July 19, 2019

Stephanie Cooper
Deputy Commissioner for Policy and Planning
Department of Environmental Protection
One Winter Street
2nd Floor
Boston, MA 02108

Re: Proposed Revisions to the Massachusetts Contingency Plan (MCP, 310 CMR 40.000), Proposed GW-1 standards and RCGW-1 Reportable Concentrations for per- and polyfluoroalkyl substances (PFAS)

Dear Deputy Commissioner Cooper:

The Chemical Products and Technology Division of the American Chemistry Council (ACC/CPTD) submits the following comments on the Massachusetts Department of Environmental Protection's (DEP) proposal to establish groundwater and soil standards and reportable concentrations for perfluorohexanesulfonic acid (PFHxS), perfluorohepatanoic acid (PFHpA), perfluorooctanoic acid (PFOA), perfluoroctane surfonate (PFOS), perfluorononanoic acid (PFNA), and perfluorodecanoic acid (PFDA). ACC/CPTD represents companies interested in ensuring that regulations related to these substances, like the DEP proposal, incorporate the best available science. ACC/CPTD is concerned about the proposal to (1) apply an additional uncertainty factor to the US Environmental Protection Agency's (USEPA) assessment of PFOS and PFOA exposure in drinking water and (2) use this revised value as a basis for combined groundwater and soil standards for PFHxS, PFHpA, PFOS, PFOA, PFNA, and PFDA.

USEPA recently proposed interim cleanup levels for PFOS and PFOA, but not for any additional per- and polyfluoroalkyl substances (PFAS), that are intended to serve as recommendations for sites in Massachusetts and throughout the nation. The establishment of separate state levels has the potential to create confusion and conflict about the applicable cleanup targets for these substances.[1] Applying any limit for PFOS and PFOA to other PFAS is not supported by the available science, moreover, and could lead to unnecessary cleanups. Finally, DEP's proposal to establish soil cleanup levels for the six PFAS is premature considering

[1] The proposed reportable concentration for the six PFAS of $2X10^{-5}$ milligrams/Liter (mg/L) is equivalent to the proposed GW-1 standard of 20 parts per trillion. Consequently, ACC's concerns with the GW-1 proposal also apply to the proposed reportable concentrations.

that USEPA has not yet developed validated techniques for measuring PFAS in soil and sediments.

DEP Should Review EPA Cleanup Levels Before Proceeding

Earlier this year, EPA's Office of Land and Emergency Management proposed interim groundwater cleanup recommendations for PFOS and PFOA. ACC and other stakeholders submitted extensive comments on the proposal which are currently under review by the Agency.[2] Once finalized the recommendations will identify cleanup levels for the two substances that are protective of human health and the environment. In the absence of a federal maximum contaminant level (MCL) or other federal standard, the pending recommendations will serve as the applicable or relevant and appropriate requirement (ARAR) for federally-administered cleanups under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA). Creating separate state levels for these two substances would create confusion about applicable cleanup targets among affected parties and levels of protection among the public.

The Application of a Database Uncertainty Factor is Inappropriate

DEP has proposed the addition of a 10-fold uncertainty factor to the reference doses (RfD) for PFOS and PFOA "to account for more sensitive toxicity endpoints." According to the proposal, the addition of a database uncertainty factor (UF_D) is suggested for potential immunotoxicity effects for PFOS and liver and mammary gland effects for PFOA. According to DEP, the proposals to add additional uncertainty factors are based on draft documents from the federal Agency for Toxic Substances and Disease Registry (ATSDR) and the New Jersey Department of Environmental Protection – neither of which have been finalized.

According to US Environmental Protection Agency guidance, the UF_D is intended to account for the potential for deriving an under-protective RfD as a result of an incomplete characterization of the chemical's toxicity. In addition to identifying toxicity information that is lacking, review of existing data may also suggest that a lower reference value might result if additional data were available. Consequently, in deciding to apply this factor to account for deficiencies in the available data set and in identifying its magnitude, the assessor should consider both the data lacking and the data available for particular organ systems as well as life stages.[3] An UF_D is generally applied when reproductive and developmental toxicity studies are missing since they have been found to provide useful information for establishing the lowest no

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[2] ACC's comments on the EPA proposal are enclosed.

