July 1, 2019

Ms. Kara Steward
Washington State Department of Ecology
Hazardous Waste & Toxics Reduction Program
300 Desmond Drive SE
Lacey, WA 98503
Submitted via http://wt.ecology.commentinput.com/?id=x2ChA

RE: Washington State Preliminary Chemical Action Plan (CAP) Recommendations for Per- and Polyfluoroalkyl Substances (PFAS) and Other Updated PFAS CAP Chapters

Dear Ms. Steward:

FluoroCouncil appreciates this opportunity to provide comments on Washington State's Preliminary Recommendations for its Per- and Polyfluoroalkyl Substances (PFAS) Chemical Action Plan (CAP), as well as for the additional updated PFAS CAP chapters. FluoroCouncil[1] is a global organization representing the world's leading manufacturers of products based on PFAS. FluoroCouncil has a fundamental commitment to product stewardship and rigorous, science-based regulation, and, as part of its mission, addresses science and public policy issues related to PFAS.

We understand the important issues currently facing Washington regarding elevated levels of certain PFAS found in multiple locations in the state. Further, we appreciate the significant efforts the departments of Ecology and Health have put into drafting the Preliminary Recommendations and additional updated chapters for the PFAS CAP. As Washington continues with these efforts, it is crucial that the state takes a science- and risk-based approach grounded in a thorough understanding of the broad family of PFAS in order to develop a set of final recommendations that will address these issues in an appropriate and effective manner.

As drafted, however, both the Preliminary Recommendations and additional updated PFAS CAP chapters remain technically inaccurate and fail to identify and focus on the true sources of concern that should be addressed under the CAP. The documents attempt to characterize the extremely broad and diverse group of chemicals referred to as "PFAS," which is a group that includes products and substances that are not PBTs and are not relevant to the contamination

[1] FluoroCouncil's member companies are AGC Inc., Daikin Industries, Ltd., Solvay Specialty Polymers, The Chemours Company LLC, Archroma Management LLC (associate), Dynax (associate), and Tyco Fire Products LP (associate).

issues in Washington. Furthermore, both the Preliminary Recommendations and additional updated PFAS CAP chapters' repeated and unsubstantiated grouping together of short-chain and long-chain PFAS is contradicted by the substantial body of data on short-chain PFAS. We again recommend that Ecology and Health refine their focus to a more narrow and appropriate scope addressing long-chain PFAS and related salts only.

Below is a summary of our comments, and attached are FluoroCouncil's specific comments on both the Preliminary Recommendations and additional updated PFAS CAP chapters, which are offered to provide technical accuracy and a more appropriate, focused scope that would support actions to address the PFAS-related issues in Washington.

A. The PFAS CAP and its Preliminary Recommendations should focus on long-chain PFAS.

1. Certain long-chain PFAS have been found in Washington at elevated levels.

The PFAS-related environmental contamination issues currently facing Washington were found to be related to certain long-chain PFAS, namely PFOS. "Long-chain" and "short-chain" is a distinction that applies to certain PFAS[2] and is recognized by regulators globally.[3] Long-chain PFAS include PFOS, PFOA, and their precursors, including long-chain fluorotelomer-based products.

The distinction between long-chain and short-chain PFAS is not based purely on chemical structure, but also on hazard characteristics, with long-chains having greater toxicity and higher bioaccumulation potential. By contrast, and contrary to both the Preliminary Recommendations and additional updated PFAS CAP chapters, a substantial body of data demonstrates that short-chain PFAS chemicals are not bioaccumulative, are not carcinogenic, and generally exhibit low toxicity. Numerous non-polymeric, long-chain PFAS, including long-chain perfluorocarboxylic acids (PFCAs) such as PFOA and long-chain perfluoroalkane sulfonic acids (PFSAs) such as PFOS, have been classified as PBT substances by regulators around the world. PFOS and its salts are the only long-chain PFAS listed as PBTs in Washington.

Through regulation, the EPA PFOA Stewardship Program and other voluntary initiatives, major manufacturers in the U.S., Europe, and Japan, including FluoroCouncil member companies, worked to successfully phase out long-chain PFAS (including precursors), virtually eliminating these chemicals from their products and facility emissions globally.[4] While PFOS, PFOA, and

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[2] Only non-polymeric PFAS and fluorotelomer-based products can be described as long-chain or short-chain. This description is irrelevant to other PFAS, including fluoropolymers.

[3] Long-chain PFAS are defined by the Organisation for Economic Co-operation and Development (OECD) as:

- PFCAs with carbon chain lengths C8 and higher, including PFOA;
- PFSAs with carbon chain lengths C6 and higher, including perfluorohexane sulfonic acid (PFHxS) and PFOS; and
- precursors of these substances that may be produced or present in products.

See https://www.oecd.org/chemicalsafety/portal-perfluorinated-chemicals/aboutpfass/.

[4] As a result of this phase-out, levels of long-chain PFCAs and PFSAs have been declining in U.S. blood levels. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, "Fourth National Report on Human Exposure to Environmental Chemicals," Updated January 2017 https://www.cdc.gov/biomonitoring/pdf/FourthReport_UpdatedTables_Volume1_Jan2017.pdf.

other long-chain PFAS are no longer produced in the U.S., their production has not stopped outside of the US, Europe, and Japan. Production, use, and sale of these substances and products containing them continues by companies that have not made similar stewardship commitments. This allows products containing long-chain compounds to enter into Washington from abroad, potentially leading to continued exposure and environmental contamination.

Recent environmental monitoring and product testing in Washington shows continued presence of long-chain PFAS. To appropriately address the PFAS-related contamination issues facing Washington, the Preliminary Recommendations should focus on those that would target any long-chain PFAS found at elevated levels in the state and their sources, including products containing those substances.

2. The CAP process is designed to address PBT substances.

The regulations that establish and govern the CAP program recognize that PBT substances present unique risk concerns that require a focused regulatory response.[5] Thus, the CAP program is specifically intended to address the presence of PBT chemicals in Washington and to mitigate the human health and environmental risks associated with those PBT substances. It is not intended to address non-PBT substances, such as fluoropolymers and short-chain PFAS.

The regulations also contain a list of specific PBT chemicals (including groups of chemicals), that are to be addressed under the CAP program. This list identifies "toxic chemicals that require further action because they remain ("persist") in the environment for long periods of time where they can bioaccumulate to levels that pose threats to human health and environment in Washington."[6]

The regulations specify that Ecology will select chemicals for CAP development from the PBT list and that any additions to the PBT list will be accomplished through rulemaking after public notice and an opportunity to comment.[7] Thus, in order to align the PFAS CAP and its Preliminary Recommendations with the purpose and intent of the governing regulations, Ecology must focus the CAP and its recommendations specifically on those PFAS chemicals that are PBT substances, including PFOS. Indeed, the only PFAS chemical included on the PBT list in the regulations is the group of chemicals referred to as "Perfluorooctane sulfonates," which is defined to consist of PFOS acid and various salts.[8] Under the express terms of the regulations, the PFAS CAP and its resulting recommendations must focus on this listed group of chemicals and should not be expanded to include the entire universe of PFAS. Such an expansion would be arbitrary and capricious.

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[5] "Persistent, bioaccumulative toxins (PBTs) are chemicals that pose a unique threat to human health and the environment in Washington State. . . . Because of the unique threat that these PBTs pose, special attention is necessary to identify actions that will reduce and eliminate threats to human health and the environment. . . . The goal of [these CAP regulations] is to reduce and phase-out PBT uses, releases and exposures in Washington." WAC § 173-333-100.

[6] *Id* at § 173-333-300.

[7] *Id*. at §§ 173-333-300 and 173-333-340.

[8] *See* WAC § 173-333-310.

3. It is not appropriate to include other PFAS in the CAP and its Preliminary Recommendations.

In order to achieve the policy objectives of the CAP program, as spelled out in the regulations, Ecology should focus its efforts on substances that are PBTs, such as PFOS and PFOA. Other categories of PFAS, including fluoropolymers and short-chain PFAS, should not be included in the PFAS CAP because they are not PBTs and they are unrelated to the contamination issues facing Washington.

Because fluoropolymers are too large to be bioavailable, they are neither toxic nor bioaccumulative. Their chemical structure and high stability under all types of environmental conditions means they are not precursors to any PFCAs or PFSAs. Therefore, they should be removed from the scope of PFAS included in the CAP. Discussion of fluoropolymers and their uses is not appropriate for the PFAS CAP or its recommendations.

