



November 21, 2016

VIA ELECTRONIC MAIL

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Drinking Water Quality Institute

RE: REQUEST FOR PUBLIC INPUT FOR PERFLUOROCTANOIC ACID

To Whom It May Concern:

On behalf of our members, the Chemistry Council of New Jersey (CCNJ) and Site Remediation Industry Network (SRIN) appreciates the opportunity to provide comments to the Drinking Water Quality Institute (DWQI) pursuant to the Institute's request for public input regarding the recently released subcommittee reports on Perfluorooctanoic Acid (PFOA). In the interest of brevity, we also would like to refer DWQI to CCNJ's earlier comments on PFOA submitted on July 28, 2014. The concerns raised in our earlier comments remain relevant to the subcommittee reports currently at issue. CCNJ/SRIN have long advocated for greater transparency and public input with respect to DWQI's activities and we appreciate the steps taken to provide this opportunity.

General Concerns

Based upon available science and data, as further detailed below, we have significant concerns that DWQI's current recommendations related to PFOA are needlessly low and could not be feasibly implemented by New Jersey water providers, and this cannot be supported by an objective analysis of the available science and data. As such, DWQI's current draft Maximum Contaminant Level (MCL) for PFOA should be held until such time that scientific evidence can support its recommendation. In the alternative, we urge DWQI to further review the detailed scientific data and literature that was either ignored or missed in its current review of PFOA before submitting a recommendation to the New Jersey Department of Environmental Protection (NJDEP). The following are specific examples, also further discussed in the comments that follow, and additional citations are noted in the reference list at the end of this letter:

- enHealth. June 2016. Statement: Interim national guidance on human health reference values for per- and poly-fluoroalkyl substances for use in site investigations in Australia. <http://www.health.nsw.gov.au/environment/factsheets/Documents/pfas-interim-health-values-ahppc.pdf>

- Government of Canada. June 2016. Perfluorooctanoic Acid (PFOA) in Drinking Water, Document for Public Consultation. <http://healthycanadians.gc.ca/health-system-systeme-sante/consultations/acide-perfluorooctanoic-acid/document-eng.php>
- Health Council of the Netherlands. 2013. Perfluorooctanoic acid and its salts - Evaluation of the carcinogenicity and genotoxicity. The Hague: Health Council of the Netherlands; publication no. 2013/32 ISBN 978-90-5549-982-3. http://www.gezondheidsraad.nl/sites/default/files/201332Perfluorooctanoic_acid_and_its_salts.pdf
- Chang ET, Adami HO, Boffetta P, Cole P, Starr TB, and Mandel JS. 2014. A critical review of perfluorooctanoate and perfluorooctanesulfonate exposure and cancer risk in humans. *Crit Rev Toxicol.* 44(S1):1-81

We believe that it is in the best interests of public policy and public health in New Jersey to review this science prior to any final PFOA MCL recommendation from DWQI. These resources will provide valuable insight to DWQI and allow for the review of the best information currently available.

Comments on each of the subcommittee reports are provided below and cover the following key points:

1. The federal United States Environmental Protection Agency (USEPA) and other agencies that have comprehensively reviewed the available scientific evidence recognize the uncertainty in the available data and do not share DWQI's perspective on potential health effects of PFOA in drinking water at the proposed MCL.
2. DWQI should explain what margin of exposure (MOE) is afforded by the total set of uncertainty factors it uses to derive the MCL.
3. DWQI compares predicted serum PFOA levels to background levels but fails to provide any context regarding the proposed Target Human Serum level.
4. Despite evidence to the contrary, DWQI classifies PFOA as a developmental toxicant, which not only adds an unwarranted uncertainty factor, but also contributes to the public's unsupported concern for increased risk for women of childbearing age and infants.
5. DWQI did not apply a true weight-of-evidence analysis of the animal toxicity study data, nor a discussion regarding the severity of effects across the range of endpoints.
6. DWQI applies a default relative source contribution (RSC) of 20 percent, without considering the data available to support a PFOA-specific value, and despite having adopted a non-default value for PFNA.

7. DWQI significantly overstated the link between PFOA exposure and carcinogenicity in humans and failed to rely on the most recent comprehensive assessments of the available science.
8. DWQI's report lacks sufficient evidence that the Practical Quantitation Level (PQL) of 6 parts per trillion (ppt) is generally achievable utilizing USEPA Method 537 and that the difference between 14 ppt (proposed MCL) and 20 ppt (federal Unregulated Contaminants Monitoring Rule (UCMR)3 Minimum Reporting Limit (MRL) for PFOA) is discernable given measurement errors at that low level of detection.
9. DWQI has not evaluated the feasibility of achieving the MCL, nor has it provided an assessment of the potential utility and efficacy of treatment technologies other than granular activated carbon (GAC).
10. DWQI does not adhere to Executive Order No. 2 (EO #2) established by Governor Christie.

Health Effects Subcommittee Report

USEPA has gathered a great deal of data, nationally, on PFOA and its potential health effects and, very recently, issued a federal protective guideline of 70 ppt for PFOA in drinking water. DWQI rejects the federal government's careful analysis and replaces it with its own approach which would lead to a far stricter guideline being imposed on the communities and businesses of New Jersey.

Unlike DWQI, USEPA and other jurisdictions recognize the lack of scientific evidence and uncertainties associated with the science related to any health effects associated with PFOA.

