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CATHERINE R. McCABE
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November 8, 2018

Sarah Pillsbury, Administrator
Drinking Water and Groundwater Bureau
New Hampshire Department of Environmental Services
Sarah.Pillsbury@des.nh.gov
RE: 10/3/18 NHDES request for input on setting MCLs for PFOA, PFOS, PFNA and PFHxS

Dear Ms. Pillsbury,

On October 3, 2018, NHDES requested technical input on setting Maximum Contaminants Levels (MCLs) for four long-chain perfluoroalkyl acids (PFAAs) - PFOA, PFOS, PFNA and PFHxS. Information was requested on approaches, data, and studies to be considered in MCL development; health effects studies and data not considered in the ATSDR (2018) Draft Toxicological Profile for Perfluoroalkyls or the USEPA (2016) PFOA and PFOS Health Advisories; and data and methodologies relevant to costs and benefits for MCLs.

The New Jersey Department of Environmental Protection (NJDEP) and the New Jersey Drinking Water Quality Institute, a legislatively-established advisory body to NJDEP, have extensively evaluated the scientific information relevant to development of MCLs for PFOA, PFOS, and PFNA, including detailed reviews of the ATSDR Draft Toxicological Profile and USEPA Health Advisories mentioned by NHDES. Citations for NJDEP and DWQI documents and peer-reviewed publications on these topics are listed at the end of this letter. The major NJDEP conclusions are shown in the attached PowerPoint presentation, which was excerpted from recent longer NJDEP presentations. These conclusions are summarized below, with citations of publications not considered by ATSDR (2018) or USEPA (2016) marked in **bold**.

General approach and conclusions about risk assessment and MCL development for long-chain PFAAs

NJDEP's general approach and overall conclusions about human health risk assessment and MCL development for long-chain PFAAs such as PFOA, PFOS, and PFNA are summarized in *Slides 3 - 14* of the attached PowerPoint presentation. These conclusions are discussed in more detail in a recent publication by **Post et al. (2017).**

In summary, NJDEP concludes that there is a need for caution about exposure to long-chain PFAAs from drinking water. Unlike other well-known persistent, bioaccumulative and toxic (PBT) chemicals such as PCBs and dioxins, long-chain PFAAs are water soluble and drinking water is an important exposure route. Ongoing ingestion of even low drinking water concentrations of these PFAAs (e.g. well below the USEPA Health Advisory of 70 ng/L) overwhelms exposures from other sources (primarily food and consumer products) prevalent in the general population. Infants, a sensitive subpopulation for developmental effects of PFAAs, receive higher exposures from breast milk or prepared formula than adults using the same contaminated drinking water source. Because these PFAAs have long human half-lives (several years), body burdens remain elevated for many years after exposure to contaminated drinking water ends.

The substantial increases in blood serum levels from low drinking water concentrations are of concern because long-chain PFAAs are associated with human health effects at blood serum levels prevalent in the general population, even without additional drinking water exposure. Although limitations in the epidemiological data preclude their use as the basis for quantitative risk assessment, these human data provide support for a public health protective approach in development of MCLs based on animal data. Long-chain PFAAs cause multiple toxicological effects in laboratory animals, including some at low doses. Evaluation of mode of action data indicates that these toxicological effects are adverse and relevant to humans. In risk assessment of long-chain PFAAs, animal-to-human extrapolations must be based on internal doses (e.g. blood serum levels), not administered doses, because of the same administered dose results in a much higher internal dose in humans than in animal species.

Factors considered in Development of New Jersey Recommended MCLs for PFOA, PFOS and PFNA

The NJ DWQI developed MCL recommendations of 13 ng/ml for PFNA in 2015, 14 ng/L for PFOA in 2017, 13 ng/L for PFOS in 2018. The MCL for PFNA was adopted by NJDEP in September 2018, and the MCL recommendations for PFOA and PFOS are currently used by NJDEP as guidance for public water systems with detections of these contaminants.

As shown in *Slide 3* of the attached PowerPoint presentation, the DWQI MCL recommendations considered three factors: health effects (Health-based MCL), analytical limitations (Practical Quantitation Level; PQL), and availability of drinking water removal treatment methods. For all three of these PFAAs, achievement of the Health-based MCL was not limited by analytical or treatment removal factors, and the Health-based MCL was therefore recommended as the MCL. Links to DWQI MCL recommendations and technical reports on the health effects, analytical and treatment removal considerations for PFOA, PFOS, and PFNA are provided in the citation list below.

