May 31, 2019

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Department of Environmental Protection
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PO Box 402
Trenton, New Jersey 08625-0402
Attn: DEP Docket Number: 02-19-03 Proposal Number: PRN 2019-042

Re: DRN comments on Proposed Health-Based Maximum Contaminant Level (MCL) in drinking water for PFOA and PFOS and related regulatory proposals

Please find enclosed Delaware Riverkeeper Network's comment and the technical analysis prepared by Fardin Oliaei, MPA, PhD, and Don Kriens, Sc.D., P.E. of Cambridge Environmental Consulting dated May 27, 2019 commissioned by Delaware Riverkeeper Network and submitted on behalf of the organization and its membership on the New Jersey Department of Environmental Protection (DEP) proposal regarding:
Discharges of Petroleum and Other Hazardous Substances Rules; **Ground Water Quality Standards Rules; Private Well Testing Act Rules; Safe Drinking Water Act Rules; and New Jersey Pollutant Discharge Elimination System Rules**
Ground Water Quality Standards and Maximum Contaminant Levels (MCLs) for Perfluorooctanoic Acid (PFOA) and Perfluorooctanesulfonic Acid (PFOS)
Proposed Amendments: N.J.A.C. 7:1E Appendix A, 7:9C Appendix Table 1, 7:9E-2.1, 7:10-5.2, and 12.30, 7:14A-4 Appendix A and 7.9

Comments by Cambridge Environmental Consulting entitled "Comments on the New Jersey Department of Environmental Protection Proposal to Amend the New Jersey Safe Drinking Water Act to Establish a Maximum Contaminant Level (MCL) of 14 nanograms per liter (ng/l) for perfluorooctanoic acid (PFOA) and a MCL of 13 ng/l for perfluoroctanesulfonate (PFOS)", pertain directly to the MCLs proposed by DEP and pertain to the other proposed regulatory changes in regards to the technical analyses that are the basis for standards employed in the related regulations.

Also enclosed is an excel worksheet regarding children's intakes. Also enclosed are the Curriculum Vitae for Dr. Oliaei and for Don Kriens, Sc.D., P.E.

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Delaware Riverkeeper Network submits these comments advocating that the public be protected from PFOA and PFOS contamination and that New Jersey's drinking water be required to be treated to a safe level based on the best available scientific evidence that protects all populations, including vulnerable age groups of children, as detailed in the enclosed excel worksheet. We support Cambridge Environmental Consulting's recommendation that PFOA and PFOS MCLs should be set for the most sensitive endpoint based on the scientific evidence presented in their enclosed report.

We support all the recommendations and findings made by Cambridge Environmental Consulting in these technical comments. We support Cambridge Environmental Consulting's proposal that MCLs for PFOA and PFOS be set at ≤ 1.0 ng/l based on immunotoxicity endpoints. Absent MCLs set at ≤ 1.0 ng/l based on immunotoxicity, the MCL for PFOA should be no greater than 6 ng/l and the MCL for PFOS no greater than 5 ng/l based on use of children age-specific body weights and water intakes for children age group 1-6.

We also include portions of Delaware Riverkeeper Network's verbal testimony that address other proposed regulatory changes included in DEP's proposal.

Sincerely,

Maya van Rossum
the Delaware Riverkeeper

Tracy Carluccio
Deputy Director

Enclosed:

"Comments on the New Jersey Department of Environmental Protection Proposal to Amend the New Jersey Safe Drinking Water Act to Establish a Maximum Contaminant Level (MCL) of 14 nanograms per liter (ng/l) for perfluorooctanoic acid (PFOA) and a MCL of 13 ng/l for perfluorooctanesulfonate (PFOS)", Fardin Oliaei, Ph.D. and Don L. Kriens, Sc.D., Cambridge Environmental Consulting, May 27, 2019

Excel Worksheet Children Intakes, Cambridge Environmental Consulting May 28, 2019

Curriculum Vitae for Dr. Oliaei and for Don Kriens, Sc.D., P.E.

Excerpts from Verbal Testimony, Tracy Carluccio, Delaware Riverkeeper Network, NJ DEP Drinking Water Standards and other Regulatory Proposals re. PFOA and PFOS, Docket 02-19-03, May 15, 2019

Comments on the New Jersey Department of Environmental Protection Proposal to Amend the New Jersey Safe Drinking Water Act to Establish a Maximum Contaminant Level (MCL) of 14 nanograms per liter (ng/l) for perfluorooctanoic acid (PFOA) and a MCL of 13 ng/l for perfluoroctanesulfonate (PFOS)

by

Fardin Oliaei, Ph.D. and Don L. Kriens, Sc.D.

Cambridge Environmental Consulting

May 27, 2019

Executive Summary

We comment on New Jersey Department of Environmental Protection proposed rule to amend the New Jersey Safe Drinking Water Act (SDWA) to establish an MCL (maximum contaminant level) for PFOA (perfluorooctanoic acid) at 14 ng/l and an MCL for PFOS (perfluorooctanesulfonic acid) at 13 ng/l in drinking water. The proposed rule also establishes the same concentrations for PFOA and PFOS as ground water quality standards, and adds PFNA (perfluorononanoic acid), PFOA, and PFOS to testing requirements under NJPDES (New Jersey Pollutant Discharge Elimination System) rules for discharge to ground water.

This submittal is an update of our prior comments. In November 2016 we commented on the New Jersey Drinking Water Quality Institute (NJDWQI) recommended MCL for PFOA of 14 ng/l, based on the non-cancer toxicity endpoint of increased liver weight, and in January 2018 we commented on the Institute's recommended MCL for PFOS of 13 ng/l, based on the non-cancer toxicity endpoint of immune system suppression.

Serious adverse health effects are related to low-level exposures to PFOS and PFOA. Although the NJDWQI completed comprehensive analyses to determine RfDs (reference doses) and MCLs for PFOA and PFOS and should be commended on leading regulatory protection efforts for these chemicals, we believe that NJDWQI chose less conservative options to establish these MCLs. The proposed MCLs do not protect all consumers of drinking water contaminated with PFOS and PFOA, and other PFAS (perfluoroalkyl substances) likely present, especially vulnerable age groups of children. We base this conclusion in two areas:

- Toxicity Endpoints and Uncertainty factors (UFs) Used
- Inaccurate Exposure Variables Used

We find that the granular activated carbon treatment technology chosen by the NJDWQI treatment subcommittee will not consistently and reliably remove PFOA, and will not efficiently remove other perfluorinated compounds (such as the short- chain perfluorinated compounds) likely to be present (and pose toxicity) in drinking waters contaminated with these chemicals. Although long-chain PFAS have similar properties, as stated by the subcommittee, there are significant differences in treatability due to differences in structural chemistry, as demonstrated at many treatment installations and research projects.

NJDWQI use of adult default exposure values of 70 kg for body weight and 2.0 l/day water intake results in excessive, and therefore unsafe, PFOS and PFOA risk to nearly all ages of children. Establishment of the proposed MCLs may result in daily doses to children age 2 months through 13 years that exceed the reference doses derived by NJDWQI. The MCLs derived should afford protection to all groups, especially young children who have greater vulnerability to environmental toxicants. Appropriate age-specific exposure values for body weights and water intakes should be used to derive MCLs for children subpopulations to enable protection.

