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November 12, 2019

Via Electronic Mail: Brian.Dougherty@FloridaDEP.gov

Mr. Brian Dougherty
Program Manager
Florida Department of Environmental Protection
Division of Waste Management
2600 Blair Stone Road, MS 4535
Tallahassee, Florida 32399-2400

RE: Comments on the Development of Surface Water Screening Levels White Papers as Presented at the September 12, 2019 Contaminated Media Forum Meeting

Dear Mr. Dougherty:

At the September 12, 2019 Contaminated Media Forum Meeting, the Center for Environmental and Human Toxicology of University of Florida presented two draft white papers concerning the development of surface water screening levels for protection of human health and for eco-based systems. After these presentations, the Florida Department of Environmental Protection (Department) sought comments on the draft white papers and asked that written comments be submitted by November 12, 2019. As such, please find the following comments prepared by a third-party toxicology consultant on behalf of Waste Management Inc. of Florida (WMIF).

We write to provide comment on the white paper "Development of Surface Water Screening Levels for PFOA and PFOS Based on the Protection of Human Health," prepared for the Florida Department of Environmental Protection (Department) by the University of Florida. In particular, we are concerned about the implications of the proposed 4 part-per-trillion concentration for PFOS regarding compliance and remediation costs, as well as the public concerns it may generate.

Our review indicates that there is a considerable degree of overprotectiveness built into the derivation. We thus suggest some alternative assumptions that we believe can be applied and still result in a surface water screening level for PFOS and PFOA that protects human health with an ample margin of safety.

We have included two attachments – with the permission and support of the authors – that focus on technical issues. Both of these attachments are comment letters that were previously submitted to the Massachusetts Department of Environmental Protection pursuant to proposed regulations in that state. The authors of these documents (Green Toxicology, LLC and Sanborn Head & Associates, Inc.) are open to questions.

The formula used to derive the surface water screening level *SWSL* based on potential risk from fish ingestion is:

$$SWSL\ (\mu g/L) = RfD \times RSC \times \left(\frac{BW}{FI \times BAF}\right) \times CF$$

where the factors are:

- *RfD*, the reference dose used to characterize PFOS and PFOA toxicity;
- *RSC*, the relative source contribution or fraction of the RfD that is allotted to the fish ingestion pathway;
- *BW*, the human body weight;
- *FI*, the fish consumption rate;
- *BAF*, the bioaccumulation factor that relates the PFOS and PFOA concentrations in fish and surface water; and
- *CF*, a units conversion factor.

We offer the following comments on several of these factors.

The Reference Dose (RfD) should be based on the most relevant animal studies

The *RfD* used to characterize PFOS and PFOA toxicity are a key factor in determining the surface water screening levels, and proposes the use of the 20 ng/kg-d established in 2016 by USEPA to support its 70 ppt Lifetime Health Advisory for drinking water. The white paper notes that the Agency for Toxic Substances and Disease Registry (ATSDR) and some states have proposed or developed *RfDs* lower than USEPA's 20 ng/kg-d value, and recommends potentially revisiting the *RfD* pending further determinations of regulatory levels.

Much of the uncertainty concerning *RfD* values stems from the reliance on studies in rats and mice to determine adverse health effects of perfluoroalkyl substances (PFAS) generally, including PFOS and PFOA. Simply put, rats and mice are bad biological models for assessing PFAS toxicity in humans because PFAS behave in markedly different manner in rats/mice and humans. In many cases it is unclear that the effects that have been noted in rat/mouse toxicity studies are even relevant to humans. The trend toward using rat/mouse studies has resulted in the use of considerable safety factors to compensate for uncertainty in extrapolating study results to humans. In addition to the need to apply an adjustment factor of about 200 to account for differing half-lives of PFAS in humans and mice, an additional safety factor of 300 is built into USEPA's *RfD* based on standard practice. The degree of protectiveness afforded by these safety factors is rarely communicated to the public, and (for PFAS especially), the principal reason that we can claim that standards are highly protective of health. The values applied for PFAS, along with some factors that suggest that they are protective (and arguably more protective than necessary) are as follows:

- A factor of 10 is applied to account for the possibility that some individuals might be more sensitive to PFAS than the finite number of animals studied in the laboratory test. USEPA

selected a developmental health study as the basis of its *RfD* – this is arguably a study of the category of individuals (pre-born infants) likely to be most sensitive to chemicals such as PFAS that are capable of crossing the placenta. Arguably, since the most sensitive population has been studied, a lower factor of 3 could have been applied to sufficiently account for the possibility that some women/fetuses within the key subpopulation being more sensitive than others.

- A factor of 10 is applied to extrapolate the Lowest Observed Adverse Effects Level (LOAEL) to an assumed No Observed Adverse Effects Level (NOAEL), *i.e.*, an assumed level of exposure that would have led to no delays in finger development and no hastened male puberty in the baby mice (the observed effects in the study). This safety factor is again standard practice and it is not uncommon to use LOAELs to derive RfDs for other chemicals. What arguably makes this factor protective – and maybe more protective than necessary – for PFOS/PFOA is the selection of the transient (non-permanent) effects as the basis of the RfD. The baby mice were ultimately not damaged and grew up normally. Hence, some toxicologists would argue that this factor of 10 is not necessary at all.

- A factor of 3 is applied under the assumption that humans might categorically be more sensitive to the effects of PFAS than the animals (mice) studied in the toxicity test. The fact that USEPA selected 3 for this factor instead of 10 reflects some knowledge/judgement that humans may not be as sensitive to PFAS than are mice. In fact, evidence indicates that mice are more sensitive to PFAS than are humans – the exact opposite of the standard assumption. As explained in Attachment A, developmental effects of PFAS in mice are mediated via the peroxisome proliferator-activated receptor alpha (PPARα). Strong evidence indicates that rats and mice are highly susceptible to the effects of chemicals that act via PPARα, while humans are resistant to these effects. Hence, application of "best science" would entail application of a modifying factor in the opposite direction, thus reducing (and not increasing) the overall safety factor.

USEPA's 20 ng/kg-d *RfD* serves as an example of programmatic resolution of uncertainties to err consistently on the side of health protection when extrapolating rate and mouse studies. It is also worth considering the basis of the *RfD* and how it relates to evidence from epidemiological studies. USEPA's *RfD* is based on a study in mice in which subtle differences were noted in the development of pups compared to control groups. The pups developed into normal adult mice, which (as noted by the study authors) indicates that the observed difference is not of sufficient significance to serve as the basis of an *RfD*. Moreover, developmental effects observed in rats and mice are not consistent with the results of the C8 Panel epidemiological studies in humans, which failed to find significant statistical associations between exposure to PFOA and birth defects in people living near the industrial facility that manufactured PFOA.

Again, a fundamental problem with PFOS and PFOA lies in the difficulty of using rat and mouse studies to gauge health effects in humans. An alternative and better approach would be to derive an *RfD* from studies in animals that more closely match human responses to PFOS and PFOA, such as studies in rabbits, guinea pigs, or monkeys. There are in fact published studies in monkeys available for this purpose. These studies can be used to derive *RfDs* of 280 ng/kg-d for PFOS and

89 ng/kg-d for PFOA. Details of these derivations are provided in Attachment A. These values represent health-protective derivations as they are based on less serious endpoints – in the case of PFOS, a slight decrease in thyroid function that did not compromise the health of the monkeys, and in the case of PFOA, liver weight increase that is likely non-permanent (reversible). Both *RfDs* also incorporate a safety factor of 30. We suggest that DEP consider this alternate *RfD* value as more appropriate for characterizing PFOS and PFOA toxicity.

The Relative Source Contribution (RSC) should be lowered to reflect available data

A value of 0.2, or 20%, is assumed for the *RSC*, which implies that "background" exposure to PFOS and PFOA from pathways other than fish ingestion could account for 80% of the *RfD*, or 16 ng/kg-d (80% × 20 ng/kg-d). An *RSC* of 20% is the default value recommended by the USEPA for contaminants when data are lacking to make a better estimate.

In the case of PFOS and PFOA, however, background exposure (and subsequently *RSC*) can be estimated from examination of blood serum data. Such an analysis is detailed in Attachment 2. As described therein, the median concentrations of serum PFOS and PFOA in Americans decreased by factors of 6 and 2.5, respectively, from 1999 to 2013 in response to the discontinued use of these compounds in the United States. Using the serum concentrations of PFOS and PFOA in combination with pharmacokinetic data, background exposure rates of 0.6 ng/kg-d for PFOS and 0.3 ng/kg-d for PFOA are estimated (as rounded to one significant digit). These background exposure rates correspond to the following *RSC* values:

- At the USEPA's *RfD* of 20 ng/kg-d,
  - for PFOS, the background exposure of 0.6 ng/kg-d represents 3% of the *RfD*, and the *RSC* is 1 – 0.03 = 0.97, and
  - for PFOA, the background exposure of 0.3 ng/kg-d represents 1.5% of the *RfD*, and the *RSC* is 1 – 0.015 = 0.985;
- At our recommended *RfD* of 280 ng/kg-d for PFOS,
  - the background exposure of 0.6 ng/kg-d represents 0.21% of the *RfD*, and the *RSC* is 1 – 0.0021 = 0.9979, and
- At our recommended *RfD* of 89 ng/kg-d for PFOA,
  - the background exposure of 0.3 ng/kg-d represents 0.34% of the *RfD*, and the *RSC* is 1 – 0.0034 = 0.9966.

Fish Ingestion Rate (FI) and Body Weight (BW)

The white paper uses values of 29 g/d and 75 kg for the *FI* and *BW*, respectively. USEPA's 2015 update of National Ambient Water Quality Criteria uses values of 22 g/d for *FI* and 80 kg for *BW*.[i] These differences are small, but for consistency with USEPA procedures, we recommend the adoption of the USEPA values.

Water to fish Bioaccumulation Factors (BAFs)

We have no specific recommendations regarding the white paper's choice of *BAFs* other than to note that values derive mainly from studies conducted in China in fish species that differ from those common in Florida.[ii] We thus recommend that DEP consider collecting fish and water samples to derive BAFs specific to Florida's environment.

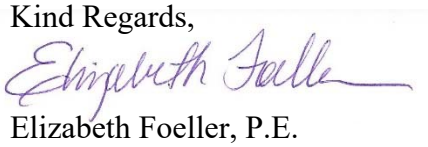
Overall Recommendations for Human Health-Based Surface Water Screening Levels

Our recommendations of alternate parameters are summarized in the following table. Using these values in the formula for SWSL results in values of 370 ng/L for PFOS and 4,700 ng/L for PFOA, respectively.

Parameter	PFOS	PFOA
Reference dose (ng/kg-d)	280	89
Body weight (kg)	80	80
Relative source contribution	0.9979	0.9966
Freshwater and estuarine finfish and shellfish consumption rate (kg/d)	0.022	0.022
Bioaccumulation factor (L/kg)	2358	68
Surface Water Screening Level (ng/L)	430	4,700

If you have additional questions, please call me anytime at 1.941.720.0564. Thank you for your time and consideration of these comments.

Kind Regards,



Elizabeth Foeller, P.E.
Area Environmental Protection Manager
Waste Management Inc. of Florida (WMIF)

cc: Chris Carey, DDO WMIF
Matt Orr, DDO WMIF
Carl Eldred, HGS

Attachments:
Comments on Massachusetts Department of Environmental Protection's (DEP's) groundwater and soil standards for perfluoroalkyl substances (PFAS) in the Department's proposed 2019 amendments to the Massachusetts Contingency Plan, Green and Couch

Comments on Proposed MCP Standards for PFAS, Callahan, July 19, 2019

[i] https://www.epa.gov/sites/production/files/2015-10/documents/human-health-2015-update-factsheet.pdf
[ii] http://www.eregulations.com/florida/fishing/freshwater/freshwater-fish-florida/

**Comments on Massachusetts Department of Environmental Protection's (DEP's)
groundwater and soil standards
for perfluoroalkyl substances (PFAS)
in the Department's proposed 2019 amendments
to the Massachusetts Contingency Plan**

Laura C. Green, Ph.D., D.A.B.T. and Edmund A.C. Crouch, Ph.D.
July 19, 2019

Introduction and Overview

The Massachusetts Department of Environmental Protection (MassDEP, 2019) proposes new standards for the sum of six perfluoroalkyl substances (PFAS):

- perfluorooactanoic acid (PFOA),
- perfluoroheptanoic acid (PFHpA),
- perfluorononanoic acid (PFNA),
- perfluorodecanoic acid (PFDA),
- perfluorooctane sulfonic acid (PFOS), and
- perfluorohexane sulfonic acid (PFHxS).

Unfortunately, MassDEP's proposed PFAS standards are not based on current evidence, but could and should be revised. Among other issues, MassDEP's currently proposed standards:

- Are not based on any reliable evidence of adverse effects in humans;
- Are instead based almost entirely on only two studies in rodents:
  - One study of PFOA in laboratory mice (Lau et al., 2006), in which minor, transient, developmental effects were reported; and
  - One study of PFOS in laboratory rats (Luebker et al., 2005) that reported "delayed eye opening" and reduced birth weights in neonates;
- Do not reflect well-established, marked differences in sensitivities to PFOA and other PFAS between and among laboratory rats, mice, monkeys, and humans;
- Ignore reliable, relevant evidence from controlled studies of PFOA and PFOS in laboratory monkeys; and
- Fail to account for recent, relevant, clinical and epidemiological studies of PFOA.

