Submitted electronically via Ecology form:
https://hwtr.ecology.commentinput.com/?id=HWQc5

January 28, 2022

Washington State Department of Ecology
Hazardous Waste and Toxics Reduction Program
PO Box 47600
Olympia, WA 98504

**Re: Safer Products for Washington Draft Report to the Legislature, Pub. No. 21-04-047**

To Whom It May Concern:

The 3M Company (3M) appreciates the opportunity to comment on the Washington State Department of Ecology's ("Ecology") Draft Regulatory Determinations Report to the Legislature on the Safer Products for Washington Phase Three Implementation ("Draft Report"), released November 2021. The Draft Report proposes regulatory determinations to restrict various substances in certain products, including limiting the use of per-and polyfluoroalkyl substances ("PFAS") in leather and textile furniture and furnishings, carpets and rugs, and aftermarket stain and water resistance treatments (i.e., indoor and outdoor textiles, outdoor apparel and gear).

3M has several concerns with the Draft Report's determinations related to PFAS. First, the Draft Report improperly makes broad determinations for PFAS as a single class, without accounting for the fact that PFAS includes thousands of substances with widely varying characteristics. Second, the Draft Report does not appear to rely on the best available science when determining the potential health effects of PFAS exposure.

## I. PFAS SHOULD NOT BE REGULATED AS A CLASS

The Draft Report defines the term "PFAS" to include "fluorinated organic chemicals containing at least one fully fluorinated carbon atom."[1] Draft Report at 25. This definition encompasses thousands of substances with distinct and widely varying properties, profiles, and uses. Generalizing all PFAS together is overbroad and not scientifically sound.[2] Different PFAS have different toxicological properties, bioaccumulation potentials, toxicity levels and effects. As the United States Environmental Protection Agency ("EPA") has noted, "PFAS vary widely in chemical and physical properties, behavior, and potential risks to human health and the environment. Differences in the chemical structure, carbon chain length, degree of fluorination, and chemical functional group(s) of individual PFAS have implications for their mobility, fate,

[1] Various regulators have proposed multiple different definitions of "PFAS," reflecting the difficulty in defining PFAS as a group.
[2] https://www.epa.gov/sites/default/files/2018-08/documents/r4_combined_presentations_.pdf, accessed November 20, 2021 (EPA noting "thinking of them [PFAS] as 'single' chemical or classes of chemicals can be problematic.")

and degradation in the environment, as well as uptake, metabolism, clearance, and toxicity in humans, plants, and other animals."[3] For example, the high molecular weight backbone polymers, such as PTFE, FEP, ETFE and PFA, have been widely cited as being of low concern (Henry et al.,[4] and ITRC[5]). The Draft Report does not account for these findings or the underlying chemical differences between PFAS molecules driving them.

A determination as to whether regulatory action is necessary under RCW 70A.350.040 requires considering toxicological endpoints, which depends on both toxicokinetic and toxicodynamic properties, and those vary widely among different PFAS. This approach is clearly understood by the authors of the Draft Report, as Table Number 19 describes several PFAS for which there are data and identifies widely differing toxicological endpoints that the authors assert are associated with those substances. Critically, however, as the Draft Report acknowledges, data is limited for many PFAS substances. *See* Draft Report at 74. Ecology nevertheless "assumes that data poor PFAS are potentially hazardous" and then "appl[ies] the hazards of the data rich chemicals in the class to determine whether the class fails to meet our minimum criteria (sic)." *Id.* The body of scientific literature does not support bridging or extrapolating data in this way. Ecology should not classify inert solids, liquids, salts and gases into a single class given the stark differences in intrinsic properties such as hazard, vapor pressure, and environmental partitioning. A chemical's persistence alone is not enough to assess present or future risks to human health and the environment. Further scientific assessment should be conducted in order to characterize the risk before adopting risk management measures.

3M supports a rigorous, science-based dialogue and review among regulators, academic researchers, manufacturers, and others to assess and research these substances. Consistent with sound environmental policy, such assessments must be based on the best available science, not unsupported assumptions and conjecture. Regulating PFAS broadly as a class based on assumptions creates an untenable situation for both the agency and the regulated community as it untethers Ecology's determination from its statutory authority. Ecology should reevaluate its determinations related to PFAS and instead make determinations for specific substances based on the scientific criteria outlined by the legislature.