We propose that MCLs for PFOA and PFOS be set at ≤ 1.0 ng/l based on immunotoxicity endpoints. Absent MCLs set at ≤ 1.0 ng/l based on immunotoxicity, the MCL for PFOA should be no greater than 6 ng/l and the MCL for PFOS no greater than 5 ng/l based on use of children age-specific body weights and water intakes for children age group 1-6.

1. Toxicity Endpoints and Uncertainty Factors Used

a. Mammary Gland Development Toxicity Endpoint - PFOA

The toxicity endpoint of delayed mammary gland development should be used as the most sensitive endpoint in the PFOA MCL derivation. This effect was shown in nine different studies (NJDWQI report 2016). As we indicated previously, delayed mammary gland development is concerning since adverse effects related to delayed mammary gland development persist into adulthood and become permanent. Several researchers indicate that delayed mammary gland growth may result in greater susceptibility to cancer later in life (Fenton 2006; Rudel et al., 2011; Fenton et al., 2012; Osborne et al. 2015). Others note that developmental exposures in sensitive time periods can result in increased risk of later disease or dysfunction (Heindel and Vandenberg, 2015). The mode of action is explained by Osborne as: "Anything that changes the timing of mammary development will affect the timing of the presence of TEBs (terminal end buds), and therefore the window of susceptibility to carcinogens. Late initiation of mammary development causes decreased longitudinal growth of the epithelium and fewer TEBs, and decreased alveolar budding at weaning. As development progresses, these glands may have more TEBs at puberty, because the pace of development is slower. It is hypothesized that factors that lengthen the period when TEBs are present lengthen the period during which the MG (mammary gland) is susceptible to carcinogens" (Osborne et al., 2015).

NJDWQI acknowledged these studies but chose not to use delayed mammary gland development RfD as the basis for a recommended Health-based MCL, not because of uncertainty about the scientific validity of doing so, but rather because of a lack of precedent to use this endpoint as the primary basis for health-based criteria for environmental contaminants. Instead, an additional 10 UF was applied to an unrelated endpoint (increased liver weight that forms the basis of the MCL derivation) to compensate. In our November 2016 report we opined "why not use the more sensitive endpoint for which adequate toxicity data already exists, including a BMDL, even if that endpoint has not previously been used, versus adding an additional uncertainty factor to an alternate endpoint to compensate for an uncertainty that is, in fact, known?"

Animal studies showing delayed mammary gland development are sufficient, peer reviewed, and appropriate to use in the PFOA MCL determination. The lack of precedent to use this endpoint does not form a basis to not use it. We calculate a MCL of 1.0 ng/l for PFOA using the delayed mammary gland development BMDL and the default adult exposure values used:

$$\text{RfD} = \frac{22{,}900 \text{ ng/L (BMDL)} \times 0.00014 \text{ L/kg/day (clearance factor)}}{30 \text{ UF}} = 0.107 \text{ ng/kg/day}$$

$$\text{MCL} = \frac{0.107 \text{ ng/kg/day (RfD)} \times 70 \text{ kg (adult body wt)} \times 0.2 \text{ (RSC)}}{2 \text{ L/day (adult water intake } 90^{th} \text{ \%)}} = 0.75 \text{ ng/L (round to 1.0 ng/l)}$$

Based on delayed mammary gland development endpoint the PFOA MCL is 1.0 ng/l.

b. Immunotoxicity Endpoint for PFOA - Epidemiologic Studies

Strong, significant epidemiologic evidence that include quantitative data for immune suppression is available to derive PFOA and PFOS BMDLs. The Grandjean and Budtz-Jorgensen study represents the greatest sensitivity to PFOA thus studied, un-confounded by exposure to other chemical contaminants (Grandjean and Budtz-Jorgensen 2013). That study found a strong association between serum PFOA and PFOS concentrations and serum antibody concentrations against tetanus and diphtheria toxoids. Regression modeling of PFOA and PFOS as independent variables along with potential confounders of sex, age, and booster type at age 5 and 7, with antibody concentrations as outcome, determined a benchmark dose (BMD) and response.

Using the Grandjean and Budtz-Jorgensen 2013 BMDL of 0.3 ng/l, a clearance factor of 0.00014 L/kg/day used by NJDWQI to apply to the target human serum level, a UF of 10 for human variation in susceptibility, and the NJDWQI default adult exposure values of 70 kg body weight, 2 L/day water intake, and a relative source contribution of 0.2, we calculated a MCL for PFOA:

$$\text{RfD} = \frac{330 \text{ ng/L} \times .00014 \text{ L/kg/day}}{\text{UF } 10} = 0.0046 \text{ ng/kg/day}$$

$$\text{MCL} = \frac{0.0046 \text{ ng/kg/day} \times 70 \text{ kg} \times 0.2}{2 \text{ L/day}} = 0.032 \text{ ng/L}$$

Based on immunotoxicity endpoint the PFOA MCL is **0.03 ng/L**.

c. Immunotoxicity Endpoint for PFOS - Epidemiologic Studies

Quantitative epidemiologic data for an immune suppression effect should also be taken into account for the PFOS MCL. We use the Grandjean and Budtz-Jørgensen (2013) study BMDL of 1.3 ng/l as the target human serum level, an uncertainty factor of 10 for human variation, a clearance factor of 8.1×10^{-5} L/kg/day derived by USEPA (USEPA 2016b), and adult default exposure values of body weight and water intakes used by NJDWQI to calculate a PFOS MCL as:

$$\text{RfD} = \frac{1.3 \text{ ng/ml} \times 1000 \text{ ml/L} \times 8.1 \times 10^{-5} \text{ L/kg/day}}{\text{UF } 10} = 0.01 \text{ ng/kg/day}$$

$$\text{MCL} = \frac{0.01 \text{ ng/kg/day} \times 70 \text{ kg} \times 0.2 \text{ RSC}}{2 \text{ L/day}} = 0.07 \text{ ng/L (round to 0.1 ng/L)}$$

Based on immunotoxicity endpoint the PFOS MCL is **0.1 ng/L**

d. PFOS Immunotoxicity – Animal Study Uncertainty Factors

The Dong et al 2009 study of decreased plaque forming cell response, predictive of immunotoxicity, resulted in the lowest (most sensitive) point of departure (POD) and was used as the NJDWQI toxicity endpoint for PFOS. We concurred with this assessment but disagree with the UF (uncertainty factor) used to determine the PFOS target human serum level. NJDWQI applied a UF of unity (1.0) for sub-chronic versus chronic testing used in Dong et al 2009 study even though this study of 60 days is of sub-chronic

duration. Sub-chronic duration is > 30 day to ≤ 90 days. A UF of 10 is normally applied when sub-chronic is used instead of chronic testing to estimate a NOAEL.

