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May 3, 2021

Tyler Saechao
Office of Environmental Health Hazard Assessment
1001 I Street
P.O. Box 4010, MS-12B
Sacramento, California 95812-4010

Submitted electronically via https://oehha.ca.gov/comments

Re: Comments on Notice of Intent to List Perfluorooctanoic Acid by the Authoritative Bodies Mechanism Under the California Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65)

Dear Mr. Saechao:

The 3M Company ("3M") appreciates the opportunity to comment on the Notice of Intent to List Perfluorooctanoic Acid ("PFOA") by the Authoritative Bodies Mechanism, which was issued by the California Environmental Protection Agency's Office of Environmental Health Hazard Assessment ("OEHHA") on March 19, 2021 (the "Notice"). As a science-based company with substantial experience, expertise and product stewardship of these chemicals, 3M is well-positioned to provide input to OEHHA in this proceeding.

3M respectfully submits that PFOA cannot be listed as a chemical known to cause cancer under Proposition 65[1] pursuant to the grounds identified in the Notice. Specifically, the Notice seeks to list PFOA on the ground that an "authoritative body has formally identified the chemical as causing cancer or reproductive harm," pursuant to 27 Code of California Regulations Section 25306. But, as detailed below, OEHHA cannot list PFOA pursuant to that regulatory provision because the strict requirements under that regulation are not met. Among other things, the record does not establish that an authoritative body has formally identified PFOA as a known carcinogen, and the single authoritative body report that OEHHA relies on in making its determination does not satisfy the "sufficient evidence" requirement under the regulation. Additionally, the weight of the evidence as a whole, which must be considered in making this listing determination, is insufficient to support the affirmative conclusion that PFOA is a known (as opposed to probable) carcinogen. Accordingly, 3M respectfully urges OEHHA to conclude that PFOA should not be listed as a known carcinogen under the authoritative body listing mechanism.

In addition, prioritizing OEHHA's limited resources on PFOA may not be warranted based on the actual prevalence (or more precisely, the absence of prevalence) of PFOA in California today. The manufacture of PFOA has been voluntarily phased out across the United States.[2] Under

[1] "Proposition 65" refers to the Safe Drinking Water and Toxic Enforcement Act of 1986.
[2] *See, e.g.*, Fact Sheet: 2010/2015 PFOA Stewardship Program ("The manufacture and import of PFOA has also been phased out in United States as part of the PFOA Stewardship program.").

the U.S. Environmental Protection Agency's ("EPA") 2010/2015 PFOA Stewardship Program, eight major chemical manufacturers and processors – including 3M – committed to reducing PFOA from facility emissions and product content by 95 percent no later than 2010, and to working toward eliminating PFOA from emissions and product content no later than 2015.[3] According to EPA, all participating companies reported having met the PFOA Stewardship Program goals.[4]

For these and the other reasons detailed below, OEHHA should conclude that PFOA should not be listed on the grounds identified in the Notice. Thank you for your consideration.

Regards,

Oyebode A. Taiwo, MD, MPH

[3] *See id.*
[4] *See id.*

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I. The Relevant Standards for Listing PFOA as a Carcinogen Under Proposition 65.

As relevant here, there are two different mechanisms by which OEHHA could list PFOA as a carcinogen under Proposition 65: (1) the State's Qualified Experts ("SQE") mechanism; or (2) the authoritative bodies mechanism.

A. The SQE Mechanism.

The SQE listing process is a very thorough, detailed process. As part of the SQE procedure, OEHHA maintains a database of chemicals that have come to its attention for carcinogenicity evaluation. Chemicals entered into the tracking database are investigated for the existence of relevant toxicity data and the potential for human exposure. Those chemicals with data suggesting that they both cause cancer and have exposure potential in California become candidate chemicals. Candidate chemicals are then screened using a focused literature review. Chemicals selected by the screen undergo preliminary toxicological evaluation to determine whether they should be proposed for Carcinogen Identification Committee ("CIC") consideration for possible preparation of hazard identification materials. The list of chemicals proposed by OEHHA for CIC consideration for potential preparation of hazard identification materials is then released for public comment. During CIC meetings, OEHHA receives advice and consultation from the CIC regarding which chemicals should undergo the development of hazard identification materials, committee review, and eventual listing. OEHHA then selects the chemicals for development of hazard identification materials and solicits information on the evidence for carcinogenicity on chemicals selected for review. Next, hazard identification materials are prepared for CIC consideration and released to the public for comment. The CIC holds a public meeting to deliberate on whether the chemical has been clearly shown to cause cancer. At the end of deliberations, the CIC will generally render an opinion as to the carcinogenicity of a chemical, as appropriate, and a listing decision will be made.