## II. ECOLOGY'S ANALYSIS OF PFAS HAZARDS DOES NOT REFLECT BEST-AVAIALBLE SCIENCE

In the Draft Report, Ecology provides a brief analysis describing the Agency's view of the health hazards purportedly associated with certain PFAS compounds. The vast body of scientific evidence, however, does not show that PFAS cause adverse health effects in humans at current exposure levels, or even at the historically higher levels found in blood prior to the U.S. phase out of PFOS and PFOA.

[3] EPA Multi-Industry PFAS Study – 2021 Preliminary Report (September 16, 2021).
[4] B. J. Henry, et al, 'A critical review of the application of polymer of low concern and regulatory criteria to fluoropolymers'. Integr Environ Assess Manag, 14, 2018, p. 316-334
[5] Interstate Technology Regulatory Council, 'PFAS – Per- and Polyflouroalkyl Substances: Chemistry, Terminology, and Acronyms', viewed on 2 June 2020 at: https://pfas-1.itrcweb.org/2-2-chemistry-terminology-and-acronyms/

Notably, two authoritative bodies have recently reviewed the body of available research and concluded that there is not strong evidence of health effects in humans. The Agency for Toxic Substances and Disease Registry ("ATSDR") recently 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*."[6]

Likewise, the Australian Expert Health Panel concluded in March 2018, "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*."[7] 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 in reaching this conclusion.[8]

3M has analyzed numerous studies regarding the potential health effects of PFAS. Specifically, 3M has assessed Ecology's concerns related to purported carcinogenicity (including testicular cancer, kidney cancer, and liver cancer) and reproductive toxicity. A summary of 3M's review of the scientific evidence on some of these purported health issues is attached as **Appendix A**. Ecology should carefully review these studies and account for the various findings in revised regulatory determinations and any subsequent regulations imposing product restrictions.

3M respectfully requests that Ecology consider these comments, and the studies in Appendix A, in all subsequent rulemakings related to PFAS.

[6] ATSDR 2021 at p. 751, https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf (emphasis added).
[7] Expert Health Panel for Per- and Poly-Fluoroalkyl Substances PFAS: Summary at 2 (emphasis added).
[8] Expert Health Panel for (PFAS), March 2018 at 382-403.

## Appendix A

## CARCINOGENICITY

### Testicular Cancer

- Specifically regarding testicular cancer, the published studies that have reported more than 5 testicular cancer cases (or deaths) with exposure to PFOA are the community worker cohort study by Barry et al. (2013)[14] (n = 17 cases) and an ecological/case-control study of the same regional area by Vieira et al. (2013)[9] (n = 18 cases). However, these two studies likely had considerable overlap in their reported cases. The amount of this overlap remains unknown today despite the former three C8 Science Panel members' recent updating of the evidence on PFOA published since their original research (Steenland et al. 2020).[10] Furthermore, neither Barry et al. nor Vieira et al. reported the histology of the testicular cancers, of which approximately 95% are of germ-cell origin in humans, as opposed to the pathophysiologically distinct Leydig cell tumors reported in some rat carcinogenicity studies of PFOA. The Leydig cell adenomas reported in two of the three Sprague Dawley rat bioassays have a mode of action (MOA) that likely involves one of two possible pathways.[11] One pathway involves activation of PPARalpha, increased CYP19a1 (aromatase) or increased interstitial estradiol, TGFalpha, and subsequent Leydig cell proliferation. The other pathway involves decreased testosterone production with compensatory increases in luteinizing hormone (LH), which could lead to increased Leydig cell proliferation. LH activity is known to be >10x greater in the rat than the human. Neither of these pathways were considered likely relevant for humans.[12] The published epidemiology of testicular cancer data (Barry et al 2013[13]; Vierra et al. 2013[15]) relative to PFOA exposure can only offer suggestive evidence, at best, as unresolved overlapping of the relatively few testicular cancer cases in these two studies, as well as the fact that Leydig cell tumors diagnoses are quite rare in humans.