Human Health Risk Assessment for PFOA, PFOS, PFNA and PFHxS

PFOA

The basis for the New Jersey Health-based MCL for PFOA is presented in the **NJ DWQI (2017)** document, "Health-Based Maximum Contaminant Level Support Document: Perfluorooctanoic Acid (PFOA)." A detailed review of the basis of the USEPA Health Advisory for PFOA is found in Appendix 2 of DWQI (2017). NJDEP comments on the ATSDR (2018) Draft Toxicological Profile include comments on PFOA. Links to these NJDEP and DWQI documents are provided in the citation list below. The NJDEP approach and conclusions for PFOA risk assessment are summarized on *Slides 16 - 23* of the attached PowerPoint presentation.

Two Reference Doses (RfDs) were developed for PFOA by DWQI (2017). The first RfD is based on delayed mammary gland development from developmental exposure in mice. This was the most sensitive endpoint for PFOA that provided dose-response data needed for RfD development. This effect is well established, as it was observed in nine separate studies and in two strains of mice (reviewed in detail on p. 130-136 of DWQI, 2017). It is considered to be adverse because structural changes in the mammary gland persisted until adulthood, and there is no reason to discount its human relevance. Furthermore, three human studies report that PFOA is associated with decreased duration of breastfeeding. As noted in NJDEP comments to ATSDR, one of these studies, **Timmermann et al. (2016)** was not cited by ATSDR. NJDEP disagrees with the USEPA and ATSDR rationales for dismissal of this endpoint from consideration for risk assessment; see DWQI (2017 - Appendix 2, p. 9-10) and p. 6-7 of NJDEP comments on the ATSDR Draft Toxicological Profile.

As presented in a peer-reviewed publication (**Post et al., 2012**), BMDLs were developed for 10% decreases in mammary gland developmental score and number of terminal endbuds (a quantitative parameter) in gestationally exposed offspring from Macon et al. (2011). The RfD based on these BMDLs is 0.11 ng/kg/day, and the Health-based MCL based on this RfD would be 0.77 ng/L. Although this endpoint and RfD were judged to be scientifically valid, this Health-based MCL was not recommended because there is no precedent for use of this endpoint as the primary basis for risk assessment.

A second RfD of 2 ng/kg/day was based on increased liver weight in mice, with an uncertainty factor of 10 for more sensitive developmental effects including delayed mammary gland development and persistent liver toxicity. A detailed review of PFOA's hepatic effects (beginning on p. 111 of DWQI, 2017) concludes that increased liver weight caused by PFOA is adverse because it co-occurs with and/or progresses to more severe types of hepatic toxicity. For this reason, NJDEP does not agree with the USEPA and ATSDR conclusion that increased liver weight from PFOA is reversible and not adverse based on the criteria of Hall et al. (2012). Additionally, a detailed review of mode of action data concludes that these hepatic effects of PFOA are relevant to humans. The NJDEP conclusions about mode of action and adversity are discussed on p. 10-14 of

the NJDEP comments on ATSDR (2018) Draft Toxicological Profile and on p. 181-190 of DWQI (2017). The Health-based MCL based on the RfD of 2 ng/kg/day is 14 ng/L.

As shown on *Slide 22* of the attached PowerPoint presentation, PFOA was classified as having suggestive evidence of carcinogenicity by NJDEP and USEPA. NJDEP developed a cancer slope factor of 0.021 $(mg/kg/day)^{-1}$ for testicular tumors in male rats from Butenhoff et al. (2012a). The Health-based MCL based on this slope factor and the one-in-one million (1×10^{-6}) risk level specified in the NJ Safe Drinking Water Act (N.J.S.A. 58: 12A) is 14 ng/L, identical to the value based on non-carcinogenic effects.

Blood serum PFOA levels are expected in increase by about 5-fold from the median U.S. level from exposure to the USEPA Health Advisory of 70 ng/L and about 2-fold from exposure to the NJ MCL of 14 ng/L; see Appendix 2, p. 8 and 13 of DWQI (2017) and *Slide 23* of attached PowerPoint presentation. DWQI (2017) concluded that since "several health effects, some with evidence supporting multiple criteria for causality, are associated with PFOA exposures at serum levels well below those that would result from exposure to 70 ng/L in drinking water," "elevations in serum PFOA levels of the magnitude expected from ongoing exposure to 70 ng/L (the USEPA Health Advisory) in drinking water are not desirable and may not be protective of public health."

