BEFORE THE ILLINOIS POLLUTION CONTROL BOARD

IN THE MATTER OF:	)	
	)	
PROPOSED AMENDMENTS TO	)	R 22-18
GROUNDWATER QUALITY	)	(Rulemaking – Public Water Supplies)
35 ILL ADM. CODE 620	)	

NOTICE OF FILING

TO: Mr. Don Brown,
Clerk of the Board
Illinois Pollution Control Board
100 West Randolph Street
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Chicago, Illinois 60601
(VIA ELECTRONIC MAIL)

Vanessa Horton,
Hearing Officer
Illinois Pollution Control Board
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(See Persons on Attached Service List)

PLEASE TAKE NOTICE that I have today filed with the Office of the Clerk of the Illinois Pollution Control Board, **THE ILLINOIS ENVIRONMENTAL PROTECTION AGENCY'S PRE-FILED ANSWERS TO THE NATIONAL WASTE AND RECYCLING ASSOCIATION**, copies of which are hereby served upon you.

Respectfully Submitted,

ILLINOIS ENVIRONMENTAL
PROTECTION AGENCY

Dated: March 8, 2022

By: /s/ Sara Terranova
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THE ILLINOIS ENVIRONMENTAL PROTECTION AGENCY'S PRE-FILED ANSWERS TO THE NATIONAL WASTE AND RECYCLING ASSOCIATION

The Illinois Environmental Protection Agency ("Illinois EPA" or "Agency"), by and through its attorneys, and pursuant to the Illinois Pollution Control Board's ("Board") Notice of Hearing dated January 13, 2022, submits the following Pre-filed Answers to the National Waste and Recycling Association ("NWRA") for hearings scheduled on March 9-10, 2022.

I. General Questions

NWRA Question 1
In its Statement of Reasons ("SOR"), at pp. 17–19, the IEPA generally explains a series of stakeholder meetings and public comment periods that it conducted, stating that it "accepted and considered all public comments regarding the proposed groundwater qualitystandards for six weeks, until June 25, 2021." For the Board to fully understand and address the significant issues in this proceeding, and to make an informed decision as to whether the proposedrules are ready for the Board to adopt as its "First Notice" proposal requiring further hearings anda specific statutory time frame for promulgation, would the IEPA please include in this record:

(a) its various versions of the proposed rules and all stakeholder comments itreceived in response to those draft proposed rules;

(b) a summary of the changes it made (or did not make) in response to thoseproposals and the reasons therefor; and

(c) any recordings or minutes or transcripts of public meetings and/or hearings thatwere held?

Agency Answer 1
Please see Attachments, 1, 2, and 3, for the various versions of the proposed rules the Agency distributed to stakeholders and the public.

Please see the Agency's December 7, 2021 Statement of Reasons, Section IV Outreach, for a general summary of the significant questions received and the Agency's response.

Please see Attachment 4 for the Agency's 2021 620 Meeting Record.

NWRA Question 2

On September 17, 2021, the Illinois Groundwater Advisory Council ("GAC") declined to recommend that the IEPA move forward with its proposed rules – and posed several concerns, in the nature of questions, to the IEPA. Statement of Reasons ("SOR"), at pp. 4976– 4977. The GAC's recommendation was followed by a September 29, 2021 letter from the Intergovernmental Coordinating Committee on Groundwater ("ICCG") pursuant to its obligationunder the Illinois Groundwater Protection Act, 415 ILCS 55/4, to provide a written response to the GAC's recommendation. The ICCG letter, SOR at pp. 4979–4980, states, in relevant part:

> *The ICCG as a whole entity does not have the expertise to answer or comment on the GAC questions/comments on the proposed changes to the 35 Ill. Adm. Code 620 Groundwater Quality standards. These changes to the Groundwater Quality standards arebeing proposed by the Illinois EPA, who has the expertise and knowledge to address this (GAC) Recommendation. Therefore, it isthe Committee's stated opinion that the GAC Recommendation should be addressed by the Illinois EPA in the Statement of Reasonsor before the Illinois Pollution Control Board. Further, this Response by ICCG does not specifically endorse or disapprove of the proposed rule changes and individual ICCG member reserves the right to provide additional comment, questions, or concerns during the rule making process.*

Additionally, Ms. Sara Terranova, Assistant Counsel, IEPA Division of Legal Counsel, provided IEPA's response to the GAC recommendation in a November 18, 2021, email to Mr. BobElvert, GAC Chairperson. SOR at 4982. The email reads:

> *The Illinois Environmental Protection Agency (Agency) has received and reviewed the Groundwater Advisory Council's (GAC)Recommendations to Proposed 35 Ill. Adm. Code 620. The Agencybelieves each applicable point of concern raised by the GAC has been sufficiently addressed in the SOR and the accompanied Testimony that is to be filed before the Illinois Pollution Control Board (Board). However, if any outstanding issues remain, each concern may be raised and further addressed during the 35 Ill. Adm.Code 620 rulemaking proceeding before the Board.*

To sufficiently address the concerns raised by GAC, and of ultimate and immediate interestto the participants in this rulemaking, NWRA requests that the IEPA address the following:

a) *Please point to where in its SOR or Testimony the IEPA has addressed, or please otherwise address in response to this question, the GAC's criticism concerning "the basis for the Illinois EPA's reluctance to work with <u>all</u> (emphasis in original) impacted*

parties during the drafting of these rules, which could have resulted in discussions answering many of the questions raised during the comment period that ended May 25, 2021."

Agency Answer 2(a)
Please see the Agency's December 7, 2021 Statement of Reasons, Section IV Outreach. The Agency held multiple comment periods, a question-and-answer session, and a public meeting. Each was open to the public and all impacted parties.

b) *Please point to where in its SOR or Testimony the IEPA has addressed, or please otherwise address in response to this question, the GAC's criticism thatthe IEPA has not yet provided sufficient information regarding "the basis for the IEPA's urgency to file these proposed rules with the IPCB without prior response to all comments submitted during the comment period that ended May.*

Agency Answer 2(b)
Please see the Agency's December 7, 2021 Statement of Reasons, Section IV Outreach. In addition, while the Agency is not required to respond comments outside of the Board's rulemaking proceeding, the Agency did consider all comments received prior to filing with the Board on December 8, 2021.

c) *Please point to where in its SOR or Testimony the IEPA has provided, or otherwise please provide in response to this question, the GAC's requested information explaining how this rule proposal compares to the federal orsurrounding state approaches, methodologies, and standards.*

Agency Answer 2(c)
Please see the Agency's December 7, 2021 Statement of Reasons, Section I Statutory Authority. Pursuant to Section 55/8 of the Illinois Groundwater Protection Act, the Agency was required to adopt groundwater quality standards. The USEPA does not have groundwater quality standards, therefore no comparison can be made. Please see Appendices A, B, and C of Part 620. These appendices establish approaches, methodologies, and standards for developing groundwater quality standards in Illinois.

d) *Please point to where in its SOR or Testimony the IEPA has addressed, or otherwise please address in response to this question:*

i. *the IEPA's rationale in proposing these rules prior to the USEPA developing its proposed approach to addressing PFAS; and*

ii. *the IEPA's rationale for proposing a stricter approach or rationale than that being considered by the USEPA and/or in place or under consideration in surrounding states.*

Agency Answer 2(d)(i) and (ii)
Please see the Agency's December 7, 2021 Statement of Reasons, Section I Statutory Authority. The Illinois Groundwater Protection Act (IGPA) contains the criteria the Agency must consider when adopting groundwater quality standards.

e) *Please point to where in its SOR or Testimony the IEPA has addressed, or otherwise please address in response to this question, "how testing will be performed in state laboratories at the levels recommended in the proposal, including calculation assumptions and technical research references."*

Agency Answer 2(e)
IEPA does not plan to perform PFAS testing in state laboratories currently. Each method includes procedures for testing, including calculation assumptions.

f) *Please point to where in its SOR or Testimony the IEPA has addressed, or otherwise provide in response to this question, sufficient justification and explanation for the methods regulated entities should use to analyze for per/polyfluoroalkyl (PFA's) substances and other materials in wastewater,biosolids, and other products.*

Agency Answer 2(f)
35 Illinois Adm. Code 620 pertains only to the setting of groundwater quality standards. Methods for wastewater, biosolids, and other products are not applicable or necessary for setting groundwater quality standards.

NWRA Question 3
In Carol Hawbaker's pre-filed testimony (the "Hawbaker Testimony"), Ms. Hawbaker asserts that the "Agency for Toxic Substances and Disease Registry ("ATSDR") Minimal Risk Levels" are "peer reviewed and are available at http://www.atsdr.cdc.gov/mrls.html on the ATSDR website." Hawbaker Testimony, p. 7. This link was not accessible (using common internet browsers including Microsoft Edge and Chrome). Would IEPA please provide either the correct internet addresses for this information, or otherwise include the information in the record?

Agency Answer 3
The correct link is: https://wwwn.cdc.gov/TSP/MRLS/mrlsListing.aspx

NWRA Question 4
In the Hawbaker Testimony, Ms. Hawbaker asserts that certain "carcinogen designations are available at: https://www.monographs.iarc.who.int/agents-classified-by-the- iarc." Hawbaker Testimony, p. 27. This link was not accessible (using common internet browsers including Microsoft Edge and Chrome). Would IEPA please provide either the correct internet addresses for this information, or otherwise include the information in the record?

Agency Answer 4
The correct link to the search page for the list of classifications is: https://monographs.iarc.who.int/list-of-classifications
The Agency recommends searching the list by CAS No.

NWRA Question 5
How has the IEPA considered the timing of federal efforts as presented in the USEPA PFAS Roadmap[1] including detailed studies that are expected to be available in the Fall of2022 and how will those efforts impact the proposed rulemaking?

Agency Answer 5
The Illinois EPA's rulemaking effort for the revision of the 620 Groundwater Quality Standards is independent of USEPA's proposed National Primary Drinking Water regulations and the "USEPA Strategic Roadmap".

NWRA Question 6
If the Board adopts the IEPA's proposed standard, does the IEPA intend to amend the standard as new research becomes available?

a) If so, what is the IEPA's plan for doing so?

Agency Answer 6
At this time, the Agency's efforts and focus are on the current revisions of the 620 Groundwater Quality Standards.

II. Questions Related to the Impact of the IEPA's Proposed Part 620 Changes on Other Important and Existing Board Regulations

NWRA Question 7
Since Part 620 has an integral impact on other longstanding Board regulations— especially those regulating the monitoring of groundwater and the treatment of waste, such as Parts724, 725, 734, 740, 742, 807, and 811—what consideration was given as to what changes will be required to these Board regulations in order to achieve consistency with the significant changes being proposed in this rulemaking?

NWRA Question 8
Does the IEPA have a timeline for proposing amendments to each of these key regulatory programs? Please explain that timeline.

NWRA Question 9
Meanwhile, how does the State intend to enforce these new standards across thesekey regulatory programs that have not yet been amended for consistency with the proposed rule?

NWRA Question 10
Will the PFAS constituents be added to the List of Leachate Monitoring Parameterscontained in Appendix C to 35 Ill. Adm. Code 811?

NWRA Question 11
If so, given significant matrix interference in leachate, what appropriate testing methods have been identified and vetted by the IEPA?

NWRA Question 12
What is the IEPA's expectation of the acceptance and treatment of leachate in light of its proposed new PFAS standards?

a) *For example, does the IEPA intend to add PFAS limits to 35 Ill. Adm. Code Part 309 or otherwise require treatment of PFAS containing leachate?*

b) *Has the IEPA conducted a cost-benefit analysis concerning the treatment of leachate that might contain PFAS at the levels proposed?*

NWRA Question 13
How will non-detects with method detections limits or practical quantitation limits ("PQL") above the Class I standard be addressed in the background statistical analysis relevant to landfills and other waste disposal units?

NWRA Question 14
If well construction accomplished pursuant to IEPA guidelines was determined to nonetheless contribute to a detection of PFAS at the limits proposed, will the IEPA require reconstruction of these wells?

a) *Has the IEPA conducted a cost-benefit analysis to address this issue?*

NWRA Question 15
What is the IEPA's expectation of changes it will require to the existing Groundwater Impact Assessment ("GIA") program to address PFAS constituents at the levels proposed?

a) *Has the IEPA conducted a cost-benefit analysis to address this issue?*

NWRA Question 16
Will the IEPA provide a mechanism to address PFAS model failures without automatically reverting to a contingent remediation program?

a) *Has the IEPA conducted a cost-benefit analysis to address this issue?*

NWRA Question 17
What potential contaminant transport models has the IEPA identified to address PFAS constituents?

NWRA Question 18
Will existing waste disposal sites with permitted contingent remediation plans need to be re-evaluated for the inclusion of PFAS?

a) *If so, when?*

b) *Has the IEPA conducted a cost-benefit analysis on this issue?*

NWRA Question 19
Will existing waste disposal sites already engaged in permitted corrective action bere-evaluated for the inclusion of PFAS?

a) If so, when?

NWRA Question 20
Will the proposed new parameters be evaluated prior to the IEPA's release of landfill sites from post-closure care?

a) Has the IEPA conducted a cost-benefit analysis on this issue?

NWRA Question 21
Does the IEPA expect to revise the guidance document LPC-PA2, or create a newdocument, related to sample retrieval and testing methods for the PFAS constituents?

a) If so, when?

NWRA Question 22
What consideration has IEPA given to the impact of its proposal on other regulatedmedia (e.g., biosolids, finished compost, and clean up residuals from contaminated sites)?

Agency Answers 7-22
The questions in this section relate to potential changes to various programs that may be needed in response to changes in Part 620. Other programs are understandably affected by changes to Part 620 because Part 620 contains the State's groundwater quality standards, which are administered through multiple programs. Changes that will be needed to these programs as a result of changes to Part 620, however, cannot be determined until any changes to Part 620 are adopted and known.

Once amendments to Part 620 are adopted, the Agency will identify and develop amendments needed in other rules. It is an iterative process that requires multiple steps. As noted earlier, the Agency has already begun outreach with the NWRA to discuss potential impacts, including potential impacts raised in these questions. However, any amendments to Part 620 must first be adopted and known before the questions can be held.

III. Questions Directly Related to IEPA's Proposed 620 Changes.

A. 35 Ill. Adm. Code § 620.110: Definitions

NWRA Question 23
What is the IEPA's justification for substitution of LCMRL or other terms that are defined and calculated based on reagent water, versus current standards that are derived from real-world samples?

Agency Answer 23
IEPA is updating these terms to align with language throughout approved methods being used. LCMRLs are utilized for drinking water methods. Questions regarding method development should be directed to USEPA.

NWRA Question 24
What is the IEPA's technical justification for the substitution of the practical quantification limit ("PQL"), derived from a rigorous, interlaboratory process that generates a valid estimate of minimum analytical capability appropriate for setting numeric standards, with terms/limits not derived from such rigorous procedures?

Agency Answer 24
IEPA is updating these terms to align with language throughout the approved methods being used. Specifically, SW-846 is moving away from the use of a PQL and now provides procedures for establishing an LLOQ. Questions regarding method development should be directed to USEPA.

NWRA Question 25
What is the technical basis for the IEPA removing reference to the PQL from the proposed rules?

a) What consideration did the IEPA give to the entirety of the state's regulatory framework by proposing such changes in these new groundwater rules?

Agency Answer 25
The LCMRL utilizes an updated statistical approach for a single laboratory to meet its Measurement Quality Objectives (MQO). This approach provides a more accurate determination of the MQO and eliminates the issue of laboratories using multiple Practical Quantitation Limit methods to determine the MQO. SW-846 Chapter 1 uses the LLOQ. IEPA has considered that future proposed updates to rules within the State's regulatory framework will need to include updated terms to remain consistent with the proposed changes to 620.

NWRA Question 26
What is the technical feasibility of replacing the PQL with the new proposed methodology?

Agency Answer 26
IEPA is proposing the new methodology align with the USEPA methods being used. IEPA is unsure what Illinois Environmental Regulatory Group means by "technical feasibility".

NWRA Question 27
What consideration has the IEPA given to a laboratory's ability to analytically quantify at a health-based level versus the PQL (or MRL)?

Agency Answer 27
As stated in Part 620.605(b)(1) of the proposed rulemaking: "If the concentration for such substance is less than the lowest appropriate LLOQ or LCMRL for the substance, incorporated by reference at Section 620.125, the guidance level is the lowest appropriate LLOQ or LCMRL."

NWRA Question 28
In Section III of the Hawbaker Testimony, Ms. Hawbaker states that, "Due to updates in analytical methods that can quantify contaminants at lower levels," many carcinogens "whose Class I standards are based on the MCL are no longer set at the practical quantitation limit("PQL"), now proposed to be referred to as the LLOQ or LCMRL." This language indicates that the PQL is equivalent to the LLOQ and LCMRL and that these terms are interchangeable. Can the IEPA explain the inconsistency of the testimony with the proposed definitions?

Agency Answer 28
The Agency concurs that this language indicates that the PQL is equivalent to the LLOQ and LCMRL and that these terms are interchangeable and should be edited to state the following: Due to updates in analytical methods that can quantify contaminants at lower levels, many carcinogens whose Class I standards are based on the MCL are no longer set at the practical quantitation limit("PQL"), now proposed to be replaced by the LCMRL.

B. 35 Ill. Adm. Code § 620.210: Class I: Potable Resource Groundwater

NWRA Question 29
In proposed Section 620.210(a)(3), the word "or" was removed from prior draft proposals that had been circulated. Does IEPA now intend that all the conditions of 620.210(a)(1-5) must be met in order for groundwater to be considered a Potable Resource Groundwater? If so, what is the IEPA's justification?

Agency Answer 29
The indicated change is a drafting error. Thank you for bringing it to the Agency's attention. The final "or" following the semi-colon after the words "Pump test" should not have been stricken. It is the Agency's intent that any of the listed conditions is Class I groundwater.

C. 35 Ill. Adm. Code § 620.410 and 420

NWRA Question 30
In its SOR, at p. 9, the IEPA "proposes to add Class I groundwater quality standardsfor ten new chemicals as they have been identified in the groundwater in Illinois and may cause a hazard to human health." These new chemicals are: (1) Aluminum, (2) Lithium, (3) HFPO-DA (hexafluoropropylene oxide dimer acid, GenX), (4) 1-Methylnaphthalene, (5) Molybdenum, (6) PFBS (perfluorobutanesulfonic acid), (7) PFHxS (perfluorohexanesulfonic acid), (8) PFNA (perfluorononanoic acid), (9) PFOA (perfluorooctanoic acid), and (10) PFOS (perfluorooctanesulfonic acid). Will the IEPA please provide all groundwater sampling and analytical data obtained and utilized for each chemical in support of their addition to the Class I (and Class II) ground water quality standards ("GQS") at the levels proposed?

Agency Answer 30
Groundwater sampling and analytical data for lithium and molybdenum are available pursuant to 40 CFR Part 257 Subpart D: Standards for the Disposal of Coal Combustion Residuals in Landfills and Surface Impoundments.Illinois-specific data are located at https://www.luminant.com/illinois-ccr/ and https://www.nrg.com/legal/coal-combustion-residuals.html.

Groundwater sampling and analytical data for PFOA, PFOS, PFBS, PFHxS, and PFNA in raw water from Public Water Supply wells in Illinois are available in Drinking Water Watch located at http://water.epa.state.il.us/dww/index.jsp. In addition, HFPO-DA has been detected in groundwater monitoring wells in Illinois from Illinois EPA Bureau of Land program sites.
Aluminum has been detected in groundwater geographically distributed across Illinois in the range of 0.00002 to 4.18 mg/L.

1-methylnaphthalene has been detected in Illinois groundwater at numerous Illinois EPA Bureau of Land program sites.

NWRA Question 31
The Hawbaker Testimony states that the IEPA "documented detections of proposed per- and polyfluoroalkyls perfluorobutanesulfonic acid ("PFBS"), PFHxS, PFOS, and PFOA in finished water of public water supplies across Illinois…." Ms. Hawbaker also stated that, "thousands more utilize groundwater from private potable wells, usually without access totreatment technologies", and that "The above-referenced PFAS were also found in community water supply wells…."

a) *As the information provided from Ms. Hawbaker is from treated water which may have been altered by the treatment process, have there been any studies to show that the treatment process itself is not the source of these constituents, or that treatment has increased the concentrations of these constituents?*

b) *What have any such studies demonstrated?*

Agency Answer 31
Illinois EPA is unaware of any studies that support the correlation between drinking water treatment processes and increased PFAS presence. However, sampling data available on Drinking Water Watch shows the presence of PFAS in raw water sampling of groundwater and surface water at public water supplies where PFAS was detected in finished water. The finished water PFAS concentrations are consistently lower than raw water concentrations when comparing samples from the same public water supply.

NWRA Question 32
The Hawbaker Testimony states that "The only way to confirm the presence of PFAS is through proper sampling and analysis."

a) *For the samples where these constituents were found, what sampling and analytical methods were utilized to ensure that the samples were free of outsideinfluences?*

Agency Answer 32(a)
Sampling and analysis of public water supplies was done in accordance with IEPA's Quality Assurance Project Plan: Per- and Polyfluoroalkyl Substances (PFAS) Sampling in Community Water Supplies which requires adherence to USEPA Method 537.1.

b) *Will the IEPA provide any and all sampling data that supports its answer to (a)?*

Agency Answer 32(b)
No.

NWRA Question 33
The Hawbaker Testimony states that, "HFPO-DA is detected in groundwaterduring sampling for purposes other than the statewide PFAS sampling initiative."

a) What other purposes is Ms. Hawbaker referring to here?

b) What sampling protocols and analytical protocols were employed to ensure thatpotential outside contamination did not occur?

Agency Answer 33
HFPO-DA was detected in groundwater at a manufacturing facility entered in the Agency's Bureau of Land Site Remediation Programs. The sampling and analytical protocols were overseen by a Licensed Professional Engineer, and the document was certified by a Licensed Professional Engineer that sampling and analytical protocols were properly utilized.

NWRA Question 34
The Hawbaker Testimony states that, "[f]or the thirty-nine constituents with current Class I standards based on procedures in Part 620 Subpart F and Appendix A, all have been recalculated using the proposed methods specified in Subpart F and Appendix A. After the recalculation of the health-based standards for the constituent, Illinois EPA compared the updated standards with LLOQs/LCMRLs for groundwater and drinking water analytical methods." As noted in Section I of the Hawbaker Testimony, drinking water methods are appropriate for analyzing Class I potable resource groundwater. Table A includes both drinking water and SW- 846 methods. Why were SW-846 analytical methods used for comparison to the LLOQs/LCMRLs as opposed to the drinking water analytical methods as it has been stated that the drinking water methods are the appropriate standards for analyzing Class I potable resource groundwater?

Agency Answer 34
The use of both of SW-846 and drinking water methods as examples is intended to show Illinois EPA will accept either groundwater or a drinking water analytical methods with an LLOQ/LCMRL less than the groundwater quality standard. SW-846 methods have been incorporated into the Part 620 regulations since its first promulgation in 1991.

NWRA Question 35
Did the IEPA consult with certified Illinois commercial laboratories to ascertain whether such laboratories have the capability to quantify and report to the low-level GQS's proposed by the IEPA?

(a) *If so, what labs were consulted and would the IEPA provide documentation ofthat consultation?*

Agency Answer 35
IEPA has identified Illinois laboratories that are capable of meeting the proposed groundwater quality standards.

NWRA Question 36
How does the IEPA justify use of LLOQ in the proposed rule (a single laboratory concept) when comparing to a numeric standard as used in the proposed rulemaking?

Agency Answer 36
Optimally, the LLOQ should be less than the regulatory action level. Justification is provided in the series of SW-846 methods.

NWRA Question 37
What process did the IEPA employ and were commercial laboratories available tothe regulated community consulted to choose the lowest quantitation limit to establish a numeric standard?

a) *Did the IEPA review all analytical methods and each individual commercial laboratory's capabilities and then just choose the lowest quantitation limit to establish a numeric standard?*

b) *If the answer to a, above, is in the affirmative, how does the IEPA consider thatapproach technically defensible or acceptable?*

Agency Answer 37
IEPA assumes that by lowest quantitation limit the NWRA means the Lower Limit of Quantitation (LLOQ) or the Lowest Concentration Minimum Reporting Level (LCMRL). The LLOQ/LCMRL is not used to establish a numeric standard. The LLOQ/LCMRL (depending on the method) is used as the numeric standard if the guidance level falls below the LLOQ/LCMRL.

NWRA Question 38
The Hawbaker Testimony states that, "Part 620, Subpart F and Appendix A, provide the basis for developing Illinois Pollution Control Board ("Board") rulemaking proposalsfor new or revised numerical standards (35 Ill. Adm. Code 620.601(c))." It further indicates that,"[a]s the standards calculated using the methods at Part 620, Subpart F and Appendix A are basedon the protection of human health from ingesting groundwater, and MCLs are promulgated for drinking water, drinking water methods are appropriate for analyzing Class I potable resource groundwater."

a) *Has the Board ever endorsed the use of MCL's as an appropriate technical basis for developing and adopting groundwater quality standards? If so, please explain when.*

Agency Answer 38(a)
MCLs have been the primary source for Class I groundwater quality standards since Part 620's promulgation in 1991. However, when MCLs are not available, Part 620 Subpart F and Appendix A provide the basis for developing Illinois Pollution Control Board ("Board") rulemaking proposals for new or revised numerical standards (35 Ill. Adm. Code 620.601(c)).

b) *Has the USEPA drinking water methodology ever been required for comparison to the Illinois GQS's and compliance with 35 Ill. Admin. Code 620?*

Agency Answer 38(b)
The Agency is unsure what is meant by "comparison". Illinois EPA uses USEPA MCLs as the basis for many groundwater quality standards. The noncancer method for calculating an HTTAC as a groundwater quality standard is the USEPA drinking water methodology for developing USEPA Health Advisory Levels for drinking water. The proposed cancer method for calculating an HNTAC as a groundwater quality standard is derived from procedures described in "Guidelines for Carcinogenic Risk Assessment" and "Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens" (both dated March 2005, by USEPA Risk Assessment Forum, included as Attachment 1F 1, located on page 2,849, and 1F 2, located on page 3,016, of the December 27, 2021, filing). The proposed Part 620 updates for calculating an HNTAC are USEPA's residential tap water ingestion equations for calculating a residential tap water screening level in USEPA's RSL calculator.

c) *If so, when?*

Agency Answer 38(c)
Please see the answer to (b).

d) *What significance does Ms. Hawbaker attribute to her reference to SW846 (Hazardous Waste Test Methods) in the regulatory references?*

Agency Answer 38(d)
SW-846 methods have been incorporated by reference since Part 620's promulgation in 1991.

NWRA Question 39
Would the IEPA please identify the data and science it relied upon to determine that the appropriate regulatory approach for Illinois is to adopt strict drinking water standards for PFAS compounds and apply them as GQS's?

Agency Answer 39
A number of the Class I groundwater standards are based on drinking water standards, not just PFAS compounds. This approach is appropriate because private wells typically do not receive any treatment before consumption by well owners. Therefore, the raw groundwater is a private well owners drinking water. Application of constituent levels equal to drinking water standards would therefore be protective of human health.

NWRA Question 40
Would the IEPA please explain how the LLOQ and LCMRL were used to established health-based limits?

Agency Answer 40
The LLOQ and LCMRL are not used to establish health-based groundwater quality standards. Health-based standards are calculated using the procedures in Part 620 Appendix A, then compared to the LLOQ or LCMRL to determine if the health-based groundwater quality standard can be quantified in analyses. Refer to Part 620.605(b)(1) for the appropriate use of an LLOQ or LCMRL in lieu of a health-based groundwater quality standard.

NWRA Question 41
Considering there are numerous laboratory terms and acronyms for reporting, detection, and quantitation limits, how did the IEPA apply such terms in setting its proposed numeric standards?

Agency Answer 41
The numerous laboratory terms and acronyms are standard terms used by IEPA, USEPA, and other agencies or research groups. These terms are not applied when calculating numeric health-based standards. If the health-based guidance level falls below the LLOQ/LCMRL, then the LLOQ/LCMRL becomes the standard.

NWRA Question 42
How has the IEPA determined, addressed, and considered the very serious issues with sample and laboratory contamination by PFAS of concern in setting its proposed numeric standards?

Agency Answer 42
Issues with sample and laboratory contamination do not apply to the process of setting numeric health-based standards. Contamination issues are addressed in the Quality Assurance Project Plans and Methods.

NWRA Question 43
Will the IEPA evaluate and eliminate data from its evaluation that are from laboratories where known contamination (e.g., method blanks and field blanks) have created excessive positive bias?

a) *What is the bias criterion for removal of data?*

Agency Answer 43
IEPA will follow the guidance outlined in the approved methods to maintain quality assurance. The criteria for removal of data depends on which method is being used. Samples with blank contamination are not to be reported and sites are to be resampled.

NWRA Question 44
Will the IEPA commit to promulgating a process (and study procedure) whereby a regulated party may demonstrate that:

a) *site-specific matrix interferences affect the testing results to such an extent that data cannot be produced at the numeric standard?*

b) *site-specific matrix interferences affect the testing results to such an extent that data produced at the numeric standard lacks significant digits?*

c) *site-specific matrix interferences affect the testing results to such an extent that does not have as many significant digits as the numeric standard?*

Agency Answer 44
No.

NWRA Question 45
What is the basis for setting numeric standards below the analytical technologies' quantitation limit and forgoing development of a PQL when a numeric standard should be based on a laboratory's ability to quantitate at that level?

Agency Answer 45
The basis for setting numeric standards is protecting human health from the adverse effects of a particular chemical or group of chemicals.

a) *What is IEPA's proposed alternative approach to account for minimum analytical capability if not developing a PQL?*

Agency Answer 45(a)
The Lowest Concentration Minimum Reporting Level (LCMRL) and the Lower Limit of Quantitation (LLOQ).

b) *Does IEPA's proposed alternative approach involve application of rigorous terms, definitions, concepts, and incorporations of interlaboratory quantitationlimits?*

Agency Answer 45(b)
The LCMRL does involve terms, definitions, concepts, and quantitation limits. The term "rigorous" depends on the opinion and level of expertise of the user. USEPA provides a freely accessible software package for calculating the LCMRL.

c) *Will the MDL be used as a replacement for the PQL even though quantitation is defined at the PQL?*

Agency Answer 45(c)
No.

d) *If the answer to c, above, is in the negative, will the IEPA be using a single- or interlaboratory denotation for the MDL?*

Agency Answer 45(d)
The Method Detection Level may be referred to as MDL. IEPA is not aware of any other denotation for MDL.

e) *Does the IEPA plan to address that these are single-laboratory concepts not appropriate replacements for a PQL?*

Agency Answer 45(e)
No.

NWRA Question 46
The IEPA cites removal efficiency rates of 75–95% for inorganic constituents in 620.420(a)(1) and 30-90% for organic constituents in 620.420(b)(1) in support of several proposed Class II

groundwater quality standards, apparently on the basis of the effectiveness in treating the constituent in groundwater. What is the source and basis of such stated removal efficiencies? More specifically, how were these removal efficiency rates derived and by whom?

Agency Answer 46
For inorganic constituents at Part 620.420(a)(1), treatment factors are applied for 2 constituents: antimony (treatment factor of 4) and thallium (treatment factor of 10). The treatment factors applied for these constituents have not changed since they were added to Part 620 in IPCB R93-27, promulgated in 1994.

For organic constituents at Part 620.420(b)(1), please refer to Carol Hawbaker Testimony, pages 28 – 30, and Attachments 1A 2, located on page 365 and 1J 1 located on page 4,854 of the December 7, 2021, filing for the source and basis of proposed updated treatment factors.

NWRA Question 47
What sampling protocols has the IEPA developed for PFAS constituents?

Agency Answer 47
Illinois EPA's SOP for Sample Collection of PFAS at CWS in Illinois.

NWRA Question 48
Will entities performing sampling be required to be accredited?

Agency Answer 48
No.

NWRA Question 49
Did the IEPA consider if analytical data should be reported below a PQL (or MRL)to avoid falsely reporting a standards exceedance when it does not exist?

Agency Answer 49
No.

NWRA Question 50
If a commercial laboratory certified in Illinois cannot achieve a PQL (or MRL), what actions will be taken by the IEPA?

Agency Answer 50
None.

a) Will this be considered non-compliance?

Agency Answer 50(a)
No.

b) *What would be the responsibility of the regulated party in these instances?*

Agency Answer 50(b)
The regulated party should contract with an alternative laboratory.

NWRA Question 51
IEPA's proposed rule uses a pre-established ranking for Tier 3 sources which is inconsistent with USEPA's 2003 directive for the selection of toxicity values (specifying that priority should be given to "sources of information that are the most current, the basis for whichis transparent and publicly available, and which has been peer reviewed.")

a) *What is the IEPA's technical rationale for proposing a pre-established ranking for Tier 3 sources in the establishment of GQS's?*

b) *Given IEPA's proposed approach, how will the IEPA ensure the most technically defensible science is being used to establish GQS's?*

Agency Answer 51
While the 2003 OSWER Directive 9285.7-53 (included as Attachment 1C 1, starting on page 513 of the December 7, 2021, filing) established an overall hierarchy for selecting toxicity values, it did not attempt to rank Tier 3 sources. On May 16, 2013, USEPA's OSWER Human Health Regional Risk Assessors Forum issued a "Tier 3 Toxicity Value White Paper" (included as Attachment 1C 2, starting on page 518 of the December 7, 2021, filing) which provided a recommended ranking of Tier 3 toxicity sources to assist in determining appropriate toxicity values. This ranking is utilized by USEPA's Regional Screening Level "RSL" Workgroup for determining appropriate toxicity values for calculating screening levels. Refer to Carol Hawbaker Testimony pages 6-9.

NWRA Question 52
The proposed rules present procedures for determining an acceptable daily exposure to be used in establishing GQS's for substances for which a reference dose is not available from the hierarchy of sources for toxicity values.

a) *What criteria for determining the quality and reliability of a study for derivingtoxicity values will be used?*

b) *How will the IEPA ensure that such derived toxicity values are technically defensible?*

Agency Answer 52
Please refer to proposed 35 Ill. Adm. Code 620 Appendix A(c) for procedures for establishing validity of data from animal studies. Any toxicity criteria developed using the procedures at Part 620 Appendix A(b) will be peer-reviewed and submitted for public comment in any proposed amendments to 35 Ill. Adm. Code 620.

NWRA Question 53
What is the IEPA's technical basis for the use of a combined uncertainty factor of 10,000 when the USEPA recommends that a maximum uncertainty factor of 3,000 be used when developing noncancer toxicity criteria?

Agency Answer 53
Please refer to Attachment 1E 1, titled, "A Review of the Reference Dose and Reference Concentration Process," beginning on page 2,546 of the Agency's December 7, 2021, filing. The maximum Uncertainty Factor (UF) for development of an oral reference dose is 10,000. The maximum UF of 3000 is used for development of an inhalation value or reference concentration.

a) *How does the IEPA plan to counter the compounding conservatism that will beintroduced into toxicity values by such a method?*

Agency Answer 53(a)
The most current USEPA guidance will be followed to apply UFs when calculating toxicity values.

b) *Is the highly uncertain reference dose that will result appropriate for establishing GQS's?*

Agency Answer 53(b)
Uncertainty was applied using the most current USEPA guidance, therefore it is the most appropriate for establishing GQSs.

NWRA Question 54
At what frequency will the rules be updated to consider new and evolving toxicology?

Agency Answer 54
Consistent with past practice, the Agency has no fixed schedule, but will propose amendments when needed, taking into account the latest chemical and toxicity data.

a) *When toxicity criteria from a preferred reference source becomes available will the GQS's be updated in a timely manner?*

Agency Answer 54(a)
When PFAS toxicity criteria from sources higher on USEPA's toxicity hierarchy become available, Illinois EPA will review the information and will consider amendments to standards.

b) *Have the toxicity criteria anticipated from USEPA's Integrated RiskInformation System in 2022 (including criteria for PFHxS and PFNA) been considered in the proposed rulemaking? If so, please explain how.*

Agency Answer 54(b)
As USEPA's Integrated Risk Information System ("IRIS") has not released draft toxicity criteria for review or issued final toxicity evaluations for PFHxS and PFNA, Illinois EPA did not consider information from IRIS.

NWRA Question 55
The IEPA specifies that the toxicity values would be from USEPA's Provisional Peer-Reviewed Toxicity Value ("PPRTV") for the compound; this source was specifically mentioned in the testimony for all PFAS compounds except PFBS.

a) Please identify what toxicity value is being proposed to establish the GQS for PFBS.

Agency Answer 55(a)
An oral reference dose of 3E-04 mg/kg-day, issued by PPRTV in April 2021, is the proposed toxicity value for PFBS. This information is included as Attachment 1I 1, starting on page 3,236, and the PFBS PPRTV toxicity profile is included as Attachment 1I 12, starting on page 4,471, of the December 7, 2021, filing.

b) If the PPRTV remains the source for this value for deriving the GQS, please explain how the selection of a benchmark dose response of one-half the controlstandard deviation by USEPA in the PPRTV for PFBS is justified?

Agency Answer 55(b)
The Illinois EPA selected USEPA's PPRTV as the toxicity source because PPRTV is a Tier 2 toxicity source developed and used by USEPA; and is a final value. Concerns regarding the basis for USEPA's PPRTV development of its toxicity value are more appropriately directed to USEPA.

NWRA Question 56
The IEPA's rule proposal is based upon MRLs from ATSDR for PFHxS and PFNA; however, ATSDR states the following regarding the databases for these specific MRLs: "these were based on marginal databases and additional dose-response studies are needed to support the basis of the MRL." How does the IEPA justify the use of MRLs from ATSDRs in its rule proposal?

Agency Answer 56
The Illinois EPA selected U.S. Health and Human Services ATSDR dose MRLs as toxicity sources, for PFHxS and PFNA because ATSDR is a Tier 3 toxicity source permitted for use within USEPA's toxicity hierarchy; and these are final values. Concerns regarding the basis for ATSDR's development of its toxicity values are more appropriately directed to ATSDR.

NWRA Question 57
ATSDR recognizes the uncertain nature of the human half-lives used to derive human equivalent doses for PFOA and PFOS. Does the IEPA agree that the uncertain nature of these half-lives introduces a substantial degree of uncertainty in the MRLs for these compounds?

a) If not, why not?

b) How does the IEPA support the use of highly uncertain MRLs for setting GQS's?

Agency Answer 57
The Illinois EPA selected U.S. Health and Human Services ATSDR dose MRLs as noncancer toxicity sources for PFOA and PFOS because ATSDR is a Tier 3 toxicity source permitted for

use within USEPA's toxicity hierarchy; and these are final values. Concerns regarding the basis for ATSDR's development of its toxicity values are more appropriately directed to ATSDR.

NWRA Question 58
Explain what criteria and methodologies are considered for setting relative source contributions ("RSC")?

Agency Answer 58
USEPA uses a default RSC value of 20% unless clear supporting documentation demonstrates the applicability of an RSC other than the default. Illinois EPA relies on USEPA's RSC value when they are available.

a) *What specific data and conditions must be met for an RSC of other than 20% to be used?*

Agency Answer 58(a)
Illinois EPA bases RSCs on USEPA RSCs.

b) *Why is the RSC default of 20% being applied for all PFAS?*

Agency Answer 58(b)
USEPA determined, and Illinois EPA agrees, that RSCs cannot be set at levels other than the default for PFAS due to insufficient data. PFAS constituents are ubiquitous in the environment outside of groundwater. Its many uses in manufacturing goods, such as clothing, furniture, carpeting, food packaging, personal care products, and a myriad of other items provides ample opportunity for exposure to PFAS from sources other than drinking water. In addition, bioaccumulation of PFAS in plants and animals used for food sources indicates significant opportunity for exposures to PFAS other than through ingestion of water. The presence of PFAS in the blood of virtually every person in the world, particularly in people living in areas where contaminated drinking water is not a source of exposure, indicates evidence of multiple exposure routes.

c) *Does the IEPA agree that the use of the default RSC of 20% overestimates thecontribution of diet and other non-drinking water sources in situations where exposure to elevated PFAS in drinking water occurs?*

Agency Answer 58(c)
No. Refer to answer (b)

d) *If the answer to c, above, is in the negative, please explain why the IEPA disagrees that the use of the default RSC of 20% overestimates the contributionof diet and other non-drinking water sources in situations where exposure to elevated PFAS in drinking water occurs.*

Agency Answer 58(d)
Refer to answer (b).

NWRA Question 59
Please describe the intended application of the proposed rules on toxic additivity.

a) Under what conditions does toxic additivity need to be considered?

Agency Answer 59(a)
Please refer to proposed 35 Ill. Adm. Code 620.615(a) and Appendix B(d) for conditions where toxic additivity must be considered. These procedures are also described in the 35 Ill. Adm. Code 742 regulations.

b) Should toxic additivity be evaluated for all potable groundwater?

Agency Answer 59(b)
Refer to answer (a).

c) If the answer to b, above, is in the affirmative, does such a procedure require the collection of a full suite of analytical data?

Agency Answer 59(c)
Analytical requirements for chemical sampling are subject to the Agency's program-specific regulations, such as permitting or cleanup.

d) Please explain IEPA's view of the technical feasibility of the regulated community's application of this Appendix.

Agency Answer 59(d)
Requirements for addressing toxic additivity have been in place since Part 620's promulgation in 1991.

Respectfully Submitted,

ILLINOIS ENVIRONMENTAL
PROTECTION AGENCY

Dated: March 8, 2022

By: /s/ Sara Terranova
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CERTIFICATE OF SERVICE

I, the undersigned, hereby certify that I have electronically served **THE ILLINOIS ENVIRONMENTAL PROTECTION AGENCY'S PRE-FILED ANSWERS TO NATIONAL WASTE AND RECYCLING ASSOCIATION** on March 8, 2022, to the attached service list. I further certify that my email address is sara.terranova@illinois.gov and that the email transmission took place before 5:00pm.

ILLINOIS ENVIRONMENTAL
PROTECTION AGENCY

Dated: March 8, 2022

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ATTACHMENT 1

Attachment 1

TITLE 35: ENVIRONMENTAL PROTECTION
SUBTITLE F: PUBLIC WATER SUPPLIES
CHAPTER I: POLLUTION CONTROL BOARD

PART 620
GROUNDWATER QUALITY

SUBPART A: GENERAL

SUBPART B: GROUNDWATER CLASSIFICATION

SUBPART C: NONDEGRADATION PROVISIONS FOR APPROPRIATE GROUNDWATERS

SUBPART D: GROUNDWATER QUALITY STANDARDS

SUBPART E: GROUNDWATER MONITORING AND ANALYTICAL PROCEDURES

SUBPART F: HEALTH ADVISORIES

AUTHORITY: Implementing and authorized by Section 8 of the Illinois Groundwater Protection Act [415 ILCS 55/8] and authorized by Section 27 of the Illinois Environmental Protection Act [415 ILCS 5/27].

SOURCE: Adopted in R89-14(B) at 15 Ill. Reg. 17614, effective November 25, 1991; amended in R89-14(C) at 16 Ill. Reg. 14667, effective September 11, 1992; amended in R93-27 at 18 Ill. Reg. 14084, effective August 24, 1994; amended in R96-18 at 21 Ill. Reg. 6518, effective May 8, 1997; amended in R97-11 at 21 Ill. Reg. 7869, effective July 1, 1997; amended in R01-14 at 26 Ill. Reg. 2662, effective February 5, 2002; amended in R08-18 at 36 Ill. Reg. 15206, effective October 5, 2012; amended in R08-18(B) at 37 Ill. Reg. 16529, effective October 7, 2013.

SUBPART B: GROUNDWATER CLASSIFICATION

Section 620.240 Class IV: Other Groundwater

Except as provided in Section 620.250, Other Groundwater is:

a) Groundwater within a zone of attenuation as provided in 35 Ill. Adm. Code 811 and 814;

b) Groundwater within a point of compliance as provided in 35 Ill. Adm. Code 724, but not to exceed a distance of 200 feet from a potential primary or secondary source.

c) Groundwater that naturally contains more than 10,000 mg/L of total dissolved solids;

d) Groundwater which has been designated by the Board as an exempt aquifer pursuant to 35 Ill. Adm. Code 730.104; or

e) Groundwater which underlies a potential primary or secondary source, in which contaminants may be present from a release, if the owner or operator of such source notifies the Agency in writing and the following conditions are met:

1) The outermost edge is the closest practicable distance from such source, but does not exceed:

A) A lateral distance of 25 feet from the edge of such potential source or the property boundary, whichever is less, and

B) A depth of 15 feet from the bottom of such potential source or the land surface, whichever is greater;

2) The source of any release of contaminants to groundwater has been controlled;

3) Migration of contaminants within the site resulting from a release to groundwater has been minimized;

4) Any on-site release of contaminants to groundwater has been managed to prevent migration off-site; and

5) No potable water well exists within the outermost edge as provided in subsection (e)(1).

f) Groundwater which underlies ~~a coal mine refuse disposal area not contained within an area from which overburden has been removed,~~ a coal combustion waste disposal area at a surface coal mine authorized under Section 21(s) of the Act, or an impoundment that contains sludge, slurry, or precipitated process material at a coal preparation plant, in which contaminants may be present, if such area or impoundment was placed into operation after February 1, 1983, if the owner and operator notifies the Agency in writing, and if the following conditions are met:

1) The outermost edge is the closest practicable distance, but does not exceed:

A) A lateral distance of 25 feet from edge of such area or impoundment, or the property boundary, whichever is less; and

B) A depth of 15 feet from the bottom of such area or impoundment, or the land surface, whichever is greater;

2) The source of any release of contaminants to groundwater has been controlled;

3) Migration of contaminants within the site resulting from a release to groundwater has been minimized;

4) Any on-site release of contaminants to groundwater has been managed to prevent migration off-site; and

5) No potable water well exists within the outermost edge as provided in subsection (f)(1)~~(e)(1)~~.

g) Groundwater within a previously mined area, unless monitoring demonstrates that the groundwater is capable of consistently meeting the standards of Sections 620.410 or 620.420. If such capability is determined, groundwater within the previously mined area shall not be Class IV.

h) Groundwater which underlies a coal mine refuse disposal area not contained within an area from which overburden has been removed, in which contaminants may be present, if such area or impoundment was placed into operation after February 1, 1983, if the owner and operator notifies the Agency in writing, and if the following conditions are met:

1) The outermost edge is the closest practicable distance, but does not exceed:

A) A lateral distance of 300 feet from the toe of the refuse disposal area, or the property boundary, whichever is less; and

B) The uppermost geological formation that is a potential contamination migration pathway and any hydraulically connected contamination migration pathways.

2) The source of any release of contaminants to groundwater has been controlled;

3) Migration of contaminants within the site resulting from a release to groundwater has been minimized;

4) Any on-site release of contaminants to groundwater has been managed to prevent migration off-site; and

5) No potable water well exists within the outermost edge as provided in subsection (h)(1).

Section 620.450 Alternative Groundwater Quality Standards

a) Groundwater Quality Restoration Standards

1) Any chemical constituent in groundwater within a groundwater management zone is subject to this Section.

2) Except as provided in subsections (a)(3) or (a)(4), the standards as specified in Sections 620.410, 620.420, 620.430, ~~and~~ 620.440, and 620.450(b) apply to any chemical constituent in groundwater within a groundwater management zone.

3) Prior to completion of a corrective action described in Section 620.250(a), the standards as specified in Sections 620.410, 620.420, 620.430, ~~and~~ 620.440, and 620.450(b) are not applicable to such released chemical constituent, provided that the initiated action proceeds in a timely and appropriate manner.

4) After completion of a corrective action as described in Section 620.250(a), the standard for such released chemical constituent is:

A) The standard as set forth in Section 620.410, 620.420, 620.430, ~~or~~ 620.440, or 620.450(b) if the concentration as determined by groundwater monitoring of such constituent is less than or equal to the standard for the appropriate class set forth in those Sections; or

B) The concentration as determined by groundwater monitoring, if such concentration exceeds the standard for the appropriate class set forth in Section 620.410, 620.420, 620.430, ~~or~~ 620.440, or 620.450(b) for such constituent, and:

i) To the extent practicable, the exceedence has been minimized and beneficial use, as appropriate for the class of groundwater, has been returned; and

ii) Any threat to public health or the environment has been minimized.

5) The Agency shall develop and maintain a listing of concentrations derived pursuant to subsection (a)(4)(B). This list shall be made available to the public and be updated periodically, but no less frequently than semi-annually. This listing shall be published in the Environmental Register.

b) Coal Reclamation Groundwater Quality Standards

1) Any inorganic chemical constituent or pH in groundwater, within an underground coal mine, or within the cumulative impact area of groundwater for which the hydrologic balance has been disturbed from a permitted coal mine area pursuant to the Surface Coal Mining Land Conservation and Reclamation Act [225 ILCS 720] and 62 Ill. Adm. Code 1700 through 1850, is subject to this Section.

2) Prior to completion of reclamation at a coal mine, the standards as specified in Sections 620.410(a) and (e), 620.420(a) and (e), 620.430 and 620.440 are not applicable to inorganic constituents and pH.

3) After completion of reclamation at a coal mine, the standards as specified in Sections 620.410(a) and (e), 620.420(a), 620.430, and 620.440 are applicable to inorganic constituents and pH, except:

A) The concentration of total dissolved solids (TDS) must not exceed:

i) The post-reclamation concentration or 3000 mg/L, whichever is less, for groundwater within the permitted area; or

ii) The post-reclamation concentration of TDS must not exceed the post-reclamation concentration or 5000 mg/L, whichever is less, for groundwater in underground coal mines and in permitted areas reclaimed after surface coal mining if the Illinois Department of Mines and Minerals and the Agency have determined that no significant resource groundwater existed prior to mining (62 Ill. Adm. Code 1780.21(f) and (g)); and

B) For chloride, iron, manganese and sulfate, the post-reclamation concentration within the permitted area must not be exceeded.

C) For pH, the post-reclamation concentration within the permitted area must not be exceeded within Class I: Potable Resource Groundwater as specified in Section 620.210(a)(4).

D) For 1,3-dinitrobenzene, 2,4-dinitrotoluene, 2,6-dinitrotoluene, HMX (high melting explosive, octogen), nitrobenzene, RDX (royal demolition explosive, cyclonite), 1,3,5-trinitrobenzene, and 2,4,6-trinitrotoluene (TNT), the post-reclamation concentration within the permitted area must not be exceeded.

4) A refuse disposal area (not contained within the area from which overburden has been removed) is subject to the inorganic chemical constituent and pH requirements of:

A) 35 Ill. Adm. Code 302.Subparts B and C, except due to natural causes, for such area that was placed into operation after February 1, 1983, and before the effective date of this Part, provided that the groundwater is a present or a potential source of water for public or food processing;

B) Section 620.440(c) for such area that was placed into operation prior to February 1, 1983, and has remained in continuous operation since that date; or

C) Subpart D of this Part for such area that is placed into operation on or after the effective date of this Part.

5) For a refuse disposal area (not contained within the area from which overburden has been removed) that was placed into operation prior to February 1, 1983, and is modified after that date

to include additional area, this Section applies to the area that meets the requirements of subsection (b)(4)(C) and the following applies to the additional area:

A) 35 Ill. Adm. Code 302.Subparts B and C, except due to natural causes, for such additional refuse disposal area that was placed into operation after February 1, 1983, and before the effective date of this Part, provided that the groundwater is a present or a potential source of water for public or food processing; and

B) Subpart D for such additional area that was placed into operation on or after the effective date of this Part.

6) For a new refuse disposal area (not contained within the area from which overburden has been removed), as defined in 35 Ill. Adm. Code 408.110, the following applies to the area:

A) Groundwater quality shall be maintained at each constituent's background concentration, at or beyond the point of compliance established pursuant to 35 Ill. Adm. 620.505. The applicable groundwater quality standard established for any constituent shall be the background concentration; and

B) Any statistically significant increase above an applicable groundwater quality standard established pursuant to subsection (b)(6)(A) that is attributable to the facility and which occurs at or beyond the point of compliance within 100 years after reclamation is a violation.

7)~~6)~~ A coal preparation plant (not located in an area from which overburden has been removed) which contains slurry material, sludge or other precipitated process material, is subject to the inorganic chemical constituent and pH requirements of:

A) 35 Ill. Adm. Code 302.Subparts B and C, except due to natural causes, for such plant that was placed into operation after February 1, 1983 and before the effective date of this Part, provided that the groundwater is a present or a potential source of water for public or food processing;

B) Section 620.440(c) for such plant that was placed into operation prior to February 1, 1983, and has remained in continuous operation since that date; or

C) Subpart D for such plant that is placed into operation on or after the effective date of this Part.

8)~~7)~~ For a coal preparation plant (not located in an area from which overburden has been removed) which contains slurry material, sludge or other precipitated process material, that was placed into operation prior to February 1, 1983, and is modified after that date to include additional area, this Section applies to the area that meets the requirements of subsection (b)(6)(C) and the following applies to the additional area:

A) 35 Ill. Adm. Code 302.Subparts B and C, except due to natural causes, for such additional area that was placed into operation after February 1, 1983, and before the effective date of this Part, provided that the groundwater is a present or a potential source of water for public or food processing; and

B) Subpart D for such additional area that was placed into operation on or after the effective date of this Part.

c) Groundwater Quality Standards for Certain Groundwater Subject to a No Further Remediation Letter under Part 740. While a No Further Remediation Letter is in effect for a region formerly encompassed by a groundwater management zone established under 35 Ill. Adm. Code 740.530, the groundwater quality standards for "contaminants of concern", as defined in 35 Ill. Adm. Code 740.120, within such area shall be the groundwater objectives achieved as documented in the approved Remedial Action Completion Report.

Section 620.505 Compliance Determination

a) Compliance with standards at a site is to be determined as follows:

1) For a structure (e.g., buildings), at the closest practical distance beyond the outermost edge for the structure.

2) For groundwater that underlies a potential primary or secondary source, the outermost edge as specified in Section 620.240(e)(1).

3) For groundwater that underlies a coal mine refuse disposal area, a coal combustion waste disposal area, or an impoundment that contains sludge, slurry, or precipitated process material at a coal preparation plant, the outermost edge as specified in Section 620.240(f)(1) or (h)(1) or location of monitoring wells in existence as of the effective date of this Part on a permitted site.

4) For a groundwater management zone, as specified in a corrective action process.

5) For groundwater, any point where monitoring is conducted using a water well, or a monitoring well that meets one of the following conditions:

A) For a potable water supply well if geologic logs exist for this well or geologic logs in the immediate 1,000-foot area of this well are representative of the hydrogeologic materials encountered by this well as determined by a licensed professional geologist or a licensed professional engineer or a WHPA has been delineated outside of an applicable setback zone of a community water well or well field in accordance with the "Guidance Document for Groundwater Protection Needs Assessments," incorporated by reference at Section 620.125, and "The Illinois Wellhead Protection Program," incorporated by reference at Section 620.125.

B) For a potable water supply well other than a community water supply well, a construction report has been filed with the Department of Public Health for such potable well, or such well has been located and constructed (or reconstructed) to meet the Illinois Water Well Construction Code [415 ILCS 30] and 77 Ill. Adm. Code 920.

C) For a potable water supply well that was constructed prior to August 20, 1965, the enactment of the Illinois Water Well Construction Code [415 ILCS 30], and meets all of the following criteria:

i) Construction must be done in a manner that will enable the collection of groundwater samples that represent in situ groundwater conditions;

ii) Casings and screens must be made from durable material resistant to expected chemical or physical degradation that do not interfere with the quality of groundwater samples being collected; and

iii) The annular space opposite the screened section of the well (i.e., the space between the bore hole and well screen) must be filled with gravel or sand if necessary to collect groundwater samples. The

annular space above and below the well screen must be sealed to prevent migration of water from adjacent formations and the surface to the sampled depth.

D) For a community water supply well, such well has been permitted by the Agency, or has been constructed in accordance with 35 Ill. Adm. Code 602.115.

E) For a water well other than a potable water supply well (e.g., a livestock watering well or an irrigation well), a construction report has been filed with the Department of Public Health or the Office of Mines and Minerals in the Department of Natural Resources for such well, or such well has been located and constructed (or reconstructed) to meet the Illinois Water Well Construction Code [415 ILCS 30] and 35 Ill. Adm. Code 920.

F) For a monitoring well, such well meets the following requirements:

i) Construction must be done in a manner that will enable the collection of groundwater samples;

ii) Casings and screens must be made from durable material resistant to expected chemical or physical degradation that do not interfere with the quality of groundwater samples being collected; and

iii) The annular space opposite the screened section of the well (i.e., the space between the bore hole and well screen) must be filled with gravel or sand if necessary to collect groundwater samples. The annular space above and below the well screen must be sealed to prevent migration of water from adjacent formations and the surface to the sampled depth.

6) Monitoring shall not be conducted for compliance determinations pursuant to subsection (a) of this Section:

A) For a water well that is:

i) Less than 15 feet in total depth from the land surface,

ii) bored or dug,

iii) constructed of permeable materials (e.g., cement, tile, stone or brick), and

iv) 36 inches or more in diameter.

B) For a water well with water quality problems due to damaged well construction materials or poorly-designed well construction;

C) For a water well in a basement or pit; or

D) For water well water from a holding tank.

b) For a spring, compliance with this Subpart shall be determined at the point of emergence.

Attachment 2

Draft dated 12-19-2019.

TITLE 35: ENVIRONMENTAL PROTECTION
SUBTITLE F: PUBLIC WATER SUPPLIES
CHAPTER I: POLLUTION CONTROL BOARD

PART 620
GROUNDWATER QUALITY

SUBPART A: GENERAL

Section

SUBPART B: GROUNDWATER CLASSIFICATION

Section

SUBPART C: NONDEGRADATION PROVISIONS FOR APPROPRIATE GROUNDWATERS

Section

SUBPART D: GROUNDWATER QUALITY STANDARDS

Section

SUBPART E: GROUNDWATER MONITORING AND ANALYTICAL PROCEDURES

SUBPART F: HEALTH ADVISORIES

AUTHORITY: Implementing and authorized by Section 8 of the Illinois Groundwater Protection Act [415 ILCS 55/8] and authorized by Section 27 of the Illinois Environmental Protection Act [415 ILCS 5/27].

SOURCE: Adopted in R89-14(B) at 15 Ill. Reg. 17614, effective November 25, 1991; amended in R89-14(C) at 16 Ill. Reg. 14667, effective September 11, 1992; amended in R93-27 at 18 Ill. Reg. 14084, effective August 24, 1994; amended in R96-

18 at 21 Ill. Reg. 6518, effective May 8, 1997; amended in R97-11 at 21 Ill. Reg. 7869, effective July 1, 1997; amended in R01-14 at 26 Ill. Reg. 2662, effective February 5, 2002; amended in R08-18 at 36 Ill. Reg. 15206, effective October 5, 2012; amended in R08-18(B) at 37 Ill. Reg. 16529, effective October 7, 2013; amended in _____at __ _ Ill. Reg. _____, effective _____.

SUBPART A: GENERAL

Section 620.110 Definitions

The definitions of the Environmental Protection Act [415 ILCS 5] and the Groundwater Protection Act [415 ILCS 55] apply to this Part. The following definitions also apply to this Part.

"Act" means the Environmental Protection Act [415 ILCS 5].

"Agency" means the Illinois Environmental Protection Agency.

"Aquifer" means saturated (with groundwater) soils and geologic materials which are sufficiently permeable to readily yield economically useful quantities of water to wells, springs, or streams under ordinary hydraulic gradients. [415 ILCS 55/3(b)]

"BETX" means the sum of the concentrations of benzene, ethylbenzene, toluene, and xylenes.

"Board" means the Illinois Pollution Control Board.

"Carcinogen" means a contaminant that is classified as a Category A1 or A2 Carcinogen by the American Conference of Governmental Industrial Hygienists; or a Category 1 or 2A/2B carcinogen by the World Health Organization's International Agency for Research on Cancer; or a "Human carcinogen" or "Anticipated Human Carcinogen" by the United States Department of Health and Human Service National Toxicological Program; or a Category A or B1/B2 Carcinogen or as "carcinogenic to humans" or likely to become carcinogenic to humans" *by the United States Environmental Protection Agency in Integrated Risk Information System or a Final Rule issued in a Federal Register notice by the USEPA.* [415 ILCS 5/58.2]

"Community water supply" means a public supply which serves or is intended to serve at least 15 service connections used by residents or regularly serves at least 25 residents. [415 ILCS 5/3.145]

"Contaminant" means any solid, liquid, or gaseous matter, any odor, or

any form of energy, from whatever source. [415 ILCS 5/3.165]

"Corrective action process" means those procedures and practices that may be imposed by a regulatory agency when a determination has been made that contamination of groundwater has taken place, and are necessary to address a potential or existing violation of the standards set forth in Subpart D.

"Cumulative impact area" means the area, including the coal mine area permitted under the Surface Coal Mining Land Conservation and Reclamation Act [225 ILCS 720] and 62 Ill. Adm. Code 1700 through 1850, within which impacts resulting from the proposed operation may interact with the impacts of all anticipated mining on surface water and groundwater systems.

"Department" means the Illinois Department of Natural Resources.

"Detection" means the identification of a contaminant in a sample at a value equal to or greater than the:

> "Method Detection Limit" or "MDL" means the minimum concentration of a substance that can be measured as reported with 99 percent confidence that the true value is greater than zero, pursuant to 40 CFR 136, appendix B (2006), incorporated by reference at Section 620.125; or
>
> "Method Quantitation Limit" or "MQL" means the minimum concentration of a substance that can be measured and reported pursuant to "Test Methods for Evaluating Solid Wastes, Physical/Chemical Methods", incorporated by reference at Section 620.125.

"Groundwater" means underground water which occurs within the saturated zone and geologic materials where the fluid pressure in the pore space is equal to or greater than atmospheric pressure. [415 ILCS 5/3.210]

"Hydrologic balance" means the relationship between the quality and quantity of water inflow to, water outflow from, and water storage in a hydrologic unit such as a drainage basin, aquifer, soil zone, lake, or reservoir. It encompasses the dynamic relationships among precipitation, runoff, evaporation, and changes in ground and surface water storage.

"IGPA" means the Illinois Groundwater Protection Act [415 ILCS 55].

"LOAEL" or "Lowest observable adverse effect level" means the lowest tested concentration of a chemical or substance that produces a statistically significant increase in frequency or severity of non-overt adverse effects between the exposed population and its appropriate control. LOAEL may be determined for a human population (LOAEL-H) or an animal population (LOAEL-A).

"Licensed Professional Engineer" or "LPE" means a person, corporation, or partnership licensed under the laws of the State of Illinois to practice professional engineering. [415 ILCS 5/57.2]

"Licensed Professional Geologist" or "LPG" means an individual who is licensed under the Professional Geologist Licensing Act *to engage in the practice of professional geology in Illinois.* [225 ILCS 745/15]

"NOAEL" or "No observable adverse effect level" means the highest tested concentration of a chemical or substance that does not produce a statistically significant increase in frequency or severity of non-overt adverse effects between the exposed population and its appropriate control. NOAEL may be determined for a human population (NOAEL-H) or an animal population (NOAEL-A).

"Non-community water supply" means a public water supply that is not a community water supply. [415 ILCS 5/3.145]

"Off-site" means not on-site.

"On-site" means on the same or geographically contiguous property that may be divided by public or private right-of-way, provided the entrance and exit between properties is at a crossroads intersection and access is by crossing as opposed to going along the right-of-way. Noncontiguous properties owned by the same person but connected by a right-of-way that he controls and that the public does not have access to is also considered on-site property.

"Operator" means the person responsible for the operation of a site, facility or unit.

"Owner" means the person who owns a site, facility or unit or part of a site, facility or unit, or who owns the land on which the site, facility or unit is located.

"Potable" means generally fit for human consumption in accordance with accepted water supply principles and practices. [415 ILCS 5/3.340]

"Potential primary source" means any unit at a facility or site not currently subject to a removal or remedial action which:

Is utilized for the treatment, storage, or disposal of any hazardous or special waste not generated at the site; or

Is utilized for the disposal of municipal waste not generated at the site, other than landscape waste and construction and demolition debris; or

Is utilized for the landfilling, land treating, surface impounding or piling of any hazardous or special waste that is generated on the site or at other sites owned, controlled or operated by the same person; or

Stores or accumulates at any time more than 75,000 pounds above ground, or more than 7,500 pounds below ground, of any hazardous substances. [415 ILCS 5/3.345]

"Potential route" means abandoned and improperly plugged wells of all kinds, drainage wells, all injection wells, including closed loop heat pump wells, and any excavation for the discovery, development or production of stone, sand or gravel. This term does not include closed loop heat pump wells using USP (U.S. Pharmacopeia) *food grade propylene glycol.* [415 ILCS 5/3.350]

"Potential secondary source" means any unit at a facility or a site not currently subject to a removal or remedial action, other than a potential primary source, which:

Is utilized for the landfilling, land treating, or surface impounding of waste that is generated on the site or at other sites owned, controlled or operated by the same person, other than livestock and landscape waste, and construction and demolition debris; or

Stores or accumulates at any time more than 25,000 but not more than 75,000 pounds above ground, or more than 2,500 but not more than 7,500 pounds below ground, of any hazardous substance; or

Stores or accumulates at any time more than 25,000 gallons above ground, or more than 500 gallons below ground, of petroleum, including crude oil or any fraction thereof which is

not otherwise specifically listed or designated as a hazardous substance; or

Stores or accumulates pesticides, fertilizers, or road oils for purposes of commercial application or for distribution to retail sales outlets; or

Stores or accumulates at any time more than 50,000 pounds of any de-icing agent; or

Is utilized for handling livestock waste or for treating domestic wastewaters other than private sewage disposal systems as defined in the Private Sewage Disposal Licensing Act [225 ILCS 225]. [415 ILCS 5/3.355]

"Practical Quantitation Limit" or "PQL" means the lowest concentration or level that can be reliably measured within specified limits of precision and accuracy during routine laboratory operating conditions in accordance with "Test Methods for Evaluating Solid Wastes, Physical/Chemical Methods", EPA Publication No. SW-846, incorporated by reference at Section 620.125.

"Previously mined area" means land disturbed or affected by coal mining operations prior to February 1, 1983.
BOARD NOTE: February 1, 1983, is the effective date of the Illinois permanent program regulations implementing the Surface Coal Mining Land Conservation and Reclamation Act [225 ILCS 720] as codified in 62 Ill. Adm. Code 1700 through 1850.

"Property class" means the class assigned by a tax assessor to real property for purposes of real estate taxes.
BOARD NOTE: The property class (rural property, residential vacant land, residential with dwelling, commercial residence, commercial business, commercial office, or industrial) is identified on the property record card maintained by the tax assessor in accordance with the Illinois Real Property Appraisal Manual (February 1987), published by the Illinois Department of Revenue, Property Tax Administration Bureau.

"Public water supply" means all mains, pipes and structures through which water is obtained and distributed to the public, including wells and well structures, intakes and cribs, pumping stations, treatment plants, reservoirs, storage tanks and appurtenances, collectively or severally, actually used or intended for use for the purpose of furnishing water for drinking or general domestic use and which serve at least 15 service connections or which regularly serve at least 25 persons at least

60 days per year. A public water supply is either a "community water supply" or a "non-community water supply". [415 ILCS 5/3.365]

"Regulated entity" means a facility or unit regulated for groundwater protection by any State or federal agency.

"Regulatory agency" means the Illinois Environmental Protection Agency, Department of Public Health, Department of Agriculture, the Office of Mines and Minerals in the Department of Natural Resources, and the Office of State Fire Marshal.

"Regulated recharge area" means a compact geographic area, as determined by the Board pursuant to Section 17.4 of the Act, *the geology of which renders a potable resource groundwater particularly susceptible to contamination.* [415 ILCS 5/3.390]

"Resource groundwater" means groundwater that is presently being, or in the future is capable of being, put to beneficial use by reason of being of suitable quality. [415 ILCS 5/3.430]

"Saturated zone" means a subsurface zone in which all the interstices or voids are filled with water under pressure greater than that of the atmosphere.

"Setback zone" means a geographic area, designated pursuant to this Act, containing a potable water supply well or a potential source or potential route having a continuous boundary, and within which certain prohibitions or regulations are applicable in order to protect groundwaters. [415 ILCS 5/3.450]

"Site" means any location, place, tract of land and facilities, including but not limited to, buildings and improvements used for the purposes subject to regulation or control by the Act or regulations thereunder. [415 ILCS 5/3.460]

"Spring" means a natural surface discharge of an aquifer from rock or soil.

"Threshold dose" means the lowest dose of a chemical at which a specified measurable effect is observed and below which it is not observed.

"Treatment" means the technology, treatment techniques, or other procedures for compliance with 35 Ill. Adm. Code, Subtitle F.

"Unit" means any device, mechanism, equipment, or area (exclusive of

land utilized only for agricultural production). [415 ILCS 5/3.515]

"USEPA" means the United States Environmental Protection Agency.

"Wellhead protection area" or "WHPA" means the surface and subsurface recharge area surrounding a community water supply well or well field, delineated outside of any applicable setback zones (pursuant to Section 17.1 of the Act [415 ILCS 5/17.1]), and pursuant to Illinois' Wellhead Protection Program, through which contaminants are reasonably likely to move toward such well or well field.

"Wellhead Protection Program" or "WHPP" means the wellhead protection program for the State of Illinois, approved by USEPA under 42 USC 300h-7.
BOARD NOTE: Derived from 40 CFR 141.71(b) (2003). The wellhead protection program includes the "groundwater protection needs assessment" under Section 17.1 of the Act [415 ILCS 5/17.1] and 35 Ill. Adm. Code 615-617.

(Source: Amended at __ Ill. Reg. _____, effective _____)

Section 620.125 Incorporations by Reference

a) The Board incorporates the following material by reference:

ASTM International. 100 Barr Harbor Drive, PO Box C700, West Conshohocken, PA 19428-2959 (610) 832-9500.

"Standard Practice for Classification of Soils for Engineering Purposes (Unified Classification System)" ASTM D2487-06.

CFR (Code of Federal Regulations). Available from the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402 (202) 783-3238.

Method Detection Limit Definition, appendix B to Part 136, 40 CFR 136, appendix B – Revision 2 (2019~~2006~~).

Control of Lead and Copper, general requirements, 40 CFR 141.80 (2019~~2006~~).

Maximum contaminant levels for organic contaminants, 40 CFR 141.61 (2019~~2006~~).

Maximum contaminant levels for inorganic contaminants, 40 CFR 141.62 (2019~~2006~~).

Maximum contaminant levels for radionuclides, 40 CFR 141.66 (2019~~2006~~).

GPO. Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20401 (202) 783-3238~~)~~.

USEPA Guidelines for Carcinogenic Risk Assessment, 51 Fed. Reg. 33992-34003 (September 24, 1986).

Illinois Environmental Protection Agency, 1020 North Grand Avenue East, P.O. Box 19276, Springfield, IL 62794-9276 (217) 785-4787.

"Guidance Document for Groundwater Protection Needs Assessments," Agency, Illinois State Water Survey, and Illinois State Geologic Survey Joint Report, January 1995.

"The Illinois Wellhead Protection Program Pursuant to Section 1428 of the Federal Safe Drinking Water Act," Agency, # 22480, October 1992.

NCRP. National Council on Radiation Protection, 7910 Woodmont Ave., Bethesda, MD (301) 657-2652.

"Maximum Permissible Body Burdens and Maximum Permissible Concentrations of Radionuclides in Air and in Water for Occupational Exposure", NCRP Report Number 22, June 5, 1959.

NTIS. National Technical Information Service, 5285 Port Royal Road, Springfield, VA 22161 (703) 605-6000.

"Methods for Chemical Analysis of Water and Wastes," March 1983, Doc. No. PB84-128677. EPA 600/4-79-020 (available online at http://nepis.epa.gov/).

"Methods for the Determination of Inorganic Substances in Environmental Samples," August 1993, PB94-120821 (referred to as "USEPA Environmental Inorganic Methods"). EPA 600/R-93-100 (available online at http://nepis.epa.gov/).

"Methods for the Determination of Metals in Environmental Samples," June 1991, Doc. No. PB91-231498. EPA 600/4-91-010 (available online at http://nepis.epa.gov/).

"Methods for the Determination of Metals in Environmental Samples − Supplement I," May 1994, Doc. No. PB95-125472. EPA 600/R-94-111 (available online at http://nepis.epa.gov/).

"Methods for the Determination of Organic Compounds in Drinking Water," Doc. No. PB91-231480. EPA/600/4-88/039 (December 1988 (revised July 1991)) (available online at http://nepis.epa.gov/).

"Methods for the Determination of Organic Compounds in Drinking Water, Supplement I," Doc. No. PB91-146027. EPA/600/4-90/020 (July 1990) (available online at http://nepis.epa.gov/).

"Methods for the Determination of Organic Compounds in Drinking Water, Supplement II," Doc. No. PB92-207703. EPA/600/R-92/129 (August 1992) (available online at http://nepis.epa.gov/).

"Methods for the Determination of Organic Compounds in Drinking Water, Supplement III," Doc. No. PB95-261616. EPA/600/R-95/131 (August 1995) (available online at http://nepis.epa.gov/).

"Methods for the Determination of Organic and Inorganic Compounds in Drinking Water" Volume I: EPA 815-R-00-014 (August 2000) (available online at http://nepis.epa.gov/).

"Prescribed Procedures for Measurement of Radioactivity in Drinking Water," Doc. No. PB80-224744. EPA 600/4-80-032, (August 1980) (available online at http://nepis.epa.gov/).

"Procedures for Radiochemical Analysis of Nuclear Reactor Aqueous Solutions," H.L. Krieger and S. Gold, Doc. No. PB222-154/7BA. EPA-R4-73-014, May 1973.

"Radiochemical Analytical Procedures for Analysis of Environmental Samples," March 1979, Doc. No. EMSL LV 053917.

"Radiochemistry Procedures Manual," Doc. No. PB-84-215581. EPA-520/5-84-006, December 1987.

"Practical Guide for Ground-Water Sampling", EPA Publication No. EPA/600/2-85/104 (September 1985), Doc. No. PB 86-137304.

"Test Methods for Evaluating Solid Waste, Physical/Chemical Methods," USEPA Publication No. SW-846, as amended by Updates I, II, IIA, IIB, III, IIIA, and IIIB (Doc. No. 955-001-00000-1) (available on line at http://www.epa.gov/epaoswer/hazwaste/test/main.htm).

United States Environmental Protection Agency, Office of Resource Conservation and Recovery.

"Statistical Analysis of Groundwater Monitoring Data at RCRA Facilities, (March 2009 Unified Guidance)", EPA 530/R-09-007.

USGS. United States Geological Survey, 1961 Stout St., Denver, CO 80294 (303) 844-4169

"Techniques of Water Resources Investigations of the United States Geological Survey, Guidelines for Collection and Field Analysis of Ground-Water Samples for Selected Unstable Constituents", Book I, Chapter D2 (1976).

b) This Section incorporates no later editions or amendments.

(Source: Amended at __ Ill. Reg. _____, effective _____)

SUBPART B: GROUNDWATER CLASSIFICATION

Section 620.210 Class I: Potable Resource Groundwater

Except as provided in Sections 620.230, 620.240, or 620.250, Potable Resource Groundwater is:

a) Groundwater located 10 feet or more below the land surface and within:

1) The minimum setback zone of a well which serves as a potable water supply and to the bottom of such well;

2) Unconsolidated sand, gravel or sand and gravel which is 5 feet or more in thickness and that contains 12 percent or less of fines (i.e., fines which pass through a No. 200 sieve tested according to ASTM Standard Practice D2487-06, incorporated by reference at Section 620.125);

3) Sandstone which is 10 feet or more in thickness, or fractured carbonate which is 15 feet or more in thickness; ~~or~~

4) Any geologic material which is capable of a:

A) Sustained groundwater yield, from up to a 12 inch borehole, of 150 gallons per day or more from a thickness of 15 feet or less; or

B) Hydraulic conductivity of 1 x 10^{-4} cm/sec or greater using one of the following test methods or its equivalent:

i) ~~Permeameter;~~

~~ii)~~ Slug test; or

ii)~~iii)~~ Pump test.

5) The wellhead protection area of a community water supply well or well field, as defined in Section 620.110 and delineated pursuant to the methods incorporated by reference in Section 620.125. For the purposes of this Subpart, when a maximum setback zone has been adopted pursuant to Section 14.3 of the Act, the WHPA includes the delineated area within the maximum setback zone.

b) Any groundwater which is determined by the Board pursuant to petition procedures set forth in Section 620.260, to be capable of potable use.

BOARD NOTE: Any portion of the thickness associated with the geologic materials as described in subsections 620.210(a)(2), (a)(3) or (a)(4) should be designated as Class I: Potable Resource Groundwater if located 10 feet or more below the land surface.

(Source: Amended at __ Ill. Reg. _____, effective _____)

Section 620.250 Groundwater Management Zone

a) Within any class of groundwater, a groundwater management zone may be established as a three dimensional region containing groundwater being managed to mitigate impairment caused by the release of contaminants from a site:

1) That is subject to a corrective action process approved by the Agency; or

2) For which the owner or operator undertakes an adequate corrective action in a timely and appropriate manner and provides a written confirmation to the Agency. Such confirmation must be provided in a form as prescribed by the Agency.

b) A groundwater management zone is established upon concurrence by the Agency that the conditions as specified in subsection (a) are met and groundwater management continues for a period of time consistent with the action described in that subsection.

c) A groundwater management zone expires upon the Agency's receipt of appropriate documentation which confirms the completion of the action taken pursuant to subsection (a) and which confirms the attainment of applicable standards as set forth in Subpart D. The Agency shall review the on-going adequacy of controls and continued management at the site if concentrations of chemical constituents, as specified in Section 620.450(a)(4)(B), remain in groundwater at the site following completion of such action. The review must take place no less often than every 5 years and the results shall be presented to the Agency in a written report.

d) Notwithstanding subsections (a) and (b) above, a groundwater management zone as defined in 35 Ill. Adm. Code 740.120 may be established in accordance with the requirements of 35 Ill. Adm. Code 740.530 for sites undergoing remediation pursuant to the Site Remediation Program. Such a groundwater management zone shall remain in effect until the requirements set forth at 35 Ill. Adm. Code 740.530(c) are met.

e) While the groundwater management zone established in accordance with 35 Ill. Adm. Code 740.530 is in effect, the otherwise applicable standards as specified in Subpart D of this Part shall not be applicable to the "contaminants of concern," as defined at 35 Ill. Adm. Code 740.120, for which groundwater remediation objectives have been approved in accordance with the procedures of 35 Ill. Adm. Code 740.

f) Notwithstanding subsection (c) above, the review requirements concerning the on-going adequacy of controls and continued management at the site shall not apply to groundwater within a three-dimensional region formerly encompassed by a groundwater management zone established in accordance with 35 Ill. Adm. Code 740.530 while a No Further Remediation Letter issued in accordance with the procedures of 35 Ill. Adm. Code 740 is in effect.

g) All groundwater management zone applications submitted pursuant to subsection (a) must contain the following:

1) Facility information, including name, address and county where the site is located.

2) Identification of specific units (operating or closed) present at the facility.

3) Maps and engineering drawings showing the facility, and units at the facility.

4) Statement of the groundwater classification(s) at the facility.

5) Identification of the chemical constituents released to the groundwater.

6) Description of how groundwater will be monitored to determine the rate and extent of the release, and if it has migrated off site.

7) Schedule for investigation of the extent of the release.

8) Results of available soil testing and groundwater monitoring associated with a release, locations and depths of samples, and monitoring well construction details with well logs.

9) Remedy

A) Description of selected remedy and why it was chosen;

B) Results of groundwater contaminant transport modeling or calculations showing how the selected remedy will achieve compliance with the applicable groundwater standards;

C) Description of the fate and transport of contaminants with selected remedy over time; and

D) A statement of how groundwater at the facility will be monitored following implementation of the remedy to ensure that the groundwater standards have been attained.

10) Information requested by the Agency, necessary for its review of the groundwater management zone application.

(Source: Amended at __ Ill. Reg. _____, effective _____)

SUBPART C: NONDEGRADATION PROVISIONS FOR APPROPRIATE GROUNDWATERS

Section 620.302 Applicability of Preventive Notification and Preventive Response Activities

a) Preventive notification and preventive response as specified in Sections 620.305 through 620.310 applies to:

1) Class I groundwater under Section 620.210(a)(1), (a)(2), or (a)(3) that is monitored by the persons listed in subsection (b); or

2) Class III groundwater that is monitored by the persons listed in subsection (b).

b) For purposes of subsection (a), the persons that conduct groundwater monitoring are:

1) An owner or operator of a regulated entity for which groundwater quality monitoring must be performed pursuant to State or Federal law or regulation (e.g., section 106 and 107 of the Comprehensive Environmental Response, Compensation and Liability Act (42 USC 9601, et seq.); sections 3004 and 3008 of the Resource Conservation and Recovery Act (42 USC 6901, et seq.); sections 4(q), 4(v), 12(g), 21(d), 21(f), 22.2(f), 22.2(m) and 22.18 of the Act; 35 Ill. Adm. Code 616, 724, 725, 730, 731, 750, 807, 811, ~~and~~ 814 and 815, and 62 Ill. Adm. Code 1780);

2) An owner or operator of a public water supply well who conducts groundwater quality monitoring;

3) A State agency that is authorized to conduct, or is the recipient of, groundwater quality monitoring data (e.g., Illinois Environmental Protection Agency, Department of Public Health, Department of Agriculture, Office of State Fire Marshal or

Department of Natural Resources); or

4) An owner or operator of a facility that conducts groundwater quality monitoring pursuant to State or federal judicial or administrative order.

c) If a contaminant exceeds a standard set forth in Section 620.410 or Section 620.430, the appropriate remedy is corrective action and Sections 620.305 and 620.310 do not apply.

(Source: Amended at __ Ill. Reg. _____, effective _____)

Section 620.310 Preventive Response Activities

a) The following preventive assessment must be undertaken:

1) If a preventive notification under Section 620.305(c) is provided by a community water supply:

A) The Agency shall notify the owner or operator of any identified potential primary source, potential secondary source, potential route, or community water supply well that is located within 2,500 feet of the wellhead.

B) The owner or operator notified under subsection (a)(1)(A) shall, within 30 days after the date of issuance of such notice, sample each water well or monitoring well for the contaminant identified in the notice if the contaminant or material containing such contaminant is or has been stored, disposed of, or otherwise handled at the site. If a contaminant identified under Section 620.305(a) is detected, then the well must be resampled within 30 days of the date on which the first sample analyses are received. If a contaminant identified under Section 620.305(a) is detected by the resampling, preventive notification must be given as set forth in Section 620.305.

C) If the Agency receives analytical results under subsection (a)(1)(B) that show a contaminant identified under Section 620.305(a) has been detected, the Agency shall:

i) Conduct a well site survey pursuant to 415 ILCS 5/17.1(d), if such a survey has not been previously conducted within the last 5 years; and

ii) Identify those sites or activities that represent a hazard to the continued availability of groundwaters for public use unless a groundwater protection needs assessment has been prepared pursuant to 415 ILCS 5/17.1(d).

2) If a preventive notification is provided under Section 620.305(c) by a non-community water supply or for multiple private water supply wells, the Department of Public Health shall conduct a sanitary survey within 1,000 feet of the wellhead of a non-community water supply or within 500 feet of the wellheads for multiple private water supply wells.

3) If a preventive notification under Section 620.305(b) is provided by the owner or operator of a regulated entity and the applicable standard in Subpart D has not been exceeded:

A) The appropriate regulatory agency shall determine if any of the following occurs for Class I: Potable Resource Groundwater:

i) The levels set forth below are exceeded or are changed for pH:

CAS No.	**Constituent**	**Criteria (mg/L)**
	~~Para-Dichlorobenzene~~	~~0.005~~
95-50-1	Ortho-Dichlorobenzene	0.01
	~~Ethylbenzene~~	~~0.03~~
1634-04-4	Methyl Tertiary-Butyl Ether (MTBE)	0.02
108-95-2	Phenols	0.001
100-42-5	Styrene	0.01
108-88-3	Toluene	0.04
1330-20-7	Xylenes	0.02

ii) A statistically significant increase occurs above background (as determined pursuant to other regulatory procedures (e.g., 35 Ill. Adm. Code 616, 724, 725 or 811) or Unified Guidance incorporated by reference in Section 620.125) for aluminum, ~~arsenic~~, beryllium, boron, cadmium, chromium, cyanide, lead, lithium, mercury, molybdenum, nitrate, perchlorate, thallium, or

vanadium (except due to natural causes); or for acenaphthene, acetone, aldicarb, anthracene, atrazine and metabolites, benzoic acid, 2-butanone (MEK), carbofuran, carbon disulfide, ~~carbofuran,~~ chlorobenzene, 2,4-D, dalapon, ~~2-butanone (MEK),~~ dicamba, dichlorodifluoromethane, 1,1-dichloroethane, 1,2-dichloroethylene, cis-1,2-dichloroethylene, trans-dichloroethylene, diethyl phthalate, di-n-butyl phthalate, dinoseb, endothall, endrin, ~~endothall~~, fluoranthene, fluorine, hexachlorocyclopentadiene, isopropylbenzene (cumene), ~~lindane (gamma-hexachloro-cyclohexane),~~ ~~2,4-D, 1,1-dichloroethylene, cis-1,2-dichloroethylene, trans-1,2-dichloroethylene,~~ MCPP (mecoprop), methoxychlor, 1-methylnaphthalene, 2-methylnaphthalene, ~~methoxychlor,~~ 2-methylphenol (o-cresol), ~~monochlorobenzene,~~ naphthalene, ~~perchlorate,~~ Perfluorobutane Sulfonic Acid (PFBS), Perfluorohexane Sulfonic Acid (PFHxS), Perfluorononanoic Acid (PFNA), Perfluorooctanoic Acid (PFOA), Perfluorooctane Sulfonic Acid (PFOS), picloram, pyrene, simazine, 2,4,5-TP (silvex), sulfate, total dissolved solids, ~~1,2,4-trichlorobenzene,~~ 1,1,1-trichloroethane, 1,1,2-trichloroethane, ~~1,1,1trichloroethane,~~ and trichlorofluoromethane.

iii) For a chemical constituent of gasoline, diesel fuel, or heating fuel, the constituent exceeds the following:

Constituent	**Criterion (mg/L)**
BETX	0.095

iv) For pH, a statistically significant change occurs from background.

BOARD NOTE: Constituents that are carcinogens have not been listed in subsection (a)(3)(A) because the standard is set at the PQL and any exceedence thereof is a violation subject to corrective action.

B) The appropriate agency shall determine if, for Class III: Special Resource Groundwater, the levels as determined by the Board are exceeded.

C) The appropriate regulatory agency shall consider whether the owner or operator reasonably demonstrates that:

i) The contamination is a result of contaminants remaining in groundwater from a prior release for which appropriate action was taken in accordance with laws and regulations in existence at the time of the release;

ii) The source of contamination is not due to the on-site release of contaminants; or

iii) The detection resulted from error in sampling, analysis, or evaluation.

D) The appropriate regulatory agency shall consider actions necessary to minimize the degree and extent of contamination.

b) The appropriate regulatory agency shall determine whether a preventive response must be undertaken based on relevant factors including, but not limited to, the considerations in subsection (a)(3).

c) After completion of preventive response pursuant to authority of an appropriate regulatory agency, the concentration of a contaminant listed in subsection (a)(3)(A) in groundwater may exceed 50 percent of the applicable numerical standard in Subpart D only if the following conditions are met:

1) The exceedence has been minimized to the extent practicable;

2) Beneficial use, as appropriate for the class of groundwater, has been assured; and

3) Any threat to public health or the environment has been minimized.

d) Nothing in this Section shall in any way limit the authority of the State or of the United States to require or perform any corrective action process.

(Source: Amended at __ Ill. Reg. _____, effective _____)

SUBPART D: GROUNDWATER QUALITY STANDARDS

Section 620.410 Groundwater Quality Standards for Class I: Potable Resource Groundwater

a) Inorganic Chemical Constituents
Except due to natural causes or as provided in Section 620.450, concentrations of the following chemical constituents must not be exceeded in Class I groundwater:

CAS No.	Constituent	Units	Standard
7429-90-5	Aluminum	mg/L	3.5
7440-36-0	Antimony	mg/L	0.006
7440-38-2	Arsenic*	mg/L	0.010
7440-39-3	Barium	mg/L	2.0
7440-41-7	Beryllium	mg/L	0.004
7440-42-8	Boron	mg/L	1.4~~2.0~~
7440-43-9	Cadmium	mg/L	0.005
16887-00-6	Chloride	mg/L	200.0
7440-47-3	Chromium	mg/L	0.1
7440-48-4	Cobalt	mg/L	0.0021~~1.0~~
7440-50-8	Copper	mg/L	0.5~~0.65~~
57-12-5	Cyanide	mg/L	0.2
16984-48-8	Fluoride	mg/L	2.0~~4.0~~
7439-89-6	Iron	mg/L	5.0
7439-92-1	Lead	mg/L	0.0075
7439-93-2	Lithium	mg/L	0.014
7439-96-5	Manganese	mg/L	0.15
7487-94-7	Mercury	mg/L	0.002
7439-98-7	Molybdenum	mg/L	0.035
7440-02-0	Nickel	mg/L	0.1
14797-55-8	Nitrate as N	mg/L	10.0
14797-73-0	Perchlorate	mg/L	0.0049
~~13982-63-3~~	~~Radium 226~~	~~pCi/L~~	~~20.0~~
~~15262-20-1~~	~~Radium 228~~	~~pCi/L~~	~~20.0~~
13982-63-3 15262-20-1	Combined Radium (226 + 228)	pCi/L	5
7782-49-2	Selenium	mg/L	0.02~~0.05~~
7440-22-4	Silver	mg/L	0.035~~0.05~~

CAS No.	Constituent	Units	Standard
14808-79-8	Sulfate	mg/L	400.0
7440-28-0	Thallium	mg/L	0.002
	Total Dissolved Solids (TDS)	mg/L	1,200
7440-62-2	Vanadium	mg/L	0.00049~~0.049~~
7440-66-6	Zinc	mg/L	5.0

*Denotes a carcinogen.

b) Organic Chemical Constituents
Except due to natural causes or as provided in Section 620.450 or subsection (d), concentrations of the following organic chemical constituents shall not be exceeded in Class I groundwater:

CAS No.	Constituent	Standard (mg/L)
83-32-9	Acenaphthene	0.42
67-64-1	Acetone	6.3
15972-60-8	Alachlor*	0.002
116-06-3	Aldicarb	0.003
120-12-7	Anthracene	2.1
	~~Atrazine~~	~~0.003~~
71-43-2	Benzene*	0.005
56-55-3	Benzo(a)anthracene*	0.00085~~0.00013~~
205-99-2	Benzo(b)fluoranthene*	0.00085~~0.00018~~
207-08-9	Benzo(k)fluoranthene*	0.0085~~0.00017~~
50-32-8	Benzo(a)pyrene*	0.0002
65-85-0	Benzoic acid	28.0
78-93-3	2-Butanone (methyl ethyl ketone)	4.2
1563-66-2	Carbofuran	0.04
75-15-0	Carbon Disulfide	0.7
56-23-5	Carbon Tetrachloride*	0.005
12789-03-6	Chlordane*	0.002
108-90-7	Chlorobenzene	0.1
67-66-3	Chloroform*	0.07
218-01-9	Chrysene*	0.085~~0.012~~
94-75-7	2,4-D	0.07
75-99-0	Dalapon	0.2
53-70-3	Dibenzo(a,h)anthracene*	0.000085~~0.0003~~
96-12-8	1,2-Dibromo-3-Chloropropane*	0.0002
1918-00-9	Dicamba	0.21
95-50-1	ortho-Dichlorobenzene	0.6
106-46-7	para-Dichlorobenzene*	0.075

CAS No.	Constituent	Standard (mg/L)
75-71-8	Dichlorodifluoromethane	1.4
75-34-3	1,1-Dichloroethane	1.4
107-06-2	1,2-Dichloroethane*	0.005
75-35-4	1,1-Dichloroethylene	0.007
156-59-2	cis-1,2-Dichloroethylene	0.07
156-60-5	trans-1,2-Dichloroethylene	0.1
75-09-2	Dichloromethane (methylene chloride)*	0.005
78-87-5	1,2-Dichloropropane*	0.005
117-81-7	Di(2-ethylhexyl)phthalate*	0.006
84-66-2	Diethyl Phthalate	5.6
84-74-2	Di-n-butyl Phthalate	0.7
99-65-0	1,3-Dinitrobenzene	0.0007
121-14-2	2,4-Dinitrotoluene*	0.00027
606-20-2	2,6-Dinitrotoluene*	0.000057
88-85-7	Dinoseb	0.007
123-91-1	1,4-Dioxane (p-dioxane)*	0.00085
145-73-3	Endothall	0.1
72-20-8	Endrin	0.002
100-41-4	Ethylbenzene*	0.7
106-93-4	Ethylene Dibromide*	0.00005
206-44-0	Fluoranthene	0.28
86-73-7	Fluorene	0.28
76-44-8	Heptachlor*	0.0004
1024-57-3	Heptachlor Epoxide*	0.0002
319-84-6	Hexachlorocyclohexane, alpha-*	0.000014
58-89-9	Hexachlorocyclohexane, gamma-(Lindane)*	0.0002
77-47-4	Hexachlorocyclopentadiene	0.05
2691-41-0	HMX (High Melting Explosive, Octogen)	1.4
193-39-5	Indeno(1,2,3-cd)pyrene*	0.00085~~0.00043~~
98-82-8	Isopropylbenzene (Cumene)	0.7
	~~Lindane (Gamma-Hexachlorocyclohexane)~~	~~0.0002~~
	~~2,4-D~~	~~0.07~~
	~~ortho-Dichlorobenzene~~	~~0.6~~
	~~para-Dichlorobenzene~~	~~0.075~~
	~~1,2-Dibromo-3-Chloropropane*~~	~~0.0002~~
	~~1,2-Dichloroethane*~~	~~0.005~~
	~~1,1-Dichloroethylene~~	~~0.007~~
	~~cis-1,2-Dichloroethylene~~	~~0.07~~
	~~trans-1,2-Dichloroethylene~~	~~0.1~~
	~~1,2-Dichloropropane*~~	~~0.005~~

CAS No.	Constituent	Standard (mg/L)
	~~Ethylbenzene~~	~~0.7~~
93-65-2	MCPP (Mecoprop)	0.007
72-43-5	Methoxychlor	0.04
90-12-0	1-Methylnaphthalene	0.49
91-57-6	2-Methylnaphthalene	0.028
95-48-7	2-Methylphenol (o-cresol)	0.35
1634-04-4	Methyl Tertiary-Butyl Ether (MTBE)	0.07
	~~Monochlorobenzene~~	~~0.1~~
91-20-3	Naphthalene	0.14
98-95-3	Nitrobenzene	0.014
	~~P-Dioxane*~~	~~0.0077~~
87-86-5	Pentachlorophenol*	0.001
375-73-5	Perfluorobutane Sulfonic Acid (PFBS)	0.14
335-46-4	Perfluorohexane Sulfonic Acid (PFHxS)	0.00014
375-95-1	Perfluorononanoic Acid (PFNA)	0.000021
108-95-2	Phenols	0.1
1918-02-1	Picloram	0.5
	~~Pyrene~~	~~0.21~~
1336-36-3	Polychlorinated Biphenyls (PCBs) (as decachloro-biphenyl)*	0.0005
	~~alpha-BHC (alpha-Benzene hexachloride)*~~	~~0.00011~~
129-00-0	Pyrene	0.21
121-82-4	RDX (Royal Demolition Explosive, Cyclonite)	0.07
122-34-9	Simazine	0.004
100-42-5	Styrene	0.1
93-72-1	2,4,5-TP (Silvex)	0.05
127-18-4	Tetrachloroethylene*	0.005
108-88-3	Toluene	1.0
8001-35-2	Toxaphene*	0.003
120-82-1	1,2,4-Trichlorobenzene*	0.07
71-55-6	1,1,1-Trichloroethane	0.2
79-00-5	1,1,2-Trichloroethane	0.005
	~~1,2,4-Trichlorobenzene~~	~~0.07~~
79-01-6	Trichloroethylene*	0.005
75-69-4	Trichlorofluoromethane	2.1
99-35-4	1,3,5-Trinitrobenzene	0.84
118-96-7	2,4,6-Trinitrotoluene (TNT)	0.014
75-01-4	Vinyl Chloride*	0.002
1330-20-7	Xylenes	10.0

*Denotes a carcinogen.

c) ~~Explosive Constituents~~
~~Concentrations of the following explosive constituents must not exceed the Class I groundwater standard:~~

~~Constituent~~	~~Standard (mg/L)~~
~~1,3-Dinitrobenzene~~	~~0.0007~~
~~2,4-Dinitrotoluene*~~	~~0.0001~~
~~2,6-Dinitrotoluene*~~	~~0.00031~~
~~HMX (High Melting Explosive, Octogen)~~	~~1.4~~
~~Nitrobenzene~~	~~0.014~~
~~RDX (Royal Demolition Explosive, Cyclonite)~~	~~0.084~~
~~1,3,5-Trinitrobenzene~~	~~0.84~~
~~2,4,6-Trinitrotoluene (TNT)~~	~~0.014~~

~~*Denotes a carcinogen.~~

~~d)~~ Complex Organic Chemical Mixtures

1) Concentrations of the following chemical constituents ~~of gasoline, diesel fuel, or heating fuel~~ must not be exceeded in Class I groundwater:

CAS No.	**Constituent**	**Standard (mg/L)**
71-43-2	Benzene*	0.005
	BETX	11.705

*Denotes a carcinogen.

2) Atrazine and Metabolites

In addition to atrazine, the following atrazine metabolites shall be analyzed, and the total concentration of atrazine and metabolites shall be compared to the atrazine Class I groundwater standard of 0.003 mg/l.

CAS No.	**Constituent**	**Standard (mg/L)**
	Total Atrazine and metabolites:	0.003

1912-24-9	Atrazine Desethyl-atrazine (DEA) Desisopropyl-atrazine (DIA) Diaminochlorotriazine (DACT)

3) The concentrations of the following constituents must not be exceeded in Class I groundwater at both the individual standards and a combined standard of 0.000021 mg/L.

CAS No.	**Constituent**	**Standard (mg/L)**
335-67-1	Perfluorooctanoic Acid (PFOA)	0.000021
1763-23-1	Perfluorooctane Sulfonic Acid (PFOS)	0.000014

d)~~e)~~ pH
Except due to natural causes, a pH range of 6.5 - 9.0 units must not be exceeded in Class I groundwater.

e)~~f)~~ Beta Particle and Photon Radioactivity

1) Except due to natural causes, the average annual concentration of beta particle and photon radioactivity from man-made radionuclides shall not exceed a dose equivalent to the total body organ greater than 4 mrem/year in Class I groundwater. If two or more radionuclides are present, the sum of their dose equivalent to the total body, or to any internal organ shall not exceed 4 mrem/year in Class I groundwater except due to natural causes.

2) Except for the radionuclides listed in subsection (f)(3), the concentration of man-made radionuclides causing 4 mrem total body or organ dose equivalent must be calculated on the basis of a 2 liter per day drinking water intake using the 168-hour data in accordance with the procedure set forth in NCRP Report Number 22, incorporated by reference at Section 620.125(a).

3) Except due to natural causes, the average annual concentration assumed to produce a total body or organ dose of 4 mrem/year of the following chemical constituents shall not be exceeded in Class I groundwater:

CAS No.	**Constituent**	**Critical Organ**	**Standard (pCi/L)**

10028-17-8	Tritium	Total body	20,000.0
7440-24-6	Strontium-90	Bone marrow	8.0

(Source: Amended at __ Ill. Reg. _____, effective _____)

Section 620.420 Groundwater Quality Standards for Class II: General Resource Groundwater

a) Inorganic Chemical Constituents

1) Except due to natural causes or as provided in Section 620.450 or subsection (a)(3) or (e) of this Section, concentrations of the following chemical constituents must not be exceeded in Class II groundwater:

CAS No.	**Constituent**	**Standard (mg/L)**
7440-36-0	Antimony	0.024
7440-38-2	Arsenic*	0.2
7440-39-3	Barium	2.0
7440-41-7	Beryllium	0.5
7440-43-9	Cadmium	0.05
7440-47-3	Chromium	1.0
7440-48-4	Cobalt	1.0
57-12-5	Cyanide	0.6
16984-48-8	Fluoride	2.0~~4.0~~
7439-92-1	Lead	0.1
7439-93-2	Lithium	2.5
7487-94-7	Mercury	0.01
7439-98-7	Molybdenum	0.05
14797-55-8	Nitrate as N	100.0
14797-73-0	Perchlorate	0.0049
7440-28-0	Thallium	0.02
7440-62-2	Vanadium	0.1

*Denotes a carcinogen.

2) Except due to natural causes or as provided in Section 620.450 or subsection (a)(3) or (e) of this Section, concentrations of the following chemical constituents must not be exceeded in Class II

groundwater:

CAS No.	Constituent	Units	Standard ~~(mg/L)~~
7429-90-5	Aluminum	mg/L	5.0
7440-42-8	Boron	mg/L	2.0
16887-00-6	Chloride	mg/L	200.0
7440-50-8	Copper	mg/L	0.5~~.65~~
7439-89-6	Iron	mg/L	5.0
7439-96-5	Manganese	mg/L	10.0
7440-02-0	Nickel	mg/L	2.0
13982-63-3 15262-20-1	Combined Radium (226 + 228)	pCi/L	5
7782-49-2	Selenium	mg/L	0.02~~0.05~~
7440-22-4	Silver	mg/L	0.035
	~~Total Dissolved Solids (TDS)~~		~~1,200.0~~
14808-79-8	Sulfate	mg/L	400.0
	Total Dissolved Solids (TDS)	mg/L	1,200.0
7440-66-6	Zinc	mg/L	10.0

3) The standard for any inorganic chemical constituent listed in subsection (a)(2) of this Section, for barium, or for pH does not apply to groundwater within fill material or within the upper 10 feet of parent material under such fill material on a site not within the rural property class for which:

A) Prior to November 25, 1991, surficial characteristics have been altered by the placement of such fill material so as to impact the concentration of the parameters listed in subsection (a)(3) of this Section, and any on-site groundwater monitoring of such parameters is available for review by the Agency.

B) On November 25, 1991, surficial characteristics are in the process of being altered by the placement of such fill material, that proceeds in a reasonably continuous manner to completion, so as to impact the concentration of the parameters listed in subsection (a)(3) of this Section, and any on-site groundwater monitoring of such parameters is

available for review by the Agency.

4) For purposes of subsection (a)(3) of this Section, the term "fill material" means clean earthen materials, slag, ash, clean demolition debris, or other similar materials.

b) Organic Chemical Constituents

1) Except due to natural causes or as provided in Section 620.450 or subsection (b)(2) or (e) of this Section, concentrations of the following organic chemical constituents must not be exceeded in Class II groundwater:

CAS No.	Constituent	Standard (mg/L)
83-32-9	Acenaphthene	2.1
67-64-1	Acetone	6.3
15972-60-8	Alachlor*	0.002~~0.010~~
116-06-3	Aldicarb	0.003~~0.015~~
120-12-7	Anthracene	10.5
	~~Atrazine~~	~~0.015~~
71-43-2	Benzene*	0.025
56-55-3	Benzo(a)anthracene*	0.0043~~0.00065~~
205-99-2	Benzo(b)fluoranthene*	0.0043~~0.0009~~
207-08-9	Benzo(k)fluoranthene*	0.043~~0.006~~
50-32-8	Benzo(a)pyrene*	0.002
65-85-0	Benzoic acid	28.0
78-93-3	2-Butanone (methyl ethyl ketone~~MEK~~)	4.2
	~~Carbon Disulfide~~	~~3.5~~
1563-66-2	Carbofuran	0.04~~0.2~~
75-15-0	Carbon Disulfide	3.5
56-23-5	Carbon Tetrachloride*	0.025
12789-03-6	Chlordane*	0.01
108-90-7	Chlorobenzene	0.1~~0.5~~
67-66-3	Chloroform*	0.35
218-01-9	Chrysene*	0.43~~0.06~~
94-75-7	2,4-D	0.07~~0.35~~
75-99-0	Dalapon	0.2~~2.0~~
53-70-3	Dibenzo(a,h)anthracene*	0.00043~~0.0015~~
96-12-8	1,2-Dibromo-3-Chloropropane*	0.0002
1918-00-9	Dicamba	0.21
95-50-1	ortho-Dichlorobenzene	0.6~~1.5~~
106-46-7	para-Dichlorobenzene*	0.075~~0.375~~

CAS No.	Constituent	Standard (mg/L)
75-71-8	Dichlorodifluoromethane	7.0
75-34-3	1,1-Dichloroethane	7.0
107-06-2	1,2-Dichloroethane*	0.005~~0.025~~
75-35-4	1,1-Dichloroethylene	0.035
156-59-2	cis-1,2-Dichloroethylene	0.35~~0.2~~
156-60-5	trans-1,2-Dichloroethylene	0.5
75-09-2	Dichloromethane (methylene chloride)*	0.005~~0.05~~
78-87-5	1,2-Dichloropropane*	0.005~~0.025~~
117-81-7	Di(2-ethylhexyl)phthalate*	0.06
84-66-2	Diethyl Phthalate	5.6
84-74-2	Di-n-butyl Phthalate	3.5
99-65-0	1,3-Dinitrobenzene	0.0007
121-14-2	2,4-Dinitrotoluene*	0.0014
606-20-2	2,6-Dinitrotoluene*	0.00029~~0.0002 8~~
88-85-7	Dinoseb	0.07
123-91-1	1,4-Dioxane (p-)*	0.00085
145-73-3	Endothall	0.1
72-20-8	Endrin	0.01
100-41-4	Ethylbenzene*	3.5~~1.0~~
106-93-4	Ethylene Dibromide*	0.00005~~0.0005~~
206-44-0	Fluoranthene	1.4
86-73-7	Fluorene	1.4
76-44-8	Heptachlor*	0.002
1024-57-3	Heptachlor Epoxide*	0.001
319-84-6	Hexachlorocyclohexane, alpha-*	0.00007
58-89-9	Hexachlorocyclohexane, gamma- (Lindane)*	0.001
77-47-4	Hexachlorocyclopentadiene	0.5
2691-41-0	HMX (High Melting Explosive, Octogen)	7.0
193-39-5	Indeno(1,2,3-cd)pyrene*	0.0043~~0.0022~~
98-82-8	Isopropylbenzene (Cumene)	3.5
	~~Lindane (Gamma-Hexachloro-cyclophexane)~~	~~0.001~~
	~~2,4-D~~	~~0.35~~
	~~Ortho-Dichlorobenze~~	~~1.5~~
	~~Para-Dichlorobenzene~~	~~0.375~~
	~~1,2-Dibromo-3-Chloropropane*~~	~~0.002~~
	~~1,2-Dichloroethane*~~	~~0.025~~
	~~1,1-Dichloroethylene~~	~~0.035~~
	~~cis-1,2-Dichloroethylene~~	~~0.2~~

CAS No.	Constituent	Standard (mg/L)
	~~Trans-1,2-Dichloroethylene~~	~~0.5~~
	~~1,2-Dichloropropane*~~	~~0.025~~
	~~Ehylbenzene~~	~~1.0~~
93-65-2	MCPP (Mecoprop)	0.007
72-43-5	Methoxychlor	0.2
90-12-0	1-Methylnaphthalene	2.5~~2.4~~
91-57-6	2-Methylnaphthalene	0.14
95-48-7	2-Methylphenol (o-cresol)	0.35
1634-04-4	Methyl Tertiary-Butyl Ether (MTBE)	0.07
	~~Monochlorobenzene~~	~~0.5~~
91-20-3	Naphthalene	0.22
98-95-3	Nitrobenzene	0.014
	~~P-Dioxane*~~	~~0.0077~~
87-86-5	Pentachlorophenol*	0.005
375-73-5	Perfluorobutane Sulfonic Acid (PFBS)	0.14
335-46-4	Perfluorohexane Sulfonic Acid (PFHxS)	0.00014
375-95-1	Perfluorononanoic Acid (PFNA)	0.000021
108-95-2	Phenols	0.1
1918-02-1	Picloram	0.5~~5.0~~
	~~Pyrene~~	~~1.05~~
1336-36-3	Polychlorinated Biphenyls (PCBs) (as decachloro-biphenyl)*	0.0025
	~~alpha-BHC (alpha-Benzene hexachloride)*~~	~~0.00055~~
129-00-0	Pyrene	1.05
121-82-4	RDX (Royal Demolition Explosive, Cyclonite)	0.07
122-34-9	Simazine	0.004~~0.04~~
100-42-5	Styrene	0.1~~0.5~~
93-72-1	2,4,5-TP (Silvex)	0.05~~0.25~~
127-18-4	Tetrachloroethylene*	0.025
108-88-3	Toluene	5.0~~2.5~~
8001-35-2	Toxaphene*	0.015
120-82-1	1,2,4-Trichlorobenzene*	0.7
71-55-6	1,1,1-Trichloroethane	1.0
	~~1,2,4-Trichlorobenzene~~	~~0.7~~
79-00-5	1,1,2-Trichloroethane	0.005~~0.05~~
79-01-6	Trichloroethylene*	0.025
75-69-4	Trichlorofluoromethane	10.5
99-35-4	1,3,5-Trinitrobenzene	4.2

CAS No.	Constituent	Standard (mg/L)
118-96-7	2,4,6-Trinitrotoluene (TNT)	0.07
75-01-4	Vinyl Chloride*	0.01
1330-20-7	Xylenes	10.0

* Denotes a carcinogen.

2) The standards for pesticide chemical constituents listed in subsection (b)(1) of this Section do not apply to groundwater within 10 feet of the land surface, provided that the concentrations of such constituents result from the application of pesticides in a manner consistent with the requirements of the Federal Insecticide, Fungicide and Rodenticide Act (7 USC 136 et seq.) and the Illinois Pesticide Act [415 ILCS 60].

c) ~~Explosive Constituents~~
~~Concentrations of the following explosive constituents must not exceed the Class II groundwater standard:~~

~~Constituent~~	~~Standard (mg/L)~~
~~1,3-Dinitrobenzene~~	~~0.0007~~
~~2,4-Dinitrotoluene*~~	~~0.0001~~
~~2,6-Dinitrotoluene*~~	~~0.00031~~
~~HMX (High Melting Explosive, Octogen)~~	~~1.4~~
~~Nitrobenzene~~	~~0.014~~
~~RDX (Royal Demolition Explosive, Cyclonite)~~	~~0.084~~
~~1,3,5-Trinitrobenzene~~	~~0.84~~
~~2,4,6-Trinitrotoluene (TNT)~~	~~0.014~~

~~*Denotes a carcinogen.~~

~~d)~~ 1) Complex Organic Chemical Mixtures
Concentrations of the following organic chemical constituents of gasoline, diesel fuel, or heating fuel must not be exceeded in Class II groundwater:

CAS No.	Constituent	Standard (mg/L)
71-43-2	Benzene*	0.025

	BETX	18.525~~13.525~~

*Denotes a carcinogen

2) In addition to atrazine, the following atrazine metabolites shall be analyzed, and the total concentration of atrazine and metabolites shall be compared to the atrazine Class II groundwater standard of 0.003 mg/l.

CAS. No	Constituent	Standard (mg/L)
	Total Atrazine and metabolites:	0.003
1912-24-9	Atrazine	
	Desethyl-atrazine (DEA)	
	Desisopropyl-atrazine (DIA)	
	Diaminochlorotriazine (DACT)	

3) The concentrations of the following constituents must not be exceeded in Class II groundwater at both the individual standards and a combined standard of 0.000021 mg/L:

CAS No.	Constituent	Individual Standard (mg/L)
335-67-1	Perfluorooctanoic Acid (PFOA)	0.000021
1763-23-1	Perfluorooctane Sulfonic Acid (PFOS)	0.000014

d)~~e)~~ pH
Except due to natural causes, a pH range of 6.5 - 9.0 units must not be exceeded in Class II groundwater that is within 5 feet of the land surface.

(Source: Amended at __ Ill. Reg. _____, effective _____)

Section 620.430 Groundwater Quality Standards for Class III: Special Resource Groundwater

Except due to natural causes, concentrations ~~Concentrations~~ of inorganic and organic chemical constituents must not exceed the standards set forth in Section 620.410, except for ~~those~~:

a) The chemical constituents for which the Board has adopted a standard pursuant to Section 620.260; and

b) The following standards set forth below for Class III Special Resource Groundwater established in accordance with Section 620.230(b) and depicted in 620.Appendix E:

1) The following standards are applicable for Pautler Cave Nature Preserve, Stemler Cave Nature Preserve, Fogelpole Cave Nature Preserve and Armin Krueger Speleological Nature Preserve:

Chloride	20 mg/L
pH	range of 7.0-9.0 Standard Units

2) The following standard is applicable for Cotton Creek Marsh Nature Preserve and Spring Grove Fen Nature Preserve:

Chloride	45 mg/L

(Source: Amended at __ Ill. Reg. _____, effective _____)

SUBPART E: GROUNDWATER MONITORING AND ANALYTICAL PROCEDURES

Section 620.510 Monitoring and Analytical Requirements

a) Representative Samples
A representative sample shall be taken from locations as specified in Section 620.505.

b) Sampling and Analytical Procedures

1) Samples must be collected in accordance with the procedures set forth in the documents pertaining to groundwater monitoring and analysis "Methods for Chemical Analysis of Water and Wastes," "Methods for the Determination of Inorganic Substances in Environmental Samples," "Methods for the Determination of Metals in Environmental Samples," "Methods for the Determination of Organic Compounds in Drinking Water," "Methods for the Determination or Organic Compounds in

Drinking Water, Supplement I," "Methods for the Determination of Organic Compounds in Drinking Water, Supplement II," "Methods for the Determination of Organic Compounds in Drinking Water, Supplement III," "Methods for the Determination of Organic and Inorganic Compounds in Drinking Water," "Prescribed Procedures for Measurement of Radioactivity in Drinking Water," "Procedures for Radiochemical Analysis of Nuclear Reactor Aqueous Solutions," "Radiochemical Analytical Procedures for Analysis of Environmental Samples," "Radiochemistry Procedures Manual," "Practical Guide for Ground Water Sampling," "Test Methods for Evaluating Solid Wastes, Physical/Chemical Methods" (SW-846), 40 CFR 136, appendix B, 40 CFR 141.80, 40 CFR 141.61, and 40 CFR 141.62, "Techniques of Water Resources Investigations of the United States Geological Survey, Guidelines for Collection and Field Analysis of Ground Water Samples for Selected Unstable Constituents," "Practical Guide for Ground-Water Sampling," "Techniques of Water Resources Investigations of the United States Geological Survey, Guidelines for Collection and Field Analysis of Ground-Water Samples for Selected Unstable Constituents," incorporated by reference at Section 620.125 or other procedures adopted by the appropriate regulatory agency.

2) Groundwater elevation in a groundwater monitoring well must be determined and recorded when necessary to determine the gradient.

3) Statistical methods used to determine naturally occurring groundwater quality background concentrations of contaminants must be conducted in accordance with "Statistical Analysis of Groundwater Monitoring Data at RCRA Facilities, (March 2009 Unified Guidance)", as incorporated by reference in Section 620.125 for use with prediction limits and all other statistical tests including, but not limited to, confidence limits and control charts.

4) The analytical methodology used for the analysis of constituents in Subparts C and D must be consistent with both of the following:

A) The methodology must have a PQL at or below the preventive response levels of Subpart C or groundwater standard set forth in Subpart D, whichever is applicable; and

B) "Methods for Chemical Analysis of Water and Wastes," "Methods for the Determination of Inorganic Substances in Environmental Samples," "Methods for the Determination of Metals in Environmental Samples," "Methods for the Determination of Organic Compounds in Drinking Water," "Methods for the Determination of Organic Compounds in Drinking Water, Supplement I," "Methods for the Determination of Organic Compounds in Drinking Water, Supplement II," "Methods for the Determination of Organic Compounds in Drinking Water, Supplement III," "Methods for the Determination of Organic and Inorganic Compounds in Drinking Water," "Prescribed Procedures for Measurement of Radioactivity in Drinking Water," "Procedures for Radiochemical Analysis of Nuclear Reactor Aqueous Solutions," "Radiochemical Analytical Procedures for Analysis of Environmental Samples," "Radiochemistry Procedures Manual," "Practical Guide for Ground Water Sampling," "Test Methods for Evaluating Solid Wastes, Physical/Chemical Methods" (SW-846), 40 CFR 136, appendix B, 40 CFR 141.80, 40 CFR 141.61, and 40 CFR 141.62, "Techniques of Water Resources Investigations of the United States Geological Survey, Guidelines for Collection and Field Analysis of Ground Water Samples for Selected Unstable Constituents," "Practical Guide for Ground-Water Sampling", "Techniques of Water Resources Investigations of the United States Geological Survey, Guidelines for Collection and Field Analysis of Ground-Water Samples for Selected Unstable Constituents", incorporated by reference at Section 620.125.

c) Reporting Requirements
At a minimum, groundwater monitoring analytical results must include information, procedures and techniques for:

1) Sample collection (including but not limited to name of sample collector, time and date of the sample, method of collection, and identification of the monitoring location);

2) Sample preservation and shipment (including but not limited to field quality control);

3) Analytical procedures (including but not limited to the method

detection limits and the PQLs); and

4) Chain of custody control.