[3] USEPA Risk Assessment Forum. A review of the reference dose and reference concentration processes. EPA/630/P-02/002F (December 2002). https://www.epa.gov/sites/production/files/2014-12/documents/rfd-final.pdf

adverse effect level.[4] If the RfD is based on animal data, a factor of 3 is often applied if either a prenatal toxicity study or a two-generation reproduction study is missing, or a factor of 10 may be applied if both are missing.

The reproductive and development data bases for PFOS and PFOA are robust, however, and do not suggest the need to account for an incomplete characterization of toxicity. Similarly, the potential immunotoxic effects of PFOS have been studied in both laboratory animals and humans. The results of these studies are inconsistent and both EPA[5] and Health Canada[6] have questioned the relevance of immune system effects observed in mice and the small antibody variations seen in epidemiology studies to adverse health effects in humans. It is inappropriate, therefore, to conclude that immunotoxic effects represent a more sensitive health effect such that a modifying factor of 10 should be included in the assessment of PFOS.

Both the liver and mammary gland effects reported in animal studies of PFOA are associated with activation of the peroxisome proliferator activated receptor alpha (PPARα) and are of questionable relevance to humans. This conclusion is supported by the conclusions of the C8 Health Project[7] and recent human data reported by Convertino *et al.* (2018)[8] which provide strong evidence that the liver effects are a rodent-specific adaptive response. Alterations in mammary gland development were not observed in a study of offspring of wild type, PPARα-null, or PPARα humanized mice following *in utero* exposure to PFOA.[9] In a multi-generational study in CD-1 mice, moreover, the investigators noted that the delay in mammary

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4 Ibid, at 4-45.

5 USEPA. Health effects support document for perfluorooctane sulfonate (PFOS). EPA 822-R-16-202 (May 2016), at 4-7.

6 Health Canada. Guidelines for Canadian Drinking Water Quality: Guideline Technical Document — Perfluorooctane Sulfonate (PFOS). Water and Air Quality Bureau, Healthy Environments and Consumer Safety Branch. Ottawa, Ontario. Catalogue No. H144-13/9-2018E-PDF. (2018). https://www.canada.ca/content/dam/canada/health-canada/migration/healthy-canadians/publications/healthy-living-vie-saine/guidelines-canadian-drinking-water-quality-guideline-technical-document-perfluorooctane-sulfonate/PFOS%202018-1130%20ENG.pdf

7 The C8 Health Project is a large epidemiological study conducted in communities surrounding a manufacturing facility in Parkersburg, West Virginia that used PFOA from the 1950s until 2002. The study included over 32,000 adult residents and facility workers. The Science Panel formed as part of this project concluded that "there is not a probable link between exposure to C8 (also known as PFOA) and liver disease. The C8 Science Panel conclusions are summarized at http://www.c8sciencepanel.org/prob_link.html.

8 Convertino M *et al.* Stochastic pharmacokinetic-pharmacodynamic modeling for assessing the systematic health risk of perfluorooctanoate (PFOA). *Toxicol Sci* 163(1) 293-306 (2018).

9 Albrecht PP *et al.* A species difference in the peroxisome proliferator-activated receptor α-dependent response to the developmental effects of perfluorooctanoic acid. *Toxicol Sci* 131:568–582 (2013).

gland development did not appear to affect lactational support based on normal survival and growth of the second generation (F2) offspring.[10]

The Available Science Does Not Support Applying a Single Value to Multiple PFAS

DEP has proposed to apply a single groundwater standard to the sum of six PFAS that vary significantly in the availability of health effects information and metabolism. While much is known about PFOS and PFOA, less data is available for the other four substances.[11] Even in the case of PFOS and PFOA, the mechanism by which exposure to these substances causes health effects in laboratory animals is unknown.

