May 6, 2015

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New Jersey Department of Environmental Protection
Trenton, New Jersey

Re: Proposed Health-Based Maximum Contaminant Level (MCL) for Perfluorononanoic Acid (PFNA, C9) in Drinking Water

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 on the Drinking Water Quality Institute's document **Proposed Health-Based Maximum Contaminant Level (MCL) for Perfluorononanoic Acid (PFNA, C9) in Drinking Water**. Also attached is a PDF 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 PFNA 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 Department of Environmental Protection and find that the proposed 13 ng/L is not protective. We support Dr. Oliaei's position that the MCL should be developed on the basis of a more vulnerable population segment (children). We support Dr. Oliaei's recommendation of 3 ng/L for age group 1 to 6 years using a 90th percentile water ingestion rate or 5 ng/l for age group 1 to 6 years using a mean water ingestion rate. We urge the Drinking Water Quality Institute and the Department to move forward with 3 ng/l or no greater than 5 ng/L based on Dr. Oliaei's analysis.

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

Sincerely,

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Attachments:

Technical Analysis of NJ Drinking Water Quality Institute Proposed Health-Based Maximum Contaminant Level (MCL) for PFNA in Drinking Water

Curriculum Vitae - Fardin Oliaei, MPA, PhD. and Don Kriens, Sc.D., P.E.

Technical Analysis of New Jersey Drinking Water Quality Institute

Proposed Health-Based Maximum Contaminant Level (MCL) for PFNA in Drinking Water

prepared by

Fardin Oliaei MPA, Ph.D.

Don Kriens Sc.D., P.E.

Cambridge Environmental Consulting

May 5, 2015

PREFACE

The opinions in this report are stated to a reasonable degree of scientific probability. The methods and principles 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.

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

by

Cambridge Environmental Consulting

Executive Summary

We conclude that the proposed drinking water MCL of 13 ng/L for PFNA is not protective. The criterion should be developed on the basis of a more vulnerable population segment (children), based on animal studies, and epidemiologic evidence that associate negative health effects in children due to PFNA exposures. We propose that the MCL at a minimum be revised to 5 ng/l based on children age group 1-6 (using a mean water ingestion rate), and preferably to 3 ng/l for children age group 1-6 (using a 90th percentile water ingestion rate). Using a 90th percentile ingestion rate is consistent with updated EPA default criteria applied to adult exposure assessments.

Introduction

This is a summary of our analysis and evaluation of the proposed health based maximum contaminant level (MCL) for PFNA 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: Perfluorononanoic Acid (PFNA), dated March 31, 2015, hereinafter referred to as the Report.

The presence of PFNA in New Jersey water supplies is of high concern because of unusual high concentrations in groundwater and surface water within the Delaware River Watershed. According to water sampling analysis conducted by the Delaware River Basin Commission (DRBC), PFNA had the highest concentrations of any PFCs sampled during monitoring of 2007-2009. The highest level of PFNA (976 ng/L) was found in the lower part of Delaware River at Paulsboro, near the Solvay plant. To our knowledge, this is the highest level of PFNA ever reported in surface water, worldwide. PFNA was also found at a very high level (96 ng/L) in a raw groundwater sample at the Paulsboro Water Department in 2009. In 2013 PFNA was found at 140 ng/L in raw water and 150 ng/L in finished water in this well in Paulsboro. To our knowledge this is these are highest levels of PFNA in drinking water reported in studies, worldwide.

Drinking water contamination is one of the most important PFNA human exposure routes. PFNA is known to be persistent and bioaccumulative with a long half-life in humans, and causes some toxic effects similar to PFOA, but at lower doses (ATSDR 2009). Human epidemiologic and animal data suggest potential health risks from drinking water exposures.
The MCL for PFNA was derived by quantifying a point of departure defined as the BMDL (benchmark dose 95th percentile lower confidence interval) in dose-response modeling, using pregnant mice PFNA serum levels (at selected PFNA dose) and increase in liver weights. The benchmark response used was a 10% increase in mean liver weight of pregnant control mice. The analysis is based on a study by Das et. al. 2015,

the only study available where dose-response data allows quantification of a BMDL (Das 2015). Using USEPA Benchmark Dose Modeling Software 2.40 a BMDL of 5200 ng/ml was selected as the POD, pursuant to finding the best fit of the model using statistical criteria in the BMD software. We concur with the approach used and also determined a 5200 ng/ml BMDL as a point of departure.

