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Public comments on the California Office of Environmental Health Hazard Assessment's proposed public health goals for perfluorooctanoic acid and perfluorooctane sulfonic acid in drinking water

Submitted via electronic docket at https://oehha.ca.gov/comments

We are writing from Silent Spring Institute to submit comments to the California Office of Environmental Health Hazard Assessment (OEHHA) in support of the proposed public health goals (PHGs) for perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS) in drinking water. We appreciate this opportunity to provide input.

Silent Spring Institute is a non-profit research organization that studies environmental chemicals and disease, with a focus on breast cancer. Chemicals in the PFAS family are of concern for many health endpoints, including breast cancer. Silent Spring previously published peer-reviewed studies on PFAS in drinking water,[1,2] food packaging,[3] and blood.[4] Silent Spring currently has four federally funded research studies on PFAS, including 1) Massachusetts PFAS and Your Health Study, part of the larger CDC/ATSDR Multi-Site Study on health effects of PFAS exposures from drinking water, 2) PFAS-REACH, which is assessing the relationship between PFAS and pediatric immunotoxicity, 3) STEEP, led by the University of Rhode Island, which is investigating environmental transport, exposure, and health effects of PFAS, and 4) a new study funded by the National Science Foundation to investigate policy responses to PFAS at multiple levels of governance. We are writing to share scientific information on PFAS exposures and health effects from our own research and other published scientific studies to inform the proposed PHGs under consideration.

We support OEHHA's rigorous analysis of cutting-edge science and the use of toxicological data to determine the public health goals (PHGs) for lifetime exposure to PFOA or PFOS. We recommend the approval of the more protective PHGs based off of cancer risks: 0.007 parts per

[1] Schaider LA, Rudel RA, Ackerman JM, Dunagan SC & Brody JG. 2014. Pharmaceuticals, perfluorosurfactants, and other organic wastewater compounds in public drinking water wells in a shallow sand and gravel aquifer. Science of the Total Environment, 468, 384-393.
[2] Hu XC, Andrews DQ, Lindstrom AB, Bruton TA, Schaider LA, Grandjean P, Lohmann R, Carignan CC, Blum A, Balan SA, Higgins CP & Sunderland EM. 2016. Detection of poly-and perfluoroalkyl substances (PFASs) in US drinking water linked to industrial sites, military fire training areas, and wastewater treatment plants. Environmental science & technology letters, 3(10), 344-350.
[3] Schaider LA, Balan SA, Blum A, Andrews DQ, Strynar MJ, Dickinson ME, Lunderburg DM, Lang JR & Peaslee GF. 2017. Fluorinated compounds in US fast food packaging. Environmental science & technology letters, 4(3), 105-111.
[4] Boronow KE, Brody JG, Schaider LA, Peaslee GF, Havas L & Cohn BA. (2019). Serum concentrations of PFASs and exposure-related behaviors in African American and non-Hispanic white women. Journal of exposure science & environmental epidemiology, 29(2), 206-217.

trillion (ppt) for PFOA based on kidney cancer in humans and 1 ppt for PFOS based on liver and pancreatic tumors in animal studies. We also recommend that OEHHA extends the PHGs beyond PFOA and PFOS and adopt a class-based approach to regulation that assumes other PFAS are similarly toxic unless you have data to the contrary.

Our following comments go more into detail on these points:

1. **We support OEHHA's rigorous analysis of cutting-edge science and the use of toxicological data to determine the public health goals (PHGs) for lifetime exposure to PFOA and PFOS**.

We found the overall scientific approach to calculating the PHGs for PFOA and PFOS to be thorough and well-reasoned. The PHGs of 0.007 ppt for PFOA and 1 ppt for PFOS are appropriate based on the weight of evidence and calculations for protection against cancer, and they should be protective against non-cancer toxic effects as well.