With regard to the first point, it remains the case that epidemiologic and/or clinical evidence has so far failed to establish that any PFAS harms human health at or near environmental exposure-levels (ATSDR, 2018). MassDEP should make this clear, but currently it does not.

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High-level, experimental exposures to some PFAS do harm the health of laboratory animals, and it is entirely appropriate to base health-protective guidelines on exposure-response data derived from laboratory animal studies (in the absence of, or in addition to, usable exposure-response data from studies of humans).

Ideally, health-based guidelines and standards should be based on controlled studies of (i) humans, (ii) monkeys, and/or (iii) other laboratory mammals known to mimic humans with regard to relevant biological responses. Unfortunately, the two studies on which MassDEP rely are in none of these three categories.

In what follows, we present constructive criticisms of MassDEP's approach, and offer alternate bases for regulation. In particular, we show that the results from studies of PFOA and PFOS in laboratory monkeys can, and should, be used to derive highly protective, evidence-based "reference doses" (essentially, acceptable daily intakes), which in turn should be used to fashion regulations intended to protect public health, with an ample margin of safety.

The evidence-based, highly conservative, reference doses that we derive herein are 89 ng PFOA per kg body weight per day and 240 ng PFOS/kg-day. We also note that reference doses for other PFAS should be based on chemical-specific evidence.

Health-risks from PFOA

Based on minor, transient, developmental effects in CD-1 mice exposed to high doses of PFOA (Lau et al., 2006), U.S. EPA, California EPA, and others (Goeden et al., 2019) assume that this PFAS poses a risk of developmental toxicity to humans. And MassDEP, by extension, assumes the same for all of the six PFAS that it proposes to regulate, despite zero such evidence for at least four of these PFAS (all but PFOS, about which more below).

As it happens, the fundamental uncertainties in this assumption render these CD-1 mouse bioassay results entirely unsuitable for purposes of assessing risks to human health — even from exposures to PFOA, let alone from exposures to the other five PFAS of interest to MassDEP. Why did MassDEP rely on this single study in CD-1 mice, when, as explained below, controlled, reliable, and relevant studies of PFOA in monkeys have been peer-reviewed, published (Butenhoff et al., 2002, 2004a, and 2004b), and serve as much better predictors of effects in humans?[1]

[1] One answer is that MassDEP decided to simply accept U.S. EPA's (2016) reference dose at face value; despite the facts that EPA's derivation of its PFOA reference dose has not been peer-reviewed and has not been relied upon by EPA for standard-setting. Moreover, environmental guidelines and standards for PFOA, as established by various regulatory expert-groups internationally, *vary by 750-fold* (Dourson et al., 2019): this alone is indication that various analysts' assumptions and subjective judgments — rather than a set of objective, verifiable, unambiguous, health-effects data — are what drive these disparate, bottom-line numbers for "acceptable" exposures to PFOA.

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The developmental (and many other) effects of PFOA in mice are mediated via the cell-nuclear hormone receptor, peroxisome proliferator-activated receptor alpha (PPARα; Abbott et al., 2012; Albrecht et al., 2013).[2] However, the activity-levels, structures, and functions of PPARα vary substantially among rodent-species and other animal-species; and, importantly, vary substantially between laboratory, "wild-type" mice (such as CD-1 mice) and humans (Bell et al., 1998; Corton et al., 2018). Abundant evidence indicates that rats and mice are highly susceptible to the effects (both adverse and beneficial) of chemicals (both endogenous and exogenous) that act via PPARα, while humans and other mammals — including guinea pigs, hamsters, rabbits, and monkeys — are relatively resistant to these effects (Klaunig et al., 2003 and 2012; Hoivik et al., 2004; Corton et al., 2018).

In addition to mice, laboratory rabbits have been used to assess the developmental effects of PFOA (Gortner et al., 1982). As just noted, rabbits can serve as faithful models for humans with regard to the actions of peroxisome proliferators on PPARα (Staels & Auwerx, 1998). In the relevant study, pregnant New Zealand White/Minikin rabbits were dosed with the ammonium salt of PFOA at 0, 1.5, 5, and 50 mg/kg-day on gestational days 6 through 18 (Gortner et al., 1982). The highest dose-rate, as expected, caused significant, temporary weight loss in the pregnant rabbits; but their fetuses at gestational day 29 showed zero indications of reproductive toxicity, embryotoxicity, or gross, skeletal, or internal malformations, or any other adverse effects, in *any* PFOA dose-group, including the highest.

MassDEP currently takes no notice of this important study. U.S. EPA also did not even mention this rabbit bioassay in its assessment of PFOA (U.S.EPA, 2016), which is surprising, since the study-report is included in EPA's Administrative Record.

Standard regulatory guidance (and common sense) dictates that when extrapolating results from developmental studies, health risk-assessors should rely on laboratory animal-species *that best mimic humans with regard to relevant biological mechanisms*. *Per* U.S. FDA (2017):

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[2] PPARs are present in all animal-species, although with different forms in different species. As explained by Hall et al. (2012):

> PPARs regulate lipid and cholesterol metabolism through induction of (peroxisome proliferator response element (PPRE)) containing target genes resulting in increased beta-oxidation of fatty acids (Xu, Li, and Kong 2005). Natural ligands for PPARα include saturated and unsaturated fatty acids, eicosinoids, and linoleic acid metabolites. However, a diverse range of xenobiotics from many classes and structures are also able to activate PPARα such as the fibrate hypolipidaemic agents (clofibrate, fenofibrate, gemfibrozil amongst others), methaphenilene, thromboxane synthetase inhibitors, dehydroepiandosterone, non-steroidal anti-oestrogens, ibuprofen, Wy-14,643, diphenyl ether herbicides, and phenoxy herbicides (Greaves 2007).

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> The rabbit has proven to be useful in identifying human teratogens that have not been detected in rodents; and the rabbit is routinely used as the non-rodent species based on the extensive historical background data, availability of animals, and practicality.

Importantly, the epidemiology on PFOA does not indicate that this chemical harms human development. As noted by ATSDR (2018):

> . . . most [epidemiological] studies found *no association* between maternal serum PFOA levels and the risk of low birth weight infants (typically defined as <2,500 g) . . . or found a *decreased* risk of low birth weight infants . . . [*emphasis* added]

And summarizing the literature on infant birth-weights *in the normal range*, ATSDR (2018) notes that although three sets of studies on women exposed to background concentrations *did* report inverse associations between maternal serum PFOA and birth weight, another *twelve similar studies found no such associations.*

Thus, although the CD-1 mouse data on the biological and toxicological effects of PFOA are of little-to-no relevance with regard to effects of PFOA on humans, more reliable and relevant data on the biological and toxicological effects of PFOA have been generated in laboratory monkeys (Butenhoff et al., 2002,[3] 2004a, and 2004b); and these primate data, combined with information from studies in humans, can be used to generate estimates of risks to human health from PFOA. We do so as follows.

Butenhoff and co-workers (2002, 2004a, and 2004b) examined the effects of the ammonium salt of PFOA (APFO) in male cynomolgus monkeys, during and after oral dosing for 6 months. The dose-rates were 3, 10, and 30 mg of APFO/kg body weight/day, although because the monkeys in the high dose-rate reduced their food intake and failed to gain weight, this highest dose-rate was reduced 20 mg/kg-day.

Doses of 30 and/or 20 mg/kg-day were plainly toxic, with evidence of liver injury in the highest dosed monkeys, but doses of 10 mg/kg-day and 3 mg/kg-day were not: no histopathologic evidence of liver injury was observed in monkeys in these middle and low dose-groups, and concentrations of liver enzymes in their blood-sera were normal.

All doses of APFO did increase the relative weights of the monkeys' livers, due to proliferation of liver mitochondria. This effect was expected, since statin drugs and other peroxisome proliferators (which act like PFOA in the liver) also cause increased biosynthesis of mitochondria. Although this is clearly a chemically-induced (and drug-induced) effect, it is not

[3] Individual animal data for this study are available in Thomford (2001) and 3M Environmental Laboratory (2001).

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clear that it is an *adverse* effect, as opposed to merely an adaptive effect (Berthiaume and Wallace, 2002; Butenhoff et al., 2002; Hall et al., 2012; Convertino et al, 2018).

Nonetheless, the authors (Butenhoff et al., 2004b) erred on the side of safety by using the relative increase in liver weight (expressed as the ratio of animals' liver weight to brain weight) to derive a benchmark concentration (BMC) for PFOA that could be used for purposes of human health risk assessment.

Their BMC analysis used mean values by dose group of concentration and liver-to-brain weight ratio, and omitted the high-dose group. However, there is substantial intraspecies variation in concentrations at fixed dose rates; for example, the two animals in the high dose group differed by almost a factor of 3 in their plasma concentrations of PFOA (averaged over weeks 20 to 26, as used by Butenhoff et al., 2004b; see Butenhoff et al., 2004a or 3M Environmental Laboratory, 2001 for individual animal concentrations in this experiment). The same sort of variation in the ratio of plasma concentration to dose can be expected in humans, since the weight-specific volume of distribution is unlikely to vary substantially between individuals while the half-life varies substantially, as seen in a cohort in Sweden and in the C8 study (Li et al., 2017, 2018).

A BMC analysis using individual animal data is sensitive to inclusion/exclusion of the monkey with highest concentration or inclusion/exclusion of the high dose animals (**Figure 1**, **Table 1**).

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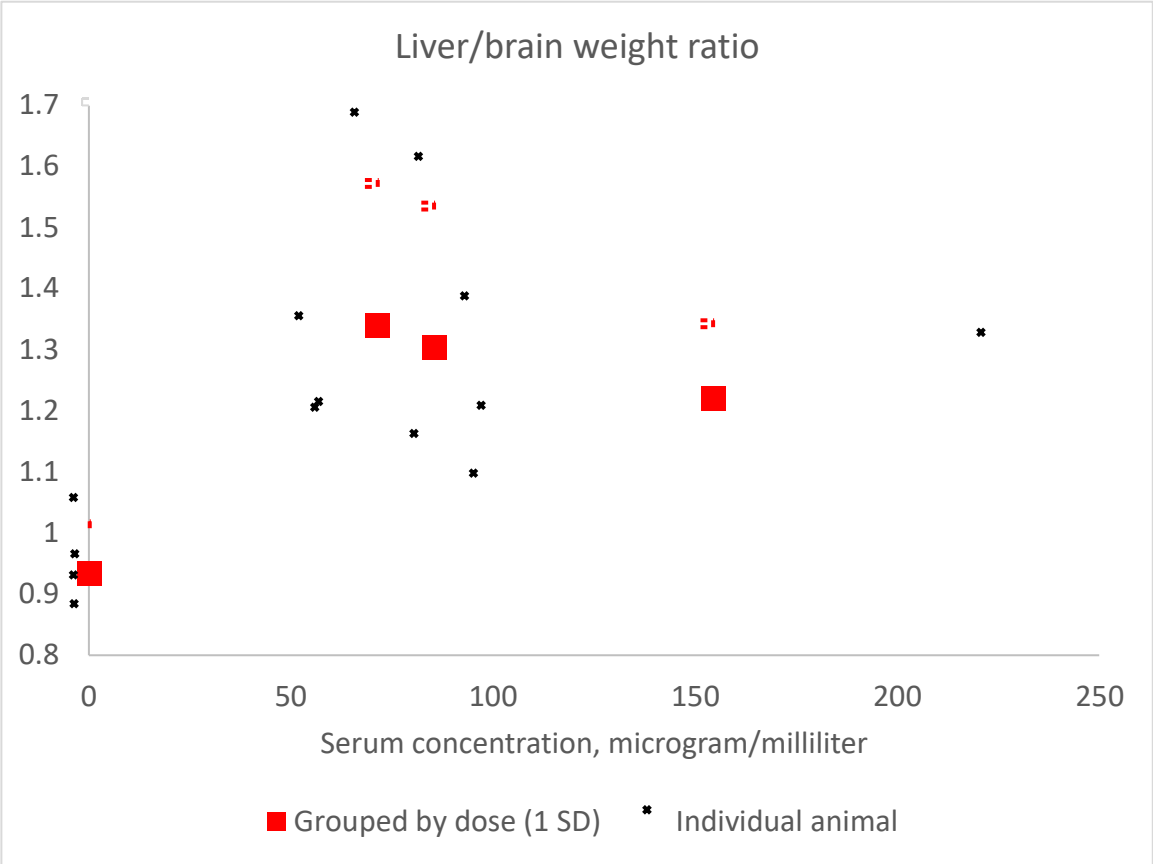

Figure 1 Liver/brain weight ratio in Butenhoff et al. (2002)

	BMCLo	BMC	BMCHi
Grouped, all doses	45.0	79.7	343.9
Grouped, omit high dose	22.6	35.5	79.8
Individual, all animals	57.5	113.2	3099.8
Individual, omit high concentration	29.9	52.4	205.1
Individual, omit high dose	28.3	49.1	178.4

Table 1 BMC estimates (serum concentrations, μg/ml) using liver/brain weight (95% confidence limits, 1 SD, linear model, constant variance)

In fact, in this experiment, the liver/bodyweight ratio provides a more sensitive endpoint (**Figure 2**, **Table 2**). The BMCLo obtained using the individual animal data is the most appropriate for cross-species extrapolation using serum concentration as the relevant metric, so we use that as the point of departure (POD).