NJDWQI asserts that an uncertainty factor to extrapolate sub-chronic to chronic is not needed because the immunotoxicity studies of sub-chronic duration did not show a greater effect (response) at longer duration (*but within the sub-chronic duration period*) among the three studies reviewed. NJDWQI notes that for the same PFOS serum concentration of 1 x
10^5 ng/ml, plaque forming cell response decreased by the same 60% in two studies despite the difference in duration between these two studies, (Zheng et al 2009) at 7 days duration and (Dong et al 2009) at 60 days duration. NJDWQI asserts, therefore, that the decrease in plaque forming cell response does not progress at longer exposure duration.

Although suggestive of a lack of progression over time, these tests are of very short duration (7-60 days) and we believe they do not fully explain whether this premise holds true at longer chronic durations of 6 months or more. Further, the mechanistic basis for the immunotoxic effect of PFOS is unknown, and whether further long-term exposures may indeed accelerate this effect.

Omission of a UF for sub-chronic-to-chronic in risk assessments should not be done on the basis of results taken solely from short term studies, especially without an understanding of the mechanism of toxicity. A sub-chronic to chronic UF should be applied. In lieu of some (limited) evidence of no increase in effect in dose-response between the 7-day and 60-day short-term sub-chronic studies we would apply a UF of 3 (versus 10) as a reasonable compromise. Applying a UF for sub-chronic to chronic is important since serum PFOS levels in the general U.S. population are already near the range of central tendency serum PFOS levels in the studies that found associations with decreased immune response (NJDWQI 2017).

We applied a UF of 3 to estimate the NOAEL for chronic testing from sub-chronic testing in the Dong et al 2009 study. An UF_{human} of 10 was used to account for general physiological and metabolic variation within the human population and, in this case, for increased sensitivity in sensitive sub-populations versus the average human population. A UF of 3 was used to account for interspecies (rodent to human) toxicodynamic differences. No UF is needed for toxicokinetic differences since the POD (point of departure), in this case the NOAEL, is based on blood serum PFOS levels. Since individual UFs are as log-units the product of 3 for sub-chronic to chronic x 3 for interspecies toxicodynamic differences is taken as 10. Therefore, the total UF we applied is 100. Using the EPA clearance factor and default adult exposure values of 70 kg adult body weight and 2.0 l/day adult water intake (90^{th} %), the PFOS MCL is:

$$\text{Target Human Serum Level} = \frac{\text{POD (NOAEL) 674 ng/ml}}{\text{UF 100}} = 6.74 \text{ ng/ml}$$

Reference dose (RfD) = 6.74 ng/ml x 1000ml/L x .000081 L/kg/day (clearance) = 0.55 ng/kg/day

$$\text{PFOS MCL} = \frac{\text{0.55 ng/kg/day x 70 kg body weight x 0.2}}{\text{2 L/day}} = 3.85 \text{ ng/L (rounded to 4 ng/l)}$$

Based on the revised UF the PFOS MCL is **4.0 ng/l**

2. Exposure Variables Used in the Risk Assessments

NJDWQI use of adult default exposure values of 70 kg for body weight and 2.0 l/day for water intake results in excessive, unsafe PFOS and PFOA risk to nearly all ages of children. This is especially disconcerting since many epidemiologic studies have shown associations between PFOS and PFOA exposure and health effects in children including adverse effects in serum lipids (high total cholesterol), delayed onset of puberty (associated with altered risk of adult disease: diabetes mellitus, heart disease, bone disease, substance abuse, and asthma), associations between renal function and serum PFC levels, and suppression of vaccine mediated antibody response. These adverse effects on children are fully described with references in our prior submittals. Animal studies have also shown a number of adverse reproductive and developmental effects, including delayed mammary gland development with increased vulnerability to later disease development, as discussed above.

Children have greater vulnerability to environmental contaminant exposures due to their rapidly growing and developing bodies. Children consume more water and food per unit of body weight than do adults. Children's metabolic rates are greater than those of adults. The greater metabolic rates of children may lead to greater doses of chemicals created by metabolic processes (breakdown). Infants and younger children metabolic differences and rates may lead to differential elimination or excretion of absorbed contaminants. For example, undeveloped liver and kidneys of infants are less effective at removing environmental contaminants than adults.

Young children are exposed to differential intakes of PFOS and PFOA because of age-specific behaviors, such as hand-to-mouth behavior, resulting in greater ingestion of house dust and dust on surfaces/products containing perfluorochemicals such as upholstered furniture, clothing, bedding, automobile fabrics, and carpets. Children are burdened with PFOS at birth. "PFOS is distributed within the body and can be transferred from pregnant women to their unborn children and offspring" (USEPA 2016b). PFOS has been quantified in umbilical cord blood, suggesting maternal transfer (Apelberg et al. 2007; Cariou et al. 2015; Tao et al. 2008; Völkel et al. 2008; Von Ehrenstein et al. 2009; USEPA 2016b). PFOS is transferred to infants at breast feeding, offloading the mother's PFOS blood serum levels (Völkel et al. 2008; USEPA 2016b; Mondal et al. 2014; Cariou et al. 2015).
We analyzed 19 children age groups ranging from 2 months through age 17 years to calculate the dose (ng/kg/day) resulting from exposure to the proposed MCL of 13 ng/l for PFOS and MCL of 14 ng/l for PFOA. We used mean body weights and 90th % water intakes for these groups, published in the USEPA Exposure Factors Handbook 2011, consistent with EPA's revised default values of mean body weights and 90th % water intakes for adults 21 years and older (USEPA 2017). We found that the resulting daily dose of PFOS to all children groups from ages 2 months through age 13 exceeded the 1.8 ng/kg/day allowable reference dose (RfD) for PFOS derived by New Jersey. Resulting PFOS doses ranged from 1.9 ng/kg/day at age 13 to 6.63 ng/kg/day at age 1. PFOS doses exceed the RfD by a factor of 1.1 times at age 13 to a factor of 3.7 times at age 1.

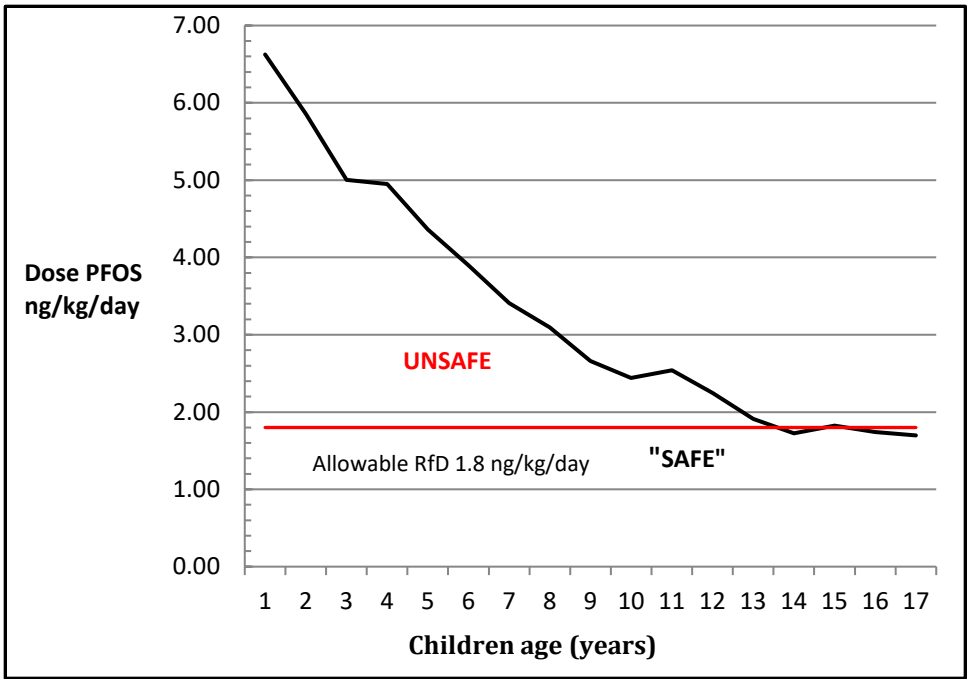
The resulting dose of PFOA to all children groups ages 2 months through age 13 exceeded the allowable RfD of 2.0 ng/kg/day for PFOA derived by New Jersey. Resulting PFOA doses ranged from 2.06 ng/kg/day at age 13 to 7.14 ng/kg/day at age 1. PFOA doses exceeded the RfD by a factor of 1.03 times at age 13 to a factor of 3.57 times at age 1.