The grouping of substances under a single standard is justified only when the substances are believed to cause health effects by the same mechanism of action.[12] This is clearly not the case for the six substances identified by DEP. Although the USEPA's lifetime Health Advisories (LHAs) for PFOS and PFOA are based on developmental effects, the critical developmental endpoints identified by EPA do not suggest a common mechanism.[13] Similar evaluations of the potential health effects of exposure to PFHxS, PFHpA, PFNA, or PFDA are not available from EPA, and the draft evaluations for PFHxS and PFNA from the Agency for Toxic Substances and Disease Registry (ATSDR) indicate that a very limited amount of data exist for these substances – particularly data related to mechanism of action. In the case of PFDA and PFHpA, ATSDR concluded that "insufficient data are available for derivation" of minimum risk levels.

Existing calculations of the risks associated with exposure to PFAS are highly dependent on estimates of the elimination half-lives of the substances. In the case of the PFAS identified by DEP, significant differences exist. While the half-life of PFHxS in humans is estimated to be on the order of 5 to 8 years, the half-life for PFHpA is estimated to be much shorter,[14] and the limited data for PFDA and PFNA do not allow for a robust estimate of their half-life.[15] While the

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[10] White SS *et al.* Gestational and chronic low-dose PFOA exposures and mammary gland growth and differentiation in three generations of CD-1 mice. *Environ Health Persp* 119(8):1070–1076 (2011).

[11] ATSDR. Toxicological profile for perfluoroalkyls - draft for public comment (June 2018). https://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=1117&tid=237

[12] EPA. Guidance for identifying pesticide chemicals and other substances that have a common mechanism of toxicity. Office of Pesticide Programs (January 26, 1999). https://www.epa.gov/pesticide-science-and-assessing-pesticide-risks/guidance-identifying-pesticide-chemicals-and-other

[13] In addition, EPA's selection of the point of departure (POD) for developmental effects for both PFOS and PFOA are not consistent with the conclusions of the authors of the papers from which they are derived.

[14] Russell MH *et al.* Inhalation and oral toxicokinetics of 6:2 FTOH and its metabolites in mammals. *Chemosphere* 120:328–335 (2015).

[15] ATSDR 2018.

estimates from occupational studies generate more conservative estimates, the half-life values derived from general population data are the most relevant to exposures considered by DEP. Using estimates from general population studies can significantly affect the estimates of the magnitude of risk associated with PFAS exposure.

Applying EPA's Drinking Water Advisory Level to Groundwater is Inappropriate and Unnecessary

The EPA LHAs for PFOS and PFOA were developed as health-based guidelines for assessing potential exposure in drinking water. They are based on a number of conservative assumptions regarding levels of water consumption, exposures among sensitive populations, and exposure to sources other than drinking water.[16] Consequently, they indicate a level of conservatism that is inappropriate and unnecessary for groundwater standards. Cleaning up groundwater to the levels proposed by DEP, moreover, is not a practical approach to protecting public health.

Although many PFAS can be readily removed from water, removal requires that the water comes in contact with granular activated carbon (GAC) or adsorbent resins. ACC-CPTD is not aware of cost-effective means for treating PFAS contamination in-situ. DEP's proposal to require cleanup of groundwater to the proposed GW-1 standard would require expensive systems to bring the groundwater to the surface for treatment or to install extensive treatment networks underground. Such systems are cumbersome and disruptive and generally must operate for extended periods of time to achieve target levels. Available evidence suggests, moreover, that short-chain PFAS like PFHpA may not be as readily adsorbed as PFOS and PFOA and may require the use of redundant beds for effective removal.[17]

Given the challenges of reducing groundwater levels of the five PFAS included in the DEP proposal, ACC-CPTD urges the Department to abandon the current proposal and base its efforts on the cleanup levels for PFOA and PFOA to be finalized by EPA.

Standard Measurement of PFAS in Soil are Still Being Developed

The DEP proposal also includes a soil cleanup limits for the same six PFAS. In addition to the concerns expressed above, ACC-CPTD notes that USEPA has not yet developed validated methods for measuring PFAS levels in soils. We urge DEP to await the results of the EPA effort before proceeding with development/revision of soil levels for these substances.

