28 October 2021

California Environmental Protection Agency
Office of Environmental Health Hazard Assessment
Submitted electronically to: https://oehha.ca.gov/comments

Re: Draft Technical Support Document for Proposed Public Health Goals for Perfluorooctanoic Acid and Perfluorooctane Sulfonic Acid in Drinking Water

Dear Sir or Madam:

Enclosed please find comments submitted on behalf of the National Association for Surface Finishing (NASF) regarding the California Office of Environmental Health Hazard Assessment (OEHHA) Draft Technical Support Document for the Proposed Public Health Goals (PHGs) for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonic Acid (PFOS) in Drinking Water.

We appreciate the opportunity to provide OEHHA with these comments. If you have any questions, would like additional information, or would like to discuss these comments, please contact me by telephone at 202-257-3756 or by email at jhannapel@thepolicygroup.com.

Respectfully submitted,

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The Policy Group
On Behalf of NASF

28 October 2021

Comments on the California Office of Environmental Health Hazard Assessment (OEHHA) Draft Technical Support Document for Proposed Public Health Goals for Perfluorooctanoic Acid and Perfluorooctane Sulfonic Acid in Drinking Water

The California Office of Environmental Health Hazard Assessment (OEHHA) has issued a draft technical support document for proposed public health goals (PHGs) for perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS) in drinking water The National Association for Surface Finishing (NASF) and its California Chapters have an established history of collaboration and cooperation with California's public health offices including the State Water Resources Control Board and OEHHA to promote environmental stewardship. We have worked, and will continue to work, to ensure that our industry is governed by environmentally sound regulations and policies that are data driven to ensure the continuation of our industry via environmentally sustainable business practices.

OEHHA has requested public comment on the proposed PHGs. Our technical consultant, GSI Environmental Inc., has put together the following comments on our behalf. We offer these comments in the continued spirit of collaboration and coordination with California's agencies, to support data-driven and sound science-based policies and regulations. Overall, we are concerned that the draft PHGs are not based on sound science or best risk assessment practices. **OEHHA's draft PHGs contain overly conservative misrepresentations of the**

PFOA/PFOS-related science, resulting in proposed PHGs that are not consistent with the available science and best standards of practice. Our detailed and specific comments are provided below.

Summary of the Surface Finishing Industry

The NASF represents the business, management, technical, and educational programs, as well as the regulatory and legislative advocacy interests, of the surface finishing industry to promote the advancement of the North American surface finishing industry globally. NASF has approximately 800 members, including surface finishing companies, surface finishing suppliers, large global industrial customers, and individual and professional members. The surface finishing industry plays a vital role in the nation's economic future. The industry's role in corrosion protection alone provides an estimated $200 billion annual economic benefit to the nation, including significant corrosion protection for military equipment that provides national defense.

Nearly all surface finishing companies are small businesses. Over 80 percent of the companies in the business employ fewer than 75 people, while nearly 40 percent employ fewer than 20 people. Most surface finishing firms are family-owned businesses, located in every state.

Summary of Comments

1. The PHGs should be based on noncancer endpoints from experimental animal data.

Epidemiological studies can provide supporting evidence regarding potential human health effects from chemical exposures. However, human data are often inadequate for direct quantitative use in risk assessment and/or establishing public health goals due to significant uncertainty and challenges associated with confounding, sample size, and exposure assessment. As such, human data are generally useful only as a qualitative line of evidence, when observations demonstrate consistency with animal data and biological plausibility for human relevance. In the case of PFOA and PFOS, the available epidemiology data do not corroborate the animal studies and are fraught with uncertainties and limitations. Therefore, PHGs should be based only on experimental animal data with observations that provide a clear dose-response and human relevant biological plausible endpoint.