(Source: Amended at __ Ill. Reg. _____, effective _____)

SUBPART F: HEALTH ADVISORIES

Section 620.605 Issuance of a Health Advisory

a) The Agency shall issue a Health Advisory for a chemical substance if all of the following conditions are met:

1) A community water supply well is sampled and a substance is detected and confirmed by resampling;

2) There is no standard under Section 620.410 for such chemical substance; and

3) The chemical substance is toxic or harmful to human health according to the procedures of Appendix A, B, or C.

b) The Health Advisory must contain a general description of the characteristics of the chemical substance, the potential adverse health effects, and a guidance level to be determined as follows:

1) If disease or functional impairment is caused due to a physiological mechanism for where there is a threshold dose below which no damage occurs, the guidance level for any such substance shall be the Maximum Contaminant Level Goal (MCLG), adopted by USEPA for such substance, 40 CFR 136, appendix B, 40 CFR 141.80, 40 CFR 141.61, and 40 CFR 141.62, incorporated by reference at Section 620.125. If there is no MCLG for the substance, the guidance level is the Human Threshold Toxicant Advisory Concentration for such substance as determined in accordance with Appendix A~~, unless the concentration for such substance is less than the lowest appropriate PQL specified in "Test Methods for Evaluating Solid Wastes, Physical/Chemical Methods", EPA Publication No. SW-846 (SW-846), incorporated by reference at Section 620.125 for the substance. If the concentration for such substance is less than the lowest appropriate PQL for the substance specified in SW-846, incorporated by reference at Section 620.125, the guidance level is the lowest appropriate PQL~~.

2) If the chemical substance is a carcinogen, the guidance level for any such chemical substance is the one-in-one-million cancer risk concentration~~, unless the concentration for such substance is less than the lowest appropriate PQL specified in "Test Methods for Evaluating Solid Wastes, Physical/Chemical Methods," EPA Publication No. SW-846 (SW-846), incorporated by reference at Section 620.125 for such substance. If the concentration for such substance is less than the lowest appropriate PQL for the substance specified in SW-846, the guidance level is the lowest appropriate PQL~~. The one-in-one-million cancer risk concentration, the Human Nonthreshold Toxicant Advisory Concentration (HNTAC), shall be determined according to the following equation:

$$\frac{HNTAC}{(mg/L)} = \frac{TR \times BW \times AT \times 365\ days/year}{SFo \times IR \times EF \times ED}$$

Where:

TR = Target Risk = 1.0E-06
BW = Body Weight = 70 kg
AT = Averaging Time = 70 years
SFo = Oral Slope Factor = Chemical-specific
IR = Daily Water Ingestion Rate = 2 liters/day
EF = Exposure Frequency = 350 days/year
ED = Exposure Duration = 30 years

(Source: Amended at __ Ill. Reg. _____, effective _____)

Section 620.Appendix B Procedures for Determining Hazard Indices for Class I: Potable Resource Groundwater for Mixtures of Similar-Acting Substances

a) This appendix describes procedures for evaluating mixtures of similar- acting substances which may be present in Class I: Potable Resource Groundwaters. Except as provided otherwise in subsection (c), subsections (d) through (h) describe the procedure for determining the Hazard Index for mixtures of similar-acting substances.

b) For the purposes of this appendix, a "mixture" means two or more substances which are present in Class I: Potable Resource Groundwater which may or may not be related either chemically or commercially, but which are not complex mixtures of related isomers and congeners which are produced as commercial products (for example, PCBs or technical grade chlordane).

c) The following substances listed in Section 620.410 are mixtures of similar acting substances:

1) Mixtures of ortho-Dichlorobenzene and para-Dichlorobenzene. The Hazard Index ("HI") for such mixtures is determined as follows:

HI = [ortho-Dichlorobenzene]\0.6+ [para-Dichlorobenzene]\0.075

2) Mixtures of 1,1-Dichloroethylene and 1,1,1-trichloroethane. The Hazard Index ("HI") for such mixtures is determined as follows:

HI= [1,1-Dichloroethylene]\0.007 + [1,1,1-trichloroethane]\0.2

d) When two or more substances occur together in a mixture, the additivity of the toxicities of some or all of the substances will be considered when determining health-based standards for Class I: Potable Resource Groundwater. This is done by the use of a dose addition model with the development of a Hazard Index for the mixture of substances with similar-acting toxicities. This method does not address synergism or antagonism. Guidelines for determining when the dose addition of similar-acting substances is appropriate are presented in Appendix C.

The Hazard Index is calculated as follows:

HI= [A]/ALA+ [BJ/ALB+ … [I]/ALI)

Where:

HI = Hazard Index, unitless.

[A], [BJ, [I]= Concentration of each similar-acting substance in groundwater in milligrams per liter (mg/L).

ALA, ALB, ALI = The acceptable level of each similar-acting substance in themixture in milligrams per liter (mg/L).

e) For substances which are considered to have a threshold mechanism of toxicity, the acceptable level is:

1) The standards listed in Section 620.410; or

2) For those substances for which standards have not been established in Section 620.410, the Human Threshold Toxicant Advisory Concentration (HTTAC) as determined in Appendix A.

f) For substances which are carcinogens, the acceptable level is:

1) The standards listed in Section 620.410; or

~~2)~~ For those substances for which standards have not been established under Section 620.410, the one-in-one-million cancer risk concentration~~, unless the concentration for such substance is less than the lowest appropriate POL specified in "Test Methods for Evaluating Solid Wastes, Physical/Chemical Methods," EPA Publication No. SW- 846, incorporated by reference at Section 620.125, for the substance, in which case the lowest appropriate POL shall be the acceptable level.~~

g) Since the assumption of dose addition is most properly applied to substances that induce the same effect by similar modes of

action, a separate HI must be generated for each toxicity endpoint of concern.

h) In addition to meeting the individual substance objectives, a Hazard Index must be less than or equal to 1 for a mixture of similar-acting substances.

(Source: Amended at Ill. Reg. , effective)

Section 620.Appendix C Guidelines for Determining When Dose Addition of Similar-Acting Substances in Class I: Potable Resource Groundwaters is Appropriate

a) Substances must be considered similar-acting if:

1) The substances have the same target in an organism (for example, the same organ, organ system, receptor, or enzyme).

2) The substances have the same mode of toxic action. These actions may include, for example, central nervous system depression, liver toxicity, or cholinesterase inhibition.

b) Substances that have fundamentally different mechanisms of toxicity (threshold toxicants vs. carcinogens) must not be considered similar-acting. However, carcinogens which also cause a threshold toxic effect should be considered in a mixture with other similar-acting substances having the same threshold toxic effect. In such a case, an Acceptable Level for the carcinogen must be derived for its threshold effect, using the procedures described in Appendix A.

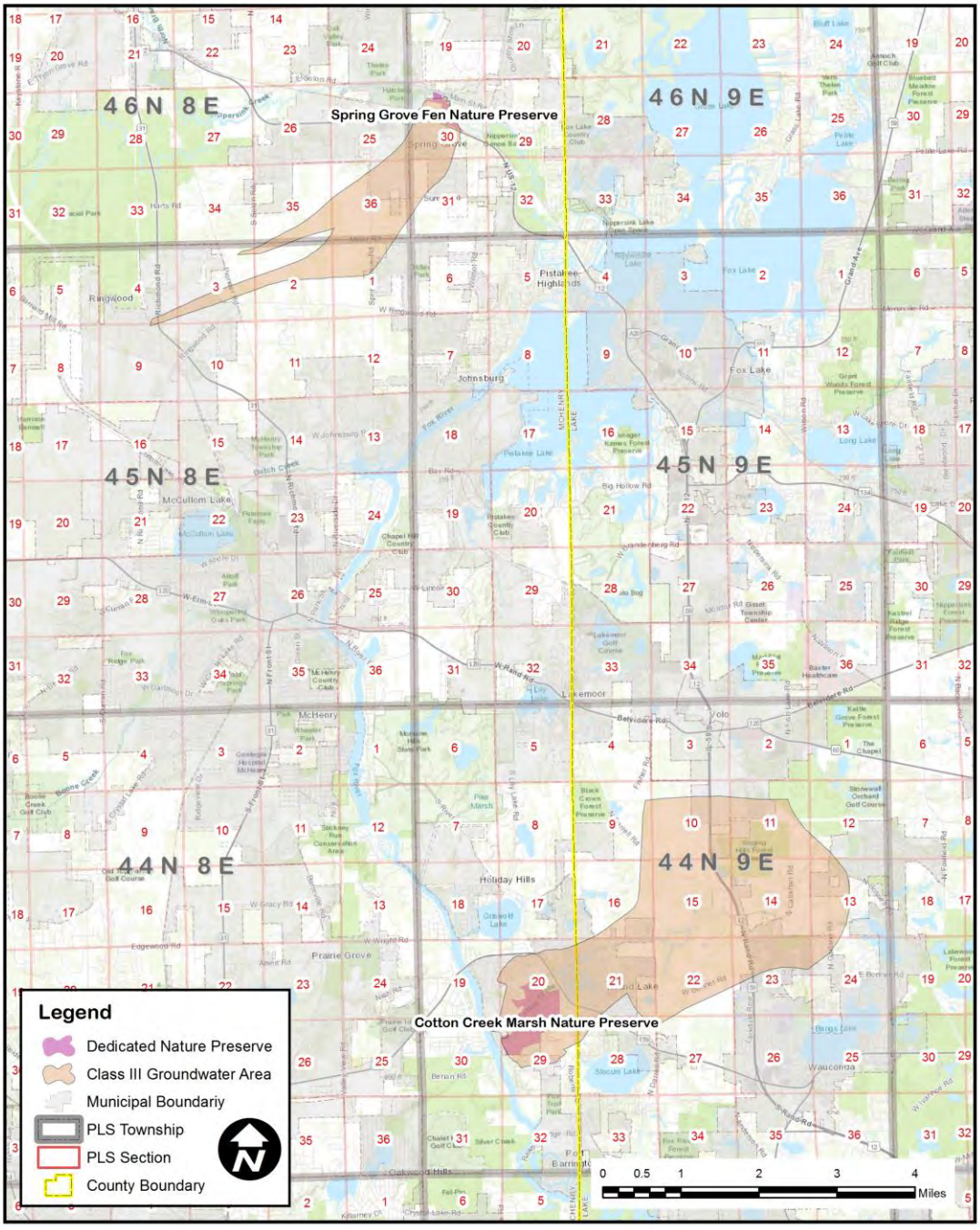
c) Substances which are components of a complex mixture of related compounds which are produced as commercial products (for example, PCBs or technical grade chlordane) are not mixtures, as defined in Appendix B. Such complex mixtures are equivalent to a single substance. In such a case, the Human Threshold Toxicant Advisory Concentration may be derived for threshold effects of the complex mixture, using the procedures described in Appendix A, if valid toxicological or epidemiological data are available for the complex mixture. If the complex mixture is a carcinogen, the Health Advisory Concentration is the one-in-one-million cancer risk

concentration~~, unless the concentration forsuch substance is less than the lowest appropriate POL specified in "Test Methods for Evaluating Solid Wastes, Physical/Chemical Methods," EPA Publication No. SW-846, incorporated by reference at Section 620.125, for the substance, in which case the lowest appropriate POL shall be the Health Advisory Concentration~~.

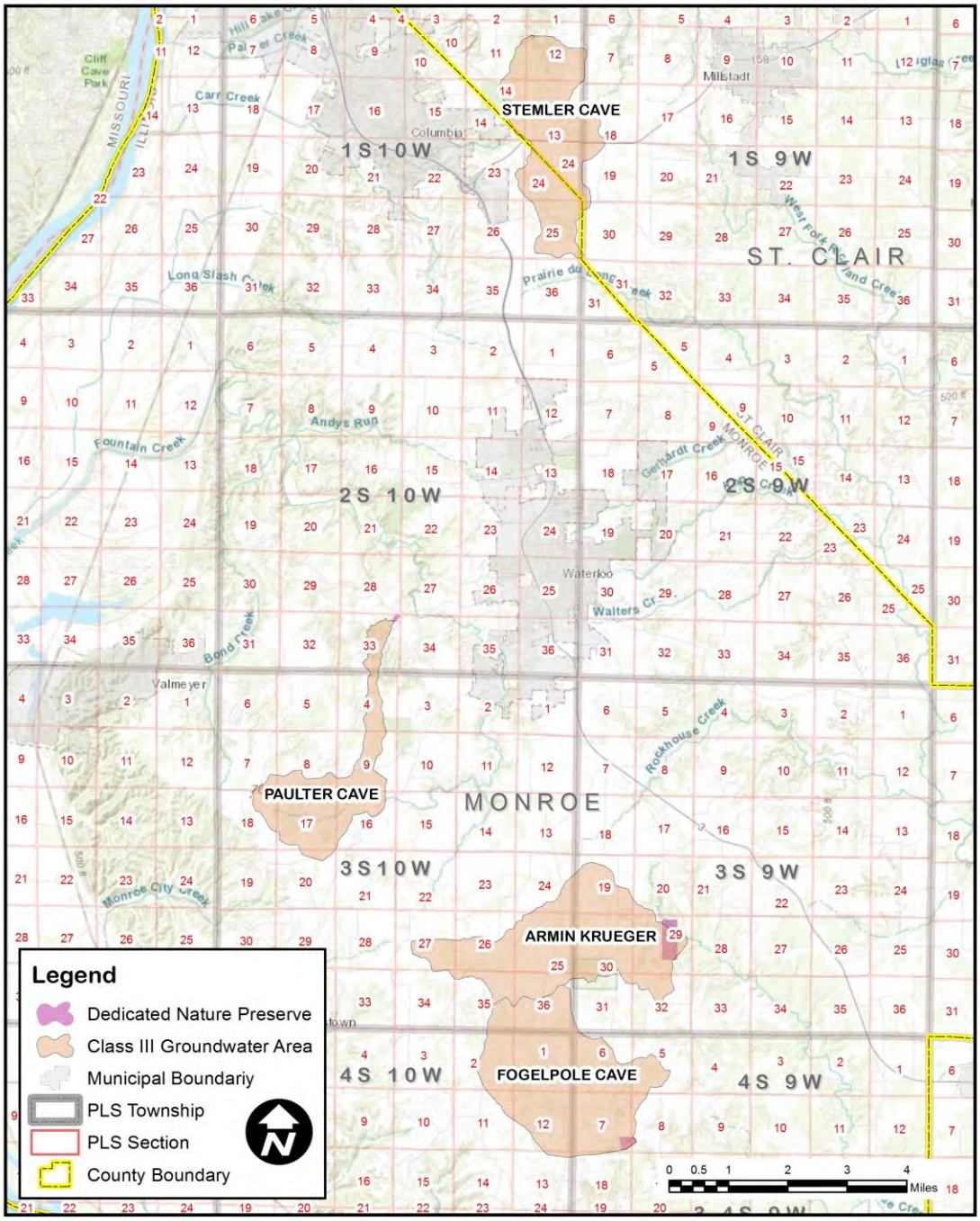
(Source: Amended at Ill. Reg. , effective)

Section 620.APPENDIX E Maps of Class III Special Resource Groundwater

SPRING GROVE FEN AND COTTON CREEK MARSH CLASS III GROUNDWATER AREAS



PAUTLER, STEMLER, ARMIN KRUEGER AND FOGELPOLE CAVES CLASS III GROUNDWATER AREAS



Attachment 3

SUBTITLE F

TITLE 35: ENVIRONMENTAL PROTECTION
SUBTITLE F: PUBLIC WATER SUPPLIES
CHAPTER I: POLLUTION CONTROL BOARD

PART 620
GROUNDWATER QUALITY

SUBPART A: GENERAL

Section

SUBPART B: GROUNDWATER CLASSIFICATION

Section

SUBPART C: NONDEGRADATION PROVISIONS
FOR APPROPRIATE GROUNDWATERS

Section

SUBPART D: GROUNDWATER QUALITY STANDARDS

Section

SUBTITLE F

SUBPART E: GROUNDWATER MONITORING AND ANALYTICAL PROCEDURES

SUBPART F: HEALTH ADVISORIES

AUTHORITY: Implementing and authorized by Section 8 of the Illinois Groundwater Protection Act [415 ILCS 55/8] and authorized by Section 27 of the Illinois Environmental Protection Act [415 ILCS 5/27].

SOURCE: Adopted in R89-14(B) at 15 Ill. Reg. 17614, effective November 25, 1991; amended in R89-14(C) at 16 Ill. Reg. 14667, effective September 11, 1992; amended in R93-27 at 18 Ill.

Reg. 14084, effective August 24, 1994; amended in R96-18 at 21 Ill. Reg. 6518, effective May 8, 1997; amended in R97-11 at 21 Ill. Reg. 7869, effective July 1, 1997; amended in R01-14 at 26 Ill. Reg. 2662, effective February 5, 2002; amended in R08-18 at 36 Ill. Reg. 15206, effective October 5, 2012; amended in R08-18(B) at 37 Ill. Reg. 16529, effective October 7, 2013; amended in _____ at ____ Ill. Reg. _____, effective _____

Section 620.110 Definitions

The definitions of the Environmental Protection Act [415 ILCS 5] and the Groundwater Protection Act [415 ILCS 55] apply to this Part. The following definitions also apply to this Part:

"Act" means the Environmental Protection Act [415 ILCS 5].

"Agency" means the Illinois Environmental Protection Agency.

"Aquifer" means saturated (with groundwater) soils and geologic materials which are sufficiently permeable to readily yield economically useful quantities of water to wells, springs, or streams under ordinary hydraulic gradients. [415 ILCS 55/3(b)]

"BETX" means the sum of the concentrations of benzene, ethylbenzene, toluene, and xylenes.

"Board" means the Illinois Pollution Control Board.

“Chemical Abstract Services Registry Number” or “CASRN” means a unique numerical identifier designated for only one substance, assigned by the Chemical Abstracts Service for that substance.

"Carcinogen" means a contaminant that is classified as a Category A1 or A2 Carcinogen by the American Conference of Governmental Industrial Hygienists; or a Category 1 or 2A/2B carcinogen by the World Health Organization's International Agency for Research on Cancer; or a "Human carcinogen" or "Anticipated Human Carcinogen" by the United States Department of Health and Human Service National Toxicological Program; or a Category A or B1/B2 Carcinogen or as “carcinogenic to humans” or “likely to become carcinogenic to humans” *by the United States Environmental Protection Agency in Integrated Risk Information System or a Final Rule issued in a Federal Register notice by the USEPA.* [415 ILCS 5/58.2]

"Community water supply" means a public supply which serves or is intended to serve at least 15 service connections used by residents or regularly serves at least 25 residents. [415 ILCS 5/3.145]

"Contaminant" means any solid, liquid, or gaseous matter, any odor, or any form of energy, from whatever source. [415 ILCS 5/3.165]

"Corrective action process" means those procedures and practices that may be imposed by a regulatory agency when a determination has been made that contamination of groundwater has taken place, and are necessary to address a potential or existing violation of the standards set forth in Subpart D.

"Cumulative impact area" means the area, including the coal mine area permitted under the Surface Coal Mining Land Conservation and Reclamation Act [225 ILCS 720] and 62 Ill. Adm. Code 1700 through 1850, within which impacts resulting from the proposed operation may interact with the impacts of all anticipated mining on surface water and groundwater systems.

"Department" means the Illinois Department of Natural Resources.

"Detection" means the identification of a contaminant in a sample at a value equal to or greater than the:

> "Method Detection Limit" or "MDL" means the minimum measured concentration of a substance that can be reported with 99% confidence that the measured concentration is distinguishable from method blank results, pursuant to 40 CFR 136, appendix B (2017), incorporated by reference at Section 620.125; or
>
> "Lower Limit of Quantitation " or "LLOQ" means the minimum concentration that can be measured or reported pursuant to "Test Methods for Evaluating Solid Wastes, Physical/Chemical Methods," incorporated by reference at Section 620.125.

"Groundwater" means underground water which occurs within the saturated zone and geologic materials where the fluid pressure in the pore space is equal to or greater than atmospheric pressure. [415 ILCS 5/3.210]

"Hydrologic balance" means the relationship between the quality and quantity of water inflow to, water outflow from, and water storage in a hydrologic unit such as a drainage basin, aquifer, soil zone, lake, or reservoir. It encompasses the dynamic relationships among precipitation, runoff, evaporation, and changes in ground and surface water storage.

“Lowest Concentration Minimum Reporting Level” or “LCMRL” means the lowest spiking concentration such that the probability of spike recovery in the 50% to 150% range is at least 99%.

"IGPA" means the Illinois Groundwater Protection Act. [415 ILCS 55]

"Lowest observable adverse effect level" or "LOAEL" means the lowest tested concentration of a chemical or substance that produces a statistically significant increase in frequency or severity of non-overt adverse effects between the exposed population and its appropriate control.

"Licensed Professional Engineer" or "LPE" means a person, corporation, or partnership licensed under the laws of the State of Illinois to practice professional engineering. [415 ILCS 5/57.2]

"Licensed Professional Geologist" or "LPG" means an individual who is licensed under the Professional Geologist Licensing Act *to engage in the practice of professional geology in Illinois.* [225 ILCS 745/15]

"Mutagen" means a carcinogenic constituent that operates by a mutagenic mode of action for carcinogenesis. Carcinogens with a mutagenic mode of action would be expected to cause irreversible changes to DNA and would exhibit greater effects in early life versus later life exposure.

"No observable adverse effect level" or "NOAEL" means the highest tested concentration of a chemical or substance that does not produce a statistically significant increase in frequency or severity of non-overt adverse effects between the exposed population and its appropriate control.

"Non-community water supply" means a public water supply that is not a community water supply. [415 ILCS 5/3.145]

"Off-site" means not on-site.

"On-site" means on the same or geographically contiguous property that may be divided by public or private right-of-way, provided the entrance and exit between properties is at a crossroads intersection and access is by crossing as opposed to going along the right-of-way. Noncontiguous properties owned by the same person but connected by a right-of-way that he controls and that the public does not have access to is also considered on-site property.

"Operator" means the person responsible for the operation of a site, facility or unit.

"Owner" means the person who owns a site, facility, or unit; part of a site, facility, or unit; or who owns the land on which the site, facility, or unit is located.

"Potable" means generally fit for human consumption in accordance with accepted water supply principles and practices. [415 ILCS 5/3.340]

"Potential primary source" means any unit at a facility or site not currently subject to a removal or remedial action which:

Is utilized for the treatment, storage, or disposal of any hazardous or special waste not generated at the site; or

Is utilized for the disposal of municipal waste not generated at the site, other than landscape waste and construction and demolition debris; or

Is utilized for the landfilling, land treating, surface impounding or piling of any hazardous or special waste that is generated on the site or at other sites owned, controlled or operated by the same person; or

Stores or accumulates at any time more than 75,000 pounds above ground, or more than 7,500 pounds below ground, of any hazardous substances. [415 ILCS 5/3.345]

"Potential route" means abandoned and improperly plugged wells of all kinds, drainage wells, all injection wells, including closed loop heat pump wells, and any excavation for the discovery, development or production of stone, sand or gravel. This term does not include closed loop heat pump wells using USP (U.S. Pharmacopeia) *food grade propylene glycol.* [415 ILCS 5/3.350]

"Potential secondary source" means any unit at a facility or a site not currently subject to a removal or remedial action, other than a potential primary source, which:

Is utilized for the landfilling, land treating, or surface impounding of waste that is generated on the site or at other sites owned, controlled or operated by the same person, other than livestock and landscape waste, and construction and demolition debris; or

Stores or accumulates at any time more than 25,000 but not more than 75,000 pounds above ground, or more than 2,500 but not more than 7,500 pounds below ground, of any hazardous substance; or

Stores or accumulates at any time more than 25,000 gallons above ground, or more than 500 gallons below ground, of petroleum, including

crude oil or any fraction thereof which is not otherwise specifically listed or designated as a hazardous substance; or

Stores or accumulates pesticides, fertilizers, or road oils for purposes of commercial application or for distribution to retail sales outlets; or

Stores or accumulates at any time more than 50,000 pounds of any de-icing agent; or

Is utilized for handling livestock waste or for treating domestic wastewaters other than private sewage disposal systems as defined in the Private Sewage Disposal Licensing Act [225 ILCS 225]. [415 ILCS 5/3.355]

"Previously mined area" means land disturbed or affected by coal mining operations prior to February 1, 1983.

BOARD NOTE: February 1, 1983, is the effective date of the Illinois permanent program regulations implementing the Surface Coal Mining Land Conservation and Reclamation Act [225 ILCS 720] as codified in 62 Ill. Adm. Code 1700 through 1850.

"Property class" means the class assigned by a tax assessor to real property for purposes of real estate taxes.

BOARD NOTE: The property class (rural property, residential vacant land, residential with dwelling, commercial residence, commercial business, commercial office, or industrial) is identified on the property record card maintained by the tax assessor in accordance with the Illinois Real Property Appraisal Manual (February 1987), published by the Illinois Department of Revenue, Property Tax Administration Bureau.

"Public water supply" means all mains, pipes and structures through which water is obtained and distributed to the public, including wells and well structures, intakes and cribs, pumping stations, treatment plants, reservoirs, storage tanks and appurtenances, collectively or severally, actually used or intended for use for the purpose of furnishing water for drinking or general domestic use and which serve at least 15 service connections or which regularly serve at least 25 persons at least 60 days per year. A public water supply is either a "community water supply" or a "non-community water supply". [415 ILCS 5/3.365]

"Regulated entity" means a facility or unit regulated for groundwater protection by any State or federal agency.

"Regulatory agency" means the Illinois Environmental Protection Agency, Department of Public Health, Department of Agriculture, the Office of Mines and Minerals in the Department of Natural Resources, and the Office of State Fire Marshal.

"Regulated recharge area" means a compact geographic area, as determined by the Board pursuant to Section 17.4 of the Act, *the geology of which renders a potable resource groundwater particularly susceptible to contamination.* [415 ILCS 5/3.390]

"Resource groundwater" means groundwater that is presently being, or in the future is capable of being, put to beneficial use by reason of being of suitable quality. [415 ILCS 5/3.430]

"Saturated zone" means a subsurface zone in which all the interstices or voids are filled with water under pressure greater than that of the atmosphere.

"Setback zone" means a geographic area, designated pursuant to this Act, containing a potable water supply well or a potential source or potential route having a continuous boundary, and within which certain prohibitions or regulations are applicable in order to protect groundwaters. [415 ILCS 5/3.450]

"Site" means any location, place, tract of land and facilities, including but not limited to, buildings and improvements used for the purposes subject to regulation or control by the Act or regulations thereunder. [415 ILCS 5/3.460]

"Spring" means a natural surface discharge of an aquifer from rock or soil.

"Threshold dose" means the lowest dose of a chemical at which a specified measurable effect is observed and below which it is not observed.

"Treatment" means the technology, treatment techniques, or other procedures for compliance with 35 Ill. Adm. Code, Subtitle F.

"Unit" means any device, mechanism, equipment, or area (exclusive of land utilized only for agricultural production). [415 ILCS 5/3.515]

"U.S. EPA" means the United States Environmental Protection Agency.

"Wellhead protection area" or "WHPA" means the surface and subsurface recharge area surrounding a community water supply well or well field, delineated outside of any applicable setback zones (pursuant to Section 17.1 of the Act [415 ILCS 5/17.1]), and pursuant to Illinois' Wellhead Protection Program, through which contaminants are reasonably likely to move toward such well or well field.

"Wellhead Protection Program" or "WHPP" means the wellhead protection program for the State of Illinois, approved by U.S. EPA under 42 USC 300h-7. BOARD NOTE: Derived from 40 CFR 141.71(b) (2003). The wellhead protection program includes the "groundwater protection needs assessment" under Section 17.1 of the Act [415 ILCS 5/17.1] and 35 Ill. Adm. Code 615-617.

(Source: Amended at Ill. Reg. , effective)

Section 620.125 Incorporations by Reference

a) The Board incorporates the following material by reference:

ASTM International. 100 Barr Harbor Drive, PO Box C700, West Conshohocken, PA 19428-2959 (610) 832-9500.

"Standard Practice for Classification of Soils for Engineering Purposes (Unified Classification System)" ASTM D2487-06.

CFR (Code of Federal Regulations). Available from the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402 (202) 783-3238.

Method Detection Limit Definition, appendix B to Part 136, 40 CFR 136, appendix B – Revision 2 (82 FR 40939, Aug. 28, 2017).

Control of Lead and Copper, general requirements, 40 CFR 141.80 (72 FR 57814, Oct. 10, 2007).

Maximum contaminant levels for organic contaminants, 40 CFR 141.61 (59 FR 34324, July 1, 1994).

Maximum contaminant levels for inorganic contaminants, 40 CFR 141.62 (69 FR 38855, June 29, 2004).

Maximum contaminant levels for radionuclides, 40 CFR 141.66 (65 FR 76748, Dec. 7, 2000) .

GPO. Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20401 (202) 783-3238.

U.S. EPA Guidelines for Carcinogenic Risk Assessment, 51 Fed. Reg. 33992-34003 (September 24, 1986).

Illinois Environmental Protection Agency, 1020 North Grand Avenue East, P.O. Box 19276, Springfield, IL 62794-9276 (217) 785-4787.

"Guidance Document for Groundwater Protection Needs Assessments," Agency, Illinois State Water Survey, and Illinois State Geologic Survey Joint Report, January 1995.

"Illinois Integrated Water Quality Report and Section 303(d) List," Agency.

"The Illinois Wellhead Protection Program Pursuant to Section 1428 of the Federal Safe Drinking Water Act," Agency, # 22480, October 1992.

NAS. National Academy of Sciences, Engineering, and Medicine, 550 5th St. NW, Washington DC (202) 334-2000.

"Water Quality Criteria", EPA.R3.73-033, (1973).

NCRP. National Council on Radiation Protection, 7910 Woodmont Ave., Bethesda, MD (301) 657-2652.

"Maximum Permissible Body Burdens and Maximum Permissible Concentrations of Radionuclides in Air and in Water for Occupational Exposure", NCRP Report Number 22, June 5, 1959.

U. S. Environmental Protection Agency, 1200 Pennsylvania Avenue, N.W., Washington, DC 20460 ~~NTIS. National Technical Information Service, 5285 Port Royal Road, Springfield, VA 22161 (703) 605-6000.~~

"Methods for Chemical Analysis of Water and Wastes," March 1983, Doc. No. PB84-128677. EPA 600/4-79-020 (available online at http://nepis.epa.gov/).

"Methods for the Determination of Inorganic Substances in Environmental Samples," August 1993, PB94-120821 (referred to as "U.S. EPA Environmental Inorganic Methods"). EPA 600/R-93-100 (available online at http://nepis.epa.gov/).

"Methods for the Determination of Metals in Environmental Samples," June 1991, Doc. No. PB91-231498. EPA 600/4-91-010 (available online at http://nepis.epa.gov/).

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"Prescribed Procedures for Measurement of Radioactivity in Drinking Water," Doc. No. PB80-224744. EPA 600/4-80-032, (August 1980) (available online at http://nepis.epa.gov/).

"Procedures for Radiochemical Analysis of Nuclear Reactor Aqueous Solutions," H.L. Krieger and S. Gold, Doc. No. PB222-154/7BA. EPA-R4-73-014, May 1973.

"Radiochemical Analytical Procedures for Analysis of Environmental Samples," March 1979, Doc. No. EMSL LV 053917.

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"Validated Test Method 8327: Per-and Polyfluoroalkyl Substances (PFAS) Using External Standard Calibration and Multiple Reaction Monitoring (MRM) Liquid Chromatography/Tandem Mass Spectrometry (LC/MS/MS)" Revision 0, June 2019.

United States Environmental Protection Agency, Office of Resource Conservation and Recovery.

"Statistical Analysis of Groundwater Monitoring Data at RCRA Facilities, (March 2009 Unified Guidance)", EPA 530/R-09-007.

USGS. United States Geological Survey, 1961 Stout St., Denver, CO 80294 (303) 844-4169

"Techniques of Water Resources Investigations of the United States Geological Survey, Guidelines for Collection and Field Analysis of Ground-Water Samples for Selected Unstable Constituents", Book I, Chapter D2 (1976).

b) This Section incorporates no later editions or amendments.

(Source: Amended at Ill. Reg. , effective)

Section 620.210 Class I: Potable Resource Groundwater

Except as provided in Sections 620.230, 620.240, or 620.250, Potable Resource Groundwater is:

a) Groundwater located 10 feet or more below the land surface and within:

1) The minimum setback zone of a well which serves as a potable water supply and to the bottom of such well;

2) Unconsolidated sand, gravel, or sand and gravel which is 5 feet or more in thickness and that contains 12% or less of fines (i.e., fines which pass through a No. 200 sieve tested according to ASTM Standard Practice D2487-06, incorporated by reference at Section 620.125);

3) Sandstone which is 10 feet or more in thickness, or fractured carbonate which is 15 feet or more in thickness;

4) Any geologic material which is capable of a:

A) Sustained groundwater yield, from up to a 12-inch borehole, of 150 gallons per day or more from a thickness of 15 feet or less; or

B) Hydraulic conductivity of 1×10^{-4} cm/sec or greater using one of the following test methods or its equivalent:

i) Slug test; or

ii) Pump test

5) The wellhead protection area of a community water supply well or well field, as defined in Section 620.110 and delineated pursuant to the methods incorporated by reference in Section 620.125. For the purposes of this Subpart, when a maximum setback zone has been adopted pursuant to Section 14.3 of the Act, the WHPA includes the delineated area within the maximum setback zone.

b) Any groundwater which is determined by the Board pursuant to petition procedures set forth in Section 620.260, to be capable of potable use.

BOARD NOTE: Any portion of the thickness associated with the geologic materials as described in subsections 620.210(a)(2), (a)(3), or (a)(4) should be designated as Class I: Potable Resource Groundwater if located 10 feet or more

below the land surface.

(Source: Amended at, effective)

Section 620.250 Groundwater Management Zone

a) Within any class of groundwater, a groundwater management zone may be established as a three-dimensional region containing groundwater being managed to mitigate impairment caused by the release of contaminants from a site:

1) That is subject to a corrective action process approved by the Agency; or

2) For which the owner or operator undertakes an adequate corrective action in a timely and appropriate manner and provides a written confirmation to the Agency. Such confirmation shall be provided in a form as prescribed by the Agency.

b) A groundwater management zone is established upon concurrence by the Agency that the conditions as specified in subsection (a) are met and groundwater management continues for a period of time consistent with the action described in that subsection.

c) A groundwater management zone expires upon the Agency's receipt of appropriate documentation which confirms the completion of the action taken pursuant to subsection (a) and which confirms the attainment of applicable standards as set forth in Subpart D. The Agency shall review the on-going adequacy of controls and continued management at the site if concentrations of chemical constituents, as specified in Section 620.450(a)(4)(B), remain in groundwater at the site following completion of such action. The review shall take place no less often than every 5 years and the results shall be presented to the Agency in a written report.

d) Notwithstanding subsections (a) and (b) above, a groundwater management zone as defined in 35 Ill. Adm. Code 740.120 may be established in accordance with the requirements of 35 Ill. Adm. Code 740.530 for sites undergoing remediation pursuant to the Site Remediation Program. Such a groundwater management zone shall remain in effect until the requirements set forth at 35 Ill. Adm. Code 740.530(c) are met.

e) While the groundwater management zone established in accordance with 35 Ill. Adm. Code 740.530 is in effect, the otherwise applicable standards as specified in Subpart D of this Part shall not be applicable to the "contaminants of concern", as defined at 35 Ill. Adm. Code 740.120, for which groundwater remediation objectives have been approved in accordance with the procedures of 35 Ill. Adm. Code 740.

f) Notwithstanding subsection (c) above, the review requirements concerning the ongoing adequacy of controls and continued management at the site shall not apply to groundwater within a three-dimensional region formerly encompassed by a groundwater management zone established in accordance with 35 Ill. Adm. Code 740.530 while a No Further Remediation Letter issued in accordance with the procedures of 35 Ill. Adm. Code 740 is in effect.

g) All groundwater management zone applications submitted pursuant to subsection (a) shall contain the following:

1) Facility information. This includes the name, address, and county where the site is located.

2) Identification of specific units (operating or closed) present at the facility.

3) Maps and engineering drawings showing the facility and units at the facility.

4) Statement of the groundwater classification(s) at the facility.

5) Identification of the chemical constituents released to the groundwater.

6) Description of how groundwater will be monitored to determine the rate and extent of the release, and if it has migrated off site.

7) Schedule for investigation of the extent of the release.

8) Results of available soil testing and groundwater monitoring associated with a release, locations and depths of samples, and monitoring well construction details with well logs.

9) Remedy

A) Description of selected remedy and why it was chosen;

B) Results of groundwater contaminant transport modeling or calculations showing how the selected remedy will achieve compliance with the applicable groundwater standards;

C) Description of the fate and transport of contaminants with selected remedy over time; and

D) A statement of how groundwater at the facility will be monitored following implementation of the remedy to ensure that the groundwater standards have been attained.

10) Information requested by the Agency, necessary for its review of the groundwater management zone application.

(Source: Amended at __ Ill. Reg. _____, effective _____)

(Source: Amended at, effective)

Section 620.302 Applicability of Preventive Notification and Preventive Response Activities

a) Preventive notification and preventive response as specified in Sections 620.305 through 620.310 applies to:

1) Class I groundwater under Section 620.210(a)(1), (a)(2), or (a)(3) that is monitored by the persons listed in subsection (b); or

2) Class III groundwater that is monitored by the persons listed in subsection (b).

b) For purposes of subsection (a), the persons that conduct groundwater monitoring are:

1) An owner or operator of a regulated entity for which groundwater quality monitoring shall be performed pursuant to State or Federal law or regulation (e.g., section 106 and 107 of the Comprehensive Environmental Response, Compensation and Liability Act (42 USC 9601, et seq.); sections 3004 and 3008 of the Resource Conservation and Recovery Act (42 USC 6901, et seq.); sections 4(q), 4(v), 12(g), 21(d), 21(f), 22.2(f), 22.2(m) and 22.18 of the Act; 35 Ill. Adm. Code 615, 616, 724, 725, 730, 731, 750, 807, 811, 814, and 815; and 62 Ill. Adm. Code 1780);

2) An owner or operator of a public water supply well who conducts groundwater quality monitoring;

3) A State agency that is authorized to conduct, or is the recipient of, groundwater quality monitoring data (e.g., Illinois Environmental Protection Agency, Department of Public Health, Department of Agriculture, Office of State Fire Marshal, or Department of Natural Resources); or

4) An owner or operator of a facility that conducts groundwater quality monitoring pursuant to State or federal judicial or administrative order.

c) If a contaminant exceeds a standard set forth in Section 620.410 or Section 620.430, the appropriate remedy is corrective action and Sections 620.305 and 620.310 do not apply.

(Source: Amended at, effective)

Section 620.310 Preventive Response Activities

a) The following preventive assessment shall be undertaken:

1) If a preventive notification under Section 620.305(c) is provided by a community water supply:

A) The Agency shall notify the owner or operator of any identified potential primary source, potential secondary source, potential route, or community water supply well that is located within 2,500 feet of the wellhead.

B) The owner or operator notified under subsection (a)(1)(A) shall, within 30 days after the date of issuance of such notice, sample each water well or monitoring well for the contaminant identified in the notice if the contaminant or material containing such contaminant is or has been stored, disposed of, or otherwise handled at the site. If a contaminant identified under Section 620.305(a) is detected, then the well shall be resampled within 30 days of the date on which the first sample analyses are received. If a contaminant identified under Section 620.305(a) is detected by the resampling, preventive notification shall be given as set forth in Section 620.305.

C) If the Agency receives analytical results under subsection (a)(1)(B) that show a contaminant identified under Section 620.305(a) has been detected, the Agency shall:

i) Conduct a well site survey pursuant to 415 ILCS 5/17.1(d), if such a survey has not been previously conducted within the last 5 years; and

ii) Identify those sites or activities that represent a hazard to the continued availability of groundwaters for public use unless a groundwater protection needs assessment has been prepared pursuant to 415 ILCS 5/17.1(d).

2) If a preventive notification is provided under Section 620.305(c) by a non-community water supply or for multiple private water supply wells, the Department of Public Health shall conduct a sanitary survey within 1,000 feet of the wellhead of a non-community water supply or within 500 feet of the wellheads for multiple private water supply wells.

3) If a preventive notification under Section 620.305(b) is provided by the owner or operator of a regulated entity and the applicable standard in Subpart D has not been exceeded:

A) The appropriate regulatory agency shall determine if any of the following occurs for Class I: Potable Resource Groundwater:

i) The levels set forth below are exceeded or are changed for pH:

CASRN	**Constituent**	**Criteria (mg/L)**
95-50-1	*o*rtho-Dichlorobenzene (1,2-dichlorobenzene)	0.01
1634-04-4	MTBE (methyl tertiary-butyl ether)	0.02
108-95-2	Phenol	0.001
100-42-5	Styrene	0.01
108-88-3	Toluene	0.04
1330-20-7	Xylenes	0.02

ii) A statistically significant increase occurs above background(as determined pursuant to other regulatory procedures (e.g., 35 Ill. Adm. Code 616, 724, 725, or 811)) for the following inorganic constituents (except due to natural causes) or for the following organic constituents:

CASRN	**Constituent**
Inorganics	
7429-90-5	Aluminum
7440-36-0	Antimony
7440-41-7	Beryllium
7440-43-9	Cadmium
7440-47-3	Chromium (total)
143-33-9	Cyanide (sodium cyanide)
7439-92-1	Lead
7487-94-7	Mercury (mercuric chloride)
7439-98-7	Molybdenum
7440-28-0	Thallium
7440-62-2	Vanadium

Organics	
83-32-9	Acenaphthene
67-64-1	Acetone
116-06-3	Aldicarb
120-12-7	Anthracene
1912-24-9	Atrazine
65-85-0	Benzoic acid
78-93-3	2-Butanone (methyl ethyl ketone)
1563-66-2	Carbofuran
75-15-0	Carbon disulfide
108-90-7	Chlorobenzene
94-75-7	2,4-D (2,4-dichlorophenoxy acetic acid)
75-99-0	Dalapon
1918-00-9	Dicamba
75-71-8	Dichlorodifluoromethane
75-34-3	1,1-Dichloroethane
75-35-4	1,1-Dichloroethylene
156-59-2	*cis*-1,2-Dichloroethylene
156-60-5	*trans*-1,2-Dichloroethylene
84-66-2	Diethyl phthalate
84-74-2	Di-*n*-butyl phthalate
88-85-7	Dinoseb
145-73-3	Endothall
72-20-8	Endrin
206-44-0	Fluoranthene
86-73-7	Fluorene
2691-41-0	HMX (octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine)
77-47-4	Hexachlorocyclopentadiene
72-43-5	Methoxychlor
90-12-0	1-Methylnaphthalene
91-57-6	2-Methylnaphthalene
95-48-7	2-Methylphenol (*o*-cresol)
91-20-3	Naphthalene
98-95-3	Nitrobenzene
375-73-5	PFBS (perfluorobutanesulfonic acid)
355-46-4	PFHxS (perfluorohexanesulfonic acid)
375-95-1	PFNA (perfluorononanoic acid)
1763-23-1	PFOS (perfluorooctanesulfonic acid)
1918-02-1	Picloram

129-00-0	Pyrene
121-82-4	RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine)
122-34-9	Simazine
118-96-7	TNT (2,4,6-trinitrotoluene)
93-72-1	2,4,5-TP (silvex)
120-82-1	1,2,4-Trichlorobenzene
71-55-6	1,1,1-Trichloroethane
79-00-5	1,1,2-Trichloroethane
75-69-4	Trichlorofluoromethane
99-35-4	1,3,5-Trinitrobenzene

iii) For a chemical constituent of gasoline, diesel fuel, or heating fuel, the constituent exceeds the following:

Constituent	**Criterion (mg/L)**
BETX	0.095

iv) For pH, a statistically significant change occurs from background.

BOARD NOTE: Constituents that are carcinogens have not been listed in subsection (a)(3)(A) because the standard is set at the MCL, LLOQ or LCMRL, and any exceedence thereof is a violation subject to corrective action.

B) The appropriate agency shall determine if, for Class III: Special Resource Groundwater, the levels as determined by the Board are exceeded.

C) The appropriate regulatory agency shall consider whether the owner or operator reasonably demonstrates that:

i) The contamination is a result of contaminants remaining in groundwater from a prior release for which appropriate action was taken in accordance with laws and regulations in existence at the time of the release;

ii) The source of contamination is not due to the on-site

release of contaminants; or

iii) The detection resulted from error in sampling, analysis, or evaluation.

D) The appropriate regulatory agency shall consider actions necessary to minimize the degree and extent of contamination.

b) The appropriate regulatory agency shall determine whether a preventive response shall be undertaken based on relevant factors including, but not limited to, the considerations in subsection (a)(3).

c) After completion of preventive response pursuant to authority of an appropriate regulatory agency, the concentration of a contaminant listed in subsection (a)(3)(A) in groundwater may exceed 50% of the applicable numerical standard in Subpart D only if the following conditions are met:

1) The exceedence has been minimized to the extent practicable;

2) Beneficial use, as appropriate for the class of groundwater, has been assured; and

3) Any threat to public health or the environment has been minimized.

d) Nothing in this Section shall in any way limit the authority of the State or of the United States to require or perform any corrective action process.

(Source: Amended at, effective)

Section 620.410 Groundwater Quality Standards for Class I: Potable Resource Groundwater

a) Inorganic Chemical Constituents
Except due to natural causes or as provided in Section 620.450, concentrations of the following chemical constituents shall not be exceeded in Class I groundwater:

CASRN	Constituent	Standard[a]
7429-90-5	Aluminum	1.9[b]
7440-36-0	Antimony	0.006[c]
7440-38-2	Arsenic[d]	0.01[c]
7440-39-3	Barium	2[c]
7440-41-7	Beryllium	0.004[c]
7440-42-8	Boron	0.77[b]
7440-43-9	Cadmium	0.005[c]
16887-00-6	Chloride	200[e]
7440-47-3	Chromium (total)	0.1[c]
7440-48-4	Cobalt	0.0012[b]
7440-50-8	Copper	0.5[f]
143-33-9	Cyanide (sodium cyanide)	0.2[c]
7681-49-4	Fluoride (sodium fluoride)	2[f]
7439-89-6	Iron	5[e]
7439-92-1	Lead	0.0075[g]
7439-93-2	Lithium	0.01[h]
7439-96-5	Manganese	0.15[i]
7487-94-7	Mercury (mercuric chloride)	0.002[c]
7439-98-7	Molybdenum	0.019[b]
7440-02-0	Nickel	0.077[b]
14797-55-8	Nitrate as N	10[c]
14797-73-0	Perchlorate	0.0027[b]
7440-14-4	Radium (combined 226+228)	5[c]
7782-49-2	Selenium	0.02[j]
7440-22-4	Silver	0.019[b]
14808-79-8	Sulfate	400[e]
	TDS (total dissolved solids)	1,200[e]
7440-28-0	Thallium	0.002[c]
7440-62-2	Vanadium	0.00027[b]
7440-66-6	Zinc	1.2[b]

Constituent Name and Groundwater Quality Standard Notations

[a] The standard units are milligrams per liter ("mg/L"), except for the radium (combined 226+228) unit of picocuries per liter ("pCi/L").
[b] The standard is calculated using the Human Threshold Toxicant Advisory Concentration ("HTTAC") procedures at Appendix A.
[c] The standard is based on the Maximum Contaminant Level ("MCL"), promulgated by U.S. EPA, Office of Water, and Illinois EPA Primary Drinking Water Standards at 35 Ill. Adm. Code 611.
[d] The constituent meets the definition of a "carcinogen" at Section 620.110.
[e] The standard is the 95% confidence concentration stated in Illinois EPA's "*Integrated Water Quality Report and Section 303(d) List"*, incorporated by reference at Section 620.125.
[f] The standard is based on beneficial use for watering livestock, per "*Water Quality Criteria*", by National Academy of Sciences, incorporated by reference at Section 620.125.
[g] The standard is 50% of the U.S. EPA "action level" of 0.015 mg/L for lead. The U.S. EPA action level applies at the service connection. The standard is reduced by 50% as a safety margin, based on the assumption that 50% of water would be treated.
[h] The standard is the "LLOQ" or "LCMRL" as defined in Section 620.110.
[i] The standard is promulgated at 35 Ill. Adm. Code 611.300.
[j] The standard is based on beneficial use for irrigation of crops, per "*Water Quality Criteria*", by National Academy of Sciences, incorporated by reference at Section 620.125.

b) Organic Chemical Constituents

Except due to natural causes or as provided in Section 620.450 or subsection (d), concentrations of the following organic chemical constituents shall not be exceeded in Class I groundwater:

CASRN	Constituent	Standard (mg/L)
83-32-9	Acenaphthene	0.23[a]
67-64-1	Acetone	3.5[a]
15972-60-8	Alachlor[b]	0.002[c]
116-06-3	Aldicarb	0.003[c]
120-12-7	Anthracene	1.2[a]
319-84-6	*alpha*-BHC (*alpha*-benzene hexachloride)[b]	0.000012[d]
71-43-2	Benzene[b]	0.005[c]
56-55-3	Benzo(a)anthracene[e]	0.00025[d]

CASRN	Constituent	Standard (mg/L)
205-99-2	Benzo(b)fluoranthene[e]	0.00025[d]
207-08-9	Benzo(k)fluoranthene[e]	0.0025[d]
50-32-8	Benzo(a)pyrene[e]	0.0002[c]
65-85-0	Benzoic acid	15[a]
78-93-3	2-Butanone (methyl ethyl ketone)	2.3[a]
1563-66-2	Carbofuran	0.04[c]
75-15-0	Carbon disulfide	0.38[a]
56-23-5	Carbon tetrachloride[b]	0.005[c]
12798-03-6	Chlordane[b]	0.002[c]
108-90-7	Chlorobenzene	0.1[c]
67-66-3	Chloroform[b]	0.07[f]
218-01-9	Chrysene[e]	0.025[d]
94-75-7	2,4-D (2,4-dichlorophenoxy acetic acid)	0.07[c]
75-99-0	Dalapon	0.2[c]
53-70-3	Dibenzo(a,h)anthracene[e]	0.000025[d]
96-12-8	1,2-Dibromo-3-chloropropane (dibromochloropropane)[e]	0.0002[c]
1918-00-9	Dicamba	0.12[a]
95-50-1	*o*-Dichlorobenzene (1,2-dichlorobenzene)	0.6[c]
106-46-7	*p*-Dichlorobenzene (1,4-dichlorobenzene)[b]	0.075[c]
75-71-8	Dichlorodifluoromethane	0.77[a]
75-34-3	1,1-Dichloroethane	0.77[a]
107-06-2	1,2-Dichloroethane[b]	0.005[c]
75-35-4	1,1-Dichloroethylene	0.007[c]
156-59-2	*cis*-1,2-Dichloroethylene	0.07[c]
156-60-5	*trans*-1,2-Dichloroethylene	0.1[c]
75-09-2	Dichloromethane (methylene chloride)[e]	0.005[c]
78-87-5	1,2-Dichloropropane[b]	0.005[c]
117-81-7	Di(2-ethylhexyl)phthalate[b]	0.006[c]
84-66-2	Diethyl phthalate	3.1[a]
84-74-2	Di-*n*-butyl phthalate	0.38[a]
99-65-0	1,3-Dinitrobenzene	0.001[g]
121-14-2	2,4-Dinitrotoluene[b]	0.001[g]
606-20-0	2,6-Dinitrotoluene[b]	0.001[g]
88-85-7	Dinoseb	0.007[c]
123-91-1	1,4-Dioxane (*p*-dioxane)[b]	0.00078[d]
145-73-3	Endothall	0.1[c]

CASRN	Constituent	Standard (mg/L)
72-20-8	Endrin	0.002[c]
100-41-4	Ethylbenzene[b]	0.7[c]
106-93-4	Ethylene dibromide (1,2-dibromoethane)[b]	0.00005[c]
206-44-0	Fluoranthene	0.15[a]
86-73-7	Fluorene	0.15[a]
58-89-9	*gamma*-HCH (*gamma*-hexachlorocyclohexane, lindane)[b]	0.0002[c]
2691-41-0	HMX (octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine)	0.77[a]
76-44-8	Heptachlor[b]	0.0004[c]
1024-57-3	Heptachlor epoxide[b]	0.0002[c]
77-47-4	Hexachlorocyclopentadiene	0.05[c]
193-39-5	Indeno(1,2,3-c,d)pyrene[e]	0.00025[d]
98-82-8	Isopropylbenzene (cumene)[b]	0.38[a]
93-65-2	MCPP (mecoprop)	0.1[g]
1634-04-4	MTBE (methyl tertiary-butyl ether)	0.038[a]
72-43-5	Methoxychlor	0.04[c]
90-12-0	1-Methylnaphthalene	0.27[a]
91-57-6	2-Methylnaphthalene	0.015[a]
95-48-7	2-Methylphenol (*o*-cresol)	0.19[a]
91-20-3	Naphthalene	0.077[a]
98-95-3	Nitrobenzene	0.0077[a]
1336-36-3	PCBs (polychlorinated biphenyls as decachloro-biphenyl)[b]	0.0005[c]
375-73-5	PFBS (perfluorobutanesulfonic acid)	0.0012[a]
355-46-4	PFHxS (perfluorohexanesulfonic acid)	0.000077[a]
375-95-1	PFNA (perfluorononanoic acid)	0.000012[a]
335-67-1	PFOA (perfluorooctanoic acid)[b]	0.000002[g]
1763-23-1	PFOS (perfluorooctanesulfonic acid)	0.0000077[a]
87-86-5	Pentachlorophenol[b]	0.001[c]
108-95-2	Phenol	1.2[a]
1918-02-1	Picloram	0.5[c]
129-00-0	Pyrene	0.12[a]
121-82-4	RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine)	0.062[a]
122-34-9	Simazine	0.004[c]
100-42-5	Styrene	0.1[c]
118-96-7	TNT (2,4,6-trinitrotoluene)	0.0077[a]

CASRN	Constituent	Standard (mg/L)
93-72-1	2,4,5-TP (silvex)	0.05[c]
127-18-4	Tetrachloroethylene[b]	0.005[c]
108-88-3	Toluene	1[c]
8001-35-2	Toxaphene[b]	0.003[c]
120-82-1	1,2,4-Trichlorobenzene	0.07[c]
71-55-6	1,1,1-Trichloroethane	0.2[c]
79-00-5	1,1,2-Trichloroethane	0.005[c]
79-01-6	Trichloroethylene[e]	0.005[c]
75-69-4	Trichlorofluoromethane	1.2[a]
99-35-4	1,3,5-Trinitrobenzene	0.46[a]
75-01-4	Vinyl chloride[e]	0.002[c]
1330-20-7	Xylenes	10[c]

Constituent Name and Groundwater Quality Standard Notations

[a] The standard is the Human Threshold Toxicant Advisory Concentration ("HTTAC"), calculated using procedures at Appendix A.
[b] The constituent meets the definition of a "carcinogen" at Section 620.110.
[c] The standard is based on the Maximum Contaminant Level ("MCL"), promulgated by U.S. EPA, Office of Water, and Illinois EPA Primary Drinking Water Standards at 35 Ill. Adm. Code 611.
[d] The standard is the Human Nonthreshold Toxicant Advisory Concentration ("HNTAC"), calculated using procedures at Appendix A.
[e] The constituent meets the definition of a "mutagen" at Section 620.110.
[f] The standard is based on the Maximum Contaminant Level Goal ("MCLG"), promulgated by U.S. EPA, Office of Water.
[g] The standard is the "LLOQ" or "LCMRL" as defined in Section 620.110.

c)

Complex Organic Chemical Mixtures

1) Concentrations of the following chemical constituents shall not be exceeded in Class I groundwater:

CASRN	Constituent	Standard (mg/L)
71-43-2	Benzene[a]	0.005[b]
	Total BETX	11.705[c]

Constituent Name and Groundwater Quality Standard Notations

[a] The constituent meets the definition of a "carcinogen" at Section 620.110.
[b] The standard is based on the Maximum Contaminant Level ("MCL"), promulgated by U.S. EPA, Office of Water, and Illinois EPA Primary Drinking Water Standards at 35 Ill. Adm. Code 611.
[c] The standard is the total combined standard of benzene, ethylbenzene, toluene, and xylenes.

2) Atrazine and Metabolites

The total concentration of Atrazine plus Atrazine metabolites shall be compared to the Atrazine Class I groundwater standard of 0.003 mg/L.

CASRN	Constituent	Standard (mg/L)
1912-24-9	Atrazine	0.003[a]
	Total Atrazine and Metabolites DEA (desethyl-atrazine) DIA (desisopropyl-atrazine) DACT (diaminochlorotriazine)	0.003

Groundwater Quality Standard Notation

[a] The standard is based on the Maximum Contaminant Level ("MCL"), promulgated by U.S. EPA, Office of Water, and Illinois EPA Primary Drinking Water Standards at 35 Ill. Adm. Code 611.

d) pH

Except due to natural causes, a pH range of 6.5 - 9.0 units shall not be exceeded in

Class I groundwater.

e) Beta Particle and Photon Radioactivity

1) Except due to natural causes, the average annual concentration of beta particle and photon radioactivity from man-made radionuclides shall not exceed a dose equivalent to the total body organ greater than 4 mrem/year in Class I groundwater. If two or more radionuclides are present, the sum of their dose equivalent to the total body, or to any internal organ shall not exceed 4 mrem/year in Class I groundwater except due to natural causes.

2) Except for the radionuclides listed in subsection (f)(3), the concentration of man-made radionuclides causing 4 mrem total body or organ dose equivalent shall be calculated on the basis of a 2 liter per day drinking water intake using the 168-hour data in accordance with the procedure set forth in NCRP Report Number 22, incorporated by reference at Section 620.125(a).

3) Except due to natural causes, the average annual concentration assumed to produce a total body or organ dose of 4 mrem/year of the following chemical constituents shall not be exceeded in Class I groundwater:

CASRN	**Constituent**	**Critical Organ**	**Standard (pCi/L)**
10028-17-8	Tritium	Total Body	20,000
10098-97-2	Strontium-90	Bone Marrow	8.0

(Source: Amended at, effective)

Section 620.420 Groundwater Quality Standards for Class II: General Resource Groundwater

a) Inorganic Chemical Constituents

1) Except due to natural causes or as provided in Section 620.450 or subsection (a)(3) or (e) of this Section, concentrations of the following chemical constituents shall not be exceeded in Class II groundwater:

CASRN	Constituent	Standard (mg/L)
7440-36-0	Antimony	0.006[a]
7440-38-2	Arsenic[b]	0.2[c]
7440-39-3	Barium	2.0[a]
7440-41-7	Beryllium	0.5[d]
7440-43-9	Cadmium	0.05[d]
7440-47-3	Chromium (total)	1.0[c]
7440-48-4	Cobalt	1[c]

CASRN	Constituent	Standard (mg/L)
143-33-9	Cyanide (sodium cyanide)	0.6[c]
7681-49-4	Fluoride (sodium fluoride)	2[c]
7439-92-1	Lead	0.1[c]
7439-93-2	Lithium	2.5[e]
7487-94-7	Mercury (mercuric chloride)	0.01[c]
7439-98-7	Molybdenum	0.05[e]
14797-55-8	Nitrate as N	100[c]
14797-73-0	Perchlorate	0.0027[a]
7440-28-0	Thallium	0.002[a]
7440-62-2	Vanadium	0.1[c]

Constituent Name and Groundwater Quality Standard Notations

[a] The Class II standard is equal to the Class I groundwater quality standard.
[b] The constituent meets the definition of a "carcinogen" at Section 620.110.
[c] The standard is based on beneficial use for watering livestock, per "*Water Quality Criteria*", by National Academy of Sciences, incorporated by reference at Section 620.125.
[d] The standard is based on beneficial use for watering livestock and irrigation of crops, per "*Water Quality Criteria*", by National Academy of Sciences, incorporated by reference at Section 620.125.
[e] The standard is based on beneficial use for irrigation of crops, per "*Water Quality Criteria,*", by National Academy of Sciences, incorporated by reference at Section 620.125.

2) Except as provided in Section 620.450 or subsection (a)(3) or (e) of this Section, concentrations of the following chemical constituents shall not be exceeded in Class II groundwater:

CASRN	Constituent	Standard[a]
7429-90-5	Aluminum	5[b]
7440-42-8	Boron	2[c]
16887-00-6	Chloride	200[d]
7440-50-8	Copper	0.5[b]
7439-89-6	Iron	5[d]
7439-96-5	Manganese	10[c]
7440-02-0	Nickel	2[c]
7440-14-4	Radium (combined 226+228)	5[e]
7782-49-2	Selenium	0.02[c]
7440-22-4	Silver	0.019[e]
14808-79-8	Sulfate	400[d]
	TDS (total dissolved solids)	1,200[d]
7440-66-6	Zinc	10[c]

Constituent Name and Groundwater Quality Standard Notations

[a] The standard units are milligrams per liter ("mg/L"), except for the radium (combined 226+228) unit of picocuries per liter ("pCi/L").
[b] The standard is based on beneficial use for watering livestock, per "*Water Quality Criteria*", by National Academy of Sciences, incorporated by reference at Section 620.125.
[c] The standard is based on beneficial use for irrigation of crops, per "*Water Quality Criteria*", by National Academy of Sciences, incorporated by reference at Section 620.125.
[d] The standard is the 95% confidence concentration stated in Illinois EPA's "*Integrated Water Quality Report and Section 303(d) List*", incorporated by reference at Section 620.125.
[e] The Class II standard is equal to the Class I groundwater quality standard.

3) The standard for any inorganic chemical constituent listed in subsection (a)(2) of this Section, for barium, or for pH does not apply to groundwater within fill material or within the upper 10 feet of parent material under

such fill material on a site not within the rural property class for which:

A) Prior to November 25, 1991, surficial characteristics have been altered by the placement of such fill material so as to impact the concentration of the parameters listed in subsection (a)(3) of this Section, and any on-site groundwater monitoring of such parameters is available for review by the Agency.

B) On November 25, 1991, surficial characteristics are in the process of being altered by the placement of such fill material, that proceeds in a reasonably continuous manner to completion, so as to impact the concentration of the parameters listed in subsection (a)(3) of this Section, and any on-site groundwater monitoring of such parameters is available for review by the Agency.

4) For purposes of subsection (a)(3) of this Section, the term "fill material" means clean earthen materials, slag, ash, clean demolition debris, or other similar materials.

b) Organic Chemical Constituents

1) Except due to natural causes or as provided in Section 620.450 or subsection (b)(2) or (e) of this Section, concentrations of the following organic chemical constituents shall not be exceeded in Class II groundwater:

CASRN	Constituent	Standard (mg/L)
83-32-9	Acenaphthene	1.2[a]
67-64-1	Acetone	3.5[b]
15972-60-8	Alachlor[c,d]	0.002[b]
116-06-3	Aldicarb[d]	0.003[b]
120-12-7	Anthracene	6[a]
319-84-6	*alpha*-BHC (*alpha*-benzene hexachloride)[c,d]	0.00006[a]
71-43-2	Benzene[c]	0.025[a]
56-55-3	Benzo(a)anthracene[e]	0.001[a]
205-99-2	Benzo(b)fluoranthene[e]	0.001[a]
207-08-9	Benzo(k)fluoranthene[e]	0.01[a]
50-32-8	Benzo(a)pyrene[e]	0.001[a]
65-85-0	Benzoic acid	15[b]

CASRN	Constituent	Standard (mg/L)
78-93-3	2-Butanone (methyl ethyl ketone)	2.3[b]
1563-66-2	Carbofuran[d]	0.04[b]
75-15-0	Carbon disulfide	1.9[a]
56-23-5	Carbon tetrachloride[c]	0.025[a]
12798-03-6	Chlordane[c]	0.01[a]
108-90-7	Chlorobenzene	0.1[b]
67-66-3	Chloroform[c]	0.35[a]
218-01-9	Chrysene[e]	0.13[a]
94-75-7	2,4-D (2,4-dichlorophenoxy acetic acid)[d]	0.07[b]
75-99-0	Dalapon	0.2[b]
53-70-3	Dibenzo(a,h)anthracene[e]	0.000125[a]
96-12-8	1,2-Dibromo-3-chloropropane[e]	0.0002[b]
1918-00-9	Dicamba[d]	0.12[b]
95-50-1	*o*-Dichlorobenzene (1,2-dichlorobenzene)	0.6[b]
106-46-7	*p*-Dichlorobenzene (1,4-dichlorobenzene)[c]	0.075[b]
75-71-8	Dichlorodifluoromethane	3.9[a]
75-34-3	1,1-Dichloroethane	3.9[a]
107-06-2	1,2-Dichloroethane[c]	0.005[b]
75-35-4	1,1-Dichloroethylene	0.035[a]
156-59-2	*cis*-1,2-Dichloroethylene	0.35[a]
156-60-2	*trans*-1,2-Dichloroethylene	0.5[a]
75-09-2	Dichloromethane (methylene chloride)[e]	0.025[a]
78-87-5	1,2-Dichloropropane[b]	0.005[b]
117-81-7	Di(2-ethylhexyl)phthalate[b]	0.03[a]
84-66-2	Diethyl phthalate	3.1[b]
84-74-2	Di-*n*-butyl phthalate	1.9[a]
99-65-0	1,3-Dinitrobenzene	0.001[b]
121-42-2	2,4-Dinitrotoluene[c]	0.005[a]
606-20-0	2,6-Dinitrotoluene[c]	0.005[a]
88-85-7	Dinoseb[d]	0.035[a]
123-91-1	1,4-Dioxane (*p*-dioxane)[c]	0.00078[b]
145-73-3	Endothall[d]	0.1[b]
72-20-8	Endrin[d]	0.01[a]
100-41-4	Ethylbenzene[c]	3.5[a]
106-93-4	Ethylene dibromide (1,2-dibromoethane)[c]	0.00005[b]
206-44-0	Fluoranthene	0.75[a]
86-73-7	Fluorene	0.75[a]
58-89-9	*gamma*-HCH (*gamma*-	0.001[a]

CASRN	Constituent	Standard (mg/L)
	hexachlorocyclohexane, lindane)[c,d]	
2691-41-0	HMX (octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine)[d]	3.9[a]
76-44-8	Heptachlor[c]	0.002[a]
1024-57-3	Heptachlor epoxide[c]	0.001[a]
77-47-4	Hexachlorocyclopentadiene	0.25[a]
193-39-5	Indeno(1,2,3-c,d)pyrene[e]	0.0013[a]
98-82-8	Isopropylbenzene (cumene)[c]	1.9[a]
93-65-2	MCPP (mecoprop)[d]	0.1[b]
1634-04-4	MTBE (methyl tertiary-butyl ether)	0.038[b]
72-43-5	Methoxychlor[d]	0.2[a]
90-12-0	1-Methylnaphthalene	0.27[b]
91-57-6	2-Methylnaphthalene	0.015[b]
95-48-7	2-Methylphenol (*o*-cresol)	0.19[b]
91-20-3	Naphthalene	0.39[a]
98-95-3	Nitrobenzene	0.0077[b]
1336-36-3	PCBs (polychlorinated biphenyls as decachloro-biphenyl)[c,d]	0.0025[a]
375-73-5	PFBS (perfluorobutanesulfonic acid)	0.0012[b]
355-46-4	PFHxS (perfluorohexanesulfonic acid)	0.000077[b]
375-95-1	PFNA (perfluorononanoic acid)	0.000012[b]
335-67-1	PFOA (perfluorooctanoic acid)[c]	0.000002[b]
1763-23-1	PFOS (perfluorooctanesulfonic acid)	0.0000077[b]
87-86-5	Pentachlorophenol[d]	0.005[a]
108-95-2	Phenol	1.2[b]
1918-02-1	Picloram[d]	0.5[b]
129-00-0	Pyrene	0.6[a]
121-82-4	RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine)[d]	0.062[b]
122-34-9	Simazine[d]	0.004[b]
100-42-5	Styrene	0.1[b]
118-96-7	TNT (2,4,6-trinitrotoluene)	0.039[a]
93-72-1	2,4,5-TP (silvex)[d]	0.05[b]
127-18-4	Tetrachloroethylene[c]	0.025[a]
108-88-3	Toluene	5[a]
8001-35-2	Toxaphene[c,d]	0.015[a]
120-82-1	1,2,4-Trichlorobenzene	0.35[a]
71-55-6	1,1,1-Trichloroethane	1[a]

CASRN	Constituent	Standard (mg/L)
79-00-5	1,1,2-Trichloroethane	0.005[b]
79-01-6	Trichloroethylene[e]	0.025[a]
75-69-4	Trichlorofluoromethane	6[a]
99-35-4	1,3,5-Trinitrobenzene	2.3[a]
75-01-4	Vinyl chloride[e]	0.01[a]
1330-20-7	Xylenes	50[b]

Constituent Name and Groundwater Quality Standard Notations

[a] A treatment factor of 5 is applied to the Class I groundwater quality standard, based on Illinois EPA's treatment efficiency determination. A constituent's treatment efficiency is based the effectiveness to treat the constituent in the groundwater at an 80% removal efficiency rate for the constituent. A treatment factor of 5 is applied to a constituent having either an organic carbon partition coefficient (K_{oc}) greater than ethylbenzene's K_{oc} of 446 L/kg for carbon adsorption efficiency, or a constituent having a dimensionless Henry's Law Constant (H') greater than dichloromethane's (methylene chloride) H' of 0.11, when set at a groundwater system temperature of 20 degrees Celsius, for air stripping efficiency.

[b] Illinois EPA's treatment efficiency determination demonstrates a treatment factor is not applicable for the constituent. The standard is equal to the Class I groundwater quality standard.

[c] The constituent meets the definition of a "carcinogen" at Section 620.110.

[d] An enthalpy of vaporization value cannot be derived for the constituent; therefore, a dimensionless Henry's Law Constant value set at 25 degrees Celsius is used for evaluation of its treatment efficiency.

[e] The constituent meets the definition of a "mutagen" at Section 620.110.

2) The standards for pesticide chemical constituents listed in subsection (b)(1) of this Section do not apply to groundwater within 10 feet of the land surface, provided that the concentrations of such constituents result from the application of pesticides in a manner consistent with the requirements of the Federal Insecticide, Fungicide and Rodenticide Act (7 USC 136 et seq.), and the Illinois Pesticide Act [415 ILCS 60].

c)

Complex Organic Chemical Mixtures

1) Concentrations of the following organic chemical constituents shall not be exceeded in Class II groundwater:

CASRN	Constituent	Standard (mg/L)
71-43-2	Benzene[a]	0.025[b]
	Total BETX	58.525[c]

Constituent Name and Groundwater Quality Standard Notations

[a] The constituent meets the definition of a "carcinogen" at Section 620.110.
[b] A treatment factor of 5 is applied to the Class I groundwater quality standard, based on Illinois EPA's treatment efficiency determination.
[c] The standard is the total combined Class II standard of benzene, ethylbenzene, toluene, and xylenes.

2) Atrazine and Metabolites

The total concentration of Atrazine plus Atrazine metabolites shall be compared to the atrazine Class I groundwater standard of 0.003 mg/L.

CASRN	Constituent	Standard (mg/L)
1912-24-9	Atrazine	0.003[a]
	Total Atrazine and Metabolites[b] DEA (desethyl-atrazine) DIA (desisopropyl-atrazine) DACT (diaminochlorotriazine)	0.003

Constituent Name and Groundwater Quality Standard Notations:

[a] Illinois EPA's treatment efficiency determination demonstrates a treatment factor is not applicable for the constituent. Therefore, the standard is a concentration equal to the Class I groundwater quality standard.

[b] An enthalpy of vaporization value cannot be derived for the constituent; therefore, a dimensionless Henry's Law Constant value set at 25 degrees Celsius is used for evaluation of its treatment efficiency.

d) pH

Except due to natural causes, a pH range of 6.5 - 9.0 units shall not be exceeded in Class II groundwater that is within 5 feet of the land surface.

(Source: Amended at, effective)

Section 620.430 Groundwater Quality Standards for Class III: Special Resource Groundwater

Except due to natural causes, concentrations of inorganic and organic chemical constituents shall not exceed the standards set forth in Section 620.410, except for:

a) The chemical constituents for which the Board has adopted a standard pursuant to Section 620.260; and

b) The following standards set forth below for Class III Special Resource Groundwater established in accordance with Section 620.230(b) and depicted in the Environmental Register as indicated for each nature preserve.

1) The following standards are applicable for Pautler Cave Nature Preserve Stemler Cave Nature Preserve (Environmental Register, May 2005, Num. 611), Fogelpole Cave Nature Preserve (Environmental Register May 2003, Num. 587), and Armin Krueger Speleological Nature Preserve (Environmental Register, December 2009, Num. 666):

Chloride	20 mg/L
pH	range of 7.0-9.0 Standard Units

2) The following standard is applicable for Cotton Creek Marsh Nature Preserve and Spring Grove Fen Nature Preserve (Environmental Register, July 2012, Num 697):

Chloride	45 mg/L

(Source: Amended at __ Ill. Reg. _____, effective _____)

Section 620.440 Groundwater Quality Standards for Class IV: Other Groundwater

a) Except as provided in subsection (b) or (c), Class IV: Other Groundwater standards are equal to the existing concentrations of constituents in groundwater.

b) For groundwater within a zone of attenuation as provided in 35 Ill. Adm. Code 811 and 814, the standards specified in Section 620.420 shall not be exceeded, except for concentrations of contaminants within leachate released from a permitted unit.

c) For groundwater within a previously mined area, the standards set forth in Section 620.420 shall not be exceeded, except for concentrations of TDS, chloride, iron, manganese, sulfates, pH, 1,3-dinitrobenzene, 2,4-dinitrotoluene, 2,6-dinitrotoluene, HMX (octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine), nitrobenzene, RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine), 1,3,5-trinitrobenzene, or TNT (2,4,6-trinitrotoluene). For concentrations of TDS, chloride, iron, manganese, sulfates, pH, 1,3-dinitrobenzene, 2,4-dinitrotoluene, 2,6-dinitrotoluene, HMX, nitrobenzene, RDX, 1,3,5-trinitrobenzene, or TNT, the standards are the existing concentrations.

(Source: Amended at, effective)

Section 620.450 Alternative Groundwater Quality Standards

a) Groundwater Quality Restoration Standards

1) Any chemical constituent in groundwater within a groundwater management zone is subject to this Section.

2) Except as provided in subsections (a)(3) or (a)(4), the standards as specified in Sections 620.410, 620.420, 620.430, and 620.440 apply to any chemical constituent in groundwater within a groundwater management zone.

3) Prior to completion of a corrective action described in Section 620.250(a), the standards as specified in Sections 620.410, 620.420, 620.430, and 620.440 are not applicable to such released chemical constituent, provided that the initiated action proceeds in a timely and appropriate manner.

4) After completion of a corrective action as described in Section 620.250(a), the standard for such released chemical constituent is:

A) The standard as set forth in Section 620.410, 620.420, 620.430, or 620.440, if the concentration as determined by groundwater monitoring of such constituent is less than or equal to the standard for the appropriate class set forth in those Sections; or

B) The concentration as determined by groundwater monitoring, if such concentration exceeds the standard for the appropriate class set forth in Section 620.410, 620.420, 620.430, or 620.440 for such constituent, and:

i) To the extent practicable, the exceedence has been minimized and beneficial use, as appropriate for the class of groundwater, has been returned; and

ii) Any threat to public health or the environment has been minimized.

5) The Agency shall develop and maintain a listing of concentrations derived pursuant to subsection (a)(4)(B). This list shall be made available to the public and be updated periodically, but no less frequently than semi-annually. This listing shall be published in the Environmental Register.

b) Coal Reclamation Groundwater Quality Standards

1) Any inorganic chemical constituent or pH in groundwater, within an underground coal mine, or within the cumulative impact area of groundwater for which the hydrologic balance has been disturbed from a permitted coal mine area pursuant to the Surface Coal Mining Land Conservation and Reclamation Act [225 ILCS 720] and 62 Ill. Adm. Code 1700 through 1850, is subject to this Section.

2) Prior to completion of reclamation at a coal mine, the standards as specified in Sections 620.410(a) and (e), 620.420(a) and (e), 620.430, and 620.440 are not applicable to inorganic constituents and pH.

3) After completion of reclamation at a coal mine, the standards as specified in Sections 620.410(a) and (e), 620.420(a), 620.430, and 620.440 are applicable to inorganic constituents and pH, except:

A) The concentration of total dissolved solids ("TDS") shall not exceed:

i) The post-reclamation concentration or 3000 mg/L, whichever is less, for groundwater within the permitted area; or

ii) The post-reclamation concentration of TDS shall not exceed the post-reclamation concentration or 5000 mg/L, whichever is less, for groundwater in underground coal mines and in permitted areas reclaimed after surface coal mining if the Illinois Department of Mines and Minerals and the Agency have determined that no significant resource groundwater existed prior to mining (62 Ill. Adm. Code 1780.21(f) and (g)); and

B) For chloride, iron, manganese, and sulfate, the post-reclamation concentration within the permitted area shall not be exceeded.

C) For pH, the post-reclamation concentration within the permitted area shall not be exceeded within Class I: Potable Resource Groundwater as specified in Section 620.210(a)(4).

D) For 1,3-dinitrobenzene, 2,4-dinitrotoluene, 2,6-dinitrotoluene, HMX (octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine), nitrobenzene, RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine), 1,3,5-trinitrobenzene, and TNT (2,4,6-trinitrotoluene), the post-reclamation concentration within the permitted area shall not be exceeded.

4) A refuse disposal area (not contained within the area from which overburden has been removed) is subject to the inorganic chemical constituent and pH requirements of:

A) 35 Ill. Adm. Code 302.Subparts B and C, except due to natural causes, for such area that was placed into operation after February 1, 1983, and before the effective date of this Part, provided that the groundwater is a present or a potential source of water for public or food processing;

B) Section 620.440(c) for such area that was placed into operation prior to February 1, 1983, and has remained in continuous operation since that date; or

C) Subpart D of this Part for such area that is placed into operation on or after the effective date of this Part.

5) For a refuse disposal area (not contained within the area from which overburden has been removed) that was placed into operation prior to February 1, 1983, and is modified after that date to include additional area, this Section applies to the area that meets the requirements of subsection (b)(4)(C) and the following applies to the additional area:

A) 35 Ill. Adm. Code 302.Subparts B and C, except due to natural causes, for such additional refuse disposal area that was placed into operation after February 1, 1983, and before the effective date of this Part, provided that the groundwater is a present or a potential source of water for public or food processing; and

B) Subpart D for such additional area that was placed into operation on or after the effective date of this Part.

6) A coal preparation plant (not located in an area from which overburden has been removed) which contains slurry material, sludge, or other precipitated process material, is subject to the inorganic chemical

constituent and pH requirements of:

A) 35 Ill. Adm. Code 302.Subparts B and C, except due to natural causes, for such plant that was placed into operation after February 1, 1983 and before the effective date of this Part, provided that the groundwater is a present or a potential source of water for public or food processing;

B) Section 620.440(c) for such plant that was placed into operation prior to February 1, 1983, and has remained in continuous operation since that date; or

C) Subpart D for such plant that is placed into operation on or after the effective date of this Part.

7) For a coal preparation plant (not located in an area from which overburden has been removed) which contains slurry material, sludge or other precipitated process material, that was placed into operation prior to February 1, 1983, and is modified after that date to include additional area, this Section applies to the area that meets the requirements of subsection (b)(6)(C) and the following applies to the additional area:

A) 35 Ill. Adm. Code 302.Subparts B and C, except due to natural causes, for such additional area that was placed into operation after February 1, 1983, and before the effective date of this Part, provided that the groundwater is a present or a potential source of water for public or food processing; and

B) Subpart D for such additional area that was placed into operation on or after the effective date of this Part.

c) Groundwater Quality Standards for Certain Groundwater Subject to a No Further Remediation Letter under Part 740. While a No Further Remediation Letter is in effect for a region formerly encompassed by a groundwater management zone established under 35 Ill. Adm. Code 740.530, the groundwater quality standards for "contaminants of concern", as defined in 35 Ill. Adm. Code 740.120, within such area shall be the groundwater objectives achieved as documented in the approved Remedial Action Completion Report.

(Source: Amended at, effective)

Section 620.510 Monitoring and Analytical Requirements

a) Representative Samples
A representative sample shall be taken from locations as specified in Section 620.505.

b) Sampling and Analytical Procedures

1) Samples shall be collected in accordance with the procedures set forth in the documents pertaining to groundwater monitoring and analysis incorporated by reference at Section 620.125 or other procedures adopted by the appropriate regulatory agency.

2) Groundwater elevation in a groundwater monitoring well shall be determined and recorded when necessary to determine the gradient.

3) Unless specified otherwise by regulations, statistical methods used to determine naturally occurring groundwater quality background concentrations of contaminants shall be conducted in accordance with "Statistical Analysis of Groundwater Monitoring Data at RCRA Facilities, (March 2009 Unified Guidance)," as incorporated by reference in Section 620.125 for use with prediction limits and all other statistical tests including, but not limited to, confidence limits and control charts.

4) The analytical methodology used for the analysis of constituents in Subparts C and D shall be consistent with both of the following:

A) The methodology shall have a LLOQ or LCMRL at or below the preventive response levels of Subpart C or groundwater standard set forth in Subpart D, whichever is applicable; and

B) "Methods for Chemical Analysis of Water and Wastes," "Methods for the Determination of Inorganic Substances in Environmental Samples," "Methods for the Determination of Metals in Environmental Samples," "Methods for the Determination of Organic Compounds in Drinking Water," "Methods for the Determination of Organic Compounds in Drinking Water, Supplement I," "Methods for the Determination of Organic Compounds in Drinking Water, Supplement II," "Methods for the Determination of Organic Compounds in Drinking Water, Supplement III," "Methods for the Determination of Organic and

Inorganic Compounds in Drinking Water," "Prescribed Procedures for Measurement of Radioactivity in Drinking Water," "Procedures for Radiochemical Analysis of Nuclear Reactor Aqueous Solutions," "Radiochemical Analytical Procedures for Analysis of Environmental Samples," "Radiochemistry Procedures Manual," "Practical Guide for Ground Water Sampling," "Test Methods for Evaluating Solid Wastes, Physical/Chemical Methods" (SW-846), 40 CFR 136, appendix B, 40 CFR 141.80, 40 CFR 141.61, and 40 CFR 141.62, "Techniques of Water Resources Investigations of the United States Geological Survey, Guidelines for Collection and Field Analysis of Ground Water Samples for Selected Unstable Constituents," "Practical Guide for Ground-Water Sampling", "Techniques of Water Resources Investigations of the United States Geological Survey, Guidelines for Collection and Field Analysis of Ground-Water Samples for Selected Unstable Constituents", incorporated by reference at Section 620.125.

c) Reporting Requirements
At a minimum, groundwater monitoring analytical results shall include information, procedures, and techniques for:

1) Sample collection (including but not limited to name of sample collector, time and date of the sample, method of collection, and identification of the monitoring location);

2) Sample preservation and shipment (including but not limited to field quality control);

3) Analytical procedures (including but not limited to the MDL, LLOQ, or theLCMRL); and

4) Chain of custody control.

(Source: Amended at, effective)

SUBPART F: HEALTH ADVISORIES

Section 620.601 Purpose of a Health Advisory

This Subpart establishes procedures for the issuance of a Health Advisory that sets forth guidance levels that, in the absence of standards under Section 620.410, shall be considered by the Agency in:

a) Establishing groundwater cleanup or action levels whenever there is a release or substantial threat of a release of:

1) A hazardous substance or pesticide; or

2) Other contaminant that represents a significant hazard to public health or the environment.

b) Determining whether the community water supply is taking its raw water from a site or source consistent with the siting and source water requirements of 35 Ill. Adm. Code604.200.

c) Developing Board rulemaking proposals for new or revised numerical standards.

d) Evaluating mixtures of chemical substances.

(Source: Amended at, effective)

Section 620.605 Issuance of a Health Advisory

a) The Agency shall issue a Health Advisory for a chemical substance if all of the following conditions are met:

1) A community water supply well is sampled and a substance is detected and confirmed by resampling;

2) There is no standard under Section 620.410 for such chemical substance; and

3) The chemical substance is toxic or harmful to human health according to the procedures of Appendix A, B, or C.

b) The Health Advisory shall contain a general description of the characteristics of the chemical substance, the potential adverse health effects, and a guidance level to be determined as follows:

1) If disease or functional impairment is caused due to a physiological mechanism for where there is a threshold dose below which no damage occurs, the guidance level for any such substance shall be the Maximum Contaminant Level Goal ("MCLG"), adopted by U.S. EPA for such substance, 40 CFR 136, appendix B, 40 CFR 141.80, 40 CFR 141.61, and 40 CFR 141.62, incorporated by reference at Section 620.125. If there is no MCLG for the substance, the guidance level is either the Human Threshold Toxicant Advisory Concentration or the Human Nonthreshold Toxicant Advisory Concentration for such substance as determined in accordance with Appendix A, whichever is less, unless the lower concentration for such substance is less than the lowest appropriate LLOQ specified in "Test Methods for Evaluating Solid Wastes, Physical/Chemical Methods", EPA Publication No. SW-846 (SW-846), incorporated by reference at Section 620.125, or the LCMRL specified in the drinking water methods incorporated by reference at Section 620.125 for the substance. If the concentration for such substance is less than the lowest appropriate LLOQ or LCMRL for the substance s, incorporated by reference at Section 620.125, the guidance level is the lowest appropriate LLOQ or LCMRL.

(Source: Amended at, effective)

Section 620.APPENDIX A Procedures for Determining Human Toxicant Advisory Concentrations for Class I: Potable Resource Groundwater

a) Calculating the Human Threshold' Toxicant Advisory Concentration for Non-Cancer Effects

For those substances for which U.S. EPA has not adopted a Maximum Contaminant Level Goal ("MCLG"), the Human Threshold Toxicant Advisory Concentration is calculated as follows:

$$HTTAC = \frac{RSC \bullet ADE}{W}$$

Where:

HTTAC = Human Threshold Toxicant Advisory Concentration in milligrams per liter ("mg/L");

RSC = Relative contribution of the amount of the exposure to a chemical via drinking water when compared to the total exposure to that chemical from all sources. Valid chemical-specific data shall be used if available. If valid chemical-specific data are not available, a value of 20% (= 0.20) shall be used;

ADE = Acceptable Daily Exposure of substance in milligrams per day ("mg/d") as determined pursuant to subsection (b); and

W = Per capita daily water consumption for a child (0-6 years of age, equal to 0.78 liters per day ("L/d").

b) Procedures for Determining Acceptable Daily Exposures for Class I: Potable Resource Groundwater

1) The Acceptable Daily Exposure ("ADE") represents the maximum amount of a threshold toxicant in milligrams per day ("mg/d"), which if ingested daily by a child from 0 to 6 years of ageresults in no adverse effects. Subsections (b)(2) through (b)(6) list, in prescribed order, methods for determining the ADE in Class I: Potable Resource Groundwater.

2) For those substances for which non-cancer toxicity values have been derived and presented in units of milligrams per kilogram per day ("mg/kg/day"), as determined by U.S. EPA's hierarchy of usable sources, the ADE equals the product of multiplying the toxicity value by 15 kilograms ("kg"), which is the assumed average weight of a child 0 to 6 years of age. The hierarchy of sources for toxicity values are listed in the following order:

A) Tier I: U.S. EPA Integrated Risk Information System ("IRIS")

B) Tier II: Provisional Peer Reviewed Toxicity Values ("PPRTV")

C) Tier III: Other peer reviewed toxicity values which are transparent and publicly available including, but not limited to:

i) Agency for Toxic Substances and Disease Registry ("ATSDR") dose Minimal Risk Level ("dose-MRL")

ii) California Environmental Protection Agency, Office of Environmental Health Hazard Assessment ("Cal EPA – OEHHA")

iii) PPRTV Appendix "Screening Toxicity Values"

iv) HEAST toxicity values

3) For those substances for which an oral reference dose is not available from the hierarchy of sources for toxicity values, the ADE equals the value of the most sensitive Point of Departure ("POD") as determined by Benchmark Dose Modeling or the NOAEL/LOAEL approach consistent with current U.S. EPA RfD guidance, followed by the derivation of a Human Equivalent Dose ("HED") using physiologically based pharmacokinetic ("PBPK") modeling or Dose Adjustment Factor ("DAF"), then divided by the total Uncertainty Factor ("UF"). The value is then multiplied by 15 kg (the assumed average weight of a child 0 – 6 years of age). The equation is depicted below:

$$ADE = \frac{POD}{Total\ UF} \bullet 15kg$$

4) Uncertainty Factors shall be applied to the Point of Departure ("POD") in

increments of 1, 3, or 10, not to exceed a total UF of 10,000, and shall be used consistent with U.S. EPA guidance. A composite UF of 3 and 10 shall be expressed as 30 whereas a composite UF of 3 and 3 shall be expressed as 10. UFs may be used to account for the following:

A) Interspecies Variability

B) Intraspecies Variability

C) Lowest Observable Adverse Effects Level ("LOAEL") to No Observed Adverse Effects Level ("NOAEL") Uncertainty

D) Database Deficiencies

E) Subchronic to Chronic Duration

c)

Calculating a Human Nonthreshold Toxicant Advisory Concentration ("HNTAC") for Cancer Risk

The Human Nonthreshold Toxicant Advisory Concentration ("HNTAC") is calculated as follows:

1) For chemicals designated by U.S. EPA as "mutagens," the HNTAC is calculated as follows:

$$HNTAC = \frac{TR \bullet \left(AT \bullet 365 \frac{days}{year}\right)}{SF_o \bullet IFWM_{adj}}$$

Where:

HNTAC	=	Human Nonthreshold Toxicant Advisory Concentration, equal to milligrams per liter (mg/L)
TR	=	Target Cancer Risk, equal to one-in-one million cancer risk (1E-06)
AT	=	Averaging Time, equal to 70 years
SF_o	=	Oral Slope Factor (chemical-specific), equal to $(mg/kg\text{-}day)^{-1}$

$IFWM_{adj}$	=	Age-Adjusted Mutagenic Drinking Water Ingestion Rate, equal to 1,019.9 liters per kilogram (L/kg)

2) For chemicals not designated by U.S. EPA as "mutagens," the HNTAC is calculated as follows:

$$HNTAC = \frac{TR \bullet \left(AT \bullet 365 \frac{days}{year}\right)}{SF_o \bullet IFW_{adj}}$$

Where:

HNTAC	=	Human Nonthreshold Toxicant Advisory Concentration, equal to milligrams per liter (mg/L)
TR	=	Target Cancer Risk, equal to one-in-one million cancer risk (1E-06)
AT	=	Averaging Time, equal to 70 years
SF_o	=	Oral Slope Factor (chemical-specific), equal to $(mg/kg\text{-}day)^{-1}$
IFW_{adj}	=	Age-Adjusted Drinking Water Ingestion Rate, equal to 327.95 liters per kilogram (L/kg)

(Source: Amended at, effective)

Section 620.APPENDIX B Procedures for Determining Hazard Indices for Class I: Potable Resource Groundwater for Mixtures of Similar-Acting Substances

a) This appendix describes procedures for evaluating mixtures of similar-acting substances which may be present in Class I: Potable Resource Groundwaters. Except as provided otherwise in subsection (c), subsections (d) through (h) describe the procedure for determining the Hazard Index for mixtures of similar-acting substances.

b) For the purposes of this appendix, a "mixture" means two or more substances which are present in Class I: Potable Resource Groundwater which may or may not be related either chemically or commercially, but which are not complex mixtures of related isomers and congeners which are produced as commercial products (for example, PCBs or technical grade chlordane).

c) The substances listed in Appendix E are similar acting substances.

d) When two or more substances occur together in a mixture, the additivity of the toxicities of some or all of the substances will be considered when determining health-based standards for Class I: Potable Resource Groundwater. This is done by the use of a dose addition model with the development of a Hazard Index for the mixture of substances with similar-acting toxicities. This method does not address synergism or antagonism. Guidelines for determining when the dose addition of similar-acting substances is appropriate are presented in Appendix C. The Hazard Index is calculated as follows:

$$HI = [A]/ALA + [B]/ALB + \ldots [I]/ALI$$

Where:

HI	=	Hazard Index, unitless.
[A], [B], [I]	=	Concentration of each similar-acting substance in groundwater in milligrams per liter (“mg/L”).
ALA, ALB, ALI	=	The acceptable level of each similar-acting substance in the mixture in milligrams per liter (“mg/L”).

e) For substances that are considered to have a threshold mechanism of toxicity, the acceptable level is:

1) The standards listed in Section 620.410; or

2) For those substances for which standards have not been established in Section 620.410, the Human Threshold Toxicant Advisory Concentration ("HTTAC") as determined in Appendix A.

f) For substances that are carcinogens, the acceptable level is:

1) The standards listed in Section 620.410; or

2) For those substances for which standards have not been established under Section 620.410, the one-in-one-million cancer risk concentration, unless the concentration for such substance is less than the lowest appropriate LLOQ specified in "Test Methods for Evaluating Solid Wastes, Physical/Chemical Methods," EPA Publication No. SW-846, incorporated by reference at Section 620.125, or the LCMRL specified in the drinking water methods incorporated by reference at Section 620.125 for the substance, incorporated by reference at Section 620.125, the guidance level is the lowest appropriate LLOQ or LCMRL.

g) Since the assumption of dose addition is most properly applied to substances that induce the same effect by similar modes of action, a separate Hazard Index shall be generated for each toxicity endpoint of concern.

h) In addition to meeting the individual substance objectives, a Hazard Index shall be less than or equal to 1 for a mixture of similar-acting substances.

(Source: Amended at, effective)

Section 620.APPENDIX C Guidelines for Determining When Dose Addition of Similar-Acting Substances in Class I: Potable Resource Groundwaters is Appropriate

a) Substances shall be considered similar-acting if:

1) The substances have the same target in an organism (for example, the same organ, organ system, receptor, or enzyme).

2) The substances have the same mode of toxic action. These actions may include, for example, central nervous system depression, liver toxicity, or cholinesterase inhibition.

b) Substances that have fundamentally different mechanisms of toxicity (threshold toxicants vs. carcinogens) shall not be considered similar-acting. However, carcinogens which also cause a threshold toxic effect should be considered in a mixture with other similar-acting substances having the same threshold toxic effect. In such a case, an Acceptable Level for the carcinogen shall be derived for its threshold effect, using the procedures described in Appendix A.

c) Substances which are components of a complex mixture of related compounds which are produced as commercial products (for example, PCBs or technical grade chlordane) are not mixtures, as defined in Appendix B. Such complex mixtures are equivalent to a single substance. In such a case, the Human Threshold Toxicant Advisory Concentration may be derived for threshold effects of the complex mixture, using the procedures described in Appendix A, if valid toxicological or epidemiological data are available for the complex mixture. If the complex mixture is a carcinogen, the Health Advisory Concentration is the one-in-one-million cancer risk concentration, unless the lower concentration for such substance is less than the lowest appropriate LLOQ specified in "Test Methods for Evaluating Solid Wastes, Physical/Chemical Methods," EPA Publication No. SW-846, incorporated by reference at Section 620.125, or the LCMRL specified in the drinking water methods incorporated by reference at Section 620.125 for the substance. If the concentration for such substance is less than the lowest appropriate LLOQ or LCMRL for the substance incorporated by reference at Section 620.125, the guidance level is the lowest appropriate LLOQ or LCMRL.

(Source: Amended at, effective)

Section 620.APPENDIX E: Similar-Acting Substances

Table A: Similar-Acting Noncarcinogenic Constituents

Adrenal Gland	
106-93-4	Ethylene dibromide (1,2-dibromoethane)
98-82-8	Isopropylbenzene (cumene)
120-82-1	1,2,4-Trichlorobenzene
Cholinesterase Inhibition	
116-06-3	Aldicarb
1563-66-2	Carbofuran
Circulatory System	
15972-60-8	Alachlor
7440-36-0	Antimony
71-43-2	Benzene
7440-48-4	Cobalt
94-75-7	2,4-D (2,4-dichlorophenoxy acetic acid)
156-59-2	*cis*-1,2-Dichloroethylene
121-42-2	2,4-Dinitrotoluene
606-20-0	2,6-Dinitrotoluene
206-44-0	Fluoranthene
86-73-7	Fluorene
14797-55-8	Nitrate as N
98-95-3	Nitrobenzene
355-46-4	PFHxS (perfluorohexanesulfonic acid)
7782-49-2	Selenium
122-34-9	Simazine
100-42-5	Styrene
99-35-4	1,3,5-Trinitrobenzene
7440-66-6	Zinc
Decreased Body Weight Gain	
1912-24-9	Atrazine
143-33-9	Cyanide (sodium cyanide)
84-66-2	Diethyl phthalate
95-48-7	2-Methylphenol (*o*-cresol)
91-20-3	Naphthalene
7440-02-0	Nickel
108-95-2	Phenol

122-34-9	Simazine
127-18-4	Tetrachloroethylene
71-55-6	1,1,1-Trichloroethane
1330-20-7	Xylenes
Developmental	
50-32-8	Benzo(a)pyrene
78-87-5	1,2-Dichloropropane
7439-93-2	Lithium
375-73-5	PFBS (perfluorobutanesulfonic acid)
355-46-4	PFHxS (perfluorohexanesulfonic acid)
375-95-1	PFNA (perfluorononanoic acid)
335-67-1	PFOA (perfluorooctanoic acid)
1763-23-1	PFOS (perfluorooctanesulfonic acid)
79-01-6	Trichloroethylene
Endocrine System	
143-33-9	Cyanide (sodium cyanide)
98-95-3	Nitrobenzene
Eye	
1336-36-3	PCBs (polychlorinated biphenyls as decachloro-biphenyl)
79-01-6	Trichloroethylene
Gastrointestinal System	
7440-41-7	Beryllium
7440-50-8	Copper
145-73-3	Endothall
7681-49-4	Fluoride (sodium fluoride)
77-47-4	Hexachlorocyclopentadiene
7439-89-6	Iron
1634-04-4	MTBE (methyl tertiary-butyl-ether)
Immune System	
156-60-5	*trans*-1,2-Dichloroethylene
7487-94-7	Mercury (mercuric chloride)
1336-36-3	PCBs (polychlorinated biphenyls as decachloro-biphenyl)
355-46-4	PFHxS (perfluorohexanesulfonic acid)
335-67-1	PFOA (perfluorooctanoic acid)

1763-23-1	PFOS (perfluorooctanesulfonic acid)
Kidney	
67-64-1	Acetone
7440-39-3	Barium
94-75-7	2,4-D (2,4-dichlorophenoxy acetic acid)
75-99-0	Dalapon
75-34-3	1,1-Dichloroethane
107-06-2	1,2-Dichloroethane
156-59-2	*cis*-1,2-Dichloroethylene
100-41-4	Ethylbenzene
206-44-0	Fluoranthene
58-89-9	*gamma*-HCH (*gamma*-hexachlorocyclohexane, lindane)
98-82-8	Isopropylbenzene (cumene)
7439-93-2	Lithium
93-65-2	MCPP (mecoprop)
375-73-5	PFBS (perfluorobutanesulfonic acid)
87-86-5	Pentachlorophenol
129-00-0	Pyrene
108-88-3	Toluene
7440-62-2	Vanadium
Liver	
83-32-9	Acenaphthene
319-84-6	*alpha*-BHC (*alpha*-benzene hexachloride)
56-23-5	Carbon tetrachloride
12798-03-6	Chlordane
108-90-7	Chlorobenzene
67-66-3	Chloroform
94-75-7	2,4-D (2,4-dichlorophenoxy acetic acid)
117-81-7	Di(2-ethylhexyl)phthalate
95-50-1	*o*-Dichlorobenzene (1,2-dichlorobenzene)
106-46-7	*p*-Dichlorobenzene (1,4-dichlorobenzene)
75-71-8	Dichlorodifluoromethane
75-35-4	1,1-Dichloroethylene
156-60-2	*trans*-1,2-Dichloroethylene
78-87-5	1,2-Dichloropropane
75-09-2	Dichloromethane (methylene chloride)
121-42-2	2,4-Dinitrotoluene
606-20-0	2,6-Dinitrotoluene

123-91-1	1,4-Dioxane (*p*-dioxane)
72-20-8	Endrin
106-93-4	Ethylene dibromide (1,2-dibromoethane)
100-41-4	Ethylbenzene
206-44-0	Fluoranthene
58-89-9	*gamma*-HCH (*gamma*-hexachlorocyclohexane, lindane)
2691-41-0	HMX (octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine)
76-44-8	Heptachlor
1024-57-3	Heptachlor Epoxide
1634-04-4	MTBE (methyl tertiary-butyl ether)
375-73-5	PFBS (perfluorobutanesulfonic acid)
355-46-4	PFHxS (perfluorohexanesulfonic acid)
375-95-1	PFNA (perfluorononanoic acid)
335-67-1	PFOA (perfluorooctanoic acid)
1763-23-1	PFOS (perfluorooctanesulfonic acid)
87-86-5	Pentachlorophenol
1918-02-1	Picloram
100-42-5	Styrene
118-96-7	TNT (2,4,6-trinitrotoluene)
127-18-4	Tetrachloroethylene
93-72-1	2,4,5-TP (silvex)
79-00-5	1,1,2-Trichloroethane
75-01-4	Vinyl chloride
Mortality	
84-74-2	Di-*n*-butyl phthalate
1330-20-7	Xylenes
Nervous System	
7429-90-5	Aluminum
143-33-9	Cyanide (sodium cyanide)
121-42-2	2,4-Dinitrotoluene
606-20-0	2,6-Dinitrotoluene
72-20-8	Endrin
7439-93-2	Lithium
7439-96-5	Manganese
95-48-7	2-Methylphenol (*o*-cresol)
1763-23-1	PFOS (perfluorooctanesulfonic acid)
7782-49-2	Selenium
127-18-4	Tetrachloroethylene

Reproductive System	
117-81-7	Di(2-ethylhexyl)phthalate
7440-42-8	Boron
78-93-3	2-Butanone (methyl ethyl ketone)
1563-66-2	Carbofuran
75-15-0	Carbon disulfide
96-12-8	1,2-Dibromo-3-chloropropane
88-85-7	Dinoseb
106-93-4	Ethylene dibromide (1,2-dibromoethane)
7439-93-2	Lithium
72-43-5	Methoxychlor
375-73-5	PFBS (perfluorobutanesulfonic acid)
335-67-1	PFOA (perfluorooctanoic acid)
1763-23-1	PFOS (perfluorooctanesulfonic acid)
108-95-2	Phenol
121-82-4	RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine)
Skin	
7440-38-2	Arsenic
1336-36-3	PCBs (polychlorinated biphenyls as decachloro-biphenyl)
7782-49-2	Selenium
7440-22-4	Silver
7440-28-0	Thallium
Spleen	
99-65-0	1,3-Dinitrobenzene
606-20-2	2,6-Dinitrotoluene
99-35-4	1,3,5-Trinitrobenzene
Thyroid	
7440-48-4	Cobalt
14797-73-0	Perchlorate
355-46-4	PFHxS (perfluorohexanesulfonic acid)
375-73-5	PFBS (perfluorobutanesulfonic acid)
335-67-1	PFOA (perfluorooctanoic acid)
8001-35-2	Toxaphene

Table B: Similar-Acting Carcinogenic Constituents

Circulatory System

71-43-2	Benzene
107-06-2	1,2-Dichloroethane
106-93-4	Ethylene dibromide (1,2-dibromoethane)
87-86-5	Pentachlorophenol
Gastrointestinal System	
56-55-3	Benzo(a)anthracene
205-99-2	Benzo(b)fluoranthene
207-08-9	Benzo(k)fluoranthene
50-32-8	Benzo(a)pyrene
218-01-9	Chrysene
53-70-3	Dibenzo(a,h)anthracene
106-93-4	Ethylene dibromide (1,2-dibromoethane)
193-39-5	Indeno(1,2,3-c,d)pyrene
Kidney	
67-66-3	Chloroform
96-12-8	1,2-Dibromo-3-chloropropane (dibromochloropropane)
100-41-4	Ethylbenzene
335-67-1	PFOA (perfluorooctanoic acid)
Liver	
319-84-6	*alpha*-BHC (*alpha*-benzene hexachloride)
117-81-7	Di(2-ethylhexyl)phthalate
56-23-5	Carbon tetrachloride
12798-03-6	Chlordane
67-66-3	Chloroform
106-46-7	*p*-Dichlorobenzene (1,4-dichlorobenzene)
75-09-2	Dichloromethane (methylene chloride)
78-87-5	1,2-Dichloropropane
121-14-2	2,4-Dinitrotoluene
606-20-0	2,6-Dinitrotoluene
123-91-1	1,4-Dioxane (*p*-dioxane)
58-89-9	*gamma*-HCH (*gamma*-hexachlorocyclohexane, lindane)
76-44-8	Heptachlor
1024-57-3	Heptachlor epoxide
87-86-5	Pentachlorophenol
1336-36-3	PCBs (polychlorinated biphenyls as decachloro-biphenyl)
335-67-1	PFOA (perfluorooctanoic acid)
127-18-4	Tetrachloroethylene

8001-35-2	Toxaphene
79-01-6	Trichloroethylene
75-01-4	Vinyl Chloride
Mammary Gland	
121-14-2	2,4-Dinitrotoluene
606-20-0	2,6-Dinitrotoluene

Attachment 4

Join from the meeting link
https://illinois.webex.com/illinois/j.php?MTID=m699b2767bc0dcdeb49dcb54296d2fbe4

Join by meeting number
Meeting number (access code): 177 631 1593
Meeting password: 83vsYMPMPr4

Tap to join from a mobile device (attendees only)
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+1-415-655-0002 US Toll
Global call-in numbers

Join from a video system or application
Dial 1776311593@illinois.webex.com
You can also dial 173.243.2.68 and enter your meeting number.

Room reserved? Sangamo room? Smaller room?

Carol and Lynn will present PowerPoint slides. Lynn Dunnaway has 6 slides, Caro with more. Sabrina to share document.

Brad and Sabrina to moderate with me

Most questions in the past were directed to BOL, so Greg Dunn will be there this time.

Will need to reserve BOL mobile equipment.

Record

Panelists up front (Sarah?), Barb

Illinois Environmental Protection Agency Proposed Updates to 35 Ill. Adm. Code 620

By: Carol Hawbaker
Environmental Risk Assessor
Illinois Environmental Protection Agency
Office of Toxicity Assessment

May 26, 2021

Proposed Updates to 35 Ill. Adm. Code 620

The presentation will cover the following topics:

- Introduction of nine new constituents.
- Addition of three metabolites to be evaluated with atrazine for compliance with groundwater quality standards (GQS).
- Combination of radium 226 and 228 to form a new combined radium (226+228) constituent.
- Addition of carcinogen designations for four existing constituents.
- Updates to constituents in the tables at Section 620.310(a)(3)(A)(i) and (ii).

Proposed Updates to 35 Ill. Adm. Code 620

The presentation will cover the following topics (continued):

- Updates of Class I GQS for three inorganic constituents from MCLs to irrigation/livestock water quality standards, based on beneficial use of groundwater.
- For constituents which Class I GQS are based on procedures found in Section 620, Subpart F and Appendix A:
  - Updates to toxicity values and relative source contribution (RSC) values;
  - Updates to exposure factors;
  - Addition of a mutagenic method for the development of carcinogen GQS for constituents with a mutagenic mode of action.

Proposed Updates to 35 Ill. Adm. Code 620

The presentation will cover the following topics (continued):

- Updates to Class II GQS.
- Introduction of tables (Appendix E) listing constituents that are similar-acting.

Introduction of New Constituents

- Aluminum
- Lithium
- 1-Methylnaphthalene
- Molybdenum

- Five Per-and Polyfluoroalkyl Substances (PFAS):
  - PFBS (Perfluorobutanesulfonic Acid)
  - PFHxS (Perfluorohexanesulfonic Acid)
  - PFNA (Perfluorononanoic Acid)
  - PFOA (Perfluorooctanoic Acid)
  - PFOS (Perfluorooctanesulfonic Acid)

Introduction of New Constituents

Proposed Class I and Class II GQS:

CASRN	Constituent	Proposed Class I GQS (mg/L)	Class I Source	Proposed Class II GQS (mg/L)	Class II Source
7429-90-5	Aluminum	1.9	Subpart F	5	Livestock
7439-93-2	Lithium	0.01	Subpart F	2.5	Irrigation
90-12-0	1-Methylnaphthalene	0.27	Subpart F	0.27	Subpart F
7439-98-7	Molybdenum	0.019	Subpart F	0.05	Irrigation

Per and Poly-Fluoroalkyl Substances (PFAS)

PFAS are a group of human-made constituents applied to many consumers products to make them waterproof, stain resistant or non-stick.

- Non-Stick Pans;
- Stain-Resistant Carpets and Textiles;
- Water-Proof Clothing and Footwear;
- Food Packaging (Pizza Boxes, Food Wrappers, Microwave Popcorn Bags, etc.);
- Fire-Fighting Foam;
- Personal Care Products (shampoos, conditioners, dental floss, cosmetics, suntan lotion, etc.);
- Paints and sealants;
- Industrial Uses (metal plating, wire coatings, automotive fluids, artificial turf;
- Firefighting Foam (AFFF).

Per and Poly-Fluoroalkyl Substances (PFAS)

PFAS are constituents of emerging concern:

- "Forever Chemicals": PFAS not degrade naturally in the environment.
- PFAS constituents have an affinity for water and can migrate long distances.
- PFAS can bioaccumulate in plants, fish and wildlife, and humans.
- PFAS are a group of chemicals consisting of over 5,000 substances.
- Toxicological studies and assessments are being conducted by several agencies.
- Limited data for most PFAS: verified toxicological data for 5 PFAS: PFBS, PFHxS, PFNA, PFOA, and PFOS.

Per and Poly-Fluoroalkyl Substances (PFAS)

Epidemiology and Animal Studies Suggest Associations Between PFAS Exposure and Several Health Effects:

- Pregnancy-Induced Hypertension/Pre-Eclampsia
- Liver Damage
- High Cholesterol
- Thyroid Disease
- Decreased Response to Vaccines
- Decreased Fertility
- Decreased Birth Weight
- Developmental Delays

Per and Poly-Fluoroalkyl Substances (PFAS)

- PFOA meets Illinois EPA's definition of a carcinogen. The International Agency for Research on Cancer (IARC) classified PFOA as a "2B" carcinogen in 2017.
- A "2B" classification means the constituent is possibly carcinogenic to humans.
- U.S. EPA concluded there was "suggestive potential" for PFOS to be carcinogenic to humans; however, PFOS does not meet Illinois EPA's definition of a carcinogen at this time.
- Possible Cancer Links:
  - Kidney
  - Testicular
  - Prostate
  - Liver
  - Pancreas

Per and Poly-Fluoroalkyl Substances (PFAS)

Proposed Class I GQS are based on proposed procedures for 35 Ill. Adm. Code 620, Subpart F and Appendix A.

CASRN	Constituent	Class I and Class II GQS (mg/L or ppm)	Class I and Class II GQS (ng/L or ppt)	Toxicity Value	Toxicity Value Source	Relative Source Contribution Value for Noncarcinogens
375-73-5	PFBS	0.0012	1,200	3E-04	PPRTV	0.2
355-46-4	PFHxS	0.000077	77	2E-05	ATSDR	0.2
375-95-1	PFNA	0.000012	12	3E-06	ATSDR	0.2
335-67-1	PFOA	0.000002	2	1.4E+02	OEHHA	Not Applicable
1763-23-1	PFOS	0.0000077	7.7	2E-06	ATSDR	0.2

PPRTV: Provisional Peer Reviewed Toxicity Values.

ATSDR: Agency for Toxic Substance and Disease Registry.

OEHHA: California EPA Office of Environmental Health Hazard Assessments.

PFBS, PFHxS, PFNA and PFOS toxicity values are oral reference doses (RfDs) for noncarcinogen effects in units of mg/kg-day.

PFOA toxicity value is an oral slope factor (SF_o) for cancer risks in units of $(mg/kg\text{-}day)^{-1}$. The GQS are the minimum reporting level, per Subpart F.

Addition of Atrazine Metabolites

The amendments propose the addition of 3 atrazine metabolites to be included when comparing atrazine concentrations to GQS.

Added Metabolites

- DEA (Desethyl-atrazine)
- DIA (Desisopropyl-atrazine)
- DACT (Diaminochlorotriazine)

Combination of Radium 226 and 228

Presently, radium 226 and radium 288 are listed separately in the Class I GQS. They are not listed in the Class II GQS.

The amendments propose radium (combined 226+228) Class I and Class II GQS.

The proposed value for the Class I and Class II GQS is based on the Federal maximum contaminant level (MCL) for radium (combined 226+228) of 5 picocuries per liter (pCi/L).

Proposed Updates to Carcinogen Designations

Carcinogen designations are updated for the following constituents:

- *p*-Dichlorobenzene (1,4-dichlorobenzene)
  - Classified “2B” by International Agency for Research on Cancer (IARC) - 1999
- Ethylbenzene
  - Classified “2B” by IARC - 2000
- *gamma*-HCH (*gamma*-Hexachlorocyclohexane, lindane)
  - Classified “1” by IARC – 2018
- Isopropylbenzene (cumene)
  - Classified “2B” by IARC - 2013

In addition, PFOA is classified “2B” by IARC – 2017; therefore, it meets the definition of a “carcinogen” per the Illinois Environmental Protection Act (415 ILCS 5/58.2).

Proposed Updates to Constituents in Tables at 35 Ill. Adm. Code 620.310(a)(3)(A)(i) and (ii) – Preventive Response Activities

The following constituents are removed from the tables due to carcinogenicity classifications, based on the Board Note at Section 620.310(a)(3)(A).

- *p*-Dichlorobenzene (1,4-dichlorobenzene)
- Ethylbenzene
- Arsenic
- *gamma*-HCH (lindane)
- Isopropylbenzene (cumene)

MCPP (mecoprop) is removed as the constituent's proposed Class I GQS is based on its lowest level of quantitation (LLOQ) or lowest concentration minimum reporting level (LCMRL).

Proposed Updates to Constituents in Tables at 35 Ill. Adm. Code 620.310(a)(3)(A)(i) and (ii) – Preventive Response Activities

Constituents Added to Tables

- Aluminum
- Molybdenum
- 1-Methylnaphthalene
- PFBS
- PFHxS
- PFNA
- PFOS
- Antimony
- HMX (octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine)
- Nitrobenzene
- RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine
- TNT (2,4,6-trinitrotoluene)
- 1,3,5-Trinitrobenzene

Proposed Updates of Class I GQS for Three Inorganic Constituents Based on More Stringent Irrigation or Livestock Values

Class I potable resource groundwater may also be used for irrigation and watering of livestock. The following constituents are proposed to be updated as follows:

CASRN	Constituent	Current Class I GQS (mg/L)	Current Source	Proposed Class I GQS (mg/L)	Proposed Source
7440-50-8	Copper	0.65	Lead/Copper Rule	0.5	Livestock
7681-49-4	Fluoride	4	U.S. EPA MCL	2	Livestock
7782-49-2	Selenium	0.05	U.S. EPA MCL	0.02	Irrigation

Proposed Updates to Subpart F and Appendix A

Out of 115 total constituents presently listed at 35 Ill. Adm. Code 620.410, 40 utilize the procedures in Subpart F and Appendix A to develop its Class I GQS:

- 30 constituents utilize the Human Threshold Toxicant Advisory Concentration (HTTAC) calculation at Appendix A(a) for noncarcinogens.