For example only, some jurisdictions include a scientific alternative that allows for an MOE or "margin of safety" approach, as detailed below (please see "Alternate Approach Proposed: Reevaluate the Margin of Exposure"), which melds the animal data with human biomonitoring data. DWQI should clearly explain why the MOE findings for PFOA set forth in the Health Canada (2012) document and, specifically, the "margin of safety" found by Canadian authorities are (or are not) appropriate scientific research to consider when protecting health in the state of New Jersey.

Moreover, the draft MCL proposed by DWQI did not properly emphasize that (1) analyses of serum PFOA remain highly variable, and (2) reported residues of PFOA do not correlate with effects. The science remains tenuous to link PFOA biomonitoring residues (as measures of exposure) to many of the specific potential human health effects under consideration. This

science remains in its infancy and, for this reason, an MOE (alternative approach) such as that used by other jurisdictions¹ is recommended. Uncertainty remains high even in PFOA analysis:

- The chemical/analytical precision and accuracy ascribed at low levels to water or serum in biomonitoring data often overshadow the tighter range of low-level biological responses. If accuracy of PFOA analyses is properly acknowledged to be less than 100% (for example, perhaps precise or accurate to only within +/- 30% of a true value) for serum, as compared to any given percent change in biological response (such as 5% for cancer or 10% for noncancer effect thresholds), the attempt to correlate analytical results to the DWQI-assessed candidate health effects is tenuous, at best. Thus, New Jersey should not be fooled into thinking that low-level analytical results are “true” and accurate when attempting to match water or serum PFOA to possible effects.
- Similarly, if additional DWQI safety factors did not drive down the reporting level of interest, particularly given that accuracy is only +/- 30% for low levels of PFOA in water (such as reported at the DWQI meeting on September 22, 2016 by one of the laboratories in attendance), the need to protect the health of New Jerseyans is still accomplished without trying to measure such extremely low levels at what would amount to unreasonably higher cost for more (and lower) analyses.

Alternate Approach Proposed: Reevaluate the Margin of Exposure

DWQI derived a far lower PFOA MCL of 14 ppt than the federal guideline by using only professional judgment to add an additional “safety factor” to account for possible mammary gland developmental effects that could occur at much lower doses than those that caused increased liver weight relied upon in the USEPA risk assessment resulting in the federal 70 ppt guideline for PFOA. Notwithstanding the weaknesses within the possible mammary gland evidence itself, as detailed below (please see “Weaknesses in Mammary Gland Body of Evidence”), the application of somewhat arbitrary “safety factors” would have impacts on NJ citizens (both in financial burden and health risk perception), and is not supported or borne out by the human evidence. Species differences explain why the DWQI conservatism is not necessary, and NJ citizens and water purveyors would be held to a more stringent (and less feasible to implement) standard than the science supports as necessary.

A specific alternate approach put forth in a scientific research assessment by another health jurisdiction¹ concluded,

Comparison of the PFOA serum levels associated with adverse effects in laboratory animals (13–77 µg/mL) with the serum or plasma levels found in non-occupationally

¹ Health Canada. 2012. Screening Assessment Report: Perfluorooctanoic Acid, its Salts, and its Precursors. http://www.ec.gc.ca/ese-ees/370AB133-3972-454F-A03A-F18890B58277/PFOA_EN.pdf

exposed adults, infants and children (0.00162–0.0195 µg/mL) results in margins of exposure greater than 660. These margins are considered to be adequately protective to account for uncertainties in the hazard and exposure databases.

Thus, using this scientific research approach, another health-protective jurisdiction did not find it necessary to apply additional “uncertainty factors” like DWQI chose to do because the MOE or “margin of safety” in biomonitoring results was sufficient. Based on Health Canada (2012)’s assessment (see Table 8¹),

The MOEs between the serum PFOA levels associated with the most sensitive effects in laboratory animals and the plasma PFOA levels in adult Canadians range from approximately 5100 to 30 600 for geometric means and from 2300 to 14 000 for 95th percentiles.

If DWQI believes that the MOE between PFOA serum levels of NJ citizens and adversely impacted laboratory animals requires the safety factors because of a specific NJ public policy, then DWQI should clearly state their calculated NJ MOE for transparency to the public and regulatory authorities regarding the “margin of safety” being included in the proposed 14 ppt PFOA MCL. CCNJ/SRIN do not believe DWQI has the data to support an MOE analysis; specifically, in the September 22, 2016 meeting materials, DWQI provided the following in a PowerPoint presentation²:

- Most recent (US NHANES) data (2011-2012) = Median: 2.1 ng/ml (parts per billion (ppb)) (serum), 95th percentile: 5.7 ng/ml (ppb) (*equal to US 0.0021 µg/mL median, and 0.0057 µg/mL 95th percentile, not much different from the Canadian values cited above¹*).
- “Biomonitoring data specific to New Jersey have not been collected.”

Therefore, because no biomonitoring data specific to New Jersey have been collected, New Jersey is unable to demonstrate to the public that the State’s citizens are any more at risk from PFOA accumulation in serum than people in other jurisdictions, so it is unclear why a more restrictive draft MCL is appropriate for its citizenry.

Comparison to Background Serum PFOA Lacks Context Regarding Target Human Serum Level

DWQI conflates two related but very distinct concepts – serum concentrations that exceed background, and serum concentrations that are within a range that may present a health risk. It

² <http://www.nj.gov/dep/watersupply/pdf/pfoa-hb-talk.pdf>

is inappropriate to present the increase in PFOA serum levels over background serum levels for exposures at select PFOA drinking water concentrations (see Figures E-1, 1 and 8 and associated test) without discussing the uncertainty and variability in the underlying assumptions and without tying serum levels to actual risk. DWQI's proposed MCL is based on their calculated Target Human Serum level of 14.5 ng/mL, but DWQI does not discuss the variability and uncertainty in the serum/plasma half-life data (Lorber et al. 2011), nor the fact that serum levels that exceed NHANES background serum levels but are below the Target Human Serum level would be interpreted as presenting a *de minimis* risk. DWQI should address these points so the public is not misled by DWQI's presentation of NHANES baseline levels.

