November 19, 2016

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

Re: Health-Based Maximum Contaminant Level Support Document: Perfluorooctanoic Acid (PFOA)

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 the organization and its membership regarding the Support Document and recommendation by the Drinking Water Quality Institute for a **Health-Based Maximum Contaminant Level for Perfluorooctanoic Acid (PFOA)**. 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 PFOA 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 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 Dr. Oliaei's finding that that the proposed drinking water MCL of 14 ng/L for PFOA based on increased relative liver weight is not adequately protective of all population segments. We support Dr. Oliaei's position that the standard may be developed based on an immunotoxic association in children or, alternatively, evidence of developmental effects shown in rodent studies. Both of these approaches provide more sensitive endpoints with quantitative data to develop an MCL, providing greater protection. We support Dr. Oliaei's analysis and final conclusion that the recommended MCL should be lowered to 1 ng/L, or alternatively, should be no higher than 6 ng/L.

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

DELAWARE RIVERKEEPER NETWORK
925 Canal Street, Suite 3701
Bristol, PA 19007
Office: (215) 369-1188
fax: (215)369-1181
drn@delawareriverkeeper.org
www.delawareriverkeeper.org

Sincerely,

Maya van Rossum
the Delaware Riverkeeper

Tracy Carluccio
Deputy Director

Attached: Technical Analyses of New Jersey Drinking Water Quality Institute Proposed Health-Based Maximum Contaminant Level for Perfluorooctanoic Acid (PFOA) in Drinking Water, Fardin Z. Oliaei, Don Kriens, Cambridge Environmental Consulting, Nov. 18, 2016

Technical Analyses of New Jersey Drinking Water Quality Institute

Proposed Health-Based Maximum Contaminant Level (MCL) for Perfluorooctanoic Acid (PFOA) in Drinking Water

prepared by

Fardin Z. Oliaei MPA, Ph.D.
Don L. Kriens* Sc.D., P.E.

Cambridge Environmental Consulting

November 18, 2016

PREFACE

The opinions in this report are stated to a reasonable degree of scientific probability. The methods and principals used in forming these opinions are generally accepted within the scientific community and are consistent with their regular application within the scientific community. Qualifications of the authors, including publications where applicable, are summarized in the attached resumes. We reserve the right to modify or supplement opinions stated in this report.

** The views expressed in this report do not necessarily reflect those of the Harvard T.H. Chan School of Public Health, Harvard University, of which the author is affiliated as a Research Fellow.*

Technical Analysis of NJDWQI Proposed Health-Based Maximum Contaminant Level (MCL) for Perfluorooctanoic Acid (PFOA)

by

Cambridge Environmental Consulting

Executive Summary

We conclude that the proposed drinking water MCL of 14 ng/L for PFOA based on increased relative liver weight is not adequately protective of all population segments. The criterion may be developed on the basis of epidemiologic evidence of a significant immunotoxic association in children or, alternatively, evidence of significant adverse developmental effects shown in rodent studies. Both of these offer more sensitive endpoints with quantitative data to develop an MCL to assure greater health protection. We calculate an approximate MCL of 0.5 ng/L based on the BMDL determined and the association found between immune suppression and serum PFOA levels in children as reported by Grandjean and Budtz-Jørgensen, or an approximate MCL of 1.0 ng/L based on the BMDL determined in the delayed mammary gland developmental effects in mice studies. Alternatively, we calculate a MCL of 6 ng/L for children group ages 1-6 using the increased liver weight endpoint, with exposure values we determined for mean weight and 90th percentile water intake in that group. We propose that NJDWQI lower the proposed MCL to 1.0 ng/L, consistent with the values found pursuant to the immunotoxic epidemiologic study and/or animal studies showing adverse developmental effects. Excluding use of these values the MCL should be no greater than 6 ng/L to assure protection of children.

Introduction

This is a summary of our analysis and evaluation of the proposed health based maximum contaminant level (MCL) for PFOA in drinking water developed by the New Jersey Drinking Water Quality Institute (NJDWQI), as described in its report Health-Based Maximum Contaminant Level Support Document: Perfluorooctanoic Acid (PFOA), dated June 27, 2016, hereinafter referred to as the NJDWQI Report.

The presence of PFOA in New Jersey water supplies is of great concern because high concentrations are found in groundwater and surface water within the Delaware River Watershed and other locations in New Jersey. According to NJDEP database as of January 2016, of 72 public water supplies (PWS) tested 47% or 66 PWS were found contaminated with PFOA at levels equal to or exceeding the reporting limit (5 ng/l). Thirty-two PWS or 45% had levels exceeding 10 ng/l, and 12 PWS or 17% had levels exceeding 40 ng/l (NJDWQI 2016). Water tested at these PWS includes both raw and finished water; negligible to no removal of PFOA is achieved in the conventional water treatment systems used at these PWS. The affected population was not listed although we would expect it to exceed 1 million. NJDEP has not

published studies of PFOA in private wells, however 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).