Daily doses by children age group at the proposed MCLs are shown in the following table:

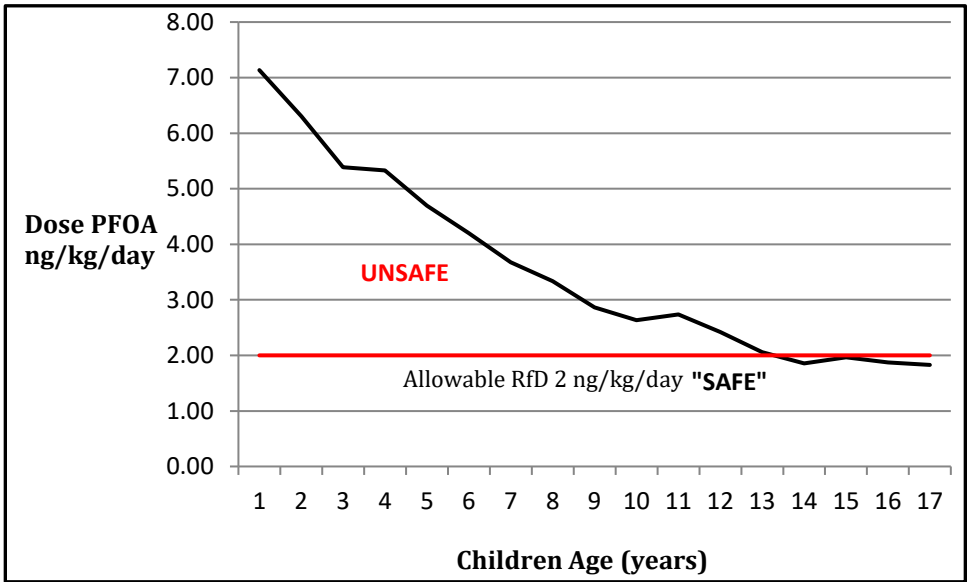
Age	Dose PFOS ng/kg/day at PFOS MCL 13 ng/l	Dose PFOA ng/kg/day at PFOA MCL 14 ng/l	Exceeds Reference Dose (RfD)?
2 to 6 months	5.62	6.05	yes
7 to 12 months	4.76	5.12	yes
1 year	6.63	7.14	yes
2 years	5.86	6.31	yes
3 years	5.00	5.39	yes
4 years	4.95	5.33	yes
5 years	4.36	4.70	yes
6 years	3.90	4.20	yes
7 years	3.41	3.67	yes
8 years	3.10	3.33	yes
9 years	2.66	2.86	yes
10 years	2.44	2.63	yes
11 years	2.54	2.73	yes
12 years	2.25	2.42	yes
13 years	1.91	2.06	yes
Reference Dose (RfD)	**1.8**	**2.0**	
14 years	1.72	1.86	no
15 years	1.82	1.97	no
16 years	1.74	1.88	no
17 years	1.70	1.83	no

Table 1. Daily dose of PFOS and PFOA by Age at Proposed MCLs

The following graphs characterize PFOS and PFOA doses in children age 1 year through age 17. Children 2 months through age 13 may receive PFOA and PFOS daily doses that exceed the allowable RfDs (reference doses) if 14 ng/l and 13 ng/l are established as MCLs for PFOA and PFOS.



Graph 1. Children Daily Dose (ng/kg/day) of PFOS at Proposed MCL 13 ng/l



Graph 2. Children Daily Dose (ng/kg/day) of PFOA at Proposed MCL 14 ng/l

a. MCLs based on Children Exposure Variables (Age Group 1-6)

The proposed MCLs of 13 ng/l for PFOS and 14 ng/l for PFOA do not protect a large segment of the population. Although an uncertainty factor applied for human variability in the risk assessment accounts for variable sensitivity within the broader population, including variability in sensitivity within children sub-populations, appropriate age-specific exposure values for body weights and water intakes should be used to derive MCLs for children subpopulations to enable protection.

We previously recommended that a composite group age 1-6 be used to establish the MCLs. We derive a MCL of 5 ng/l for PFOS using the PFOS RfD of 1.8 ng/kg/day, a body weight of 16.8 kg, and a water intake of 1.19 l/day for children group ages 1-6. For PFOA we calculate a MCL of 6 ng/l using the PFOA RfD of 2

ng/kg/day and the same exposure values for this group.

MCL PFOS = $\frac{1.8 \text{ ng/kg/day} \times 16.8 \text{ kg} \times 0.2 \text{ RSC}}{1.19 \text{ L/day}}$ = **5.08 ng/L (round to 5 ng/L)**

MCL PFOA = $\frac{2.0 \text{ ng/kg/day} \times 16.8 \text{ kg} \times 0.2}{1.19 \text{ L/day}}$ = **5.65 ng/L (rounded to 6 ng/L)**

Mean weight for the age group 1-6 were determined using EPA 2011 Exposure Factor Handbook data for these ages, taking smaller increments of age groups and gender, combined by weighting the means of group increments, and pooling variances to determine means and standard deviations. We determined a 1.19 L/day composite water intake rate for children 1-6 at the 90th percentile, based on the lognormal distribution of water intakes for this combined age group.

PFOA and PFOS MCLs should be set for the most sensitive endpoint. That is epidemiologic studies of immunotoxicity in children by Grandjean and Budtz-Jørgensen (2013), which developed BMDLs for PFOA and PFOS. We calculate MCLs <1.0 ng/l based on immunotoxicity in that study. We propose, therefore, that the MCLs for PFOA and PFOS be set at ≤ 1.0 ng/l.

Absent the establishment of MCLs set at ≤ 1.0 ng/l, the MCL for PFOA should be no greater than 6 ng/l and the MCL for PFOS no greater than 5 ng/l based on appropriate use of age-specific body weights and water intakes for the children age group 1-6.