Weaknesses in Mammary Gland Body of Evidence

DWQI derived a far lower PFOA MCL of 14 ppt by citing the work of Post et al. (2012). However, the Post et al. (2012) review manuscript³ would not meet most jurisdictions' quality standards, such as USEPA standards, for acceptance if it was conducted by industry. The Post et al. (2012) study fails to demonstrate any dose/response relationship in the reported data tables, and repeats data already summarized by the USEPA (2016) document⁴. For most of the original studies reviewed, group sizes are far too small (i.e. the Macon et al. 2011⁵ data set had n = 3 to 5 measurements of mammary glands per exposure group) to conclude a biological effect. When USEPA finalized its PFOA review in 2016, it did not find the Post et al. (2012) paper informative, as all key (original research) studies cited by Post et al. (2012) had been included in the USEPA prior assessment document.

In addition, the individual studies on mammary gland developmental delays often had significant weaknesses, such as lack of statistical adverse effects "due to interindividual variance and multiple criteria used to calculate mammary gland development scores"⁵. Finally, basing a regulatory action on this endpoint in a highly variable species (mice) is, as DWQI acknowledged, not done by other jurisdictions. In part, this may be because details that suggest that the murine mammary gland delayed development is not of biological significance in humans (e.g. the White et al. 2011⁶ data had demonstrated that no significant dose-related differences were found in

³ Post, G.B., P.D. Cohn, and K.R. Cooper. 2012. Perfluorooctanoic acid (PFOA), an emerging drinking water contaminant: a critical review of recent literature. *Environmental Research* 116:93–117.

⁴ USEPA. May 2016. *Health Effects Support Document for Perfluorooctanoic Acid (PFOA)*. Office of Water. EPA 822-R-16-003.

⁵ Macon, M.B., Villanueva, L.R., Tatum-Gibbs, K., Zehr, R.D., Strynar, M.J., Stanko, J.P., White, S.S., et al. 2011. Prenatal perfluorooctanoic acid exposure in CD-1 mice: low dose developmental effects and internal dosimetry. *Toxicol.Sci.* 122:134–145.

⁶ White, S.S., J.P. Stanko, K. Kato, A.M. Calafat, E.P. Hines, and S.E. Fenton. 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(8):1070–1076.

the ability of the CD-1 mice given 1 mg/kg/day to provide nourishment to their young as reflected in measurements of body weight in F1 and F2 pups across a 63-day postnatal period, as the USEPA (2016) document pointed out). This could mean that the gland developmental delay is inconsequential, or biologically insignificant. DWQI failed to properly discuss the inconsistency present in the various studies addressing this endpoint and failed to disclose the fact that there was no effect on puberty, reproduction, or nursing capabilities of the mice with delayed mammary gland development (Macon et al. 2011). If, overall, the underlying scientific data related to mammary gland development is insufficient and not robust enough to be the basis of a regulation, then forcing an additional uncertainty factor to be applied to account for the endpoint is not scientific or logical. Not only does consideration of possible PFOA-mediated developmental effects result in a lower MCL recommendation by the unwarranted extra uncertainty factor, the classification of PFOA as a developmental toxicant contributes to the public's unsupported concern for increased risk for women of childbearing age and infants.

Misrepresentation of Variability in Point of Departure for Non-cancer Endpoints

DWQI presented results from toxicity studies linking PFOA exposure to multiple noncancer endpoints of concern and claimed that they yield relatively consistent points of departure (POD) for calculating a human equivalent dose. However, DWQI did not apply a true weight-of-evidence analysis of the study data, nor a discussion regarding the severity of effects across the range of endpoints. Please refer to the attachment for a discussion of specific examples of limitations in DWQI's evaluation of immunotoxicity, mammary gland development, and liver toxicity. A more transparent discussion is needed regarding the variability and uncertainty in findings, and positions stated by regulatory agencies regarding the lack of suitability of endpoints for quantifying risks to humans.

Inconsistency in Relative Source Contribution

DWQI applied a default RSC of 20 percent based on the conclusion that there are insufficient data to develop a chemical-specific RSC for PFOA. This decision is not scientific and is illogical and inconsistent. Not only is there sufficient information to derive a chemical-specific RSC, DWQI chose to adopt a non-default RSC of 50% for PFNA, despite significantly less PFNA exposure data than is available for PFOA. Available data strongly support a higher RSC, similar to the situation for PFNA. Some of the analysis conducted by DWQI in Appendix 2 can be used to demonstrate a higher RSC for drinking water exposure. Furthermore, the very same publication used by DWQI to support the PFOA clearance factor, Lorber et al. (2011), provides data to support a RSC of between 60 and 70 percent. It is illogical for DWQI to utilize data from Lorber (2011) for a parameter that impacts the health-based MCL by over 100 fold (i.e. the clearance factor), and not use this same publication and data therein to support a PFOA-specific RSC.