Drinking water represents a significant portion of total human exposure to PFOA. The relative portion depends significantly upon the concentration of PFOA in drinking water. One study found that drinking water (at 9.66 ng/l) represented 24% of total exposure (Thompson et al 2011). Using NHANES 2003/2004 data, Lorber and Egeghy also determined a relative contribution of drinking water to total intake at 24%. They note that this rate is similar among adults and children (Lorber and Egeghy 2011). Others have found that drinking water represents a much higher portion of total exposure (Noorlander et al. 2011). A 20% contribution to total intake is used as a default value for relative source contribution (RSC) in this risk analyses.

PFOA exists predominantly in anionic form in drinking water sources. PFOA is non-volatile and therefore inhalation exposures to PFOA during showering and bathing and other domestic uses are negligible. PFOA does not cross the skin barrier and therefore PFOA is not absorbed into circulation via the skin, based on skin permeability of PFOA (Franko et al. 2012).

PFOA may escape water via aerosolization. In a laboratory study aerosols generated from deionized, fresh, and ocean waters spiked with PFO were found to have significantly higher concentrations of PFO than the parent water body, ≤ 80 times for ocean waters (McMurdo et al. 2008). Aerosols are produced by breaking waves on surface waters to generate air bubble beneath the surface which, when bursting at the surface, eject aerosol droplets into the atmosphere. This study also suggests that gas-phase evolution of PFOA from the aerosol-bound PFO into the atmosphere likely occurs due to the short aerosol-to-gas phase transfer half-life, about 7 seconds. Aerosol generation may also account for long-range air transport of PFOA, in addition to pathways of atmospheric transport of volatile precursors (8:2 FTOH) and transport of PFOA via the ocean.

Localized surface and groundwater PFOA contamination is primarily caused by wastewater discharges, air transport and deposition from PFOA emission sources, and groundwater plume migration. The extent of PFOA-laden aerosols via short-range air transport and potential direct exposure to humans is unknown but may help to explain, in part, PFOA concentrations in ground and surface waters in some locales proximate to factory sources, such as those found in Minnesota (Oliaei et al 2012).

We calculated that exposure to PFOA from drinking water source aerosols produced during typical showering conditions are likely to be negligible at a range of source water concentrations, based on equations we used in inhalation studies of aerosol particulates during showering (Cowen and Ollison, 2006; Zhou et al. 2010).

Calculation of MCL Using Quantitative Epidemiologic Data (Immunotoxicity)

The National Toxicology Program (NTP) supports a conclusion that PFOA alters human immune function (NTP 2016). A number of studies have shown PFOA immunotoxicity in that PFOA suppresses immune response. Four studies assessing associations with antibody concentrations following vaccination had prospective study designs that allowed temporality assessment. Among these, a prospective birth cohort study in Norway found strong evidence of decreased rubella-induced antibodies with increasing PFOA maternal serum concentrations in 99 pregnant women with a subsequent follow-up of 56 children at 3 years of age (Granum et al. 2013). Although no statistically significant associations were found with responses to vaccines for Influenza Type B or Influenza Type A H1N1, a large prospective cohort study of 411 adults in the mid-Ohio valley found decreasing antibody concentrations following Influenza A H3N2 vaccination (Looker et al., 2014). A large prospective cohort of 656 consecutive singleton births in the Faroe Islands with prospective follow-up of 587 cohort members at ages 5 and 7 years, found a strong association between serum PFC concentrations (PFOA and PFOS) and serum antibody concentrations against tetanus and diphtheria toxoids (Grandjean and Budtz-Jørgensen 2013).

The NJDWQI report acknowledged that "data from other human studies and toxicology studies provides support for biological plausibility of decreased immune system response to vaccines in humans" (NJDWQI Report 2016). The Report cites Fletcher et al. (2009), which "reported several statistically significant associations between several markers of immune function (decreased IgA; decreased IgE in females only; increased anti-nuclear antibody; decreased C-reactive protein) and serum PFOA levels in communities with drinking water exposure to PFOA in a C8 Science Panel status report" (NJDWQI 2016).

There is concordance with animal studies showing suppression of immune response. As noted in the NJDWQI report these include (in mice) decreased absolute and relative spleen and thymus weights, decreased thymocyte and splenocyte counts, decreased immunoglobulin response, and changes in total numbers and/or specific populations of lymphocytes in the spleen, thymus, peripheral blood, and bone marrow" (NJDWQI report).