Uncertainty Factors (UFs)

In its interim draft PFNA groundwater criterion the New Jersey Department of Environmental Protection (NJDEP) had proposed a cumulative UF (CUF) of 300. This is revised in the NJDWQI MCL for drinking water to a CUF of 1000. This is based on a UF of 10 for intraspecies differences (human variation), a UF of 10 for extrapolation from non-chronic to chronic, a UF of 3 for incomplete database (notably for the lack of carcinogenic studies), and a UF of 3 (3.16) for extrapolation from animal to human (interspecies) for toxicodynamic differences.

Given that the target tissue is blood serum level we concur that toxicokinetic differences between species (human and test animals) is accommodated and therefore no UF is needed for toxicokinetic interspecies extrapolation. This is consistent with EPA's position: "interspecies differences in TK are defined as differences in the external dose
producing the same level of the dose metric in the target tissue of interest in test
animals" (USEPA 2014).

A UF of 10 for the lack of data versus a UF of 3 could be viewed as appropriate since there is a lack of carcinogenic test information. If we use a 10 for lack of data then the CUF would be 3000. However, uncertainty values chosen are inherently subject to bias and a resultant calculation can go both ways – either towards a conservative or a less conservative result. We have no scientific basis to assign a more conservative value for lack of data, underscoring use of professional judgment where a UF of 3 and 10 are often equivalently applied in risk assessments for lack of data. In this case we concur with a UF of 3 for lack of data and a CUF of 1000, which is consistent with CUF's commonly applied in other health risk assessments for non-carcinogenic endpoints.

Serum:Water Ratio

We disagree with the use of a central tendency (median) value for the serum:drinking water ratio, presumably using the PFOA ratio of 100:1 and multiplying by a factor of 2, based on limited data that the human half-life of PFNA is at least 2 times that of PFOA. NJDWQI Report also indicates that the half-life of PFOA is higher in children. The central tendency of 200:1 used here is inconsistent with upper percentile exposure values used by USEPA in its assessments. Therefore, 200:1 represents a less protective and non-conservative ratio.

Relative Source Contribution Factor

Although derivation of a RSC based on chemical-specific exposure data improves accuracy, we disagree with the basis used in NJDWQI's analysis to determine an RSC of 0.50 for PFNA. We find that potential PFNA exposures from local sources other than drinking water, such as locally grown vegetables, recreationally caught fish, and indoor contamination, in areas and regions with known PFC contamination, were not fully taken into account.

NJDWQI proposes to use the upper tail (95th percentile) of the U.S. population distribution of PFNA serum concentration (NHANES 2011-2012) as a surrogate for non-drinking water sources, including food, soil, air, water, and consumer products. Although the 95th percentile is an upper percentile of PFNA serum distribution in the normal population (uninfluenced by contaminated drinking water), it is not necessarily representative of individuals exposed to non-drinking water sources of PFCs in known "local" PFC contaminated regions/areas. The variability of national PFNA serum levels is likely due to within-population pharmacokinetic differences. Humans respond differently to the same or similarly dosed chemical exposures based on exogenous and intrinsic factors, as well as life stages, which would affect PFNA serum levels. Therefore, the 95th percentile serum PFNA may not be singularly representative of an upper level of serum concentrations associated with non-drinking water inputs of PFNA. In addition, the 95th percentile serum as a surrogate for non-drinking water inputs is very unlikely to be representative in areas where PFC contamination has been shown to be present.

NJDWQI formulates a basis that non-drinking water PFNA sources in the area/region are negligible because "the most recent data (PFNA analysis of white perch and channel catfish from locations on the Delaware River in the vicinity of communities where drinking water is contaminated with PFNA) do not suggest elevated exposures from recreationally caught fish in communities where PFNA is present in drinking water" (NJDWQI 2015). Based solely on this analysis NJDWQI assumes that the 95th percentile U.S. population PFNA serum level of 2.54 ng/mL represents a reasonable and protective estimate of total non-drinking water exposure. This reasoning is not supportable. First, we note that only two species (white perch and channel catfish) were tested for PFNA in the Delaware River, hardly representative of all recreational fish potentially contaminated with PFNA and consumed. Presumably, analysis was limited to white perch and channel catfish since they are on fish consumption advisories for other contaminants (PCBs) in the Delaware River. In fact, the Delaware River Basin Commission (DRBC) states that data collected for these fish are used to track the progress of PCB TMDLs established by the U.S. EPA in 2003 (DRBC 2012).

