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VIA ELECTRONIC MAIL
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Attn: DEP Docket No. 02-19-03
Office of Legal Affairs
Department of Environmental Protection
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RE: COMMENTS ON NJDEP PROPOSED AMENDMENTS TO VARIOUS RULES, GROUND WATER QUALITY STANDARDS AND MAXIMUM CONTAMINANT LEVELS FOR PERFLUOROOCTANOIC ACID AND PERFLUOROOCTANESULFONIC ACID (DEP DOCKET NO. 02-19-03, PROPOSAL NO. PRN 2019-042)

Dear Mr. Knapick:

On behalf of our members, the Chemistry Council of New Jersey (CCNJ) appreciates the opportunity to provide the following comments to the New Jersey Department of Environmental Protection (NJDEP, the Department) on the proposed amendments to various rules, including Discharges of Petroleum and Other Hazardous Substances (DPHS) Rules, Ground Water Quality Standards (GWQS) Rules, Private Well Testing Act (PWTA) Rules, Safe Drinking Water Act (SDWA) Rules, and New Jersey Pollutant Discharge Elimination System (NJPDES) Rules, for Perfluorooctanoic Acid (PFOA) and Perfluorooctanesulfonic Acid (PFOS) published in the New Jersey Register on April 1, 2019.

CCNJ has serious concerns with the Maximum Contaminant Levels (MCLs) and GWQS proposed by the NJDEP, which include (but are not limited to):

- the Department's **failure to assess** whether the proposed MCLs and GWQS are "practical and feasible" for municipalities and affected communities;
- the Department's **inappropriate and overly conservative** basis for the proposed MCLs regarding the toxicity and potential for exposure to these substances;

- the Department's **failure to consider** information for the proposed MCLs that was submitted in response to the Drinking Water Quality Institute (DWQI) summary documents that are the basis of this rule proposal;

- the fact that the Department's proposed monitoring thresholds for the two substances are **below the reporting limits** of currently accepted detection methods, which may lead to the use of methods that have **not been sufficiently validated**; and

- the Department's **inaccurate and misleading** proposed language for consumer confidence reporting for public notification in the event of a detection of either of the two substances.

In addition, we continue to have concerns regarding the transparency of this rulemaking process. There was one (1) stakeholder meeting on January 18, 2019 to discuss rule concepts and issues related to the regulation of PFOA and PFOS. Specifically, the NJDEP discussed the addition of PFOA and PFOS to the DPHS List of Hazardous Substances; however, the Department did not discuss the addition of the acidic and anionic forms, as well as the salts and esters, of PFOA, PFOS, and Perfluorononanoic Acid (PFNA). Only the day before comments are due did the NJDEP provide CCNJ with a web link to the "extensive list of PFNA, PFOA, and PFOS salts and esters, as well as mixtures that contain these substances, that may be or have been used or stored commercially and industrially" (mentioned on page 35 of the rule proposal). Stakeholders were not given adequate time to thoroughly review and consider/evaluate the implications of this list.

Below are our comments in detail.

The NJDEP has not fully assessed the economic impacts of the proposed MCLs/GWQS.

In addition to the medical, scientific, and technological feasibility of the proposed MCLs, the SDWA also requires that the NJDEP consider the "limits of practicability and feasibility" when establishing standards for substances such as PFOA and PFOS. The Department estimates that 207 public water systems in the state have levels of PFOA above the proposed MCL, and that PFOS levels in 97 systems exceed the proposed MCL. Assuming that there is some overlap between the two lists, we conservatively estimate the total number of potentially affected systems to be in excess of 200. Based on the weighted average capital costs for treatment technology of $1.3 million estimated by the New York State Department of Health (NYSDOH),[1] total capital costs to comply with the proposed MCLs would exceed $260 million.[2] Based on the NJDEP's estimate of $80,000 per year, total operating costs would exceed $16 million annually. The Department's proposal makes no attempt to assess the impacts of these costs on the affected communities, nor does it provide sufficient information to allow stakeholders to estimate these impacts. While the draft indicates that the costs to comply with the proposed MCLs "will be ultimately passed on to consumers," it fails to consider what those costs may be and whether they are practicable and feasible.

Similarly, the NJDEP provides no estimate for how many water systems have already installed, or are installing, granular activated carbon (GAC) systems for the treatment of Perfluorononanoic Acid (PFNA)

[1] These estimates were presented at a meeting of the New York State Drinking Water Council held on December 18, 2018. A recording of this presentation can be found at the Council's website; the cost discussion occurs at minute 32 of the recording. https://www.health.ny.gov/environmental/water/drinking/dwqc/

[2] The NJDEP estimate of capital costs range from $0.5 to $16 million, depending on the size of the treatment plant. The estimate from the NYSDOH is on the lower end on this range, and may understate the costs of compliance with the NJDEP proposal.

and will, therefore, incur "little to no additional cost for the treatment of PFOA and/or PFOS."[3] In fact, the available evidence suggests that the number of systems incurring little to no cost would be small since PFOA and PFOS were not found at the four public water systems where PFNA was reported in the United States Environmental Protection Agency (USEPA)'s Unregulated Contaminant Monitoring Rule (UCMR) Occurrence Database.[4] The Department also provides no evidence to support its contention that "the costs of treatment are likely to decrease over time."[5]

Also, recommending one particular technology does not allow consideration of other equally suitable technologies that are readily available; for example, please refer to the following Interstate Technology & Regulatory Council (ITRC) comparison table: https://pfas-1.itrcweb.org/wp-content/uploads/2018/05/ITRCPFASFactSheetRemediationComparisonTablesApril18.xlsx. In addition, GAC is known to be less effective for removal of short chain PFAS. If additional criteria are formulated for short chain PFAS components after installation of GAC, a different treatment system may be selected. CCNJ recommends that the NJDEP remove the single technology reference and recognize that many new products are entering the market.

Since these capital and maintenance costs will ultimately be passed onto the customers of the water systems, it is imperative that the NJDEP evaluates how the cost of compliance with the proposed MCLs will impact the households served by the systems. In addressing the costs for individual households, the USEPA's National Drinking Water Advisory Council (NDWAC) recommends that a given drinking water standard be considered affordable if the annual cost per customer to meet the standard does not exceed 1.0 percent of the median household income for the median system in each drinking water system size category.[6] Without estimating the increased cost to households served by the affected water systems, the Department cannot determine whether its proposed MCLs are affordable, and thus whether they can be considered practicable and feasible.[7]

This rule proposal further estimates that nearly 5,500 active groundwater remediation sites[8] in the state could be potentially impacted by the proposed GWQS. Based on the NJDEP's estimate of the percent of public water systems that exceed the proposed MCL for PFOA (17 percent), one can estimate that more than 900 of the active sites will be required to conduct sampling, laboratory analysis, and treatment for PFOA and PFOS contamination. Although some of these sites may already be impacted by the groundwater standard for PFNA, the drinking water data suggest that the number of sites already conducting per- and polyfluoroalkyl substances (PFAS) remediation will be small.