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[16] EPA. Drinking water health advisory for perfluorooctanoic acid (PFOA). EPA 822-R-16-005 (May 2016); Drinking water health advisory for perfluorooctane sulfonate (PFOS). EPA-822-R-16-004 (May 2016).

[17] McCleaf P *et al.* Removal efficiency of multiple poly- and perfluoroalkyl substances (PFASs) in drinking water using granular activated carbon (GAC) and anion exchange (AE) column tests. *Water Res* 120:77-87 (2017).

In light of the concerns discussed above, ACC-CPTD urges DEP to withdraw its current proposal in light of the pending EPA announcement of cleanup levels for PFOS and PFOA. Please feel free to contact me at (202) 249-6727 or srisotto@americanchemistry.com if you would like to discuss the information presented above.

Sincerely,

Steve Risotto

Stephen P. Risotto
Senior Director

Enclosure

June 10, 2019

Mr. Stiven Foster
Science Advisor
Office of Land and Emergency Management
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, D.C. 20460

Re: Draft Interim Recommendations for Addressing Groundwater Contaminated with Perfluorooctanoic Acid and Perfluorooctane Sulfonate; Docket No. EPA-HQ-OLEM-2019-0229

Dear Mr. Foster:

The American Chemistry Council[1] appreciates the opportunity to comment on the draft Interim Recommendations for Addressing Groundwater Contaminated with Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS). ACC is deeply concerned about the scientific basis for the draft recommendations, as well as the process that led to their issuance.

The draft guidance proposes the following recommendations –

- a preliminary remediation goal (PRG) of 70 parts per trillion (ppt) for PFOA and PFOS in groundwater used or potentially used as drinking water, based on EPA's 2016 lifetime health advisories (LHAs), and
- a groundwater screening level of 40 ppt for these two substances based on a Hazard Quotient (HQ) of 0.1.

The LHAs have not been subject to sufficiently robust peer review to support their use as PRGs. The Agency proposal to set the screening level based on an HQ of 0.1, moreover, is not supported by the available science and is not consistent with Agency policy. For the reasons outlined below, we encourage EPA to ensure that cleanup levels for PFOA and PFOS – whether issued as recommendations or as enforceable limits – be based on the weight of the best

[1] ACC represents the leading companies engaged in the business of chemistry. ACC members apply the science of chemistry to make innovative products and services that make people's lives better, healthier and safer. ACC is committed to improved environmental, health and safety performance through Responsible Care®, common sense advocacy designed to address major public policy issues, and health and environmental research and product testing.

available scientific evidence, be consistent with current EPA guidance, and be subject to robust review.

Preliminary Remediation Goals

In releasing the LHAs for PFOA and PFOS in 2016, EPA cautioned that health advisories "are non-enforceable and non-regulatory" and are intended to "provide technical information to states agencies and other public health officials on health effects, analytical methodologies, and treatment technologies associated with drinking water contamination." As such, it is inappropriate to use these values as remediation goals where no applicable or relevant and appropriate requirements (ARARs) exist – particularly in light of the concerns identified below.

EPA's Office of Water released draft health effects documents supporting the development of LHAs for PFOA and PFOS in February 2014.[2] A scientific peer review meeting was subsequently held in August 2014 during which the reviewers raised a number of questions and concerns about the draft documents.[3] Among the concerns expressed were the selection of the health end point used as a basis for deriving the reference dose (RfD) and the use of an older physiologically based pharmacokinetic (PBPK) model for predicting human serum levels.

As a result of the comments received, the Office of Water revised its selection of the key health endpoints but did not recirculate the revised analysis for appropriate peer review before finalizing the LHAs. In finalizing the advisories, the Water Office also added a recommendation that "the health advisory guideline be applied as the sum of the concentrations" of PFOA and PFOS – a recommendation that was not subject to public comment or peer review.