2. **The weight of evidence for cancer effects for PFOS is that the compound is unlikely to present a carcinogenic risk, especially at low levels, therefore, the PHG should be based on PFOS noncancer health effects.**

    (a) **The available PFOS laboratory animal and epidemiology data evaluating cancer risk is largely negative, with no clear association between exposure and increased cancer risk noted, therefore, the animal study should not be used to support an OEHHA PHG.**

The animal data evaluating PFOS carcinogenicity are "suggestive", not definitive, based on the available data (only one chronic animal bioassay) and numerous authoritative agency reviews (e.g., USEPA, European Food Safety Authority, Health Canada). OEHHA's evaluation contradicts these other recent authoritative and comprehensive reviews. The rodent liver tumors from Butenhoff et al. (2012) are of questionable human relevance due to potential species-specific mode of action considerations (non-human relevant mechanisms involving xenobiotic nuclear receptors, such as PPAR-alpha) (Elcombe et al., 2012). Furthermore, the liver tumors noted with statistical significance were actually benign adenomas - no statistically significant increases in hepatocellular carcinomas were observed in either the male or female rats - with no clear dose response. These data are not strong enough to suggest that PFOS is carcinogenic to humans at low doses, and no other data provide PFOS carcinogenicity evidence in animal bioassays.

Epidemiology studies have not reported a consistent or clear increase in cancers for occupational workers, impacted communities, or general population cohorts exposed to PFOS. Of the numerous cancer types evaluated, only community studies of breast cancer have reported mixed (some studies with an association and other studies with no observed association) results. Many of the studies evaluating a possible association between PFOS and breast cancer have experimental design limitations including small cohort size, co-exposure to other chemicals, and challenges with exposure assessment. Thus far, the potential cancer risk associated with PFOS exposure has not been demonstrated.

(b) OEHHA's conclusion that PFOA and PFOS are genotoxic conflicts with conclusions by numerous state, federal, and international organizations. Genotoxicity is not a mode of action relevant for PFOA or PFOS and should not be used as a supporting line of evidence for PHGs based on cancer.

In the Risk Characterization section of the draft PFOA and PFOS PHGs, OEHHA states that "*genotoxicity cannot be dismissed as a possible mode of action for PFOA and PFOS"* (OEHHA, 2021). This conclusion mischaracterizes the overall weight-of-evidence of PFOA and PFOS genotoxicity. Multiple studies provide clear evidence that neither PFOA nor PFOS are mutagenic or cause direct genotoxicity as the mechanism for inducing cancer. OEHHA's interpretation of PFOA and PFOS genotoxicity data also conflicts with the available data and recent conclusions of numerous state, federal, and international health agencies despite no substantial new genotoxicity publications. Additionally, OEHHA's conclusion on genotoxicity of PFOA and PFOS is at odds with a recent Environmental Working Group publication on the key characteristics of carcinogens as they apply to PFAS. Temkin et al. (2020) stated that *"Given the lack of direct genotoxicity for PFAS chemicals, any carcinogenic hazard is likely due to mechanisms other than direct DNA damage*".

Specific discrepancies between OEHHA's conclusions regarding PFOA and PFOS genotoxicity and those of other environmental regulatory agencies and scientific organizations are detailed below.

<u>PFOA</u>

OEHHA's representation of the weight-of-evidence of PFOA genotoxicity contradicts recent conclusions by Minnesota, New Jersey, and the International Agency for Research on Cancer (IARC). In their recent derivations of health-based drinking water values, both Minnesota and New Jersey clearly state PFOA is unlikely to be genotoxic. New Jersey's 2019 Technical Support Documents for Interim Specific Groundwater Criterion for PFOA (NJDEP, 2019b) concluded:

> *"PFOA is not chemically reactive. Thus, it is not metabolized to reactive intermediates and does not covalently bind to nucleic acids and proteins. Consistent with these properties, **available data indicate that it is not genotoxic**."* (Emphasis added)

New Jersey's findings are consistent with Minnesota's interpretation of available PFOA genotoxicity data, which concluded in their August 2020 Toxicology Summary for PFOA (MNDOH, 2020b) that "*PFOA is not genotoxic*". Lastly, OEHHA's conclusion on PFOA genotoxicity contradicts the IARC, which stated "*it is widely accepted that PFOA is not directly genotoxic*" in the 2017 PFOA Monograph (IARC, 2017). IARC also determined the following:

> "*PFOA is not DNA-reactive, and gives negative results in an overwhelming number of assays for direct genotoxicity. Therefore, **there is strong evidence that direct genotoxicity is not a mechanism of PFOA carcinogenesis**."* (Emphasis added)