- 10 constituents utilize the Human Nonthreshold Toxicant Advisory Concentration (HNTAC) calculation at 35 Ill. Adm. Code 620.605(b)(2), for carcinogens. Of these 10, 7 constituents utilize a practical quantitation limit (PQL), because the calculated HNTAC is less than the PQL.

Proposed Updates to Appendix A

Illinois EPA's Hierarchy for Determining Toxicity Values

Basis for hierarchy is derived from U.S. EPA OSWER Directive 9285.7-53, dated December 5, 2003, and discussed in the Illinois Pollution Control Board Rulemaking R08-18: Proposed Amendments to Groundwater Quality Standards, 35 Ill. Adm. Code 620.

- Tier 1 Toxicity Value Source: Integrated Risk Information System (IRIS)
- Tier 2 Toxicity Value Source: Provisional Peer Reviewed Toxicity Values (PPRTV)
- Tier 3: Other Toxicity Values

"Priority given to sources of information that are the most current, the basis for which is transparent and publicly available, and which has been peer-reviewed."

OSWER Directive 9285.7-53

Proposed Updates to Appendix A

Additional Guidance Regarding the Selection of Tier 3 Toxicity Values derived from U.S. EPA OSWER Directive 9285.7-86, dated May 16, 2013. Tier 3 sources are ranked as follows:

1. Agency for Toxic Substances and Disease Registry (ATSDR) minimal risk levels.
2. California EPA, Office of Environmental Health Hazard Assessment (OEHHA), toxicity values.
3. PPRTV Appendix "Screening Toxicity Values".
4. Health Effect Assessment Summary Table (HEAST) toxicity values.

Proposed Updates to Appendix A

Updates Procedures for Determining an Oral Reference Dose (RfD) When an RfD is Not Available from the Listed Toxicity Values Sources.

- Proposes to update the procedures found at 35 Ill. Adm. Code 620, Appendix A(b)(3)-(c) for when there is no “verified” RfD, due to outdated methodology.
- The proposed updated method is based on the methodology used by IRIS, the Tier 1 toxicity source.
- There is only 1 constituent (MTBE) that utilized the methodology at Appendix A(b)(3)-(c) for developing an RfD.

Proposed Updates to Appendix A

Updates to Exposure Factors in the HTTAC calculation

(updates are proposed for a more sensitive receptor population – children)

Current Exposure Factors	Proposed Exposure Factors
Body Weight (BW) = 70 kg (equivalent for an average adult)	Body Weight (BW) = 15 kg (equivalent for a child 0 – 6 years) of age
Daily Water Ingestion Rate (W) = 2 L/day (equivalent for an average adult)	Daily Water Ingestion Rate (W) = 0.78 L/day (equivalent for a child 0 – 6 years of age)

Proposed Updates to Appendix A

Updates to HNTAC Calculation

(moved from 35 Ill. Adm. Code 620.605(b) to Appendix A)

HNTAC calculation for carcinogens is based on methodology found in U.S. EPA's Risk Assessment Guidance for Superfund (RAGs), Part B.

Supplemental Guidance from U.S. EPA updates the carcinogen calculation to account for age-adjusted daily water ingestion rates, as opposed to adult only water ingestion rates currently used in the calculation.

Supplemental Guidance also applies adjustment factors to the age-adjusted daily water ingestion rates for to account for toxicokinetic differences between children of various age groups and adults for carcinogens with a mutagenic mode of action for carcinogenesis.

Updated equations used by U.S. EPA Regional Screening Level (RSL).

Proposed Updates to Appendix A

Updates to HNTAC Calculation

Illinois EPA proposes to update the HNTAC calculation by incorporating updated guidance to adjust for childhood exposures to carcinogens. This includes:

- Updating the HNTAC carcinogen calculation, including updating exposure factors.
- Adding a HNTAC mutagen calculation for carcinogen constituents which operate by a mutagenic mode of action for carcinogenesis. 11 constituents are classified as mutagens; 6 rely on the HNTAC calculation to determine Class I GQS.

Proposed Updates to Appendix A

Current HNTAC Calculation

$$HNTAC(mg/L) = \frac{TR \bullet BW \bullet AT \bullet 365 \frac{days}{year}}{SF_o \bullet IR \bullet EF \bullet ED}$$

Where:

Symbol (units)	Parameter	Existing Value
TR (unitless)	Target Cancer Risk – 1 in 1 Million Risk	1.0E-06
BW (kg)	Body Weight	70
AT (years)	Averaging Time for Carcinogens	70
SF_o $((mg/kg\text{-}day)^{-1})$	Oral Slope Factor – Toxicological Value	Chemical-Specific
IR (L/day)	Daily Water Ingestion Rate	2
EF (days/year)	Exposure Frequency	350
ED (year)	Exposure Duration	30

Proposed Updates to Appendix A

Proposed Updated HNTAC Calculation

$$HNTAC\ (mg/L) = \frac{TR \bullet \left(AT \bullet 365\,\frac{days}{year}\right)}{SF_o \bullet IFW_{adj}}$$

Where:

Symbol (units)	**Parameter**	**Proposed Value**
TR (unitless)	Target Cancer Risk – 1 in 1 million	1.0E-06
AT (years)	Averaging Time for Carcinogens	70
SF_o $((mg/kg\text{-}day)^{-1})$	Oral Slope Factor – Toxicological Value	Chemical-Specific
IFW_{adj} (L/kg)	Age-Adjusted Daily Water Ingestion Rate	327.95

Proposed Updates to Appendix A

IFW_{adj} Calculation

$$IFW_{adj}(327.95\ L/kg) = \left[\left(\frac{EF_{child} \bullet ED_{child} \bullet IRW_{child}}{BW_{child}}\right) + \left(\frac{EF_{adult} \bullet ED_{adult} \bullet IRW_{adult}}{BW_{adult}}\right)\right]$$

Where:

Symbol (units)	**Parameter**	**Value**
EF all (days/year)	Exposure Frequency	350
ED_{child} (years)	Exposure Duration – child (0 – 6 years)	6
IRW_{child} (L/day)	Daily Water Ingestion Rate – child	0.78
BW_{child} (kg)	Body Weight – child	15
ED_{adult} (year)	Exposure Duration – adult	20
IRW_{adult} (L/day)	Daily Water Ingestion Rate – adult	2.5
BW_{adult} (kg)	Body Weight – adult	80

Proposed Updates to Appendix A

Proposed Introduction of an HNTAC Calculation for Mutagens

$$HNTAC_{MUT}\ (mg/L) = \frac{TR \bullet \left(AT \bullet 365\,\frac{days}{year}\right)}{SF_o \bullet IFWM_{adj}}$$

Where:

Symbol (units)	**Parameter**	**Value**
TR (unitless)	Target Cancer Risk – 1 in 1 million	1.0E-06
AT (years)	Averaging Time for Carcinogens	70
SF_o $((mg/kg\text{-}day)^{-1})$	Oral Slope Factor – Toxicological Value	Chemical-Specific
$IFWM_{adj}$ (L/kg)	Age-Adjusted Daily Water Ingestion Rate for Mutagens	1,019.9

Proposed Updates to Appendix A

$IFWM_{adj}$ Calculation

$$IFWM_{adj}\,(1019.9\,L/kg) = \left[\left(\frac{EF_{0-2} \bullet ED_{0-2} \bullet IRW_{0-2} \bullet 10}{BW_{0-2}}\right) + \left(\frac{EF_{2-6} \bullet ED_{2-6} \bullet IRW_{2-6} \bullet 3}{BW_{2-6}}\right) + \left(\frac{EF_{6-16} \bullet ED_{6-16} \bullet IRW_{6-16} \bullet 3}{BW_{6-16}}\right) + \left(\frac{EF_{16-26} \bullet ED_{16-26} \bullet IRW_{16-26} \bullet 1}{BW_{16-26}}\right)\right]$$

Adjustment Factors of 10, 3 and 1 are used to account for different risks from exposure during different life stages.

Proposed Updates to Appendix A

$IFWM_{adj}$ Calculation

$IFWM_{adj}$ Parameter Values:

Symbol	**Parameter**	**Proposed Value**
EF - all (days/year)	Exposure Frequency	350
ED_{0-2} (years)	Exposure Duration: 0-2 years of age	2
ED_{2-6} (years)	Exposure Duration: 2-6 years of age	4
ED_{6-16}, ED_{16-26} (years)	Exposure Duration: 6-16 and 16-26 years of age	10
IRW_{0-2}, IRW_{2-6} (L/day)	Daily Water Ingestion Rate: 0-2 and 2-6 years of age	0.78
IRW_{6-16}, IRW_{16-26} (L/day)	Daily Water Ingestion Rate: 6–16 and 16-26 years of age	2.5
BW_{0-2}, BW_{2-6} (kg)	Body Weight: 0-2 and 2-6 years of age	15
BW_{6-16}, BW_{16-26} (kg)	Body Weight: 6-16 and 16-26 years of age	80

Updates to Class II: General Resource Groundwater Quality Standards (Section 620.420)

In addition to the new constituents, updated Class II GQS are proposed for 74 constituents or mixtures currently listed in Section 620.420. Proposed updated standards are based on the following factors:

- Updated Class I Groundwater Quality Standards
- Irrigation or Livestock Criteria
- Updated Treatment Factors

Updated Treatment Factors

Treatment Factors are applied based on the effectiveness to treat the constituent in the groundwater at an 80% removal efficiency rate:

- For removal via air stripping, an 80% removal efficiency rate is assumed for constituents having a Dimensionless Henry's Law Constant (H') value greater than methylene chloride's (H') value of 0.111 at a 20 ^{0}C Groundwater System Temperature.

OR

- For removal via carbon adsorption, an 80% removal efficiency rate is assumed for constituents having an Organic Carbon Partition Coefficient (K_{oc}) value greater than ethylbenzene's (K_{oc}) value of 446 L/kg.

If a constituent's chemical/physical values meet either of the criteria, a Treatment Factor of 5 is applied to the Class I Groundwater Quality Standard to calculate a Class II Groundwater Quality Standard.

- Source of Chemical/Physical Values: U.S. EPA Regional Screening Levels
- Source of Treatment Factor Criteria: Illinois Pollution Control Board R08-18

Addition of Tables at Appendix E for Similar-Acting Chemicals

Code	35 Ill. Adm. Code 620, Appendix B and Appendix C provide procedures for mixtures of similar-acting substances within the groundwater.
Table	Table A lists similar-acting constituents based on noncarcinogenic health effects or target organs.
Table	Table B lists similar-acting constituents based on cancer effects.

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35 Ill. Adm. Code 620; Groundwater Quality
Pre-Filing Public Comment Period
Factsheet and Overview of Proposed Changes

Draft Proposed Rules

The Illinois EPA is proposing draft language to update 35 Ill. Adm. Code 620. The proposed updates include nine new chemicals, three new atrazine metabolites, and procedures for selecting toxicity values consistent with current federal guidance. Definitions are updated and references are consistent with those criteria and practices as incorporated. Site specific groundwater standards for designated Class III Special Resource Groundwater are also added. Exposure factors are updated, and the Human Non-Threshold Toxicant Advisory Concentration model is updated. Tables for similar-acting constituents are added. Finally, this proposal includes groundwater quality standards for five Per- and Polyfluoroalkyl Substances (PFAS): perfluorooctanoic acid (PFOA), perfluorooctanesulfonic acid (PFOS), perfluorononanoic acid (PFNA), perfluorohexanesulfonic acid (PFHxS), and perfluorobutanesulfonic acid (PFBS).

A summary of the key provisions is below. More information concerning the draft proposed rule may be found at
https://www2.illinois.gov/epa/about-us/rules-regs/water/Pages/620-Groundwater-Quality.aspx

Public Comment

Prior to submitting proposed rules to the Illinois Pollution Control Board for review and final adoption, the Illinois EPA is entertaining public comment on draft proposed rules. The Illinois EPA will accept written public comment until **June 25, 2021**. Comments should be submitted to **EPA.620.rulemaking@illinois.gov**

All comments, including proposed alternative language, received by Illinois EPA will be considered prior to the Agency filing the proposed rule with Illinois Pollution Control Board. Questions about the process or rulemaking should be submitted to the e-mail address above.

Public Meeting

The Illinois EPA will host a virtual public meeting to review the proposed changes and answer questions concerning the proposal. **The meeting will be held at 1:00 pm on May 26, 2021.**
The meeting link is:
https://illinois.webex.com/illinois/j.php?MTID=m19e9dc943bb9f835453fc6b6e8823826
Computer and telephone connection instructions are provided at the bottom of this Notice. If you have questions about connecting to the meeting, contact Jeff Guy at (217) 785-8724 or by submitting an e-mail to **EPA.620.rulemaking@illinois.gov.**

Key Provisions

1. Updates the methodology located in Appendix A for developing oral reference doses (RfDs), when a verified RfD is not available. The updated methodology is the method used by U.S. EPA Integrated Risk Information System (IRIS), the Tier 1 source for selecting toxicity criteria.
2. Provides the hierarchy for selecting a verified RfD from various sources. The hierarchy is in Appendix A.
3. Updates the Exposure Factors used in the Human Threshold Toxicant Advisory Concentration (HTTAC) equation and the Human Non-Threshold Toxicant Advisory Concentration (HNTAC) equations for both carcinogens and mutagens to be consistent with the U.S. EPA Exposure Factors Handbook (2011) and U.S. EPA Regional Screening Level calculator. Updates the exposure population from an average adult to a child ages 0-6 years for the HTTAC equation.
4. Updates Class I groundwater quality standards in tables at Part 620.410, based on updates to toxicity values, exposure factors and other methodologies.
5. Updates Class II groundwater quality standards in tables at Part 620.420, based on updates to Class I groundwater quality standards and updates to treatment factors, based on updates to dimensionless Henry's Law Constants when calculated at 20 ^{o}C and organic carbon partition coefficients.
6. Establishes groundwater quality standards for nine new chemicals, adds three metabolites as a mixture to atrazine, and moves atrazine and its metabolites tables to Part 620.410(c)(2) and Part 620.420(c)(2) for complex mixtures. Combines Radium 226 and 228 to form CASRN 7440-14-4: Radium (combined 226+228), updates the Class I groundwater quality standard for radium (combined 226+228) to an updated standard of 5 pCi/L, equal to the U.S. EPA Drinking Water MCL, and adds a Class II groundwater quality standard for radium (combined 226+228) at Part 620.420(a)(2). Establishes a Class II groundwater quality standard for silver and adds it to the table at Part 620.420(a)(2).
7. Updates constituent tables to include Chemical Abstract Services Registry Numbers (CASRNs) as additional identifiers for the constituents.
8. Adds footnotes to tables identifying the sources or methods for determining the groundwater quality standards.
9. Removes the explosive constituents at Parts 620.410(c) and 620.420(c); integrates the constituents into Parts 620.410(b) and 620.420(b).
10. Adds Appendix E, providing tables for similar-acting non-carcinogenic constituents by health effect (Table A) and similar-acting carcinogen constituents by cancer effect (Table B).
11. Updates the names of eleven constituents.
12. Adds carcinogen designations for four existing chemicals and one new chemical.
13. Adds mutagen designations for eleven chemicals.
14. Updates toxicity values for the constituents whose groundwater quality standards are based on the Human Threshold Toxicant Advisory Concentration (HTTAC) equation for noncarcinogens or the Human Nonthreshold Toxicant Advisory Concentration (HNTAC) equation for carcinogens.

A detailed list of Key Provisions can be found at
https://www2.illinois.gov/epa/about-us/rules-regs/water/Pages/620-Groundwater-Quality.aspx

Proposed Changes to 620 Sub Part A-C

Sub Part	Section	Proposed Changes
Part A	**620.110**	Adds definition of "Chemical Abstract Service Registry Numbers (CASRN)", "Lowest Concentration Minimum Reporting Level", and "Mutagen". Updates definition of "Carcinogen" to be consistent with updates to terminology used by U.S. EPA Integrated Risk Information System, and definition of "Detection" to language currently used in test methods. Removes the definition of "Practical Quantitation Level".
	620.125	Updates CFR references to most recent iteration of the code. Adds Illinois EPA "Integrated Water Quality Report and Section 303(d) List" and National Academy of Science "Water Quality Criteria" (1973) to incorporated references and updates several test methods. Adds references from the U.S. EPA Office of Research and Development, National Center for Environmental Assessment, and reference from U.S. EPA Office of Resource Conservation and Recovery. Updated for groundwater guidance from USEPA 2017.
Part B	**620.210**	Removes permeameter as an acceptable means to determine hydraulic conductivity. Adds the wellhead protection area of a community water supply well or well field as a specific area to which Class I groundwater quality standards are applicable.
	620.250	Lists a standard set of documentation that must be included with all groundwater management zone applications.
Part C	**620.302**	Adds to the list of examples of persons who do groundwater monitoring.
	620.310	Updates table at Part 620.310(a)(3)(A)(i) to include CASRN for each constituent; and removes para-dichlorobenzene and ethylbenzene from the table due to their updated carcinogen classification and the Board Note for 620.310(a)(3)(A). Adds a table at Part 620.310(a)(3)(A)(ii) depicting the constituents in the subsection; and removes *gamma*-HCH (*gamma*-hexachlorocyclohexane, lindane) and isopropylbenzene (cumene) due to their updated carcinogen classification and the Board Note for 620.310(a)(3)(A). Amends Board Note for 620.310(a)(3)(A) to revised outdated language.

Proposed Changes to 620 Sub Part D-F

Sub Part	Section	Proposed Changes
Part D	**620.410**	Adds Class I groundwater quality standards for nine new chemicals. Updates constituent tables to add CASRN for each constituent. Adds footnotes detailing the sources of the standards. Updates Class I groundwater quality standards as applicable. Removes explosive constituents table at 620.410(c) and integrates the constituents into table at 620.410(b). Moves atrazine from 620.410(b) to the complex chemical mixtures tables at 620.410(c) with the addition of atrazine metabolites.
	620.420	Adds Class II groundwater quality standards for nine new chemicals and two chemicals listed in 620.410 without prior Class II groundwater quality standards. Updates constituent tables to add a CASRN for each constituent, and update Class II groundwater quality standards as applicable. Adds footnotes detailing the sources of the standards. Removes explosive constituents table at 620.420(c) and integrates the constituents into table at 620.420(b). Moves atrazine from 620.420(b) to the complex chemical mixtures tables at 620.420(c) with the addition of atrazine metabolites.
	620.430	Establishes site specific Class III groundwater quality standards for chloride and pH at four dedicated nature preserves, which are caves, pursuant to 620.230(b). Establishes site specific Class III groundwater quality standards for chloride at two dedicated nature preserves, which are wetlands, pursuant to 620.230(b).
	620.440	Updates names of explosive constituents.
	620.450	Updates names of explosive constituents.
Part E	**620.510**	Requires that the 2009 Unified Guidance be used to determine background groundwater quality unless other methods are specified by regulation. Replaces the use of the PQL with the LLOQ, LCMRL or MDL, as appropriate to the nature of the chemical.
Part F	**620.601**	(b)-Updates code reference to 604.200.
	620.605	(b)(1) Designates the more stringent toxicity value of the (Human Threshold Toxicant Advisory Concentration (HTTAC) or Human Nonthreshold Toxicant Advisory Concentration (HNTAC) as the guidance value in the absence of a Maximum Contaminant Level (MCL) or Maximum Contaminant Level Goal (MCLG).
		(b)(2) Removes the Human Nonthreshold Toxicant Advisory Concentration (HNTAC) language and equation and relocates it to Appendix A.

Proposed Changes to 620 Appendices

Appendix	Section	Proposed Changes
A	**(a)**	Updates exposure factors representative of a child for the HTTAC model, which is consistent with Illinois Administrative Code Part 742 and U.S. EPA Regional Screening Levels (per capita daily water consumption = 0.78 liters per day, assumed average weight of a child 0-6 years of age = 15 kg).
	(b)(2)	Incorporates U.S. EPA's hierarchy of toxicity sources from "*Tier 3 Toxicity Value White Paper*", dated May 16, 2013, by U.S. EPA Office of Solid Waste and Emergency Response Human Health Regional Risk Assessors Forum (OSWER) for determining an appropriate verified oral reference dose.
	(b)(3)	Revises methodology used to calculate guidance values when a verified oral reference dose is not available to make language consistent with U.S. EPA Reference Dose Guidance.
	(b)(4)	Clarifies usage of uncertainty factors.
	(c)(1)	Adds equation for calculating HNTAC guidance level for chemicals designated as mutagens.
	(c)(2)	Updates equation for calculating HNTAC guidance levels for chemicals designated as carcinogens that are not designated as mutagens.
B	**(c)**	Removes language specific to mixtures of ortho-dichlorobenzene and para-dichlorobenzene, and 1,1-dichloroethane and 1,1,1-trichloroethane, and adds reference to Appendix E.
E		Provides tables of similar acting non-carcinogenic and carcinogenic constituents.

Proposed Changes to 35 Ill. Adm. Code Part 620

May 2021

Section 620.110 Definitions

- Definitions have been added to reflect updated terminology
- Delete obsolete terms

Section 620.125 Incorporations by Reference

- Update reference to USEPA documents
- New and updated analytical methods
- Update sample collection procedures

Section 620.210 Class I: Potable Resource Groundwater

- Added delineated wellhead protection areas as Class I groundwater areas
- Eliminated permeameters as a method to determine hydraulic conductivity for groundwater classification

Section 620.250 Groundwater Management Zone

- Added a list of information that must be provided with a GMZ application

Section 620.302 Applicability of Preventive Notice and Preventive Response Activities

- Added additional examples of programs conduction groundwater monitoring

Section 620.310 Preventive Response Activities

- Tabulated lists of chemicals
- Added chemical abstract numbers for reference
- Eliminated chemicals which are now considered carcinogens
- Added proposed chemicals to which Preventive Response will apply
- Replaced outdated analytical references with updated references

Section 620.410 Groundwater Quality Standards for Class I: Potable Resource Groundwater

- Tabulated lists of chemicals
- Added chemical abstract numbers for reference
- Added proposed chemicals
- Updated numerical groundwater standards to reflect MCLs
- Update numerical groundwater standards withd the proposed criteria for establishing health-based concentrations (Carol Hawbaker will discuss these proposed changes further)
- Added footnotes describing the origin of the numerical groundwater standard

Section 620.420 Groundwater Quality Standards for Class II: General Resource Groundwater

- Tabulated lists of chemicals
- Added chemical abstract numbers for reference
- Added proposed chemicals
- Updated numerical groundwater standards to reflect updated treatment efficiencies
- Added footnotes describing the origin of the numerical groundwater standard

Section 620.430 Groundwater Quality Standards for Class III: Special Resource Groundwater

- Site specific standards for chloride and pH within the designated Class III Groundwater areas of four Dedicated Nature Preserves that are cave systems

- Site specific standards for chloride within the designated Class III Groundwater areas of two Dedicated Nature Preserves that are wetlands

Section 620.440 Groundwater Quality Standards for Class IV: Other Groundwater

- Updated names of previously regulated chemicals

Section 620.450 Alternative Groundwater Quality Standards

- Updated names of previously regulated chemicals

Section 620.510 Monitoring and Analytical Requirements

- Simplify citation to Section 620.125
- Add new subsection for statistical methods document contained in Section 620.125
- Update analytical method references

Section 620.601 Purpose of a Health Advisory

- Update citation to applicable regulations

Section 620.605 Issuance of a Health Advisory

- Update references to guidance
- Update analytical method references

Illinois EPA Moderator Opening Statements

Public Meeting on 35 Ill. Adm. Code 620 – Groundwater Quality

Good afternoon and welcome to today's meeting; we appreciate your attendance today. My name is Jeff Guy and I will be moderating today's meeting. As the moderator, I intend to treat everyone in a respectful manner, and I ask that Agency staff and the public please do the same. If you have connection or audio issues, please attempt to reconnect. Also, please keep your lines muted at this time.

The Illinois EPA is proposing to update 35 Illinois Administrative Code Part 620: Groundwater Quality. These regulations are the state standards that set acceptable levels for various pollutants in groundwater. Prior to submitting proposed rules to the Illinois Pollution Control Board for review and final adoption, the Illinois EPA is accepting written public comments on the proposed rules: The Illinois EPA will accept written public comments until June 25, 2021; please submit your comments to EPA.620.rulemaking@illinois.gov. We will take all comments into consideration before filing with the Illinois Pollution Control Board. This email address and other pertinent information regarding the draft proposed rules can be found on the Agency's general public notice webpage. The general notice webpage includes the following: 'Notice of Comment Period and Public Meeting', 'Factsheet and Overview of Proposed Changes', and the Agency's slideshow presentations that will be shown in a few minutes.

The purpose of today's meeting is to give an overview of the proposed changes to the 620 regulations and to answer questions you may have related to the proposed changes. The Agency's panel today consists of myself, Lynn Dunaway (in the Bureau of Water), Michael Brown (in the Bureau of Water), Carol Hawbaker (in the Office of Toxicity Assessment), Kyle Rominger (in the Bureau of Land), Greg Dunn (in the Bureau of Land), and Sara Terranova (in the Division of Legal Counsel). Additional Agency staff present include Brad Frost and Sabrina Bailey in the Office of Community Relations. The agenda for today consists of the Agency's opening remarks, followed by an overview of proposed changes presented by

Lynn Dunaway and Carol Hawbaker. Then we will answer questions related to the proposed rule changes, as part of the question and answer session.

At this time, Agency staff will present an overview of the proposed changes. This will be followed by additional instructions from me on how we will be taking questions during the Q&A session. First, we have Lynn Dunaway, followed by Carol Hawbaker.

Overview of Proposed Changes

Thank you, Mr. Dunaway. Next, we have Ms. Carol Hawbaker.

Logistics for Q&A

Now I will cover the logistics for the Q&A portion of today's meeting. You have the opportunity to ask questions in two ways: You can use the Webex chat feature on the computer by clicking the speech balloon icon and typing your question into the box. Please include your name and affiliation (if any). Agency staff will read questions that are submitted through the chat feature.

Or you can ask a question using the "raise hand" feature. If you are connected by computer and want to ask a question, click the "raise hand" icon next to your name. If you called in to today's meeting and want to ask a question, hit *3 to raise your hand. Please wait to speak until I call on you. When it is your turn to speak, please ensure to unmute your line and provide your name and affiliation (if any). If you called in today, you can mute and unmute your line by using *6. On the computer, click the microphone icon next to your name to mute and unmute your line. The Q&A session will now begin.

That concludes our public meeting. Thank you for your participation today. Again, the Illinois EPA will accept written public comments until June 25, 2021; please submit your comments to EPA.620.rulemaking@illinois.gov. We take all comments into consideration before filing with the Illinois Pollution Control Board. Thank you.

Notes:

Example: Non-chat question (hand signal: No. 1)
Me: Brad, who is the first speaker.
Brad: It is phone number starting with area code _____
Me: Ok, whoever has phone number _____ please unmute your line and proceed with your question.

Example: Chat question (hand signal: No. 2)
Me: At this time, we will read one of our chat questions, Sabrina?

Other: “Please submit your comments in writing. Today, we are only accepting questions.”

NOTE: Complete a write-up for Heather afterwards

Illinois Environmental Protection Agency
Notice of Comment Period and Public Meeting
35 Ill. Adm. Code 620; Groundwater Quality

The Illinois EPA is proposing to update 35 Ill. Adm. Code 620: Groundwater Quality. The rules are the state standards that set acceptable levels for various pollutants in groundwater. Prior to submitting proposed rules to the Illinois Pollution Control Board for review and final adoption, the Illinois EPA is soliciting public comment on draft proposed rules.

The Illinois EPA will accept written public comment until **June 25, 2021**. Comments should be submitted to **EPA.620.rulemaking@illinois.gov.** All comments, including proposed alternative language, received by Illinois EPA will be considered prior to the Agency filing the proposed rule with Illinois Pollution Control Board. Questions about the process or rulemaking should be submitted to the e-mail address above.

The Illinois EPA will host a virtual public meeting to review the proposed changes and answer questions concerning the proposal. **The meeting will be held at 1:00 pm on May 26, 2021.**
The meeting link is:
https://illinois.webex.com/illinois/j.php?MTID=m19e9dc943bb9f835453fc6b6e8823826
Computer and telephone connection instructions are provided at the bottom of this Notice. If you have questions about connecting to the meeting, contact Jeff Guy at (217) 785-8724 or by submitting an e-mail to **EPA.620.rulemaking@illinois.gov.**

The proposed updates include nine new chemicals, three new atrazine metabolites, and procedures for selecting toxicity values consistent with current federal guidance. Definitions are updated and references are consistent with those criteria and practices as incorporated. Site specific groundwater standards for designated Class III Special Resource Groundwater are also added. Exposure factors are updated, and the Human Non-Threshold Toxicant Advisory Concentration model is updated. Tables for similar-acting constituents are added. Finally, this proposal includes groundwater quality standards for five Per- and Polyfluoroalkyl Substances (PFAS): perfluorooctanoic acid (PFOA), perfluorooctanesulfonic acid (PFOS), perfluorononanoic acid (PFNA), perfluorohexanesulfonic acid (PFHxS), and perfluorobutanesulfonic acid (PFBS).

More information concerning the draft proposed rule may be found at
https://www2.illinois.gov/epa/about-us/rules-regs/water/Pages/620-Groundwater-Quality.aspx

Meeting Connection Instructions

Cisco Webex Meeting Information
Date: Wednesday, May 26, 2021
Time: 1:00 p.m. CT
Meeting Number: 177 758 5798
Meeting Password: E2TePWPcg25

Connect by Computer

1. Select this link, which will direct you to the Webex webpage for the meeting:

https://illinois.webex.com/illinois/j.php?MTID=m19e9dc943bb9f835453fc6b6e8823826

2. Enter your information (name and address) and select "Join Now". You may be prompted for a Meeting Number or Meeting Password, above.

3. An audio connection is required. The best connection option is "Call Me" (from the "Select Audio Connection" drop down, select "Call Me"). Input or select your telephone number.

Connect by Dial-in Phone

1. Call +1-312-535-8110

2. You will be prompted to enter the access code or meeting number. Enter the Meeting Number, above, and select the # sign.

Tips

- Find a quiet location with a power source for your device.
- Close all background applications or browser sessions.
- Reduce distractions and practice good meeting etiquette.
- Non-smartphone cellular (mobile) phones or landlines provide an audio-only experience.
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- Reduce distractions and practice good meeting etiquette.
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- Smartphone, iPad or Tablets use the Webex mobile application.

From: Frost, Brad
To: Guy, Jeff; Zeivel, Christine
Subject: RE: Equip update
Date: Wednesday, September 23, 2020 12:48:55 PM
Attachments: image001.png

Thanks Jeff, appreciate it

From: Guy, Jeff <Jeff.Guy@Illinois.gov>
Sent: Wednesday, September 23, 2020 11:54 AM
To: Zeivel, Christine <Christine.Zeivel@illinois.gov>
Cc: Frost, Brad <Brad.Frost@Illinois.gov>
Subject: Equip update

Christine,

I had an opportunity to use the BOL equipment this morning. I set up a Webex meeting with Carol H. and the video and audio worked fine. The mobile system includes a hard drive, keyboard, monitor, mouse and external webcam that has a built in microphone. You simply plug in a few power chords, plug in internet ethernet cable, log in, enter the meeting, and adjust video/audio settings. Log in runs slow, especially the first time. Plan on 10-15 minutes – when you do the actual hearing. For the test run on Monday, I will just log in my account.

Jeffrey J. Guy
Illinois EPA
Office of Community Relations
(217) 785-8724
Jeff.Guy@illinois.gov

Illinois Environmental Protection Agency
Proposed Updates to 35 Ill. Adm. Code 620

Written comments must be received by the Illinois EPA by **June 25, 2021**.

Comments must be submitted to **EPA.620.rulemaking@illinois.gov.**

Illinois Environmental Protection Agency

Agenda

- **Opening Remarks**
- **Overview of Changes**
  - Bureau of Water-Lynn Dunaway
  - Associate Director's Office (Toxicology)-Carol Hawbaker
- **Q&A with Panelist**
  - Department of Legal Counsel- Sara Terranova
  - Bureau of Land-Greg Dunn
  - Bureau of Water- Michael Brown
  - Bureau of Water-Lynn Dunaway
  - Associate Director's Office (Toxicology)-Carol Hawbaker
- **Closing Remarks**

Illinois Environmental Protection Agency

Illinois Environmental Protection Agency
Proposed Updates to 35 Ill. Adm. Code 620

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Illinois Environmental Protection Agency

Agenda

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- **Overview of Changes**
  - Bureau of Water-Lynn Dunaway
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  - Associate Director's Office (Toxicology)-Carol Hawbaker
- **Closing Remarks**

Illinois Environmental Protection Agency

Proposed Changes to 35 Ill. Adm. Code Part 620

May 2021

Illinois Environmental Protection Agency

Agenda

- **Opening Remarks**
- **Overview of Changes**
  - Bureau of Water-Lynn Dunaway
  - Associate Director's Office (Toxicology)-Carol Hawbaker
- **Q&A with Panelist**
  - Department of Legal Counsel- Sara Terranova
  - Bureau of Land-Greg Dunn
  - Bureau of Water-Lynn Dunaway
  - Associate Director's Office (Toxicology)-Carol Hawbaker
- **Closing Remarks**

Illinois Environmental Protection Agency

Proposed Changes Subparts A and B

Section 620.110 Definitions

- Definitions have been added to reflect updated terminology
- Delete obsolete terms

Section 620.125 Incorporations by Reference

- Update reference to USEPA documents
- New and updated analytical methods
- Update sample collection procedures

Section 620.210 Class I: Potable Resource Groundwater

- Added delineated wellhead protection areas as Class I groundwater areas
- Eliminated permeameters as a method to determine hydraulic conductivity for groundwater classification

Section 620.250 Groundwater Management Zone

- Added a list of information that must be provided with a GMZ application

Illinois Environmental Protection Agency

Proposed Changes Subpart C

Section 620.302 Applicability of Preventive Notice and Preventive Response Activities

- Added additional examples of programs conducting groundwater monitoring

Section 620.310 Preventive Response Activities

- Tabulated lists of chemicals
- Added chemical abstract numbers for reference
- Eliminated chemicals which are now considered carcinogens
- Added proposed chemicals to which Preventive Response will apply
- Replaced outdated analytical references with updated references

Illinois Environmental Protection Agency

Proposed Changes Subpart D

Section 620.410 Groundwater Quality Standards for Class I: Potable Resource Groundwater

- Tabulated lists of chemicals
- Added chemical abstract numbers for reference
- Added proposed chemicals
- Updated numerical groundwater standards to reflect MCLs
- Update numerical groundwater standards with the proposed criteria for establishing health-based concentrations (Carol Hawbaker will discuss these proposed changes further)
- Added footnotes describing the origin of the numerical groundwater standard

Section 620.420 Groundwater Quality Standards for Class II: General Resource Groundwater

- Tabulated lists of chemicals
- Added chemical abstract numbers for reference
- Added proposed chemicals
- Updated numerical groundwater standards to reflect updated treatment efficiencies
- Added footnotes describing the origin of the numerical groundwater standard

Proposed Changes Subpart D

Section 620.430 Groundwater Quality Standards for Class III: Special Resource Groundwater

- Site-specific standards for chloride and pH within the designated Class III Groundwater areas of four Dedicated Nature Preserves that are cave systems

- Site-specific standards for chloride within the designated Class III Groundwater areas of two Dedicated Nature Preserves that are wetlands

Section 620.440 Groundwater Quality Standards for Class IV: Other Groundwater

- Updated names of previously regulated chemicals

Section 620.450 Alternative Groundwater Quality Standards

- Updated names of previously regulated chemicals

Illinois Environmental Protection Agency

Proposed Changes Subparts E and F

Section 620.510 Monitoring and Analytical Requirements

- Simplify citation to Section 620.125
- Add new subsection for statistical methods document contained in Section 620.125
- Update analytical method references

Section 620.601 Purpose of a Health Advisory

- Update citation to applicable regulations

Section 620.605 Issuance of a Health Advisory

- Update references to guidance
- Update analytical method references

Illinois Environmental Protection Agency

Written comments must be received by the Illinois EPA by **June 25, 2021**.

Comments must be submitted to **EPA.620.rulemaking@illinois.gov.**

Thank You For Your Participation!

Illinois Environmental Protection Agency

Illinois Environmental Protection Agency Proposed Updates to 35 Ill. Adm. Code 620

By: Carol Hawbaker
Environmental Risk Assessor
Illinois Environmental Protection Agency
Office of Toxicity Assessment

May 26, 2021

Proposed Updates to 35 Ill. Adm. Code 620

The presentation will cover the following topics:

- Introduction of nine new constituents.
- Addition of three metabolites to be evaluated with atrazine for compliance with groundwater quality standards (GQS).
- Combination of radium 226 and radium 228 to form a new constituent: radium (combined 226+228).
- Addition of carcinogen designations for four existing constituents.
- Updates to constituents in the tables at Section 620.310(a)(3)(A)(i) and (ii).

Proposed Updates to 35 Ill. Adm. Code 620

The presentation will cover the following topics (continued):

- Updates of Class I GQS for three inorganic constituents from MCLs to irrigation/livestock water quality standards, based on beneficial use of groundwater.
- For constituents which Class I GQS are based on procedures found in Section 620, Subpart F and Appendix A:
  - Updates to toxicity values and relative source contribution (RSC) values;
  - Updates to exposure factors;
  - Addition of a mutagenic method for the development of a carcinogen Class I GQS for constituents with a mutagenic mode of action.

Proposed Updates to 35 Ill. Adm. Code 620

The presentation will cover the following topics (continued):

- Updates to Class II GQS.
- Introduction of tables (Appendix E) listing constituents that have similar-acting health effects or affect the same target organ.

Introduction of New Constituents

- Aluminum
- Lithium
- 1-Methylnaphthalene
- Molybdenum

- Five Per-and Polyfluoroalkyl Substances (PFAS):
  - PFBS (Perfluorobutanesulfonic Acid)
  - PFHxS (Perfluorohexanesulfonic Acid)
  - PFNA (Perfluorononanoic Acid)
  - PFOA (Perfluorooctanoic Acid)
  - PFOS (Perfluorooctanesulfonic Acid)

Introduction of New Constituents

Proposed Class I and Class II GQS:

CASRN	Constituent	Proposed Class I GQS (mg/L)	Class I Source	Proposed Class II GQS (mg/L)	Class II Source
7429-90-5	Aluminum	1.9	Subpart F	5	Livestock
7439-93-2	Lithium	0.01	Subpart F	2.5	Irrigation
90-12-0	1-Methylnaphthalene	0.27	Subpart F	0.27	Subpart F
7439-98-7	Molybdenum	0.019	Subpart F	0.05	Irrigation

Per and Poly-Fluoroalkyl Substances (PFAS)

PFAS are a group of human-made constituents applied to many consumers products to make them waterproof, stain resistant or non-stick.

- Food packaging - fast food containers, lunch meat paper, disposable plates and bowls, and oil-, water- and grease-resistant coatings on food packaging (pizza boxes);
- Commercial household products - non-stick coated cookware (Teflon), cleaning products, waxes, polishes, and adhesives;
- Clothing and fabric textiles - stain- and water-resistant carpeting and upholstery, water repellant clothing, tents, umbrellas, shoes, and leather goods;
- Cosmetics and personal care products - shampoos, conditioners, sunscreens, cosmetics, and dental floss;
- Building and exterior use products - paints and sealants;
- Industrial use - metal plating and finishing, wire coatings, automotive fluids, and the manufacture of artificial turf;
- Firefighting foam - aqueous film-forming foam (AFFF).

Per and Poly-Fluoroalkyl Substances (PFAS)

PFAS are constituents of emerging concern:

- "Forever Chemicals": PFAS not degrade naturally in the environment.
- PFAS constituents have an affinity for water and can migrate long distances.
- PFAS can bioaccumulate in plants, fish and wildlife, and humans.
- PFAS are a group of chemicals consisting of over 5,000 substances.
- Toxicological studies and assessments are being conducted by several agencies.
- Limited toxicological data for most PFAS.

Per and Poly-Fluoroalkyl Substances (PFAS)

Epidemiology and Animal Studies Suggest Associations Between PFAS Exposure and Several Health Effects:

- Pregnancy-Induced Hypertension/Pre-Eclampsia
- Liver Damage
- High Cholesterol
- Thyroid Disease
- Decreased Response to Vaccines
- Decreased Fertility
- Decreased Birth Weight
- Developmental Delays

Per and Poly-Fluoroalkyl Substances (PFAS)

- PFOA meets Illinois EPA's definition of a carcinogen. The International Agency for Research on Cancer (IARC) classified PFOA as a "2B" carcinogen in 2017.
- A "2B" classification means the constituent is possibly carcinogenic to humans.
- U.S. EPA concluded there was "suggestive potential" for PFOS to be carcinogenic to humans; however, PFOS does not meet Illinois EPA's definition of a carcinogen at this time.
- Possible Cancer Links:

  -Kidney

  -Testicular

  -Prostate

  -Liver

  -Pancreas

Per and Poly-Fluoroalkyl Substances (PFAS)

Proposed Class I GQS are based on proposed procedures for 35 Ill. Adm. Code 620, Subpart F and Appendix A.

CASRN	Constituent	Class I and Class II GQS (mg/L or ppm)	Class I and Class II GQS (ng/L or ppt)	Toxicity Value	Toxicity Value Source	Relative Source Contribution Value for Noncarcinogens
375-73-5	PFBS	0.0012	1,200	3E-04	PPRTV	0.2
355-46-4	PFHxS	0.000077	77	2E-05	ATSDR	0.2
375-95-1	PFNA	0.000012	12	3E-06	ATSDR	0.2
335-67-1	PFOA	0.000002	2	1.4E+02	OEHHA	Not Applicable
1763-23-1	PFOS	0.0000077	7.7	2E-06	ATSDR	0.2

PPRTV: Provisional Peer Reviewed Toxicity Values.

ATSDR: Agency for Toxic Substance and Disease Registry.

OEHHA: California EPA Office of Environmental Health Hazard Assessments.

PFBS, PFHxS, PFNA and PFOS toxicity values are oral reference doses (RfDs) for noncarcinogen effects in units of mg/kg-day.

PFOA toxicity value is an oral slope factor (SF_o) for cancer risks in units of $(mg/kg\text{-}day)^{-1}$. The GQS is the minimum reporting level, per Subpart F.

The amendments propose the addition of 3 atrazine metabolites to be included when comparing atrazine concentrations to GQS.

Added Metabolites

-DEA (Desethyl-atrazine)

-DIA (Desisopropyl-atrazine)

-DACT (Diaminochlorotriazine)

Combination of Radium 226 and 228

Presently, radium 226 and radium 228 have individual Class I GQS. They are not listed in the Class II GQS.

The amendments propose radium (combined 226+228) Class I and Class II GQS.

The proposed value for the Class I and Class II GQS is based on the Federal maximum contaminant level (MCL) for radium (combined 226+228) of 5 picocuries per liter (pCi/L).

Proposed Updates to Carcinogen Designations

Carcinogen designations are updated for the following constituents:

p-Dichlorobenzene (1,4-dichlorobenzene)

- Classified "2B" by International Agency for Research on Cancer (IARC) - 1999

Ethylbenzene

- Classified "2B" by IARC - 2000

gamma-HCH (*gamma*-hexachlorocyclohexane, lindane)

- Classified "1" by IARC – 2018

Isopropylbenzene (cumene)

- Classified "2B" by IARC - 2013

Proposed Updates to Constituents in Tables at 35 Ill. Adm. Code 620.310(a)(3)(A)(i) and (ii) – Preventive Response Activities

The following constituents are removed from the tables due to carcinogenicity classifications, based on the Board Note at Section 620.310(a)(3)(A).

- *p*-Dichlorobenzene (1,4-dichlorobenzene)
- Ethylbenzene
- Arsenic
- *gamma*-HCH (lindane)
- Isopropylbenzene (cumene)

MCPP (mecoprop) is removed as the constituent's proposed Class I GQS is based on its lowest level of quantitation (LLOQ) or lowest concentration minimum reporting level (LCMRL), formerly termed practical quantitation limit (PQL).

Proposed Updates to Constituents in Tables at 35 Ill. Adm. Code 620.310(a)(3)(A)(i) and (ii) – Preventive Response Activities

Constituents Added to Tables

- Aluminum
- Molybdenum
- 1-Methylnaphthalene
- PFBS
- PFHxS
- PFNA
- PFOS
- Antimony
- HMX (octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine)
- Nitrobenzene
- RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine
- TNT (2,4,6-trinitrotoluene)
- 1,3,5-Trinitrobenzene

Proposed Updates of Class I GQS for Three Inorganic Constituents Based on More Stringent Irrigation or Livestock Values

Class I potable resource groundwater may also be used for irrigation and watering of livestock. The following constituents are proposed to be updated as follows:

CASRN	Constituent	Current Class I GQS (mg/L)	Current Source	Proposed Class I GQS (mg/L)	Proposed Source
7440-50-8	Copper	0.65	Lead/Copper Rule	0.5	Livestock
7681-49-4	Fluoride	4	U.S. EPA MCL	2	Livestock
7782-49-2	Selenium	0.05	U.S. EPA MCL	0.02	Irrigation

Proposed Updates to Subpart F and Appendix A

Out of 115 total constituents presently listed at 35 Ill. Adm. Code 620.410, 40 utilize the procedures in Subpart F and Appendix A to develop Class I GQS (35% of constituents):

- 30 constituents utilize the Human Threshold Toxicant Advisory Concentration (HTTAC) calculation at Appendix A(a) for noncarcinogens.

- 10 constituents utilize the Human Nonthreshold Toxicant Advisory Concentration (HNTAC) calculation at 35 Ill. Adm. Code 620.605(b)(2), for carcinogens. Of these 10, 7 constituents utilize a practical quantitation limit (PQL), because the calculated HNTAC is less than the PQL.

Proposed Updates to Appendix A

Illinois EPA's Hierarchy for Determining Toxicity Values

Basis for hierarchy is derived from U.S. EPA OSWER Directive 9285.7-53, dated December 5, 2003, and discussed in the Illinois Pollution Control Board Rulemaking R08-18: Proposed Amendments to Groundwater Quality Standards, 35 Ill. Adm. Code 620.

- Tier 1 Toxicity Value Source: Integrated Risk Information System (IRIS)
- Tier 2 Toxicity Value Source: Provisional Peer Reviewed Toxicity Values (PPRTV)
- Tier 3 Other Toxicity Values:

"Priority given to sources of information that are the most current, the basis for which is transparent and publicly available, and which has been peer-reviewed."

OSWER Directive 9285.7-53

Proposed Updates to Appendix A

Additional Guidance Regarding the Selection of Tier 3 Toxicity Values derived from U.S. EPA OSWER Directive 9285.7-86, dated May 16, 2013. Tier 3 sources are ranked as follows:

1. Agency for Toxic Substances and Disease Registry (ATSDR) minimal risk levels.
2. California EPA, Office of Environmental Health Hazard Assessment (OEHHA), toxicity values.
3. PPRTV Appendix "Screening Toxicity Values."
4. Health Effect Assessment Summary Table (HEAST) toxicity values.

Proposed Updates to Appendix A

Updates Procedures for Determining an Oral Reference Dose (RfD) When an RfD is Not Available from the Listed Toxicity Values Sources.

- Due to outdated methodology, proposes to update the procedures found at 35 Ill. Adm. Code 620, Appendix A(b)(3)-(c) for when there is no “verified” RfD, The proposed updated method is based on the methodology used by IRIS, U.S. EPA’s Tier 1 toxicity source.

- Only 1 constituent (MTBE) utilized the methodology at Appendix A(b)(3)-(c) for developing an RfD.

Proposed Updates to Appendix A

Updates to Exposure Factors in the HTTAC calculation

(updates are applied for a more sensitive receptor population – children)

Current Exposure Factors	Proposed Exposure Factors
Body Weight (BW) = 70 kg (equivalent for an average adult)	Body Weight (BW) = 15 kg (equivalent for a child 0 – 6 years) of age
Daily Water Ingestion Rate (W) = 2 L/day (equivalent for an average adult)	Daily Water Ingestion Rate (W) = 0.78 L/day (equivalent for a child 0 – 6 years of age)

Proposed Updates to Appendix A

Updates to HNTAC Calculation

(moved from 35 Ill. Adm. Code 620.605(b) to Appendix A)

HNTAC calculation for carcinogens is based on methodology found in U.S. EPA's Risk Assessment Guidance for Superfund (RAGs), Part B.

Supplemental Guidance from U.S. EPA updates the carcinogen calculation to account for age-adjusted daily water ingestion rates, as opposed to adult only water ingestion rates currently used in the calculation.

Supplemental Guidance also applies adjustment factors to the age-adjusted daily water ingestion rates for to account for toxicokinetic differences between children of various age groups and adults for carcinogens with a mutagenic mode of action for carcinogenesis.

Updated equations used to calculate U.S. EPA Regional Screening Levels (RSLs) for ingestion of tapwater.

Proposed Updates to Appendix A

Updates to HNTAC Calculation

Illinois EPA proposes to update the HNTAC calculation by incorporating updated guidance to adjust for childhood exposures to carcinogens. This includes:

- Updating the HNTAC carcinogen calculation, including updating exposure factors.

- Adding a HNTAC mutagen calculation for carcinogen constituents which operate by a mutagenic mode of action for carcinogenesis. 11 constituents are classified as mutagens; 6 rely on the HNTAC calculation to determine Class I GQS.

Proposed Updates to Appendix A

Current HNTAC Calculation

$$HNTAC(mg/L) = \frac{TR \bullet BW \bullet AT \bullet 365 \frac{days}{year}}{SF_o \bullet IR \bullet EF \bullet ED}$$

Where:

Symbol (units)	**Parameter**	**Existing Value**
TR (unitless)	Target Cancer Risk – 1 in 1 Million Risk	1.0E-06
BW (kg)	Body Weight	70
AT (years)	Averaging Time for Carcinogens	70
SF_o ((mg/kg-day)$^{-1}$)	Oral Slope Factor – Toxicological Value	Chemical-Specific
IR (L/day)	Daily Water Ingestion Rate	2
EF (days/year)	Exposure Frequency	350
ED (year)	Exposure Duration	30

Proposed Updates to Appendix A

Proposed Updated HNTAC Calculation

$$HNTAC\ (mg/L) = \frac{TR \bullet \left(AT \bullet 365\,\frac{days}{year}\right)}{SF_o \bullet IFW_{adj}}$$

Where:

Symbol (units)	**Parameter**	**Proposed Value**
TR (unitless)	Target Cancer Risk – 1 in 1 million	1.0E-06
AT (years)	Averaging Time for Carcinogens	70
SF_o $((mg/kg\text{-}day)^{-1})$	Oral Slope Factor – Toxicological Value	Chemical-Specific
IFW_{adj} (L/kg)	Age-Adjusted Daily Water Ingestion Rate	327.95

Proposed Updates to Appendix A

IFW_{adj} Calculation

$$IFW_{adj}(327.95\ L/kg) = \left[\left(\frac{EF_{child} \bullet ED_{child} \bullet IRW_{child}}{BW_{child}}\right) + \left(\frac{EF_{adult} \bullet ED_{adult} \bullet IRW_{adult}}{BW_{adult}}\right)\right]$$

Where:

Symbol (units)	**Parameter**	**Value**
EF all (days/year)	Exposure Frequency	350
ED_{child} (years)	Exposure Duration – child (0 – 6 years)	6
IRW_{child} (L/day)	Daily Water Ingestion Rate – child (0 – 6 years)	0.78
BW_{child} (kg)	Body Weight – child (0 – 6 years)	15
ED_{adult} (year)	Exposure Duration – adult	20
IRW_{adult} (L/day)	Daily Water Ingestion Rate – adult	2.5
BW_{adult} (kg)	Body Weight – adult	80

Proposed Updates to Appendix A

Proposed Introduction of an HNTAC Calculation for Mutagens

$$HNTAC_{MUT}\ (mg/L) = \frac{TR \bullet \left(AT \bullet 365\frac{days}{year}\right)}{SF_o \bullet IFWM_{adj}}$$

Where:

Symbol (units)	**Parameter**	**Value**
TR (unitless)	Target Cancer Risk – 1 in 1 million	1.0E-06
AT (years)	Averaging Time for Carcinogens	70
SF_o $((mg/kg\text{-}day)^{-1})$	Oral Slope Factor – Toxicological Value	Chemical-Specific
$IFWM_{adj}$ (L/kg)	Age-Adjusted Daily Water Ingestion Rate for Mutagens	1,019.9

Proposed Updates to Appendix A

$IFWM_{adj}$ Calculation

$$IFWM_{adj}\,(1019.9\,L/kg) = \left[\left(\frac{EF_{0-2} \bullet ED_{0-2} \bullet IRW_{0-2} \bullet 10}{BW_{0-2}}\right) + \left(\frac{EF_{2-6} \bullet ED_{2-6} \bullet IRW_{2-6} \bullet 3}{BW_{2-6}}\right) + \left(\frac{EF_{6-16} \bullet ED_{6-16} \bullet IRW_{6-16} \bullet 3}{BW_{6-16}}\right) + \left(\frac{EF_{16-26} \bullet ED_{16-26} \bullet IRW_{16-26} \bullet 1}{BW_{16-26}}\right)\right]$$

Adjustment Factors of 10, 3 and 1 are used to account for different risks from exposure during different life stages.

Proposed Updates to Appendix A

$IFWM_{adj}$ Calculation

$IFWM_{adj}$ Parameter Values:

Symbol	**Parameter**	**Proposed Value**
EF - all (days/year)	Exposure Frequency	350
$ED_{0\text{-}2}$ (years)	Exposure Duration: 0-2 years of age	2
$ED_{2\text{-}6}$ (years)	Exposure Duration: 2-6 years of age	4
$ED_{6\text{-}16}$, $ED_{16\text{-}26}$ (years)	Exposure Duration: 6-16 and 16-26 years of age	10
$IRW_{0\text{-}2}$, $IRW_{2\text{-}6}$ (L/day)	Daily Water Ingestion Rate: 0-2 and 2-6 years of age	0.78
$IRW_{6\text{-}16}$, $IRW_{16\text{-}26}$ (L/day)	Daily Water Ingestion Rate: 6–16 and 16-26 years of age	2.5
$BW_{0\text{-}2}$, $BW_{2\text{-}6}$ (kg)	Body Weight: 0-2 and 2-6 years of age	15
$BW_{6\text{-}16}$, $BW_{16\text{-}26}$ (kg)	Body Weight: 6-16 and 16-26 years of age	80

Updates to Class II: General Resource Groundwater Quality Standards (Section 620.420)

In addition to the new constituents, updated Class II GQS are proposed for 74 constituents or mixtures currently listed in Section 620.420. Proposed updated standards are based on the following factors:

-Updated Class I Groundwater Quality Standards

-Irrigation or Livestock Criteria

-Updated Treatment Factors

Updated Treatment Factors

Treatment Factors are applied based on the effectiveness to treat the constituent in the groundwater at an 80% removal efficiency rate:

- For removal via air stripping, an 80% removal efficiency rate is assumed for constituents having a Dimensionless Henry's Law Constant (H') value greater than methylene chloride's (H') value of 0.111 at a 20°C groundwater system temperature.

OR

- For removal via carbon adsorption, an 80% removal efficiency rate is assumed for constituents having an Organic Carbon Partition Coefficient (K_{oc}) value greater than ethylbenzene's (K_{oc}) value of 446 L/kg.

If a constituent's chemical/physical values meet either of the criteria, a Treatment Factor of 5 is applied to the Class I Groundwater Quality Standard to calculate a Class II Groundwater Quality Standard.

- Source of Chemical/Physical Values: U.S. EPA Regional Screening Levels
- Source of Treatment Factor Criteria: Illinois Pollution Control Board R08-18

Proposed Addition of Tables at Appendix E for Similar-Acting Chemicals

35 Ill. Adm. Code 620, Appendix B and Appendix C provide procedures for mixtures of similar-acting substances within the groundwater.

- Table A lists similar-acting constituents based on noncarcinogenic health effects or target organs.
- Table B lists similar-acting constituents based on cancer effects.

Illinois Environmental Protection Agency Proposed Updates to 35 Ill. Adm. Code 620

By: Carol Hawbaker
Environmental Risk Assessor
Illinois Environmental Protection Agency
Office of Toxicity Assessment

May 26, 2021

Proposed Updates to 35 Ill. Adm. Code 620

The presentation will cover the following topics:

- Introduction of nine new constituents.
- Addition of three metabolites to be evaluated with atrazine for compliance with groundwater quality standards (GQS).
- Combination of radium 226 and radium 228 to form a new constituent: radium (combined 226+228).
- Addition of carcinogen designations for four existing constituents.
- Updates to constituents in the tables at Section 620.310(a)(3)(A)(i) and (ii).

Proposed Updates to 35 Ill. Adm. Code 620

The presentation will cover the following topics (continued):

- Updates of Class I GQS for three inorganic constituents from MCLs to irrigation/livestock water quality standards, based on beneficial use of groundwater.
- For constituents which Class I GQS are based on procedures found in Section 620, Subpart F and Appendix A:
  - Updates to toxicity values and relative source contribution (RSC) values;
  - Updates to exposure factors;
  - Addition of a mutagenic method for the development of a carcinogen Class I GQS for constituents with a mutagenic mode of action.

Proposed Updates to 35 Ill. Adm. Code 620

The presentation will cover the following topics (continued):

- Updates to Class II GQS.
- Introduction of tables (Appendix E) listing constituents that have similar-acting health effects or affect the same target organ.

Introduction of New Constituents

- Aluminum
- Lithium
- 1-Methylnaphthalene
- Molybdenum

- Five Per-and Polyfluoroalkyl Substances (PFAS):
  - PFBS (Perfluorobutanesulfonic Acid)
  - PFHxS (Perfluorohexanesulfonic Acid)
  - PFNA (Perfluorononanoic Acid)
  - PFOA (Perfluorooctanoic Acid)
  - PFOS (Perfluorooctanesulfonic Acid)

Introduction of New Constituents

Proposed Class I and Class II GQS:

CASRN	Constituent	Proposed Class I GQS (mg/L)	Class I Source	Proposed Class II GQS (mg/L)	Class II Source
7429-90-5	Aluminum	1.9	Subpart F	5	Livestock
7439-93-2	Lithium	0.01	Subpart F	2.5	Irrigation
90-12-0	1-Methylnaphthalene	0.27	Subpart F	0.27	Subpart F
7439-98-7	Molybdenum	0.019	Subpart F	0.05	Irrigation

Per and Poly-Fluoroalkyl Substances (PFAS)

PFAS are a group of human-made constituents applied to many consumers products to make them waterproof, stain resistant or non-stick.

- Food packaging - fast food containers, lunch meat paper, disposable plates and bowls, and oil-, water- and grease-resistant coatings on food packaging (pizza boxes);
- Commercial household products - non-stick coated cookware (Teflon), cleaning products, waxes, polishes, and adhesives;
- Clothing and fabric textiles - stain- and water-resistant carpeting and upholstery, water repellant clothing, tents, umbrellas, shoes, and leather goods;
- Cosmetics and personal care products - shampoos, conditioners, sunscreens, cosmetics, and dental floss;
- Building and exterior use products - paints and sealants;
- Industrial use - metal plating and finishing, wire coatings, automotive fluids, and the manufacture of artificial turf;
- Firefighting foam - aqueous film-forming foam (AFFF).

Per and Poly-Fluoroalkyl Substances (PFAS)

PFAS are constituents of emerging concern:

- "Forever Chemicals": PFAS not degrade naturally in the environment.
- PFAS constituents have an affinity for water and can migrate long distances.
- PFAS can bioaccumulate in plants, fish and wildlife, and humans.
- PFAS are a group of chemicals consisting of over 5,000 substances.
- Toxicological studies and assessments are being conducted by several agencies.
- Limited toxicological data for most PFAS.

Per and Poly-Fluoroalkyl Substances (PFAS)

Epidemiology and Animal Studies Suggest Associations Between PFAS Exposure and Several Health Effects:

- Pregnancy-Induced Hypertension/Pre-Eclampsia
- Liver Damage
- High Cholesterol
- Thyroid Disease
- Decreased Response to Vaccines
- Decreased Fertility
- Decreased Birth Weight
- Developmental Delays

Per and Poly-Fluoroalkyl Substances (PFAS)

- PFOA meets Illinois EPA's definition of a carcinogen. The International Agency for Research on Cancer (IARC) classified PFOA as a "2B" carcinogen in 2017.
- A "2B" classification means the constituent is possibly carcinogenic to humans.
- U.S. EPA concluded there was "suggestive potential" for PFOS to be carcinogenic to humans; however, PFOS does not meet Illinois EPA's definition of a carcinogen at this time.
- Possible Cancer Links:

  -Kidney

  -Testicular

  -Prostate

  -Liver

  -Pancreas

Per and Poly-Fluoroalkyl Substances (PFAS)

Proposed Class I GQS are based on proposed procedures for 35 Ill. Adm. Code 620, Subpart F and Appendix A.

CASRN	Constituent	Class I and Class II GQS (mg/L or ppm)	Class I and Class II GQS (ng/L or ppt)	Toxicity Value	Toxicity Value Source	Relative Source Contribution Value for Noncarcinogens
375-73-5	PFBS	0.0012	1,200	3E-04	PPRTV	0.2
355-46-4	PFHxS	0.000077	77	2E-05	ATSDR	0.2
375-95-1	PFNA	0.000012	12	3E-06	ATSDR	0.2
335-67-1	PFOA	0.000002	2	1.4E+02	OEHHA	Not Applicable
1763-23-1	PFOS	0.0000077	7.7	2E-06	ATSDR	0.2

PPRTV: Provisional Peer Reviewed Toxicity Values.

ATSDR: Agency for Toxic Substance and Disease Registry.

OEHHA: California EPA Office of Environmental Health Hazard Assessments.

PFBS, PFHxS, PFNA and PFOS toxicity values are oral reference doses (RfDs) for noncarcinogen effects in units of mg/kg-day.

PFOA toxicity value is an oral slope factor (SF_o) for cancer risks in units of $(mg/kg\text{-}day)^{-1}$. The GQS is the minimum reporting level, per Subpart F.

Addition of Atrazine Metabolites

The amendments propose the addition of 3 atrazine metabolites to be included when comparing atrazine concentrations to GQS.

Added Metabolites

-DEA (Desethyl-atrazine)

-DIA (Desisopropyl-atrazine)

-DACT (Diaminochlorotriazine)

Combination of Radium 226 and 228

Presently, radium 226 and radium 228 have individual Class I GQS. They are not listed in the Class II GQS.

The amendments propose radium (combined 226+228) Class I and Class II GQS.

The proposed value for the Class I and Class II GQS is based on the Federal maximum contaminant level (MCL) for radium (combined 226+228) of 5 picocuries per liter (pCi/L).

Proposed Updates to Carcinogen Designations

Carcinogen designations are updated for the following constituents:

p-Dichlorobenzene (1,4-dichlorobenzene)

- Classified "2B" by International Agency for Research on Cancer (IARC) - 1999

Ethylbenzene

- Classified "2B" by IARC - 2000

gamma-HCH (*gamma*-hexachlorocyclohexane, lindane)

- Classified "1" by IARC – 2018

Isopropylbenzene (cumene)

- Classified "2B" by IARC - 2013

Proposed Updates to Constituents in Tables at 35 Ill. Adm. Code 620.310(a)(3)(A)(i) and (ii) – Preventive Response Activities

The following constituents are removed from the tables due to carcinogenicity classifications, based on the Board Note at Section 620.310(a)(3)(A).

- *p*-Dichlorobenzene (1,4-dichlorobenzene)
- Ethylbenzene
- Arsenic
- *gamma*-HCH (lindane)
- Isopropylbenzene (cumene)

MCPP (mecoprop) is removed as the constituent's proposed Class I GQS is based on its lowest level of quantitation (LLOQ) or lowest concentration minimum reporting level (LCMRL), formerly termed practical quantitation limit (PQL).

Proposed Updates to Constituents in Tables at 35 Ill. Adm. Code 620.310(a)(3)(A)(i) and (ii) – Preventive Response Activities

Constituents Added to Tables

- Aluminum
- Molybdenum
- 1-Methylnaphthalene
- PFBS
- PFHxS
- PFNA
- PFOS
- Antimony
- HMX (octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine)
- Nitrobenzene
- RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine
- TNT (2,4,6-trinitrotoluene)
- 1,3,5-Trinitrobenzene

Proposed Updates of Class I GQS for Three Inorganic Constituents Based on More Stringent Irrigation or Livestock Values

Class I potable resource groundwater may also be used for irrigation and watering of livestock. The following constituents are proposed to be updated as follows:

CASRN	Constituent	Current Class I GQS (mg/L)	Current Source	Proposed Class I GQS (mg/L)	Proposed Source
7440-50-8	Copper	0.65	Lead/Copper Rule	0.5	Livestock
7681-49-4	Fluoride	4	U.S. EPA MCL	2	Livestock
7782-49-2	Selenium	0.05	U.S. EPA MCL	0.02	Irrigation

Proposed Updates to Subpart F and Appendix A

Out of 115 total constituents presently listed at 35 Ill. Adm. Code 620.410, 40 utilize the procedures in Subpart F and Appendix A to develop Class I GQS (35% of constituents):

- 30 constituents utilize the Human Threshold Toxicant Advisory Concentration (HTTAC) calculation at Appendix A(a) for noncarcinogens.

- 10 constituents utilize the Human Nonthreshold Toxicant Advisory Concentration (HNTAC) calculation at 35 Ill. Adm. Code 620.605(b)(2), for carcinogens. Of these 10, 7 constituents utilize a practical quantitation limit (PQL), because the calculated HNTAC is less than the PQL.

Proposed Updates to Appendix A

Illinois EPA's Hierarchy for Determining Toxicity Values

Basis for hierarchy is derived from U.S. EPA OSWER Directive 9285.7-53, dated December 5, 2003, and discussed in the Illinois Pollution Control Board Rulemaking R08-18: Proposed Amendments to Groundwater Quality Standards, 35 Ill. Adm. Code 620.

- Tier 1 Toxicity Value Source: Integrated Risk Information System (IRIS)
- Tier 2 Toxicity Value Source: Provisional Peer Reviewed Toxicity Values (PPRTV)
- Tier 3 Other Toxicity Values:

"Priority given to sources of information that are the most current, the basis for which is transparent and publicly available, and which has been peer-reviewed."

OSWER Directive 9285.7-53

Proposed Updates to Appendix A

Additional Guidance Regarding the Selection of Tier 3 Toxicity Values derived from U.S. EPA OSWER Directive 9285.7-86, dated May 16, 2013. Tier 3 sources are ranked as follows:

1. Agency for Toxic Substances and Disease Registry (ATSDR) minimal risk levels.
2. California EPA, Office of Environmental Health Hazard Assessment (OEHHA), toxicity values.
3. PPRTV Appendix "Screening Toxicity Values."
4. Health Effect Assessment Summary Table (HEAST) toxicity values.

Proposed Updates to Appendix A

Updates Procedures for Determining an Oral Reference Dose (RfD) When an RfD is Not Available from the Listed Toxicity Values Sources.

- Due to outdated methodology, proposes to update the procedures found at 35 Ill. Adm. Code 620, Appendix A(b)(3)-(c) for when there is no “verified” RfD, The proposed updated method is based on the methodology used by IRIS, U.S. EPA’s Tier 1 toxicity source.

- Only 1 constituent (MTBE) utilized the methodology at Appendix A(b)(3)-(c) for developing an RfD.

Proposed Updates to Appendix A

Updates to Exposure Factors in the HTTAC calculation

(updates are applied for a more sensitive receptor population – children)

Current Exposure Factors	Proposed Exposure Factors
Body Weight (BW) = 70 kg (equivalent for an average adult)	Body Weight (BW) = 15 kg (equivalent for a child 0 – 6 years) of age
Daily Water Ingestion Rate (W) = 2 L/day (equivalent for an average adult)	Daily Water Ingestion Rate (W) = 0.78 L/day (equivalent for a child 0 – 6 years of age)

Proposed Updates to Appendix A

Updates to HNTAC Calculation

(moved from 35 Ill. Adm. Code 620.605(b) to Appendix A)

HNTAC calculation for carcinogens is based on methodology found in U.S. EPA's Risk Assessment Guidance for Superfund (RAGs), Part B.

Supplemental Guidance from U.S. EPA updates the carcinogen calculation to account for age-adjusted daily water ingestion rates, as opposed to adult only water ingestion rates currently used in the calculation.

Supplemental Guidance also applies adjustment factors to the age-adjusted daily water ingestion rates for to account for toxicokinetic differences between children of various age groups and adults for carcinogens with a mutagenic mode of action for carcinogenesis.

Updated equations used to calculate U.S. EPA Regional Screening Levels (RSLs) for ingestion of tapwater.

Proposed Updates to Appendix A

Updates to HNTAC Calculation

Illinois EPA proposes to update the HNTAC calculation by incorporating updated guidance to adjust for childhood exposures to carcinogens. This includes:

- Updating the HNTAC carcinogen calculation, including updating exposure factors.

- Adding a HNTAC mutagen calculation for carcinogen constituents which operate by a mutagenic mode of action for carcinogenesis. 11 constituents are classified as mutagens; 6 rely on the HNTAC calculation to determine Class I GQS.

Proposed Updates to Appendix A

Current HNTAC Calculation

$$HNTAC(mg/L) = \frac{TR \bullet BW \bullet AT \bullet 365 \frac{days}{year}}{SF_o \bullet IR \bullet EF \bullet ED}$$

Where:

Symbol (units)	**Parameter**	**Existing Value**
TR (unitless)	Target Cancer Risk – 1 in 1 Million Risk	1.0E-06
BW (kg)	Body Weight	70
AT (years)	Averaging Time for Carcinogens	70
SF_o $((mg/kg\text{-}day)^{-1})$	Oral Slope Factor – Toxicological Value	Chemical-Specific
IR (L/day)	Daily Water Ingestion Rate	2
EF (days/year)	Exposure Frequency	350
ED (year)	Exposure Duration	30

Proposed Updates to Appendix A

Proposed Updated HNTAC Calculation

$$HNTAC\ (mg/L) = \frac{TR \bullet \left(AT \bullet 365\,\frac{days}{year}\right)}{SF_o \bullet IFW_{adj}}$$

Where:

Symbol (units)	**Parameter**	**Proposed Value**
TR (unitless)	Target Cancer Risk – 1 in 1 million	1.0E-06
AT (years)	Averaging Time for Carcinogens	70
SF_o ((mg/kg-day)$^{-1}$)	Oral Slope Factor – Toxicological Value	Chemical-Specific
IFW_{adj} (L/kg)	Age-Adjusted Daily Water Ingestion Rate	327.95

Proposed Updates to Appendix A

IFW_{adj} Calculation

$$IFW_{adj}(327.95\ L/kg) = \left[\left(\frac{EF_{child} \bullet ED_{child} \bullet IRW_{child}}{BW_{child}}\right) + \left(\frac{EF_{adult} \bullet ED_{adult} \bullet IRW_{adult}}{BW_{adult}}\right)\right]$$

Where:

Symbol (units)	**Parameter**	**Value**
EF all (days/year)	Exposure Frequency	350
ED_{child} (years)	Exposure Duration – child (0 – 6 years)	6
IRW_{child} (L/day)	Daily Water Ingestion Rate – child (0 – 6 years)	0.78
BW_{child} (kg)	Body Weight – child (0 – 6 years)	15
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IRW_{adult} (L/day)	Daily Water Ingestion Rate – adult	2.5
BW_{adult} (kg)	Body Weight – adult	80

Proposed Updates to Appendix A

Proposed Introduction of an HNTAC Calculation for Mutagens

$$HNTAC_{MUT}\ (mg/L) = \frac{TR \bullet \left(AT \bullet 365\frac{days}{year}\right)}{SF_o \bullet IFWM_{adj}}$$

Where:

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AT (years)	Averaging Time for Carcinogens	70
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$IFWM_{adj}$ (L/kg)	Age-Adjusted Daily Water Ingestion Rate for Mutagens	1,019.9

Proposed Updates to Appendix A

$IFWM_{adj}$ Calculation

$$IFWM_{adj}\,(1019.9\,L/kg) = \left[\left(\frac{EF_{0-2} \bullet ED_{0-2} \bullet IRW_{0-2} \bullet 10}{BW_{0-2}}\right) + \left(\frac{EF_{2-6} \bullet ED_{2-6} \bullet IRW_{2-6} \bullet 3}{BW_{2-6}}\right) + \left(\frac{EF_{6-16} \bullet ED_{6-16} \bullet IRW_{6-16} \bullet 3}{BW_{6-16}}\right) + \left(\frac{EF_{16-26} \bullet ED_{16-26} \bullet IRW_{16-26} \bullet 1}{BW_{16-26}}\right)\right]$$

Adjustment Factors of 10, 3 and 1 are used to account for different risks from exposure during different life stages.

Proposed Updates to Appendix A

$IFWM_{adj}$ Calculation

$IFWM_{adj}$ Parameter Values:

Symbol	**Parameter**	**Proposed Value**
EF - all (days/year)	Exposure Frequency	350
ED_{0-2} (years)	Exposure Duration: 0-2 years of age	2
ED_{2-6} (years)	Exposure Duration: 2-6 years of age	4
ED_{6-16}, ED_{16-26} (years)	Exposure Duration: 6-16 and 16-26 years of age	10
IRW_{0-2}, IRW_{2-6} (L/day)	Daily Water Ingestion Rate: 0-2 and 2-6 years of age	0.78
IRW_{6-16}, IRW_{16-26} (L/day)	Daily Water Ingestion Rate: 6–16 and 16-26 years of age	2.5
BW_{0-2}, BW_{2-6} (kg)	Body Weight: 0-2 and 2-6 years of age	15
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-Updated Treatment Factors

Updated Treatment Factors

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- For removal via air stripping, an 80% removal efficiency rate is assumed for constituents having a Dimensionless Henry's Law Constant (H') value greater than methylene chloride's (H') value of 0.111 at a 20°C groundwater system temperature.

OR

- For removal via carbon adsorption, an 80% removal efficiency rate is assumed for constituents having an Organic Carbon Partition Coefficient (K_{oc}) value greater than ethylbenzene's (K_{oc}) value of 446 L/kg.

If a constituent's chemical/physical values meet either of the criteria, a Treatment Factor of 5 is applied to the Class I Groundwater Quality Standard to calculate a Class II Groundwater Quality Standard.

- Source of Chemical/Physical Values: U.S. EPA Regional Screening Levels
- Source of Treatment Factor Criteria: Illinois Pollution Control Board R08-18

Proposed Addition of Tables at Appendix E for Similar-Acting Chemicals

35 Ill. Adm. Code 620, Appendix B and Appendix C provide procedures for mixtures of similar-acting substances within the groundwater.

- Table A lists similar-acting constituents based on noncarcinogenic health effects or target organs.
- Table B lists similar-acting constituents based on cancer effects.

Proposed Changes to 35 Ill. Adm. Code Part 620

May 2021

Illinois Environmental Protection Agency

Agenda

- **Opening Remarks**
- **Overview of Changes**
  - Bureau of Water-Lynn Dunaway
  - Associate Director's Office (Toxicology)-Carol Hawbaker
- **Q&A with Panelist**
  - Department of Legal Counsel- Sara Terranova
  - Bureau of Land-Greg Dunn
  - Bureau of Water-Lynn Dunaway
  - Associate Director's Office (Toxicology)-Carol Hawbaker
- **Closing Remarks**

Illinois Environmental Protection Agency

Proposed Changes Subparts A and B

Section 620.110 Definitions

- Definitions have been added to reflect updated terminology
- Delete obsolete terms

Section 620.125 Incorporations by Reference

- Update reference to USEPA documents
- New and updated analytical methods
- Update sample collection procedures

Section 620.210 Class I: Potable Resource Groundwater

- Added delineated wellhead protection areas as Class I groundwater areas
- Eliminated permeameters as a method to determine hydraulic conductivity for groundwater classification

Section 620.250 Groundwater Management Zone

- Added a list of information that must be provided with a GMZ application

Illinois Environmental Protection Agency

Proposed Changes Subpart C

Section 620.302 Applicability of Preventive Notice and Preventive Response Activities

- Added additional examples of programs conducting groundwater monitoring

Section 620.310 Preventive Response Activities

- Tabulated lists of chemicals
- Added chemical abstract numbers for reference
- Eliminated chemicals which are now considered carcinogens
- Added proposed chemicals to which Preventive Response will apply
- Replaced outdated analytical references with updated references

Illinois Environmental Protection Agency

Proposed Changes Subpart D

Section 620.410 Groundwater Quality Standards for Class I: Potable Resource Groundwater

- Tabulated lists of chemicals
- Added chemical abstract numbers for reference
- Added proposed chemicals
- Updated numerical groundwater standards to reflect MCLs
- Update numerical groundwater standards with the proposed criteria for establishing health-based concentrations (Carol Hawbaker will discuss these proposed changes further)
- Added footnotes describing the origin of the numerical groundwater standard

Section 620.420 Groundwater Quality Standards for Class II: General Resource Groundwater

- Tabulated lists of chemicals
- Added chemical abstract numbers for reference
- Added proposed chemicals
- Updated numerical groundwater standards to reflect updated treatment efficiencies
- Added footnotes describing the origin of the numerical groundwater standard

Proposed Changes Subpart D

Section 620.430 Groundwater Quality Standards for Class III: Special Resource Groundwater

- Site-specific standards for chloride and pH within the designated Class III Groundwater areas of four Dedicated Nature Preserves that are cave systems

- Site-specific standards for chloride within the designated Class III Groundwater areas of two Dedicated Nature Preserves that are wetlands

Section 620.440 Groundwater Quality Standards for Class IV: Other Groundwater

- Updated names of previously regulated chemicals

Section 620.450 Alternative Groundwater Quality Standards

- Updated names of previously regulated chemicals

Illinois Environmental Protection Agency

Proposed Changes Subparts E and F

Section 620.510 Monitoring and Analytical Requirements

- Simplify citation to Section 620.125
- Add new subsection for statistical methods document contained in Section 620.125
- Update analytical method references

Section 620.601 Purpose of a Health Advisory

- Update citation to applicable regulations

Section 620.605 Issuance of a Health Advisory

- Update references to guidance
- Update analytical method references

Written comments must be received by the Illinois EPA by **June 25, 2021**.

Comments must be submitted to **EPA.620.rulemaking@illinois.gov.**

Thank You For Your Participation!

Illinois Environmental Protection Agency

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3M

January 31, 2020

Stephanie Flowers, Part 620
Illinois Environmental Protection Agency
1021 North Grand Avenue East
P.O. Box 19276
Springfield, IL 62794-9276

3M Comments on Proposed Changes to 35 Ill. Adm. Code 620: Groundwater Quality

Dear Ms. Flowers,

3M is pleased to submit comments to the Illinois Environmental Protection Agency (Illinois EPA) regarding its proposed changes to the language of 35 Ill Adm. Code 620: Groundwater Quality. In its letter to certain business and industrial group stakeholders dated December 24, 2019, the Illinois EPA noted that it is proposing to amend Section 620.410 Groundwater Quality Standards for Class I Potable Resource Groundwater by, among other things, adding standards for Perfluorbutane Sulfonic Acid (PFBS), Perfluorhexane Sulfonic Acid (PFHxS), Perfluorononanoic Acid (PFNA), Perfluorooctanoic Acid (PFOA), and Perfluorooctane Sulfonic Acid (PFOS).

The statement in Illinois EPA's December 24, 2019 letter that it is "using the methodology developed under Part 620 Subpart F with PFAS oral reference doses drafted by the Agency for Toxic Substances and Disease Registry (ATSDR)," appears to confuse the concept of minimum risk levels (MRLs) identified by ATSDR with the concept of reference doses used by the United States EPA. As the ATSDR Toxicological Profile for Perfluoroalkyls notes, "MRLs are derived using a modified version of the risk assessment methodology that the Environmental Protection Agency (EPA) provides (Barnes and Dourson 1988) to determine reference doses (RfDs) for lifetime exposure." ATSDR, Toxicological Profile for Perfluoroalkyls: Draft for Public Comment at C-1 (June 2018) ("Draft ATSDR Profile"). In addition, although ATSDR has not identified an MRL for PFBS, Illinois EPA is proposing a Groundwater Quality Standard for that substance without citing any basis other than ATSDR.

There are numerous problems with adopting ATSDR's MRLs for the listed substances. 3M submitted extensive comments in response to ATSDR's Draft Toxicological Profile for Perfluoroalkyls. Those comments, which address significant deficiencies in the science and studies relied upon by ATSDR in developing its PFOA, PFOS, and PFHxS MRLs, are enclosed at Attachment A. Some of the key deficiencies include:

- Greater consideration should have been given to non-human primate studies that exist in the literature for PFOA and PFOS. In addition, ATSDR selection for PFOA and PFOS did not consider more recently available human and non-human primate studies;

- ATSDR selected inappropriate studies to serve as the basis for the MRL for PFOA;
- PFOA, PFOS, and PFHxS MRLs are biased (downward) because ATSDR used serum half-lives that do not accurately reflect the most reliable and current evidence on human serum half-lives applicable to the general population;
- ATSDR applied scientifically flawed uncertainty factors that lowered the MRLs by as much as an order of magnitude or more;
- Significant new studies were not considered by ATSDR;
- There was a lack of transparency in ATSDR's synthesis of its weight-of-the-evidence review for the eight epidemiological associations or key toxicological endpoints; and
- ATSDR failed to address declining levels of PFOS and PFOA in the general population.

Each of these key deficiencies is explained in detail in Attachment A.

Even assuming its MRLs are scientifically sound, which they are not, ATSDR acknowledged that "[t]he available human studies have identified some potential targets of toxicity; however, cause and effect relationships have not been established for any of the effects, and the effects have not been consistently found in all studies." Draft ATSDR Profile at 636. ATSDR has also stated that MRLs "are not intended to define clean up or action levels." Draft ATSDR Profile at A-1.

Given the apparent confusion between the concept of reference dose and MRLs, the significant deficiencies in ATSDR's MRLs, and the lack of causal connection to human health effects, 3M urges Illinois EPA to re-evaluate its proposed Groundwater Quality Standards for PFBS, PFHxS, PFNA, PFOA, and PFOS.

3M appreciates the opportunity to provide comments in advance of the proposed rule. Thank you for your consideration.

Regards,

Oyebode A. Taiwo, MD, MPH

ATTACHMENT A

John Banovetz, Ph.D.
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August 20, 2018

Division of Toxicology and Human Health Sciences,
Agency for Toxic Substances and Disease Registry,
1600 Clifton Rd. NE, MS F–57
Atlanta, GA 30329
Attn: Docket No. ATSDR– 2015–0004

Subject: 3M Company's Comments of ATSDR Draft Toxicological Profile for Perfluoroalkyls

The 3M Company (3M) appreciates the opportunity to review and provide comment on ATSDR's "Draft Toxicological Profile for Perfluoroalkyls" (Draft Profile). As we highlight here and address in our detailed comments, we believe there are major shortcomings with the current draft, especially with the proposed minimal risk levels (MRLs). Considering the strong interest by the general public and others, it is important that this profile reflect the best science and full weight of evidence known about these chemicals. At present, it does not.

3M's Voluntary Phase out and Declining PFOA, PFOS, and PFHxS

As a science-based company, 3M has substantial experience and expertise with the breadth of topics addressed by the Draft Profile. In fact, numerous 3M scientists are authors or contributors to many of the studies referenced in the report, especially in the areas of toxicology, pharmacokinetics, biomonitoring, and epidemiology. 3M also was first to sponsor the development of several physiologically-based pharmacokinetic models (PBPK) regarding perfluoroalkyls.

As you know, 3M announced in 2000 that it would voluntarily phase out the manufacture and use of PFOS and PFOA (and their related materials). This was completed worldwide by about 2008. 3M phased out these chemicals due to their persistence. We did not believe there was evidence of actual adverse health effects in humans at that time, and the body of literature available to date, when properly assessed, continues to confirm this position.

After 3M announced that it would voluntarily phase out of these chemistries, other manufacturers began to phase out of production and use of PFOA under EPA's Stewardship plan. As a result of the phase-out, the levels of PFOS and PFOA in the blood of the general population in the US have declined and are expected to continue to decline. Data from the American Red Cross show that, as of 2015, levels of PFOS and PFOA among these study subjects had declined 70-80% since 2000. Similar percentage have declined in the general U.S. population through 2013 – 2014 as published by NHANES. This is important

information for the public, which is absent in the current Draft Toxicological Profile for Perfluoroalkyls. Because people may erroneously equate presence with harm, levels found in the environment must be understood in the context of the weight of the evidence showing the lack of harm from perfluoroalkyl exposure at such levels.

The body of scientific evidence does not show adverse health effects in humans from perfluoroalkyls

The vast body of scientific evidence does not show that PFOS or PFOA cause any adverse health effects in humans at current exposure levels, or even at the historically higher levels found in blood. ATSDR acknowledges that there is no cause and effect, when it states: "The available human studies have identified some potential targets of toxicity; however, cause and effect relationships have not been established for any of the effects, and the effects have not been consistently found in all studies." However, ATSDR does not present this critical point until page 636 of the draft profile.

A recently released review of studies involving perfluoroalkyls exposed populations commissioned by the Australian government also supports the lack of evidence of harm. That May 2018 report by the Australian Expert Health Panel stated, "The Panel concluded there is mostly limited or no evidence for any link with human disease from these observed differences. Importantly, there is no current evidence that supports a large impact on a person's health as a result of high levels of perfluoroalkyl exposure." The report further stated: "After considering all the evidence, the Panel's advice to the Minister on this public health issue is that the evidence does not support any specific health or disease screening or other health interventions for highly exposed groups in Australia, except for research purposes."

ATSDR's Public Health Role Mandates that it Revise the Draft Toxicological Profile for Perfluoroalkyls

ATSDR's states that the "primary purpose" of the draft Toxicological Profile for Perfluoroalkyls is to provide "public health officials, physicians, toxicologists" and others "with an overall perspective on the toxicology of perfluoroalkyls" (p. 21). ATSDR does not meet this goal, especially with respect to the MRL development, because it relies on flawed and incomplete data and because the conclusions it draws are unjustified by the data on which it relies. These errors require a wholesale revision of the draft Toxicological Profile and a new round of comments on any revised profile.

For many stakeholders, MRLs may be the most important component of the draft Toxicological Profile for Perfluoroalkyls. Media accounts clearly show there is already great confusion among the public, Congress, the media and NGOs as to their meaning and how MRLs should or should not be used. Some erroneously believe that MRLs are a bright line between safe and unsafe. It is imperative, therefore, that ATSDR clearly educate readers on the use and meaning of MRLs in Chapter One, where the MRLs are first presented, and not where they currently appear, over 600 pages later deep in the technical appendices.

Stakeholders reading the draft profile need to clearly understand that ATSDR has said that:

- MRLs "are not intended to define clean up or action levels"
- MRLs are "intended only to serve as a screening tool"
- MRLs "are below levels that might cause adverse health effects in the people most sensitive to such chemical-induced effects"
- "Exposure to a level above the MRL does not mean that adverse health effects will occur."
- An "MRL may be as much as 100-fold below levels that have been shown to be nontoxic in laboratory animals"
- "If someone is exposed to an amount above the MRLs, it does not mean that health problems will happen."

The Proposed MRLs Fail to Reflect the Best Available Science

Overall, the provisional MRLs proposed by ATSDR for PFOA, PFOS, and PFHxS were not derived using best available science. There were many deficiencies and unnecessarily conservative and scientifically flawed assumptions associated with these MRLs. They should be withdrawn or revised to reflect a more realistic and scientifically supported risk assessment. As more fully set forth in our comments, key concerns with these MRLs include, but not are limited to:

- Greater consideration should be given by ATSDR to the non-human primate studies that exist in the literature for PFOA and PFOS, as was done by ATSDR in 2015. In addition, ATSDR selection for PFOA and PFOS did not consider the more recently available human and non-human primate studies. 3M believes ATSDR should seriously consider these studies in their approach to MRL as they either do or more closely represent human physiology; and, have relevance to questions regarding thyroid, cholesterol, and liver evaluations. These include a Phase 1 clinical trial in humans for PFOA (Convertino et al. 2018) and a one-year evaluation of clinical chemistries in non-human primates for PFOS (Chang et al. 2017).

- ATSDR selected inappropriate studies to serve as basis for the proposed MRL for PFOA which ***lacked fundamental scientific rigor***, including such shortcomings as: (1) use of only single dose level, making it impossible to confirm a dose-response effect, or to determine the point of departure level; (2) involved too few animals to generate reliable results; (3) provided no details on the reproductive nor the developmental hallmarks; (4) litter bias; (5) used non-standard testing methods; and (5) provided no internal serum PFOA dosimetry data. The corresponding study results should not be used in any meaningful risk assessment for humans and are wholly inadequate to form the basis for a PFOA MRL.

- PFOA, PFOS, and PFHxS MRLs are biased (downward) because ATSDR used serum half-lives that do not accurately reflect the most reliable and current evidence on human serum half-lives applicable to the general population. Had it done so MRL

values would have ranged between 9 - 40% higher for PFOA, 12 – 38% higher for PFOS, and 14-38% higher for PFHxS;

- ATSDR applied scientifically flawed uncertainty factors that lowered the MRLs by as much as an order of magnitude or more, including: (1) use of an uncertainty factor of three for interspecies extrapolation (animal to human) for PFOA, PFOS and PFHxS, even though that rodents are known to be more sensitive than humans to the effects at issue; (2) use of an uncertainty factor of 10 in its PFOS and PFHxS MRL derivations to account for potential immunological effects that was arbitrary, not justified by toxicology and epidemiologic studies, and contrary to ATSDR's acknowledgement that the human evidence for immune effects is insufficient to support causation; and (3) use of an inappropriate uncertainty factor of 10 for PFOA for a LOAEL-to-NOAEL extrapolation because the study design was so deficient so as to preclude even establishing any LOAEL or NOAEL values.

Epidemiological Associations Claimed by ATSDR are Not Supported by the Science

In addition, the draft Toxicological Profile for Perfluoroalkyls identified eight potential epidemiological associations between perfluoroalkyl exposure and health outcomes. The relevant body of science for these chemicals does not support ATSDR's position. As our detailed comments show, the scientific evidence clearly refutes the claimed associations and shows that ATSDR must revisit its analysis. In addition, ATSDR actually acknowledges that none of these associations indicate causality (see above comment on page 2 of this letter). To minimize undue public misperceptions and undue fears, ATSDR must place this admission prominently at the beginning of the report, before any discussion of the alleged epidemiological associations between perfluroroalkyl exposure and health outcomes.

Many Other Concerns and Deficiencies Require Revisions to the Draft

Our detailed comments outline many other concerns with the draft Toxicological Profile for Perfluoroalkyls, including, but not limited to: (1) significant new studies were not considered by ATSDR; (2) a lack of transparency in ATSDR's synthesis of its weight-of-the-evidence review for the eight epidemiological associations or key toxicological endpoints; and (3) a failure to address declining levels of PFOS and PFOAs in the general population.

Finally, because of the 852-page length of the draft profile, along with its nearly 300-page supporting document, the 60-days provided to the public for review and comment was not adequate for detailed review and comment on every aspect of the draft Toxicological Profile for Perfluoroalkyls. Accordingly, the lack of comment on any particular detail or section within this ATSDR document does not necessarily imply agreement by 3M with that content.

ATSDR Must Further Review and Revise the DRAFT Toxicological Profile for Perfluoroalkyls

3M appreciates the opportunity to provide its comment on the draft Toxicological Profile for Perfluoroalkyls. The document represents a significant undertaking by ATSDR, but it needs

to be based on current, relevant and reliable scientific information to be accurate and useful to multiple stakeholders. As highlighted here and in our detailed comments, the shortcomings with the current draft, including the proposed MRLs require that ATSDR perform additional work to assure that the profile reflect the best science and full weight of evidence known about these perfluoroalkyls.

If there are questions or comments concerning this matter, please contact me.

Sincerely,

John Banovetz

John Banovetz, Ph.D.
Senior Vice President and Chief Technology Officer

Executive Summary of 3M's Comments

The 3M Company (3M) appreciates the opportunity to review and comment on the "Draft Toxicological Profile for Perfluoroalkyls". As authors or a sponsor of many of the human epidemiology and toxicology studies discussed in the draft documents, we offer these detailed comments for Health Effects in assisting with that effort. Given the magnitude of scientific literature that have become available since the last Draft was released in 2015, the following important scientific comments should be considered by ATSDR with the overall data integration.

A. **The Public Comment Period was Too Short**. The Draft Toxicological Profile is 852 pages long. Its support document is nearly 300 pages long. The 60-days provided to the public for review and comment was not adequate for detail review and comment on every aspect of the draft Toxicological Profile. Accordingly, the lack of comment on any particular detail or section within this ATSDR document does not necessarily imply agreement with that content.

B. **MRL Meaning and Limitations Not Prominently Presented.** ATSDR should be aware that for the public and regulators the Minimum Risk Levels (MRLs) will be an important component of the draft Toxicological Profile. Yet, ATSDR defers any explanation of what the MRLs mean and the limits on their use until deep in the technical appendices of this document (e.g., page 713 in Appendix A and page in Appendix C). Accordingly, it is very important that ATSDR features this information in Chapter 1, where ATSDR presents the MRL values. ATSDR should recognize that most readers will not go any further than this opening chapter. Media accounts show there is already great confusion among the general public, Congress, the media and NGOs as to what MRLs values mean and how they should or should not be used. There is a clear misperception that MRLs represent a line between safe and unsafe exposure to a chemical, which is incorrect.

   ATSDR should include the following statements from the technical appendices in Chapter 1.

   From Appendix A (page A-1, page 713 of the profile), ATSDR should include:

   - *An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be* ***without appreciable risk of adverse noncancer health effects*** *over a specified route and duration of exposure. These substance-specific estimates, which are* ***intended to serve as screening levels****, are used by ATSDR health assessors to identify contaminants and potential health effects that may be of concern at hazardous waste sites. It is important to note that* ***MRLs are not intended to define clean-up or action levels****.*
   - *They are* ***below levels that might cause adverse health effects in the people most sensitive*** *to such chemical-induced effects.*
   - *MRLs are generally* ***based on the most sensitive substance-induced endpoint*** *considered to be of relevance to humans. Serious health effects (such as irreparable damage to the liver or kidneys, or birth defects) are not used as a basis for establishing MRLs.* ***Exposure to a level above the MRL does not mean that adverse health effects will occur****.*

- *MRLs are **intended only to serve as a screening tool** to help public health professionals decide where to look more closely.*
- *In the absence of evidence to the contrary, ATSDR assumes that humans are more sensitive to the effects of hazardous substance than animals and that certain persons may be particularly sensitive. Thus, **the resulting MRL may be as much as 100-fold below levels that have been shown to be nontoxic** in laboratory animals*

From Appendix C (page C-1, page 835 of the profile), ATSDR should include:

- *These MRLs are **not meant to support regulatory action**, but to acquaint health professionals with exposure levels **at which adverse health effects are not expected to occur** in humans.*
- *MRLs should help physicians and public health officials determine the safety of a community living near a hazardous substance emission, given the concentration of a contaminant in air or the estimated daily dose in water. MRLs are based largely on toxicological studies in animals and on reports of human occupational exposure.*

Finally, ATSDR's website includes a description of MRLs for the general public, which should also be included to help the lay public:

- *An MRL is an estimate of the amount of a chemical a person can eat, drink, or breathe each day **without a detectable risk to health**. MRLs are developed for health effects other than cancer. **If someone is exposed to an amount above the MRLs, it does not mean that health problems will happen**. When health assessors find exposures higher than the MRLs, it means that they may want to look more closely at a site.*

C. The PFOA, PFOS, and PFHxS MRLs are Critically Flawed, Lower than Appropriate or Necessary, Unsupported by the Science, and should be Withdrawn or Revised. Due to time limitations, 3M's review focused on the provisional Minimum Risk Levels (MRLs) for three perfluoroalkyls (PFOA, PFOS, and PFHxS). The selection of the critical toxicological endpoints and the derivation process in establishing these provisional MRLs lacked scientific rigor and that the best available science was not applied. The improper uses of studies and overly conservative assumptions used by ATSDR resulted in MRL values that are significantly lower than supported by the science. Key concerns with ATSDR's MRL development are presented below:

1) Toxicological endpoints and human relevance

Among the toxicological endpoints chosen by ATSDR for MRL calculations, they have not been observed in humans. ATSDR should explain the relevance of these effects, if any, to human health to avoid undue public misperception. Specifically, published mode of action data on xenosensor nuclear receptors have suggested that rodents may not be the most appropriate species for the hazard assessment of perfluoroalkyls on developmental toxicity in humans. In addition, rodent hepatocytes appeared to be more sensitive to xenosensor nuclear receptor activations than human hepatocytes. Therefore, ATSDR

should take this into consideration when performing human risk assessment using rodent data.

2) Best available science not applied

There are many technical uncertainties associated with the current MRL derivations for PFOA, PFOS, and PFHxS (all based on rodent studies), and ATSDR did not appear to apply the best available science. Specifically:

- For PFOA, the two studies selected by ATSDR lacked fundamental scientific rigor (*e.g.*, a single dose study without any dose-response, small sample size with only 6 pregnant dams; no details on the reproductive nor the developmental hallmarks, litter bias, non-standard testing methods, no internal serum PFOA dosimetry data…etc.). The corresponding study results should not be used in any meaningful risk assessment for humans. ATSDR is encouraged to consider evaluating a published phase I clinical trial data with PFOA in 49 human subjects for its assessment (Convertino et al. 2018).

- For PFOS, ATSDR should take maternal toxicity influence as well as human relevance under consideration. ATSDR is encouraged to consider evaluating a published clinical chemistry study with monkeys with PFOS for its risk assessment, given these non-human primates have much similar physiological resemblance to humans than those of rodents, and the effects of PFOS on 27 clinical chemistry parameters as well as the corresponding serum PFOS levels were followed for more than 400 days (Chang et al. 2017).

- For PFHxS, the thyroid histology finding in rats cannot be replicated in another rodent species (mice) under similar study conditions hence there is no conclusive evidence to suggest that PFHxS impacts thyroid homeostasis in rodents. ATSDR is encouraged to consider evaluating a published reproductive and developmental study in mice with PFHxS for its assessment (Chang et al. 2018). In addition, ATSDR should recognize that there are distinct differences in thyroid hormone regulations between rodents and humans; and similar to PPARα- or CAR/PXR-mediated hepatocellular hypertrophy noted in rats, thyroid findings in rodents are usually rodent-specific, usually not applicable to humans, and it requires careful (weight-of-evidence) interpretation when extrapolating to human risk assessment.

3) Excessive and unnecessary adjustment factors applied for point of departure (POD)

It is scientifically unjustified for ATSDR to apply a combined adjustment factor of 300 for PFOA, PFOS, and PFHxS MRLs in addition to the (large) dosimetric TK adjustments that had already been incorporated. The (very) large dosimetric adjustment factors (10,000, 14,400, and 15,500 for PFOA, PFOS, and PFHxS, respectively) more than adequately compensate for the difference between rodents and humans. The additional combined factor of 300 reflected an overall adjustment factor of 3,000,000 for PFOA, 4,320,000 for PFOS, and 4,650,000 for PFHxS from the point of departure (POD). The

extent of these adjustments, on the order of 10E6, is not made transparent by ATSDR and is excessive.

Specific uncertainty factors that are not scientifically justified include: (a) factor of 10 for immunotoxicity (PFOS, PFHxS); and (b) factor of 10 for use of LOAEL (PFOA)

4) Toxicokinetics and half-lives in humans

In their MRL calculations, ATSDR chose to use the arithmetic mean serum elimination half-life estimates for PFOA, PFOS, and PFHxS from Olsen et al. (2007) because the study of these retirees had a longer follow-up time. These retirees averaged 66 years of age at the end of the study. ATSDR was concerned that, based on a study by Seals et al. (2011), slower kinetics is likely to constitute a larger contribution to the terminal half-life. Olsen et al. had reservations of using arithmetic means to describe their data because of its right skewness; ATSDR chose to not acknowledge this limitation. In addition, ATSDR chose not to consider serum elimination half-lives that are dependent on other factors such as age of the study subjects, and not just follow-up time, because age is associated with the glomerular filtration rate (GFR). Renal clearance of perfluoroalkyls is largely a sum of three processes involving glomerular filtration, renal tubular secretion, and renal tubular reabsorption. Because PFOA and other perfluoroalkyls vary in their affinities to bind plasma proteins, glomerular filtration of perfluoroalkyls is a product of the unbound fraction of the perfluoroalkyls and GFR. Thus, the lower estimates of serum elimination half-lives based on the younger ages in the other study populations (Bartell et al. 2010; Li et al. 2018) may be due to the higher GFR of these younger study subjects. ATSDR also did not recognize that the proportion of the general population age $\geq$ 65 years old is approximately 15%. Therefore, other serum elimination half-lives should be considered in ATSDR's MRL calculations to reflect the overall general population and its greater GFR. At a minimum, ATSDR should present sensitivity analyses using these collective data (see below).

5) Underestimation of HEDs and MRLs by ATSDR using slower half-life

For PFOA, PFOS, and PFHxS, the corresponding HEDs (and subsequent MRLs) were likely to have been underestimated because ATSDR used the most conservative half-lives reported. These half-lives were based on a cohort of retired fluorochemical workers whose exposure source was occupational and the elimination profile was dependent upon a GFR reflective of older adults. ATSDR should use half-lives more closely matching the general population demographics and their GFR. This will correspond to increases in MRLs ranging between 9 - 40% higher for PFOA; 12 – 38% higher for PFOS, and 14-38% higher for PFHxS.

6) Chronic toxicology studies are available for PFOA and PFOS

Scientifically pertinent data such as 2-year chronic studies with PFOS (Butenhoff et al. 2012a) and PFOA (Butenhoff et al. 2012c) should be included by ATSDR for the weight-of-evidence consideration. In addition (to rodent data), in considering selection of

"chronic" studies, there are internationally-recognized guidance which states that "studies of 6 months duration in non-rodents are acceptable according to Council Directive 75/318/EEC, as amended" (EMEA 1999a). Therefore, non-human primate studies with PFOA (Butenhoff et al. 2002) and PFOS (Chang et al. 2017; Seacat et al. 2002) should also be considered by ATSDR. Most importantly, these studies not only encompassed extended study period (i.e., chronic exposure) but also illustrated similar toxicological endpoints.

D. Lack of comprehensive interpretation and synthesis of the epidemiological associations concluded by ATSDR

3M respectfully disagrees with the interpretation of the epidemiological associations concluded by ATSDR and offers scientific evidence to refute these opinions. Most importantly, 3M disagrees with the lack of highlighting by ATSDR that none of these associations indicate causality, as acknowledged by ATSDR (*cf.* pages 24 and 635-636). This (the absence of causation) should be highlighted on page 5 in front of the associations that ATSDR ultimately listed to minimize undue public misperception.

1) Epidemiological association: Pregnancy-induced hypertension and pre-eclampsia

ATSDR combined pregnancy-induced hypertension and pre-eclampsia into a single health outcome without providing scientific justification for combining these two distinct pregnancy outcomes. The evidence for an association between preeclampsia and PFOA/PFOS exposure was limited to three epidemiologic studies with inconsistent findings; the strongest study methodologically reported no association. Similarly, only three studies examined the association between PFOA exposure and pregnancy-induced hypertension and also reported mixed results. The majority of studies, for both preeclampsia and pregnancy-induced hypertension, used unvalidated, self-reported pregnancy outcomes and could not establish temporality due to the cross-sectional study design. Overall, given these limitations and the inconsistencies in findings across studies, there is insufficient evidence for an association between preeclampsia and pregnancy-induced hypertension and PFOA/PFOS.

2) Epidemiological association: Hepatic enzymes

In citing an increase in liver enzymes is associated with PFOA, ATSDR neglected to simultaneously state there was no increased risk for liver disease, including enlarged liver, fatty liver, or cirrhosis. Thus, there is no liver disease-related causation with exposure to PFOA or PFOS. Furthermore, ATSDR grossly over interpreted the magnitude of influence of ALT by using the words "liver damage" associated with ALT at the concentrations reported in the literature. ALT is a leakage enzyme and may be increased due to necrosis, injury or repair. The human half-life of ALT is approximately 47 hours. Based on the recommendations of numerous regulatory authorities, increases in ALT activity of two-to threefold should be considered indicative of "hepatocellular damage." Those epidemiological studies that have suggested an elevation of ALT

associated with PFOA or PFOS remain well-within the expected physiologic range of ALT, not 2 - 3 fold higher. Therefore, ATSDR's use of the term 'liver damage" is highly misleading. Furthermore, it is well-recognized in clinical pathology it is possible to have statistically significant modest increases in ALT that are not toxicologically relevant. Finally, ATSDR did not adequately mention the many confounding factors that should be considered in evaluating liver enzymes including age, sex, race, a reliable measure of obesity (not measured as just BMI), alcohol, diet, other diseases including diabetes, and genetics.

3) Epidemiological association: Increased serum total cholesterol and LDL

The ATSDR did not provide a rationale behind its suggestion of a possible biphasic response of serum cholesterol and PFOA (or likely PFOS). Although ATSDR recognized the preliminary abstract results of a phase 1 clinical trial of PFOA (ammonium salt) published in 2010 that stated observed reductions in LDL-cholesterol were consistent with a pharmacodynamic effect, ATSDR was unaware of the actual results from the clinical chemistry assessment from this phase 1 trial that have been publicly available via its Advance Access in Toxicological Sciences in February 2018 with final publication in the May 2018 issue (Convertino et al. 2018). ATSDR is strongly encouraged to carefully consider the Convertino et al. (2018) publication and its ramification(s) in ATSDR's weight of evidence review for PFOA related to cholesterols (as well as liver enzymes and thyroid hormones). The findings from this human phase 1 clinical trial showing that cholesterol is lowered at high doses of PFOA are consistent with some animal models and the hypolipidemic activity of the xenosensor nuclear receptor PPARα agonist PFOA. ATSDR should assess plausible noncausal roles of biology and physiology at the very low PFOA concentration (4+ orders of magnitude lower than Convertino et al.) that have been reported in the conflicting observational studies.

4) Epidemiological association: Increased risk of thyroid disease

There are no consistent associations reported across the studies found in the epidemiologic literature regarding thyroid hormones or specific thyroid disease (hypothyroidism, hyperthyroidism) as related to exposure to PFOA or PFOS. ATSDR's review of the thyroid literature is disjointed and provides minimal rationale to how ATSDR reached a decision that an association exists between PFOA/PFOS and increased risk of thyroid disease. This confusion is caused, in part, by the highly inconsistent evidence presented in the epidemiologic literature. Therefore, in the draft 2018 Toxicological Profile, ATSDR should acknowledge the lack of consistent evidence of an association.

5) Epidemiological association: Decreased antibody response to vaccines

Among the epidemiologic studies cited by ATSDR, antibody responses to 8 distinct vaccines were measured. Given that observed changes in antibody response to a particular vaccine type should not be interpreted as consistent with changes in the

antibody response to another vaccine type, the ATSDR should consider immune responses to individual vaccines as distinct health outcomes. Mostly null findings were reported across all studies for PFOA, PFOS, PFHxS, and PFDeA. Furthermore, most studies have found no association between PFAS levels and increased incidence of infectious disease (or lower ability to resist or respond to infectious disease). As such, the absence of clinical immunosuppression along with inconsistent findings both within and across studies, do not support the ATSDR conclusion "*suggestive of a link between serum PFOA, PFOS, PFHxS, and PFDeA levels and decreased antibody responses to vaccines*".

6) Epidemiological association: Increased risk of asthma diagnosis

Prospective cohort studies have consistently reported no association between PFOA and asthma. Conversely, cross-sectional and case-cohort studies have reported inconsistent findings and were limited by temporal ambiguity, and unvalidated, self-reported asthma diagnosis. NTP (2016) recognized these limitations and concluded that "*there is low confidence that exposure to PFOA during childhood is associated with increased hypersensitivity responses based on the available studies*". The rationale for this conclusion was "*primarily due to the cross-sectional nature of the studies and uncertainty as to whether exposure levels reflect exposure prior to the development of hypersensitivity.*" Therefore, collectively, the existing epidemiologic evidence does not support an association between PFOA exposure and asthma risk.

7) Epidemiological association: Increased risk of decreased fertility

ATSDR incorrectly concluded an association exists between increased perfluoroalkyls (PFOA, PFOS) and decreased fertility based on epidemiologic studies. In its 2018 draft Toxicological Profile, ATSDR failed to discuss methodological issues that have been repeatedly discussed in the published epidemiology literature, in particular, those surrounding the metric of time-to-pregnancy and the amount of interpregnancy time for reaccumulation of PFOA or PFOS. Women with longer interpregnancy intervals would have longer time for reaccumulation; thus the potential for reverse causation to be observed in parous women with time to pregnancy. As reviewed in their systematic review of the reproductive epidemiology literature regarding perfluoroalkyls, Bach et al. (2016) reported of the 8 epidemiologic studies reviewed related to time to pregnancy, only one study found an association when restricted to nulliparous women; 4 studies reported an association with parous women that Bach et al. (2016) concluded was not causal but likely the result of reverse causation and unmeasured confounding related to prior pregnancies and childbirths that could influence the measurement of PFAS.

8) Epidemiological association: Small decreases in birthweight

ATSDR incorrectly concluded that an association exists between lower birthweight (< 20 gm) and PFOA. ATSDR very briefly discussed two meta-analyses published by Johnson et al. (2014) and Verner et al. (2015). Unfortunately, several important issues were not discussed via the historical context of these two meta-analyses, including understanding

the relationship between maternal glomerular filtration and fetal growth. In addition, ATSDR was not aware of two more recent meta-analyses (Negri et al. 2017; Steenland et al. 2018). Negri et al. questioned the lack of a quantitative toxicological evidence to support the biological plausibility of a causal association in humans. The study abstract from Steenland et al. was recently published on-line in the journal *Epidemiology*. Based on their meta-analysis of 25 studies (that included one previously excluded large study), Steenland et al. reported an association of -1.0 grams (95% CI -2.4, 0.4) per ng/mL PFOA. Restricting the studies to where blood samples for PFOA measurement were collected in early pregnancy (or even shortly before conception), the time period identified by Verner et al. in their PBPK simulations where confounding by maternal glomerular filtration rate would be of least concern, Steenland et al. reported a meta-analysis nonsignificant estimate of -3.3 gm (95% CI -9.6, 3.0) per ng/mL PFOA; thus further indicating a lack of an association between lower birthweight and PFOA.

Detailed Comments on PFOA MRL

ATSDR position (page A-16)

MRL Summary: A provisional intermediate-duration oral MRL of $3x10^{-6}$ mg/kg/day was derived for PFOA based on altered activity at 5–8 weeks of age and skeletal alterations at 13 and 17 months of age in the offspring of mice fed a diet containing PFOA on GD 1 through GD 21 (Koskela et al. 2016; Onishchenko et al. 2011). The MRL is based on a HED LOAEL of 0.000821 mg/kg/day and a total uncertainty factor of 300 (10 for use of a LOAEL, 3 for extrapolation from animals to humans with dosimetric adjustments, and 10 for human variability).

Selection of the Critical Effect: Intermediate-duration oral studies of PFOA in animals indicate that the liver, immune system, reproductive system, and the developing organism are the primary targets of toxicity because adverse outcomes were observed at lower doses than other effects and have been consistently observed across studies.

3M Conclusion

A. Studies by Onishchenko et al. (2011) and Koskela et al. (2016) should not be used to derive the PFOA MRL
B. The critical effects cited by ATSDR for the PFOA MRL derivation (altered activity and skeletal alterations in offspring in mice) were not supported by the available animal data, and they contradicted ATSDR's own evaluation of epidemiological data
C. PFOA does not affect the reproductive system in laboratory animals
D. The developmental effects reported in laboratory animals for PFOA were primarily mediated by maternal effects
E. Liver findings in rodents are not relevant for human risk assessment
F. Immune findings in rodents are not consistent; they lack concordance with epidemiological observation data
G. A study with one single dose group is not adequate in estimating point-of-departure
H. Serum PFOA concentrations in pups should be considered for POD instead of dams because critical effects chosen by ATSDR were based on (developing) pups
I. HED cannot be reliably estimated in the absence of serum concentration data
J. HED for PFOA will be higher when considering faster half-life
K. Wambaugh benchmark dose model used by ATSDR was not optimized
L. Uncertainty factors by ATSDR were conservative and not supported by scientific data
    1. Incorrect use of "10" for a LOAEL.
    2. Use of "3" for animal-to-human, in addition to large dosimetric TK adjustment, is conservative because humans are less sensitive than rodents with exposure to PFOA

ATSDR's overall interpretation on both toxicology and epidemiology data are inconsistent with the most current knowledge. Its application of uncertainty factors is not scientifically justified and the proposed PFOA MRL is not supported by the scientific data. The PFOA MRL derived for the human-health risk assessment is therefore inappropriate and not justified by an adequate scientific foundation.

3M Comments (Details):

A. Studies by Onishchenko et al. (2011) and Koskela et al. (2016) should not be used to derive PFOA MRL. The toxicology database for PFOA is quite comprehensive. Many of these studies included detailed information on the reproductive and developmental toxicity with these compounds across different PFOA dose levels as well as valuable insights on the role of maternal effects and its attribution to the developmental outcomes in laboratory animals. Comprehensive review on the potential developmental toxicity of PFOA in laboratory animals was reported in 2004 (Kennedy et al. 2004; Lau et al. 2004) and updated subsequently (Abbott 2015; Andersen et al. 2008; Lau 2012; Lau et al. 2007). Despite the wealth of data available, ATSDR chose mouse developmental studies reported by Onishchenko et al. (2011) and Koskela et al. (2016) as reference studies for its derivation of PFOA MRL (based on altered activity and skeletal alterations seen in offspring in mice).

   ATSDR's assessments on these studies (and the corresponding reported critical effects) failed to make clear to the public that the proposed MRL did not reflect the absence of an association between PFOA exposure and musculoskeletal outcomes or neurological outcomes in humans (cf. pages 141 – 145; pages 293-296). Furthermore, there are major technical concerns associated with these studies that preclude the results (from these studies) to be meaningful in any human risk assessment. They include:

   1. They are the same study. Albeit published five years apart, these two publications actually originated from one single study. From the same pregnant dams treated with dietary PFOA during gestation, the pups evaluated by Onishchenko et al. (2011) were litter-mates of the pups evaluated by Koskela et al. (2016). As such, it was really one study (in essence) and the corresponding outcomes (from both studies) should be consolidated when discussed.

   2. A single dose experiment cannot address (any) dose-response relationship. There was only one PFOA dose group used in these two studies and as such, it is impossible to interpret the experimental data reported by these authors in terms of any dose-response. Considering the inherent variations in biological responses in any animal study, the nature of a single-dose study simply does not allow any specific evaluation of any dose-and-effect responses or biological plausibility inference.

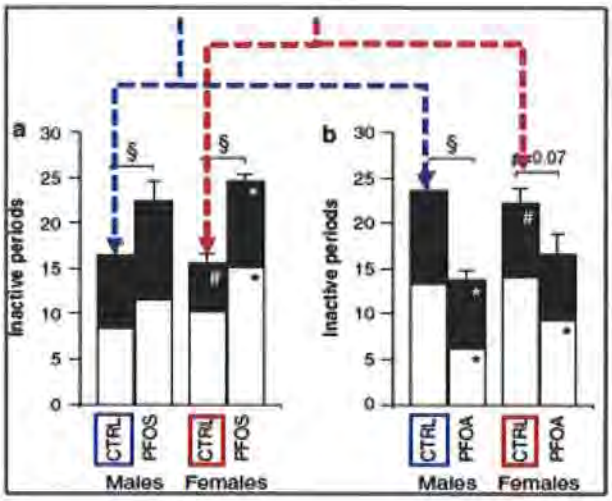
      Using a study that evaluated a single PFOA dose group was in absolute contradiction of what ATSDR stated in its MRL approach. On page A-6 of the draft profile, ATSDR explicitly stated that one of the MRL approach was to "*Identify laboratory animal studies that have evaluated dose-response relationship for toxicity targets identified in epidemiology studies*".

      Hence for PFOA, not only did ATSDR not identify musculoskeletal or neurological outcomes as sensitive endpoints in humans; it did not select a laboratory animal study that appropriately addressed or evaluated dose-response relationship.