Short-chain PFAS, including short-chain PFCAs, short-chain PFSAs, and their precursors, should also be excluded from the scope of the PFAS CAP. Short-chain PFAS offer similar or superior product performance as long-chain PFAS, but with improved environmental and biological profiles. These short-chain PFAS have been reviewed and approved for use by regulators around the world based on extensive toxicological and environmental testing. The extensive body of research supporting short-chain PFAS shows that, unlike their long-chain counterparts, they have not been classified as PBTs, and consequently are not appropriate to include in the CAP, based on the underlying Washington State regulations. Furthermore, they are not precursors for long-chain PFAS and are not contributing to the long-chain contamination issues in Washington. Therefore, short-chain PFAS should also not be within the scope of the PFAS CAP or its recommendations.

B. PFAS cannot be addressed as a broad class.

PFAS includes a wide variety of chemical substances and polymers with very diverse properties. The term "PFAS" simply means that a substance is highly fluorinated. "PFAS" as a term is too general to be useful for communication purposes and is insufficient to describe a regulatory class. Because there is so much variation among the many chemicals in the PFAS category,[9] no scientifically sound rationale exists for treating them all the same as a matter of public policy.

PFAS vary significantly in their hazard profiles. As discussed above, certain PFAS have been classified as PBTs. However, not all PFAS and related products are persistent, bioaccumulative, and/or toxic, particularly at concentrations typically present in the environment. While some PFAS remain in the environment for years, other PFAS are short-lived and convert to other substances in a matter of hours or days. Not all PFAS persist in biological tissues. While some long-chain PFAS have half-lives in humans that extend for years, other PFAS compounds,

[9] *See* OECD, Summary Report on Updating the OECD 2007 List of Per- and Polyfluoroalkyl Substances (PFASs), www.oecd.org/officialdocuments/publicdisplaydocumentpdf/?cote=ENV-JM-MONO(2018)7&doclanguage=en.

including short-chains, are readily eliminated and do not bioaccumulate.[10] Kinetics studies in animals further demonstrate that the persistence of PFAS compounds decreases with decreasing chain length.[11]

All PFAS also do not share a common toxicity profile. For example, toxicity testing on some PFAS substances shows carcinogenic potential (e.g., PFOA) while similar testing on other substances (e.g., PFHxA) does not show any evidence of carcinogenicity.[12] In addition, even when toxicity testing of PFAS substances may show some similarity of effects, the doses associated with those effects can vary by orders of magnitude from substance to substance.[13]

Sound science dictates that when multiple chemicals have differing toxicity characteristics, they cannot be grouped together for risk assessment purposes.[14] Given the wide variations in toxicities and other hazard characteristics exhibited by different PFAS chemicals, it is scientifically inappropriate to group all PFAS together for purposes of risk assessment.

The broad family of PFAS includes some substances that have been developed and are actually used in commercial applications; however, a large number have not been developed and not all PFAS compounds cited in the referenced OECD report are items in commerce. Additionally, it is important to understand that those PFAS with commercial uses are not used interchangeably. Different PFAS impart different properties, and those in the marketplace have been designed for specific uses, making it essential for public policy to be based on the risks associated with exposure to individual substances in particular uses. For example, fluoropolymers are not used to make grease-resistant food wrappers, and fluorotelomers are not used to make wire and cable coatings. Consequently, the life-cycle impact of any particular compound within the PFAS category can differ by orders of magnitude.

As a result of this significant diversity within the family of PFAS, it is inappropriate to address PFAS as a broad class. Rather, regulatory and policy measures should be substance-specific.

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[10] Chengelis C.P., J.B. Kirkpatrick, N.R. Myers, M. Shinohara, P.L. Stetson, and D.W. Sved. 2009a. Comparison of the toxicokinetic behaviour of perfluorohexanoic acid (PFHxA) and nonafluorobutane-1-sulfonic acid (PFBS) in cynomolgus monkeys and rats. Reprod Toxicol, 27(3-4):342-351. Gannon S.A., T. Johnson, D.L. Nabb, T.L. Serex, R.C. Buck, S.E. Loveless. 2011. Absorption, distribution, metabolism, and excretion of [1-14C]-perfluorohexanoate ([14C]-PFHx) in rats and mice. Toxicology, 283: 55–62. Iwai H. 2011. Toxicokinetics of ammonium perfluorohexanoate. Drug and Chem. Toxicol. 34: 341–346.

[11] Chang S-C, K. Das, D. Ehresman, M.E. Ellefson, G.S. Gorman, J.A. Hart, P.E. Noker, Y-M Tan, P.H. Lieder, C. Lau, G.W. Olsen, and J.L. Butenhoff. 2008. Comparative pharmacokinetics of perfluorobutyrate in rats, mice, monkeys, and humans and relevance to human exposure via drinking water. Tox. Sci. 104: 40-53. Kudo, N., E. Suzuki-Nakajima, A. Mitsumoto, and Y. Kawashima. 2006. Responses of the liver to perfluorinated fatty acids with different carbon chain length in male and female mice: In relation to induction of hepatomegaly, peroxisomal beta-oxidation and microsomal 1-acylglycerophosphocholine acyltransferase. Biol. Pharm. Bull. 29:1952–57. Ohmori, K., N. Kudo, K. Katayama, and Y. Kawashima. 2003. Comparison of the toxicokinetics between perfluorocarboxylic acids with different carbon chain length. Toxicology 184:135–40.

[12] Klaunig, J.E., M. Sinohara, H. Iwai, C. Chengelis, J. Kirkpatrick, Z. Wang, and R. Bruner. 2015. Evaluation of the chronic toxicity and carcinogenicity of perfluorohexanoic acid (PFHxA) in Sprague-Dawley rats. Tox. Pathology 43:209-220.

[13] ATSDR. 2015. Draft toxicological profile for perfluoroalkyls. Agency for Toxic Substances and Disease Registry. U.S. Department of Health and Human Services Public Health Service, August.

[14] As OECD notes, equating the risks of various chemicals for which there are known differences in toxicity is not "scientifically warranted." *See* http://www.oecd.org/officialdocuments/publicdisplaydocumentpdf/?cote=env/jm/mono(2014)4&doclanguage=en at 18. Similarly, if analysis of one chemical using information about another does not create "an accurate and credible assessment of the hazards for the substance in question," then it is inappropriate to read-across between the substances. http://www.ecetoc.org/wp-content/uploads/2014/08/ECETOC-TR-116-Category-approaches-Read-across-QSAR.pdf at 44.

C. Recommendations regarding PFAS in products should ensure an appreciable public health benefit.

The Preliminary Recommendations include funding in the millions of dollars to identify priority consumer products and recommend actions to PFAS in those products. Noting that the PFAS in use in consumer products today have met stringent regulatory requirements that are protective of public health and the environment, such efforts are likely to result in minimal public health benefit. We question whether this is the best use of substantial State resources. We recommend focusing on efforts that would address imported consumer products that are made with or contain long-chain PFAAs, which is no longer state-of the art practice in the U.S., Europe, and Japan.

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FluoroCouncil understands Washington's need to address the PFAS-related contamination issues in the state. It is critical that the approach taken to address those issues be focused on the chemicals found at elevated levels that are within the scope of the CAP process (PBTs): long-chain PFAS.

FluoroCouncil welcomes the opportunity to continue working with the departments to refine the PFAS CAP to ensure it results in a targeted set of recommendations supported by a scientifically and economically sound foundation.

Sincerely,

Jessica S. Bowman
Executive Director
FluoroCouncil

Enclosures:

- FluoroCouncil detailed comments re: Preliminary CAP Recommendations for PFAS and Other Updated PFAS CAP Chapters

Preliminary Recommendations

Draft PFAS CAP (introductory paragraphs) (page 3)

1st paragraph

"PFAS are a group of over 4,700 synthetic organic chemicals. They are used in…."

The above statements imply all 4,700+ PFAS are in commercial use, which is inaccurate. In the recently released EPA PFAS Action Plan, EPA indicated only 602 PFAS were "active" chemicals, and many of those have been phased out under the EPA PFOA Stewardship Program.

2nd paragraph

"Some PFAS have been identified as persistent, bioaccumulative and toxic."

The CAP should be completely clear about the fact that the only PFAS that has been listed as a PBT in WA State is PFOS. A limited number of other long-chain perfluoroalkyl acids (PFAAs), such as PFOA, have been identified as PBTs by other regulatory agencies outside of WA State, and that is worth noting. As we have previously commented, the CAP should focus on PBTs as is the mandate of the CAP program.

4th paragraph

"Because new PFAS continue to be developed for use in products, we included proposals to evaluate and ensure their safety."

Given that the US EPA has not approved a Pre-manufacture Notice (PMN) to allow new PFAS chemistries to enter the market since 2016, this statement is irrelevant.

Why are we concerned About PFAS (page 3)

1st paragraph

"Ecology and DOH are concerned about the class of PFAS…."