NJDWQI notes that a "review of epidemiologic studies provides evidence of consistent findings among studies of decreased antibody concentrations following vaccination and PFOA. However, while there is epidemiologic evidence of temporality, evidence of an exposure-response is limited" (NJDWQI 2016). We disagree. We believe that where there is strong, significant epidemiologic evidence that includes quantitative data to enable derivation of a BMDL, such data should be taken into account in derivation of the MCL.

The Grandjean and Budtz-Jørgensen study represents the greatest sensitivity to PFOA thus studied, un-confounded by exposure to other chemical contaminants. In this study regression modeling of PFC concentrations (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, allowed determination of benchmark response (BMR) and benchmark dose (BMD).

The lower one-sided 95% CL (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, in spite of this the BMDL developed does provide a strong epidemiologic basis to develop a MCL.

Based on the immunotoxic effects shown in this study we propose that a 0.33 ng/ml BMDL for PFOA be used as a target human serum level. Assuming a serum:water ratio of 100:1 and an uncertainty factor (UF) of 10 to account for human variation in susceptibility, we calculate a MCL as follows:

$$\text{MCL} = \frac{0.33 \text{ ng/ml}}{\text{UF 10 x 100 serum:water ratio}} = \frac{330 \text{ ng/L}}{1000} = 0.33 \text{ ng/L (rounded to 0.5 ng/L)}$$

Alternatively, the NJDWQI methodology uses a clearance factor of 0.00014 L/kg/day to apply to the Target Human Serum Level. Using that methodology, a BMDL of 0.3 ng/ml as the POD (point of departure) for RfD determination, and a UF of 10 for human variation in susceptibility to determine the Target Human Serum Level, the RfD is:

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

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:

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

Based on the above we propose that the MCL for PFOA be 0.5 ng/L.

Calculation of MCL based on Delayed Mammary Gland Development (Animal Studies)

Delayed mammary gland development in mice resulting from developmental exposures to PFOA is a sensitive endpoint. This toxicity effect has been shown in nine different studies (NJDWQI report 2016). Delayed mammary gland development is especially concerning since adverse effects including histological changes 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). Mode of action is explained by Osborne: "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 is susceptible to carcinogens" (Osborne et al., 2015).

NJDWQI acknowledged these studies, which may result in increased susceptibility to cancer later in life. The NJDWQI states that "The Health Effects Subcommittee chose not to use this (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 lack of precedent for use of this endpoint as the primary basis for health-based criteria for environmental contaminants. Instead the Subcommittee arbitrarily applied an additional 10 UF to an unrelated endpoint (increased liver weight that forms the basis for their MCL derivation) to compensate for the more sensitive endpoint (delayed mammary gland development). This is confusing. 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?

We propose that the MCL be determined using the sensitive endpoint BMDL for delayed mammary gland development, clearance factor, and default adult exposure values per NJDWQI analyses, as follows:

Summary of variables used and values

BMDL	POD of 22.9 ng/ml (22,900 ng/L)
total UF	30 (10 human variation, 3 animal-to-human extrapolation)
RSC	0.20
clearance factor	0.00014 L/Kg/day
default adult body weight	70 kg per NJDWQI report
default adult intake	2.0 L per NJDWQI report

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

$$MCL = \frac{0.107 \text{ ng/kg/day} \times 70 \text{ kg} \times 0.2}{2 \text{ L/day}} = 0.75 \text{ ng/L (rounded to 1 ng/L)}$$

Based on the above we propose that the MCL for PFOA be 1 ng/L.

Children, PFOA Exposure, and Use of Adult Default Exposure Values

There is evidence that young children are exposed to greater levels of PFOA than adults. This

may occur because of age-specific behaviors such as hand-to-mouth behaviors resulting in greater ingestion of house particulates, and more time spent on floors with treated carpets. Using NHANES data, Lorber and Egeghy found that incidental ingestion of dust is far less important among adults than among children (Lorber and Egeghy 2011). Children's 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).

Peak serum PFOA concentrations occur during the first year of life, in part due to "off-loading" from the mother at birth. As noted in NJDWQI report, levels remain elevated for at least several additional years. Blood serum levels have been found to be higher in children. Higher serum levels were observed in children ages 2-5 versus older children and adults in Little Hocking, Ohio residents who have been exposed to PFOA in drinking water (Emmet et al 2006). Toxicity effects to children during this developmental period may persist into adulthood and become permanent.

Children therefore represent a special case. They have greater drinking water and food consumption on a body weight basis. Using adult default exposure values is inappropriate since a priori use of adult default values for body weight and water intake omits protection to children, the population's most vulnerable exposure group. Calculation of a MCL using adult default values results in a RfD to children (age group 1-6) that significantly exceeds that deemed allowable by NJDWQI based on the increased liver weight toxicity endpoint.