PFOS

The basis for the New Jersey Health-based MCL for PFOS is presented in the **NJ DWQI (2018)** document, "Health-Based Maximum Contaminant Level Support Document: Perfluorooctane Sulfonate (PFOS)." A detailed review of the basis of the USEPA Health Advisory for PFOS is found in Appendix 2 of DWQI (2018). NJDEP comments on the recent ATSDR PFOS risk assessment are included in NJDEP comments on the ATSDR (2018) Draft Toxicological Profile for Perfluoroalkyls. The NJDEP approach and conclusions for PFOS risk assessment are summarized on *Slides 25 - 27* of the attached PowerPoint presentation.

Suppression of immune response to a foreign antibody, as indicated by decreased plaque forming cell response, was identified by DWQI (2018) and NJDEP (**Pachkowski, 2018**) as the most sensitive toxicological endpoint for PFOS. The NJDEP RfD for PFOS of 1.8 ng/kg/day is based on suppression decreased plaque forming cell response in mice in Dong et al. (2009), and the basis for this RfD is presented in a recent peer-reviewed publication (Pachkowski et al., 2018). Decreased plaque forming cell response in mice has been reported in four studies of PFOS. It is well-established as an endpoint for risk assessment was used as the basis for RfDs developed by USEPA IRIS. As discussed in DWQI (2018) and Pachkowski et al. (2018), associations of PFOS with decreased vaccine response (the analogous human effect) and increased incidence of infectious disease support the human relevance of this effect.

Suppression of immune response has also been identified as a sensitive effect of PFOS by several other federal, state and international agencies and academic researchers including **NTP (2016)**, ATSDR (2018), **MDH (2017)**, **EFSA (2018- draft)**, **Dong et al. (2017)**, and **Lilienthal et al.**

(2017). DWQI (2017 – Appendix 2, p. 305-306) and NJDEP (Pachkowski et al., 2018) conclude that USEPA did not provide a supportable rationale for use of a less sensitive endpoint, decreased rat offspring body weight (Luebker et al., 2005), as the basis for their PFOS RfD.

The NJDEP comments on ATSDR (2018) note that **Keil et al. (2008)** was not included in the discussion of studies decreased plaque forming cell response in mice caused by PFOS, two recent studies, **Impinen et al. (2018)** and **Goudarzi et al. (2017)** that reported epidemiological associations of infectious disease with PFAAs, including PFOS, were not cited.

PFOS was classified as having suggestive evidence of carcinogenicity by NJDEP and USEPA. DWQI (2018) developed a slope factor based on the incidence of hepatocellular tumors in female rats in Butenhoff (2012b). Although the slope factor was judged too uncertain to use as the basis for the MCL, the cancer risk at the MCL of 13 ng/L based on the RfD for immune system suppression was estimated as 3 in one million, which is close to the New Jersey cancer risk goal of one-in-one million.

Blood serum PFOS levels are expected in increase by about 3.7-fold from the median U.S. level from exposure to the USEPA Health Advisory of 70 ng/L and about 1.5-fold from exposure to the NJ MCL of 13 ng/L; see DWQI (2018) - Appendix 2, p. 304 and p. 311, and *Slide 27* of attached PowerPoint presentation. DWQI (2018) concluded that "there is substantial evidence from epidemiology studies that decreased vaccine response occurs at levels of serum PFOS prevalent in the general population," and that "exposure to PFOS in drinking water at the USEPA Health Advisory of 70 ng/L is predicted to increase serum PFOS concentrations to the upper end of this range and higher. Therefore, the magnitude of elevations in serum PFOS levels expected from ongoing exposure to PFOS in drinking water at the USEPA Health Advisory level are not desirable and may not be protective of public health."

PFNA

The basis for the New Jersey Health-based MCL for PFOA is presented in the **NJ DWQI (2015)** document, "Health-Based Maximum Contaminant Level Support Document: Perfluorononanoic Acid (PFNA)," and in the NJDEP PFNA MCL rule proposal (**NJDEP, 2017**) and adoption (**NJDEP, 2018**) documents. NJDEP approach and conclusions for PFNA risk assessment are summarized on *Slides 29 - 39* of the attached PowerPoint presentation.

