Oyebode A. Taiwo
Corporate Medical Director

3M Corporate Occupational Medicine

3M Center, Building 0220-06-W-08
St. Paul, MN 55144-1000 USA
Office: 651 736 2350
Mobile: 651 285 2983
Fax: 651 733 9066
Email: oataiwo@mmm.com

January 22, 2021

VIA ELECTRONIC SUBMISSION

Washington State Department of Ecology
Washington State Department of Health

To Whom It May Concern:

The 3M Company (3M) appreciates the opportunity to review and provide comments on the Per- and Polyfluoroalkyl Substances Draft Chemical Action Plan (Draft CAP). 3M is a science-based company with substantial experience, expertise, and product stewardship related to per- and polyfluoroalkyl substances (PFAS). It is with that background in mind that 3M offers comments on the Draft CAP.

The body of scientific evidence does not demonstrate that PFAS cause adverse effects in humans. The available peer-reviewed scientific literature do not support the health concerns cited in the Draft CAP. In addition, the evidence does not support the Draft CAP's premature conclusion that any PFAS, individually or as a group, should be classified as hazardous substances under the Model Toxics Control Act. Likewise premature are the Draft CAP's suggestions that it will impose monitoring requirements or effluent limitations on wastewater treatment plants or that it will move to limit the PFAS content in certain products.

3M requests that the Department of Ecology (Ecology) and Department of Health (Health) consider and incorporate 3M's comments when finalizing the CAP.

I. Sound Science Must Form the Basis of Policy and Regulatory Action

a. *The body of scientific evidence does not show adverse effects in humans from PFAS*

The vast body of scientific evidence does not show that PFAS – either individually or as a group – cause adverse health effects in humans. While there remains some uncertainty in the science, the evidence available today does not support the statements made in the Draft CAP. 3M has provided extensive comments to other agencies, including the Agency for Toxic Substances and Disease Registry (ATSDR), regarding the lack of scientific support and consensus around claimed impacts on fetuses and infants, cancer, antibody response, and other

issues. 3M will provide those comments to Ecology and Health and participate in a technical discussion if helpful.

In 2018, ATSDR concluded regarding perfluoroalkyls: "The available human studies have identified some potential targets of toxicity; however, cause and effect relationships have not been established for any of the effects, and the effects have not been consistently found in all studies." ATSDR 2018 Analysis at 635-36 (emphasis added).[1]

Another authoritative body, the Australian Expert Health Panel, concluded in March 2018 that "there is mostly limited or no evidence for any link with human disease from these observed differences. Importantly, there is no current evidence that supports a large impact on a person's health as a result of high levels of PFAS exposure." Expert Health Panel for PFAS: Summary at 2 (emphasis added).[2] The report further stated: "After considering all of the evidence, the Panel's advice … is that the evidence does not support any specific health or disease screening or other health interventions for highly exposed groups in Australia, except for research purposes." *Id* (emphasis added). Like ATSDR, the Australian Expert Health Panel analyzed hundreds of studies when reaching this conclusion. Expert Health Panel for Per- and Poly-Fluoroalkyl Substances (PFAS), March 2018 at 382-403.[3]

Finally, the information on "The C8 Health Project" included as an introduction to and premise for the Draft CAP's Epidemiology section is misleading and outdated. *See* Draft CAP at 296. Scientists and collaborators who formed the "C8 Science Panel" recently reviewed the current literature with respect to each of the health conditions potentially linked to PFOA.[4] The article concludes the epidemiological evidence remains limited and questions the broader implications drawn from their prior work, noting that it assessed a single population and that additional studies would be expected to vary. The article's findings include:

- **Increased blood cholesterol** – the authors reviewed additional studies regarding the effects of PFOS and PFOA on serum cholesterol levels. While these more recent studies did generally support an association between exposure and increased levels of cholesterol, the magnitude of the cholesterol effect is inconsistent across different exposure levels in the epidemiologic studies and is not supported in the toxicological studies. The article notes there is not consistent evidence that exposure translates to an increase in cardiovascular disease risk.

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[1] Available at https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf.
[2] Available at https://www1.health.gov.au/internet/main/publishing.nsf/Content/C9734ED6BE238EC0CA2581BD00052C03/$File/summary-panels-findings.pdf.
[3] Available at https://www1.health.gov.au/internet/main/publishing.nsf/Content/C9734ED6BE238EC0CA2581BD00052C03/$File/expert-panel-report.pdf.
[4] *See* Kyle Steenland, Tony Fletcher, Cheryl R. Stein, Scott M. Bartell, Lyndsey Darrow, Maria-Jose Lopez-Espinosa, P. Barry Ryan, David A. Savitz, "Review: Evolution of evidence on PFOA and health following the assessments of the C8 Science Panel," Environment International, Volume 145, 2020 (*available at* https://doi.org/10.1016/j.envint.2020.106125).

