January 31, 2018

watersupply@dep.nj.gov
New Jersey Department of Environmental Protection
Trenton, New Jersey

Re: Health-Based Maximum Contaminant Level Support Document: Perfluorooctane Sulfonate (PFOS) 11 (CAS #: 1763-23-1; Chemical Formula: C8HF17O3S)

Please find enclosed a technical analysis prepared by Fardin Oliaei, MPA, PhD, and Don Kriens, Sc.D., P.E. of Cambridge Environmental Consulting commissioned by Delaware Riverkeeper Network and submitted on behalf of this organization and its membership regarding the Support Document and recommendation by the New Jersey Drinking Water Quality Institute (NJDWQI) for a **Health-Based Maximum Contaminant Level for Perfluorooctane Sulfonate (PFOS)**. Also attached are two PDFs containing the Curriculum Vitae for Dr. Oliaei and for Don Kriens, Sc.D., P.E.

Delaware Riverkeeper Network submits these comments advocating that the public be protected from 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.

We support all the recommendations and findings made by Dr. Oliaei and Don Kriens of Cambridge Environmental Consulting in this technical analysis. We advocate that an appropriately protective MCL be recommended to and acted upon by the New Jersey Department of Environmental Protection and agree with Cambridge Environmental Consulting's finding that that the NJDWQI's recommended drinking water MCL of 13 ng/L for PFOS is not adequately protective of all population segments. We support Cambridge Environmental Consulting's position that the standard should be calculated based on children's exposure values. We agree that it is of utmost importance to assure protection of children's health since the developing fetus, infants, and young children are particularly sensitive to PFOS exposure during early sensitive periods. As explained by Dr. Oliaei and Don Kriens, even at very low doses, early life exposure of children to PFOS may affect risk for disease later in life. **We support Cambridge Environmental Consulting's analysis and final conclusion that the recommended MCL should be lowered to 5 ng/L.**

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We also support Cambridge Environmental Consulting's conclusion that when found combined with PFOA in water, the combination of PFOS and PFOA concentrations in water supplies should be no higher than 13 ng/L.

Thank you for proposing a recommended MCL for PFOS, an action that is critically needed to remove this toxic compound from New Jersey's drinking water supplies.

Sincerely,

Maya van Rossum
the Delaware Riverkeeper

Tracy Carluccio
Deputy Director

Attached: **Technical Analysis of New Jersey's Proposed Health-Based Maximum Contaminant Level (MCL) for Perfluorooctane Sulfonate (PFOS)**, Fardin Oliaei, Ph.D. and Don L. Kriens, Sc.D., Cambridge Environmental Consulting, submitted January 2018.

Technical Analysis of New Jersey's Proposed Health-Based Maximum Contaminant Level (MCL) for Perfluorooctane Sulfonate (PFOS)

by

Fardin Oliaei, Ph.D. and Don L. Kriens, Sc.D.
Cambridge Environmental Consulting
submitted January 2018

Executive Summary

The RfD (reference dose) for PFOS (perfuorooctane sulfonate) derived by the NJDWQI (New Jersey Drinking Water Quality Institute) is the most stringent and protective in the U.S., based on a rigorous analysis of all available PFOS animal and epidemiologic toxicological studies. However, in its use of adult default exposure values to determine a maximum contaminant level (MCL), younger children would not be protected since younger children dose intakes would exceed the allowable RfD. This is disconcerting since existing PFOS serum levels in children in the normal population are already within or near the serum PFOS levels associated with immunotoxic effects found in epidemiologic studies. In addition, other toxic effects found associated with children and PFOS exposure may lead to increased potential for later disease manifestation. It is essential, therefore, that the NJDWQI depart from the typical use of adult default exposure values and use children's values. Using appropriate children exposure values, we recommend a ***MCL for PFOS of 5 ng/L***.

Alternatively, we recommend a MCL such that the combination of PFOS and PFOA concentrations in water supplies be no higher than 13 ng/L, ***MCL [PFOA + PFOS] ≤ 13 ng/l.***

Introduction

The presence of PFOS and PFOA (perfluoro-octanoic acid) in New Jersey is of public health concern because relatively high concentrations have been found in public water supplies (PWS), in some instances at levels greater than the existing 14 ng/l MCL for PFOA and the proposed 13 ng/l MCL for PFOS. Ten of 80 PWS tested by the New Jersey DEP (Department of Environmental Protection) had PFOS at levels ≥ 20 ng/l with 7 PWS containing PFOS at levels exceeding 40 ng/l (NJDEP 2017). Testing of 175 New Jersey PWS, including 165 large community systems and 10 small community systems completed under the UCMR3, found 6 PWS with PFOS levels exceeding 40 ng/L and 18 PWS with PFOA levels exceeding 20 ng/L (UCMR3 2017, NJDWQI 2017). Ingestion of any level of PFOS and PFOA contaminated water could further increase residents' PFOS blood serum levels beyond that already found in epidemiologic studies to be associated with adverse health effects (immunotoxicity) in the general U.S. population.

A large number of animal studies indicate a wide range of PFOS toxicity effects. These include decreased body weight, increased liver weight with microscopic lesions in monkeys and rats, increased incidence of hepatocellular adenomas in rats, developmental effects on rats and mice including neonatal mortality, decreased gestation length, lower birth weights, and developmental delays. For post-gestation and lactational exposure these include: developmental neurotoxicity, changes in thyroid and reproductive hormones, altered lipid and glucose metabolism, and decreased immune function (USEPA 2016b).

Among non-cancer endpoints the NJDWQI concludes that, for adult animals, the most sensitive endpoints (lowest LOAELs based on serum PFOS concentrations) are described in 9 studies and include: "endocrine/metabolic effects (e.g., decreases in thyroid hormone and increased incidence of thyroid follicular cell adenomas), changes in immune parameters (e.g., increased relative number of macrophages and decreased plaque forming cell response), and increased liver weight and liver histopathology" (NJDWQI 2017). For post-natal or gestational exposures, NJDWQI concludes the most sensitive non-cancer endpoints (lowest LOAELs based on serum PFOS concentrations) are described in 11 studies and include: "decreased body weight, changes in endocrine/metabolic parameters (i.e., fasting levels of serum glucose and insulin, markers of insulin resistance, and thyroid hormone levels), increased liver weight, changes in lung morphology, and increased mortality" (NJDWQI 2017).

NJDWQI examined these 20 toxic endpoints in terms of the timing of biological significance and suitability for dose-response analysis, and determined 4 endpoints suitable to calculate a (POD) point of departure. The immunotoxic effect shown in the Dong et al. (2009) study was chosen as the most sensitive POD (point of departure) at 674 ng/ml.

The immunotoxic endpoint chosen to develop an MCL based on decreased plaque forming cell response, a predictor of immunosuppression, in animal studies is supported by epidemiologic studies that found associations between PFOS and PFOA blood serum levels in humans and decreases in immune function.

A study by Grandjean and Budtz-Jørgensen (2013) found an association between increases in serum PFOS and PFOA levels and decreases in serum antibody concentrations against tetanus and diphtheria toxoids. In this study regression modeling of PFOA and PFOS concentrations as independent variables along with potential confounders of sex, age, and booster type at age 5 and 7, with antibody concentrations as outcome, allowed determination of benchmark response (BMR) and benchmark dose (BMD). The lower one-sided 95% confidence limit of the BMD, the BMDL (benchmark dose level), was determined in this study to be approximately 0.33 ng/ml for PFOA and 1.3 ng/ml for PFOS, based on the linear slope model of the regression. The study notes strong correlation between PFOS and PFOA, making mutual adjustment in the regression difficult. However, the BMDL developed provides a strong epidemiologic basis using an immunosuppression endpoint to develop a MCL.