Weakness in Link between PFOA Exposure and Cancer Outcomes

DWQI significantly overstated the link between PFOA exposure and carcinogenicity in humans and failed to rely on the most recent comprehensive assessments of the available science. DWQI cites conclusions from an USEPA Science Advisory report published in 2006⁷, rather than the more recent peer-reviewed evaluation of PFOA carcinogenicity conducted by USEPA Office of Water (2016)⁸. In addition, DWQI cites the IARC cancer evaluation for PFOA, despite the shortcomings of IARC's antiquated classification scheme noted by international scientists⁹, and did not consider the recent Health Council of the Netherlands evaluation that concluded that the data are insufficient to make any conclusions regarding the carcinogenicity of PFOA and its salts¹⁰.

In addition, DWQI derives a human equivalent dose from an administered dose in rats (Butenhoff et al. 2012), rather than a study that reports the internal dose (serum PFOA)¹¹. This is inconsistent with the data usability criteria applied by DWQI for non-cancer studies (pg. 205-206): "Only those studies that provide serum PFOA data were considered for dose-response modeling for non-carcinogenic effects. A risk assessment approach based on measured serum PFOA levels is less uncertain than one based on pharmacokinetic modeling of estimated serum PFOA levels or an approach in which interspecies extrapolations is based on interspecies half-life differences." DWQI should thoroughly discuss the uncertainties and limitations in their quantitative cancer analysis.

Testing Subcommittee Report

Although DWQI recommended a PQL of 6 ppt and referenced a PowerPoint presentation¹² on September 22, 2016 that stated this low PQL is "achievable" by laboratories and "the Testing Subcommittee decided to use the method for deriving the PFOA PQL that takes into consideration both the precision and accuracy of the analytical method", no reported values

⁷ USEPA. 2006. United States Environmental Protection Agency. Science Advisory Board Review of EPA's Draft Risk Assessment of Potential Human Health Effects Associated with PFOA and Its Salts, May 30.

⁸ USEPA. 2016. United States Environmental Protection Agency. Drinking Water Health Advisory for Perfluorooctanoic Acid (PFOA). Office of Water. EPA 822-R-16-005. May.

⁹ Boobis, A.R., Cohen, S.M., Dellarco, V.L., Doe, J.E., Fenner-Crisp, P.A., Moretto, A., Pastoor, T.P., Schoeny, R.S., Seed, J.G. and Wolf, D.C., 2016. Classification schemes for carcinogenicity based on hazard-identification have become outmoded and serve neither science nor society. *Reg. Toxicol. Pharm.* Oct. 22.

¹⁰ Health Council of the Netherlands. 2013. Perfluorooctanoic acid and its salts - Evaluation of the carcinogenicity and genotoxicity. The Hague: Health Council of the Netherlands; publication no. 2013/32 ISBN 978-90-5549-982-3. http://www.gezondheidsraad.nl/sites/default/files/201332Perfluorooctanoic_acid_and_its_salts.pdf

¹¹ Butenhoff, J. L., Kennedy, G. L. Jr., Chang, S. C. Olsen, G. W. (2012). Chronic dietary toxicity and carcinogenicity study with ammonium perfluorooctanoate in Sprague-Dawley rats. *Toxicology* 298: 1-13

¹² <http://www.nj.gov/dep/watersupply/pfoa-test.pptx>

were given by the summarized laboratories to actually allow the public to evaluate “both the precision and accuracy” of the analytical method. Instead, many of the laboratories’ PQLs reported in the materials actually do not meet the 6 ppt proposed PQL utilizing USEPA Method 537, making it unclear that this is truly an achievable or worthy target.

This unnecessary 6 ppt PQL is supported further by the fact that even the UCMR sampling for PFOA was generally unable nationwide to satisfactorily quantify PFOA (see most recent July 2016 UCMR database release, downloadable from USEPA) below the MRL of 0.02 ppb (20 ppt) using USEPA Method 537. In addition, USEPA states in the May 2, 2012 Federal Register, “while particular laboratories may be able to meet MRLs lower than those proposed, the selected MRLs reflect those achievable by the national array of laboratories that support the program.” DWQI setting a 14 ppt MCL (with PQL of 6 ppt) is unprecedented given the UCMR3 dataset for New Jersey indicate that all 176 water supplies have detected PFOA less than 20 ppt MRL and 18 water supplies detected slightly above the 20 ppt MRL. There is no way to distinguish if any of the 176 water supplies that detected below 20 ppt MRL would be in compliance with the proposed 14 ppt MCL. Based on UCMR New Jersey water supplies, PFOA data indicate sufficiently below the federal protective guideline of 70 ppt. If DWQI has laboratory datasets demonstrating there is a detectable (within significant figures and precision ranges) difference between 14 ppt MCL and 20 ppt MRL utilizing USEPA Method 537 reliably with field samples (vs. laboratory-created samples), these data sets should be shared. Requiring a more stringent PFOA MCL is an unnecessary action given available data to date, regardless if the draft MCL of 14 ppt is adopted, as the current USEPA Method 537 MRL of 20 ppt is (within scientific significant figures) unlikely to be discernible from 14 ppt given the precision and accuracy of the methods being used.

