, 2nd Floor
Boston, MA 02108

Re: 2019 Proposed PFAS MCP Revisions

Dear Commissioner Suuberg,

Thank you to the Massachusetts Department of Environmental Protection for the opportunity to provide comments on the proposed revisions to the Massachusetts Contingency Plan to develop standards for PFAS chemicals in water. We are glad to see the Commonwealth of Massachusetts moving forward with the development of PFAS standards.

We are co-directors and project leaders of the Sources, Transport, Exposure & Effects of PFASs (STEEP) Superfund Research Program, led by the University of Rhode Island in partnership with the Harvard T.H. Chan School of Public Health's Department of Environmental Health and Silent Spring Institute. STEEP's team members contribute decades of interdisciplinary experience in developing methods for chemical detection in the environment, determining health impacts of chemical compounds and where in the body these compounds accumulate, training the next generation of scientists, engaging communities to improve well water quality and awareness, and communicating complex science to a variety of audiences. Here in Massachusetts, we are conducting a private well water testing program on Cape Cod to characterize exposures from well water and identify potential sources, and we are studying the fate and transport of PFAS compounds as they move through Cape groundwater and ponds and potential bioaccumulation and ecological effects.

We agree that there are serious health concerns arising from the exposure of the general public to PFASs, and that the reference doses (RfDs) developed by EPA for PFOS and PFOA are not adequately protective. Recent studies by STEEP researchers and many others indicate the potential for harmful effects resulting from low-dose exposures according to both toxicological and epidemiological research.

Importance of early-life exposures and evidence for low-dose effects

STEEP research is focusing on risks to human health from early-life exposures that may occur during pregnancy or through breastfeeding. PFASs can pass the placental barrier,[1] thereby allowing a mother's PFAS body burden to be transferred to her child. The shared exposure continues postnatally, as PFASs

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are transferred through breast milk, and longer durations of breastfeeding result in increased serum PFAS concentrations in children.[2] Since adverse effects on the next generation, for instance on the development of the immune system, may have long-term adverse health implications, we believe that a substantial amount of precaution is appropriate to protect the most vulnerable part of the population.

Epidemiological research led by STEEP researcher Philippe Grandjean on the Faroe Islands has shown associations between PFAS exposures in young children and suppressed antibody response to vaccines.[3] Based on benchmark dose calculations of immunotoxic effects, Grandjean and Budtz-Jorgensen[4] suggested that 0.1 ng/mL serum would be an appropriate benchmark dose level for PFOS and PFOA, which corresponds to 1 ng/L when converted to drinking water concentrations, assuming a ratio of 1:100. More recently, Budtz-Jorgensen and Grandjean[5] extended these benchmark dose calculations to simultaneously account for exposures to the five long-chain PFAS chemicals included in the draft standard.

The Minnesota Department of Health developed a toxicokinetic model that accounts for accumulation of PFASs *in utero* and transfer of PFAS compounds via breast milk.[6] The New Hampshire Department of Environmental Services recently issued revised draft MCLs that accounted for this model. The resulting draft MCLs, which are lower than initially proposed, are 12 ng/L PFOA, 15 ng/L PFOS, 11 ng/L PFNA, and 18 ng/L PFHxS. Still, the model does not appropriately take into regard that exposure during prenatal or infancy development can cause lasting impairment of organ functions with associated disease risks. This concern suggests that further lowering of the MCLs is needed.

Current evidence on rodent models have shown that low-dose PFOA exposures can impair mammary gland development.[7-10] Altered breast development associated with low-dose PFOA exposure is concerning because of the potential to disrupt lactation. PFOA exposure in mice was associated with reduced mammary differentiation and altered milk protein gene expression.[9] In humans, elevated serum PFOA was associated with early termination of breastfeeding in a cohort of U.S. mothers[11] and PFOA, PFOS, PFNA, and PFDA serum concentrations were associated with shorter duration of breastfeeding in a cohort of mothers in the Faroe Islands.[12] As noted by researchers at Silent Spring Institute and others, altered mammary gland development may increase breast cancer susceptibility later in life.[7, 13] The New Jersey Department of Environmental Protection (NJDEP) noted that delayed mammary gland development, along with increased liver weight, were the two most sensitive non-carcinogenic endpoints associated with PFOA exposure.[14] NJDEP concluded that the target serum concentration to be protective of delayed mammary gland development was below the median serum PFOA level in the general population. While NJDEP's recommended MCL was not based on this endpoint due to a lack of precedent for using this endpoint as the basis for risk assessment, NJDEP applied an extra uncertainty factor to account for this and other sensitive endpoints.