3. The study design was flawed and insufficient to support a NOAEL or LOAEL. Again, given that there was only PFOA dose group used, the study design did not follow the fundamental practice of toxicology testing such as evaluation of a dose response relationship. Hence, given the lack of any dose-response, it is scientifically impossible to establish a realistic NOAEL and/or LOAEL for the data reported.

4. Limited sample size. There were only 6 dams that received PFOA diet to produce the pup cohort, and there was a total of 10 dams that received control diet; however, the control animals spanned from two (separate) blocks of individual experiments. The sample size for the study was quite small and given that only a single PFOA dose group was used, it is impossible to properly address biological plausibility (if any) and background variability.

   For example, regardless of sex, Onishchenko et al. (2011) reported a statistically significant difference between control and PFOA pups for the number of inactive periods (Figure 3b). However, on the accompanying graph (Figure 3a), they also reported a statistically significant difference between control and female pups from PFOS dose group for the number of inactive periods. Without looking at the treatment groups and just comparing the sex-matched control responses alone between Figure 3a and Figure 3b (see illustration below), it became very apparent the large variations exist even in the sex-matched control animals. This large variation (on the background control alone) most likely attributed to the statistical significance when compared to the treatment groups (either PFOS or PFOA).



Another similar example is on the body weight. The absence of statistical power to address inherent biological variations due to the limited study design did not allow for a valid comparison of biological responses between control and treatment. While Koskela et al. (2016) reported an increase in the body weight in the female pups from PFOA-

treated group with statistical significance at 13 months and 17 months; however, the difference was already present at birth (as stated by the authors) hence the reported difference may well have reflected normal variation which cannot be adequately demonstrated as there were insufficient animals and litters.

5. Lack of reproduction (pregnancy) outcome information. Given the study design included the gestation and lactation periods, it was perplexing that very little information on the pregnancy or lactation outcomes were discussed by the authors (*e.g.*, gestation length, number of implantation, litter size, sex ratio, or lactation performance). All these are critical in evaluating the quality of the study.

6. Lack of litter outcome information. Given the study design included the developmental phase of pups, it was also perplexing as to why the authors did not disclose any detailed litter outcomes from dams received PFOA treatment (*e.g.*, survival, birth weight, anogenital distance, nipple retention, onset of number of implantation, gestation length, litter size, sex ratio, onset of sexual maturation…etc.) All these are critical in evaluating the quality of the study.

7. Questionable pup selection bias / litter bias. It was unclear as to how the pups were selected for the evaluations. To rule out litter-related effects, it is a standard practice for pups from the same litter to be evaluated as one single unit (rather than individual pups) in the assessment of reproductive and developmental outcomes in laboratory animals (OECD 2007, 2016). Given that there were only 6 dams that received PFOA treatment, therefore, the maximum number of pups from PFOA dose group should be 6 (*i.e.*, one pup per litter). Depending on the endpoints, the authors reported the data based on 6 – 10 pups, which would indicate that the pup selection was confounded by litter effect; and subsequently, the study findings were also confounded by litter effects.

8. Questionable dietary preparation. In the studies by Onishchenko et al. and Koskela et al., pregnant dams were administered with dietary PFOA throughout gestation for a total of 21 daily doses (as described by Koskela et al. 2016). According to the study authors, PFOA was dissolved in 95% ethanol first and then applied on food pellet. The pellets were kept on the bench for 2 hours (presumably at room temperature) to allow for ethanol evaporation prior to feeding them to the animals.

   This was a very crude method of preparing a dietary formulation – there were no information on the final PFOA concentration achieved in the diet and there was no information on the homogeneity distribution of PFOA in the diet. All these parameters were essential in contributing to a good dietary study and none of the information was available or explained by the study authors.

9. Possible residual ethanol present in the dietary PFOA chow. In addition to the crude dietary preparation method, the study authors assumed that the 95% ethanol used to dissolve PFOA would have been completely evaporated within 2 hours after sitting on the bench (presumably at room temperature), however, there were no supporting data to prove this. It is well-known that pure ethanol does evaporate faster than water on the

basis of higher vapor pressure, lower boiling point, and less hydrogen bonds (Innocenzi et al. 2008). When ethanol is mixed with water, more hydrogen bonds are created; and when ethanol-in-water mixture is further mixed with PFOA as well as applied onto the surface of food chow (such as this study), the additional intramolecular forces (between ethanol and water, ethanol-in-water and PFOA, and, ethanol-in-water and PFOA and food chow ingredients) would have reduced the overall volatility of ethanol. The authors should have obtained a quantitative measurement of the PFOA/chow mixture to demonstrate the absence of ethanol after 2-hour evaporation.

This verification step was critical for this study because the authors evaluated and reported neurobehavior endpoints as findings. Albeit the control animals also received food chow diet that had been applied with 95% ethanol followed by evaporation, however, the intramolecular force between ethanol, water and food chow (i.e., control food chow) would be different than the intramolecular force between ethanol, water, PFOA, and food chow (i.e., PFOA food chow). Given that ethanol is well-known for its effects on the central nervous system (Boschen and Klintsova 2017; Harrison et al. 2017) and 95% ethanol was used in the study, any ethanol that had not evaporated and remained on the food chow could have confounded the study results, especially on the neurobehavior parameters.

10. There were no serum PFOA data reported in these studies. ATSDR has determined that, rather than relying on external dose, serum PFOA concentration (internal dosimetry) is the appropriate exposure matrix when determining a point-of-departure (POD) for the MRL derivation with PFOA (*cf.* page A-16 and Table A-7 on page A-24 of the draft profile). Neither Onishchenko et al. (2011) or Koskela et al. (2016) reported any information on the serum PFOA concentrations; and this was a major deficiency of the study. Even though ATSDR "estimated" the time-weighted-average serum PFOA concentration based on its PBPK model, the absence of serum PFOA data preluded the verification of the ATSDR PBPK model, in addition to the other unknowns associated with the study (*i.e.*, no dose-response and no dose verification).

    It is also worth noting that the study authors had the technical capability to perform PFOA analysis because Onishchenko et al. (2011) reported PFOA concentrations in a subset of pup brain and liver samples.

11. Timing of behavior assessments in pups were not appropriate. In the study data reported by Onishchenko et al. (2011), numerous neurobehavior endpoints were evaluated by the study authors. Given that the study was done under non-GLP protocols and by a university research lab(s), most of the timings and behavior assessment procedures (as described by the study authors) did not appear to follow the conventional recommendations and methodology. As a result, it is difficult to determine the quality of the data that had been reported. For instance, compared to the OECD 426 test guideline (TG) for developmental neurotoxicity study (OECD 2007), these authors did not follow standardized timeline recommended to FOB evaluations for the developing pups. The table below is a side-by-side comparison between the OECD 426 TG recommendation timeline vs. what Onishchenko et al. did. It was apparent that Onishchenko et al. had

missed critical windows for the assessments on many key parameters (i.e., no behavior assessments were done prior to weaning) and there were no specific references or rationales to explain or justify their study design.

	OECD 426 TG Recommendation for developmental neurotoxicity study	Study by Onishchenko et al. 2011
Dosage	Control + 3 dose levels	Control + 1 dose level
Animal number	20 litters / group	6 litters / group
Detailed clinical observation	20 pups /sex (1 / sex/ litter)	6 – 10 pups / sex
Brain weight PND 11-22	10 pups / sex (1 / litter)	No data reported
Brain weight PND 70	10 pups / sex (1 / litter)	No data reported
Neuropathology PND 11-22	10 pups / sex (1 / litter)	No data reported
Neuropathology PND 70	10 pups / sex (1 / litter)	No data reported
Sexual maturation	20 pups /sex (1 / sex/ litter)	No data reported
Behavioral ontogeny (e.g., righting and reflex)	2X prior to weaning at PND 21	No data reported
Motor activity	1-3X prior to weaning at PND 21; 1X during PND 60-70	None prior to weaning; 1X during PND 35 – 56;
Motor and sensory function	1X during PND 23-27; 1X during PND 60-70	None prior to weaning; 1X during PND 90 - 120
Learning and memory (~ PND 23-27 and 60-70)	1X during PND 23-27; 1X during PND 60-70	None prior to weaning; 1X during PND 35 – 56;

12. Non-standard behavior assessment procedures used in pups. Among the behavior endpoints evaluated by Onishchenko et al., given that the study was done under non-GLP by university research lab(s) and it did appear that the tests were done on a single day without further repeat(s) later, it raised the question as to the overall reliability and reproducibility of the instruments and the corresponding data generated.

    For instance, to measure and record circadian activity in the home cage, the TrafficCage™ used by Onishchenko et al. is shown in the picture below (obtained from manufacturer's website). Compared to the conventional 3-D photo beam boxes where movements were recorded in vertical, horizontal, and lateral directions, the TrafficCage™ system lacks the ability to measure any vertical movements. In addition, the TrafficCage™ system has several "dead spots" without any sensors. The validity of the instrument and the corresponding results generated (circadian activity) are questionable.

Illustration of TrafficCage™

(Source: https://www.tse-systems.com/product-details/phenoworld/trafficage?open=3806#trafficage-3806)

13. No information on background data for bone morphology and bone density. Koskela et al. (2016) reported that female offspring from PFOA-treated dams had increased femoral periosteal area and decreased mineral density of tibias, hence ATSDR concluded that "skeletal alterations in offspring" was a critical effect with PFOA exposure in mice.

    Bone morphology is a collective description on the shapes (geometry) of the bones, such as long bones (*e.g.,* femur and tibia), short bones (*e.g.*, bones of the feet and hands), or flat bones (*e.g.*, calvaria or breast bones). There are many factors contributing to the morphological sizes of the bones. The morphology of bone is not a "fixed" static structure, rather, it is a composite structure that will continue to evolve like other organs in the body. While the components of the bones are maintained in a balanced manner, there are also inherent biological variability within each component that needs to be taken into account when determining the overall homeostatic status of the bones (Boskey and Coleman 2010; Jepsen 2009).

    It is well-known that age and body weight are two factors in establishing the size, mass, and strength of the bones (Iwaniec and Turner 2016). In the data reported by Koskela et al., there was a pre-existing difference in body weight in female pups at birth where higher body weight was consistently observed in these female pups from PFOA-treated groups; and that difference reached statistical significance at 13 months and 17 months (*vide supra*). Therefore, it should not be a surprise that increased bone sizes in offspring with higher body weight (*e.g.*, offspring from PFOA-treated dams) had increased periosteal and medullary areas in both femurs and tibias. On the other hand, given the small sample size of the animals used in this study, the inherent background variation cannot be ruled out. For example, compared to control, the study authors also reported a decrease in mineral density in tibias in offspring born from PFOA-treated dams. The extent of decrease was very minor (only 2.5%) and it was only observed in tibias, not in femurs. Because the study authors did not have any additional information on the

background data with regards to these parameters, this minor difference may be well within the normal biological variations (again, especially with such small sample size).

14. Mechanical determinants of bone functions were not affected in pups from PFOA-treated dams. Based on study data reported by Koskela et al. (2016), ATSDR concluded that there were skeletal alterations in offspring from PFOA-treated dams and deemed it to be a critical health effect. However, in the same cohort of pups, Onishchenko et al. (2011) reported motor and sensory function assessments (muscle grip strength and rotarod test) and found no differences in the outcomes between control and PFOA-treated groups. Given that muscle force is a strong determinant of bone integrity, the slight morphological difference noted by ATSDR possibly reflected the normal background variations in this strain of mice and not likely due to PFOA.

15. Lack of supporting evidence on the effect of PFOA and bone development. If PFOA exposure does have a direct (causal) effect on the bone development, then one would expect such effect to be even more pronounced under longer (repeated) dose conditions. This was not the case, as long-term toxicology studies in rodents and non-human primates have not identified bone as a target tissue with exposure to PFOA (Biegel et al. 2001; Butenhoff et al. 2002; Butenhoff et al. 2012b).

16. Other technical comments about the study data by Koskela et al. (2016).

    - In addition to the likely litter-bias that has been discussed earlier, it is unclear why Koskela et al. only included female offspring in their evaluation but not male offspring.

    - PFOA has a high affinity to binding with serum albumins and given that bone marrow is the hemopoietic origin of blood, one should not be surprised to find trace level of PFOA in the bone. Albeit Koskela et al. claimed that bone marrow had been flushed out and only the hard bones were powdered and analyzed for PFOA content, it is important to recognize that the bone consists of "live" mesenchymal cells with lots of protein components (chondrocytes, osteoblasts, and osteocytes), not just marrow (Boskey and Coleman 2010; Iwaniec and Turner 2016; Jepsen 2009).

    - The study authors only evaluated long bone morphology but not others. If bone is indeed a target tissue with exposures to PFOA, other bones (in addition to femur and tibia) also need to be included in the evaluation.

    - It is well-known that there are large inter-species differences in bone composition, density, quality, as well as genetic variability within the same species (Aerssens et al. 1998). Again, if bone is indeed a target tissue with exposures to PFOA, such cause-and-effect needs to be demonstrated in a dose-response fashion within the same animal model as well as other species.

- Other factors that can affect bone morphology and density should also be comprehensively evaluated before drawing a conclusion. For example, endocrine effects such as estrogen and IGF-1, essential nutrient status such as calcium and vitamin D3.

- The use of imaging devices in the assessment of bone morphology is not a new concept, and CT images have been used in both clinical settings as well as research settings. However, similar to the comments provided above on the behavior assessments provided above, Koskela et al. should have demonstrated that the validity of the micro-CT scanning technique used in their facility as well as their competency in using the instrument. Given the fact that a very small magnitude of surface area was being reported as a "statistically significant" change (in the range of 0.2 – 0.3 mm^2), it is important to validate the sources of these measurements. For example, was the instrument calibrated? Were the operator(s) trained in using the equipment? Were the acquired images analyzed by qualified radiologists who are trained in doing image interpretation?

- For any imaging-based scanning, it is absolutely critical that the object (or subject) remained steady for the duration of the scanning acquisition. Any movement during the scanning process will deviate the result. The study authors described that the bone was "*wrapped in a PBS-moistened tissue paper and inserted into a plastic tube, with the proximal end pointing upwards. The container was then placed into the chamber of the microCT device*". The description did not address attempts to prevent any movement of the bone (inside the plastic tube) during the scanning process. Given the asymmetrical shape of femurs and tibias, it is important to immobilize the bone inside the tube and any slight shift will artificially affect the image data during scanning.

Overall, the studies by Onishchenko et al. (2011) and Koskela et al. (2016) lacked scientific rigors to properly address the selected developmental endpoints and they should not be used for any human risk assessment.

B. The critical effects cited by ATSDR for PFOA MRL derivation (altered activity and skeletal alterations in offspring in mice) were not supported by available animal data and contradicted ATSDR's own evaluation of epidemiological data. There is insufficient evidence for an association between PFOA exposure and musculoskeletal outcomes or neurological outcomes in humans (cf. pages 141 – 145; pages 293-296). ATSDR should offer a plausible explanation as to why it believes these effects are relevant to human risk assessment.

C. PFOA does not affect the reproductive system in laboratory animals. It is incorrect for ATSDR to conclude that the reproductive system is one of the primary targets of toxicity with exposure to PFOA (cf. page A-16).

On the contrary, PFOA did not affect the functional aspects of male or female reproduction in laboratory animals. These included estrous cycles, sperm parameters, mating index, fertility index, and reproductive organ morphology. A number of studies on the reproductive

and developmental effects of PFOA in laboratory animals have been published (Abbott et al. 2007; Albrecht et al. 2013; Butenhoff et al. 2004; Gortner 1981, 1982; Lau et al. 2006; Staples et al. 1984; Yahia et al. 2010). Many of these studies included detailed information on the reproductive and developmental toxicity with these compounds across different PFOA dose levels as well as valuable insights on the role of maternal effects and its attribution to the developmental outcomes in laboratory animals.

The potential of PFOA to influence reproductive performance has been evaluated in mice, rats, and rabbits. Gestational exposure to ammonium PFOA did not affect the number of uterine implantation sites in various strains of mice such as CD-1, Sv129, PPARα knockout, and humanized PPARα (Abbott et al. 2007; Albrecht et al. 2013; Lau et al. 2006; White et al. 2007). At inhalation dose up to 25 mg/m^3/day of ammonium PFOA or oral doses up to 100 mg/kg/day given during gestation to rats did not affect mating, pregnancy, and implantation (Staples et al. 1984). Oral administration of ammonium PFOA up to 150 mg/kg/day in rats or 50 mg/kg/day in rabbits during GD 6 – 15 (period of organogenesis) also caused reduced body-weight gain, however, they did not affect the ovaries or the reproductive contents of the dams (Gortner 1981, 1982). In a two-generation reproduction/developmental study in rats (Butenhoff et al. 2004), the reproductive outcome was not affected with daily oral ammonium PFOA administrations up to 30 mg/kg/day (the highest dose used in the study). There were no effects on the mating or fertility indices in either male or female rats. Male rats had normal sperm parameters (count, motility, morphology) and female rats had regular estrous cycling with normal gestation lengths, and microscopic examination did not reveal any abnormalities in sex organs. Furthermore, effects of PFOA on reproductive organ morphologies in male non-human primates were evaluated from a six-month oral study and results indicated no abnormalities (Butenhoff et al. 2002).

D. <u>The developmental effects reported in laboratory animals for PFOA were primarily mediated by maternal effects</u>. While ATSDR concluded that developing organisms are primary targets of toxicity with exposure to PFOA (cf. page A-16), there are strong experimental evidences demonstrating that developmental effects associated with PFOA exposures in offspring are observed <u>only</u> where there were significant effects in the maternal animals. Because neither Onishchenko et al. (2011) nor Koskela et al. (2016) reported detailed maternal-related endpoints with regards to reproduction, no maternal influence discussion is possible. However, observations involving maternal effects in the outcome of the developmental toxicity, as seen in the disruption of maternal homeostasis, include the following examples:

Using the mouse developmental study data reported by Lau et al. (2006), which was the critical study chosen by U.S. EPA Office of Water for the derivation of the Lifetime Water Health Advisory for PFOA issued in 2016, there were statistically significant ($p \leq 0.05$), dose-related increases in maternal liver weight observed at doses 1 mg/kg/day ammonium PFOA or higher (the corresponding serum PFOA concentration was 21,900 ng/mL at the end of gestation). Various developmental effects were reported (*e.g.*, decreased postnatal survival, decreased body weight at birth and body-weight gain thereafter, and delays in eye openings) and they were only for litters from dams receiving 3 mg/kg/day or higher. Maternal responses clearly were present at doses that affected the fetus/neonate. In addition,

because the influence of body weight on sexual maturation is well-described in the literature, it is not surprising that Lau et al. noted altered pubertal maturations in the offspring.

The developmental toxicity of ammonium PFOA has also been studied in rats (Butenhoff et al. 2004; Gortner 1981; Staples et al. 1984) and rabbits (Gortner 1982). In these studies, no increase in malformations relative to controls was observed at oral doses up 150 mg/kg/day in rats and 50 mg/kg/day in rabbits, as well as inhalation concentrations up to 25 mg/m^3/day (6 hours/day). In the studies by Gortner and by Staples et al., any effects on fetal or pup body weight were present at dose levels equivalent to or higher than those causing effects such as body weight in the maternal animals. In a two-generation reproduction and developmental study in rats (Butenhoff et al. 2004), F1-generation pups from the highest dose group (30 mg/kg) had decreased birth weight and reduced viability that were in apparent relationship to the corresponding reduced body weight at birth and weaning. These latter effects are similar to those observed in mice by others (Abbott et al. 2007; Lau et al. 2006; Yahia et al. 2010). Even though similar to the observation by Lau et al. (2006) in that sexual maturation were slightly delayed (at the highest dose group only), there was no significant difference in F1 pups when days to sexual maturation was adjusted by (reduced) body weight.

Based on data from the large scale 2-generation reproductive and developmental studies (which are considered as the most comprehensive test by various agencies for evaluating endocrine functions), PFOA clearly did not alter the reproductive functions as the reproductive performances in both males and females were normal (*vide supra*). In addition, there is sufficient evidence in experimental animals (mammals) to suggest that rodents may not be the best model in evaluating the reproductive-related outcomes for human risk assessment. PFOA is a known activator for xenosensor nuclear receptors such as PPARα, constitutive androstane receptor (CAR), and pregnane X receptor (PXR) (Corton et al. 2014; Elcombe et al. 2010; Elcombe et al. 2014; Klaunig et al. 2003; Klaunig et al. 2012). It is well documented that PFOA causes hepatomegaly in rodents as a result of PPARα activation with some contribution from CAR and PXR. It is well-known that human liver is less responsive to the pleiotrophic effects of activation of PPARα or CAR (Gonzalez and Shah 2008; Klaunig et al. 2003; Klaunig et al. 2012; Lake 2009; Ross et al. 2010). Thus, with respect to PPARα and CAR-mediated effects in the liver and related metabolism, the human response is either attenuated or absent as compared to that of the rodents.

Mechanistic studies have demonstrated that many of the observed effects upon PFOA exposure, including those observed in developing mice, can be explained, in part, by the activation of PPARα. Many of the developmental effects were either absent or attenuated when PFOA was administrated to PPARα knockout mouse. The influence of PPARα on the fetal developmental effects of PFOA in the Sv/129 mouse strain (wild-type vs. PPARα knockout) was investigated by Abbott et al. (2007) and Albrecht et al. (2013). While it is not possible to rule out completely the contribution of other modes of action(s), many of the developmental effects with PFOA described above were attenuated and/or improved with PPARα knockout mice such as post-natal survival and body weight effects. Given that rodents are more responsive and susceptible than humans to PPARα-mediated biological effects (*vide supra*) and PPARα may not play a critical role in normal development

(Braissant et al. 1996; Lee et al. 1995), it calls into question the relevance of nuclear receptor-mediated effects in rodents and their biological significance to humans. Therefore, the developmental effects reported in the laboratory animals for PFOA were primarily mediated by maternal effects and based on the recent mode of action data, rodents may not be the most appropriate species for the hazard assessment of PFOA on developmental toxicity in humans.

E. Liver findings in rodents are not relevant for human risk assessment. While it is commonly acknowledged that liver is a primary target organ with exposure to PFOA, it is important to recognize that the liver effects observed in laboratory animals were adaptive in nature and there was no conclusive evidence to support that liver findings observed in laboratory animals with exposure to PFOA are relevant for human risk assessment. Given the known knowledge on the nuclear receptor activation and species relevance discussed earlier (*vide supra*), liver findings cited by ATSDR should not be deemed relevant for human risk assessment. For instance, in the study by Butenhoff et al. (2004), increased liver weights were reported in male rats of both the P and F1 generations at all dose levels.

The corresponding increases in liver weight in laboratory animals with exposure to perfluoroalkyls reflected the adaptive nature of liver, which is a natural phenomenon due to cytochrome P450 enzyme inductions in the liver. Given that PFOA is a known activator for several xenosensor nuclear receptors (as discussed above), microscopic changes in the liver of some PFOA-treated male rats such as hepatocellular hypertrophy and focal to multifocal necrosis were consistent with activation of these receptors and as discussed earlier, it is well-known that human liver is less responsive than rodents to the pleiotrophic effects of activation of these receptors (Gonzalez and Shah 2008; Klaunig et al. 2003; Klaunig et al. 2012; Lake 2009; Ross et al. 2010). Thus, with respect to PPARα and CAR-mediated effects in the liver and related metabolism, the human response is either attenuated or absent as compared to that of the rodents. Another federal agency, USEPA (in its assessments of PFOA in 2009 and again in 2016), as well as other international regulatory authorities such as European Chemical Agency Risk Assessment Committee (2015), European Food and Safety Authority (2018), and Australian Expert Health Panel (2018) also considered the liver weight findings in laboratory animal studies with PFOA (or other perfluoroalkyls) to be irrelevant for human risk assessments.

It should be noted that, acetylsalicylic acid (commonly known as aspirin) and alcohol can also elicit increased liver weight in laboratory animals similar to the observations reported with perfluoroalkyls in rodents (EMEA 1999b).

F. Mammary gland development findings in mice are inconsistent: Despite that the availability of several studies that have investigated the potential effects of PFOA on the developing mammary glands in mice as a consequence of exposure during either the *in utero* or postnatal/peripubertal (Albrecht et al. 2013, Tucker et al. 2014, White et al. 2007, White et al. 2009, White et al. 2011, Yang et al. 2009, Zhao et al. 2010), ATSDR is correct that this endpoint *cannot be consistently* described and quantified in mouse models. Given that 1) to

date, there is no standardized method or guideline of evaluating rodent mammary gland; and 2) there is a lack of concordance among all the available data on mammary gland development in mice as well as an absence of such findings in human epidemiological studies calls for question on the biological significance of this phenotype and its relevance to human health. This conclusion is consistent with the assessments from another federal agency, USEPA (in its assessments of PFOA in 2009 and again in 2016), as well as other international regulatory authorities such as European Chemical Agency Risk Assessment Committee (ECHA 2015), European Food and Safety Authority (EFSA 2018), and Australian PFAS Expert Health Panel (2018).

It should be noted that there are three epidemiologic studies that have examined the potential association between maternal PFAS exposure and shorter duration of breastfeeding or greater risk of stopping breastfeeding (Fei et al. 2010b; Romano et al. 2016; Timmermann et al. 2016). Fei et al (2010) measured PFOA and PFOS concentrations of 1400 women during early pregnancy. Self-reported data on the duration of breastfeeding (any and exclusive) were collected around 6 and 18 months after birth. While the study reported significant associations between PFOA concentrations and shorter duration of breastfeeding (before 3 and 6 months) among multiparous women, no significant associations were observed among primiparous women. The authors note that multiparous women who breastfed during prior pregnancies or breastfed longer may have had lower serum PFOA levels through excretion via breast milk. Consequently, reverse causation could not be excluded. The second study (Romana et al. 2016), observed a significant association between PFOA exposure and ending "any" breastfeeding by 3 and 6 months; however, no association was observed between PFOA exposure and ending "exclusive" breastfeeding by 3 and 6 months. More importantly, when stratified by parity, associations between PFOA and ending "any" breastfeeding at 3 and 6 months were largely attenuated for nulliparous women. Like Fei et al (2010), the significant associations observed among multiparous women were likely attributed to reverse causation. The third study (Timmerman et al. 2016), examined the potential association between PFOA exposure and duration of breastfeeding (both total and exclusive) among 1092 Faroese women with general population PFOA levels (median = 2.40 ng/mL). The authors reported that a doubling of maternal serum PFOA was significantly associated with a reduction in exclusive breastfeeding of 0.5 months. This association was observed among both primiparous and multiparous women (excluding the role of reverse causation). One important limitation of this study, worth noting, is that self-reported breastfeeding duration was collected 5 years after birth and was likely prone to misclassification error.

Finally, it is important to recognize that reduced breastfeeding duration in humans is not equivalent to "delayed mammary gland development" in rodents. In humans, numerous factors can influence breastfeeding duration other than diminished milk production (e.g., lack of prenatal education, inadequate lactation support from healthcare providers after delivery, medications incompatible with breastfeeding, lack of spousal/family support, short maternity leave, sore nipples/breasts, infant intolerance to breast milk, and individual choice). These factors were not considered in the epidemiology studies, and may have influenced the observed associations.

G. Immune findings in rodents are not consistent; and they lack concordance with epidemiological observation data. With exposure to PFOA, ATSDR also concluded that immunotoxicity is a primary target of toxicity based on decreased antigen-specific antibody responses in mice reported by DeWitt et al. (DeWitt et al. 2008; DeWitt et al. 2016) where PFOA suppressed T cell-dependent IgM antibody response (TDAR) but not the secondary IgG response. While ATSDR concluded that such findings were consistent with human epidemiology studies with regards to vaccine responses (see epidemiology discussion below), it is important to recognize that the humoral immune response to vaccinations, as measured in the human epidemiology studies, is mainly a secondary IgG memory response.

While suppression of the IgM response by PFOA was demonstrated in several studies where administered doses also induced signs of overt toxicity (i.e., reductions in body and lymphoid organ weight), the levels of IgG were not suppressed (either unchanged or enhanced). It is difficult to interpret why the primary IgM response was suppressed in mice by PFOA and yet the secondary IgG response was either not affected or enhanced. Collectively, human and animal bodies of evidence for antibody response are divergent. Mouse studies showed suppression of the IgM response with no impairment of the secondary antigen specific IgG response, which is in contrast to the epidemiological associations which suggested suppression by PFOA of IgG-mediated antibody titers to vaccinations in some studies for certain vaccines. Therefore, the weight of evidence and the lack of concordance between animal and human epidemiological data do not support the claim that PFOA induces immunotoxicity or caused decreased antibody response to certain vaccines. Finally, as noted above, the fact that the epidemiological data does not reveal a consistent association between exposure and response across all vaccines is further evidence that the animal and human data are not consistent.

Contrary to what ATSDR stated "the potential immunotoxicity of PFOA has not been investigated in chronic-duration studies" (*cf.* page A-30), it should be noted that the primary immune organs were evaluated microscopically in rats after 2 years of dietary treatment containing ammonium PFOA (Butenhoff et al. 2012c). In this study, representative primary immune organs were collected (mesenteric lymph node, spinal cord, bone marrow, and spleen) and evaluated microscopically by a board-certified veterinary pathologist at the end of a 2-year period. There were no neoplastic or non-neoplastic lesions observed in these immune organs. This is important because it demonstrated the absence of a direct effect on primary immune organs with chronic PFOA exposures in the rats. In addition, PFOA-treated rats had similar or higher percent survival compared to controls, which is contrary to chronic immunosuppression-mediated toxicity such as cyclosporin (a known immunosuppressant) that ultimately resulted in increased mortality in rats (Ryffel and Mihatsch 1986).

H. A study with one dose group is not adequate in estimating point-of-departure. ATSDR selected two mouse studies with developmental endpoints (Onishchenko et al 2011 and Koskela et al 2016) for the point-of-departure (POD) to derive the MRL value for PFOA (endpoints were altered activity and skeletal alterations in offspring of C57Bl/6 mice). These studies tested only a control group and one dose of 0.3 mg/kg, which was chosen as the LOAEL. As only one dose was tested, a dose-relationship cannot be evaluated.

Selection of studies with no information on dose-response for effects is not acceptable to establish a point-of-departure. ATSDR should follow its own guidance (as stated in pages A-6).

I. Serum PFOA concentrations in pups should be considered for POD instead of dams because critical effects chosen by ATSDR were based on (developing) pups. The studies chosen by ATSDR examined developmental endpoints that were measured in offspring, which are used as the basis for the MRL. In order to estimate steady-state plasma concentrations of PFOA, ATSDR used the Wambaugh model for PFOA that is parameterized for adult animals and cannot be used to predict concentrations in fetuses or pups. This model also does not account for life stage differences in physiology or pharmacokinetics, and can potentially over-predict as well as under-predict the area-under-the-curve (AUC). In addition, AUC and steady-state concentration are probably different in the offspring than in the dam. Overall internal exposure (as estimated by calculation of the AUC) may change with growth, and there could be a period of peak exposure. Use of the Wambaugh model (and thus use of the maternal plasma concentration as a surrogate for the offspring) introduces uncertainty in the MRL derivation as the offspring plasma concentration may be different that than of the maternal animals. Use of a physiologically-based model that incorporates fetal and pup compartments would provide an estimate of fetal and pup internal exposure (rather than use of the maternal concentration as a surrogate), which would reduce the uncertainty in the MRL value.

J. HED cannot be reliably estimated in the absence of serum concentration data. As discussed above, studies by Onishchenko et al. (2011) and Koskela et al. (2016) did not have any analytical verification on either the dietary PFOA level or the resulting serum PFOA concentrations in the mice. With the questionable reliability of the study design as well as the data gathered, there were a great number of inherent uncertainties associated with attempting to predict the mean serum concentrations using modeling approach.

Confirming that it is inappropriate to derive an MRL where there is an absence of serum concentration data, in its current draft profile for other perfluoroalkyls, ATSDR stated in several places that "…. Database was considered inadequate for derivation of an MRL … because … study did not measure serum [perfluoroalkyl] levels, which are needed to calculate / estimate HEDs" (cf. pages A-14, A-56, A-65, A-72, A-109).

K. HED for PFOA will be higher when considering faster half-life. In the MRL calculations, ATSDR chose to use the arithmetic mean serum elimination half-life estimate for PFOA from Olsen et al. (2007) over other studies because Olsen et al. had a longer follow up time and ATSDR was concerned that based on a study by Seals et al. (2011), slower kinetics is likely to constitute a larger contribution to the terminal half-life. For example, whereas Olsen et al. had an average follow-up of 5 years, Bartell et al. had a follow-up of a year and Li et al. had a follow-up of 2.3 years among those studies that followed individuals and were not cross-sectional analyses of populations. However, this line of reasoning by ATSDR for selection of the arithmetic mean from the Olsen et al. study fails to take into account several factors that likely biased upwards the ATSDR MRL estimates. These include the following points.

1. The ATSDR chose not to use the geometric mean estimate that was discussed in the Olsen et al. paper. Given the right skewness of their data, Olsen et al. were more favorable to use the geometric mean for a measure of central tendency. ATSDR provided no explanation as to why they chose the arithmetic mean vs. the geometric mean in this study. This decision is interesting (and curious) because ATSDR chose to report median initial and final concentrations in Table A2 rather than the arithmetic mean initial and final concentrations in Table A2. A median concentration would be better represented by a half-life estimate based on the geometric mean.

2. The Olsen et al. 2007 study comprised 26 retirees (end of study average age = 66 years) who likely would have had an average glomerular filtration rate lower than those calculated from younger ages as reported in Bartell et al. (average age 55) and Li et al. (age range 15 – 55). The average estimated glomerular filtration rate declines with age as shown in the table below.

Age range	Estimated GFR (ml/min/1.73 m^2)	Source:
1-6 months	77	Heilbron et al. 1991 Pediatr Nephrol. Jan; 5(1):5-11.
6-12 months	103	
12-19 months	127	
2-12 years	127	
20–29	116	https://www.kidney.org/sites/default/files/docs/11-10-1813_abe_patbro_gfr_b.pdf
30–39	107	
40–49	99	
50–59	93	
60–69	85	
70+	75	

Renal clearance of perfluorocarboxylates (and perfluorosulfonates) is largely a sum of three processes involving glomerular filtration, renal tubular secretion, and renal tubular reabsorption (Han et al. 2012). Because PFOA and other perfluorocarboxylates vary in their affinities to bind plasma proteins, glomerular filtration of perfluorocarboxylates (and perfluorosulfonates) is a product of the unbound fraction of the perfluorocarboxylate and the glomerular filtration rate (GFR). Thus, the higher estimates of GFR based on the younger ages in the other study populations, especially the younger Li et al. study which had approximately 50% of the follow-up time of Olsen et al., may be due to the age differences of the subjects, and not necessarily the shorter follow-up period considered in these studies. Thus, the serum elimination half-lives of other studies are likely equally valid for consideration in MRL calculations.

3. The Olsen et al. study had to consider, during the course of their follow-up, the possibility of retirees reentering the 3M Decatur and Cottage Grove manufacturing plants. Indeed, this resulted in Olsen et al. eliminating 1 study subject entirely, and truncating follow-up times for two retirees. This would have biased estimates upwards for the serum elimination half-lives due to the increased exposure. It is not likely that

ambient general population level concentrations would have biased these retiree's estimates substantially as discussed by Bartell et al. 2012. On the other hand, although Bartell et al. and Li et al. had shorter follow-up times, the primary exposure in these populations was through drinking water. Installation of GAC filters in these populations' affected municipal water supply would have immediately ceased their primary exposure to PFOA, PFOS, and PFHxS.

4. ATSDR suggests the Seals et al study indicates a lower clearance rate may occur as subjects are followed long-term post exposure; thus, the decision by ATSDR to use the study that had the longest follow-up time (Olsen et al. 2007). However, ATSDR did not mention the main limitations of the Seals et al. study: 1) the cross-sectional nature of the analysis. Individual subjects were not followed. Model-based estimates were instead calculated based on the initial concentrations; 2) there was the added assumption that there was uniform exposure based on the concentration of PFOA measured in each water district; and 3) subjects with initial PFOA concentrations < 15 ng/mL were excluded which maximized the probability of analyzing individuals with sufficiently high baseline PFOA concentrations that would not be at ambient levels. Seals et al. surmised their findings indicated the half-life for PFOA was between 2.3 and 3.8 years, not at the end of this range, as chosen by ATSDR via the arithmetic mean estimate from Olsen et al. Seals et al. did show their modeled estimates in clearance rates between low- and high-exposure water districts could suggest a possible concentration-dependent or time-dependent clearance process but could not rule out inadequate adjustment for background exposures.

5. Given the above additional considerations (beyond that of ATSDR's consideration about the length of follow-up), the MRLs, assuming same PODs from the same studies, are recalculated in the table below using the different serum elimination half-life values for PFOA, PFOS, and PFHxS that are reported in Olsen et al., Bartell et al., Li et al., and Seals et al. Accordingly, the percent of the MRL that might be overestimated by the ATSDR using in their most conservative serum elimination value (arithmetic means from Olsen et al. 2007) would then result in a range of overestimations of the MRL for PFOA between 9 and 40 percent. This type of sensitivity analysis is definitely needed in Appendix A for the MRL calculations to take into account the variation of serum elimination half-life estimates that have been reported in the literature that will be, in part, a function of the GFRs from the population studied. Given the fact that ATSDR has used developmental studies to calculate the PODs for their MRLs, it is therefore not justified to use the arithmetic mean half-life estimate based solely on retirees, in part, because the GFRs of older adults are markedly lower than adults of much younger age and people 65 years of age or older represent only approximately 15% of the general population Therefore the estimated half-lives should reflect the entire population, not just the upper tail, which can be a reflection of lower GFRs that occur with age. Thus, calculation of serum elimination half-lives may be age, sex, and concentration-dependent. MRLs, based in part on half-lives, should reflect this diversity of inputs in their calculations as shown in the table below.

Reference Study	Estimated Half-life		MRL (mg/kg/d)	% MRL over current ATSDR MRL
	Years	Days		
*ATSDR Estimate (arithmetic Mean from Olsen et al. 2007)	3.8	1400	2.74E-06	--
Olsen et al. 2007 (geometric mean)	3.5	1278	3.00E-06	9
Seals et al. 2011	2.9	1058	3.62E-06	24
Li et al. 2018	2.7	985.5	3.89E-06	30
Bartell et al. 2010	2.3	839	4.56E-06	40

As illustrated above, because HED and MRL are dependent of the clearance rate used, the resulting MRL for PFOA can differ substantially and could be 9 to 40% higher than the current provisional MRL proposed by ATSDR.

L. Uncertainties associated with Wambaugh benchmark dose model used by ATSDR. ATSDR relied on an animal PBPK model to predict subsequent POD of MRL derivation, but on the other hand, it has also explicitly stated that "Although physiologically based pharmacokinetic (PBPK) models have been developed for rodents and humans, these models are not sufficient to allow for comparisons between administered doses in laboratory animals and serum concentrations in humans" (*cf.* page 5 of draft profile). This statement indicated a great amount of uncertainty associated with the PBPK model used hence ATSDR needs to acknowledge this fact in its summary.

The supplementary information from Wambaugh et al. (2013) contains a table (Supplemental Table 3) that compares the agreement of the predicted final plasma concentration of PFOA with those measured from several animal studies. The plasma concentrations resulting from higher doses appear to be better predicted than those resulting from lower doses. For many of the studies that tested lower doses, a plasma concentration measurement was not available for comparison. However, one mouse study (Lau et al 2006) did have measured plasma concentrations available at lower doses; for these, the predicted values appear to overestimate the final plasma concentrations at the lower doses of 1 and 3 mg/kg/day. The predicted values are almost three times higher than those measured (a factor of 2 is generally accepted for model-predicted values). This introduces uncertainty around model predictions at these lower doses, which are closer to the dose used by ATSDR for derivation of the MRL than the higher values that appear to be better predicted by the model. Although ATSDR used the model to estimate serum concentrations at higher doses, the POD for derivation of the MRL was a dose of 0.3 mg/kg/day. As a result, the model predictions for serum concentration could be more uncertain in this low dose range. Although model predictions were not compared to measured steady-state concentrations by Wambaugh et al 2013, which was what was used to derive the POD plasma concentration, the overestimated predictions in the low dose range still introduces uncertainty into the assessment.

Although the Wambaugh model was used to estimate maternal serum concentrations from developmental datasets (Lau et al. 2006; White et al. 2009; Wolf et al. 2007), the model was not specifically parameterized for this, which is another factor contributing to the uncertainty in using this model to estimate an MRL for a developmental endpoint. The Wambaugh PFOA model was parameterized for male and female cynomolgus monkeys, male and female

SD rats, and female CD1 and C57Bl/6 mice. ATSDR states that they could not model some of the studies due to lack of parameters among different mouse models: Cheng et al. 2013 (Wistar rats), Loveless et al. 2008 (CD1 male mice), and Abbott et al. 2007 and Abrecht et al. 2013 (129S1/SvlmJ wild-type mice). While there are well-known differences in pharmacokinetics for male and female rats for PFOA and differences across species, ATSDR provides no evidence or support for sex or strain differences in pharmacokinetics for mice or differences in pharmacokinetics for different strains of rats. As ATSDR modeled only certain strains, this limits the studies it can use when relying on this model and introduces further uncertainty into the MRL value as several studies could not be considered.

In performing the benchmark dose modeling on the DeWitt et al. studies (2008; 2016), ATSDR used the Wambaugh model to estimate steady-state plasma concentrations of PFOA. These studies were conducted in C57Bl/6N mice, for which the Wambaugh model was not parameterized. ATSDR is not consistent in their modeling approaches with the Wambaugh model (i.e., they did not model some studies due to lack of strain-specific parameters but they modeled the DeWitt studies, which were conducted in a strain that the model was not parameterized for).

M. Uncertainty factors chosen by ATSDR were overly conservative and not supported by best available scientific data. They include:

1. Incorrect use of "10" for a LOAEL. ATSDR concluded that the studies by Onishchenko et al. (2011) and Koskela et al. (2016) did not have a NOAEL hence assigned an uncertainty factor of 10 for LOAEL to NOAEL extrapolation. However, given that there was only one PFOA dose group used in the study (in addition to the fact that there were very few animals studied), it was impossible to establish any meaningful dose-response relationship. ATSDR should recognize this limitation as a critical design flaw and it should also recognize that a NOAEL or LOAEL cannot be established under the study condition. This factor of "10" is not scientifically justified and should be removed by ATSDR should it insist on using the same dataset for its MRL derivation on PFOA.

2. Use of "3" for animal-to-human, in addition to large dosimetric TK adjustment, is not scientifically justified. While 3M agrees with ATSDR to adjust for toxicokinetic difference between human and rodent serum clearance of PFOA, 3M does not agree with the serum elimination half-life chose by ATSDR for the calculation (see toxicokinetic discussion above). In addition, while this TK clearance adjustment represented a factor of 10,000 based on ATSDR's derivation, 3M does not agree with ATSDR that an additional factor of "3" is needed to account for uncertainty in using laboratory animal data to derive human exposure levels. This, in fact, represents an adjustment of 30,000 when taking dosimetry into account. The use of an additional factor of 3 to account for rodent-to-human toxicodynamic difference is not necessary.

   More specifically, ATSDR has derived its proposed MRL based on the rodent developmental data. Because humans are considerably less sensitive to the pleiotrophic effects of xenosensor nuclear receptors such as PPARα, CAR/PXR activation compared to rodents (Corton et al. 2014; Elcombe et al. 2014; Gonzalez and Shah 2008; Klaunig

et al. 2003; Klaunig et al. 2012; Lake 2009; Ross et al. 2010), the qualitative differences brings into question the relevance of rodent developmental effects with exposure to PFOA and their biological significance to humans. For example, many of the developmental effects observed noted in wildtype mice when exposed to PFOA were attenuated when PPARα genes were knocked out (Abbott et al., 2007). This further supported the qualitative difference and human relevance between rodents and humans. Thus, the very large dosimetric adjustment of 10,000 more than adequately compensates for the additional factor of 3 for difference between rodents and humans. ATSDR should not apply another factor of 3 for animal to human extrapolation when this uncertainty is already embedded in the large adjustment for the dosimetric difference.

3. Additional factor of "10" for human variability is overly conservative. For PFOA MRL, ATSDR included a factor of 10 for human variability. If ATSDR could have developed a more appropriate PBPK model that accounted for life stage differences in humans (rather than relying on rodent model), this factor of 10 for human variability could potentially be reduced.

Detailed Comments on PFOS MRL

ATSDR Position (page A-36)

MRL Summary: A provisional intermediate-duration oral MRL of $2x10^{-6}$ mg/kg/day was derived for PFOS based on delayed eye opening and transient decrease in F2 body weight during lactation in the offspring of rats administered PFOS via gavage in a 2-generation study (Luebker et al. 2005a). The MRL is based on a HED NOAEL of 0.000515 mg/kg/day and a total uncertainty factor of 30 (3 for extrapolation from animals to humans with dosimetric adjustments and 10 for human variability) and a modifying factor of 10 for concern that immunotoxicity may be a more sensitive endpoint than developmental toxicity).

Selection of the Critical Effect: The most sensitive targets of PFOS toxicity in laboratory animals are similar to those identified in longer term epidemiology studies. These effects include liver damage and increases in serum lipids, decreased antibody response to vaccines, and small decreases in birth weight; epidemiology studies have not consistently found neurological effects to be associated with serum PFOS levels.

3M Conclusion

A. The critical effect concluded by ATSDR with PFOS exposure (decreased pup body weight and delayed eye opening in rats) has been not shown in humans
B. ATSDR should recognize rodent-specific effects and their relevance to humans
C. PFOS does not affect the reproductive system in laboratory animals
D. The developmental effects reported in the laboratory animals for PFOS were primarily mediated by maternal effects
E. Liver findings in rodents are not relevant for human risk assessment
F. PFOS does not cause increase in serum lipid in laboratory animals
G. The nervous system is not a primary target organ with exposure to PFOS
H. Inconsistent immune findings in rodents were confounded by systemic toxicity
I. Inconclusive immune findings in human epidemiological data do not support ATSDR conclusions
J. Serum PFOS concentrations in pups should be considered for POD instead of dams because critical effects chosen by ATSDR were based on (developing) pups
K. HED for PFOS will be higher when considering faster half-life
L. Wambaugh benchmark dose model used by ATSDR was not optimized
M. Uncertainty factors by ATSDR were conservative and not supported by scientific data
    1. Use of "3" for animal-to-human, in addition to large dosimetric TK adjustment, is conservative because humans are less sensitive than rodents based on *in vitro* hepatocyte data (Bjork and Wallace 2009)
    2. Scientifically unjustified use of "10" for concerns on immunotoxicity

ATSDR's overall interpretation on both toxicology and epidemiology data are inconsistent with the most current knowledge. Its application of uncertainty factors is not scientifically justified and the proposed PFOS MRL is not supported by the scientific data. The PFOS MRL derived for the human-health risk assessment is therefore conservative and not scientifically justified.

3M Comments (Details):

A. The critical effect concluded by ATSDR with PFOS exposure (decreased pup body weight and delayed eye opening in rats) has been not shown in humans (see epidemiology discussion above). ATSDR should offer a plausible explanation as to why it believes these effects are relevant to human risk assessment.

B. ATSDR should recognize rodent-specific effects and their relevance to humans. For PFOS, the critical effect chosen by ATSDR are delayed eye opening and decreased pup body weight, based on the results from a 2-generation reproduction study in rats with PFOS (Luebker et al. 2005a). While the text of the proposed MRL derivation fails to make clear that none of the listed effects has been shown in humans (see epidemiology discussion above), the inclusion of some of the effects is incorrect even based on animal data alone. Many "effects" included by ATSDR are specific to rodents and often contrary to the current published literature. For instance, mechanistic research has shown that many metabolic effects to PFOS exposures in rodents can be explained by the activation of xenosensor nuclear receptors such as PPARα, constitutive androstane receptor (CAR), and pregnane X receptor (PXR) in the liver (Bjork et al. 2011; Bjork and Wallace 2009; Elcombe et al. 2012a; Elcombe et al. 2012b; Vanden Heuvel et al. 2006). Given that humans are considerably less sensitive to the pleiotrophic effects of PPARα or CAR/PXR activation compared to rodents (Corton et al. 2014; Elcombe et al. 2014; Gonzalez and Shah 2008; Klaunig et al. 2003; Klaunig et al. 2012; Lake 2009; Ross et al. 2010), the qualitative differences calls into question the relevance of rodent developmental effects and their biological significance to humans. For example, neonatal survival actually improved in mice when PPARα knockout mice were exposed to PFOS when compared to the wildtype (Abbott 2009; Abbott et al. 2009).

C. PFOS does not affect the reproductive system in laboratory animals. It is incorrect for ATSDR to conclude that reproductive system is one of the primary targets of toxicity with exposure to PFOS (cf. page A-36).

   A number of experimental animal (mammalian) toxicological studies on the reproductive and developmental effects of PFOS have been published (Abbott et al. 2009; Butenhoff et al. 2009b; Case et al. 2001; Gortner et al. 1980; Grasty et al. 2005; Lau et al. 2003; Luebker et al. 2005a; Thibodeaux et al. 2003). These studies included detailed information on the developmental toxicity with these compounds as well as valuable insights on the role of maternal effects and its attribution to the developmental outcomes in laboratory animals. Comprehensive review on the potential developmental toxicity of the perfluoroalkyl acids was reported in 2004 (Lau et al. 2004) and updated subsequently (Abbott 2015; Andersen et al. 2008; Lau et al. 2004).

   Overall, PFOS did not affect the functional aspects of male or female reproductive functions in the laboratory animals. These included estrous cycles, sperm parameters, mating index, fertility index, and reproductive organ morphology. The potential of PFOS to influence reproductive performance was evaluated in mice (Abbott et al. 2009; Thibodeaux et al. 2003), rats (Butenhoff et al. 2009; Luebker et al. 2005a), and rabbits (Case et al. 2001).

Gestational exposure to PFOS did not affect the number of embryonic implantation sites in several strains of mice (CD-1, Sv129, or PPARα knockout) (Abbott et al. 2009; Thibodeaux et al. 2003). Similarly, implantations were not affected in rabbits either when exposed up to 3.75 mg/kg-d during GD 7 – 20 (period of organogenesis) albeit decreased body-weight gain and food consumption were observed (Case et al. 2001). In rats, oral administration of PFOS up to 10 mg/kg-d during GD 6 – 15 (period of organogenesis) also caused reduced body-weight gain, however, they did not affect the ovaries or the reproductive contents of the dams (Gortner 1980).

In a two-generation reproduction/developmental study in rats (Luebker et al. 2005), potassium PFOS (given as potassium salt) doses as high as 3.2 mg/kg-d given to male and female rats for 6 weeks prior to mating, through mating and, for females, through gestation and lactation. PFOS did not adversely affect mating or fertility parameters in male or females, including fertility and pregnancy indices, estrous cycling, number of pregnancies per number of matings, number of days to inseminate, number of matings during the first week of cohabitation, epididymal sperm maturation, litter averages for corpora lutea, implantations, viable embryos, non-viable embryos, and reproductive organ histology. In particular, there were no statistically significant differences between control and potassium PFOS-treated females in the mean number of estrous cycles, rats with ≥6 consecutive days of diestrus or estrous during the 28-day evaluation period. In a developmental neurotoxicity study with PFOS, pregnant female rats received PFOS doses up to 1 mg/kg/day from gestation to lactation. No PFOS treatment-related effects were noted on maternal health or reproductive outcomes (Butenhoff et al. 2009). Furthermore, the morphologic effects of PFOS on reproductive organs in non-human primates were evaluated from a six-month oral study and results indicated no abnormalities (Seacat et al. 2002).

D. The developmental effects reported in laboratory animals for PFOS were primarily mediated by maternal effects. While ATSDR concluded that developing organisms are primary targets of toxicity with exposure to PFOS (cf. page A-36), there is strong experimental evidence demonstrating that developmental effects associated with PFOS exposures in offspring are observed only where there were significant effects in the maternal animals. Experimental evidence demonstrates that developmental effects associated with PFOS exposures in offspring are observed when maternal animals were affected such as body weights. Evidence involving maternal effects in the outcome of the developmental toxicity includes the following examples.

PFOS developmental toxicity has been evaluated in several laboratory species. In rabbits, oral PFOS administration ranging from 0.1 – 3.75 mg/kg/day was given from GD 6 – 20 and decreased maternal body-weight gain was observed at 1 mg/kg dose group or higher. No abnormal fetal effects were noted except decreased fetal body weight, which was observed with 2.5 and 3.75 mg/kg/day dose groups only. Study authors concluded that "The fetal effects occurred at maternally toxic dose levels and no fetal changes were present at nontoxic maternal doses" (Case et al. 2001). In mice, there was a statistically significant ($p \leq 0.05$), dose-related increase in maternal liver weight when pregnant dams were treated during gestation at a dose as low as 1 mg/kg potassium PFOS (Thibodeaux et al. 2003). Various develpmental effects were reported (e.g., decrased postnatal survival and growth deficits) but

primarily for litters from dams receiving 10 mg/kg/day potassium PFOS or higher (Lau et al. 2003). In addition to mice, the developmental toxicity of PFOS has also been evaluated in rats. Oral administration of PFOS during gestation to pregnant rats caused reduced maternal body-weight gain and fetal body-weight gain at 2 mg/kg-d maternal dose group or higher (Lau et al. 2003). In a two-generation reproduction/developmental study in rats by Luebker et al. (2005), described in detail above, the authors reported reduced body weight and body weight-gain at parental generation at 0.4 mg/kg or higher. Developmental hallmarks similar to those previously reported by others (*i.e.*, decreased fetal body weight, decreased postnatal survival, and developmental delays) were observed in pups from 1.6 mg/kg/day maternal dose groups or higher. Therefore, the developmental effects reported in the laboratory animals for PFOS were primarily mediated by maternal effects and based on the recent mode of action data, rodents may not be the most appropriate species for the hazard assessment of PFOS on developmental toxicity in humans.

E. Liver findings in rodents are not relevant for human risk assessment. The comments to follow are related to ATSDR's identification of "liver damage' in laboratory animal studies as sensitive target with exposure to PFOS. Similar to the comments provided earlier on PFOA, liver findings in rodents warrant careful consideration. Given that it is well recognized that there is distinct difference in mode-of-action between rodents and humans when it comes to liver changes mediated by xenosensor nuclear receptors, liver effects observed in rodents are scientifically unjustified and inappropriate for use as a critical effect for human risk assessment.

There is a well-established body of experimental evidence for activation of PPARα and CAR/PXR as a major factor in the rodent hepatic response to exposure to PFOS. As Elcombe et al. (Elcombe et al. 2012a; Elcombe et al. 2012b) point out, the hypertrophic and hyperplastic response of rat liver to PFOS exposure has clearly been demonstrated to be consistent with the criteria used to establish PPARα/CAR/PXR activation as a mode of action. The transcriptional signature (mRNA) for PPARα/CAR/PXR activation was also observed in livers from PND 21 male rat pups exposed via maternal gavage in the developmental neurotoxicology study reported by Butenhoff et al. (2009b) and Chang et al. (2009) as well as in adult male wild-type mice (Rosen et al. 2010). In the E3L.CETP mouse transgenic mouse model, dietary PFOS exposure of adult males resulted in transcriptional gene expression profiles and changes in lipid parameters consistent with activation of PPARα and PXR (Bijland et al. 2011). Rosen et al. (2009) observed the same transcriptional signature consistent with activation of PPARα/CAR/PXR in CD-1 mouse fetal liver after maternal exposure to PFOS during gestation.

There are fundamental differences between the responses of human and rodent liver from exposure to agents that increase activation of PPARα and CAR/PXR (Corton et al. 2014; Elcombe et al. 2014). The basis for the fundamental differences between the rodent and human liver response from exposure to agents that activate these receptors has become clearer with development of receptor knock-out and humanized receptor knock-in transgenic mouse models and the increased availability of human primary hepatocytes. When exposed to PPARα and CAR/PXR agonists, mice that have been genetically modified by removal of the natural mouse receptors and replacement with the natural human forms of the receptors

do not have the hyperplastic response observed in wild-type mice (Gonzalez and Shah 2008; Ross et al. 2010). Key differences between rodent and human hepatocytes, especially the lack of a hyperplastic response in human hepatocytes exposed to PPARα and CAR activators, have also been demonstrated (Elcombe et al. 1996; Goll et al. 1999; Hirose et al. 2009; Parzefall et al. 1991; Perrone et al. 1998).

As noted above, human hepatocytes respond to PPARα agonists differently than rodent hepatocytes, and activation of human PPARα does not appear to result in the characteristic hyperplastic response observed in rats and mice (Corton et al. 2014; Gonzalez and Shah 2008). Bjork and Wallace (2009), working with primary rat and human hepatocytes as well as the HepG2 human liver cell line in culture, demonstrated major differences between primary rat hepatocytes and human hepatocytes in response to exposure to PFOS in culture. In comparison to the large increase over control in mRNA for peroxisomal enzymes Cte/Acot1 and Acox, the human hepatocytes showed essentially no increase in transcripts. However, consistent with observations with other peroxisome proliferators, CYP4A11 mRNA was increased by PFOS exposure in human as well as Cyp4A1 in rat hepatocytes.

In addition to PPARα, Bjork et al. (2011) characterized the activation of several other hepatic nuclear receptors (PXR, CAR, the liver X receptor α (NR1H3 or LXRα), and the farnesoid X receptor (NR1H4 or FXR) by PFOS in primary rat and human hepatocytes. In rat hepatocytes, they demonstrated multiple nuclear receptors participate in the metabolic response to PFOS exposure, resulting in a substantial shift from carbohydrate metabolism to fatty acid oxidation and hepatic triglyceride accumulation. They concluded that, "while there is some similarity in the activation of metabolic pathways between rat and humans, particularly in PPARα regulated responses; the changes in primary human cells were subtle and possibly reflect an adaptive metabolic response rather than an overt metabolic regulation observed in rodents." Supporting this, the potential activation of human CAR3 isoform and human PXR has been studied. PFOS was not shown to activate directly either human nuclear receptor at concentrations up to 33 μM, with slight activation (much less than for positive control substances) of CAR3 and PXR occurring only at 100 μM (Ehresman et al. 2014).

Collectively, the established mode-of-action supports the liver hypertrophic effects in rodents from exposure to PFOS. The experimental evidence also shows the lack of a response, or a markedly reduced response, in human liver cells as compared to rodent liver. Furthermore, there were no adverse liver effects noted in humans (see epidemiology discussion above). The observational human data as well as a significant body of mechanistic experimental data that relates to the liver response to exposure to PFOS strongly suggests that use of rodent liver findings as an endpoint for the human-health risk assessment of PFOS is not scientifically justified. Other federal agency such as USEPA (in its assessments of PFOA in 2009 and again in 2016), as well as other international regulatory authorities such as European Chemical Agency Risk Assessment Committee (2015), European Food and Safety Authority (2018), and Australian Expert Health Panel (2018) also considered the liver weight findings in laboratory animal studies with PFOA (or other perfluoroalkyls) to be irrelevant for human risk assessments.

It should be noted that, acetylsalicylic acid (commonly known as aspirin), one of the most common over-the-counter drugs used in the world, can also elicit increased liver weight in laboratory animals similar to the observations reported with perfluoroalkyls in rodents (EMEA, 1999).

F. PFOS does not cause increase in serum lipid in laboratory animals. It is incorrect for ATSDR to conclude that "increases in serum lipid" is a sensitive target associated with exposure to PFOS. To the contrary, exposure to PFOS in laboratory animals has been consistently shown to decrease serum lipids (Butenhoff et al. 2012a; Chang et al. 2017; Elcombe et al. 2012a; Elcombe et al. 2012b; Seacat et al. 2003; Seacat et al. 2002). PFOS has been established as a hypolipidemic agent in mechanistic studies and reduction in serum cholesterol has been shown to be an early effect related to dosing with PFOS in toxicological studies with rodents and primates (Bijland et al., 2011; Elcombe et al., 2012a; Seacat et al., 2002, 2003). The hypolipidemic activity of PFOS occurs via the activation of xenosensor nuclear receptors peroxisome proliferator-activated receptor alpha (PPARα) and pregnane X receptor, which can influence fatty acid β-oxidation and lipid synthesis (Bijland et al. 2011; Bjork et al. 2011; Elcombe et al. 2012a; Elcombe et al. 2012b). Mechanistic study has elucidated how PFOS modulates the hypolipidemic responses. Using ApoE*3.Leiden.CETP mice, a humanized model having attenuated clearance of ApoB-containing lipoprotein and exhibiting human-like lipoprotein metabolism on a Western-type diet (ApoE*3 model paper), Bijland et al. (2011) demonstrated that high dietary doses of PFOS resulted in lower serum cholesterol by reducing VLDL production with enhanced triglyceride clearance (mediated by lipoprotein lipase) as well as decreased production of apolipoprotein B. PFOS also affected the rate of apolipoprotein A1 synthesis which ultimately resulted in the reduction of circulating HDL.

In a more recent study with non-human primates, Chang et al. (2017) confirmed the potential associations between serum PFOS and changes in serum lipid over a period of more than 1 year. With the highest serum PFOS achieved at approximately 165 ug/ml, only a slight reduction in serum cholesterol (primarily the high-density lipoprotein fraction), although not toxicologically significant, was observed and the corresponding lower-bound fifth percentile benchmark concentrations ($BMCL_{1sd}$) were 74 and 76 ug/ml for male and female monkeys, respectively.

Therefore, there is no evidence to suggest that PFOS causes an increase in serum lipid.

G. The nervous system is not a primary target organ in laboratory animals with exposure to PFOS. ATSDR also suggests that nervous system is a sensitive targets with exposure to PFOS per observations reported by Butenhoff et al. (2009b), this is incorrect.

In Butenhoff et al. (2009), the "increased motor activity and decreased habituation" was observed as a single, transient observation in male pups from 1.0 mg/kg-d maternal dose group on postnatal day (PND) 17. ATSDR failed to account for the lack of evidence for developmental neurological effects observed in the study as well as other corroborating studies. The use of this single, transient observation as a critical endpoint when more significant data are available as part of the same study (as well as other studies mentioned

below) that demonstrate normal neurological development is at odds with guidance for data interpretation for developmental neurotoxicity studies (Francis et al. 1990; USEPA 1998)These guidelines state that a weight of evidence approach and expert judgment should be used. It is evident that this has not been the case for PFOS.

Locomotor activity was one of many developmental neurotoxicological endpoints evaluated in the study by Butenhoff et al. (2009). While habituation (a primitive form of learning) and higher learning and memory were evaluated in three phases of the Biel maze swimming assessment on PNDs 22 through 28. The tri-phasic Biel maze swimming trial test paradigm to evaluate learning and memory did not reveal an effect of PFOS on the studied parameters in pups (20 / sex / dose groups). There were no other observations among the many recorded that were suggestive of a neurotoxicological effect of PFOS on development through the PND 66 observation period. A functional observation battery (FOB) was performed with the same sets of 20 rats per sex per group on PNDs 4, 11, 21, 35, 45, and 60; and it included various stages of development permitting: ease of cage removal; ease of handling in hand; lacrimation/chromodacryorrhea; salivation; piloerection; appearance of fur; palpebral closure; respiratory rate/character; red, crusty deposits; mucous membranes/skin color; eye prominence; eye color; mobility; muscle tone; convulsions/tremors; hindlimb extension; grooming; arousal; bizarre/stereotypic behavior; urination/defecation; pupillary response; backing; forelimb/hindlimb grip strength; tail pinch response; gait; and air righting. None of these FOB endpoints was affected by treatment with PFOS.

The lack of an effect on learning and memory is also supported by the results of Lau et al. (2003) and Luebker et al. (2005a). In the study by Lau et al., PND 22 rat pups from dams given 3.0 mg/kg/d throughout gestation did not differ from controls when tested using a T-maze with alternation. In the study by Luebker et al., F_1-generation pups were tested for learning, short-term retention, and memory in a passive avoidance paradigm beginning on PND 24, and, beginning on approximately PND 70, were evaluated in a water-filled M-maze for neuromuscular coordination, swimming ability, learning, and memory. No effects of treatment were observed.

H. Inconsistent immune findings in rodents which were confounded by systemic toxicity. With exposure to PFOS, ATSDR also concluded that immunotoxicity (as decreased antibody responses to vaccines) is one of the most sensitive targets. Similar to the discussion with PFOA, these are based on the decreased antigen-specific antibody responses in mice where PFOS suppressed T cell-dependent IgM antibody response (TDAR) but not the secondary IgG response (Dong et al. 2011; Dong et al. 2009; Guruge et al. 2009; Peden-Adams et al. 2008). A key principle in conducting a robust immunotoxicity study is to avoid / minimize systemic toxicity, including body weight loss.

Toxicological studies cited by ATSDR for reduced immune findings are confounded by overt toxicity and should not be included in the interpretation of immune findings. For example, in the studies by Dong et al. (2009; 2011), exposure to PFOS has also been associated with suppression of NK cell activity, a dose-dependent decrease in IgM PFC responses, but no evidence in IgG suppression were noted. It is important to note that the reported suppressions with exposures to PFOS appeared to be a high dose phenomenon where

systemic effects (i.e., body weight reduction) were present. This confounded the overall study interpretation in the immunotoxicity studies because reduced body weight as well as increased corticosterone serum levels were known immunosuppressive factors. The data presented by Dong et al. also lacked scientific validity to support the conclusion that PFOS suppresses immune responses. Concordance between several key immune parameters should be systematically illustrated in these immunotoxicity studies. Again, using the study by Dong et al. (2009) as an example, they did not properly address the following:

1. It is well known that body weight plays a critical role in studying immune response and any factors that can influence body weight will likely indirectly affect immune responses. Although Dong et al. claimed that body weight was not affected in the first two lower dose groups (0.5 and 5 mg/kg TAD), in looking at Table 1 in the Dong et al. paper, there appeared to be a difference in mean body weight change between the control group (3.10) and the 0.5 mg/kg group (2.58). By taking the summary data for each treatment group to replicate the ANOVA and Dunnett's t tests by computing 1-sided critical values for Dunnett's test, the final body weights in the 0.5 mg/kg treatment group were significantly lower than the control group at $\alpha=0.10$ ($0.05 < p < 0.10$).

2. It is also well known that the antibody titers to vaccinations are secondary IgG antibody isotype. The study data reported by Dong et al. (as well as others) was the primary IgM antibody response only, which did not reflect what the status of the secondary (memory) IgG antibody was.

3. It is important to emphasize that, not only was the secondary IgG response not measured by Dong et al, it was not appropriately induced to elicit a *bona fide* memory response as antigen was challenged only once in the study.

4. As an extension from above, Dong et al. did not evaluate the production of other immunoglobulin isotypes and they did not take the time-based progression of IgM → IgG antibody class switching into consideration. The normal progression of antibody development involves the IgM production by B cells first as primary immune response. The B cells will subsequently proliferate and become activated when further challenged by antigen, which, ultimately leads to antibody class switching to produce IgG, which is the clinical measurement for the assessment of antibody titer.

5. While Dong et al. claimed that the antibody response was reduced based on IgM PFCR data; the IgM PFCR activity was only evaluated in spleen cells only. The authors should have also looked at thymus and serum for IgM levels to illustrate that the responses are consistent.

6. By way of similar rationale listed in point #3, Dong et al. should have looked at IgG in addition to IgM, as well as evaluated IgG levels in thymus and serum.

7. While the immune cell populations were reported by Dong et al. in spleen and thymus, they did not look at these cell populations in another key immune organ: bone marrow. That was a major omission by the study authors.

8. While Dong et al. reported NK cell activity in their study for the spleen, they did not examine the thymus.

9. The LDH assay is not a standard assay used to assess NK cell activity and the LDH values reported by Dong et al. should not be interpreted as NK cell activity data. LDH measurement is associated with cell membrane integrity and it is a non-specific assay. The standard assay for NK cell activity is flow cytometry, which Dong et al. did not perform.

10. Dong et al. reported a negative effect of PFOS and the splenic lymphocyte proliferation as a way of demonstrating that the immune cells were not "proliferating" upon challenge. However, the specific problem with this piece of data is that MTT assay is not a measurement of cell proliferation. It is simply an indicator of cell's mitochondrial respiration state and it does not reflect any proliferative responses at all. The standard assay for cell proliferation would be something like BrDU assay, which was not evaluated by Dong et al.

11. The antigen challenge substance used by Dong et al. was sheep red blood cell (SRBC) and in the field of immunology, responses from SRBC challenge are very crude and non-specific to T cell activation. There are many T-cell dependent antigens available for use in the immunology research (i.e., ovalbumin) and Dong et al. failed to recognize this.

12. No information on blood lymphocyte counts was provided (part of the standard CBC panel parameters).

13. No histological evidence for thymus, spleen, or bone marrow was provided.

14. Dong et al. only evaluated male mice; they should have also looked at female mice to rule out any gender-specific difference in the immune response.

As discussed above, antibody response is IgG isotype, not IgM. If PFOS was truly an immunosuppressing agent, one would expect similar suppressive immune responses to be observed in major key organs such as decreased IgM and IgG in spleen, thymus, and serum concurrently. Dong et al. evaluated IgM in spleen only but did not provide any concurrent IgM status in other key organs such as thymus or serum. As an immunosuppressing agent, one would expect decreased immune cell populations in spleen, thymus, blood, and bone marrow and Dong et al. only looked at spleen and thymus. As an immunosuppressing agent, one would expect decreased proliferation in immune cells and Dong et al. did not use the correct methods to evaluate these responses. If one is to rely on Dong et al. data as the basis for their evaluation, they need to justify why, when compared to the concurrent control with an overall body weight gain of 3. 1 g over 60-day dosing period, a significant lower overall body weight-gain of 2.58 g in the lowest dose group mice (0.5 mg/kg/ TAD) did not confound the immunological responses reported.

Peden-Adams et al. (2008) reported increased lymphatic NK cell activity was seen in male B6C3F1 mice but not females; however, NK cell activity was not measured in other key immune organs such as spleen, thymus, or serum. They also reported suppression of IgM but did not evaluate IgG. The study by Guruge et al. (2009) reported that exposure to PFOS was associated with reduced ability of animals to respond to infectious disease, which was based on the resistance of female B6C3F1 mice to influenza virus A/PR/8/34 (H1N1) after exposure to PFOS. However, the study was confounded by mortality.

Collectively, these studies cannot be conclusively interpreted as demonstrating an effect of PFOS on immune functions and there is no robust scientific evidence to support the claim that PFOS is associated with immune suppression in mice.

On page A-44 of the draft Toxicological Profile (for PFOS MRL), contrary to what ATSDR stated that "Immune function was not examined following chronic-duration oral exposure in laboratory animal studies", it should be noted that the primary immune organs were evaluated microscopically in rats after 2 years of dietary treatment containing potassium PFOS (Butenhoff et al. 2012a). In this study, representative primary immune organs were collected (femur with bone marrow, lymph node (mesenteric), spinal cord (cervical, thoracic, and lumbar); spleen; sternum with bone marrow, and thymus) and evaluated microscopically by a board-certified veterinary pathologist at the end of a 2-year period. There were no statistically significant findings (neoplastic or non-neoplastic) for these immune organs in either male or female rats fed potassium PFOS in diet when compared with respective control group rats. This is important because it demonstrated the absence of a direct effect on primary immune organs with chronic PFOS exposures in the rats. In addition, PFOS-treated rats had similar or higher percent survival compared to controls, which is contrary to chronic immunosuppression-mediated toxicity such as cyclosporin (a known immunosuppressant) that ultimately resulted in increased mortality in rats (Ryffel and Mihatsch 1986).

I. Inconclusive immune findings in human epidemiological data. While ATSDR concluded that such findings in rodents were consistent with human epidemiology studies with regards to vaccine responses (see epidemiology discussion above), it is important to recognize that the humoral immune response to vaccinations, as measured in the human epidemiology studies, is mainly a secondary IgG memory response, not IgM. While suppression of the IgM response by PFOS was demonstrated in several animal studies where administered doses also induced signs of overt toxicity (i.e., reductions in body and lymphoid organ weight), it is difficult to interpret why the primary IgM response was suppressed in mice by PFOS and yet the secondary response was either not affected or enhanced. Collectively, the aforementioned studies suggest that PFOS impairs immune cell activity in laboratory animals at very high doses which may be mediated in part by overt toxicity as suggested by increased corticosterone serum levels, decreased body and lymphoid organ weights and decreased lymphoid tissue cellularity. The animal studies do not support that PFOS suppresses immune cell activity in the absence of overt toxicity.

J. Serum PFOS concentrations in pups should be considered for POD because critical effects chosen by ATSDR were based on (developing) pups. ATSDR selected a rat 2-generation study (Luebker et al. 2005a) for the point-of-departure to derive the MRL value for PFOS

(endpoints were decreased pup bodyweight and delayed eye opening in offspring of SD rats). Similar to PFOA, the study chosen by ATSDR for the PFOS POD examined developmental endpoints that were measured in offspring, which are used as the basis for the MRL. In order to estimate steady-state plasma concentrations of PFOS, ATSDR used the Wambaugh model for PFOS, which is parameterized for adult animals and cannot be used to predict concentrations in fetuses or pups. This model also does not account for life stage differences in physiology or pharmacokinetics. The area-under-the-curve (AUC) and steady-state concentration are probably different in the offspring than in the dam. Overall internal exposure (as estimated by calculation of the AUC) may change with growth, and there could be a period of peak exposure. Use of the Wambaugh model introduces uncertainty in the MRL derivation as the offspring plasma concentration may be different that than of the maternal animals. Use of a physiologically-based model that incorporates fetal and pup compartments would provide an estimate of fetal and pup internal exposure (rather than use of the maternal concentration as a surrogate), which would reduce the uncertainty in the MRL value.

K. HED for PFOS will be higher when considering faster half-life. In the MRL calculations, ATSDR chose to use the arithmetic mean serum elimination half-life estimate for PFOA from Olsen et al. (2007) over other studies because Olsen et al. had a longer follow up time and ATSDR was concerned that based on a study by Seals et al. (2011), slower kinetics is likely to constitute a larger contribution to the terminal half-life. For example, whereas Olsen et al. had an average follow-up of 5 years, Bartell et al. had a follow-up of a year and Li et al. had a follow-up of 2.3 years among those studies that followed individuals and were not cross-sectional analyses of populations. However, this line of reasoning by ATSDR for selection of the arithmetic mean from the Olsen et al. study fails to take into account several factors that likely biased upwards the ATSDR MRL estimates. These include the following points.

1. The ATSDR chose not to use the geometric mean estimate that was discussed in the Olsen et al. paper. Given the right skewness of their data, Olsen et al. were more favorable to use the geometric mean for a measure of central tendency. ATSDR provided no explanation as to why they chose the arithmetic mean vs. the geometric mean in this study. This decision is interesting (and curious) because ATSDR chose to report median initial and final concentrations in Table A2 rather than the arithmetic mean initial and final concentrations in Table A2. A median concentration would be better represented by a half-life estimate based on the geometric mean.

2. The Olsen et al. 2007 study comprised 26 retirees (end of study average age = 66 years) who likely would have had an average glomerular filtration rate lower than those calculated from younger ages as reported in Bartell et al. (average age 55) and Li et al. (age range 15 – 55). The average estimated glomerular filtration rate declines with age as shown in the table below.

Age range	Estimated GFR (ml/min/1.73 m^2)	Source:
1-6 months	77	Heilbron et al. 1991 Pediatr Nephrol. Jan;5(1):5-11.
6-12 months	103	
12-19 months	127	
2-12 years	127	
20–29	116	https://www.kidney.org/sites/default/files/docs/11-10-1813_abe_patbro_gfr_b.pdf
30–39	107	
40–49	99	
50–59	93	
60–69	85	
70+	75	

Renal clearance of perfluorocarboxylates (and perfluorosulfonates) is largely a sum of three processes involving glomerular filtration, renal tubular secretion, and renal tubular reabsorption (Han et al. 2012). Because PFOA and other perfluorocarboxylates vary in their affinities to bind plasma proteins, glomerular filtration of perfluorocarboxylates (and perfluorosulfonates) is a product of the unbound fraction of the perfluorocarboxylate and the glomerular filtration rate (GFR). Thus, the higher estimates of GFR based on the younger ages in the other study populations, especially the younger Li et al. study which had approximately 50% of the follow-up time of Olsen et al., may be due to the age differences of the subjects, and not the shorter follow-up period considered in these studies. Thus, the serum elimination half-lives are likely equally valid for consideration in MRL calculations.

3. The Olsen et al. study had to consider, during the course of their follow-up, the possibility of retirees reentering the 3M Decatur and Cottage Grove manufacturing plants. Indeed, this resulted in Olsen et al. eliminating 1 study subject entirely, and truncating follow-up times for two retirees. This would have biased estimates upwards for the serum elimination half-lives due to the increased exposure. It is not likely that ambient general population level concentrations would have biased these retiree's estimates substantially as discussed by Bartell et al. 2012. On the other hand, although Bartell et al. and Li et al. had shorter follow-up times, the primary exposure in these populations was through drinking water. Installation of GAC filters in these populations' affected municipal water supply would have immediately ceased their exposure to PFOA, PFOS, and PFHxS.

4. ATSDR suggests the Seals et al study indicates a lower clearance rate may occur as subjects are followed long-term post exposure; thus, the decision by ATSDR to use the study that had the longest follow-up time (Olsen et al. 2007). However, ATSDR did not mention the main limitations of the Seals et al. study: 1) the cross-sectional nature of the analysis. Individual subjects were not followed. Model-based estimates were instead calculated based on the initial concentrations; 2) there was the added assumption that there was uniform exposure based on the concentration of PFOA measured in each water district; and 3) subjects with initial PFOA concentrations < 15 ng/mL were excluded

which maximized the probability of analyzing individuals with sufficiently high baseline PFOA concentrations that would not be at ambient levels.

5. Given the above additional considerations (beyond that of ATSDR's consideration about the length of follow-up), the MRLs, assuming same PODs from the same studies, are recalculated in the table below using the different serum elimination half-life values for PFOA, PFOS, and PFHxS that are reported in Bartell et al., Li et al., and Seals et al. Accordingly, the percent of the MRL that might be overestimated by the ATSDR using in their most conservative serum elimination value (arithmetic means from Olsen et al. 2007) would then result in a range of overestimations of the MRL for PFOS between 12 and 38 percent. This type of sensitivity analysis is definitely needed in Appendix A for the MRL calculations to take into account the variation of serum elimination half-life estimates that have been reported in the literature that will be, in part, a function of the GFRs from the population studied. Given the fact that ATSDR has used developmental studies to calculate the PODs for their MRLs, it is therefore not justified to use the arithmetic mean half-life estimate based solely on retirees, in part, because the GFRs of older adults are markedly lower than adults of much younger age and people 65 years of age or older represent only approximately 15% of the general population Therefore the estimated half-lives should reflect the entire population, not just the upper tail, which can be a reflection of lower GFRs that occur with age. Thus, calculation of serum elimination half-lives may be ages, sex, and concentration-dependent. MRLs, based in part on half-lives, should reflect this diversity of inputs in their calculations.

Reference Study	Estimated Half-life		MRL (mg/kg/d)	% MRL over current ATSDR MRL
	Years	Days		
*ATSDR Estimate. (arithmetic Mean from Olsen et al. 2007)	5.4	2000	1.72E-06	--
Olsen et al. 2007 (geometric mean)	4.8	1752	1.96E-06	12
Li et al. 2018	3.4	1241	2.77E-06	38

As illustrated above, because HED and MRL are dependent of the clearance rate used, the resulting MRL for PFOS can differ substantially and could be 12 to 38% higher than the current provisional MRL proposed by ATSDR.

L. Wambaugh benchmark dose model used by ATSDR was not optimized. ATSDR relied on animal PBPK model to predict subsequent POD of MRL derivation, but on the other hand, it has also explicitly stated that "Although physiologically based pharmacokinetic (PBPK) models have been developed for rodents and humans, these models are not sufficient to allow for comparisons between administered doses in laboratory animals and serum concentrations in humans" (*cf.* page 5 of draft profile). This statement indicated a great amount of uncertainty associated with the PBPK model used hence ATSDR needs to acknowledge this fact in its summary.

Although the Wambaugh model was used to estimate final maternal plasma concentrations in rats from developmental datasets (Butenhoff et al. 2009b; Chen et al. 2012; Luebker et al. 2005a; Luebker et al. 2005b; Thibodeaux et al. 2003), the model was not specifically

parameterized for this, which is another factor contributing to the uncertainty in using this model to estimate an MRL for a developmental endpoint.

The Wambaugh PFOS model was parameterized for male and female cynomolgus monkeys, male and female SD rats, and male and female CD1 mice. ATSDR states that they could not model some data sets as the studies were conducted in strains that the model was not parameterized for. Specifically, they state that they could not model the following studies: Long et al. 2013 (C57BL/6 mice), Dong et al 2009 and 2011 (C57BL/6 mice), Guruge et al. 2009 (B6C3F1 mice), Peden-Adams et al. 2008 (B6C3F1 mice), Wang et al. 2015 (Wistar rats), Onishchenko et al. 2011 (C57BL/6 mice), and Yahia et al. 2008 (ICR mice). ATSDR provides no evidence of sex or strain differences in pharmacokinetics for mice or rats. As ATSDR modeled only certain strains, this limits the studies they can use when relying on this model and introduces further uncertainty in MRL values.

M. Uncertainty factors by ATSDR were overly conservative and not supported by scientific data. They include:

1. Use of "3" for animal-to-human, in addition to large dosimetric TK adjustment, is not scientifically justified. While 3M agrees with ATSDR that adjusting for toxicokinetic difference between human and rodent serum clearance of PFOS is appropriate; 3M does not agree with the serum elimination half-life chose by ATSDR for the calculation (see toxicokinetic discussion above). While this represented a factor of 14,400 based on ATSDR's MRL derivation, 3M does not agree with ATSDR that an additional factor of "3" is needed to account for uncertainty in using laboratory animal data to derive human exposure levels. This, in fact, represents an adjustment of 43,000 when taking dosimetry into account. The use of an additional factor of 3 to account for rodent-to-human toxicodynamic difference is not scientifically justified and unnecessary.

   More specifically, ATSDR has derived its proposed MRL based on the rodent developmental data. Because humans are considerably less sensitive to the pleiotrophic effects of xenosensor nuclear receptors such as PPARα, CAR/PXR activation compared to rodents (Corton et al., 2014; Elcombe et al., 2014; Gonzalez and Shah, 2008; Klaunig et al., 2003; Klaunig et al., 2012; Lake, 2009; Ross et al., 2010), the qualitative differences brings into question the relevance of rodent developmental effects with exposure to PFOS and biological significance to humans. Thus, the very large dosimetric adjustment of 14,400 more than adequately compensates for the additional factor of 3 for difference between rodents and humans. ATSDR should not apply another factor of 3 for animal to human when this uncertainty is already embedded in the large adjustment for the dosimetric difference.

2. Additional factor of "10" for human variability is overly conservative. For PFOS MRL, ATSDR included a factor of 10 for human variability. If ATSDR could have developed a more appropriate PBPK model that accounted for life stage differences in humans (rather than relying on rodent model), this factor of 10 for human variability could potentially be reduced.

3. Scientifically unjustified use of "10" for concerns on immunotoxicity. As discussed earlier, to the extent that exposure to PFOS influences immune cell activities at very high doses in laboratory animals and as such, these systemic effects indirectly affect immune responses. In addition, long-term subchronic studies in non-human primates (Chang et al. 2017; Seacat et al. 2002) as well as 2-year chronic study in rats (Butenhoff et al. 2012a) did not identify the immune system being the target organs. As a matter of fact, the survival rates in the 2-year chronic study in PFOS-treated rats were higher than the concurrent control. The animal studies do not support that PFOS suppresses immune cell activity in the absence of overt toxicity and an uncertainty factor of "10" is not scientifically justified and should be removed by ATSDR.

   [NOTE: It should be noted that the 2-generation reproductive and developmental study in rats with exposure to PFOS (Luebker et al. 2005) was the same critical study chosen by U.S. EPA Office of Water for the derivation of the Lifetime Water Health Advisory for PFOS issued in 2016. EPA's conclusion on the immunotoxicity is included below:]

   > *"Both human and animal studies have demonstrated the potential impact of PFOS on the immune system; however, uncertainties exist related to MOA and the level, duration, and/or timing of exposure that are not yet clearly delineated. The animal immunotoxicity studies support the association between PFOS and effects on the response to sheep red blood cells as foreign material and on the natural killer cell populations; however, the doses with effects are inconsistent across studies for comparable endpoints. When both males and females were evaluated, the males responded at a lower dose than the females. Because of these uncertainties, EPA did not quantitatively assess this endpoint."*

Detailed Comments on PFHxS MRL

ATSDR Position (page A-49)

MRL Summary: A provisional intermediate-duration oral MRL of 2x10-5 mg/kg/day was derived for PFHxS based on thyroid follicular cell damage in adult male rats administered via gavage PFHxS for a minimum of 42 days (Butenhoff et al. 2009a; Hoberman and York 2003). The MRL is based on a HED NOAEL of 0.0047 mg/kg/day and a total uncertainty factor of 30 (3 for extrapolation from animals to humans with dosimetric adjustments and 10 for human variability) and a modifying factor of 10 for database limitations.

Selection of the Critical Effect: Two intermediate-duration studies in laboratory animals have been identified for PFHxS. In a developmental toxicity study, increased incidences of thyroid follicular cells hypertrophy, and hyperplasia were observed in F0 male rats administered ≥3 mg/kg/day (Butenhoff et al. 2009a; Hoberman and York 2003). Increased liver weight and centrilobular hepatocellular hypertrophy were also observed in the males at ≥3 mg/kg/day. No reproductive or developmental effects were reported. Liver effects (decreases in serum lipids, increases in hepatic triglyceride levels, and increases in liver weight) were also observed in mice exposed to 6 mg/kg/day PFHxS in the diet for 4–6 weeks (Bijland et al. 2011). Using the Hall et al. (2012) criteria (see Section 2.9 for a discussion of the criteria), the liver effects were not considered relevant for human risk assessment. Thus, the lowest LOAEL identified in intermediate-duration studies was 3 mg/kg/day for thyroid effects.

3M Conclusion

A. The critical effect concluded by ATSDR with PFHxS exposure (thyroid follicular cell damage) has been not shown in humans
B. No conclusive evidence to suggest that PFHxS impacts thyroid homeostasis in rodents
C. ATSDR should recognize rodent-specific thyroid effects and their relevance to humans
D. HED for PFHxS will be higher when considering faster half-life
E. Wambaugh benchmark dose model used by ATSDR was not optimized
F. Uncertainty factors by ATSDR were overly conservative and not supported by scientific data
    1. Use of "3" for animal-to-human, in addition to large dosimetric TK adjustment, is conservative because humans are less sensitive than rodents based on *in vitro* hepatocyte data (Bjork and Wallace 2009)
    2. Scientifically unjustified use of "10" for concerns on database limitations, especially on immunotoxicity and general toxicity

ATSDR's overall interpretation on both toxicology and epidemiology data are inconsistent with the most current knowledge. Its application of uncertainty factors is not scientifically justified and the proposed PFHxS MRL is not supported by the scientific data. The PFHxS MRL derived for the human-health risk assessment overly conservative and not supported by adequate scientific foundation.

3M Comments (Details):

A. The critical effect concluded by ATSDR with PFOA exposure (thyroid follicular cell damage) has been not shown in humans. ATSDR needs to offer a plausible explanation as to why it believes these effects are relevant to human risk assessment.

B. No conclusive evidence to suggest that PFHxS impacts thyroid homeostasis in rodents. Based on findings from a reproductive and developmental study with PFHxS in rats (Butenhoff et al. 2009a), ATSDR concluded that the thyroid follicular cell damage findings in rats was the critical effect and used that as the basis for its derivation of PFHxS MRL. This is not the correct interpretation.

It is incorrect for ATSDR to conclude that there was "thyroid follicular cell damage" based on the study findings reported by Butenhoff et al. (2009a). The descriptor "increased incidence of thyroid follicular epithelium hypertrophy/hyperplasia" does not mean "thyroid follicular cell damage". In that study where rats received daily doses of potassium PFHxS at either 0, 0.3, 1, 3, or 10 mg/kg/day, increased incidence of thyroid follicular epithelium hypertrophy/hyperplasia was noted in the 10 mg/kg/day dose group male rats after 42 days of treatment (see table below). Because histomorphometrically, there is a distinct difference between hypertrophy (increases in cell size) vs. hyperplasia (increases in cell number), it is impossible to determine whether there was actual thyroid hyperplasia associated with PFHxS exposure in the rats because, following standard practice at the time of the study, both hypertrophy and hyperplasia were reported as one category by the original study pathologist.

		Potassium PFHxS Doses (mg/kg/day)				
		0 (control)	**0.3**	**1.0**	**3.0**	**10**
Number of F_0 male rats evaluated		10	10	10	10	10
Microscopic Thyroid hypertrophy/hyperplasia (follicular epithelium)	Minimal	0	1	1	2	0
	Mild	2	2	1	2	3
	Moderate	0	0	0	0	4
	Total Incidence	**2**	**3**	**2**	**4**	**7**

Given that the systemic circulating thyroid hormones levels were not measured in that study, as stated by the study authors, the overall thyroid hormone status was difficult to interpret because the combined histological categorization added additional uncertainty. In addition, because thyroid gland dysfunction could potentially affect the reproductive functions in the animals, but yet there were no treatment-related effects on mating or fertility in any of the PFHxS-treated rats, there was no strong evidence to support thyroid-related effects based on this study.

In addition, ATSDR should recognize that in rodents, increased hepatocellular hypertrophy due to activation of hepatic nuclear receptors is often accompanied by increased thyroid follicular epithelial hypertrophy/hyperplasia (Capen 1997). This is a well-documented in rodents and it is primarily due to the increased hepatocyte mass (hypertrophy) overall will result in an increase in overall liver metabolism. The increased liver metabolism is capable of directing the circulating thyroid hormone for rapid turnover (with increased hepatic UDP-glucuronyl transferase). Consequently, to compensate for the higher turnover rate of thyroid hormones, there will be an increase in thyroid gland activity hence it is common to see hepatocellular hypertrophy and thyroid hypertrophy concurrently. Again, this observation is particularly well-known phenomenon in rodents but not in humans (see detailed discussion below) (Capen 1997; Curran and DeGroot 1991). Therefore, the observed increase in mild to moderate thyroid follicular epithelial hypertrophy and hyperplasia in the 10 mg/kg-d treatment group males was consistent with the increase in centrilobular hepatocellular hypertrophy associated with exposure to PFHxS. Again, it reflected the activation of xenosensor nuclear receptor activation in rats when exposed to PFHxS (Bijland et al. 2011; Bjork et al. 2011; Bjork and Wallace 2009; Chang et al. 2018).

Recognizing this uncertainty as well as the difference in serum toxicokinetics between female rats and female mice, a separate OECD 422 study was reported by Chang et al. (2018) and they demonstrated that thyroid hormone status in mice exposed to PFHxS (based on TSH levels and thyroid histopathology) was not altered. In that study, there was no effect of PFHxS on TSH in the adult F_0 mice or in the F_1 pups when serum TSH was measured at multiple times during their development; and, most importantly, there were no effect on thyroid histopathology. Therefore, there is no evidence to suggest that PFHxS impacts thyroid homeostasis.

C. ATSDR should recognize rodent-specific thyroid effects and their relevance to humans. In addition, there are significant differences exist in thyroid hormone physiology between rodents and humans. In human and non-human primates, circulating thyroid hormones are bound primarily to thyroid binding globulin (TBG) and this high-affinity binding protein is absent in rodents (Oppenheimer et al. 1995). Rodents mainly rely on serum albumin, which has lower affinity than TBG, as thyroid hormone carriers. The plasma thyroid hormone half-life is considerably shorter (12 – 24 hours) than in humans (5 – 9 days) (Capen 1997). It has been well demonstrated that, between rodents and humans, these difference in plasma half-lives of thyroid hormones and binding affinity to carrier proteins attribute to a greater sensitivity of rodents (but not humans) in developing hypertrophic and hyperplastic lesions (Capen 1997; Curran and DeGroot 1991).

In summary, ATSDR should recognize that there are distinct differences in thyroid hormone regulations between rodents and humans; and similar to hepatocellular hypertrophy noted in rats, thyroid findings in rodents require careful (weight-of-evidence) interpretation when extrapolating to human risk assessment.

D. HED for PFHxS will be higher when considering faster half-life. In the MRL calculations, ATSDR chose to use the arithmetic mean serum elimination half-life estimate for PFOA from Olsen et al. (2007) over other studies because Olsen et al. had a longer follow up time and ATSDR was concerned that based on a study by Seals et al. (2011), slower kinetics is likely to constitute a larger contribution to the terminal half-life. For example, whereas Olsen et al. had an average follow-up of 5 years, Bartell et al. had a follow-up of a year and Li et al. had a follow-up of 2.3 years among those studies that followed individuals and were not cross-sectional analyses of populations. However, this line of reasoning by ATSDR for selection of the arithmetic mean from the Olsen et al. study fails to take into account several factors that likely biased upwards the ATSDR MRL estimates. These include the following points.

1. The ATSDR chose not to use the geometric mean estimate that was discussed in the Olsen et al. paper. Given the right skewness of their data, Olsen et al. were more favorable to use the geometric mean for a measure of central tendency. ATSDR provided no explanation as to why they chose the arithmetic mean vs. the geometric mean in this study. This decision is interesting (and curious) because ATSDR chose to report median initial and final concentrations in Table A2 rather than the arithmetic mean initial and final concentrations in Table A2. A median concentration would be better represented by a half-life estimate based on the geometric mean.

2. The Olsen et al. 2007 study comprised 26 retirees (end of study average age = 66 years) who likely would have had an average glomerular filtration rate lower than those calculated from younger ages as reported in Bartell et al. (average age 55) and Li et al. (age range 15 – 55). The average estimated glomerular filtration rate declines with age as shown in the table below.

Age range	Estimated GFR (ml/min/1.73 m^2)	Source:
1-6 months	77	Heilbron et al. 1991 Pediatr Nephrol. Jan;5(1):5-11.
6-12 months	103	
12-19 months	127	
2-12 years	127	
20–29	116	https://www.kidney.org/sites/default/files/docs/11-10-1813_abe_patbro_gfr_b.pdf
30–39	107	
40–49	99	
50–59	93	
60–69	85	
70+	75	

Renal clearance of perfluorocarboxylates (and perfluorosulfonates) is largely a sum of three processes involving glomerular filtration, renal tubular secretion, and renal tubular reabsorption (Han et al. 2012). Because PFOA and other perfluorocarboxylates vary in their affinities to bind plasma proteins, glomerular filtration of perfluorocarboxylates (and perfluorosulfonates) is a product of the unbound fraction of the perfluorocarboxylate

and the glomerular filtration rate (GFR). Thus, the higher estimates of GFR based on the younger ages in the other study populations, especially the younger Li et al. study which had approximately 50% of the follow-up time of Olsen et al., may be due to the age differences of the subjects, and not the shorter follow-up period considered in these studies. Thus, the serum elimination half-lives are likely equally valid for consideration in MRL calculations.

3. The Olsen et al. study had to consider, during the course of their follow-up, the possibility of retirees reentering the 3M Decatur and Cottage Grove manufacturing plants. Indeed, this resulted in Olsen et al. eliminating 1 study subject entirely, and truncating follow-up times for two retirees. This would have biased estimates upwards for the serum elimination half-lives due to the increased exposure. It is not likely that ambient general population level concentrations would have biased these retiree's estimates substantially as discussed by Bartell et al. 2012. On the other hand, although Bartell et al. and Li et al. had shorter follow-up times, the primary exposure in these populations was through drinking water. Installation of GAC filters in these populations' affected municipal water supply would have immediately ceased their exposure to PFOA, PFOS, and PFHxS.

4. ATSDR suggests the Seals et al study indicates a lower clearance rate may occur as subjects are followed long-term post exposure; thus, the decision by ATSDR to use the study that had the longest follow-up time (Olsen et al. 2007). However, ATSDR did not mention the main limitations of the Seals et al. study: 1) the cross-sectional nature of the analysis. Individual subjects were not followed. Model-based estimates were instead calculated based on the initial concentrations; 2) there was the added assumption that there was uniform exposure based on the concentration of PFOA measured in each water district; and 3) subjects with initial PFOA concentrations < 15 ng/mL were excluded which maximized the probability of analyzing individuals with sufficiently high baseline PFOA concentrations that would not be at ambient levels.

5. Given the above additional considerations (beyond that of ATSDR's consideration about the length of follow-up), the MRLs, assuming same PODs from the same studies, are recalculated in the table below using the different serum elimination half-life values for PFOA, PFOS, and PFHxS that are reported in Bartell et al., Li et al., and Seals et al. Accordingly, the percent of the MRL that might be overestimated by the ATSDR using in their most conservative serum elimination value (arithmetic means from Olsen et al. 2007) would then result in a range of overestimations of the MRL for PFHxS between 14 and 38 percent. This type of sensitivity analysis is definitely needed in Appendix A for the MRL calculations to take into account the variation of serum elimination half-life estimates that have been reported in the literature that will be, in part, a function of the GFRs from the population studied. Given the fact that ATSDR has used developmental studies to calculate the PODs for their MRLs, it is therefore not justified to use the arithmetic mean half-life estimate based solely on retirees, in part, because the GFRs of older adults are markedly lower than adults of much younger age and people 65 years of age or older represent only approximately 15% of the general population Therefore the estimated half-lives should reflect the entire population, not just the upper tail, which can

be a reflection of lower GFRs that occur with age. Thus, calculation of serum elimination half-lives may be age, sex, and concentration-dependent. MRLs, based in part on half-lives, should reflect this diversity of inputs in their calculations.

Reference Study	Estimated Half-life		MRL (mg/kg/d)	% MRL over current ATSDR MRL
	Years	Days		
*ATSDR Estimate (arithmetic Mean from Olsen et al. 2007)	8.5	3100	1.57E-05	--
Olsen et al. 2007 (geometric mean)	7.3	2665	1.82E-05	14
Li e al. 2018	5.3	1935	2.51E-05	38

As illustrated above, because HED and MRL are dependent of the clearance rate used, the resulting MRL for PFHxS can differ substantially and could be 14 to 38% higher than the current provisional MRL proposed by ATSDR.

E. Wambaugh benchmark dose model used by ATSDR was not optimized. Similar to comments provided above for PFOS and PFOA, the MRL is largely based on uncertainty rather than on supportable science derived from Wambaugh model. Again, ATSDR relied on animal PBPK model to predict subsequent POD of MRL derivation, but on the other hand, it has also explicitly stated that "Although physiologically based pharmacokinetic (PBPK) models have been developed for rodents and humans, these models are not sufficient to allow for comparisons between administered doses in laboratory animals and serum concentrations in humans" (*cf.* page 5 of draft profile). This statement indicated a great amount of uncertainty associated with the PBPK model used hence ATSDR needs to acknowledge this fact in its summary.

F. Uncertainty factors used by ATSDR were overly conservative and not supported by scientific data. They include:

1. Use of "3" for animal-to-human, in addition to large dosimetric TK adjustment, is not scientifically justified. While 3M agrees with ATSDR in principle to adjust for toxicokinetic difference between human and rodent serum clearance of PFHxS, which represented a factor of 15,500 based on ATSDR's derivation, 3M does not agree an additional factor of "3" is needed to account for uncertainty in using laboratory animal data to derive human exposure levels. This, in fact, represents an adjustment of 46,000 when taking dosimetry into account. The use of an additional factor of 3 to account for rodent-to-human toxicodynamic difference is unnecessary and not scientifically justified.

   More specifically, ATSDR has derived its proposed MRL based on the rodent developmental data. Because humans are considerably less sensitive to the pleiotrophic effects of xenosensor nuclear receptors such as PPARα, CAR/PXR activation compared to rodents (Corton et al. 2014; Elcombe et al. 2014; Gonzalez and Shah 2008; Klaunig et al. 2003; Klaunig et al. 2012; Lake 2009; Ross et al. 2010), the qualitative differences brings into question the relevance of rodent developmental effects with exposure to PFHxS and biological significance to humans. Thus, the very large

dosimetric adjustment of 15,500 more than adequately compensates for the additional factor of 3 for difference between rodents and human extrapolation. ATSDR should not apply another factor of 3 for animal to human when this uncertainty is already embedded in the large adjustment for the dosimetric difference.

2. Additional factor of "10" for human variability is overly conservative. For the PFHxS MRL, ATSDR included a factor of 10 for human variability. If ATSDR could have developed a more appropriate PBPK model that accounted for life stage differences in humans (rather than relying on rodent model), this factor of 10 for human variability could potentially be reduced.

3. Scientifically unjustified use of "10" for concerns on database limitations, especially on immunotoxicity and general toxicity. ATSDR stated that there is limited toxicology database on PFHxS, especially with regards to immunotoxicity and general toxicity. This is not correct.

   Albeit the number of publications on PFHxS is fewer than PFOS or PFOA, the available studies (to date) on PFHxS have addressed many key toxicity endpoints such as liver and cholesterol under repeated dose conditions following comprehensive macroscopic and microscopic examinations (Bijland et al. 2011; Butenhoff et al. 2009a; Chang et al. 2018). ATSDR is incorrect in stating that there are limited "general toxicity" information on PFHxS.

   Furthermore, with regards to the immunotoxicity, ATSDR has not justified the relevance of existing studies to human risk assessment. Studies by Butenhoff et al. (2009a) and Chang et al. (2018), repeated oral treatments of PFHxS to either adult male rats or mice for 42 days, and, pregnant dams from the beginning of gestation to the end of lactation, had no effects on the weights (absolute or relative) or the histology of the primary immune organs, including thymus, spleen, lymph nodes, or bone marrow. These data clearly support an absence of effects on immune function, which was the conclusion by ATSDR (on Table 2-5 of the draft profile).

   Therefore, the default database uncertainty factor of "10" is not scientifically justified and should be removed by ATSDR.

Detailed Comments on Pregnancy-induced hypertension / pre-eclampsia (PFOA, PFOS)

ATSDR Position

ATSDR concluded there is "*suggestive epidemiological evidence for an association between serum PFOA and PFOS and pregnancy-induced hypertension/pre-eclampsia.*" For PFOA, evidence was based on 6 studies: 4 cross-sectional (Nolan et al. 2010; Savitz et al. 2012a; Savitz et al. 2012b; Stein et al. 2009) 1 prospective cohort (Darrow et al. 2013) and 1 case-cohort (Starling et al. 2014). For PFOS, evidence was based on 3 studies (Stein et al. 2009; Darrow et al. 2013; Starling et al. 2014).

3M Comments on Preeclampsia

It is unclear why ATSDR combined pregnancy-induced hypertension and pre-eclampsia into a single health outcome. While both diseases are defined by new onset of hypertension that develops after the 20th week of pregnancy, preeclampsia is a far more serious complication of pregnancy often characterized by proteinuria and/or signs of clinical pathology to another organ system. Further, the American College of Obstetricians and Gynecologists recognizes pregnancy-induced hypertension and pre-eclampsia as two distinct types of hypertensive disorders with differing diagnostic criteria and disease management strategies (American College of Obstetricians and Gynecologists 2013). The ATSDR provided no scientific justification for combining these two distinct pregnancy outcomes.

Of the 6 studies referenced by ATSDR, only 3 specifically evaluated preeclampsia in relation to maternal exposure levels of PFOA and/or PFOS (Stein et al. 2009; Savitz et al. 2012a; Starling et al. 2014). These studies differed by several important factors (which were not addressed in the ATSDR draft profile) including study design, exposure assessment and preeclampsia assessment. These differences are discussed below.

Both Stein et al. (2009) and Savitz et al. (2012a) were cross-sectional studies of a highly exposed community population in the Mid-Ohio Valley region (C8 Health Study). In both studies, self-reported preeclampsia was obtained via questionnaire. This was a major deficiency of these studies given that self-reported preeclampsia has a low positive predictive value (~50-60%) when validated against medical records (Stuart et al. 2013). Further, study participants were aware of their exposure status (i.e. PFOA and PFOS levels), which likely introduced some level of recall bias. In addition, Stein et al. (2009) obtained self-reported preeclampsia outcomes between 2000-2006, which preceded PFOA, and PFOS serum measurements by approximately 5 years (*i.e.*, temporality would be difficult to establish). Savitz et al. (2012a), on the other hand, examined pregnancy outcomes from 1990 to 2004 in relation to modeled PFOA exposure. The model was based on serum PFOA measurements in 2005, residential histories, historical information on PFOA releases, environmental distribution and pharmacokinetic modeling. The authors reported an overall correlation of 0.67 between predicted (modeled) and observed serum PFOA levels measured in 2005-2006 and stated that "our estimates undoubtedly

introduced some misclassification" (Savitz et al. 2012a). This study observed a significant positive association for risk of preeclampsia when modeled PFOA was analyzed per 100 ng/mL increase (OR = 1.08, 95%CI: 1.01-1.15); however, no significant findings were observed when estimated serum PFOA concentrations were evaluated in quintiles (i.e., no dose-response) or per interquartile increase in the log transformed estimates. (Note: The ATSDR did not cite these null findings in the draft profile). Additionally, Stein et al. (2009) reported no significant association between self-reported preeclampsia and measured PFOA levels. Preeclampsia was, however, significantly associated with PFOS levels above the median (OR = 1.3, 95%CI: 1.1-1.7) and levels above the 90th percentile (OR = 1.6: 95%CI: 1.2-2.3), but not for levels below the 90th percentile or when PFOS was examined per increase from the 25th to the 75th percentile. (Note: Again, ATSDR failed to cite these findings in the draft profile).

The most recent study (Starling et al. 2014) to examine the potential association between preeclampsia and PFAS levels was a case-cohort study of 976 women enrolled in the Norwegian Mother and Child Cohort. Unlike studies by Stein et al. (2009) and Savitz et al. (2012a), Starling et al. (2014) was the only study to measure maternal plasma PFOA levels during mid pregnancy. Furthermore, it was the only study to use medically validated preeclampsia cases (466 cases and 510 non-cases) and include nulliparous women. Since parity is an important risk factor for preeclampsia, the exclusion of parous women was a notable strength of the study. Moreover, the inclusion of nulliparous women ensured that measured PFAS levels were not affected by recent declines in body burden due to prior pregnancies and lactation (Starling et al. 2014). This study reported no significant associations between risk of preeclampsia and measured PFOA and PFOS when analyzed in quartiles and as a continuous variable. It is important to note that while PFOA and PFOS levels in this study represented general population levels, the median PFOS concentration was approximately equal to the Mid-Ohio River Valley levels reported by Stein et al (2009).

3M Conclusion on preeclampsia

The evidence for an association between preeclampsia and PFOA and PFOS exposure is limited to 3 epidemiologic studies with inconsistent findings. When considering the important limitations of 2 studies (Stein et al. 2009; Savitz et al. 2012a), and the null findings of the methodologically strongest study (Starling et al. 2014), there is insufficient evidence of an association between preeclampsia and PFOA and PFOS exposure.

3M Comments on pregnancy-induced hypertension

Like the preeclampsia studies, only 3 studies specifically examined the association between pregnancy-induced hypertension (PIH) and PFOA and PFOS levels: 2 cross-sectional studies (Nolan et al. 2010; Savitz et al. 2012b) and one prospective cohort, with some cross-sectional analysis (Darrow et al. 2013). All three studies examined a highly exposed community population in the Mid-Ohio Valley region. Again, the ATSDR draft profile failed to acknowledge notable limitations (or strengths) of these studies and

provided no interpretation of the results. As such, study limitations and overall findings are briefly discussed below.

Nolan et al. (2009) examined the relationship between PIH and residential drinking water with elevated PFOA levels from the Little Hocking Water Association (LHWA). While this study was strengthened by use of medically validated cases of PIH, it was severely limited by lack of individual PFOA exposure measurements. Rather, water service category (LHWA only versus partial LHWA) served as a proxy for high versus low PFOA exposure. The study reported a nonsignificant unadjusted OR = 1.2, 95% CI: 0.7-2.0 and concluded that PFOA was not associated with an increased risk of maternal risk factors (Nolan et al. 2009).

Savitz et al. (2012b) examined the potential relationship between modeled serum PFOA estimates and PIH obtained from birth records in two separate analyses. Both analyses used modeled serum PFOA of the mother at 4 months of gestation. As stated previously, the study authors acknowledged that this modeling approach "undoubtedly introduced some misclassification" of PFOA exposure (Savitz et al. 2012a). In the first analysis (Study 1), models were based exclusively on the residential address listed on birth certificates. In the second analysis (Study 2), birth records were linked with lifetime residential history based on self-reported survey data. In Study 1, the authors reported "no consistent evidence of an association between estimated PFOA exposure and still birth, pregnancy-induced hypertension, preterm birth, or indices of fetal growth" and in Study 2, the authors reported that "PFOA was unrelated to pregnancy-induced hypertension" (Savitz et al. 2012b).

Darrow et al. (2009) was a prospective analysis of measured maternal PFOA and PFOS serum levels (2005-2006) and PIH cases (n=106) ascertained from birth records between 2005 and 2010). It is important to note, however, that 25% of the births preceded PFOA and PFOS serum measurements. Furthermore, PFAS levels measured in 2005-2006 may not have reflected PFAS levels at the time of follow-up (2008-2011), especially among women with reduced PFAS body burden due to multiple pregnancies and lactation. PFOA and PFOS were analyzed as continuous variables (per unit increase and per interquartile increase), and as quintiles among all births and separately for the first pregnancy conceived after serum measurement among nonpregnant women. For PFOA, among all births, significant associations were observed between PIH and PFOA analyzed as per in unit increase and as quintiles (with a significant dose-response). No associations were observed when PFOA was analyzed as per interquartile increase. More importantly, no significant associations were observed for any PFOA metric among first pregnancies conceived after serum measurement. (Note: this information was not cited in the ATSDR draft profile). For PFOS, among all births, significant associations were observed between PIH and PFOS analyzed as per in unit increase and as quintiles (with no significant dose-response), but not when PFOS was measured as per interquartile increase. Among first pregnancies conceived after serum measurement, significant associations were observed for both continuous variables and for quintile 3 only with no significant trend. Overall, inconsistent results were observed within the study and no evidence of a monotonic increase in risk was reported. The authors concluded that

"*results provide some evidence of positive associations between measured serum perfluorinated compounds and pregnancy-induced hypertension*" but also acknowledge that "*more refined outcome classification is warranted*".

3M Conclusion on Pregnancy-induced Hypertension

Only three studies have examined the association between PFOA exposure and PIH and have reported mixed results. Although Darrow et al. (2013) observed significant positive associations, the other two studies (Nolan et al. 2009; Savitz et al. 2012b) did not. Given the inconsistency in findings within the Darrow et al. (2013) study and across all 3 studies, and the fact that no independent confirmation of these findings outside the community population in the Mid-Ohio Valley region exists, the evidence of an association between PIH and PFOA exposure is limited. Further, given that Darrow et al. (2013) is the only study to have examined PIH in relation to PFOS exposure and reported mixed findings with no significant trend, therefore there is insufficient evidence of an association between PIH and PFOS exposure.

Detailed Comments on Hepatic Enzymes (alanine aminotransferase, ALT)

ATSDR position.

On page 5, ATSDR wrote, "Although a large number of epidemiology studies have examined the potential of perfluoroalkyl compounds to induce adverse health effects, most of the studies are cross-sectional in design and do not establish causation. Based on a number of factors including the consistency of findings across studies, the available epidemiology studies suggest associations between perfluoroalkyl exposure and several health outcomes."

According to the ATSDR, this includes "liver damage, as evidenced by increases in serum enzymes and decreases in serum bilirubin levels (PFOA, PFOS, PFHxS)." Noted on page 147, ATSDR wrote, "Occupational exposure and community studies did not find increased risk of liver disease associated with PFOA or PFOS. As assessed by serum enzyme and bilirubin levels, the epidemiology studies provide suggestive evidence of liver damage. Increases in aspartate aminotransferase (AST), alanine aminotransferase (ALT), and gamma-glutamyl transferase (GGT) levels and decreases in serum bilirubin levels have been reported in occupational, community and/or general population studies. Although there is considerable variability across studies, the evidence is adequate for PFOA, PFOS, and PFHxS, particularly for ALT levels." Presented on pages 148-149 is Table 2-10, which displays a summary of liver disease in humans. On pages 150-156 is a summary of alterations in serum hepatic enzymes and bilirubin levels in humans. There were 13 cross-sectional studies (not counting duplicate references) and 3 longitudinal studies. [Note: Some of these studies are mislabeled as cohort studies in the draft Supporting Document for Epidemiological Studies when they are, in fact, cross-sectional studies. See Table 7 (Gilliland and Mandel 1996; Mundt et al. 2007; Olsen et al. 2000, 2003; Olsen et al. 1999) (both cross-sectional and cohort).] Liver disease and hepatic enzyme findings are discussed for PFOA on pages 170-172 with summary on page 186 where ATSDR wrote, "Exposure to PFOA does not appear to be associated with increased risks of liver disease in workers or highly exposed community members. The epidemiology studies have found associations between serum PFOA levels and increases in serum ALT, AST, and GGT enzyme levels and decreases in serum bilirubin levels. However, the results have not been consistently found, and serum enzyme levels were typically within normal range. Four studies examined the risk of serum enzyme levels outside of the normal range; the results were mixed for the risk of elevated ALT, with two studies finding and increased risk and two studies finding no association." For PFOS, the discussion of liver disease and hepatic serum enzymes and bilirubin is found on pages 187-188 with the ATSDR summary on page 196 where ATSDR wrote, "The available occupational exposure studies or general population studies do not consistently suggest an association between PFOS exposure and increases in the risk of liver disease or biliary tract disorders. A small number of occupational exposure studies have not found associations between serum PFOS levels and increases in ALT, AST, or GGT levels." The only mention of PFHxS is on page 197 were ATSDR cited the Lin et al. (Lin et al.

2010) study and that they did not find associations between ALT and GGT levels with PFHxS levels in the NHANES data set that they analyzed.

3M Comments

ATSDR mischaracterized the epidemiological data as it relates to ALT and PFOA and its use of the phrase "liver damage". ALT is a "leakage" enzyme and may be increased due to necrosis, injury or repair (Cattley and Cullen 2013). Increases of two- to four-fold in rodents, canines, non-human primates, and humans indicate hepatic injury. As defined by (Hall et al. 2012),"Based on the recommendations of regulatory authorities, (EMEA 2010; FDA 2009; HED 2002) increases in ALT activity of two-to threefold should be considered as indicated of 'hepatocellular damage.' As will be discussed below, those studies that have suggestion of an elevation of ALT remain well-within the expected physiologic range of measured ALT. Using the term 'damage' in this context is therefore highly misleading. It is also possible to have quite modest but statistically significant increases in ALT that are not toxicologically relevant (Cattley and Cullen, 2013). It should be noted that the human half-life of ALT is approximately 47 hours with significant variation of 10 – 30% on a day-to-day basis with significant circadian variation (Cordoba et al. 1999; Kim et al. 2008). ATSDR failed to mention this when cohort studies are conducted examining estimated serum PFOA concentrations over time when there is only a single ALT value reported. Finally, it should be noted that nonalcoholic fatty liver disease is the most common cause of mild elevations of liver enzymes (Giannini et al. 2005).

Several studies are worthy of careful evaluation in this ATSDR Toxicological Profile as it relates to ALT and PFOA either because: 1) the size of the population studied that was exposed to PFOA via the drinking water, 2) the study concerned occupational populations, or 3) the study was experimental and based on a phase 1 clinical trial in humans designed to ascertain the maximum tolerated dose of PFOA (ammonium salt). Three studies concerning exposure to PFOA via drinking water were from the C8 Science Panel (one cross-sectional (Gallo et al. 2012), and the other two were cross-sectional and longitudinal based on an estimated cumulative serum (ng/mL-year) model (Darrow et al. 2016). Four studies were occupational studies including two cross-sectional studies (Olsen and Zobel 2007; Sakr et al. 2007a) and two longitudinal studies (Olsen et al. 2012; Sakr et al. 2007b). One study was an experimental phase 1 clinical trial (Convertino et al. 2018). Collectively, these studies do not suggest "liver damage" (see above 2 to 4-fold increase) as measured by ALT associated with increasing serum concentrations of PFOA. Although some studies' regression coefficients for PFOA may be statistically significant, the percent variation explained of ALT by PFOA is minimal, at best, and the elevation of ALT very modest (generally an increase of 1 to 3 IU ALT). Nor is there any evidence of increased mortality from increased liver disease in epidemiologic analyses of community-based exposure to PFOA (Darrow et a. 2016) or in occupational cohort mortality studies (Steenland and Woskie 2012); (Raleigh et al. 2014).