- **Ulcerative colitis** – the authors reviewed four additional published studies and concluded that while the evidence still supports a possible link, more studies are needed to reach definitive conclusions.

- **Thyroid function** – the authors concluded the evidence of an association of PFOA with thyroid disease has, in fact gotten weaker. The review focused on studies of a 2019 Swedish community regarding exposure to PFOS and PFOA.

- **Testicular cancer** – based on their review, the authors concluded that as a general matter, the evidence does not support PFOA being considered carcinogenic for any given site. Specific to testicular cancer, the authors noted that the evidence for an association is suggestive but noted it is a rare type of cancer, limiting possible conclusions.

- **Kidney cancer** – likewise, the authors concluded the evidence for an association between exposure to PFOA and kidney cancer remains suggestive. They cautioned, however, that this determination is inconsistent with newer studies, including a 2014 study of high-exposure workers.

- **Pre-eclampsia and elevated blood pressure during pregnancy** – the authors determined the C8 Science Panel conclusions were relatively insensitive to potential errors in exposure and toxicokinetic models. Two new studies reviewed proved inconclusive as to an association between PFOA and pre-eclampsia.

The broad assertions made in the Draft CAP regarding potential health effects of PFAS are inconsistent with peer-reviewed science and government publications. The Draft CAP studies and publications cited do not include the most recent data and studies and do not support the broad claims of health impacts. The document relies on a number of publications by other states and federal agencies, many of which are flawed, in draft form, or otherwise problematic in this context. For instance, the document relies on state and federal materials, including those published by New Jersey, New Hampshire, Minnesota, and EPA, upon which 3M has already provided extensive technical comments. In short, the State should be cautious in the Draft CAP not to simply duplicate erroneous and incomplete work done by other agencies.

When finalizing the CAP, Ecology and Health should review and incorporate the latest scientific research and rely primarily on peer-reviewed information that has been published in its final form, taking into account comments from the public and experts. In addition to the information above from the Steenland report, 3M notes:

- PFOS and PFOA do not cause increase in serum lipid in laboratory animals. Several observational epidemiological studies have reported an association between PFOA exposure and increased cholesterol levels but the magnitude of effect is entirely inconsistent across exposure levels. These findings are inconsistent with experimental studies which have observed <u>decreased</u> cholesterol levels with markedly higher PFOA concentrations. These experimental studies

now include a Phase 1 clinical trial in humans (Convertino et al. 2018) and a transgenic mouse model that mimics human lipoprotein metabolism (Pouwer et al. 2019). There is no evidence of increased risk of cardiovascular mortality in the highest exposed occupational cohorts (Steenland and Woskie 2012; Raleigh et al. 2014) based on worker analyses in these studies which minimized the healthy work effect.

- There is no known association between PFOA or PFOS with human liver disease including enlarged liver, fatty liver, cirrhosis, or liver cancer. Small percentage changes in alanine aminotransferase (ALT) have been reported, albeit inconsistently in epidemiology studies across vastly different perfluoroalkyl concentrations but are within normal physiological ranges. This small magnitude of change, if it is even present, does not indicate liver damage by any standard clinical practice of medicine. *See* 3M Comments on ATSDR Draft Toxicological Profile for Perfluoroalkyls (August 20, 2018) (hereinafter "3M ATSDR Comments").

- The absence of clinical immunosuppression along with inconsistent findings both within and across studies do not support a link between PFAS levels and decreased antibody responses to vaccines in humans. There is highly inconsistent evidence to suggest an association of PFAS with an increased risk of infection in children. *See* 3M ATSDR Comments; 3M Comments on European Food Safety Authority Draft Scientific Opinion on the "Risk to human health related to the presence of perfluoroalkyl substances in food" (April 20, 2020).

- There is insufficient evidence in the literature to conclude that an association between thyroid disease and exposure to PFAS exists in humans. *See* 3M ATSDR Comments; Li et al. 2021;[5] Andersson et al. 2019.