We appreciated the discussion of PFOA and PFOS effects on hormone levels and on the mammary gland. However, we noted that the discussion of reproductive and developmental toxicity failed to include several epidemiological and animal studies demonstrating impaired ability to breastfeed in mothers and delayed breast development in girls. For example, several well-powered epidemiological studies have documented associations between serum PFAS (including PFOA and PFOS) and reduced duration of breastfeeding.[5,6,7] While early termination of breastfeeding can be related to several sociocultural factors, the most common reasons are concerns about insufficient milk supply, milk quality, and pain,[8,9,10,11] which are all related to hormonal imbalances. The lowest serum concentrations observed to have a significant odds ratio (OR) for reduced breastfeeding time was 1 ng/mL PFOA and 10 ng/mL PFOS.[8] These are

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[5] Fei C, McLaughlin JK, Lipworth L, Olsen J. 2010. Maternal concentrations of perfluorooctanesulfonate (pfos) and perfluorooctanoate (pfoa) and duration of breastfeeding. Scandinavian Journal of Work, Environment & Health: 413-421.
[6] Timmermann CAG, Budtz-Jørgensen E, Petersen MS, Weihe P, Steuerwald U, Nielsen F, et al. 2017. Shorter duration of breastfeeding at elevated exposures to perfluoroalkyl substances. Reproductive toxicology (Elmsford, NY) 68:164-170.
[7] Timmermann CAG, Andersen MS, Budtz-Jorgensen E, Boye H, Nielsen F, Jensen RC, et al. 2021. Pregnancy exposure to perfluoroalkyl substances, prolactin concentrations and breastfeeding in the odense child cohort. J Clin Endocrinol Metab.
[8] Ahluwalia IB, Morrow B, Hsia J. 2005. Why do women stop breastfeeding? Findings from the pregnancy risk assessment and monitoring system. Pediatrics 116:1408-1412.
[9] Gianni ML, Bettinelli ME, Manfra P, Sorrentino G, Bezze E, Plevani L, et al. 2019. Breastfeeding difficulties and risk for early breastfeeding cessation. Nutrients 11:2266.
[10] Lechosa-Muniz C, Paz-Zulueta M, Cayon-De Las Cuevas J, Llorca J, Cabero-Perez MJ. 2021. Declared reasons for cessation of breastfeeding during the first year of life: An analysis based on a cohort study in northern spain. Int J Environ Res Public Health 18.
[11] Stuebe AM, Horton BJ, Chetwynd E, Watkins S, Grewen K, Meltzer-Brody S. 2014. Prevalence and risk factors for early, undesired weaning attributed to lactation dysfunction. J Womens Health (Larchmt) 23:404-412.

substantially lower than the NOAEC level of 9.8 ng/mL PFOA for elevated ALT and LOAEC of 16.4 ng/mL PFOS for elevated cholesterol cited in the OEHHA draft PHG document. The potential for PFAS chemicals to impair breastfeeding is supported by studies in rodents showing that PFOA exposure during pregnancy impairs lactation through reduced epithelial differentiation and altered milk protein production,[12,13] resulting in dose-dependent increases in pup morbidity but not maternal toxicity.[12,14]

Multiple studies in rodents have also demonstrated that prenatal exposure to PFOA delays mammary gland development.[12,13,15,16] A dose of 0.01 mg PFOA/kd/day in CD-1 mouse dams for under 40% of pregnancy (gestation day 10-17) was capable of inducing significant delays in ductal elongation and branching and terminal end bud formation in pups, with effects that persisted into adulthood.[20] The PFOA-induced delays in mammary gland development and lactation-related defects were evident across three generations of mice,[13] underscoring the importance of addressing this effect in public health guidance documents to prevent additive or synergistic effects of continued PFAS exposure across generations of humans.

As additional epidemiological evidence of how PFAS may impact pubertal development in girls, perinatal exposure to PFOA has been associated with delays in menstruation (> 4.4 – 19.8 ng/mL PFOA in serum)[17] and breast development;[18,19] while peripubertal serum levels of PFOA (> 11.4 ng/mL) and PFOS (> 23 ng/mL) were both associated with delayed menstruation.[20] Such epidemiological evidence illustrates that more women-relevant endpoints should be considered

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[12] White SS, Calafat AM, Kuklenyik Z, Villanueva L, Zehr RD, Helfant L, et al. 2007. Gestational pfoa exposure of mice is associated with altered mammary gland development in dams and female offspring. Toxicol Sci 96:133-144.

[13] White SS, Stanko JP, Kato K, Calafat AM, Hines EP, Fenton SE. 2011. Gestational and chronic low-dose pfoa exposures and mammary gland growth and differentiation in three generations of cd-1 mice. Environmental Health Perspectives 119:1070-1076.