Additionally, the proposed PQL of 6 ppt, which was derived with an ideal matrix in the laboratory setting, does not account for the real-life challenges of obtaining representative samples at ppt levels for PFOA. Although this problem is not unique to PFOA, it is greatly exacerbated by the extremely low ppt levels being proposed for regulation and the ubiquity of PFCs, including PFOA, in the environment. PFOA is known to be distributed widely in the environment as a result of atmospheric deposition and other natural cycles. In addition, PFCs, including PFOA, are present in many products, consumer and otherwise, that may be sources of false positives related to cross-contamination; these include Teflon[®], water-resistant field books, markers (including Sharpie[®] markers), Post-it-Notes, Gor-Tex[™] and Tyvek[®] garments, fabric softeners, and “blue ice” packs. USEPA Method 537.1 attempts to mitigate false positives by proscribing the use of Teflon[®] laboratory ware; however, the potential for cross-contamination during sample collection and handling has not received adequate attention and may significantly impair the acquisition of

representative samples at very low levels. Several organizations, such as the US Navy¹³, have advised caution and issued guidance on this topic, but neither USEPA nor DWQI have considered the issue. It is wholly unreasonable for DWQI to recommend an MCL for PFOA at a concentration that may often be detected due to cross-contamination rather than actual presence in the water supply, and it is premature to regulate PFCs (e.g. PFNA, PFOA) at such incomparably low levels without further study regarding the feasibility of obtaining representative data.

Also, it is of critical importance to have data from laboratories that have an intuitive understanding of and conduct work in accordance with NJDEP requirements. An appropriate phase-in period must be allowed for laboratories to be certified in New Jersey so that there is adequate capacity and geographic coverage.

Treatment Subcommittee Report

Regarding treatment options for PFOA, the Health Effects Subcommittee Report correctly states (first paragraph on page 4 and last paragraph on page 30) that, while PFOA and other perfluorinated compounds (PFCs) are not effectively removed from drinking water by standard treatment processes, they can be removed from drinking water by GAC or reverse osmosis. However, the report fails to indicate that treatment via anion exchange resin (stand-alone or as a polish to GAC) may also offer significant improvement over stand-alone GAC treatment in terms of both treatment performance and cost effectiveness, particularly for PFOA and PFOS compounds. Since the promise of anion exchange as a treatment option is discussed in the Addendum to Appendix C: Recommendation on Perfluorinated Compound Treatment Options for Drinking Water, CCNJ/SRIN recommend that this treatment option be included in the general discussion in the noted places within the Health Effects Subcommittee Report.

In addition, DWQI does not evaluate the feasibility of water suppliers of all kinds and types across the State implementing carbon or other treatment on their water supplies. This failure means that DWQI has not evaluated the feasibility of implementing the MCL it recommends. This will result in water suppliers to increase costs to treat the PFOA water and hence consumers in the state of New Jersey.

The 2015 Appendix C document states (page 10) that “USEPA notes that “incineration of the concentrated wastes would be needed for the complete destruction of PFCs” (2014)”, which is only a best management practice; there is no discussion of regulatory basis for how this waste

¹³ Department of the Navy. 2015. Navy Drinking Water Sampling Policy for Perfluorochemicals PFOS and PFOA. Letter from Director, Energy and Environmental Readiness (OPNAV N45) to Commander, Navy Installations (N4). September 14, 2015.

may be classified under RCRA. Any discussion of availability and viability of treatment must consider and discuss regulatory disposal requirements (vs. recommendations) of any waste streams.

Compliance with Executive Order No. 2

CCNJ/SRIN must point to the "Common Sense Principles" established by Governor Christie in EO #2 issued on January 20, 2010. Among the principles outlined in the Order were the following:

- Employ the use of cost/benefit analyses, **as well as scientific and economic research from other jurisdictions**, including but not limited to the federal government when conducting an economic impact analysis on a proposed rule. [emphasis added]
- Detail and justify every instance where a proposed rule exceeds the requirements of federal law or regulation. State agencies shall, when promulgating proposed rules, **not exceed the requirements of federal law except when required by State statute or in such circumstances where exceeding the requirements of federal law or regulation is necessary in order to achieve a New Jersey specific public policy goal**. [emphasis added]

DWQI must ensure that these principles are followed with respect to its PFOA regulatory actions. The two bolded passages from EO #2, above, are particularly relevant to the DWQI activity regarding PFOA. We would like to refer DWQI to the fact that scientific research from other jurisdictions (at the federal and international scale) is available and had not been considered. Second, the draft NJ MCL will "exceed the requirements of federal law" without providing any additional proven health protection, and without the necessary cost/benefit analysis. As such, CCNJ/SRIN believe strongly that the draft recommendation failed to give appropriate consideration to EO #2.

Summary

As discussed above, DWQI is proposing an MCL for New Jersey that is far lower than the guideline the federal government recently determined is protective for drinking water. CCNJ/SRIN strongly suggest that DWQI reevaluate the quality of the mammary gland developmental study data and reconsider that the "margin of safety" afforded by the USEPA federal PFOA guideline of 70 ppt for the citizens of New Jersey is sufficient. As other jurisdictions have found, the MOE approach is protective for its citizens; therefore, they have not based excessive safety factors on top of lower-quality findings, and New Jersey should reject the draft MCL DWQI proposed. No additional known health protection is achieved, suggesting the DWQI proposal does not overcome the additional cost and reporting/regulatory burden that would unnecessarily hinder economic growth and success in New Jersey.

DWQI must be mindful of the mandates established by Governor Christie in EO #2 and look to the science being developed in other states and by the federal government. The works completed by other states/countries and the USEPA are also informative to DWQI's PFOA review. It is imperative that the Institute review these works, as they clearly help identify the flaws in New Jersey's current scientific literature regarding PFOA. In situations where urgency is required and federal guidance is available, it is a sound policy for the State to rely on the federal guidance and allow the scientific process to develop data to support MCLs and other NJ environmental standards.

Thank you for the consideration of our comments on this very important issue. We look forward to working with DWQI as it continues its work in recommending drinking water quality standards in New Jersey. If I can be of further assistance, please let me know.