3. Treatment

The Treatment Subcommittee recommends granular activated carbon (GAC) to remove PFOS and PFOA. The subcommittee states that treatment options are expected to be the same for the long-chain PFAS (perfluroalkyl substances) because of the compounds' similar properties (for example persistence in the environment, water solubility, similar structure, strong-carbon fluorine bonds, and high polarity. We disagree. There are differences in chemical structure and polarity among long-chain perfluoralkyl substances that leads to, at times, significant differences in removal capability by GAC. Specifically, the charged functional group, carboxylic or sulfonic acid, affects the adsorption capability of activated carbon. PFSAs (perfluoroalkyl sulfonic acids, e.g. PFOS) are stronger acids and more hydrophobic and thus more strongly adsorbed onto carbon compared to PFCAs (pefluoroalkyl carboxylic acids, e.g. PFOA, PFBA). Therefore, perfluoroalkyl sulfonic acids (e.g. PFOS) tendency to adsorb onto activated carbon is greater. (We note that persistence in the environment usually has little relationship to treatability by GAC.)
Differences in GAC capability to remove long-chain PFAS, including PFOS and PFOA, and short-chain PFAS, including PFBS and PFBA, has been demonstrated at a number of peer-reviewed research projects and full-scale installations. Reverse osmosis has been found to be superior to GAC in removal of PFOS, PFOA, and other PFAS, including short-chain PFAS such as PFBS and PFBA. Although GAC is generally efficient at removing PFOS, it is less efficient to reliably remove PFOA and is relatively inefficient to remove shorter-chain PFAS such as PFBA, PFBS, PFHxA, and PFPeA.

We previously described some of these studies and briefly summarize here:

A study by Du et al. (2014) found that carbon sorption capacities of PFSAs (e.g. PFOS) onto activated carbon or other adsorbents were higher than PFCAs (e.g. PFOA) with the same carbon numbers due to

greater hydrophobicity of PFSAs versus PFCAs (Du et al. 2014).

Activated column experiments by Ostlund (2015) found higher removal efficiency of PFSAs than PFCAs, comparing the same number of carbons in the perfluorocarbon chain length, indicating that that functional group (carboxylic or sulfonic) affects removal efficiencies of PFASs; “sulfonic group resulted in higher removal efficiency compared to carboxylic group” (Ostlund 2015).

The Water Research Foundation (WRF) study of 15 full-scale water treatment systems in the U.S., including two potable reuse treatment systems, found that full-scale anion exchange and GAC column treatments were more effective at removing long-chain PFSAs (e.g. PFOS) than PFCAs (e.g PFOA, PFBA) (Water Research Foundation 2016). This study also found that full-scale reverse osmosis systems demonstrated significant removal for all perfluorinated compounds, including the smallest, perfluorobutanoic acid (PFBA). The WRF study further evaluated nanofiltration (NF) for removal of PFCAs and PFSAs and noted that NF “has been deemed potentially effective (> 95%) in bench-scale experiments using NF270 membranes” (WRF 2016; Steinle-Darling and Reinhard 2008). WRF (2016) indicated that NF may be as capable of rejecting (treating) perfluorinated compounds as reverse osmosis at lower cost.

PFOS was found to be strongly adsorbed by GAC in a study of sorption onto GAC, zeolite, and sludge, but PFOA and PFBS were removed by GAC but to a lesser extent. The authors noted “that the length of the fluorocarbon chain and the nature of the functional group influenced sorption of the anionic surfactants" (Ochoa-Herrera and Reyes-Sierra 2008).

PFOS and PFOA were removed to low ppt levels using reverse osmosis at two water reclamation plants (treating domestic effluents as influent) in Southeast Queensland, Australia. PFOA was removed to less than 1.4 ng/L, from influent levels ranging from 15 to 27 ng/L, and PFOS was removed to less than reporting with influent PFOS levels ranging from 23 to 39 ng/L. However, in the treatment plant using biologically activated carbon, PFOA and PFOS were ineffectively removed (Thompson et al., 2011). (It should be noted that poor PFOS and PFOA removals via the carbon system may have been related to carbon age or contact time.)

PFOA was not effectively removed at a water treatment plant in Amsterdam using GAC. The authors found greater removals of PFOS and PFNA. This study also found that PFBA, PFPeA, PFHxA, PFOA, and PFBS were not well removed by the GAC system. In general, the authors found that PFOA decreased by only 50% using GAC (Eschauzier et al. 2012).

A study in Spain suggests that although GAC alone was reasonably effective to remove PFOS, reverse osmosis was needed to achieve efficient PFOA removal. In this study 2 separate stages following conventional water treatment (GAC, or Ultrafiltration followed by Reverse Osmosis) were evaluated. The system supplies 100 million gallons per day drinking water to 1 million inhabitants. Ultrafiltration and reverse osmosis removed PFOS and PFOA by ≥ 99%, but GAC removed PFOS and PFOA by only 64 ± 11% and 45 ± 19%, respectively (Flores et al. 2013).

In a study monitoring drinking water treatment facilities across the U.S., a utility that used microfiltration and reverse osmosis for indirect potable reuse in wastewater treatment reduced total perfluorinated compound influent levels of 80 ng/L and influent PFOS of 41 ± 18 ng/L to no reportable levels. Minimum reporting levels were 1.0 ng/L for all perfluorinated compounds monitored except PFOA, where the

minimum reporting level was 5 ng/L (Quinones and Snyder 2009).

The Northwest Water Treatment Plant of Brunswick County, North Carolina studied treatment technologies including GAC, ion exchange, reverse osmosis, ozone with biofiltration, and UV/AOP to remove organic chemicals including PFAS in its drinking water supply source, the Cape Fear River. The water treatment plant will supply about 36 million gallons per day. The study found that reverse osmosis was the best technology for removal of PFAS, and was also the most robust technology for protecting against unidentified contaminants (CDM Smith 2018).

PFOS and PFOA do not occur in isolation in drinking water supplies. Other PFAS frequently co-occur with PFOS and PFOA and include PFNA, PFHxS, PFBS, PFHpA, and PFBA, among others. Focusing on treatment or treatability of only PFOS and PFOA overlooks the complexity of PFAS compounds present in most water supplies, their potential interactions, and overall toxicity.

Short chain PFAS, such as PFBA and PFBS are highly persistent and very mobile in water. PFBS and PFBA often co-occur with PFOS and PFOA in PFAS contaminated water supplies. PFBA and PFBS are poorly removed, or not removed at all, through GAC systems. For example, PFBA passes through untreated at relatively high levels at the City of Oakdale, Minnesota GAC plant, which treats PFOS and PFOA in PFAS contaminated drinking water.

Little research is available on the toxic effects related to exposure to short chain PFAS. However, emerging studies suggests toxicity with some short-chain PFAS. In its draft toxicity assessment of two GenX chemicals (hexafluoropropylene oxide (HFPO) dimer acid and its ammonium salt) and PFBS, the EPA noted that PFBS is associated with thyroid and kidney effects (USEPA Nov 2018). Decreases in thyroid hormone were accompanied by other effects indicative of delayed maturation or reproductive development (e.g., vaginal patency and eyes opening).
It is highly likely that a number of PFAS co-exist in affected New Jersey water supplies. In addition, as acknowledged by NJDWQI, the potential for additive toxicity of PFOS and PFOA and other PFAS was not considered in development of the MCLs. Although GAC adequately removes PFOS, PFOA is at times marginally removed, and other PFAS may be poorly removed. Reverse osmosis offers the most robust technology to consistently remove all PFAS potentially present, including PFOA and short chain PFAS (PFBA and PFBS), as well as unidentified contaminants in the drinking water. Reverse osmosis or equivalent high pressure membrane technology (such as nanofiltration) should be required as Best Available Technology to remove PFAS in New Jersey drinking water supplies and in discharges to ground water. In some cases pre-treatment with GAC may be appropriate to reduce the mass load of PFAs in the reverse osmosis reject, depending upon the reject discharge location.