In no way do these recommendations or the remaining chapters support the above statement. The broad class of PFAS is not limited to long-chain and short-chain PFAAs, but also includes other PFAS such as fluoropolymers which present no significant toxicity concerns. The CAP chapters generally focus on PFAAs. No justification is provided regarding potential concern for other types of PFAS.

1st-3rd paragraphs

In explaining why Ecology and DOH are concerned about PFAS, these paragraphs seem focused on justifying why short-chains are included in the scope of the CAP. We again note that short-chain PFAS are not PBTs, and thus should not be included in the scope of the CAP. Such inclusion is arbitrary and capricious. Given the presence of long-chain PFAAs in the environment at several locations in WA State at levels exceeding EPA's lifetime health advisory, we would expect this section to discuss why the agencies are concerned about those substances.

PFAS concerns (page 4)

2. Some PFAS have been shown to have harmful health effects.

This is section should be clear about which specific PFAAs have been shown to have links to the listed health outcomes. Citations should be provided. Studies showing potential effects from long-chain PFAAs should not be broadly used to justify concerns with other PFAAs or other PFAS, particularly when studies on those other PFAS show they do not cause the listed health effects.

3. Everyone in Washington is likely exposed to PFAS.

Please be specific about which PFAS have been found in blood in national testing. Again, the presence of some PFAAs in blood should not be used to justify concerns with all PFAS. For example, PFHxA was dropped from CDC NHANES testing because it was not being found. Some PFAS, such as fluoropolymers, are not bioavailable, so will not be present in blood. It is also important to note that levels of PFOA and PFOS in the general population have declined significantly with the phase out of those substances. Are there data to support the statement that some populations may have higher exposures? If so, it should be included to substantiate the statement.

5. PFAS levels in some Washington drinking water supplies exceed recommended levels for public health protection.

Again, please be specific about which PFAS have been found at elevated levels (PFOS and PFOA). These blanket statements about PFAAs are highly misleading and inaccurate.

CAP Recommendations (pages 8-22)

1.0 Ensure drinking water is safe (pp. 8-12)

This section indicates testing of drinking water systems has been extremely limited, but also notes that "exposures to PFAS-contaminated water are disproportionately borne by populations experiencing cumulative impacts, health disparities, and environmental justice considerations." Can this conclusion be drawn if testing has been so limited? Does the limited testing conducted to date support this statement?

1.3 Biomonitoring (pp. 11-12)

We recommend that any biomonitoring program be developed in conformity to protocols developed by the Centers for Disease Control (CDC).

3.2 Identify priority consumer products based on PFAS releases and exposures (pp. 17-18)

There does not appear to be a consideration of hazard based on PFAS used in specific products. We recommend prioritization, including a consideration of hazard. We also recommend discussing how the product testing for PFAS already conducted in WA State will be factored into the prioritization. Furthermore, we recommend the use of validated, quantitative analytical methods for any testing to be conducted.

Please clarify: will two full time Ecology staff and DOH support be devoted directly to this recommended work on PFAS or is that for implementation of the entire law?

3.3 Implement reduction actions for PFAS in priority consumer products (pp. 19-20)

This section discusses one PFAS-related SNUR developed by EPA, including a 2015 proposed (but not yet finalized) SNUR. There are a number of other SNURs for PFAS, and we recommend adding those to the document.

As with the previous section, are the cost estimates for PFAS actions only or for full implementation of SS5135?

Sources and Uses

Summary (page 2)

4th paragraph

All AFFF contains PFAS.

6th paragraph

Only some of the listed household products contain PFAS.

1.0 Introduction (page 3)

1st paragraph

It may be helpful and illustrative to provide examples for the statement, "[s]ome PFAS are highly persistent and mobile in the environment and bioaccumulative in humans," rather than the current open-ended statement.

It is unclear what is meant by "contamination," as the map included is comprised of sites where any amount of PFAS was detected.

2.0 Secondary Manufacturing (page 4)

General comment on uses

The list appears to be missing an essential use for many PFAS end uses – lubrication. *See* IC2 webinar: *The PFSA Universe: Uses, Classification, and Degradation*, http://www.theic2.org/ic2_webinar_the_pfas_universe.

3.0 Fire Fighting Foam (page 6)

1st italicized paragraph

Consider including the following reference:

Perfluoroalkyl Substances in the Environment: Theory, Practice and Innovation. DM Kempisty, Y Xing and L Racz Editors. 2019; Boca Raton, FL; CRC Press; Environmental and Occupational Health Series. *See* Chapter 1, pp 3-34.

We recommend striking NYSPPI 2018 as a core reference, as this document appears to be under revision, due to inaccuracies.

3.1 Fire departments and fire training (page 7)

2nd paragraph

"Use of non-fluorinated foam is recommended, more research is needed to identify safer alternatives to PFAS."

This blanket statement needs to be qualified to make it consistent with the recent law enacted by WA State with clear exemptions: While non-fluorinated foams

may perform sufficiently for some situations, AFFF is the only foam that meets performance needs for some critical Class B high hazard flammable liquid fire-fighting emergencies, including chemical plants and refineries.

Same comment applies to **Section 3.5 page 12, paragraph 1 and Section 3.6 page 12, paragraph 1.**

3rd paragraph

The data presented in Darwin 2011 (below) should be considered for PFOS-based AFFF inventories, which is actually one of the cited references, instead of solely relying on the 2004 report currently cited.

Darwin, RL. 2011. Estimated Inventory of PFOS-based Aqueous Film Forming Foam (AFFF). 2011 update to the 2004 report entitled "Estimated Quantities of Aqueous Film Forming Foam (AFFF) in the United States." Prepared for the Fire Fighting Coalition. July 2011. UNEPPOPS-POPRC13FU-SUBM-PFOA-FFFC-3-20180112.En.pdf.

This same comment applies to **Section 3.2, page 8, Section 3.3 page 9 and Section 3.4, page 10.**

4.3 Landfilled products (page 18)

3rd paragraph

References to the state landfill data cited in the chapter should be provided so the reader can examine the cited information. Also, it is not clear how relevant the German and Chinese data are to Washington State's situation.

Carpet (p. 19)

1st paragraph

Perfluoroalkane sulfonyl fluorides (PASF) are not fluoropolymers; this needs to be corrected. PASF are intermediates or building blocks to make a variety of products but not fluoropolymers per se; however, they are fluorinated polymers.

2nd paragraph

A reference for the statement "Between 50 and 90 percent of carpet is treated…" should be provided. In addition, references are also needed for the broad and blanket statement that "Chemicals used to treat carpet are widely dispersed in areas…"

3rd paragraph, 2nd bullet

The KEMI report, while it may well be a citable reference, significantly mischaracterizes the amount of fluorochemical products used to treat carpets to obtain the historical benefits of oil, water, soil and stain repellency. Typically and historically, approximately 1000 ppm (0.1%) of PFAS product was used to treat and provide the noted benefits. This is 150 times less than the value cited by KEMI. In fact, today's use rates are well below the 0.1% product level. Please contact the Carpet & Rug Institute (CRI) for more information.

Furniture (p. 19)
1st paragraph

"PFBS-based products" (rather than "PFBS fluoride") is a more accurate description of which PFAS have been used to treat leather and upholstered furniture after 2003.

Textiles (pp. 19-20)
1st paragraph

"Current polymer chemistry used for textiles includes polyfluorinated/perfluorinated (meth)acrylate polymers (C2-C20)."

It should be noted that perfluorinated polymers of this type are not used to make these acrylates; however, polyfluorinated are. It also should be documented here that the US, EPA PFOA Stewardship program participants (that are in US, Europe and Japan) do not use C8-C20 polymers and have not used such compounds since the end of 2015 when the industry fully shifted to largely C6-based acrylate products. If long-chains such as C8-type products are making their way into WA State, they are coming from countries such as China and others, not Stewardship program participants. Also, please note in-line text correction in red.

Food packaging (p. 20)
2nd paragraph

Current products on the US FDA food contact notification (FCN) list are short-chain fluorotelomer-based polymers and perfluoropoly ethers, not PAPs and diPAPs. Therefore, the statement containing "(C4-C20)" is incorrect for US FDA FCN products. In addition, the C4-C9 sulfonamide sentence is also incorrect, as are the other statements with products phased out at the end of 2015 in the US EPA Stewardship program.

3rd paragraph, 2nd bullet

Please check the KEMI report to ensure 1.5 percent by weight PFCA in treated paper products. That is a massive amount of perfluorocarboxylic acids; given levels generally reported and/or found are in ppm, not percentages.

Summary (p. 21)
Table 11

Where appropriate, please check and revise the calculations based on the comments provided here on treatment levels, especially in carpets.