Several types of studies are discussed below.

Community studies (n = 2)

Gallo et al. (2012). Gallo et al. reported on the C8 Health Project cross-sectional data collected in 2005-2006. They found a positive association between PFOA and serum ALT. Based on 3 different regression models, Gallo et al. reported statistically significant ln-PFOA (ng/mL) beta coefficients in models where lnALT was the independent variable. What is most important to note is that these three models had an increasing number of covariates (2, 7, and 11) besides PFOA in each model. The R^2 of these three models were 0.170, 0.174, and 0.265, respectively. However, the partial R^2 for PFOA (difference between R^2 including and excluding PFOA) remained 0.002, 0.001, and 0.002 for these three models, respectively. This clearly does not suggest that PFOA was a substantive contributor to the increase of ln ALT as it only explained between 0.1 and 0.2 percent of the variance of ln ALT, although the coefficient was statistically significant because of the study sample size (N = 47,092). The ATSDR failed to mention this very low partial R^2 in the regression modeling that was done by Gallo et al. Based on their fitting values of ALT by deciles of PFOA (given the mean values of the covariates), Gallo et al. showed a mean (untransformed) ALT of approximately 20.9 IU/L reported at 6 ng/mL PFOA that increased to approximately an ALT of 22.2 IU/L at 30 ng/mL PFOA (+1.3 IU/L increase in ALT) but plateaued thereafter. The highest decile was 23 IU/L ALT associated with approximately 320 ng/ml PFOA. It should be noted that the upper reference range (depending on laboratory) for ALT is approximately 45 IU/L.

Darrow et al. (2016). In their cross-sectional analysis, they suggested the results of the C8 Science Panel's community worker cohort study were consistent with the Gallo et al. (above) showing an increasing trend in the β coefficients across quintiles where estimated serum PFOA in 2005-2006 was Quintile 1 (2.6-<5.8 ng/mL PFOA; Quintile 2 5.8-<11.4 ng/mL; Quintile 3 11.4-<26.7 ng/mL PFOA; Q4 26.7-<81.5 ng/mL PFOA; and Q5 81.5-3558.8 ng/ml PFOA. There were up to 11 covariates in these models, which were the same as model 3 in Gallo et al. Darrow et al. did not provide R^2 or partial R^2 values in these cross-sectional analyses.

In their analysis of estimated cumulative exposure of PFOA in the C8 Science Panel's community and worker study on liver function and disease (Darrow et al. 2016), Table S1 (see supplement) of Darrow et al. provided the linear regression coefficients for ln-transformed ALT per ln PFOA. These coefficients for PFOA for the 3 models were Model 1 (β = 0.003); Model 2 (β =0.012); and Model 3 (β = 0.011) adjusted for the same number of covariates in addition to PFOA (2, 7, and 11). The R^2 for these 3 models were 0.15, 0.232, and 0.235 respectively, similar in magnitude to Gallo et al. (see above paragraph) of 0.170, 0.174, and 0.265 for the same models adjusted for the covariates in their cross-sectional analysis, although PFOA in Darrow was an estimated cumulative ng/mL-year metric versus

measured (ng/mL). However, unlike Gallo et al., Darrow did not show the partial R^2 for PFOA. Because the coefficients of determination for the Darrow et al. models 1, 2, and 3 are very similar to Gallo et al. (despite a different metric for PFOA), it is highly likely the partial R^2 for PFOA in the Darrow et al. study also remained in the extremely low range of 0.001 (0.1%) to 0.002 (0.2%), thus ln PFOA (ng/ml-years) probably explained very little of the variance of ln ALT in the Darrow et al. paper in Table S1.

Darrow et al. also estimated, via modeling, the estimated cumulative serum PFOA concentration (ln ng/mL-year) and reported (compared to the reference quintile) the following percent change in ALT per increased quintiles of estimated cumulative PFOA where: Quintile 1 (reference); Quintile 2 (191.2-<311.3 ng/mL-years PFOA) 2.3%; Quintile 3 (311.3-<794.1 ng/mL-years PFOA) 3.6%; Quintile 4 (791.4-<3997.6 ng/mL-years PFOA) 4.0%; and Quintile 5 (3997.6-205667.3 ng/mL-years PFOA 6%. In other words, at least a 10X (one order of magnitude or higher) increase in estimated cumulative PFOA in this C8 Science Panel's community workers cohort study resulted in a 6% increase (95% CI 4% to 7.9%) in the ALT. For example, if Quintile 1 reference had an ALT value of 25 IU/L, the ALT value for Quintile 5 would be 26.5 IU/L, adjusted for the 11 covariates. If the ALT value would have been 45 IU/L (upper end of normal) for ALT for Quintile 1 adjusted for the 11 covariates, the corresponding ALT value for Quintile 5 (at least an order of magnitude higher in cumulative PFOA concentration) would be 47.7 IU/L. Given the very slight change in these ALT values over a large range (at least 10X) of estimated cumulative serum PFOA concentrations, a change of just 6% in an ALT would be, for all purposes, considered clinically insignificant. This point should be emphasized by ATSDR because Darrow et al. did not report any increased risk for any liver disease or the subcategory of enlarged liver, fatty liver or cirrhosis as related to PFOA in this community worker cohort study. Based on a 10-year lagged exposure, the hazard ratios (95% CI) for these three liver diseases were Quintile 1 (reference); Quintile 2: 1.04 (0.82, 1.50); Quintile 3: 0.91 (0.64, 1.31); Quintile 4: 0.84 (0.59, 1.21); and quintile 5: 0.87 (0.61, 1.25). The hazard ratio for those prospectively followed since 2006 were Quintile 1 (reference); Quintile 2 (1.19 (0.75, 1.88); Quintile 3: 1.02 (065, 1.61), Quintile 4 (0.94 (0.60, 1.48), and Quintile 5: 0.92 (0.58, 1.47).

Thus, it would be highly inappropriate for ATSDR to continue to suggest that the enzyme findings from the Darrow et al. (or Gallo et al.) suggest "liver damage" is associated with PFOA. In fact, the C8 Science Panel (2012) stated the obvious as they interpreted their own research,

> "*From our studies of patterns of diagnosed liver disease there is no evidence of any increased risk of liver disease in relation to PFOA exposure. Based on our studies of liver enzymes and inconsistent findings in reported literature there is some evidence of small shifts in liver function, mainly within the normal physiologic range, being associated with increasing PFOA exposure. It is uncertain if PFOA is the cause of*

the association, but if so there is no evidence that this is reflected in any increase in overall incidence of diagnosed liver disease. Therefore, the Science Panel does not find a probable link between exposure to PFOA and liver disease."

Furthermore, this line of reasoning by the C8 Science Panel is in agreement with the ATSDR Toxicological Profile (page 24), which stated,

"It should be noted that although the data may provide strong evidence of an association, it does not imply that the observed effect is biologically relevant because the magnitude of the chance may be within the normal limits or not indicative of an adverse health outcome."

[NOTE: The C8 Science Panel findings were based on "probable link" assessments that were defined as part of a settlement agreement and do not indicate causation (Steenland et al. 2014)]

Occupational Studies (n = 4)

Sakr et al. (2007a) conducted a cross-sectional analysis of 1,025 active workers at the DuPont Washington Works plant. Median serum PFOA concentrations among 259 of the workers assigned in PFOA (ammonium salt) production areas was 494 ng/mL (range 17 – 9,550). Lesser exposed groups with more intermittent or past exposures had median PFOA concentrations ranging from 114 to 195 ng/mL. Based on a linear regression analysis with 6 other covariates (model R2 = 0.276), the regression coefficient for ALT was not statistically significant (β= 0.023, p = 0.124). Examining only those workers not taking cholesterol lowering medications (n = 840), the regression coefficient became β = 0.031, p = 0.071.

Sakr et al. (2007b) also conducted a longitudinal analysis of ALT and PFOA that involved 231 workers and their measured ALT. The regression coefficient for PFOA was not statistically significant (β= 0.54, 95% CI -0.46, 1.54).

Olsen and Zobel (2007) reported on a cross-sectional study of 506 male 3M workers, not taking cholesterol lowering medications, working at 3 different production sites. Analyzed by deciles, they reported the adjusted mean of the 1st decile was 29 IU/L (95% CI 25 – 33) compared to the mean of the 10th decile (95% CI 30 – 38). These means were not statistically significantly different. The median PFOA concentrations were 60 ng/mL (range 7 – 130) in the first decile compared to 4,940 (range 3,710 – 92,030) in the 10th decile. An adjusted (age, BMI, alcohol) regression analysis that examined ln ALT and ln PFOA resulted in a coefficient for ln PFOA of 0.0249 (p-value 0.06). A different analysis that substituted triglycerides for BMI resulted in an adjusted coefficient of 0.0115 (p-value 0.40). The latter was examined because ALT can also be elevated due to dyslipidemia (see below discussion).

Olsen et al. (2012) conducted a longitudinal analysis of workers who were engaged in the decommissioning, demolition and removal of production buildings that were involved with the production of perfluoroctanesulfonyl fluoride (POSF) and PFOA. This remediation work occurred over a 2-year time period although not all workers were engaged for that period of time. Baseline clinical chemistries and perfluoroalkyl measurements were taken before a worker became involved with the project, which was followed by similar end-of-project measurements. Of 120 workers with baseline concentrations < 15 ng/mL PFOA and < 50 ng/mL PFOS, their median increase at end-of-project was 5.3 ng/mL (mean 44.2 ng/mL) ($p < 0.0001$) and 0.7 ng/mL PFOS (median 4.2 ng/mL) ($p<0.0001$). Given these modest increases in serum PFOA or PFOS concentrations, there was no change in median ALT and the mean ALT change was -0.7 IL/L ($p = 0.53$).

Experimental study (n = 1)

Convertino et al (2018). A 6-week phase one clinical trial was conducted in Scotland to determine the maximum tolerated dose that could be provided with the weekly oral administration of PFOA (ammonium salt) for ultimately evaluating the chemotherapeutic potential of PFOA in solid tumors (Convertino et al. 2018). The study was a standard 3+3 dose escalation phase 1 study. Forty-nine subjects participated. Subjects received PFOA (ammonium salt) on a single weekly dose as high as 1200 mg week. Monitoring of clinical chemistries, including ALT, AST, GGT, alkaline phosphatase and total bilirubin were done. Based on analysis of the probability distribution functions, ALT was unchanged for any categorization with the highest PFOA category at 870 – 1530 μM (~360,000 – ~632,000 ng/mL) where a reduction of serum cholesterol consistent with a pharmacodynamic effect was evident. Given the study conditions, these authors concluded liver enzymes were not altered at PFOA concentrations that are 5 orders of magnitude greater than the general population measurements of PFOA.

General Population (NHANES) studies

It should be noted that several of the studies reported by ATDSR analyzed NHANES data. The challenges of using NHANES biomonitoring data to incorporate into any form of risk assessments has been well-described by Sobus et al. (2015). In this regard, both Lin et al. (2010) and Gleason et al. (2015) have analyzed multiple 2-year cycle NHANES cross-sectional data with liver enzymes and PFOA or PFOS. Due to its study design, ATSDR is well-aware that temporality cannot be determined in these NHANES cross-sectional studies. However, an equally important methodological limitation that has not been addressed by either Lin et al. or Gleason et al. with their analysis of NHANES data, or this ATSDR Toxicological Profile, relates to the analysis of liver enzyme data in relation with serum lipids. As shown by Deb et al. (2018), in their

analysis of NHANES data from 1999-2012 there is an association between measured liver enzymes and lipid levels. Deb et al. reported that LDL was associated with a 2-fold increase in odds of an elevated ALT and AST measurements. Thus, any association between perfluoroalkyls measurements and liver enzymes should consider at least adjusting for age, sex, race/ethnicity, and lipids. If lipids are associated with liver enzymes then lipids might be a confounder in studying the association between perfluoroalkyls and liver enzymes. However, some may suggest PFOA may be associated with lipids (at lower PFOA concentrations). Therefore, lipids, at low concentrations, might be on the causal path between the exposure (perfluoroalkyls) and increased liver enzymes. On the other hand, there is less evidence to suggest this path (higher lipids) exists at substantively higher perfluoroalkyl concentrations (see Convertino et al. 2018). Thus, the intermediate path of serum lipids might need to be considered in studying the association between perfluoroalkyls and liver enzymes. ATSDR offered no insights into this issue between perfluoroalkyls, lipids, and liver enzymes. What is certain, however, is there has not been reported to be an increased risk of self-reported liver disease in NHANES data (Melzer et al. 2010), in the Canadian Health Measures Survey (Fisher et al. 2013) as well as with medically validated liver disease with exposure to PFOA in the C8 Health Panel study (Darrow et al. 2016), including fatty liver disease. In this regard, with a lack of any increased risk for liver disease, it is inappropriate to infer very weak associations with ALT and measured perfluoroalkyls in populations whose serum PFAS concentrations can be orders of magnitude different. Thus, numerous confounding factors must be considered in analyses of ALT, including age, sex, body mass index (preferably waist-to-hip ratio as a measure of abdominal obesity), triglyceride level, total cholesterol, alcohol, glucose (women), physical activity, and smoking (the latter two are negatively correlated) (Kim et al. 2008).

3M Conclusion

There is no association between either PFOA or PFOS and liver disease including enlarged liver, fatty liver, or cirrhosis. Small percentage changes in ALT have been reported, albeit inconsistently in epidemiology studies across vastly different perfluoroalkyl concentrations, but are within normal physiological ranges. This small magnitude of change, if it is even present, does not indicate liver damage by any standard clinical practice of medicine. Confounding cannot be ruled out as a possible explanation for this observation due to the many factors that can influence ALT. Thus, there is insufficient evidence of an association with ALT.

Detailed Comments on Cholesterol

ATSDR position on PFOA and cholesterol

On page 5, the ATSDR wrote, "Although a large number of epidemiology studies have examined the potential of perfluoroalkyl compounds to induce adverse health effects, most of the studies are cross-sectional in design and do not establish causation. Based on a number of factors including the consistency of findings across studies, the available epidemiology studies suggest associations between perfluoroalkyl exposure and several health outcomes." According to ATSDR, this included "increases in serum lipids, particularly total cholesterol and low-density lipoprotein (LDL) cholesterol (PFOA, PFOS, PFNA, PFDeA)." On pages 156-169 is Table 2-12, which provides a summary of serum lipid outcomes in humans. For various studies: Figure 2-9 is a graph of percent change in total cholesterol relative to PFOA levels; Figure 2-10 provides elevated cholesterol adjusted risk relative to PFOA; Figure 2-11 is a graph of percent change in LDL relative to PFOA levels; Figure 2-12 provides elevated LDL adjusted risk relative to PFOA. Based on these figures and studies presented in the ATSDR text (pages 172, 177-182), ATSDR concluded (page 186), "studies examining the change in cholesterol per change in serum PFOA levels have found greater increases in serum cholesterol levels associated with serum PFOA levels at the lower range of PFOA levels and the dose-response curve suggests a biphasic relationship. Positive associations have also been observed for LDL cholesterol, although associations have not been consistently found. In general, no consistent associations were found between serum PFOA and HDL cholesterol or triglyceride levels." On page 187, ATSDR recognized "In contrast to the results observed in epidemiology studies, an experimental study in humans exposed to PFOA (MacPherson et al. 2011) and human exposure to other PPARα agonists, such as fibrates (Roy and Pahan 2009), suggest that hypolipidemic effects, similar to those observed in rodents, may occur in humans exposed to PFOA, although humans may not be as sensitive as rodents."

3M Comments on PFOA and Cholesterol

The ATSDR recognized (pages 181, 187) the preliminary results of a phase 1 clinical trial of PFOA (ammonium salt) that was published in 2010 as an abstract by MacPherson et al. (2011) in the J Clinical Oncology. The abstract stated "Reductions in LDL-cholesterol consistent with a PD effect were observed." The phase 1 trial was a dose escalation study with the highest weekly dose administered at 1200 mg PFOA (range 50mg – 1200 mg). ATSDR was not certain whether this effect occurred at all dose levels as such clarification was not present in the abstract. ATSDR was not aware that the results from the clinical chemistry assessment from this phase 1 trial have been available via Advance Access and published on February 16, 2018 in *Toxicological Sciences* with hardcopy publication in the May 2018 issue, (Convertino et al. 2018). ATSDR is strongly encouraged to carefully consider the Convertino et al. (2018) publication and its ramification(s) in ATSDR's weight of evidence review for PFOA as related to lipids (as well as liver enzymes and thyroid hormones).

According to Convertino et al. (2018), this phase 1 dose-escalation study assessed the chemotherapeutic potential of perfluorooctanoate (ammonium salt). There were 49 primarily solid-tumor cancer patients who failed standard therapy that received weekly doses of PFOA (50 – 1200 mg) for 6 weeks. The primary purpose of this study was to determine the dose limiting toxicity of PFOA. However, no more than one subject demonstrated a dose limiting toxicity at any dose level so a maximum tolerated dose was not reached. The 1000 mg weekly dose was the recommended phase 2 dose based on tolerability. Standard clinical chemistry measurements were performed at baseline examination and weekly thereafter. Not all subjects took the weekly dose so measured serum PFOA concentration, internal dosimetry, not dose administered, was considered the metric of choice. Statistical analyses included generalized estimating equations a probabilistic analysis using probability distribution functions at various PFOA concentrations, and a 2-compartment pharmacokinetic/pharmacodynamic model. According to Convertino et al., total cholesterol (and free T4 – see under thyroid) showed a negative trend with increased serum PFOA concentrations with a clear transition in shape and range of the probability distribution functions for a decrease in total cholesterol at approximately 420 and 565 μM PFOA (approximately 175,000 – 230,000 ng/mL PFOA). The effect observed involved LDL, not HDL, and is consistent with the toxicological evidence in rodents observed at approximately an order of magnitude lower concentration. The PFOA concentrations, however, reported by Convertino et al. in the phase 1 clinal trial are several orders of magnitude higher than those reported to occur in workers, an exposed West Virginia community, and the general population.

Based on the study abstract that was available to ATSDR (Macpherson et al. 2010), ATSDR speculated about the possibility of a biphasic response in the human with decreased cholesterol reported at higher PFOA concentrations and elevated cholesterol at markedly lower levels. However, the ATSDR did not offer any possible modes of action explanation for a biphasic response whereas Convertino et al. did. The ATSDR should offer their explanations for a biphasic response. At the high concentrations of PFOA administered and measured where the decrease became clear with total cholesterol, Convertino et al. suggested this hypolipidemic response was consistent with a xenosensor nuclear receptor PPARα-mediated mode of action. They then suggested the inconsistency with the observational epidemiological studies showing positive associations between cholesterol and markedly lower PFOA concentrations are likely the consequence of one or more noncausal biological explanations. These would include the inherent variability in the glomerular filtration rate which confounds other associations that have been reported with PFOA including lower birthweight and chronic kidney disease; organic transporters in the gastrointestinal tract that may share binding affinity with lipids and PFOA; saturation of an underling physiologic mechanism given the nonlinear association observed n between PFOA and cholesterol reported by Steenland et al. (2009) and Frisbee et al. (2010) that was also mentioned by the ATSDR (page 181); and PFOA binding to lipoproteins (also mentioned by ATSDR on page 181). Convertino et al. cautioned that the latter may not have been thoroughly examined as Butenhoff et al. (2012d) had an extremely low sample size (n = 1) and should be replicated in much larger numbers. Convertino et al. also urged examination of plausible biologic modes of action that could support the hypercholesterolemia positive association reported at low ng/mL

PFOA. They wrote, "these observational studies have reported contrary associations, but currently understood biology does not support the existence of such conflicting effects." And, in fact, many of the authors of the papers cited in Figures 2-9 through 2-12 discounted the contrary animal data as not being relevant to humans. This can no longer be accepted practice in the literature given the publication of Convertino et al. (2018). Clearly, more cross-sectional studies are highly unlikely to be enlightening to any scientific understanding. ATSDR agrees with this recommendation when they wrote on page 635, "Interpretation of the human data is limited by the reliance of cross-sectional studies, which do not establish causality, and the lack of exposure data."

ATSDR also wrote on page 635, "Studies of serum lipids suggest that the dose-response curve is steeper at lower concentrations and flattens out at higher serum perfluoroalkyl concentrations (Steenland et al. 2010), additional studies that could be used to establish dose-response relationships would be valuable. Mechanistic studies examining the association between perfluoroalkyl exposure and serum lipid level would also provide insight." Therefore, ATSDR and the scientific community (both toxicologists and epidemiologists) are urged to reassess the dose response curve in humans based on the one and only experimental study done in humans (Convertino et al. 2018).

In this regard, ATSDR should consider whether the associations observed in many epidemiologic studies (primarily cross-sectional) at the much lower general population and community levels for PFOA may actually be a reflection of underlying, yet-to-be identified, physiological processes that result in a noncausal lipid/PFOA biological associations. This includes ATSDR's desire, so stated above, to describe the mode of action likely at these low doses that results in the association with higher cholesterol that is entirely inconsistent with the animal and human toxicological evidence that has demonstrated at sufficiently high concentrations of PFOA results in hypolipidemia. Convertino et al. offered several possible noncausal explanations (see above) but other possibilities are also worthy of investigation. For example, not stated by Convertino et al., is the fact that thyroid disease and chronic kidney disease can both affect GFR. Both of these conditions are also associated with dyslipidemia. All three may affect the glomerular filtration rate. Dyslipidemia, itself, has also been associated with altered GFR. Therefore, a lowered GFR may maintain a higher amount of PFOA – creating the association observed in some epidemiology studies.

In summary, given the recent publication of Convertino et al., the ATSDR should acknowledge the consistency of pharmacodynamic effects (decreased cholesterol and LDL) in both animals and humans with high exposure to PFOA. It is therefore inaccurate to have written what ATSDR provided on page 634 when stated, "The effects observed in rodents differ from those observed in humans. In humans, exposure to PFOA, PFOS, PFNA, and PFDeA appear to result in increases in serum lipid levels, particularly total cholesterol levels."

3M Conclusion on PFOA and cholesterol

There is no association between PFOA and coronary artery disease, cerebrovascular disease (stroke), and hypertension. Very high concentrations of PFOA will unequivocally result in lowered serum total cholesterol involving LDL, not HDL cholesterol in experimental studies in both animals and humans. The mode of action is likely via PFOA acting on xenosensor nuclear receptors, including PPARα, which is common to many species, including humans. Fibrate pharmaceuticals that lower serum cholesterol in humans also bind to this same nuclear receptor family. The contrary association of higher cholesterol associated with low PFOA concentrations, as reported in several but not all observational epidemiology studies, remains yet to be understood as to its biological (causal or noncausal) plausibility.

ATSDR position on PFOS and cholesterol

ATSDR presented information on PFOS and cholesterol on pages 188-196, with figures presented on total cholesterol change (%) relative to serum PFOS level in Figure 2-13, risk of abnormal cholesterol with PFOS levels in Figure 2-14, and LDL cholesterol change (%) relative to serum PFOS level in Figure 2-14. Unlike PFOA, there are fewer studies presented in these figures for PFOS. Neither the occupational studies nor the community study (which was not exposed to PFOS in the drinking water) are presented in these figures. The ATSDR wrote there were positive associations reported between PFOS and cholesterol with the occupational (page 188) and community (page 188-189) studies but the results were mixed in the general population studies (page 193-194).

3M Comments on PFOS and Cholesterol

ATSDR cited the Olsen et al. 2003a study as well as Steenland et al. 2009 study as evidence for positive associations reported between PFOS and cholesterol. Not discussed by the ATSDR was the concern expressed by both investigators that although PFOS may have been significant predictors of lipid levels, PFOS did contribute much to the variance of the prediction. For example, Steenland et al. wrote, "It should be noted that although PFOA and PFOS are highly significant predictors of lipid levels (our study had high power to detect statistically significant differences compared with prior smaller studies), the perfluorinated compounds themselves did not explain a large portion of the variance in lipids." For total cholesterol, the most important predictors were age, gender, and body mass index, not serum levels of PFOS. Olsen et al. stated for their model of cholesterol where the $R^2 = 0.06$, the partial R^2 for PFOS was < 0.01.

Similar to the PFOA phase 1 clinical trial discussed above, the ATSDR should recognize (which it has not) the findings from Chang et al. (2017) regarding a non-human primate study where a slight reduction in serum cholesterol (primarily HDL) was reported with administration of PFOS (potassium salt) in a 6-month study of non-human primates. The corresponding lower bound 5th percentile benchmark concentration was 74,000 and 86,000 ng/mL for these male and female monkeys (cynomolgus), respectively. This

finding would suggest that at sufficiently high concentrations, PFOS is likely to result in lower (HDL, not LDL) serum cholesterol concentrations in humans.

3M Conclusion on PFOS and cholesterol

There is insufficient evidence to conclude an association exists between PFOS and lipids in the epidemiology literature.

Detailed Comments on Thyroid Disease

ATSDR position

On page 5 and 6, ATSDR wrote, "Although a large number of epidemiology studies have examined the potential of perfluoroalkyl compounds to induce adverse health effects, most of the studies are cross-sectional in design and do not establish causation. Based on a number of factors including the consistency of findings across studies, the available epidemiology studies suggest associations between perfluoroalkyl exposure and several health outcomes." According to the ATSDR, this includes "increased risk for thyroid disorders. (PFOA, PFOS)". Similar statement was provided on page 25. ATSDR provides Table 2-15 (pages 223-237) as a summary of thyroid outcomes in humans. This table contains both studies that reported both thyroid hormones as well as thyroid disease (self-reported as well as medically validated) in occupational, community-based and general populations. Study designs are not listed in these tables and the reader is referred to the supporting information. For PFOA (correcting for the study design misidentification discussed earlier in the supporting information), it appears that of the 21 studies listed in Table 2-15, 20 are cross-sectional with one study a cohort. For PFOS, 18 studies in Table 2-15 were cross-sectional and 1 study had a cohort component. ATSDR did not comment on this preponderance of cross-sectional studies as they discussed thyroid. The text presents a mixture of findings but no rationale of understanding provided by ATSDR. Unlike other sections, there are no summary statements in the thyroid section for either PFOA or PFOS.

3M Comments

ATSDR's review of the thyroid is disjointed and did not explain how it decided that an "association" exists between PFOA/PFOS and an increased risk of thyroid disease. This confusion is caused, in part, by the inconsistent evidence presented in the scientific literature. The lack of a summary statement by ATSDR indicate the lack of scientific support for the conclusion that ATSDR makes.

Primary hypothyroidism is clinically characterized by a high serum thyrotropin (TSH) concentration and a low serum free thyroxine fT4 concentration. Subclinical hypothyroidism is generally defined as a normal Ft4 in the presence of an elevated TSH. Hyperthyroidism is defined as a decreased TSH level and elevated free T4 and free T3 levels. Measuring specific antibodies, such as anti-TSH-receptor antibodies in Graves' disease, or anti-thyroid peroxidase in Hashimoto's thyroiditis — a common cause of hypothyroidism — may also contribute to the diagnosis.

As ATSDR wrote (page 238), there were "no associations between serum PFOA and TSH or T4 levels found in the general population studies except for Lewis et al. (2015). On page 222, ATSDR also wrote, "the occupational exposures do not suggest an association between serum PFOA and alterations in thyroid hormone levels." Further, ATSDR conceded that although TSH, T3 or T4 have been reported, "*the results are not consistent across studies (page 222).*" Thus, on a population analysis basis, trends in

thyroid hormone levels, in particular TSH (the primary clinical diagnostic indicator to diagnose hypo-or hyperthyroidism), is lacking with exposure to PFOA or PFOS.

In the abovementioned phase 1 clinical trial of PFOA (ammonium salt) (Convertino et al. 2018), the physicians examined for TSH and free T4, the usual two thyroid tests done for clinical thyroid assessment. The phase 1 trial study is described above in the lipids section. Based on the probability distribution functions, there was no change in TSH even at the highest concentrations of PFOA measured (highest category range was 870 µM - 1530 µM (µM (~360,000 ng/mL – ~632,000 ng/mL) PFOA. There appeared to be an increase in free T4 (fT4) at a higher PFOA transition point than reported for cholesterol. This increase with no apparent effect on TSH suggested to Convertino et al. that the increase in fT4 was not clinically significant but may be due to displacement of the thyroid bound hormone by PFOA. Such an effect is reported for PFOS in rats where displaced thyroxine from binding proteins transiently increases free thyroxine without altering overall thyroid hormone homeostasis (Chang et al. 2007,20008; Weiss et al. 2009).

In their analysis of NHANES data, Melzer et al. (2010) reported associations for females categorized as having "*current thyroid disease with thyroid medication*". However, they did not delineate by type of thyroid disorder (hypothyroidism, hyperthyroidism). Given the high prevalence of hypothyroidism in females, it can be presumed the majority of these prevalent female cases were hypothyroid. This finding was not supported by Winquist and Steenland (2014) in their analysis of the mid-Ohio river valley population who were exposed to drinking water that contained PFOA. Winquist and Steenland (2014) wrote in their study Abstract:

> *"Associations were observed for hyperthyroidism and hypothyroidism among women."*

However, this was not supported by their Discussion section where they wrote:

> *"We found evidence of an association between PFOA exposure and functional thyroid disease, especially for hyperthyroidism among women (in retrospective analyses) and for hypothyroidism among men (in prospective analyses)."*

This quote, however, is not supported by the ATSDR review of Winquist and Steenland (2014) where the ATSDR wrote on page 238, "*No associations between cumulative serum PFOA and hyperthyroidism or hypothyroidism were found in retrospective analysis (Winquist and Steenland 2014b). However, in prospective analysis, an association between cumulative serum PFOA and hypothyroidism was found in men (Winquist and Steenland 2014b).*"

Indeed, analysis of the Winquist and Steenland 2014 supporting information tables (see the eTable 1 through eTable 6 in Winquist and Steenland 2014) reported no statistically significant trends ($P < 0.05$) for hypothyroidism in women in either their retrospective, retrospective qualifying year, or prospective analyses. (This would be in direct conflict

with the findings from Melzer et al.). Altogether, there were 12 trend test analyses conducted (log linear model trend test p-values) in these supporting tables. For hypothyroidism, there were 0 trend tests among women with p-values < 0.05; 1 trend test with a p-value >= 0.05 and < 0.1; 3 trend tests with a p-value between >= 0.1 and < 0.2; and 8 trend tests with a p-value >= 0.2. These observations do not support an association between PFOA and hypothyroidism among women.

On the other hand, for hyperthyroidism among women, there were 4 trend tests with a p-value < 0.05; 2 trend tests with a p-value between >= 0.05 and < 0.1; 4 trend tests with a p-value between 0.1 and < 0.2; and 2 trend tests with a p-value >= 0.2. Among males, there were 4 trend tests with a p-value < 0.05 for hypothyroidism but none for hyperthyroidism.

ATSDR also reported (see page 222) that in a study published in 2015, Steenland et al. "did not find an association between serum PFOA and the risk of thyroid disease in male or female workers at the Washington Works facility," In fact, what Steenland et al. wrote, was "there was a positive non-significant trend for male hypothyroidism" where the 10 year lag trends in relative risk were 1.00 reference, 1.64, 1.13, 2.16 (p value trend via categories p = 0.06), however, their table presented this information as "thyroid disease" not differentiated to the type. Not discussed by Steenland et al. or by ATSDR, is the fact that there was an equally negative trend (not significant) in women for thyroid disease where the 10-year lag trends in relative were 1.0 reference, 0.79, 0.87, and 0.23; p value trend via categories p = 0.13).

3M Conclusion on thyroid disease

Given the inconsistencies in the literature regarding associations of thyroid hormones and thyroid disease, there is insufficient evidence to conclude an association exists as related to exposure to PFOA or PFOS.

Detailed Comments on Decreased Antibody Response to Vaccines (PFOA, PFOS, PFHxS, PFDeA)

ATSDR Position

The ATSDR draft document concluded that "*evidence is suggestive of a link between serum PFOA, PFOS, PFHxS, and PFDeA levels and decreased antibody responses to vaccines*". Evidence for this conclusion comes from 8 epidemiologic studies (4 cross-sectional and 4 prospective cohort) in which antibody titers to vaccinations were quantified in combination with measurements of serum PFOA, PFOS and other PFAS levels, coupled with supportive animal studies. Among the epidemiologic studies, antibody responses to 8 distinct vaccines (i.e., diphtheria, tetanus, mumps, measles, rubella, influenza A/H1N1, influenza A/H3N2 and influenza B) were measured. The most commonly studied vaccine response was to the tetanus vaccine with 5 studies (Grandjean et al. 2012; Grandjean et al. 2017; Granum et al. 2013; Kielsen et al. 2016; Mogensen et al. 2015) followed by 4 diphtheria studies (Grandjean et al. 2012; Mogensen et al. 2015; Kielsen et al. 2016; Grandjean et al. 2017), two rubella and measles studies (Granum et al. 2013; Stein et al. 2016b) and two influenza A/H3N2 studies (Looker et al. 2014; Stein et al. 2016a)). Antibody responses to mumps (Stein et al., 2016b), *H. influenza* (Granum et al., 2013), influenza B and influenza A/H1N1 (Looker et al., 2014) were each examined in only 1 study.

3M Comments

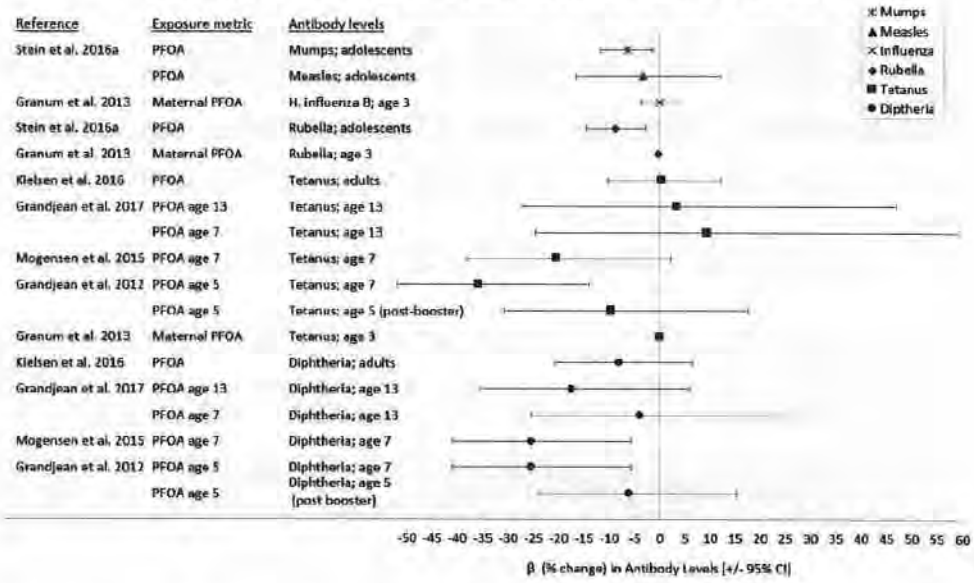
It is inappropriate for ATSDR to interpret antibody responses to these 8 distinct vaccines as a single health outcome (i.e., "decreased antibody responses to vaccines"). Commercially available vaccines differ depending on the nature of the vaccine antigen. Tetanus and diphtheria, for example, are toxoid vaccines whereas measles, mumps and rubella are live attenuated vaccines. Influenza vaccines are inactivated (killed), conjugate or live attenuated depending on the strain and method of administration (e.g., intranasal, injectable). Consequently, each vaccine type elicits an immune response through various molecular and cellular mechanisms of the immune system. Additionally, all vaccines contain various excipients including adjuvants to improve the antibody response, preservatives, stabilizers, and vehicles for delivering the vaccine which may differ substantially depending on the vaccine (Baxter 2007).

The National Toxicology Program acknowledged the differences in immune response across vaccines, and stated that "*The strength of an antibody response in terms of antibody level and length of time that an elevated/effective antibody response is maintained is known to differ across vaccines*" (NTP 2016). Granum et al (2013), a study cited in the ATSDR draft profile, also concluded that "*different vaccines may stimulate different components of the immune system, which can explain the vaccine-dependent differences in the effect of PFAS exposure*". Therefore, observed changes in antibody response to a particular vaccine should not be interpreted as consistent with

changes in the antibody response to another vaccine. **The ATSDR draft document should consider immune responses to individual vaccines as distinct health outcomes.**

The ATSDR draft profile graphically presents epidemiologic study findings (i.e., the changes in antibody levels relative to serum PFAS levels) in Figures 2-19 (PFOA), 2-21 (PFOS), 2-23 (PFHxS), 2-25 (PFNA) and 2-27 (PFDeA). These figures clearly illustrate the heterogeneity in results both within and across the 8 studies reviewed by ATSDR. For example, Figure 2-19 (below), shows that of the 5 studies that examined antibody responses to the tetanus vaccine relative to serum PFOA levels, only one study reported a significant decrease in antibody levels (Grandjean et al., 2012). The other 4 studies, including a follow-up study of Grandjean et al., 2012, did not observe a significant decrease in tetanus antibody levels (Grandjean et al., 2017).

Figure 2-19. Antibody Responses Relative to Serum PFOA Levels in Epidemiology Studies
(Presented as percent difference in antibody concentration per 2-fold increase in serum PFOA)



(Note: Not included in Figure 2-19 are results from two influenza studies with mostly null findings (Looker et al. 2014; Stein et al. 2016b). While both studies are cited in the draft profile, ATSDR should acknowledge that results from these two studies were omitted from the Figure and provide reasons for their omission.)

Similar to the results observed for PFOA, inconsistent results were also observed for PFOS, PFHxS and PFDeA. None of the 5 studies reported a significant association between tetanus antibody levels and PFNA. In addition, findings across all vaccine types were also inconsistent. As presented in Figure 2-19, for example, only 5 of the 18 associations between PFOA and a change in antibody levels were statistically significant. Similar inconsistencies across all vaccine types are also apparent for PFOS, PFHxS, PFNA, and PFDeA. Considering the inconsistent (and mostly non-significant) findings

across the 8 published studies, the available epidemiologic evidence of an effect of PFOA, PFOS, PFHxS and PFDeA on antibody response to vaccines is weak at best. Moreover, ATSDR failed to recognize that small changes in antibody response do not necessarily translate to an increased risk of infectious disease. Six epidemiologic studies ((Dalsager et al. 2016; Fei et al. 2010a; Leonard et al. 2008; Looker et al. 2014; Okada et al. 2014) have examined PFAS levels and infectious disease outcomes (i.e., occurrence of common colds and otitis media, mortality from infectious and parasitic diseases, and hospitalizations from infectious diseases). Most of these studies reported no association between PFAS levels and increased risk of infectious disease outcomes. As noted in the ATSDR draft profile (page 268), the NTP (2016) concluded that there is low confidence that exposure to PFOA and PFOS is associated with increased incidence of infectious disease (or lower ability to resist or respond to infectious disease). Other regulatory bodies have reached similar conclusions (FSANZ 2017; USEPA 2016a, b). Given the absence of increased infectious disease susceptibility, it is questionable whether the observed decreases in antibody response are clinically relevant.

Finally, the ATSDR did not provide an interpretation of the epidemiologic evidence or a conclusion regarding the potential association between PFAS levels and decreased antibody response to vaccines. Instead, ATSDR quoted the 2016 NTP conclusion (page 268) that "*exposure to PFOA or PFOS is presumed to be an immune hazard to humans*" while ignoring conclusions from other regulatory bodies and expert health panels. These conclusions (provided below) should be included in the ATSDR draft profile to provide readers with a more balanced and thorough interpretation of the epidemiologic evidence. It is inappropriate for ATSDR to cite a single conclusion from one regulatory body and not cite others with divergent conclusions.

Other regulatory have made the following conclusions regarding PFAS and immunotoxicity:

Australia Expert Health Panel (2018):
"*The strongest evidence for a link between PFAS and clinically important immunological effects is for impaired vaccine response. However, the human dose-response/threshold for potential immune effects is very poorly characterized, and the overall human evidence is weak.*"

Food Standards Australia New Zealand, FSANZ (2016):
A literature review commissioned by FSANZ concluded that *"there are both positive and negative studies showing associations for increasing PFOS and PFOA concentrations to compromise antibody production in humans. However, to date there is no convincing evidence for increased incidence of infective disease associated with PFOS or PFOA effects on human immune function".*

Health Canada (2017a):
"Studies in environmentally-exposed populations have identified associations between PFOS levels and decreased antibodies against various illnesses, but the influence of PFOS exposure on clinical immunosuppression (i.e., incidence of illnesses) appears to be

more tenuous." Health Canada further commented that "*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.*" Health Canada reached similar conclusions regarding PFOA (Health Canada, 2017b).

National Institute for Public Health and the Environment (RIVM, 2016):
RIVM concluded that "*associations have been found between exposure to PFOA and a decreased vaccination response*", but the "*evidence is unclear*".

New Jersey Drinking Water Quality Institute (DWQI, 2017):
"Review of epidemiologic studies provides evidence of consistent findings among studies of decreased antibody concentrations following vaccination and PFOA. There is epidemiologic evidence of temporality. However, there are a limited number of comparisons across the same vaccination types, making consistency/specificity difficult to evaluate."

3M Conclusion on decreased antibody responses to vaccines

The inconsistent findings both within and across studies, along with the absence of clinical immunosuppression, do not support the ATSDR conclusion "*suggestive of a link between serum PFOA, PFOS, PfHxS, and PFDeA levels and decreased antibody responses to vaccines*".

Detailed Comments on Increased Risk of Asthma Diagnosis (PFOA)

ATSDR Position

The ATSDR draft profile concluded there is a "*possible link between serum PFOA levels and an increased risk of asthma diagnosis*". The draft profile cites 8 epidemiologic studies (2 prospective cohort studies, 2 case-control studies and 4 cross-sectional studies) that examined the relationship between PFOA exposure and self-reported asthma. ATSDR provided no interpretation of the epidemiologic evidence or rationale for their conclusion of a "possible link". In fact, the only conclusion ATSDR provided in the document is the following statement: "*In tests of hypersensitivity, there is some evidence of an association between serum PFOA and asthma diagnosis in children and adults, although this finding was not consistent across studies; increased risk of allergy or allergic sensitization does not appear to be associated with serum PFOA (page 276).*"

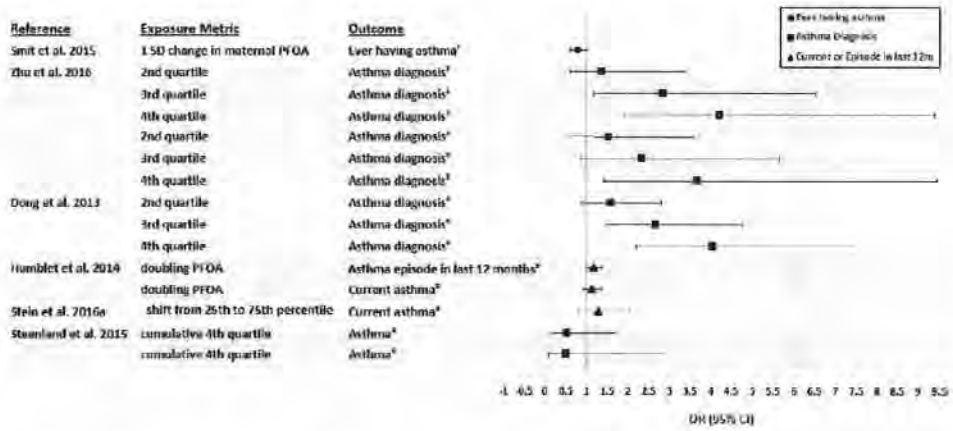
3M Comments

The ATSDR draft profile cited the NTP (2016) conclusion that "*there is low confidence that exposure to PFOA during childhood is associated with increased hypersensitivity responses based on the available studies*" (page 279). The ATSDR draft profile, however, does not include NTP's stated rationale for the conclusion of "low confidence" which was "*primarily due to the cross-sectional nature of the studies and uncertainty as to whether exposure levels reflect exposure prior to the development of hypersensitivity.* (NTP, 2016)". The ATSDR failed to recognize these important limitations or other methodological issues in the draft document. The following comments are provided to offer this insight.

Five of the 8 referenced epidemiologic studies used self-reported asthma (Anderson-Mahoney et al. 2008; Granum et al. 2013; Humblet et al. 2014; Smit et al. 2015; Stein et al. 2016b). The validity of self-reported asthma is largely unknown. However, a review of asthma questionnaires reported a mean sensitivity of 68% and specificity of 94% for self-reported asthma when compared with a clinical diagnosis of asthma (Toren et al. 1993). Consequently, studies using self-reported asthma diagnosis are subject to some degree of measurement error, which may bias the study results.

Asthma diagnosis was medically validated in 3 studies ((Dong et al. 2013); (Steenland et al. 2015); (Zhu et al. 2016)). It is important to note that 2 of these studies (Dong et al. 2013; Zhu et al. 2016) each reported on results from a single case-control study of the same population (456 Taiwanese children enrolled in the Genetic and Biomarkers study of Childhood Asthma (GBCA) study). While, the ATSDR document acknowledged in Table 2-16 that the same group of children (231 asthmatic and 225 non-asthmatic) were evaluated by both authors, the ATSDR did not address this in the text or in Figure 2-20 (below). This gives readers the false impression that these are two distinct studies with consistent findings.

Figure 2-20. Risk of Asthma Diagnosis Relative to PFOA Levels (Presented as Adjusted Odds Ratios)



Dong et al. (2013) reported a significant association and exposure trend between serum PFOA levels and asthma diagnosed in the last 12 months among children aged 10-15 years (OR for highest versus lowest quartile of serum PFOA = 4.05, 95% CI: 2.21, 7.42, P_{trend} = <0.001). However, no significant association between serum PFOA levels and asthma severity score was reported (p=0.119). Zhu et al. (2016), observed significant associations and exposure trends in both males and females in a stratified analysis of the same study population. An important limitation in the study by Dong et al (2013) and Zhu et al (2016), not mentioned in the ATSDR draft profile, is that asthma diagnosis preceded serum PFOA measurements. The third study (Steenland et al. 2015), examined the potential association between occupational exposure to PFOA and validated asthma with reported current medication. However, only study participants who self-reported having asthma were asked to give consent for medical records review to validate cases. Of the 138 self-reported asthma cases, 108 (78%) provided consent for medical records review; 82 cases were validated and included in the statistical analysis. Therefore, asthma diagnosis was validated only among study participants who self-reported having asthma and not for participants whose medical records were not reviewed. In contrast to findings reported by Dong et al (2013) and Zhu et al (2016), Steenland et al.(2015) observed no significant association between PFOA exposure and risk of medicated asthma..

Two additional studies, published since 2016, should be included in the ATSDR draft profile ((Impinen et al. 2018; Timmermann et al. 2017). Study by Timmerman et al. used a cross-sectional design to examine the potential association between pre- and postnatal PFAS exposure and self-reported childhood asthma in a cohort of Faroese children. Among 22 MMR-unvaccinated children, a doubling of serum PFOA levels (measured at age 5) was significantly associated with increased odds of asthma at age 5 (OR = 10.37, 95%CI: 1.06, 101.93) and 13 (OR = 9.92, 95%CI: 1.06, 93.22). No significant associations were observed among MMR-vaccinated children. Additionally, no associations were observed between maternal PFOA exposure and childhood asthma at age 5 and 13 years. Due to the small sample size, precision of the estimates was poor as evident by the wide confidence intervals. Study by Impinen et al. was a well-designed prospective cohort study of 641 children enrolled in the Norwegian Environment and

Childhood Asthma (ECA) birth cohort which examined the association between PFAS measurement from cord blood and medically validated asthma diagnosis in children 2 and 10 years of age. Investigators found no significant associations between prenatal exposure to PFOA and asthma related outcomes. This study was strengthened by its prospective exposure assessment and validated asthma diagnosis.

3M Conclusion on increased risk of asthma diagnosis

Prospective cohort studies have consistently reported no association between PFOA and asthma. Conversely, cross-sectional and case-cohort studies are limited by temporal ambiguity, lack of consistent findings, and unvalidated outcome assessment. Collectively, the existing epidemiologic evidence does not support an association between PFOA exposure and asthma risk.

Detailed Comments on Increased Risk of Decreased Fertility

ATSDR position

On page 5 and 6, ATSDR wrote, "Although a large number of epidemiology studies have examined the potential of perfluoroalkyl compounds to induce adverse health effects, most of the studies are cross-sectional in design and do not establish causation. Based on a number of factors including the consistency of findings across studies, the available epidemiology studies suggest associations between perfluoroalkyl exposure and several health outcomes." According to the ATSDR, this included increased risk of decreased fertility (PFOA, PFOS). This was reiterated on page 24 where ATSDR wrote, "A suggestive link between serum PFOA and PFOS levels and an increased risk of decreased fertility has been found." Table 2-21 (pages 318-320) provided point estimates for selected categorically-defined PFOA or PFOS serum concentrations that are sometimes stratified by the subgroups parous or nulliparous. Page 325-326 is ATSDR's written description of the epidemiology studies that describe effects on fertility as related to PFOA. On page 326 is Figure 2-29. This figure provides adjusted fecundability ratios (95% CI) form PFOA for 13 references. These ratios were stratified by parity status. On page 327 is Figure 2-30. This figure provides infertility (95% CI) relative to PFOA for 16 references. This was stratified by parity status. On page 332, paragraph 3. ATSDR provides its written description of the epidemiology studies that describe effects on fertility as related to PFOS. On page 333 is Figure 2-31. This figure provides adjusted fecundability ratios (95% CI) from PFOS for 13 references. These ratios were stratified by parity status. On page 334 is Figure 2-32. This figure provides infertility (95% CI) relative to PFOS for 16 references. This figure was stratified by parity status. Within the framework of the text on pages 325-326 for PFOA or page 332 for PFOS, there is no discussion on how ATSDR evaluated the weight of the evidence to arrive at its conclusion that there was an association with "increased risk of decreased fertility (PFOA, PFOS)."

3M Comments

ATSDR failed to offer a critical assessment of the epidemiology literature and the study methods used related to fertility and exposure to PFOA and PFOS. ATSDR neglected to discuss the very important methodological issues surrounding the metric time to pregnancy and when measured serum perfluoroalkyl concentrations are taken in nulliparous and parous women. This has been a topic of considerable interest and controversy as extensively discussed in the perfluoroalkyl literature since 2009. In this regard, ATSDR never explained why the studies discussed on pages 325-326 (PFOA), page 332 (PFOS), and their associated figures and tables, are stratified by nulliparous or parous status. This reflects ATSDR's failure to properly assess the reproductive epidemiology literature and its methods regarding PFOA and PFOS, which preclude a conclusion for finding an association between an increased risk of decreased fertility with exposures to PFOA and PFOS.

While Fei et al. (2009) reported an association (the first to do so) between PFOA and a decrease in fecundability and an increase in infertility with women in the Danish National Birth Cohort (page 330), they did not stratify their data by parity. This stratified analysis was published 3 years later (see (Fei et al. 2012). Commentary. Perfluorinated chemicals and time to pregnancy: A link based on reverse causation? Epidemiology 23:264-266). This stratified analysis was prompted by a review of the original Fei et al. 2009 publication by (Olsen et al. 2009) (Note: Olsen et al. 2009 was never cited by ATSDR. For Olsen et al. 2009 see Perfluoroalkyl chemicals and human fetal development: An epidemiologic review with clinical and toxicological perspectives. Reprod Toxicol 27:212-230). Olsen et al. wrote (see page 228 of their paper.) the following describing their suspected methodological question of Fei et al. 2009:

> *"Another troubling issue depicted in Fig. 6 (see obtained copyright figure below) is that parity is both an outcome of fecundity and a cause of PFC concentration: this induces a cyclic change that violates the conditions of causal inference. Although this is an artificial cycle that arises from not explicitly representing the variation of PFC level over time, it highlights the conundrum of trying to make do with a current PFC level, when the actual level may be an earlier and somewhat different level, even with compounds that may have long serum elimination half-lives such as PFOS or PFOA. For example, under the reasonable assumption that PFC levels will be lower after a pregnancy, a longer interval between births would result in more time for a woman to absorb PFCs that could replace the loss incurred from the birth. Women who begin with comparable PFC concentrations and equal parity may have different PFC concentrations at their next birth based on the time that passed between births. All else being equal, those women with longer TTP will have longer intervals of time between births and so may have higher PFC levels prior to the next pregnancy. This would result in longer TTP measurements associated with PFC levels, but the direction of the causality would be backwards: it would be the longer time between births (including the TP) that resulted in higher PFC concentrations. This illustrates the complexity of situation that could be encountered when a causal model (Fig 6) has an unelaborated time-dependent cyclic chain."*

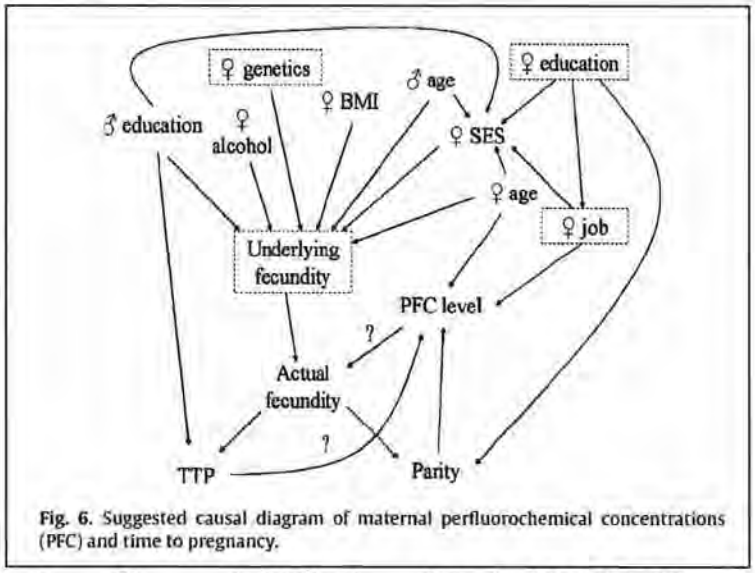


Fig. 6. Suggested causal diagram of maternal perfluorochemical concentrations (PFC) and time to pregnancy.

From Olsen et al. 2009. Reprod Toxicol 27:212-230.

Given this methodological interpretation and question raised by Olsen et al. (2009), Whitworth et al. (2012) examined this issue on fecundability and infertility with their use of the Norwegian Mother and Child Cohort Study (MOBA) database. While Whitworth et al. also found an association with decrease fecundability and exposure to PFOA and PFOS; however, when they stratified their data by parity (nulliparous, parous), the association was only observed among parous women. Whitworth et al. wrote the following in their discussion:

> *"The discrepant results we observed among parous and nulliparous women may be explained by factors related to pregnancy history. As noted earlier, there is a complex relation between a woman's pregnancy history and current levels of environmental toxicants, particularly when exposures to the toxicant vary over time. Due to the pharmacokinetics of PFCs during pregnancy and lactation, an apparent association between PFCs and subfecundity may be produced even when a causal association does not exist. It is possible that following the decrease in maternal PFC levels observed during pregnancy, deliver, and lactation, the levels again increase to baseline. Therefore, as mentioned earlier, a long interval between the birth of the previous child and the start of the next pregnancy attempt will allow for a longer time during which levels can increase-potential resulting in a noncausal association between subfecundity and PFC levels. Results from women with no previous pregnancies may be more informative regarding toxic effects of these compounds. Based on the nulliparous women in our study, we found no evidence of an adverse effect on subfecundity at the PFC levels in our population."*

In 2012, Fei et al. published their stratified analysis by pregnancy history of their 2009 paper because of the question raised by Olsen et al. 2009) and regarding the timing of the

measurement of perfluorinated compounds. Fei et al. (2012) wrote in their Introduction the following:

> *"In 2008, we reported that high maternal levels of perfluorooctatnoate (POFA) and perfluorooctane sulfonate (PFOS) were associated with longer time to pregnancy (TTP) in the Danish National Birth Cohort. Reverse causality is a possible explanation for the association, as has been pointed out by Olsen and colleagues. Even with age adjustment, past pregnancies and deliveries may serve to lower stored levels of PFOA and PFOS. On average, women with longer TTP will have had more time to reaccumulate perfluorinated chemicals (PFCs). "*

Furthermore, Fei et al. (2012) wrote,

> *"A directed acylic graph (DAG) representing the relationships among these factors is shown in the Figure. (provided by Fei et. al 2012). Present and past fecundability share common determinants, and those determinants confound the relationship between PFOA/PFOS and present fecundability. Adjusting for parity should serve to block that pathway and hence control confounding. However, a subtlety not capture by the DAG is that PFOA/PFOS were not measured at the beginning of the attempt at conception (which would have been ideal), but at the end, after a pregnancy had been achieved. Thus, in the available data, the measurement of PFOA/PFOS can potentially be influenced by TTP for parous women through reaccumulation of the chemicals. Such influence produces a cycle in the graph through the arrow from TTP to the measured PFOA/PFOS. However, for nulliparous women, that arrow does not exist in a model that adjusts for age."*

As the ATSDR (page 325) displayed in their subsequent figures, when the women were then categorized by parity, decreased fecundability OR and increased infertility ORs were more often found in the parous women and these risks attenuated more towards the null among nulliparous women. [Note: the association remained after stratification for parity with PFOS in the Fei et al. 2012 study.] Fei et al. surmised their study showed limited evidence for reverse causation as an explanation for their results and welcomed further studies.

ATSDR was correct that there were additional analyses of this particular Danish National Birth Cohort by Bach et al. (2015). There was an updated analysis of the original sample n = 1161 as well as an additional 440 women included. Bach et al. wrote "*the pooled analyses (both samples) were driven by the larger old sample, but we did not corroborate our previous finding of an association between high PFOS and longer TTP in the new sample. The tendency towards an association for PFOA and TTP in parous women may be due to reverse causation.*" In ATDSR's discussion (see page 325), ATSDR failed to recognize this issue of 'reverse causation' among parous women with TTP and PFOA.

Additional studies were forthcoming including, as ATSDR notes (page 328), studies by, (Jorgensen et al. 2014) and (Vestergaard et al. 2012)that reported no associations.

ATSDR did not include the preplanner study by Buck Louis et al (2013) which showed no association with fecundability for PFOA (adjusted odds ratio 0.94, 95% CI 0.81 – 1.10) or PFOS (adjusted odds ratio 0.99 (95 CI 0.85 – 1.17). Buck Louis et al. did show an association with PFOSA (the primary amide of PFOS) but this finding was difficult to interpret because 90% of the measurements for PFOSA were below the limit of detection. Another study by Whitworth (2016) only reported a weak decreased fecundability odds ratio with PFOSA (interquartile distance was 0.91 (95% CI 0.71 – 1.17) among primiparous women. Neither of these studies (Buck Louis 2013 or Whitmore 2016) were cited in the draft ATSDR 2018 document.

Finally, Vélez et al. (2015) concluded there was reduced fecundity with PFOA (not PFOS) in the MIREC study. Unlike many other studies discussed above, however, Vélez et al. chose not to adjust or stratify their analyses for parity when studying the potential adverse reproductive effects (decreased fecundability, infertility) as they reasoned that conditioning on parity would introduce over adjustment through collider stratification bias. Vélez et al. maintained this argument in a letter to the editor (not cited by ATSDR) when they criticized Bach et al. (2015) by having restricted their analyses of serum perfluoroalkyl acids and TTP to 1,372 women from the Aarhus Birth Cohort. In this study, Bach et al. reported there was no evidence of an association between TTP and serum levels of PFOA (odds ratio 1.10; 95% CI 0.93-1.30) and PFOS (odds ratio 1.09; 95% CI -0.95-1.29). Bach et al. (2016) (not cited by ATSDR) argued that if parity is not conditioned on, reverse causality may still be a spurious association between PFAS levels and TTP in parous women due to reaccumulation issues addressed above. Subsequently, Bach et al. (2016b) (not cited by ATSDR) conducted a systematic review of PFAS and measures of human fertility, including fecundability and infertility. They reported 8 studies that examined the association between PFAS and TTP. Only one study found an association when restricted to nulliparous women; 4 studies reported an association with parous women. Bach et al. concluded the latter was likely not causal but a result of reverse causation and unmeasured confounding related to prior pregnancies and childbirths that could influence the measurement of PFAS.

Given the above discussion in the literature and the omission by ATSDR of discussion of these above methodological issues, ATSDR does not appear to have documented or conducted an appropriate weight-of-the-evidence assessment. These methodological issues, analyses and insights have been extensively discussed since 2009. ATSDR should reconsider its assessment as there is an insufficient basis to conclude that there is an "increased risk of decreased fertility (PFOA, PFOS)" based on a thorough examination of this published epidemiology literature.

3M Conclusion on increased risk of decreased fertility

There is no association of an increase in decreased fertility, when analyzed as the metric time to pregnancy, in nulliparous women for PFOA or PFOS exposure. A longer time period between the birth of the previous child and the start of the next pregnancy attempt will allow for a greater potential for reaccumulation of PFOA or PFOS. This could

potentially result in noncausal associations observed in parous women when assessing subfecundity by the metric of time to pregnancy with PFOA or PFOS.

Detailed Comments on Lower Birth Weight

ATSDR position

On page 5 and 6, ATSDR wrote, "Although a large number of epidemiology studies have examined the potential of perfluoroalkyl compounds to induce adverse health effects, most of the studies are cross-sectional in design and do not establish causation. Based on a number of factors including the consistency of findings across studies, the available epidemiology studies suggest associations between perfluoroalkyl exposure and several health outcomes." According to the ATSDR, this includes "small (<20 g or 0.7 ounces per 1 ng/mL increase in blood perfluoroalkyl level) decreases in birth weight (PFOA, PFOS)." Similar statement was provided on page 25. Table 2-23 provides a summary of epidemiologic studies that evaluated birth outcomes in humans. On page 377, ATSDR states, "mixed results have been found for birth outcomes, particularly birth weight. Some epidemiology studies have found associations between maternal PFOA or PFOS exposure and decreases in birth weight, and meta-analyses of these data have found that increases in maternal PFOA or PFOS were associated with 15-19 g or 5 g decreases in birth weight, respectively; accounting for maternal glomerular filtration rate attenuated these results by about 50%." On page 381, ATSDR briefly discussed the meta-analyses of Johnson et al. (2014) for PFOA and Verner et al. (2015) for PFOA and PFOS. In the Johnson et al. meta-analysis, they reported an estimate of -18.9 g (95% CC -29.8, -7.9) change in birth weight per 1 ng/mL increase in serum or plasma PFOA. Using not quite the same number of studies, Verner et al. provided an estimate of a -14.72 g change in birth weight (95% CI -21.66, - 7.78) per ng/mL PFOA. Through PBPK model simulations, they estimated that taking into account the maternal glomerular filtration rate would reduce this estimate to -7.92 g change (95% CI -9.42, -6.43) per ng/mL PFOA measured at delivery and -7.13 g change (95% CI -8.46, -5.80) per ng/mL PFOA measured in cord blood. For PFOS, Johnson did not provide a meta-analysis estimate but Verner et al. did at -5.00 g change (95% CI -8.92, -1.09) per ng/mL PFOS that would attenuate to -1.46 g change (-181, -1.11) per ng/mL PFOS measured at delivery and -2.72 g change (95% CI -3.40, -2.04) per ng/mL PFOS measured in cord blood.

3M Comments

ATSDR briefly discussed two meta-analyses conducted by Johnson et al. (2014) and Verner et al. (2015). ATSDR provided no historical context to these two studies. Unfortunately, several important issues were not discussed by ATSDR that are critical to deciding whether sufficient information exists to even describe whether an association exists. In addition, two additional meta-analyses were not considered by ATSDR ((Negri et al. 2017; Steenland et al. 2018). The latter was recently released in abstract form in the journal *Epidemiology* and is critical to understanding whether an association between lower birth weight and PFOA is likely to even exist, let alone be biologically relevant (see ATSDR Toxicological Profile, page 573.

First, as a minor point, ATSDR stated there were 7 papers included in the meta-analysis by Johnson et al. (2014) whereas there were 9 papers. Not cited by ATSDR were the

Washino et al. (2009) and Whitworth et al. (2012) publications considered by Johnson et al. Thus, the only difference between Johnson et al. (2014) and Verner et al. (2015) meta analyses were the inclusion of the Fromme et al. (2010) and Kim et al. (2011) papers by Johnson but not by Verner et al. (2015). Fromme et al. (2010) and Kim et al. (2011) were small studies whose point estimates for reported birth weights were large but highly imprecise (see Figure 5 in Johnson et al.). Verner et al. did not consider these two papers and subsequently Verner reported a lower meta-analysis point estimate of 14.7 gm (95% CI -21.66, -7.78) birth per ng/mL PFOA in their meta-analysis than did Johnson et al. who reported -18.91 (95% CI -29.8 to -7.9) birth per ng/mL PFOA.

A more critically important difference between the Johnson et al. and Verner et al. papers was the fact that Johnson et al. (see also (Lam et al. 2014)) stated they found "limited and inconsistent data that were inadequate to draw conclusions on the association between fetal growth and glomerular filtration rate (GFR)." ATSDR should also include the Lam et al. (2014) paper for the background that led to this conclusion as well as their systematic review of fetal growth and maternal GFR by Vesterinen et al. (2015) (which included most of the authors of Johnson et al (2014) and Lam et al. (2014). The hypothesis (discussed by both Johnson et al. and Verner et al.) was that the increase in plasma volume expansion that occurs in early to first trimester will result in an increase in the maternal glomerular filtration rate, but less so in mothers of lower weight births (compared to mothers of higher weight births during their pregnancy). As a result, the former would have higher PFAS concentrations retained due to less PFAS eliminated via the kidney because of the comparably lower maternal GFR.

Thus, GFR would be an important confounder that could influence the association between birth weight and measured PFOA or PFOS in maternal or cord blood. In their systematic review of fetal growth and maternal GFR, Vesterinen et al. did not include the largest published study (Morken et al. 2014) to examine this relationship because it was published after their review. Morken et al. examined a subcohort of 953 selected women (470 women with and 483 women without preeclampsia in the Norwegian Mother Child Cohort study) and reported an association between maternal GFR during pregnancy and infant birth weight thus showing GFR could, indeed, confound selected epidemiologic associations. [Note: this one study by Morken et al. equaled the entire size of the database that Vesterinen et al. reviewed in their meta-analysis of 16 very small studies that were published in the scientific literature on fetal growth and maternal GFR. As with very small studies, they lacked statistical power.]

Because the association between fetal growth and maternal GFR was shown in Morken et al., Verner et al. then utilized an established PBPK model to examine the influence that GFR may have on simulated maternal serum concentrations based on the epidemiologic data. They subsequently reported that the association between simulated maternal and cord plasma PFOA levels and birth weight was dependent on the time elapsed after conception. This critical issue was not mentioned by the ATSDR. The association was not seen with PFOA measured in the first trimester and strongest at term where they reported an -7.92 g (95% CI -9.42, -6.43) reduction in birthweight per ng/mL PFOA measured at delivery. As stated above, simulation of measured cord blood PFAS resulted

in a -7.13 g birth weight per ng/ml PFOA. Verner et al. concluded a "substantial proportion of the association between prenatal PFAS and birth weight may be attributable to confounding by GFR which would be more important to examine in those studies with sample collection later in pregnancy".

Based on the analyses by Verner et al. showing maternal GFR may substantially confound any association between PFOA or PFOS and fetal growth (measured as birth weight), the available data do not permit ATSDR to conclude that there is an association between PFOA or PFOS and lower birth weight in this regard, especially without listing the caveats (confounding) known to date, let alone the unknown multitude of other physiologic changes occurring during the course of a pregnancy that have yet to be accounted for in any epidemiologic analyses.

The next most recent meta-analysis performed was published in 2017 by Negri et al. They included 16 studies in their meta-analysis. The additional studies not considered by Johnson et al. (2014) included the publications by Wu et al. (2012), Darrow et al. (2013), Bach et al. (2016a)), Lenters et al. (2016), Robledo et al. (2015)), and Lee et al. (2016).

The Negri et al meta-analyses used both the untransformed and natural log transformations of PFOA and PFOS. For PFOA, they reported a -12.8 g untransformed birthweight (95% CI -23.21, -2.38) and -27.12 (95 % CI -50.64, -3.6) g (natural log transformed) change per ng/mL PFOA. For PFOS, they reported a -0.92 g untransformed birthweight (95% CI -3.43, 1.60) and -46.09 g (natural log transformed) (95% CI -80.33, -11.85) per ng/mL PFOS. Based on their sensitivity analyses, there were stronger associations from studies conducted in Asia and significant heterogeneity was observed when the measurement of PFOA/PFOS was done later in the pregnancy or using cord blood. The latter is consistent with the simulation PBPK modeling done by Verner et al. (2015) as it relates to the potential confounding influence of maternal GFR with the timing of when PFOA is measured during pregnancy. Negri et al. also examined the laboratory animal data (results not reported here) and concluded the animal data showed similar dose-response trends but the effective serum concentrations in rodents were 100 to 1000 times higher than in humans based on the epidemiological evidence. This led Negri et al. to increase their degree of uncertainty as to the biological plausibility of a causal relationship between PFOA or PFOS exposure and lower birthweight in humans. This doubt led these authors to suggest there might be some, not yet identified, confounding factors that lead to this spurious association of lower birth weight and perfluoroalkyl measurements in humans. For reasons not explained, Negri et al. chose not to reference the Verner et al. (2015) PBPK simulation study who aptly demonstrated the potential confounding of maternal GFR, the timing of measurement of PFOA/PFOS during and through pregnancy, and reported birth weight.

Published in abstract form in August 2018 is a fourth meta-analysis authored by Steenland et al. (Epidemiology 2018). It is anticipated the full study will be available on-line in 60 to 90 days. These investigators conducted a meta-analysis of 24 studies, which

examined the association between lower birth weight and PFOA. (PFOS was not part of this meta-analysis.) The additional nine new studies (not identified in the abstract) added 6019 births to the 6937 births examined by Negri et al. in their meta-analysis. They included another large study (not identified in abstract) that was excluded from previous analyses, in a sensitivity analysis. Overall, they found a change of birthweight of -10.5 grams (95% CI -16.7, -4.4) per ng/ml PFOA in maternal or cord blood. After adding the one previously excluded large study, Steenland et al. found "little" evidence of an association (-1.0 grams, 95% CI -2.4, 0.4) per ng/mL PFOA. Restricting to the studies where blood was sampled from mothers early in the pregnancy or shortly before conception (5393 births), they reported "little" association of PFOA with birthweight (-3.3 grams (95% CI -9.6, 3.0)). In studies where blood was sampled late in the pregnancy (7563 pregnancies), lower birthweight was associated with PFOA (-17.8 g (95% CI -25.0, -10.6)/ ng/mL PFOA. Steenland et al. concluded the present human evidence provides only modest support for decreased birthweight with increasing PFOA. Critically important to understand is the time interval when perfluoroalkyls were measured.

Steenland et al. concluded "*studies with a wide range of exposure and studies with blood sampled early in pregnancy showed little or no association of PFOA with birthweight. These are the studies in which confounding and reverse causality would be of less concern.*" This conclusion is consistent with the findings from Verner et al. [Note: ATSDR also concluded in its draft Toxicological Profile on page 517 (without citing Negri et al. or Steenland et al. meta-analyses) that "the decreases in birth weight were small and not likely biologically relevant."]

3M Conclusion on lower birth weight

There is no association between low birth weight (<2500 g) in humans and exposure to PFOA or PFOS. Taking into account 1) confounding by the increased maternal glomerular filtration rate that increases during early pregnancy, 2) the time period when PFOA/PFOS are measured before, during or after pregnancy, and 3) the possibility of reverse causation, there is insufficient epidemiologic evidence to conclude an association exists between lower birth weight (i.e., several grams) and PFOA or PFOS concentration (per ng/ml).

Additional Comments

General note:

There is no authorship by chapters or sections within chapters.

Page v.

- The role of SRC, Inc. as it relates to this Toxicological Profile needs to be described on this page under Chemical Manager Team.

- Dr. Emmett has served as a peer reviewer selected by ATSDR on the 2009, 2015, and now 2018 draft Toxicological Profiles for Perfluoroalkyls. Dr. David Savitz's role as a peer reviewer on the draft 2009 Toxicological Profile should be acknowledged as well as ATSDR's request that Dr. Savitz provide publicly available comments on the draft 2015 ATSDR Toxicological Profile. Dr. Cory-Slechta has served as: 1) the chairperson of the 2005 U.S. Environmental Protection Agency Science Advisory Board Perfluorooctanoic Acid (PFOA) Risk Assessment Review Panel; 2) a peer reviewer (and the chairperson) on the U.S. EPA draft 2014 health effects document for PFOA; 3) a peer reviewer (and the chairperson) on the U.S. EPA draft 2014 health effects document for PFOS; 4) a peer-reviewer of the draft 2015 ATSDR Toxicological Profiles on Perfluoroalkyls; and 5) a peer-reviewer of the draft 2018 ATSDR Toxicological Profiles on Perfluoroalkyls. Dr. DeWitt was one of 20 members of the 2014 IARC Workshop that reviewed PFOA; a peer reviewer on the U.S. EPA draft 2014 health effects document for PFOA; and a peer reviewer on the U.S. EPA draft 2014 health effects document for PFOS.

  To have repeatedly selected these reviewers minimizes the peer-review process of receiving comments that could have been made available to ATSDR.

- Dr. Jamie DeWitt was paid by plaintiff attorneys in the case of State of Minnesota vs. 3M. This financial conflict of interest with another governmental agency should be noted in this draft 2018 ATSDR Toxicological Profile. Dr. DeWitt should not have been chosen as a peer reviewer to a federal government agency given this paid financial conflict of interest regarding another governmental agency. Any other financial conflicts of interest by Dr. DeWitt should also be listed as to her funded role in any litigation effort, to the present date, regarding perfluoroalkyls.

Page 1:

- ATSDR used the term "perfluoroalkyls" for the 14 compounds that it has evaluated. While it is acceptable to use this general nomenclature in some parts of the discussion, it is not applicable for topics such as major applications listed under section 1.1.

- For clarity most of the 14 perfluoroalkyl substances that are the focus of this report have limited commercial utility. PFOS, PFOA and PFOA pre-cursors have been used extensively.
- On a technical definition, ATSDR should make note to differentiate that the following two compounds (among the 14 evaluated) are polyfluoroalkyls, not perfluoroalkyls.
    - 2-(N-Methyl-perfluorooctane sulfonamide) acetic acid (Me-PFOSA-AcOH)
    - 2-(N-Ethyl-perfluorooctane sulfonamide) acetic acid (Et-PFOSA-AcOH)
- The ATSDR draft profile cites a 2003-2004 NHANES study (Calafat et al, 2007). More recent NHANES biomonitoring data was published in the CDC's "Fourth National Report on Human Exposure to Environmental Chemicals" in 2018.

Page 2:

- The ATSDR draft profile recognized that serum levels of PFOA and PFOS in the U.S. general population have "decreased dramatically in recent years". For further clarification, from 1999-2000 to 2013-2014 mean blood levels of PFOS and PFOA have decreased by approximately 84% and 63%, respectively, based on NHANES data. A more recent study, using data from the American Red Cross, reported an 88% and 77% decline in serum PFOS and PFOA levels, respectively, from 2000-2001 to 2015 (Olsen et al., 2017). These reductions are largely attributed to the concerted efforts by industry and the U.S. EPA to decrease the use of these chemicals in manufacturing and releases to the environment.
- ATSDR should revise the last paragraph on this page. Contaminated drinking water near fluoropolymer manufacturing facility in southeastern Ohio and West Virginia did not have high levels of exposure to PFOS.
- Page 2, Paragraph 1. The statement that PFOS and PFOA are no longer imported is not entirely accurate. PFOS, FC-98 and a few other PFOS-precursor substances are not TSCA prohibited, and may be imported.
- ATSDR stated: "Volatile *fluorotelomer* alcohols may be *broken down* into substances like PFOA, and atmospheric deposition can lead to contamination of soils and leaching into groundwater away from point sources." There is no description of what fluorotelomers are. "Broken down" is inappropriate scientific terminology.
- There is no definition of the word "high". "High" is relative to some other value and is subjective language The ATSDR should substitute this word "high" throughout this document for the specific concentrations referred to when "high" or "low" are used and be specific whether these values are arithmetic means, geometric means, or medians, as well as offer a measure of variation to the point estimates (e.g., standard deviation, standard error, 95% confidence interval, or a range minimum/maximum). Also, it is important to refer to the year in which these perfluoroalkyl values were actually measured (not just the author and reference year) because of the declining trends over the past 15+ years in most general populations not exposed to an environmental point source of exposure.

Page 3:

- ATSDR should provide the actual median value and corresponding year-dependent NHANES median value. ATSDR should provide the percentage decline as well in these geometric mean values for PFOS (decline of 83.6%) and PFOA (decline of 62.7%) between 1999-2000 and 2013-2014.
- In the last paragraph, ATSDR reported breast milk concentrations, but does not indicate when such concentrations were measured. This is important because breast milk concentrations have declined similar to serum concentrations in adults. See above comment on incomplete paragraph 1 on page 3. Concentrations have also declined in children. See Olsen et al. (2005) who reported on children (2 – 12) serum measurements made in 1994-1995 to those measurements recently reported by Ye et al (2018) who reported, in a nationally representative sample of children age 3-11, that their concentrations were comparable to adults measured also in 2013-2014. The measured concentrations in these children were substantially lower in other non-representative samples of 597 children reported by Olsen et al. (measured in 1994-1995). Therefore, breast milk concentrations have also likely declined over time.
- There are additional studies on human breast milk biomonitoring studies, ATSDR should reference and summarize studies by: Sundstrom et al. 2011 Environ Int 37 178-183; Karrman et al 2009 Environ Int 35 712-17; Llorca et al 2010 Environ Int 36 584-592; Mosch et al. 2010 J Chromatog B 878 2652-2658; Kang et al. 2016 Environ Res 148 351-359; Cariou et al. 2015 Environ Int 84 71-81; Al-sheyab et al. 2015 Environ Sci Pollut Res 22 12415-12423; Lankova et al. 2013 Talanta 117 318-25; Pratt et al. 2013 Food Addit Contam A 30 1788-1798; Guerranti et al. 2013 Food Chem 140 197-203; Antignac et al. 2013 Chermosphere 91 802-808; Barbarossa et al. 2013 Environ Int 51 27-30; Croes et al. 2012 Chemosphere 89 988-994; Domingo et al. 2012 Food Chem 135 1575-1582; Thomsen et al. 2010 Environ Sci Technol 44 9550-9556.

Page 4:

- ATSDR used the term "perfluoroalkyls" to describe the 14 compounds that are listed on page 1 (including Perfluorooctane sulfonamide (PFOSA), 2-(N-Methyl-perfluorooctane sulfonamide) acetic acid (Me-PFOSA-AcOH), and 2-(N-Ethyl-perfluorooctane sulfonamide) acetic acid (Et-PFOSA-AcOH)). Accordingly, ATSDR cannot make the blanket statement that perfluoroalkyls "are not metabolized in humans or laboratory animals" because these 3 compounds can and do metabolize in laboratory animals.
- Table 1-1. The estimated elimination half-life of PFOA in humans is clearly not 8 years. This estimate is not found in the Olsen et al. 2007a paper. More importantly, similar to the data reported in rats and mice, there are available ranges of the estimated elimination half-lives of PFOA, PFOS, and PFHxS. There are several high-quality and more recent studies of populations whose exposure was mitigated by installation of GAC filters that have shown the serum elimination half-life of PFOA to be between 2.3 years (95% CI) (Bartell 2013) and 2.8 years (95% CI) (Li et al. 2018). Similarly, the serum elimination half-life for PFOS of 5.4 years is the highest estimate of 6 studies.

Page 5:

- It is incorrect for ATSDR to state that "In general, epidemiology studies use serum perfluoroalkyl levels as a biomarker of exposure, which contrasts experimental studies that utilize dose, expressed in mg/kg/body weight/day units". As difference in toxicokinetics have been well-recognized, it is the serum levels in the animals (resulted from doses given) that should be used for data interpretation; and many toxicological studies have been measuring and reporting serum levels in the laboratory animals as internal dose metrics (ng/mL) as well as benchmark lower bound internal serum concentrations.
- ATSDR relied on animal PBPK model to predict subsequent POD of MRL derivation, but on the other hand, it has also explicitly stated that "Although physiologically based pharmacokinetic (PBPK) models have been developed for rodents and humans, these models are not sufficient to allow for comparisons between administered doses in laboratory animals and serum concentrations in humans". This statement indicated a great amount of uncertainty associated with the PBPK model used hence ATSDR needs to reflect and acknowledge this fact in its summary.
- It is inappropriate to solely consider the Emmett et al. (2006a) mean PFOA estimate of 423 ng/mL as the mean estimate of PFOA level in highly exposed residents for the community surrounding the DuPont Washington Works facility in west Virginia because other data are available. Furthermore, Sakr et al. 2007a did not provide the most appropriate estimate for the average PFOA concentration for the workers (Woskie et al. 2012 Ann Occup Hyg 56 1025-1037).
- Throughout this draft toxicological profile, ATSDR stated that most epidemiology studies were of the cross-sectional design. However, nowhere does ATSDR provide the actual quantitative number of epidemiological studies by the type of study design. Furthermore, in most tables reported in Chapter 2, ATSDR never provides the type of study design of the author. It assumes the reader will look at more detail in the abridged abstracts of these studies presented in the Supporting Document. This is highly unfortunate and a major shortcoming of the ATSDR report. All studies listed in tables should be listed as to their study design.
- It is highly misleading for ATSDR to state on page 5, paragraph 2, prior to identifying associations between PFAS exposure and eight health outcomes, that "Based on a number of factors including the consistency of findings across studies, the available epidemiology studies suggest associations between perfluoroalkyl exposure and several health outcomes" because on page 635/636 (chapter on the adequacy of the database), it makes the following contradictory statement: "The available human studies have identified some potential targets of toxicity; however, cause-and-effect relationships have not been established for any of the effects, and the effects have not been consistently found in all studies." Indeed, there is not consistency of findings in the epidemiology data across these 8 associations. Moreover, ATSDR does a disservice to the scientific literature to suggest that there is consistency. Therefore, it is imperative that the statement found on page 635/636 be placed either in front of or immediately after the

listing of the 8 associations provided on page 5/6 in Chapter 1. Otherwise, these "associations" may be misperceived to reflect causality by scientists as well as the public reading this Toxicological Profile.

Pages 6 – 9:

Figures 1-1, 1-2, and 1-3 are misleading. The studies compiled in each figure have different study designs with different animal models used and different dosing regimens; they simply do not reflect final body burden achieved. These figures should either be removed or revised by taking toxicokinetic into consideration.

Page 10:

Under liver effects: ATSDR should also cite other key studies such as Elcombe et al 2010 Arch Toxicol 84 787-798; Albrecht et al. 2013 Toxicol Sci 131 568-582; and Butenhoff et al. 2012 Reprod Toxicol 33 513-530.

Page 11:

- ATSDR should also include other nuclear receptors in its discussion, such as CAR/PXR. It should include studies by Elcombe et al 2010 Arch Toxicol 84 787-798; Vanden Heuvel et al. 2006 Toxicol Sci 92 476-489; Albrecht et al. 2013 Toxicol Sci 131 568-582; Bjork & Wallace 2009 Toxicol Sci 111 89-99; and Bjork et al. 2011 Toxicology 288 8-17.
- ATSDR is incorrect stating that increased hepatic palmitoyl CoA oxidase activity was increased in PFOS-treated monkeys in Seacat et al. (2002) study (see Table 6 of Seacat et al. manuscript).
- ATSDR should also cite another relevant study for the serum lipid change in monkeys (Chang et al. 2017 Toxicol Sci 156 387-401), which followed a cohort of monkeys for 400+ days and their serum lipid profiles were characterized before and after PFOS treatments. The lower benchmark concentration was around 75 µg/mL (75000 ng/mL) in the serum where a decrease in serum cholesterol occurred in these monkeys.

Page 12:

- ATSDR should provide compelling scientific data to explain why they concluded the following:

> "*Specific effects reported include prenatal loss, reduced neonate weight and viability, neurodevelopment toxicity, and delays in mammary gland differentiation, eye opening, vaginal opening, and first estrus (Abbott et al. 2007; Albrecht et al. 2013; Cheng et al. 2013; Johansson et al. 2008; Koskela et al.*

2016; Lau et al. 2006; Macon et al. 2011; Ngo et al. 2014; Onishchenko et al. 2011; Sobolewski et al. 2014; White et al. 2007, 2009, 2011; Wolf et al. 2007; Yahia et al. 2010). These effects occurred generally in the absence of overt maternal toxicity."

In the studies cited by ATSDR above, there were compelling supporting data to illustrate developmental toxicity with PFOA exposure under maternal influences. In addition, there was no standardized method evaluating mammary gland during pup developments and the delayed mammary gland conclusions reported by White et al. (2007, 2009, 2011) and Macon et al. (2011) contradicted with the conclusions reported by others (Albrecht et al. 2014, Yang et al. 2009 Reproduct Toxicol 27 299-306; Hardisty et al 2010 Drug Chem Toxicol 33 131-137) where strain-specific responses cannot be ruled out.

- Study outcomes reported by Onishchenko et al. (2011) had many technical issues and its data lacked scientific rigors necessary for it to be used in any meaningful human risk assessment.

- Brain and nervous system have not been identified as target organs in long-term toxicological studies, including 2-year bioassays in rats (Butenhoff et al. 2012 Toxicology 298 1-13; Biegel et al 2001 ToxSci 60 44-55), 13-week study in rats (Perkins et al. 2004 Drug Chem Toxicol 27 361-378), 2-generation in rats (Butenhoff et al 2004 Toxicology 196 95-116), or 6-month study in monkeys (Butenhoff et al 2002 ToxSci 69 244-257).

Pages 13 and 14:

- Similar to comments provided on PFOA, there were compelling supporting data to illustrate developmental toxicity with PFOS exposure was mediated by maternal toxicity. In addition, the neurodevelopmental alterations in mice cited by ATSDR were confounded by poor study design (Onishchenko et al. 2011, where only a single PFOS dose was used) or unexplained non-PFOS-related stress such as restraining during pregnancy (Fuentes et al. 2007a). Evaluation of immune parameters based on the results reported by Keil et al. (2008) was not comprehensive in that normal response to immunization is based on IgG titer, not IgM; and that Keil et al. did not evaluate the subpopulation in other key immune organs such as bone marrow and blood.

- Study by Dong et al. (2009) also had numerous deficiencies which precluded its data to be used in a proper human risk assessment. The data presented by Dong et al. lacked scientific validity to support the conclusion that PFOS suppresses immune responses. There should be concordance between several key immune parameters (as discussed below) and the study by Dong et al. failed to demonstrate such many important aspects of immunotoxicity study. Briefly, antibody response is IgG isotype, not IgM, and as an immunosuppressing agent, one would expect similar suppressive immune responses to be

observed in major key organs such as decreased IgM and IgG in spleen, thymus, and serum. Dong et al. evaluated IgM in spleen only but did not provide any concurrent IgM status in other key organs such as thymus or serum. As an immunosuppressing agent, one would expect decreased immune cell populations in spleen, thymus, blood, and bone marrow and Dong et al. only looked at spleen and thymus. As an immunosuppressing agent, one would expect decreased proliferation in immune cells and Dong et al. did not use the correct methods to evaluate these responses and improperly reported their data. Collectively, the study by Dong et al. did not provide any robust or compelling scientific evidence to support the claim that PFOS is associated with immune suppression in mice.

Page 21:

As stated previously, the ATSDR draft profile cited a 2003-2004 NHANES study (Calafat et al, 2007). More recent NHANES biomonitoring data was published in the CDC's "Fourth National Report on Human Exposure to Environmental Chemicals" in 2018.

Page 22:

ATSDR stated that "*For studies in which the population was divided into perfluoroalkyl exposure categories, such as quartiles, the risk ratio reported in the summary table is for the lowest exposure category with a statistically significant association; risk ratios for higher exposure categories are presented in the Supporting Document for Epidemiological Studies for Perfluoroalkyls*". This approach is problematic for several reasons. First, readers will likely refer only to the ATSDR draft profile and not the Supporting Document. As such, readers will not be informed of all findings including those exposure categories with non-significant findings and evidence (or lack thereof) of a dose-response. Second, results from continuous exposure metrics and other statistical measures are not reported in Summary tables or in the Supporting Document. It is inappropriate for ATSDR to include only categorical results and not present all the available evidence (both significant and non-significant findings).

Page 23:

ATSDR stated that "*The discussion of the available data for each health effect is divided into several subsections. Each health effect section begins with an overview, which contains a brief discussion of the available data and conclusions that can be drawn from the data*". However, the section overview, for most health effects, failed to provide any conclusions that can be drawn from the data or any discussion beyond presenting overall study findings. Of the 18 health effects reviewed in draft profile, ATSDR did not provide their overall conclusion for 10 health effects, including death (page 106), body weight (page 109), respiratory (page 121), cardiovascular (page 123), gastrointestinal (page 135),

hematological (page 137), dermal (page 219), ocular (page 220), neurological (page 293) and cancer (page 418).

Page 24:

ATSDR reported that a "weight-of-evidence" approach was used to evaluate whether the available data support a link between perfluoroalkyl exposure and a particular health outcome. Further, ATSDR stated that "*this weight-of-evidence approach takes into consideration the consistency of the findings across studies, the quality of the studies, dose-response and plausibility*". However, ATSDR failed to 1) cite the "weight-of-evidence" approach that was used, and 2) provide scientific justification or documentation of the underlying evidence used to reach a conclusion. Given that a "weight-of-evidence approach" requires use of scientific judgment, the ATSDR must be transparent in all steps of the evaluation process and all conclusions drawn. For example, on the 8 associations listed on page 25, the ATSDR has failed to explain to the reader how it reached such a collective conclusion for each one given the quality (often cross sectional) of the studies reviewed, the lack of dose-responses, and lack of any known biological plausibility in the human, especially when such plausibility was either not shown or known to result in contradictory findings in the human.

Page 25:

- The term "links" does not have a precise scientific meaning. This word is not standard scientific language taught in epidemiology courses in Schools of Public Health. Therefore, the ATSDR should delete throughout this document the word "link or links" and replace with the word "association or associations."
- See comments for Page 5, Paragraph 2. It is not possible to discuss associations without explicitly stating the admission by ATSDR, found on page 635/636 of the chapter on the adequacy of the database, the following statement (see section on Epidemiology and Human Dosimetry Studies): "The available human studies have identified some potential targets of toxicity; however, cause-and-effect relationships have not been established for any of the effects, and the effects have not been consistently found in all studies." This statement should immediately precede or follow the associations whenever the associations are listed; otherwise these "associations" may be erroneously assumed to reflect causality by non-epidemiologists as well as the public-at-large or others that may read this Toxicological Profile or parts therein.

Page 108:

OECD (2002) document cited on this page is public information and can be found on the following web link:

https://www.oecd.org/env/ehs/risk-assessment/2382880.pdf

Pages 109 – 433:

For each of the endpoints listed here, ATSDR reported the study findings for each compound under each effect but did not provide its overall assessment. The data presentation (spanning 300+ pages) was on the who/how/what of the selected epidemiological and toxicological studies. It lacked overall conclusion and there was no "synthesis" on the selected data presented by ATSDR in this section. A conclusion or position statement by ATSDR at the end of each endpoint will be helpful to the readers.

Page 131:

ATSDR incorrectly stated that *"Another"* study (Darrow et al, 2013) found significant increases in odds ratios for pregnancy-induced hypertension. This study is the same study that is cited in the previous sentence.

Pages 244-300 (Section 2.14):

Two additional studies (Timmermann et al. 2017; Impinen et al. 2018) have been published since 2016 and should be included in the ATSDR draft profile.

Pages 245-250, Table 2-16:

- ATSDR did not cite the study by Anderson-Mahoney et al (2008). It is, however, cited in the Supporting Document (page 105, Table 10).
- ATSDR did not cite a study (Leonard et al., 2008) of PFOA/PFOS exposure and mortality from infectious and parasitic diseases. While this study was cited in Section 2.2, it should also be included in Section 2.14 (as other studies have been cited in more than one section).

Pages 268 - 281:

ATSDR cited several National Toxicology Program (NTP 2016) conclusions on immunosuppression outcomes without providing the NTP rationale for reaching such conclusions. For example, on page 269, in a separate paragraph, ATSDR states "*NTP (2016b) concluded that there is moderate confidence that exposure to PFOA is associated with suppression of the antibody response based on the available human studies. NTP (2016b) also concluded that there is low confidence that exposure to PFOA is associated with increased incidence of infectious disease (or lower ability to resist or respond to infectious disease.*" ATSDR should describe NTPs confidence ratings in more detail (i.e. inadequate, low, moderate, high) and provide the rationale for reaching each conclusion.

Pages 270, Figure 2-19:

The "percent difference in antibody concentration per 2-fold increase in serum PFOA" is presented in Figure 2-19. However, findings from two influenza studies (Looker et al. 2014; Stein et al. 2016b) that used other measures of association, and reported null findings, were not included. Although both studies were cited in the draft profile (page 269), the ATSDR should acknowledge that results from these two studies were omitted from Figure 2-19 and provide reasons for their omission.

Pages 272, Figure 2-20:

Results from asthma studies reporting adjusted odds ratios are presented in Figure 2-20. Similar to the previous comment, results from two studies (Anderson-Mahoney et al 2008; Granum et al 2013) which reported different measures of association were not included in the Figure. The ATSDR should acknowledge that results from these two studies were omitted from Figure 2-20 and provide reasons for their omission.

Pages 272 (Figure 2-20), 280 (Figure 2-22), 285 (Figure 2-24), 288 (Figure 2-26), and 292 (Figure 2-28):

The ATSDR should clearly acknowledge that results from Zhu et al (2016) and Dong et al (2013) were from a single case-control study of the same population (456 Taiwanese children). As currently presented, it gives readers a false impression that these are two distinct studies with consistent findings, which they are not.

Pages 277, Figure 2-21:

The "percent difference in antibody concentration per 2-fold increase in serum PFOS" is presented in Figure 2-21. However, findings from two influenza studies (Looker et al. 2014; Stein et al. 2016b), which used different measures of association, and reported null findings, were not included. The results by Looker et al (2014) were cited in the draft profile (page 277), but not the results from Stein et al (2016b). The ATSDR should acknowledge that results from these two studies were omitted from Figure 2-21 and provide reasons for their omission.

Pages 289-291 and Figure 2-27:

ATSDR offered no explanation for how it concluded that there is an association between PFDeA and decreased antibody responses to vaccines given that only 3 studies have examined this potential association and have reported mixed results. This conclusion is not scientifically supported given the limited and inconsistent evidence.

Pages 433 – 449:

Among all the mechanisms listed here, ATSDR failed to highlight the lipid mechanism. Albeit it was discussed under hepatic toxicity mechanism, it should be emphasized because lipid-lowering is a hallmark biological event with exposures to many of the perfluoroalkyls (at relatively high doses). The lipid-lowering mechanism has been elucidated for PFBS, PFHxS, and PFOS using ApoE3*Leiden.CETP mice (Bijland et al. 2011 Tox Sci 123 290-303). The hypolipidemia has been extensively discussed with PFOA by others (which are cited by ATSDR on page 11).

Pages 434 – 438:

For PPARalpha-dependent mechanism, ATSDR should offer a summary or a position statement on PPARalpha-mediated effects reported in animals and their lack of relevance to humans.

Pages 438 – 441:

Similarly, ATSDR should offer a summary or a position statement on PPARalpha-independent effects reported in animals and their relevance to humans.

Pages 441 - 443:

The liver toxicity mechanism in rodents, in part, has been well-documented and ATSDR should offer a summary or a position statement on the rodent liver effects and their relevance to humans.

Pages 443-444:

Research on immunotoxicity has produced only inconclusive evidence, as acknowledged by EPA in its 2016 Health Effects Document for PFOS, where it stated that:

> *"Both human and animal studies have demonstrated the potential impact of PFOS on the immune system; however, uncertainties exist related to MOA and the level, duration, and/or timing of exposure that are not yet clearly delineated. The animal immunotoxicity studies support the association between PFOS and effects on the response to sheep red blood cells as foreign material and on the natural killer cell populations; however, the doses with effects are inconsistent across studies for comparable endpoints. When both males and females were evaluated, the males responded at a lower dose than the females. Because of these uncertainties, EPA did not quantitatively assess this endpoint."*

Page 445:

Although many toxicological studies had reported endocrine disturbance potential with PFOA and PFOS exposures, specifically on the thyroid hormones, it is important to realize that most of these studies were done either under *in vitro* conditions (to which high concentrations of PFOA or PFOS were employed) or *in vivo* but only with a limited set of endpoints evaluated such as selected gene expressions (D'Orazio et al. 2014; Dankers et al. 2013; Dixon et al. 2012; Du et al. 2012; Du et al. 2013; Gao et al. 2013; Kraugerud et al. 2011; Sales et al. 2013; Sonthithai et al. 2015; Wens et al. 2013; White et al. 2011a; Feng et al. 2015; Lopez-Doval et al. 2015; Lopez-Doval et al. 2014; Pereiro et al. 2014; Wang et al. 2011).

In the study cited by ATSDR, Ren et al. (2015) evaluated perfluoroalkyl bindings using a computer software model to simulate thyroid hormone binding; and their in vivo portion of the study was on tadpoles, not in mammalian species. The endocrine system is very complicated and evaluation of endocrine functions is a very highly specialized field (this is especially true in human clinical medicine). Given that PFOA and PFOS are strong surfactants, the toxicity effects reported from the typical mono-layered *in vitro* tissue culture system offered very little insight and scientific value because the data were often comprised by the surfactant-induced toxicity. Similarly, gene expressions do not represent functionality and endocrine function is an intricate network.

Based on data from the large scale 2-generation reproductive and developmental studies (which are considered as the most comprehensive test by various agencies for evaluating endocrine functions), PFOA and PFOS clearly did not alter the reproductive functions as the reproductive performances in both males and females were normal (*vide supra*). If they were indeed endocrine disrupting compounds, then one would expect it to directly activate endocrine receptors such as estrogen receptors or thyroid receptors.

Ishibashi et al. (2007) reported that PFOA or PFOS did not activate human estrogen receptor α or β. Likewise, Yao et al. (2014) did not report that PFOA can activate mouse or human estrogen receptors. Yao et al. also showed a lack of change in the histomorphology of uterine/cervix and vaginal tissues in female mice after receiving oral ammonium PFOA treatments. Furthermore, while triiodothyronine (T3, the active form of thyroid hormone) elicits a dose-response activation of human thyroid receptor α from 0.000001 – 0.01 uM, under the same study condition, there was no activation of human thyroid receptor α when exposed to ammonium PFOA or PFOS up to 100 uM (Ehresman et al. 2014 The Toxicologist (abstract 1135) 138 302).

Under in vitro condition, Chang et al. had extensively evaluated the effects of PFOS and thyroid hormone status in rodents (Chang et al 2007 Toxicology 234 21-33; Chang et al 2008 Toxicology 243 330-339; Chang et al 2009 Reproduct Toxicol 27 387-399) and

monkeys (Chang et al. 2017 Toxicol Sci 156 387-401) and did not observe any toxicological relevant alterations in functional aspects of thyroid hormone homeostasis. Furthermore, Convertino et al. (2018) reported that, in a phase 1 clinical trial study with 49 human subjects that received large doses of PFOA where serum PFOA level was up to 600,000 ng/mL (5 orders of magnitude higher than general population in the US), there was no alteration in serum TSH level in these human subjects (TSH is the key serum diagnostic parameter for thyroid hormone status used by the physicians).

Overall, the weight-of-evidence does not support that PFOS or PFOA can cause endocrine disruption and ATSDR should recognize and acknowledge this conclusion.

Pages 447 – 449:

The genotoxicity summary by Butenhoff et al. (2014 Toxicology Reports 1 252-270) should be included in the discussion.

Page 450:

Given that the perfluoroalkyls are highly bound to serum albumins, ATSDR should recognize that the distribution patterns in tissues are bloodborne-based.

Page 450:

- As stated earlier, because ATSDR used the term "perfluoralkyls" that included Perfluorooctane sulfonamide (PFOSA), 2-(N-Methyl-perfluorooctane sulfonamide) acetic acid (Me-PFOSA-AcOH), and 2-(N-Ethyl-perfluorooctane sulfonamide) acetic acid (Et-PFOSA-AcOH)), it cannot state that perfluoroalkyls "are not metabolized in humans or laboratory animals" because these 3 compounds listed above can and do metabolize in laboratory animals.

- An inhalation study for 2-(N-Ethyl-perfluorooctane sulfonamide) acetic acid (Et-PFOSA-AcOH) is available in rats and the study data indicated that Et-PFOSA-AcOH can be metabolized to form PFOS via inhalation (see Chang et al. 2017 Environ Res 155 307-313)

Page 514:

ATSDR wrote: 'Assuming a terminal elimination t1/2 of 1,400 days for PFOA in humans (Olsen et al. 2007a), a constant rate of intake for 17 years would be required to achieve 95% of steady state.' This is only applicable with a constant rate of daily (PFOA) intake for 17 years, which is an untenable assumption for any population whether occupational

(inhalation, oral, dermal) or affected communities (primarily oral via drinking water) or general population (primarily oral via diet).

Page 518:

- Given the findings reported by Convertino et al. (2018), the following statement is highly speculative and has no basis of fact, and should be deleted.

  "Increase in serum cholesterol may result in a greater health impact in individuals with high levels of cholesterol or with other existing cardiovascular risk factors."

- Given the fact that ATSDR did not find perfluoroalkyl associated with uric acid, the following statement is highly speculative and has no basis of fact. It should be deleted.

  "Increases in uric levels have been observed in individuals with higher perfluoroalkyl levels. Increased uric acid may be associated with an increased risk in high blood pressure and individuals with hypertension may be at greater risk."

Page 539, Figure 5-2:

Title of Figure 5-2: Timeline of Important Events in the History of Polyfluorinated Compounds

This figure, taken from the copyrighted paper of Lindstrom et al., is factually inaccurate as to what was stated in a 1976 publication of an abstract by Taves et al. (1976). In the figure that ATSDR secured copyright permission to display from a journal, the figure states "1976 - Taves et al. tentatively identified PFOA in pooled blood." This is not true and does not reflect what was stated in the study abstract by Taves et el. Furthermore, it ignores the limitations of the analytical procedures used, including the complex analytical processes and biases that were employed at the time (See Guy WS. 1979. Inorganic and organic fluorine in human blood. In (eds) Johansen E, Taves DR, Olsen TO. AAAS Selected Symposium 11. Pages 125-14. Westview Press; Boulder, Colorado). Thus, ATSDR needs to change this figure accordingly to reflect the technical details of the abstract.

Page 541:

The statement "Similarly, 3M and other manufacturers are using various perfluoropolyethers in fluoropolymer manufacturing and have reformulated surface treatment products to employ short-chain substances that are not as bioaccumulative as the long-chain perfluoroalkyls." Should be revised to state "3M and other manufacturers

are using various poly and ~~perfluoropolyethers~~ perfluoroether acid salts fluoropolymer manufacturing …"

Page 581:

The µg/L concentration discussed by Chang et al (2008) was only based on one sample. This should be so noted in this sentence.

Page 596:

Percentage declines should be provided in addition to modifiers such as "dramatic" or "clear" trend.

Page 633:

ATSDR should identify how many of the 400 epidemiological studies were cross-sectional.

Page 636:

As discussed elsewhere, the statement – "The available human studies have identified some potential targets of toxicity; however, cause-and-effect relationships have not been established for any of the effects, and the effects have not been consistently found in all studies" should be included up front on page 5 before the potential associations are discussed.

Additional comments:

- **Consolidate Epidemiological Study Information into Chapter 2.** ATSDR included a 277-page draft Supporting Document for Epidemiological Studies on Perfluoroalkyls. This provided the references, study populations, exposures, and outcomes for these epidemiological studies. While this information is helpful, it was burdensome to go from the figures and tables in Chapter 2 to this draft supporting document to identify the study designs identified in figures and tables in Chapter 2. Therefore, the study designs must be provided in tables and figures in Chapter 2 because the vast majority of the studies cited are cross-sectional where temporality cannot be determined.

- The draft Toxicological Profile mischaracterized the C8 Science Panel studies as having reported "cumulative PFOA exposure" when these estimates were based on an exposure model and not actually measured cumulative PFOA concentrations since they are reported as ng/mL-year. Therefore, ATSDR should consistently insert the word 'estimated' or 'modeled' in front of the word 'cumulative' throughout this document when referring to their data. Provided below are the references and page numbers where

these corrections must be made. This may not be exhaustive so ATSDR should do its own assessment of this mischaracterization. This issue also has to be addressed in the Draft Supporting Information for Epidemiologic Studies for Perfluoroalkyls (see below) where ATSDR usually acknowledges the word 'estimate' or 'modeled' in the Exposure Column of the C8 Science Panel references but rarely does the ATSDR use the words 'estimated' or 'modeled' in the Outcomes column.

	Study	Page
Reference	Steenland et al. 2015	10
	Steenland et al. 2015	14
	Simpson et al. 2015	18
	Winquist et al. 2014	19
	Steenland et al. 2015	31
	Steenland et al 2015	42
	Darrow et al. 2016	43
	Darrow et al. 2016	44
	Winquist et al. 2014a	46
	Steenland et al. 2015	71
	Steenland et al. 2015	84
	Winquist and Steenland 2014b	86
	Winquist and Steenland 2014b	87
	Steenland et al. 2015	105
	Steenland et al. 2015	106
	Steenland et al. 2015	237
	Karnes et al. 2014	239
	Steenland et al. 2015	253

Draft Supporting Document for Epidemiological Studies for Perfluoroalkyls	Study	Additional Note	Page
	Steenland et al. 2015		10
	Steenland et al. 2015		14
	Simpson et al. 2013		18
	Winquist and Steenland 2014a		19
	Olsen et al. 1998a.	Should be cross-sectional study	29
	Steenland et al. 2015		31
	Gilliland and Mandel 1996	Should be cross-sectional study	38
	Olsen et al. 2000	Should be cross-sectional study	39
	Olsen and Zobel 2007	Should be cross-sectional study	40
	Steenland et al. 2015		42
	Darrow et al. 2016		43
	Winquist and Steenland 2014a		46
	Olsen et al. 1999	Should be cross-sectional study	52
	Olsen et al. 2003	Should be cross-sectional study	53
	Mundt et al. 2007	Should be cross-sectional study	63
	Lundin et al. 2009	Should be cross-sectional study	69
	Steenland et al. 2015		71
	Olsen et al. 1998a	Should be cross-sectional study	76
	Olsen et al. 1998b	Should be cross-sectional study	83
	Olsen and Zobel 2007	Should be cross-sectional study	83
	Steenland et al. 2015		84
	Steenland and Winquist 2014b		86
	Olsen et al. 1998a	Should be cross-sectional study	90
	Mundt et al. 2007	Should be cross-sectional study	98
	Steenland et al. 2015		105
	Olsen et al. 1998b	Should be cross-sectional study	140
	Dhingra et al. 2016a		141
	Dhingra et al. 2016a		142
	Bach et al. 2016	Should be cohort study	143
	Olsen et al. 1998a	Should be cross-sectional study	152
	Bach et al. 2016	Should be cohort study	152
	Bach et al. 2016	Should be cohort study	168
	Whitworth et al. 2012a.	Should be cohort study	182
	Bach et al. 2016	Should be cohort study	225
	Bach et al. 2016	Should be cohort study	229
	Steenland et al 2015		237
	Steenland and Woskie et al. 2012		238
	Lundin et al. 2009.	Should be retrospective cohort study	251
	Steenland et al. 2015		253
	Steenland and Woskie et al. 2012		253

Citations:

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From: Curtis, James R.
To: Terranova, Sara
Cc: Stitt, Scott E; Liniger, Douglas E.
Subject: 35 Ill. Adm. Code 620 Outreach Extension and Stakeholder Q&A Session, IDOT response
Date: Friday, January 31, 2020 8:35:51 AM

Sara, The Illinois Department of Transportation provides the following comment regarding the proposed changes to the language in 35 Ill. Adm. Code 620: Groundwater Quality.

IDOT's Bureau of Design and Environment recommends permeameter studies remain an approved option for calculating hydraulic conductivity as part of a Class 1 groundwater determination, at least for road construction-related projects conducted by IDOT. Removal of this option will hinder the department's ability to efficiently conduct road construction and increase cost.

IDOT is a unique user of the permeameter study during investigations as part of road construction projects. Permeameter studies are a valid option because they are based on "undisturbed samples" collected from a boring, are laboratory controlled, follow ASTM methods, and are representative of the parent materials that are being targeted. A pump test is also reliable method of calculating hydraulic conductivity so long as the test is conducted for sufficient length of time. A pump test is, however, generally infeasible for evaluating groundwater characteristics on a typical road construction project. Slug testing, while field expedient and cost-effective, often lends itself to having the most error-prone data because results are based on where the user draws the line on the output curve. Many professionals incorrectly identify draining the sand pack as actual well recharge skewing results towards an inappropriate higher conductivity value.

When IDOT must manage groundwater during a road construction project, a Class II standard would be typically used. With the changes to the proposed regulations, IDOT would need to install a well, run slug or pump tests, and then characterize the groundwater based on those parameters as either Class I or Class II. This is impractical within the context of road construction.

Under the proposed regulations, IDOT would need to mobilize a minimum of 3 times to a project site to install a permanent well, characterize the groundwater and remove the well. The proposed changes would cause IDOT additional challenges in terms of time and cost in characterizing our wastes for management during road construction projects. A permeameter test option reduces the number of mobilizations by technical staff, the required time and the costs associated with the evaluation of groundwater. IDOT can collect the sample and have it run on a single mobilization.

In summary, IDOT recommends permeameter studies remain an approved option for calculating hydraulic conductivity as part of a Class 1 groundwater determination for road construction-related construction projects.

Jim Curtis
Chief, Geologic & Waste Assessment Unit

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From: Curtis, James R.
Sent: Thursday, January 30, 2020 2:36 PM
To: Terranova, Sara <Sara.Terranova@Illinois.gov>
Subject: RE: 35 Ill. Adm. Code 620 Outreach Extension and Stakeholder Q&A Session

Sara, We changed our mind. We will have a comment or two to provide to IEPA. Thanks, Jim

Jim Curtis, 217-558-4653, IDOT Central Office, Geologic & Waste Assessment Unit,
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From: Curtis, James R.
Sent: Tuesday, January 28, 2020 2:52 PM
To: Terranova, Sara <Sara.Terranova@Illinois.gov>
Subject: RE: 35 Ill. Adm. Code 620 Outreach Extension and Stakeholder Q&A Session

Sara, The IDOT Bureau of Design and Environment will not have comments to the proposed 620 regs. Thanks, Jim

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Cc: Sofat, Sanjay <Sanjay.Sofat@Illinois.gov>; Diers, Stefanie <Stefanie.Diers@Illinois.gov>; Zeivel, Christine <Christine.Zeivel@illinois.gov>; Cobb, Rick <RICK.COBB@Illinois.gov>; Hawbaker, Carol <Carol.Hawbaker@Illinois.gov>; Nifong, Heather <Heather.Nifong@Illinois.gov>; Rettig, Todd <Todd.Rettig@illinois.gov>; Dunaway, Lynn <LYNN.DUNAWAY@Illinois.gov>; Buscher, Bill <BILL.BUSCHER@Illinois.gov>; Hill, Christopher <Christopher.Hill@Illinois.gov>; Lake, Paul <Paul.Lake@Illinois.gov>; Falco, Charlene <Charlene.Falco@Illinois.gov>; Peters, Christopher M <Christopher.M.Peters@illinois.gov>
Subject: 35 Ill. Adm. Code 620 Outreach Extension and Stakeholder Q&A Session
Importance: High

Dear Stakeholders:

The Illinois Environmental Protection Agency (Illinois EPA or Agency) is extending the comment period regarding the proposed changes to the language in 35 Ill. Adm. Code 620: Groundwater Quality to **Friday, February 28, 2020**. The Illinois EPA will also be holding a stakeholder Q&A session regarding the proposed changes to the language in Part 620, in particular on Illinois EPA's development of the proposed PFAS groundwater quality standards on **Thursday, February 13, 2020**.

At the Q&A session, the Agency will be explaining the methodology used in the development of the PFAS groundwater quality standard and fielding any questions from the group.

Details regarding the Q&A session location, starting time, and a call-in number for those who cannot attend in person, **will be provided following this email**. To aid in this discussion, the Agency is attaching Part 620 Appendix A; *Procedures for Determining Human Threshold Toxicant Advisory Concentration for Class I: Potable Resource Groundwater* and the document: *Illinois EPA's Development of Proposed PFAS Groundwater Quality Standards for 35 Illinois Adm. Code Part 620.*

Please direct all questions and comments to me, Sara Terranova at:
Sara.Terranova@illinois.gov
217-558-3098

Thank you!

Sara G. Terranova
Assistant Counsel
Division of Legal Counsel
Illinois Environmental Protection Agency
1021 North Grand Avenue East, P.O. Box 19276
Springfield, Il 62794-9276
Phone: 217-782-5544 / Fax: 217-782-9807
Sara.Terranova@illinois.gov

From: Katie Pelch, PhD
Organization: Natural Resources Defense Council
1) Are there technical support documents available for the proposed groundwater quality standards?
2) how are these related to the health based guidance levels available at: https://www2.illinois.gov/epa/topics/water-quality/pfas/Documents/HA%20PFOS.pdf Some of the values are different between the health based values and the groundwater quality standards and I'd like to better understand where this difference derives from.
3) If you have information on the health based values (or know who I should contact), I'm curious why there isn't a value for PFNA, though it is mentioned on the page and there was a draft value for PFNA available in January 2020 and there seems to be a groundwater quality standard recommended for PFNA?

Comment: I'm unclear if these questions will be addressed or not at tomorrow's public meeting and would appreciate any further clarification you could provide.

From: Daniel Lombardi, Principal Hydrologist
Organization: St. John-Mittelhauser & Associates, Inc.,

1) What was the basis for having the same groundwater quality criteria for the five new PFA compounds and 1,4-Dioxane be the same for **both** Class I and Class II groundwater?

Comment: These new Class II standards should not be subject to the same Class I standards for those occurrences where groundwater is not used for potable sources of drinking water. I believe there would be a lower risks relating to Class II groundwater and the new criterial should be changed to account for it.

620 Questions and Comments

From: Katie Pelch, PhD
Organization: Natural Resources Defense Council
Date: 5/25/21

1) Are there technical support documents available for the proposed groundwater quality standards?
2) how are these related to the health based guidance levels available at: https://www2.illinois.gov/epa/topics/water-quality/pfas/Documents/HA%20PFOS.pdf Some of the values are different between the health based values and the groundwater quality standards and I'd like to better understand where this difference derives from.
3) If you have information on the health based values (or know who I should contact), I'm curious why there isn't a value for PFNA, though it is mentioned on the page and there was a draft value for PFNA available in January 2020 and there seems to be a groundwater quality standard recommended for PFNA?

Comment: I'm unclear if these questions will be addressed or not at tomorrow's public meeting and would appreciate any further clarification you could provide.

From: Daniel Lombardi, Principal Hydrologist
Organization: St. John-Mittelhauser & Arachel Bretzssociates, Inc.,
Date:5/25/21

1) What was the basis for having the same groundwater quality criteria for the five new PFA compounds and 1,4-Dioxane be the same for **both** Class I and Class II groundwater?
Comment: These new Class II standards should not be subject to the same Class I standards for those occurrences where groundwater is not used for potable sources of drinking water. I believe there would be a lower risks relating to Class II groundwater and the new criterial should be changed to account for it.