- The levels of PFOS or PFOA causing a potential reproductive or developmental toxicity in rodents are several orders of magnitude higher than the levels experienced by the general human population, demonstrating an ample margin of safety. *See* 3M Comments to California Office of Environmental Health Hazard Assessment on Prioritization of PFOS (October 19, 2020) and PFHxS (November 16, 2020). In laboratory animals, fetal effects generally occurred at maternally toxic dose levels and no fetal changes were present at nontoxic material doses. *Id*. Similarly, EPA has been unable to establish a causal relationship between PFOS or PFOA and reproductive toxicity in humans. The evidence from two meta-analyses now indicate a non-causal association with lower birthweight for PFOA (Steenland et al. 2018) and PFOS (Dzierlenga et al. 2020) as it is likely due to confounding related to the maternal timing of the blood measurement and the physiological changes in pregnancy between first and second/third trimesters as related to the glomerular filtration rate.

[5] Prepublication draft available at https://www.sciencedirect.com/science/article/abs/pii/S0013935120315449.

b. The Draft CAP should account for the phase-outs of PFOA and PFOS, as well as the decline of those chemicals in blood serum

As the State has recently recognized, PFOA and PFOS have been voluntarily phased out across the United States, and the presence of certain PFAS have been declining in blood serum. Draft Recommended State Action Levels for PFAS in Drinking Water: Approach, Methods and Supporting Information (Updated August 2020) at 9, 26 (Supporting Information). 3M was one of the main manufacturers of PFOS in the United States. The company initiated a voluntary phase-out of these chemicals in 2000. That phase-out was largely complete in the United States by the end of 2002 – a full 18 years ago. After 3M ceased the manufacture of PFOS, EPA promulgated federal regulations that prevent other manufacturers (as well as 3M) from manufacturing or importing PFOS or PFOS precursors, subject to a handful of very narrow critical use exceptions with limited exposure potential approved by EPA. These regulations have been in place for nearly two decades. EPA's rules allowed the continuation of a few specifically limited, highly technical uses of these chemicals for which no alternatives were available, and which were characterized by very low volume, low exposure, and low releases. Any other uses of these chemicals would require prior notice to and review by EPA.

The Supporting Information points to no evidence that production or frequent discharges of PFOA and PFOS continue in the United States in general, or in Washington State in particular. For instance, EPA has published data indicating that production and import of PFOA and PFOS have halted or dropped below Chemical Data Reporting Program reporting thresholds. *See* 85 Fed. Reg. at 14115. PFOS has not been reported to EPA as manufactured or imported into the United States since at least 2006.[6] In addition, countless countries have signed onto the international Stockholm Convention, including China, which now requires the elimination of PFOS in essentially all consumer and other goods originating in member countries. And, significant federal action relating to PFOS and PFOS precursors has been underway since 2002, and EPA has imposed and continues to ratchet up strong restrictions on the manufacture, import, and use of PFOS and PFOS precursors pursuant to its Significant New Use Rule authority under the Toxic Substances Control Act.

Since the phase-out of PFOS began in 2000, there has been an unmistakable downward trend in residues of PFOS in human blood.[7] Studies show that from 1999 to 2014, blood PFOS levels in the United States have declined by more than 80% and PFOA levels have declined by more than 60%. Agency for Toxic Substances and Disease Registry, PFAS in the U.S. Population, https://www.atsdr.cdc.gov/pfas/health-effects/us-population.html (last accessed December 28, 2020); *see also* 85 Fed. Reg. 14115 (EPA reporting a decrease of over 75% in the 95th percentile serum PFOS concentrations between the 1999-2000 cycle and the 2015-2016 cycle).

[6] Available at https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/fact-sheet-20102015-pfoa-stewardship-program#mfg.

[7] The mere presence of PFOS in blood serum, without a full understanding of the broader influencing factors, provides only a limited view of exposure risk.

The State must consider these documented declines more thoroughly in determining whether and how to move forward. Washington must present a full and accurate picture of the state of the science, use, and exposure potential for each PFAS it considers regulating.

c. *PFAS are not known to occur in public water systems at levels of public health concern*

The State should consider comments submitted by 3M to the State Board of Health (Board) on September 30, 2020 concluding that PFAS do not occur in public water systems at levels of public health concern. For this reason, the Board's proposed State Action Levels (SALs) for Perfluorbutane Sulfonic Acid (PFBS), Perfluorhexane Sulfonic Acid (PFHxS), Perfluorononanoic Acid (PFNA), Perfluorooctanoic Acid (PFOA), and Perfluorooctane Sulfonic Acid (PFOS) are an improper mechanism to address the scope and scale of the State's concern.