B. The Authoritative Bodies Mechanism.

In contrast to the thorough SQE listing process, the authoritative bodies mechanism provides a shortcut for listing a chemical and allows OEHHA to rely on the analysis and determination of an outside party. The CIC has designated certain organizations, including the National Toxicology Program ("NTP"), as "authoritative bodies." A chemical is added to the Proposition 65 list if an authoritative body formally identifies it as causing cancer or reproductive harm. The criteria for determining if an authoritative body has formally identified a chemical as causing cancer are detailed in the applicable regulations at 27 Code of California Regulations ("CCR") Section 25306 and described in Section II below.

Because listing a chemical via the authoritative bodies mechanism is already a much less involved process than listing a chemical via the SQE mechanism, it is critically important that every criterion in Section 25306 is met. As explained below, several studies that were not

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considered by the NTP in the NTP Report (defined below) show that the required causal relationship between PFOA and cancer in humans does not exist. As such, PFOA should not be listed as a carcinogen under the authoritative bodies mechanism.

II. The NTP Report Does Not Satisfy the Criteria Established to List PFOA as a Known Carcinogen Under the Authoritative Bodies Mechanism at 27 CCR § 25306.

As is described above, the authoritative bodies mechanism, which OEHHA is relying on to support its listing determination in the subject Notice, contains several elements that must be satisfied before a chemical can be added to the Proposition 65 list. Of relevance here, the applicable regulation mandates the following:

1. OEHHA is required to determine which chemicals have been "formally identified" by an authoritative body "as causing cancer";

2. A chemical is "formally identified" by an authoritative body if OEHHA determines that the chemical: (1) has been included on a list of chemicals causing cancer issued by the authoritative body; (2) is the subject of a report that is published by the authoritative body and that concludes that the chemical causes cancer or reproductive toxicity; or (3) has otherwise been identified as causing cancer or reproductive toxicity by the authoritative body in a document that indicates that such identification is a final action;

3. "As causing cancer" means "sufficient evidence" of carcinogenicity exists from studies in human or experimental animals; and

4. OEHHA must find that a chemical does not satisfy the definition of "as causing cancer" if scientifically valid data that was not considered by the authoritative body clearly establishes that the chemical does not satisfy the carcinogen criteria, enumerated above.

27 CCR § 25306(c), (e)-(f).

OEHHA's Notice relies on an NTP Report, entitled *NTP Technical Report on the Toxicology and Carcinogenesis Studies of Perfluorooctanoic Acid (CASRN 335-67-1) Administered in Feed to Sprague Dawley (Hsd:Sprague Dawley® SD®) Rats* ("NTP Report"), which presents the results of animal testing, as a basis for listing PFOA as a known carcinogen under the authoritative body listing mechanism described above. The NTP Report and associated record as a whole do not satisfy the governing criteria for the reasons described below.

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A. PFOA Has Not Been "Formally Identified" by the NTP Within the Meaning of the Applicable Regulatory Standards.

As described above, a chemical is "formally identified" by an authoritative body under the governing regulation at 27 CCR § 25306(c) if OEHHA determines that the chemical: (1) has been included on a list of chemicals causing cancer issued by the authoritative body; (2) is the subject of a report that is published by the authoritative body and that concludes that the chemical causes cancer or reproductive toxicity; or (3) has otherwise been identified as causing cancer or reproductive toxicity by the authoritative body in a document that indicates that such identification is a final action**.**

First, PFOA has not been added to the NTP Report on Carcinogens, and NTP has not proposed to do so.[5] Second, though the NTP Report does note evidence of PFOA carcinogenic activity of PFOA in certain rats, the NTP Report ***does not*** specifically ***conclude*** that PFOA causes cancer, as suggested in the Notice. Instead, the NTP Report's "Conclusions" section finds that "there was clear evidence of carcinogenic activity of PFOA in male Hsd:Sprague Dawley® SD® rats" and "some evidence of carcinogenic activity of PFOA in female Hsd:Sprague Dawley® SD® rats."[6] The terms "clear evidence" and "some evidence" are defined in the Report as follows:

> Clear evidence of carcinogenic activity is demonstrated by studies that are interpreted as showing a dose-related (i) increase of malignant neoplasms, (ii) increase of a combination of malignant and benign neoplasms, or (iii) marked increase of benign neoplasms if there is an indication from this or other studies of the ability of such tumors to progress to malignancy.
>
> Some evidence of carcinogenic activity is demonstrated by studies that are interpreted as showing a chemical-related increased incidence of neoplasms (malignant, benign, or combined) in which the strength of the response is less than that required for clear evidence.[7]

Third, OEHHA's Notice does not identify any other document in which the NTP has taken final action to formally identify PFOA as causing cancer. As such, the NTP has not "formally identified" PFOA as a carcinogen within the meaning of the governing regulation.

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[5] *See* 14th Report on Carcinogens, https://ntp.niehs.nih.gov/whatwestudy/assessments/cancer/roc/index.html.
[6] *See NTP Technical Report on the Toxicology and Carcinogenesis Studies of Perfluorooctanoic Acid (CASRN 335-67-1) Administered in Feed to Sprague Dawley (Hsd:Sprague Dawley® SD®) Rats* at p. 92, https://ntp.niehs.nih.gov/ntp/htdocs/lt_rpts/tr598_508.pdf, last accessed May 2, 2021.
[7] *Id.* at xviii.

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B. The Weight of the Evidence in the Record Does Not Support the Determination That PFOA Is a Known Carcinogen.

California courts interpreting Proposition 65 have repeatedly stressed that "[o]nly those chemicals that are ***known***, and not merely suspected, of causing cancer or reproductive toxicity must be [placed] on the [Proposition 65] list." *W.n Crop Prot. Ass'n v. Davis*, 80 Cal. App. 4th 741, 749 (2000) quoting *AFL-CIO v. Deukmejian*, 212 Cal. App. 3d 425, 436-37 (1989) (emphasis added) (alterations in *W. Crop Prot. Ass'n*). In this regard, the record as a whole, including all scientifically valid data, must be considered in determining whether to add a chemical to the Proposition 65 list as a known carcinogen. *See, e.g.*, 27 CCR § 25306(f). Here, there is significant evidence that was not before nor considered by the NTP, thereby precluding reliance on the NTP Report in these streamlined proceedings. *Id.*

For these reasons, 3M presents evidence illustrating why PFOA should not be determined by OEHHA to be a known carcinogen, based on the record before it.

Steenland and Winquist (2020)[8] recently published a scoping review of the epidemiologic evidence of per- and polyfluoroalkyl substances ("PFAS") (which includes PFOA) and cancer. They identified 16 cohort or case-cohort studies, 10 case-control studies (four nested within cohorts), one cross-sectional, and one ecologic study. Each study was critiqued for its strengths and weaknesses. The authors concluded the evidence for an association between PFAS and cancer remains "sparse."[9] They concluded that the cancer sites with the "most evidence" for an association with PFAS (i.e., PFOA) were testicular and kidney cancer.[10] There were some "suggestions" in a few studies for an association with prostate cancer, but the data were inconsistent.[11] These authors did not describe the epidemiologic evidence with PFOA as "likely" to cause cancer. Presented below are 3M's brief descriptions of some of the epidemiological evidence regarding liver, pancreatic, testicular, kidney, and prostate cancer:

- **Liver cancer.** The two largest occupational PFOA exposure cohort studies by Steenland and Woskie (2012)[12] and Raleigh et al. (2014)[13] 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 seven liver cancer

[8] Kyle Steenland and Andrea Winquist, *PFAS and Cancer, a Scoping Review of the Epidemiologic Evidence*, 194 *Env't Resch.*110690 (2021), https://doi.org/10.1016/j.envres.2020.110690.
[9] *Id.* at 1.
[10] *Id.*
[11] *Id.*
[12] Kyle Steenland and Susan Woskie, *Cohort Mortality Study of Workers Exposed to Perfluorooctanoic Acid*, 176 *Am. J. Epidemiology* 909 (2012), https://pubmed.ncbi.nlm.nih.gov/23079607.
[13] Katherine Raleigh et al., *Mortality and Cancer Incidence in Ammonium Perfluorooctanoate Production Workers*, 71 *Occupational & Env't Med.* 500 (2014), https://oem.bmj.com/content/oemed/71/7/500.full.pdf.