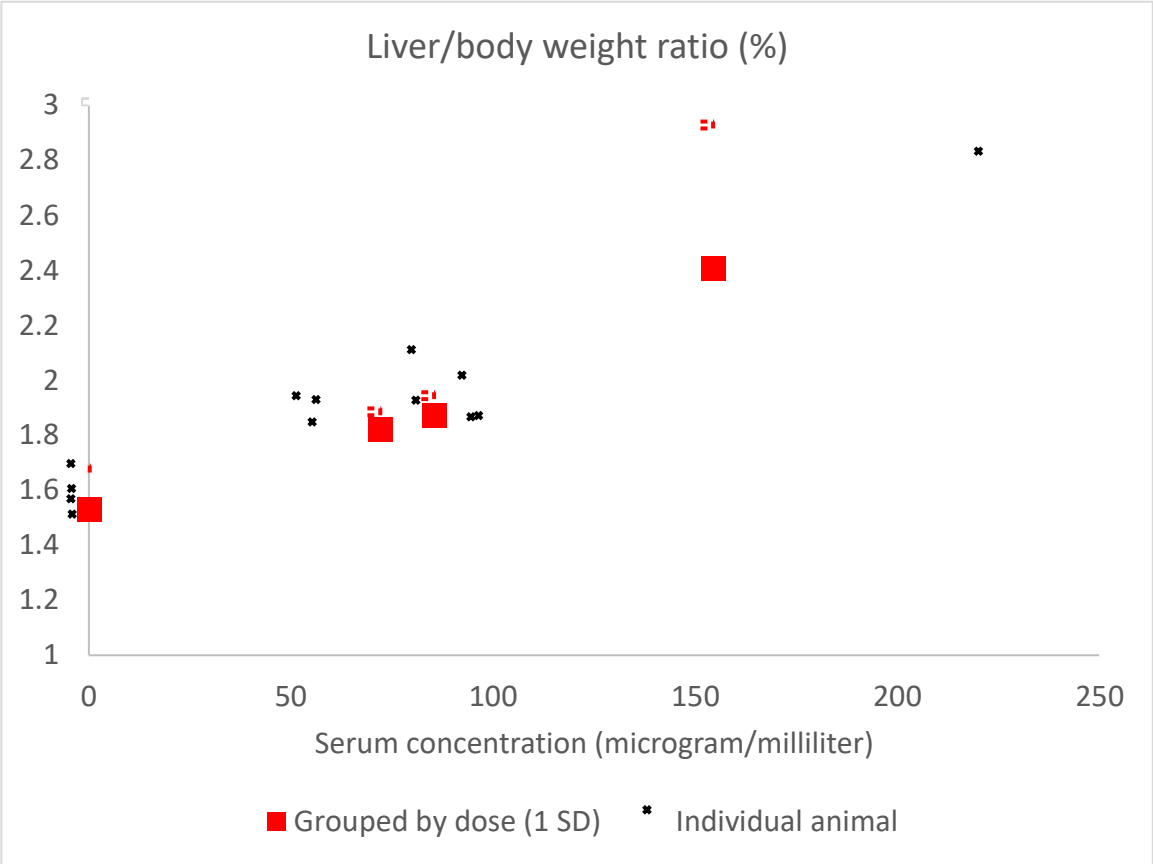

Figure 2 Liver/bodyweight ratio in Butenhoff et al. (2002).

	BMCLo	BMC	BMCHi
Grouped, all doses	26.0	50.9	88.5
Individual, all animals	19.0	32.5	57.4

Table 2 BMC estimates (serum concentrations, μg/ml) using liver/body weight ratio (95% confidence limits, 1 SD, restricted power model, constant variance)

Extrapolating this POD to humans using an interspecies factor of 3 and an intraspecies factor of 10 (compared with the 3-fold difference from 5th to 95th percentile expected solely from the variation in half-lives, Li et al., 2017, 2018), leads to a human plasma concentration of 633 ng/ml. The potential effects of PFOA exposure are seen with short induction times, so no factor is required for extrapolation from subchronic to chronic exposure. Assuming a distribution volume of 0.2 L/kg (ATSDR 2018, Table A-4) and a median half-life of 2.7 years for humans (Li et al., 2017, 2018) gives a reference dose of 89 ng/kg-day.

This primate results-based, reference dose is highly conservative, since, as noted, it assumes that liver weight gain in PFOA-exposed monkeys, in the absence of any indication of liver damage, is an adverse, as opposed to simply adaptive, effect.

Of course, risk assessment is intended to err on the side of safety, so this conservatism is, we believe, appropriate. We recommend that MassDEP consider using this more reliable and relevant value for PFOA as it continues to refine its approach for the regulation of this chemical.

We would add that we think it quite important for risk assessors to communicate that chemicals, such as PFOA, with very small reference doses based on laboratory animal study-results (with multiple safety factors applied) are *not necessarily* highly toxic to humans. Indeed, analysts should make plain that PFAS are *categorically* different from chemicals such as arsenic, lead, mercury, benzene, 2,3,7,8-TCDD, and a multitude of other environmental contaminants for which adverse effects in humans have long been well-established.

As it happens, PFOA has been found to combat certain tumor-types, and has actually, perhaps surprisingly, been administered at extremely large dose-rates — up to 1.2 grams per patient per week, which is about 2,300,000 ng PFOA/kg-day! — to cancer patients in a phase I trial (Convertino et al., 2018). The resulting blood-serum concentrations of PFOA in these phase I study patients were, as noted by Convertino et al. (2018) "the highest ever reported in humans." Yet their serum liver enzyme levels remained normal, and there was otherwise no indication of organ toxicity.[4]

Health-risks from PFOS

Next, PFOS has been studied in laboratory rats, rabbits and monkeys (Case et al., 2001; Seacat et al., 2002; Chang et al., 2012 and 2017); and here again the monkey data can be used to estimate risks to human health.

In developmental toxicity studies in both rabbits and rats (Case et al., 2001), the highest dose rates of PFOS caused frank maternal toxicity, which in turn led to some fetal losses and reversible, delayed ossification. However, per the study-authors, "detailed external gross, soft tissue, and skeletal fetal examinations failed to reveal any compound-related malformations in either species," giving a NOEL for developmental toxicity of 1 mg/kg-d. Moreover, "[t]he

[4] Interestingly, at these high doses, the apparent half-life of PFOA in these patients was on the order of only weeks (Dourson et al., 2019) — substantially lower than the median half-life of 2.7 years that has been derived from people exposed only environmentally (via contaminated drinking water), who have vastly lower plasma concentrations of PFOA.

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finding that PFOS was not a selective developmental toxicant to rabbit fetuses concurs with results of previously conducted rat developmental toxicology studies."

Chang et al. (2017) dosed male and female cynomolgus monkeys with one, two, or three doses of PFOS at various times during a 422 day experiment, examining clinical chemistry parameters and measuring serum PFOS concentrations. PFOS serum concentrations at the highest extreme reached values close to those demonstrating overtly toxic effects in an earlier bioassay (Seacat et al., 2002): nonetheless, all clinical chemistry parameters remained within normal biological limits during the experiment. As expected, serum concentrations of two exposure-markers, total thyroxine (TT4) and high density lipoprotein (HDL), did decrease with PFOS treatment, although these varied only within the normal range. Moreover, again as expected, the PFOS-associated decreases in serum TT4 (due presumably to competitive binding) were not accompanied by alterations in serum concentrations of thyroid stimulating hormone (TSH), thus indicating no toxicologically significant effect of PFOS on thyroid function (Chang et al., 2017).

A benchmark concentration (BMC) analysis using individual animal data, based on the conservative assumption that the slight decrements in serum HDL were adverse, yielded a BMCLo (1 SD) of 74,259 and 76,373 ng/ml for males and females respectively. Once again, as in the case of PFOA, evaluation using individual animal data is essential since standard analyses (not shown) based on the published grouped data provide substantially different results (both higher and lower, depending on the assumptions made), presumably because of the large variation in serum concentration to dose ratios.

Extrapolating an average point of departure of 75,300 ng/ml to humans, using an interspecies factor of 3 and an intraspecies factor of 10 (again, larger than the expected major component of such intraspecies factor, the dose-to-serum concentration ratio, which is approximately a factor of 3 between 5th and 95th percentiles, Li et al., 2017, 2018), leads to a human plasma concentration of 2,510 ng/ml. All potential effects of PFOS exposure in animal models are seen with short induction times, so no factor is required for extrapolation from subchronic to chronic exposure. Assuming a distribution volume of 0.2 L/kg (ATSDR 2018, Table A-4) and a human half-life of 3.4 years (Li et al., 2017, 2018) gives a reference dose for PFOS of 280 ng/kg-day.

We recommend that MassDEP consider using this more reliable and relevant value for PFOS as it continues to refine its approach for the regulation of this chemical. MassDEP should also note that this most sensitive effect — a slight reduction in serum HDL — was, as noted by the study-authors, *of no significance to the health of the test-animals*. Indeed, serum lipid levels decreased overall with PFOS-exposure, and this is not adverse.

Risks from other PFAS

In deriving its proposed PFAS standards, MassDEP applies an extra safety factor of 4 (further reducing U.S. EPA's reference doses for PFOA and PFOS from 20 ng/kg-day to 5 ng/kg-day), to account for what DEP claims is the possibility that all six PFAS could harm people's immune

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systems at or near these miniscule dose-rates. This factor of 4 is entirely arbitrary, and is not justified by MassDEP by any holistic analysis of the weight of scientific evidence. We would note that such an holistic analysis has been peer-reviewed and published (Chang et al., 2016), and it concludes:

> With few, often methodologically limited studies of any particular health condition, generally inconsistent results, and an inability to exclude confounding, bias, or chance as an explanation for observed associations, the available epidemiologic evidence is insufficient to reach a conclusion about a causal relationship between exposure to PFOA and PFOS and any immune related health condition in humans. When interpreting such studies, an immunodeficiency should not be presumed to exist when there is no evidence of a clinical abnormality.

We would also note that the two rodent bioassays on which U.S. EPA's reference doses for PFOA and PFOS are based reported no effects on the exposed animals' immune systems.

More generally, ATSDR (2018) has extensively reviewed studies of immune system effects for several of the PFAS of interest: the Agency finds no compelling evidence that PFAS-exposure compromises people's immune systems.

With regard to PFOA, ATSDR (2018) notes that "no consistent associations" have been "found between serum PFOA and disease resistance, as measured by episodes of the common cold, cough, fever, or hospitalization for infectious disease."

With regard to PFOS, ATSDR (2018) notes, "Mixed results have been observed in studies evaluating infectious disease resistance. Similarly, inconsistent results have been examined in studies evaluating associations between serum PFOS and hypersensitivity outcomes, such as asthma; no associations were found for eczema, dermatitis, food allergies/sensitizations."

With regard to PFHxS, ATSDR (2018) notes, "In general, the available studies do not suggest an association between serum PFHxS and decreased infectious disease resistance."

And with regard to PFNA, ATSDR (2018) notes, "Most studies examining a possible association between serum PFNA levels and immunosuppression have not found associations."

We would add that MassDEP should regulate each individual PFAS based on the chemical, biological, and toxicological evidence for that specific PFAS — rather than simply, and counterfactually, assuming that all six PFAS (i) act identically and (ii) pose identical risks to public health. Clearly, they do not.

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Concluding remarks

Assessing risks to public health from PFAS is not straight-forward, and there is no one best approach. Nonetheless, we believe that MassDEP can and will improve upon its draft assessment.

The currently proposed PFAS regulations are both inordinately stringent and unusually poorly justified. We believe that when MassDEP takes the time it needs to evaluate the relevant scientific evidence, from studies in humans and non-human primates alike, the Department will conclude that these six PFAS do not pose the extreme health-threat implied by the currently proposed standards.

Acknowledgements

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Note: Copies of the EPA Administrative Record AR-226 may be requested on CD-ROM from the EPA Docket Office by calling 202-566-0280 or sending an email request to: oppt.ncic@epa.gov.

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SANBORN HEAD

Building Trust. Engineering Success.

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July 19, 2019

Submitted via e-mail to: bwsc.information@mass.gov

Re: Comments on Proposed MCP Standards for PFAS

Dear Ms. Callahan:

Sanborn Head has prepared this letter to transmit our comments on the proposed groundwater and soil standards for per- and polyfluoroalkyl substances (PFAS) specified within the proposed 2019 amendments to the Massachusetts Contingency Plan.

Sanborn Head is an environmental and engineering consulting firm with offices in Massachusetts and other states. Our staff comprises Massachusetts Licensed Site Professionals, Professional Engineers, environmental scientists, risk assessors, and staff with other related expertise. We believe we are well-qualified based on our PFAS knowledge and experience to provide constructive input on the proposed PFAS standards.