<u>PFOS</u>

In Section 6.2.2 of the PHG document, OEHHA states "*There is evidence to indicate that PFOS is genotoxic*". This statement is inconsistent with conclusions made by New Jersey, USEPA, and the Agency for Toxic Substances and Disease Registry (ATSDR) on PFOS genotoxicity. New Jersey's 2019 Technical Support Document for Interim Specific Groundwater Criterion for PFOS (NJDEP, 2019a) found:

> "*PFOS is not chemically reactive. Thus, it is not metabolized to reactive intermediates and does not covalently bind to nucleic acids and proteins. **Consistent with these properties, available data indicate that it is not genotoxic**."* (Emphasis added)

Consistent with New Jersey's findings, ATSDR concluded in their 2021 Toxicological Profile for PFAS (ATSDR, 2021) that "*Results do not provide evidence for genotoxicity of PFOS, except for one in vitro study showing cell transformation and one report of increased micronuclei formation following in vivo exposure*". USEPA has also reached similar conclusions on PFOS genotoxicity (USEPA, 2016). In the 2016 PFOS Health Advisory, USEPA summarized available evidence of PFOS genotoxicity by stating:

> "*All genotoxicity studies including an Ames test, mammalian-microsome reverse mutation assay, an in vitro assay for chromosomal aberrations, an unscheduled DNA synthesis assay, and mouse micronucleus assay were negative*"

OEHHA appears to rationalize the contradiction between their findings on the genotoxicity of PFOS and the conclusions of numerous state, federal, and international agencies by suggesting that OEHHA identified four additional genotoxicity studies that were not reviewed by other agencies. However, a recent European Food Safety Authority (EFSA) publication reviewed three of the additional genotoxicity publications noted by OEHHA, and still concluded, "*For PFOS and PFOA, no evidence for a direct genotoxic mode of action was identified*" (EFSA et al., 2020). The fourth study is a PFOS biochemical study with no direct relevance to evaluation of genotoxicity.

(c) OEHHA misapplied IARC's key characteristics of carcinogens; the key characteristics of carcinogens should not be used as supporting evidence for cancer-based PHGs.

Ten key characteristics of carcinogens (KCs) were described by the IARC Working Group as characteristics exhibited by known human carcinogens (Smith et al., 2016). However, it is important to note that the IARC working group did not evaluate incidences of KCs for chemicals that did not cause cancer, and did not quantitatively evaluate the association between the dose-response of a specific carcinogenic effect and the KCs (Doe et al., 2019). KCs are highly qualitative and are ineffective at utilizing available data for causal analysis (Becker et al., 2017).

In large datasets on specific KCs or proposed mechanisms of action such as genotoxicity, it is not uncommon to have some positive results despite an overwhelming majority of negative findings (Becker et al., 2017). While KCs may occur in the formation of tumors, they only serve as one line of evidence for carcinogenicity. Their occurrence alone should not inherently result in the classification of the chemical as a carcinogen (Doe et al., 2019). Additional considerations should include quantitative dose and temporal evaluations of the chemical- and tumor-specific sequences of molecular events leading to tumor development against a human relevance framework.

KCs were used by OEHHA in the draft PFOA and PFOS PHG document to identify and discuss plausible mechanisms of carcinogenesis, and at least in part, to justify linear extrapolation in the derivation of the cancer slope factors. OEHHA determined both PFOA and PFOS had relevant data for five of the ten KCs. However, OEHHA did not apply any quantitative scoring method to evaluate confidence in these characteristics and failed to take into account the well-established understanding of cancer etiology and progression along a dose- and time-response continuum.

3. OEHHA's use of epidemiological data to derive the PFOA cancer slope factor is highly uncertain.

Translating two epidemiological studies - Shearer et al. (2021) and Vieira et al., (2013) - into a PFOA cancer slope factor is a relatively complex, yet consequential step in the derivation of the PFOA PHG. While there is precedent for using epidemiological studies in the derivation of cancer slope factors (OEHHA, 2004; USEPA, 2011), OEHHA does not appropriately contextualize shortcomings in the use of Shearer et al., (2021) for the derivation of the PFOA cancer slope factor and subsequent draft PHG. Additionally, the statistical values selected by OEHHA from Shearer et al., (2021) for derivation of a cancer slope factor are inconsistent with best practices for human health risk assessment.