A number of fish species need to be tested in rivers to determine the extent of PFC contamination and risk to consumers. Researchers have found widely varying PFC levels in fish within and between species, and bioaccumulation factors for PFCs (PFOS) vary greatly from study to study and among species within studies (Oliaei 2006; MPCA 2010; Oliaei 2012). Researchers have found that PFC concentrations do not necessarily increase with trophic position. In Minnesota the following levels of increasing levels of PFOS have been found in some of the fish tested: (channel catfish < walleye < carp <bluegill < white bass < smallmouth bass) (McCann 2007). For example, bluegill in Mississippi River locations are generally low in environmental contaminants (PCB, Hg) but have relatively high PFOS levels, generally much higher PFOS levels than fish at higher trophic levels. In Alabama (Bakers Creek and the Tennessee River) PFOS in channel catfish were 7 to 886 times lower than PFOS found in largemouth bass (Sass).

NJDWQI analysis disregards other non-drinking water sources in its calculation of a RSC. In areas with known PFC contamination, researchers have also found a significant positive association between serum PFC (PFOA) levels and home-grown vegetable consumption after adjusting for water (PFOA) concentrations, suggesting that locally grown food may be an important source of exposure (Hoffman 2011). This association was also found in other studies (Bartell 2010; Steenland 2009). We would expect a similar pattern with PFNA.

It is illogical to conclude that the lack of PFNA in only two fish species tested is representative of all non-drinking water inputs (locally grown food, fish consumed, indoor air, etc.) in areas where known PFC contamination has occurred. An RSC of 0.50, based solely on the assumption that background U.S. PFNA serum levels (95th percentile) represent non-drinking water sources, is cursory and overlooks other potential local PFNA inputs.

We conclude that NJDWQI has not supported a data-driven RSC alternative to the default RSC of 0.20, and therefore the default RSC of 0.20 should continue be used in the MCL calculations, until such time data is available to formulate a data-driven RSC.

PFNA Toxicity and Unknowns

PFOA exposures have been associated (probable links) in epidemiologic studies with several health endpoints including increased cholesterol, ulcerative colitis, thyroid disease, reduction in vaccine response, and hyperuricemia (Steenland 2009; Steenland 2013; Lopez-Espinosa 2012; Steenland 2010; Looker 2013). PFCs, including PFOA and PFNA, have been found to be associated with a lower percentage of sperm with coiled tails, a measure of sperm quality (Louis 2015). *In utero* exposure to PFOA has also been found to be associated with lower adjusted sperm concentration and total sperm count (Vested 2013). Although PFNA is a close homologue of PFOA we do not know whether these human health endpoints are also associated with PFNA exposure.

We also do not know whether PFNA causes cancer in test animals because carcinogenic studies have not been undertaken, although PFOA and PFOS have been shown to cause tumors in rats (Sibinski, 1987; Biegel 2001; Thomford 2002). We do have epidemiologic evidence of significant associations between higher PFOA serum levels and testicular, kidney, prostate, and ovarian cancers and non-Hodgkin lymphoma (Vieira 2013). We also know that at least for some testing endpoints, such as reproductive and developmental, that PFNA, a one-carbon higher carboxylate than PFOA, is a more potent toxicant than PFOA (Das 2015; Wolf 2010), which, as the NJDWQI Report states, “is likely related to its greater intrinsic potency and longer persistence in the body” (NJDWQI 2015).

Studies that include PFNA have found it to be significantly associated with increases in total cholesterol. As stated in the NJDWQI Report; “epidemiologic data provide evidence of consistency, specificity, and exposure-response for PFNA and increased total cholesterol, although data on temporal relationship and strength of an association are limited.” The NJDWQI Report further states: “The possibility that PFNA causes increased cholesterol is further supported by evidence from epidemiology studies of PFOA, a closely related compound with similar toxicological effects. The epidemiology database for PFOA includes multiple studies of different designs in the general population, communities with drinking water exposure, and workers with occupational exposure, and suggests that a causal relationship may exist between PFOA and increased cholesterol” (NJDWQI 2015). We note that although a conclusion of causality between PFNA and increased cholesterol cannot be made since the evidence is based on epidemiologic studies, the evidence for the association is nevertheless very strong.