[14] Lau C, Thibodeaux JR, Hanson RG, Narotsky MG, Rogers JM, Lindstrom AB, et al. 2006. Effects of perfluorooctanoic acid exposure during pregnancy in the mouse. Toxicol Sci 90:510-518.

[15] Macon MB, Villanueva LR, Tatum-Gibbs K, Zehr RD, Strynar MJ, Stanko JP, et al. 2011. Prenatal perfluorooctanoic acid exposure in cd-1 mice: Low-dose developmental effects and internal dosimetry. Toxicol Sci 122:134-145.

[16] Tucker DK, Macon MB, Strynar MJ, Dagnino S, Andersen E, Fenton SE. 2015. The mammary gland is a sensitive pubertal target in cd-1 and c57bl/6 mice following perinatal perfluorooctanoic acid (pfoa) exposure. Reprod Toxicol 54:26-36.

[17] Kristensen SL, Ramlau-Hansen CH, Ernst E, Olsen SF, Bonde JP, Vested A, et al. 2013. Long-term effects of prenatal exposure to perfluoroalkyl substances on female reproduction. Hum Reprod 28:3337-3348.

[18] Kale A, Deardorff J, Lahiff M, Laurent C, Greenspan LC, Hiatt RA, et al. 2015. Breastfeeding versus formula-feeding and girls' pubertal development. Matern Child Health J 19:519-527.

[19] Pinney SM, Biro FM, Windham GC, Herrick RL, Yaghjyan L, Calafat AM, et al. 2014. Serum biomarkers of polyfluoroalkyl compound exposure in young girls in greater cincinnati and the san francisco bay area, USA. Environ Pollut 184:327-334.

[20] Lopez-Espinosa MJ, Fletcher T, Armstrong B, Genser B, Dhatariya K, Mondal D, et al. 2011. Association of perfluorooctanoic acid (pfoa) and perfluorooctane sulfonate (pfos) with age of puberty among children living near a chemical plant. Environ Sci Technol 45:8160-8166.

in the formation of the non-cancer PHGs for PFOS and PFOA, and if they were the PHGs would likely be lower than the proposed 2ppt for PFOS and 3ppt for PFOA.

In summary, we found that adverse non-cancer effects on the mammary gland are evident for impaired lactation at 1 ng/mL PFOA and 10 ng/mL PFOS, which are lower than the NOAEC of 9.8 ng/mL PFOA for elevated ALT and LOAEC of 16.4 ng/mL PFOS for elevated cholesterol used by OEHHA. Inclusion of effects on lactation/breastfeeding would have yielded more protective non-cancer-based guidelines. However, the final PHG of 0.007 ppt for PFOA and 1 ppt for PFOS (selected based on cancer endpoints) should be protective against adverse effects on the mammary gland as well. We commend OEHHA for their careful and health protective approach.

As we discuss below, since the public is exposed to many additional PFAS chemicals that have similar toxic effects, using a class-based approach that assumes all PFAS have similar toxicity as PFOA and PFOS seems appropriate. This class-based approach is more likely to be protective against both cancer and non-cancer toxic effects for the vast mixture of PFAS in the environment, including many that have yet to be rigorously evaluated in epidemiological and toxicological studies.

2. **We urge OEHHA to develop a class-based PHG for all PFAS and recommend to the State Water Resources Control Board that they set class-based MCLs for PFAS to provide more holistic public health protection.**

The EPA has identified over 9,000 compounds classified as PFAS,[21] and many have been associated with industrial uses and consumer products.[22] This class of chemicals is associated with a wide range of adverse health outcomes, including cancer, immunotoxicity, reproductive toxicity, developmental effects on the mammary gland, neurotoxicity, and thyroid, liver, and kidney effects.[23] However, given the size of this chemical family, conducting human health risk assessments for each individual PFAS compound is not possible. Despite the high diversity of the class, PFAS are all alike in that they contain perfluoroalkyl moieties that are extremely resistant to environmental and metabolic degradation, and this high persistence means that their continual release will result in accumulating environmental concentrations and increasing

[21] U.S. Environmental Protection Agency. 2020. *PFAS Master List of PFAS Substances (Version 2).* Available from: https://comptox.epa.gov/dashboard/chemical_lists/pfasmaster.