A study in Norway evaluated the effect of prenatal exposure to PFAS (perfluoroalkyl substances) on responses to pediatric vaccines and immune-related health outcomes in

children up to 3 years of age (Granum et al. 2013). In this study blood samples were taken from the mothers at time of delivery and children at 3 years age. The study found an inverse association between the level of anti-rubella antibodies in the children's serum at age 3 years and the concentrations of four PFAS compounds (PFOS, PFOA, PFNA, and PFHxS). The strength of the association between rubella antibody-levels and PFAS concentrations were PFNA>PFOA>PFH_xS>PFOS.

A cross-sectional study of 1191 children 12-19 years old using NHANES data (1999-2000 and 2003-2004) found that increased exposure to several PFAS compounds was associated with lower levels of mumps and rubella antibody concentrations, especially among seropositive individuals (Stein et al. 2016). This study found that a doubling of PFOS serum concentration was associated with a 7.4% (95% CI: -12.8, -1.7) decrease in mumps antibodies. A doubling of PFOS serum concentration was also associated with a 13.3% decrease in rubella antibodies; this association occurred among seropositive individuals. Decreases in rubella antibodies were also found to be associated with PFOA and PFH_xS. The authors found that children with higher PFOS levels were less likely to be sensitized to allergens, but children with higher PFOS levels were more likely to be sensitized to mold (IQR OR 1.33, 95% CI: 1.06, 1.69).

A small study in Denmark evaluated reduced antibody responses to perfluorochemical exposures (Kielsen et al. 20016). Twelve adults were boosted with tetanus and diphtheria toxoids with antibody responses and followed in a subsequent one-month period. Participants had been vaccinated with diphtheria and tetanus toxoids in childhood, but no boosters had been received within the last 5 years. The study found that at a doubling of PFOS exposure, based on serum obtained from participants 10 days post-vaccination, the relative increase in the diphtheria antibody concentration decreased by an average of about 12%, $p = 0.044$. Significant diptheria antibody concentration reductions were also found for PFNA, PFDA, PFU_nDA, and PFD_oDA.

A study of 411 adults in the mid Ohio region of Ohio and West Virginia, where the drinking water supply had been contaminated with PFOA (Dupont plant), evaluated antibody response and PFOS and PFOA levels in blood serum following vaccination with influenza vaccine. The study found that elevated PFOA serum concentrations were "associated with reduced antibody titer rise, particularly to A/H3N2 influenza virus, and an increased risk of not attaining the antibody threshold considered to offer long-term protection" (Looker et al. 2014). However, in this study no association was found between PFOS serum levels and reductions in antibody rise.

The National Toxicology Program concluded that "exposure to PFOS is ***presumed to be an immune hazard to humans*** based on a high level of evidence that PFOS suppressed the antibody response from animal studies and a moderate level of evidence from studies in humans". NTP also concludes that "PFOS is suspected to suppress infectious disease resistance and NK cell activity in humans, and these conclusions are based on moderate level of evidence from animal studies and low or inadequate level of evidence from human studies" (NTP 2016).

Reference Dose and Uncertainty Factors - Immunotoxicity Endpoint

Human epidemiologic data have current limitations and are not used as a quantitative basis for a health-based RfD and MCL. However, a RfD based on quantitative epidemiologic data for an immunotoxic effect should be taken into account. In our review of the New Jersey 2016 proposed standard for PFOA we derived a MCL based on the Grandjean and Budtz-Jørgensen (2013) study where benchmark calculations based on regression modeling enabled a determination of a BMDL. Based on that study's BMDL we calculated a 0.03 MCL for PFOA (Oliaei and Kriens 2016). Using the same methodology, we derive a MCL for PFOS as follows:

The lower one-sided 95% confidence limit of the BMD, the BMDL (benchmark dose level) determined in the Grandjean and Budtz-Jørgensen (2013) study, is approximately 1.3 ng/ml for PFOS, based on the linear slope model of the regression. Based on the immunotoxic effects shown in this study we propose a 1.3 ng/ml BMDL for PFOS as the target human serum level. An uncertainty factor of 10 for human variation in susceptibility is applied. A clearance factor of 8.1×10^{-5} L/kg/day derived by USEPA for PFOS (USEPA 2016b) is applied to the target human serum Level to calculate an RfD.

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

Using NJDWQI default adult exposure values of 70 kg body weight, 2 L/day water intake, and a relative source contribution of 0.2, the MCL is:

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

Absent application of epidemiologic data, NJWQI's rigorous methodology and criteria used to select a BMDL, or NOAEL if applicable, is scientifically sound and conservative. Of the 4 final studies chosen by NJWQI for dose-response modeling, 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). As discussed in the report, the NOAEL of the study was used as the POD, or 674 ng/L, since BMDS software modeling would not calculate a BMDL for this study's dose-response data (NJDWQI 2017). After application of uncertainty factors the target human serum level calculated was the lowest among the 4 final studies, and resulted in the lowest RfD of 1.8 ng/kg/day.

This RfD is considerably more protective than the USEPA RfD of 20 ng/kg/day based on animal developmental effects (e.g. decreased pup body weight) (USEPA 2016b). However, because of potential increased susceptibility during pregnancy and lactation EPA used drinking water intake and body weight exposure values at the 90% distribution for lactating women, moderating the calculation (versus use of adult default weight and intake) to 70 ng/l for a lifetime health advisory (HA) or "MCL" for this target population.

Although the RfD determined by NJDWQI is a significant departure from the much less

protective RfD of 20 ng/kg/day developed by USEPA, we disagree with the UF (uncertainty factor) used to determine the target human serum level. NJDWQI applied a UF of unity (1.0) for sub-chronic versus chronic testing used in Dong et al (2009) 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 1×10^5 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 would 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 accelerate this effect.

Omission of a UF for sub-chronic-to-chronic in risk assessment 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 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 applying a UF of 3 versus 10 is reasonable.

As indicated by NJDWQI, “serum PFOS levels in the general U.S. population are currently near or within the range of central tendency serum PFOS levels in the studies that found associations with decreased immune response (NJDWQI 2017). Median and 95% serum PFOS concentrations are 5.2 ng/ml and 19 ng/ml, respectively, in the general U.S. population (CDC-NHANES 2017). Decreases in vaccine response were found at serum levels 6 – 27 ng/ml (Grandjean et al. 2012; Granum et al. 2013; Kielsen et al. 2016; Stein et al., 2016), within the range of serum levels in the general population. Therefore, contribution of any additional PFOS from exposure to contaminated drinking water, irrespective of the MCL level chosen, may be inadequate to assure protection for these toxicity effects (immunotoxicity), especially in sensitive individuals and vulnerable segments like infants and children. This uncertainty is broadly reflected in applying UFs to calculate MCLs.

Derivation of RfD and MCL – Adding an Uncertainty Factor to Adjust Sub-chronic to Chronic

A UF of 3 should be applied to extrapolate from sub-chronic to chronic testing in the Dong et al. (2009) study, to calculate a RfD and MCL, as follows:

A UF_{human} of 10 was used to account for increased sensitivity in sensitive sub-populations versus the average human population, and for general physiological and metabolic variation within the 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. A UF of 3 is applied to estimate the NOAEL for chronic testing from sub-chronic testing used. Since individual UFs are as log-units the product of 3 x 3 is taken as 10. Therefore, the total UF applied is 100.

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

The RfD (reference dose) is calculated as: target human serum level x clearance factor, where the clearance factor is the constant 1.8×10^{-5} derived by USEPA (EPA 2016b).