7.0 Global estimate: Washington proportion (pages 26-27)

1st paragraph (p. 26)

In addition to the cited references, two other important publications are listed below that are important to understanding a global inventory. Please see these articles:

- Wang Z, et al, Global emission inventories for C4–C14 perfluoroalkyl carboxylic acid (PFCA) homologues from 1951 to 2030, Part I: production and emissions from quantifiable sources..., Environment International (2014), http://dx.doi.org/10.1016/j.envint.2014.04.013.
- Global emission inventories for C4–C14 perfluoroalkyl carboxylic acid (PFCA) homologues from 1951 to 2030, part II: The remaining pieces of the puzzle

Zhanyun Wang, Ian T. Cousins, Martin Scheringer, Robert C. Buck, Konrad Hungerbühler, Environment International 69(2014)166–176.

3rd paragraph (p. 27)

Please note that the Prevedouros et al. publication largely dealt with PFOA emissions and global inventory. FTOH, although discussed, was not a major part of this manuscript.

Table 16

Please note that the data from the Prevedouros et al. study were best estimates as of 2005 (14 years ago). Given the complete change in the industry manufacture and use patterns from 2005 onward (US EPA Stewardship program started in 2006), expectations would be that emissions from WA State may well be significantly lower today than those reported in the Table. For reference, see also the two articles cited above for a more current perspective.

Chemistry

Summary (page 2)

1st paragraph

While it is true that the unique properties of PFAS arise from the strength of the carbon-fluorine bond, in many cases these fluorinated products are both hydrophobic and oleophobic and allow for the development of very low surface tension solutions, as well as the creation of formulations for surface treatment that cannot be obtained with hydrocarbons.

3rd paragraph

The transition from legacy, long-chain to new short-chain chemistries occurred over a 10+ year period (not rapid), as supply chains had to be re-engineered, and customer re-qualification of new products took years in some cases. In addition, a sufficient body of both toxicological and environmental data had to be developed to permit these new products to enter the markets through global regulatory processes.

Modifications for PFAS Chemical Function (pages 8-9)

2nd paragraph (p. 8)

Most often, the fluorosurfactant tail is short rather than long. Historically, fluorosurfactant mixtures in the fluorotelomer-based products (for example) were a homologue mixture with a majority of C6-chain lengths. It is clear that the primary fluorosurfactant used for polymer polymerizations was the ammonium salt of PFOA (7 fluorinated carbons).

Figure 6

"Space" should be replaced with "Spacer" in the figure.

2nd paragraph (p. 9)

The first sentence should read, "Very similar fluorinated monomer structures are used in the production…," as fluorinated monomers are used to make the side-chain products described here.

Figure 7

"Fluorinated monomer" should be replaced with "Fluorinated Side Chain" in the figure.

Electrochemical Fluorination (ECF) (page 11)

1st paragraph

It should be noted in this paragraph that the ECF process produces odd and even chains as well as branched and linear mixtures.

Telomerization (page 11)

From a historical viewpoint, telomerization chemistry was practiced fully from the 1970s onward. While it is true that there was a void when the major producer of ECF materials phased out of the longer-chain materials, shorter-chain ECF products became available in the 2000s. Moreover, it is well-documented that ECF production of long-chains was started in Asian countries such as China to fill the void left by the major global manufacturer who exited production.

Technical Quality and Implications for Environmental Impacts (page 13)

1st paragraph

A more definitive and true statement is: "Suflon®111 was discontinued."; please remove "presumably."

Trends in Per- and Polyfluorinated Substance Design (page 18)

Page number is missing.

1st paragraph

The first sentence may read better if it said, "PFOS and PFOA, both of which are characterized as long chains, have dominated the literature…"

Characteristic Product Uses of PFAS (page 20)

Table 6 (Use Category)

"Carpet…"

The Example Current-Use Products should be clarified: "≤C6 FTI/FTOH."

"Paper and Packaging Treatment"

"FTOH-based PAPs" should be removed from the Example Current-Use Products, as none of these type of products are on the US FDA's current FCN listing as approved products. Also, "≤C6" should be added as a modifier preceding "Side-chain fluorinated polymers."

"Polymer Processing Aids"

The Example Current-Use Product should note "Ammonium salts of PFOA" as an example, not the plain acid.

Carpet and Textile Surface Treatment (page 21)

2nd paragraph

The last sentence should read, "6:2 fluorotelomer products have replaced the longer chain legacy products in the US, Europe, Japan and elsewhere globally."

Paper and Packaging Treatment (page 22)

2nd bullet

Please include a note to the effect that none of these types of products (fluorotelomer-based PAPs) are on the US FDA's current FCN listing as approved products, or consider striking this bullet reference altogether.

Environment

Abstract (page 2)

1st paragraph

The first sentence would read better if it said “Major environmental pathways…have been identified as manufacturing emissions, stormwater…” to be consistent with 1.0 Introduction.

2.4 Surface Water (page 5)

Figure 2

It would be helpful if there were units (e.g. ng/L) in the figure like what was done with Figure 3 on page 7.

3.0 Wildlife studies outside of Washington (page 14)

It may be more useful if current data can be located rather than the 2011 Houde et al. citation to give a more realistic view of what today’s values might be.

List of chemicals discussed (page 5, Appendix)

For consistency, it may be helpful to have all the carboxylic acid names the same. Whereas, most of the acids are named as alkanoic acids, two are named as alkanoates: PFTrDA and PFUnA.

Health

Abstract (page 2)

1st paragraph

The last sentence refers to EPA's lifetime health advisory level of 0.07 ppb. Recommend listing PFOA and PFOS specifically, and for the exceedances in the state of Washington that are referenced, clarify if that was for the sum of PFOA + PFOS, or for individual compounds (or both).

4th paragraph

The last sentence is not correct. It states, "The available epidemiologic studies suggest links between PFAAs exposure and several health outcomes including increases in cholesterol levels, reduction in birth weight, reduction in immune antibody response to childhood vaccines and increases in rates of some cancers such as kidney and testicular." See more detailed comments below for examples of why the interpretation of epidemiological data does not accommodate such a black-and-white statement, even in an abstract. A more supportable summary statement would be: "The available epidemiologic studies are not conclusive with respect to links between PFAAs exposure and health outcomes. Collectively, the strongest links to adverse outcomes appear to include dyslipidemia (particularly total cholesterol and LDL cholesterol), vaccine response, renal function, and age at menarche."

While it is true that some epidemiological studies show links, in fact, a large number of studies indicate there is no consistent link, for example, between prenatal PFAS exposure (as indicated by maternal serum levels during pregnancy) and adverse health outcomes in offspring during early childhood and later developmental periods. While some studies show positive associations (i.e., a change that would be interpreted as adverse), others for the same PFAS compounds show no association, negative associations, or non-monotonic relationships.

ATSDR (2018) and Rappazzo et al. (2017) report that the strongest evidence for a relationship between PFAS exposure and health outcomes in children comes from studies of dyslipidemia (particularly total cholesterol and LDL cholesterol), vaccine response, renal function, and age at menarche. Both references describe studies of asthma as less consistent and only suggestive of a link, given a broader range of study designs and quality. There is less agreement among studies for a link between prenatal PFAS exposure and effects on cardiometabolic function based on anthropogenic measures (e.g., body weight, BMI), serum levels of insulin, glucose, and adiponectin, diagnosis of metabolic syndrome, and incidence of infections and disease during early childhood. The evidence is weak and indicates no clear association between PFAS and IgE antibodies, allergies, thyroid hormones (and incidence of congenital hypothyroidism), and neurodevelopment and attention.

1.0 PFAS contamination of drinking water in Washington state (page 4)

Table 2

In the title, replace "Military detections of PFOS and PFOA…" with "Detections of PFOS and PFOA…"

2.2 Children's exposure (page 16)

1st paragraph

The sentence beginning, "However, children had substantially higher 95th percentile values…" should be reworded to give the x-fold factor higher 95th percentile value, rather than using the phrase "substantially higher" which is unnecessarily subjective. The phrase "x-fold" is appropriately used later in this section, in the second to last sentence.

2.3 Firefighters (page 18)

1st paragraph

In the first sentence, replace "…appear to be slightly above the general population,…" with "…appear to be slightly greater than the general population,…"

3.1 Drinking water (page 20)

1st paragraph

For the last sentence, add the concept that serum levels also depend on non-drinking water (i.e., baseline) exposures (see underlined phrase): "Levels in serum are likely to relate to how long the drinking water exposure occurred, the timing of serum sampling relative to when the exposure occurred, individual consumption and use patterns of drinking water, exposure to food (Section 3.2), consumer products (Section 3.3), and other potential PFAS sources, and other unknown factors."