The New Hampshire Department of Environmental Services (NHDES) estimates capital costs of up to $2.2 million for waste sites to treat PFOA and PFOS contamination, plus as much as $1.0 million in annual

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3 NJDEP Proposal No. PRN 2019-042, page 46

4 Unregulated Contaminant Monitoring Rule (UCMR) Occurrence Database. Occurrence data for the most recent data collection is available at https://www.epa.gov/dwucmr/occurrence-data-unregulated-contaminant-monitoring-rule#3.

5 NJDEP Proposal No. PRN 2019-042, page 46

6 USEPA. Recommendations of the National Drinking Water Advisory Council to U.S. EPA on its National Small Systems Affordability Criteria (July 2003). https://www.epa.gov/sites/production/files/2015-11/documents/report_ndwac_affordabilitywg_final_.08-08-03.pdf

7 It is also likely that the initial and ongoing sampling costs associated with the NHDES proposal will be passed onto customers and should be included in the NHDES' affordability calculation.

8 The proposed regulation indicates that 40 percent of the 13,707 active site remediation cases in the state (~5,500 sites) involved groundwater contamination.

maintenance costs.[9] Using these estimates, the total cost impact of the proposed groundwater standards is staggering – as much as $2 billion in capital costs and $900 million in annual operating costs.

Currently, the only practical groundwater remediation technology is pump-and-treat. It is indicated that, at active remediation sites, the new MCLs will be used as criteria. At all 14 sites investigated, PFOS was identified above the criteria and additional remedial effort is required. The data shows the widespread diffuse nature of these compounds and elevated background concentrations. Given the ubiquitous nature of PFOA and PFOS, no practical remedial endpoint can be achieved other than infinite pump-and-treat, and it will be infeasible in many cases to attribute PFAS components in groundwater to individual site owners and sources; we request that the NJDEP considers this consequence when adopting the MCLs as GWQS for active and new remediation projects.

In relation to this, it is noted that there is currently no groundwater in-situ treatment technology available that is proven at field scale, and groundwater remediation will rely on pump-and-treat systems. Given the ubiquitous nature of the compounds, large scale extraction of groundwater will be required, which may cause significant depletion of groundwater resources and may negatively impact groundwater resources. We request that the NJDEP considers both quality as well as quantity (volumes) aspects of groundwater resources when setting targets.

It is further noted that reduction to drinking water MCLs in many of these active remediation projects will not lead to further mitigation of human health or ecological risks, as the groundwater extracted and treated will be discharged and not used for drinking water purposes. CCNJ recognizes the importance of setting MCLs for drinking water and GWQS for PFOA/PFOS, but request that the NJDEP allows a site-specific risk-based approach for defining a remedial strategy for PFOA/PFOS in line with the ASTM RBCA approach. This process should balance risks for human health and ecology on the basis of both current and potential future uses of a site, recognizing the ubiquitous nature of PFAS.

In addition, given the extremely low proposed standards, operation of such a pump-and-treat system will result in true and significant economic hardship for small businesses. These materials were used by small businesses under the assumption that they were safe, and, in fact, were often used for the purpose of employee safety. System operation and treatment media disposal costs will be significant over time. We recommend that the NJDEP include hardship provisions in these rules to protect small businesses from financial ruin.

As with the consideration of the affordability of the proposed MCLs, the NJDEP must consider the potential cost impacts on residents for compliance with the groundwater standards. Many of the active remediation sites, and sites that will be subsequently identified, are owned by municipalities who will be required to bear the cost of compliance with the standards. They will, in turn, be required to pass those higher costs onto residents through higher local taxes or fees. Given the diverse and diffuse nature of the historic use of PFOA and PFOS, it often may not be possible to identify a responsible party.

[9] NHDES. Summary report on the New Hampshire Department of Environmental Services development of maximum contaminant levels and ambient groundwater quality standards for PFOS, PFOA, PFNA, and PFHxS. R-WD-19-01 (January 4, 2019). https://www.des.nh.gov/organization/commissioner/pip/publications/documents/r-wd-19-01.pdf

The NJDEP's proposed standards for PFOA are based on animal effects of questionable relevance to humans.

As noted in the DWQI summary document for PFOA[10] that is the basis for the proposed standards, systemic effects have been observed in experimental animals exposed to PFOA, including effects on liver, immune system, and developmental effects. However, not all of the observed animal effects are adverse, and not all animal adverse effects are relevant to humans.

Liver effects have not been reported in human studies.

Increased relative liver weight is a common effect of PFOA in animal studies that has been reported to occur at lower levels of exposure than those causing effects on other organ systems. Extrapolation of liver effects seen in animals to humans must be approached with caution, however, in light of the conclusions of the C8 Health Project and recent human data reported by Convertino *et al.* (2018) and strong evidence for rodent-specific adaptive responses.

The C8 Health Project is a large epidemiological study conducted in communities surrounding a manufacturing facility in Parkersburg, West Virginia that used PFOA from the 1950s until 2002. The study included over 32,000 adult residents and facility workers. The Science Panel formed as part of this project concluded that "there is not a probable link between exposure to C8 (also known as PFOA) and liver disease."[11]

The conclusions of the C8 Science Panel are supported by the recent work of Convertino *et al.* who reported no differences in clinical measures (including triglycerides, urea, glucose, AST, GGT, alkaline phosphatase, total bilirubin, fibrinogen, PTT and aPTT) at weekly PFOA doses as high as 1200 milligrams (about 16 milligrams/kilogram (mg/kg)), among a sensitive sub-population of cancer patients.[12] The authors concluded that the disparity between animal and human liver endpoint studies, emphasizing a lack of risk of human enlarged liver, fatty liver, or cirrhosis, can be attributable to mode of action differences. Increased liver weight due to hepatocellular hypertrophy can be an adaptive (protective) effect in animals due to up-regulation of detoxification enzymes, leading toxicologists to revisit key liver endpoint studies.[13] Research has shown that many metabolic effects of exposure to PFOA and PFOS in rodents can be explained by the activation of xenosensor nuclear receptors such as the peroxisome proliferator activated receptor (PPARα) in the liver.[14] These effects are of questionable relevance for risk assessment since the associated proliferative response in mice has not been observed in humans.[15]

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[10] NJ Drinking Water Quality Institute. Health-based maximum contaminant level support document: perfluorooctanoic acid (PFOA). Health Effects Subcommittee (February 15, 2017).