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

Summary of variables

NOAEL (POD)	674 ng/ml
total UF	100 (10 UF_{human}, $3UF_{subcronic\text{-}chronic}$, $3UF_{interspecises\ toxicodynamic}$)
Target human serum level	6.74 ng/ml
RSC	0.20
clearance factor	0.000081 L/Kg/day
default adult body weight	70 kg per NJDWQI
default adult water intake	2.0 L/day per NJDWQI

To compare with NJDWQI in its derivation, the MCL is calculated using adult default exposure values of weight and intake:

$$\text{MCL} = \frac{0.55 \text{ ng/kg/day} \times 70 \text{ kg} \times 0.2}{2 \text{ L/day}} = \textbf{\textit{3.85 ng/L (rounded to 4 ng/L)}}$$

Adjusting the total UF to 100, the MCL calculated using NJDWQI variables should be 4 ng/L.

Children Exposure and Risk

There is evidence that 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. These exposures are

generally in addition to normal PFOS exposures from food and water, packaging, and a range of consumer products.

Using NHANES data, Lorber and Egeghy found that incidental ingestion of dust is far less important among adults than children (Lorber and Egeghy 2011). Children dust intakes are highly variable due to the distribution of dust PFOA concentrations in homes; the 95th percentile intake from dust ingestion is about three times the intake from food ingestion (Lorber and Egeghy 2011). In another study by Egeghy and Lorber, the authors estimated that under typical exposure conditions, where exposure media concentrations are representative of background conditions, the median PFOS intake (sum of the median route-specific intakes) for 2-year-old children under typical exposure conditions was 50 ng/day (Egeghy and Lorber 2011). In the typical scenario for 2-year olds the contribution from ingested dust and ingested water were found to be nearly the same at 36% and 42%, respectively. Alternatively, for adults the contributions to PFOS daily intakes were much different, 6% for dust ingestion and 72% for food.

Under a typical scenario Egeghy and Lorber estimated a median total PFOS intake at 160 ng/day for adults. Under a contaminated environment scenario (contaminated water supply) they estimate median total PFOS intakes of 640 ng/day in 2-year olds and 2200 ng/day in adults. In either the typical scenario or a contaminated water scenario the authors estimated that the contribution of water to total PFOS intake is about the same in adults as in 2-year olds, about 20%. Using the authors median PFOS intake data and median weight for 2-year olds of about 13 kg (Table 8-12, Exposure Factors Handbook 2011), we calculate the median PFOS daily dose to 2-year olds at 3.85 ng/kg/day, and the median daily PFOS dose to adults (using a default 70 kg adult weight) at 2.29 ng/kg/day under the typical scenario. The 2-year old children PFOS daily exposure dose is therefore about 70% higher than adults. ***Both adults and children (2-year olds) median daily dose under the typical exposure scenario would exceed the allowable reference dose (RfD) of 1.8 ng/kg/day proposed by NJDWQI for PFOS, double the proposed RfD in 2-year olds***.

In addition to greater environmental exposures than adults, children are burdened with PFOS at birth. “Evidence shows that 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). One study found PFOS at a mean of 1.28 ng/ml in 99 of 100 samples of cord blood (Cariou et al. 2015).

PFOS is also transferred to children via breast milk. Cariou et al. 2015 found PFOS in 82% of breast milk samples at a mean concentration of 0.04 ng/ml. In a study of 70 human breast milk samples in patients from Germany and Hungary PFOS concentrations ranged from 0.028 to .309 ng/ml (Völkel et al. 2008; USEPA 2016b). PFOS transfer to infants during breast feeding lowers the mother’s PFOS blood serum levels. In a study by Mondal et al. (2014) of 633 women and 49 infants each month of breast feeding was found to lower maternal serum PFOS levels by 3%

and increase infant serum levels by 4%. Using the Cariou et al. (2015) mean breast milk PFOS concentration of 0.04 ng/ml and an upper percentile daily milk intake of 951 ml/day (table 15-3, USEPA 2008), we calculated daily PFOS intake from breast milk to breast fed infants 0 < 1 years at about 38 ng/day. Based on a 90th percentile body weight of 10.8 kg for infants 6 < 12 months age (table 8-3 USEPA 2008), ***the daily dose intake for this infant group of 0 < 1 years from breast milk is estimated at 3.5 ng/kg/day***.

In summary, age-specific behaviors (e.g. hand-to-mouth) and exposures from placental transfer and breastfeeding, in addition to normal exposures from ingested water and food, increase the PFOS body burdens in young children.

Children Toxicity Studies

Epidemiologic studies have shown many associations between PFOS and PFOA exposure and health effects in children. A systematic review by Rappazzo et al. (2017) summarized the epidemiologic evidence (literature) for relationship between prenatal/childhood perfluorochemical exposure and health outcomes in children. They conclude: “there is evidence for positive associations between PFAS (perfluoroalkyl substances) and dyslipidemia, immunity (including vaccine response and asthma), renal function, and age at menarche”, as described below (Rappazzo et al. 2017).

A study by Geiger et al. (2014) in adolescents from NHANES data, found increases in PFOA, PFOS, or total PFAS serum concentrations positively associated with high total cholesterol (>170 mg/dL) and high LDL-C. Results in a study of 12,476 children and adolescents found that PFOA was significantly associated with increased total cholesterol and LDL-C, and PFOS was significantly associated with increased total cholesterol, HDL-C, and LDL-C (Frisbee et al. 2010). Several other studies support dyslipidemia effects from exposure to PFCs in children (Rappazzo et al. 2017).

Delayed onset of puberty has been associated with altered risk of adult disease: diabetes mellitus, heart disease, bone disease, substance abuse, and asthma" (Rappazzo et al 2017). A C8 cross-sectional analysis of 3076 boys and 2931 girls found later age of puberty in both boys and girls associated with serum PFOS and PFOA levels (Lopez-Espinosa et al. 2011). For boys in that study “there was a relationship of reduced odds of reached puberty (raised testosterone) with increasing PFOS (delay of 190 days between the highest and lowest quartile)”. In girls, “higher concentrations of PFOA or PFOS were associated with reduced odds of postmenarche (130 and 138 days of delay, respectively)”. Delayed onset of puberty associated with PFOS and PFOA levels in epidemiologic studies is supported by animal studies. For example, PFOA was found to cause delayed mammary gland development in female mice offspring (White et al. 2011).

A limited number of studies have shown associations between renal function and serum PFC levels. Decrements in estimated glomerular filtration rate (eGFR) were found to be associated with increases in PFOA and PFOS concentrations in a large community study of 9660 children

age 1<18 years (Watkins et al. 2013). The study population was children and adolescents highly exposed to PFOA from contaminated water supplies, but exposed to levels typical of PFOS, PFNA, and PFHxS in the normal population. Another cross-sectional analyses of NHANES 2003-2010 data of 1960 adolescents aged 12-19 years found PFOS and PFOA associated with a reduction in kidney function and increased uric acid levels (Kataria et al. 2015). The authors found that adolescents in the highest PFOA and PFOS quartile had a lower eGFR (estimated glomerular filtration), 6.84 mL/min/1.73 m^2 (95 % CI: 2.19 to 11.48) and 9.69 mL/min/1.73 m^2 (95 % CI: -4.59 to 14.78), respectively, compared to the lowest quartile. However, the authors note that reverse causality and residual confounding could explain their findings.