3.2 Food (page 20)

1st paragraph

For the third sentence, add the word "some": "In the U.S. and Canada, PFOA and PFOS have been detected in some snack foods, vegetables, …"

3rd paragraph

In the subsequent Section 3.3., there is a reference to DNELs for selected chemicals in carpets. Recommend adding a similar concept at the end of this section: "Based on recently derived human health-based chronic toxicity thresholds for PFHxA (Anderson et al., 2019; Luz et al., 2019; ANSES 2017), one of the PFAS compounds associated with environmental occurrence of side-chain fluorinated polymers, there is a larger margin of safety (e.g., 200,000 to 320,000) for even the most sensitive subpopulation, such as infants potentially exposed through breastmilk, cereals, and formula."

4.1 PFAA concerns (pages 23-25)

Liver toxicity and cholesterol levels (p. 23)

In this subsection, only positive associations between serum levels of PFAS and cholesterol are noted. Rappazzo et al. (2017) provides a comprehensive overview of available human data for these endpoints and offers a more balanced perspective. Given that most of the epidemiology studies are cross sectional, temporality in exposure

cannot be established and potential confounders (e.g., diet, disease) may lead to false inferences about causality. Under these conditions, and particularly when the key studies have wide confidence intervals for odds ratios, the biological plausibility of findings should be more fully explored. For example, regarding dyslipidemia, Convertino et al. (2018) question if there is a low-dose causal relationship between PFAS and serum cholesterol, noting it is more biologically plausible that PFOA exposure mediates a *reduction* rather than an elevation in serum cholesterol via a PPARα mode of action. It should be noted that several factors may contribute to reverse causality (i.e., simultaneously affecting an increase serum levels of both cholesterol and PFAS), such as:

1) Conditions that result in elevated serum lipoproteins may increase binding sites for PFNA in serum.
2) Individuals with increased enterohepatic circulation may have higher serum levels of lipids and PFNA due to elevated levels of organic anion transporters in the liver and gastrointestinal tract.
3) Conditions that reduce glomerular filtration rates can result in reduced renal elimination of PFAS.

In addition, although biomarkers such as elevated cholesterol are risk factors for cardiovascular disease, it should be noted that there appears to be no association between serum PFAS and any additional cardiovascular conditions (e.g., Lin et al., 2013; Mattsson et al. 2015; Starling et al. 2014).

Additional notes regarding studies cited by Rappazzo et al. (2017; see the original paper for full references corresponding to citations noted in brackets [ ]).

<u>Dyslipidemia (↑ HDL, LDL, triglycerides)</u>

- U.S. NHANES, adolescents [62, 63]: + for PFOA, PFOS, and total PFAS serum levels and high total cholesterol and LDL-C; null for HDL-C and triglycerides
- Ohio Valley C8 population (children and adults?): + for PFOA, PFOS and high total cholesterol, LDL-C; negative association for PFOS and HDL-C
- Denmark, children [60]: null for PFOA, PFOS and triglycerides in overweight children (ages?)
- Taiwan: + for PFOA, PFOS, and PFNA and total cholesterol, LDL-C, triglycerides
- Avon ALSPAC: + for maternal serum PFAS and total cholesterol, LDL-C, though nonlinear and non-monotonic

Immune toxicity and hypersensitivity reactions

The presentation for this endpoint is more balanced; however, the evidence from human data is weak and indicates no clear association between PFAS and IgE antibodies, allergies, and thyroid hormones (and incidence of congenital hypothyroidism). Also note that Rappazzo et al. (2017) refer to published studies of three populations:

a. Faroe Islands, children ages 5 and 7 years, increasing serum PFAS levels were associated with lower serum antibody titers for tetanus and diphtheria. [e.g., citation 122 in the Feb 2019 report]. Note that there is no indication that the statistical analysis included a control for possible confounding effects of methyl mercury developmental exposure, despite that fact that there is an indication that serum methyl mercury exhibits a moderate positive correlation with prenatal PFOA (i.e., r=0.3) and PFOS serum levels prenatal (i.e., r=0.3) and at age 7 (r=0.4) (Osuna et al. 2014, Supplemental Table 1).

b. Norway, children ages 3 years, increasing maternal plasma PFAS levels were associated with lower serum antibody titers for rubella (for PFOA, PFOS, PFNA, and PFHxS) and also measles (PFOA and PFOS), but the results were not adjusted for potential confounders.
c. U.S. general population, adolescents, increasing serum PFOA and PFOS associated with lower serum antibodies for rubella and mumps [e.g., citation 124 in the Feb 2019 report].

Additional notes regarding studies cited by Rappazzo et al. (2017; see the original paper for full references corresponding to citations noted in brackets [ ]).

Asthma, infection, immunity (n = 13): Immunity (vaccine-mediated antibody response, IgE antibodies):

- Faroe Islands: + for serum PFAS and ↓serum antibody titers for tetanus, diphtheria at 5 and 7 years
- Norway [77]: + for maternal plasma PFOA, PFOS, PFNA, PFHxS and ↓serum antibody titers for rubella at 3 years; also + for PFOA, PFOS and measles vaccine antibody, but unadjusted for potential confounders
- U.S. NHANES adolescents [78]: + for PFOA, PFOS and ↓serum antibody for rubella and mumps
- Norway: null for PFAS and serum IgE antibodies (atopic status)
- Canada [87]: null for maternal serum PFAS and immune function markers in newborns
- Japan [85]: negative for maternal serum PFOA and cord blood IgE for females (null for males); null for PFOS; null for infant allergies and infectious diseases at infant age 18 months.
- Taiwan [86]: + for cord blood PFOA, PFOS and cord blood IgE for males (null for females); null for cord blood PFOA, PFOS, PFNA and serum IgE at 2 years old;
- Taiwan [79]: + for serum PFOA, PFOS, PFDA, PFDoA, PFNA, PFTA and serum IgE
- Zhu et al., asthmatic boys [82]: + for serum PFOS, PFOA, PFDA and serum IgE and cytokines Th1 and Th2

Infections/Disease:

- Denmark, infants 1 year [88]: null for maternal serum PFOA, PFOS and hospitalizations for infectious disease
- Norway, newborn to 3 years [87]: + for maternal plasma PFOA, PFNA and common cold incidence; and + for PFOA, PFHxS and gastroenteritis; but study did not adjust for potential confounders

Quoting and paraphrasing from Rappazzo et al. 2017:

- *Studies of individual health outcomes are limited in number, therefore conclusions should be made with caution.*
- *Current evidence indicates that antibody response to vaccination may be influenced by PFAS; asthma studies also show positive associations, but are less consistent and include a broader range of study designs and quality.*
- *There is no evidence for relationships between PFAS and IgE levels, allergy, and infection.*

- *One study for a cohort in Norway [77] examined a range of outcomes and observed + associations that collectively may indicate that prenatal PFAS exposure is linked to childhood humoral immunomodulation.*

Developmental toxicity (p. 24)

2nd paragraph

Add a final sentence to include information on PFBA and PFHxA:

"Developmental toxicity does not occur at doses of PFHxA below which maternal systematic toxicity occurs, based on a recent reanalysis of results of a mouse developmental toxicity study (Iwai et al. 2019). Similar to PFBA, exposure to PFHxA during pregnancy does not affect survival, growth, or sexual maturation of newborn pups throughout lactation and postweaning developmental periods."

Cancer (p. 25)

Add a final paragraph:

"The collective epidemiologic evidence from 18 studies does not support the hypothesis of a causal association between PFOA or PFOS exposure and cancer in humans, although statistically significant positive associations have been reported in some studies, for example, with cancers of the prostate, kidney, testis, and thyroid (Chang et al. 2014)."

4.3 Toxicology and health effects of short-chain PFAAs (page 27)

PFHxA

1st paragraph

Begin paragraph with:

"Based on a recent comprehensive review of the available animal toxicology data, effects caused by PFHxA exposure are largely limited to potential kidney effects, are mild and/or reversible, and occur at much higher doses than observed for PFOA (Luz et al. 2019)."

Following the 2nd sentence of the paragraph, add a sentence that states:

"Hepatocellular hypertrophy and increased hepatic peroxisomal beta oxidation were reported in male (100 and 500 mg/kg-day groups) and female rats (500 mg/kg-day group)."

A second 90-day subchronic toxicity study has also been conducted with perfluorohexanoic acid (Chengelis et al. 2009), and discussion of this study should be added. Text is provided below.

"In a second 90-day subchronic toxicity study (Chengelis et al. 2009), male and female rats were oral gavage dosed with 0, 10, 50, or 200 mg/kg-day perfluorohexanoic acid. Liver effects were limited to the high-dose (200 mg/kg-day) male group, and included a small increase in relative liver weight, increased incidence of hepatocellular hypertrophy, and increased peroxisomal beta-oxidation. These effects were reversible with a 28-day recovery period, and are consistent with mild liver effects reported in Loveless et al. 2009."