[11] The C8 Science Panel conclusions are summarized at http://www.c8sciencepanel.org/prob_link.html.

[12] Convertino M *et al.* Stochastic pharmacokinetic-pharmacodynamic modeling for assessing the systematic health risk of perfluorooctanoate (PFOA). *Toxicol Sci* 163(1) 293-306 (2018). https://academic.oup.com/toxsci/article/163/1/293/4865972

[13] Hall, A.P. et al. (2012). Liver Hypertrophy: A Review of Adaptive (Adverse and Non-Adverse) Changes-Conclusions from the 3rd International ESTP Expert Workshop. Toxicological Pathology. 40:971-994. https://journals.sagepub.com/doi/pdf/10.1177/0192623312448935

[14] See for example: Bjork JA *et al.* Multiplicity of nuclear receptor activation by PFOA and PFOS in primary human and rodent hepatocytes. *Toxicol* 288: 8-17 (2011).

[15] An understanding of the biological functions and role in chemical effects of PPARα has been facilitated by the use of a mouse model that lacks a functional PPARα (the PPARα-null mouse). Many of the effects of peroxisome proliferators have been shown to be mediated by PPARα as these effects were not observed in similarly treated PPARα-null mice.

The uncertainty regarding the relevance of the liver effects reported in animal studies to humans, notwithstanding, the study by Loveless *et al.* (2006) is not the most appropriate study for assessing liver effects in the laboratory studies. Although increases in hepatocellular hypertrophy and liver weight were observed at slightly lower doses in other animal studies, Perkins *et al.* (2004) is the more relevant study to use for this endpoint. One major advantage is that Perkins *et al.* is one of the few studies to report a no observed adverse effect level (NOAEL). The studies by Loveless *et al.* and most others did not identify a NOAEL. Instead, they were limited by their design and could only report a lowest observed adverse effect level (LOAEL), which means that further mathematical conversions (safety factors) to derive a NOAEL send the resulting level lower than necessary.[16]

A further advantage of the Perkins *et al.* study over the other low-dose studies is the longer duration of the study, with exposure durations of up to 13 weeks. In addition to *ad libitum* controls, moreover, the study provided pair-fed controls to ensure that effects did not result from differences in food consumption across dose groups. Finally, peroxisome proliferator activated receptor α (PPAR-α) activity was measured in the Perkins *et al.* study. This is important because it provides insight into a possible biological basis for the increase in liver weight. PPAR-α is a nuclear receptor and its activation is one possible mechanism for liver hypertrophy in rodents. However, in the Perkins *et al.* study, there was only a slight increase in PPAR-α activity at doses greater than 1.94 mg/kg per day (mg/kg/day) indicating that the hepatocellular hypertrophy observed was not resulting from peroxisome proliferation.

Since humans are much less responsive to PPAR-α activation than rodents, the findings from the Perkins *et al.* study are relevant to the assessment of health effects in humans. For the reasons mentioned previously (i.e. a human study that found no liver effects and the potential for hepatocellular hypertrophy to not be adverse), however, using the findings from the Perkins study should be considered extremely precautionary.

Benchmark dose (BMD) modeling of the data from Perkins *et al.* produces a reference dose (RfD) of 0.00015 mg/kg/day, nearly 30 times higher than the RfD derived from the Loveless *et al.* data.[17]

<u>Mammary gland and testicular cancer effects should not be used as a basis for proposed standards.</u>

In addition to assessing liver effects, DWQI considered health-based MCLs based on evidence of delayed mammary gland development and testicular cancer in laboratory studies. As indicated above, many metabolic effects of exposure to PFOA in rodents, including developmental effects, are associated with a proliferative response in mice that has not been observed in humans. While the study by Macon *et al.* (2011),[18] used by DWQI as the basis for an alternative RfD, observed a delay in mammary gland development in CD-1 mice, the results in other mouse studies are equivocal and support a PPARα-activated mechanism of questionable relevance to humans. Albrecht *et al.* (2013) did not find

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See Corton JC *et al.* Mode of action framework analysis for receptormediated toxicity: the peroxisome proliferator-activated receptor alpha (PPARα) as a case study. Crit Rev Toxicol 44(1):1-49 (2014).

[16] A similar NOAEL of 0.05 mg/kg/day can be obtained from Kennedy *et al.* (1987) when standard assumptions for food intake and bodyweight in rats are used, but the authors did not provide actual values of measured doses. Kennedy GL. Increase in mouse liver weight following feeding of ammonium perfluorooctanoate and related fluorochemicals. Toxicol Lett 39(2-3):295-300 (1987).

[17] USEPA. Health effects support document for perfluorooctanoic acid (PFOA). EPA 822-R-16-003. Office of Water (May 2016).

[18] Macon MB *et al.* Prenatal perfluorooctanoic acid exposure in CD-1 mice: low dose developmental effects and internal dosimetry. *Toxicol Sci* 122: 134-45 (2011).

alterations in mammary gland development in offspring of wild type, PPARα-null, or PPARα humanized mice following *in utero* exposure to PFOA.[19] In a multi-generational study in CD-1 mice, moreover, no clear dose-response was reported and the investigators noted that the delay in mammary gland development did not appear to affect lactational support based on normal survival and growth of the second generation (F2) offspring.[20]

In assessing the association between PFOA exposure and testicular cancer, DWQI focuses on the same study as that considered by the USEPA in its 2016 evaluation,[21] but generates a potency factor that is 36x lower than that calculated by the USEPA.[22] The disparity results from DWQI's decision to use a dose adjustment factor based on biological half-life rather than the default body-weight adjustment chosen by the USEPA. While DWQI cites the USEPA's cancer risk guidelines for the need to adjust for pharmacokinetic differences between species, it provides no rationale for abandoning the default approach, nor does DWQI attempt to compare its conclusion with that reached by the USEPA.

By adjusting the dose for biological half-life instead of body weight, DWQI's analysis places PFOA among the more potent chemicals for which cancer potency factors have been calculated.[23] Such a conclusion is not consistent with the animal data which suggest a modest cancer response in rats exposed up to 14.2 mg/kg/day, or with the information available from the C8 Health Project.[24] In its analysis, the C8 Science Panel noted that the association with testicular cancer was stronger in community residents than among workers, whose exposures were higher, and that there was little evidence of increasing risk among the residents when compared to the US population.[25]

Application of a database uncertainty factor is inappropriate.