In summary, toxicological database for PFNA is considerable and is sufficient for development of a Health-based MCL. PFNA causes hepatic, immune system, developmental, renal, and male reproductive toxicity; chronic carcinogenicity studies have not been conducted. The toxicological effects and mode of action of PFNA are generally similar as PFOA. However, as compared to PFOA, PFNA is more biologically persistent (longer half-life), and its effects occur at lower doses and are more severe, in some cases.

Based on rodent half-life studies and limited human half-life data based on urinary excretion, the human half-life of PFNA was estimated to be twice that of PFOA. As is the case for PFOA

(discussed above), mode of action studies indicate that increased liver weight caused by PFNA is adverse and relevant to humans. The Health-based MCL is based on increased liver weight in pregnant mice (Das et al., 2015), the only study providing the numerical serum PFNA data needed for dose-response analysis. Hepatic necrosis occurred at much lower doses and serum PFNA levels, below those that increased liver weight, in another study (**Stump et al., 2008**), but this study could not be used for quantitative risk assessment because numerical serum PFNA data were not provided. As such, an uncertainty factor of 3 was applied to protect for more sensitive effects at lower doses.

Two rat studies that provide important toxicological data for PFNA (**Stump et al., 2008; Mertens et al., 2010**) were not cited by ATSDR (2018), and NJDEP commented to ATSDR that these studies should be considered. These are the longest duration toxicological studies of PFNA (13 weeks and 18-21 weeks), and they report toxicity at a much lower administered dose and serum PFNA levels shorter duration studies. In these studies, rats were dosed with a technical mixture of PFAS (Surflon S-111) consisting primarily of PFNA (74%), with smaller percentages of other perfluorocarboxylic acids (perfluoroundecanoic acid (C11), 20%; perfluorotridecanoic acid (C13), 5%; PFOA, perfluorodecanoic acid, and perfluorododecanoic acid (C12), <1%). A detailed evaluation of the data from these studies concluded that toxicological effects were primarily caused by PFNA; see p. 39-41 of DWQI (2015).

An additional recent study of male reproductive toxicity of PFNA (**Singh and Singh, 2018**) was not available to DWQI (2015) or ATSDR (2015) and should be considered. The citation is provided below.

PFHxS

Although New Jersey has not developed a formal risk assessment for PFHxS, NJDEP scientists have reviewed the current toxicological literature for this compound. The following key studies on toxicity of PFAS were not cited by ATSDR (2018) and should be considered: **Das et al. (2017), Chang et al. (2018)** and **Ramhøj et al. (2018).** Citations are provided below.

We hope that this information is helpful to NHDES in its development of MCLs for PFOA, PFOS, PFNA, and PFHxS. If you have questions or need additional information, please feel free to contact Dr. Gloria Post of the NJDEP Division of Science and Research at gloria.post@dep.nj.gov.

Sincerely,

Gary A. Buchanan, Ph.D.
Director

Attachments

<u>Citations</u> (Those not cited by ATSDR, 2018 are in **bold.**)

ATSDR (2018). Agency for Toxics Substances and Disease Registry. Toxicological Profile for Perfluoroalkyls. Draft for Public Comment. June 2018.

Butenhoff, J. L., Kennedy, G. L. Jr., Chang, S. C. Olsen, G. W. (2012a). Chronic dietary toxicity and carcinogenicity study with ammonium perfluorooctanoate in Sprague-Dawley rats. Toxicology 298: 1-13.

Butenhoff JL, Chang SC, Olsen GW, Thomford PJ. (2012b). Chronic dietary toxicity and carcinogenicity study with potassium perfluorooctanesulfonate in Sprague Dawley rats. Toxicology. 293:1-15.

Chang S, Butenhoff JL, Parker GA, Coder PS, Zitzow JD, Krisko RM, Bjork JA, Wallace KB, Seed JG. (2018). Reproductive and developmental toxicity of potassium perfluorohexanesulfonate in CD-1 mice. Reprod Toxicol. 78:150-168.

Das, K.P., Grey, B.E., Rosen, M.B., Wood, C.R., Tatum-Gibbs, K.R., Zehr, R.D., Strynar, M.J., Lindstrom, A.B., Lau, C. (2015). Developmental toxicity of perfluorononanoic acid in mice. Reproductive Toxicology 51:133-144.