Our overall comments and recommendations are summarized in the following two points, with more detailed comments and explanation provided on the ensuing pages of this letter:

1. Based on our comprehensive review of the available health study data, the GW-1 groundwater standard would be protective of human health and the environment if set at 70 parts per trillion (ppt) consistent with the U.S.EPA's Lifetime Health Advisory (LHA) level. The LHA already contains a considerable degree of health protectiveness, and the U.S.EPA's position is that the LHA is set at a safe level. Importantly, the MassDEP's proposed lower level of 20 ppt is not based on scientific data demonstrating adverse health effects at 20 ppt, but it is instead the result of an additional safety factor that is not robustly evidence-based and goes beyond the already-protective assumptions used to reach the 70 ppt LHA level.

2. The proposed S-1/GW-1 soil standard of 0.2 parts per billion (ppb) for the sum of six PFAS compounds is likely lower than background conditions in soil. A study of background PFAS in Massachusetts soils would provide the necessary data to establish an appropriate level that accounts for anthropogenic background, but one has not been performed. In the meantime, based on a recent study of background PFAS in shallow soils in Vermont, the S-1/GW-1 soil standard could be set at 4.2 ppb, which is the 90th percentile value of the summed concentrations of six PFAS compounds measured in the Vermont study (please see our detailed comments attached). In addition, given the lack of a published and recognized method for

analyzing PFAS in soil, an analytical method should also be specified for PFAS in soil, and a study made of the ability of commercial laboratories to generate reliable data from the method.

We greatly appreciate the opportunity to comment and are happy to discuss our comments at greater length, so please do not hesitate to contact us with questions.

Thank you again for this opportunity to participate in this process.

Very truly yours,
SANBORN, HEAD & ASSOCIATES, INC.

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Attachments: Comments on Proposed MCP Standards for PFAS (following pages)
Excel spreadsheet "VTBackgroundSoilData.xlsx" with PFAS soil data

Comments on the proposed groundwater and soil standards for per- and polyfluoroalkyl substances (PFAS) specified within the proposed 2019 amendments to the Massachusetts Contingency Plan

Sanborn Head respectfully submits these comments to the Massachusetts Department of Environmental Protection (MassDEP) for its consideration regarding the establishment of Massachusetts Contingency Plan (MCP) Method 1 soil and groundwater standards for per- and polyfluoroalkyl substances (PFAS). We recognize and support MassDEP's responsible actions to protect public health and the environment, and we applaud the focus and attention MassDEP has dedicated to this issue. We also recognize the concerns of the regulated community regarding the potentially very high costs of meeting extremely low concentration standards for PFAS, especially if these standards are more stringent than the levels necessary to protect public health, as supported by existing toxicological and epidemiological data. It is thus imperative, from our perspective, that MassDEP set MCP standards for PFAS at levels that reflect scientifically sound evaluation of adverse health effects based on a holistic analysis of available data.

COMMENT ON THE PROPOSED GW-1 GROUNDWATER STANDARD OF 20 PPT

Based on our review of available scientific studies and information related to PFAS, and considering this information in aggregate, insufficient scientific evidence has been developed to compel establishing a GW-1 standard for PFAS at 20 parts per trillion (ppt), equivalent to 20 nanograms per liter (ng/l), in place of using the U.S.EPA 70 ppt Lifetime Health Advisory (LHA) level. The LHA was established as MassDEP's Drinking Water Guideline and thus far MassDEP's *de facto* level of concern. Current, important, scientific evidence (some not available when U.S. EPA established its guideline of 70 ppt) demonstrates that concentrations this low pose no significant threat to public health. We urge MassDEP to carefully review and consider comments submitted by Green Toxicology that discuss this new evidence.

There is a considerable degree of health protectiveness built into the U.S.EPA's LHA that receives insufficient attention and acknowledgment. Recently, in announcing the PFAS Action Plan in February 2019, the U.S.EPA stated its position that the 70 ppt LHA is a safe level (https://www.youtube.com/watch?v=xaRgWcwwmXc), in direct response to a question on the lower levels being established by certain states such as New Jersey.

The U.S.EPA has not been compelled to recommend lower advisory levels for PFAS. A principal reason to believe that 70 ppt is a "safe level" stems from the safety factor of 300 built into the underlying reference dose (RfD) of 20 nanograms per kilogram body weight per day (ng/kg-d). The combined safety factor of 300 is based on (i) the most sensitive effect identified, in (ii) the most sensitive test species (laboratory mice), and (iii) includes a safety factor of 3 to account for the possibility that people are more sensitive than laboratory rodents to effects from PFAS exposure. While this is a common standard "default" assumption for deriving reference doses, evidence related to PFAS effects mediated via the PPAR- alpha receptor (which effects include actions on the liver and on development) indicates precisely the opposite from the default. PFOA is now known to be much more toxic to

mice and rats than it is even to other rodents, such as guinea pigs and hamsters, let alone to monkeys and, importantly, humans.[1] It would thus be scientifically justifiable, and based on the evidence more technically correct, to either remove this safety factor of 3 or to apply the factor in the opposite sense (and by doing so *increase* the LHA by a factor of about 10).

There are additional degrees of protectiveness built into the U.S. EPA's 20 ng/kg-d reference dose that MassDEP should clearly communicate to the public and consider in their own standard development process. The safety factor of 300 also includes a factor of 10 to protect sensitive subpopulations. This factor is arguably unnecessary because the subpopulation thought to be most sensitive to PFAS – developing infants – is explicitly accounted for in the derivation of the LHA from the RfD – which is designed to protect the developing fetus and nursing infant, via the child's nursing mother. The assumed drinking water ingestion rate of 0.054 liters per kilogram body weight per day (L/kg-d) for a nursing mother is almost twice as large as the 0.029 L/kg-d ingestion rate typically used to derive Maximum Contaminant Levels (MCLs) and health advisories.[2]

The final safety factor of 10 that contributes to the overall safety factor of 300 is used to extrapolate the Lowest Observed Adverse Effects Level (LOAEL) to an assumed No Observed Adverse Effects Level (NOAEL) because effects on the mice offspring were observed in the lowest dose group tested in the toxicity study. This is again standard default procedure in RfD derivation, but is arguably over protective in the case of PFAS because the observed effects in the toxicity study were transient in nature, *i.e.*, the observations of delayed ossification of phalanges in the offspring and hastened puberty in male pups did not permanently affect the health of the baby mice and prevent them from developing into normal adults.[3] Many toxicologists would argue that more serious and permanent effects, such as cellular damage, should serve as the basis of RfDs used for regulatory purposes. By basing its RfD on transient effects, the U.S.EPA has incorporated yet another health protective safety factor.

We also note that the U.S.EPA chose a developmental toxicity study in laboratory mice as the basis of its RfD even though no developmental health effects were linked to PFOA in the C8 Studies[4] (the most comprehensive epidemiological studies conducted to date on people exposed to high levels of PFOA in their drinking water with approximately 70,000 respondents). Specifically, these studies found no associations between exposures to PFOA (whether measured in water or assessed according to concentrations in people's blood) and rates of birth defects, miscarriages, stillbirths, and/or preterm/low birth weight.

As correctly noted by MassDEP, there is yet another factor of safety built into the procedural basis of deriving GW-1 standards. The target hazard quotient of 0.2 that serves as the basis

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1 See for example: Tyagi S, Gupta P, Saini AS, Kaushal C, Sharma S. The peroxisome proliferator-activated receptor: A family of nuclear receptors role in various diseases. J Adv Pharm Technol Res. 2011 Oct;2(4):236-40

2 0.029 l/kg-d = 2 L/d of water consumption by a 70 kg individual.

3 Lau, C., J.R. Thibodeaux, R.G. Hanson, M.G. Narotsky, J.M. Rogers, A.B. Lindstrom, and M.J. Strynar. 2006. Effects of perfluorooctanoic acid exposure during pregnancy in the mouse. Toxicological Science 90:510–518.

4 http://www.c8sciencepanel.org/

of GW-1 standards allows for background exposure (from pathways other than drinking water) to contribute up to 80% of the safe exposure level. But recent blood serum data collected by the Center for Disease Control indicate that current background exposure to PFAS is much smaller than 16 ng/kg-d (80% of the RfD). Our calculations, which are based on serum levels of several PFAS in human subpopulations over time and are described in Appendix A, indicate that current background exposure to four of the PFAS compounds of interest to MassDEP is only about 1 ng/kg-d, meaning that almost all the 80% assumed exposure via background is unnecessary (and hence highly protective) for a typical person. In other words, because PFOA and PFOS have not been manufactured and used in the U.S. for almost two decades now, our body burdens of these compounds are much smaller than they were even as recently as the year 2000. To the extent that PFOA and PFOS pose a potential threat to public health, that threat is already far smaller than it once was, both here in Massachusetts and throughout the U.S. These recent data and evidence-based trends should also be taken into account by MassDEP in development of their standards.

MassDEP has proposed to add another safety factor of 4 to the U.S. EPA's RfD to reduce the level from 20 ng/kg-d to 5 ng/kg-d to account for potential immunotoxicity effects. Based on the protective factors described above, the extra factor of 4 is not necessary, and MassDEP should simply adopt the U.S.EPA's 70 ppt LHA as the GW-1 standard and await further change (if any) from the U.S.EPA to re-evaluate the merits of such change. We note that the U.S.EPA also considered immunotoxicity effects in establishing its RfD and LHA, and a relevant discussion is provided in the Drinking Water Health Advisory for PFOA document.[5] At present, the U.S. EPA does not find consistent evidence to warrant any additional factor to account for possible immunotoxicity effects of PFOA or related compounds.

Moreover, MassDEP's stated basis of the additional factor of 4 reflects concern over potential immunotoxicity effects, which differs from the developmental basis of the U.S.EPA RfD. This is a non-standard and unjustified approach for RfD derivation. If MassDEP wishes to base its RfD on immunotoxicity, then a toxicological study based on immunotoxicity should be used as the basis of the RfD derivation. If instead no scientifically reliable immunotoxicity study can be identified, as is apparently the case here, then no "accounting" for "immunotoxicity" can or should be offered. In the absence of a scientifically reliable study, the additional safety factor of 4 is entirely arbitrary.

In summary, the 70 ppt LHA that remains supported by the U.S.EPA contains a systematic series of protective assumptions and biases that, when considered in aggregate, impart a high degree of health protectiveness. There is no reliable scientific evidence that these, yet alone lower levels of exposure, actually harm human health. We therefore recommend that MassDEP adopt the 70 ppt concentration as the PFAS GW-1 standard (and subsequently as the state MCL) subject to reevaluation if there is any further modification by the U.S.EPA.

COMMENT ON THE PROPOSED S-1/GW-1 SOIL STANDARD OF 0.2 PPB

We believe that MassDEP's proposed S-1/GW-1 standard is both impractical and unnecessarily low because the underlying assumptions in its selection do not consider or

[5] https://www.epa.gov/sites/production/files/2016-05/documents/pfoa_health_advisory_final-plain.pdf

account for key information. Specifically, our comments below support an increase in the proposed standard because: (1) the proposed standard is less than likely background levels in shallow soils, (2) the proposed standard, set at the MassDEP's proposed reporting limits for the six PFAS, is less than common commercial laboratory reporting limits for those six PFAS, and (3) the proposed standard should be based on the already-protective concentration of 70 ppt in groundwater.

Because the proposed S-1/GW-1 PFAS soil standard is unnecessarily low and PFAS occurrence in background soils is potentially widespread, the proposed standard could result in reportable conditions at any site in the state where soil is sampled for PFAS, leading to unnecessary groundwater sampling and remedial actions throughout the state.

The MassDEP proposed Method 1 Soil Standard is 0.0002 micrograms per gram (µg/g) ∑PFAS for S-1 Soils, where ∑PFAS is the sum of six PFAS (PFHpA, PFOA, PFNA, PFDA, PFHxS, and PFOS). The 0.0002 µg/g value is equivalent to 0.2 ppb in soil. This value is based on the anticipated reporting limit (RL) for the six PFAS rather than a leaching-based value, because MassDEP's calculated leaching-based value is less than the anticipated RL. It should be noted that we refer to the "anticipated RL" since this is based on MassDEP conversations with several commercial laboratories who stated that they could meet an RL of 0.2 ppb but this is not the current practice at these same laboratories where the RLs are currently higher than 0.2 ppb. From documentation provided in MassDEP's 2019 MCP Revision Spreadsheets, we understand the following methodologies were used for calculating a leaching-based value and then selecting the anticipated RL.

- **The leaching-based value** is based on the proposed GW-1 standard and a dilution attenuation factor (DAF). The ∑PFAS leaching-based value was calculated from an assumed/default dilution attenuation factor (DAF) of 1 and the target GW-1 standard of 20 ppt, resulting in a value of 0.02 ppb based strictly on leaching from soil. Documentation of the DAF is unclear. The MassDEP apparently did not model the DAF for ∑PFAS or the DAFs for individual PFAS using its standard MCP approach. Further comments on the DAF are provided below.