From Rachel Bretz, Director of Water Quality and Environmental Compliance
Organization: Illinois American Water
Date: 6/9/21
Comment:

- included PFAS (PFBS, PFHxS, PFNA, PFOA, PFOS) in both Class I and II groundwater limits
- Levels are slightly different than the drinking water HALs they established (Table below) ☐

Acronym		**Health-Based Guidance Level**	Groundwater Quality Standard **Proposed**
		(ng/L)	**(ng/L)**
Perfluorobutanesulfonic acid	PFBS	2,100*	1200
Perfluorohexanesulfonic acid	PFHxS	140	77
Perflurooctanesulfonic acid	PFOS	14	7.7
Perfluorooctanoic acid	PFOA	2	2
Perfluorohexanoic acid	PFHxA	560,000	NONE
PFNA (perfluorononanoic acid)	PFNA	NONE	12

From Carol Hawbaker
Organization: Illinois EPA
Date:6/11/21
Comment:

ITRC which has the most comprehensive information on it regarding other states data. It is located at: https://pfas-1.itrcweb.org/fact-sheets/
Under the "Regulations" bullet (PFAS Water and Soil Values Table Excel File). The Excel file units are in µg/L (and cover many chemicals not included in the proposed updates to 620, so I'll condense here):

☐

Region 5 State	Type (GW/DW)	Promulgated Rule (Y/N)	PFBS (ng/L)	PFHxS (ng/L)	PFNA (ng/L)	PFOA (ng/L)	PFOS (ng/L)
Illinois Proposed	GW		1,200	77	12	2	7.7
Indiana	GW	Y (2019)	400,000	----	----	----	----
Michigan	DW/GW	Y (2021)	420	51	6	8	16
Minnesota	DW/GW	*See Note Below	2,000	47	----	35	15
Ohio	DW	N (2019)	140,000	140	21	70**	70**
Wisconsin	GW	N	450,000	40	30	20**	20**

ota has promulgated rules (2018) with chronic Health Risk Limit (HRL) values for PFOA = 35 ng/L, PFOS = 300 ng/L and PFBS = 7,000 ng/L. In 2019, Minnesota proposed updated Health Based Values (HBVs) for PFOS = 15 ng/L and PFBS = 2,000 ng/L and introduced an HBV for PFHxS = 47 ng/L. The proposed HBVs are not promulgated.
** Guidance levels based in individual or combined FPOA/PFOS level of 70 ng/L for Ohio, and 20 ng/L for Wisconsin.

Note, the units in the above table are ng/L or ppt. For reference:
mg/L = ppm
µg/L = ppb
ng/L = ppt

The proposed values use the recently released final toxicity values for PFBS (PPRTV in May 2021), PFHxS, PFNA, PFOA, and PFOS (all ATSDR in May 2012) for non-cancer evaluations. However, in the case of PFOA, the only PFAS meeting the Act's definition of a carcinogen, the cancer value is more stringent than the non-cancer value. Therefore, the PFOA cancer value, using California EPA's cancer toxicity value, is more stringent.

From:	Bailey, Sabrina
To:	Terranova, Sara; Brown, Michael L.; Dunaway, Lynn; Frost, Brad; Lieberoff, Barb; Wake, Elizabeth; Guy, Jeff; Nifong, Heather; Diers, Stefanie; Sofat, Sanjay; Ankney, Clayton; Martin, Lauren; Hawbaker, Carol; Woods, Teschlyn; Irlam, Justin; Shaw, Melinda; Wilson, Nicole; Dunn, Greg; Summers, Michael
Subject:	620 Questions and Comments 6/22/21
Date:	Tuesday, June 22, 2021 2:47:38 PM

From: Donna Campbell, Client Relations Manager
Organization: Eurofins TestAmerica
Date: 6/22/21

Comments.

- The new standard for Vanadium of 0.00027 mg/l is not achievable by 6020A ICP-MS, which is the industry-standard for meeting lower level metals limits. This limit is over 10x lower than what can typically be met with this methodology.

- Dibenzo(a,h)anthracene at 0.000025 mg/l is not achievable by 8270D, 8270D LL or 8270D SIM. Again, this limit is over 10x lower than what can typically be met with these methodologies. **Question:** Is it possible that one to many zeros to the right of the decimal place were added?

Sabrina Bailey, PhD
Office of Community Relations
Illinois EPA
(847) 294-4394
Sabrina.Bailey@illinois.gov

From: Bailey, Sabrina <Sabrina.Bailey@Illinois.gov>
Sent: Wednesday, June 9, 2021 8:33 AM
To: Terranova, Sara <Sara.Terranova@Illinois.gov>; Brown, Michael L. <Michael.L.Brown@Illinois.gov>; Dunaway, Lynn <LYNN.DUNAWAY@Illinois.gov>; Frost, Brad <Brad.Frost@Illinois.gov>; Lieberoff, Barb <Barb.Lieberoff@Illinois.gov>; Wake, Elizabeth <Elizabeth.Wake@Illinois.gov>; Guy, Jeff <Jeff.Guy@Illinois.gov>; Nifong, Heather <Heather.Nifong@Illinois.gov>; Diers, Stefanie <Stefanie.Diers@Illinois.gov>; Sofat, Sanjay <Sanjay.Sofat@Illinois.gov>; Ankney, Clayton <Clayton.Ankney@Illinois.gov>; Martin, Lauren <Lauren.Martin2@Illinois.gov>; Hawbaker, Carol <Carol.Hawbaker@Illinois.gov>; Woods, Teschlyn <Teschlyn.Woods@Illinois.gov>; Irlam, Justin <Justin.Irlam@Illinois.gov>; Shaw, Melinda <Melinda.Shaw@illinois.gov>; Wilson, Nicole <Nicole.Wilson@Illinois.gov>; Dunn, Greg <Greg.Dunn@Illinois.gov>; Summers, Michael <Michael.Summers@Illinois.gov>
Subject: Re: 620 Questions and Comments 6/9/21

Good Morning All,
Below are comments from Illinois American Water.

From Rachel Bretz, Director of Water Quality and Environmental Compliance
Organization: Illinois American Water

Comment:

- included PFAS (PFBS, PFHxS, PFNA, PFOA, PFOS) in both Class I and II groundwater limits
- Levels are slightly different than the drinking water HALs they established (Table below)

Acronym		**Health-Based Guidance Level**	Groundwater Quality Standard **Proposed**
		(ng/L)	**(ng/L)**
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Perfluorooctanoic acid	PFOA	2	2
Perfluorohexanoic acid	PFHxA	560,000	NONE
PFNA (perfluorononanoic acid)	PFNA	NONE	12

Sabrina Bailey, PhD
Office of Community Relations
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From: Bailey, Sabrina
Sent: Wednesday, May 26, 2021 11:35 AM
To: Terranova, Sara <Sara.Terranova@Illinois.gov>; Brown, Michael L. <Michael.L.Brown@Illinois.gov>; Dunaway, Lynn <LYNN.DUNAWAY@Illinois.gov>; Frost, Brad <Brad.Frost@Illinois.gov>; Lieberoff, Barb <Barb.Lieberoff@Illinois.gov>; Wake, Elizabeth <Elizabeth.Wake@Illinois.gov>; Guy, Jeff <Jeff.Guy@Illinois.gov>; Nifong, Heather <Heather.Nifong@Illinois.gov>; Diers, Stefanie <Stefanie.Diers@Illinois.gov>; Sofat, Sanjay <Sanjay.Sofat@Illinois.gov>; Ankney, Clayton <Clayton.Ankney@Illinois.gov>; Martin, Lauren <Lauren.Martin2@Illinois.gov>; Hawbaker, Carol <Carol.Hawbaker@Illinois.gov>; Woods, Teschlyn <Teschlyn.Woods@Illinois.gov>; Irlam, Justin <Justin.Irlam@Illinois.gov>; Shaw, Melinda <Melinda.Shaw@illinois.gov>; Wilson, Nicole <Nicole.Wilson@Illinois.gov>; Dunn, Greg <Greg.Dunn@Illinois.gov>; Summers, Michael <Michael.Summers@Illinois.gov>
Subject: 620 Questions and Comments

Good Morning All,
Attached are comments and questions concerning 620 proposed changes. I will send a daily update of the comments in word, and they will be added to an excel spreadsheet that will be updated weekly and shared.

Sabrina Bailey, PhD
Office of Community Relations
Illinois EPA

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Sabrina.Bailey@illinois.gov

From: Diers, Stefanie
To: Terranova, Sara
Subject: 620
Date: Friday, February 28, 2020 2:24:05 PM

Ken Liss (it is with Andrews Engineering) called and was asking some questions on 620. He is with a Site Remedial Council. They are concerned about economic impacts of this rule and wanted to know if the Agency has considered those impacts.

Stefanie N. Diers
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Division of Legal Counsel, Illinois EPA
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The PFAS Regulatory Coalition
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February 28, 2020

VIA ELECTRONIC AND REGULAR MAIL
Stephanie Flowers
Illinois Environmental Protection Agency
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P.O. Box 19276
Springfield, Illinois 62794-9276
stephanie.flowers@illinois.gov

Re: Comments of the PFAS Regulatory Coalition on Proposed Rulemaking on Section 620.410 Groundwater Quality Standards for Class I Potable Resource Groundwater

Dear Sir or Madam:

The PFAS Regulatory Coalition (Coalition) appreciates the opportunity to file comments regarding the proposed rulemaking on Section 620.410 Groundwater Quality Standards for Class I Potable Resource Groundwater.

I. The Coalition's Interest

The Coalition is a group of industrial companies, municipal entities, agricultural parties, and trade associations that are directly affected by the State's development of policies and regulation related to per- and polyfluoroalkyl substances (PFAS). Coalition membership includes entities in the automobile, coke and coal, iron and steel, municipal, paper, petroleum, and other sectors. None of the Coalition members manufacture PFAS compounds. Coalition members, for purposes of these comments, include: American Coke and Coal Chemicals Institute; American Forest and Paper Association; American Iron and Steel Institute; Barr Engineering; Brown & Caldwell; Gary Sanitary District (IN); Illinois Association of Wastewater Agencies; Lowell, MA; Pueblo, CO; Tempe, AZ; Toyota; Trihydro, and Yucaipa Valley Water District (CA).

Coalition members support the State's efforts to identify potential sources of those individual PFAS that pose risks to human health and the environment, and to prioritize the protection of drinking water sources for vulnerable populations. In the State's pursuit of such regulations, the Coalition urges State regulators to ensure that final standards are scientifically supported, cost-effective, and achievable.

II. Proposed Rulemaking

On December 24, 2019, the Illinois Environmental Protection Agency (IEPA or State) sent letters to a limited number of "stakeholders," proposing changes to the State's groundwater quality standards to protect potential sources of drinking water and proposing to add new contaminants (with related standards), including certain perfluoroalkyl substances (PFAS) compounds. In proposing new standards, the State relied heavily on the "Minimum Risk Levels" drafted by the United States Agency for Toxic Substances and Disease Registry (ATSDR) and the U.S. Environmental Protection Agency's (USEPA) Provisional Peer Reviewed Toxicity Values. The proposed rulemaking designates five PFAS compounds with corresponding groundwater standards, as follows:

- Perfluorobutane Sulfonic Acid (PFBS): 0.14 mg/L
- Perfluorohexane Sulfonic Acid (PFHxS): 0.00014 mg/L
- Perfluorononanoic Acid (PFNA): 0.000021 mg/L
- Perfluorooctanoic Acid (PFOA): 0.000021 mg/L
- Perfluorooctane Sulfonic Acid (PFOS): 0.000014 mg/L

The proposed rulemaking also contains a combined PFOA and PFOS groundwater standard of 0.000021 mg/L. Additionally, Section 620.310 requires preventive response activities, including preventive notification mandates.

The PFAS Regulatory Coalition has general concerns with the State's decision to notice only a limited number of affected stakeholders, as well as the derived standards it is proposing for the various PFAS compounds. Because of the limited outreach insofar as the proposal, the Coalition did not even learn about the proposed standards until almost half way into the short comment period. The Coalition appreciates the comment period was extended but is still concerned that notice of such significant regulatory changes should have been more widely distributed.

Regarding the proposal itself, the proposed standards raise significant questions about their scientific basis and justification. The Coalition does not believe that groundwater monitoring and cleanup standards should be based on the ATSDR oral reference doses, which are derived for purposes other than environmental regulation, such as those being considered and developed by USEPA.

As discussed below, the Coalition requests that the State reconsider its new proposed standards and work more closely with all stakeholders to develop appropriate standards that provide necessary protection of the State's groundwater resources without unreasonably burdening the regulated community with unnecessarily stringent standards.

III. Coalition Analysis and Recommendations

In the comments below, the Coalition recognizes and summarizes some of the challenges that the State faces in attempting to promulgate enforceable regulations, as well as some of the challenges that Coalition members face if states promulgate standards that vary from any existing or future federal standards. The Coalition appreciates the State's desire to act to protect its citizens from potential risks associated with exposure to certain PFAS compounds, but urges Illinois and other states to work with the federal government to develop a cohesive national strategy to help ensure national uniformity. The prospect of a patchwork set of state-specific standards that vary widely is likely to cause significantly more confusion and overwhelming challenges for Coalition members that operate in multiple states or nationwide.

A. The Scientific Community Does Not Agree on Human Health Toxicity Values for PFAS

The term "PFAS" refers to a group of man-made chemicals that include perfluorooctanoic acid (PFOA), perfluorooctane sulfonic acid (PFOS), GenX,[1] and other fluorinated compounds. The most prevalent and available science regarding the incidence and potential health effects of PFAS is based on PFOA and PFOS, two compounds that are no longer manufactured in the United States due to voluntary phase outs. For replacement chemicals, industry has begun using shorter-chain PFAS that have different physical, chemical, and toxicological properties from the long-chain PFOA and PFOS. The scientific understanding of how PFAS impacts people and the environment is still developing and, for thousands of PFAS compounds, much remains unknown. From a toxicological perspective, regulatory agencies must have adequate science for determining health-based values before promulgating individual compound standards, limits, and related regulations.

Toxicologists, whether they work for various state agencies, USEPA, international standards-setting organizations, academia, or in private practice, have not yet established specific methodologies, resources, or even agreed on which of the hundreds of studies of PFAS compounds are the appropriate or critical studies that must or should support appropriate regulatory "standards." Different methodologies, levels of experience, procedural prerequisites to standards-setting, and even local political pressures are leading to consideration of very different standards in various states and at USEPA. Accordingly, the Coalition urges states to work with one another and with USEPA to continue

[1] Note that GenX is a trade name for a specific PFAS compound, ammonium, 2,3,3,3-tetrafluoro-2-(heptafluoropropoxy) propanoate. ITRC "Naming Conventions and Physical and Chemical Properties of Per- and Polyfluoroalkyl Substances (PFAS)," at 12, *available at* https://pfas-1.itrcweb.org/wp-content/uploads/2018/03/pfas_fact_sheet_naming_conventions__3_16_18.pdf (last visited January 23, 2020). More generically, GenX can be denoted by the abbreviation, "HFPO-DA."

developing science and methodologies to inform and encourage a more uniform approach to federal and state PFAS regulatory mandates.

B. Federal Action on PFAS

USEPA has issued "Interim Recommendations for Addressing Groundwater Contaminated with PFOA and PFOS."[2] Those recommendations provide clear and consistent guidance for federal cleanup sites being evaluated and addressed under federal programs, including the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) and the Resource Conservation and Recovery Act (RCRA). The screening levels followed under such cleanups are risk-based values that are used to determine if levels of contamination may warrant further investigation at a site. The recommendations are intended to be used as guidance for states to evaluate state cleanup and corrective action sites. The interim guidance recommends in relevant part:

- Using a screening level of 40 parts per trillion (ppt) to determine if either PFOA, or PFOS, or both, is present at a site and may warrant further attention.
- Using USEPA's PFOA and PFOS Lifetime Drinking Water Health Advisory level of 70 ppt as the preliminary remediation goal (PRG) for contaminated groundwater that is a current or potential source of drinking water, where no state or tribal MCL or other applicable or relevant and appropriate requirements (ARARs) are available or sufficiently protective.

In addition, USEPA is focusing significant resources on developing appropriate regulatory mechanisms specific to various PFAS compounds. For example, USEPA has developed a PFAS Action Plan, which provides a multi-media, multi-program, national research, and risk communication plan to address emerging PFAS challenges.[3] Part of USEPA's PFAS Action Plan involves expanding the scientific foundation for understanding and managing risk from PFAS, including researching improved detection and measurement methods, generating additional information about PFAS presence in the environment and drinking water, improving the understanding of effective treatment and remediation methods, and developing more information regarding the potential toxicity of a broader set of PFAS. In turn, USEPA expects that this information will help states and others better manage PFAS risks.

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[2] USEPA Office of Land and Emergency Management, OLEM Directive No. 9283.1-47 (December 19, 2019), *available at* https://www.epa.gov/sites/production/files/2019-12/text_version_epas_interim_recommendations_for_addressing_groundwater_contaminated_with_pfoa_and_pfos_dec_2019.txt.

[3] *See* USEPA "EPA's Per- and Polyfluoroalkyl Substances (PFAS) Action Plan" (February 2019) *available at* https://www.epa.gov/sites/production/files/2019-02/documents/pfas_action_plan_021319_508compliant_1.pdf.

EPA is also moving towards possible Maximum Contaminant Level (MCL) standards for PFOA and PFOS—two of the most well-known and prevalent PFAS chemicals. On February 20, 2020, EPA released a prepublication version of its Regulatory Determination for Contaminants on the Fourth Drinking Water Contaminant Candidate List. The Regulatory Determination supports regulating under PFOA and PFOS under the Safe Drinking Water Act, meaning EPA is proposing to move forward with setting MCLs for this two PFAS compounds. In making this determination, EPA also relied on the reference dose of 0.00002 mg/kg/day for both compounds.[4] EPA has stated that, "[p]roposing a regulatory determination is the next step in the maximum contaminant level [] rulemaking process under the Safe Drinking Water Act; it enables the USEPA to propose and solicit comment on information critical to regulatory decision-making towards protecting public health and communities across the nation."[5] Additionally, USEPA is gathering and evaluating information to determine if similar regulations are appropriate for a broader number of PFAS compounds.

While USEPA is working through its long-established processes and rulemaking procedures, Congress is considering ways to expedite and fund various national standards-setting approaches. Recently, the U.S. House of Representatives passed the PFAS Action Act (H.R. 535), which would require, among other things, that USEPA promulgate a national primary drinking water regulation for certain PFAS and a health advisory for other PFAS not subject to a national primary drinking water regulation. Also, Congress passed and then the President signed into law the National Defense Authorization Act (NDAA) (P.L. 116-92) that mandates additional federal actions to regulate and manage various risks associated with many PFAS. While we recognize that not all states and stakeholders can agree on specific priorities or approaches to PFAS regulations, these congressional actions, combined with USEPA's efforts, are important national developments that should be supported by the states through their contribution of expertise, resources, and efforts as the Nation works to respond to PFAS exposure risks.

Indeed, a patchwork of 50 different state solutions is unworkable and contrary to how the U.S. has previously addressed similar emerging contaminant issues. While some limited variations related to groundwater, surface water, or soil cleanup levels may be expected and appropriate, the highly variable regulatory health advisories, action levels, and drinking water standards currently being developed or under consideration across the country create unnecessary confusion and complexity for the public and the regulated community.

The Coalition recognizes that states have elected to utilize different methods and processes for communicating risks to their populations. However, standards-setting must reflect more national and uniform collaboration and cohesion. We must work to avoid the

[4] This Regulatory Determination had not yet been published in the *Federal Register* at the time of drafting of these comments, but is available at: https://www.epa.gov/sites/production/files/2020-02/documents/ccl_reg_det_4_preliminary_frn.webposting.pdf.
[5] *Id.*

undesirable solution of 50 separate state rules, particularly with regard to drinking water standards. With this in mind, we urge the states to work closely with USEPA to establish science-based and peer-reviewed federal standards that serve as the basis for comparable state standards. Such an approach is consistent with how USEPA and the states have addressed environmental and human health risks since the creation of USEPA.

In addition, the Coalition can foresee challenges to states that choose to develop their own unique and varying drinking water standards. Many jurisdictions have existing laws or rules that prohibit the state from promulgating regulations that are more stringent than the federal rules. When USEPA does promulgate national primary drinking water regulations, such states may be in conflict with their legislature's clearly stated policy. These states may be required to amend their state-specific PFAS regulations when USEPA completes its work in this regard. And, state antibacksliding provisions may complicate their abilities to change their standards to conform with federal rules.

Considering the above, implementation of any future federal standards likely will be more complex and resource-consuming for states that set their own limits in advance of federal action. Indeed, the purpose of federal law is to protect against a patchwork of state law. Accordingly, the State should clearly articulate how forthcoming federal drinking water standards may impact this State-specific proposed rulemaking, how the State will help to foster consistency and uniformity with neighboring states, and how the State will defer to federal standards or revise standards based on future federal action and improved scientific understanding about exposure, dose, and toxicology.

The Coalition urges the State to use its resources to support the development of sound science upon which USEPA can base its federal standards, heed the non-binding recommendations of USEPA's Federal Health Advisory of 70 ppt (for PFOA and PFOS combined) and, ultimately, work to implement any forthcoming national primary drinking water standards. This will protect the State from expending resources on establishing and enforcing individual PFAS drinking water standards that are inconsistent both with other states and with federal science-based and peer-reviewed standards.

C. Reliance on the ATSDR Values

The ATSDR, part of the federal Center for Disease Control, and many states have reviewed the toxicity information available for PFOA and PFOS and opined on appropriate dosages that reflect highly conservative assumptions designed to protect human health, including the most susceptible subpopulations. ASTDR values are derived through different methods than USEPA's MCL (and Health Advisory) values and the two are not directly comparable.[6] These variabilities in how various health recommendations are

[6] *See* ATSDR Public Health Assessment Guidance Manual (2005) at Appendix F: Derivation of Comparison Values (https://www.atsdr.cdc.gov/hac/phamanual/appf.html) ("MCLs represent more realistic assumptions about toxicity and contain fewer uncertainty factors than the very conservative ATSDR environmental guidelines.")

derived must be considered and addressed to ensure that any final standards are scientifically justified and corroborated. [7]

Moreover, ATSDR has only finalized the Toxicological Profile for two PFAS compounds, PFOA and PFOS. The profiles for two additional PFAS—Hexafluoropropylene Oxide (HFPO) Dimer Acid, more commonly referred to as the "GenX Chemicals," and Perfluorobutane Sulfonic Acid/Potassium Perfluorobutane Sulfonate, referred to as PFBS—are still only in draft form. ATSDR made the Toxicological Profiles for these additional PFAS available for public comment in 2018, and the Profiles have not yet been finalized.

Considering the above, the Coalition recommends that the State base any rulemaking on any forthcoming national primary drinking water standards, rather than the draft ATSDR report. Further, according to Part 620 Subpart F, for substances that USEPA has not established a Maximum Contaminant Level Goal (MCLG), IEPA should base its highest priority approach for calculating the Advisory Concentration on the reference oral dose for humans as derived by USEPA. USEPA has not established MCLGs for any of the five compounds, but it has set a Health Advisory level of 70 ppt for PFOA and PFOS, individually or combined, based on oral reference doses of 0.00002 mg/kg/day for both compounds. Accordingly, IEPA should use the most current USEPA reference doses, such as those used for establishing the Health Advisory level for PFOA and PFOS, rather than establishing standards based on the ATSDR values, some of which are still in draft form.

And, even if the State still seeks to base its rulemaking on the ASTDR reference doses, the Coalition recommends that it wait until ATSDR finalizes its Toxicological Profiles, as the science supporting ATSDR's reference doses is not fully developed nor has the scientific community generally agreed on the science. Moreover, ATSDR has not even drafted profiles for some of the compounds that the State is proposing to regulate.

The State, at best, must avoid underpinning regulations on information that the scientific community is still debating, or using science not yet fully developed enough for ATSDR to draft recommendations. USEPA is actively working on developing its own assessments for these and other PFAS compounds and, consequently, final standards-setting is still premature.

D. Specificity in the Type of Regulated PFAS

Generally, PFAS regulations should clearly specify the individual compounds of PFAS that they seeks to regulate. Given the wide variations in toxicities and other characteristics exhibited by different PFAS chemicals, it is not scientifically appropriate to

[7] For a thorough discussion on possible confusion created by comparing ATSDR and EPA standards, *see* ECOS White Paper (*Processes & Considerations for Setting State PFAS Standards*) Appendix A, *available at*: https://www.ecos.org/documents/ecos-white-paper-processes-and-considerations-for-setting-state-pfas-standards/ (last accessed Feb. 28, 2020).

group all PFAS together for purposes of risk assessment or to assume that exposures to mixtures of PFAS necessarily bioaccumulate in one's body in interchangeable 1:1 ratios.

Accordingly, the Coalition supports the proposed rulemaking's specificity in identifying which PFAS compounds are regulated and recommends that the regulation of individual PFAS substances reflect peer-reviewed science regarding the physical, chemical, and toxicological properties of each compound. Similarly, the Coalition recommends against including any combined PFAS standards or limits unless science clearly demonstrates that the mixture of the PFAS compounds subject to the combined limit results in bioaccumulation in hazardous concentrations.

E. Validated Test Methods for PFAS

The State should regulate only those PFAS comopounds for which there are validated analytical test methods. USEPA's main validated test methods for PFAS, Methods 537 and 537.1, apply only to 18 PFAS compounds in samples derived from drinking water. USEPA recently issued Method 533 that can be used to measure an additional 11 "short-chain" PFAS compounds (and only 14 of the 18 PFAS covered by Method 537.1), again only for use in testing drinking water. Therefore, the entirety of USEPA's approved test methods can measure no more than 29 different PFAS compounds, and multiple methods would have to be used to obtain results from all 29 compounds.

No validated USEPA test methods exist for testing PFAS compounds in any other environmental media. USEPA has received comments on a draft non-potable water test method (SW-846 Method 8327), but that method is only considered "guidance" at this time. USEPA also is working with the Department of Defense's Naval Seas Systems Command Laboratory Quality and Accreditation Office to validate a solid-phase extraction/isotope dilution method to include solid matrices (*i.e.,* for soil, sediment, fish tissue, biosolids), as well as non-potable water sources, but that effort may not be completed until 2021.[8]

Accordingly, the Coalition recommends that the proposed rulemaking recognize the limits of the available USEPA validated test methods and choose a specific test method to be referenced by any standards being adopted. Limitations on test methods and the lack of any validated method by USEPA for anything except drinking water create major challenges for the State's efforts to regulate non-potable water or other matrices.

[8] *See* PFAS Methods Technical Brief *available at* https://www.epa.gov/sites/production/files/2020-01/documents/pfas_methods-sampling_tech_brief_7jan2020-update.pdf.

F. Testing Capabilities and Reliability

The Coalition urges the State to consider the capabilities and reliability of laboratories that test for PFAS. There is limited capacity nationally to perform all of the analytical laboratory work and limited reliability on any given sample result due to potential lab error, cross contamination, or other factor that could impact results in the very low parts per trillion levels being considered. There is little doubt that the closer the State sets a limit or standard to the detection limit, analytical sampling and related lab results become increasingly unreliable.

For example, Coalition members who have sent split samples to multiple labs report receiving highly variable results. Such anecdotal evidence demonstrates the potential difficulty and unreliability of performing testing at limits that approach the detection limit. Considering that the State can potentially impose fines, costly corrective action, or other penalties for failing to meet regulatory limits, the regulated community must have the ability to accurately measure PFAS to demonstrate compliance. Subjecting the regulated community to fines, corrective action, and other penalties based on potentially unreliable testing raises due process concerns. Accordingly, the Coalition urges the State to consider testing capability and reliability, and set limits and impose a regulatory scheme that accounts for the variability in and limits of current laboratory testing.

G. Availability of Testing and Disposal

A limited number of established laboratories in the country have robust experiecnce testing and reporting PFAS results. The State's rulemaking should account for the limited number of testing laboratories in the region. The Coalition recommends, for example, that in regions where testing capacity is limited that the rule provide for a delayed effective date or phased implementation that allows for laboritories to develop the expertise necessary to reliably accommodate the increased testing that the rule will require.

Similarly, treatment technologies for PFAS are still being developed, and there is limited capacity for the disposal of byproducts from newly-developed technologies. For example, absorption technologies such as granular activated carbon (GAC) are being developed as potential response measures to achieve compliance with new drinking water standards for PFAS. The regulated community will need to safely dispose of the byproducts of such treatment technologies used to treat PFAS in drinking water. Again, this is another area where USEPA is taking action.

Congress, in the NDAA, mandated that USEPA, not later than one year after enactment, "publish interim guidance on the destruction and disposal of perfluoroalkyl and polyfluoroalkyl substances and materials containing perfluoroalkyl and polyfluoroalkyl substances," which includes guidance on "spent filters, membranes, resins, granular

carbon, and other waste from water treatment."[9] The Coalition urges the State to use its resources to support the development of USEPA's interim guidance documents prior to independently establishing MCLs.

H. The State Should Consider the Rulemaking's True Costs

The proposed rulemaking should account for the developing nature of treatment technologies and availability of disposal or other treatment endpoints. Information exists regarding the variable costs of treatment systems installed at locations around the country, and the State should consider that information in establishing remediation standards. Though information exists regarding the costs of treatment alternatives, there is signifcant uncertainty regarding how to handle byproducts from PFAS treatment.

For example, a remediating party may not be able to find a landfill to take the spent media, and incineration of the media is currently subject to criticism and further study. As stated in Section G above, Congress has directed USEPA to develop guidance to specially address these issues.

These remediation standards could also affect sites being remediated under federal programs, such as Superfund. For Department of Defense (DOD) sites, for example, the NDAA requires that cooperative agreements with states include that the DOD "shall meet or exceed the most stringent . . . standards for PFAS in any environmental media." NDAA Sec. 332(a)(2).

The states, municipalities, and private parties that are conducting these cleanups will incur substantial costs as a result. Accordingly, the State should consider the costs to remediate to these proposed standards in its regulatory analysis.

In sum, if this regulation will become final before there is more certainty regarding the underlying questions of treatment and disposal, then the State should conduct a more robust cost analysis to account for the potential costs, including remediation and the range of true disposal and ongoing operation and maintenance costs.

[9] NDAA Sec. 7631(4).

V. Conclusion

The Coalition appreciates the opportunity to submit these comments concerning the proposed rulemaking. We look forward to working closely with the State regarding developing appropriate, reasonable, and scientifically-defensible groundwater protection standards. Please feel free to call or e-mail if you have any questions, or if you would like any additional information concerning the issues raised in these comments.

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February 28, 2020

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Re: Proposed changes to 35 Illinois Administrative Code 620: Groundwater Quality

Dear Ms. Flowers:

The Chemical Products and Technology Division of the American Chemistry Council (ACC/CPTD) submits the following comments on the Illinois Environmental Protection Agency's (IEPA) proposal to establish groundwater quality standards for perfluorobutane sulfonic acid (PFBS), perfluorohexanesulfonic acid (PFHxS), perfluorooctanoic acid (PFOA), perfluorooctane sulfonic acid (PFOS), and perfluorononanoic acid (PFNA). ACC/CPTD represents companies interested in ensuring that regulations related to these substances, like the IEPA proposal, incorporate the best available science. As described below, we oppose the Agency's proposal to –

- establish groundwater quality standards for PFOA and PFOS that are below the interim recommendations established by the Office of Land and Emergency Management (OLEM) of the US Environmental Protection Agency (USEPA),
- establish standards for PFHxS and PFNA in the absence of recommendations from USEPA and without providing a rationale for how the proposed levels were selected, and
- set the same standards for the PFAS and other substances for both potable (Class I) and general resource (Class II) groundwater.

PFOA and PFOS

In setting groundwater quality standards, IEPA has historically sought to maintain consistency with federal levels – specifically using maximum contaminant levels (MCLs) established by USEPA for those substances for which drinking water standards have been established. It is concerning, therefore, that the proposed standards for PFOA and PFOS are not consistent with USEPA recommendations for groundwater cleanup recently finalized by the

Office of Land and Emergency Management (OLEM)[1] or the lifetime Health Advisories (HAs) established by the Office of Water in 2016. While only recommendations, the value of 70 part per trillion (ppt) developed by the two USEPA offices provide a consistent approach to addressing PFOA and PFOS contamination in groundwater – based on a very recent review of the available scientific information. Establishing a different standard in Illinois will create conflicting targets for cleanup at the various locations within the state and cause significant confusion about the appropriate cleanup level. ACC/CPTD urges IEPA to establish the groundwater standards for PFOA and PFOS at the levels recommended by USEPA.

PFHxS and PFNA

Unlike PFOA and PFOS, USEPA has not provided guidance on the appropriate drinking water or groundwater levels of PFHxS and PFNA.[2] While the Agency for Toxic Substances and Disease Registry (ATSDR) has released a draft evaluation for these two substances, ATSDR did not propose recommendations for drinking or groundwater. Available data for PFHxS and PFNA are limited, moreover, and it is not clear how IEPA developed the proposed groundwater standards for these two substances. Prior to proposing standards for these two substances, IEPA should make its derivation of the proposed values available for review and comment.

Proposed Values for Class I and Class II Groundwater

IEPA's proposal would establish the same standards for Class I and Class II groundwater for several substances, including the five PFAS, even though only Class I groundwater is considered potable by the state. The approach taken in the proposal is inconsistent with the Agency's historic practice of setting the standard for Class II groundwater higher than the standard for Class I groundwater. In the case of substances for which a federal MCL exists, for example, the Class II standard is set five times higher. IEPA has not provided a rationale for why it is proposing to change its approach or explained the public benefit that it provides. Classifying groundwater as to its appropriate use allows the state, local jurisdictions, and affected companies to focus resources on priority contamination. Establishing groundwater standards that disregard the specified use achieves little if any public benefit and could significantly increase the cost and complexity of ensuring groundwater quality. It would likely

[1] USEPA. Interim recommendations to address groundwater contaminated with perfluorooctanoic acid and perfluorooctane sulfonate. Memo from Peter C. Wright, Assistant Administrator, Office of Land and Emergency Management (December 19, 2019). https://www.epa.gov/pfas/interim-recommendations-addressing-groundwater-contaminated-pfoa-and-pfos,

[2] USEPA has released a draft toxicity assessment for the fifth PFAS included in the IEPA proposal, PFBS. The proposed groundwater standard for this substance appears consistent with the USEPA assessment, but IEPA's analysis should also be made available for public review.

delay compliance which further erodes public confidence that the state is taking effective action to protect public health.

ACC/CPTD urges the Agency to revise the draft regulation prior to formal proposal. Please contact me at srisotto@americanchemistry.com or at 202-249-6727 if you have questions about the information provided above.

Sincerely,

Steve Risotto

Stephen P. Risotto
Senior Director

January 31, 2020

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Sent via USPS and e-mail

Re: Proposed Amendment to 35 Ill. Adm. Code Part 620: Groundwater Quality

Dear Ms. Flowers:

The Illinois Attorney General's Office welcomes the opportunity to comment on the Illinois Environmental Protection Agency's proposed changes to Illinois' groundwater quality regulations (35 Ill. Adm. Code 620). We strongly support promulgating regulations to address the severe health risks presented by per- and polyfluoroalkyl substances (PFAS).

We have previously supported federal legislative efforts to address PFAS.[1] Unfortunately, the federal government has yet to adopt adequate PFAS legislation or regulations, making state regulations even more important.

Illinois must act to protect its citizens from the health risks of PFAS, particularly in light of the federal government's reluctance. For this reason, the Illinois Attorney General's Office strongly supports regulating PFAS in groundwater. Though PFAS contamination is a wide-ranging problem and cannot be solved by a single set of regulations, strong groundwater standards are necessary to protect the public.

Below, we have questions concerning the details of IEPA's proposal.

1) Why did IEPA choose a standard of 0.00014 mg/L for PFHxS?

IEPA proposed that concentrations of perfluorohexane sulfonic acid (PFHxS) shall not exceed 0.00014 milligrams per liter (mg/L) in Class I groundwater. 35 Ill. Adm. Code 620.410(b) (proposed). Other states have adopted different PFHxS standards. For example, Massachusetts adopted a much lower groundwater standard for PFHxS: 0.02 micrograms per liter (ug/L) (equivalent to 0.00002 mg/L).[2]

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[1] *See* Letter from Attorney General Kwame Raoul, et al. regarding Federal PFAS legislation, http://www.illinoisattorneygeneral.gov/pressroom/2019_07/Multistate_PFAS_Legislative_Letter_73019.pdf (July 30, 2019).

[2] Massachusetts Dep. of Environmental Protection, Final PFAS-Related Revisions to the MCP, https://www.mass.gov/lists/final-pfas-related-revisions-to-the-mcp-2019#final-regulations---promulgated-december-27,-2019- (Dec. 27, 2019).

How did IEPA choose its PFHxS standard (0.00014 mg/L)? Did IEPA consider a 0.00002 mg/L standard?

2) Why did IEPA choose a standard of 0.14 mg/L for PFBS?

IEPA proposed that concentrations of perfluorobutane sulfonic acid (PFBS) shall not exceed 0.14 mg/L in Class I groundwater. 35 Ill. Adm. Code 620.410(b) (proposed). By comparison, Minnesota has adopted a protective guidance standard for PFBS in drinking water at the level of 2 parts per billion (equivalent to 0.002 mg/L), which is much lower than IEPA's proposed groundwater standard.[3]

How did IEPA choose its PFBS standard (0.14 mg/L)? Was 0.002 mg/L considered?

3) Why did IEPA propose a combined standard for just PFOS and PFOA, rather than all five listed PFAS chemicals?

IEPA proposed limits for perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS) in Class I groundwater: PFOA concentrations shall not exceed 0.000021 mg/L; PFOS concentrations shall not exceed 0.000014 mg/L, and PFOA and PFOS combined shall not exceed 0.000021 mg/L. 35 Ill. Adm. Code 620.410(c)(3) (proposed). Other states have adopted combined standards that include additional PFAS. For example, Massachusetts adopted a groundwater standard of 0.02 ug/L (equivalent to 0.00002 mg/L) that applies to PFDA, PFHpA, PFHxS, PFNA, PFOS, and PFOA combined.[4]

How did IEPA choose 0.000021 mg/L for its combined standard? How did IEPA choose which PFAS to include in its combined standard? Did IEPA consider including substances in addition to PFOA and PFOS in its combined standard?

4) Did IEPA consider proposing a standard for additional PFAS?

IEPA proposed limits for five substances: PFOA, PFOS, PFBS, PFHxS, and perfluorononanoic acid (PFNA). Other states have adopted regulations for additional PFAS. For instance, Massachusetts also regulates perfluorodecanoic acid (PFDA) and perfluoroheptanoic acid (PFHpA) in groundwater.[5] Michigan has proposed limits on hexafluoropropylene oxide dimer acid (HFPO-DA, a.k.a. GenX) and perfluorohexanoic acid (PFHxA) in drinking water.[6] Furthermore, Congress recently adopted a law adding GenX to the toxics release inventory.[7]

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[3] Minn. Dept. of Health, Human Health-Based Water Guidance Table: https://www.health.state.mn.us/communities/environment/risk/guidance/gw/table.html.
[4] *Supra* at n.2.
[5] *Id.*
[6] Michigan Dept. of Environment, Great Lakes, and Energy, Drinking Water Rule Promulgation, https://www.michigan.gov/egle/0,9429,7-135-3313_3675_3691-9647--,00.html.
[7] P.L. 116-92 § 7321, https://www.congress.gov/116/bills/s1790/BILLS-116s1790enr.pdf (Dec. 20, 2019).

Lastly, using the U.S. Environmental Protection Agency analytical methods, laboratories can detect 29 PFAS in drinking water.[8]

How did IEPA choose the five PFAS it proposes to regulate (PFOA, PFOS, PFBS, PFHxS, and PFNA)? Did IEPA consider other PFAS (*e.g.*, PFDA, PFHpA, GenX, PFHxA, or other PFAS detectable under USEPA's analytical methods)? Did IEPA consider regulating PFAS as a class of chemicals, rather than individually?

5) What resources did IEPA use to develop its proposed standards?

IEPA's letter to the AGO from January 7, 2020 states that the proposed PFAS standards use "the methodology developed under Part 620 Subpart F with PFAS oral reference doses drafted by the Agency for Toxic Substances and Disease Registry (ATSDR)." Please fully cite the ATSDR resources relied upon for this proposal. Did the Agency rely on any other resources in developing its proposal? If so, please cite these additional resources.

6) Is IEPA considering additional PFAS regulations (air, drinking water, surface water, sampling, disclosure, etc.)?

Other states have used many different types of regulatory approaches in order to address PFAS contamination. For instance, several states have proposed or adopted regulations for PFAS in drinking water. States have also proposed or adopted regulations for PFAS in surface water, air, and other media. Additionally, states have adopted or proposed regulations and legislation for PFAS sampling and disclosure, PFAS manufacture, and use of PFAS-containing materials. Is IEPA considering additional regulations to address PFAS contamination?

8) What is the status of the Agency's PFAS sampling?

IEPA's 2018 groundwater and drinking program review[9] states that the Agency plans to study PFAS in community water supplies and surface water intakes. Can IEPA provide an update on this study's status? Does IEPA still expect the sampling to begin in early 2020 and expect the study to finish within a year?

Thanks again for the opportunity to weigh in on this important work.

Very truly yours,

/s/ Jason E. James
Jason E. James
Assistant Attorney General
Environmental Bureau

[8] U.S. Environmental Protection Agency, EPA PFAS Drinking Water Laboratory Methods, https://www.epa.gov/pfas/epa-pfas-drinking-water-laboratory-methods.
[9] Illinois EPA, Annual Groundwater and Drinking Water Program Review for Calendar Year 2018, https://www2.illinois.gov/epa/Documents/iepa/compliance-enforcement/drinking-water/2019_Groundwater_Drinking_Water_Program_Review_CY18_Report_Final.pdf (Dec. 2019).

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February 28, 2020

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Sent via USPS and e-mail

Re: Proposed Amendment to 35 Ill. Adm. Code Part 620: Groundwater Quality

Dear Ms. Flowers:

The Illinois Attorney General's Office adds this supplemental comment to the Illinois Environmental Protection Agency's proposed changes to groundwater quality regulations (35 Ill. Adm. Code 620), which include proposals concerning per- and polyfluoroalkyl substances (PFAS). We submitted our initial comment on January 31, 2020. Since then, we have reviewed IEPA's methodology fact sheet and participated in the February 13, 2020 stakeholder question and answer session, prompting the following questions.

1) Why did IEPA propose a combined standard for just PFOS and PFOA, rather than all five PFAS chemicals it proposes to regulate?

As described in our initial comment, IEPA proposed a combined limit for perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS). 35 Ill. Adm. Code 620.410(c)(3) (proposed). IEPA does not propose to include other PFAS in this combined limit.

IEPA has not directly addressed how it developed the combined standard. How did IEPA choose 0.000021 mg/L for PFOA and PFOS combined? Did IEPA consider adding the other three PFAS it proposes to regulate into the combined standard? If so, how did IEPA decide against including those other PFAS in the combined standard?

2) Why did IEPA choose a standard of 0.00014 mg/L for PFHxS?

IEPA proposed that concentrations of perfluorohexane sulfonic acid (PFHxS) shall not exceed 0.00014 milligrams per liter (mg/L). 35 Ill. Adm. Code 620.410(b) (proposed). All other states that have addressed PFHxS in groundwater have adopted significantly more stringent standards:

- Massachusetts: 0.00002 mg/L (7 times lower)[1]

[1] Massachusetts Dept. of Environmental Protection, Final PFAS-Related Revisions to the MCP, https://www.mass.gov/lists/final-pfas-related-revisions-to-the-mcp-2019#final-regulations---promulgated-december-27,-2019- (Dec. 27, 2019).

- Minnesota: 0.000047 mg/L (about 3 times lower)[2]
- New Hampshire: 0.000018 mg/L (about 7.75 times lower)[3]
- Texas: 0.000093 mg/L (about 1.5 times lower)[4]
- Vermont: 0.00002 mg/L (7 times lower)[5]

IEPA has provided general information about its methodology. Please provide more detail on how IEPA chose its PFHxS standard. How does IEPA's analysis differ from other states' analysis?

3) Did IEPA review underlying scientific studies on PFAS?

IEPA stated that it based its proposed standards on the Agency for Toxic Substances and Disease Registry's June 2018 draft toxicological profile for perfluoroalkyls[6] and U.S. EPA's Provisional Peer-Reviewed Toxicity Values for potassium perfluorobutane sulfonate (PFBS).[7] Did IEPA review the scientific studies underlying these documents (*e.g.*, Lao 2006,[8] Lieder 2009[9])? If so, please list the specific scientific studies that IEPA reviewed when drafting the proposed standards.

Did IEPA review any scientific studies concerning types of PFAS that it does *not* propose to regulate? For example, Massachusetts reviewed scientific studies on perfluorodecanoic acid (PFDA) that were released after the ATSDR assessment.[10] Please list specific studies or other resources that IEPA reviewed which concern PFAS that IEPA does not propose to regulate.

Thanks again for the opportunity to weigh in on this important work.

Very truly yours,

/s/ Jason E. James

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Environmental Bureau
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[2] Minnesota Dept. of Health, Toxicological Summary for Perflourohexane sulfonate, https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfhxs.pdf (April 2019).
[3] New Hampshire Code of Adm. Rules, Env-Or 600, https://www.des.nh.gov/organization/commissioner/legal/rules/documents/env-or600.pdf.
[4] Texas Commission on Environmental Quality, Toxicology Evaluation of Perfluoro Compounds, https://www.tceq.texas.gov/assets/public/implementation/tox/evaluations/pfcs.pdf (Jan. 4, 2016).
[5] Vermont General Assembly Act 21 (S.49), https://legislature.vermont.gov/bill/status/2020/S.49 (May 15, 2019).
[6] ATSDR, Toxicological Profile for Fluoroalkyls Draft for Public Comment, https://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=1117&tid=237 (June 2018).
[7] USEPA, Provisional Peer-Reviewed Toxicity Values for Potassium Perfluorobutane Sulfonate, https://cfpub.epa.gov/ncea/pprtv/recordisplay.cfm?deid=339119 (July 17, 2104).
[8] *See supra* n. 6 at Chapter 8, References.
[9] *See supra* n. 7 at Appendix D, References.
[10] *See* Mass. Dept. of Envt'l Prot., Technical Support Document for PFAS at Appendix 2, p32-35, https://www.mass.gov/doc/per-and-polyfluoroalkyl-substances-pfas-an-updated-subgroup-approach-to-groundwater-and/download (Dec. 26, 2019).

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Clean Air Act Permit Program (CAAPP)

Applicant submits CAAPP or CAAPP Renewal Application

IEPA conducts an administrative completeness review, makes decision within 60 days.

Notice of Incompleteness issued to applicant

Request for additional information issued to applicant

Notice of Completeness issued to applicant and technical review begins

For facilities in EJ Areas of Concern, EJ Notification process commences, informs IEPA of potential public interest

Technical review finishes, IEPA prepares draft of CAAPP permit, Statement of Basis, and other relevant materials or Notice of Intent to Deny

Public notice and 30-day comment period commences

Most follow this track

No public comments received

Written comments received, no request for hearing and/or other outreach taken (e.g. public meeting)

Comments received, public hearing requested and held at the authorization of the director

IEPA drafts Responsiveness Summary and proposed CAAPP permit

IEPA submits draft Responsiveness Summary and proposed CAAPP permit to USEPA

Following 45 day review by USEPA, IEPA takes final action on permit application

Public meetings or other communication between IEPA and stakeholders can occur any time throughout the permit process

CAAPP permits must be renewed every five years. Applications for renewals must be submitted at least 9 months in advanced of the current permit's expiration date.

CAAPP Permits, More to Know!

What is a CAAPP permit?

Under Title V of the 1990 Clean Air Act Amendments, any major source that has actual or potential emissions at or above the major source threshold for any major pollutant must receive a Title V permit. The USEPA authorizes states to administer these permits. In Illinois, this permit is a CAAPP permit.

What is a Major Source?

Major source thresholds are set by the Clean Air Act. For any air pollutant, 100 tons/year is the default, but lower thresholds apply in non-attainment areas (based on National Ambient Air Quality Standards). Major source thresholds for Hazardous Air Pollutants (HAP) are 10 tons/year for a single HAP or 25 tons/year for any combination of HAP. Sources with a FESOP must remain below all of these thresholds.

If a source has submitted an application in a timely manner, it can continue to operate under the conditions of the existing permit (permit shield). Additionally, it must comply with any new applicable requirements during the pending period of the application.

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ILLINOIS ENVIRONMENTAL PROTECTION AGENCY

OFFICE OF PUBLIC UTILITIES
CITY OF SPRINGFIELD, ILLINOIS
JAMES O. LANGFELDER, MAYOR

January 28, 2020

Stephanie Flowers
Illinois Environmental Protection Agency
1021 North Grand Avenue East
P.O. Box 19276
Springfield, IL 62794-9276

Re: Proposed changes to 35 Ill. Adm. Code Part 620

Dear Ms. Flowers,

I am writing on behalf of the City of Springfield, Office of Public Utilities -- also known as City Water, Light and Power (CWLP). CWLP appreciates the opportunity provided by the Illinois Environmental Protection Agency (Agency) to submit pre-filing comments on proposed changes to 35 Ill. Adm. Code Part 620. We have reviewed the proposed changes and noted a number of differences in the Class I and Class II groundwater quality standards in relation to the current standards and also in relation to the changes that were proposed in the last draft we are aware of from 2017. I've attached a chart summarizing those changes we noted as Attachment A.

Both CWLP's Electric Division and Water Division have interest in this proceeding and would like to provide meaningful input to the Agency. However, the rule language alone, without an accompanying reference to the sources of new toxicological information that are being relied on makes us unable to provide that input at this time.

We hope there will either be an opportunity for further input once that information is available to be shared with stakeholders or that the Agency establishes some type of process for the technical experts to explain the basis for the changes and respond to stakeholder questions or concerns prior to filing with the Pollution Control Board.

Thank you for keeping the public informed on the Agency's plans and we look forward to participating in this process as it develops. If you have questions or would like to provide additional background information, please feel free to contact me at deborah.williams@cwlp.com or 217-789-2116 ext. 2628.

Sincerely,

Deborah J. Williams

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ATTACHMENT A

Chemical	CLASS I				CLASS II			
	Current	Units	September 2017 draft	December 2019 draft	*Current*	Units	September 2017 draft	December 2019 draft
Inorganic								
Aluminum	n/a	mg/L	3.5	3.5	n/a	mg/L	5	5
Antimony	0.006	mg/L	0.006	0.006	0.024	mg/L	0.024	0.024
Arsenic*	0.01	mg/L	0.01	0.01	0.2	mg/L	0.2	0.2
Barium	2	mg/L	2	2	2	mg/L	2	2
Beryllium	0.004	mg/L	0.004	0.004	0.5	mg/L	0.5	0.5
Boron	2	mg/L	2	1.4	2	mg/L	2	2
Cadmium	0.005	mg/L	0.005	0.005	0.05	mg/L	0.05	0.05
Chloride	200	mg/L	200	200	200	mg/L	200	200
Chromium	0.1	mg/L	0.1	0.1	1	mg/L	1	1
Cobalt	1	mg/L	0.002	0.0021	1	mg/L	1	1
Copper	0.65	mg/L	0.2	0.5	0.65	mg/L	0.2	0.5
Cyanide	0.2	mg/L	0.2	0.2	0.6	mg/L	0.6	0.6
Fluoride	4	mg/L	4	2	4	mg/L	4	2
Iron	5	mg/L	5	5	5	mg/L	5	5
Lead	0.0075	mg/L	0.0075	0.0075	0.1	mg/L	0.1	0.1
Lithium		mg/L		0.014		mg/L		2.5
Manganese	0.15	mg/L	0.15	0.15	10	mg/L	10	10
Mercury	0.002	mg/L	0.002	0.002	0.01	mg/L	0.01	0.01
Molybdenum		mg/L		0.035		mg/L		0.05
Nickel	0.1	mg/L	0.1	0.1	2	mg/L	2	2
Nitrate as N	10	mg/L	10	10	100	mg/L	100	100
Perchlorate	0.0049	mg/L	0.0049	0.0049	0.0049	mg/L	0.0049	0.0049
Radium-226	20	pCi/l	5	n/a				

Radium-228	20	pCi/l	5	n/a				
Combined Radium	N/A	pCi/l	5	5		pCi/l		5
Selenium	0.05	mg/L	0.05	0.02	0.05	mg/L	0.05	0.02
Silver	0.05	mg/L	0.05	0.035		mg/L		0.035
Sulfate	400	mg/L	400	400	400	mg/L	400	400
Thallium	0.002	mg/L	0.002	0.002	0.02	mg/L	0.02	0.02
Total Dissolved Solids (TDS)	1,200	mg/L	1,200	1200	1200	mg/L	1200	1200
Vanadium	0.049	mg/L	0.00049	0.00049	0.1	mg/L	0.1	0.1
Zinc	5	mg/L	5	5	10	mg/L	10	10
	CLASS I				**CLASS II**			
	Current		September 2017 draft	December 2019 draft	Current		September 2017 draft	December 2019 draft
Organic								
Acenaphthene	0.42	mg/L	0.42	0.42	2.1	mg/L	2.1	2.1
Acetone	6.3	mg/L	6.3	6.3	6.3	mg/L	6.3	6.3
Alachlor*	0.002	mg/L	0.002	0.002	0.01	mg/L	0.01	0.002
Aldicarb	0.003	mg/L	0.003	0.003	0.015	mg/L	0.015	0.003
Anthracene	2.1	mg/L	2.1	2.1	10.5	mg/L	10.5	10.5
Atrazine	0.003	mg/L	0.003	n/a	0.015	mg/L	0.015	n/a
Benzene*	0.005	mg/L	0.005	0.005	0.025	mg/L	0.025	0.025
Benzo(a)anthracene*	0.00013	mg/L	0.00085	0.00085	0.00065	mg/L	0.0043	0.0043
Benzo(b)fluoranthene*	0.00018	mg/L	0.00085	0.00085	0.0009	mg/L	0.0043	0.0043
Benzo(k)fluoranthene*	0.00017	mg/L	0.0085	0.0085	0.006	mg/L	0.043	0.043
Benzo(a)pyrene*	0.0002	mg/L	0.0002	0.0002	0.002	mg/L	0.002	0.002
Benzoic acid	28	mg/L	28	28	28	mg/L	28	28
2-Butanone (MEK)	4.2	mg/L	4.2	4.2	4.2	mg/L	4.2	4.2
Carbofuran	0.04	mg/L	0.04	0.04	0.2	mg/L	0.2	0.04

Carbon Disulfide	0.7	mg/L	0.7	0.7	3.5	mg/L	n/a	n/a
Carbon Tetrachloride*	0.005	mg/L	0.005	0.005	0.025	mg/L	0.025	0.025
Chlordane*	0.002	mg/L	0.002	0.002	0.01	mg/L	0.01	0.01
Chlorobenzene	n/a	mg/L	0.1	0.1	n/a	mg/L	0.5	0.1
Chloroform*	0.07	mg/L	0.07	0.07	0.35	mg/L	0.35	0.35
Chrysene*	0.012	mg/L	0.085	0.085	0.06	mg/L	0.43	0.43
2,4-D	0.07	mg/L	0.07	0.07	n/a	mg/L	0.35	0.07
Dalapon	0.2	mg/L	0.2	0.2	2	mg/L	2	0.2
Dibenzo(a,h)anthracene*	0.0003	mg/L	0.000085	0.000085	0.0015	mg/L	0.00043	0.00043
1,2-Dibromo-3-Chloropropane*	0.0002	mg/L	0.0002	0.0002	0.002	mg/L	0.002	0.0002
Dicamba	0.21	mg/L	0.21	0.21	0.21	mg/L	0.21	0.21
ortho-Dichlorobenzene	0.6	mg/L	0.6	0.6	1.5	mg/L	1.5	0.6
para-Dichlorobenzene	0.075	mg/L	0.075	0.075	0.375	mg/L	0.375	0.075
Dichlorodifluoromethane	1.4	mg/L	1.4	1.4	7	mg/L	7	7
1,1-Dichloroethane	1.4	mg/L	1.4	1.4	7	mg/L	7	7
1,2-Dichloroethane*	0.005	mg/L	0.005	0.005	0.025	mg/L	0.025	0.005
1,1-Dichloroethylene	0.007	mg/L	0.007	0.007	0.035	mg/L	0.035	0.035
cis-1,2-Dichloroethylene	0.07	mg/L	0.07	0.07	0.2	mg/L	0.2	0.35
trans-1,2-Dichloroethylene	0.1	mg/L	0.1	0.1	0.5	mg/L	0.5	0.5
Dichloromethane (methylene chloride)*	0.005	mg/L	0.005	0.005	0.05	mg/L	0.05	0.005
1,2-Dichloropropane*	0.005	mg/L	0.005	0.005	0.025	mg/L	0.025	0.005
Di(2-ethylhexyl)phthalate*	0.006	mg/L	0.006	0.006	0.06	mg/L	0.06	0.06
Diethyl Phthalate	5.6	mg/L	5.6	5.6	5.6	mg/L	5.6	5.6
Di-n-butyl Phthalate	0.7	mg/L	0.7	0.7	3.5	mg/L	3.5	3.5
1,3-Dinitrobenzene	0.0007	mg/L	0.0007	0.0007	0.0007	mg/L	0.0007	0.0007
2,4-Dinitrotoluene*	0.0001	mg/L	0.00027	0.00027	0.0001	mg/L	0.0014	0.0014
2,6-Dinitrotoluene*	0.00031	mg/L	0.000057	0.000057	0.00031	mg/L	0.00028	0.00029
Dinoseb	0.007	mg/L	0.007	0.007	0.07	mg/L	0.07	0.07

1,4-Dioxane (p-dioxane)	n/a	mg/L	0.00085	0.00085	0.0077	mg/L	0.00085	0.00085
Endothall	0.1	mg/L	0.1	0.1	0.1	mg/L	0.1	0.1
Endrin	0.002	mg/L	0.002	0.002	0.01	mg/L	0.01	0.01
Ethylbenzene	0.7	mg/L	0.7	0.7	1	mg/L	1	3.5
Ethylene Dibromide*	0.00005	mg/L	0.00005	0.00005	0.0005	mg/L	0.0005	0.00005
Fluoranthene	0.28	mg/L	0.28	0.28	1.4	mg/L	1.4	1.4
Fluorene	0.28	mg/L	0.28	0.28	1.4	mg/L	1.4	1.4
Heptachlor*	0.0004	mg/L	0.0004	0.0004	0.002	mg/L	0.002	0.002
Heptachlor Epoxide*	0.0002	mg/L	0.0002	0.0002	0.001	mg/L	0.001	0.001
Hexachlorocyclohexane, alpha-*	n/a	mg/L	0.000014	0.000014	n/a	mg/L	0.00007	0.00007
Hexachlorocyclohexane, gamma (Lindane)	0.0002	mg/L	0.0002	0.0002	0.001	mg/L	0.001	0.001
Hexachlorocyclopentadiene	0.05	mg/L	0.05	0.05	0.5	mg/L	0.5	0.5
HMX (High Melting Explosive, Octogen)	1.4	mg/L		1.4	1.4	mg/L	7	7
Indeno(1,2,3-cd)pyrene*	0.00043	mg/L	0.00085	0.00085	0.0022	mg/L	0.0043	0.0043
Isopropylbenzene (Cumene)	0.7	mg/L	0.7	0.7	3.5	mg/L	3.5	3.5
MCPP (Mecoprop)	0.007	mg/L	0.007	0.007	0.007	mg/L	0.007	0.007
Methoxychlor	0.04	mg/L	0.04	0.04	0.2	mg/L	0.2	0.2
1-Methylnaphthalene	n/a	mg/L	0.49	0.49	n/a	mg/L	2.4	2.5
2-Methylnaphthalene	0.028	mg/L	0.028	0.028	0.14	mg/L	0.14	0.14
2-Methylphenol (o-cresol)	0.35	mg/L	0.35	0.35	0.35	mg/L	0.35	0.35
Methyl Tertiary-Butyl Ether (MTBE)	0.07	mg/L	0.07	0.07	0.07	mg/L	0.07	0.07
Monochlorobenzene	0.1	mg/L	n/a	n/a	0.5	mg/L	n/a	n/a
Naphthalene	0.14	mg/L	0.14	0.14	0.22	mg/L	0.22	0.22
Nitrobenzene	0.014	mg/L		0.014	0.014	mg/L		0.014
P-Dioxane*	0.0077	mg/L	n/a	n/a	0.0077	mg/L	n/a	n/a

Pentachlorophenol*	0.001	mg/L	0.001	0.001	0.005	mg/L	0.005	0.005
Perfluorobutane Sulfonic Acid (PFBS)	n/a	mg/L	n/a	0.14	n/a	mg/L	n/a	0.14
Perfluorohexane Sulfonic Acid (PFHxS)	n/a	mg/L	n/a	0.00014	n/a	mg/L	n/a	0.00014
Perfluorononanoic Acid (PFNA)	n/a	mg/L	n/a	0.000021	n/a	mg/L	n/a	0.000021
Phenols	0.1	mg/L	0.1	0.1	0.1	mg/L	0.1	0.1
Picloram	0.5	mg/L	0.5	0.5	5	mg/L	5	0.5
Polychlorinated Biphenyls (PCBs) (as decachloro-biphenyl)*	0.0005	mg/L	0.0005	0.0005	0.0025	mg/L	0.0025	0.0025
alpha-BHC (alpha-Benzene hexachloride)*	0.00011	mg/L	n/a	n/a	0.00055	mg/L	n/a	n/a
Pyrene	0.21	mg/L	0.21	0.21	1.05	mg/L	1.05	1.05
RDX (Royal Demolition Explosive, Cyclonite)	0.084	mg/L	0.084	0.07		mg/L	0.084	0.07
Simazine	0.004	mg/L	0.004	0.004	0.04	mg/L	0.04	0.004
Styrene	0.1	mg/L	0.1	0.1	0.5	mg/L	0.5	0.1
2,4,5-TP (Silvex)	0.05	mg/L	0.05	0.05	0.25	mg/L	0.25	0.05
Tetrachloroethylene*	0.005	mg/L	0.005	0.005	0.025	mg/L	0.025	0.025
Toluene	1	mg/L	1	1	2.5	mg/L	2.5	5
Toxaphene*	0.003	mg/L	0.003	0.003	0.015	mg/L	0.015	0.015
1,2,4 Trichlorobenzene	0.07	mg/L	0.07	0.07	0.7	mg/L	0.7	0.7
1,1,1-Trichloroethane	0.2	mg/L	0.2	0.2	1	mg/L	1	1
1,1,2-Trichloroethane	0.005	mg/L	0.005	0.005	0.05	mg/L	0.05	0.005
Trichloroethylene*	0.005	mg/L	0.005	0.005	0.025	mg/L	0.025	0.025
Trichlorofluoromethane	2.1	mg/L	2.1	2.1	10.5	mg/L	10.5	10.5
1,3,5-Trinitrobenzene	0.84	mg/L	0.84	0.84	0.084	mg/L	4.2	4.2
2,4,6-Trinitrotoluene (TNT)	0.014	mg/L	0.014	0.014	0.84	mg/L	0.07	0.07
Vinyl Chloride*	0.002	mg/L	0.002	0.002	0.01	mg/L	0.01	0.01
Xylenes	10	mg/L	10	10	10	mg/L	10	10

	CLASS I				CLASS II			
	Current		September 2017 draft	December 2019 draft	Current		September 2017 draft	December 2019 draft
Explosive Constituents - this section consolidated with organics								
1,3-Dinitrobenzene	0.0007	mg/L						
2,4-Dinitrotoluene*	0.0001	mg/L			0.0001	mg/L		
2,6-Dinitrotoluene*	0.00031	mg/L			0.00031	mg/L		
HMX (High Melting Explosive, Octogen)	1.4	mg/L		1.4	1.4	mg/L	7	
Nitrobenzene	0.014	mg/L			0.014	mg/L		
RDX (Royal Demolition Explosive, Cyclonite)	0.084	mg/L		0.07		mg/L	0.084	
1,3,5-Trinitrobenzene	0.84	mg/L			0.084	mg/L	4.2	
2,4,6-Trinitrotoluene (TNT)	0.014	mg/L			0.84	mg/L	0.07	
Complex Organic Chemical Mixtures								
Benzene*	0.005	mg/L	0.005	0.005	0.025	mg/L	0.025	0.025
BETX	11.705	mg/L	11.705	11.705	13.525	mg/L	13.525	18.525
Total Atrazine and metabolites: *Atrazine Desethyl-atrazine (DEA) Desisopropyl-atrazine (DIA) Diaminochlorotriazine (DACT)*	n/a	mg/L	0.003	0.003	0.015	mg/L	0.015	0.003
Perfluorooctanoic Acid (PFOA)	n/a	mg/L	0.000007	0.000021	n/a	mg/L	0.00007	0.000021
Perfluorooctyl Sulfonate (PFOS)	n/a	mg/L	0.000007	0.000014	n/a	mg/L		0.000014
Beta Particle & Photon Radioactivity								
Tritium (Total body)	20,000.00	(pCi/L)	20,000.00	20,000.00	n/a		n/a	n/a
Strontium-90 (Bone marrow)	8	(pCi/L)	8	8	n/a		n/a	n/a

Comments of Dynegy Midwest Generation, LLC; Kincaid Generation, LLC; Illinois Power Resources Generating Company; Illinois Power Generating Company; and Electric Energy Inc.

Submitted to

The Illinois Environmental Protection Agency

February 28, 2020

Dynegy Midwest Generation, LLC; Kincaid Generation, LLC; Illinois Power Resources Generating, LLC; Illinois Power Generating Company; and Electric Energy Inc. (collectively, "Dynegy") submit these comments in response to the Illinois Environmental Protection Agency's ("IEPA" or the "Agency") request for input regarding the Agency's December 2019 draft proposed changes to the language of 35 Ill. Admin. Code 620: Groundwater Quality ("Part 620 Draft"). These comments address several concerns Dynegy has regarding a subset of the proposed Class I and II groundwater standards in §§ 620.410 and 620.420 of the Part 620 Draft. As explained below, Dynegy believes these standards should take into account background levels when appropriate, use realistic and data-backed assumptions (particularly for relative source contribution), be consistent with United States Environmental Protection Agency ("USEPA") maximum contaminant level goals ("MCLGs") and/or regional screening levels ("RSLs"), and take into account risk profiles as supported in various literature.

Dynegy appreciates IEPA's efforts in crafting the Part 620 Draft and looks forward to the Agency's consideration of these comments prior to finalizing its proposal.

I. Boron

The draft proposed Class I standard of 1.4 mg/L for boron is too low given existing federal standards and guidance that are protective of human health and the environment. Under the Safe Drinking Water Act ("SDWA"), USEPA sets primary standards, called MCLGs, for contaminants which "may have an adverse effect on the health of persons" and secondary standards for contaminants at levels "requisite to protect the public welfare." 42 U.S.C. § 300f(1),(2). Notably, USEPA has not established primary or secondary drinking water standards for boron.[1] Additionally, USEPA derives RSLs to serve as guidance for and assist with initial screening and risk assessment for contaminants at CERCLA remediation sites. RSLs are risk-based concentrations and are set at levels intended to be protective of human health, including sensitive populations, over a lifetime.[2] USEPA has derived an RSL for boron in drinking water of 4.0 mg/L.[3] Further, USEPA's current life-time health advisory for boron is 6 mg/L.[4]

Moreover, in crafting the draft proposed boron standard, the relative source contribution assigned by IEPA to boron from drinking water is too low. It appears the Agency calculated the standard by using USEPA's Integrated Risk Information System ("IRIS") reference dose for boron and applying a relative source contribution of 20%. This assumes that drinking water makes up 20% of a person's boron intake and that other (namely, dietary) sources make up 80% of a person's

[1] USEPA, National Drinking Water Reference Table, available at https://www.epa.gov/sites/production/files/2016-06/documents/npwdr_complete_table.pdf.

[2] *See* https://www.epa.gov/risk/regional-screening-levels-frequent-questions; *See also*, USEPA, Regional Screening Levels - Users Guide, November 2019, available at https://www.epa.gov/risk/regional-screening-levels-rsls-users-guide#intro.

[3] USEPA, Regional Screening Levels – Generic Tables, November 2019, available at https://www.epa.gov/risk/regional-screening-levels-rsls-generic-tables.

[4] USEPA, 2018 Edition of the Drinking Water Standards and Health Advisories Tables, available at https://www.epa.gov/sites/production/files/2018-03/documents/dwtable2018.pdf

boron intake. IEPA should consider data and other information that more accurately reflects exposure and supports applying a higher relative source contribution percentage when calculating a Class I standard for boron. By way of example, USEPA assigned a relative source contribution of 80% to drinking water when calculating its life-time health advisory level for boron.[5] As another example, using IEPA's calculation methodology for acceptable daily intake in 35 Ill. Admin. Code Section 620.Appendix A, acceptable daily intake of boron is 14 mg. Data from the Agency for Toxic Substances and Disease Registry ("ATSDR") suggests that mean daily intake of boron for male and female adults is 1.28 mg and 1.0 mg, respectively.[6] Even assuming the entirety of that intake is from dietary sources (which it is not[7]), this data supports approximately 9% (1.28mg/14mg) of a person's boron intake coming from dietary sources, suggesting 91% of the daily intake of 14 mg can come from drinking water.

II. Cobalt

The draft proposed Class I standard of 0.002 mg/L for cobalt is also too low given that it is not in line with federal standards or guidance. USEPA has not issued a standard for cobalt under the SDWA,[8] and IEPA's proposal is lower than the drinking water RSL for cobalt, which is 0.006 mg/L.[9]

Additionally, IEPA should consider the impact of background levels and detection limits for cobalt. Setting a standard close to or below background levels or detection limits can be impractical for many reasons, including for purposes of assigning responsibility and conducting remediation. Illinois groundwater data from the USGS National Water Information System[10] demonstrates that all of the samples from the database tested for "total" cobalt detected cobalt at levels above 0.002 mg/L or had detection limits that were above 0.002 mg/L. Further, approximately 32% of filtered cobalt samples from the database had detections above 0.002 mg/L or detection limits above 0.002 mg/L.

Furthermore, IEPA should reconsider certain assumptions that appear to have been used to calculate the proposed cobalt standard. IEPA appears to have used a chronic oral reference dose for cobalt of 0.0003 mg/kg-day, derived by USEPA as a Provisional Peer Reviewed Toxicity Value.[11] However, other studies and sources have concluded that a more accurate reference dose

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[5] USEPA, Drinking Water Health Advisory for Boron, May 2008, available at https://www.epa.gov/sites/production/files/2014-09/documents/drinking_water_health_advisory_for_boron.pdf.

[6] ATSDR, Toxicological Profile for Boron, November 2010, available at https://www.atsdr.cdc.gov/toxprofiles/tp26.pdf.

[7] Id.

[8] USEPA, National Drinking Water Reference Table, available at https://www.epa.gov/sites/production/files/2016-06/documents/npwdr_complete_table.pdf

[9] USEPA, Regional Screening Levels – Generic Tables, November 2019, available at https://www.epa.gov/risk/regional-screening-levels-rsls-generic-tables.

[10] USGS Groundwater Data for the Nation, available at https://waterdata.usgs.gov/nwis/gw.

[11] USEPA, the Provisional Peer Reviewed Toxicity Values (PPRTV) for Cobalt, 2008, available at https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=338894

for cobalt is higher, presenting values that are up to 100 times higher.[12] IEPA also appears to have used a relative source contribution of 20% when calculating its proposed cobalt standard. Here again, IEPA should consider available information and data that will result in a standard that accurately reflects exposure, rather than relying upon a default 20% contribution value. For example, ATSDR data suggests the average person consumes about 0.011 mg/day of cobalt from dietary sources.[13] Using IEPA's calculation methodology for acceptable daily intake in 35 Ill. Admin. Code Section 620.Appendix A, acceptable daily intake of cobalt is 0.021 mg/day. This suggests dietary sources make up only approximately half of the acceptable daily intake of cobalt (0.011mg/0.021mg), supporting a relative source contribution from drinking water that is much higher than 20%.

III. Lithium

The draft proposed Class I standard of 0.014 mg/L for Lithium is too low due to the fact that USEPA has not set a standard for lithium under the SDWA,[14] it has not issued a health advisory or developed an IRIS value for lithium, and the drinking water RSL for lithium is 0.04 mg/L.[15] Additionally, for the same reasons discussed above, IEPA should take into account background levels of lithium in connection with any proposed lithium standards. Illinois groundwater data for filtered samples from the USGS National Water Information System[16] demonstrates that a significant amount of the 434 samples tested, approximately 33%, exceeded the proposed 0.014 mg/L standard.

IV. Molybdenum

The draft proposed Class I and II standards for molybdenum are too low. For comparison, USEPA has not set a standard for molybdenum under the SDWA and both standards are more stringent than the drinking water RSL for molybdenum, which is 0.1 mg/L.[17] Again, IEPA appears to have used a conservative relative source contribution of 20% when calculating the proposed 0.035 mg/L standard for molybdenum. Dynegy recommends using available information and data that more accurately reflects exposure to molybdenum. For example, the ASTDR notes that adults

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[12] Finley, BL, Monnot, AD, Paustenbach, DJ, & Gaffney, SH. 2012. Derivation of a chronic oral reference dose for cobalt. Regulatory Toxicology and Pharmacology. 64(3):491–503; European Food Safety Authority (EFSA). 2009. Scientific opinion: Assessment of the safety of cobalt(II) chloride hexahydrate added for nutritional purposes as a source of cobalt in food supplements and the bioavailability of cobalt from this source. The EFSA Journal. 1066:1-8.

[13] ATSDR, Toxicological Profile for Cobalt, April 2004, available at https://www.atsdr.cdc.gov/ToxProfiles/tp33.pdf.

[14] USEPA, National Drinking Water Reference Table, available at https://www.epa.gov/sites/production/files/2016-06/documents/npwdr_complete_table.pdf

[15] USEPA, Regional Screening Levels – Generic Tables, November 2019, available at https://www.epa.gov/risk/regional-screening-levels-rsls-generic-tables.

[16] USGS Groundwater Data for the Nation, available at https://waterdata.usgs.gov/nwis/gw.

[17] USEPA, Regional Screening Levels – Generic Tables, November 2019, available at https://www.epa.gov/risk/regional-screening-levels-rsls-generic-tables.

in the United States ingest on average 0.076 to 0.109 mg of molybdenum per day.[18] Using IEPA's calculation methodology for acceptable daily intake in 35 Ill. Admin. Code Section 620.Appendix A, acceptable daily intake of molybdenum is 0.35 mg/day. Thus, ASTDR's average daily dietary intake estimates make up only approximately 30% of this acceptable daily intake, supporting a much higher relative source contribution for drinking water.

V. Fluoride and Selenium Standards

The draft proposed Class I and II fluoride and selenium standards are too low. Both sets of standards are lower than the respective MCLGs for fluoride and selenium. Appendix A of Part 620 suggests that IEPA should follow the MCLGs when setting Class I standards, employing the calculation methodology set forth in Part 620 only for "those substances for which USEPA has not adopted an MCLG." 35 Ill. Admin. Code Section 620.Appendix A(a). The MCLG for fluoride is 4.0 mg/L, as opposed to the proposed 2.0 mg/L standard.[19] The MCLG for selenium is 0.05 mg/L, as opposed to the proposed 0.02 mg/L standard.[20]

[18] ATSDR, Toxicological Profile for Molybdenum, April 2017, available at https://www.atsdr.cdc.gov/ToxProfiles/tp212.pdf

[19] USEPA, 2018 Edition of the Drinking Water Standards and Health Advisories Tables, available at https://www.epa.gov/sites/production/files/2018-03/documents/dwtable2018.pdf.

[20] Id.

eurofins | Environment Testing TestAmerica

01/31/2020

Stephanie Flowers
Illinois Environmental Protection Agency
1021 North Grand Avenue East
PO Box 19276
Springfield, IL 62794-9276

Dear Ms. Flowers:

Eurofins TestAmerica is a network of environmental testing laboratories with 21 locations within the US, including one in Chicago IL. Several of our laboratories outside Illinois analyze samples from clients operating within Illinois.

As invited in your letter of December 24, we are commenting on proposed changes to the language of 35Ill. Adm. Code 620: Groundwater Quality. Please feel free to contact me if you have any questions regarding our comments.

Sincerely.

Richard Burrows, Ph.D.

Corporate Technical Director

Richard.burrows@testamericainc.com

(303)885-3348

eurofins | Environment Testing TestAmerica

Eurofins TestAmerica comments on proposed changes to the language of 35Ill. Adm. Code 620: Groundwater Quality.

We are primarily concerned regarding item 1) c., "Eliminate practical quantitation limits (PQLs) as standards due to updated analytical methodologies."

Historically, compliance limits have been set as the higher of (i) the health-based limit and (ii) the PQL, with the PQL representing the lowest quantitation level that could be reliably measured within specified limits of precision and accuracy during routine laboratory operating conditions.

Elimination of the PQL from consideration clearly results in at least the possibility that a compliance limit could be set that is below the level at which a well performing laboratory can actually measure the contaminant within reasonable limits of precision and accuracy.

Illinois EPA appears to believe that advancements in analytical measurement technology means that in all cases, laboratories are able to meet health-based limits under routine operating conditions, and therefore, use of the PQL is no longer relevant.

Relevant EPA methods (for example methods 608/8081, 624/8260, 65/8270 and 200.8/6020) have certainly been updated several times in the last 20 years. However, in virtually all cases, the fundamental measurement technology remains the same. For example, method 8081, for the analysis of chlorinated pesticides, utilized gas chromatography with electron capture detection 30 years ago, and continues to utilize gas chromatography with electron capture detection to this day. As a result, PQLs have not really changed that much. A casual perusal of the scientific literature will reveal that various methods are available that can achieve much lower PQLs than method 8082 (for example triple quadrupole negative chemical ionization GCMS) – unfortunately these are not EPA methods and therefore not routinely available for compliance monitoring.

It is also possible that health-based limits will change in the future, potentially lowering below levels at which labs can routinely meet reasonable precision and accuracy limits.

There are also a few compounds that will require adoption of non-standard methods to meet the compliance limits listed for Class 1 groundwater. For example, 2,6-dinitrotoluene, limit 0.000057 mg/L. This is 2 orders of magnitude below our routine quantitation limit using method 8270 or 625. We can achieve the limit by using LC/MS/MS technology, but this is available at a relatively limited number of laboratories. We also note that the limit for vanadium in Class 1 groundwater has been reduced by a factor of 100 (to 0.00049 mg/L) but is unchanged in class 2 groundwater. There is now a 200X difference in compliance level between vanadium in Class 1 and class 2 groundwaters, much greater than any other metal. A limit if 0.00049 mg/L is achievable by ICP/MS, but the analyte is subject to interferences that will probably result in false positives at this low level.

We therefore suggest that IEPA retain use of PQL in setting compliance limits. If IEPA believes that lower PQLs are now possible, a good start would be to survey Illinois laboratories for their current quantitation limits. Even better would be to evaluate the levels of precision and accuracy that the laboratories are achieving at these limits.

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The topic of quantitation is critical to the application of the Part 620 rules because measurements are used in statistical evaluations and in comparison to numeric standards; and, both of these activities presume that the measurement results are of known and controlled precision and bias. A review of several state programs has revealed varying degrees to which agencies attempt to meet the requirements of 40 Code of Federal Regulation (CFR) Section (§)258.53(h)(5) that requires "any practical quantitation limit (PQL) that is used in the statistical method shall be the lowest concentration level that can be reliably achieved within specified limits of precision and accuracy during routine laboratory operating conditions that are available to the facility". The state of Texas appears to have applied the most rigorous scientific approach to establishing acceptable limits of quantitation for its monitoring programs (https://www.tceq.texas.gov/assets/public/permitting/waste/msw/msw-pqls.pdf). TCEQ's objectives were to develop a mechanism to implement the rule in a way that was protective of the human health and the environment, require quality standards for data that they receive, and establish benchmarks reflective of the capabilities of the commercial laboratories available to the regulated community. TCEQ requested laboratories, which routinely generate monitoring data submitted by regulated parties to the TCEQ, participate in an inter-laboratory study to collect data for target analytes at various concentrations using the commonly referenced methods from EPA SW846. After the data collection process was complete, TCEQ applied the Inter-Laboratory Quantitation Estimate (IQE) Standard (ASTM D6512) statistical process to the data to arrive at "benchmark" quantitation limits. During this process TCEQ established expected quality requirements for precision (in the form of %RSD) and accuracy (in the form of %recovery) for each class of analytes and introduced these quality requirements into the facility permits. This study demonstrated what levels of quantitation the commercial laboratories available to the regulated parties were able to achieve. The TCEQ subsequently published the results of the study and the "benchmark" quantitation limits that the regulated parties were expected to achieve.

ILLINOIS AMERICAN WATER

Randolph Pankiewicz
Manager Water Quality &
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Illinois American Water
800 N Front Street
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February 26, 2020

Sara G. Terranova
Assistant Counsel
Division of Legal Counsel
Illinois Environmental Protection Agency
1021 North Grand Avenue East, P.O. Box 19276
Springfield, Il 62794-9276

RE: 35 Ill. Adm. Code Part 620 - Outreach Meeting

Dear Ms.Terranova,

Illinois American Water Company (ILAWC) would like to offer the following comments to the proposed part 620 regulations presented at the outreach meeting held in Springfield on February 13, 2020.

- Protection of the groundwater resources of Illinois is of critical importance for the future, as these resources are limited and provide for the potable water of many communities within the state.
- In determining the values of the parameters to be regulated, the best verified and accepted scientific information currently available should be used.
- The federal EPA is in the process of determining appropriate drinking water MCLs for the PFAS chemicals. These MCLs should be considered in the development of the groundwater standards.
- Protection of our customers requires consideration of the impact on the water that will be available for potable water from both a health and cost perspective. If drinking water limits are set too far below health levels, this will increase costs associated with all sources and may prohibit the use of a given, needed, source.
- There was some discussion of potential future application of standards for PFAS chemicals in wastewater treatment plant effluents and biosolids and drinking water residuals. ILAW feels that more research is needed in determining at what levels any impact may occur from these sources on groundwater or soils.
- In general ILAW will work with you and the federal EPA to provide the guidance on what limits should be considered for setting limits for the PFAS group of chemicals.

If you would like to discuss any of these comments please reach out to me.

Sincerely,

Randolph Pankiewicz

Cc: Elizabeth Matthews
Sean Flynn

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Illinois Environmental Regulatory Group
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February 28, 2020

Sara G. Terranova
Assistant Counsel
Division of Legal Counsel
Illinois Environmental Protection Agency
1021 North Grand Avenue East, P.O. Box 19276
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Submitted by email to:
Sara.Terranova@illinois.gov

Ms. Terranova:

Please accept the below comments on behalf of the members of the Illinois Environmental Regulatory Group ("IERG") regarding the Agency's proposed changes to the language of 35 Ill. Adm. Code Part 620: Groundwater Quality, that were shared on December 24, 2019 ("the Dec. 2019 language") and which were the subject of the Agency's February 13, 2020 Stakeholders Meeting. IERG participated in that meeting and appreciates the Agency's efforts to address questions raised, however some concerns and questions remain.

It was clear that there were numerous participants at the February 13th meeting that had questions regarding the Agency's intent for implementation of the draft standards that were unanswered. IERG is generally concerned regarding how the new standards will be implemented, including, but not limited to, the potential impact on previously remediated sites, sites in various stages of ongoing remediation, and the potential to negatively impact beneficial reuse of coal combustion residuals.

Additionally, IERG is in receipt of the Agency's *Illinois EPA Tables Describing the Basis for Developing the Proposed Class I and Class II Groundwater Quality Standards (35 Ill. Admin. Code 620.410 and 620.420)* that was distributed to stakeholders on February 25, 2020. IERG appreciates the Agency providing this information, but its review of the document has only just begun and meaningful input cannot be provided in this comment in accordance with your established February 28 deadline.

Once IERG has had an opportunity to review the additional information described above, I hereby request an opportunity to meet with the appropriate Agency staff to discuss the findings of that review and IERG's and other stakeholders' concerns regarding implementation. I look forward to your response.

Sincerely,

Alec Davis
Executive Director

cc: Sanjay Sofat

February 28, 2020

Sent via email to sara.terranova@illinois.gov

Sara Terranova
1021 North Grand Ave East
P.O. Box 19276
Springfield, IL 62794-9276

Re: Proposed changes to 35 Ill. Adm. Code 620: Groundwater Quality

Dear Ms. Terranova,

We the signers, applaud the efforts by the Illinois EPA to set enforceable groundwater standards for PFAS chemicals, which will be necessary for identifying and cleaning up contaminated groundwater resources in the state. However we are very concerned that the proposed groundwater standards are not strong enough to fully protect human health when it is used for human consumption. Several states have set more protective water standards for PFAS by considering the special vulnerability to PFAS exposure during gestation and infancy, and by basing risk evaluations on the most sensitive target organs like the mammary gland. Other states have also accounted for increased ingestion of drinking water during pregnancy, and the fact that infants are exposed to PFAS via sources other than water used to mix baby formula.

We urge Illinois to ensure that Illinois groundwater be regulated at levels protective enough to ensure that women and children could safely drink this water without any risk of harmful effects from PFAS, particularly the risk calculations for PFHxS and PFBS. The state should also account for the strong evidence of additive and synergistic effects of exposure to mixtures of PFAS chemicals by setting additional standards for groups of PFAS chemicals that share similar toxicological targets.

This request is consistent with Illinois' nondegradation provisions for groundwater found at 35 Ill. Adm. Code 620.301 that prohibit the release of any contaminant to a resource groundwater such that: 1) Treatment or additional treatment is necessary to continue an existing use or to assure a potential use of such groundwater; or 2) An existing or potential use of such groundwater is precluded.

Recommendation 1 - Groundwater standards should ensure that water is safe for ingestion by women, infants and children

Because of widespread contamination of food and drinking water, most Americans have measurable levels of PFAS in their bodies from the early stages of pregnancy onward. The developing fetus and infant have more intense exposure than adults and are also more sensitive to the harms caused by PFAS.[1] Compounding the issue of increased exposure, fetuses, infants, and children are also more vulnerable to exposure-related health effects than adults. The young may be more sensitive to the effects of PFAS due to their immature, developing biological systems (such as the immune system), and rapid body growth during development.[2] For example, exposure to PFAS before birth and/or in early childhood may result in decreased birthweight, decreased immune responses, and hormonal effects later in life.[3]

Decisions made when developing a health benchmark, such as evaluation of data gaps, the selection of uncertainty factors, and choice of exposure parameters to use, should be made to be protective of the most vulnerable populations, particularly developing fetuses, infants, and children. In fact, the National Academy of Sciences (NAS) has recommended the use of an additional uncertainty factor of 10 to ensure protection of fetuses, infants and children who often are not sufficiently protected from toxic chemicals such as pesticides by the traditional intraspecies (human variability) uncertainty factor.[4] Congress adopted this requirement in the Food Quality Protection Act for pesticides in foods.[5] **Considering the many health effects linked to PFAS that affect this vulnerable population and the substantial data gaps on exposure and toxicity of these compounds in complex mixtures, we recommend the use of this uncertainty factor when deriving health-protective benchmarks for PFAS.**

Furthermore, Illinois states that its proposed levels, that use adult exposure assumptions, are also protective of infants. However, that assumption is based on the use of a 100% relative sources contribution. That means that Illinois is assuming that 100% of infant exposure comes from tap water. However infants may also ingest PFAS in maternal milk and have significant exposure through oral and dermal contact with

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[1] Goeden HM, et al., 2019.

[2] Landrigan P and Goldman L, 2011. Children's Vulnerability to Toxic Chemicals: A Challenge and Opportunity to Strengthen Health and Environmental Policy. *Health Affairs* 30(5):842-850.

[3] Kristen M. Rappazzo et al., 2017. Exposure to Perfluorinated Alkyl Substances and Health Outcomes in Children: A Systematic Review of the Epidemiologic Literature, *Int J Environ Res Public Health* 14(7):691.

[4] National Academy of Sciences (NAS), 1993. Pesticides in the Diets of Infants and Children, National Research Council. https://www.nap.edu/catalog/2126/pesticides-in-the-diets-of-infants-and-children.

[5] Food Quality Protection Act for pesticides in foods. 21 U.S.C. § 346a(b)(2)(C)(ii)(II).

PFAS-treated items like carpeting and other textiles.[6] For example, researchers estimated exposure to PFOA and PFOS from hand-to-mouth transfer from treated carpets to be 40–60% of the total uptake in infants, toddlers, and children.[7] **In order to protect this more vulnerable group we recommend both using infant exposure assumptions and lowering the relative source contribution from water to at least 50%, if not to the more conservative 20%.**

Recommendation 2 - Illinois should consider the additive effects of individual chemicals on human health

Illinois EPA should account for the fact that people are exposed to complex mixtures of PFAS daily in their food, water and consumer products.

Biomonitoring studies demonstrate that Americans have chronic exposure to multiple PFAS chemicals throughout their lifetimes. CDC's NHANES studies reveal that nearly every American has PFOS, PFOA, PFHxS and PFNA detected in their bloodstream, including young children.[8] At least seven other compounds are detected by NHANES studies: MeFOSAA, PFDeA, PFUA, PFHpA, PFBS, FOSA, EtFOSAA and PFDoA. Most other PFAS chemicals are not routinely included in biomonitoring studies.

Those PFAS chemicals that have been studied for their toxicological impacts share many similar targets, including harms to reproduction and development, the immune and endocrine systems, and liver, blood and lipids. The Natural Resources Defense Council compiled information about the potential additive effects of the better-studied PFAS chemicals from the ATSDR draft assessment of PFAS.[9]

Table 1: Summary of potential additive effects of PFAS[8]

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[6] Gyllenhammar K, et al., 2018. Perfluoroalkyl Acids (PFAAs) in serum from 2-4-month-old infants: Influence of maternal serum concentration, gestational age, breast-feeding, and contaminated drinking water. *Environ Sci Technol* 52:7101-7110; Llorca M, et al., 2010. Infant exposure of perfluorinated compounds: levels in breast milk and commercial baby food. *Environ Int* 36(6):584-592.

[7] Trudel D, et al., 2008. Estimating consumer exposure to PFOS and PFOA. Risk Anal, 28(2), 251-269.

[8] *Environ. Sci. Technol.* 2007, 41, 7, 2237-2242. https://doi.org/10.1021/es062686m

[9] Reade A, et al., 2019. NRDC Report: Scientific and Policy Assessment for Addressing Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water. https://bit.ly/2LN1T4f citing Agency for Toxic Substances and Disease Registry (ATSDR), 2018. Toxicological Profile for Perfluoroalkyls: Draft for Public Comment (June 2018). https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf

	Immune system	Reproduction and development	Lipids	Liver	Endocrine sysemt	Body Weight	Blood
PFOA	x	x	x	x	x	x	x
PFOS	x	x	x	x	x	x	x
PFHxS	x			x			x
PFNA	x		x			x	
PFDeA	x	x	x	x	x	x	
PFDoA	x	x				x	
PFUA	x	x				x	x
PFHxA		x					x
PFBA		x		x	x		x
PFBS				x			x

In recognition of the potential for additive effects of multiple PFAS chemicals in water, several states have proposed or enacted water standards that address the sum total of several PFAS chemicals.

Table 2: Many states have set more health protective screening levels for multiple PFAS chemicals in groundwater

State	Water standard	Chemical	Value
Vermont	Ground and drinking water	PFOS + PFOA + PFNA+ PFHxS + PFHpA	20 ppt
Massachusetts	Groundwater (*proposed*)	Sum of PFOA + PFOS + PFNA + PFHxS + PFHpA + PFDA	20 ppt

Wisconsin	Groundwater (*proposed*)	PFOS + PFOA	20 ppt

Illinois EPA should explore this route by **adding a group groundwater standard for the sum of all quantifiable PFAS due to shared toxicity targets**. We propose this be in addition to its values for individual chemicals.

Recommendation 3 - Illinois EPA must reduce its recommended groundwater standard level fo PFHxS

The 6-carbon chain fluorosulfonate PFHxS is still legally added to scores of consumer products and industrial processes. As a result it poses a serious threat to groundwater. PFHxS shares many toxicological properties to the longer-chain chemicals (PFOS and PFOA) that have been voluntarily withdrawn from commerce. It is recommended for full phase out in the UN's Stockholm Convention on Persistent Organic Pollutants.

Several states have proposed or set more protective standards for PFHxS in ground and drinking water. These levels are justified by additional modeling of data or safety factors to address the vulnerability of the developing fetus and breastfed infant. We urge the Illinois EPA to ensure that groundwater can safely be consumed and reduce its proposed standards for PFHxS.

Table 3 Many states have proposed or enacted more protective standards for PFHxS

State	**Water standard**	**Value**
Minnesota	Groundwater (*proposed*)	47 ppt
New Hampshire	Ground and drinking water	18 ppt
Massachusetts	Groundwater value	20 ppt
Vermont	Groundwater value	20 ppt
Michigan	Drinking water (*proposed*)	51 ppt

Recommendation 4: Illinois EPA must reduce its recommended groundwater standard level for PFBS.

PFBS is a replacement chemical for PFOS and remains in widespread use. It is used in household products like carpeting and carpet cleaners, floor wax and car wax as well as

food packaging. PFBS is more mobile in ground water meaning that it travels further and faster than longer-chain compounds.

In 2018 EPA's Office of Research and Development released a draft risk assessment for PFBS which calculated four different reference doses for PFBS based on the length of exposure. The values range from 10 to 100 ng/kg-day. NGO comments[10] identified significant shortcomings in the draft risk assessment, including an insufficient margin of safety when considering the major gaps in our understanding of PFBS' toxicological properties. EPA failed to adequately reflect the uncertainty about low dose toxicity of PFBS and proposed a surprisingly low uncertainty factor of 3 to account for the lack of thorough toxicity testing, yet the database for PFBS is quite limited and does not have robust data for critical endpoints including developmental impacts to the immune and neurological systems. Furthermore, the EPA used the Body Weight3/4 allometric scaling approach to calculate a human equivalent oral dose from an animal-based point of departure. The Body Weight3/4 allometric scaling approach is based on body surface area and basal metabolic rate in adults. Importantly, EPA stated in the draft assessment that the Body Weight3/4 approach is not suitable for estimating an equivalent dose in infants and children. EPA derived RfD based on kidney effects in adult rats and thyroid effects in newborn mice. Given the lack of toxicokinetic information available in humans, rats, and mice, especially at different life points, it is unclear how appropriate the default Body Weight3/4 scaling approach is for estimating human equivalent doses. This uncertainty should at minimum be acknowledged, though it would be more health protective if Illinois explored alternative approaches to extrapolating from animal to human doses that better take into account the significant differences in elimination rates between animals in humans generally seen for PFAS chemicals.

This importance of fully addressing uncertainty and accounting for the differences in toxicokinetics between animals and humans is demonstrated by examining how Michigan derived its significantly lower proposed MCL for PFBS of 420 ppt.[11]The dose adjustment factor Michigan used for PFBS was based on the ratio of human to animal half-lives for PFBS, not the Body Weight3/4 allometric scaling approach. Michigan states, "As that [half-life-based dose adjustment factor] allowed conversion of the point of departure to a human equivalent dose using chemical-specific information, the SAW [Science Advisory Workgroup] selected this approach over the allometric scaling used in the draft USEPA (2018) PFBS toxicity assessment." Although the half-life of PFBS is significantly shorter than long-chain PFAS (665 hours vs. 1241 days for PFOS), the half-life in humans is still much longer than in animals (665 hours in humans vs 2.1 hours mice). The dose adjustment factor for PFBS was 316, orders of magnitude greater than an adjustment factor based on body weight differences.

[10] https://www.nrdc.org/sites/default/files/comments-assessments-of-pfbs-and-genx-01222019.pdf
[11]https://www.michigan.gov/documents/pfasresponse/Health-Based_Drinking_Water_Value_Recommendations_for_PFAS_in_Michigan_Report_659258_7.pdf

Recommendation 5: Illinois EPA must reduce its recommended groundwater standard level for PFOA and PFOS

In August of 2019, California's Office of Environmental Health Hazard Assessment developed reference levels PFOA and PFOS in drinking water for both cancer and non-cancer effects.[12] The cancer effect reference level is based on the concentration of the chemical in drinking water that would not pose more than a one in one million cancer risk over a lifetime. For PFOA, OEHHA derived a reference level of 0.1 ppt based on pancreatic and liver tumors found in male rats in a new National Toxicology Program study.[13] For PFOS, OEHHA derived a reference level of 0.4 ppt based on liver tumors in male rats and the structural and biological similarity of PFOS to PFOA. We urge Illinois to examine OEHHA's risk assessment on PFOA and PFOS as it is significantly stricter than what it has proposed.

Recommendation 6 - Illinois must investigate the total burden of PFAS contamination in groundwater

We appreciate the state's initiative to investigate groundwater contamination and ensure the protection of water resources from harmful PFAS compounds. In addition to numerical standards for better-studied PFAS chemicals, the state should explore methods to document the total burden of organofluorine chemicals in contaminated areas using novel testing methods like TOP Assay or Total Organic Fluorine or Extractable Organic Fluorine assays to determine the full magnitude of synthetic fluorochemicals in water resources.

Summary of more protective choices that can be made for the PFAS Illinois is proposing to regulate.

	Current value (ppt)	Infant exposure assumptions* + 20% RSC	With UF to protect fetuses, infants and children
PFOA	21	3.4	0.3

[12]Office of Environmental Health Hazard Assessment. Notification Level Recommendations. Perfluorooctanoic Acid and Perfluorooctane Sulfonate in Drinking Water. August 2019. Accessed at: https://oehha.ca.gov/media/downloads/water/chemicals/nl/final-pfoapfosnl082119.pdf

[13] National Toxicology Program. TR-598: Technical Report Pathology Tables and Curves - PFOA. 2018. Assessed at: https://tools.niehs.nih.gov/cebs3/views/?action=main.dataReview&bin_id=13658

PFOA (CA)	0.1	-	0.01
PFOS	14	2.3	0.2
PFOS (CA)	0.4	-	0.04
PFNA	21	3.4	0.3
PFNA(MI)	6	-	0.6
PFHxS	140	23	2.3
PFHxS (NH)	18	-	1.8
PFBS	140,000	22,857	2,286
PFBS (MI)	420	-	42

*Vermont infant ingestion rate of 0.175 L/kg/d

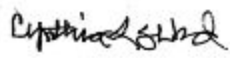
Considering the above information, Illinois should at minimum set a combined standard for PFOA, PFOS, PFNA, and PFHxS of 2 ppt, the reporting limit these chemicals, and a MCL of 42 ppt for PFBS. However, PFAS share similar structure and properties, including extreme persistence and high mobility in the environment. Many PFAS are also associated with similar health endpoints, some at extremely low levels of exposure. There is additionally potential for additive or synergistic toxicity among PFAS. Given the similarity among chemicals of the PFAS class and the known risk of the well-studied PFAS, there is reason to believe that other members of the PFAS class pose similar risk. Therefore, health-protective standards for PFAS should be based on the known adverse effects of the well-studied members of the PFAS class. **We therefore recommend that Illinois set a combined standard for all quantifiable PFAS at 2 ppt.**

The structure of the fluorine-carbon bond and the impacts documented on the studied PFAS already available support concern over the health impacts of the entire class. This is supported by the constant exposure to short-chain chemicals, even if they have a relatively short presence in the body, as well as the fact that in many cases the use of these chemicals may be much higher than their long-chain cousins. Furthermore, many PFAS can convert into PFAAs (a PFAS subgroup, which includes PFOA and PFOS, that is linked to many adverse health effects) or PFAAs are used in their manufacture and can be contaminants in their final product. A goal of zero PFAS in drinking water is needed to provide an adequate margin of safety to protect public health from a class of chemicals that is characterized by extreme persistence, high mobility, and is associated with a multitude of different types of toxicity at very low levels of exposure. **We therefore urge Illinois to explore in the near future the establishment of a treatment technique for PFAS** - a minimum treatment requirement or a necessary

methodology or technology that a public water supply must follow to ensure control of a contaminant.

Thank you for considering these important ways to ensure greater protection for Illinois residents. Please take these urgent and defensible actions to strengthen groundwater protections from PFAS to ensure that Illinois ground water resources.

Sincerely,



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February 27, 2020

VIA e-mail: sara.terranova@illinois.gov
Ms. Sara G. Terranova
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RE: Comments Regarding Proposed Amendments to 35 Ill. Adm. Code 620: Groundwater Quality

Dear Ms. Terranova:

We are the owner of Quad Cities Landfill in Milan, IL and are compelled to submit the comments below regarding proposed changes to the groundwater quality rules in 35 Ill. Adm. Code 620.

Background

Several Per- and Poly-Fluoroalkyl Substances (PFAS) compounds [man-made hydrophobic chemicals] are being proposed as additions to the potable (Class I) and general resource (Class II) groundwater quality lists. Specifically, the following compounds and groundwater standards are being proposed:

- Perfluorobutane Sulfonic Acid (PFBS) 140,000 ng/L (0.14 mg/L)
- Perfluorohexane Sulfonic Acid (PFHxS) 140 ng/L
- Perfluorononanoic Acid (PFNA) 21 ng/L
- Perfluorooctanoic Acid (PFOA) 21 ng/L
- Perfluorooctane Sulfonic Acid (PFOS) 14 ng/L

The amendments propose both individual and combined values for PFOA (21 ng/L) and PFOS (14 ng/L), which combined are not to exceed 21 ng/L.

Documentation suggests the range of PFOA + PFOS concentrations in landfills generally vary from 500 to 5,000 ng/L depending on the facility's acceptance of industrial waste or biosolids from wastewater treatment plants (WWTP). Continued acceptance of biosolids from WWTP will progressively concentrate PFAS compound mass.

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Concerns for Current Compliance

There are several concerns for active solid waste landfills that are currently regulated under 35 IAC Part 811 that should be accounted for if the new drinking water standards are adopted.

Groundwater Monitoring

One of the biggest concerns is the effect of adding PFAS compounds to the groundwater monitoring lists and the interferences (false positives) that will occur from sampling from the existing groundwater monitoring systems. Landfills regulated under 35 IAC 811 have established leak detection monitoring systems. Detection monitoring systems are based on conservative constituents (e.g., chloride) that are even more mobile that PFAS compounds; additional monitoring wells will not be required. However, many (if not most) active landfills have dedicated submersible sampling pumps that are permanently installed in the observation wells that make up the monitoring network of upgradient and downgradient wells. The Sampling and Analysis Plans (SAP) for permitted landfills have IEPA approval regulated under 35 IAC 811.318. Unfortunately, countless landfills (such as Quad Cities Landfill IV) have existing leak detection monitoring systems in place that are not suitable for sampling of PFAS compounds since the dedicated sampling tubing are lined with Teflon™ for its hydrophobic properties to prevent adsorption of constituents during sampling. Teflon is specifically identified as one of three materials approved for use (along with Stainless Steel 304 & 316) as durable, corrosion-resistant material allowed by IEPA for water sampling as outlined in IEPA's *Instructions for the Groundwater Protection Evaluation for Putrescible and Chemical Waste Landfills* [Appendix C to LPC-PA2].

Entire monitoring well networks may contain pumps with Teflon bladders, gaskets, discharge tubing, and Teflon-coated wire, all in direct contact with the groundwater samples. Teflon tape is commonly used on the threads of pumps and possibly at joints of well screens and casings. Therefore, the dedicated monitoring wells and tubing of solid waste facilities may be subject to significant burden of demonstration that alternate sources are the cause for false positive results.

If PFAS sampling is limited in its adoption to solid waste facilities, such as a single sampling event confirming detects less than drinking water standards (similar to the addition of new volatile organic compounds to the 620 standards), temporary removal of the sampling pumps, followed by redevelopment of the monitoring wells prior to PFAS sampling may be a work around. However, these procedures would be substantially burdensome if they had to continue long-term.

Groundwater impact Assessment (GIA)

The requirements of 35 IAC 811.317 is a unique permitting element to the Solid Waste Regulations in Illinois. Groundwater contaminant transport (GCT) modeling results must demonstrate predicted concentrations of all constituents in leachate outside the zone of attenuation are less than applicable groundwater standards within 100 years of closure of the

unit. Addition of PFAS compounds to the Part 620.410 and 620.420 groundwater standards and their subsequent addition to GIA's will result in countless landfills having GCT models that will no longer meet the requirements of 811.819(b) and be out of compliance. There are several reasons for the concerns with the GIA modeling that are outlined below:

- Leachate Source Characterization – This is a concern that is similar to the groundwater monitoring network, in that leachate collection and distribution components may contain PFAS compounds (including Teflon-bearing plumber's tape, as well as other gaskets, washers, and o-rings within leachate pumps, values, and tubing). This equipment is not readily replacable. Biased high results from system components in leachate would have direct effect on the GIA since these are required as conservative source concentrations in the GCT modeling.

  Should characterization of PFAS in leachate be required for the GIA, a reasonable alternative would be an allowance of average PFAS concentrations. Currently, leachate concentrations are required to be at least in the upper 95% confidence interval of detected concentrations. Since leachate concentrations will likely be biased high due to PFAS compounds being present in leachate collection and conveyance components, allowance of an averaged source concentration is appropriate.

- Transport and Fate Properties – Components for fate and transport of chemical-specific groundwater modeling of the PFAS compounds are uncertain. Preliminary data indicate that these compounds are known to be soluble, very stable, and non-volatile. PFAS compounds that are most commonly detected in the environment typically have competing tendencies of the head and the tail. The tail is hydrophobic (tends to repel water), whereas the head groups are polar and hydrophilic (tend to mix with water). The variations in tail lengths lead to a wide distribution in the environment (https://pfas-1.itrcweb.org/).

  Given heterogeneous subsurface environments, other geochemical factors such as pH, and presence of polyvalent cations, multiple partitioning mechanisms should be considered when characterizing PFAS fate and transport (Guelfo and Higgins 2013; McKenzie et al. 2016; Brusseau 2018). This statement suggests that accurate (or average) site conditions be considered in GCT modeling versus the most conservative assumptions that are currently required for GIA inputs. For example, at relevant environmental pH values, some PFAS constituents are typically present as organic anions and therefore tend to associate with the organic carbon fraction that may be present in the subsurface. Instead of calculating migration with accurate (average) organic content values, an overly conservative input of the lower 95% confidence interval is currently required for GCT modeling. This requirement could be relaxed for PFAS compounds so that average site conditions are represented for complicated PFAS migration processes and recent uncertainties in GCT results.

  Organic carbon-water partition coefficients (Koc values) are being established for many commonly detected PFAS compounds that are often detected at release sites (https://pfas-1.itrcweb.org/). However, diffusivity properties of PFOS compounds are still in development. PFOS diffusion in groundwater appears not to have been a priority in initial

migration studies since diffusion rates are significantly slower relative to advection processes. However, in composite or clay-lined landfills in clay-rich subsurface environments that are common in Illinois, knowledge of diffusion rates is required. Thus, implementation of GCT modeling requirements that will be triggered by new 35 IAC 620 groundwater standards is worrisome for solid waste owners and operators.

Alternatives to the current configuration of the solid waste regulations have been added to the discussions above. Additional alternatives for the IEPA to consider are reducing PFAS constituents pending investigations and elimination for the GIA requirement.

- Consider eliminating or reducing requirements for certain PFAS constituents that may be detected as false-positives as a result of cross-contamination from existing (and permitted) groundwater monitoring systems and/or approved standard landfill design guidelines.
- Consider eliminating the GIA. The requirement of a GCT model is unique to Illinois and is not necessary if minimum design considerations are met. The GIA serves no material practical purpose for the construction of landfills. It is well demonstrated that the standard Subtitle D landfill design has served to provide environmental protection. Regarding landfills, the GIA serves no material benefit to environmental projection. It is time to eliminate the GIA.

We are grateful for the opportunity to submit these comments. Please do not hesitate to contact me if you have any questions or comments.

Sincerely,

Millennium Waste Incorporated

Dominic J Remmes, PE
Region Engineer

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February 28, 2020

Via Email: sara.terranova@illinois.gov

Ms. Sara G. Terranova
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Illinois Environmental Protection Agency
1021 North Grand Avenue East
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Springfield, IL 62794-9276

Re: Comments Regarding Amendments To 35 Ill.Adm.Code 620: Groundwater Quality

Dear Ms. Terranova:

This firm represents the Illinois Chapter of The National Waste & Recycling Association (NWRA). As you know, NWRA submitted comments to the Illinois Environmental Protection Agency on the above-referenced amendments on February 10, 2020. In addition, several members of NWRA attended the stakeholder session on February 13, 2020.

The following members of NWRA have prepared supplemental comments, which are attached:

- PDC Technical Services, Inc.
- Andrews Engineering (on behalf of Republic Services)
- Millennium Waste Incorporated
- Waste Management

Please be advised that the membership of the Illinois Chapter of NWRA endorses and adopts the comments that accompany this letter.

If you have any questions regarding this submission, please contact me.

Sincerely,

James M. Morphew

James M. Morphew
JMM/jf
Attachments

4727166 2/28/2020

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February 27, 2020

Sara Terranova, Part 620
Illinois Environmental Protection Agency
1021 North Grand Avenue East
P.O. Box 19276
Springfield, IL 62794-9276

Dear Sara:

We are pleased to submit our enclosed comments regarding the proposed changes to the language of 35 Ill. Adm. Code 620: Groundwater Quality. If you have any questions regarding our comments please feel free to contact us. We appreciate the Agency allowing us the opportunity to comment on the proposed changes and we look forward to the next step in the rulemaking process.

Sincerely,

George L. Armstrong P.E.
Vice President—Engineering and Consulting Services

Charles Hostetler, Ph.D.
Director of Environmental Services

Enclosure: PDC Technical Services, Inc. Comments

PDC Technical Services, Inc.'s comments on the draft Part 620, dated 12-19-2019, are as follows:

Section 620.410 QWQS for Class I: Potable Resource Groundwater and Section 620.420 GWQS for Class II: General Resource Groundwater

1. Proposed PFAS standards will affect existing practices and procedures used by the solid waste industry

   In the state of Illinois, Municipal Solid Waste Landfills (i.e. landfills regulated under 35 IAC 811) must complete a Groundwater Impact Assessment (GIA) prior to initial permitting to demonstrate that the landfill will have no effect on groundwater quality for a period extending 100 years following landfill closure. The IEPA requires that the GIA models used to permit landfills assume that the landfill has a defective liner system. A key component of GIAs is the concentration of each constituent in leachate. The initial GIA is based on assumed concentrations of a long list of pollutants in leachate. Actual leachate concentrations are reviewed as part of each 5-year permit renewal application and, if they are greater than assumed in the initial GIA, additional modeling or computations are required. It has been reported that PFAS in landfill leachate have been detected at levels greater than 3,500 ppt (Lang, et al. 2017). Considering the proposed PFAS standards, and the anticipated high concentrations of PFAS in leachate, until the PFAS compound fate and transport mechanisms are better understood, we have concern whether or not any landfill GIA would pass under the assumption of a defective liner system, or if the typical models used for GIAs are stable to the proposed concentrations. Further, it is reasonable to assume high laboratory reporting limits of PFAS in leachate due to analytical (matrix) interferences. Industry practice is to assume that leachate parameters that were not detected are present at the reporting limit. Will GIA models demonstrate satisfactory results if the high reporting limits are used, or only at relatively low PFAS concentrations?

   Illinois is the only state in the United States which requires a GIA in a landfill's siting, initial operating permit, and permit renewal application processes. Therefore, the concerns described above are unique to the solid waste industry in the state. It is acknowledged that the proposed PFAS standards are not as conservative as those promulgated by some state agencies (e.g., Michigan, New Jersey); however, it is unclear if the agency accounted for the state-specific requirements and the implementability of the proposed standards for the solid waste industry.

Older, closed sanitary landfills are regulated under 35 IAC 807. Many of these landfills are owned by municipalities, and are nearing the end of their post-closure care period. Prior to being released from post-closure care, the IEPA Bureau of Land requires that the groundwater monitoring wells be sampled and analyzed for all constituents for which a groundwater quality standard has been established at 35 IAC 620. Considering the very low concentration standards that are being proposed and the ubiquitous nature of PFAS compounds, detection of PFAS at concentrations greater than the 620 standards will likely result in significant additional costs to these legacy landfill owners, even after years of satisfactory groundwater monitoring results.

2. There is significant uncertainty associated with the environmental health risks associated with PFAS compounds and, in particular Acceptable Daily Exposure (ADE) values used in calculating the Human Threshold Toxicant Advisory Concentration (HTTAC), as described in 35 Ill. Adm. Code 620, Appendix A.

The Centers for Disease Control and Prevention states "The human health effects from exposure to low environmental levels of PFOA are unknown....More research is needed to assess the human health effects of exposure to PFOA" (https://www.cdc.gov/biomonitoring/PFOA_FactSheet.html, downloaded 2/14/2020). Similarly, the National Institute of Environmental Health Sciences states "More research is needed to fully understand all sources of exposure, and if and how they cause health problems", "The research conducted to date reveals possible (emphasis added) links between human exposures to PFAS and adverse health outcomes.", and "While knowledge about the potential health effects of PFAS has grown, many questions remain unanswered" (https://www.niehs.nih.gov/health/topics/agents/pfc/index.cfm, accessed February 19, 2020).

The available research regarding exposure to PFAS has created a large disparity in the federal and state advisory levels promulgated by governing agencies. The variation is largely related to the different definitions of critical health effects and relative scarcity of human and mammalian studies. Human health studies are largely limited to populations of individuals who 1) have been occupationally exposed during the production or use of PFAS, 2) live in a community with high levels of PFAS measured in drinking water, or 3) have been exposed to background levels of PFAS. Mammalian studies are limited due to the difficulty of extrapolating results from a small animal population provided a controlled exposure dose to the human population in an uncontrolled environment. Further, the mammalian studies which have been conducted have not consistently defined the same critical health effects, making it more difficult to accurately determine an ADE value.The uncertainty associated with ADE values can dramatically shift groundwater standards. IEPA should review the endpoints of reference doses and critical health effects in available literature to determine the magnitude of differences between ADE values.

3. PFAS cleanup objectives are not provided as part of the proposed standards

IEPA does not provide cleanup objectives with the proposed standards. In an instance where PFAS compounds are detected in groundwater at a landfill, what are the expectations for corrective action? Will acceptable background concentrations be considered if PFAS is detectable in upgradient locations?

4. There has been an apparent lack of due process in the establishment of the proposed groundwater standards

It is not immediately apparent if peer reviews have been conducted on the proposed groundwater standards. If not, it should be considered imperative that IEPA conduct a peer review of their proposed standards to ensure that the Agency's standard development procedure is consistent with other regulatory agencies.

5. IEPA should consider the ubiquitous extent of PFAS compounds in groundwater and complete a statewide groundwater survey prior to promulgating regulatory standards

Considering the apparent extent of PFAS in the environment, it is possible that non-attributable concentrations of PFAS compounds will be detected in groundwater upgradient and downgradient of landfills. The state of Illinois is currently conducting a statewide survey of drinking water systems. It is reasonable to conduct a statewide groundwater survey, similar in scope, to determine areas of known PFAS contamination. Development of such a database would provide data to support background analyses and support alternate source demonstrations in scenarios where detectable PFAS concentrations are not attributable to the landfill. At a minimum, IEPA should clarify how the presence of PFAS will be handled in situations not commonly associated with landfill operations (i.e., detectable concentrations in upgradient monitoring wells).

6. There is currently a lack of established analytical methods for more complex leachate, soil and groundwater matrices.

Sampling and laboratory analysis methods have not been established for more complex leachate, soil and groundwater matrices. Laboratories must rely on their own modified analytical methods for analyzing these matrices. Modified methods vary from laboratory to laboratory. As of the date of this submittal, the solid waste industry is waiting on SW-486 method 8328 and Office of Water method 1600 to be issued by the USEPA. Inconsistent results between laboratories could result in analytical results that are not reproducible or defensible.

7. Drinking water standards should be promulgated before groundwater standards.

The purpose of the Class I (Potable Resource) Groundwater Standards is to protect drinking water supplies. The IEPA should not propose Class I Groundwater Standards until after drinking water standards are established. Additionally, the proposed Class I Groundwater Standards are based on concentrations in water that is consumed, and does not factor the probability of whether or not groundwater classified as Class I at any one location will ever be consumed as drinking water, nor does it factor contaminant fate and transport mechanisms. This is overly conservative considering that the vast majority of groundwater that is classified as Class I will never be used for drinking water.

8. Class II Groundwater is not used as a source of drinking water.

The proposed Class II (General Resource) Groundwater Standards are identical to the proposed Class I (Potable Resource) Groundwater Standards. Class II Groundwater is generally not suitable for a drinking water supply. Class II Groundwater Standards should not be based on direct consumption, but rather should be based on protecting other drinking water supplies considering location and fate and transport mechanisms.

9. All regulated landfills in the State must either routinely test for all parameters for which 620 standards are established, or will be required to test for them prior to ending post-closure care.

   Considering the apparent ubiquitous extent of PFAS compounds, it is probable that PFAS compounds will be detected in groundwater upgradient and downgradient of landfills. Because it is a VOC, landfill gas could not automatically be ruled out as a contributor of PFAS in groundwater upgradient of a landfill. As a result, it could be a very expensive and lengthy process to demonstrate that the landfill is not the source of PFAS compounds in groundwater that will never be ingested.