As discussed in the NJDWQI Report, although only one study found evidence of a significant positive association with thyroid stimulating hormone (TSH) and PFNA serum levels (Webster 2014), and 10 other studies found a null association, in our view the Webster study is more meaningful since it was a prospective birth cohort study. Considering the hierarchy of evidence, cohort studies, which establish a

temporal relationship between exposures and outcome, are considerably more valuable than cross-sectional studies in determining outcomes.

Applying the MCL to a Vulnerable Group

As stated in USEPA guidance, in part, sensitive life stages should be considered explicitly in the risk assessment when sufficient data are available (USEPA 2005).

PFCs have been shown to be significantly associated with some health effects in children. In a epidemiologic study by Lopez-Espinosa of thyroid function and PFAAs in children living near a chemical plant, serum PFOS and PFNA concentrations were significantly associated with slightly higher levels of thyroid hormone TT_4 in children 1-17 (Lopez-Espinosa 2012). For PFNA the study found that interquartile contrasts of 1.2 to 2.0 ng/mL were both associated with a 1.1% increase in TT_4 (95% CI: 0.7, 1.5) in children 1-17. The association remained after adjustment for PFOS, also found to be associated with increased TT_4 levels. It should be noted that increased TT_4 in this study was not associated with subclinical hypothyroidism. Yet an association between PFNA and increases in thyroid hormones is of concern, considering their importance to cognitive function in children and the adolescent brain. This study also found a significant association between thyroid disease (usually hypothyroidism) and serum PFOA levels (OR 1.44; CI 1.02, 2.03).

Other studies on children have shown health impacts from PFC exposures. Significant increases in total cholesterol and LDL cholesterol were linearly and positively associated ($p < .001$) with PFOA and PFOS serum concentrations in a large study of 12,476 children (Frisbee 2010). PFNA was not evaluated in that study. However, again, as noted in the NJDWQI Report "epidemiologic data provide evidence of consistency, specificity, and exposure-response for PFNA and increased total cholesterol" (NJDWQI 2015). It is reasonable, therefore, to expect similar associations in increased total cholesterol and PFNA exposures in children.

Given that the carcinogenic potential of PFNA is unknown and that the MCL is based solely on one dose-response study in pregnant mice, we believe that a margin of safety should be applied to the MCL derivation. This is further supported by a finding in another study (with no dose-response data) that suggests a more sensitive endpoint than increased liver weight in the mice study and greater toxicity to the liver (rats) due to histological changes, including necrosis (Stump 2008).

We believe that the MCL should be calculated on the basis of the more vulnerable group of children. This is based, in part, on the potential vulnerability to early childhood exposures of contaminants with later manifestation of health impacts, epidemiologic evidence that PFNA is associated with level of hormones in children, and our inability to observe and quantify developmental exposures and their impact on later life disease incidence.

Accordingly, we propose that the MCL be derived for a children group 1-6 as follows.

Proposed Revision for MCL

Summary of variables used and values

BMDL	POD of 5200 ng/ml
CUF	1000
RSC	0.20
Serum:water ratio	200:1
default adult body weight	80 kg per USEPA
default adult intake	3L at 90th percentile per USEPA
children body weight	16.8 kg
children intake	0.69 L mean, 1.19 L 90th percentile

Adult Calculation

Based on a BMDL (POD) of 5200 ng/l, cumulative UF of 1000, the default RSC 0f 0.20 versus a proposed RSC of 0.50, and a central tendency serum:water ratio of 200:1, we calculate an adult MCL as:

target serum level = $\frac{5200 \text{ ng/ml}}{1000 \text{ UF}}$ = 5.2 ng/ml

Increase in human serum level that can result from drinking water exposure only:

5.2 ng/ml x 0.20 RSC = 1.04 ng/ml (1040 ng/L)

MCL = $\frac{1040 \text{ ng/L}}{200:1 \text{ serum:water}}$ = **5.2 ng/L (5 ng/l) ADULT**

Children (age 1-6)

Using a 5.2 ng/L MCL adult, revised EPA default values of daily water intake at 3L/day (90th percentile) and adult mean body weight at 80 kg (EPA 2014), the daily allowable mass intake of PFNA is calculated as:

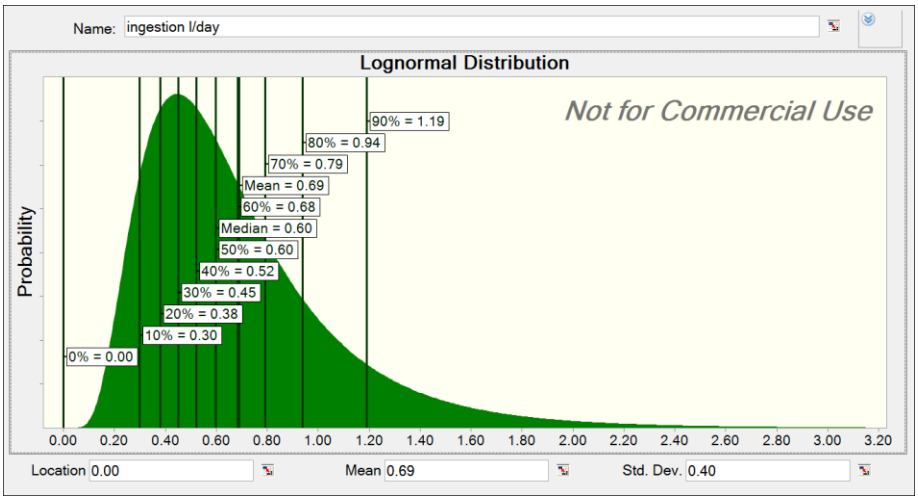
$\frac{5.2 \text{ ng/L x 3L/day default}}{80 \text{ kg x 0.20 RSC}}$ = 0.975 ng/kg/day allowable daily intake

To extrapolate to children age group 1-6 we use the same allowable mass intake of 0.975 ng/kg/day and calculate a MCL using a mean child body weight of 16.8 kg and mean child water intake of .69 L/day. (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.)

$\frac{0.975 \text{ ng/kg/day x 16.8 kg x 0.20 RSC}}{.69 \text{ L/day (mean value)}}$ = **4.75 ng/l MCL (5 ng/l) CHILDREN 1-6**

Following the EPA's default criteria of the 90th percentile distribution of water intake, we found a 1.19

L/day water ingestion rate for children 1-6 at the 90th percentile, based on our derivation of a lognormal distribution of water intakes for this combined age group, as shown in the graph below.



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

Accordingly,

$$\frac{0.975 \text{ ng/kg/day} \times 16.8 \text{ kg} \times 0.20 \text{ RSC}}{1.19 \text{ L/day } (90^{th} \%)} = \textbf{2.75 ng/l MCL (3 ng/l) CHILDREN 1-6}$$

We propose that the MCL be revised to 5 ng/l, and preferably to 3 ng/l, based on protection of children 1-6.

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PROFILE

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

EDUCATION

Harvard University School of Public Health, Boston, MA
Audited several courses: Air Pollution; Water Pollution; and Risk Assessment

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

Western Michigan University, Kalamazoo, MI
PhD in Environmental Sciences
- Dissertation title: Acid Rain and Lake Acidification Impacts on Aquatic Life

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

National University of Iran, Tehran, Iran
BS Chemistry, Minor Biology

PROFESSIONAL EXPERIENCE

Cambridge Environmental Consulting, LLC., Boston, MA 2006 - Present
Senior Scientist and President
- "Visiting Professor" at the Iranian National Institute of Oceanography (INIO) - conducted training workshops for INIO staff/scientist and coastal management professionals on the policy aspects of

coastal zone management and its implications. The training was tailored to the local cultural characteristics, government structure, resource integrity, and management needs of the country (2012).