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deaths (Girardi and Merler 2019).[14] They reported large, but extremely imprecise, unadjusted relative risks of liver cancer across tertiles of estimated high cumulative serum PFOA exposure.[15] Among the mid-Ohio river community worker cohort study, Barry et al. reported nine liver cancers cases (eight in community members), with no exposure response trend in estimated cumulative serum PFOA. The Danish case-cohort study by Eriksen et al. (2009)[16] studied 67 liver cancer cases of which 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.

- **Pancreatic cancer.** The epidemiological evidence does not indicate pancreatic cancer is associated with exposure to PFOA. Steenland and Woskie (2012)[17] and Raleigh et al. (2014)[18] each reported 18 pancreatic cancer deaths in their occupational cohorts within manufacturing operations. Neither study reported significantly increased risk for pancreatic cancer mortality. Girardi and Merler (2019)[19] did not report any pancreatic cancer deaths within their paper. In the Barry et al. (2013)[20] study, there was no significant trend with their estimated cumulative serum PFOA exposure metric that involved 21 pancreatic cancer cases in the community and six pancreatic cancer cases in the workers. In the Danish case-cohort study, Eriksen et al. (2009)[21] did not find a significant trend for the 128 pancreatic cancer cases reported in this cohort. The mode of action ("MOA") of the pancreatic acinar cell tumors in the rats exposed to PFOA is likely through increased cholecystokinin ("CCK") as a consequence of cholestasis. While CCK promotes acinar cell hyperplasia in the rats, this MOA is not considered to be relevant to human risk because of the difference in MOA.[22] The causal mechanism in the development of the human pancreatic (ductule) adenocarcinomas is neurogenically dependent and not via the CCK pathway.

- **Testicular cancer.** The only published studies that have reported more than five testicular cancer cases (or deaths) with exposure to PFOA are the community worker cohort study by Barry et al. (2013) (n = 17 cases) and an ecological/case-control study of the same

[14] Paolo Girardi and Enzo Merler, *A Mortality Study on Male Subjects Exposed to Polyfluoroalkyl Acids with High Internal Dose of Perfluorooctanoic Acid*, 179 Env't Resch. 108743 (2019), https://www.sciencedirect.com/science/article/abs/pii/S0013935119305407.
[15] *Id.*
[16] Kirsten Eriksen et al., *Perfluorooctanoate and Perfluorooctanesulfonate Plasma Levels and Risk of Cancer in the General Danish Population*, 101 *J. of Nat'l Cancer Inst.* 605 (2009), https://academic.oup.com/jnci/article/101/8/605/899923.

[17] Steenland and Woskie, *supra* note 11.
[18] Raleigh et al., *supra* note 12.
[19] Girardi and Merler, *supra* note 13.
[20] Barry et al. 2013 Environ Health Perspect 121 1313-1318.
[21] Eriksen et al., *supra* note 16.
[22] Myer et al. 2014 Toxicol Pathol 42 260-274.

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regional area by Vieira et al. (2013)[23] (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 and Winquist 2021).[24] 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 MOA that likely involves one of two possible pathways.[25] 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 more than 10 times greater in the rat than in humans. Neither of these pathways were considered likely relevant for humans.[26] The published epidemiology testicular cancer data (Barry et al. 2013;[27] Vieira et al. 2013[28]) regarding PFOA 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 the Leydig cell tumors diagnoses are quite rare in humans.

- **Kidney cancer.** Barry et al.[29] and Vieira et al.[30] also had considerable overlap in their kidney cancer cases (105 and 94 kidney cancer cases, respectively). The extent of this overlapping, like testicular cancer, has yet to be clarified (Steenland and Winquist 2021).[31] As part of their study, Barry et al. stratified their analyses further by whether the case lived in the community without DuPont work experience or had worked at the local Dupont fluoropolymer manufacturing plant (87 and 18 cases, respectively).[32] Lagging exposure by 10 years, the trend in hazard ratio for the estimated log cumulative serum PFOA concentrations were p = 0.17 and p = 0.94, respectively.[33] Analyzing all 105 cases, the trend in the hazard ratio was p = 0.15.[34] Thus, the paper by Barry et al. provides little evidence of an association when lagged exposure is accounted for in the analyses. The