10. IEPA prescribed groundwater monitoring device construction and practices may not be compatible with obtaining representative groundwater quality data consistent with the proposed standards.

    Many of the dedicated groundwater monitoring well sampling bailers and pumps that are currently in use were likely manufactured with PFAS-containing compounds, specifically Teflon. In some cases, Teflon well casing might also have been used, and/or other well construction materials might have inadvertently contained PFAS compounds. It is possible that PFAS compounds from this equipment could have leached into groundwater making it difficult to distinguish the source of extremely low concentrations of PFAS compounds. Additionally, it would be very costly to replace all dedicated sampling pumps, and possibly groundwater monitoring wells themselves, using equipment and supplies that can be certified free of PFAS compounds.

11. PFAS-containing waste acceptance criteria are little understood.

    A better understanding of which wastestreams exhibit high concentrations of PFAS compounds (e.g. remediation wastes, municipal and industrial wastewater sludges, etc.) is needed before imposition of the groundwater standards. Unduly stringent groundwater standards could create an inappropriate lack of disposal capacity for such wastes.

ANDREWS ENGINEERING

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February 28, 2020

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re: Comments for 35 Ill. Adm. Code 620 Proposed Revisions

Dear Ms. Terranova:

On behalf of Republic Services, submitted herein are comments pertaining to the proposed revisions to 35 Ill. Adm. Code 620.

Should you have any questions or require additional information, please contact Eric Ballenger at 224-970-1128 or me at 217-787-2334. Thank you.

Sincerely,

Brad J. Hunsberger

Brad J. Hunsberger, LPG
Vice President

BJH:bjh

w/attachments

cc: Peggy Macenas (NWRA) – email
Kenn Liss (Andrews Engineering) - email

PROPOSED REVISIONS TO 35 IAC PART 620

AFFECTS TO THE GROUNDWATER IMPACT ASSESSMENT

The purpose of the Groundwater Impact Assessment is to provide an integrated evaluation of the acceptability of the physical setting and design of the landfill units through contaminant transport modeling. The impacts of leachate seepage from the unit must be addressed (i.e. modeled) in a systematic fashion using the techniques described in 35 IAC 811.317 and 812.316 [Appendix C to LPC-PA2]. The statutory requirements for the GIA are provided in 35 IAC 811.317 for a waste disposal facility complying with the regulations of 35 IAC Part 812 - Subpart C, and Part 814 - Subpart C.

The proposed revisions to the regulations of 35 IAC Part 620 will have a significant effect to the results of the Groundwater Impact Assessment (GIA) process for solid waste disposal units; specifically the proposed addition of Section 620.410(d)(3). The proposed addition states:

> *1) The concentrations of the following constituents must not be exceeded in Class I groundwater at both the individual standards and a combined standard of 0.000021 mg/L.*
>
> | *CAS No.* | *Constituent* | *Standard (mg/L)* |
> |---|---|---|
> | *335-67-1* | *Perfluorooctanoic Acid (PFOA)* | *0.000021* |
> | *1763-23-1* | *Perfluorooctane Sulfonic Acid (PFOS)* | *0.000014* |

The extremely low proposed standards and relatively non-attenuative properties of the PFOA and PFOS constituents make for a worst-case scenario with respect to an acceptable GIA. The GIA is conducted for all new waste units and is evaluated at least once every five years (35 IAC 813.304) pursuant to the permit renewal process contained in 35 IAC Part 813, Subpart C for existing units. Therefore, all 38 active landfill facilities (2018 Illinois Landfill Disposal Capacity Report) will be economically impacted by this rulemaking.

The parameters listed in 35 IAC 620.410 automatically become part of the GIA process as those are referenced in (at a minimum):

Section 811.315(e)(1)(G)(i) – background concentrations must be established for "*Any constituent for which there is a standard at 35 Ill. Adm. Code 620 established by the Board and which is expected to appear in the leachate, and*"

Section 811.317(a)(2) – "*The concentration of constituents in the leachate shall be determined from actual leachate samples from the waste or similar waste, or laboratory derived extracts.*" This regulation infers the 620 parameters via Section 811.315(e)(1)(G)(i).

Section 811.317(a)(3) – *"A contaminant transport model meeting the standards of subsection (c) shall be utilized to estimate the concentrations of the leachate constituents over time and space. The Agency must review a groundwater*

contaminant transport model for acceptance in accordance with 35 Ill. Adm. Code 813.111."

Section 811.320(a)(3(B) – Applicable Groundwater Quality Standards – For the purposes of this Part: ""*Board established standard" is the concentration of a constituent adopted by the Board as a groundwater quality standard adopted by the Board pursuant to Section 14.4 of the Act or Section 8 of the Illinois Groundwater Protection Act."*

There are multiple complexities within the GIA process that arise as part of the subject proposed rule revisions. Those are discussed individually below:

1. Establishment of AGQSs

The GIA through contaminant transport modeling provides predicted model concentrations that are compared to AGQS values derived pursuant to Section 811.320(d). If all predicted model concentrations fall below the AGQS, the GIA is deemed acceptable. However, derivation of accurate AGQSs for PFOA and PFOS constituents will be difficult at best, and may be suspect due to many factors.

Establishment of background concentrations require at least four quarters of good data (the timing and number of sampling intervals may be altered if approved by the Illinois EPA). Good data is dependent upon sampling and testing methods, as well as a monitor well network free of PFOA and PFOS constituents. Sampling methods have to some extent been established. However, many laboratory testing methods are in draft stages and are specific to clean water, not for samples that may contain turbidity, or with respect to leachate - probable matrix interference issues.

Cross contamination from the wells is also a potential due to well construction methods. Illinois EPA documentation (Appendix C to LPC-PA2 (Instructions for the Groundwater Protection Evaluation for Putrescible and Chemical Waste Landfills)) specifically recommended well materials that are known PFAS sources. Section IV.B of Appendix C states:

The application must provide detailed documentation of the monitoring well and piezometer construction. Casing and screen material must be inert to avoid contributing contamination or causing interference with the analysis of the water sample. Teflon, Stainless Steel 316, and Stainless Steel 304 are recommended as durable, corrosion- resistant materials. Since plastic (PVC) may have a significant effect on the ability to obtain a "representative" sample, the Agency only allows the use of plastic casing for piezometers or through the unsaturated zone for wells.

Entire monitor well networks contain pumps with Teflon bladders, gaskets, discharge tubing, and Teflon-coated wire, all in direct contact with the groundwater samples (potential for direct cross contamination). In addition, Teflon seals or tape were commonly used on the threads of the well screens and casings. Packaging for well materials may have contained PFAS, including bags and containers for sand (screen sand pack) and bentonite, cross contaminating the well unaffiliated with the waste unit. Also, the Illinois EPA requires that potable water be used in construction of the wells. Most water supplies for well installation and equipment decontamination are obtained from city supply lines or bulk stations that may contain PFAS compounds. Potable water sources will need to be located that can be certified free of PFAS,

otherwise, any well installed may be cross contaminated by the potable water supply. The source of low level PFAS concentrations may never be identified with the potential for cross contamination from numerous sources. It is unreasonable to assume that all wells will need to be replaced that show low level PFAS contamination because of potential cross contamination when the well was installed pursuant to IEPA guidelines. For the installation of new wells, testing may be necessary throughout each phase of installation. This would include the potable water supply, the drilling contractor equipment (including water tanks, lines, hoses, and pumps), and well materials.

Upon approval and implementation of the proposed rules, it will likely be difficult to identify the source of PFOA and PFOS constituents if detected in any well, upgradient or downgradient. More time and effort will be spent trying to validate the data such that it is useable and meaningful. Alternate sources will be evaluated as part of this process, which will require significant additional time. If the AGQS values are suspect, the GIA process may be of little to no use for the PFOA and PFOS constituents.

2. Source Concentration

The source concentration is probably the single most important model input parameter. A high source concentration for particularly sensitive parameters (largely non-attenuative) such as ammonia, chloride, or boron, normally result in initial failure of the GIA baseline model. Pursuant to Section 811.317(a)(2), leachate samples from the applicable waste units will require analyses for PFOA and PFOS constituents once the rule revisions are approved. The constituents will be utilized as source concentrations for the contaminant transport model, resulting in a predicted model concentration used to determine if the GIA is acceptable.

Analyses of the subject parameters in the leachate will be difficult due to probable matrix interference. This will likely increase the Practical Quantitation Limit (PQL), which can artificially increase the source concentration resulting in a higher predicted model concentration and likely resulting in failing model results. Laboratory analytical methods have not been advanced sufficiently to provide accurate results from a leachate matrix.

The source concentration must be accurate. Similar cross contamination issues described above apply to obtaining a representative leachate sample. The leachate collection system within a modern waste unit consists of collection and conveyance lines, sealing materials, and numerous pump systems that can contribute PFOA and PFOS constituents to the leachate samples. Detection of low level concentrations in the leachate will be suspect and the source concentration likely inaccurate. Cross contamination of PFAS may be sufficient to cause failure of the GIA, or failure of the original assumptions of the GIA in the case of a permit renewal application.

The Illinois EPA Bureau of Land should revise the guidance document (LPC-PA2) or create a new document to standardize sample retrieval and testing methods for leachate.

3. Potential Design Changes

Each operational landfill and many closed waste units (35 IAC Part 814, Subpart C) maintain approved GIAs. Pursuant to 35 IAC 813.304, the GIA must be re-evaluated at least every five years (permit renewal process) or sooner if changes to the facility or its operations would

result in an increased probability of exceeding a groundwater quality standard beyond the zone of attenuation.

The GIA of record for any facility was completed utilizing site specific data (hydrogeologic and leachate analyses) or as otherwise approved by the Illinois EPA as being representative of the facility setting. In many cases, the initial baseline model runs for the new waste units were borderline or even failed. To address those, design changes were incorporated to include thicker liner systems, revision the slope and leachate collection system to reduce the leachate head (seepage rate), revision to the liner system placement within the hydrogeologic setting (relocate the liner elevations to provide additional in-situ low hydraulic conductivity deposits between the liner invert and uppermost aquifer), and/or revision to the final cover system design to decrease the precipitation infiltration into the waste unit. The model also incorporated partitioning coefficients for specific surrogate groups which aided in reduction of the predicted model concentrations for typically problematic constituents, resulting in an acceptable model.

Regulatory constraints and guidance for the contaminant transport models have been largely consistent since the mid to late 1990s. Design and cell construction have been permitted for all active facilities, as well as final closure for many waste units. The final cover systems were designed based on HELP modeling which was used to determine seepage rate for the input to the contaminant transport model.

The addition of PFOA and PFOS constituents through the 35 IAC 620 rule revisions has the potential to cause failure of many permitted GIAs which are acceptable under the current requirements. It would have been possible during the initial design stage to address results of the PFOA and PFOS constituents through design changes. However, the potential for design changes to existing waste units are very limited, with only the final cover system realistically remaining for redesign to lower infiltration to the waste unit during post closure, thus possibly reducing the leachate head on the liner system.

Design changes for future cells (already permitted) yet to be constructed may be necessary if the results of the contaminant transport model fail due to the addition of the PFOA and PFOS constituents. However, this will be highly dependent upon the geologic setting and may be restricted by the local siting resolution pursuant to Section 39.2 of the Act. If the Illinois EPA is to go forward with the revisions as proposed, a mechanism needs to be created allowing existing facilities a way to address GIA failures without automatically reverting to a contingent remediation program.

4. Appropriate Contaminant Transport Models

The GIA is a determination of the time and distance dependent potential impact of a landfill unit on local groundwater chemistry. The GIA is based on a site-specific solute transport model of the actual design, site-specific hydrogeology, and conservative performance standards for the liner system, leachate management system and final cover system. The GIA is considered acceptable if the groundwater contaminant transport model predicts that the concentrations of all leachate constituents outside of the zone of attenuation are less than the Applicable Groundwater Quality Standards (AGQS) of 35 IAC 811.320 within 100 years of closure of the unit.

Typically contaminant transport models associated with the GIA have been generally simplistic, being one- and/or two-dimensional, such as POLLUTE and MIGRATE. The conceptual model assumes:

- all geologic units and soil liners are homogeneous and isotropic with respect to all lithologic and hydrologic parameters,
- that all layers are laterally extensive and the thickness of each layer is uniform,
- all layers are fully saturated,
- the external stresses on the system are constant through time,
- the source concentration is constant over the entire modeling period, and
- baseline surrogates were prepared in which no retardation or decay occurs.

Allowing the use of more reasonable model parameters would help reduce the model prediction factor and increase the probability of an acceptable model. The model input parameters are typically the most conservative across the board. When combined with conservative parameters for use in the HELP modeling, the end result is an ultraconservative model where surrogate groups are often needed to achieve an acceptable model. This would be a policy change for the Bureau of Land, not a regulatory change.

Under fully saturated conditions (bottom of the liner system to the bottom of the upper most aquifer), the models utilized for the approved GIAs are likely adequate for evaluation of the PFOA and PFOS constituents. However, settings where unsaturated conditions exist or a vadose zone exists beneath the liner system, a more complex model would better simulate transport of the PFOA and PFOS constituents as transport through such deposits are significantly less. Recent studies have shown PFOA and PFOS constituents are substantially retained in unsaturated deposits via solid phase adsorption, and also at the air-water interface. Differing models may simulate this characteristic better than the typical one- and two-dimensional models used for previous GIAs. Most contaminant transport models are incapable of working with the small-scale changes for these parameters that are seen within many geologic materials. The introduction of other contaminant transport models to deal specifically with the PFOA and PFOS constituents will be costly and time consuming not only for the facility but for review purposes by the Bureau of Land's Permit Section.

5. Bureau of Land Guidance

Even though the proposed 620 rule changes are being driven by the Bureau of Water, ramifications to the Bureau of Land programs are paramount. Prior to sending the proposed rule changes to the Illinois Pollution Control Board, the Bureau of Land should vet the potential ramifications to the regulations of 35 IAC Parts 811-815. The Bureau of Land should then provide a draft update to Appendix C to LPC-PA2 (Instructions for the Groundwater Protection Evaluation for Putrescible and Chemical Waste Landfills) for review and comment by the waste disposal industry. The Illinois EPA has provided two revisions to Appendix C based on what was learned over time during the permitting process. It is reasonable to expect the Bureau of Land should do the same with respect to implications to existing solid waste disposal facilities for revision of the 620 rules. Topics that should be addressed include but are not limited to:

a. Legacy impacts (cross contamination) to groundwater quality – what if the wells already exhibit PFOA and PFOS concentrations in excess of the proposed standards
    i. Well construction issues
    ii. Pump materials
    iii. Impacts to AGQS determination
b. Sampling protocols for groundwater and leachate
c. Laboratory analyses test methodology and limitations – how can "draft" methods be placed into a state regulations
d. GIA – It is a tool
    i. Computer models – provide insight on potential other models for use
    ii. Input parameters
        - Use of more realistic values versus overly conservative values
        - Use of averages or statistical derivations, not the maximum or minimum
        - Update Attachment 1 to Appendix C to include PFOS and PFOA constituents
    iii. Surrogate Modeling for PFOA and PFOS constituents
        - Retardation allowances
        - Sensitivity analyses constraints
e. Use of contingent remediation programs to address predicted exceedences
f. Permitted Contingent Remediation Plans – will all of these need to be re-evaluated with the inclusion of PFOA and PFOS constituents
g. Impacts to permitted waste units in corrective action (35 IAC 807 and 814 Subpart C and D)
h. Impacts to permitted waste units conducting corrective action pursuant to consent orders and/or in conjunction with the US EPA or other entities
i. Sites finishing post closure care (the Affidavit for Certification of Completion of Post-Closure Care has been submitted) – will PFOA and PFOS constituents require analyses prior to release
j. Reasonable dates and timelines for implementation
k. Regulatory exclusion – if the proposed rules are passed, while a facility evaluates its water and leachate quality, the Illinois EPA must provide temporary exclusion from Section 18 of the Act, or others that may apply

Once a new standard is promulgated in Part 620, it is then incorporated into the relevant programs administered by the Bureau of Land. As described above, the process to evaluate potential contaminants are imposed through permits issued by the Bureau of Land. The mere detection of the PFOA and PFOS constituents at a landfill monitor well requires the owner/operator to disprove the potential of a release to the environment. Considering the current body of scientific knowledge, facilities will likely be thrust into the environmental investigation process. That process leads to corrective action. No economic impact study has been conducted to evaluate the cost or the value of expending resources on this path.

The next public meeting should include members from the Bureau of Land prepared to discuss implications to the existing permitted landfill facilities. These issues should be considered prior to submittal of the proposed rule revisions to the Illinois Pollution Control Board for approval.

MWI
Millennium Waste Incorporated

February 27, 2020

VIA e-mail: sara.terranova@illinois.gov
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Division of Legal Counsel
Illinois Environmental Protection Agency
1020 North Grand Avenue East
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RE: Comments Regarding Proposed Amendments to 35 Ill. Adm. Code 620: Groundwater Quality

Dear Ms. Terranova:

We are the owner of Quad Cities Landfill in Milan, IL and are compelled to submit the comments below regarding proposed changes to the groundwater quality rules in 35 Ill. Adm. Code 620.

Background

Several Per- and Poly-Fluoroalkyl Substances (PFAS) compounds [man-made hydrophobic chemicals] are being proposed as additions to the potable (Class I) and general resource (Class II) groundwater quality lists. Specifically, the following compounds and groundwater standards are being proposed:

- Perfluorobutane Sulfonic Acid (PFBS) 140,000 ng/L (0.14 mg/L)
- Perfluorohexane Sulfonic Acid (PFHxS) 140 ng/L
- Perfluorononanoic Acid (PFNA) 21 ng/L
- Perfluorooctanoic Acid (PFOA) 21 ng/L
- Perfluorooctane Sulfonic Acid (PFOS) 14 ng/L

The amendments propose both individual and combined values for PFOA (21 ng/L) and PFOS (14 ng/L), which combined are not to exceed 21 ng/L.

Documentation suggests the range of PFOA + PFOS concentrations in landfills generally vary from 500 to 5,000 ng/L depending on the facility's acceptance of industrial waste or biosolids from wastewater treatment plants (WWTP). Continued acceptance of biosolids from WWTP will progressively concentrate PFAS compound mass.

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Concerns for Current Compliance

There are several concerns for active solid waste landfills that are currently regulated under 35 IAC Part 811 that should be accounted for if the new drinking water standards are adopted.

Groundwater Monitoring

One of the biggest concerns is the effect of adding PFAS compounds to the groundwater monitoring lists and the interferences (false positives) that will occur from sampling from the existing groundwater monitoring systems. Landfills regulated under 35 IAC 811 have established leak detection monitoring systems. Detection monitoring systems are based on conservative constituents (e.g., chloride) that are even more mobile that PFAS compounds; additional monitoring wells will not be required. However, many (if not most) active landfills have dedicated submersible sampling pumps that are permanently installed in the observation wells that make up the monitoring network of upgradient and downgradient wells. The Sampling and Analysis Plans (SAP) for permitted landfills have IEPA approval regulated under 35 IAC 811.318. Unfortunately, countless landfills (such as Quad Cities Landfill IV) have existing leak detection monitoring systems in place that are not suitable for sampling of PFAS compounds since the dedicated sampling tubing are lined with Teflon™ for its hydrophobic properties to prevent adsorption of constituents during sampling. Teflon is specifically identified as one of three materials approved for use (along with Stainless Steel 304 & 316) as durable, corrosion-resistant material allowed by IEPA for water sampling as outlined in IEPA's *Instructions for the Groundwater Protection Evaluation for Putrescible and Chemical Waste Landfills* [Appendix C to LPC-PA2].

Entire monitoring well networks may contain pumps with Teflon bladders, gaskets, discharge tubing, and Teflon-coated wire, all in direct contact with the groundwater samples. Teflon tape is commonly used on the threads of pumps and possibly at joints of well screens and casings. Therefore, the dedicated monitoring wells and tubing of solid waste facilities may be subject to significant burden of demonstration that alternate sources are the cause for false positive results.

If PFAS sampling is limited in its adoption to solid waste facilities, such as a single sampling event confirming detects less than drinking water standards (similar to the addition of new volatile organic compounds to the 620 standards), temporary removal of the sampling pumps, followed by redevelopment of the monitoring wells prior to PFAS sampling may be a work around. However, these procedures would be substantially burdensome if they had to continue long-term.

Groundwater impact Assessment (GIA)

The requirements of 35 IAC 811.317 is a unique permitting element to the Solid Waste Regulations in Illinois. Groundwater contaminant transport (GCT) modeling results must demonstrate predicted concentrations of all constituents in leachate outside the zone of attenuation are less than applicable groundwater standards within 100 years of closure of the

unit. Addition of PFAS compounds to the Part 620.410 and 620.420 groundwater standards and their subsequent addition to GIA's will result in countless landfills having GCT models that will no longer meet the requirements of 811.819(b) and be out of compliance. There are several reasons for the concerns with the GIA modeling that are outlined below:

- Leachate Source Characterization – This is a concern that is similar to the groundwater monitoring network, in that leachate collection and distribution components may contain PFAS compounds (including Teflon-bearing plumber's tape, as well as other gaskets, washers, and o-rings within leachate pumps, values, and tubing). This equipment is not readily replacable. Biased high results from system components in leachate would have direct effect on the GIA since these are required as conservative source concentrations in the GCT modeling.

  Should characterization of PFAS in leachate be required for the GIA, a reasonable alternative would be an allowance of average PFAS concentrations. Currently, leachate concentrations are required to be at least in the upper 95% confidence interval of detected concentrations. Since leachate concentrations will likely be biased high due to PFAS compounds being present in leachate collection and conveyance components, allowance of an averaged source concentration is appropriate.

- Transport and Fate Properties – Components for fate and transport of chemical-specific groundwater modeling of the PFAS compounds are uncertain. Preliminary data indicate that these compounds are known to be soluble, very stable, and non-volatile. PFAS compounds that are most commonly detected in the environment typically have competing tendencies of the head and the tail. The tail is hydrophobic (tends to repel water), whereas the head groups are polar and hydrophilic (tend to mix with water). The variations in tail lengths lead to a wide distribution in the environment (https://pfas-1.itrcweb.org/).

  Given heterogeneous subsurface environments, other geochemical factors such as pH, and presence of polyvalent cations, multiple partitioning mechanisms should be considered when characterizing PFAS fate and transport (Guelfo and Higgins 2013; McKenzie et al. 2016; Brusseau 2018). This statement suggests that accurate (or average) site conditions be considered in GCT modeling versus the most conservative assumptions that are currently required for GIA inputs. For example, at relevant environmental pH values, some PFAS constituents are typically present as organic anions and therefore tend to associate with the organic carbon fraction that may be present in the subsurface. Instead of calculating migration with accurate (average) organic content values, an overly conservative input of the lower 95% confidence interval is currently required for GCT modeling. This requirement could be relaxed for PFAS compounds so that average site conditions are represented for complicated PFAS migration processes and recent uncertainties in GCT results.

  Organic carbon-water partition coefficients (Koc values) are being established for many commonly detected PFAS compounds that are often detected at release sites (https://pfas-1.itrcweb.org/). However, diffusivity properties of PFOS compounds are still in development. PFOS diffusion in groundwater appears not to have been a priority in initial

migration studies since diffusion rates are significantly slower relative to advection processes. However, in composite or clay-lined landfills in clay-rich subsurface environments that are common in Illinois, knowledge of diffusion rates is required. Thus, implementation of GCT modeling requirements that will be triggered by new 35 IAC 620 groundwater standards is worrisome for solid waste owners and operators.

Alternatives to the current configuration of the solid waste regulations have been added to the discussions above. Additional alternatives for the IEPA to consider are reducing PFAS constituents pending investigations and elimination for the GIA requirement.

- Consider eliminating or reducing requirements for certain PFAS constituents that may be detected as false-positives as a result of cross-contamination from existing (and permitted) groundwater monitoring systems and/or approved standard landfill design guidelines.
- Consider eliminating the GIA. The requirement of a GCT model is unique to Illinois and is not necessary if minimum design considerations are met. The GIA serves no material practical purpose for the construction of landfills. It is well demonstrated that the standard Subtitle D landfill design has served to provide environmental protection. Regarding landfills, the GIA serves no material benefit to environmental projection. It is time to eliminate the GIA.

We are grateful for the opportunity to submit these comments. Please do not hesitate to contact me if you have any questions or comments.

Sincerely,

Millennium Waste Incorporated

Dominic J Remmes, PE
Region Engineer

WASTE MANAGEMENT

Comments on 35 Ill. Adm. Code 620 Proposed Amendments
February 28, 2020

A. Detection and Quantification

The proposed Part 620 amendments include many standards listed at levels that will be difficult for commercial laboratories to quantify. Any proposed standards must consider a commercial laboratory's ability to quantify and report at these levels (i.e., at the practical quantification limit - PQL). The purpose of the PQL is to adjudicate between a health-based level and a laboratory's ability to quantify at that level. Any proposed standard must consider the commercial laboratory's capability to quantify at that level to avoid falsely reporting a standards exceedance when it does not exist. The following points are critical for IEPA to address prior to finalizing proposed standards in Part 620.

1. **Section 620 [All Subsections] - The use of PQLs in setting numeric standards needs to be retained or added:** Setting of numeric standards must consider analytical capability and variability to represent legal and attainable regulatory limits. Fundamental to any regulatory establishment of numeric standards is the evaluation and adjustment of health-based 'goals', where needed, to create standards that analytical technology can reliably quantify. It is this 'adjudication' of the environmental 'goal' to what is practically achievable that provides the technical foundation for the Agency to regulate, and for regulated parties to comply with regulation. Where a numeric standard is set without this adjudication, the establishment of a standard is arbitrary and capricious. A direct example of federal adjudication to analytical technology's limitations is MCLGs being adjusted to MCLs. [See Fed. Reg. 54, #97, May 22, 1989 page 22100 for a discussion of MCLs and the use of PQLs].

   For RCRA landfill groundwater regulations at the federal level, the PQL model is defined in regulation and is used for this adjudication. Although the current IEPA Title 35 Part 620 includes a definition for PQL, how it is used in the rule is unclear. The seeming removal of PQL from the Agency 's numeric standard setting process ignores the vital role of the PQL in establishing 'reasonableness' in setting regulatory levels. In this scenario, regulated parties must use analytical technology to measure and compare to a regulatory standard, which is inconsistent with RCRA and environmental regulation in general.

2. **IEPA needs to address how PQLs are utilized in the setting of standards:** and consider the analytical minimum capabilities (sensitivity) and variability (precision and accuracy) of the commercial laboratories in making regulatory decisions. Setting and using PQLs and assuring the upper end of the uncertainty bounds at the health-based numeric value when less than PQL are needed. Use of the PQL will prevent arbitrary and discriminatory enforcement of standards for those who apply them.

3. **A clear definition of the PQL is needed in Part 620:** USEPA initially defined PQL at 50 Fed. Reg. 46906 (Nov. 13, 1985) under the Clean Drinking Water Act. PQL is defined as *"the lowest level achievable by good laboratories within specified limits during routine operating conditions."* 620.110, Definitions, defines PQL as *"the lowest concentration or level that can be reliably measured within specified limits of precision and accuracy during routine laboratory operating conditions in accordance with "Test Methods for Evaluating Solid Wastes, Physical/Chemical Methods", EPA Publication No. SW-846, incorporated by reference at Section 620.125."* In 40 CFR

Part 257.23.g.5 (page 16 of 81), the PQL is *"the lowest concentration level that can be reliably achieved within specified limits of precision and accuracy during routine laboratory operating conditions that are available to the facility."*

Most importantly, the Statistical Analysis of Groundwater Monitoring Data at RCRA Facilities (Unified Guidance - 2009) which is referenced in Part 620 states on p. 2-7 that *"Any practical quantification limit (pql) approved by the Regional Administrator under §264.97(h) [or §258.53(g)] that is used in the statistical method shall be the lowest concentration level that can be reliably achieved within specified limits of precision and accuracy during routine laboratory operating conditions available to the facility."* It is recommended that the Unified Guidance definition be incorporated into Part 620.

While these definitions may vary somewhat, the key elements are:

- PQLs must be established using laboratories available to the regulated facilities
- Routine operating conditions must be in place at the time the data are being developed for the determination of the PQLs.
- Specified limits of (aka known and controlled) precision and accuracy requires the Agency to select precision and accuracy values. These values represent the allowable Relative Measurement Error (RME) and, thus, significant digits (or parts thereof) required before quantitation is established.
- Marginal changes to the definition do not change the science or the Agency responsibility to select criteria.

The application of statistics also requires that other requirements be specified and met such as sample size, normality and confidence.

B. Addition of 5 PFAS Compounds (PFOA, PFOS, PFBS, PFHxS, and PFNA)

The IEPA should address and resolve key scientific uncertainties before developing standards for PFAS. Existing literature demonstrates significant scientific uncertainty where standards are being developed with insufficient technical knowledge. Developing standards for PFAS at this time will lead to flawed rulemaking and will impose unwarranted, unfair, and oppressive legal, economic, and operational burdens on the regulated community. Standards developed would be scientifically unsound, fundamentally unfair, and create confusion and unintended societal consequences in the future.

1. **The accelerated pace to established PFAS standards does not allow the time needed to adequately assess the potential toxicity of a given compound:** let alone to develop MCLs that consider economic and technological factors. By way of comparison, and focusing solely on the toxicity component alone, USEPA has been assessing the potential toxicity of dioxin and furans – a group of merely 210 compounds, a much smaller group than the 4,000 unique PFAS compounds – since 1985. USEPA's assessment of dioxin-like compounds has been reviewed by USEPA Science Advisory Boards on four separate occasions, has been reviewed by the National Academy of Sciences, and has undergone multiple rounds of public comment. It took USEPA more than 20

years to reach consensus on the noncancer effects of dioxin and furans, and, even after such technical scrutiny, USEPA still has not reached a consensus on dioxin's cancer potency. This is for a group of chemicals for which the mode of toxic action and the relative potency among congeners is well-developed. None of these conditions hold for PFAS, and yet IEPA wants to establish standards for five PFAS when significant data gaps exist. The current science does not support standards establishment. Standards should not be developed until much greater scientific certainty and technical understanding is gained.

2. **The development of PFAS standards should start with properly assessing human health risks** not with assuming that the science is settled. Over 2,000 studies have been conducted on either PFOA or PFOS in laboratory animals, including mice, rats, and primates, plus over 400 human epidemiological studies have been published on PFAS, primarily on PFOA and PFOS. Although many independent studies have been performed, results are inconsistent and scientific consensus is lacking on what the data mean regarding human health risks and PFAS toxicity. It is imperative to keep the state of the science in the forefront to ensure technically defensible standards are developed and are appropriate for the long term.

3. **IEPA should consider interim, conditional, or similar alternatives to prematurely establishing formal standards** given the inadequate technical information and scientific consensus regarding PFAS health risks. For example, delaying rulemaking until adequate technical information is available by performing state-funded studies and reviewing ongoing studies being performed worldwide to fill data gaps would not only be procedurally justified but would be a more responsible use of funding. A reasonable approach for IEPA is to consider reevaluation of PFAS health effects on an annual basis following review of new studies and other scientific developments.

C. Concerns with Approach in Development of PFAS Groundwater Quality Standards

It is clear from the literature that too little is known currently to derive reliable standards for PFAS. Standards for PFAS based on the current state of knowledge are driven by uncertainty that needs to be responsibly addressed within the scientific community. If standards development is going to move forward, the process needs to be more rigorous and transparent so that better values can be supported. It is again emphasized that IEPA should consider interim, conditional, or similar alternatives to prematurely establishing formal standards.

1. **The state of knowledge on PFAS toxicology is still developing and many uncertainties exist. IEPA should refrain from developing PFAS groundwater standards until these scientific uncertainties are better explored.** Discrepancies exist between effects observed in rodent toxicology studies and human epidemiology studies. Furthermore, the adversity of many effects observed in human epidemiology studies is an area of active debate. Given the uncertainty in underlying data, there is wide variation in guideline/regulatory values that have been developed by different organizations.

Comments on 35 Ill. Adm. Code 620 Proposed Amendments
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2. **Use of ATSDR's draft MRLs results in overly conservative groundwater quality standards that are driven by uncertainty. If IEPA moves forward with developing groundwater quality standards, they should consider using different Tier III toxicity values.** In developing their draft minimum risk levels (MRLs), ATSDR applied dosimetric adjustment factors of 10,000 (PFOA), 14,400 (PFOS), 15,500 (PFHxS), and 6,500 (PFNA) to account for differences between humans and rodents. Additionally, a combined uncertainty factor of 300 was applied to each PFAS such that the total adjustment factors used were 3,000,000 for PFOA, 4,300,000 for PFOS, 4,650,000 for PFHxS, and 1,950,000 for PFNA. Application of such large adjustment factors is excessive and is the main driver of IEPA's proposed low groundwater quality standards. As outlined further below, the chronic oral reference doses (RfDs) developed by USEPA Office of Water in 2016 for PFOA and PFOS should be used by IEPA to develop groundwater quality standards.
3. **If IEPA moves forward with developing groundwater quality standards for PFAS, they need to document which Tier III toxicity values they considered and how they determined which values were the most appropriate.** ATSDR's PFAS MRLs are still in draft format. During the stakeholder meeting on February 13, 2020, a number of scientific and technical issues were raised regarding some of ATSDR's draft MRLs. As discussed further below, more scientifically supportable Tier III toxicity values are available and should be considered by IEPA if they move forward with developing groundwater quality standards for these compounds.

4. **If IEPA moves forward with developing groundwater quality standards for PFOS, USEPA's chronic oral reference dose should be used.** ATSDR developed an intermediate-duration MRL of 2 ng/kg-day for PFOS based upon results from a two-generation reproductive toxicity study (Luebker et al. 2005), which is the same key study underlying the chronic oral RfD (20 ng/kg-day) developed by USEPA Office of Water in 2016. Both USEPA and ATSDR used similar approaches to develop their toxicity values. Both USEPA and ATSDR applied UFs of 10 to account for human variability and 3 to account for interspecies variability; however, ATSDR applied an additional modifying factor (MF) of 10 to their PFOS MRL to account for uncertainty that immunotoxicity may be a more sensitive endpoint than developmental toxicity. Application of an additional MF of 10 is excessive when a total adjustment factor of 430,000 had already been applied. Furthermore, USEPA considered immunotoxicity endpoints in their PFOS assessment and determined that due to uncertainties related to mode of action and the level, duration, and time of exposure it is not appropriate to quantitatively assess immunotoxicity[1].

5. **ATSDR used low validity studies to develop their PFOA MRL. If IEPA moves forward with developing groundwater quality standards for PFOA, US EPA's chronic oral reference dose should be used.** ATSDR developed the PFOA MRL using results from two developmental toxicity studies (Koskela et al. 2016; Onishchenko et al. 2011). Both studies utilized a single dose level (i.e., 0.3 mg/kg-day) and had a small sample size (i.e., n=5-10 animals per dose). According to 35

[1] See Section 8.3 (Consideration of Immunotoxicity) in Drinking Water Health Advisory for Perfluorooctane Sulfonate (PFOS). EPA Document number 822-R-16-004.

Ill. Adm. Code 620 App. A, the studies utilized by ATSDR are low validity studies. To avoid developing groundwater quality standards based on low validity studies, IEPA should use the chronic oral RfD developed by USEPA Office of Water in 2016. USEPA's PFOA RfD is the basis of USEPA's 2016 drinking water Health Advisory Level[2] and USEPA's 2019 interim recommendations for addressing PFOA contaminated groundwater[3]. Use of USEPA's PFOA RfD would align IEPA with current federal practices. Use of USEPA's RfD for PFOA and supports a groundwater quality standard of 140 ng/L for PFOA.

6. **USEPA's draft chronic oral RfD for PFBS may be the most appropriate toxicity value for developing a groundwater quality standard.** Currently, the proposed groundwater quality standard of 140,000 ng/L PFBS is based upon USEPA's 2014 provisional peer-reviewed toxicity value of 0.02 mg/kg-day PFBS. However, in November 2018 USEPA's Office of Research and Development issued a draft chronic oral RfD of 0.01 mg/kg-day PFBS[4]. Although this value is not yet finalized, it received favorable peer-reviews and it represents the best available science for PFBS. IEPA should consider using USEPA's 2018 draft RfD for PFBS, which supports a groundwater quality standard of 70,000 ng/L PFBS.

7. **Use of a default relative source contribution (RSC) value of 20% is not appropriate for developing groundwater quality standards for all PFAS.** The use of the default RSC of 20% likely overestimates the contribution of diet and other non-drinking water sources in situations where exposure to elevated PFAS in drinking water occurs. For example, based on chemical-specific data, the New Jersey Drinking Water Quality Institute determined that a RSC of 50% was most appropriate for developing a drinking water MCL for PFNA. Use of a RSC of 50% for PFNA would increase the proposed drinking water standard from 21 ng/L to 53 ng/L. IEPA should evaluate the relative source contributions for each of the PFAS for which it develops a groundwater quality standard. Chemical-specific data is available for some of the PFAS for which IL EPA has proposed groundwater quality standards.

D. Basis for Standards Development

Overall, more information is needed to document the technical basis for each of the proposed standards. The procedures described in the regulations, including Appendices A and B of Title 35, are not sufficient to understand the basis of the proposed standards. It is not possible to track the basis of all of the numbers simply by following what is documented in the subject Appendices of the regulations.

1. **We recommend that Illinois document the toxicity value and any other values used to calculate the standards** and provide that supporting information in the regulation so that the technical

[2] https://hero.epa.gov/hero/index.cfm/reference/details/reference_id/3603279
[3] https://www.epa.gov/pfas/interim-recommendations-addressing-groundwater-contaminated-pfoa-and-pfos
[4] PFBA Draft Toxicity Assessment. Available from: https://www.epa.gov/pfas/genx-and-pfbs-draft-toxicity-assessments

basis for each standard is transparent. For example, if the standard is based on the MCL or MCLG, IEPA should document the value and its source. If the value is calculated using a toxicity criterion and a relative source contribution (RSC) factor, IEPA should document the toxicity value used in the calculation, the source of that value, and the assumed RSC. IEPA also should provide the rationale for and basis of any differences between Class I and Class II standards. Without this information, the technical soundness of the criteria cannot be fully evaluated.

For example:

- IEPA states that MCLs/MCLGs are used as the basis for Class I standards, when available. Yet that is not the case for the proposed Class I standards for fluoride, lead, selenium, and copper, which differ from (and are lower than) the MCLs. The rationale for those differences should be clearly documented.

- IEPA states that it uses USEPA noncancer reference doses and a default RSC of 0.2 to calculate Class I standards when MCLs/MCLGs are not available. Yet, a simple comparison of the proposed Class I standards to risk screening levels (RSLs) developed by USEPA for drinking water exposures shows that has not been done consistently. Given the differences in the ingestion rate and body weight assumed by USEPA and IEPA and the additional application of an RSC term by IEPA, the proposed Class I standards would be lower than USEPA's ingestion only RSLs, however this is not the case for several noncarcinogens (e.g., 1,3,5-trinitrobenzene and HMX). The basis for these differences should be documented.

- IEPA should document the sources of and the values used for the cancer slope factors (CSFs) included in the calculations for potential carcinogenic chemicals. The information provided in the Appendices does not identify the preferred source for CSFs used for deriving Class I criteria for carcinogens. A simple comparison of the proposed Class I standards to USEPA's drinking water RSL (ingestion only) values for potential carcinogens indicates that IEPA either did not use USEPA CSFs or used some other undefined factors in their calculation (e.g., Class 1 criteria for carcinogenic PAHs are more than 3-fold higher than EPA's RSL for ingestion only).

- IEPA should provide an explanation of how the endpoint of concern (i.e., cancer vs. noncancer) is selected when deriving Class I criteria. One would assume that when toxicity criteria are available from USEPA for cancer and noncancer endpoints that the more sensitive endpoint would be selected as the basis for calculating a Class I criteria. This does not appear to be the case in all instances. For example, the Class I criteria for 2,4,6-Trinitrotoluene appears to be based on the noncancer endpoint, however a Tier 1 EPA CSF for this compound is available and results in a lower risk-based level (as indicated by USEPA's RSL for this compound).

2. **IEPA should provide a chemical-specific analysis to support its use of an RSC of 0.2** in calculation of Class I values. Appendix A states that a default relative source contribution (RSC) of 20% should

be used in calculating Class I criteria for non-carcinogens, unless chemical-specific data are available. The RSC is based on the assumption that only 20% of a person's daily exposure to a chemical is via drinking water, and that the rest is from other sources, such as diet. There is no evidence that this assumption is valid for any of the regulated chemicals, and it is likely that a chemical-by-chemical analysis would document that it is invalid in many instances. For example, many chemicals are unlikely to be present in the diet, which is an important source of non-drinking water exposure factored into the RSC. In these cases, drinking water exposures could represent up to 100% of the exposures, and the calculated Tier 1 standards could be 5 times higher. A reconsideration of the use of a default RSC of 0.2 is warranted.

3. **IEPA should provide an explanation of how Class II (for General Resource groundwater) criteria** are derived. No explanation is provided, nor does there appear to be consistency in the criteria proposed. For some chemicals with an MCL available the MCL appears to be adopted for the Class II criteria, whereas for others it is not. Some Class II criteria look to be set equal to the Class I criteria, whereas others are lower, and still others are higher.

E. Background Update/Unified Guidance

Reference to the Unified Guidance (2009) is a good addition to the rule as long as it allows the regulated entity to apply any of the methods that are applicable within the guidance document.

1. **Using the Unified Guidance (2009) should be strongly advocated by IEPA** as its use will provide statistical flexibility and more up to date statistical approaches to be followed both when developing and updating background at a facility and developing the most appropriate statistical limits to satisfy permit conditions. Finally, the application of the Unified Guidance (2009) procedures should be synchronized with other State regulatory programs such as Rule 811 to be as effective as possible to all the regulated community.

F. References

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January 28, 2020

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Dear Stephanie,

We are pleased to submit our enclosed comments regarding the proposed changes to the language of 35 Ill. Adm. Code 620: Groundwater Quality. If you have any questions regarding our comments please feel free to contact us. We appreciate the Agency allowing us the opportunity to comment on the proposed changes and we look forward to the next step in the rulemaking process.

Sincerely,

Julie Rada
Laboratory Director

Scott D. Siders
Scott Siders
Director of Quality Assurance

Enclosure

PDC Laboratories, Inc.'s Comments on Draft Part 620

PDC Laboratories, Inc.'s comments on the draft Part 620, dated 12-19-2019, are as follows:

1. (Section 620.110 Definitions)

The definition for "Method Detection Limit" is obsolete and needs replaced with the current definition in Appendix B to Part 136:

"The method detection limit (MDL) is defined as the minimum measured concentration of a substance that can be reported with 99% confidence that the measured concentration is distinguishable from method blank results."

The definition for and use of the term "Method Quantitation Limit" or "MQL" may not be relevant to Part 620 as it does not appear to be used in the document and is a rather dated term.

The definition for and use of the term "Practical Quantitation Limit" or "PQL" may not be relevant to Part 620 as it is a dated term and SW-846 has moved to using the term "Lower Limit of Quantitation" or "LLOQ".

2. (Section 620.605 Issuance of a Health Advisory)

Removing the below language regarding PQL is a positive change, as laboratories will not be constrained by the somewhat arbitrary (one size fits all) and outdated PQLs found in the older versions of SW-846 methods. This proposed change is especially helpful since SW-846 has only one draft PFAS testing method and other methods are still under development by the USEPA. The consumer of testing services will need to make sure the laboratory they use has MDLs and verified LLOQs (SW-846) or LOQ (2016 TNI Standard) for each constituent that meets their testing and reporting needs and those of the applicable regulatory program, like Part 620.

~~unless the concentration for such substance is less than the lowest appropriate PQL specified in "Test Methods for Evaluating Solid Wastes, Physical/Chemical Methods," EPA Publication No. SW-846 (SW-846), incorporated by reference at Section 620.125 for such substance. If the concentration for such substance is less than the lowest appropriate PQL for the substance specified in SW-846, the guidance level is the lowest appropriate PQL.~~

3. (Section 620.410 QWQS for Class I: Potable Resource Groundwater)

PDC Laboratories, Inc. can meet all of the proposed Inorganic Chemical Constituents reporting levels (standard). However, the Vanadium Class I report level of 0.00049 mg/L is close to our current MDL of 0.00025 mg/L. Additional method development may allow us to achieve lower sensitivity and MDL for Vanadium and hence a lower reporting level. However, we request the IEPA provide a justification or explanation of the basis for lowering the standard for Vanadium from its current standard to the proposed 0.00049 mg/L. This is a 100X lowering of the standard and the closer a report level gets to the MDL (<3X MDL) the greater uncertainty or confidence in the test result.

4. (Section 620.410 QWQS for Class I: Potable Resource Groundwater and Section 620.420 GWQS for Class II: General Resource Groundwater)

PDC Laboratories, Inc. has only commented on the Organic Chemical Constituents report levels that have the potential for requiring some changes or further method development at our laboratory. Every other reporting level (standard) on the list can be assumed to be something our laboratory should be able to achieve with existing instrumentation and methodology. Our examination of the proposed Organic Chemical Constituents was approached from two perspectives, do we internally have a way to produce results at the proposed levels and what might our internal changes need to be to accommodate. A laboratory would also need to internally look at the cost of making some of the needed changes should they be required (e.g. an additional extraction/analysis required by additional methods in order to meet required limits. The four Organic Chemical Constituents we identified as such are:

A. Dibenzo(a,h)anthracene (Class I: 0.000085 mg/L, Class II: 0.00043 mg/L)
    1. Class 1 limit: We do calibrate below this level by SW-846 method 8310 so could achieve if required. At most would require changes in MDL study spiking level. Would likely not be attainable by SW-846 method 8270.
    2. Class II limit: Could be achieved with current SW-846 method 8310 analysis. Level is between our MDL and the minimum reporting level for SW-846 method 8270.
B. Hexachlorocyclohexane, alpha (Class I: 0.000014 mg/L)
    1. Below current low calibration standard level (0.05 ug/L), but with increased sensitivity of our new gas chromatogram this should be a level we could attain. Would require further testing to confirm.
    2. Would not be attainable by SW-846 method 8270 analysis.
C. 1-Methylnaphthalene (Class I: 0.49 mg/L, Class II: 2.5 mg/L)
    1. Unknown, as we do not currently analyze for this compound. Reasonable to expect this would be something we could do and at this level. Would require standards for further testing.
D. Diaminochlorotriazine (Under Total Atrazine and metabolites)

1. Not something, we currently analyze for. Based on preliminary searches it appears availability of material/standards for testing is currently very limited.

5. Sampling and Analytical Methodologies

Sampling and laboratory analytical methods have not been established for more complex leachate, soil and groundwater matrices. Laboratories must rely on their own modified analytical methods for analyzing these matrices. Modified methods vary from laboratory to laboratory.

Need to establish sampling and laboratory analytical methods for leachate, soil and groundwater that will ensure data of sufficient quality and consistent results. As of the date of this submittal, the industry as a whole is waiting on SW-846 method 8328 and Office of Water method 1600 to be issued by the USEPA.

PDC Technical Services, Inc.
4349 Southport Road, P.O. Box 9071
Peoria, Illinois 61615
309.676.4893
www.pdcarea.com

February 27, 2020

Sara Terranova, Part 620
Illinois Environmental Protection Agency
1021 North Grand Avenue East
P.O. Box 19276
Springfield, IL 62794-9276

Dear Sara:

We are pleased to submit our enclosed comments regarding the proposed changes to the language of 35 Ill. Adm. Code 620: Groundwater Quality. If you have any questions regarding our comments please feel free to contact us. We appreciate the Agency allowing us the opportunity to comment on the proposed changes and we look forward to the next step in the rulemaking process.

Sincerely,

George L. Armstrong P.E.
Vice President—Engineering and Consulting Services

Charles Hostetler, Ph.D.
Director of Environmental Services

Enclosure: PDC Technical Services, Inc. Comments

PDC Technical Services, Inc.'s comments on the draft Part 620, dated 12-19-2019, are as follows:

Section 620.410 QWQS for Class I: Potable Resource Groundwater and Section 620.420 GWQS for Class II: General Resource Groundwater

1. Proposed PFAS standards will affect existing practices and procedures used by the solid waste industry

   In the state of Illinois, Municipal Solid Waste Landfills (i.e. landfills regulated under 35 IAC 811) must complete a Groundwater Impact Assessment (GIA) prior to initial permitting to demonstrate that the landfill will have no effect on groundwater quality for a period extending 100 years following landfill closure. The IEPA requires that the GIA models used to permit landfills assume that the landfill has a defective liner system. A key component of GIAs is the concentration of each constituent in leachate. The initial GIA is based on assumed concentrations of a long list of pollutants in leachate. Actual leachate concentrations are reviewed as part of each 5-year permit renewal application and, if they are greater than assumed in the initial GIA, additional modeling or computations are required. It has been reported that PFAS in landfill leachate have been detected at levels greater than 3,500 ppt (Lang, et al. 2017). Considering the proposed PFAS standards, and the anticipated high concentrations of PFAS in leachate, until the PFAS compound fate and transport mechanisms are better understood, we have concern whether or not any landfill GIA would pass under the assumption of a defective liner system, or if the typical models used for GIAs are stable to the proposed concentrations. Further, it is reasonable to assume high laboratory reporting limits of PFAS in leachate due to analytical (matrix) interferences. Industry practice is to assume that leachate parameters that were not detected are present at the reporting limit. Will GIA models demonstrate satisfactory results if the high reporting limits are used, or only at relatively low PFAS concentrations?

   Illinois is the only state in the United States which requires a GIA in a landfill's siting, initial operating permit, and permit renewal application processes. Therefore, the concerns described above are unique to the solid waste industry in the state. It is acknowledged that the proposed PFAS standards are not as conservative as those promulgated by some state agencies (e.g., Michigan, New Jersey); however, it is unclear if the agency accounted for the state-specific requirements and the implementability of the proposed standards for the solid waste industry.

Older, closed sanitary landfills are regulated under 35 IAC 807. Many of these landfills are owned by municipalities, and are nearing the end of their post-closure care period. Prior to being released from post-closure care, the IEPA Bureau of Land requires that the groundwater monitoring wells be sampled and analyzed for all constituents for which a groundwater quality standard has been established at 35 IAC 620. Considering the very low concentration standards that are being proposed and the ubiquitous nature of PFAS compounds, detection of PFAS at concentrations greater than the 620 standards will likely result in significant additional costs to these legacy landfill owners, even after years of satisfactory groundwater monitoring results.

2. There is significant uncertainty associated with the environmental health risks associated with PFAS compounds and, in particular Acceptable Daily Exposure (ADE) values used in calculating the Human Threshold Toxicant Advisory Concentration (HTTAC), as described in 35 Ill. Adm. Code 620, Appendix A.

The Centers for Disease Control and Prevention states "The human health effects from exposure to low environmental levels of PFOA are unknown….More research is needed to assess the human health effects of exposure to PFOA" (https://www.cdc.gov/biomonitoring/PFOA_FactSheet.html, downloaded 2/14/2020). Similarly, the National Institute of Environmental Health Sciences states "More research is needed to fully understand all sources of exposure, and if and how they cause health problems", "The research conducted to date reveals possible (emphasis added) links between human exposures to PFAS and adverse health outcomes.", and "While knowledge about the potential health effects of PFAS has grown, many questions remain unanswered" (https://www.niehs.nih.gov/health/topics/agents/pfc/index.cfm, accessed February 19, 2020).

The available research regarding exposure to PFAS has created a large disparity in the federal and state advisory levels promulgated by governing agencies. The variation is largely related to the different definitions of critical health effects and relative scarcity of human and mammalian studies. Human health studies are largely limited to populations of individuals who 1) have been occupationally exposed during the production or use of PFAS, 2) live in a community with high levels of PFAS measured in drinking water, or 3) have been exposed to background levels of PFAS. Mammalian studies are limited due to the difficulty of extrapolating results from a small animal population provided a controlled exposure dose to the human population in an uncontrolled environment. Further, the mammalian studies which have been conducted have not consistently defined the same critical health effects, making it more difficult to accurately determine an ADE value.The uncertainty associated with ADE values can dramatically shift groundwater standards. IEPA should review the endpoints of reference doses and critical health effects in available literature to determine the magnitude of differences between ADE values.

3. PFAS cleanup objectives are not provided as part of the proposed standards

IEPA does not provide cleanup objectives with the proposed standards. In an instance where PFAS compounds are detected in groundwater at a landfill, what are the expectations for corrective action? Will acceptable background concentrations be considered if PFAS is detectable in upgradient locations?

4. There has been an apparent lack of due process in the establishment of the proposed groundwater standards

It is not immediately apparent if peer reviews have been conducted on the proposed groundwater standards. If not, it should be considered imperative that IEPA conduct a peer review of their proposed standards to ensure that the Agency's standard development procedure is consistent with other regulatory agencies.

5. IEPA should consider the ubiquitous extent of PFAS compounds in groundwater and complete a statewide groundwater survey prior to promulgating regulatory standards

   Considering the apparent extent of PFAS in the environment, it is possible that non-attributable concentrations of PFAS compounds will be detected in groundwater upgradient and downgradient of landfills. The state of Illinois is currently conducting a statewide survey of drinking water systems. It is reasonable to conduct a statewide groundwater survey, similar in scope, to determine areas of known PFAS contamination. Development of such a database would provide data to support background analyses and support alternate source demonstrations in scenarios where detectable PFAS concentrations are not attributable to the landfill. At a minimum, IEPA should clarify how the presence of PFAS will be handled in situations not commonly associated with landfill operations (i.e., detectable concentrations in upgradient monitoring wells).

6. There is currently a lack of established analytical methods for more complex leachate, soil and groundwater matrices.

   Sampling and laboratory analysis methods have not been established for more complex leachate, soil and groundwater matrices. Laboratories must rely on their own modified analytical methods for analyzing these matrices. Modified methods vary from laboratory to laboratory. As of the date of this submittal, the solid waste industry is waiting on SW-486 method 8328 and Office of Water method 1600 to be issued by the USEPA. Inconsistent results between laboratories could result in analytical results that are not reproducible or defensible.

7. Drinking water standards should be promulgated before groundwater standards.

   The purpose of the Class I (Potable Resource) Groundwater Standards is to protect drinking water supplies. The IEPA should not propose Class I Groundwater Standards until after drinking water standards are established. Additionally, the proposed Class I Groundwater Standards are based on concentrations in water that is consumed, and does not factor the probability of whether or not groundwater classified as Class I at any one location will ever be consumed as drinking water, nor does it factor contaminant fate and transport mechanisms. This is overly conservative considering that the vast majority of groundwater that is classified as Class I will never be used for drinking water.

8. Class II Groundwater is not used as a source of drinking water.

   The proposed Class II (General Resource) Groundwater Standards are identical to the proposed Class I (Potable Resource) Groundwater Standards. Class II Groundwater is generally not suitable for a drinking water supply. Class II Groundwater Standards should not be based on direct consumption, but rather should be based on protecting other drinking water supplies considering location and fate and transport mechanisms.

9. All regulated landfills in the State must either routinely test for all parameters for which 620 standards are established, or will be required to test for them prior to ending post-closure care.

   Considering the apparent ubiquitous extent of PFAS compounds, it is probable that PFAS compounds will be detected in groundwater upgradient and downgradient of landfills. Because it is a VOC, landfill gas could not automatically be ruled out as a contributor of PFAS in groundwater upgradient of a landfill. As a result, it could be a very expensive and lengthy process to demonstrate that the landfill is not the source of PFAS compounds in groundwater that will never be ingested.

10. IEPA prescribed groundwater monitoring device construction and practices may not be compatible with obtaining representative groundwater quality data consistent with the proposed standards.

    Many of the dedicated groundwater monitoring well sampling bailers and pumps that are currently in use were likely manufactured with PFAS-containing compounds, specifically Teflon. In some cases, Teflon well casing might also have been used, and/or other well construction materials might have inadvertently contained PFAS compounds. It is possible that PFAS compounds from this equipment could have leached into groundwater making it difficult to distinguish the source of extremely low concentrations of PFAS compounds. Additionally, it would be very costly to replace all dedicated sampling pumps, and possibly groundwater monitoring wells themselves, using equipment and supplies that can be certified free of PFAS compounds.

11. PFAS-containing waste acceptance criteria are little understood.

    A better understanding of which wastestreams exhibit high concentrations of PFAS compounds (e.g. remediation wastes, municipal and industrial wastewater sludges, etc.) is needed before imposition of the groundwater standards. Unduly stringent groundwater standards could create an inappropriate lack of disposal capacity for such wastes.

PDC Laboratories, Inc.
2231 W. Altorfer Drive • Peoria, IL 61615
(309) 692-9688 • (800) 752-6651 • FAX (309) 692-9689

January 28, 2020

Sara Terranova, Part 620
Illinois Environmental Protection Agency
1021 North Grand Avenue East
P.O. Box 19276
Springfield, IL 62794-9276

Dear Sara,

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Julie Rada

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Laboratory Director

Scott D. Siders

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From:	Bailey, Sabrina
To:	Terranova, Sara; Brown, Michael L.; Dunaway, Lynn; Frost, Brad; Lieberoff, Barb; Wake, Elizabeth; Guy, Jeff; Nifong, Heather; Diers, Stefanie; Sofat, Sanjay; Ankney, Clayton; Martin, Lauren; Hawbaker, Carol; Woods, Teschlyn; Irlam, Justin; Shaw, Melinda; Wilson, Nicole; Dunn, Greg; Summers, Michael
Subject:	Re: 620 Questions and Comments End of Comment Period
Date:	Monday, June 28, 2021 8:27:19 AM
Attachments:	620 Comments and Questions.docx 620 Comments and Questions.zip

Good Morning All,

There is a total of sixteen responses to the proposed 620 rule changes. The word document attached contains a list of comments/ questions and file names for each of the respondents. The folder contains the pdf files with comments and supplemental information sent by eleven of the sixteen respondents.

Sabrina Bailey, PhD
Office of Community Relations
Illinois EPA
(847) 294-4394
Sabrina.Bailey@illinois.gov

From: Bailey, Sabrina <Sabrina.Bailey@Illinois.gov>
Sent: Tuesday, June 22, 2021 2:47 PM
To: Terranova, Sara <Sara.Terranova@Illinois.gov>; Brown, Michael L. <Michael.L.Brown@Illinois.gov>; Dunaway, Lynn <LYNN.DUNAWAY@Illinois.gov>; Frost, Brad <Brad.Frost@Illinois.gov>; Lieberoff, Barb <Barb.Lieberoff@Illinois.gov>; Wake, Elizabeth <Elizabeth.Wake@Illinois.gov>; Guy, Jeff <Jeff.Guy@Illinois.gov>; Nifong, Heather <Heather.Nifong@Illinois.gov>; Diers, Stefanie <Stefanie.Diers@Illinois.gov>; Sofat, Sanjay <Sanjay.Sofat@Illinois.gov>; Ankney, Clayton <Clayton.Ankney@Illinois.gov>; Martin, Lauren <Lauren.Martin2@Illinois.gov>; Hawbaker, Carol <Carol.Hawbaker@Illinois.gov>; Woods, Teschlyn <Teschlyn.Woods@Illinois.gov>; Irlam, Justin <Justin.Irlam@Illinois.gov>; Shaw, Melinda <Melinda.Shaw@illinois.gov>; Wilson, Nicole <Nicole.Wilson@Illinois.gov>; Dunn, Greg <Greg.Dunn@Illinois.gov>; Summers, Michael <Michael.Summers@Illinois.gov>
Subject: 620 Questions and Comments 6/22/21

From: Donna Campbell, Client Relations Manager
Organization: Eurofins TestAmerica
Date: 6/22/21

Comments.

- The new standard for Vanadium of 0.00027 mg/l is not achievable by 6020A ICP-MS, which is the industry-standard for meeting lower level metals limits. This limit is over 10x lower than what can typically be met with this methodology.

- Dibenzo(a,h)anthracene at 0.000025 mg/l is not achievable by 8270D, 8270D LL or 8270D SIM. Again, this limit is over 10x lower than what can typically be met with these methodologies. **Question:** Is it possible that one to many zeros to the right of the

decimal place were added?

Sabrina Bailey, PhD
Office of Community Relations
Illinois EPA
(847) 294-4394
Sabrina.Bailey@illinois.gov

From: Bailey, Sabrina <Sabrina.Bailey@Illinois.gov>
Sent: Wednesday, June 9, 2021 8:33 AM
To: Terranova, Sara <Sara.Terranova@Illinois.gov>; Brown, Michael L. <Michael.L.Brown@Illinois.gov>; Dunaway, Lynn <LYNN.DUNAWAY@Illinois.gov>; Frost, Brad <Brad.Frost@Illinois.gov>; Lieberoff, Barb <Barb.Lieberoff@Illinois.gov>; Wake, Elizabeth <Elizabeth.Wake@Illinois.gov>; Guy, Jeff <Jeff.Guy@Illinois.gov>; Nifong, Heather <Heather.Nifong@Illinois.gov>; Diers, Stefanie <Stefanie.Diers@Illinois.gov>; Sofat, Sanjay <Sanjay.Sofat@Illinois.gov>; Ankney, Clayton <Clayton.Ankney@Illinois.gov>; Martin, Lauren <Lauren.Martin2@Illinois.gov>; Hawbaker, Carol <Carol.Hawbaker@Illinois.gov>; Woods, Teschlyn <Teschlyn.Woods@Illinois.gov>; Irlam, Justin <Justin.Irlam@Illinois.gov>; Shaw, Melinda <Melinda.Shaw@illinois.gov>; Wilson, Nicole <Nicole.Wilson@Illinois.gov>; Dunn, Greg <Greg.Dunn@Illinois.gov>; Summers, Michael <Michael.Summers@Illinois.gov>
Subject: Re: 620 Questions and Comments 6/9/21

Good Morning All,
Below are comments from Illinois American Water.

From Rachel Bretz, Director of Water Quality and Environmental Compliance
Organization: Illinois American Water
Comment:

- included PFAS (PFBS, PFHxS, PFNA, PFOA, PFOS) in both Class I and II groundwater limits
- Levels are slightly different than the drinking water HALs they established (Table below)

Acronym		Health-Based Guidance Level	Groundwater Quality Standard **Proposed**
		(ng/L)	**(ng/L)**
Perfluorobutanesulfonic acid	PFBS	2,100*	1200
Perfluorohexanesulfonic acid	PFHxS	140	77
Perfluorooctanesulfonic acid	PFOS	14	7.7
Perfluorooctanoic acid	PFOA	2	2
Perfluorohexanoic acid	PFHxA	560,000	NONE
PFNA (perfluorononanoic acid)	PFNA	NONE	12

Sabrina Bailey, PhD
Office of Community Relations
Illinois EPA
(847) 294-4394
Sabrina.Bailey@illinois.gov

From: Bailey, Sabrina
Sent: Wednesday, May 26, 2021 11:35 AM
To: Terranova, Sara <Sara.Terranova@Illinois.gov>; Brown, Michael L. <Michael.L.Brown@Illinois.gov>; Dunaway, Lynn <LYNN.DUNAWAY@Illinois.gov>; Frost, Brad <Brad.Frost@Illinois.gov>; Lieberoff, Barb <Barb.Lieberoff@Illinois.gov>; Wake, Elizabeth <Elizabeth.Wake@Illinois.gov>; Guy, Jeff <Jeff.Guy@Illinois.gov>; Nifong, Heather <Heather.Nifong@Illinois.gov>; Diers, Stefanie <Stefanie.Diers@Illinois.gov>; Sofat, Sanjay <Sanjay.Sofat@Illinois.gov>; Ankney, Clayton <Clayton.Ankney@Illinois.gov>; Martin, Lauren <Lauren.Martin2@Illinois.gov>; Hawbaker, Carol <Carol.Hawbaker@Illinois.gov>; Woods, Teschlyn <Teschlyn.Woods@Illinois.gov>; Irlam, Justin <Justin.Irlam@Illinois.gov>; Shaw, Melinda <Melinda.Shaw@illinois.gov>; Wilson, Nicole <Nicole.Wilson@Illinois.gov>; Dunn, Greg <Greg.Dunn@Illinois.gov>; Summers, Michael <Michael.Summers@Illinois.gov>
Subject: 620 Questions and Comments

Good Morning All,
Attached are comments and questions concerning 620 proposed changes. I will send a daily update of the comments in word, and they will be added to an excel spreadsheet that will be updated weekly and shared.

Sabrina Bailey, PhD
Office of Community Relations
Illinois EPA
(847) 294-4394
Sabrina.Bailey@illinois.gov

620 Questions and Comments

From: Katie Pelch, PhD
Organization: Natural Resources Defense Council
Date: 5/25/21

1) Are there technical support documents available for the proposed groundwater quality standards?
2) how are these related to the health based guidance levels available at: https://www2.illinois.gov/epa/topics/water-quality/pfas/Documents/HA%20PFOS.pdf Some of the values are different between the health based values and the groundwater quality standards and I'd like to better understand where this difference derives from.
3) If you have information on the health based values (or know who I should contact), I'm curious why there isn't a value for PFNA, though it is mentioned on the page and there was a draft value for PFNA available in January 2020 and there seems to be a groundwater quality standard recommended for PFNA?

Comment: I'm unclear if these questions will be addressed or not at tomorrow's public meeting and would appreciate any further clarification you could provide.

From: Daniel Lombardi, Principal Hydrologist
Organization: St. John-Mittelhauser & Associates, Inc.
Date: 5/25/21

1) What was the basis for having the same groundwater quality criteria for the five new PFA compounds and 1,4-Dioxane be the same for **both** Class I and Class II groundwater?
Comment: These new Class II standards should not be subject to the same Class I standards for those occurrences where groundwater is not used for potable sources of drinking water. I believe there would be a lower risks relating to Class II groundwater and the new criterial should be changed to account for it.

From Rachel Bretz, Director of Water Quality and Environmental Compliance
Organization: Illinois American Water
Date: 6/9/21
Comment:

- included PFAS (PFBS, PFHxS, PFNA, PFOA, PFOS) in both Class I and II groundwater limits
- Levels are slightly different than the drinking water HALs they established (Table below) ☐

Acronym		**Health-Based Guidance Level**	Groundwater Quality Standard **Proposed**
		(ng/L)	**(ng/L)**
Perfluorobutanesulfonic acid	PFBS	2,100*	1200
Perfluorohexanesulfonic acid	PFHxS	140	77
Perfluorooctanesulfonic acid	PFOS	14	7.7
Perfluorooctanoic acid	PFOA	2	2
Perfluorohexanoic acid	PFHxA	560,000	NONE
PFNA (perfluorononanoic acid)	PFNA	NONE	12

From Ground Water Advisory Council Member
Organization:
Date:6/11/21
Questions:

- Information on how the proposed changes compares to other Region 5 states?
- Information on how the proposed PFAS values compare to recent work from USEPA?

Response: Carol Hawbaker
ITRC which has the most comprehensive information on it regarding other states data. It is located at: https://pfas-1.itrcweb.org/fact-sheets/
Under the "Regulations" bullet (PFAS Water and Soil Values Table Excel File). The Excel file units are in µg/L (and cover many chemicals not included in the proposed updates to 620, so I'll condense here):

☐

Region 5 State	Type (GW/DW)	Promulgated Rule (Y/N)	PFBS (ng/L)	PFHxS (ng/L)	PFNA (ng/L)	PFOA (ng/L)	PFOS (ng/L)
Illinois Proposed	GW		1,200	77	12	2	7.7
Indiana	GW	Y (2019)	400,000	----	----	----	----
Michigan	DW/GW	Y (2021)	420	51	6	8	16
Minnesota	DW/GW	*See Note Below	2,000	47	----	35	15
Ohio	DW	N (2019)	140,000	140	21	70**	70**
Wisconsin	GW	N	450,000	40	30	20**	20**

ota has promulgated rules (2018) with chronic Health Risk Limit (HRL) values for PFOA = 35 ng/L, PFOS = 300 ng/L and PFBS = 7,000 ng/L. In 2019, Minnesota proposed updated Health Based Values (HBVs) for PFOS = 15 ng/L and PFBS = 2,000 ng/L and introduced an HBV for PFHxS = 47 ng/L. The proposed HBVs are not promulgated.
** Guidance levels based in individual or combined FPOA/PFOS level of 70 ng/L for Ohio, and 20 ng/L for Wisconsin.

Note, the units in the above table are ng/L or ppt. For reference:
mg/L = ppm
µg/L = ppb
ng/L = ppt

The proposed values use the recently released final toxicity values for PFBS (PPRTV in May 2021), PFHxS, PFNA, PFOA, and PFOS (all ATSDR in May 2012) for non-cancer evaluations. However, in the case of PFOA, the only PFAS meeting the Act's definition of a carcinogen, the cancer value is more stringent than the non-cancer value. Therefore, the PFOA cancer value, using California EPA's cancer toxicity value, is more stringent.

From: Donna Campbell, Client Relations Manager
Organization: Eurofins TestAmerica
Date: 6/22/21

Comments:

- The new standard for Vanadium of 0.00027 mg/l is not achievable by 6020A ICP-MS, which is the industry-standard for meeting lower level metals limits. This limit is over 10x lower than what can typically be met with this methodology.

- Dibenzo(a,h)anthracene at 0.000025 mg/l is not achievable by 8270D, 8270D LL or 8270D SIM. Again, this limit is over 10x lower than what can typically be met with these methodologies. **Question:** Is it possible that one to many zeros to the right of the decimal place were added?

From: Mike Travis, Corporate Director of Quality Assurance & Julie Rada Lab Director
Organization: PDC Laboratories Inc.
Date: 6/24/21
Comments: See file Mike Travis PDC Laboratories

From: Charles Hostetler, PhD, Director of Environmental Sciences
Organization: PDC Technical Services Inc.
Date: 6/24/21
Comments: See file Charles Hostetler PDC Technical Services

From: Janet Anderson, PhD, Principal Toxicologist
Organization: GSI Environmental Inc
Date: 6/25/21
Comments: See file Janet Anderson GSI Environmental

From: Sandra Carey, HSE Executive
Organization: International Molybdenum Association (IMOA)
Date: 6/25/21
Comments: See file Sandra Carey IMOA

From: Steve Risotto, Senior Director
Organization: American Chemistry Council, Senior Director
Date: 6/25/21
Comments: See 4 files
Steve Risotto 1 Comments American Chemistry Council
Steve Risotto 2 Comments American Chemistry Council
Steve Risotto 3 Lit Article American Chemistry Council
Steve Risotto 4 Lit Article American Chemistry Council

From: Alec Davis, Executive Director
Organization: Illinois Environmental Regulatory Group (IERG)
Date:6/25/21
Comments: **See** file Alec Davis IERG Comments

From: James Morphew, Attorney
Organization: Sorling Northrup Attorneys/National Waste and Recycling Association
Date:6/25/21
Comments: **See f**ile Sorling Law and National Waste Recycling Assoc

From: Ashley Parr. Associate
Organization: Barnes & Thornburg LLP / PFAS Regulatory Coalition
Date:6/25/21
Comments: See file Ashley Parr Barnes Thornburg PFAS Reg Coalition

From: Kristen Gale, Partner
Organization: Nijman Franzetti LLP/Midwest Generation LLC
Date:6/25/21
Comments: See file Kristen Gale Nijman Franzetti Midwest LLC **(Same as Janet Anderson)**

From: Iyana Simba, Clean Water Policy Director
Organization: Natural Resources Defense Council/ Sierra Club
Date:6/25/21
Comments: See file Iyana Simba Natural Resource Defense Council Sierra Club

From: Richard Burrows, PhD, Technical Director
Organization: Eurofins Environmental Test America
Date:6/25/21
Comments: See file Richard Burrows Eurofins Environmental TestAmerica

Illinois Environmental Regulatory Group
An Affiliate of the Illinois Chamber of Commerce

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June 25, 2021

Brad Frost
Manager
Office of Community Relations
Illinois Environmental Protection Agency
1021 North Grand Avenue East, P.O. Box 19276
Springfield, IL 62794-9276

Submitted Electronically to:
EPA.620.rulemaking@illinois.gov

Mr. Frost:

Please accept the below comments on behalf of the members of the Illinois Environmental Regulatory Group (IERG) regarding the Illinois EPA's draft proposed amendments to 35 Ill. Adm. Code Part 620: Groundwater Quality Standards, shared on May 12, 2021, and presented during the Agency's May 26th stakeholder meeting. IERG participated in the meeting and appreciates the Agency's efforts to address questions raised, however some concerns and questions remain. IERG's remaining concerns and recommendations are outlined below.

Justification of Draft PFAS Standards

Based on a comparison of the draft PFAS standards to standards adopted in other states, it is apparent that Illinois would have some of, if not the most stringent standards for some PFAS chemicals in the country. IERG questions whether the Agency has identified some new science or understanding of the threats posed by these PFAS chemicals that were unknown to other states, or if some assumptions were made differently in calculating the draft PFAS standards than was the case in other states. IERG is concerned that the level of the standards proposed could potentially result in many detections above that level throughout the state, creating potential liability for numerous entities and raising questions and concerns from the general public about threats to their health.

It was clear from the May 26 stakeholder meeting that there are numerous outstanding questions regarding the rationale and analysis behind the draft PFAS standards. The Agency indicated that the information would be made available with the proposal to the Illinois Pollution Control Board (Board). **IERG suggests it would be advantageous to the Agency and helpful to the regulated community to share its basis and rationale for its draft PFAS standards prior to proposing the standards to the Board.**

Measuring Draft PFAS Standards

IERG is aware that there are questions regarding the ability of Illinois commercial laboratories to detect some PFAS chemicals reliably at the levels contained in the Agency's draft standards. IERG is concerned that some of the detection limits that are theoretically achievable based on published methods may be difficult to achieve in practice, and that methods adopted for certain sample types (i.e., drinking water) may not be applicable or accurate when used for other types of samples (i.e., raw water or potentially polluted groundwater). **IERG requests that the Agency provide information regarding the availability of existing laboratories to accurately and reliably test samples and provide the regulated community with results relative to the draft PFAS standards levels.**

PFAS in Illinois' Environment

IERG has been closely following the Illinois EPA's community water supply sampling program for PFAS chemicals, and applauds the Agency for its efforts to better understand the scope of PFAS chemicals' presence in the environment. IERG does, however, have some concerns that the study has not yet been completed, and that preliminary results indicate that PFAS chemicals are fairly widespread in the environment such that it is being found in treated drinking water. As of the date of this comment, the Agency's sampling program has detected PFAS chemicals at a detectable level at nearly 9% of sampled water supplies across the state. Of those, approximately half of the detections were found to be above the Agency's "Health Based Guidance Level." A finding that 5-10% of water supplies across the state have PFAS chemicals is significant. IERG is not aware of efforts to study the presence of PFAS chemicals in untreated water or non-community water supply waters.

IERG has also recently become aware of ongoing work studying levels of PFAS chemicals found in precipitation, and that although publication of those studies has not yet occurred, based on preliminary reporting, concentrations are being found far in excess of the Agency's draft standards. This appears to indicate that PFAS are pervasive in the environment and identifying a source or background values when PFAS are found to be exceeded may prove difficult. IERG encourages the Agency to proceed with caution in adopting groundwater quality standards that will invite questions and concern from the general public about the health effects of rainwater but also complicate determinations of the source or fate of PFAS chemicals found in surface and groundwaters. See, for example:

https://cen.acs.org/acs-news/acs-meeting-news/US-rainwater-contains-new-and-phased-out-PFAS/99/web/2021/04

https://grist.org/science/its-raining-forever-chemicals-in-the-great-lakes/

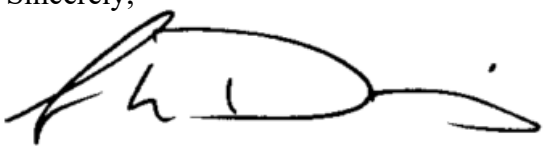
In sum, IERG is concerned that not enough is yet known about PFAS chemicals' presence in the environment in Illinois to understand the practical ramifications, including addressing the questions and concerns of the general public, of adopting the draft standards. **IERG encourages the Illinois EPA to consider concluding its statewide sampling study and to further investigate the prevalence of PFAS in Illinois prior to proposing groundwater quality standards.**

Federal PFAS Standard Development

Finally, U.S. EPA has recently announced a number of developments regarding PFAS chemicals and drinking water. Specifically, on February 22, 2021, U.S. EPA finalized its determination to regulate PFOA and PFOS under the Safe Drinking Water Act. *See* 86 Fed. Reg. 12272 (March 3, 2021). U.S. EPA has also announced expanded monitoring nation-wide for 29 additional PFAS chemicals under the Unregulated Contaminant Monitoring Rule (UCMR). *See* 86 Fed. Reg. 13846 (March 11, 2021). IERG is concerned that there is a high probability for public confusion regarding the State's efforts to regulate PFAS chemicals under Part 620 and federal action regarding the same chemicals. Additionally, in the event that inconsistent state and federal requirements are adopted and applicable to a given site or entity, IERG is concerned that the regulated community will be burdened with duplicative, overlapping, inconsistent requirements. **IERG recommends that the Illinois EPA reach out to and coordinate its efforts to regulate PFAS with U.S. EPA to understand its timeline and intent and better avoid the confusion and burdens described.**

Thank you for your consideration of the above comments.

Sincerely,



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June 25, 2021

VIA ELECTRONIC MAIL
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Re: Comments of the PFAS Regulatory Coalition on Proposed Rulemaking to Revise the Part 620 Groundwater Quality Regulations

Dear Sir or Madam:

The PFAS Regulatory Coalition (Coalition) appreciates the opportunity to file comments regarding the proposed revisions to Illinois' Part 620 groundwater quality regulations.

I. The Coalition's Interest

The Coalition is a group of industrial companies, municipal entities, agricultural parties, and trade associations that are directly affected by the State's development of policies and regulation related to per- and polyfluoroalkyl substances (PFAS). Coalition membership includes entities in the automobile, coke and coal chemicals, iron and steel, municipal, paper, petroleum, and other sectors. None of the Coalition members manufacture PFAS compounds. Coalition members, for purposes of these comments, include: Airports Council International – North America; American Coke and Coal Chemicals Institute; American Forest and Paper Association; American Fuel and Petrochemical Manufacturers; American Iron and Steel Institute; Barr Engineering; Brown & Caldwell; Gary Sanitary District (IN); Illinois Association of Wastewater Agencies; Lowell, MA; Pueblo, CO; Toyota; Trihydro, and Yucaipa Valley Water District (CA).

Coalition members support the State's efforts to set groundwater standards for those individual PFAS that pose risks to human health and the environment. In the State's pursuit of such regulations, the Coalition urges State regulators to ensure that final standards are scientifically supported, cost-effective, and achievable.

II. Proposed Rulemaking

On May 12, 2021, the Illinois Environmental Protection Agency (IEPA or State or Agency) proposed draft language to update 35 Ill. Adm. Code 620. The proposed updates include the addition of nine new chemicals, three new atrazine metabolites, and procedures for selecting toxicity values consistent with current federal guidance. The Coalition's comments address only the proposed revisions relating to PFAS compounds and IEPA's methodologies underlying the groundwater standards for PFAS. Notably, the proposal includes groundwater quality standards for the following PFAS:

- Perfluorobutane Sulfonic Acid (PFBS): 0.0012 mg/L
- Perfluorohexane Sulfonic Acid (PFHxS): 0.000077 mg/L
- Perfluorononanoic Acid (PFNA): 0.000012 mg/L
- Perfluorooctanoic Acid (PFOA): 0.000002 mg/L
- Perfluorooctane Sulfonic Acid (PFOS): 0.0000077 mg/L

Additionally, the proposed revisions to Section 620.310 include preventive response activities, including preventive notification mandates.

The PFAS Coalition has significant concerns and questions relating to the proposed standards, which are orders of magnitude lower than the standards the State initially proposed in December 2019. The Coalition recognizes that IEPA has updated its methodology for developing oral reference doses (RfDs), established a hierarchy for selecting verified RfDs, and updated exposure factors to reflect exposure of a child from 0 to 6 years of age as opposed to exposure of an average adult.[1] The Coalition appreciates IEPA's prioritization of USEPA data, where available, but the Agency's brief discussion of the changes to the rule is insufficient to explain the drastic difference from the standards proposed in December 2019 and the standards proposed currently. The Agency's discussion of the changes do not provide an adequate explanation of IEPA's methodology that would allow the public to independently evaluate the proposal. In this regard, the insufficiency of IEPA's proposal undermines the public's ability to comment and participate meaningfully in the rulemaking process.

As discussed below, the Coalition requests that the State reconsider its new proposal standards, through a more transparent process, towards developing standards that provide necessary protection of the State's groundwater resources without unreasonably burdening the regulated community with unnecessarily stringent standards.

[1] The Coalition disagrees with IEPA's decision to include age-adjusted water intake factors to account for increase cancer risk from childhood exposure for substances suspected of being mutagenic carcinogens. The oral slope factor (SFo) used in calculating the HNTAC is based on a default linear, low-dose extrapolation using a mutagenic mode of action. The Agency does not need to use age-adjusted exposure factors, as that level of conservatism is already included in the SFo derivation.

III. Coalition Analysis and Recommendations

In the comments below, the Coalition discusses some of the challenges that the State faces in attempting to promulgate enforceable regulations, as well as some of the challenges that Coalition members face if states promulgate standards that vary from any existing or future federal standards. The Coalition appreciates the State's desire to act to protect its citizens from potential risks associated with exposure to certain PFAS compounds, but urges Illinois and other states to work with the federal government to develop a cohesive national strategy to help ensure national uniformity. A patchwork set of state-specific standards that vary widely would likely cause significantly more confusion and overwhelming challenges for Coalition members that operate in multiple states or nationwide.

A. The Scientific Community Does Not Agree on Human Health Toxicity Values for PFAS

The term "PFAS" refers to a group of man-made chemicals that include perfluorooctanoic acid (PFOA), perfluorooctane sulfonic acid (PFOS), GenX,[2] and other fluorinated compounds. The most prevalent and available science regarding the incidence and potential health effects of PFAS is based on PFOA and PFOS, two compounds that are no longer manufactured in the United States due to voluntary phase outs over a decade ago. For replacement chemicals, industry has begun using shorter-chain PFAS that have different physical, chemical, and toxicological properties from long-chain PFOA and PFOS. The scientific understanding of how PFAS impacts people and the environment is still developing and, for thousands of PFAS compounds, much remains unknown. From a toxicological perspective, regulatory agencies must have adequate science for determining health-based values before promulgating individual-compound standards, limits, and related regulations.

Toxicologists, whether they work for various state agencies, USEPA, international standards-setting organizations, academia, or in private practice, have not yet established specific methodologies, resources, or even agreed on which of the hundreds of studies of PFAS compounds are the appropriate or critical studies that must or should support appropriate regulatory "standards." Different methodologies, levels of experience, procedural prerequisites to standards-setting, and even local political pressures are leading to consideration of very different standards in various states and at USEPA. The Coalition urges states to work with one another, and with USEPA, to continue developing science and methodologies to inform and encourage a more uniform approach to federal and state PFAS regulatory mandates.

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[2] Note that GenX is a trade name for a specific PFAS compound, ammonium, 2,3,3,3-tetrafluoro-2-(heptafluoropropoxy) propanoate. ITRC "Naming Conventions and Physical and Chemical Properties of Per- and Polyfluoroalkyl Substances (PFAS)," at 12, *available at* https://pfas-1.itrcweb.org/fact_sheets_page/PFAS_Fact_Sheet_Naming_Conventions_April2020.pdf (last visited June 24, 2021). More generically, GenX can be denoted by the abbreviation, "HFPO-DA."

B. Federal Action on PFAS

USEPA issued "Interim Recommendations for Addressing Groundwater Contaminated with PFOA and PFOS" in December 2019[3] Those recommendations provide clear and consistent guidance for federal cleanup sites being evaluated and addressed under federal programs, including the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) and the Resource Conservation and Recovery Act (RCRA). The screening levels recommended for such cleanups are risk-based values that are used to determine if levels of contamination may warrant further investigation at a site. The recommendations are intended to be used as guidance for states to evaluate state cleanup and corrective action sites. The interim guidance recommends in relevant part:

- Using a screening level of 40 parts per trillion (ppt) to determine if either PFOA, or PFOS, or both, are present at a site and may warrant further attention.
- Using USEPA's PFOA and PFOS Lifetime Drinking Water Health Advisory level of 70 ppt as the preliminary remediation goal (PRG) for contaminated groundwater that is a current or potential source of drinking water, where no state or tribal MCL or other applicable or relevant and appropriate requirements (ARARs) are available or sufficiently protective.

In addition, USEPA is focusing significant resources on developing appropriate regulatory mechanisms specific to various PFAS compounds. For example, USEPA has developed a PFAS Action Plan, which provides a multi-media, multi-program, national research and risk communication plan to address emerging PFAS challenges.[4] Part of USEPA's PFAS Action Plan involves expanding the scientific foundation for understanding and managing risk from PFAS, including researching improved detection and measurement methods, generating additional information about PFAS presence in the environment, improving the understanding of effective treatment and remediation methods, and developing more information regarding the potential toxicity of a broader set of PFAS. In turn, USEPA expects that this information will help states and others better manage PFAS risks. To bolster this work, USEPA Administrator Regan established the PFAS Action Council on April 27, 2021.[5]

While we recognize that not all states and stakeholders can agree on specific priorities or approaches to PFAS regulations, USEPA and Congress are leading important

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[3] USEPA Office of Land and Emergency Management, OLEM Directive No. 9283.1-47 (December 19, 2019), *available at* https://www.epa.gov/sites/production/files/2019-12/text_version_epas_interim_recommendations_for_addressing_groundwater_contaminated_with_pfoa_and_pfos_dec_2019.txt.

[4] *See* USEPA "EPA's Per- and Polyfluoroalkyl Substances (PFAS) Action Plan" (February 2019) *available at* https://www.epa.gov/sites/production/files/2019-02/documents/pfas_action_plan_021319_508compliant_1.pdf.

5 *See* Memorandum Regarding Per- and Polyfluoroalkyl Substances (April 27, 2021) *available at* https://www.epa.gov/pfas/memo-epa-council-pfas.

national initiatives that states should support through their contribution of expertise, resources, and efforts as the United States works to respond to PFAS exposure risks. Indeed, a patchwork of 50 different state solutions is unworkable and contrary to how the U.S. has previously addressed similar emerging-contaminant issues. While some limited variations related to groundwater, surface water, or soil cleanup levels may be expected and appropriate, the highly variable regulatory health advisories, action levels, and numeric standards currently being developed or under consideration across the country create unnecessary confusion and complexity for the public and the regulated community.

The Coalition recognizes that states have elected to utilize different methods and processes for communicating risks to their populations. However, standards-setting must reflect more national and uniform collaboration and cohesion. We must work to avoid the undesirable solution of 50 separate state rules. With this in mind, we urge the states to work closely with USEPA to establish science-based and peer-reviewed federal standards that serve as the basis for comparable state standards. Such an approach is consistent with how USEPA and the states have addressed environmental and human health risks since the creation of USEPA.

C. Transparency of IEPA's Proposal

It is not possible to discern from IEPA's proposal how the Agency arrived at the proposed standards. Although the Agency has provided updated equations and values, it does not explain how these updates translate into the new standards proposed. In particular, the proposal does not explain how or why the latest proposed standards are orders of magnitude lower than the standards proposed in December 2019. Not only is IEPA's methodology not clearly explained, the sources from which IEPA has derived its information are different for the various PFAS compounds. The Agency should support USEPA's development of defensible data for each of the PFAS compounds it seeks to regulate and base it groundwater quality standards on updated, sound USEPA-derived values, when available.

IEPA must provide a more detailed methodology, and explanation of how it derived the proposed standards using that methodology, to allow for meaningful public comment. From our review of the proposal and the available support documents, it appears that the Agency is deriving these standards using an assumption that various substances will appear together in mixtures. Then, it is assumed that if several compounds act on the same organ, or produce a similar effect to a given system (e.g, the nervous system), their potential risks as to that organ or effect can be combined. Then, the potential cancer or non-cancer risks to various organs or systems can be combined to yield an overall risk. And somehow, all of those issues are factored in together to result in a specific standard for each substance. However, nowhere does IEPA provide the calculations that yield those proposed standards. Also, the Agency has not provided technical support for the assumptions that provide the basis for the standards, including as to whether (1) it is appropriate to assume that various compounds will occur in mixtures, or (2) that the risks to a given organ or system from several substances can be combined in an additive fashion, or (3) that cancer or non-cancer risks to several different organs or systems can be similarly combined. That information needs to be provided as to

each of the substances covered by the proposal, including as to which studies are being relied on for each toxicity endpoint. Without such information, one cannot determine if the proposed standards are scientifically supported. Stakeholders need to have the opportunity to review that information, and provide comments to the Agency concerning that information, before this proposal can proceed further.

D. Hierarchy of Sources

The Coalition appreciates IEPA's prioritization of USEPA-developed or USEPA-approved sources and values, such as USEPA's IRIS and USEPA's Provisional Peer-Reviewed Toxicity Value (PPRTV). The Coalition disagrees with IEPA reliance on certain of the Tier III sources for toxicity values, including the Agency for Toxic Substances and Disease Registry (ATSDR) and CalEPA. The ATSDR, part of the federal Center for Disease Control, and many states have reviewed the toxicity information available for PFOA and PFOS and opined on appropriate dosages that reflect highly conservative assumptions designed to protect human health, including the most susceptible subpopulations. ATSDR values are derived through different methods than USEPA's MCL (and Health Advisory) values and the two are not directly comparable.[6] These variabilities in how various health recommendations are derived must be considered and addressed to ensure that any final standards are scientifically justified and corroborated. [7]

Accordingly, the Coalition recommends that the State base any rulemaking on the forthcoming national primary drinking water standards, rather than the ATSDR report. Further, according to 35 Ill. Adm. Code Part 620 Subpart F, for substances that USEPA has not established a Maximum Contaminant Level Goal (MCLG), IEPA should base its highest priority approach for calculating the Advisory Concentration on the reference oral dose for humans as derived by USEPA. USEPA has not established MCLGs for any of the five compounds that are the subject of this rulemaking, but it has set a Health Advisory level of 70 ppt for PFOA and PFOS, individually or combined, based on oral reference doses of 0.00002 mg/kg/day for both compounds. IEPA should use the most current USEPA reference doses, such as those used for establishing the Health Advisory level for PFOA and PFOS, rather than establishing standards based on the ATSDR values.

For example, we note that one of five standards for PFAS, PFBS, was based on the PPRTV, which, for the reasons described above, is preferable to the ATSDR value. Notably, the standard for PFBS is also a far higher standard than any of the other PFAS standards. The fact that the PFBS standard, which is the only standard based on the more

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[6] *See* ATSDR Public Health Assessment Guidance Manual (2005) at Appendix F: Derivation of Comparison Values (https://www.atsdr.cdc.gov/hac/phamanual/appf.html) ("MCLs represent more realistic assumptions about toxicity and contain fewer uncertainty factors than the very conservative ATSDR environmental guidelines.")

[7] For a thorough discussion on possible confusion created by comparing ATSDR and EPA standards, *see* ECOS White Paper (*Processes and Considerations for Setting State PFAS Standards*) Appendix A, *available at*: https://www.ecos.org/documents/ecos-white-paper-processes-and-considerations-for-setting-state-pfas-standards/ (last accessed Feb. 28, 2020).

appropriate PPRTV value, is significantly higher than the other PFAS standards further supports the notion that the State should wait for USEPA to develop scientifically substantiated values, rather than promulgating its own standards based on underdeveloped science, which are unnecessary and unduly burdensome.

Additionally, PFOA is the only PFAS compounds for which the State has developed a standard based on cancer risk. USEPA has chosen not to regulate PFOA based on cancer risk. Also, CalEPA's study of PFOA is based on questionable science, which USEPA has not adopted or substantiated. Ultimately, the CalEPA study yields a much more stringent standard that is not derived from a sound or widely-accepted cancer risk assessment.

The State must avoid underpinning regulations on information that the scientific community is still debating, or using science that is not yet fully developed. USEPA is actively working on developing its own assessments for these and other PFAS compounds and, consequently, final standards-setting by the State is still premature. Illinois should not promulgate standards that are unjustifiably much more stringent than the eventual USEPA values.

E. Specificity in the Type of Regulated PFAS

In this current proposal, IEPA appears to have removed the combined PFOS and PFOA limit that the Agency initially included in the December 2019 proposal. The Coalition previously recommended against including any combined PFAS standards or limits and appreciates this revision in the current proposal.

PFAS regulations should clearly specify the individual compounds of PFAS that they seeks to regulate. Given the wide variations in toxicities and other characteristics exhibited by different PFAS chemicals, it is not scientifically appropriate to group all PFAS together for purposes of risk assessment or to assume that exposures to mixtures of PFAS necessarily bioaccumulate in one's body in interchangeable 1:1 ratios. Generally, the Coalition supports the proposed rulemaking's specificity in identifying which PFAS compounds are regulated and recommends that the regulation of individual PFAS substances reflect peer-reviewed science regarding the physical, chemical, and toxicological properties of each compound. Similarly, the Coalition reiterates its recommendation against including any combined PFAS standards or limits unless science clearly demonstrates that the mixture of the PFAS compounds subject to the combined limit results in hazardous concentrations.

F. Validated Test Methods for PFAS in Groundwater

There are no USEPA validated test methods for groundwater. As a general approach, the State should regulate only those PFAS compounds for which there are validated, approved analytical test methods. Here, though, IEPA is seeking to set groundwater limits without a validated test method. USEPA's main validated test methods

for PFAS, Methods 537 and 537.1, apply only to 18 PFAS compounds in samples derived from drinking water. USEPA recently issued Method 533 that can be used to measure an additional 11 "short-chain" PFAS compounds (and only 14 of the 18 PFAS covered by Method 537.1), again only for use in testing drinking water. Therefore, the entirety of USEPA's approved test methods can measure no more than 29 different PFAS compounds, and multiple methods would have to be used to obtain results for all 29 compounds.

No validated, approved USEPA test methods exist for testing PFAS compounds in any other environmental media. USEPA is developing a draft non-potable water test method (SW-846 Method 8327), but that method has not yet been formally incorporated into the SW-846 Compendium. Similarly, USEPA is working with the Department of Defense's (DOD) Naval Seas Systems Command Laboratory Quality and Accreditation Office to validate a solid-phase extraction/isotope dilution method to include solid matrices (*i.e.,* for soil, sediment, fish tissue, biosolids), as well as non-potable water sources, but that effort has not yet been completed.

The Coalition recommends that the proposed rulemaking recognize the limits of the available USEPA validated test methods and choose a specific test method to be referenced by any standards being adopted. Limitations on test methods and the lack of any validated, approved method by USEPA for anything except drinking water creates major challenges for the State's efforts to regulate non-potable water or other matrices. Considering that the State can potentially impose fines, costly corrective action, or other penalties for failing to meet regulatory limits, the regulated community must have the ability to accurately measure PFAS to demonstrate compliance. Subjecting the regulated community to fines, corrective action, and other penalties based on potentially unreliable testing or lack of available testing raises due process concerns. Accordingly, the Coalition urges the State to consider testing capability and reliability, and set limits and impose a regulatory scheme that accounts for the variability in and limits of current laboratory testing.

G. Availability of Treatment and Disposal Options

Similarly, treatment technologies for PFAS are still being developed, and there is limited capacity for the disposal of byproducts from newly-developed technologies. For example, adsorption technologies such as granular activated carbon (GAC) are being developed as potential response measures to achieve compliance with new standards for PFAS. The regulated community will need to safely dispose of the byproducts of such treatment technologies used to treat PFAS. If IEPA issues very low standards based on limited or deficient toxicology data, and the site data is generated by non-validated analytical methods, the regulated community will expend unnecessary resources on already limited remediation options. IEPA should account for the availability, feasibility, and cost of treatment and disposal options in setting standards to ensure that the regulated community has the ability to comply with the regulations.

Again, this is another area where USEPA is taking action. Congress, in the latest National Defense Authorization Act (NDAA), mandated that USEPA, not later than one

year after enactment, "publish interim guidance on the destruction and disposal of perfluoroalkyl and polyfluoroalkyl substances and materials containing perfluoroalkyl and polyfluoroalkyl substances," which includes guidance on "spent filters, membranes, resins, granular carbon, and other waste from water treatment."[8] In December 2020, USEPA released the new interim guidance for public comment, noting that considerable further research must be done to better characterize PFAS-containing materials; to measure and assess the effectiveness of existing methods for destruction; and to develop other technologies that may be employed instead of or with existing technologies.[9] The Coalition urges the State to use its resources to support the development of USEPA's interim guidance documents prior to establishing groundwater quality standards that will require disposal.

H. The State Should Consider the Technical Feasibility and Economic Reasonableness of the Rulemaking

The Illinois Pollution Control Board (Board) ultimately will need to adopt the groundwater quality standards that IEPA issues. The Board's enabling legislation requires that it take into account, among other factors, "the technical feasibility and economic reasonableness of measuring or reducing the particular type of pollution." 415 ILCS 5/27(a). Accordingly, IEPA should specifically address the technical feasibility and economic reasonableness of measuring and reducing PFAS in the environment in this rulemaking. Specifically, the rulemaking should account for the developing nature of treatment technologies and availability of disposal or other treatment endpoints. Information exists regarding the variable costs of treatment systems at locations around the country, and the State should consider that information in establishing remediation standards. Though some information exists regarding the costs of treatment alternatives IEPA must consider the signifcant uncertainty surrounding the handling of byproducts from PFAS treatment.

For example, a remediating party may not be able to find a landfill to take spent media. Additionally, incineration of spent media is thesubject to criticism and requires further study. As discussed in Section G above, Congress has directed USEPA to develop guidance to specially address these issues.

These remediation standards could also affect sites being remediated under federal programs, such as Superfund. For example, at DOD sites, the NDAA requires that cooperative agreements with states include that DOD "shall meet or exceed the most stringent . . . standards for PFAS in any environmental media." NDAA Sec. 332(a)(2). As a result, the states, municipalities, and private parties that are conducting cleanups may

[8] NDAA Sec. 7631(4).
[9] 85 Fed. Reg. 83554 "Interim PFAS Destruction and Disposal Guidance; Notice of Availability for Public Comment" (December 21, 2020).

incur substantial additional costs. The State should consider the costs to remediate to these proposed standards in its regulatory analysis.

Additionally, the rulemaking proposal does not appear to account for background concentrations of PFAS in the environment. Because the Agency has proposed such stringent levels, it is possible that background concentrations of certain PFAS already exceed the standards proposed. Of course, the higher the background concentrations of PFAS, the more costly and technically challenging it will be to remediate to the levels proposed. The rulemaking should include an analysis and determination regarding background levels of PFAS to inform the evaluation of technical feasibility and economic reasonableness of remediating to the levels proposed.

In summary, if this regulation will become final before there is more certainty regarding the underlying questions of treatment, disposal, and background concentrations then the State should conduct a more robust analysis of the technical feasibility and economic reasonableness to account for the potential costs, including remediation and the range of true disposal and ongoing operation and maintenance costs.

V. Conclusion

The Coalition appreciates the opportunity to comment concerning the proposed rulemaking. We look forward to working closely with the State regarding developing appropriate, reasonable, and scientifically-defensible groundwater protection standards. Please feel free to call or e-mail if you have any questions, or if you would like any additional information concerning the issues raised in these comments.

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Technical Comments of

Anna Reade, PhD

Katherine Pelch, PhD

Natural Resources Defense Council

in collaboration with the

Illinois Environmental Council

Sierra Club, Illinois Chapter

to the

Illinois Environmental Protection Agency

Re 35 Ill. Adm. Code 620; Groundwater Quality

Pre-Filing Public Comment Period

June 25, 2021

To whom it concerns,

We the signers, applaud the efforts by the Illinois EPA to set enforceable groundwater standards for PFAS chemicals, which will be necessary for identifying and cleaning up contaminated groundwater resources in the state. We previously submitted comments on the original proposal for groundwater standards in February 2020. Since then we are glad to see the IEPA has used more protective exposure estimates which have resulted in stronger health guidelines especially for perfluorooctanoic acid (PFOA), perfluorooctanesulfonic acid (PFOS), and perfluorononanoic acid (PFNA). However, we are still very concerned that some of the proposed groundwater standards, specifically those for perfluorohexane sulfonic acid (PFHxS) and perfluorobutanesulfonic acid (PFBS) are still not strong enough to fully protect human health. Several states have set more protective water standards for PFAS by considering the special vulnerability to PFAS exposure during gestation and infancy, and by basing risk evaluations on the most sensitive health effects linked to a particular PFAS. Other states have used a transgenerational toxicokinetic model to estimate exposure over a lifetime, including the increased consumption of water by infants and very young children, which leads to an increased body burden of PFAS during the most sensitive period of life.

The following comments lay out our concerns over IEPA's overall risk assessment process, in addition to comments on the chemical specific risk assessments performed. We urge IEPA to ensure that Illinois groundwater be regulated at levels protective enough to ensure that women and children could safely drink this water without any risk of harmful effects from PFAS. Finally, we urge IEPA to move beyond a chemical-by-chemical approach, to acknowledge the risks posed by the entire class, including cumulative exposures to mixtures of PFAS.

General Comments on IEPA's Risk Assessment Process

Risk assessments should be based on the current best available science, including the use of any chemical specific parameters available, and should be protective of all populations. Federal and state agencies that conduct independent risk assessments can evaluate current data to determine the appropriate parameters that should be used to arrive at a final value that is protective of those populations most vulnerable to exposure to a specific chemical or group of chemicals.

In contrast, IEPA is proposing to use an *a priori* determined hierarchy to guide development of its risk assessments. This hierarchy consists of three tiers from which to choose an existing toxicity value: 1) Integrated Risk Information System, 2) Provisional Peer Reviewed Toxicity Values, and 3) other toxicity values from sources where the risk assessment has been peer-reviewed. On one hand, this is beneficial to the state agency in that it streamlines the development of water standards, thereby allowing for their more rapid development. On the other hand, it limits the agency from conducting its own independent review of the existing literature and may limit the agency from utilizing risk assessments conducted by other state agencies. We also note that the procedures outlined in Appendix A leave little room and flexibility to incorporate chemical specific parameters. Further it is unclear how the hierarchy

takes into consideration how up-to-date various toxicity values are, or how new information can be considered.

Given the constraints imposed by the process used by IEPA for setting groundwater standards, we support the use of a RSC of 20% (= 0.2) in the absence of chemical specific data, which was outlined in Appendix A, Section (a) Calculating the Human Threshold' Toxicant Advisory Concentration for NonCancer Effects. Further, we feel that this RSC was appropriately applied in the risk assessments for PFAS prepared by IEPA.

However, we do not support the use of W=Per capita daily water consumption for a child (0 to 6 years of age, equal to 0.782 liters per day ("L/d") (Appendix A, Section (a)). Several states have used the more protective drinking water exposure estimate for very young infants 0 to 1 year of age (0.142 L/kg/day), and we encourage IEPA to do the same. Infants are particularly susceptible to the harmful effects of environmental chemical exposures due to the rapid growth and development that occurs during early life. Infants also consume more water on a per body weight basis than adults (0.029 L/kg/day), lactating women (0.054 L/kg/day), and even children aged 0 to 6 years (0.052 L/kg/day). Note that the drinking water exposure estimate for infants 0 to 1 year of age is more than double the estimate for children 0 - 6 years old.

Further we point out that the requirement to use the methodology outlined in Appendix A, Section (a) precludes the use of more sophisticated toxicokinetic modeling for estimating exposure through drinking water. For example, the procedure for "Calculating the Human Threshold' Toxicant Advisory Concentration for NonCancer Effects" proposed in Administrative Code 620 does not allow for the use of the peer reviewed transgenerational toxicokinetic model developed by Minnesota Department of Health scientists that more accurately models serum levels of persistent chemicals, such as PFAS, over a lifetime of consumption.[1, 2] Importantly, the transgenerational toxicokinetic model and supporting documentation highlight the need to protect the very young, as serum levels of PFOA and related chemicals spike (i.e. are elevated) in the first two years of life.

We also note that the hierarchy of sources of toxicity values described in Appendix A, section (b), subsection (2) does not allow for needed flexibility in responding to the rapidly evolving science related to PFAS. It is unclear how IEPA will make use of the hierarchy of toxicity values when new information becomes available, especially given that some of the listed agencies in Subsection (2), parts A-C are not required to regularly update their assessments. It is possible that these resources could become out of date as new scientific literature becomes available. Without the option to conduct its own risk assessment or to make use of risk assessments conducted by other state agencies IEPA risks developing standards that are out of date and not health protective.

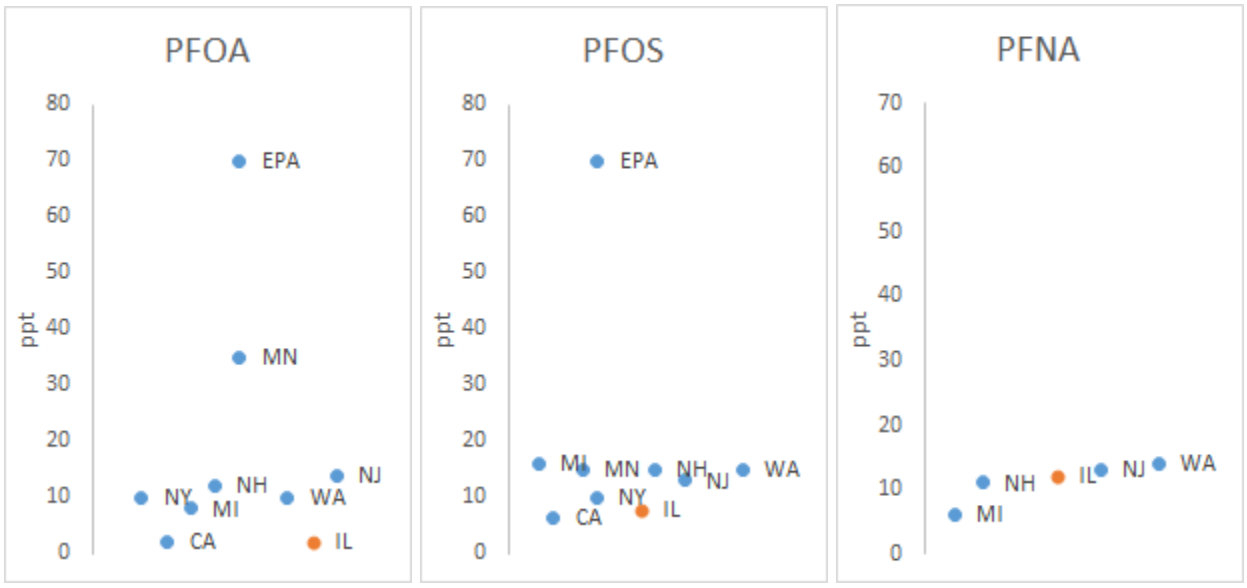
As noted in an EPA memorandum from December 1993 entitled "Use of IRIS Values in Superfund Risk Assessment" (OSWER Directive 9285.7-16, December 21, 1993):

> "...IRIS is not the only source of toxicology information, and in some cases more recent, credible and relevant data may come to the Agency's attention. In particular, toxicological information other than that in IRIS may be brought to the Agency by outside parties. Such information should be considered along with the data in IRIS in selecting toxicological values; ultimately, the Agency should evaluate risk based upon its best scientific judgement and consider all credible and relevant information available to it."[3]

However, it is unclear if IEPA has always followed the above cited guidance and how IEPA will do so moving forward. For example, in an earlier draft of the groundwater standard for PFBS, IEPA had relied upon Tier II data - a PPRTV from EPA from 2014, which was already considered out of date by other state and federal agencies conducting risk assessment on PFBS. At the time of IEPA's draft there was already an existing draft human health toxicity value derived by US EPA[4], and toxicity values derived by Michigan's Science Advisory Workgroup[5] and Minnesota's Department of Health[6]. We are pleased to see that IEPA is now relying on the new human health toxicity value for PFBS released by US EPA on April 28, 2021, but it remains unclear in Administrative Code 620 how the age of the data is considered when deciding which toxicity value to use and/or when to update existing standards.

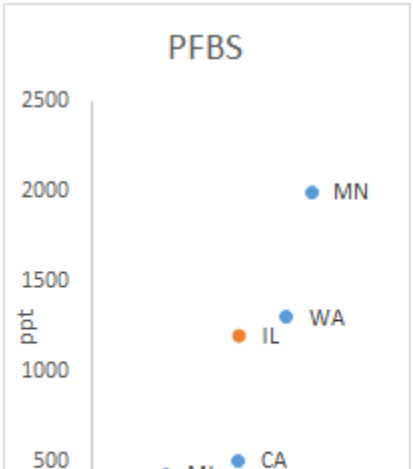
Chemical-specific Comments

We support IEPA's decision to set the groundwater quality standard for PFOA at 2 ppt, as this value is health protective based on current evidence. We generally support IEPA's decision to set the groundwater quality standards for PFOS at 7.7 ppt, and PFNA at 12 ppt.[7] Although our own analysis suggests that these values could be slightly more health protective, they are in line with values derived by other reputable states and agencies.



However, as discussed in detail below, we do not agree with IEPA that the values for PFHxS (77 ppt) and PFBS (1,200 ppt) are health protective groundwater standards, thus highlighting the need to make further changes to the Administrative Code as described above.

PFBS



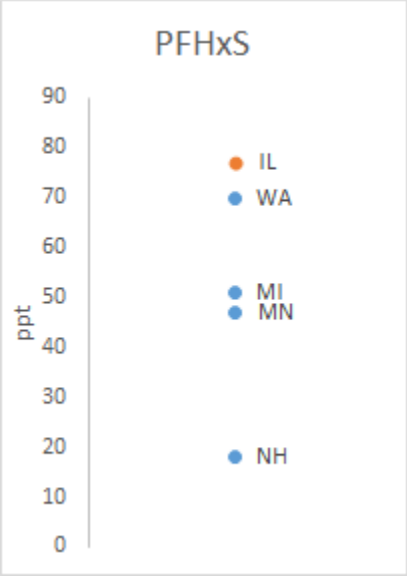
IEPA used the reference dose (RfD) of 300 ng/kg/day derived by the US EPA. The RfD was also used by Michigan and Washington in setting health-based values in those states. California also based it's RfD on the same critical study, yet calculated a RfD of 500 ng/kg/day. Michigan and California, each arrived at more health protective final values than IEPA: 420 ppt in Michigan and 500 ppt in California compared to IEPA's 1,200 ppt. The nearly two- to three-fold difference in

final values is the result of choosing to protect very young infants who are most vulnerable.

Michigan used a drinking water ingestion estimate specific for infants (birth to <1 year old) of 0.142 L/kg/day based on the 95th percentile of water intake for consumers only (direct and indirect consumption) per Table 3-1 in USEPA Exposure Factors Handbook, 2019. Similarly, California used a drinking water ingestion estimate specific for infants 0-6 months old of 0.237 L/kg/day. In contrast, IEPA has chosen to use a drinking water ingestion estimate for children up to 6 years old of 0.052 L/kg/day. While this drinking water estimate is significantly more protective than drinking water ingestion estimated for adults (0.029 L/kd/day), it is not as protective as drinking water ingestion estimates for infants or for nursing and lactating women (0.054 to 0.055 L/kg/day), both of which have often been used by agencies engaged in PFAS risk assessment.

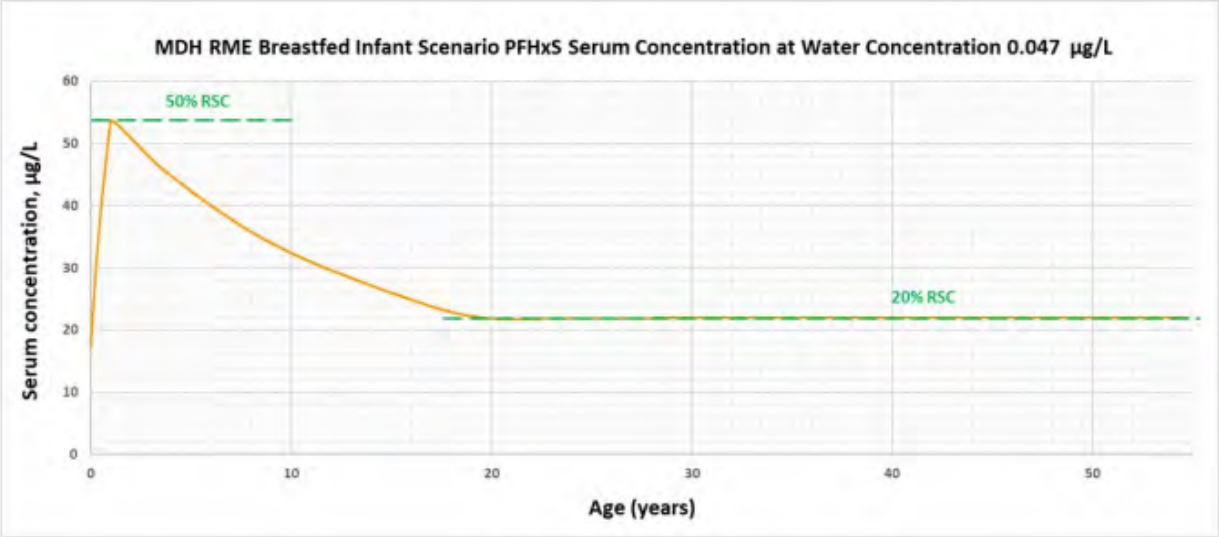
IEPA has chosen to base its risk assessment for PFBS on the critical effect of decreased total serum T4 in newborn animals. However, by using a drinking water ingestion estimate for children of an older age, it is questionable if the final value achieved will actually be protective of this effect or not. We encourage IEPA to acknowledge that infants 1 year of age and younger are a particularly vulnerable and sensitive population when it comes to PFAS exposure by choosing to use a drinking water ingestion estimate for infants 0 to 1 years old in all of it's PFAS risk assessments unless there is strong evidence that an effect is more sensitive in another population. We note above that this should be addressed by updating Appendix A, Section (a).

PFHxS



IEPA used the RfD originally derived by ATSDR in June 2018,[8] which qualifies as a Tier 3 Toxicity Value in the proposed hierarchy described in Appendix A, Section (b), Subsection (2) of Administrative Code 620. This RfD of 20 ng/kg/day is based on thyroid follicular cell damage in adult rats, and was finalized without any updates in May 2021.[9] Other state agencies, namely Michigan, Minnesota, and Washington, that conducted risk assessment for PFHxS subsequent to the publication of the ATSDR Draft Toxicological Profile did not base their assessments on the same endpoint.[5, 9, 11] Rather, these state agencies based the risk assessment on decreased free T4 observed in adult male rats in the National Toxicology Program's (NTP) TOX96 Report from 2018.[12] The resulting RfD for this endpoint used by Michigan, Minnesota, and Washington is 9.7 ng/kg/day. New Hampshire also conducted risk assessment for PFHxS subsequent to publication of the ATSDR Draft Toxicological Profile and chose a critical effect of impaired female reproduction, specifically reduced litter size in exposed mice, resulting in a RfD of 4.0 ng/kg/day.[13] Importantly, the work utilized by New Hampshire was published in a peer reviewed document,[14] which would qualify it for use as a Tier 3 Toxicity Value according to the hierarchy described in Appendix A, Section (b), Subsection (2) of Administrative Code 620. It is unclear if these newer toxicity values could be used by IEPA given the hierarchy of toxicity values outlined in Appendix A.

Further, nearly all state agencies that have conducted risk assessment for PFHxS have relied upon the peer reviewed transgenerational toxicokinetic model[1] for estimating exposure to PFHxS.[5, 9, 11,13] As noted in Figure 3 from the risk assessment document provided by Minnesota,[10] serum levels of PFHxS are expected to spike in breastfed infants within the first two years of life, further highlighting the deficiency of the drinking water exposure estimate for children 0 to 6 years of age proposed for use by IEPA.



We recognize that the IEPA has strengthened its proposed groundwater quality standards for most of the PFAS chemicals however considering the above information, Illinois should lower its groundwater quality standard for perfluorohexane sulfonic acid (PFHxS) and perfluorobutanesulfonic acid (PFBS) to be on par with those set by Michigan and California in order to protect the most vulnerable populations to PFAS exposure. This can be accomplished by using the most up to date toxicity values and drinking water exposure estimates that are protective of the most vulnerable and susceptible populations.