- **A RL** of 0.2 ppb was selected by the MassDEP for ∑PFAS and for individual PFAS, in soil, and in the spreadsheet documentation, the MassDEP noted that the RL for the individual PFAS were based on a "Reporting Limit (RL) from MassDEP Wall Experiment Station recommendation". In the summary of the proposed revision, MassDEP indicated the RL, was "based on a survey of several laboratories currently conducting PFAS analysis". Technical documentation supporting the anticipated RL has not been provided for review and comment.

The proposed S-1/GW-1 standard for ∑PFAS appears to be based solely on the reported analytical capabilities of laboratories; neither chemical-specific fate and transport information nor toxicological information (e.g., via the proposed GW-1 standard) are the basis. Although not noted in the documentation, the approach suggests that the MassDEP has insufficient fate and transport information for PFAS to model leaching from soil to derive chemical-specific DAFs, or perhaps, the MassDEP believes the model would not sufficiently describe PFAS leaching. While the science regarding PFAS is rapidly evolving and may

sometimes be uncertain, we urge the MassDEP to consider the available information on PFAS in soil and to modify the proposed PFAS standard accordingly.

Background Levels

Published studies indicate detectable concentrations of PFAS in surface soils collected around the world, including the Northeast United States. One global study (n=60, Strynar et al. 2012[6]) estimated global median "background" concentrations of 0.124 ppb and 0.472 ppb for PFOA and PFOS, respectively. Another study (n=62, Rankin et al. 2016[7]) included "background" samples from across the US and across the globe, including Antarctica (0.048 ppb PFOA and 0.007 ppb PFOS) and the Arctic Circle in Canada (0.270 ppb PFOA and 0.018 ppb PFOS). Every soil sample had quantifiable concentrations of PFAS, with PFOA and PFOS being the most prevalent. The reported mean concentrations for North America were 1.82 ppb for the sum of perfluoroalkyl carboxylic acids (which includes PFHpA, PFOA, PFNA, and PFDA) and 0.410 ppb for the sum of perfluoalkyl sulfonic acids (which includes PFHxS and PFOS). These studies indicate a global background distribution of PFAS in soils, with mean and median concentrations of summed PFAS in North America likely exceeding the proposed S-1/GW-1 standard.

In addition to the global studies, a study of PFAS concentrations in Vermont shallow soils was recently published by the Vermont Department of Environmental Conservation (VTDEC).[8] The study was conducted by the University of Vermont and Sanborn Head with partial funding and support provided by VTDEC. Soil samples were collected from 66 properties with no known potential sources of PFAS (primarily municipal or state-owned parks, forests, greens, or lawns). Because PFAS is anthropogenically sourced, it is reasonable to suspect that background data collected from largely-rural Vermont may be indicative of, or perhaps underpredict, background concentrations that may be detected in Massachusetts.[9] The VT Background Soil Study data for the six PFAS included in the proposed MCL standards are provided with these comments as an excel spreadsheet named "VTBackgroundSoilData.xlsx".

Several PFAS were detected in greater than 50% of the soil samples collected in Vermont, including the six PFAS proposed to be included in the S-1/GW-1 standard. A summary of the

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6 Mark J. Strynar, Andrew B. Lindstrom, Shoji F. Nakayama, Peter P. Egeghy, Laurence J. Helfant. (2012). Pilot scale application of a method for the analysis of perfluorinated compounds in surface soils. Chemosphere, 86, 252-257.

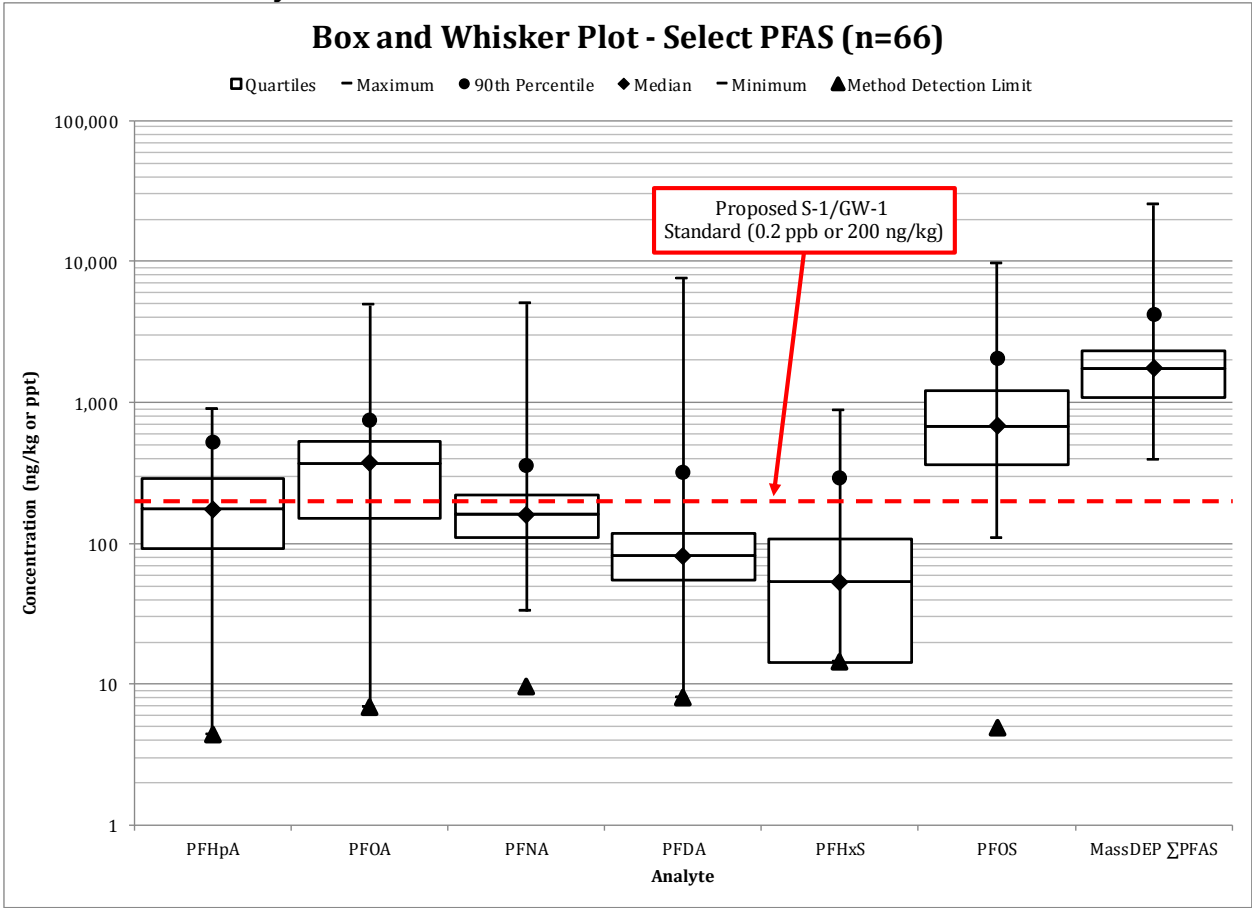
7 Rankin, K., Mabury, S. A., Jenkins, T. M., & Washington, J. W. (2016). A North American and global survey of perfluoroalkyl substances in surface soils: Distribution patterns and mode of occurrence. Chemosphere, 161, 333–341.

8 Badireddy, A.R, Zhu, W., Zemba, S. G., Roakes, H. (2019). PFAS Background in Vermont Shallow Soils. Available for download: https://anrweb.vt.gov/PubDocs/DEC/PFOA/Soil-Background/PFAS-Background-Vermont-Shallow-Soils-03-24-19.pdf

9 Vermont is known to have a "point" source that released PFOA and impacted groundwater wells in and near Bennington via atmospheric deposition. Studies of the area indicate facility-related impacts to soil and water extending several miles from the point of PFOA emissions. While it is likely that emissions from this facility have deposited to soils at some levels at greater distances, the speciation and distribution of PFAS suggest atmospheric deposition from other (probably multiple) sources have more greatly affected the shallow soils sampled in the VT background soil study. The other cited background soil studies corroborate the significance of longer-range transport of PFAS from multiple sources to the environment.

data is provided in Exhibit 1, below. The proposed S-1/GW-1 standard of 0.2 ppb, or 200 ng/kg, is plotted on the exhibit for reference.

Exhibit 1. Summary of Vermont Shallow Soil PFAS Data



Note: Estimated values are used for the data detected above the method detection limit but below the laboratory reporting limit.

The detected background concentrations of individual PFAS compounds often exceed the proposed S-1/GW-1 standard. For example, over 95% of the samples had PFOS concentrations greater than 0.2 ppb. The sum of the six PFAS exceeds the proposed S-1/GW-1 standard in all samples. Clearly, we do not present the comparison to suggest that all soil in Vermont presents a potential leaching concern because it is greater than MassDEP's, or other, proposed soil screening values for the protection of groundwater. On the contrary, the comparison provides evidence that the proposed S-1/GW-1 standard is inconsistent with environmental occurrence data and that "below detection" is not a reasonable threshold for assessing the leaching potential of PFAS in soils.

Thus, MassDEP should either use available data to assign background levels to PFAS in soils or engage in a state-specific study of background levels in Massachusetts. Consistent with MassDEP policies under the MCP, background levels should be set at upper percentile levels (e.g., 90th percentile) and should also consider potential differences in urban and rural areas.

Finally, the implication of the proposed 0.2 ppb S-1/GW-1 standard is that, if background PFAS levels are considerably greater than the 0.2 ppb value proposed by MassDEP as an S-1/GW-1 standard, as suggested by the Vermont soil study results, then one might expect PFAS levels in groundwater should be ubiquitously greater statewide than the 20 ppt level of concern as proposed by MassDEP. This is because the leaching models used by MassDEP, based on the 20 ppt GW-1 standard, resulted in a target soil value of 0.02 ppb. The proposed 0.2 ppb soil standard, based on the anticipated RL, is ten-times greater than the modeled soil value; through application of the same leaching model, the proposed 0.2 ppb soil standard would be associated with 200 ppt in groundwater (i.e., ten-times greater than the 20 ppt GW-1 standard). Because anthropogenic background is likely much higher than the proposed 0.2 ppb standard, the model suggests PFAS in background groundwater should be above even 200 ppt. Although paired groundwater data was not collected as part of the Vermont soil study, the implied, ubiquitous, elevated concentrations of PFAS in groundwater are inconsistent with our understanding of PFAS occurrence in background groundwater based on sampling at multiple sites in VT, NH, and MA.

In addition to considering the occurrence of PFAS in background soils, the MassDEP should consider the proposed S-1/GW-1 standard in the context of empirical relationships between PFAS in soil and groundwater. PFAS leaching from soil to groundwater is difficult to generically model due to complex interactions and sorption processes, including an affinity for the air-water interface in vadose zone soil. Proposed standards should be compared with actual soil and groundwater data, including background studies, to support the feasibility and appropriateness.

Dilution Attenuation Factor Determination for PFAS

MassDEP elected not to use its leaching model of PFAS from soils because the model predictions were much lower than detectable concentrations of PFAS in soil. Hence the proposed S-1 standard of 0.2 ppb represents the analytical reporting limit that MassDEP believes is reliably achievable. MassDEP can and should explore more realistic leaching models in developing S-1/GW-1 standards. While we recognize that the use of MassDEP's standard leaching model likely does not account for the complexities of PFAS fate and transport, MassDEP should at a minimum apply its standard modeling approach as described in its the Background Documentation for the Development of the MCP Numerical Standards (April 1994) technical guidance to estimate a Dilution Attenuation Factor (DAF).

The only chemical-specific data provided in the guidance was for PFOS. Henry's Law Constant (K_H^{pc}) and soil organic carbon-water partitioning coefficient (K_{OC}) were reported for PFOS as 0.011 $\frac{atm-m^3}{mol}$ and 370 $\frac{ml-aqueous}{g-soil}$, respectively. References for these values were not provided. Per a relatively simple MassDEP guidance model, these values correspond to a DAF of 130.[10] Applying this DAF of 130 would result in a leaching-based soil standard of 2.6 ppb. We note that chemical-specific data are also available for the other PFAS (*e.g.*, see the ITRC PFAS fact sheets). While chemical-specific data may not be available for the typical

[10] Estimated from DAF = 6207*H + 0.166*Koc, as provided in MassDEP's 1994 documentation.

model used by MassDEP for DAF calculation, sufficient information is available to calculate DAF from the more simple MassDEP model.

We also suggest that MassDEP could modify and improve its standard approach to account for the unusual properties of PFAS. Shortcomings of MassDEP's model with respect to PFAS will likely lie in the difficulty of estimating partitioning to the air-water interface and the inadequacy of using K_{oc} alone to model PFAS partitioning to solids. As described in a recent paper by Anderson et al. 2016,[11] PFAS partitioning in soil depends on additional factors not included in MassDEP's model. We suggest that MassDEP review the available literature and propose a different model to estimate PFAS leaching potential. Similar to models used for some metals, it may be more practical and appropriate to estimate DAFs from soil-water distribution coefficients based on empirical factors and data.