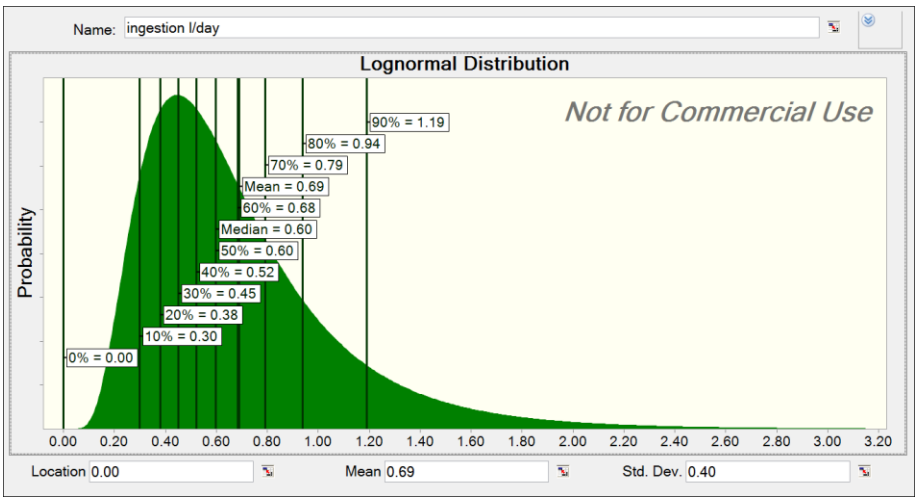
As described in the Introduction, three epidemiologic studies found suppression of vaccine-mediated antibody response to be associated with PFOS and PFOA exposure in children. The study by Grandjean and Budtz-Jørgensen (2013) found an association between increases in serum PFOS and PFOA levels and decreases in serum antibody concentrations against tetanus and diphtheria toxoids in young children (follow-up of a Faroese birth cohort). A study in Norway of 99 participants found an inverse association between the level of anti-rubella antibodies in children's serum at age 3 years and the concentrations of PFOS, PFOA, PFNA, and PFHxS (Granum et al. 2013). A recent large cross-sectional study by Stein et al (2016) of 1191 children 12-19 years old using NHANES data (1999-2000 and 2003-2004) found that a doubling of PFOS serum concentration was associated with a 7.4% (95% CI: -12.8, -1.7) decrease in mumps antibodies. A doubling of PFOS serum concentration was also associated with a 13.3% decrease in rubella antibodies; this association occurred among seropositive individuals (Stein et al. 2016).

PFOS serum levels in children associated with these immunosuppressive effects, found in these studies, are within or close to the PFOS serum levels found in the normal population.

Calculation of MCL Based on Children-Specific Exposure Variables

Some of the PFOS toxic endpoints to children have lasting effects and may subject children to later disease development. Deriving a MCL based on adult weights and water intakes results in a RfD imposed on children in excess of the maximum allowable 1.8 ng/kg/day. The uncertainty factor for sensitivity in the human population applied in the derivation accounts only for human variability in sensitivity to effect. To assure protection of children it is important that children-specific weight and water intake exposure values be used in the MCL calculation.

Body weight and water intakes of children ages 1-6 are used herein to determine a MCL. We use a mean body weight for this group of 16.8 kg and water intakes of 0.69 L/day mean, 1.19 L/day 90^{th} percentile. Mean weight for the 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 90^{th} percentile, based on the lognormal distribution of water intakes for this combined age group, shown in the graph below.



Lognormal Distribution of Water Intakes for Children Group Ages 1-6

To compare the difference in MCLs derived by NJDWQI with that derived herein using children weight and water intakes, we use the same RfD of 1.8 ng/kg/day derived by NJDWQI (which excludes applying the uncertainty factor of 3 we used to estimate NOAEL chronic from sub-chronic testing).

Summary of variables used and values

RfD	1.8 ng/kg/day
RSC	0.20
children body weight	16.8 kg
children intake	1.19 L/day 90th percentile

Children Group (age 1-6)

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

(Using a mean water intake of 0.69 L/day results in a MCL of 8.8 ng/L)

The MCL for PFOS should be 5 ng/l.

If a UF of 3 to estimate chronic NOAEL from sub-chronic is included the MCL becomes:

$$MCL = \frac{0.55\ ng/kg/day \times 16.8\ kg}{1.19\ L/day} \times 0.2\ RSC = 1.55\ ng/L\ (round\ to\ 2\ ng/L)$$

Other states have recognized children and infants as a more vulnerable population segment. Vermont used a 95th percentile body weight-adjusted water intake rate of 0.175 L/kg/day for the first year of life in its MCL calculation to determine a MCL of 20 ng/l. The MCL is applied as a

sum combination, [PFOS + PFOA] ≤ 20 ng/l (Vermont 2016). In 2017 the Minnesota Department of Health (MDH) updated its earlier Health Risk Limit (HRL) for PFOS in drinking water. MDH used the USEPA RfD of 20 ng/kg/day based on animal developmental effects but incorporated a database uncertainty factor of 3 in recognition of immunotoxicity shown in animal studies. This resulted in a RfD of 5.1 ng/kg/day. MDH modeled two scenarios and found a breast-fed infant exposure scenario as most limiting, and determined a PFOS limit of 27 ng/L (MDH 2017).

Conclusion

NJDWQI's evaluation of animal and epidemiologic PFOS toxicity studies was comprehensive and rigorous. The New Jersey Department of Environmental Protection has been progressive in efforts to protect public health from PFOS, PFOA, and other perfluorochemical exposures. We concur with the process used to derive a PFOS reference dose, concluding with the Dong et al (2009) study showing an immunotoxic effect in test animals.

However, all population segments must be protected. Our analysis finds that at the proposed 13 ng/l MCL, PFOS daily intakes by body weight posed to young children 1-6 would be more than double the PFOS dose of 1.8 ng/kg/day deemed allowable by NJDWQI. This is disconcerting since existing serum PFOS levels in children in the population are already within or near serum PFOS levels associated with immunotoxic effects found in epidemiologic studies. The developing fetus, infants, and young children are particularly sensitive to PFOS and PFOA exposures during early sensitive periods. Early PFOS exposures in children, even at low doses of 1.8 ng/kg/day, may affect risk for later disease manifestation. To assure protection of children's health NJDWQI should depart from using adult default exposure values and use children-specific exposure values in its MCL derivation, as described in this review.

We recommend an MCL of 5 ng/L, as calculated above based on children exposure values.

Alternatively, due to PFOS and PFOA co-occurrence in water supplies and additivity concerns, we recommend that the combination of PFOS and PFOA concentrations in water supplies be no higher than 13 ng/L. [PFOA + PFOS] ≤ 13 ng/L

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January 31, 2018

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Re: Comment on the DWQI Draft Second Addendum to Appendix C: Recommendation on Perfluorinated Compound Treatment Options for Drinking Water (PFOS), November 2017

Please find enclosed a technical analysis prepared by Don Kriens, Sc.D., P.E. of Cambridge Environmental Consulting commissioned by Delaware Riverkeeper Network and submitted on behalf of this organization and its membership on the Drinking Water Quality Institute's document **Draft Second Addendum to Appendix C: Recommendation on Perfluorinated Compound Treatment Options for Drinking Water (PFOS), November 2017.**

Also enclosed is a copy of the Curriculum Vitae for Don Kriens, Sc.D., P.E.

Delaware Riverkeeper Network submits these comments advocating that the public be protected from 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 and the most effective treatment technologies.

We support the recommendations and findings made by Don Kriens of Cambridge Environmental Consulting in this technical analysis regarding the Draft Second Addendum to the Treatment Options Report by the DWQI. We support the utilization of the most effective methods of removing perfluorinated compounds (PFCs), including PFOS, considering the highly toxic properties of these compounds.

Cambridge Environmental Consulting **recommends granular activated carbon (GAC) combined with reverse osmosis technology (RO)** to completely remove all PFCs from drinking water to the proposed safe drinking water standards. Dr. Oliaei and Don Kriens recommend further research on nanofiltration, which may demonstrate adequacy to remove PFOA, PFOS and lower carbon number PFCs as a substitute for RO. Delaware Riverkeeper Network supports these findings and advocates for the use of the best available technology that will be capable of removing both PFOS and PFOA as well as other PFCs such as shorter chain PFCs with a goal of providing safe drinking water to the public.

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Thank you for the opportunity to comment on the Treatment Options to remove PFCs, including PFOS.

Sincerely,

Maya van Rossum
the Delaware Riverkeeper

Tracy Carluccio
Deputy Director

Attachments: **Review of New Jersey Draft Second Addendum to Appendix C: Recommendation on Perfluorinated Compound Treatment Options**, Don L. Kriens Sc.D., P.E., Cambridge Environmental Consulting, January 2018.