The limited discussion of occurrence of PFOA, PFOS, PFHxS, PFNA, and PFBS in the Supporting Information issued with the proposed SALs appears to be contrary to a finding of occurrence in public water systems at any meaningful frequency. According to the Supporting Information, 132 public water systems in Washington, including all Class A systems, were monitored for six PFAS, and only one water system had a well that exceeded EPA's lifetime health advisory level for PFOA and PFOS. Supporting Information at 7. The Supporting Information also cites to two surveys of U.S. drinking water that "show low percentages of drinking water systems with significant PFAS contamination." Supporting Information at 18.

Furthermore, the data that the Board relied on was not particularly current. The Washington data was collected from 2013 to 2015, and the federal data is, at minimum, 5 years old. Not only has the occurrence of many PFAS likely declined since the cited data was collected, it should continue to decline, and the Board should have accounted for these declines in its evaluation of the occurrence of the PFAS at issue here.

II. PFAS Should Not Be Classified As Hazardous Substances Under the Model Toxics Control Act

The Draft CAP repeatedly prejudges that PFAS will in the future be declared "hazardous substances" under the Model Toxics Control Act (MTCA). This is premature, because the Draft CAP contains no legal or technical analysis in this regard. No individual PFAS meet the statutory criteria for a hazardous substance. Furthermore, it would be improper to evaluate and classify PFAS as a group under the MTCA – and even if the State were to do this, PFAS would not meet the statutory criteria.

The relevant statutory definition of "hazardous substance" is technical and complex, yet the Draft CAP does not specify how any PFAS would fall under it. The definition is:

> Any dangerous or extremely hazardous waste as defined in RCW 70A.300.010 (1) and (7), or any dangerous or extremely dangerous waste designated by rule pursuant to chapter 70A.300 RCW; (b) Any hazardous substance as defined in RCW 70A.300.010(10) or any hazardous substance as defined by rule pursuant to

> chapter 70A.300 RCW; (c) Any substance that, on March 1, 1989, is a hazardous substance under section 101(14) of the federal cleanup law, 42 U.S.C. Sec. 9601(14); (d) Petroleum or petroleum products; and (e) Any substance or category of substances, including solid waste decomposition products, determined by the director by rule to present a threat to human health or the environment if released into the environment. The term hazardous substance does not include any of the following when contained in an underground storage tank from which there is not a release: Crude oil or any fraction thereof or petroleum, if the tank is in compliance with all applicable federal, state, and local law.

RCW 70A.305.020. In turn, the relevant definitions of "dangerous waste" and "extremely hazardous waste" are similarly complex. "Dangerous waste" is defined as:

> any discarded, useless, unwanted, or abandoned substances, including but not limited to certain pesticides, or any residues or containers of such substances which are disposed of in such quantity or concentration as to pose a substantial present or potential hazard to human health, wildlife, or the environment because such wastes or constituents or combinations of such wastes: (a) Have short-lived, toxic properties that may cause death, injury, or illness or have mutagenic, teratogenic, or carcinogenic properties; or (b) Are corrosive, explosive, flammable, or may generate pressure through decomposition or other means.

RCW 70A.300.010. "Extremely hazardous waste" is defined as:

> any dangerous waste which: (a) Will persist in a hazardous form for several years or more at a disposal site and which in its persistent form (i) Presents a significant environmental hazard and may be concentrated by living organisms through a food chain or may affect the genetic makeup of human beings or wildlife, and (ii) Is highly toxic to human beings or wildlife (b) If disposed of at a disposal site in such quantities as would present an extreme hazard to human beings or the environment.

Id. The term "hazardous substance" is defined for purposes of RCW 70A.300.010(10) as:

> any liquid, solid, gas, or sludge, including any material, substance, product, commodity, or waste, regardless of quantity, that exhibits any of the characteristics or criteria of hazardous waste as described in rules adopted under this chapter.

The Draft CAP does not specify which part of the MTCA "hazardous substance" definition any PFAS may fall under, nor how the State would go about that evaluation. For the reasons described in the previous sections and in 3M's prior comments to the State, however, 3M believes based on available peer-reviewed literature and government publications that no PFAS fall under the definition. If the State does choose to make such an evaluation, it should be performed at the individual substance level, not for PFAS as a group. As the Draft CAP acknowledges, different PFAS have widely varying physical and chemical characteristics, so it

would be improper to treat them as a group. With this variability in physical and physiological characteristics, it is important to ensure that there is adequate scientific support for each action proposed in the Draft CAP for each specific chemical. Additionally, any future consideration of PFAS must be intentional and should entail evaluation of the specific traits of each PFAS involved.