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Don Kriens **Curriculum Vitae**

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AREAS OF EXPERTISE

- Professional engineer - range of civil and environmental engineering projects, and design.
- Exposure and risk assessments for human health.
- Project manager - toxic contaminant cleanup projects.
- Design of water/wastewater treatment systems, hydro-geologic studies, remediation projects, stormwater control, and hazardous waste cleanups (Superfund).
- Industrial technologies and processes, pollution prevention, industrial process chemistry, and application of emerging treatment technologies to industries.
- HAZMAT trained.
- Regulatory enforcement, civil and criminal. Skilled in technical writing and presentation, and negotiation. Knowledge of federal and state environmental regulatory programs.
- Global water scarcity problems, environmental policy and justice, climate change impacts, energy, and engineering economic analysis.
- Modeling exposure and risk of chemicals, including disinfection byproducts and contaminants in drinking water supplies.

EDUCATION

HARVARD UNIVERSITY, Cambridge, MA

Sc.D. Environmental Health

Concentration - Exposure Sciences

HARVARD UNIVERSITY, Cambridge, MA

M.S. Environmental Health

UNIVERSITY OF IOWA, Iowa City, Iowa.

M.S. Environmental Engineering

UNIVERSITY OF IOWA, Iowa City, Iowa.

B.S. Sciences

AWARDS

Bush Foundation Leadership Fellow 2008

U.S. EPA Civil and Criminal Investigation Award

Harvard University Andelot Scholarship

Harvard University Water Initiative Fellow

PROFESSIONAL EXPERIENCE

1978-2008 MINNESOTA POLLUTION CONTROL AGENCY, St. Paul, MN

Principal Engineer

- Lead agency technical expert for water projects. Mentor to engineers, hydro-geologists, and other technical staff.
- Research projects to assess ecological and health impacts of contaminants. Evaluated emerging technologies to resolve pollution problems.
- Conducted major civil and criminal environmental investigations with MN Attorney General staff, U.S. Attorney's Office, USEPA Region V. Expert witness.
- Developed major industrial environmental permits, determined technologies required to comply. Assessed economic impact of regulations.
- Technical expert for water/wastewater treatment, remediation and hazardous waste, water supplies.
- Technical expert for emergency response regarding toxics and resolution. Project manager and/or engineer for remediation of various toxic waste sites.

1996-2008 Kriens Engineering, Oakdale, MN

Consulting Engineer and Owner

- Design of Individual Sewage Treatment Systems. Groundwater (well) analysis and water consulting.

Castek Consulting Engineering Services

Engineer

- Operation, design, and process chemistry evaluations of wastewater treatment plants; air pollution studies; indoor air quality assessments.

TEACHING EXPERIENCE

Harvard University

- Teaching Assistant in water pollution and risk assessment. Lecturer in water scarcity at Harvard Extension School.

Kirkwood Community College, Cedar Rapids, Iowa

- Instructor; wrote courses in chemistry/advanced chemistry of wastewater treatment.

University of Iowa Department of Civil and Environmental Engineering, Iowa City, Iowa

Research Scientist and Environmental Engineering Laboratory Supervisor

- Supervised laboratory conducting biological and chemical analyses, including GC and GC/MS. Conducted field studies. Occasional teaching assistant.

LICENSES AND PROFESSIONAL AFFLILIATIONS

- Registered Professional Engineer
- Individual Sewage Treatment System Designer (Minnesota)
- Certification in air quality inspections (California Air Resources Board)
- Certification in Stormwater Treatment and Erosion Design
- Member, Minnesota Government Engineers Council
- Member, Society of Professional Engineers

PAPERS AND PUBLICATIONS

Listing available on request

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PROFILE

- Accomplished scientist with years of experience in creating innovative solutions to challenging environmental problems related to public health, policy development and environmental sustainability.
- Experienced project manager with skills in the application of analytical methods and techniques necessary for working within the framework of state/federal environmental and public health organizations.
- Registered independent consultant in the UNEP and UNIDO experts' roster for U-POPs and New-POPs and implementation of the Stockholm Convention on Persistent Organic Pollutants.
- Rigorous researcher and team leader experienced in spearheading all phases of (planning, budgeting, developing, conducting, and directing) of environmental project management.
- Effective communicator with ability to translate complex scientific data into coherent material in order to inform audiences with varying degrees of knowledge about environmental issues.
- Conscientious professional with experience presenting expert witness testimony in litigation cases involving a wide range of environmental problems and related public health issues.
- Experienced college instructor developing and teaching natural sciences and environmental science and public health policy courses.

EDUCATION

Harvard University School of Public Health, Boston, MA

Audited several courses: Air Pollution; Water Pollution; and Risk Assessment

Harvard University John F. Kennedy School of Government, Cambridge, MA

Master in Public Administration

Concentration*:* Leadership and International Env. Health Policy and Management

Western Michigan University, Kalamazoo, MI

PhD in Environmental Sciences

- Dissertation title: Acid Rain and Lake Acidification Impacts on Aquatic Life

MS in Biology

- Thesis title: Drinking Water Quality and Waterborne Diseases in Rural Iran

National University of Iran, Tehran, Iran

BS Chemistry, Minor Biology

PROFESSIONAL EXPERIENCE

Cambridge Environmental Consulting, LLC., Boston, MA 2006 - Present

Senior Scientist and President

- "Visiting Professor" at the Iranian National Institute of Oceanography (INIO) - conducted training workshops for INIO staff/scientist and coastal management professionals on the policy aspects of coastal zone management and its implications. The training was tailored to the local cultural characteristics, government structure, resource integrity, and management needs of the country (2012).
- Invited by the Iranian Governor's Officials to visit and evaluate the environmental impacts of a historically contaminated site caused by the largest landfill located near the Caspian Sea. Developed an integrated solid waste management plan for implementation, including an assessment of all environmental risks, and the development of mitigation efforts required to minimize the adverse impacts on Public health and the environment (2012).
- Participated and presented two papers at Dioxin 2010 - 30th International Symposium on Halogenated Persistent Organic Pollutants (POPs) on 1) Presence of PBDEs in Minnesota Landfills – Environmental Releases and Exposure Potential, and 2) Investigation of PFOS/PFCs Contamination from a PFC Manufacturing Facility in Minnesota – Environmental Releases and Exposure Risks (2010).
- Chaired the "New POPs" Section (Implication of Stockholm Convention of New POPs) of the11th International HCH and Pesticide Forum, Cabala, Azerbaijan (2012).
- Serve as expert witness in environmental litigation pertaining to release of industrial toxic contaminants.
- Conduct evaluations of toxic contaminants (including New POPs) and use dispersion modeling (groundwater, surface water, soils and air) to evaluate contaminants' environmental impacts and public health risks.
- Review and evaluate EPA documents related to the issuance of new source National Pollutant Discharge Elimination System (NPDES) permits to industrial activities.