Sincerely,



Dennis Hart
Executive Director

Attachment

References

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USEPA. 2016. *Drinking Water Health Advisory for Perfluorooctanoic Acid (PFOA)*. Office of Water. EPA 822-R-16-005. May.

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COMMENTS ON NEW JERSEY DRINKING WATER QUALITY INSTITUTE HEALTH-BASED MAXIMUM CONTAMINANT LEVEL FOR PERFLUOROOCCTANOIC ACID (PFOA); JUNE 2016

The following are comments on the *Public Review Draft Health-Based Maximum Contaminant Level Support Document: Perfluorooctanoic Acid (PFOA)* developed by the New Jersey Drinking Water Quality Institute (DWQI) Health Effects Subcommittee, dated June 27, 2016. DWQI is recommending a maximum contaminant level (MCL) for PFOA of 0.014 µg/L or parts per billion (ppb), equivalent to 14 parts per trillion (ppt). The MCL, however is based on a number of uncertainties that significantly undermine the scientific merit of DWQI's recommendation, most notably:

- 1) the decision to select a health effect endpoint in rodents that toxicologists generally concur is not likely to extend to humans;
- 2) application of an additional uncertainty factor for an effect endpoint that did not actually result in adverse effect in rodents and has uncertain relevance to humans; and
- 3) an assumption about the relative source contribution of drinking water to total PFOA exposure that is both illogical and contrary to earlier recommendations by the same committee for the health-based MCL for perfluorononanoic acid (PFNA).

This document discusses these specific shortcomings, which we would request that DWQI consider if their final recommendation for a health-based MCL for PFOA is intended to reflect the best available science.

General Comment

Overall, we find the support document to be extremely biased in its characterization of PFOA risks. DWQI did not appropriately handle the uncertainty and inconsistency within the database for health effects associated with PFOA exposure – regarding both the animal toxicity study data and human epidemiology data. DWQI appears to have used data selectively to support its analysis, rather than considering the full weight of evidence.

Studies and findings that support DWQI's conclusions were presented with high confidence and little mention of weaknesses or limitations, while studies and findings that demonstrate inconsistent and/or conflicting interpretations were either not provided or were presented in a manner that over emphasized weaknesses and limitations in those studies. This misrepresentation of PFOA toxicity is found throughout the document and is extremely concerning. The failure to conduct a comprehensive weight-of-evidence review on the full range of data and the selection of a disproportionately low regulatory value for

PFOA, results in unwarranted public concern and economic impacts. The adverse impacts of which to the public have recently been discussed by the Australian Government Department of Health in their selection of interim drinking water guidance values for perfluoroalkyl substances (enHealth 2016).

Specific Comments

The following specific comments highlight the key technical deficiencies in DWQI's approach.

1. Justification is needed for the selected noncancer endpoints, including transparent and fair representation of the actual adversity of selected endpoints.

DWQI presented results from toxicity studies linking PFOA exposure to multiple noncancer endpoints of concern and claimed that they yield relatively consistent points of departure (POD) for calculating a human equivalent dose. However, DWQI did not apply a true weight-of-evidence analysis of the study data, nor a discussion regarding the severity of effects across the range of endpoints.

A more comprehensive analysis is needed on the following endpoints:

- a. *Immunotoxicity.* Published research findings should be cited that provide the range of "normal" antibody levels in children to establish adversity of the epidemiological studies. Additionally, DWQI should discuss potential confounding factors (i.e., factors other than PFOA exposure) that may contribute to changes in antibody levels. A more transparent discussion is needed regarding the weight-of-evidence of positive and negative findings for this endpoint.
- b. *Mammary gland development.* There is a wide range of findings for this endpoint. DWQI should consider and better present inconsistencies in the database regarding developing mammary gland effects, including negative studies, studies that showed inhibition of mammary gland development, and the studies that showed a stimulation of glandular development. A more thorough analysis is needed regarding the biological significance of the effects, including the relevance of the lack of concurrent adverse effects on reproduction and nursing capabilities for studies for which inhibition of mammary gland development is noted (Macon et al. 2011) (see comment 5 below).
- c. *Liver toxicity.* Several regulatory agencies have clearly stated that rodent toxicity studies that demonstrate liver hypertrophy in the absence of cell toxicity or other evidence of potential impairment of liver function are not

suitable for quantifying risks to humans (TOXSAC 2002; Hall et al. 2012). We strongly agree, and recommend that DWQI provide a more transparent presentation of the data showing PFOA-mediated effects on the rodent liver, including a discussion on where actual liver toxicity is or is not observed along the dose-response continuum (see comment 2 below).

- 2. At the selected point of departure, liver weight increases in rodents are indicative of exposure rather than effects and there has not been evidence of liver disease in PFOA exposed humans, therefore, liver weight is not an appropriate critical effect from which to derive a health-based MCL.**

DWQI derived the health-based MCL on increased liver weights in rodents, even though there is strong evidence that at this low dose, the measured change in the liver is an adaptive response associated with normal liver functioning, and not evidence of an adverse effect. Increased liver weight without histopathological changes indicative of cell damage (at the same dose in the same study) should be considered non-adverse. A report by the European Society of Toxicologic Pathology evaluated adverse versus adaptive changes in the liver and concluded that liver weight increases up to 150% of control values may still be considered non-adverse in the context of chemical safety evaluations (Hall et al., 2012).