The final CAP should not contain statements assuming that the State will make a hazard determination for any PFAS under the MTCA. *See, e.g.*, Draft CAP at 11, 46, 343 ("Once PFAS water contaminants are officially classified as hazardous substances…they can be addressed under the state [MTCA] framework."); 12, 344 (noting that some actions may occur "when PFAS are declared hazardous substances under MTCA.").

III. PFAS monitoring requirements for wastewater treatment plant influent and effluent and landfill leachate are premature

There currently exists no EPA-approved validated methods that are broadly reliable for a wide range of PFAS for a number of media. This includes wastewater treatment plant (WWTP) influent and effluent, as well as landfill leachate. In the Draft CAP, the State acknowledges that only approved validated methods recommended by EPA should be used for targeted PFAS analysis. Draft CAP at 115. 3M agrees.

The Draft CAP confirms that there exists no EPA-approved validated methods that are broadly reliable for a wide range of PFAS in media other than drinking water. SW-846 Method 8327 is designed to be used for reagent water, surface water, groundwater, and wastewater effluent. However, as the Draft CAP acknowledges, for many of the target analytes, there are "known difficulties with reproducibility, response, recovery, stability, and or chromatography that may reduce the overall quality or confidence in the result when using this method." Draft CAP at 121. This is the case for nearly half of the target PFAS analytes the test is designed for (11 out of 24). *Id*. at 121-122. A test method with such serious "quality or confidence" reliability concerns for nearly half of the target analytes should not be broadly mandated for use by the state. The State should wait until a more reliable test method is validated and approved for non-drinking water samples before promulgating testing requirements. Statements on pages 24-25 and 66-67 of the Draft CAP, as well as similar statements throughout the document, should be amended to reflect this.

In addition, the Draft CAP acknowledges that "EPA-approved methods for monitoring compliance with [wastewater] effluent limits for PFAS have not yet been developed and adopted by EPA." Draft CAP at 165. The Draft CAP also admits that any monitoring requirements "should include consideration of whether EPA has developed approved analytical methods for PFAS suitable for WWTP effluent." *Id*. at 183. The Draft CAP identifies no EPA-approved and validated methods for soil matrices or solid matrices. No such testing should be mandated from these media either until reliable and widely-available test methods are developed.

IV. The State should properly consider costs when contemplating regulation of PFAS

Any future regulation of PFAS the State may contemplate must properly take into account the costs, broadly defined, of any such action. The Draft CAP discusses the potential for granular activated carbon (GAC) to remove PFAS for water systems. Draft CAP at 215. However, any requirement to treat wastewater with granular activated carbon (GAC) or other methods could have much greater monetary and environmental costs than the state anticipates.

For example, unlike drinking water, wastewater contains high levels of contaminants likely to quickly "blind" activated carbon. This would require the carbon to be replaced or regenerated often. Replacement of carbon would create a large amount of solid waste, and regeneration would consume a significant amount of energy. Additionally, replacing or regenerating activated carbon would likely require the use of diesel trucks, and regenerating is an energy-intensive process. The State's discussion of GAC should be supplemented to reflect the limitations of using GAC on water streams other than drinking water.

V. The State should not presume which product categories will be included in the second Safer Products for Washington Cycle

The Draft CAP lists several product categories it may consider in the second Safer Products for Washington cycle. Draft CAP at 21. It would be premature for the State to presume now that any of these categories should be included in the second cycle – or that the second cycle should address PFAS at all. The law is structured so that Ecology learns about chemicals in products first, and then makes regulatory determinations.

Reasons not to include the use of PFAS in products in the second Safer Products for Washington cycle include voluntary phase-outs and declining industry uses of PFAS. The State must also properly account for the fact that the body of scientific evidence does not show adverse health effects in humans from perfluoroalkyls. Finally, the State must satisfy its statutory obligation to consider the availability and feasibility of safer alternatives. The State failed to do any of these things in the first cycle in connection with carpets and rugs, leather and textile furnishings, and aftermarket stain and water resistance treatments. See 3M Comments on Priority Consumer Products Draft Report to the Legislature (March 2, 2020).

3M appreciates the opportunity to comment and would be pleased to provide additional information or copies of previous comments upon request.

Regards,

Oyebode A. Taiwo, MD, MPH

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