Although the MCL should be based on human immunotoxicity and/or the delayed mammary gland development shown in test animals, as calculated above, we believe that at a minimum MCL calculations using increased liver weight as an endpoint should be based on children exposure values for body weight and drinking water intakes. Using children group ages 1-6 we determined the MCL as follows:

Summary of variables used and values

BMDL	POD of 4351 ng/ml (4,351,000 ng/L)
CUF	300 (10 human variation, 3 animal-to-human extrapolation, 10 for delayed mammary gland development
RSC	0.20
Children body weight[a]	16.8 kg
Children intake[b]	0.69 L/day mean, 1.19 L/day 90th percentile

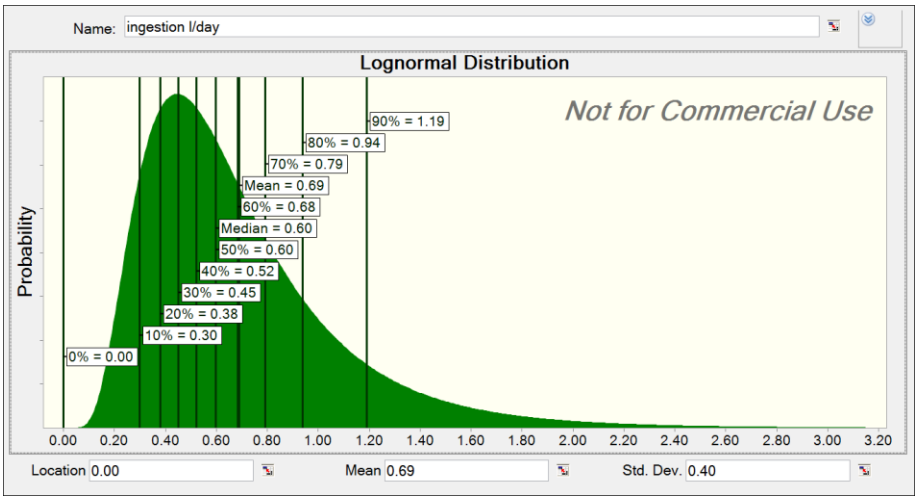
Children Group (age 1-6)

$$RfD = \frac{4{,}351{,}000 \text{ ng/L} \times 0.00014 \text{ L/kg/day}}{300 \text{ UF}} = 2.03 \text{ ng/kg/day}$$

$$MCL = \frac{2 \text{ ng/kg/day} \times 16.8 \text{ kg} \times 0.2}{1.19 \text{ L/day}} = 5.65 \text{ ng/L (rounded to 6 ng/L)}$$

a. These values were determined using EPA 2011 Exposure Factor Handbook data, 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.)

b. Following EPA's default criteria of 90th percentile distribution of water intake, we found a 1.19 L/day water intake rate for children 1-6 at the 90th percentile, based on derivation of a lognormal distribution of water intake for this combined age group, shown in the graph below.



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

Based on the above the MCL for PFOA should be 6 ng/L.

Conclusion

NJDWQI's Health Effects Subcommittee's work in developing a MCL for PFOA demonstrates a considerably sounder scientific basis than EPA's recent drinking water advisory for PFOA, where a 70 ng/L MCL is developed (USEPA 2016). However, NJDWQI's reliance upon Increased Relative Liver Weight in animal studies as an endpoint to develop a RfD disregards more sensitive toxicity endpoints. We believe that animal studies showing significant delayed mammary gland development are sufficient and appropriate to use in the MCL determination, irrespective of whether there is absence of precedence, where benchmark dose modeling allows calculation of an approximate MCL of 1 ng/L. Substantial epidemiological evidence showing a range of toxic effects should also be taken into account versus reliance solely upon animal studies. One such study, the Immunotoxicity study by Grandjean and Budtz-Jørgensen 2013 showing a significant association between PFOA and suppression of antibody responses in children, provides benchmark dose response data to calculate a MCL of ≤1 ng/L.

In addition, the proposed MCL of 14 ng/l calculated using adult default values for body weight results in a PFOA dose to children (ages 1-6) that is 50% higher at mean water intake levels, and 2½ times higher at 90th percentile water intake levels, than the reference dose (RfD) allowed to assure that serum levels remain below a protective maximum target level. Thus, the proposed

MCL of 14 ng/L using default adult exposure values is not protective of all age groups. This is concerning since, based on animal developmental studies that likely relate to humans, toxic effects from PFOA exposures in early childhood may persist into adulthood and could result in more profound disease in later life.

Absent lowering the proposed MCL to 1 ng/L, the MCL should be no higher than 6 ng/L.

** The views expressed in this report do not necessarily reflect those of the Harvard T.H. Chan School of Public Health, Harvard University, of which one of the authors is affiliated as a Research Fellow.*

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