Additionally, the EPA 2002 guidance document from the Health Effects Division Toxicology Science Advisory Council (TOXSAC 2002) notes that a statistically significant increase in liver size alone (i.e., in the absence of histopathological evidence) is not a reliable indicator of hepatic toxicity. In conclusion, the existing data do not clearly support the liver as an appropriate target organ following low level PFOA exposures. Finally, using established toxicology guidance, the data suggest that liver weight changes in the rodents from Loveless et al. (2006) are adaptive, non-adverse effects.

Additionally, we maintain that the existing data do not clearly link low-level human exposures to PFOA-mediated adverse liver effects in humans. There have not been consistent associations of PFOA with liver biomarkers or liver changes in human studies, including those with serum concentrations equivalent to and much greater than levels used to calculate the proposed MCL. Data from studies of workers with high exposures to PFOA have not demonstrated evidence of increased liver disease and data on liver enzyme parameters have been inconsistent (for example, see Leonard 2003; Sakr et al. 2007; Leonard et al. 2008; Lundin et al. 2009; Steeland and Woskie, 2012). We recommend that DWQI select an alternative effect endpoint for which to base their PFOA MCL.

- 3. Stronger justification is needed to support the use of rodent toxicity studies with PFOA to infer comparable dose-response relationship in humans.**

DWQI relied on rodent data despite the consensus among the scientific community regarding the high uncertainty in extrapolating PFOA health effect levels from rodents to humans. We disagree with DWQI's assessment of the PFOA mode of action, specifically regarding the peroxisome proliferator-activated receptor-alpha (PPAR-alpha) and other nuclear receptors, and with DWQI's overall conclusion that rodent data are appropriate for assessing human risk. DWQI's use of rodent liver data is clearly inconsistent with conclusions reached by ATSDR (2015) and other health and environmental regulatory agencies. It is well known that PFOA mediates many of its cellular effects through PPAR-alpha, constitutive androstane receptor (CAR), and pregnane X receptor (PXR). It is also well known that humans are less responsive to the effects of these nuclear receptor signaling pathways. While it may not be currently possible to completely rule out the contribution of other (non-PPAR-alpha) modes of action for many PFOA-mediated effects (especially effects occurring in organs other than the liver), given that rodents are more responsive than humans to many of these nuclear receptor activations, the quantitative relevance of rodent data is dubious at best.

Animal-to-human extrapolation uncertainty is common in regulatory toxicology. However, with PFOA, the science is clear that rodent data have severe limitations in terms of human relevance. Rather than ignoring this fact, a more scientifically supportable approach would be for DWQI to assess this uncertainty quantitatively. Specifically, if rodent data are to be used to quantify a human threshold value for PFOA, species-specific differences in toxicokinetics and toxicodynamics should be assessed using available modeling tools. DWQI failed to accurately account for the key species-specific differences in a quantitative manner.

4. The limited of evidence on carcinogenic potential of PFOA should be presented more clearly and accurately.

DWQI significantly overstated the potential link between PFOA exposure and carcinogenicity in humans.

- a. Throughout the support document, DWQI highlighted the outdated and unofficial EPA Science Advisory Board (SAB) conclusions from 2006, over the more recent EPA Office of Water cancer description (USEPA 2016). All references to the EPA SAB report should be deleted. The SAB report is not a peer-reviewed final report representing EPA positions. Furthermore, it is not the most recent EPA evaluation of PFOA carcinogenicity and it is disingenuous to continue to reference this document and diminish the more recent and peer-reviewed EPA Office of Water Health Advisory document (USEPA 2016), which followed the same EPA cancer guidelines (USEPA 2005).

- b. Additionally, DWQI included the recent IARC cancer evaluation for PFOA, but did not include the recent Health Council of the Netherlands evaluation that concluded that the data are insufficient to make any conclusions regarding the carcinogenicity of PFOA and its salts. Importantly, IARC has recently come under attack from international scientists for their antiquated and inaccurate cancer assessments that do not include a weight-of-evidence approach, do not include all relevant regulatory studies, and that cause unnecessary health scares in the public (Boobis et al. 2016). We recommend a more balanced and fair evaluation of the PFOA carcinogenicity data, taking into consideration the evaluation of the Health Council of the Netherlands (Health Council of the Netherlands 2013), using a true weight-of-evidence analysis, and using only authoritative and recent regulatory references.
- c. Despite significant evidence demonstrating no link between human exposures to PFOA and cancer, DWQI summarized the positive studies and ignored conflicting evidence and limitations of studies that do not support DWQI's conclusions. Although the cancer endpoint was not explicitly used to derive the draft MCL, DWQI's cancer discussion for PFOA could contribute to the overall narrative of PFOA as a "cancer causing chemical" in the public perception. We recommend that DWQI provide a more fair and balanced analysis of PFOA potential carcinogenicity, which would lend to the conclusion that there is not a causal association between PFOA exposure and cancer in humans (see also Chang et al. 2014).
- d. DWQI's methodology for developing the human equivalent dose from the administered dose in rats from Butenhoff et al. (2012) is in direct conflict with their study usability criteria for noncancer effects. DWQI stated on pg. 205-206, "Only those studies that provide serum PFOA data were considered for dose-response modeling of non-carcinogenic effects. A risk assessment approach based on measured serum PFOA levels is less uncertain than one based on pharmacokinetic modeling of estimated serum PFOA levels or an approach in which interspecies extrapolations is based on interspecies half-life differences." However, to calculate the cancer-based MCL, the dose in rats corresponding to a 1×10^{-6} cancer risk level was converted to the human equivalent dose using a pharmacokinetic adjustment based on the ratio of half-lives in the two species. In the noncancer portion of their assessment, DWQI acknowledged the extreme uncertainty with this method of dosimetric conversion, and used this criteria to justify excluding numerous studies from evaluation for noncancer effects. However, nowhere in the document did DWQI acknowledge this inconsistency nor discuss the highly uncertain nature of their cancer-based

MCL. We recommend that DWQI apply the same study usability criteria, consistent for both cancer and noncancer evaluations. We also recommend that DWQI thoroughly discuss the uncertainties and limitations in their quantitative cancer analysis.