[22] Glüge J, Scheringer M, Cousins IT, DeWitt JC, Goldenman G, Herzke D, Lohmann R, Ng CA, Trier X & Wang Z. 2020. An overview of the uses of per- and polyfluoroalkyl substances (PFAS). Environmental Science-Processes & Impacts, 22(12): p. 2345-2373.

[23] Agency for Toxic Substances & Disease Registry (ATSDR). 2019. Toxicological Profile for Perfluoroalkyls. https://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=1117&tid=237.

probabilities of the occurrence of irreversible harms.[24] Due to the toxic, mobile, persistent, ubiquitous and diverse nature of these chemicals, it is well recognized that effective regulation of PFAS will need to use a class-based approach that assumes similar toxicity across the class, unless there is empirical information to the contrary.

The American Public Health Association[25] and a number of expert scientists including Dr. Linda Birnbaum, former head of the National Institute for Environmental Health Sciences, have recommended approaching PFAS as a class based on their shared chemical properties and have urged reducing overall use of PFAS.[26,27,28,29,30] Past examples (such as flame retardants[31] and CFCs) have shown that a one-chemical-at-a-time approach has not been effective at protecting public health and the global environment. The OECD already recognizes PFAS as a class,[32] and in 2019 the European Union recommended an action plan to eliminate all non-essential uses of PFAS as a class,[33] indicating regulatory agencies are already moving in this direction.

In fact, California's own Department of Toxic Substances Control is already regulating PFAS as a chemical class, citing this approach as "logical" and "necessary" given that all PFAS, and their

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[24] Cousins IT, DeWitt J, Glüge J, Goldenman G, Herzke D, Lohmann R, Miller M, Ng CA, Scheringer M, Vierke L & Wang Z. 2020. Strategies for grouping per- and polyfluoroalkyl substances (PFAS) to protect human and environmental health. Environ. Sci.: Processes Impacts, 22:1444-1460. doi: 10.1039/D0EM00147C.
[25] American Public Health Association. 2016. *Reducing Human Exposure to Highly Fluorinated Chemicals to Protect Public Health*.
[26] Birnbaum L, Southerland B, & Sussman R. 2021, July 30. EPA must protect public health by regulating PFAS as a class. The Hill. https://thehill.com/opinion/energy-environment/565528-epa-must-protect-public-health-byregulating-pfas-as-a-class.
[27] Birnbaum L. 2019. Testimony for hearing on "Examining the Federal Response to the risks associated with per- and polyfluoroalkyl substances (PFAS)." Testimony before the Senate Committee on Environmental and Public Works, March 28, 2019. Department of Health and Human Services, National Institutes of Health, National Institute of Environmental Health Sciences: Washington, DC.
[28] Birnbaum LS and Grandjean P. 2015. Alternatives to PFASs: Perspectives on the science. Environmental Health Perspectives, 123(5): p. A104-A105.
[29] Kwiatkowski CF, Andrews DQ, Birnbaum LS, Bruton BA, DeWitt JC, Knappe DRU, Maffini MV, Miller MF, Pelch KE, Reade A, Soehl A, Trier X, Venier M, Wagner CC, Wang Z & Blum A. 2020. Scientific basis for managing PFAS as a chemical class. Environmental Science & Technology Letters, 7(8): p. 532-543.
[30] Blum A, Balan SA, Scheringer M, Trier X, Goldenman G, Cousins IT, Diamond M, Fletcher T, Higgins C, Lindeman AE, Peaslee G, de Voogt P, Wang Z, Weber R. 2015. The Madrid statement on poly- and perfluoroalkyl substances (PFASs). Environmental Health Perspectives, 123(5): p. A107-A111.
[31] National Academies of Sciences, Engineering, and Medicine. *A Class Approach to Hazard Assessment of Organohalogen Flame Retardants.* Washington, DC: The National Academies Press, 2019. doi:doi:10.17226/25412.
[32] OECD. 2021. *Reconciling Terminology of the Universe of Per- and Polyfluoroalkyl Substances: Recommendations and Practical Guidance*. OECD Series on Risk Management, No. 61. OECD Publishing: Paris, France.
[33] Council of the European Union. 2019. *Towards a Sustainable Chemicals Policy Strategy of the Union*. Brussels, Belgium.

degradation, reaction, or metabolism products, display common hazardous traits.[34] Silent Spring's own research on PFAS exposures to firefighters and office workers in California's Bay Area[35] found that 70% of study participants had detectable levels of 7 out of the tested 12 target PFAS analytes, and 100% have detectable levels of 4 PFAS—pointing to the fact that simply regulating 2 individual PFAS, PFOS and PFOA, is not enough.