In its analysis, DWQI includes a composite or total uncertainty factor (UF_{total}) of 300 in the derivation of the MCL for PFOA.[26] The proposed UF_{total} includes a 10-fold uncertainty to account for variability in susceptibility across the human population (UF_H), a factor of 3 to account for the toxicodynamic differences between humans and animals (UF_A), and an additional factor of 10 for database uncertainties (UF_D). The UF_{total} is applied to adjust the human-equivalent dose (HED) to add conservatism to the calculation of a RfD from which the MCL is calculated. According to its summary report, DWQI applied a UF_D of 10 to account for "sensitive effects that are not otherwise considered," specifically citing

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19 Albrecht PP *et al.* A species difference in the peroxisome proliferator-activated receptor α-dependent response to the developmental effects of perfluorooctanoic acid. *Toxicol Sci* 131:568–582 (2013).

20 White SS *et al.* Gestational and chronic low-dose PFOA exposures and mammary gland growth and differentiation in three generations of CD-1 mice. *Environ Health Persp* 119(8):1070–1076 (2011).

21 USEPA. Health effects support document for perfluorooctanoic acid (PFOA). EPA 822-R-16-003 (May 2016). https://www.epa.gov/sites/production/files/2016-05/documents/pfoa_hesd_final_508.pdf

22 USEPA calculates the cancer slope factor for PFOA to be to be 0.07 per mg/kg/day, versus DWQI's calculation of 2.52 $(mg/kg/day)^{-1}$.

23 The cancer slope factor of 2.52 $(mg/kg/day)^{-1}$ suggested by DWQI for PFOA exceeds that for all of the polychlorinated biphenyls (PCBs) and all but a handful of substances for which USEPA has estimated cancer potencies. http://www.popstoolkit.com/tools/HHRA/SF_USEPA.aspx

24 Barry V *et al.* 2103. Perfluorooctanoic acid (PFOA) exposures and incident cancers among adults living near a chemical plant. *Environ Health Persp* 121:1313-1318. https://ehp.niehs.nih.gov/doi/10.1289/ehp.1306615

25 Overall incidence of testicular cancer in the C8 population was below incidence within the US population.

26 DWQI. Health based MCL support document for PFOA, at 214

mammary gland development and hepatic toxicity not associated with liver weight.

According to the USEPA, the UF_D is intended to account for the potential for deriving an under-protective RfD as a result of an incomplete characterization of the chemical's toxicity. In addition to identifying toxicity information that is lacking, review of existing data may also suggest that a lower reference value might result if additional data were available. Consequently, in deciding to apply this factor to account for deficiencies in the available data set and in identifying its magnitude, the assessor should consider both the data lacking and the data available for particular organ systems as well as life stages.[27] An UF_D is generally applied when reproductive and developmental toxicity studies are missing since they have been found to provide useful information for establishing the lowest NOAEL.[28] If the RfD is based on animal data, a factor of 3 is often applied if either a prenatal toxicity study or a two-generation reproduction study is missing, or a factor of 10 may be applied if both are missing.[29]

The reproductive and development databases for PFOA are robust, however, and do not suggest the need to account for an incomplete characterization of toxicity. As discussed above, evidence of mammary gland developmental effects in mice are equivocal and support a PPARα-activated mechanism of questionable relevance to humans. Similarly, DWQI's concern about liver toxicity is misplaced in light of the available epidemiological evidence and the likely contribution of PPARα activation.

The NJDEP's assessment of PFOS ignores the conclusions of the USEPA and Health Canada.

As noted in DWQI's summary report,[30] which is the basis of the NJDEP's proposal, the USEPA issued a lifetime health advisory (LHA) of 0.07 micrograms per liter (µg/L) for PFOS in May 2016 under the federal SDWA.[31] In late 2018, Health Canada finalized its recommended maximum acceptable concentration (MAC) of 0.6 µg/L for PFOS in drinking water, which was originally proposed in 2016.[32] Both of these guidelines were developed after a careful review of the available animal and human evidence. Yet, DWQI's summary document dismisses these recommendations in lieu of a value based on inconsistent findings of immunotoxicity that have been thoroughly reviewed and rejected by both the US and Canada. In defending its conclusion, DWQI's primary rationale appears to be that "immune system toxicity is a more sensitive endpoint" than the effects used by USEPA and Health Canada."

CCNJ is deeply concerned with the DWQI's disregard for US and Canadian guidance and the best available science, and with its decision to base its proposal on the animal evidence for immunotoxicity without

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27 USEPA Risk Assessment Forum. A review of the reference dose and reference concentration processes. EPA/630/P-02/002F (December 2002). https://www.epa.gov/sites/production/files/2014-12/documents/rfd-final.pdf

28 Ibid, at 4-45.

29 Dourson ML *et al.* (1996) Evolution of science-based uncertainty factors in noncancer risk assessment. *Regul Toxicol Pharmacol* 24:108–120 (1996).

30 DWQI. Health-based maximum contaminant level support document: perfluorooctane sulfonate (PFOS). Health Effects Subcommittee (June 5, 2018).

31 USEPA. Drinking water health advisory for perfluorooctane sulfonate (PFOS). EPA 822-R-16-004 (May 2016). https://www.epa.gov/sites/production/files/2016-05/documents/pfos_health_advisory_final_508.pdf

32 Health Canada. Guidelines for Canadian Drinking Water Quality: Guideline Technical Document — Perfluorooctane Sulfonate (PFOS). Water and Air Quality Bureau, Healthy Environments and Consumer Safety Branch. Ottawa, Ontario. Catalogue No. H144-13/9-2018E-PDF. (2018). https://www.canada.ca/content/dam/canada/health-canada/migration/healthy-canadians/publications/healthy-living-vie-saine/guidelines-canadian-drinking-water-quality-guideline-technical-document-perfluorooctane-sulfonate/PFOS%202018-1130%20ENG.pdf

providing a substantive basis for asserting its significance to human health. It is neither sufficient nor appropriate for the NJDEP to base its proposed MCL on a recommendation for which DWQI acknowledges that it "does not understand the reasoning" behind specific criticisms of its approach offered by the USEPA.[33]

Animal immunological data are inconsistent.