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Additional compounds for consideration

We applaud MassDEP's approach for the proposed standard that includes the sum concentrations of PFOS, PFOA, and four additional PFAS compounds. This approach recognizes the extreme persistence of PFAS compounds as a class and the long human half-lives of PFASs, especially long-chain compounds. Beyond the six compounds in the draft standard, we note that there are many more PFASs of concern.

There is ample evidence that MRLs ought to be considered for PFUnDA (C11) and PFDoDA (C12), along with PFDA that has already been added to the original five PFAS compounds in the proposed GW-1 standard. In addition to the continual exposures to these compounds, the health effects and toxicokinetic behavior of the C10-C12 compound all show similarities to the behavior of PFNA. The human half-lives for PFCAs generally increase with chain length. Geometric mean human half-lives for PFDA and PFUnDA were estimated to be 7.1 and 7.4 years, respectively, in males and older females, more than twice the estimated half-life for PFNA.[15] As noted in the ATSDR draft toxicological profile, PFDA, PFUnDA, and PFDoDA have been associated with thyroid disorders and adverse birth outcomes in epidemiological studies.[16] PFDA and PFUnDA have been linked with serum lipid outcomes, neurodevelopmental outcomes and prostate cancer. PFUnDA and PFDoDA have been linked to suppressed antibody response to vaccines and decreases in childhood growth. PFDA has been linked with male reproductive outcomes, and adverse pregnancy outcomes, and PFUnDA has been linked to diabetes. Mother-child transfer efficiencies for these compounds are often greater than PFNA, as indicated by the low maternal-fetal and maternal-infant ratios reported in the recent ATSDR toxicological profile on PFASs.

In addition, emerging research demonstrates that select shorter-chain alternatives may bioaccumulate to the same extent or to a greater degree than legacy compounds such as PFOA or PFOS.[17-21] Pharmacokinetic models suggest that shorter-chain alternatives may be equally toxic compared to legacy compounds after adjusting for differences in toxicokinetics.[22] Shorter-chain alternatives replacing legacy PFASs continue being produced and show widespread environmental occurrence. Perfluoroalkyl ether carboxylic acids (i.e. HFPO-DA or "GenX"), polyfluoroalkyl carboxylic acids, and polyfluorinated alkanesulfonates and sulfates persist in air, surface water, and drinking water downstream from release sources.[23-27]

Support for Relative Source Contribution of 20%

A new analysis led by STEEP researcher Elsie Sunderland supports the applicability of the 20% default relative source contribution estimate.[28] Using pharmacokinetic modeling and blood and drinking water samples archived from 1989-1990, these authors estimated that contributions of drinking water to overall PFAS exposures ranged from around 3% for PFOS to 34% for PFHxS.

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Consideration of a class-based approach

As a class, PFAS compounds are united in their extreme persistence and mobility. In recognition of concerns about chemicals that are very persistent and very mobile, the European Union has proposed adding a "very persistent very mobile" (vPvM) criteria to the European chemical regulatory program REACH. To the extent possible, PFASs should be considered as a class, or relevant subclasses, rather than attempting to regulate them one at a time. The scientific community has repeatedly acknowledged similar physicochemical characteristics linking >4,000 PFASs and has suggested PFASs be considered and regulated as a group or as subgroups.[29-31] Most recently, the governments of the EU countries have urged the European Commission to generate a joint strategy on PFASs, treating all the many individual compounds as a group and recommending that they be approved only for essential uses.

The current regulatory paradigm essentially assigns zero toxicity to PFAS not included in GW/MCL standards. While setting a total PFAS standard will be difficult to establish, it would be advisable to include a measure of total PFAS on a regular basis to be able to assess how abundant non-targeted PFASs are. This approach would allow Mass DEP to be alerted to the presence of other PFASs that might become threats to public health.

Analytical considerations of the proposed standards

Based on improvements of analytical instrumentation over the last few years, the proposed standards can easily be quantified using common HPLC-MS/MS based instrumentation. While the U.S. EPA only asked laboratories to report at levels above 20-90 ng/L, depending on the compound, during the UCMR3 testing in 2013-2015, lower reporting limits are easily achieved. Even at the time of the UCMR3 reporting, major analytical laboratories had much lower internal reporting limits, leading to substantial underestimates of PFAS abundance in U.S. public water systems.[32] Since then, reporting limits in the lower ng/L range are easily and routinely achieved by commercial, regulatory, and academic laboratories.

Thank you, once again, for inviting our comments. Please contact us if you wish to discuss any of the above issues further.

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