- 5. An extra uncertainty factor of 10 was applied to account for potential developmental toxicity; however, this factor should be removed given the weak evidence for developmental toxicity.**

Each uncertainty factor applied by DWQI results in a lower MCL. DWQI applied an extra uncertainty factor of 10 to account for potential developmental effects in humans. DWQI based the decision on a PFOA study in rodents in which delayed mammary gland development was observed following perinatal exposure. As DWQI acknowledged, this is a highly uncertain endpoint, with limited support in the rodent toxicology literature and no clear link to human adverse effects.

DWQI failed to properly discuss the inconsistency present in the various studies addressing this endpoint and failed to disclose the fact that there was no effect on puberty, reproduction, or nursing capabilities of the mice with delayed mammary gland development (Macon et al. 2011), further indicating that the endpoint is highly uncertain and is not biologically significant or adverse.

Not only does consideration of possible PFOA-mediated developmental effects result in a lower MCL recommendation by the unwarranted extra uncertainty factor, the classification of PFOA as a developmental toxicant contributes to the public's unsupported concern for increased risk for women of childbearing age and infants. DWQI should remove this unnecessary uncertainty factor.

- 6. Consistent with EPA guidance (USEPA 2000) and DWQI's analysis for PFNA, DWQI should use PFOA-specific exposure information rather than a default Relative Source Contribution (RSC) of 20 percent.**

The default RSC of 20% for drinking water is used to calculate health-based MCLs in the absence of additional chemical-specific or site-specific information. DWQI applied this default based on the conclusion that there are insufficient data to develop a chemical-specific RSC for PFOA (pg 215).

This decision is illogical and inconsistent. Not only is there sufficient information to derive a chemical-specific RSC, DWQI chose to adopt a non-default RSC of 50% for PFNA, despite significantly less PFNA exposure data than is available for PFOA.

We disagree with DWQI's rationale for the default RSC for PFOA because available data strongly support a higher RSC, similar to the situation for PFNA. Some of the

analysis conducted by DWQI in Appendix 2 can be used to demonstrate a higher RSC for drinking water exposure. Furthermore, the very same publication used by DWQI to support the PFOA clearance factor, Lorber et al. 2011, provides data to support a RSC of between 60 and 70%. It is illogical for DWQI to utilize data from Lorber (2011) for a parameter that impacts the health-based MCL by over 100 fold (i.e., the clearance factor), and not use this same publication and data therein to support a PFOA-specific RSC.

We strongly recommend that DWQI base the RSC on an analysis of PFOA exposure data rather than a default assumption. Furthermore, we recommend that DWQI demonstrate the impact of various RSCs on the final MCL recommendation, using summary statistics from NHANES (e.g., median or 95th percentile) and the Target Human Serum level chosen for the health-based MCL as comparison points.

7. **DWQI appears to conflate two related but very distinct concepts – serum concentrations that exceed background, and serum concentrations that are within a range that may present a health risk. It is inappropriate to present the increase in PFOA serum levels over background serum levels for exposures at select PFOA drinking water concentrations (see Figures E-1, 1 and 8 and associated test) without discussing the uncertainty and variability in the underlying assumptions and without tying serum levels to actual risk.**

DWQI presented the increase in serum levels over background for exposures at a range of PFOA drinking water concentrations. This discussion does not contribute any meaningful information to the potential health risks related to PFOA exposure and should not be provided (numerous times) in the health-based MCL support document for the following reasons:

- a. This analysis relies exclusively on the assumptions within Lorber et al. (2011) and DWQI's analysis does not demonstrate or even mention the variability and uncertainty in this analysis. For example, DWQI's Table 4 presents a range of serum/plasma half-lives, however, the assumptions within Lorber et al. (2011) rely on only one single PFOA half-life estimate. More explanation is needed regarding the basis for the range.
- b. DWQI's focus on exceedance of background levels is at odds with USEPA's definition of toxicity reference values used to derive criteria protective of adverse health effects. USEPA (2002) recommends the following definition for a noncancer threshold value, "an estimate of an exposure, designated by duration and route, to the human population (including susceptible subgroups) that is likely to be without an appreciable risk of adverse health effects over a lifetime." Accordingly, for the MCL for PFOA that is calculated from a Target Human Serum level, there is high confidence that

serum concentrations below this level present a *de minimis* risk, even if such concentrations are elevated above NHANES baseline levels. Furthermore, without additional evaluation of dose-response relationships for PFOA using serum data, no specific health risk statements can be made regarding human serum levels that exceed the Target Human Serum level.

- c. Nowhere does DWQI discuss the interval between NHANES background serum levels and their calculated Target Human Serum level of 14.5 ng/mL. This is an egregious risk communication error. Even though an increase in serum PFOA level above baseline does not equate with a health risk to an individual, the public may incorrectly draw this conclusion. We recommend that DWQI remove the calculation of PFOA serum level increases for various drinking water concentrations from the support document entirely. At the very least, this analysis should be relegated to an appendix and not have such a prominent discussion multiple times throughout the main support document, and should include transparent discussion of where target human serum levels for the critical effect fall relative to NHANES.

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