[23] Veronica Vieira et al., *Perfluorooctanoic Acid Exposure and Cancer Outcomes in a Contaminated Community: a Geographic Analysis*, 121 *Env't Health Persp.* 318 (2013), https://pubmed.ncbi.nlm.nih.gov/23308854/.
[24] Steenland and Winquist, *supra* note 7, at 2.
[25] Klaunig et al. 2012 Reprod Toxicol 33 410-418.
[26] J Christopher Corton et al., *Mode of Action Framework Analysis for Receptor-Mediated Toxicity: The Peroxisome Proliferator-Activated Receptor Alpha (PPARα) as a Case Study*,
44 *Critical Revs. in Toxicology* 1 (2014), https://pubmed.ncbi.nlm.nih.gov/24180432/.
[27] Barry et al., *supra* note 15, at 3.
[28] Vieira et al., *supra* note 24.
[29] Barry et al., *supra* note 15.
[30] Vieira et al., *supra* note 24.
[31] Steenland and Winquist, *supra* note 7, at 2.
[32] Barry et al., *supra* note 15.
[33] *Id.*
[34] *Id.*

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cohort mortality study of the DuPont workforce by Steenland and Woskie (2012)[35] reported an association between PFOA and kidney cancer (mortality) based on 12 deaths, of which eight were in the highest quartile of estimated cumulative serum PFOA. However, these investigators did not adjust for tetrafluoroethylene ("TFE") exposure in this workplace for which PFOA is used as a processing aid in its polymerization to make PTFE. TFE is a known renal carcinogen in rats. In their analysis of six PTFE production plants, including the DuPont plant, Consonni et al. (2013)[36] could not disentangle the kidney cancer association between exposure to TFE and PFOA. In a cohort that manufactured PFOA in the near absence of exposure to TFE, Raleigh et al. (2014)[37] did not find an association between PFOA and kidney cancer incidence, or one between PFOA and kidney cancer mortality. All of these aforementioned studies are smaller than that reported by Shearer et al. (2020),[38] who 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 their study subjects (cases and controls) were from the general population and therefore, like the Danish study by Eriksen et al., this study (Shearer et al. 2020) lacked the exposure response contrasts seen in the occupational cohort 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. The collective epidemiologic and toxicologic evidence for kidney cancer remains, at best, suggestive.

- **Prostate cancer.** The two largest cohort studies, with sizable PFOA exposure differences in these two populations, investigated prostate cancer incidence (Barry et al. 2013 and Raleigh et al. 2014). Barry et al. and Raleigh et al. had a combined 887 prostate cancer cases and did not provide any evidence of an association with PFOA exposure. The one large case-cohort study of the general Danish population (n = 713 prostate cancer cases) provided minimum exposure contrast. Ducatman et al. (2015)[39] concluded there was no evidence of an association between elevated PSA tests and PFOA concentrations measured among 25,412 C8 Health Project male participants. The studies that have reported associations had far fewer study subjects including Steenland et al. (2015),[40] which showed

[35] Steenland and Woskie, *supra* note 11, at 3.
[36] Dario Consonni et al., *Cancer Risk Among Tetrafluoroethylene Synthesis and Polymerization Workers* 178 Am. J. of Epidemiology 350 (2013), https://pubmed.ncbi.nlm.nih.gov/23828249/.
[37] Raleigh et al., *supra* note 12, at 3.
[38] Joseph Shearer et al., *Serum Concentrations of Per- and Polyfluoroalkyl Substances and Risk of Renal Cell Carcinoma*, 143 *J. of Nat'l Cancer Inst.* (2020), https://doi.org/10.1093/jnci/djaa143.
[39] Ducatman et al. 2015 J Occup Environ Med 57 111-114.
[40] Kyle Steenland et al., *A Cohort Incidence Study of Workers Exposed to Perfluorooctanoic Acid (PFOA)* 72 *Occupational & Env't Med.* 373 (2015), .

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a non-monotonic, non-significant trend among 108 cases, and Hardell et al. (2014),[41] who published a case-control study (n = 201 cases) and inferred a modest association of prostate cancer and PFOA among first-degree relatives. Because prostate cancer is a highly survivable cancer, the mortality analyses for prostate cancer conducted by several studies (Steenland and Woskie et al. (2012),[42] Leonard et al. (2008),[43] Lundin et al. (2009),[44] and Gilliland and Mandel (1993)[45] have limited relevance. None of the two-year bioassays in Sprague Dawley rats reported prostate neoplasia. Neither Butenhoff et al. (2012)[46] nor the NTP Report reported evidence of prostate neoplasia in male Sprague Dawley rats when these tissues were examined histologically during or at the end of the two-year study periods.