Five studies have investigated potential effects on the immune system (natural killer (NK) cell activity and sheep red blood cell (SRBC) response) in mice exposed to PFOS.[34] Although the studies reported immune effects, the USEPA concluded that the differences in the levels at which effects were reported (and conflicts in the direction of the effects) "highlight the need for additional research to confirm the NOAEL and LOAEL for the immunological endpoints."[35] Health Canada reached a similar conclusion noting that "[f]urther exploration should be performed to address the nearly two orders of magnitude difference in LOAELs in the studies before these endpoints can be reliably considered as a basis for risk assessment."[36] The inconsistency of these study results is detailed below.

The 2008 study by Peden-Adams *et al.* (2008)[37] identified decreased SRBC response in male B6C3F1 mice exposed to 0.0017 mg/kg/day after 28 days of treatment, although no overt signs of toxicity were observed at doses up to 0.166 mg/kg/day. Additionally, the study observed enhanced NK cell activity at the lowest PFOS doses, but suppressed activity at higher doses. In the study by Keil *et al*., also published in 2008,[38] B6C3F1 mice exposed during gestation had decreased NK cell activity in males (at 1 mg/kg/day) and females (at 5 mg/kg/day) at postnatal week 8 – the opposite of the effect reported by Peden Adams *et al*. SRBC response was suppressed in males, but at doses several orders of magnitude higher (5 mg/kg/day) than in the study by Peden-Adams *et al.* No SRBC response was reported in females. A 2009 study by Zheng *et al.*[39] reported decreased NK cell activity in male C56BL/6 mice exposed to 1 mg/kg/day over 7 days. Additionally, SRBC response was observed in males at 5 mg/kg/day, consistent with the report from Keil *et al.*

In the mouse study by Dong *et al.* (2009),[40] NK cell activity was reported to increase at 0.083 mg/kg/day and to decrease at doses 10-fold higher (0.833 mg/kg/day) after 60 days. Decreased SRBC response also was reported in C57BL/6 males at 0.083 mg/kg/day, well below the LOAEL reported in the Keil study.

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33 DWQI. PFOS support document, at 312.

34 Immune effects in the lone rat study occurred at exposures several orders of magnitude higher than in the mouse studies (3.21 mg/kg/day). Lefebvre DE *et al.* Immunomodulatory effects of dietary potassium perfluorooctane sulfonate (PFOS) exposure in adult Sprague - Dawley rats. *J Toxicol Environ Health A* 71:1516-1525 (2008).

35 USEPA. Health Effects Support Document for Perfluorooctane Sulfonate (PFOS). EPA 822-R-16-202 (May 2016), at 4-7.

36 Health Canada. Guidelines for Canadian drinking water quality - PFOS (2018), at 69.

37 Peden-Adams MM *et al.* Suppression of humoral immunity in mice following exposure to perfluorooctane sulfonate. *Toxicol Sci* 104(1): 144–154 (2008).

38 Keil DE *et al.* Gestational exposure to perfluorooctane sulfonate suppresses immune function in B6C3F1 mice. *Toxicol Sci* 103(1): 77–85 (2008).

39 Zheng L *et al.* Immunotoxic changes associated with a 7-day oral exposure to perfluorooctanesulfonate (PFOS) in adult male C57BL/6 mice. *Arch Toxicol* 83(7): 679–689 (2009).

40 Dong GH *et al.* Chronic effects of perfluorooctanesulfonate exposure on immunotoxicity in adult male C57BL/6 mice. *Arch Toxicol* 83(9): 805–815 (2009).

In a subsequent study, however, Dong *et al.* (2011) observed no SRBC response at 0.0167 mg/kg/day.[41]

Human immunological data are inconsistent.

Five key epidemiology studies evaluated potential impacts of PFOS exposure on immune suppression (infectious disease and vaccine response). As with the animal data, the human data are inconsistent, as noted by Health Canada, which concluded that "associations are observed between PFOS levels and decreases in antibodies against some (but not all) illnesses and the influence of PFOS exposure on clinical immunosuppression (i.e., incidence of illnesses) appears to be more tenuous."[42] Health Canada further noted that, while the available animal and human data may indicate immune system changes, "it is unclear whether small variations in these measures are sufficient to result in adverse health effects in humans."

A study in children of the Faroe Islands found an inverse relationship in immune response with exposure to perfluorinated alkyl acids (Grandjean *et al.* (2012), Grandjean and Budtz-Jørgensen (2013)),[43,44] with maternal cord PFOS levels negatively correlated with anti-diphtheria antibody concentration at 5 years. Children in this population demonstrated increased odds of not reaching protective antibody levels for diphtheria after vaccination at 7 years old (Grandjean *et al.* (2012)). The relevance of these findings to other populations is questionable, however, as increased exposure to other potential immunosuppressants was not accounted for in the study.

Increased PFOS exposure was associated with decreased antibodies against rubella in children from a prospective birth cohort of pregnant women from Norway in a 2013 study by Granum *et al*.[45] In contrast, prenatal exposure to PFOS was not associated with hospitalizations for infections in a 2010 Danish cohort study by Fei *et al.*,[46] nor with episodes of common cold, gastroenteritis, eczema or asthma in the Norwegian cohort (Granum *et al.* (2013)). In a Taiwanese cohort study, the median serum PFOS concentration was significantly higher in asthmatic children (Dong *et al.* (2013)),[47] and prenatal exposure to PFOS was positively correlated with cord blood Immunoglobulin E (IgE) levels, particularly in male children. However, Wang *et al.* (2011)[48] found no association with atopic dermatitis. Cord blood IgE levels, food allergy, eczema, wheezing, or otitis media were not associated with maternal PFOS in female

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41 Dong *et al.* Sub-chronic effect of perfluorooctanesulfonate (PFOS) on the balance of type 1 and type 2 cytokine in adult C57BL6 mice. *Arch Toxicol* 85(10): 1235–1244 (2011).

42 Health Canada. Guidelines for Canadian drinking water quality - PFOS (2018), at 69.

43 Grandjean *et al.* Serum vaccine antibody concentrations in children exposed to perfluorinated compounds. *J Am Med Assoc* 307(4): 391–397. Comment in: *J Am Med Assoc* 307(18): 1910; author reply 1910–1. Erratum in: *J Am Med Assoc* 307(11): 1142 (2012).

44 Grandjean P and Budtz-Jørgensen E. Immunotoxicity of perfluorinated alkylates: calculation of benchmark doses based on serum concentrations in children. Environ. Health, 12: 35 (2013).