### Kidney Cancer

- The collective epidemiologic and toxicologic evidence for kidney cancer in relation to PFOS and PFOA exposure remains, at best, suggestive. Shearer et al. (2020)[14] conducted a matched case-control study (n = 324 cases) that showed an imprecise odds ratio (OR) of 2.6 (95% CI 1.33 – 5.20) in kidney cancer risk at the highest exposure level which became non-statistically significant when adjusted with other PFAS compounds (OR = 2.19, 95% CI 0.86 - 5.61). All of the study subjects (cases and controls) were from the general population and therefore, like the Danish study by Eriksen et al., this study lacked the exposure response contrasts seen in the occupational cohort

[9] Vieira et al. 2013 Environ Health Perspect 121 318-323.
[10] *Id*. at page 2.
[11] Klaunig et al. 2012 Reprod Toxicol 33 410-418.
[12] Corton et al. 2014 Crit Rev Toxicol 44 1-49.
[13] *Id*. at page 3.
[14] Shearer et al. 2020 J Natl Cancer Inst, see https://doi.org/10.1093/jnci/djaa143.

and community studies. In fact, all 324 subjects would likely have been included in the reference groups in these other studies. It should be noted that an excess of kidney tumors was not observed in the three Sprague Dawley lifetime bioassays. Renal papilla hyperplasia, however, was observed in the NTP female rats (not male rats). This was considered to be the consequence of the rapid elimination of high doses of PFOA in these female Sprague Dawley rats.

**Liver Cancer**

- The two largest occupational PFOA exposure cohort studies by Steenland and Woskie (2012)[15] and Raleigh et al. (2014)[16] reported no associations between PFOA and liver cancer mortality (combined total of 17 deaths). Between these two studies, there was only one liver cancer death reported in the highest quartile exposure categorizations. A third, much smaller occupational cohort study of the Miteni plant, located in the Veneto region of Italy, observed quite different findings based on a total of 7 liver cancer deaths (Girardi and Merler 2019).[17] They reported large, but extremely imprecise, unadjusted relative risks of liver cancer across tertiles of estimated high cumulative serum PFOA exposure. Among the mid-Ohio river community worker cohort study, Barry et al. reported 9 liver cancers cases (8 in community members), with no exposure response trend in estimated cumulative serum PFOA. The Danish case-cohort study by Eriksen et al. (2009)[18] studied 67 liver cancer cases whose serum PFOA concentrations were consistent with time-dependent general population levels observed in the United States. Eriksen et al. did not observe a trend with liver cancer across the limited range of exposures of the study subjects.

**REPRODUCTIVE TOXICITY**

- In a two-generation study in Sprague Dawley rats,[19] PFOS had no significant effects on estrous cycles. In gestational developmental studies with Sprague Dawley rats and CD-1 mice,[20] PFOS did not affect estrous cycles.
- Data from large scale 2-generation reproductive and developmental studies (which are considered the most comprehensive test by various agencies for evaluating potential endocrine disruption), show that PFOS does not affect reproductive functions or performances in either males or females across multiple generations.

[15] Steenland and Woskie 2012 Am J Epidemiol 176 909-917.
[16] Raleigh et al. 2014 Occup Environ Med 71 500-506.
[17] Giradi and Merler 2019 Environ Res 179:108743.
[18] Eriksen et al. 2009 JNCI 101 605-609.
[19] Deanna Luebker et al., *Neonatal Mortality from In Utero Exposure to Perfluorooctanesulfonate (PFOS) in Sprague–Dawley Rats: Dose-Response, and Biochemical and Pharamacokinetic Parameters,* 215 *Toxicology* 149 (2005), https://www.sciencedirect.com/science/article/abs/pii/S0300483X05003471.
[20] Christopher Lau et al., *Exposure to Perfluorooctane Sulfonate during Pregnancy in Rat and Mouse. II: Postnatal Evaluation,* 74 Toxicological Sciences, Issue 2 (August 2003)382-392.

- Ishibashi et al. (2007) reported that PFOS cannot directly activate human estrogen receptor α or β.[21]

[21] Ishibashi, H., Ishida, H., Matsuoka, M., Tominaga, N., and Arizono, K. (2007). Estrogenic effects of fluorotelomer alcohols for human estrogen receptor isoforms alpha and beta in vitro. *Biol Pharm Bull*. 30, 1358-9.