Shearer et al. (2021) categorized PFOA serum concentrations into quartiles, then utilized the lowest quartile (<4 ng/ml) as the reference group to derive PFOA serum concentrations and renal cell carcinoma (RCC) odds ratios (ORs). Importantly, Shearer et al., (2021) estimated exposure to PFOA and other PFAS from a single blood sample. As summarized by Steenland and Winquist (2021), while PFAS serum levels can serve as useful biomarkers of exposure, PFAS serum levels collected at a single timepoint may not accurately represent historical exposure. Therefore, the single blood sample values in Shearer et al. (2021) should be treated with a low degree of confidence when used to represent lifetime PFAS exposure.

Shearer et al. (2021) identified ORs for PFOA serum concentrations and RCC that were corrected for numerous confounders, including age, sex, study center, race, blood sample year, BMI, smoking habits, EGFR, freeze-thaw cycles, and hypertension history. Only the OR for the highest quartile was statistically significant for PFOA. However, once the authors adjusted the PFOA concentrations and RCC ORs for co-exposure to PFOS and PFHxS, no OR remained statistically significant, only the continuous variable for PFAS adjusted ORs was significant. Nevertheless, OEHHA elected to use the unadjusted ORs for the calculation of the PFOA cancer slope factor instead of the ORs that were adjusted for co-exposure to other PFAS. This is a scientifically unjustified approach.

The use of Shearer et al., (2021) to quantify PFOA's PHG is highly uncertain. OEHHA should consider the fact that Shearer et al., (2021) is inadequate for quantitative assessment of human health cancer risk based on an estimate of exposure from a single blood sample and the lack of quartile statistical significance once ORs were adjusted for co-exposure.

4. **The weight of evidence better supports use of noncancer data for both PFOA and PFOS PHGs, however, the draft noncancer PHGs should be revised.**

    a. **The immunotoxicity database shows inconsistent associations between PFOA and PFOS and immune endpoints, including largely negative associations with actual infections or symptoms, such that an additional uncertainty factor to cover immune databases is unnecessary.**

OEHHA applied an uncertainty factor (UF) for intraspecies variation of three to the point of departure (POD) for noncancer effects to calculate the acceptable daily dose (ADD) for both PFOA and PFOS. For PFOA, OEHHA indicated that the UF is justified, in part, by the "strong evidence for immunotoxicity of PFOA", and "the potential for immunotoxicity to occur below the [no-observed-adverse-effect concentration] for elevated ALT [alanine aminotransferase] levels". Given the large number of studies that have evaluated this endpoint, and yet the relatively weak and inconsistent associations noted by OEHHA and others, this additional justification for the UF is not warranted.

OEHHA identified dozens of studies that examined potential immunotoxicity of PFOA and PFOS, including over a dozen epidemiological studies, numerous animal bioassays, and many supporting in vitro analyses. The database continues to demonstrate weak and inconsistent results, for many of which the biological relevance is unclear. There is natural variation in antibody responses in the human population and epidemiological studies have not reported antibody levels that were below those that are considered protective against disease or that failed to provide a supportive level of immunity. Furthermore, epidemiology studies have reported inconsistent associations between both PFOA and PFOS and common infections or symptoms (see discussion in ATSDR (2021) and Steenland et al. (2020)) and the National Toxicology Program scored both compounds as "low confidence" for association with infection disease outcomes (NTP, 2016).

b. (b) The total cholesterol endpoint should not be selected as the critical effect, based on current data and understanding of potential modes of action.

In addition to immunotoxicity, OEHHA identified alterations in lipid metabolism as potential category of relevant noncancer health effects for PFOS. OEHHA stated lipid metabolism yields a slightly lower health-protective concentration (HPC) and specifically selected increased total cholesterol (TC) from an epidemiological study (Steenland et al., 2009) as the critical effect endpoint. This determination is a deviation from OEHHA's prior evaluation in 2019 in which OEHHA based a noncancer reference level of 7 ppt for PFOS on immunotoxicity in mice (OEHHA, 2021, p. 11, footnote 1). Importantly, despite the availability of Steenland et al. 2009, OEHHA did not cite or utilize the publication in their 2019 Notification Level Recommendations.