45 Granum B *et al.* Pre-natal exposure to perfluoroalkyl substances may be associated with altered vaccine antibody levels and immune-related health outcomes in early childhood. *J Immunotox* 10(4): 373–379 (2013).

46 Fei *et al.* Prenatal exposure to PFOA and PFOS and risk of hospitalization for infectious diseases in early childhood. *Environ Res* 110: 773–777 (2010).

47 Dong *et al.* Serum polyfluoroalkyl concentrations, asthma outcomes, and immunological markers in a case–control study of Taiwanese children. *Environ Health Perspect* 121(4): 507–513 (2013).

48 Wang Y *et al.* Modulation of dietary fat on the toxicological effects in thymus and spleen in BALB/c mice exposed to perfluorooctane sulfonate. *Toxicol Lett* 204(2–3): 174–182 (2011).

infants in a prospective cohort study of pregnant women in Japan (Okada *et al.* (2012)).[49]

Finally, a cohort of 411 adult members of the C8 Health Project in West Virginia was evaluated to determine whether there was an association between serum PFOS levels and antibody response following vaccination with an inactivated trivalent influenza vaccine (Looker *et al.* (2014)).[50] Vaccine response, as measured by geometric mean antibody titer rise, was not affected by PFOS exposure. After reviewing the available human data, Health Canada concluded the following:

> Although some effects on the antibody response have been observed, conflicting results were common in the dataset, which remains relatively small. A low level of consistency was observed across studies, with variations between genders, specific microbial immunoglobins, infections, mother vs. child exposure, and child years, amongst other characteristics. Moreover, the risk of residual confounding, bias, and chance cannot be discarded. These flaws impede concluding on a causative mechanism, and the nature of the association remains unclear.[51]

In considering these data, the USEPA cautioned that "lack of human dosing information . . . precludes the use of these immunotoxicity data in setting the [reference dose]."[52]

<u>The NJDEP's conclusions on the relevance of animal and human evidence are not supported by the available information.</u>

In support of the proposed MCL for PFOS, the NJDEP and DWQI assert the relevance of reduced SRBC response observed in mice to reduced resistance to infection in humans. Yet, the human studies generally report no increase in infection in children or adults, and both the USEPA and Health Canada have questioned whether the small variations in the antibodies observed in the available studies are sufficient to result in adverse health effects in humans. As the National Toxicology Program (NTP) notes in its review of PFOS, the "effects on diverse endpoints such as suppression of the antibody response and increased hypersensitivity may be unrelated."[53] Moreover, while asserting that the SRBC response in mice are "analogous" to decreased vaccine response in humans, the Committee offers no supporting information and neither the USEPA nor Health Canada have reached a similar conclusion.

The 2016 NTP systematic review of the animal data concluded that it cannot be confident in the outcome assessment of the Dong *et al.* 2009 study that is the basis for the proposed MCL.[54] As described above, the results of the NJDEP's key study conflict with those reported by other researchers and with a 2011 study conducted by the same research group. The decision to use the Dong *et al.* 2009 data is further invalidated by the results of DWQI's BMD modeling, which reveals that the SRBC response data failed to provide an acceptable fit to any of the dose-response models included in the USEPA's BMD software.

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[49] Okada E *et al.* Prenatal exposure to perfluorinated chemicals and relationship with allergies and infectious diseases in infants. *Environ Res* 112: 118–125 (2012).

[50] Looker C *et al.* Influenza vaccine response in adults exposed to perfluorooctanoate and perfluorooctanesulfonate. *Toxicol Sci* 138: 76–88 (2014).

[51] Health Canada. Guidelines for Canadian drinking water quality - PFOS (2018), at 40.

[52] USEPA. Health effects support document for perfluorooctane sulfonate (PFOS), at 4-7.

[53] NTP. Monograph on Immunotoxicity Associated with Exposure to Perfluorooctanoic acid (PFOA) or Perfluorooctanoic Sulfonate (PFOS). Office of Health Assessment and Translation. (September 2016), at 1.

[54] Ibid, at 133 (Appendix 3. Risk of Bias Heatmaps).

The inability of BMD modeling to yield a valid Point of Departure (POD) suggests that the SRBC response data reported in the Dong *et al.* 2009 study are not sufficiently robust.

The NJDEP's decision to focus on immune system effects as the basis for its proposed MCL runs directly counter to the specific concerns expressed about these data by both the USEPA and Health Canada. The Department's proposal offers little support for the relevance of the available animal and human data, which NTP is clear to caution may not be related to actual health effects in humans. It also fails to provide its rationale for selecting the SRBC response data from Dong *et al.* (2009) to generate the MCL when they conflict with findings reported by the same group in a subsequent study and by other researchers. The NJDEP's analysis is similarly silent on its inability to fit the SRBC data from Dong *et al.* (2009) to any of the dose-response models included in the USEPA's BMD software.

The NJDEP has underestimated the contribution of drinking water to overall exposure.

CCNJ is concerned with the extreme conservancy associated with the NJDEP's 20 percent RSC assumption. In developing the proposed MCLs, the Department assumes a relative source contribution (RSC) of 20 percent, despite acknowledging that PFOA and PFOS use has "decreased substantially."[55] Although 20 percent is often used as a default assumption for the exposure resulting from drinking water, the available evidence suggest that other sources of potential exposure to PFOA and PFOS have declined drastically. According to data collected by the Center for Disease Control and Prevention (CDC), mean serum levels of PFOS declined by 85 percent in the US population between 1999 and 2016.[56] According to CDC, mean serum levels of PFOA declined by 60 percent over the same timeframe (see Figure 1). Given those dramatic declines, it is inappropriate to assume that 80 percent of exposure to these substances comes from sources other than drinking water. While a few other states have assumed an RSC of 50 or 60 percent, it is likely that the contribution of drinking water to overall exposure is even higher, particularly in areas where drinking water contamination has been detected.