Women's Environmental Institute (WEI), St. Paul, MN 2006 - 2012

Principal Scientific Consultant

- Served as a WEI Board Member and later, as the principal scientific consultant, developed environmental justice education program to promote environmental awareness, sustainability, and health disparity.
- Directed and managed projects on environmental issues related to public health and environmental quality.
- Analyzed the effectiveness and efficiency of existing environmental and public health programs for the implementation and administration of programs best fit the affected communities. Identified and presented to public policy makers the problems affecting concerned communities.
- Evaluated the impact of toxic pollutants on the growth and development of exposed children. Developed multimedia outreach programs to inform families about toxic exposure and consequences.
- Developed culturally specific environmental training and educational seminars for exposed communities through different radio stations and newspapers.

Mote Marine Laboratory, Sarasota, FL 2007- 2008

Associate Scientist

- Designed health risk assessment framework to evaluate potential exposure pathways and toxicity effects of contaminants in Florida manatees. Contributed to development of research proposals.
- Evaluated public and environmental regulatory policies and proposed effective mitigation tools

Minnesota Pollution Control Agency (MPCA), St. Paul, MN 1989 - 2006

Senior Scientist, Project Manager, and Emerging Contaminants Program Coordinator

- Developed policy, program analysis methods, and multimedia strategy to assess health impact of toxic chemicals.
- Initiated and led the Emerging Contaminants Program for the competent authority (MPCA).
- Prepared Environmental Impact Assessments (EIS) for major projects in MN and communicated the results, including the potential social, and economic impacts of these projects with authorities and public.
- Represented the MPCA as a scientific expert, liaison, and critical state contact in the PCBs, Dioxin, and emerging contaminants activities of the US EPA, Great Lakes Binational Strategy (GLBNS) and in other related national and international programs.
- Worked closely with diverse array of clientele and stakeholders (federal and state governments, industry,

grass root organizations, affected communities, and the state legislators) to develop progressive environmental policies and educational materials.

- Presented at international conferences and gave presentations regarding environmental issues in public meetings, legislative hearings and governmental agencies.
- Managed contracts and secured federal/state grants and awards for health impacts of contaminant in Minnesota.
- Developed statewide air toxics monitoring/bio-monitoring network using mass balance and integrated air exposure-effect models.
- As the technical coordinator and MPCA liaison, built partnership between PCA and other sister agencies (MN Department of Health, MN Department of Natural Resources, and MN Department of Agriculture), USA EPA, and MN university researchers for ongoing efforts to identify, evaluate, control, regulate, and reduce the emerging pollutants with endocrine disruptive characteristics (PFOS and PFOA, PBDEs, and pharmaceuticals).
- Assessed the current regulations and programs already in place that may be addressing reduction of toxic contaminants of concern, identified unregulated emerging contaminants of greatest potential risk to human health and the MN environment, rationale of why these contaminants need to be regulated.

TEACHING EXPERIENCE

Teach biology, chemistry, environmental science, health and policy-related courses (Elements of Health and Wellness, Foundations of Research, Public Policy Planning and Implementation), part-time at:

• **University of Phoenix** – Adjunct Faculty	Boston, MA	2010 - Present
• **Regis College** – Adjunct Professor	Weston, MA	2012 - 2013
• **Hamline University –** Adjunct Assistant Professor	St. Paul, MN	2002 - 2003
• **St. Paul College –** Adjunct Assistant Professor	St. Paul, MN	1998 - 2002
• **Inver Hills Community College** – Adjunct Faculty	St. Paul, MN	1996 - 2002
• **Minnesota Department of Corrections**	Various locations	1998 - 2000
• **Normandale Community College** – Adjunct Faculty	Bloomington, MN	1990 - 1998
• **Northland College** – Assistant Professor	Ashland, WI	1986 - 1989
• **Western Michigan University** – Teaching Assistant	Kalamazoo, MI	1980 - 1985

PROFESSIONAL AFFILIATIONS

- Member, **PCB Elimination Network (PEN)** of the Stockholm Convention 2011 - Present
- Member, **Society of Environmental Toxicology and Chemistry** 1990 - Present
- Member, Board of Directors, **Women's Environmental Institute** 2003 - Present
- Member, **Aquatic Biogeochemistry Research Group,** Harvard University, Harvard School of Public Health (HSPH) 2010 - 2012
- Member, **American Chemical Society** 1992 - 2010
- Member, **Air and Waste Management Association** 1998 - 2010

LANGUAGE SKILLS

- Fluent in English and Farsi (Persian)

PUBLICATIONS

- Brambilla, G., d'Hollander, W. Oliaei, F., Stahl, T., and Weber, R. Pathways and factors for food safety and food security at PFOS contaminated sites within a problem based learning approach, Accepted for publication at Chemosphere, 2014.
- Oliaei, F., Weber, R., Watson, A., and Kriens, D. Review of Environmental Releases and Exposure Risk of PFOS/PFAS Contamination from a PFOS Production Plant in Minnesota. Environmental Science and Pollution Research, 2013.
- Oliaei, F., Weber, R., and Watson, A. Landfills and Wastewater Treatment Plants as Sources and Reservoir of Polybrominated Diphenyl Ether (PBDE) Contamination. Environmental Science and Pollution Research, 2012.
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- Oliaei, F., and Kriens, D. *Environmental Releases of Perfluoroalkyl compounds from Two Landfills at the PFOS/PFC Production Site in Minnesota.* EPA – PFAA Day III, 2010.
- Oliaei, F., and Kriens, D. *Discovery of PFOS/PFC Contamination in Fish Near a PFOS/PFC Manufacturing Plant in Minnesota.* EPA – PFAA Day III, 2010.
- Oliaei, F., Kriens, D., and Kessler, K. *Perfluorochemical (PFC) Investigation in Minnesota: Phase One.* Minnesota Pollution Control Agency (MPCA). Legislative Report 2006. (79 pages).
- Oliaei, Fardin. *The presence and Distribution of Perfluorochemicals (PFCs) in Minnesota.* The EPA, Federal-State Toxicology and Risk Analysis Committee Meeting (FSTRAC), 2005.
- Oliaei, Fardin. *Flame Retardant: Polybrominated Diphenyl Ethers (PBDEs) in Minnesota.* Minnesota Pollution Control Agency (MPCA). Legislative Report 2005. (34 pages).
- Oliaei, Fardin. *The Presence and Distribution of PBDEs in MN's Landfills, Wastewaters and the Environment.* Minnesota Pollution Control Agency (MPCA). Annual Report of the Closed Landfill Program (CLP). 2004
- Oliaei, F., and Hamilton, C. *PBDE congener profiles in fish with different feeding behaviors from major rivers in Minnesota.* Organohalogen Comp. 64, 356-359, 2003.
- Oliaei, F., King, P., and Phillips, L. *Occurrence and Concentrations of Polybrominated Diphenyl Ethers (PBDEs) in Minnesota Environment.* Organohalogen Comp. 58, 185-188, 2002.
- Pratt, G., Oliaei, F., Wu, C., Palmer, K., and Fenske, M. *An Assessment of Air Toxics in Minnesota.* Environmental Health Perspective. 108(9), 815-825, 2002.
- Oliaei, Fardin. *Flame Retardants: Persistent, Bioaccumulative and Toxic Chemicals.* The EPA, Federal-State Toxicology and Risk Analysis Committee Meeting (FSTRAC). 2000.
- Oliaei, Fardin. *Toxic Air Pollutant Update.* Minnesota Pollution Control Agency (MPCA). 1999.
- Oliaei, Fardin. *Minnesota Air: Air Quality and Emissions Trends.* Minnesota Pollution Control Agency (MPCA). 1997, (215 pages).
- Pratt G., Gerbec, P., Livingston S., Oliaei F., Bollweg G., Paterson S., and Mackay D. *An indexing system for comparing toxic air pollutants based upon their potential environmental impacts.* Chemosphere 27(8), 1359-1379, 1993.