Notably, the decision to rely on increased serum TC to establish a quantitative relationship between PFOS exposure and adverse effect is an outlier when compared with the broad range of public health agencies in the U.S. (both federal and state) (see Table 1 below, based on (ITRC, 2020) and internationally (*e.g.*, Health Canada, Australia/New Zealand) that have reviewed the same human and animal study data on PFOS and selected an alternative endpoint as the basis for health advisories, guidance values, and drinking water maximum contaminant levels for PFOS.

The choice of increased TC as a biomarker of effects from PFOS exposure is questionable from both an empirical basis and a mechanistic basis (i.e., mode of action [MoA]). Numerous epidemiological studies and animal toxicity studies provide data to examine potential associations between serum PFOS levels and blood chemistry (serum and plasma) measures of liver function, such as levels of lipids and liver enzymes. Comprehensive evaluations of the literature, including the summary presented by OEHHA in the draft PFOA and PFOS PHG document, clearly illustrate the inconsistencies in the empirical data.

ATSDR (2018) provides illustrative graphical summaries of the literature on associations between serum PFOS and TC (Figures 2-13 and 2-14) and low-density lipoprotein (LDL) cholesterol (Figures 2-15 and 2-16), as well as a discussion of the data on high-density lipoprotein (HDL) cholesterol and triglycerides. While two large-scale epidemiological studies of participants in the C8 Science Panel studies indicate a positive and statistically significant relationship between risk of abnormal cholesterol levels and PFOS levels (presented as adjusted ratios) in adults (Steenland et al., 2009) and children and adolescents (Frisbee et al., 2010), inverse and/or non-statistically significant relationships have been observed in a variety of general population studies (Château-Degat et al., 2010; Eriksen et al., 2013; Fisher et al., 2013; Fu et al., 2014; Nelson et al., 2010).

Epidemiologists are aware of the potential for reverse causation associated with and confounding of the serum PFOS/serum lipid relationships, particularly among participants who report using cholesterol-lowering medication. The inconsistent relationship between serum PFOS and TC across studies persists even after controlling for this factor. Australia and New Zealand (FSANZ, 2017) noted that common study limitations of the cross-sectional studies include possible confounding effects (and no adjustments for): 1) co-exposure to PFOA or other PFAS; 2) diet; and 3) glomerular filtration rate (as an index of kidney function).

To date, a biologically plausible MoA has not yet been established to explain how increased exposure to PFOS and other PFAAs could cause an elevation in serum (or plasma) cholesterol levels in humans. The role of PPARα in lipid metabolism is well established and suggests that, for PPAR agonists like PFOA and PFOS, which inhibit the secretion of LDL and cholesterol from the liver, an inverse relationship with serum cholesterol is more likely (Corton et al., 2014). Prolonged activation of PPARα leads to increased lipid metabolism, thereby *reducing* cholesterol levels.

As noted by OEHHA (2021, p.101), in rodent models, including a transgenic mouse model that possesses human-like lipid metabolism, exposure to PFOA tends to *reduce* cholesterol levels (Pouwer et al., 2019). Health Canada (2018, Section 9.2.2.3 Serum Lipid Effects) provides a synthesis of the animal toxicity study data in monkeys, mice, and rats, the vast majority of which demonstrate an inverse relationship between serum PFOS and TC, LDL cholesterol, HDL cholesterol, and triglycerides. Similar findings were observed in NTP's 28-day toxicity studies of PFAA exposure in rats (Goodrum et al., 2020; NTP, 2019). This hypothesis is further supported by a recent phase I clinical trial with PFOA, which demonstrated that when human serum levels of PFOA are comparable to the relatively high levels achieved in rodent studies, cholesterol levels *decline* rather than increase (Convertino et al., 2018).