In finalizing the Health Advisories, moreover, the Office of Water did not agree with the recommendation to use more recent models to predict human serum levels, despite a reviewer's caution that –

> The choice of using the empirical model over the more recent physiological models may be a weakness [as] our understanding of transporters advance. The evolution of chemical-specific PBPK models for use in risk assessment and regulatory applications has repeated itself several times. [That] is, the first empirical non-physiological model(s) or PBPK models contain hypotheses generating ideas and later models test some of these hypotheses, especially if

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2 http://www.gpo.gov/fdsys/pkg/FR-2014-02-28/pdf/2014-04455.pdf

3 Versar. Peer review summary report – external peer review of EPA's draft health effects documents for perfluorooctanoic acid and perfluorooctane sulfonate (PFOS). Prepared for EPA Office of Water, Office of Science and Technology (November 17, 2014). https://www.regulations.gov/document?D=EPA-HQ-OW-2014-0138-0027

> additional experimental data become available. In the case of PFOA and PFOS, the EPA selected not to use the most recent PBPK models for PFOA and PFOS, but instead use a computational empirical based model . . . that was the first attempt to quantitatively interpret the kinetics of PFOA and PFOS across species of laboratory animals.[4]

In its response, the Office of Water noted that "a [PBPK] model for PFAS would be preferable because it would allow extrapolation between species, provide better estimates of chemical-specific parameters, and allow estimation of chemical concentration in the specific tissues for which toxicity is observed."[5] The response reasoned, however, "that the state of the science has not yet developed such that extrapolation between species is possible." This is no longer the case.

Since the LHAs were finalized, two significant scientific work products have become available that advance our understanding of the pharmacokinetics of PFOA and PFOS and allow for a more effective extrapolation between species. The first of these is the recently completed assessments of maximum allowable concentrations for PFOA and PFOS in drinking water from Health Canada that incorporate the newer PBPK models recommended by the peer reviewer.[6] The second new source of information is the availability of data from a human clinical trial conducted to explore the potential therapeutic action of ammonium perfluorooctanoate.[7] The clinical data includes dose-response, time-course measurements of PFOA that allow better scientific estimates of the half-life of PFOA in humans. From the standpoint of using knowledge of the kinetics in humans as the foundation for developing LHAs, these recent studies are the best available science.

Large pharmacokinetic differences exist between humans and animals for PFOA and PFOS, with lower clearance (i.e., higher half-life values) reported for humans than for rats, mice, and non-human primates. These differences can result in higher target tissue doses in humans when exposed to the same external doses as laboratory animals. To better account for these interspecies toxicokinetic differences in developing the LHAs, the Office of Water calculated a

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4 Ibid, at 39.

5 EPA. EPA response to external peer review comments on EPA draft documents; health effects support document for perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS). Office of Water, Office of Science and Technology (May 2016). https://www.regulations.gov/document?D=EPA-HQ-OW-2014-0138-0036

6 https://www.canada.ca/en/health-canada/services/environmental-workplace-health/reports-publications/water-quality.html

7 This work is summarized in Convertino M *et al.* Stochastic pharmacokinetic-pharmacodynamic modeling for assessing the systemic health risk of perfluorooctanoate (PFOA). *Toxicol Sci* 163(1):293–306 (2018). https://doi.org/10.1093/toxsci/kfy035

human equivalent dose (HED) by adjusting the serum concentration in rodents exposed via the drinking water route by the rate of estimated clearance (CL) of the substance from the human body. The CL was calculated using the estimated volume of distribution and serum elimination half-life estimated from empirical data.[8]

Compared to the approach that relied on older scientific data, the internal-dose ratios predicted by the more recent PBPK models indicate that the interspecies extrapolations for PFOA and PFOS are highly dose dependent and result from nonlinear toxicokinetics.[9] Evidence that the half-life in humans is dose-dependent indicates that a single interspecies extrapolation factor such as that used by EPA's Office of Water is not scientifically supportable for either PFOA or PFOS. Instead, using the new PBPK model to derive data for environmentally relevant exposure levels is a more scientifically appropriate approach for addressing the issue of nonlinear toxicokinetics and its impact on interspecies extrapolation.