Review of New Jersey
Second Addendum to Appendix C: Recommendation on Perfluorinated Compound Treatment Options

prepared by
Don L. Kriens Sc.D., P.E.
Cambridge Environmental Consulting
January 2018

Executive Summary

The treatment system must be capable of removing both PFOA and PFOS, as well as providing best treatment technology available to remove other perfluorinated compounds, such as PFBA, that pose some toxicity. GAC alone does not remove low carbon number perfluorinated carboxylic acids (e.g. PFBA) and, at times, low carbon number perfluorinated sulfonic acids (e.g. PFBS). There are differential removal efficiencies among perfluorinated compounds through GAC systems. As described in the full-scale GAC operating systems and research discussed herein, PFOA is often marginally removed by GAC alone. Granular activated carbon (GAC) followed by reverse osmosis technology is needed at public water treatment systems to assure removal of all perfluorinated compounds. Further testing of nanofiltration, as discussed in the Subcommittee's Recommendation on Perfluorinated Compound Treatment Options (2015), may demonstrate adequacy to remove PFOA and low carbon number perfluorinated compounds, as a substitute for reverse osmosis.

Prevalence of PFCs in New Jersey Drinking Water

PFOS and PFOA are found in New Jersey water supplies at relatively high concentrations, in some instances at levels greater than the existing 14 ng/l MCL for PFOA and the proposed 13 ng/l MCL for PFOS. We expect that in excess of one million people are ingesting PFOS and PFOA at these levels. PFOS levels in some New Jersey PWS (public water supplies) are such that ingestion of this contaminated water further increases residents' PFOS blood serum levels beyond those already found in epidemiologic studies in the U.S. population to be associated with adverse health effects (immunotoxicity).

Following discovery of PFOA levels in PWS (public water systems) at levels up to 190 ng/l in groundwater and 64 ng/l in tap or finished drinking water, New Jersey Department of Environmental Protection completed two studies, in 2006 and 2009-2010, to test for PFOS, PFOA, and other perfluorinated compounds in 53 PWS (NJDWQI 2017).

PFOS was found in the 2006 study in 30% of 23 PWS tested at or above the minimum reporting limit (MRL) of 4 ng/L (NJDWQI 2017), with the highest PFOS level at 19 ng/L (NJDWQI 2017). In the 2009-2010 study raw water was tested in 30 PWS in 19 of New Jersey's 21 counties. PFOS was found in 8 of 29 PWS sampled at levels up to 12 ng/L in 5 PWS using groundwater, and up

to 43 ng/L in 3 PWS using surface water (NJDWQI 2017). Finished drinking water in these PWS would be expected to contain the same concentrations, since minimal to no removals of perfluorinated compounds are achieved through conventional water treatment technologies.

Testing of 175 New Jersey PWS, including 165 large community systems and 10 small community systems, completed under the UCMR3 (Third Unregulated Contaminant Monitoring Rule) found 6 PWS with PFOS levels exceeding 40 ng/L, and 18 PWS with PFOA levels exceeding 20 ng/L (UCMR3 2017, NJDWQI 2017). Based on the New Jersey DEP database we note that 10 of 76 PWS tested were found to contain PFOS ≥ 20 ng/l, with 7 PWS containing PFOS at levels exceeding 40 ng/L.

The 80 PWS tested represent about 14% of the total community water supplies in New Jersey. In 2016 New Jersey had 581 community water systems (NJ DEP Division of Water Supply and Geoscience 2017), serving about 91% of the total population, of which 42% were medium to large systems, and 58% were small systems.

State-wide studies of PFOA and PFOS in private wells have not been conducted in New Jersey. About 12% of New Jersey's population obtains drinking water from private wells (NJDEP Division of Water Supply and Geoscience 2017). Although it is likely the majority of these wells are not contaminated, groundwater at locations proximate to industrial activities using perfluorinated compounds or where AFFF (aqueous fire fighting foam) has been used may exhibit levels of PFOS and PFOA. PFOA has been found at levels exceeding 40 ng/L (maximum >400 ng/L), in 59 private wells within 2 miles of a New Jersey industrial source (NJDWQI 2016 Report, DuPont, 2009). PFOS was found in private wells at levels above the USEPA advisory of 70 ng/L (PFOS + PFOA) and the proposed MCL of 13 ng/L near sites contaminated with fire fighting foam (NJDWQI 2017).

Treatment Technologies and Capabilities, Chemistry of PFSAs versus PFCAs

The NJDWQI Treatment Subcommittee states in its 2015 report, Appendix C: Recommendation on Perfluorinated Compound Treatment Options for Drinking Water: "the treatment options are not expected to differ from compound to compound due to their similar properties (e.g. persistence, water solubility, similar structure, strong carbon-fluorine bonds, and high polarity)". The NJDWQI subcommittee does not diverge from this position in the Appendices to the 2015 Report. We disagree with this position. Peer-reviewed studies show that treatment options differ in removal capability among perfluorinated compounds.

Although perfluorinated compounds have somewhat similar structure, polarity, and solubility, there are differences in structural chemistry that affect removal among treatment options. 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 compared to PFCAs (pefluoroalkyl carboxylic acids, e.g. PFOA, PFBA). Therefore, their tendency to adsorb onto activated carbon is greater.
In a review of adsorption behavior of perfluorinated compounds (mostly PFOS and PFOA) by Du

et al. (2014) sorption capacities of PFSAs (e.g. PFOS) onto activated carbon or other adsorbents were observed to be higher than PFCAs (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 affects removal efficiencies of PFASs; “sulfonic group resulted in higher removal efficiency compared to carboxylic group” (Ostlund 2015). This study also found that branched isomers (for PFOS) were less efficiently removed by GAC (granular activated carbon) compared to linear PFOS isomers. We note this finding could, in part, account for differences in PFOS removal among locations using GAC.

The Water Research Foundation 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 perfluorinated compounds and PFSAs (e.g. PFOS) versus PFCAs (e.g PFOA, PFBA) (Water Research Foundation 2016). Full-scale reverse osmosis systems demonstrated significant removal for all perfluorinated compounds, including the smallest, perfluorobutanoic acid (PFBA).

The WRF (2016) study further evaluated nanofiltration (NF) for removal of a suite of PFCAs and PFSAs and notes 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.

A study of sorption onto GAC, zeolite, and sludge found that PFOS is strongly adsorbed by GAC; PFOA and PFBS were also 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).

Perfluorinated compound removal was studied at two water reclamation plants (treating domestic effluents as influent) in Southeast Queensland, Australia. In the treatment plant using reverse osmosis, PFOA was removed to less than reporting level to 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. In the treatment plant using biologically activated carbon, PFOA and PFOS were ineffectively removed, although lack of removal may have been due to the age of the carbon or short contact times (Thompson et al., 2011).

In a study at a water treatment plant in Amsterdam using GAC, PFOA was not effectively removed, with a final (treated) mean PFOA concentration of 5.3 ng/L (range 0.8 ng/L - 9.4 ng/L) versus a mean influent (raw) PFOA concentration of 4.4 ng/L (range 3.8 ng/L - 5.2 ng/L). The authors found greater removals of PFOS and PFNA with a mean final (treated) water level of <0.23 ng/L and <0.24 ng/L, respectively, versus influent (raw) levels of 6.7 to 10 ng/L for PFOS and 0.5 to 0.8 ng/L for PFNA (Eschauzier et al. 2012). This study also found that PFBA, PFPeA,

PFHxA, PFOA, and PFBS were not well removed by the (operating) GAC filtration. In general, the authors found that PFOA decreased by only 50% using GAC.