Mild liver effects observed in Loveless et al. (2009) and Chengelis et al. (2009) are consistent with a non-adverse, adaptive response following PPARa activation as

discussed in the diagnostic framework in Hall et al. (2012). A discussion of this should be included.

> "Importantly, mild liver effects reported in the two 90-day sub-chronic toxicity studies are consistent with a non-adverse, adaptive response following PPARα activation and are unlikely to be relevant to humans (Hall et al. 2012).

Following discussion of thyroid effects in the paragraph, add the following sentence:

> "However, a recent weight of evidence analysis demonstrated that PFHxA is not an endocrine disruptor (Borghoff et al. 2018)."

2nd paragraph

Replace paragraph (sentence) with the paragraph below:

> "Human data have been reported for four cross-sectional human epidemiology studies (Luz et al., 2019). Overall, the studies provide some evidence of statistical associations between serum PFHxA levels and testosterone (Zhou et al., 2016), thyroid antibody markers (Li et al., 2017), and Gilbert's syndrome (Fan et al.,2014), and no association with immunological markers or asthma in children (Dong et al. 2013). However, all identified studies are cross-sectional in nature (i.e., can be used to identify associations only, not causal relationships), had other methodological weaknesses, and individuals had co-exposures to other PFAAs that were not controlled for in analyses. Therefore, the available human literature to date does not show a definitive association between PFHxA exposure and any human health disease."

6.0 Health-based guidance values (page 34)

Table 7

Check units in RfD column for consistency – switches between ng/kg-day to ug/kg-day; and ug/kg and ppb and mg/kg and ng/g.

References

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Ecological Toxicology

2.0 Summarizing Ecological Risk (pages 2-3)

No mention of exposure (routes, likelihood) or dose here, in terms of factors that help determine potential ecological risk. Consider adding bullet point with regards to these factors.

2.1 Assessing Ecological Risk Based on Grouping (Short- vs. Long-Chain PFAS) (page 3)

Short-Chain PFAS

1st paragraph

Consider providing examples of short-chain PFCAs (e.g., PFBA, PFHxA) in parentheses within statement in the first bullet point.

Table 1

Consider supplementing the table with more recent studies examining bioaccumulation, in addition to Conder et al. (2008). See comments below for potential additional studies.

2.2 PFAS Representative Substances (page 5)

Consider replacing phrase "the most extensively produced" to "the most commonly detected long-chain PFAS--"

3.0 Persistence and Bioaccumulation within the Organism (pages 5-7)

1st paragraph (p. 5-6)

Consider re-wording introductory paragraph: "The fluorine-carbon bonds present in PFAS compounds confer high chemical and thermal stability, which contributes to their persistence in the environment and resistance to natural degradation."

1st paragraph (p. 6)

Consider adding both citations and additional context with regards to sentence one, which refers to the low-level detections of PFOA, PFNA, and PFDA world-wide (i.e., ng/L vs. mg/L detections).

2nd paragraph

Consider listing other PFCAs and PFSAs that were examined in Reiner and Place (2015) for context, as well as why chemical detects that are close to detection limits come with a degree of uncertainty.

3rd paragraph

This summary of terrestrial bioaccumulation leans heavily on one study by Rich et al. (2015). Consider adding additional literature review summaries to this section (e.g., Das et al. 2015; Morikawa et al. 2006; Mohammadi 2015).

Table 2 (p. 7)

It is unclear whether "Whole unit" in the "Tissue" column refers to whole body samples. There are also some studies examining marine fish and aquatic invertebrates – consider review and inclusion of these as well, such as Jeon et al. (2010), Yoo et al. (2009), Taniyasu et al. (2003).

Consider inclusion of additional studies that examined bioaccumulation in fish and other aquatic receptors, such as Inoue et al. (2012), Houde et al. (2008), and Shi et al. (2018).

4.0 Toxicokinetics and Toxicological Response (pages 7-8)

1st paragraph (p. 7-8)

Notably, the mechanism of action for PFOS and PFOA is still not well understood, despite the fact that these two compounds have been studied extensively compared to other PFAS compounds.

2nd paragraph (p. 8)

Consider removing words such as "undoubtedly" and "avidly."

3rd paragraph

Replace "gender" with "sex."

4.1 Ecological Receptors in the Aquatic Environment (pages 9-10)

Table 3 (p. 9)

Consider separating freshwater and salt water/marine species into separate groups within the table for clarity, and consistency with subsequent subsections.

Consider adding additional literature examining chronic effects on fish, such as Ankley et al. (2005) and OECD (2002).

Marine Environment (p. 9-10)

1st paragraph

This subsection reads more like a summary of occurrence studies, as opposed to an examination or summary of toxicological effects of PFOA and PFOS on marine ecological receptors.

4.2 Ecological Receptors in the Upland Environment (pages 10-11)

Upland Wildlife (and surrogate species) (p. 10)

Consider adding additional literature summaries of publications on terrestrial avian receptors by Newsted et al. (2005) and Molina et al. (2006).

Table 4 (p. 11)

Consider adding NOAEL and LOAEL values from cited sources.

5.0 Summary (page 12)

This summary reverts back to generalizing across all short- and long-chain PFAS for its conclusions. The summary should be specific to what is known about particular chemicals, for consistency with Section 4, and what is understood about their potential toxicity. Extrapolating known toxicological effects from a particular long-chain PFAS chemical to another less well-understood long-chain PFAS chemical introduces a high degree of uncertainty.

References

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Fate and Transport

Abstract (page 2)

Transformation

1st bullet

"All PFAS are either PFAAs or PFAA precursors."

This statement is too broad, making it inaccurate as it would include high molecular weight fluoropolymers as well as perfluoroinerts that are neither PFAAs nor PFAA precursors.

4th bullet

Need to define "PFAS polymer," as the comment and assertions related to it need references to support such comments. It is unclear whether "PFAS polymer" means fluorinated polymers. If so, then define and reference.

Fate

3rd bullet

"…most PFAS are water soluble…"

This very broad statement is inaccurate. While many PFAS compounds are water soluble, many are not due to their chain length and end group structure/functionality.

8th bullet

"PFAS can bio-accumulate in plants and animals…"

This also is a very broad statement that is inaccurate and misleads the reader, as only a finite number of PFAS compounds have been shown to transpire in plants and move from the soil to the roots, stems, leaves, fruit, etc.

2.0 Transformation (page 3)

1st paragraph

Covered by Abstract: Transformation comment above. This statement is too broad and untrue.

2nd paragraph

Need to consider rewriting the last sentence that now says, "PFAAs 'dead end' chemicals … and will most likely exist longer than humans can observe," as it is unclear what this means.

3rd paragraph

"However, current research suggests that all PFAS ever produced will eventually transform into a PFAA…"

This statement is overly broad and thus inaccurate as all PFAS will not transform into a PFAA. For example, fluoropolymers are highly stable and do not degrade into PFAAs. Please correct to clarify not all PFAS can transform into a PFAA.

3.3 Consequences of Chemical Transformation (page 5)

1st paragraph

The paragraph needs to be updated, as PFOS production was not phased out globally in 2002 and PFOA was not phased out globally in 2015. Phase out did occur by the US EPA PFOA Stewardship program participants for their operations globally, but not other companies, such as those based in China and other countries. All Stewardship Program participating manufacturers moved to short-chain chemistries (≤C6 fluorotelomers and <C6 sulfonates) by the end of 2015. Therefore, the blanket statement that "Manufacturers continue to make precursor compounds, which will change into PFOS, [and] PFOA…" is not correct for Stewardship program participants (i.e., all US manufacturers).

In addition, analytical methods have improved dramatically over the past 5 years such that many precursors can be measured. It is true, though, that more analytical capability is needed and is being developed.

4.0 Polymers (page 6)

1st paragraph

It is correct that the biodegradation of fluorotelomer-based acrylate polymers has created a bit of controversy over the years, as the degradation half-life has been reported to vary greatly. The finding is still somewhat unsettled as of today. The Russell et al. work did show slow degradation indicating a half-life of 1200-1700 years for the acrylate polymer studied.

Additional work has been reported at recent conferences, such as Dioxin 2018, that indicate the half-life of a C6 fluorotelomer-based acrylate polymer is greater than 3000 years.

2nd paragraph

As noted earlier, "PFAS polymers" needs to be defined, as it is unclear what it encompasses.

3rd paragraph

There appears to be a lack of understanding of the PFAS chemistry being discussed and described. If PFAS polymers are fluorinated polymers such as fluorotelomer-based acrylate polymers, then the statement, "…the production of PFAS polymer requires the use of monomers and processing aids" is not correct. Processing aids are used in the manufacture of fluoropolymers but not fluorinated fluorotelomer-based polymers. The terminology in this chapter needs to be better described and clarified.

