

SCHOOL OF PUBLIC HEALTH

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August 19, 2016

John Regan NH Department of Environmental Services 29 Hazen Dr.; P.O. Box 95 Concord, NH 03302-0095 john.regan@des.nh.gov

RE: Comments on Rulemaking Notice for Env-Or 603.03(b), (c) intro, & Table 600-1 pertaining to PFOA & PFOS

Dear Mr. Regan,

I appreciate the opportunity to comment on the proposed rule for PFOA & PFOS. I serve as a scientific advisor for the ATSDR Pease Advisory Panel and am an environmental epidemiologist at the Harvard T.H. Chan School of Public Health.

I fully support the adoption of an Ambient Groundwater Quality Standard (AGQS) for PFOS and PFOA in the State of New Hampshire as there is clear evidence that these contaminants are harmful to human health. However, I am concerned that the proposed AGQS may not be sufficiently protective for all health endpoints including immune suppression and cancer, for early life exposure, and of cumulative exposure to multiple perfluoroalkyl substances (PFASs) in addition to PFOA and PFOS.

It has come to my attention that the NJ Drinking Water Quality Institute (NJDWQI), an advisory body which recommends drinking water standards to NJDEP, conducted an extensive evaluation of current health effect information on PFOA and is developing recommendations for a health-based drinking water value. It is anticipated that the draft document presenting their health effects review and health-based drinking water level recommendation will be posted for public comment in the near future. I urge NHDES to consider the forthcoming report from NJDWQI in adoption of AGQS for PFOA and PFOS.

My concerns with the proposed AGQS are detailed in the attachment.

Thank you for the opportunity to comment on the proposed rule. Please feel free to contact me at <u>carignan@hsph.harvard.edu</u> if you have any questions or need further information.

Sincerely,

Courtney Carignan

Courtney Carignan, Ph.D. Postdoctoral Fellow

Comments

The proposed AGQS may not be sufficiently protective of immune function.

The proposed AGQS does not consider findings of a recent systematic review from the National Toxicology Program (NTP) of immunotoxicity associated with exposure to PFOA and PFOS (NTP 2016). The NTP concluded that PFOA is presumed to be an immune hazard to humans based on two separate lines of evidence: (1) the high level of evidence that PFOA suppressed the antibody response from animal studies and the moderate level of evidence from studies in humans, and (2) high level of evidence that PFOA increased hypersensitivity-related outcomes from animal studies and low level of evidence in humans. The NTP also concluded that PFOS is presumed to be an immune hazard to humans based on a high level of evidence that PFOS suppressed the antibody response from animal studies in humans.

The animal and epidemiologic studies taken together indicate a causal link between PFAS exposure and decreased immune function. The human data indicate that an AWQS of 1 ng/L (or 0.001 μ g/L) would be more appropriate for protection of immune function (Grandjean et al 2013).

The proposed AGQS may not be sufficiently protective of early life.

The proposed AGQS does not consider the neonate, infant or fetus as a susceptible subpopulation. It is important to consider exposures during this timeframe because developmental effects (i.e., immune function) are known to be sensitive endpoints for the toxicity of PFOA and PFOS. Additionally, drinking water exposures for this subpopulation are higher than for adults because of the higher intake per unit of body weight. For example, serum levels in breast-fed infants whose mothers drink contaminated water, and infants consuming formula prepared with contaminated water, will be much higher than in the mother or other adults consuming the same water. PFASs are present in breast milk, and infants and children consume much more liquid on a body weight basis than adults (Fromme et al. 2010; Mogensen et al. 2015; Verner et al. 2016).

The proposed AGQS may not sufficiently consider persistence.

The proposed AGQS does not consider the increased persistence (longer biological halflives) of PFOS and PFOA in extrapolating from animal studies to humans. With ongoing drinking water exposure there is an expected 100 to 1 ratio between PFAS concentrations in serum and drinking water (Post et al. 2012; NJDWQI 2016). For example, chronic exposure to drinking water with PFOA concentrations at the proposed AGQS of 0.07 $\mu g/L$ (70 ng/L) is expected to result in an average increase of at least 7 ng/ml in blood serum. This can be compared to the median serum PFOA concentration from the most recent NHANES of ~2 ng/ml. This increase is expected to be even greater for PFOS, which has a longer half-life than PFOA.

The proposed AGQS may not be sufficiently protective against cancer.

The proposed AGQS does not appear protective against cancer based on findings from epidemiologic studies conducted in populations exposed to PFASs in drinking water such as Barry et al. (2013) and Vieira et al. (2013).

The proposed AGQS may not be sufficiently protective against cumulative effect of PFASs.

The proposed AGQS does not consider exposure to multiple PFASs other than PFOS and PFOA. Given the similar structure of PFASs and that they often co-occur at contaminated sites, it is precautious to assume they can have cumulative effects on health.

References

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