MassDEP's assumed DAF of 1 is inconsistent with reasonable models for PFAS in the environment. A DAF of 1 has been used by MassDEP as a lower limit for chemicals that, based on modeling by MassDEP, are highly soluble and tend not to partition to solids (e.g. K_{oc} values less than 40 $\frac{ml-aqueous}{g-soil}$), and therefore, flush through soils. The six PFAS are the only chemicals in the MassDEP spreadsheets for which a DAF of 1 was assumed without modeling. The K_{oc} values reported in the ITRC PFAS fact sheets range on the order of 40 to 5,000 $\frac{ml-aqueous}{g-soil}$ across the six PFAS, so the broad assumption that there is very little adsorption of the six PFAS to soil is not appropriate. In addition to neglecting sorption of the PFAS to soil, the DAF of 1 does not include dilution that can be anticipated from groundwater dilution and flow within a typical aquifer system. The result is an unrealistic leaching scenario that is not based on any chemical-specific information or hydrogeologic model.

Reporting Limit (RL) Selection

In the MassDEP's 2019 MCP Revision Spreadsheets, the MassDEP referenced the "reporting Limit (RL) from MassDEP Wall Experiment Station recommendation" as the basis for the proposed selection of the RL for PFAS. Further, in the MassDEP's "Summary of Proposed MCP Method 1 Standards Revision, March 2019," it was described that the RL "was established by [the MassDEP] based on a survey of several laboratories currently conducting PFAS analysis." However, as summarized in Exhibit 2 below, the selected RL is less than common laboratory reporting limits for soil, as reported in laboratory reports prepared by reputable commercial laboratories and provided in reports to us.

Exhibit 2. Summary of Common Laboratory Reporting Limits (RLs)

Laboratory	Report Date	Method	RL (min.-max.) (ppb)
Commercial Lab A	2019 QAPP	Modified EPA 537 with Isotope Dilution	1
Commercial Lab A	Spring 2019		0.976 – 2.00
Commercial Lab B	2019 QAPP		2
Commercial Lab B	Fall 2018		2.00
Commercial Lab C	2019 QAPP		0.2 – 0.5

[11] R. Hunter Anderson, Dave T. Adamson, Hans F. Stroo. (2019). Partitioning of poly- and perfluoroalkyl substances from soil to groundwater within aqueous film-forming foam source zones. Journal of Contaminant Hydrology, 220, 59-65.

Commercial Lab C	Summer 2018		0.21 - 0.60
Commercial Lab D	Fall 2017		~0.1 – 5

Results at the lowest ends of the RL spectrum may be less reliable, lack precision, be more subject to cross contamination, and more commonly result in false positive detections or qualified, estimated values. False positive detections, whether from cross-contamination or laboratory methods, are especially problematic when laboratory reporting limits are at or near the S-1 standard. This concern is amplified by the lack of standard laboratory methodologies for PFAS in soil analysis and the great potential from cross-contamination issues where PFAS are present in many consumer products.

MassDEP has thus not determined that commercial laboratories can reliably detect PFAS at levels as low as 0.2 ppb. There is, to our knowledge, no commonly accepted analytical method for determining PFAS levels in soils. We suggest that MassDEP provide a recommended analytical method for determining PFAS in soils, and then engage in a multi-lab study to determine whether commercial labs are reliably able to quantify PFAS concentrations at the S-1/GW-1 level proposed by MassDEP. Further, MassDEP should also provide guidance on handling combinations of detections, non-detections, and estimated values with respect to calculating the sum of six PFAS compounds and comparing the result to the proposed standard.

Closing Comments for the Proposed S-1/GW-1 Soil Standard

In consideration of the above information, MassDEP should reconsider the 0.2 ppb proposed S-1/GW-1 Soil Standard for ∑PFAS. The table below demonstrates that the 0.2 ppb value for ∑PFAS is not practical given expected background levels of PFAS in soil (based on the Vermont shallow soils study) and typical commercial laboratory reporting limits for PFAS.

PFAS	Leaching-Based Value Based on Modeling or Empirical Data	90th Percentile from VT Background Soil Study	Typical Commercial Laboratory Reporting Limit
PFHpA	Not Calculated	0.53 ppb	1 ppb
PFOA		0.75 ppb	1 ppb
PFNA		0.36 ppb	1 ppb
PFDA		0.32 ppb	1 ppb
PFHxS		0.30 ppb	1 ppb
PFOS		2.1 ppb	1 ppb
∑six PFAS		4.2 ppb[12]	6 ppb

Based on the above, MassDEP should at least consider background soil concentrations and common laboratory reporting limits in establishing the PFAS standard for soil. Further, MassDEP should consider development of leaching-based values using modeling and/or empirical data. Because modeling may not account for the complexities of PFAS fate and

[12] The 90th percentile of the sum of six PFAS does not equal the sum of the 90th percentile values of the individual PFAS as the PFAS concentrations do not correlate perfectly between samples.

transport, we urge that a proposed standard based on modeling be made available for public comment prior to finalizing.

A soil background study should be completed in Massachusetts to understand anthropogenic background of PFAS in soil and to develop soil standards that are protective of human health and the environment, but that are also more likely indicative of leaching potential of PFAS to groundwater. MassDEP could consider using the VT Background Soil Study results to develop interim S-1/GW-1 standards. The table above suggests that a S-1/GW-1 standard of 4.2 ppb for the sum of six PFAS could be used as an interim standard until a background study can be completed in Massachusetts. The accompanying spreadsheet file "VTBackgroundSoilData.xlsx" contains the individual sample results and derivation of the 90th percentile value.

APPENDIX A ESTIMATION OF PFAS BACKGROUND EXPOSURE

By regulation, MCP standards based on non-cancer health endpoints correspond to a Hazard Quotient of 0.2, meaning that the allowable exposure is only 20% of the safe reference dose, thereby allowing up to 80% additional exposure from other exposure pathways. MassDEP states that, in the case of PFAS, this is likely a conservative/protective allowance as typical background exposure is likely smaller than 80% of the reference dose. MassDEP's observation is indeed supported by a, time trend analysis of the PFAS serum concentration data collected by the Center for Disease Control (CDC) under the National Health and Nutrition Examination Survey (NHANES). The NHANES data indicate that Americans are at present excreting more PFOA, PFOS, PFHxS, and PFNA than they are taking in. Better estimates of PFAS Relative Source Contributions (RSCs) can be calculated using the NHANES time trend data and other parameters documented by New Hampshire Department of Environmental Services (NH DES)[13] in their recently proposed Maximum Contaminant Levels.

The draft Toxicological Profile for Perfluoroalkyls issued by the Agency for Toxic Substances and Disease Registry (ATSDR) provides a framework for estimating background exposure to PFAS based on the observation that concentrations of many PFAS have been decreasing in blood in the general U.S. population.[14] Heuristically:

Rate change in PFAS body burden = Background intake rate of PFAS – PFAS excretion rate

Adapting the nomenclature in Appendix A of the ATSDR Toxicological Profile, and assuming (as does ATSDR) 100% absorption of PFAS intake exposure:

$$\frac{d}{dt}(C_b V_d) = D_{back} - k_e C_b V_d$$

$$k_e = \frac{\ln(2)}{t_{1/2}}$$

where the terms are:

C_b	Arithmetic average concentration of PFAS in serum (blood) (ng/l);
V_d	Apparent volume of PFAS distribution (l/kg);
D_{back}	Background exposure to PFAS (ng/kg-d);
k_e	PFAS elimination constant (d^{-1}); and
$t_{1/2}$	PFAS half-life in the body (d).

[13] https://www.des.nh.gov/organization/commissioner/pip/publications/documents/r-wd-19-01.pdf

[14] https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf The fact that serum levels of many PFAS are decreasing in the general U.S. population is an important point worthy of greater emphasis in the face of growing concerns over adverse health effects. We recommend the incorporation of graphics similar to Figure 1 and Figure 2 within the ATSDR report, along with additional discussion of the declining trends.

PFAS concentrations have been measured in blood in the general U.S. population over several periods as part of the NHANES, the earliest in 1999, and the latest in 2013 (https://www.atsdr.cdc.gov/pfas/pfas-blood-testing.html). Assuming (1) PFAS concentrations in blood of C_{b1999} and C_{b2013} in the earliest and latest periods, (2) independence between the variables C_b and V_d, and (3) constant background exposure to PFAS over the period of exposure (T = 14 yrs = 5133.5 d),[15] the differential equation can be solved and rearranged to yield the following expression for estimating the background exposure D_{back}:

$$D_{back} = \frac{k_e V_d (C_{b2013} - C_{b1999} e^{-k_e T})}{1 - e^{-k_e T}}$$

We apply this equation to four of the six PFAS that MassDEP includes in its PFAS sum (PFOA, PFOS, PFHxS, and PFNA). Arithmetic average serum PFAS concentrations, which are appropriate for the model, are not directly available from ATSDR in the draft toxicity profile. As such, the values of the 50th, 75th, 90th, and 95th percentile levels have been extracted from CDC[16], curve-fit to estimate parameters for assumed log-normal distributions, and the parameters have been used to estimate arithmetic means. A spreadsheet with the calculations to estimate these values is provided as an attachment to our comments.

Applying the following parameters for PFOA:

C_{b1999}	5,625 ng/l (estimated arithmetic mean, U.S. residents, 1999-2000);
C_{b2013}	2,337 ng/l (estimated arithmetic mean, U.S. residents, 2013-2014);
V_d	0.17 l/kg (NH DES)[13];
$t_{1/2}$	2.7 yr = 985.5 d (NH DES)[13]; and
T	5133.5 d (14 years)

yields a background PFOA dose estimate of 0.268 ng/kg-d.

Applying the following parameters for PFOS:

C_{b1999}	33,405 ng/l (estimated arithmetic mean, U.S. residents, 1999-2000);
C_{b2013}	6,408 ng/l (estimated arithmetic mean, U.S. residents, 2013-2014);
V_d	0.23 l/kg (NH DES)[13];
$t_{1/2}$	3.4 yr = 1,241 d (NH DES)[13]; and
T	5133.5 d (14 years)

yields a background PFOS dose-estimate of 0.612 ng/kg-d.

[15] The pattern of serum PFNA does not indicate a steady decline since 1999, but rather an increase from 1999 through 2009, followed by a subsequent decline. The equation to consider background is thus considered over the period from 2009 to 2013 for PFNA.

[16] https://www.cdc.gov/exposurereport/

Added together, PFOA and PFOS background exposure are predicted to be 0.88 ng/kg-d, or 4.4% of EPA's reference dose of 20 ng/kg-d for the sum of PFOA and PFOS.

Similar estimates can be developed for PFHxS and PFNA using the blood serum data and parameters reported by ATSDR. However, unlike PFOA and PFOS, concentrations of PFHxS and PFNA (Figure 1) have not declined as rapidly in blood as those of PFOA and PFOS (Figure 2). In fact, from 1999 to 2009, concentrations of PFNA increased (Figure 1).

Applying the following parameters for PFHxS:

C_{b1999}	2,645 ng/l (estimated arithmetic mean, U.S. residents,1999-2000);
C_{b2013}	1,350 ng/l (estimated arithmetic mean, U.S. residents, 2013-2014);
V_d	0. 287 l/kg (NH DES)[13];
$t_{1/2}$	5.3 yr = 1934.5 d (NH DES)[13]; and
T	5133.5 d (14 years)

yields a background PFHxS dose estimate of 0.167 ng/kg-d.

Applying the following parameters for PFNA, but adjusting the equation to cover only the recent decay period from 2009 to 2013:

C_{b2009}	1,418 ng/l (estimated arithmetic mean, U.S. residents, 2009-2010);
C_{b2013}	801 ng/l (estimated arithmetic mean, U.S. residents, 2013-2014);
V_d	0. 2 l/kg (NH DES)[13];
$t_{1/2}$	2.5 yr = 912.5 d (NH DES)[13]; and
T	1461 d (4 years)

yields a background PFNA dose estimate of 0.0757 ng/kg-d.

The total background dose estimate for the sum of the four PFAS is:

0.268 ng/kg-d + 0.612 ng/kg-d + 0.167 ng/kg-d + 0.0757 ng/kg-d = 1.1 ng/kg-d,

which represents 5.6% of the U.S. EPA' reference dose of 20 ng/kg-d, a value far less than the default allowance of 80% under the MCP regulatory formula.

A more complex analysis that considers time-varying background and other factors, or a sensitivity study could be constructed to test the variability introduced by different parameter choices. But barring extreme changes in parameter values, large differences in estimated background exposure estimates are not likely.