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 treats 100 million gallons per day of surface river water to supply over 1 million inhabitants. The authors found that ultrafiltration/reverse osmosis removed PFOS and PFOA by ≥ 99%, but GAC alone 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).

Granular Activated Carbon Treatment Systems – Removal Efficiency

NJDWQI subcommittee describes 2 facilities in New Jersey, one in Pennsylvania, and one in Minnesota where GAC is used to treat perfluorinated compounds in public water supplies.

In the New Jersey Penns Grove GAC treatment system PFOS was reported at levels lower than the reporting limit of 5 ng/l in finished water, although the highest PFOS level in the raw water was 13 ng/L. At the New Jersey Logan System Birch Creek GAC system PFOS was reported at levels lower than the reporting limit of 5 ng/l in finished water, although the average PFOS raw water concentration was only slightly above the reporting limit, at 7 ng/L. We believe that PFOS and PFOA removal performance may be much different at other locations, where PFOS and PFOA are present in raw water at higher levels and/or where source water contains greater natural organic matter.

The Subcommittee notes the Horsham Water and Sewer Authority (HWSA) in Horsham, Pennsylvania, where GAC was recently installed to treat well water contaminated with perfluorinated compounds. Wells are believed contaminated from fire fighting foam used at the nearby Naval Air Station Joint Reserve Base - Willow Grove. NJDWQI subcommittee states that HSWA raw (well) water samples collected between January and March 2017 “show a range of PFOS concentrations from 230 - 1297 ng/L and an average of 629.3 ng/L”, and indicates PFOS was not detected in finished water. We characterize HSWA well data differently, based on our review of HWSA active well data (HWSA, PFOS PFOA Active Source Monitoring Results with Charts, 2017), as summarized in Table 1 below.

We observed that, during limited periods, post-GAC treatment in 4 HSWA contaminated wells

did not remove PFOS or PFOA to levels below the reporting limit of 5 ng/L. This appeared to occur just prior to carbon change-out, as shown in the HSWA data. However, it should be noted that GAC treated water is also blended with other HSWA wells and water sources, as shown in HSWA system schematics (HSWA June 2016), to assure that levels are below applicable standards and protective. As of December 2017 the HSWA indicates "the combined concentration of PFOS/PFOA from all sources currently supplying the public system is approximately 4 ppt (ng/l)" (HSWA Dec 2017 Update). This is below the USEPA health advisory level applied of 70 ppt (ng/l) for combined concentration (PFOA + PFOS).

Based on HSWA data as of December 19, 2017, the following table summarizes results for active HSWA wells (HWSA, PFOS PFOA Active Source Monitoring Results with Charts, 2017).

HSWA well number	Period	PFOS lowest	PFOS highest	PFOA lowest	PFOA highest
7	May 5, 2016 - Dec 19, 2017	ND	11	3.1	11
10*	Jan 19, 2016 - April 5, 2017	16	76	12	48
17**	Jan 19, 2016 - Dec 22, 2016	50	110	20	37
21***	Jan 2016 - Jan 11, 2017	5.1	14	8.4	13
26****	Jan 25, 2016 - March 8, 2017	340	1297	640	1765
40*****	Jan 11, 2017 - March 24, 2017	230	1203	33	88

Table 1. Summary Table of HSWA Raw Water PFOS and PFOA Concentrations (ng/L)

ND – non detect at reporting limit 5 ng/L

*After April 5, well 10 was treated through GAC system with N.D results.

** After December 22, 2016 well 17 was treated with GAC. Results post GAC treatment in 2017 mostly ND (reporting levels 5 ng/L), except for 6 positive PFOS values ranging from 3.1 to 159 ng/L, and 5 positive PFOA values ranging from 13 ng/L to 29 ng/L.

*** After January 2017 well 21 was treated with GAC. Results post GAC treatment ND (reporting levels 5 ng/L), except one PFOS value of 4.2 ng/L, and 5 PFOA values ranging from 3.3 to 7.8 ng/L.

**** After March 8, 2017 well #26 was treated with GAC. Results post GAC treatment ND, except for one PFOS value of 5 ng/l and one value of 5 ng/L for PFOA

***** After March 24, 2017 well #40 treated with GAC. Results post GAC treatment ND, except 4 PFOS

values ranging from 2.5 ng/L – 131 ng/L and 3 PFOA values ranging from 3.1 ng/L to 5.3 ng/L

The Second Addendum discusses the city of Oakdale, Minnesota GAC system used to remove perfluorinated compounds, including PFOS, from well (drinking) water. NJDWQI subcommittee notes that the Oakdale PFOS method detection limit is 0.5 ng/L and the Minimum Reporting Limit (MRL) is 5 ng/L. However, these limits were only recently put into effect by the Minnesota Department of Health (Rinker communication, Jan 5, 2018). (A MRL for PFOA of 5 ng/L was also recently put into effect.) Prior RLs (reporting limits) for PFOS and PFOA at Oakdale were 25 ng/L and 35 ng/L, respectively. The Subcommittee states that "samples taken after GAC treatment show no detection of PFOS". However, these non-detects are based on the prior RLs (25 ng/L and 35 ng/L), not 5 ng/L. It is yet unknown whether the Oakdale GAC system will remove PFOS or PFOA to ≤ 5 ng/L.

Based on Oakdale's perfluorinated compound data (Bachmeier 2017), PFOS was non-detect in finished drinking water for the period November 2015 through November 2017, at a RL of 50 ng/L for the period November 2015 through July 2016, and a RL of 25 ng/L for the period August 2016 to present. PFOA was non-detect for all samples at a RL of 50 ng/L for the period November 2015 through July 2016, and a RL of 35 ng/L for the period August 2016 to present. We observed that PFBA (perfluorobutanoic acid) remains at relatively high levels in Oakdale's drinking water, typically at around 1400 ng/L, consistent with other installations and research showing poor or no removal by GAC of low carbon number PFCAs. Generally PFBA passes through unchanged in concentration through the Oakdale GAC system. However, PFBA levels in Oakdale's finished water are well below the Minnesota Department of Health HRL (health risk limit) for PFBA of 7000 ng/L.

The 3M Cottage Grove manufacturing plant in Cottage Grove, Minnesota operates a GAC system to treat wastewater discharged to the Mississippi River, installed in 2004 pursuant to requirement by the NPDES (National Pollutant Discharge Elimination System) permit. 3M was the primary global producer of PFOS-related perfluorinated compounds, and PFOA, and manufactured these chemicals at its two U.S. plants in Decatur, Alabama and Cottage Grove, Minnesota, and in Europe at its plant in Antwerp, Belgium. 3M perfluorochemical production began at the Minnesota plant around 1950 (Oliaei et al. 2006).

Based on one sampling event in 2006 by Minnesota Pollution Control Agency staff, the 3M GAC treatment facility removed PFOS by 95% and PFOA by 79%. The GAC treatment plant was less effective at removal of carboxylic perfluorinated compounds (PFCAs). In the 2006 sampling of post-GAC treated wastewater perfluorinated compound concentrations were very high: PFOA 1670 ng/L, PFOS 1330 ng/L, PFBS 169,000 ng/L, and PFBA 58,100 ng/L (Oliaei et al. 2006).

Since 2006 levels of perfluorinated compounds in the 3M discharge (post-GAC treated wastewater) are considerably lower, but remain elevated, as summarized in the following table for the period October 2015 through November 2017 (NPDES 3M data provided by Marco Graziani, Minnesota Pollution Control Agency, January 2018).