5.2 Water (page 8)

1st paragraph

While it is acknowledged that the historical use of AFFF has resulted in the release of PFAS chemicals into the environment, it should be noted that these releases are localized to the training and testing areas and where emergency fires were fought.

5th paragraph

Please verify the accuracy and add reference for the statement "…the chemical constituents of the flammable materials onto which the AFFF is applied may influence transport of PFAS through the soil and groundwater."

5.3 Solids (page 9)

2nd paragraph

As noted earlier, the first sentence needs to be re-written/qualified, as the following is not a true statement: "Due to the high solubility of most PFAS…"

6.0 Bioaccumulation (page 10)

1st paragraph

The first sentence needs to be re-written to clarify what is meant by: "Levels of PFSAs and PFCAs in organisms are consistently highest among PFAS…" Does this mean to convey that when organisms are studied, the tissue and blood results show that certain PFSAs and PFCAs are most often found and at higher levels than others?

Also, it is well documented that PFHxS is considered a long-chain due to its toxicological and pharmacokinetic behavior, as well as the OECD definition.

2nd paragraph

The discussion appears to conclude that continuous exposure will occur simply because a compound (e.g., PFHxA) is persistent, and that this is the cause for sustained exposure concerns, despite the low biomonitoring results shown to date globally. It is not clear how this conclusion was reached; therefore, it should be substantiated.

It also seems premature to highlight the Persistent, Mobile, Toxic (PMT) concept, given the significant concerns many have about its current validity and applicability, especially around short-chains. If the PMT concept is included, it must be qualified, as it is part of a discussion that is at this time not resolved and/or accepted. In addition, the concept that PMT compounds would be an equivalent level of concern (ELoC) to Persistent, Bioaccumulative, Toxic (PBT) compounds is very far from both being resolved and/or accepted.

4th paragraph

As noted earlier, which PFAS are found in plants should be specified rather than the broad general statement as written.

Biosolids

WWTP Residuals: PFAS Analysis Methods, Concentration, and Trends (pages 5-6)

6th paragraph (p. 5)

It is correct to state that the TOP assay is an aggressive oxidation method, but further explanation should be provided to clarify that the method does not identify the precursor compounds present and, thus, has limited applicability in an application such as described here.

2nd paragraph (p. 6)

It may be helpful to stay consistent with the use of PFAS for the description of per- and polyfluorinatedalkyl substances rather than use "perfluorochemicals."

Literature Review of Biosolids Land Application Effects (pages 7-8)

1st paragraph (p. 7)

Decatur should be Decatur, Alabama, not Georgia.

2nd paragraph (p. 8)

It is noted that the cited study shows a yield increase from biosolids applications. The next sentence infers that the presence of trace PFAS in biosolids could adversely affect crop yields. Please provide a reference for this assertion.

Biosolids Policy Discussion (page 12)

3rd paragraph

"The *perception of risk* resulting from extremely low concentrations that may not have scientifically demonstrated human health risks could have adverse impacts on generators."

We appreciate the position taken and noted in the above sentence.

Analytical Methods and Techniques

Summary (page 2)

1st paragraph

To date, few methods have been both validated and verified by multiple laboratories. The paragraph should include a discussion of the importance of multi-laboratory validation and rugged methods, which must be developed to be reproducible from laboratory to laboratory and from analyst to analyst. Currently, this significantly limits the testing methods and laboratories available for PFAS analysis.

1st - 2rd paragraphs

The last sentence of introductory paragraph should be combined with the 2nd paragraph (see below). Also, please note in-line text correction in red.

> "A multi-laboratory validated method, USEPA method 537.1 version 1.0 (USEPA 2018) was published in November 2018 for the analysis of 18 PFAS analytes in drinking water. Method 537.1 is a solid phase extraction (SPE) liquid chromatography/tandem mass spectrometry (LC/MS/MS). Surrogate and internal standards are used to monitor for analyte loss due to sample preparation, instrument drifts, or matrix effects. This method is limited to the analysis of selected PFAS in drinking water samples."

Non-specific methods for PFAS analysis (page 9)

4th paragraph

"Measuring PFAS as a class (total PFAS) due to their persistent nature and toxicity is probably a more appropriate way for assessing exposure and risk to human health and the environment."

The above statement is not accurate and cannot be substantiated; further, it appears to contradict earlier discussion in the same section. The 2nd paragraph (p. 9) discusses how short-chain PFAS products such as PFHxA are less toxic or hazardous to human health than long-chains. Furthermore, in the 1st paragraph (p. 9), it is noted that "The majority of the estimated 4,700 PFAS currently on the world market have very limited or no toxicity information (Wang et al., 2017), indicating a critical data gap on the full extent of PFAS." It is also important to note that some PFAS (e.g., fluoropolymers) are not water soluble or bioavailable; thus it is not possible to measure for PFAS as a class.

Non-standard analytical techniques for measuring PFAS (pages 9-10)

For Combustion ion chromatography (CIC); Extractable organic fluorine (EOF); and Adsorbable organic fluorine (AOF) methods, there may be a limit on use of these analytical techniques due to limits of detection in different sample matrices. This is briefly mentioned on page 9, but may warrant additional discussion on the specific limit of detection for each method on page 10. Many health advisory limits for PFAS are being set to ppt levels and these methods may only be suitable for measuring ppb.

EPA update on PFAS analytical method development and validation efforts (page 13)

1st paragraph

"Currently, there are no standard EPA methods for analyzing PFAS in surface water, non-potable groundwater, wastewater, or solids. For non-drinking water samples, some U.S. laboratories are using modified methods based on EPA Method 537. These modified methods have no consistent sample collection guidelines and have not been validated or systematically assessed for data quality."

This is a very important statement regarding currently available analytical methods and techniques and should be generally discussed in the Summary section. Furthermore, on June 21, 2019, EPA released SW-846 Update VII, Phase II – Method 8327: Per- and Polyfluoroalkyl Substances (Using External Standard Calibration and Multiple Reaction Monitoring (MRM) Liquid Chromatography/Tandem Mass Spectrometry (LC/MS/MC) for public comment. While not finalized, this draft method would allow for the analysis of 24 PFAS analytes in reagent water, surface water, groundwater, and wastewater matrices. Therefore, it should also be discussed in this section.

Overall, the Analytical Methods and Techniques chapter was well-written and provided a rather thorough description of the analytical methods and techniques currently available for PFAS and fluorine analysis. However, at this time, there are many analytical challenges and questions facing PFAS analysis in regards to risk assessment, and this should be emphasized and discussed briefly in the Summary section. These challenges include establishing defined objectives of the analysis (e.g., measuring Total Fluorine vs. individual PFAS for human risk assessment). Other factors that should be considered include:

- What methods are robust and rugged enough for generating reliable data that can be compared from laboratory to laboratory and analyst to analyst?
- How to choose a lab for specific types of testing; will they need to be certified?
- What constituents to test for (all PFAS vs. specific PFAS)?
- How much will the analysis cost?
    - For example, PIGE analysis is limited to academic and non-commercial research facilities (as mentioned on page12) and may not be a practical technique for commercial laboratories. Cost and turnaround time for PIGE is relatively inexpensive and fast; however, instrumentation cost is significant and may only be used for PFAS analysis in a commercial lab.

Currently, there is a lack of guidance on detection and reporting limits from regulatory agencies. Once these guidelines have been established, it may limit the analytical methods and techniques available for testing.

Regulatory

Summary (page 2)

3rd paragraph

"Many other states developed standard or guidance for ~~PFAS~~ in drinking water, groundwater…

- Air release of ~~PFAS~~:
- Consumer products to restrict the use of ~~PFAS~~…"

The use of the term PFAS in the above excerpt is incorrect and overly broad. A suggested, more appropriate revision would be "a short list of non-polymeric PFAS compounds, primarily PFOS & PFOA."

1.1 Washington state laws (page 3)

Chapter 70.95G RCW

1st paragraph

"Packages Containing Metals and Toxic Chemicals law prohibits PFAS in paper or paperboard food packaging where safer alternatives for specific applications have been determined to exist. PFAS in this law is defined as a class of fluorinated organic chemicals containing at least one fully fluorinated carbon atom. Ecology is required to identify ~~that~~ whether safer alternatives to PFAS in food packaging are available, through the completion of an alternatives assessment. ~~The~~ A ban on PFAS in food packaging takes effect after two years only if Ecology identifies that safer alternatives exist. The earliest date ~~the~~ a ban would take effect is January 2022."

This statement incorrectly summarizes the law and requires the above clarifications (in red).