Das KP, Wood CR, Lin MT, Starkov AA, Lau C, Wallace KB, Corton JC, Abbott BD. (2017). Perfluoroalkyl acids-induced liver steatosis: Effects on genes controlling lipid homeostasis. Toxicology 378:37-52.

Dong GH, Zhang YH, Zheng L, Liu W, Jin YH, He QC. 2009. Chronic effects of perfluorooctanesulfonate exposure on immunotoxicity in adult male C57BL/6 mice. Arch Toxicol. 83:805-815.

Dong Z, Bahar MM, Jit J, Kennedy B, Priestly B, Ng J, Lamb D, Liu Y, Duan L, Naidu R. 2017. Issues raised by the reference doses for perfluorooctane sulfonate and perfluorooctanoic acid. Environ Int. 105:86-94.

EFSA (2018). Draft - Risk to human health related to the presence of perfluorooctanoic acid and perfluorooctane sulfonic in food (draft). EFSA Journal 15(5): 5194. Posted at: https://www.documentcloud.org/documents/4620589-EFSA-PFOS-and-PFOA-Draft.html

Goudarzi H, Miyashita C, Okada E, Kashino I, Chen CJ, Ito S, Araki A, Kobayashi S, Matsuura H, Kishi R. (2017). Prenatal exposure to perfluoroalkyl acids and prevalence of infectious diseases up to 4years of age. Environ Int.104:132-138.

Impinen A, Nygaard UC, Lødrup Carlsen KC, Mowinckel P, Carlsen KH, Haug LS, Granum B. (2018). Prenatal exposure to perfluoralkyl substances (PFASs) associated with respiratory tract infections but not allergy- and asthma-related health outcomes in childhood. Environ Res. 160:518-523.

Keil DE, Mehlmann T, Butterworth L, Peden-Adams MM. 2008. Gestational exposure to perfluorooctane sulfonate suppresses immune function in B6C3F1 mice. Toxicol Sci. 103:77-85.

Lilienthal H, Dieter HH, Hölzer J, Wilhelm M. Recent experimental results of effects of perfluoroalkyl substances in laboratory animals - Relation to current regulations and guidance values. 2017. Int J Hyg Environ Health. 220:766-775.

Luebker DJ, Case MT, York RG, Moore JA, Hansen KJ, Butenhoff JL. (2005). Two-generation reproduction and cross-foster studies of perfluorooctanesulfonate (PFOS) in rats. Toxicology.215:126-148.

Macon, M.B., Villanueva, L.R., Tatum-Gibbs, K., Zehr, R.D., Strynar, M.J., Stanko, J.P., White, S.S., Helfant, L., Fenton, S.E. (2011). Prenatal perfluorooctanoic acid exposure in CD-1 mice: low dose developmental effects and internal dosimetry. Toxicol. Sci. 122: 134-45.

MDH. 2017. Minnesota Department of Health. Health Based Guidance for Water Health Risk Assessment Unit, Environmental Health Division. Toxicological Summary for: Perfluorooctane Sulfonate. http://www.health.state.mn.us/divs/eh/risk/guidance/gw/pfos.pdf

Mertens, J.J., Sved, D.W., Marit, G.B., Myers, N.R., Stetson, P.L., Murphy, S.R., Schmit, B., Shinohara, M., Farr, C.H. (2010). Subchronic toxicity of S-111-S-WB in Sprague Dawley rats. Int. J. Toxicol. 29, 358−371.

NJDEP (2017). New Jersey Department of Environmental Protection. Maximum Contaminant Levels (MCLs) for Perfluorononanoic Acid and 1,2,3-Trichloropropane; Private Well Testing for Arsenic, Gross Alpha Particle Activity, and Certain Synthetic Organic Compounds. Proposed Amendments: N.J.A.C. 7:9E-2.1; 7:10-5.2, 5.3, and 12.30; and 7:18-6.4. August 7, 2017. https://www.nj.gov/dep/rules/proposals/20170807b.pdf

NJDEP (2018). New Jersey Department of Environmental Protection. Maximum Contaminant Levels (MCLs) for Perfluorononanoic Acid and 1,2,3-Trichloropropane; Private Well Testing for Arsenic, Gross Alpha Particle Activity, and Certain Synthetic Organic Compounds. Adopted Amendments: N.J.A.C. 7:9E-2.1; 7:10-5.2, 5.3, and 12.30; and 7:18-6.4. September 4, 2018. https://www.nj.gov/dep/rules/adoptions/adopt_20180904a.pdf