Period	PFOA ng/L	PFBA ng/L	PFNA ng/L	PFOS ng/L	PFBS ng/L
Oct-15	126	13100	<25	<46.4	94.7
Nov-15	<48	102000	<25	<23.2	1170
Dec-15	61.9	8470	<25	<23.2	1410
Jan-16	38.2	36500	<25	<46.4	1660
Feb-16	166	27500	<25	<23.2	210
Mar-16	329	15400	<25	27.8	217
Apr-16	584	55700	<25	58.1	434
May-16	<24	28100	<25	<23.2	353
Jun-16	75.9	6040	<25	<23.2	120
Jul-16	193	83700	<25	<23.3	817
Aug-16	116	380000	<25	<46.4	644
Sep-16	81.6	21200	<25	<23.2	1240
Oct-16	90.3	13400	<25	<23.3	469
Nov-16	194	18200	<25	43.3	848
Dec-16	370	19200	<25	69.3	286
Jan-17	77.2	7470	<25	25.4	195
Feb-17	108	6870	<25	26.6	201
Mar-17	157	12800	<25	37.5	6240
Apr-17	265	10700	<25	44.9	676
May-17	165	11000	<25	38.6	465
Jun-17	113	14100	<25	71.4	1340
Jul-17	170	21700	<25	28.7	3740
Aug-17	328	13900	<25	60.3	2580
Sep-17	50.6	4090	<25	<23.2	1780
Oct-17	129	11600	<25	29.4	18700
Nov-17	133	7680	<25	<46.4	6920
~ mean	**172**	**36555**	**<25**	**43**	**2031**

Table 2. 3M Perfluorinated Compound Discharge (SD001) Post-GAC Treatment ng/L

Mean concentrations in Table 2 are means of positive values, excluding non-detects. The post-GAC discharge perfluorinated compound discharge concentrations for October 2015 to present remain high: mean PFOA 172 ng/L, mean PFBA 36,555 ng/L, mean PFOS 43 ng/L, and mean PFBS 2031 ng/L. However, these concentrations are much lower than those discharged during the period January 2007 though July 2010: mean PFOA 2989 ng/L, mean PFBA 54,098 ng/L, mean PFOS 595 ng/L, and mean PFBS 18,673 ng/L. The 3M data indicate that low carbon perfluoroalkyl compounds (such as PFBS and PFBA) and PFCAs (e.g. PFBA, PFOA) are not removed by this GAC system to low levels.

Recommended Treatment - GAC Followed by Reverse Osmosis

GAC alone has not been shown in most cases to consistently remove PFOA to low ng/L levels. Therefore, GAC followed by reverse osmosis (RO) is required to remove PFOA and, in some cases PFOS, to assure consistent removal. GAC followed by RO will also enable removal of low carbon number perfluorinated compounds such PFBA. PFBA has been shown in animal studies to cause toxicity. The Minnesota Department of Health identified critical toxic effects of "liver weight changes, morphological changes in liver and thyroid gland, decreased TT4, decreased red blood cells, decreased hematocrit and hemoglobin, and subcritical toxic effects of increased relative thyroid weight, decreased serum TT4 and dFT4, decreased cholesterol, and delayed eye opening (rat)", in PFBA animal studies as the basis to derive a HRL (health risk limit) for PFBA (MDH 2017 Perfluorobutyrate).

A consideration using GAC/RO in large municipal treatment systems is disposal of the RO reject. Technologies to treat RO reject are generally limited to evaporative technologies applied to high salt concentrating RO systems, to eliminate the reject discharge. Evaporative systems require excessive energy input and are often prohibitively expensive. RO reject evaporative systems may be relatively cost effective, however, in arid climate locations to allow lined evaporation ponds, or where untreated reject water does not pose a concern or environmental impact (such as RO systems used for drinking water treatment in coastal areas, where the salt RO reject is discharged to the ocean). Such is not the case for the temperate geographic region under consideration in New Jersey. Thus, RO rejects at large GAC/RO plants would likely require direct discharge to a receiving water.

The GAC system, however, will enable removal of a significant mass of PFOS, and to a lesser extent, PFOA. A primary concern with waters receiving perfluorinated compounds is uptake of PFOS in fish, which bioaccumulates, and subsequent consumption of PFOS-contaminated recreationally caught fish. PFOA does not bioaccumulate. In any case, the mass of perfluorinated compounds, including PFOS and PFOA, discharged to a receiving water in a GAC followed by reverse osmosis system (reject) would not be greater than the mass discharged by a GAC system alone.

NJDWQI briefly discusses Point of Use (POU) drinking water systems, for use in individual homeowners on private wells, and certification by NSF (National Sanitation Foundation). NSF developed protocol NSF P473 to evaluate drinking water treatment device capability to reduce PFOA and PFOS in drinking water. The NSF certified a number of systems using GAC or GAC/RO that meet the EPA "standard" (combined PFOS and PFOA) of 70 ng/L. The NSF protocol included "challenge" of influent level of 1500 ng/L [5 parts PFOA and 10 parts PFOS by weight] to the GAC and GAC/RO systems. Studies by NSF showed good removal performance by GAC with highest performance in POU systems using GAC followed by reverse osmosis (NSF personal communication, E. Valentine, Jan 4, 2018)

We concur that GAC/RO POU systems offer a treatment solution to homeowners on private wells. Perfluorinated compounds do not volatize and therefore inhalation via showering and bathing do not pose an exposure pathway, versus other compounds such as DBPs (disinfection byproducts) where inhalation in showering may comprise a significant portion of total

exposure. In addition, perfluorinated compounds do not cross the dermal barrier. However, further testing is needed to assure that POU GAC/RO systems remove PFOS, PFOA, and PFNA to low ppt levels. Use of POU GAC/RO systems is somewhat complicated by the requirement of homeowner management of carbon change-out and proper operation.

Conclusion

The proposed New Jersey MCL of 13 ng/L, our recommended MCL in this review of 5 ng/L, the Minnesota limit (HRL) of 27 ng/L, and the Vermont drinking water advisory of 20 ng/L (combined) for PFOS are within the same very small "ballpark". Changing an uncertainty factor or exposure value used in these derivations obscures the difference in values. Accordingly, further emphasis should be placed on treatment and removal.

In addition to shorter chain perfluorinated compounds such as PFBS and PFBA, PFOA usually co-exists with PFOS in water supplies. Although the shorter chain perfluorinated compounds (e.g. PFBA, PFBS) are less toxic and excreted faster than longer chain perfluorinated compounds (e.g. PFOS, PFOA), they remain persistent, as demonstrated in the Oakdale, MN drinking water supply. The toxicity of shorter chain perfluorinated compounds is not fully understood, although toxicity of PFBA has been shown in animal testing and, in the case of Minnesota, a health risk limit (limitation) has been derived. There are differential removal efficiencies among perfluorinated compounds in GAC systems. PFOA is often poorly removed by GAC alone, as described in the full-scale GAC operating systems and research discussed above. Short chain perfluorinated carboxylic acids (e.g. PFBA) generally remain unchanged (not removed) through GAC systems.

The treatment system chosen for removal of perfluorinated compounds must be capable of removing both PFOA and PFOS, as well as providing best treatment technology available to remove other perfluorinated compounds, such as PFBA, that pose some toxicity. Removal of shorter chain perfluorinated compounds requires reverse osmosis in addition to GAC. Granular activated carbon (GAC) followed by reverse osmosis, or nanofiltration if pilot-scale studies demonstrate efficiency, is needed as a combined option to adequately remove both PFOS and PFOA, as well as other perfluorinated compounds that may be present.

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DELAWARE RIVERKEEPER NETWORK

January 31, 2018

watersupply@dep.nj.gov
New Jersey Department of Environmental Protection
Trenton, New Jersey

Re: Report on the Development of a Practical Quantitation Level for Perfluorooctanesulfonic Acid (PFOS) in Drinking Water, NJ Drinking Water Quality Institute Testing Subcommittee

Please find enclosed a technical analysis prepared by Fardin Oliaei, MPA, PhD, and Don Kriens, Sc.D., P.E. of Cambridge Environmental Consulting commissioned by Delaware Riverkeeper Network and submitted on behalf of this organization and its membership on the Drinking Water Quality Institute's document **Report on the Development of a Practical Quantitation Level for Perfluorooctanesulfonic Acid (PFOS) in Drinking Water.**

Also attached are copies of the Curriculum Vitae for Dr. Oliaei and for Don Kriens, Sc.D., P.E.