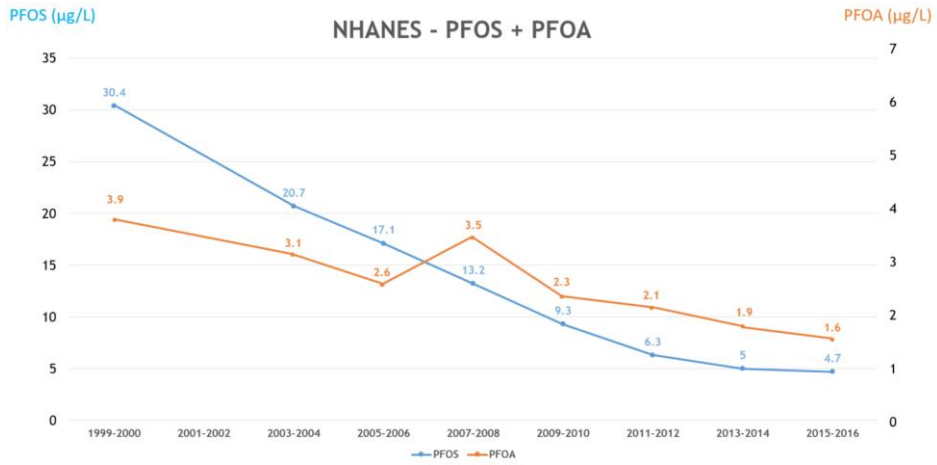

Figure 1. Serum levels of PFOA and PFOS, 1999-2016.[57]

[55] In fact, the manufacture of PFOA and PFOS has been eliminated in the US, Europe, and Japan, and imports of articles containing either substance have been significantly curtailed.

[56] CDC. Fourth national report on human exposure to environmental chemicals, update tables (January 2019). https://www.cdc.gov/exposurereport/index.html

[57] Human exposure monitoring is conducted as part of CDC's National Health and Nutrition Examination Survey (NHANES).

The proposed monitoring requirements for water utilities are inconsistent with current detection limits.

The NJDEP is proposing a monitoring threshold of 0.002 µg/L for PFOA and PFOS as part of the monitoring requirements for community water systems under N.J.A.C. 7:10-5.2(a)(5). While detection techniques and limits of detection will no doubt continue to improve over time, it is not clear that levels of these substances can be reliably detected at such a low level. For its latest national sampling results under the UCMR, for example, the USEPA listed minimum reporting limits of 0.01 µg/L or higher for these two substances. The most recent version of the USEPA's methodology for measuring PFAS in drinking water (Method 537.1) indicates that, while detection limits for the four substances range from 0.00053 to 0.0014 µg/L, "accurate quantification is not expected at [these] levels."[58]

More importantly, however, the NJDEP itself has determined practical quantification limits (PQLs) for PFOA and PFOS of 0.004 and 0.006 µg/L, respectively. The PQL is defined as the minimum concentration to which the contaminant can be reliably quantified within acceptable limits of uncertainty. It is not clear how community water systems would be able to comply with the proposed monitoring threshold that is below the practical detection limits determined by both the NJDEP and the USEPA. This is particularly problematic given the limitations of certified laboratories. Setting the threshold below the PQL will increase monitoring costs, generate inaccurate information, and likely encourage the use of unvalidated testing methods without providing any clear benefit.

The SDWA at N.J.S.A. 58:12A-13.b requires MCLs to be established within the limits of medical, scientific, and technological feasibility, with the DWQI Testing Subcommittee evaluating the limits of testing methodologies to ensure the levels consider these limitations. The USEPA's most recent PFAS methodology (537.1) reports the lower method detection limits, as the PQLs spelled out in the proposed rule are arrived at statistically and samples at these concentrations will likely not meet precision and accuracy criteria. Following the requirement of these PQLs will likely generate inaccurate data for local water purveyors and, further, it may drive laboratories to use unvalidated methods in an attempt to achieve reporting limits (leading to reporting data of unknown quality). Our recommendation is to set the threshold at 10 nanograms per liter (ng/L) or higher, allowing certified laboratories to follow validated testing methods.

The proposed Consumer Confidence Report language is inaccurate.

The proposed amendments to N.J.A.C. 7:10-5.2(b)(4) regarding the language to be included in Consumer Confidence Reports provided by community water systems that have detections of PFOA or PFOS contains several inaccurate statements about the potential health effects of the substances. The proposed language implies a level of certainty as to the causative nature of PFOA and PFOS exposure that does not exist. While notification of the public is an important aspect of ensuring the public's confidence in the drinking water supply, it is essential that the information provided be accurate and avoid inflammatory and misleading statements.

[58] https://www.epa.gov/newsreleases/epa-releases-new-tools-test-and-treat-additional-pfas-including-genx-drinking-water

Additional Comments

Appendix A List of Hazardous Substances – Addition of acidic and anionic forms, as well as salts and esters, of PFOA, PFOS, and PFNA

On April 25, 2019, CCNJ requested that the NJDEP provide the "extensive list of PFNA, PFOA, and PFOS salts and esters, as well as mixtures that contain these substances, that may be or have been used or stored commercially and industrially" (mentioned on page 35 of the rule proposal), which the Department stated they intend to make available electronically. On May 8, 2019, the NJDEP responded that the "Department intends to make available a finalized list of the multiple forms of PFOA, PFOS, and PFNA to the Department's Division of Science and Research website, in addition to sending it through the applicable Department listservs. Please note that, as indicated in the notice of proposal, in order to assist owners and operators of major facilities who store PFNA, PFOA, and PFOS in identifying related salts and esters that must be reported to the Department in accordance with the DPHS rules, the Department has developed an illustrative list of compounds identified as salts and esters of PFNA, PFOA, and PFOS. This list is not intended to be all-inclusive; regulated facilities must report all salts and esters of PFNA, PFOA, and PFOS regardless of whether they are included in the list. The Department will update this list as it becomes aware of additional compounds. The Department looks forward to receiving your comments on its proposed rules."

On May 30, 2019, the NJDEP sent an email to CCNJ with a web link to the aforementioned extensive list of PFNA, PFOA, and PFNA information, which they pointed out is located on the Department's Compliance and Enforcement page (vs. Science and Research). Unfortunately, we did not have sufficient time to review the list with our CCNJ members, and we request that the NJDEP provide an explanation regarding how an owner or operator is expected to test their materials to determine if they have a regulated PFNA, PFOA, and/or PFOS acidic/anionic forms, salts or esters.

De Minimis

CCNJ has concerns regarding products that contain de minimis levels of regulated compounds as these will likely increase with the addition of PFAS to the Discharge Prevention, Containment and Countermeasure (DPCC) list. De minimis levels have been an on-going issue in New Jersey for many years and, though the NJDEP has acknowledged that some regulated mixtures would actually be considered non-hazardous, the Department has never appropriately addressed this issue by developing de minimis exemptions. Regulating substances down to extremely low levels merely increases costs and administrative requirements while offering no additional protection to public health or the environment.

We would also like to highlight the potential DPCC compliance issue pertaining to de minimis if products do not identify that they contain low levels of PFAS compounds (e.g. paint, sealants, cleaning products) as Safety Data Sheets (SDS) generally will not include if they are only a small percentage of the product mixture.