Accordingly Health Canada compared dose metrics predicted by the various animal PBPK models to calculate a CL ratio between species (CL_{animal}/CL_{human}).[10] Using the model data to derive the CL ratio allows for a more appropriate comparison of exposures and doses of the same magnitude.[11] Based on this approach, Health Canada's analysis indicates that the EPA significantly underestimates the human clearance rate in deriving the LHAs.[12] As a result, the HEDs that EPA derived to calculate the 2016 LHAs are up to 500 times lower than what the new, best available science indicate.[13]

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[8] The volume of distribution is defined as the volume of blood (in milliliters per kilogram) in which the amount of a chemical would need to be uniformly distributed to produce the observed blood concentration. Half-life is a measure of the time (in days) required to eliminate one half of a quantity of a chemical from the body.

[9] Loccisano AE *et al.* Comparison and evaluation of pharmacokinetics of PFOA and PFOS in the adult rat using a physiologically based pharmacokinetic model. *Reprod Toxicol* 33(4):452–467 (2012). https://doi.org/10.1016/j.reprotox.2011.04.006

[10] For each species, the PBPK model was used to predict internal doses for a broad range of oral doses. Model simulations were continued until steady-state conditions or expected lifetimes were reached (Loccisano *et al.* 2012).

[11] Health Canada. Guidelines for Canadian Drinking Water Quality: Guideline Technical Document - Perfluorooctane Sulfonate. Ottawa, Ontario (2018), at 36.

[12] Based on the clearance ratio calculation, Health Canada adjusted the animal doses by factors of 0.0104 and 0.071 for PFOA and PFOS, respectively, compared to EPA's adjustment factor of 0.00014 for PFOA and 0.000081 for PFOS.

[13] Other assumptions may have changed since the 2016 LHAs were developed. For example, the water consumption rate for lactating women, on which the LHAs were based, was reduced from 55 to 47 milliliters per kilogram per day in the February 2019 update of Chapter 3 of EPA's Exposure Factors Handbook. https://www.epa.gov/expobox/about-exposure-factors-handbook

The conclusions reached by Health Canada also are supported by recent observations from a clinical study that explored the potential therapeutic action of PFOA (see Figure 1). These studies indicate that PFOA levels in humans may reach steady state after only about 25 weeks of exposure and suggest that half-lives in humans may be as short as 5 weeks[14] – in contrast to the 2.3 years assumed by EPA for the 2016 LHA.[15] While these newer human clinical data should be interpreted with caution, since the kinetics from these studies may not reflect the average population, these scientific investigations represent one of the few longitudinal studies of plasma levels in humans currently available.

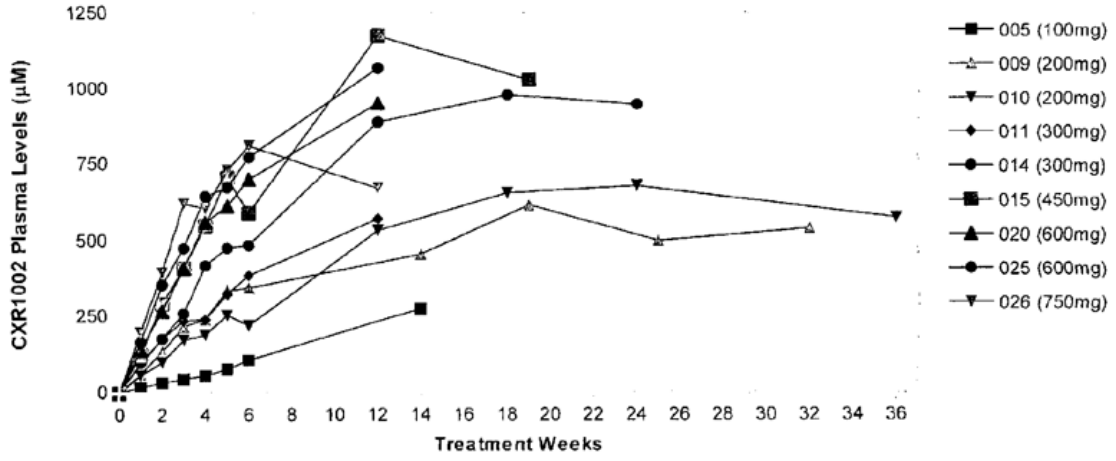

Figure 1. Plasma levels of PFOA after administration of ammonium perfluorooctanoate.[16]