Delaware Riverkeeper Network submits these comments advocating that the public be protected from 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.

We support the recommendations and findings made by Dr. Oliaei and Cambridge Environmental Consulting in this technical analysis regarding a Practical Quantitation Level (PQL) for PFOA. We support Cambridge Environmental Consulting's concurrence with the DWQI Testing Subcommittee's analysis that determined a PQL on the basis of multiplication of the method detection limit by a factor of 5, resulting in a PQL of 4.2 ng/L for PFOS. The PQL of 4.2 ng/L is below the MCL of 5 ng/L that Cambridge Environmental Consultants recommends as more protective. Thank you for the opportunity to comment on the PQL for PFOS.

Sincerely,

Maya van Rossum
the Delaware Riverkeeper

Tracy Carluccio
Deputy Director

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Attachments:

Review of NJDWQI Report on the Development of a Practical Quantitation Level for PFOS in Drinking Water, Fardin Oliaei Ph.D. and Don L. Kriens Sc.D., Cambridge Environmental Consulting, January 2018

Curriculum Vitae for Fardin Oliaei, MPA, PhD

Curriculum Vitae for Don Kriens, Sc.D., P.E.

Review of NJDWQI Report on the Development of a Practical Quantitation Level for PFOS in Drinking Water

prepared by
Fardin Oliaei Ph.D. and Don L. Kriens Sc.D.
Cambridge Environmental Consulting
January 2018

The NJDWQI Testing Subcommittee determined that use of low calibration standards for PFOS among laboratories was the most appropriate performance standard to base a practical quantitation limit (PQL). Using low calibration standards of 19 laboratories that used either EPA Method 537, modified EPA Method 537, or a proprietary method, the Subcommittee applied bootstrap analysis (bootstrap estimate of a confidence interval of the mean) to determine an upper confidence limit of 95% for PFOS at 4.2 ng/L. (One laboratory with a low calibration standard above the upper confidence level (95%) was excluded.) The same methodology was applied to 12 laboratories using EPA Method 537 to determine an upper confidence limit (95%) of 3.8 ng/l. (Two laboratories were excluded from that analysis since their low calibration standards were outside the 95% confidence interval.)

The Subcommittee's methodology appropriately diverts from the prior or traditional use of developing a PQL on the basis of multiplication of the MDL (method detection limit) by a factor of 5, consistent with USEPA "trend" not to use MDLs to develop a PQL.

We concur with the Subcommittee's analysis that determined a PQL of 4.2 ng/l for PFOS. This PQL concentration is below the proposed MCL of 13 ng/l, and below the 5 ng/l that we assert is more protective.

Fardin Zoe Oliaei

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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 2010 - 2012
Auditing several courses: Air Pollution; Water Pollution; and Risk Assessment

Harvard University John F. Kennedy School of Government, Cambridge, MA
Master in Public Administration 2009
- Concentration*:* Leadership and International Environmental Health Policy and Management

Bush Foundation Leadership Fellow (MN) to pursue studies at Harvard University 2008 - 2010

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 in Chemistry Minor in Biology

PROFESSIONAL EXPERIENCE

DST Health Solutions, LLC, Home Office East 2014 - Present
Sr. Project Management Specialist
- Evaluating the global pattern of disease and morbidity in growing population and identifying social determinants of population health as a tool for managing healthier population and creating effective health care systems.

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.
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Don L. Kriens **Curriculum Vitae**

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Oakdale, MN 55128 dlk810@mail.harvard.edu

AREAS OF EXPERTISE

- Professional engineer - civil and environmental engineering design.
- Modeling exposure/risk of environmental contaminants, including disinfection byproducts and contaminants in drinking water supplies.
- Global water scarcity, climate change impacts on water supplies
- Toxic contaminant cleanup.
- Design of water/wastewater treatment systems, remediation, stormwater.
- Industrial processes, pollution prevention, industrial process chemistry.
- Emerging treatment technologies.
- Engineering economic analysis.
- Regulatory enforcement, civil and criminal.
- Environmental policy and justice

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

Research Fellow, Harvard T.H. Chan School of Public Health, Harvard University

Principal Engineer: 1978-2008 Minnesota Pollution Control Agency
- Lead Agency technical expert for water projects. Mentor engineers and technical staff.
- Research projects - ecological and health impacts of contaminants. Evaluate emerging technologies.
- Conducted major civil and criminal investigations with MN Attorney General staff, U.S. Attorney's Office, USEPA Region V. Expert witness.
- Developed major industrial permits, technologies to comply. Economic impact.
- Technical expert for water/wastewater treatment, remediation and water supplies.
- Technical expert for emergency response regarding toxics and resolution.

Consulting Engineer and Owner: 1996-2008 Kriens Engineering, Oakdale, MN
• Design of Individual Sewage Treatment Systems. Groundwater (well) analysis and water consulting.

Engineer: Castek Consulting Engineering Services
• Operation, design, process chemistry of wastewater plants; indoor air quality studies.

Research Scientist and Environmental Engineering Laboratory Supervisor, University of Iowa
Department of Civil and Environmental Engineering

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

TEACHING EXPERIENCE

Harvard University
• Teaching Assistant in water pollution and risk assessment. Lecture in "Farm to Fork" course at Harvard Extension School.

Kirkwood Community College, Cedar Rapids, Iowa
- Instructor; wrote courses in chemistry/advanced chemistry of wastewater treatment.

LICENSES AND PROFESSIONAL AFFLILIATIONS

- Registered Professional Engineer
- Individual Sewage Treatment System Designer (Minnesota)
- Certification Air Quality Inspections (California Air Resources Board)
- Certification Stormwater Treatment and Erosion Design
- Member, Minnesota Government Engineers Council

PUBLICATIONS

Oliaei F, Kriens D, Kessler K. Investigation of Perfluorochemical Contamination (PFC) in Minnesota, MN Senate Report. Feb 2006.

Discovery of PFOS Contamination in Fish Near a PFC Manufacturing Plant in Minnesota, presentation at EPA Conference on PFCs and Perfluorocarbons, Research Triangle Park, North Carolina, June, 2010

Environmental Releases of Perfluoroalkyl Compounds from Two Landfill Sites in Minnesota, presentation at EPA Conference on PFCs and Perfluorocarbons, EPA Research Triangle Park, North Carolina, June, 2010

Discovery and Investigation of PFOS/PFCs Contamination from a PFC Manufacturing Facility in Minnesota, Kriens D, Oliaei F, The International Symposium on Halogenated Persistent Organic Pollutants – Dioxin 2010, San Antonio, Texas, August 2010

Estimating the Fraction of Methyl Mercury Exposures from Locally Caught Fish: A Case Study of U.S. Gulf Coast Residents", International Conference on Mercury, Halifax, Nova Scotia, July, 2011.

Contamination of Drinking Water and the Environment by Production and Industrial Use of Perfluoralkyl Compounds (PFCs) ", Oliaei F, Weber R, Kriens D, Watson A, The 31st International Symposium on Halogenated Persistent Organic Pollutants – Dioxin 2011, August 2011, Brussels, Belgium.

Contamination of Drinking Water and the Environment by Production and Industrial Use of Perfluoralkyl Compounds (PFCs), Oliaei F, Weber R, Kriens D, Watson A, presentation at 11th International HCH and Pesticides Conference, Gabala, Azerbaijan, September, 2011

Sunderland E, von Stackelberg K, Kriens D. Pilot Analysis of Gulf of Mexico State Residents' Methylmercury Exposure to Commercial and Locally Caught Fish. Harvard Center for Risk Analysis. March 2012.

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