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

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

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

Mote Marine Laboratory, Sarasota, FL 2007- 2008
Associate Scientist

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

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

Senior Scientist, Project Manager, and Emerging Contaminants Program Coordinator

- Developed policy, program analysis methods, and multimedia strategy to assess health impact of toxic chemicals.
- Initiated and led the Emerging Contaminants Program for the competent authority (MPCA).
- Prepared Environmental Impact Assessments (EIS) for major projects in MN and communicated the results, including the potential social, and economic impacts of these projects with authorities and public.
- Represented the MPCA as a scientific expert, liaison, and critical state contact in the PCBs, Dioxin, and emerging contaminants activities of the US EPA, Great Lakes Binational Strategy (GLBNS) and in other related national and international programs.
- Worked closely with diverse array of clientele and stakeholders (federal and state governments, industry, grass root organizations, affected communities, and the state legislators) to develop progressive environmental policies and educational materials.
- Presented at international conferences and gave presentations regarding environmental issues in public meetings, legislative hearings and governmental agencies.
- Managed contracts and secured federal/state grants and awards for health impacts of contaminant in Minnesota.
- Developed statewide air toxics monitoring/bio-monitoring network using mass balance and integrated air exposure-effect models.
- As the technical coordinator and MPCA liaison, built partnership between PCA and other sister agencies (MN Department of Health, MN Department of Natural Resources, and MN Department of Agriculture), USA EPA, and MN university researchers for ongoing efforts to identify, evaluate, control, regulate, and reduce the emerging pollutants with endocrine disruptive characteristics (PFOS and PFOA, PBDEs, and pharmaceuticals).
- Assessed the current regulations and programs already in place that may be addressing reduction of toxic contaminants of concern, identified unregulated emerging contaminants of greatest potential risk to human health and the MN environment, rationale of why these contaminants need to be regulated.

TEACHING EXPERIENCE

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

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

PROFESSIONAL AFFILIATIONS

• Member, **PCB Elimination Network (PEN)** of the Stockholm Convention	2011 - Present
• Member, **Society of Environmental Toxicology and Chemistry**	1990 - Present

- Member, Board of Directors, **Women's Environmental Institute** 2003 - Present
- Member, **Aquatic Biogeochemistry Research Group,** Harvard University, Harvard School of Public Health (HSPH) 2010 - 2012
- Member, **American Chemical Society** 1992 - 2010
- Member, **Air and Waste Management Association** 1998 - 2010

LANGUAGE SKILLS

- Fluent in English and Farsi (Persian)

PUBLICATIONS

- Brambilla, G., d'Hollander, W. Oliaei, F., Stahl, T., and Weber, R. Pathways and factors for food safety and food security at PFOS contaminated sites within a problem based learning approach, Accepted for publication at Chemosphere, 2014.
- Oliaei, F., Weber, R., Watson, A., and Kriens, D. Review of Environmental Releases and Exposure Risk of PFOS/PFAS Contamination from a PFOS Production Plant in Minnesota. Environmental Science and Pollution Research, 2013.
- Oliaei, F., Weber, R., and Watson, A. Landfills and Wastewater Treatment Plants as Sources and Reservoir of Polybrominated Diphenyl Ether (PBDE) Contamination. Environmental Science and Pollution Research, 2012.
- Weber, R., Watson, A., and Oliaei, F. *The Stockholm Convention Listing of New POPs – Implications and Follow Up Activities.* 11th International HCH and Pesticide Forum, Cabala, Azerbaijan, 2011.
- Oliaei, F., Weber, R., and Watson, A. *Landfills and Wastewater Treatment Plants as Sources of Polybrominated Diphenyl Either (PBDE) Contamination.* 11th International HCH and Pesticide Forum, Cabala, Azerbaijan, 2011.
- Oliaei, F., Weber, R., and Watson, A. Contamination of Drinking Water and the Environment by Production and Industrial Use of Perfluoroalkyl Compounds (PFCs). 11th International HCH and Pesticide Forum, Cabala, Azerbaijan, 2011.
- Weber, R., Watson, A., Forter, M., and Oliaei, F. *Persistent Organic Pollutants and Landfills – A Review of Past Experiences and Future Challenges.* Journal of Waste Management & Research, 29(1), 107-121, 2011.
- Oliaei, F., Weber, R., and Watson, A. *Presence of PBDEs in Minnesota Landfills – Environmental Releases and Exposure Potential.* Organohalogen Comp. 72, 1346-1349, 2010. http://www.dioxin20xx.org/pdfs/2010/10-1509.pdf
- Oliaei, F, Kriens, D, and Weber, R. *Investigation of PFOS/PFCs Contamination from a PFC Manufacturing Facility in Minnesota – Environmental Releases and Exposure Risks.* Organohalogen Comp. 72, 1338-1341, 2010. http://www.dioxin20xx.org/pdfs/2010/10-1507.pdf.
- Oliaei (2010), *Update on PFC Investigation and Health Risks*, http://www.w-e-i.org/update-pfc-investigation-and-health-risks-fardin-oliaei-2010
- Oliaei, F., and Kriens, D. *Environmental Releases of Perfluoroalkyl compounds from Two Landfills at the PFOS/PFC Production Site in Minnesota.* EPA – PFAA Day III, 2010.
- Oliaei, F., and Kriens, D. *Discovery of PFOS/PFC Contamination in Fish Near a PFOS/PFC Manufacturing Plant in Minnesota.* EPA – PFAA Day III, 2010.
- Oliaei, F., Kriens, D., and Kessler, K. *Perfluorochemical (PFC) Investigation in Minnesota: Phase One.* Minnesota Pollution Control Agency (MPCA). Legislative Report 2006. (79 pages).