1.2 Washington state rules (page 3)

Chapter 173-303 WAC

1st paragraph

"This regulation includes a category of state-only dangerous waste based on toxicity and persistence. Halogenated organic compounds are state-only persistent wastes. All PFAS are halogenated, therefore any waste containing PFAS at concentrations above 100 parts per million designates as a state-only dangerous waste and must be handled and disposed as required by the Dangerous Waste Regulations."

All PFAS do not exhibit the properties of both 'toxicity and persistence' therefore it is not clear how any waste containing PFAS above 100ppm would qualify as dangerous waste.

1.3 Executive orders (pages 4-5)

Executive Order 04-01

1st paragraph

"Several state purchasing efforts have focused on reducing the presence of PBTs in state products. State purchasing preferences efforts related to PFAS have focused on PFAS-free carpet and food packaging."

This statement is in error. This particular executive order specifies PBT criteria and does not include any statement about PFAS.
https://www.governor.wa.gov/sites/default/files/exe_order/eo_04-01.pdf

2.1 Environmental Protection Agency (page 5)

PFOS voluntary phase-out

1st paragraph

"The 3M Company, the only U.S. manufacturer of PFAS voluntarily discontinued use of PFOS in the United States in 2000 and phased out PFOS chemistries globally by 2002 (USEPA 2019a)."

This statement incorrectly states that 3M manufactures PFAS. No company manufactures PFAS, an OECD definition that covers a vast array of compounds. 3M does manufacture certain of PFAS products based on the electrofluorination process. 3M was the only U.S. manufacturer of PFOS.

2.2 Food and Drug Administration (page 6)

1st paragraph

"In 2011, the FDA and several manufacturers reached a voluntary agreement to stop interstate distribution of products containing long-chain PFAS (USFDA, 2017). In 2016, because the industry had discontinued the use, two PFAS were removed from the list of approved substances for oil and water repellants for paper and paperboard for use in contact with food (USFDA 2016)."

This statement is incorrect, and needs the following clarifications (in red):

In 2011, the FDA and several manufacturers reached a voluntary agreement to stop interstate distribution of products containing long-chain PFAS surfactants (USFDA, 2017). In 2016, because the industry had discontinued the use, two long-chain PFAS surfactants that were no longer in commerce were removed from the list of approved substances for oil and water repellants for paper and paperboard for use in contact with food (USFDA 2016).

3.2 Firefighting Foam (page 7)

1st paragraph

Please add the following sentence at the end of the paragraph, which is also currently missing a period:

"Additionally, responsible legislation that codifies industry best practices to ban the use of AFFF in training and testing, while allowing for emergency fire-fighting situations has been passed in Virginia, Kentucky, Georgia, Arizona, and others."

Economic Analysis

Introduction (page 1)

The goal of the economic analysis is supposed to be to estimate the costs of implementing the CAP - both those borne by WA State and those borne by others. The assessment in this chapter barely does that and, consequently, does not provide a realistic view of the cost to implement the CAP.

While this chapter presents an assessment, it is not appropriately coined an economic assessment, as it is noted that the assessment is based on a lot of estimating and a fair amount of qualitative analyses. A comprehensive economic assessment should be done before carrying out the ambitious set of programs described in the CAP.

Recommended actions analyzed (pages 2-3)

Action 2.3 bullet (p. 2-3)

It is not clear whether WA State has AFFF manufacturing processes. It may also be unrealistic to "require" industrial sites to collect and dispose of all AFFF in an emergency fire situation. Having plans in place is one thing, but a full requirement to collect and treat may not be feasible or practical.

Action 3.1 bullet (p. 3)

This is an aggressive stance without an first determining if appropriate and conducting an upfront alternatives analysis (discussed later) and may be the first step in banning all treated carpet products in WA State.

Action 3.3 bullet

This is an aggressive stance without an upfront alternatives analysis (discussed later) and may be the first step in banning all of these consumer products in WA State.

Costs of recommended actions (pages 10-20)

Action 2.3: Work to prevent PFAS releases from firefighting foam use and manufacturing processes.

Runoff collection plan (p. 10)

It would be useful to better understand where WA State obtained their costs estimates for setting up a collection plan, which seem low. Costs to set up and implement this plan could be more costly depending on if using internal resources and not hiring from the outside.

Existing stored product (p. 12)

"In lieu of ongoing use of AFFF, facilities have options of either disposal or other removal of the product, and replacement with an alternative PFAS-free product. Other removal options include selling to facilities in states with less-restrictive regulations, or transporting to another facility under the same organization (such as another military facility)."

Facilities need to assess if they need AFFF and how they will comply with the WA State law when it enters into force. Also, it is not clear whether it is a good idea for WA State to shift its AFFF burden to another state.

Action 3.1: Reduce PFAS exposure from carpet and carpet care products

2nd paragraph (p. 13)

It is noted that "[r]ecent and previous research has shown carpet to be a repository for pollutants and that indoor air quality declines when carpeted areas are disturbed."

Please clarify whether ordinary use and care of home carpets constitutes an applicable disturbance and substantiation for this statement.

"According to the Carpet and Rug Institute (CRI), carpet accounts for 51 percent of the U.S. flooring market. PFAS, largely used for stain repellent in carpet, were worth close to $1 billion worldwide in 2006 for this use."

CRI should be contacted so more current figures for this information can be obtained and included.

2nd paragraph (p. 17)

"Facing higher unit prices, buyers may substitute away from carpet and choose other floor coverings that are PFAS-free instead of PFAS-free carpet. This could lower overall costs, though we note that carpet may be chosen for safety, acoustics, or other qualities, instead of other floor coverings."

The conclusions drawn here seem a bit excessive with not enough information to substantiate them. People and businesses choose treated carpets for a variety of benefits – soil and stain resistance, noise reduction, aesthetics, etc. These and other qualities are not available from other surfaces.

Action 3.2 Implement reduction actions for PFAS in priority consumer products (pp. 17-20)

While there is substantial discussion in this section, specific PFAS were not identified in any of the priority consumer products category, nor were the location of where specific PFAS may be used in those products or what the benefit might lost in the removal of the specific PFAS. This section should be supported with this additional information.

<u>Costs of other options analyzed</u> (page 22)

Cost if Ecology assess alternatives

While a comprehensive alternatives analysis (AA) may be an appropriate path forward before determining whether a product should be substituted, it may not always be a good use of WA State funds to conduct AAs on all the listed applications, given some may be very minor uses in WA State and provide even smaller benefit by switching. Furthermore, it is still not clear what WA State will ultimately do, so clarification should be provided.

Benefits of recommended actions (page 24)

Human health and wellbeing benefits

"Despite the emerging nature of PFAS health impact literature, several trends in human health conditions associated with PFAS exposure are easily identified within the literature. These include: Increased risk of thyroid disease and endocrine system disruptions. Increased risk of certain cancers. Higher cholesterol levels. Reduced antibody response to vaccinations."

The above statement can and will be readily taken out of context and needs to be qualified. The qualifications must include that these are "associations" and not cause-effect relationships, and they should be specific on what PFAS, rather than inaccurately stating "PFAS" broadly.

Costs from likely PFAS-related health conditions (pages 24-27)

This entire section should be removed, as it is a gross misrepresentation of the available data and overly sensationalistic. The specific PFAS associations noted above have not been shown to cause the diseases cited. In each section, the discussion is qualified with the following (or a similar) sentence:

"It is not, however, possible at this time to determine the proportion of [insert health condition] cases related to PFAS exposure."

This is not an appropriate nor helpful way to qualify or frame the discussion, and, therefore, the section should not be included unless the information can be appropriately substantiated.

Ecosystem services (page 28)

2nd paragraph

"In the case of PFAS emissions, the persistence and bioaccumulation of the chemicals are shown to have negative impacts on the health of water ecosystems impacts to organisms in the environment"

If a broad statement such as the one above is included, it must be qualified with specific references and multiple specific examples.

Persistence and Bioaccumulation (page 29)

1st paragraph

"Both short- and long-chain PFAS are distributed throughout the environment."

While certain PFAS are in the environment, it is an over-exaggeration to say "throughout" and refer to all PFAS without clear specificity.

"PFAS are stable in anaerobic and aerobic conditions and are not biodegradable."

As written, the above statement is incorrect. Some PFAS compounds – e.g. precursors - do indeed biodegrade to stable metabolites.

2nd paragraph

"The high potential for persistence and distribution, and the soluble nature of PFAS has allowed their accumulation throughout the food chain with adverse impacts to individuals, populations, and communities."

The above statement is overbroad and misleading and should be corrected accordingly with specificity.

"Although fluorinated alternatives to long-chain PFAS may be less bioaccumulative, their density in products may be higher as their performance is less, which may nullify their benefit of being less bioaccumulative."

The above statement is not true and should be removed in its entirety.