Screening Levels

The description of EPA's derivation of the draft screening level is brief, but indicates that the Agency used the RfDs generated by the Office of Water and an HQ of 0.1. In explaining the rationale for not using the default HQ of 1.0 per Superfund guidance, the draft points to the specific and limited purpose of a screening level, the additive toxicity of PFOA and PFOS, and the possible co-location of other PFAS compounds for which toxicity values may not currently be available.

[14] Shinya I. Pharmacokinetics 101. *Paediatr Child Health* 16(9): 535-536 (2011). https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3223885/

[15] https://www.epa.gov/sites/production/files/2016-05/documents/pfoa_health_advisory_final_508.pdf

[16] Elcombe CR *et al.* Compositions comprising perfluoroctanoic acid. US Patent application publication no. US 2013/0029928 Al (January 31, 2013). Enclosed.

As explained in the previous section, the RfDs calculated as part of the derivation of the LHAs for PFOA and PFOS do not reflect the best and most current science and should be evaluated in light of the information that has become available since they were developed. While Superfund guidance does suggest that an HQ of 0.1 may be used as an initial screening target, the guidance explains that such an approach should be applied "where more than one chemical with the same toxic endpoint might be present."[17] Such an assumption clearly cannot be made for the other PFAS for which toxicity values are not available. It should be vigorously evaluated in light of a substantial body of evidence to support differences across PFAS in terms of physical-chemical properties, biological activity, and clearance rates.[18] Even for PFOA and PFOS, the Office of Water's conclusion that the concentrations of the two substances can be combined has not been subject to appropriate, independent peer review and is not substantiated by the scientific literature.

In addressing the presence of multiple contaminants at a location, EPA's Superfund guidance provides the following advice for modifying the target hazard quotient (THQ) to generate the appropriate screening level –

> The THQ input . . . can be modified from the default of 1. How much it should be modified is a user decision, but it could be based upon the number of contaminants being screened together. For example, if one is screening two contaminants together, then the THQ could be modified to 0.5. If ten contaminants are being screened together, then the THQ could be modified to 0.1.[19]

Notwithstanding the need for appropriate peer review the Office of Water's suggestion that PFOA and PFOS be treated as equally potent in terms of toxicity, the THQ used for developing the screening levels for PFOA and PFOS when found together should be no lower than 0.5.

In addition, consistent with existing practice when developing risk-based screening levels, the screening level at a HQ of 1 should also be presented. For example, EPA Regional Screening Level tables are released in two versions. One at a cancer risk of 1×10^{-6} and a HQ of 0.1, and one at a cancer risk of 1×10^{-6} and a HQ of 1. This allows the public to understand that the lower screening level is not appropriate for screening all sites and that under many conditions screening with a HQ of 1 is applicable.

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17 EPA. Regional Screening Levels (RSLs) - User's Guide. Office of Land and Emergency Management. Washington, DC (May 2019). https://www.epa.gov/risk/regional-screening-levels-rsls-users-guide (emphasis added)

18 Patlewicz G *et al.* A Chemical Category-Based Prioritization Approach for Selecting 75 Per- and Polyfluoroalkyl Substances (PFAS) for Tiered Toxicity and Toxicokinetic Testing. *Environ Health Perspec* 127(1):1-5 (2019). https://ehp.niehs.nih.gov/doi/10.1289/EHP4555

19 EPA RSLs 2019.

ACC urges EPA to conduct a robust review of the best available science related to PFOA and PFOS and to the application of the OLEM guidance for identifying remediation goals and screening levels before making recommendations on these two substances. Please feel free to contact me at srisotto@americanchemistry.com or at 202-249-6727 if you would like to discuss these issues further.

Sincerely,

Steve Risotto

Stephen P. Risotto
Senior Director

Enclosure