Verbal Testimony
Tracy Carluccio, Delaware Riverkeeper Network
NJ DEP Drinking Water Standards
and other Regulatory Proposals re. PFOA and PFOS
Docket 02-19-03
May 15, 2019

Delaware Riverkeeper Network supports the proposed rulemaking package that will require through the adoption of a safe drinking water standard or maximum contaminant level (MCL) the removal of Perfluorooctanoic Acid (PFOA) and perfluorooctanesulfonic acid (PFOS) from drinking water. The proposed regulatory changes will, in addition to providing safe drinking water, aid in the identification of PFAS contaminated locations and the remediation of sites contaminated with PFOA, PFOS and perfluorononanoic acid (PFNA). We consider this to be of great importance for the people of New Jersey and the state's environment. We know these compounds are highly toxic, do not break down in the environment, are water soluble and mobile, build up in human blood, and are known to be connected to serious negative health effects, including cancer. While, due to its high toxicity and extremely elevated concentration in communities, particularly in the Delaware River Watershed, an MCL was adopted for PFNA last year, this rulemaking has been years in the making for PFOA and PFOS, the two most prevalent of the poly- and perfluoroalkyl substances (PFAS) found in New Jersey.

We strongly support the addition of PFOA, PFOS, and PFNA to the list of contaminants that must be tested for by private well owners under the Private Well Testing Act. Under the Act, treatment is not required for private wells but point of entry treatment systems are available and effective in removing these compounds and DRN encourages all well owners to provide treatment if these compounds are present. We also fully support that nonpublic water systems be tested for PFOA, PFOS, and PFNA and that treatment be provided if the compounds are present. It is essential that all people are equally protected from these compounds in their drinking water, which is why we objected to PFNA not being included in the rulemaking for the MCL last year and we appreciate its inclusion now. Anything less would be unjust.

Regarding the sampling schedule for PFOA and PFOS, we advocate that sampling of public water systems start earlier than 2021. Since PFNA sampling began in January 2019, labs have already had time to purchase equipment and training so we do not support an entire year of delay and advocate that PFOA and PFOS sampling begin in the first quarter after adoption of the rule. People have been exposed far too long to these toxic compounds that build up in human blood, even from tiny amounts in drinking water, increasing risk of disease; there is no good reason to allow exposure to continue after adoption.

Discharges to Petroleum and Other Hazardous Substances Rules –
DRN supports the proposal to add PFOA, PFOS, (as acids, anions, salts, and esters) and the anionic form of PFNA to the Hazardous Substances List under the Spill Compensation and Control Act. This listing will require stricter handling to prevent discharges, impose strict liability for timely investigation and cleanup, and connect to a Spill Fund that is used for remediation,

cleanup, and removal, among other actions. Public water systems as well as private and noncommunity water wells are able to seek reimbursement for remediation costs under this Fund.

Groundwater Quality Standards –
We consider PFOA and PFOS groundwater standards to be essential to protect drinking water sources and to provide minimum remediation standards for cleanup of contaminated sites but we advocate for stricter standards. Based on an independent toxicological analysis that we submitted to DEP on the interim specific groundwater criteria, DRN urges that the proposed standard be lowered to no more than 5ppt for PFOS. The report points out that even at very low doses, early life exposure of children to PFOS may affect risk for disease later in life. For PFOA, we urge that the proposed standard be lowered to 1ppt, or alternatively, should be no higher than 6ppt. This is recommended in the report we commissioned and submitted to DEP because endpoints that are more sensitive can be utilized to develop an MCL, providing greater protection. We also advocate for no higher than 11ppt combined for PFOA and PFOS when both are present.

New Jersey Pollution Discharge Elimination System (NJPDES) –
DRN considers it very important that groundwater is monitored for PFOA, PFOS, and PFNA in discharges. We advocate that all NJPDES permits be modified now to require this, considering the many locations across the state where these compounds have been found and the likelihood of more contaminated sites being found due to the permanent legacy of pollution and mobility of these compounds in water that signifies these highly toxic PFAS compounds.

We also urge DEP to regulate PFOA, PFOS, and PFNA under Surface Water Quality regulations to provide restrictions on the discharge of these pollutants. Such changes to the NJ Surface Water Quality Standards is not being proposed by DEP in this rulemaking but we advocate that they should be. Soil, sediment and air must also be monitored and releases from those media must be controlled. PFAS compounds have been moved around the state, sometimes far from the original source of contamination, by discharges from treatment facilities, sewage plants, and sewage sludge and biosolids applications on agricultural fields. Unless the pathways of pollution are found and closed off through monitoring all potential media, these “forever chemicals” that will never go away through natural processes will continue to fan out into the environment, threatening all people in New Jersey, even those who may be remote from the original contamination.

New Jersey has a huge PFAS problem with sampling showing it has been found more frequently in New Jersey’s drinking water than other states. 224 of NJ’s public water systems have been sampled as of the start of 2019. DEP reports that 506 public community water systems and 715 public nontransient noncommunity water systems will have to monitor for PFOA and PFOS. In other words, most of New Jersey’s drinking water has not been tested. That means that many New Jerseyans could still be drinking water contaminated with PFAS and they do not even know it. The urgency of this issue cannot be exaggerated. DEP has finally come to this moment with all the information about occurrence, monitoring and treatment technologies, health effects, and exposure pathways that is needed, provide by nationally recognized top caliber scientists in NJ’s emply, and far too much time has passed during which people, including our most vulnerable populations, have been exposed to the risk of developing debilitating diseases linked to these compounds.

DEP's rulemaking, with DRN's recommended changes, should be adopted immediately. If the rulemaking would need to be re-published in order to reduce the MCLs to DRN's recommended standards, DRN recommends that the DEP's proposed regulatory package is adopted as proposed and a re-proposal issued immediately after adoption to lower the MCLs for PFOS and PFOS and related regulations to DRN's recommendations.

Thank you for the opportunity to comment on this important regulatory package.

NOTE: Final enclosure is the "Excel worksheet, children intakes, Cambridge Environmental Consulting, May 28, 2019", which is attached separately since it is an excel sheet and not a WORD document.