There is no clear pattern that explains the inconsistency in associations between serum PFAS and TC in both human and animal studies. Factors that may contribute to variability and uncertainty in the study outcomes for PFOS include the dose range, species, sex, and age group. In contrast to most regulatory agencies and independent science advisory panels that examined the same body of science on serum lipid effects, OEHHA appears to have adopted the perspective that a sufficient number of studies with PFOS demonstrate a *change* in lipid homeostasis, and that collectively these associations are indicative of an adverse effect:

- regardless of whether or not the effect manifests as an increase or decrease in TC;
- despite uncertainty in the clinical significance of the magnitude of the change;
- regardless of whether or not there is consistency within the same study in the associations with other biomarkers of lipid metabolism and liver function; and
- absent supporting animal toxicity data to demonstrate a clear and consistent dose-response relationship.

Table 1. Critical Noncancer Effect Endpoints for PFOS Selected by Regulatory Agencies.

Noncancer Critical Effect Endpoint for PFOS	Regulatory Agency	Key Studies
Developmental Effects	USEPA (2016) ATSDR (2018) Australia/FSANZ (2017) Alaska DEC (2016) Mass. DEP (2019) Michigan DHHS (2019) Vermont DEC/DOH (2018)	(Luebker et al., 2005)
Immunological Effects	Minnesota DOH (2020a) New Jersey DWQI (2018)	(Dong et al., 2011) (Dong et al., 2009)
Hepatic Effects, Serum Chemistry (↑ Total Cholesterol)	OEHHA (2021)	(Steenland et al., 2009)
Hepatic Effects, Morphology (Liver Hypertrophy accompanied by Cytoplasmic Vacuolation)	Health Canada (2018)	(Butenhoff et al., 2012)
Neurodevelopmental Effects	Texas CEQ (2016)	(Zeng et al., 2011)

c. **There are no biologically plausible genetic reasons why race or ethnicity would represent a more sensitive subgroup for PFOA or PFOS; an additional uncertainty factor to cover human variability is unnecessary.**

As noted by OEHHA, the Gallo et al. (2012) study involved a very large number of adults whom likely "included a diverse group of people in terms of ages, health status, smoking and other chemical exposures, nutrition, socioeconomic status and other factors" (OEHHA, 2021, p.182). Although the study did not include children, there is no evidence that children would be more susceptible to changes in alanine transaminase or other effect endpoints related to PFOA or PFOS exposure. The consideration of potential additional sensitivity related to race or ethnicity or age to justify an additional UF for intraspecies variation of three is unjustified.

5. **The use of the default Relative Source Contribution (RSC) for the noncancer PHGs is inconsistent with currently available data and best practices.**

OEHHA incorrectly defines the RSC term and fails to take into account U.S. and California-specific human data that informs the potential background exposure to PFOA and PFOS. The RSC is defined by OEHHA (OEHHA, 2021, p. 20) as: *"the proportion of exposures to a chemical attributed to tap water, as part of total exposure from all sources (including food and air)."* This is inaccurate. USEPA's guidance on the RSC states that the RSC is "the percentage of total exposure typically accounted for by drinking water… **applied to the RfD**…"(USEPA, 2000, p. 1-7; emphasis added). Therefore, the RSC term is used to account for the proportion of **allowable total daily exposure** (*i.e.*, the toxicity value represented by the ADD, also called the reference dose (RfD) by the USEPA, in mg/kg-day) that is attributed or allocated to drinking water in calculating acceptable levels of chemicals in water. The remainder of the total daily exposure is attributed to other exposure pathways. Therefore, in contrast to OEHHA's assertion, one does not need to be able to calculate individual exposures from all pathways, but rather, one needs to be able to understand how exposure to the general population compares to the ADD, such that the rest of the allowable exposure can be allocated to the drinking water pathway.

USEPA guidance suggests a range of 0.2 to 0.8 for the RSC term (USEPA, 2000). The low-end default value of 0.2 is applied in the absence of chemical-specific data on exposure. It assumes that 80 percent of the target dose can be attributed to exposures other than drinking water and attributes the remaining 20 percent to exposure to drinking water. When data on chemical-specific exposures are available (*i.e.*, the contribution of non-drinking water pathways to total dose) it should be used to develop an alternative RSC.