- Oliaei, Fardin. *The presence and Distribution of Perfluorochemicals (PFCs) in Minnesota.* The EPA, Federal-State Toxicology and Risk Analysis Committee Meeting (FSTRAC), 2005.
- Oliaei, Fardin. *Flame Retardant: Polybrominated Diphenyl Ethers (PBDEs) in Minnesota.* Minnesota Pollution Control Agency (MPCA). Legislative Report 2005. (34 pages).
- Oliaei, Fardin. *The Presence and Distribution of PBDEs in MN's Landfills, Wastewaters and the Environment.* Minnesota Pollution Control Agency (MPCA). Annual Report of the Closed Landfill Program (CLP). 2004
- Oliaei, F., and Hamilton, C. *PBDE congener profiles in fish with different feeding behaviors from major rivers in Minnesota.* Organohalogen Comp. 64, 356-359, 2003.
- Oliaei, F., King, P., and Phillips, L. *Occurrence and Concentrations of Polybrominated Diphenyl Ethers (PBDEs) in Minnesota Environment.* Organohalogen Comp. 58, 185-188, 2002.
- Pratt, G., Oliaei, F., Wu, C., Palmer, K., and Fenske, M. *An Assessment of Air Toxics in Minnesota.* Environmental Health Perspective. 108(9), 815-825, 2002.
- Oliaei, Fardin. *Flame Retardants: Persistent, Bioaccumulative and Toxic Chemicals.* The EPA, Federal-State Toxicology and Risk Analysis Committee Meeting (FSTRAC). 2000.
- Oliaei, Fardin. *Toxic Air Pollutant Update.* Minnesota Pollution Control Agency (MPCA). 1999.
- Oliaei, Fardin. *Minnesota Air: Air Quality and Emissions Trends.* Minnesota Pollution Control Agency (MPCA). 1997, (215 pages).
- Pratt G., Gerbec, P., Livingston S., Oliaei F., Bollweg G., Paterson S., and Mackay D. *An indexing system for comparing toxic air pollutants based upon their potential environmental impacts.* Chemosphere 27(8), 1359-1379, 1993.

Don Kriens **Curriculum Vitae**

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Oakdale, MN 55128 dlk810@mail.harvard.edu

AREAS OF EXPERTISE

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

EDUCATION

HARVARD UNIVERSITY, Cambridge, MA
Sc.D. Environmental Health
Concentration - Exposure Sciences

HARVARD UNIVERSITY, Cambridge, MA
M.S. Environmental Health

UNIVERSITY OF IOWA, Iowa City, Iowa.
M.S. Environmental Engineering

UNIVERSITY OF IOWA, Iowa City, Iowa.
B.S. Sciences

AWARDS
Bush Foundation Leadership Fellow 2008
U.S. EPA Civil and Criminal Investigation Award
Harvard University Andelot Scholarship
Harvard University Water Initiative Fellow

PROFESSIONAL EXPERIENCE

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

Principal Engineer
- Lead agency technical expert for water projects. Mentor to engineers, hydro-geologists, and other technical staff.

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

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

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

TEACHING EXPERIENCE

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

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

University of Iowa Department of Civil and Environmental Engineering, Iowa City, Iowa
Research Scientist and Environmental Engineering Laboratory Supervisor
- Supervised laboratory conducting biological and chemical analyses, including GC and GC/MS. Conducted field studies. Occasional teaching assistant.

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

PAPERS AND PUBLICATIONS
Listing available on request