NJPDES Permits

Revisiting the comments made above, and as the NJDEP has indicated in the proposal, many of the referenced compounds are ubiquitous in the environment and arise from various historical sources. Where the permitted discharge to groundwater is stormwater and not a process wastewater, the

presence of the referenced compounds could be unrelated to the permitted facility and due to off-site sources, such as precipitation, that are outside the control of the permitted facility. Their presence in groundwater might also be due to historical activities unrelated to the permitted discharge to groundwater. The permitted facility might, therefore, be held accountable for costly removal or treatment of the referenced compounds unrelated to the permitted discharge to groundwater. CCNJ recommends that these compounds not be incorporated into the NJPDES DGW permitting program at this time because of ubiquitous nature of these compounds and likelihood their presence would be unrelated to the permitted discharges to groundwater.

Regarding NJPDES permit application requirements, CCNJ recommends that the NJDEP not require monitoring if PFAS may only be expected due to their presence in source waters (i.e. none added through the actual manufacturing process).

In view of the possibility that these compounds may be present from sources other than the permitted discharge to groundwater, establishment of background concentrations of these compounds in background monitoring wells and statistical evaluations should also be considered and incorporated as appropriate into DGW permits. The NJDEP should at least allow for background concentrations and statistical evaluations, as warranted, in DGW Permits at facilities that are currently NJDEP Site Remediation projects and under Licensed Site Remediation Professional (LSRP) oversight.

N.J.A.C. 7:14A-4 Appendix A is applicable to all NJPDES permit applications. However, only Discharge to Groundwater (DGW) permitting is within the described intended scope of the proposed rules. The NJDEP did not consider the effect their rule proposal would have on other NJPDES permits and, therefore, must address the impact of the proposed rules on these other permits. If the proposed NJPDES rule amendments stay in, we recommend either adding wording to limit the scope to DGW permits or re-propose the rules with an analysis that addresses other affected types of permits.

NJPDES DGW permits, Category J (Surface Impoundment, Industrial) are associated with lined or unlined surface impoundments. Unlined surface impoundments have no permeability requirement and require groundwater monitoring to verify that pollutants are not discharged to groundwater above acceptable levels. Many unlined surface impoundments were not designed to transmit pollutants to groundwater but were instead designed to hold stormwater runoff from all or a portion of the facility prior to a discharge to surface water under an individual or general NJPDES DSW permit. Possible sources of impacted stormwater could include on-site and/or off-site soil and/or groundwater or precipitation. These facilities are typically already regulated under the NJDEP Site Remediation Program and are under the oversight of an LSRP, so many existing DGW permits specify "report only" for certain pollutants in unlined surface impoundments or groundwater monitoring wells located in the vicinity of unlined surface impoundments.

The costs associated with the design, construction, installation and permitting of a treatment system to remove pollutants from stormwater below the proposed GWQS could be extremely high. In particular, the treatment system would have to be designed and sized to address the maximum storm event. The treatment of generally uncontaminated stormwater that discharges to surface water at large facilities is not typical and is instead regulated through best management practices (BMPs) and design criteria under an individual or general NJPDES Discharge to Surface Water (DSW) permit. Requiring treatment for otherwise uncontaminated stormwater would require significant cost that was not considered in the supporting evaluation for the proposed rules. The need for treatment is best determined under the NJDEP Site Remediation Program rather than the DGW permitting program.

CCNJ recommends that the NJDEP exclude those DGW permitted facilities that are already addressing the referenced compounds in the NJDEP Site Remediation Program under oversight by an LSRP from the proposed rules. If the proposed rules are promulgated without modification, we recommend that the Department grant an additional three-year compliance schedule pursuant to N.J.A.C. 7:14A-6.4 to design, construct, install and permit a treatment system if it is determined through the DGW permitting process that removal of PFAS pollutants from the waste stream (i.e. stormwater) is warranted; we also recommend that the referenced compounds be specified in DGW permits as "report only" if the facility is under the NJDEP Site Remediation Program.

In addition, regarding inclusion of PFOA and PFOS in the NJPDES rules, there are currently no widely accepted analytical methods for groundwater and effluent matrices. USEPA Method 537.1 is a drinking water method that has not been developed and validated for the analysis of groundwater and effluents. These matrices are prone to interferences from other natural or manmade constituents. Given the proposed extremely low standards, the regulated community will not be able to provide reliable data of known quality in the absence of appropriate analytical techniques. The analytical determination error resulting from the application of inappropriate analytical techniques will render compliance with these standards impossible, and the NJDEP should wait until the USEPA has published appropriate analytical methods.

Other

In addition to the comments detailed above, CCNJ recommends that the NJDEP postpone the promulgation of their proposed MCLs and GWQS for PFOA and PFOS for the following reasons:

- The CDC and the Agency for Toxic Substances and Disease Registry (ATSDR) have just announced that they will be conducting exposure assessments in communities near current or former military bases and that are known to have had PFAS in their drinking water. This information will provide the basis for development of future studies on the effects of PFAS on human health, and the NJDEP should wait until a proper study of the effects of PFAS health is completed and the data have been properly evaluated.
- Detection of contaminants in groundwater in concentrations above the proposed standards will require remediation identification and remediation of the source area. Not only are there no widely accepted and USEPA-validated methods for analyzing environmental matrices other than drinking water, there are no standards or guidance values in place to guide remediation of source areas, including soil and waste materials. Furthermore, there are no widely agreed upon toxicological parameter values that can be used for the development of cleanup goals by the regulated community and the parties performing remediation. The NJDEP should wait until standards for all affected media are developed or, at least, until they develop toxicological parameters that can be used by the remediating party for the development of cleanup goals where corresponding Department standards do not exist.
- PFAS have been used in numerous consumer and industrial applications and have been identified in municipal drinking water systems and municipal wastewater treatment systems. Therefore, these compounds can be detected at facilities where they have never been used, but where they could have been introduced by use of contaminated municipal water, exfiltration from sewer lines, etc. The cost of addressing PFAS detections that are in no way related to facility operations can be significant and can result in hardship or even closure for small businesses. The NJDEP should wait until proper procedures for addressing such conditions have been developed.

We would like the record to reflect our support of any comments submitted by CCNJ members, as well as the American Chemistry Council.

Thank you for your consideration of our comments on this very important issue. We look forward to continuing to work with the NJDEP on this and other matters of critical importance to CCNJ members. If I can be of further assistance, please let me know.

Sincerely,

Dennis Hart
Executive Director