There have been several studies of dietary, dust, and inhalation exposure to PFOA and PFOS (reviewed in Sunderland et al., 2019), none of which suggest that exposures other than drinking water are likely to add up to 80 percent of the allowable daily intakes defined as OEHHA's RfDs. In fact, recent evaluations by several states have applied USEPA's "exposure decision tree method" to derive RSC values of 0.5 for both PFOA and PFOS (Dewitt et al., 2019; Garnick, 2021; MNDOH, 2020b; NHDES, 2019). Additionally, most recently, Garnick et al. (2021) estimated an "actual RSC" for PFOA and PFOS of 0.95 based on the 95th percentile background exposures for women based on a 2011 study (Lorber & Egeghy, 2011) and national serum concentration data from the National Health and Nutrition Examination Survey (NHANES).

Importantly, through the Biomonitoring California program, California-specific data are available. According to Minnesota, biomonitoring results such as the NHANES data set can be used to represent non-water or background exposures (MNDOH, 2020a). Drinking water exposure is likely the predominant source of PFOA and PFOS exposure and, moreover, chemical-specific data exists for OEHHA to use rather than the default value.

6. **OEHHA traditionally utilizes numerous extremely conservative science policy decisions in the derivation of PHGs. Compounding conservative assumptions produce drinking water limits that are a challenge for California drinking water providers to meet, and yet offer no increased public health protective measures.**

For deriving public health criteria such as a PHG, default, conversative exposure and toxicity assumptions are typically used. However, the conservative and uncertain toxicity and exposure decisions utilized by OEHHA within the PFOA and PFOS PHG are considerable.

- Applying a 95th percentile consumers-only drinking water intake of 0.053 L/kg-day (OEHHA, 2012). Selection of 95th percentile as the basis of drinking water intake rate is highly conservative. The drinking water intake rate of 0.053 L/kg-day is approximately 21% higher than the 90th percentile consumers-only drinking water intake of 0.043 L/kg-day identified from the same dataset. Additionally, 0.053 L/kg-day is 70% higher than the equivalent USEPA-recommended drinking water intake for an 80 kg adult.
- Use of linear extrapolation to define the cancer slope factor for both PFOA and PFOS.
- Use of the one in a million (10^{-6}) cancer risk level, the lower end of the "target cancer risk range". For comparison, the USEPA Office of Water lifetime Health Advisories for carcinogenic compounds are based on a 10^{-4} cancer risk level (USEPA, 2018).

The use of the most conservative default science policy decisions in combination with conservative interpretations of the science for PFOA and PFOS results in highly conservative yet imprecise draft PHGs for PFOA and PFOS.

Conclusion

NASF appreciates the opportunity to communicate our concerns to OEHHA on this important regulatory development within the state. OEHHA's draft document, in many ways, represents the most comprehensive and robust recent compilation of relevant information related to PFOA and PFOS potential human health risks. However, OEHHA's draft PHGs contain overly conservative misrepresentations of the PFOA/PFOS-related science, resulting in proposed PHGs that are not consistent with the available science and best standards of practice.

- The PHGs should be based on noncancer endpoints.
- The weight-of-evidence for cancer effects for PFOS demonstrates that the compound is unlikely to present a carcinogenic risk at low levels.
- OEHHA's conclusions that PFOA and PFOS are genotoxic conflicts with conclusions from numerous other organizations and the available data.
- OEHHA misapplies the IARC key characteristics of carcinogens and should not use them as supporting evidence for cancer-based PHGs.
- The human data used to derive the PFOA PHG is highly uncertain.
- The use of the default RSC for the noncancer PHGs is inconsistent with currently available data and best practices.
- The use of conservative default science policy decisions in combination with conservative interpretations of the science for PFOA and PFOS results in highly conservative yet imprecise draft PHGs.

We encourage OEHHA to use sound science based on best standards of practice, to derive PHGs that are adequately protective of public health and look forward to reviewing the revised PHG support document. If you have any questions or would like additional information regarding these comments, please contact Jeff Hannapel with NASF at jhannapel@thepolicygroup.com.

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