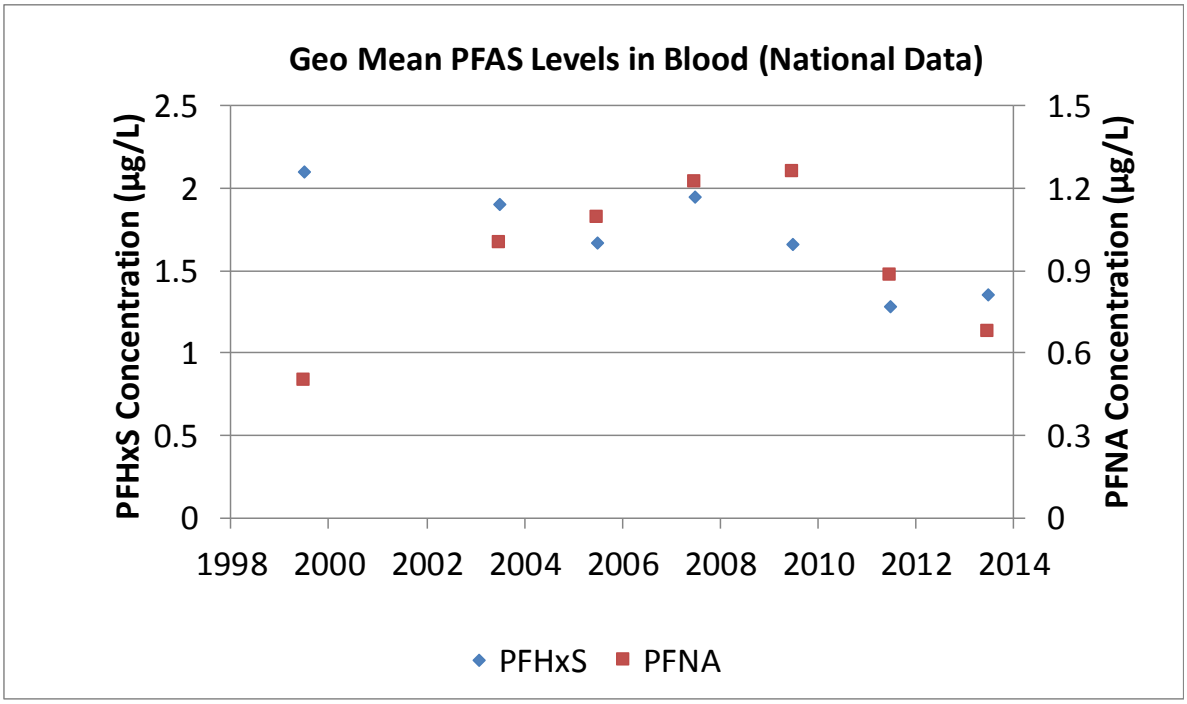


Figure 1 Geometric mean concentrations of serum PFHxS and PFNA reported for the U.S. population, from Table 5-22 of the draft ATSDR Toxicity Profile

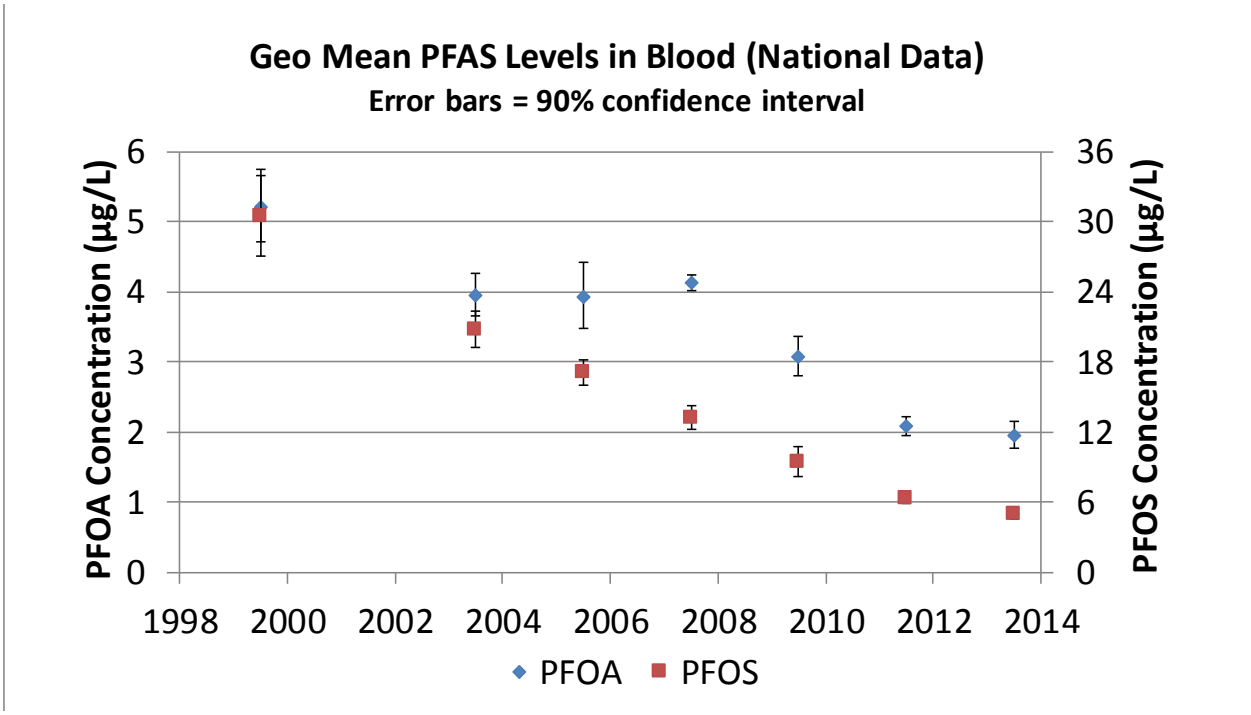


Figure 2 Geometric mean concentrations of serum PFOA and PFOS reported for the U.S. population, from Table 5-21 of the draft ATSDR Toxicity Profile. Bars represent the 5th and 95th percentile concentrations, obtained from the more detailed NHANES data available online.

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November 11, 2019

Brian Dougherty
Program Manager
Division of Waste Management and Business Support Program
Florida Department of Environmental Protection
Via E-mail: Brian.Dougherty@FloridaDEP.gov

Dear Mr. Dougherty:

On behalf of Florida Pulp and Paper Association Environmental Affairs ("FPPAEA" or "the Association"), thank you for the opportunity to submit comments related to the recent Contaminated Media Forum on September 12, 2019, which presented provisional cleanup target levels and surface water screening levels for perfluorooctanoic acid ("PFOA") and perfluorooctane sulfonate ("PFOS"). We appreciate the work of the Florida Department of Environmental Protection (the "Department") on this issue and the opportunity to comment. Our letter first presents general questions and comments regarding the overall purpose and procedure being followed by the Department, and then specifically addresses the screening levels offered by the Department, including the surface water screening levels in "Development of Surface Water Screening Levels for PFOA and PFOS Based on the Protection of Human Health" ("White Paper").

FPPAEA is the State trade association for Florida's forest products industry, representing pulp, paper, packaging, and wood products manufacturers, and forest landowners. The forest products industry is ranked in the top 5 manufacturing sector employers for both number of jobs and employee compensation. The industry is also Florida's leading manufacturer in sustainability and providing green jobs. The industry employs over 30,000 Floridians in high-paying jobs, leads the way on recycling and renewable energy generation, and sustainably manages Florida's forests. FPPAEA member mills are located throughout North Florida and, as entities heavily regulated by the Department, we have a significant interest in regulatory changes by the Department, particularly changes related to surface waters.

Comments Regarding the Purpose and Procedure

The FPPAEA recognizes that the Contaminated Media Forum is an informal meeting, and appreciates the candor and exchange that occur in such a setting. Because of the informal exchange between the Department and the public at the Contaminated Media Forum, FPPAEA is not completely certain if we correctly understood certain aspects of the surface water screening levels and clean-up target levels. Before proceeding further, therefore, we believe the Department should specifically outline the process the Department intends to pursue to

implement the screening levels and the clean-up target levels and the purpose for which the screening levels and clean-up target levels will be used.

As a general matter, the Department's reason and purpose for adopting the clean-up target levels and surface water screening levels remains unclear. Does the Department intend to apply these clean-up target levels and screening levels to sites and waters outside firefighter training locations and federal installations? Comments from the Department suggest that it will do so, but perhaps not until next year. Will surface water screening levels be used to assess whether people can eat fish from or swim in the surface water? If so, then a screening level sounds like water quality criteria adopted in chapter 62-302, Fla. Admin. Code, being used to determine if a water's designated use is impaired according to chapter 62-303, Fla. Admin. Code.

The vagueness makes it difficult to determine whether the FPPAEA will be affected by these clean-up target levels or screening levels. We recommend a clear statement of purpose be provided.

Normally, such a clear statement of purpose would be known based on the Department's rulemaking. In this case, however, we were left with the impression that the surface water screening levels and clean-up target levels would be applied to surface waters and contaminated sites without any rulemaking. We are concerned that such use is contemplated, based in part on the Department's comment in response to questions, that it might use such surface water screening levels to advise the public about whether it was safe to fish or swim in a water exceeding the surface water screening level. The Department also commented that it intended to apply the clean-up target levels to sites requiring remediation under chapter 72-780, Fla. Admin. Code.

We believe that any regulatory use, including the use of surface water screening levels and clean-up target levels described above, would constitute application of unadopted rules in violation of chapter 120, Fla. Stat. There can be little doubt that the clean-up levels and surface water screening levels are statements of general applicability implementing, interpreting, or prescribing law or policy. *See* s. 120.52(16), Fla. Stat. Use of such surface water screening levels to determine whether fish were safe to eat would not only constitute an unadopted rule, but also flout the requirements in chapters 62-302 and 62-303, Fla. Admin. Code, for setting water quality criteria and identifying impaired waters.

The Department also appeared to suggest that no rulemaking would be required to apply clean-up target levels because the equation used to develop the clean-up target levels was already contained in chapter 62-777, Fla. Admin. Code. While rule 62-777.170(1)(b), Fla. Admin. Code, provides for use of an equation in that chapter, there is no showing that the specific numbers used for each variable in the equation is logical or scientifically defensible, as would be required by rule making.

We believe that the Department and the public would be well served to address these questions about their plans, purpose and process to correct any misunderstandings at the next Contaminated Media Forum and before taking any further action on PFOA and PFOS clean-up target levels and surface water screening levels.

Comments Regarding Departmental Screening Levels and the White Paper

The Department presented several screening levels at the Contaminated Media Forum. Each such screening level appears to use a methodology similar to that provided in the White Paper. We provided detailed comments on the White Paper as requested, but believe that these comments should be applied to development of all screening levels to the extent they relied on a similar approach to the methodology in the White Paper.

The White Paper relies on several variables in a modified equation from the U.S. Environmental Protection Agency ("USEPA") for the calculation of fish consumption limits based on concentrations of PFOA and PFOS in fish tissue. FPPAEA has comments and questions regarding the values used for relative source contribution ("RSC"), freshwater and estuarine finfish and shellfish consumption rate ("FCR"), and the bioaccumulation factor ("BAF").

The White Paper states that the Department used USEPA's default RSC of 0.2 for PFOA and PFOS, which USEPA derived using the Exposure Decision Tree methodology. The use of USEPA's default, according to the White Paper is because there is "not enough information to characterize the exposure quantitatively." If there is truly not enough information to calculate an RSC, then – especially given the abundance of other pathways for PFOA and PFOS – it is inappropriate to use a default number. USEPA's decision tree does not solve this problem because there is no basis provided by USEPA for the selection of 0.2. In this context, the use of the default criteria is essentially a random number generated by USEPA.

The White Paper calculates an FCR by relying on USEPA summaries of the NHANES 2003-2010 fish consumption data. There are several flaws with such an approach. First, the Department is being forced to rely on summaries without access to the survey questions and the Department does not know the types and amounts of specific finfish and shellfish purportedly represented by the summaries. Consequently, the Department cannot explain or defend the accuracy or appropriateness of the survey. Second, the Department recognizes that use of national data "does not appear appropriate" but there is no evidence suggesting that the use of NHANES regional data for the South, Gulf of Mexico, and Atlantic regions is any more predictive of FCR in Florida than national data. Florida's extensive coastline and unique species and ecosystems suggest dramatically different consumption patterns compared to these three regions. Third, NHANES survey data is known to reflect significantly higher consumption compared to Florida-specific landings data, which represents the measured amount of finfish and shellfish caught or harvested in Florida. The inappropriateness of the NHANES data suggests that fish landing data is the better source. Fourth, after withdrawing the rule designed to implement human-health based water quality criteria, the Department initiated and is

currently undertaking a Florida-specific fish consumption survey. We question why the Division of Waste Management believes it must forge ahead with this relatively inaccurate FCR for chemicals still being actively researched and never before subject to rule making in Florida, when the Division of Environmental Assessment and Restoration is currently working to identify a more accurate FCR and has suspended rule making on water quality criteria while this study is conducted.

The BAFs selected for PFOA and PFAS raise many questions. First, the Department acknowledges that the studies used to calculate freshwater BAFs "include fish not present in Florida (e.g., rainbow trout) and fish not usually consumed (e.g., minnows, whitebait)." The studies in the Appendix appear to heavily rely on marine species to calculate freshwater BAFs. The food webs between the species used to derive the BAFs and those in Florida would be dramatically different. Overall, there are appears to be very little connection between the BAFs calculated in the White Paper and those that might actually be present in Florida freshwater species. Second, FPPAEA believes further research is warranted based on the difference in BAF between PFOA and PFOS. Given that these chemicals generally belong to the same family, it is surprising that the BAFs are different by two orders of magnitude. Third, FPPAEA would like to know what assumptions were used to develop the BAFS, including whether the Department assumed that the fish and shellfish metabolized PFOA and PFOS, and whether the Department relied on USEPA modeling in the development of the BAFs.