The above summary of studies that 3M has reviewed pertaining to potential carcinogenicity associated with exposure to PFOA does not support OEHHA listing PFOA as a known carcinogen. As such, the weight of the evidence before OEHHA does not support the determination that PFOA is a known carcinogen.

III. OEHHA's Devotion Of Public Resources On A Potential PFOA Listing Is Unwarranted Due To the Absence of Exposure Potential in California.

OEHHA should not list PFOA as a carcinogen because there is no evidence of Proposition-65 regulable discharges or exposures in California. PFOA has been effectively phased out of the U.S. as is demonstrated by declining, residual PFOA serum levels in California and nationwide.

A. The Absence of PFOA Production in the United States.

In May 2000, 3M announced that it was voluntarily phasing out the production of PFOA. This goal was largely reached by 2002, and fully achieved by 2008.

In 2006, the EPA invited eight major fluoropolymer and telomer manufacturers, including 3M/Dyneon, to join a PFOA global stewardship program with two goals: 1) to commit to achieve, no later than 2010, a 95 percent reduction, measured from a year 2000 baseline, in both facility emissions to all media of PFOA, precursor chemicals that can break down to PFOA, and related

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[41] Elin Hardell et al., *Case-Control Study on Perfluorinated Alkyl Acids (PFAAs) and the Risk of Prostate Cancer*, 63 *Env't Int'l* 35 (2014), https://pubmed.ncbi.nlm.nih.gov/24246240/.
[42] Steenland and Woskie, *supra* note 11, at 3.
[43] Robin Leonard et al., *Retrospective Cohort Mortality Study of Workers in a Polymer Production Plant Including a Reference Population of Regional Workers*, 18 *Annals of Epidemiology* 15 (2008), https://pubmed.ncbi.nlm.nih.gov/17900928/.
[44] Jessica Lundin et al., *Ammonium Perfluorooctanoate Production and Occupational Mortality*, 20 *Epidemiology* 921 (2009), https://pubmed.ncbi.nlm.nih.gov/19797969/.
[45] Gilliland and Mandel 1993 JOM 35 950-954
[46] John Butenhoff et al., *Chronic Dietary Toxicity and Carcinogenicity Study with Ammonium Perfluorooctanoate in Sprague–Dawley Rats*, 298 *Toxicology* 1 (2012), https://www.sciencedirect.com/science/article/abs/pii/S0300483X12001151.

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higher homologue chemicals, and product levels of these chemicals; and 2) to commit to working toward elimination of these chemicals from emissions and product by 2015. To achieve these goals, these companies either stopped the manufacture and import of long-chain PFAS and transitioned to alternative chemicals or exited the PFAS industry altogether.[47] The EPA reports that the manufacture and import of PFOA has been phased out in the United States due to the PFOA stewardship program.[48]

In addition, the EPA has issued regulations, known as Significant New Use Rules ("SNURs"), that prevent other manufacturers (as well as 3M) by law from manufacturing or importing PFOA or PFOA precursors, subject to a handful of very narrow critical use exceptions with limited exposure potential approved by EPA.[49] 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 the Agency. These actions effectively phased out these chemicals in the United States.[50] In other words, California citizens' exposure to PFOA from Proposition 65-regulatable discharges and exposures is essentially non-existent. This fact is confirmed by the *declining* residual levels of PFOA in nationwide and California citizens' blood levels.

B. Residual Levels of PFOA in Blood in the United States General Population Have Declined and Continue to Consistently Decline Since Production Ceased. Similar Very Low Concentrations Have Been Reported in California.

According to the National Health and Nutrition Examination Survey (United States Centers for Disease Control (CDC) National Center for Environmental Health) ("NHANES"), which is a nationally representative sample of the U.S. population (non-institutionalized), the concentration of PFOA in the serum (blood) of the general population has declined significantly since the phase-out of production activities as reported for the geometric mean and the 95th percentile (see table below). This decline in PFOA is observed across males and females, age, and race/ethnicity. These data can be found in the Fourth National Report on Human Exposure to Environmental Chemicals, Volumes 1 and 2, pages 449-450 (See https://www.cdc.gov/exposurereport/pdf/FourthReport_UpdatedTables_Volume1_Mar2021-508.pdf) and the data for PFOA is excerpted the below table. NHANES has also provided further data from 2011-2018 in advance of the next scheduled National Report update. This data is also provided in the below table. (See https://www.cdc.gov/exposurereport/pfas_early_release.html).