NJDEP (2018). New Jersey Department of Environmental Protection. Comments on Agency for Toxic Substances and Disease Registry (ATSDR) Draft Toxicological Profile for

Perfluoroalkyls. August 15, 2018. https://www.regulations.gov/document?D=ATSDR-2015-0004-0047

NJ Drinking Water Quality Institute (DWQI) MCL Recommendations

(Documents hotlinked below and posted at https://www.state.nj.us/dep/watersupply/g_boards_dwqi.html)

Perfluorooctane Sulfonate (PFOS), June 2018 – Recommendation Document.

Appendix A – Health-Based Maximum Contaminant Level Support Document for PFOS

Appendix B – Report on the Development of a Practical Quantitation Level for PFOS in Drinking Water

Appendix C – Second Addendum to Appendix C: Recommendation on Perfluorinated Compound Treatment Options for Drinking Water

Appendix D – Responses to Comments on DWQI Health Effects Subcommittee Report: "Public Review Draft - Health-Based Maximum Contaminant Level Support Document: PFOS"

Perfluorooctanoic Acid (PFOA), March 2017 – Recommendation Document.

Appendix A – Health-Based Maximum Contaminant Level Support Document" PFOA

Appendix B – Report on the Development of a Practical Quantitation Level for PFOA in Drinking Water

Appendix C – Addendum to Appendix C: Recommendation on Perfluorinated Compound Treatment Options for Drinking Water

Appendix D – Responses to Comments on DWQI Health Effects Subcommittee Report: "Public Review Draft-Health-Based Maximum Contaminant Level Support Document: PFOA"

Perfluorononanoic Acid (PFNA), July 2015 – Recommendation Document.

Appendix A – Health-Based Maximum Contaminant Level Support Document: PFNA

Appendix B – Report on the development of a Practical Quantitation Level for PFNA

Appendix C – Recommendation on Perfluorinated Compound Treatment Options for Drinking Water

NTP (2016). National Toxicology Program. Systematic review of immunotoxicity associated with exposure to perfluorooctanoic acid (PFOA) or perfluorooctane sulfonate (PFOS). September 2016. https://ntp.niehs.nih.gov/ntp/ohat/pfoa_pfos/pfoa_pfosmonograph_508.pdf

Pachkowski, B., Post, G.B., Stern, A.H. (2018). The derivation of a Reference Dose (RfD) for perfluoroctane sulfonate (PFOS) based on immune suppression. Env. Research (accepted manuscript is online at https://www.sciencedirect.com/science/article/pii/S0013935118304286).

Post, G.B., Gleason, J.A., Cooper, K.R. (2017). Key scientific issues in developing drinking water guidelines for perfluoroalkyl acids: Contaminants of emerging concern. PLoS Biol. 15(12):e2002855. Open access at https://journals.plos.org/plosbiology/article/file?id=10.1371/journal.pbio.2002855&type=printable

Post, G.B., Cohn, P.D., and Cooper, K.R. (2012). Perfluorooctanoic acid (PFOA), an emerging drinking water contaminant: a critical review of recent literature. Env. Res. 116: 93-117.

Ramhøj L, Hass U, Boberg J, Scholze M, Christiansen S, Nielsen F, Axelstad M. (2018). Perfluorohexane sulfonate (PFHxS) and a mixture of endocrine disrupters reduce thyroxine levels and cause antiandrogenic effects in rats. Toxicol Sci. 163(2):579-591.

Singh S, Singh SK. (2018). Chronic exposure to perfluorononanoic acid impairs spermatogenesis, steroidogenesis and fertility in male mice. J Appl Toxicol. doi: 10.1002/jat.3733. [Epub ahead of print]

Stump, D.G., Holson, J.F., Murphy, S.R., Farr, C.H., Schmit, B., Shinohara, M. (2008). An oral two-generation reproductive toxicity study of S-111-S-WB in rats. Reprod. Toxicol. 25, 7−20.

Timmermann CAG, Budtz-Jørgensen E, Petersen MS, Weihe P, Steuerwald U, Nielsen F, Jensen TK, Grandjean P. (2017). Shorter duration of breastfeeding at elevated exposures to perfluoroalkyl substances. Reprod Toxicol. 68:164-170.