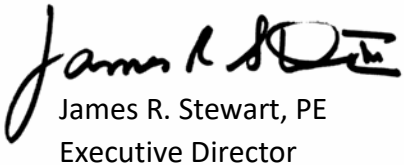
Aside from the variables discussed above, significant questions remain unanswered about the Department's methodology. For example, what concentration of PFOA and PFOS were assumed as starting points in the fish tissue and how was the concentration determined? Was this concentration assumed to drop over time or was it held constant? Were any adjustments made to account for the many fish that spend part of their life in marine waters? Overall, FPPAEA believes that substantially more research and more information is necessary before the Department attempts to utilize these screening levels in any way.

If you have any questions regarding our comments, please do not hesitate to contact me. I can be reached by telephone at (813) 215-8856 or by email at rstewart@fppaea.org.

Thank you for your time and attention to our concerns.

Best Regards,

Florida Pulp and Paper Association EA, Inc.



James R. Stewart, PE
Executive Director

cc: Greg Munson, Gunster Law
FPPAEA Board Members

NCASI
IMPACT. SCIENCE. SOLUTIONS.

Dr. Brian Dougherty
Program Manager
Division of Waste Management and Business Support Program
Florida Department of Environmental Protection
Via E-mail: Brian.Dougherty@FloridaDEP.gov

NOVEMBER 11, 2019

RE: Comments on the Contaminated Media Forum September 12, 2019 presentation on provisional cleanup target levels and surface water screening levels for perfluorooctanoic acid ("PFOA") and perfluorooctane sulfonate ("PFOS").

Dear Dr. Dougherty,

The National Council for Air and Stream Improvement (NCASI) greatly appreciates the opportunity to submit comments on the proposed provisional cleanup target levels and surface water screening levels for perfluorooctanoic acid ("PFOA") and perfluorooctane sulfonate ("PFOS"). NCASI is a research organization engaged in conducting research on environmental topics relevant to the forest products industry. Over its 76-year history, NCASI has conducted studies in a variety of areas related to aquatic biology and human health water quality criteria. NCASI has been an active participant at the state and federal levels in technical and scientific aspects of water quality criteria development for many decades. Our technical work is supported by NCASI Members which include nearly all companies engaged in pulp and paper production in Florida.

It is with this experience that we respectfully submit technical comments for your consideration while the proposed provisional cleanup target levels and surface water screening levels for PFOA and PFOS continue to be developed. Our comments are specifically related to the White Paper "Development of Surface Water Screening Levels for PFOA and PFOS Based on the Protection of Human Health."

The White Paper represents a significant effort to explore potential parameter values for the development of provisional screening values for these two substances; however, there are further scientific resources to consider in the development of these provisional screening values to ensure they represent scientifically defensible outcomes that accurately characterize the exposure pathways and relative toxicity associated with PFOA and PFOS.

NATIONAL COUNCIL FOR AIR
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Specifically, our comments are as follows:

Relative Source Contribution (RSC)

In the White Paper, an RSC of 20% is selected for PFOA and PFOS in deriving their respective provisional screening levels. The state of Florida has historically elected to evaluate substances that may have exposure pathways dominated by drinking water intake for literature that provides an evidenced based RSC in lieu of using the EPA default value of 20%. In fact, 26 compounds that Florida currently has derived Human Health Water Quality Criteria standards for have had literature reviewed for evidence based RSCs in lieu of the EPA default of 20%. This practice produces more scientifically defensible criteria or provisional screening values and would better inform the development of provisional screening values for PFOA and PFOS. This is of particular interest for these substances given that current environmental concern over these substances surrounds contaminated drinking water sources and excludes a substantive evidence-based air exposure pathway. NCASI recommends that further literature review be conducted to derive an evidenced based RSC for PFOA and PFOS.

Freshwater and Estuarine Finfish and Shellfish Consumption Rate (FCR)

In the White Paper, FCR is estimated by relying on the NHANES 2003-2010 fish consumption data. Unfortunately, this database is a federal level database capable of providing resolution to regional levels only, and likely does not reflect the fish consumption rates specific to Floridians, or likewise, cannot provide data on species specific consumption rates for this state. This becomes particularly important considering the need to apply a BAF to PFOA and PFOA to reflect the potential bioaccumulation through the food chain. At a minimum, state specific data on fish consumption rates would greatly improve the scientific quality in the provisional screening values. In the BAF and Probabilistic Risk Assessment sections of these comments, we will also discuss alternative options for more scientifically robust approaches for addressing this issue for PFOA and PFOS.

Selected Bioaccumulation Factors (BAF)

The literature reviewed in the White Paper for studies deriving field BAFs for PFOA and PFOS appears limited to a few articles with the vast majority of BAF measurements originating from a single Chinese study that likely had differences in species and environmental conditions, impacting the generalizability of these results for the purpose of deriving Florida BAFs. A point that highlights the uncertainty of the resultant BAFs is the stark difference between the values arrived at for PFOA and PFOS, two substances with similar molecular structures and toxicokinetic profiles. Expanding the literature search for laboratory and field derived BAFs for PFOA and PFOS would provide the opportunity to explore BAF values that are more relevant to the Florida aquatic environment and phylogeny. As well, conducting an original experimental BAF study using conditions relevant to Florida would increase the scientific defensibility of BAF selection.

Probabilistic Risk Assessment (PRA)

Human Health Water Quality Criteria previously developed by the state of Florida relied on an advanced risk assessment approach known as probabilistic risk assessment (PRA). Under a PRA framework, many parameters used to develop criteria (or, in this case, provisional screening values) may be input as distributions rather than single upper-bound estimates. The benefit of PRA in the development of criteria or screening values is that it can be used to characterize the exposure of a large sample of the population, and not soley a theoretical, upper-bound exposed population segment that may not actually exist. The result of employing PRA is a more transparent achievement of stated health protection targets by taking the whole distribution of parameters into account at once, as compared to the deterministic method that compounds upper bound estimates of exposure one factor at a time (e.g. estimating exposure and risk for someone who has the upper bound estimate of drinking water AND the upper bound estimate of fish consumption AND the upper bound estimate of exposure duration, etc., etc.). NCASI recommends that Florida continue to employ best science practices in their approach to risk assessment and rely on a probabilistic risk assessment approach in the derivation of provisional screening values for PFOS and PFOA.

Please feel free to contact NCASI regarding any of the comments and suggestions presented in these comments.

Submitted Respectfully,

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November 12, 2019

Dr. Brian J. Dougherty
Program Manager, District & Business Support
Florida Department of Environmental Protection
Division of Waste Management
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Re: Provisional cleanup target levels and screening levels for perfluorooctanoic acid and perfluorooctane sulfonate

Dear Dr. Dougherty:

The Chemical Products and Technology Division of the American Chemistry Council (ACC/CPTD) appreciates the opportunity to submit comments on the proposed provisional cleanup target levels (CTLs) and screening levels for perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS). ACC/CPTD represents companies interested in ensuring that regulations and guidance related to these substances, like Florida DEP's proposal, incorporate the best available science. We are concerned about the proposal to apply the Lifetime Health Advisories (LHA) for PFOA and PFOS in drinking water developed by the U.S. Environmental Protection Agency (EPA) to groundwater cleanup levels for the two substances and to establish soil and surface water levels for these substances.

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

The EPA LHAs for PFOA and PFOS 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.[1] Consequently, they indicate a level of conservatism that is inappropriate and unnecessary for groundwater cleanup standards. Cleaning up groundwater to the levels proposed by DEP, moreover, is not the most effective approach to protecting public health.

[1] 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).

Using EPA's tool for developing regional screening levels (RSLs) for chemical contaminants under the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA, or Superfund),[2] and an oral reference dose of 0.00002 mg/kg-day from the EPA Office of Water's derivation of the LHAs, generates RSLs of 400 parts per trillion (0.4 micrograms per liter) for PFOA and PFOS.[3] These values are more appropriate as provisional groundwater CTLs for the two substances.

Although PFOA and PFOS can be removed from water, treatment of groundwater for these substances can be challenging. Removal requires that the water comes in contact with granular activated carbon (GAC) or adsorbent resins. ACC-CPTD is not aware of an effective means for treating PFAS contamination in-situ. DEP's proposal to require cleanup of groundwater to the LHA generally would require "pump and treat" systems whereby the groundwater is brought to the surface, pumped through GAC beds, and subsequently discharged. Such systems are cumbersome and disruptive and generally must operate for extended periods of time to achieve target levels.

The Available Science Does Not Support Combining PFOA and PFOS Concentrations

DEP has proposed to apply the groundwater CTL to the combined concentration of PFOA and PFOS. The grouping of substances under a single value, however, is justified only when the substances are believed to cause health effects by the same ***mechanism*** of action, which is not equivalent to "similarity in effect".[4] Although EPA's LHAs for PFOA and PFOS are based on the lowest doses at which EPA identified developmental effects, these effects are unlikely to be biologically related. The developmental endpoint for PFOS was decreased body weight in rat pups. This differs from the developmental endpoints for PFOA in mouse pups of reduced ossification in males and females (a skeletal effect) and accelerated puberty in males. As such, the critical developmental endpoints identified by EPA do not suggest a common ***mechanism***.[5] Whatever CTLs are used, therefore, they should be applied separately to each substance.

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[2] https://www.epa.gov/risk/regional-screening-levels-rsls.

[3] Based on a Hazard Quotient of 1.0.

[4] 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

[5] 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.

Proposed Soil Leachability CTLs are Not Appropriate for Many Florida Sites

The proposed CTL for soil leachability is based on worst-case assumptions that are unlikely to apply to many – perhaps most – locations in the state. The CTL calculation relies on models that are dependent on several climate- and soil-specific criteria, as well as assumptions about the depth-to-water and flow rates characteristics of the soil. The inputs for these parameters can vary widely, even for simple physical/chemical properties, which can have far-reaching implications for any responsible party facing a soil investigation and potential cleanup.

Specifically, soil leachability screening levels for groundwater protection can be deconstructed to ascertain the appropriateness of the assumptions and level of inherent conservativeness in each input, which are then multiplied for further conservatism. Assumptions about the extent of binding of PFOA and PFOS to soil can vary significantly and likely are the reason why other states and countries[6] have developed levels that are many orders of magnitude greater than those proposed by DEP. For example,

- Several values for the organic carbon-water partition coefficient (Koc) for PFOA and PFOS are found in the recent peer-reviewed scientific literature, including multiple studies with log Koc ranging up to 3.7.[7] These empirical data provide a more appropriate basis for calculating the soil leachability CTL than the Koc value that DEP has adopted from the EPIWIN estimation program. In particular, the presence of iron and other co-contaminants in the soil can significantly affect the Koc, which is likely relevant at many Florida settings.

- The soil type at any given site is likely to have far more than 0.2% organic carbon content (foc) assumed by DEP in calculating the leachability CTLs. While 0.2% is a standard default, consideration of the depth at which a conceptual release of PFOA or PFOS has occurred dictates that information on the relevant soil type and fraction of organic carbon in the soil be considered. Measurements of foc in Florida soils have varied from 1.4% in Orlando to 0.13% from a sand aquifer in Tampa.[8] The CTL for soil leaching at each of these sites should be commensurate with the fOC.

- Consideration of the underlying groundwater quality (whether potable or nonpotable due to high total dissolved solids or salinity) at any site under assessment should also be a determinant in whether or not the leaching pathway is complete and whether assumptions in the spoil leachability CTL are valid.

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6 https://www.rijksoverheid.nl/documenten/rapporten/2019/07/08/tijdelijk-handelingskader-voor-hergebruik-van-pfas-houdende-grond-en-baggerspecie

7 Source Interstate Technology and Regulatory Council. https://pfas-1.itrcweb.org/fact-sheets/

8 https://www.itrcweb.org/DNAPL-ISC_tools-selection/Content/Appendix%20I.%20Foc%20Tables.htm

The proposed CTL for leachability will cause delineation costs to soar as a result of the need for sampling to such low levels and likely need for removal of soil. DEP should ensure a cost-benefit mindset is applied when selecting model inputs (whether default or site-specific) to derive a proposed leaching-protective soil concentration.

Standard Methods for Measurement of PFAS in Soil are Still Being Developed

In addition to our concerns about the overly conservative assumptions used to derive the soil leachability CTL, ACC/CPTD is concerned that validated consensus methods for the extraction and measurement of PFAS levels in soils do not currently exist. We understand that EPA currently is developing validated methods for measuring PFOA and PFOS in soil and encourage DEP to await the results of the EPA effort before proceeding with development of soil levels for these substances. Even under the best conditions, the use of non-standardized methods by contract laboratories are likely to create a high likelihood of false positives and/or increased cost due to the need to differentiate background PFOA and PFOS from source-linked releases. In our judgment, establishing numerical criteria that cannot be used for reliable comparison to inconsistently derived or inadequately supported soil analytical data serves only to confuse the public and increase costs which are borne by the regulated community.

In light of the concerns discussed above, ACC/CPTD urges DEP to align its groundwater cleanup target levels with standard practice and the best available science and to postpone implementation of target levels for soil until validated analytical methods are available.

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