In fact, other regulatory agencies in California have already recognized that PFOA and PFOS are not the only chemicals of concern in California's drinking water. While California's State Water Resources Control Board set notification levels for PFOA and PFOS in drinking water in 2018, they recently added notification levels for PFBS in January 2021,[36] and are looking into developing notification levels for PFHxS, PFHxA, PFHpA, PFNA, PFDA, and ADONA.[37]

Regarding drinking water particularly, recent research has shown that less than half the total organic fluorine measured in treated drinking water were accounted for by the sum of individually identified PFAS.[38] This indicates that there are likely many more PFAS in the water than would be identified by a targeted analysis, and points to the need to monitor, report, and regulate PFAS as a class. Therefore, it is critical that OEHHA sets a public health goal (PHG) for PFAS as a class.

As a initial step towards regulating PFAS as a class, the state could measure total PFAS in various environmental and biological media on a regular basis to assess the abundance of non-regulated PFAS. Meanwhile, other regulatory bodies are proposing innovative solutions to the challenges of regulating PFAS as a group in drinking water. Recently, the EU proposed two drinking water guidelines based on differing groupings of PFAS compounds: 100 ng/L for the sum of 20 PFAS compounds (perfluorocarboxylic acids and perfluorosulfonic acids), and 500 ng/L for the sum of all total PFAS. These values will apply once technical guidelines for monitoring these parameters are developed in accordance with Article 13(7). Member States may then decide to use either one or both of the parameters. This proposal by the EU is an example of

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[34] Bălan SA, Mathrani VC, Guo DF & Algazi AM. 2021. Regulating PFAS as a chemical class under the California Safer Consumer Products Program. Environmental Health Perspectives, 129(2), 025001.

[35] Trowbridge J, Gerona RR, Lin T, Rudel RA, Bessonneau V, Buren H, and Morello-Frosch R. (2020). Exposure to Perfluoroalkyl Substances in a Cohort of Women Firefighters and Office Workers in San Francisco. Environmental Science & Technology, 54 (6), 3363-3374. DOI: 10.1021/acs.est.9b05490.

[36] California State Water Resources Control Board. 2021. Drinking Water Notification Levels. Accessed October 28th. https://www.waterboards.ca.gov/drinking_water/certlic/drinkingwater/NotificationLevels.html.

[37] California State Water Resources Control Board. 2021. Perfluorooctanoic acid (PFOA) and Perfluorooctanesulfonic acid (PFOS). Accessed October 28th. https://www.waterboards.ca.gov/drinking_water/certlic/drinkingwater/PFOA_PFOS.html.

[38] Hu XC, Tokranov AK, Liddie J, Zhang X, Grandjean P, Hart JE, Laden F, Sun Q, Yeung LWY, Sunderland EM. (2019, Jun 6). Tap Water Contributions to Plasma Concentrations of Poly- and Perfluoroalkyl Substances (PFAS) in a Nationwide Prospective Cohort of U.S. Women. Environ Health Perspect., 127(6): 67006. doi: 10.1289/EHP4093.

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how regulating a large group of drinking water contaminants such as PFAS. We urge OEHHA to perform similar action towards creating PHGs that address PFAS in drinking water as a class.

In summary, the available science supports the final OEHHA PHG of 0.007 ppt for PFOA and 1 ppt for PFOS. Exposure to highly persistent PFAS have been associated with a range of health hazards and we commend OEHHA for taking this critical step towards protecting Californians. Setting a PHG for PFAS as a class would be a more protective public health measure and is in line with the action taken by other regulatory agencies and recommendations by prominent experts in the field. We hope OEHHA will continue to work towards this goal.

Thank you for the opportunity to provide comments.
Best regards,

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