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[47] *See* https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/fact-sheet-20102015-pfoa-stewardship-program.
[48] *Id.*
[49] 40 C.F.R. pt. 721, subpt. E.
[50] *Significant New Use Rules: Long-Chain Perfluoroalkyl Carboxylate and Perfluoroalkyl Sulfonate Chemical Substances*, 80 Fed. Reg. 2885 (Jan. 21, 2015) (to be codified at 40 C.F.R. pt. 721) ("The Agency previously determined that the 271 PFAS chemical substances identified in 40 CFR 721.9582(a)(1) were no longer being manufactured for any use in the United States, other than for the uses listed under 40 CFR 721.9582(a)(3), (a)(4), and (a)(5).").

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Serum Perfluorooctanoic acid (PFOA) (1999 – 2010)

CAS Number 335-67-1

Geometric mean and selected percentiles of serum concentrations (in µg/L) for the U.S. population from the National Health and Nutrition Examination Survey.

Demographic Categories	Survey (years)	Geometric mean (95% CI)	50th Percentile (95% CI)	75th Percentile (95% CI)	90th Percentile (95% CI)	95th Percentile (95% CI)	Sample Size
Total population	99-00	5.21 (4.72-5.74)	5.20 (4.80-5.90)	6.90 (6.30-7.80)	9.40 (8.20-11.1)	11.9 (10.9-13.5)	1562
Total population	03-04	3.95 (3.65-4.27)	4.10 (3.80-4.40)	5.80 (5.30-6.40)	7.80 (6.70-9.60)	9.80 (7.40-14.1)	2094
Total population	05-06	3.92 (3.48-4.42)	4.20 (3.80-4.50)	6.20 (5.40-7.20)	9.00 (7.40-11.2)	11.3 (8.80-14.5)	2120
Total population	07-08	4.12 (4.01-4.24)	4.30 (4.20-4.50)	5.90 (5.70-6.20)	7.90 (7.50-8.30)	9.60 (8.90-10.1)	2100
Total population	09-10	3.07 (2.81-3.36)	3.20 (2.90-3.50)	4.60 (4.10-5.10)	6.00 (5.30-7.20)	7.50 (6.20-9.70)	2233

Serum Perfluorooctanoic acid (PFOA) (2011 – 2018)

Sum of linear and branched PFOA isomers

Geometric mean and selected percentiles of serum concentrations (in µg/L) for the U.S. population from the National Health and Nutrition Examination Survey.

Demographic Categories	Survey (years)	Geometric mean (95% CI)	50th Percentile (95% CI)	75th Percentile (95% CI)	90th Percentile (95% CI)	95th Percentile (95% CI)	Sample size
Total population	11-12	2.08 (1.95-2.22)	2.08 (1.96-2.26)	3.03 (2.76-3.27)	4.35 (3.82-4.85)	5.68 (5.02-6.49)	1904
Total population	13-14	1.94 (1.76-2.14)	2.07 (1.87-2.20)	3.07 (2.67-3.37)	4.27 (3.57-5.17)	5.57 (4.60-6.27)	2165
Total population	15-16	1.56 (1.47-1.66)	1.57 (1.47-1.77)	2.47 (2.27-2.57)	3.37 (3.07-3.57)	4.17 (3.87-4.67)	1993
Total population	17-18	1.42 (1.33-1.52)	1.47 (1.37-1.57)	2.07 (1.97-2.30)	2.97 (2.77-3.37)	3.77 (3.17-5.07)	1929

As a result of the EPA PFOA product stewardship program, it is anticipated that PFOA serum concentrations will continue to decline. Due to this anticipated continued decline, and because there is no showing of any present or foreseeable water discharge of PFOA, further review of PFOA is not necessary to accomplish the goals of Proposition 65.

Thank you, again, for providing 3M with the opportunity to comment on the Notice. For each of the reasons described above, 3M respectfully disagrees with OEHHA's determination to list PFOA as a known carcinogen under Proposition 65. The record does not establish that an authoritative body has formally identified PFOA as a known carcinogen, the NTP Report that OEHHA relies on in making its determination does not satisfy the applicable standard, and the weight of the evidence as a whole is insufficient to support the conclusion that PFOA is a known (as opposed to merely probable) carcinogen. 3M respectfully urges OEHHA to conclude that PFOA should not be listed as a known carcinogen.