Moving Beyond a Chemical by Chemical Approach

Perhaps more importantly, the structure of the fluorine-carbon bond and the hazards documented for PFAS support concern over the environmental and health impacts of the entire class. It is important to note that all of these individual risk assessments do not account for cummulative exposures to mixtures of PFAS, and thus could be vastly underestimating the risk posed by PFAS exposures. Yet, virtually all people living in the US have multiple PFAS in their bodies.[15] The magnitude of this problem demands a more efficient and effective approach, which is why prominent scientists and medical organizations from around the world are urging a class-based approach for managing PFAS.[16,17] A goal of zero PFAS in drinking water is needed to provide an adequate margin of safety to protect public health from a class of chemicals that is characterized by extreme persistence, high mobility, and is associated with a multitude of different types of toxicity at very low levels of exposure.[7]

Multiple resources are available to guide IEPA in developing class-based approaches for regulating PFAS. In previous technical comments we have outlined a hierarchy of class-based

approaches for regulating PFAS in ground and drinking water, from most health protective to least, that should be further considered by IEPA to protect Illinois residents from undo PFAS exposure.[18] The most health protective approach being regulating the full class based on persistence, or the "P-sufficiency" approach, and setting a treatment technique for the class. We therefore urge Illinois to explore in the near future the establishment of a treatment technique for PFAS - a minimum treatment requirement or a necessary methodology or technology that a public water supply must follow to ensure control of a contaminant.

Thank you for considering these important ways to ensure greater protection for Illinois residents. Please take these urgent and defensible actions to strengthen groundwater protections from PFAS to ensure that Illinois groundwater resources remain safe and clean.

Sincerely,

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June 25, 2021

Illinois Environmental Protection Agency

Sent via email: EPA.620.rulemaking@illinois.gov
Subject: 35 Ill. Adm. Code 620 Proposed Updates, Public Comment

RE: ILLINOIS EPA DRAFT PROPOSED UPDATES TO 35 ILL. ADM. CODE 620; GROUNDWATER QUALITY

Thank you for the opportunity to provide these comments on behalf of Midwest Generation, LLC, on the draft proposed changes to the Illinois Part 620 Groundwater Quality rules. I am a Diplomate of the American Board of Toxicology and Principal Toxicologist with GSI Environmental, Inc. (GSI). I have over 15 years of experience providing toxicology, risk assessment, and risk management support to federal and state regulatory agencies, municipalities, and private industries. A copy of my CV is attached. I provide these comments based on my experience and expertise with regulatory toxicology and risk-based regulations.

OVERALL COMMENTS

In response to the Illinois Environmental Protection Agency (Illinois EPA)'s request for public comment on the proposed draft language updates to 35 Ill. Adm. Code 620 ("Proposed Part 620"), GSI has reviewed the information made available on the IL EPA website (https://www2.illinois.gov/epa/about-us/rules-regs/water/Pages/620-Groundwater-Quality.aspx) and participated in the May 26, 2021 virtual public meeting. Based on review of these materials and my expertise in regulatory toxicology and risk management strategies, I offer the following comments on the Proposed Part 620:

> **The application and use of default exposure parameters for children, and misinterpretation and misapplication of the U.S. Environmental Protection Agency's (USEPA) policy and guidance on toxicity value hierarchy, does not allow Illinois EPA or the regulated community to use the best available science to support sound decision making on a chemical-by-chemical basis.**
>
> **The proposed default relative source contribution (RSC) of 0.2 for boron is not supported by best available science and is inconsistent with conclusions rendered by the USEPA.**

These comments are explained in further detail below.

1. **The application and use of default exposure parameters for children, and misinterpretation and misapplication of the U.S. Environmental Protection Agency's (USEPA) policy and guidance on toxicity value hierarchy, does not allow Illinois EPA or the regulated community to use the best available science to support sound decision making on a chemical-by-chemical basis.**

The Proposed Part 620 proposes a significantly different, and not scientifically supported, methodology for adopting groundwater quality standards. The current 35 Ill. Adm. Code 620 ("Part 620") adopts as groundwater quality standards (GQS) risk-based or water quality-based values from federal agencies such as the USEPA. The Proposed Part 620 will allow Illinois EPA to calculate their own GQS for both noncarcinogens ("Human Threshold Toxicant Advisory Concentration") and carcinogens ("Human Nonthreshold Toxicant Advisory Concentration"), as described in Section 620 Appendix A. The Proposed Part 620 also will require the use of child body weight, child water consumption rate, and a relative source contribution (RSC) of 0.2. It also requires the use of USEPA Integrated Risk Information System (IRIS) toxicity values, if available, for the chemical of interest, as IL EPA claims this is USEPA's "hierarchy of usable sources". However, collectively, these new requirements for calculations are too prescriptive and do not allow for best available and sound science to be used on a chemical-by-chemical basis.

Default use of childhood exposure parameters results in compounded conservatism and is not consistent with USEPA Guidance. Generally, in any risk analysis, risk is calculated as toxicity of a chemical combined with exposure to the chemical, including time and concentration. Thus, to calculate risk for deriving regulatory threshold criteria, such as GQS, default, conservative exposure parameters are often used, which provide an assumption of the worst-case exposure scenario. The exposure parameters include estimates of the magnitude, frequency, and duration of exposure to the chemical in the relevant environmental media (in this case, groundwater). Exposure parameters specific for various receptors (e.g., body weight and drinking water ingestion rate) can be used such as for a child, an average adult, or a pregnant woman. The inherent variability in exposure for individuals is addressed by using high-end exposure estimates for the receptor subgroup.

The use of a distinct equation combining a toxicity value with exposure parameters can be mandated by legislative authority and regulatory paradigms. However, requiring the application and use of conservative receptor exposure parameters, such as the child body weight, child drinking water ingestion rates, and the lowest RSC, often results in compounded conservatism and inaccurate prediction of risk.

Here, in the Proposed Part 620, Illinois EPA essentially adopts the USEPA Regional Screening Levels methodology for noncarcinogen chemicals, except Illinois EPA only uses child parameters. Illinois EPA's proposal to require the use of the child exposure assumptions in the Proposed Part 620 GQS derivation methods are not consistent with USEPA guidance. In general, childhood exposures are most often used to address shorter duration exposure or to define conservative, initial screening levels, while adult exposure assumptions are used for

lifetime or chronic exposures and more commonly used for regulatory decision making. The USEPA screens noncarcinogen chemicals in groundwater sites using both child and adult parameters to ensure conservative protective assumptions are used to identify impacted groundwater that may need further evaluation. Due to how noncancer risk-based standards for potable water are traditionally calculated, use of a child's body weight and water ingestion rate result in a "high-end" exposure estimate and, therefore, a lower, more stringent GQS, because the child is likely to receive a greater dose on a milligram per kilogram per body weight basis. Use of the most conservative child exposure assumptions provide a first step screen, to determine if follow-up investigations may be necessary. However, USEPA Regional Screening Levels are not cleanup levels and should not be interpreted as regulatory criteria that cannot be exceeded. USEPA clearly states "**It should be emphasized that [screening levels] are *not cleanup standards*.**" (emphasis added)[1]. In fact, for most noncarcinogen chemicals, USEPA's Drinking Water Standards and Lifetime Health Advisories rely on adult body weight and water consumption rate; only the one-day and ten-day advisories utilize child exposure parameters (USEPA 2018).

In some cases, it may not only be overly conservative, but also technically incorrect to utilize a child's exposure parameters when deriving a GQS. The USEPA guidance on developmental toxicity, for example, states that the final risk characterization for a chemical needs to include information on exposure route, timing and duration of exposure specific for the toxicity value (RfD), and "…it would be inappropriate in developmental toxicity risk assessments to use [] adjustment of exposure over a different time frame than that actually encountered…" (USEPA 1991, p. 45). In other words, it is inappropriate to mismatch the exposure parameters for one receptor with a toxicity value (RfD) derived from a different receptor's exposure scenario.

The USEPA RfD used by Illinois EPA for the proposed GQS for boron is based on a development effect that occurs *in utero*. When deriving the lifetime Health Advisory for boron, USEPA explicitly stated that "the target population is pregnant women because the *in utero* development endpoint is the most sensitive." (USEPA 2008a). Therefore, the USEPA RfD for boron should be combined with exposure parameters specific for the pregnant woman (consistent with the USEPA Office of Water). The Illinois EPA should not use child exposure parameters with an *in utero* effect. Illinois EPA should have the flexibility to apply the best available science that is technically sound, and make sure that the most sensitive effect for a chemical matches the receptor parameters used in the GQS calculation.

Automatic use of USEPA IRIS toxicity values conflicts with USEPA policy and guidance and with risk assessment best practices. Beginning with USEPA Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part A), USEPA guidance recommends selecting toxicity criteria based on the most recent data (USEPA 1989, p. 7-15). This recommendation has since been implemented in numerous USEPA directives (USEPA 1993, 2003) that further establish a hierarchy and process for selecting toxicity criteria. The USEPA IRIS assessments are generally considered the top-tiered choice, based on their use of

[1] https://www.epa.gov/risk/regional-screening-levels-rsls-users-guide

standardized methods and rigorous peer review; however, IRIS toxicity values for each chemical are not always recent and up to date. Importantly, comparison of available toxicity values across multiple sources to ensure validity of the value is now a routine part of regulatory toxicology best practices (USEPA 2003, 2013a; ECOS 2007; Illinois EPA Part 320 Section 302.606[2]). Evaluation of multiple sources of toxicity information ensures that the information used is current, peer reviewed, transparent, and the best available information. This flexibility recognizes that new chemical-specific information may become available and that risk assessment practices are continually evolving; therefore, selection of a toxicity value should be based on the most recent, credible, and relevant data, as well as, incorporating the best risk assessment methods available. As stated in the original USEPA 1993 directive, "…in some cases more recent, credible and relevant data may come to the Agency's attention. … [T]he Agency should evaluate risk based upon its best scientific judgment and consider all credible and relevant information available to it" (USEPA 1993, p. 2).

Here, the Illinois EPA's proposal is to limit the toxicity criteria to the USEPA IRIS toxicity values regardless of whether there is updated information in another appropriate source. This is against best practices because it could rely upon outdated data. Instead, the Part 620 rule should allow the Illinois EPA to look to all credible and relevant information available instead of only the USEPA IRIS toxicity values.

Combined, the Proposed Part 620 adopts GQS and a methodology for deriving GQS that does not allow the Illinois EPA or regulated community to use sound science to support decision making. It locks users into default values and methods without consideration of chemical-specific best available information. The Proposed Part 620 GQS and methods may result in compounded conservatism, which creates GQS that are unnecessarily low.

2. The default relative source contribution (RSC) of 0.2 for boron is not supported by best available science and is inconsistent with conclusions rendered by the USEPA.

The regulatory concept underlying the use of RSC is that the criterion set for a single exposure pathway such as drinking water needs to also allow for the potential for exposure to occur from other pathways (e.g., diet, ingestion, dermal absorption). By determining the fraction of total exposure attributable to non-drinking water pathways, one can determine the "balance" of the exposure that cannot be exceeded from the drinking water pathway alone. Current USEPA guidance recommends determining an appropriate RSC value within the range of 20 to 80 percent (USEPA 2013b; 2000). The low-end value of 20 percent is a health protective assumption that is applied in the absence of chemical-specific data on exposure. It assumes that 80 percent of the target dose can be attributed to (or allocated to) exposures other than drinking water, while the remaining 20 percent is due to exposure via drinking water. USEPA strongly encourages States to consider available data to derive chemical-specific RSC estimates (USEPA 2000). Rather than requiring the default RSC of 0.2 for all chemicals, Illinois EPA should adopt USEPA's guidance for using the Exposure Decision Tree approach described in the

[2] Section 302.606 of the Illinois Pollution Control Board ("Board") Rules requires that the Agency reviews all data used in calculating water criteria based on "validity, applicability and completeness". 35 Ill. Adm. Code 302.606.

Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (USEPA 2000).

Specifically, the proposed GQS for boron is inconsistent with the current state-of-the-science for boron. Illinois EPA should fully consider the relevant chemical-specific information currently available for boron, and allow for the best science, rather than default parameters, to be used when deriving the GQS. Evaluations conducted by the USEPA demonstrate that a RSC of 0.8 for boron is consistent with the state-of-the-science and would result in a GQS that is still protective of human health (USEPA 2008a). USEPA determined that data were available to describe anticipated exposures to boron from different sources, including diet. The USEPA Office of Water Health Effects Support Document for Boron (USEPA 2008b) summarized data describing the exposure to boron by the general public from food, air, soil, and insecticide use, and were able to use available data to calculate a chemical-specific RSC, following USEPA guidance and best practice (USEPA 2000).

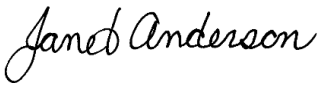
As summarized by several authoritative agency documents, there are quality studies that have quantified exposure levels for boron to various population groups (e.g., ATSDR 2010, USEPA 2004, IOM 2001). The National Academies Institute of Medicine (IOM) 2001 dietary report concludes that dietary sources represent the main background intake for boron and provide the quantitative support for the USEPA boron-specific calculation. IOM (2001) summarized the available literature as of 2001, including data collected by the Centers for Disease Control and Prevention, National Health and Nutrition Examination Survey (NHANES), and established mean boron intakes per day from dietary and supplement sources (see IOM 2001 Appendix C Tables C-12 and C-13). The information specific to pregnant women intake (to match the USEPA IRIS RfD associated with developmental effects *in utero*) of boron from dietary sources was used by the USEPA to calculate a chemical-specific RSC for boron of 0.8 (USEPA 2008a).

The ability within the GQS process to use the best available science is the most true and accurate application of USEPA policy and guidance. Specifically for boron, an analysis of the available data clearly demonstrates that the RSC should be 0.8.

CONCLUSION

To make a health-based GQS for any chemical without conducting a thorough evaluation of the current state-of-the-science, would be arbitrary. Indeed, Section 302.606 of the Board Rules requires that the Agency reviews all data used in calculating water quality criteria based on "validity, applicability and completeness". 35 Ill. Adm. Code 302.606. Accordingly, we suggest that the Illinois EPA revise the draft proposed update to include use of best available chemical-specific information. In doing so, Illinois EPA should also use the authoritative and peer-reviewed analysis of boron exposure through non-drinking water sources and USEPA's calculation of a chemical-specific RSC of 0.8. These revisions to the Proposed Part 620 would result in the use of reasonable, scientifically valid parameters that can be used to derive public health protective and technically sound GQS.

Respectfully submitted,



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IOM.2001. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. The National Academies Press Institute of Medicine, Washington, DC.

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USEPA. 1993. Use of IRIS values in Superfund risk assessment. Directive 9285.7-16. U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, DC.

USEPA. 2000. Methodology for deriving ambient water quality criteria for the protection of human health. EPA/822/B-00/004. U.S. Environmental Protection Agency, Office of Water and Office of Science and Technology, Washington, DC. October.

USEPA. 2003. Human health toxicity values in Superfund risk assessments. Directive 9285.7-53. U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, DC. February.

USEPA-IRIS. 2004. Chemical Assessment Summary, Boron and Compounds; CASRN 7440-42-8. U.S. Environmental Protection Agency, National Center for Environmental Assessment, Washington, DC.

USEPA. 2008a. Drinking Water Health Advisory for Boron. EPA-822-R-08-013. Health and Ecological Criteria Division, Office of Science and Technology, Office of Water, U.S. Environmental Protection Agency, Office of Water, Washington, DC. May.

USEPA. 2008b. Health Effects Support Document for Boron. EPA-822-R-08-002. Health and Ecological Criteria Division, Office of Science and Technology, Office of Water, U.S. Environmental Protection Agency, Office of Water, Washington, DC. January.

USEPA. 2013a. Tier 3 Toxicity Value White Paper. Regional Tier 3 Toxicity Value Workgroup. OSWER 9285.7-86. Office of Solid Waste and Emergency Response Human Health Regional Risk Assessors Forum, U.S. Environmental Protection Agency, Office of Water, Washington, DC. May.

USEPA. 2013b. Human health ambient water quality criteria and fish consumption rates: Frequently Asked Questions. Available: https://www.epa.gov/sites/production/files/2015-12/documents/hh-fish-consumption-faqs.pdf U.S. Environmental Protection Agency, Office of Water, Washington, DC. January.

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Biographical Summary

Dr. Janet Anderson is a Principal human health toxicologist and environmental risk assessor with 15 years of experience providing toxicology expertise and consultation to federal agencies and industry. She specializes in the translation of human health toxicology data into state and federal regulatory policy decisions and performs critical reviews of federal and state risk assessment guidance and regulations. She has also provided litigation consulting support and served as an expert witness.

Dr. Anderson is a nationally recognized leader in unregulated and emerging chemicals, such as per- and polyfluoroalkyl substances (PFAS), 1,4-dioxane, and 1,2,3-trichloropropane. With in-depth knowledge of federal and state environmental guidance and policies pertaining to this class of compounds, she has developed strategies to mitigate their human health impacts and address associated environmental liability for both private and public sector clients. She tracks the dynamic regulatory changes for emerging chemicals in the U.S. and internationally, offering clients the technical basis for disparate guidelines worldwide. She has extensive experience developing risk management strategies for multi-stakeholder groups.

Previously, as a civilian government employee, Dr. Anderson led the U.S. Air Force (USAF) Emerging Issues and Contaminants Program, where she developed programmatic recommendations on environmental regulations and cleanup standards and assisted with site-specific remediation. She also served as a member of the federal interagency review team providing consultation and expert review on toxicology assessments and/or guidance documents produced by EPA, the National Toxicology Program (NTP), and the Agency for Toxic Substances Disease Registry (ATSDR). As a postdoctoral fellow for the EPA Office of Research and Development National Center for Environmental Assessment, she managed numerous Superfund chemical assessments and served as a team member for Integrated Risk Information System (IRIS) assessments.

Dr. Anderson is a diplomate of the American Board of Toxicology and an active member of the Society of Toxicology. A skilled communicator, she is often an invited speaker at high-level scientific conferences, regulatory meetings, webinars, and community stakeholder meetings.

Education

Ph.D., Molecular and Cancer Biology, University of Cincinnati, College of Medicine, Cincinnati, Ohio, 2007

B.A., Biology and Women's Studies, Wittenberg University, Springfield, Ohio, 2000

Post-Doctoral Fellow, EPA Office of Research and Development National Center for Environmental Assessment, Cincinnati, Ohio, 2007–2010

Diplomate, American Board of Toxicology, 2012–present

Professional Background

Principal, GSI Environmental Inc., Houston, Texas, 2020 – current

Senior Associate, GSI Environmental Inc., Houston, Texas, 2019

Senior Consultant, Integral Consulting Inc., San Antonio, Texas, 2015 – 2019

Emerging Issues and Contaminants Program Manager, Subject Matter Specialist – Toxicology, US Air Force Civil Engineer Center, San Antonio, Texas, 2010 – 2015

Professional Certifications and Affiliations

Member of Society of Toxicology, Risk Assessment Specialty Section, Women in Toxicology Special Interest Group, and Lone Star Regional Group

Interstate Technology and Regulatory Council (ITRC) Workgroup on Per- and Polyfluoroalkyl substances (2017–present)

Interstate Technology and Regulatory Council (ITRC) Workgroup on 1,4-Dioxane (2019–present)

Ad Hoc Panelist for Alliance for Risk Assessment, "Beyond Science and Decisions"

Continuing Education and Training

Linkage, Women in Leadership Training (2020)

Physiologically Based Pharmacokinetic Modeling to Support Modernized Chemical Safety Assessment, Society of Toxicology Continuing Education Course (2018)

EPA Environmental Risk Assessment Guidance Training, EPA Environmental Response Training Program (2011)

Environmental Negotiations Workshop, Naval Civil Engineer Corps Officers School (2010)

The Hamner Institute's Computational Systems Biology, Research Triangle Park (2008)

Physiologically Based Pharmacokinetic Modeling, Dr. Ray Yang (2008)

International Life Sciences Institute Human Relevance Framework for Weight of Evidence Workshop, Tolerance Reassessment Advisory Committee (2008)

Toxicology Excellence for Risk Assessment Dose-Response Boot Camp (2007)

EPA's Benchmark Dose Training (2007)

Project Experience

Litigation Expert and Consulting Services

Expert Services, Aqueous Film Forming Foam (AFFF) and PFAS Toxicological History and Regulations—On behalf of the U.S. Department of Justice, Environmental & Natural Resources Division, provide consultation and expert opinion on the regulatory and human health toxicological history of AFFF and PFAS. *(Penna v. The United States of America, in the United States Court of Federal Claims,* Case No. 16-1545L).

Expert Services, Carbon Monoxide, California—On behalf of ITW Food Equipment Group, LLC, served as expert testifying witness on the regulatory toxicity values for, and process for human health risk evaluation of, carbon monoxide. (*Julie Lee, Julie Lacey, Lourdes Munoz, Martha Silva, Brandon Adams, Lafayette Wallace, Joshlynn Jarboe, Yolanda Rodriguez, Peter Lee and Mark Rodriguez v. Hobart Corporation, Wayne Home Equipment, A Scott Feitzer Co., A.M. Wighton & Sons, Inc., DBA A&J Refrigeration. In the Superior Court of Santa Barbara, Cook Division, California,* Case Number: 1389541).

Expert Services, 1,2,3-Trichloropropane, California—On behalf of Shell and Dow Chemical Co., served as expert on the use and interpretation of regulatory standards and toxicity values for 1,2,3-trichloropropane.

Expert Services, Dieldrin and Aldrin, Florida—On behalf of Shell Oil Company, served as expert testifying witness and authored a detailed expert report on the regulatory toxicity values for, and human health risk evaluation of, dieldrin and aldrin. (*Janice Potter, Brian Potter, David Stepp, Debra Stepp, Renee Bolton, Yvonne Hopp, Herman Osterloh, Morgan Canada and Lauren Kelly, Class Representatives v. Shell Oil Company and DeLand Golf Course, Inc. In the Circuit Court, Seventh Judicial Circuit, in and for Volusia County, Florida,* Case Number: 2011-11036-CIDL Division).

Expert Services, Portland, Maine—On behalf of Mallinckrodt U.S., LLC, served as expert testifying witness regarding human health risks and related remedial action of methylmercury in biota and sediments in the Lower Penobscot River and Estuary. *(Natural Resources Defense Council et al. v. HoltraChem Manufacturing Company, LLC et al., U.S District Court, District of Maine,* Civil Action No. 1:00-cv-00069-JAW*).*

Litigation Support Services, Alaska—On behalf of Williams Alaska Petroleum, provided technical support to the testifying expert on the appropriate toxicity values for site-specific risk assessment on sulfolane. (*State of Alaska et al. vs. Williams Alaska Petroleum et al., Shook, Hardy, and Bacon In the Superior Court for the State of Alaska Fourth Judicial District Court,* Case No. 4FA-14-01544CI).

Consulting Support Services, Perfluoroalkyl Substance Contamination—For a confidential client, provided technical and strategic support related to the regulatory processes and toxicological assessments for PFAS.

Consulting Support Services, p-Chlorobenzenesulfonic Acid Contamination—For a confidential client, provided regulatory support and toxicology assessment.

Dispute Resolution, U.S. Air Force, Lackland, Texas—Provided technical support to USAF legal offices and program managers engaged in federal and state dispute resolution related to emerging issues and contaminants, including trichloroethylene, tetrachloroethylene, 1,4-dioxane, and perfluorinated compounds.

Emerging Chemicals Strategies and Management

Regulatory Tracking and Analyses, United States. Serves as a regulatory toxicology subject matter expert for emerging chemicals such as PFAS and 1,4-dioxane. Is responsible for tracking the toxicological data and regulatory assessments and decisions internationally; providing summaries and impact assessments for clients; engaging with regulatory authorities to ensure sound scientific basis of regulatory decisions; and advising and developing risk management strategies to minimize effects of changing information and regulations, to ensure public and employee safety and health. Numerous clients.

Strategic Support Related to Management of Aqueous Film Forming Foam (AFFF) Use and Replacement. Provides confidential client with state-of-the-science updates and technical support related to regulatory and human health/environmental risks associated with AFFF use at oil and gas facilities.

Technical and Regulatory Support for the National Association for Surface Finishing. Provides technical consulting support, including toxicology, exposure, chemistry, training, and science communication, to, and on behalf of, the metal and surface finishing industry within the United States. Represents client in regulatory and legislative meetings and ensure that the human and environmental risks associated with metal plating processes is accurately understood and communicated to internal and external stakeholders.

Risk Communication and Regulatory Support Related to Contamination of Public Drinking Water, Confidential Municipality, U.S. Provides technical and strategic regulatory toxicology risk communication support to a U.S. drinking water municipality with unregulated and emerging chemicals present in source water. Ensures the municipality understands the human health risks and regulatory actions. Represents client in regulatory, and public meetings and ensure that toxicology and human health risk information is accurately communicated to and by stakeholders.

Risk Communication and Regulatory Support, 1,4-Dioxane. Confidential Publicly Owned Landfill, U.S. Provides regulatory toxicology, site-specific risk assessment reviews, and risk communication support to a publicly owned landfill with 1,4-dioxane in leachate. Ensures the human health risk assessments and regulatory actions are technically sound.

Emerging Issues and Contaminants Program Management, U.S. Air Force Civil Engineer Center, Lackland, Texas. Served as program manager of an emerging contaminants program with a $1.2 million annual budget. Oversaw support contractors, wrote documents, delivered presentations, led internal management briefings, and led department training sessions. Identified gaps in scientific knowledge that underlies USAF and DOD efforts to protect human and environmental health. Specific topics included

vapor intrusion, PFAS, 1,4-dioxane, chlorinated solvents (trichloroethylene, tetrachloroethylene) and pesticides. Also monitored evolving regulatory and political arenas to identify changes that could impact environmental cleanup costs, schedules, and procedures and policies. Developed programmatic recommendations for budget and resource needs to address environmental regulations and cleanup standards.

State-by-State Survey, United States. Participated in a survey of state and federal regulatory programs and initiatives to assess the level of activity and process by which emerging chemicals, such as PFASs, are prioritized and regulated. Analyses include understanding how state regulatory and public health agencies identify, prioritize, and develop strategies and standards to manage emerging chemicals. Work resulted in a compendium of all state-level initiatives related to emerging chemicals, which allows users to understand trends and state-specific interests.

Risk Communication, U.S. Air Force Civil Engineer Center, Lackland, Texas. Provided risk communication materials such as factsheets and informational seminars to the general public interested in understanding PFAS site-specific environmental risk and cleanup strategies affecting their community. Served as technical support to USAF public affairs officials working within a community directly impacted by PFAS-contaminated drinking water. Crafted risk communication tools and products, coordinated public meetings and agenda topics, and ensured that complex toxicology information was translated appropriately to the public.

Data Gap Analysis, U.S. Air Force Civil Engineer Center, Lackland, Texas. Identified gaps in scientific knowledge needed for the USAF and DOD to protect human and environmental health. Specific topics were vapor intrusion, chlorinated solvents, trichloroethylene, perfluorinated chemicals, 1,4-dioxane, and pesticides.

Toxicology

PFAS Product Stewardship, United States. Provides technical consulting support on short-chain PFAS and related fluorochemical products to the FluoroCouncil. Conducts scientific assessment and assist with stakeholder communications related to the health and environmental risk of short-chain PFAS and fluorotelomers.

Technical Peer Review of Federal and State Agency Guidance Documents, United States. Provides technical peer review of toxicology assessments, risk assessments, and guidance documents on behalf of clients, including peer reviews of EPA IRIS and Toxic Substances Control Act (TSCA) assessments, ATSDR toxicological profiles, California Office of Human Health and Environmental Assessment documents, and other state regulatory agency assessments. Numerous clients and chemicals.

Technical Review and Comment on the New York Department of Health Proposed Rulemaking for 1,4-Dioxane Maximum Contaminant Level (MCL) (I.D. NO. HLT-30-19-00006-P). Conducted a review of the current toxicological data related to 1,4-dioxane's carcinogenic human health risks and authored a comment letter to the New York State Department of Health on the technical validity of their proposed MCL.

Short-chain PFAS and Fluoropolymer Toxicology and Regulatory Support, United States. Provides toxicology support to a confidential client working to obtain regulatory approval for current PFAS-containing products.

Federal Toxicology and Risk Assessment Reviews, United States. Served as a member of the federal interagency review team providing consultation and expert review on nearly all toxicology assessments and/or guidance documents produced by EPA, NTP, and ATSDR. Assessed the technical validity, transparency of decisions, adherence to agency and other federal guidance, and overall technical competency of the risk assessments. Work included submitting detailed written comments and participating in interagency teleconferences and working meetings.

Technical Review and Comment on the New Jersey Drinking Water Quality Institute (DWQI) Maximum Contaminant Level Recommendation for 1,2,3-Trichloropropane (1,2,3-TCP), New Jersey. Conducted a critical review of the toxicology, epidemiology, toxicokinetic, and other studies relevant to 1,2,3-TCP

human health effects. Technical comments are part of the administrative record and continue to be considered by DWQI.

Technical Review of Pentachlorophenol Epidemiology Data in Response to proposed California Proposition 65 Listing, California. Provided critical review and analysis of the developmental and reproductive epidemiology data on pentachlorophenol in response to the California Developmental and Reproductive Toxicant Identification Committee review and proposed listing under Proposition 65.

Toxicology Evaluation of Remedial Action Objectives, California. Conducted an in-depth assessment of outdated remedial action objectives for a confidential contaminated site in California. Reevaluated the toxicology and quantitative risk assessment for a specific unregulated contaminant of concern at the site. Calculated new screening levels based on updated risk assessment methodologies to ensure that remediation actions remain protective of public health.

Human Health Chemical Hazard Identification and Dose-Response, Federal Agencies, Cincinnati, Ohio. Provided management and scientific expertise for chemical assessments performed under Superfund, IRIS, and other programs. Served on high-performance, interdisciplinary scientific teams for dioxin reassessment, computational toxicology, phthalate cumulative risk, and mode of action.

Human Health Risk Assessment (HHRA)

Baseline Human Health Risk Assessment for PFAS, Confidential Location. Serves as project manager and technical lead for a PFAS baseline human health and ecological risk assessment. Develops conceptual site models for assessing human and ecological receptor exposures. Manages selection of toxicity values and review of literature to identify primary mechanisms of action for toxicity relevant to site-specific human exposure pathways.

1,4-Dioxane Site-Specific Risk Assessment and Consulting Support Related to Public Drinking Water System Contamination, Confidential Location. Provides risk assessment and technical support related to the regulatory basis and public health impacts of 1,4-dioxane in a public drinking water system.

EPA Toxic Substances and Control Act, Low Volume Exemption Application, Confidential Client. Provided human health toxicology and exposure assessment to support a low volume exemption (LVE) application to EPA TSCA program. Conduct analysis, develop report, and assist with in-person presentation to EPA TSCA technical staff.

Environmental Risk Assessment Oversight, U.S. Air Force Civil Engineering Center, Lackland, Texas. Provided toxicology expertise and oversight of risk assessments conducted for the USAF Environmental Restoration Program's CERCLA and RCRA activities. Using EPA's risk assessment guidance, interpreted toxicology data to assess risks to human health and the environment, and reviewed site-specific risk assessments conducted at USAF installations nationwide.

PUBLICATIONS

(J.K. Anderson also published as J.K. Hess-Wilson)

Articles and Peer-Reviewed Publications

Goodrum, P.E., Anderson, J.K., Luz, A.L. and Ansell, G.K., 2020. Application of a Framework for Grouping and Mixtures Toxicity Assessment of PFAS: A Closer Examination of Dose Additivity Approaches. *Toxicological Sciences*.

Mohr, T.K., DiGuiseppi, W.H., Hatton, J.W. and Anderson, J.K., 2020. *Environmental investigation and remediation: 1, 4-dioxane and other solvent stabilizers*. CRC Press.

Iwai, H., A.M. Hoberman, P.E. Goodrum, E. Mendelsohn, and J.K. Anderson. 2019. Addendum to Iwai and Hoberman (2014) – Reassessment of developmental toxicity of PFHxA in mice. *Internat J Tox.* 38(3):183-191.

Anderson, J.K., A.L. Luz, and P. Goodrum. 2019. Response to "Overgeneralization by Anderson et al. and Luz et al. regarding safety of fluorotelomer-base chemistry". *Reg Tox Pharm*. 105:100-101.

Anderson, J.K., A.L. Luz, P. Goodrum, and J. Durda. 2019. Perfluorohexanoic acid toxicity, part II: application of human health toxicity value for risk characterization. *Reg Tox Pharm.* 103: 10-20.

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Anderson, J., J. Wilhelm, and P. Goodrum. 2016. Emerging contaminants: An analysis of inconsistent U.S. regulations. *Daily Environment Report*. Bloomberg Bureau of National Affairs. August.

Anderson, R.H., G.C. Long, R.C. Porter, and J.K. Anderson. 2016. Occurrence of select perfluoroalkyl substances at U.S. Air Force aqueous film-forming foam release sites other than fire-training areas: field-validation of critical fate and transport properties. *Chemosphere.* 150:678–685.

Anderson, R.H., J.K. Anderson, and P.A. Bower. 2012. Co-occurrence of 1,4-dioxane with trichloroethylene in chlorinated solvent groundwater plumes at U.S. Air Force installations; fact or fiction. *Integr Environ Assess Manag.* 8(4):731–737.

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Thomas, R.S., H.C. Clewell, B.C. Allen, S.C. Wesselkamper, N.Y. Wang, J.C. Lambert, J.K. Hess-Wilson, Q.J. Zhao, and M.E. Andersen. 2011. Application of transcriptional benchmark dose values in quantitative cancer and noncancer risk assessment. *Toxicol Sci*. 120(1):194–205.

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Hess-Wilson, J.K. 2009. Bisphenol A may reduce the efficacy of androgen deprivation therapy in prostate cancer. *Cancer Causes and Control.* 20(7):1029–1037.

Shah, S., J.K. Hess-Wilson, S. Webb, H. Daly, S. Godoy-Tundidor, J. Kim, J. Boldison, Y. Daaka, and K.E. Knudsen. 2008. 2,2-Bis(4-chlorophenyl)-1,1-dichloroethylene stimulates androgen independence in prostate cancer cells through combinatorial activation of mutant androgen receptor and mitogen-activated protein kinase pathways. *Mol Cancer Res.* 6(9):1507–1520.

Hess-Wilson, J.K., S.L. Webb, H.K. Daly, Y. K. Leung, J. Boldison, C.E.S. Comstock, M.A. Sartor, S.M. Ho, and K.E. Knudsen. 2007. Unique bisphenol A transcriptome in prostate cancer: novel effects on ERβ expression that correspond to AR mutation status. *Environ Health Perspect.* 115(11):1646–1653.

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White Paper – Human health risks to perfluorinated compound exposure through drinking water and appropriate risk-based screening values. March 2015.

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Emerging Issues/Contaminants Program – Preliminary evaluation and background report on lead. June 2013.

Interim AF guidance on sampling and response actions for perfluorinated compounds at active and BRAC installations. September 2012.

Perchlorate – Background on the EPA MCLG proposal and industry challenges. July 2012.

Position Paper – Impact analysis and cost impact of AF environmental liability to perfluorinated compounds. April 2012.

Position Paper – TCE impact assessment. April 2012.

Bullet Background Paper – The potential impact of USEPA's dioxin non-cancer assessment on AF installations and PBR efforts. February 2012.

Emerging Issues/Contaminants Program – Background and preliminary assessment on hexavalent chromium. November 2011.

Bullet Background Paper – Health impact of the final EPA TCE toxicity values. October 2011.

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USEPA. 2010. Recommended toxicity equivalence factors (TEFs) for human health risk assessments of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and dioxin-like compounds. EPA/100/R 10/005. U.S. Environmental Protection Agency, Risk Assessment Forum, Washington, DC. Coauthor.

SELECTED PRESENTATIONS and POSTERS

Anderson, J.K., and P. Goodrum. 2019. PFAS: Toxicology and Regulatory Actions. Webinar to the ACC Public Health Advisory Board. November 7, 2019

Luz, A., C. Hutchings, J. Anderson, P. Goodrum, J. Field. 2019. A Novel Approach for Assessing Hazard Associated with Firefighting Foams. Poster at the SETAC North American 40th Annual Meeting, Toronto Ontario, Canada. November 4.

Anderson, J.K. 2019. Federal and State Environmental Guidance/Policies that Impact Remedial Decisions for PFAS. Platform presentation at the Washington State Advanced Superfund Conference. September 12, Seattle, WA.

Anderson, J.K. 2019. PFAS: Risk Characterization Panel. Invited panelist to the Society of Environmental Toxicology and Chemistry North America, Focused Technical Meeting on PFAS. Durham, NC. August.

Anderson, J.K., A. Luz, and P. Goodrum. 2019. Chronic human health toxicity value for perfluorohexanoate (PFHxA) and risk assessment relevant to current fluorotelomer-based chemistries. Poster for the Society of Toxicology 58th Meeting and ToxExpo, March 10–14, Baltimore, MD.

Goodrum, P., J.K. Anderson, and A. Luz. 2019. Perfluoroalkyl acid mixtures—Data analysis steps to uncover clues hidden in biomonitoring data. Poster for the Society of Toxicology 58th Meeting and ToxExpo, March 10–14, Baltimore, MD.

Luz, A., J.K. Anderson, and P. Goodrum. 2019. Approaches for Assessing Perfluoroalkyl Acid Mixture Toxicity. Poster for the Society of Toxicology 58th Meeting and ToxExpo, March 10–14, Baltimore, MD.

Opdyke, D., J. Benaman, J.K. Anderson, and J. Durda. 2019. An introduction to PFAS at contaminated sediment sites: Scientific and regulatory overview. Short course at Tenth International Conference on the Remediation and Management of Contaminated Sediments, February 11–14, New Orleans, LA.

Wilhelm, J., J.K. Anderson, A. Luz, and P. Goodrum. 2018. PFAAs and ecorisk: Development of a hazard ranking system by evaluating functional groups vs. chain lengths as primary risk drivers for ecological receptors. Poster presentation. SETAC North American 39th Annual Meeting, November 4–7, Sacramento, CA.

Luz, A.L., L. Tolbert, J.K. Anderson, P. Goodrum, D. Farrar, and S. Korzeniowski. 2018. PFHxA human health risks, margin of safety, and comparison with PFOA. Platform presentation. Society of Environmental Toxicology and Chemistry North America 39th Annual Meeting. November 4–8. Sacramento, CA.

Anderson, J.K. 2018. Emerging contaminants—per-and polyfluoroalkyl substances: A case study. Invited speaker. Texas Environmental Superconference, August, Austin, TX.

Anderson, J.K., and P. Goodrum. 2018. Internal and external dosimetry—the holy grail to decoding perfluoroalkyl acid toxicity? Poster presented at the Emerging Contaminants Summit, March 6–7, Westminster, CO.

Anderson, J.K., and P. Goodrum. 2018. What does that blood level mean? The assumptions underlying interpretations of health effects from internal doses. Poster presented at the Society of Toxicology 57th Annual Meeting and ToxExpo, March 11–15, San Antonio, TX.

Goodrum, P., and J.K. Anderson. 2018. Application of internal dosimetry for perfluoroalkyl acids and methods to assess uncertainty factors used in risk assessment. Poster presented at the Society of Toxicology 57th Annual Meeting and ToxExpo, March 11–15, San Antonio, TX.

Anderson, J.K. 2017. Uncertainty in the science of toxicology and emerging contaminants. Remediation of Emerging Contaminants: Trends in Science and Regulations. Montclair State University Continuing Education Course. June.

Anderson, J.K. 2017. Why the inconsistent and dynamic state and federal chemical regulatory landscape. RTM Communications Conference, Philadelphia, PA. April.

Anderson, J.K. 2016. Inconsistent and dynamic state and federal chemical regulations: Roadmap to success. Consumer Specialty Product Association annual conference. December.

Anderson, J.K. 2016. How did we get here from there? State and Federal regulatory actions for PFAS. AEHS Annual East Coast Conference. October.

Frankel, A., P.E. Goodrum, J.K. Anderson, and K. Tsitonaki. 2016. Water quality standards for perfluoroalkyl compounds—Cross roads between regulatory toxicology and remedy selection. Platform presentation, Battelle 10th International Conference on Remediation of Chlorinated and Recalcitrant Compounds, Palm Springs, CA.

Anderson, J.K., N. Edlin, and S. Herman. 2016. Keeping a watchful eye on emerging contaminants. Environmental and Emerging Claim Managers Association annual conference. April.

Anderson, J.K. 2016. Emerging contaminants: analytical, toxicity, regulatory, and legal frontiers. Invited panelist to the Emerging Contaminants Summit. March.

Anderson, J.K., and P.E. Goodrum. 2016. Emerging contaminants: crossroads of uncertain science and risk management. Integral Webinar Series. February.

Anderson, J.K., and P.E. Goodrum. 2015. Status of regulatory decisions for perfluoroalkyl compounds: is the level of protection to the general public worth the uncertainty and cost? Poster presented at Society for Risk Analysis, Washington, DC.

Anderson, J.K. 2015. Overview of regulatory toxicology in the development of federal and state MCLs for perfluoroalkyl compounds. AEHS Annual East Coast Conference. October.

Anderson, J.K. 2014. AF approach to emerging issues & contaminants. Webinar presented to Society of Military Engineers. November.

Anderson, J.K. 2014. AF Emerging Issues & Contaminants Program: 1,4-dioxane and PFCs. Webinar presented to State Risk Assessors Teleconference. October.

Anderson, J.K. 2014. AF Emerging Issues & Contaminants Program: 1,4-dioxane and PFCs. Presented to Air Force Institute of Technology. October.

Anderson, J.K. 2014. Air Force Civil Engineering Center (AFCEC) Emerging Issues & Contaminants Program. Air Force Institute for Technology training sessions, Wright-Patterson Air Force Base, OH. August.

Philips, J.K., and J.K. Anderson. 2013. Challenges associated with practical environmental restoration risk assessment and management decisions for perfluoroalkyl substances (PFASs). Poster presented at Society for Risk Analysis Annual Meeting, Baltimore, MD. December.

Bodour, A., and J.K. Anderson. 2013. AFCEC Emerging Contaminants & Broad Agency Announcement Programs. Webinar presented to Federal Remediation Technology Roundtable, Arlington, VA. November.

Woodward, D., G. Hohenstein, J. Field, J. Phillips, D. Chiang and J.K. Anderson. 2012. Emerging contaminants: perfluorinated compounds (PFCs). Webinar presented to Society of American Military Engineers, Continuing Education. November.

Anderson, J.K. 2012. The AF Emerging Issues Program: the curious derivation of toxicity values for perfluorinated compounds. Presented to Tri-Service Toxicology Consortium, Dayton, OH. January.

Anderson, J.K., and A. Bodour. 2011. AFCEE research activities related to 1,4-dioxane—emerging issues program and broad agency announcement overview. Presented at Tucson International Airport Area Superfund Site Annual Information Exchange, Tucson, AZ. September.

Anderson, J.K. 2011. Air Force Emerging Issues/Emerging Contaminants Program. Presented at Restoration and Technology Transfer Workshop, San Antonio, TX. April.

Anderson, J.K. 2010. Cancer classification and mode of action for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). Presented at the 30th International Symposium on Halogenated Persistent Organic Pollutants, San Antonio, TX. September.

Anderson, J.K. 2010. EPA's provisional human health risk assessment process. Presented at Restoration and Technology Transfer Workshop, San Antonio, TX. April.

Anderson, J.K. 2009. TCDD cancer dose response background information and discussion. Session chair. TCDD and cancer dose response. Dioxin Workshop, Cincinnati, OH. February.

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June 25, 2021

VIA E-MAIL

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Re: Proposal for Update to Part 620, Groundwater Quality Regulations

Midwest Generation LLC ("MWG") appreciates the opportunity to provide comments on the proposed update to the Part 620 Groundwater Quality Regulations ("Proposed Part 620 Rule"). MWG's comments primarily address Illinois EPA's methodology in developing the proposed changes to the Class I and Class II groundwater standards in sections 620.410 and 620.420.

To prepare these comments, MWG obtained the expert assistance of Dr. Janet K. Anderson, PH.D., D.A.B.T., of GSI Environmental, Inc. (GSI). Dr. Anderson has over 15 years of experience providing toxicology, risk assessment, and risk management support to federal and state regulatory agencies, municipalities, and private industries. A copy of her curriculum vitae is attached to her enclosed report which sets forth her review and comments on the proposed changes to the Class 1 and Class II groundwater standards. Specifically for metals, Dr. Anderson makes the following conclusions:

- The application and use of default exposure parameters for children and misinterpretation and misapplication of the U.S. Environmental Protection Agency's (USEPA) policy and guidance on toxicity value hierarchy does not allow Illinois EPA or the regulated community to use the best available science to support sound decision making on a chemical-by-chemical basis.
- The proposed default relative source contribution (RSC) of 0.2 for boron is not supported by best available science and is inconsistent with conclusions rendered by the USEPA.

A detailed explanation of these conclusions is contained in Dr. Anderson's enclosed report.

We appreciate the opportunity afforded by the Agency to submit these comments. If you have any questions or need additional information, please contact me.

Very truly yours,

Kristen L. Gale

CC: Sharene Shealey, Midwest Generation, LLC

June 25, 2021

Illinois Environmental Protection Agency

Sent via email: EPA.620.rulemaking@illinois.gov
Subject: 35 Ill. Adm. Code 620 Proposed Updates, Public Comment

RE: ILLINOIS EPA DRAFT PROPOSED UPDATES TO 35 ILL. ADM. CODE 620; GROUNDWATER QUALITY

Thank you for the opportunity to provide these comments on behalf of Midwest Generation, LLC, on the draft proposed changes to the Illinois Part 620 Groundwater Quality rules. I am a Diplomate of the American Board of Toxicology and Principal Toxicologist with GSI Environmental, Inc. (GSI). I have over 15 years of experience providing toxicology, risk assessment, and risk management support to federal and state regulatory agencies, municipalities, and private industries. A copy of my CV is attached. I provide these comments based on my experience and expertise with regulatory toxicology and risk-based regulations.

OVERALL COMMENTS

In response to the Illinois Environmental Protection Agency (Illinois EPA)'s request for public comment on the proposed draft language updates to 35 Ill. Adm. Code 620 ("Proposed Part 620"), GSI has reviewed the information made available on the IL EPA website (https://www2.illinois.gov/epa/about-us/rules-regs/water/Pages/620-Groundwater-Quality.aspx) and participated in the May 26, 2021 virtual public meeting. Based on review of these materials and my expertise in regulatory toxicology and risk management strategies, I offer the following comments on the Proposed Part 620:

> **The application and use of default exposure parameters for children, and misinterpretation and misapplication of the U.S. Environmental Protection Agency's (USEPA) policy and guidance on toxicity value hierarchy, does not allow Illinois EPA or the regulated community to use the best available science to support sound decision making on a chemical-by-chemical basis.**
>
> **The proposed default relative source contribution (RSC) of 0.2 for boron is not supported by best available science and is inconsistent with conclusions rendered by the USEPA.**

These comments are explained in further detail below.

1. **The application and use of default exposure parameters for children, and misinterpretation and misapplication of the U.S. Environmental Protection Agency's (USEPA) policy and guidance on toxicity value hierarchy, does not allow Illinois EPA or the regulated community to use the best available science to support sound decision making on a chemical-by-chemical basis.**

The Proposed Part 620 proposes a significantly different, and not scientifically supported, methodology for adopting groundwater quality standards. The current 35 Ill. Adm. Code 620 ("Part 620") adopts as groundwater quality standards (GQS) risk-based or water quality-based values from federal agencies such as the USEPA. The Proposed Part 620 will allow Illinois EPA to calculate their own GQS for both noncarcinogens ("Human Threshold Toxicant Advisory Concentration") and carcinogens ("Human Nonthreshold Toxicant Advisory Concentration"), as described in Section 620 Appendix A. The Proposed Part 620 also will require the use of child body weight, child water consumption rate, and a relative source contribution (RSC) of 0.2. It also requires the use of USEPA Integrated Risk Information System (IRIS) toxicity values, if available, for the chemical of interest, as IL EPA claims this is USEPA's "hierarchy of usable sources". However, collectively, these new requirements for calculations are too prescriptive and do not allow for best available and sound science to be used on a chemical-by-chemical basis.

Default use of childhood exposure parameters results in compounded conservatism and is not consistent with USEPA Guidance. Generally, in any risk analysis, risk is calculated as toxicity of a chemical combined with exposure to the chemical, including time and concentration. Thus, to calculate risk for deriving regulatory threshold criteria, such as GQS, default, conservative exposure parameters are often used, which provide an assumption of the worst-case exposure scenario. The exposure parameters include estimates of the magnitude, frequency, and duration of exposure to the chemical in the relevant environmental media (in this case, groundwater). Exposure parameters specific for various receptors (e.g., body weight and drinking water ingestion rate) can be used such as for a child, an average adult, or a pregnant woman. The inherent variability in exposure for individuals is addressed by using high-end exposure estimates for the receptor subgroup.

The use of a distinct equation combining a toxicity value with exposure parameters can be mandated by legislative authority and regulatory paradigms. However, requiring the application and use of conservative receptor exposure parameters, such as the child body weight, child drinking water ingestion rates, and the lowest RSC, often results in compounded conservatism and inaccurate prediction of risk.

Here, in the Proposed Part 620, Illinois EPA essentially adopts the USEPA Regional Screening Levels methodology for noncarcinogen chemicals, except Illinois EPA only uses child parameters. Illinois EPA's proposal to require the use of the child exposure assumptions in the Proposed Part 620 GQS derivation methods are not consistent with USEPA guidance. In general, childhood exposures are most often used to address shorter duration exposure or to define conservative, initial screening levels, while adult exposure assumptions are used for

lifetime or chronic exposures and more commonly used for regulatory decision making. The USEPA screens noncarcinogen chemicals in groundwater sites using both child and adult parameters to ensure conservative protective assumptions are used to identify impacted groundwater that may need further evaluation. Due to how noncancer risk-based standards for potable water are traditionally calculated, use of a child's body weight and water ingestion rate result in a "high-end" exposure estimate and, therefore, a lower, more stringent GQS, because the child is likely to receive a greater dose on a milligram per kilogram per body weight basis. Use of the most conservative child exposure assumptions provide a first step screen, to determine if follow-up investigations may be necessary. However, USEPA Regional Screening Levels are not cleanup levels and should not be interpreted as regulatory criteria that cannot be exceeded. USEPA clearly states "**It should be emphasized that [screening levels] are *not cleanup standards*.**" (emphasis added)[1]. In fact, for most noncarcinogen chemicals, USEPA's Drinking Water Standards and Lifetime Health Advisories rely on adult body weight and water consumption rate; only the one-day and ten-day advisories utilize child exposure parameters (USEPA 2018).

In some cases, it may not only be overly conservative, but also technically incorrect to utilize a child's exposure parameters when deriving a GQS. The USEPA guidance on developmental toxicity, for example, states that the final risk characterization for a chemical needs to include information on exposure route, timing and duration of exposure specific for the toxicity value (RfD), and "…it would be inappropriate in developmental toxicity risk assessments to use [] adjustment of exposure over a different time frame than that actually encountered…" (USEPA 1991, p. 45). In other words, it is inappropriate to mismatch the exposure parameters for one receptor with a toxicity value (RfD) derived from a different receptor's exposure scenario.

The USEPA RfD used by Illinois EPA for the proposed GQS for boron is based on a development effect that occurs *in utero*. When deriving the lifetime Health Advisory for boron, USEPA explicitly stated that "the target population is pregnant women because the *in utero* development endpoint is the most sensitive." (USEPA 2008a). Therefore, the USEPA RfD for boron should be combined with exposure parameters specific for the pregnant woman (consistent with the USEPA Office of Water). The Illinois EPA should not use child exposure parameters with an *in utero* effect. Illinois EPA should have the flexibility to apply the best available science that is technically sound, and make sure that the most sensitive effect for a chemical matches the receptor parameters used in the GQS calculation.

Automatic use of USEPA IRIS toxicity values conflicts with USEPA policy and guidance and with risk assessment best practices. Beginning with USEPA Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part A), USEPA guidance recommends selecting toxicity criteria based on the most recent data (USEPA 1989, p. 7-15). This recommendation has since been implemented in numerous USEPA directives (USEPA 1993, 2003) that further establish a hierarchy and process for selecting toxicity criteria. The USEPA IRIS assessments are generally considered the top-tiered choice, based on their use of

[1] https://www.epa.gov/risk/regional-screening-levels-rsls-users-guide

standardized methods and rigorous peer review; however, IRIS toxicity values for each chemical are not always recent and up to date. Importantly, comparison of available toxicity values across multiple sources to ensure validity of the value is now a routine part of regulatory toxicology best practices (USEPA 2003, 2013a; ECOS 2007; Illinois EPA Part 320 Section 302.606[2]). Evaluation of multiple sources of toxicity information ensures that the information used is current, peer reviewed, transparent, and the best available information. This flexibility recognizes that new chemical-specific information may become available and that risk assessment practices are continually evolving; therefore, selection of a toxicity value should be based on the most recent, credible, and relevant data, as well as, incorporating the best risk assessment methods available. As stated in the original USEPA 1993 directive, "…in some cases more recent, credible and relevant data may come to the Agency's attention. … [T]he Agency should evaluate risk based upon its best scientific judgment and consider all credible and relevant information available to it" (USEPA 1993, p. 2).

Here, the Illinois EPA's proposal is to limit the toxicity criteria to the USEPA IRIS toxicity values regardless of whether there is updated information in another appropriate source. This is against best practices because it could rely upon outdated data. Instead, the Part 620 rule should allow the Illinois EPA to look to all credible and relevant information available instead of only the USEPA IRIS toxicity values.

Combined, the Proposed Part 620 adopts GQS and a methodology for deriving GQS that does not allow the Illinois EPA or regulated community to use sound science to support decision making. It locks users into default values and methods without consideration of chemical-specific best available information. The Proposed Part 620 GQS and methods may result in compounded conservatism, which creates GQS that are unnecessarily low.

2. The default relative source contribution (RSC) of 0.2 for boron is not supported by best available science and is inconsistent with conclusions rendered by the USEPA.

The regulatory concept underlying the use of RSC is that the criterion set for a single exposure pathway such as drinking water needs to also allow for the potential for exposure to occur from other pathways (e.g., diet, ingestion, dermal absorption). By determining the fraction of total exposure attributable to non-drinking water pathways, one can determine the "balance" of the exposure that cannot be exceeded from the drinking water pathway alone. Current USEPA guidance recommends determining an appropriate RSC value within the range of 20 to 80 percent (USEPA 2013b; 2000). The low-end value of 20 percent is a health protective assumption that is applied in the absence of chemical-specific data on exposure. It assumes that 80 percent of the target dose can be attributed to (or allocated to) exposures other than drinking water, while the remaining 20 percent is due to exposure via drinking water. USEPA strongly encourages States to consider available data to derive chemical-specific RSC estimates (USEPA 2000). Rather than requiring the default RSC of 0.2 for all chemicals, Illinois EPA should adopt USEPA's guidance for using the Exposure Decision Tree approach described in the

[2] Section 302.606 of the Illinois Pollution Control Board ("Board") Rules requires that the Agency reviews all data used in calculating water criteria based on "validity, applicability and completeness". 35 Ill. Adm. Code 302.606.

Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (USEPA 2000).

Specifically, the proposed GQS for boron is inconsistent with the current state-of-the-science for boron. Illinois EPA should fully consider the relevant chemical-specific information currently available for boron, and allow for the best science, rather than default parameters, to be used when deriving the GQS. Evaluations conducted by the USEPA demonstrate that a RSC of 0.8 for boron is consistent with the state-of-the-science and would result in a GQS that is still protective of human health (USEPA 2008a). USEPA determined that data were available to describe anticipated exposures to boron from different sources, including diet. The USEPA Office of Water Health Effects Support Document for Boron (USEPA 2008b) summarized data describing the exposure to boron by the general public from food, air, soil, and insecticide use, and were able to use available data to calculate a chemical-specific RSC, following USEPA guidance and best practice (USEPA 2000).

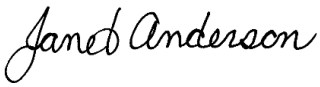
As summarized by several authoritative agency documents, there are quality studies that have quantified exposure levels for boron to various population groups (e.g., ATSDR 2010, USEPA 2004, IOM 2001). The National Academies Institute of Medicine (IOM) 2001 dietary report concludes that dietary sources represent the main background intake for boron and provide the quantitative support for the USEPA boron-specific calculation. IOM (2001) summarized the available literature as of 2001, including data collected by the Centers for Disease Control and Prevention, National Health and Nutrition Examination Survey (NHANES), and established mean boron intakes per day from dietary and supplement sources (see IOM 2001 Appendix C Tables C-12 and C-13). The information specific to pregnant women intake (to match the USEPA IRIS RfD associated with developmental effects *in utero*) of boron from dietary sources was used by the USEPA to calculate a chemical-specific RSC for boron of 0.8 (USEPA 2008a).

The ability within the GQS process to use the best available science is the most true and accurate application of USEPA policy and guidance. Specifically for boron, an analysis of the available data clearly demonstrates that the RSC should be 0.8.

CONCLUSION

To make a health-based GQS for any chemical without conducting a thorough evaluation of the current state-of-the-science, would be arbitrary. Indeed, Section 302.606 of the Board Rules requires that the Agency reviews all data used in calculating water quality criteria based on "validity, applicability and completeness". 35 Ill. Adm. Code 302.606. Accordingly, we suggest that the Illinois EPA revise the draft proposed update to include use of best available chemical-specific information. In doing so, Illinois EPA should also use the authoritative and peer-reviewed analysis of boron exposure through non-drinking water sources and USEPA's calculation of a chemical-specific RSC of 0.8. These revisions to the Proposed Part 620 would result in the use of reasonable, scientifically valid parameters that can be used to derive public health protective and technically sound GQS.

Respectfully submitted,



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References

ATSDR. 2010. Toxicological Profile for Boron. U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. Atlanta, GA. November.

Environmental Council of the States (ECOS). 2007. Identification and Selection of Toxicity Values/Criteria for CERCLA and Hazardous Waste Site Risk Assessments in the Absence of IRIS Values. ECOS-DoD Sustainability Work Group, Washington, D.C.

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USEPA. 2008b. Health Effects Support Document for Boron. EPA-822-R-08-002. Health and Ecological Criteria Division, Office of Science and Technology, Office of Water, U.S. Environmental Protection Agency, Office of Water, Washington, DC. January.

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USEPA. 2013b. Human health ambient water quality criteria and fish consumption rates: Frequently Asked Questions. Available: https://www.epa.gov/sites/production/files/2015-12/documents/hh-fish-consumption-faqs.pdf U.S. Environmental Protection Agency, Office of Water, Washington, DC. January.

January 2020

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Biographical Summary

Dr. Janet Anderson is a Principal human health toxicologist and environmental risk assessor with 15 years of experience providing toxicology expertise and consultation to federal agencies and industry. She specializes in the translation of human health toxicology data into state and federal regulatory policy decisions and performs critical reviews of federal and state risk assessment guidance and regulations. She has also provided litigation consulting support and served as an expert witness.

Dr. Anderson is a nationally recognized leader in unregulated and emerging chemicals, such as per- and polyfluoroalkyl substances (PFAS), 1,4-dioxane, and 1,2,3-trichloropropane. With in-depth knowledge of federal and state environmental guidance and policies pertaining to this class of compounds, she has developed strategies to mitigate their human health impacts and address associated environmental liability for both private and public sector clients. She tracks the dynamic regulatory changes for emerging chemicals in the U.S. and internationally, offering clients the technical basis for disparate guidelines worldwide. She has extensive experience developing risk management strategies for multi-stakeholder groups.

Previously, as a civilian government employee, Dr. Anderson led the U.S. Air Force (USAF) Emerging Issues and Contaminants Program, where she developed programmatic recommendations on environmental regulations and cleanup standards and assisted with site-specific remediation. She also served as a member of the federal interagency review team providing consultation and expert review on toxicology assessments and/or guidance documents produced by EPA, the National Toxicology Program (NTP), and the Agency for Toxic Substances Disease Registry (ATSDR). As a postdoctoral fellow for the EPA Office of Research and Development National Center for Environmental Assessment, she managed numerous Superfund chemical assessments and served as a team member for Integrated Risk Information System (IRIS) assessments.

Dr. Anderson is a diplomate of the American Board of Toxicology and an active member of the Society of Toxicology. A skilled communicator, she is often an invited speaker at high-level scientific conferences, regulatory meetings, webinars, and community stakeholder meetings.

Education

Ph.D., Molecular and Cancer Biology, University of Cincinnati, College of Medicine, Cincinnati, Ohio, 2007

B.A., Biology and Women's Studies, Wittenberg University, Springfield, Ohio, 2000

Post-Doctoral Fellow, EPA Office of Research and Development National Center for Environmental Assessment, Cincinnati, Ohio, 2007–2010

Diplomate, American Board of Toxicology, 2012–present

Professional Background

Principal, GSI Environmental Inc., Houston, Texas, 2020 – current

Senior Associate, GSI Environmental Inc., Houston, Texas, 2019

Senior Consultant, Integral Consulting Inc., San Antonio, Texas, 2015 – 2019

Emerging Issues and Contaminants Program Manager, Subject Matter Specialist – Toxicology, US Air Force Civil Engineer Center, San Antonio, Texas, 2010 – 2015

Professional Certifications and Affiliations

Member of Society of Toxicology, Risk Assessment Specialty Section, Women in Toxicology Special Interest Group, and Lone Star Regional Group

Interstate Technology and Regulatory Council (ITRC) Workgroup on Per- and Polyfluoroalkyl substances (2017–present)

Interstate Technology and Regulatory Council (ITRC) Workgroup on 1,4-Dioxane (2019–present)

Ad Hoc Panelist for Alliance for Risk Assessment, "Beyond Science and Decisions"

Continuing Education and Training

Linkage, Women in Leadership Training (2020)

Physiologically Based Pharmacokinetic Modeling to Support Modernized Chemical Safety Assessment, Society of Toxicology Continuing Education Course (2018)

EPA Environmental Risk Assessment Guidance Training, EPA Environmental Response Training Program (2011)

Environmental Negotiations Workshop, Naval Civil Engineer Corps Officers School (2010)

The Hamner Institute's Computational Systems Biology, Research Triangle Park (2008)

Physiologically Based Pharmacokinetic Modeling, Dr. Ray Yang (2008)

International Life Sciences Institute Human Relevance Framework for Weight of Evidence Workshop, Tolerance Reassessment Advisory Committee (2008)

Toxicology Excellence for Risk Assessment Dose-Response Boot Camp (2007)

EPA's Benchmark Dose Training (2007)

Project Experience

Litigation Expert and Consulting Services

Expert Services, Aqueous Film Forming Foam (AFFF) and PFAS Toxicological History and Regulations—On behalf of the U.S. Department of Justice, Environmental & Natural Resources Division, provide consultation and expert opinion on the regulatory and human health toxicological history of AFFF and PFAS. *(Penna v. The United States of America, in the United States Court of Federal Claims,* Case No. 16-1545L).

Expert Services, Carbon Monoxide, California—On behalf of ITW Food Equipment Group, LLC, served as expert testifying witness on the regulatory toxicity values for, and process for human health risk evaluation of, carbon monoxide. (*Julie Lee, Julie Lacey, Lourdes Munoz, Martha Silva, Brandon Adams, Lafayette Wallace, Joshlynn Jarboe, Yolanda Rodriguez, Peter Lee and Mark Rodriguez v. Hobart Corporation, Wayne Home Equipment, A Scott Feitzer Co., A.M. Wighton & Sons, Inc., DBA A&J Refrigeration. In the Superior Court of Santa Barbara, Cook Division, California,* Case Number: 1389541).

Expert Services, 1,2,3-Trichloropropane, California—On behalf of Shell and Dow Chemical Co., served as expert on the use and interpretation of regulatory standards and toxicity values for 1,2,3-trichloropropane.

Expert Services, Dieldrin and Aldrin, Florida—On behalf of Shell Oil Company, served as expert testifying witness and authored a detailed expert report on the regulatory toxicity values for, and human health risk evaluation of, dieldrin and aldrin. (*Janice Potter, Brian Potter, David Stepp, Debra Stepp, Renee Bolton, Yvonne Hopp, Herman Osterloh, Morgan Canada and Lauren Kelly, Class Representatives v. Shell Oil Company and DeLand Golf Course, Inc. In the Circuit Court, Seventh Judicial Circuit, in and for Volusia County, Florida,* Case Number: 2011-11036-CIDL Division).

Expert Services, Portland, Maine—On behalf of Mallinckrodt U.S., LLC, served as expert testifying witness regarding human health risks and related remedial action of methylmercury in biota and sediments in the Lower Penobscot River and Estuary. *(Natural Resources Defense Council et al. v. HoltraChem Manufacturing Company, LLC et al., U.S District Court, District of Maine,* Civil Action No. 1:00-cv-00069-JAW*).*

Litigation Support Services, Alaska—On behalf of Williams Alaska Petroleum, provided technical support to the testifying expert on the appropriate toxicity values for site-specific risk assessment on sulfolane. (*State of Alaska et al. vs. Williams Alaska Petroleum et al., Shook, Hardy, and Bacon In the Superior Court for the State of Alaska Fourth Judicial District Court,* Case No. 4FA-14-01544CI).

Consulting Support Services, Perfluoroalkyl Substance Contamination—For a confidential client, provided technical and strategic support related to the regulatory processes and toxicological assessments for PFAS.

Consulting Support Services, p-Chlorobenzenesulfonic Acid Contamination—For a confidential client, provided regulatory support and toxicology assessment.

Dispute Resolution, U.S. Air Force, Lackland, Texas—Provided technical support to USAF legal offices and program managers engaged in federal and state dispute resolution related to emerging issues and contaminants, including trichloroethylene, tetrachloroethylene, 1,4-dioxane, and perfluorinated compounds.

Emerging Chemicals Strategies and Management

Regulatory Tracking and Analyses, United States. Serves as a regulatory toxicology subject matter expert for emerging chemicals such as PFAS and 1,4-dioxane. Is responsible for tracking the toxicological data and regulatory assessments and decisions internationally; providing summaries and impact assessments for clients; engaging with regulatory authorities to ensure sound scientific basis of regulatory decisions; and advising and developing risk management strategies to minimize effects of changing information and regulations, to ensure public and employee safety and health. Numerous clients.

Strategic Support Related to Management of Aqueous Film Forming Foam (AFFF) Use and Replacement. Provides confidential client with state-of-the-science updates and technical support related to regulatory and human health/environmental risks associated with AFFF use at oil and gas facilities.

Technical and Regulatory Support for the National Association for Surface Finishing. Provides technical consulting support, including toxicology, exposure, chemistry, training, and science communication, to, and on behalf of, the metal and surface finishing industry within the United States. Represents client in regulatory and legislative meetings and ensure that the human and environmental risks associated with metal plating processes is accurately understood and communicated to internal and external stakeholders.

Risk Communication and Regulatory Support Related to Contamination of Public Drinking Water, Confidential Municipality, U.S. Provides technical and strategic regulatory toxicology risk communication support to a U.S. drinking water municipality with unregulated and emerging chemicals present in source water. Ensures the municipality understands the human health risks and regulatory actions. Represents client in regulatory, and public meetings and ensure that toxicology and human health risk information is accurately communicated to and by stakeholders.

Risk Communication and Regulatory Support, 1,4-Dioxane. Confidential Publicly Owned Landfill, U.S. Provides regulatory toxicology, site-specific risk assessment reviews, and risk communication support to a publicly owned landfill with 1,4-dioxane in leachate. Ensures the human health risk assessments and regulatory actions are technically sound.

Emerging Issues and Contaminants Program Management, U.S. Air Force Civil Engineer Center, Lackland, Texas. Served as program manager of an emerging contaminants program with a $1.2 million annual budget. Oversaw support contractors, wrote documents, delivered presentations, led internal management briefings, and led department training sessions. Identified gaps in scientific knowledge that underlies USAF and DOD efforts to protect human and environmental health. Specific topics included

vapor intrusion, PFAS, 1,4-dioxane, chlorinated solvents (trichloroethylene, tetrachloroethylene) and pesticides. Also monitored evolving regulatory and political arenas to identify changes that could impact environmental cleanup costs, schedules, and procedures and policies. Developed programmatic recommendations for budget and resource needs to address environmental regulations and cleanup standards.

State-by-State Survey, United States. Participated in a survey of state and federal regulatory programs and initiatives to assess the level of activity and process by which emerging chemicals, such as PFASs, are prioritized and regulated. Analyses include understanding how state regulatory and public health agencies identify, prioritize, and develop strategies and standards to manage emerging chemicals. Work resulted in a compendium of all state-level initiatives related to emerging chemicals, which allows users to understand trends and state-specific interests.

Risk Communication, U.S. Air Force Civil Engineer Center, Lackland, Texas. Provided risk communication materials such as factsheets and informational seminars to the general public interested in understanding PFAS site-specific environmental risk and cleanup strategies affecting their community. Served as technical support to USAF public affairs officials working within a community directly impacted by PFAS-contaminated drinking water. Crafted risk communication tools and products, coordinated public meetings and agenda topics, and ensured that complex toxicology information was translated appropriately to the public.

Data Gap Analysis, U.S. Air Force Civil Engineer Center, Lackland, Texas. Identified gaps in scientific knowledge needed for the USAF and DOD to protect human and environmental health. Specific topics were vapor intrusion, chlorinated solvents, trichloroethylene, perfluorinated chemicals, 1,4-dioxane, and pesticides.

Toxicology

PFAS Product Stewardship, United States. Provides technical consulting support on short-chain PFAS and related fluorochemical products to the FluoroCouncil. Conducts scientific assessment and assist with stakeholder communications related to the health and environmental risk of short-chain PFAS and fluorotelomers.

Technical Peer Review of Federal and State Agency Guidance Documents, United States. Provides technical peer review of toxicology assessments, risk assessments, and guidance documents on behalf of clients, including peer reviews of EPA IRIS and Toxic Substances Control Act (TSCA) assessments, ATSDR toxicological profiles, California Office of Human Health and Environmental Assessment documents, and other state regulatory agency assessments. Numerous clients and chemicals.

Technical Review and Comment on the New York Department of Health Proposed Rulemaking for 1,4-Dioxane Maximum Contaminant Level (MCL) (I.D. NO. HLT-30-19-00006-P). Conducted a review of the current toxicological data related to 1,4-dioxane's carcinogenic human health risks and authored a comment letter to the New York State Department of Health on the technical validity of their proposed MCL.

Short-chain PFAS and Fluoropolymer Toxicology and Regulatory Support, United States. Provides toxicology support to a confidential client working to obtain regulatory approval for current PFAS-containing products.

Federal Toxicology and Risk Assessment Reviews, United States. Served as a member of the federal interagency review team providing consultation and expert review on nearly all toxicology assessments and/or guidance documents produced by EPA, NTP, and ATSDR. Assessed the technical validity, transparency of decisions, adherence to agency and other federal guidance, and overall technical competency of the risk assessments. Work included submitting detailed written comments and participating in interagency teleconferences and working meetings.

Technical Review and Comment on the New Jersey Drinking Water Quality Institute (DWQI) Maximum Contaminant Level Recommendation for 1,2,3-Trichloropropane (1,2,3-TCP), New Jersey. Conducted a critical review of the toxicology, epidemiology, toxicokinetic, and other studies relevant to 1,2,3-TCP

human health effects. Technical comments are part of the administrative record and continue to be considered by DWQI.

Technical Review of Pentachlorophenol Epidemiology Data in Response to proposed California Proposition 65 Listing, California. Provided critical review and analysis of the developmental and reproductive epidemiology data on pentachlorophenol in response to the California Developmental and Reproductive Toxicant Identification Committee review and proposed listing under Proposition 65.

Toxicology Evaluation of Remedial Action Objectives, California. Conducted an in-depth assessment of outdated remedial action objectives for a confidential contaminated site in California. Reevaluated the toxicology and quantitative risk assessment for a specific unregulated contaminant of concern at the site. Calculated new screening levels based on updated risk assessment methodologies to ensure that remediation actions remain protective of public health.

Human Health Chemical Hazard Identification and Dose-Response, Federal Agencies, Cincinnati, Ohio. Provided management and scientific expertise for chemical assessments performed under Superfund, IRIS, and other programs. Served on high-performance, interdisciplinary scientific teams for dioxin reassessment, computational toxicology, phthalate cumulative risk, and mode of action.

Human Health Risk Assessment (HHRA)

Baseline Human Health Risk Assessment for PFAS, Confidential Location. Serves as project manager and technical lead for a PFAS baseline human health and ecological risk assessment. Develops conceptual site models for assessing human and ecological receptor exposures. Manages selection of toxicity values and review of literature to identify primary mechanisms of action for toxicity relevant to site-specific human exposure pathways.

1,4-Dioxane Site-Specific Risk Assessment and Consulting Support Related to Public Drinking Water System Contamination, Confidential Location. Provides risk assessment and technical support related to the regulatory basis and public health impacts of 1,4-dioxane in a public drinking water system.

EPA Toxic Substances and Control Act, Low Volume Exemption Application, Confidential Client. Provided human health toxicology and exposure assessment to support a low volume exemption (LVE) application to EPA TSCA program. Conduct analysis, develop report, and assist with in-person presentation to EPA TSCA technical staff.

Environmental Risk Assessment Oversight, U.S. Air Force Civil Engineering Center, Lackland, Texas. Provided toxicology expertise and oversight of risk assessments conducted for the USAF Environmental Restoration Program's CERCLA and RCRA activities. Using EPA's risk assessment guidance, interpreted toxicology data to assess risks to human health and the environment, and reviewed site-specific risk assessments conducted at USAF installations nationwide.

PUBLICATIONS

(J.K. Anderson also published as J.K. Hess-Wilson)

Articles and Peer-Reviewed Publications

Goodrum, P.E., Anderson, J.K., Luz, A.L. and Ansell, G.K., 2020. Application of a Framework for Grouping and Mixtures Toxicity Assessment of PFAS: A Closer Examination of Dose Additivity Approaches. *Toxicological Sciences*.

Mohr, T.K., DiGuiseppi, W.H., Hatton, J.W. and Anderson, J.K., 2020. *Environmental investigation and remediation: 1, 4-dioxane and other solvent stabilizers*. CRC Press.

Iwai, H., A.M. Hoberman, P.E. Goodrum, E. Mendelsohn, and J.K. Anderson. 2019. Addendum to Iwai and Hoberman (2014) – Reassessment of developmental toxicity of PFHxA in mice. *Internat J Tox.* 38(3):183-191.

Anderson, J.K., A.L. Luz, and P. Goodrum. 2019. Response to "Overgeneralization by Anderson et al. and Luz et al. regarding safety of fluorotelomer-base chemistry". *Reg Tox Pharm*. 105:100-101.

Anderson, J.K., A.L. Luz, P. Goodrum, and J. Durda. 2019. Perfluorohexanoic acid toxicity, part II: application of human health toxicity value for risk characterization. *Reg Tox Pharm.* 103: 10-20.

Luz, A.L., J.K. Anderson, P. Goodrum, and J. Durda. 2019. Perfluorohexanoic acid toxicity, part I: development of a chronic human health toxicity value for use in risk assessment. *Reg Tox Pharm.* 103: 41-55.

Anderson, J., J. Wilhelm, and P. Goodrum. 2016. Emerging contaminants: An analysis of inconsistent U.S. regulations. *Daily Environment Report*. Bloomberg Bureau of National Affairs. August.

Anderson, R.H., G.C. Long, R.C. Porter, and J.K. Anderson. 2016. Occurrence of select perfluoroalkyl substances at U.S. Air Force aqueous film-forming foam release sites other than fire-training areas: field-validation of critical fate and transport properties. *Chemosphere.* 150:678–685.

Anderson, R.H., J.K. Anderson, and P.A. Bower. 2012. Co-occurrence of 1,4-dioxane with trichloroethylene in chlorinated solvent groundwater plumes at U.S. Air Force installations; fact or fiction. *Integr Environ Assess Manag.* 8(4):731–737.

Wang, N.C.Y., Q.J. Zhao, S.C. Wesselkamper, J.C. Lambert, D. Peterson, and J.K. Hess-Wilson. 2012. Application of computational toxicological tools and approaches in human health risk assessment I. A tiered surrogate approach. *Regul Toxicol Pharmacol*. 63:10–19.

Thomas, R.S., H.C. Clewell, B.C. Allen, S.C. Wesselkamper, N.Y. Wang, J.C. Lambert, J.K. Hess-Wilson, Q.J. Zhao, and M.E. Andersen. 2011. Application of transcriptional benchmark dose values in quantitative cancer and noncancer risk assessment. *Toxicol Sci*. 120(1):194–205.

Mazur, C.S., J.F. Kenneke, J.K. Hess-Wilson, and J.L. Lipscomb. 2010. Differences between human and rat intestinal and hepatic bisphenol A glucuronidation and the influence of alamethicin on *in vitro* kinetic measurements. *Drug Metab Dispos*. 38(12):2232–2238.

Hess-Wilson, J.K. 2009. Bisphenol A may reduce the efficacy of androgen deprivation therapy in prostate cancer. *Cancer Causes and Control.* 20(7):1029–1037.

Shah, S., J.K. Hess-Wilson, S. Webb, H. Daly, S. Godoy-Tundidor, J. Kim, J. Boldison, Y. Daaka, and K.E. Knudsen. 2008. 2,2-Bis(4-chlorophenyl)-1,1-dichloroethylene stimulates androgen independence in prostate cancer cells through combinatorial activation of mutant androgen receptor and mitogen-activated protein kinase pathways. *Mol Cancer Res.* 6(9):1507–1520.

Hess-Wilson, J.K., S.L. Webb, H.K. Daly, Y. K. Leung, J. Boldison, C.E.S. Comstock, M.A. Sartor, S.M. Ho, and K.E. Knudsen. 2007. Unique bisphenol A transcriptome in prostate cancer: novel effects on ERβ expression that correspond to AR mutation status. *Environ Health Perspect.* 115(11):1646–1653.

Sharma, A., E.S. Knudsen, J.K. Hess-Wilson, L.M. Morey, J. Barrera, and K.E. Knudsen. 2007. Retinoblastoma tumor suppressor status is a critical determinant of therapeutic response in prostate cancer cells. *Cancer Res*. 67(13):6192–6203.

Hess-Wilson, J.K., H.K. Daly, W.A. Zagorski, C.P. Montville, and K.E. Knudsen. 2006. Mitogenic action of the androgen receptor sensitizes prostate cancer cells to taxane-based cytotoxic insult. *Cancer Res*. 66(24):11998–12008.

Wetherill, Y.B.,* J.K. Hess-Wilson,* C.E.S. Comstock, S.A. Shah, C.R. Buncher, L. Sallans, P.A. Limbach, S. Schwemberger, G.F. Babcock, and K.E. Knudsen. 2006. Bisphenol A facilitates bypass of androgen ablation therapy in prostate cancer. *Mol Cancer Ther*. 5(12):3181–3190. *Co-first authors.

Hess-Wilson, J.K., J. Boldison, K.E. Weaver, and K.E. Knudsen. 2006. Xenoestrogen action in breast cancer: impact on ER-dependent transcription and mitogenesis. *Breast Cancer Res Treat.* 96(3):279–292.

Hess-Wilson, J.K., and K.E. Knudsen. 2006. Endocrine disrupting compounds and prostate cancer. *Cancer Lett*. 241(1):1–12—Invited review.

Selected Internal Department of Defense Documents

White Paper – Human health risks to perfluorinated compound exposure through drinking water and appropriate risk-based screening values. March 2015.

Emerging Issues/Contaminants Program – Preliminary evaluation and background report on 1-bromopropane. February 2014.

Interim AF guidance on sampling and response actions for 1,4-dioxane at operational and BRAC installations. August 2013.

Emerging Issues/Contaminants Program – Preliminary evaluation and background report on lead. June 2013.

Interim AF guidance on sampling and response actions for perfluorinated compounds at active and BRAC installations. September 2012.

Perchlorate – Background on the EPA MCLG proposal and industry challenges. July 2012.

Position Paper – Impact analysis and cost impact of AF environmental liability to perfluorinated compounds. April 2012.

Position Paper – TCE impact assessment. April 2012.

Bullet Background Paper – The potential impact of USEPA's dioxin non-cancer assessment on AF installations and PBR efforts. February 2012.

Emerging Issues/Contaminants Program – Background and preliminary assessment on hexavalent chromium. November 2011.

Bullet Background Paper – Health impact of the final EPA TCE toxicity values. October 2011.

Emerging Issues/Contaminants Program – Background and preliminary assessment on 1,4-dioxane. August 2011.

EPA Documents

USEPA. 2011. Volume I. EPA's re-analysis of key issues related to dioxin toxicity and response to NAS comments. Final review draft. EPA/600/R-10/038F. U.S. Environmental Protection Agency, Washington, DC. Contributing author.

USEPA. 2010. Recommended toxicity equivalence factors (TEFs) for human health risk assessments of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and dioxin-like compounds. EPA/100/R 10/005. U.S. Environmental Protection Agency, Risk Assessment Forum, Washington, DC. Coauthor.

SELECTED PRESENTATIONS and POSTERS

Anderson, J.K., and P. Goodrum. 2019. PFAS: Toxicology and Regulatory Actions. Webinar to the ACC Public Health Advisory Board. November 7, 2019

Luz, A., C. Hutchings, J. Anderson, P. Goodrum, J. Field. 2019. A Novel Approach for Assessing Hazard Associated with Firefighting Foams. Poster at the SETAC North American 40th Annual Meeting, Toronto Ontario, Canada. November 4.

Anderson, J.K. 2019. Federal and State Environmental Guidance/Policies that Impact Remedial Decisions for PFAS. Platform presentation at the Washington State Advanced Superfund Conference. September 12, Seattle, WA.

Anderson, J.K. 2019. PFAS: Risk Characterization Panel. Invited panelist to the Society of Environmental Toxicology and Chemistry North America, Focused Technical Meeting on PFAS. Durham, NC. August.

Anderson, J.K., A. Luz, and P. Goodrum. 2019. Chronic human health toxicity value for perfluorohexanoate (PFHxA) and risk assessment relevant to current fluorotelomer-based chemistries. Poster for the Society of Toxicology 58th Meeting and ToxExpo, March 10–14, Baltimore, MD.

Goodrum, P., J.K. Anderson, and A. Luz. 2019. Perfluoroalkyl acid mixtures—Data analysis steps to uncover clues hidden in biomonitoring data. Poster for the Society of Toxicology 58th Meeting and ToxExpo, March 10–14, Baltimore, MD.

Luz, A., J.K. Anderson, and P. Goodrum. 2019. Approaches for Assessing Perfluoroalkyl Acid Mixture Toxicity. Poster for the Society of Toxicology 58th Meeting and ToxExpo, March 10–14, Baltimore, MD.

Opdyke, D., J. Benaman, J.K. Anderson, and J. Durda. 2019. An introduction to PFAS at contaminated sediment sites: Scientific and regulatory overview. Short course at Tenth International Conference on the Remediation and Management of Contaminated Sediments, February 11–14, New Orleans, LA.

Wilhelm, J., J.K. Anderson, A. Luz, and P. Goodrum. 2018. PFAAs and ecorisk: Development of a hazard ranking system by evaluating functional groups vs. chain lengths as primary risk drivers for ecological receptors. Poster presentation. SETAC North American 39th Annual Meeting, November 4–7, Sacramento, CA.

Luz, A.L., L. Tolbert, J.K. Anderson, P. Goodrum, D. Farrar, and S. Korzeniowski. 2018. PFHxA human health risks, margin of safety, and comparison with PFOA. Platform presentation. Society of Environmental Toxicology and Chemistry North America 39th Annual Meeting. November 4–8. Sacramento, CA.

Anderson, J.K. 2018. Emerging contaminants—per-and polyfluoroalkyl substances: A case study. Invited speaker. Texas Environmental Superconference, August, Austin, TX.

Anderson, J.K., and P. Goodrum. 2018. Internal and external dosimetry—the holy grail to decoding perfluoroalkyl acid toxicity? Poster presented at the Emerging Contaminants Summit, March 6–7, Westminster, CO.

Anderson, J.K., and P. Goodrum. 2018. What does that blood level mean? The assumptions underlying interpretations of health effects from internal doses. Poster presented at the Society of Toxicology 57th Annual Meeting and ToxExpo, March 11–15, San Antonio, TX.

Goodrum, P., and J.K. Anderson. 2018. Application of internal dosimetry for perfluoroalkyl acids and methods to assess uncertainty factors used in risk assessment. Poster presented at the Society of Toxicology 57th Annual Meeting and ToxExpo, March 11–15, San Antonio, TX.

Anderson, J.K. 2017. Uncertainty in the science of toxicology and emerging contaminants. Remediation of Emerging Contaminants: Trends in Science and Regulations. Montclair State University Continuing Education Course. June.

Anderson, J.K. 2017. Why the inconsistent and dynamic state and federal chemical regulatory landscape. RTM Communications Conference, Philadelphia, PA. April.

Anderson, J.K. 2016. Inconsistent and dynamic state and federal chemical regulations: Roadmap to success. Consumer Specialty Product Association annual conference. December.

Anderson, J.K. 2016. How did we get here from there? State and Federal regulatory actions for PFAS. AEHS Annual East Coast Conference. October.

Frankel, A., P.E. Goodrum, J.K. Anderson, and K. Tsitonaki. 2016. Water quality standards for perfluoroalkyl compounds—Cross roads between regulatory toxicology and remedy selection. Platform presentation, Battelle 10th International Conference on Remediation of Chlorinated and Recalcitrant Compounds, Palm Springs, CA.

Anderson, J.K., N. Edlin, and S. Herman. 2016. Keeping a watchful eye on emerging contaminants. Environmental and Emerging Claim Managers Association annual conference. April.

Anderson, J.K. 2016. Emerging contaminants: analytical, toxicity, regulatory, and legal frontiers. Invited panelist to the Emerging Contaminants Summit. March.

Anderson, J.K., and P.E. Goodrum. 2016. Emerging contaminants: crossroads of uncertain science and risk management. Integral Webinar Series. February.

Anderson, J.K., and P.E. Goodrum. 2015. Status of regulatory decisions for perfluoroalkyl compounds: is the level of protection to the general public worth the uncertainty and cost? Poster presented at Society for Risk Analysis, Washington, DC.

Anderson, J.K. 2015. Overview of regulatory toxicology in the development of federal and state MCLs for perfluoroalkyl compounds. AEHS Annual East Coast Conference. October.

Anderson, J.K. 2014. AF approach to emerging issues & contaminants. Webinar presented to Society of Military Engineers. November.

Anderson, J.K. 2014. AF Emerging Issues & Contaminants Program: 1,4-dioxane and PFCs. Webinar presented to State Risk Assessors Teleconference. October.

Anderson, J.K. 2014. AF Emerging Issues & Contaminants Program: 1,4-dioxane and PFCs. Presented to Air Force Institute of Technology. October.

Anderson, J.K. 2014. Air Force Civil Engineering Center (AFCEC) Emerging Issues & Contaminants Program. Air Force Institute for Technology training sessions, Wright-Patterson Air Force Base, OH. August.

Philips, J.K., and J.K. Anderson. 2013. Challenges associated with practical environmental restoration risk assessment and management decisions for perfluoroalkyl substances (PFASs). Poster presented at Society for Risk Analysis Annual Meeting, Baltimore, MD. December.

Bodour, A., and J.K. Anderson. 2013. AFCEC Emerging Contaminants & Broad Agency Announcement Programs. Webinar presented to Federal Remediation Technology Roundtable, Arlington, VA. November.

Woodward, D., G. Hohenstein, J. Field, J. Phillips, D. Chiang and J.K. Anderson. 2012. Emerging contaminants: perfluorinated compounds (PFCs). Webinar presented to Society of American Military Engineers, Continuing Education. November.

Anderson, J.K. 2012. The AF Emerging Issues Program: the curious derivation of toxicity values for perfluorinated compounds. Presented to Tri-Service Toxicology Consortium, Dayton, OH. January.

Anderson, J.K., and A. Bodour. 2011. AFCEE research activities related to 1,4-dioxane—emerging issues program and broad agency announcement overview. Presented at Tucson International Airport Area Superfund Site Annual Information Exchange, Tucson, AZ. September.

Anderson, J.K. 2011. Air Force Emerging Issues/Emerging Contaminants Program. Presented at Restoration and Technology Transfer Workshop, San Antonio, TX. April.

Anderson, J.K. 2010. Cancer classification and mode of action for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). Presented at the 30th International Symposium on Halogenated Persistent Organic Pollutants, San Antonio, TX. September.

Anderson, J.K. 2010. EPA's provisional human health risk assessment process. Presented at Restoration and Technology Transfer Workshop, San Antonio, TX. April.

Anderson, J.K. 2009. TCDD cancer dose response background information and discussion. Session chair. TCDD and cancer dose response. Dioxin Workshop, Cincinnati, OH. February.

PDC Laboratories, Inc.
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(309) 692-9688 • (800) 752-6651 • FAX (309) 692-9689

June 24, 2021

Illinois Environmental Protection Agency
Submitted via email to EPA.620.rulemaking@illinois.gov

RE: PDC Laboratories, Inc.'s Comments on Proposed Updates to 35 Illinois Administrative Code 620: Groundwater Quality

Dear Sir or Madam:

We are pleased to submit our comments regarding the proposed changes to the language of 35 Ill. Adm. Code 620: Groundwater Quality. If you have any questions regarding our comments, please feel free to contact either Julia Rada at jrada@pdclab.com (309-683-1739) or Michael A. Travis at mtravis@pdclab.com (309-683-1744). We appreciate the opportunity to comment on the proposed changes and look forward to the next step in the rulemaking process.

Sincerely,

Julie Rada
Laboratory Director

Michael A. Travis
Corporate Director of Quality Assurance

Enclosure

PDC Laboratories, Inc.'s Comments on Draft Part 620

PDC Laboratories, Inc.'s comments on the proposed updates to 35 Illinois Administrative Code 620: Groundwater Quality are as follows:

1. (Section 620.110 Definitions)

"Detection" means the identification of a contaminant in a sample at a value equal to or greater than the:

"Lower Limit of Quantitation ~~Method Quantitation Limit~~" or "LLOQ~~MQL~~" means the minimum concentration ~~of a substance~~ that can be measured or ~~and~~ reported pursuant to "Test Methods for Evaluating Solid Wastes, Physical/Methods," incorporated by reference at Section 620.125.

Based on the reference in Section 620.125 (p.14)

"Test Methods for Evaluating Solid Waste, Physical/Chemical Methods." U.S. EPA Publication No. SW-846, Third Edition, Final Updates I (1993), II (1995), IIA (1994), IIB (1995), III (1997), IIIA (1999), IIIB (2005), IV (2008), V (2015), VI Phase I (2017), VI Phase 2 (2018), VI 3 (2019) and VII Phase I (2020).

http://www.epa.gov/hw-sw846/sw-846-compendium

~~as amended by Updates I, II, IIA, IIB, III, IIIA and IIIBV (Doc. No. 955-001– 00000-1) (available online at http://www.epa.gov/epaoswer/hazwaste/test/main.htm).~~

PDC Comments:

The definition of "Lower Limit of Quantitation or LLOQ" should be updated to read

The lowest point of quantitation, which in most cases is the lowest concentration in the calibration curve. The LLOQ is initially verified by spiking a clean control material (e.g., reagent water, method blanks, Ottawa sand, diatomaceous earth, etc.) at the LLOQ and process through all preparation and determinative steps of the method. Laboratory-specific recovery limits should be established when sufficient data points exist. Individual methods may recommend procedures for verifying the LLOQ and acceptance limits for use until the laboratory has sufficient data to determine acceptance limits. LLOQs should be determined at a frequency established by the method, laboratory's quality system, or project.

SW-846 Update V – Chapter One - page 20 - Revision 2 – July 2014

2. (Section 620.125 Incorporation by Reference)

a) The Board incorporates the following material by reference:

U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment

Shoemaker, J. and Dan Tettenhorst. Method 537.1: Determination of Selected Per- and Polyfluorinated Alkyl Substances in Drinking Water by Solid Phase Extraction and Liquid Chromatography/Tandem Mass Spectrometry (LC/MS/MS). U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment, Washington, DC. Version 1.0, November 2018.

PDC Comments:

Method 537.1 is a solid phase extraction (SPE) liquid chromatography/tandem-mass spectrometry (LC/MS/MS) method for the determination of selected per- and polyfluorinated alkyl substances (PFAS) in **drinking water**. The method was developed and validated for the analysis of finished drinking water from both groundwater and surface water sources. Test samples evaluated during method development included groundwater samples from challenging water matrices. The groundwater sample matrices had very high total dissolved (TDS)/hardness (up to 300 mg/L). The evaluation of the groundwater matrices generated acceptable method performance data that met stringent, method-defined quality control criteria. The method was deemed effective for analyzing PFAS in ambient groundwater samples that may be used as drinking water.

Reference: https://www.epa.gov/pfas/epa-pfas-drinking-water-laboratory-methods.

However, the general Illinois statewide ranges in chemical parameters from the bedrock aquifer (Pennsylvanian, shallow dolomites and limestones, and deep sandstones) are total dissolved solids (TDS), 350-3000 mg/L; hardness, 150-1000 mg/L; sulfates, 25-600 mg/L; nitrates, 0-5 mg/L; chlorides, 0-1000mg/L; and iron 0.3 – 5.0 mg/L.

In its present form, Method 537.1 would not be robust enough to deal with the much higher mineral content found in Illinois groundwater compared to the level used when the method was validated (TDS up to 300 mg/L versus a range of 350 to 3000 mg/L) during method development. Additional matrix interferences may be caused by contaminants that are co-extracted from the sample. The extent of matrix interferences will vary considerably from source to source, depending upon the nature of the water. Humic and/or fulvic material can be co-extracted during SPE and high levels can cause enhancement and/or suppression in the electrospray ionization source or low recoveries on the SPE sorbent.

The Detection limit (DL) of an analyte is defined as the statistically calculated minimum concentration that can be measured with 99% confidence that the reported value is greater than zero. The DL is **compound dependent** and is **dependent on extraction volume, extraction efficiency, sample matrix, fortification concentration, and instrument performance.**

Method 537.1 would be inappropriate for groundwater use at this point-in-time and should not be listed as a reference for groundwater use. The method was developed and validated for finished drinking water. The cumulative effects from each of the above listed limitations would raise the detection limits by at least a factor of five above the groundwater limits proposed by the Illinois EPA making the method unsuitable for this application.

> "Validated Test Method 8327: Per-and Polyfluoroalkyl Substances (PFAS) Using External Standard Calibration and Multiple Reaction Monitoring (MRM) Liquid Chromatography/Tandem Mass Spectrometry (LC/MS/MS)" Revision 0, June 2019.

PDC Comments:

The EPA has issued a draft method for PFAS analysis, EPA SW-846 Method 8327 designed to measure a group of 24 PFAS compounds in reagent water, groundwater, surface water, and wastewater effluent samples using liquid chromatography/tandem mass spectrometry (LC/MS/MS). This method has been validated and is available for use but has not yet been formally in incorporated into the SW-846 Compendium.

On June 21, 2019, the USEPA released SW-846 Update VII, Validation Phase II – Method 8327 for public comment. Key performance issues and shortcomings with the proposed method are listed below.

1. The target analyte list was evaluated for 24 compounds. Difficulties with reproducibility, response, recovery, stability and/or chromatography were noted for 11 of the tested analytes in the validation study. However, the Executive Summary states based on the Statistical Report and Data Validation Summary, states that the method is "generally acceptable". If an analytical method reveals problems or inconsistences to this extent, the method must not be used until it can provide the scientific confidence needed for use.
2. The suggested lower limits of quantitation (LLOQ) for PFOA and PFOS are 10 ng/L - above the limits for the proposed Class I and Class II GQS for PFOA (2 ng/L) and PFOS (7.7 ng/L).The method does not provide for LLOQs that are low enough to evaluate proposed compliance with these levels.
3. Sample preparation procedures call for the sample to be filtered after the addition of methanol. PFAS are surface active, and compound loss to the filter is likely even with the use of a 50% organic co-solvent. Due to low recoveries, filtering of samples should not be recommended as part of the method.
4. Section 2.1 of the method states that acetic acid is added because it improved sensitivity for some target analytes. The method does not state which compounds were enhanced by the addition of the acid or the level of signal enhancement.
5. The method uses external standard quantitation. The use of external standards does not allow the method to correct for variability coming from sample preparation or analytical conditions. The method should be re-evaluated using isotope dilution techniques to determine if precision, accuracy, and sensitivity would be improved.

6. EPA Method 537.1 calls for all branched isomers to be included in calibrations. Method 8327 states, “PFAS targets can be calibrated using a summation of the responses for all of the branched and linear peaks if present in quantitative standards **OR** by calibrating with only the linear isomer.” To reduce variability between methods and laboratories, Method 8327 should use the same procedures for quantitation of branched isomers as Method 537.1.
7. The method and study instructions specified preparation of analysis of one or more LLOQ verification samples with each batch of 20 or fewer samples. LLOQ verification samples were recommended to be prepared at concentrations of 10 and/or 20 ng/L in 5 mL water, but some of the test laboratories included LLOQ verification QC samples at 40 and/or 80 ng/L. The recovery criterion for LLOQ verification samples is 50 – 150% of the expected (prepared) concentrations.

   The frequency of target analytes meeting LLOQ verification acceptance criteria was higher at 20 ng/L than at 10 ng/L for all target analytes. At a concentration of 20 ng/L, only a few target analytes did not meet the LLOQ verification criteria at a frequency >90%. The LLOQ verification criterion of 20 ng/L is ten times the proposed PFOA limit of 2 ng/L, making this method unsuitable for this application.

   Method 8327 exhibits poor performance for selected compounds, may introduce low bias by applying sample filtration, uses external calibration, which has inferior precision, accuracy and sensitivity when compared to isotope dilution, lacks specificity in the quantitation of branched isomers and cannot achieve required low detection limits. Use of this method as it stands should be limited to <u>screening only</u> and NOT used for the collection of definitive data.

   The IEPA must not move forward with incorporating SW-846 Method 8327 into the 35 Illinois Administration Code 620: Groundwater Quality Standard by reference until there is additional testing and sufficient scientific confidence and precision to resolve the problems associated with sample preparation, contamination, type of calibration and instrument sensitivity.

eurofins | Environment Testing TestAmerica

June 24, 2021

Via e-mail: sara.terranova@illinois.gov
Ms. Sara G. Terranova
Assistant Counsel
Division of Legal Counsel
Illinois Environmental Protection Agency
1020 North Grand Avenue East
PO Box 19276
Springfield, IL 62794

RE: Comments Pertaining to Proposed Amendments to 35 Ill. Adm Code 620 – Groundwater Quality

Dear Ms. Terranova:

We appreciate the opportunity to submit our comments on proposed changes to 35 Ill. Adm. Code 620 Groundwater Quality. Eurofins TestAmerica is a network of environmental testing laboratories with 21 locations within the US, including one in Chicago IL. Several of our laboratories outside Illinois analyze samples from clients operating within Illinois.

We are pleased to see that you have added consideration of method quantitation limits (for example the LLOQ and LCMRL) in the setting of Class 1 and Class 2 groundwater limits. However, many of the limits chosen are from drinking water methods – these quantitation limits may not be achievable for more complex sample matrices. Please take into consideration that the LLOQ and LCMRL are single laboratory concepts, their values will vary among laboratories, and they do not take into consideration any sample matrix effects as they are routinely developed using reagent grade water.

Ideally, the achievable quantitation limit would be based on a multi lab study of the quantitation limits currently in use at laboratories reporting data to the State of Illinois, specifically for the methods (SW-846) that would be used for groundwater monitoring.

A good start would be to survey Illinois laboratories for their current quantitation limits. Even better would be to evaluate the levels of precision and accuracy that the laboratories are achieving at these limits.

The topic of quantitation is critical to the application of the Part 620 rules because measurements are used in statistical evaluations and for comparison to numeric standards; and, both of these activities presume that the measurement results are of known and controlled precision and bias.

A review of several state programs has revealed varying degrees to which agencies attempt to meet the requirements of 40 Code of Federal Regulation (CFR) Section (§)258.53(h)(5) that requires “any practical quantitation limit (PQL) that is used in the statistical method shall be the lowest concentration level that can be reliably achieved within specified limits of precision and accuracy during routine laboratory operating conditions that are available to the facility”. The state of Texas appears to have applied the most rigorous scientific approach to establishing acceptable limits of quantitation for its monitoring programs (https://www.tceq.texas.gov/assets/public/permitting/waste/msw/msw-pqls.pdf). TCEQ’s

objectives were to develop a mechanism to implement the rule in a way that was protective of human health and the environment, require quality standards for data that they receive, and establish benchmarks reflective of the capabilities of the commercial laboratories available to the regulated community. TCEQ requested laboratories, which routinely generate monitoring data submitted by regulated parties to the TCEQ, participate in an inter-laboratory study to collect data for target analytes at various concentrations using the commonly referenced methods from EPA SW846. After the data collection process was complete, TCEQ applied the Inter-Laboratory Quantitation Estimate (IQE) Standard (ASTM D6512) statistical process to the data to arrive at "benchmark" quantitation limits. During this process TCEQ established expected quality requirements for precision (in the form of %RSD) and accuracy (in the form of %recovery) for each class of analytes and introduced these quality requirements into the facility permits. This study demonstrated what levels of quantitation the commercial laboratories available to the regulated parties were able to achieve. The TCEQ subsequently published the results of the study and the "benchmark" quantitation limits that the regulated parties were expected to achieve.

Our review of the proposed changes revealed a number of constituents for which Eurofins is unable to the achieve proposed GQS standards using established LLOQs. Please see the attached table of proposed GQS standards that our laboratory does not currently routinely meet. Achieving these GQS limits with a quantitation limit would require additional methods to be developed and implemented. In some cases (vanadium in particular) the limits seem lower than warranted by health-based concerns and would create a serious risk of false positives. For the metabolites of atrazine [Desethyl-atrazine (DEA), Desisopropyl-atrazine (DIA), Diaminochlorotriazine (DACT)], the laboratory has not yet established an appropriate method of analysis for these new analytes; so, it is unclear if the proposed GQS values are achievable by a quantitation limit.

CAS	620 Constituent	620 Standards	620 Unit	Method	Eurofins Buffalo LLOQ	Eurofins Buffalo MDL	LLOQ Supports Standard
7439-93-2	Lithium	0.01	mg/L	6010B	0.03	0.01	No
7440-62-2	Vanadium	0.00027	mg/L	6020A	0.004	0.0012	No
319-84-6	alpha-BHC (alpha-benzene hexachloride)	0.000012	mg/L	8081B	0.00005	7.7E-06	No
96-12-8	1,2-Dibromo-3-chloropropane (dibromochloropropane	0.0002	mg/L	8260C	0.001	0.00039	No
123-91-1	1,4-Dioxane (p-dioxane)	0.00078	mg/L	8260C	0.04	0.00932	No
106-93-4	Ethylene dibromide (1,2-dibromoethane)	0.00005	mg/L	8260C	0.001	0.00073	No
99-65-0	1,3-Dinitrobenzene	0.001	mg/L	8270D	0.02	0.00082	No
121-14-2	2,4-Dinitrotoluene	0.001	mg/L	8270D	0.005	0.000447	No
15972-60-8	Alachlor	0.002	mg/L	8270D	0.01	0.000635	No
56-55-3	Benzo(a)anthracene	0.00025	mg/L	8270D	0.005	0.00036	No
50-32-8	Benzo(a)pyrene	0.0002	mg/L	8270D	0.005	0.00047	No
205-99-2	Benzo(b)fluoranthene	0.00025	mg/L	8270D	0.005	0.00034	No
207-08-9	Benzo(k)fluoranthene	0.0025	mg/L	8270D	0.005	0.00073	No
53-70-3	Dibenzo(a,h)anthracene	0.000025	mg/L	8270D	0.005	0.00042	No
88-85-7	Dinoseb	0.007	mg/L	8270D	0.01	0.002936	No
193-39-5	Indeno(1,2,3-c,d)pyrene	0.00025	mg/L	8270D	0.005	0.00047	No
87-86-5	Pentachlorophenol	0.001	mg/L	8270D	0.01	0.0022	No
122-34-9	Simazine	0.004	mg/L	8270D	0.01	0.0014	No
1912-24-9	Total Atrazine and Metabolites DEA (desethyl-atrazine) DIA (desisopropyl-atrazine) DACT (diaminochlorotriazine)	0.003	mg/L	8270D	0.005	0.00046	No
606-20-2	2,6-Dinitrotoluene	0.001	mg/L	8270D	0.005	0.0004	No
111-42-2	DEA (desethyl-atrazine)	0.003	mg/L	NA	DNS*	DNS*	DNS*
1007-28-9	DIA (desisopropyl-atrizine	0.003	mg/L	NA	DNS*	DNS*	DNS*
3397-62.4	DACT (diaminochlorotriazine	0.003	mg/L	NA	DNS*	DNS*	DNS*

DNS: Do not currently support

INTERNATIONAL MOLYBDENUM ASSOCIATION
THE VOICE OF THE MOLYBDENUM INDUSTRY

25 June 2021

For the kind attention of Illinois EPA

Re: Proposed amendments to 35 III. Admin. Code 620: Groundwater Quality

Substance: Molybdenum

The International Molybdenum Association (IMOA) has very recently become aware of the ongoing Illinois EPA (IEPA) ground water standards proposals for molybdenum, and wishes to participate in the stakeholder group, contributing data and dialogue, and likewise this submission during the public commenting period.

Beyond the minimal information indicated in the on-line Excel sheet tab 'Class 1 GQS' on the IEPA website, we have not been able to identify any scientific support documentation specific to molybdenum that clearly articulates the convincing scientific support for the proposed standard. If we are not mistaken, US EPA's IRIS is the primary data source. In this respect, we would like to share the following concerns with you for your consideration:

- US EPA's IRIS for molybdenum has not been updated for the last 29 years, since it was written in 1992, based on scant data available at that time.

- IMOA has commissioned many environmental and human health studies between 2007-2020, where the initial driver for those studies was compliance with the EU REACH Regulation that required detailed hazard and risk assessment of substances, based on robust data from studies conducted in accordance with internationally accepted protocols. These studies are available free-of-charge to regulatory authorities, and indeed those already available by 2014 are accredited to the OECD Mutual Acceptance of Data scheme. The 2014 OECD SIAP for highly soluble molybdenum salts is accessible via: https://hpvchemicals.oecd.org/UI/SIDS_Details.aspx?id=5c88d62f-4401-4cad-b521-521a4bd710f3 The OECD-generated profile (called the Screening Information Dataset [SIDS] Initial Assessment Profile [SIAP]) contains brief summaries of SIDS endpoints as well as the major conclusions of the hazard assessment. The USA was one of the OECD country reviewers prior to the accreditation being awarded, which amongst other things is an endorsement of the quality of the dataset, having passed peer-review by multiple OECD-member countries.

- The key study in US EPA's IRIS for the molybdenum reference dose is the Koval'skiy study (1961)[1], which for many years now is widely recognised by the regulatory community as unsuitable for regulatory purposes. And recently a summary of the significant shortcomings and uncertainties of that study are now publicly documented in the May 2020 US ATSDR

[1] Koval'skiy VV, Yarovaya GA, Shmavonyan DM. 1961. Changes of purine metabolism in man and animals under conditions of molybdenum biogeochemical provinces. Zh Obshch Biol 22(3):179-191.

International Molybdenum Association, 454-458 Chiswick High Road, Chiswick, London, W4 5TT, UK
Email: info@imoa.info Tel : + 44 20 8747 6120 W: www.imoa.info & www.molybdenumconsortium.org

Toxicological Profile for Molybdenum. Likewise the NAS Institute of Medicine 2001[2] publication concluded the Koval'skiy study is unreliable science, and this is also reflected by US ATSDR in its publication.

- For regulatory compliance purposes, between 2011-2017 three higher-tier human health studies using laboratory animals, each an OECD guideline-compliant GLP study, were commissioned using the highly soluble salt sodium molybdate, all conducted by USA-based laboratories: 90-day repeated dose toxicity, prenatal developmental toxicity, and 2-generation reproduction toxicity studies. The US ATSDR Tox Profile for Molybdenum critically assesses and takes account of each of those studies, and ultimately selected the 90-day repeated dose toxicity study as the key study and basis for its intermediate oral MRL derivation. The derived intermediate oral MRL screening value is 0.06 mg/kg-d. The ATSDR Toxicological Profile and the MRL underwent an Inter-Agency peer review that included representatives from the US EPA Office of Water. ATSDR also explicitly notes that screening values can be as much as 100-fold below levels shown to be non-toxic in laboratory animal studies[3], and consequently even screening level MRL's are not an appropriate basis for state groundwater quality standards.

- The US EPA IRIS database for metals in many cases has not been updated for decades. Whilst we completely understand that resourcing constraints mean that other higher priority substances receive attention and updating, it does also mean that the US EPA IRIS database cannot be the 'go to' database it once was, because enhanced global chemicals management legislation circa 2007 onwards has resulted in the availability of high-quality robust datasets that are not in the US EPA IRIS database, meaning that the underlying scientific rigor of outdated US EPA IRIS evaluations certainly warrants review. The North American Metals Council dialogued with the US EPA IRIS offices in 2018/2019 about this highly relevant disconnect. In 2020 IRIS introduced a second tab 'Other EPA Information' which links to the US EPA Chemistry Dashboard where newer data can be sourced. Another useful source is the publicly accessible EU REACH database.

In relation to the data shown on the Excel sheet and methodological information made available on-line by IEPA we note that:

- The HTTAC methodology bases the water standard on an assumed 15 kg body weight & drinking water consumption of 0.78L/day for a *0-6 year old child*. This is nearly twice as conservative as the usual approach of using the values for an adult. We are concerned as to the suitability of the adopted approach for standard-setting for a whole population, not least on the basis that molybdenum is a recognised bio-essential trace nutrient for humans, (animals & plants), and we are unable to discern whether the essentiality of molybdenum was factored in to your proposed value of just 0.019 mg Mo/L. We note this is the same value IEPA is proposing for silver (Ag), whereas the toxicity of the two substances differs significantly and Ag is not an essential trace element.

[2] NAS. 2001. Molybdenum. In: Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc. Washington, DC: National Academies Press, 420-439.
[3] *US ATSDR Toxicological Profile for Molybdenum, Appendix A, page A-1*

INTERNATIONAL MOLYBDENUM ASSOCIATION
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A further concern is whether the economic considerations relating to the proposed value of 0.019 mg Mo/L have been adequately addressed in terms of the ability for the impacted facilities to actually achieve such a highly challenging mandatory standard? That in turn feeds back into the concern to transparently demonstrate the compelling scientific support for the proposed standard.

The IEPA Excel file also has a 'Class II GQS' tab, proposing 0.05 mg Mo/L, which does not appear to provide further insights into how the value was derived and for which purpose (e.g. forage or non-forage). More detail would be appreciated for the sake of transparency and enhanced understanding.

In light of the above rationale and multiple concerns, IMOA will welcome further dialogue with Illinois EPA, particularly in relation to the proposed groundwater standard value of 0.019 mg Mo/L. We are available and keen to engage in discussion about these matters, and to provide the available molybdate datasets and information sources for your review and consideration with a view to appropriate revision of the current molybdenum proposals.

With kind regards.

Sandra Carey

Sandra Carey
HSE Executive

Response Email: sandracarey@imoa.info

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121 West Wacker Drive,
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June 25, 2021

Submitted via Email to: EPA.620.rulemaking@illinois.gov

Illinois Environmental Protection Agency

Re: National Waste and Recycling Association Comments on Proposed Amendments to 35 Ill. Adm. Code 620: Groundwater Quality

Dear Sir or Madam:

Attached please find the National Waste and Recycling Association's comments on the proposed amendments to 35 Ill. Adm. Code 620: Groundwater Quality.

If there are any questions regarding these comments, please feel free to contact me.

Sincerely,

James M. Morphew

James M. Morphew
JMM/dc

Enclosure

NWRA – IL Chapter 's Comments on 35 Ill. Adm. Code 620 Proposed Amendments

General Comment

The May 12, 2021 proposed changes/updates to 35 IAC Part 620 did not allow for sufficient input from the regulated community. The regulated community is well aware of the health concerns associated with per- and polyfluorinated alkyl substances (PFAS) and supports updating the regulated code to address the potential health risks associated with PFAS. However, there remains a lot of uncertainty regarding the concentrations at which PFAS would pose a health risk and the ability for laboratories to reliably detect PFAS at the concentration levels proposed in this amendment to the Part 620 rules. Many of the businesses in the regulated community have operations throughout the US and have an understanding of how other states and the USEPA are developing the appropriate regulations to limit the health impacts associated with PFAS in the environment. That knowledge base could have been helpful in providing input to ensure that the proposed updates to the Part 620 rules can be reliably implemented and effective in protecting the public health without being unduly burdensome on the regulated community. In addition, the proposed changes do not consider the impacts that it would have on other portions of the Illinois Administrative Code. For example, elimination of the practical quantitation limits (PQLs) would have an effect on 35 IAC Parts 724, 740 and 811 which contains references to the PQL. Therefore, we would propose that the Illinois EPA consider developing a working group that includes the regulated community to allow further refinements to the proposed Part 620 rules prior to filing them for a rulemaking.

Detection and Quantification

The proposed Part 620 rules includes many standards listed at levels that will be difficult for commercial laboratories to quantify. In addition, the only approved method for detection of PFAS is for drinking water and there is no currently approved method for groundwater which in Illinois often contains a high level of dissolved solids which will interfere in a laboratory's ability to detect PFAS using the referenced method 537.1.

Any proposed standard must consider a commercial laboratory's ability to quantify and report at these levels (i.e., the PQL). The purpose of the PQL is to adjudicate between a health-based level and a laboratory's ability to quantify at that level. Any proposed standard must consider the commercial laboratory's capability to quantify at that level to avoid falsely reporting a standards exceedance when it does not exist.

Fundamental to any regulatory establishment of numeric standards is the evaluation and adjustment of health-based 'goals', where needed, to create standards that analytical technology can reliably quantify. It is this 'adjudication' of the environmental 'goal' to what is practically achievable that provides the technical foundation for an agency to regulate, and for regulated parties to comply with regulation. Where a numeric standard is set without this adjudication, the establishment of a standard is arbitrary and capricious.

A clear definition of the PQL is needed in Part 620. The Statistical Analysis of Groundwater Monitoring Data at RCRA Facilities (Unified Guidance - 2009) which is referenced in Part 620 states on p. 2-7 that "Any practical quantification limit (PQL) approved by the Regional Administrator under §264.97(h) [or §258.53(g)] that is used in the statistical method shall be the lowest concentration level that can be reliably achieved within specified limits of precision and accuracy during routine laboratory operating

conditions available to the facility." It is recommended that the Unified Guidance definition be incorporated into Part 620.

Addition of Five PFAS Compounds (PFOA, PFOS, PFBS, PFHxS, and PFNA)

The Agency should address and resolve key scientific uncertainties before developing groundwater standards for PFAS compounds. Existing literature demonstrates significant scientific uncertainty regarding the level at which PFAS pose a health risk. Developing standards prior to the establishment of an established and widely accepted risk level or approved analytical method will lead to flawed rulemaking and will impose scientifically unsound, unfair, and oppressive legal, economic, and operational burdens on the regulated community.

The accelerated pace to establish PFAS standards does not allow the time needed to adequately assess the potential toxicity of a given compound, let alone to develop MCLs that consider economic and technological factors. By comparison, and focusing solely on the toxicity component alone, USEPA has been assessing the potential toxicity of dioxin and furans – a group of merely 210 compounds, a much smaller group than the 4,000 unique PFAS compounds – since 1985. USEPA's assessment of dioxin-like compounds has been reviewed by USEPA Science Advisory Boards on four separate occasions, has been reviewed by the National Academy of Sciences, and has undergone multiple rounds of public comment. It took USEPA more than 20 years to reach consensus on the noncancer effects of dioxin and furans, and, even after such technical scrutiny, USEPA still has not reached a consensus on the cancer potency of dioxin. This is for a group of chemicals for which the mode of toxic action and the relative potency among congeners is well known. None of these conditions hold for PFAS, and yet the Agency wants to establish standards for five PFAS compounds when significant data gaps exist. The current science does not support standards establishment at this time and standards should not be developed until much greater scientific certainty and technical understanding is gained.

Although many independent studies have been performed, results are inconsistent and scientific consensus is lacking on what the data mean regarding human health risks and PFAS toxicity. It is imperative to keep the state of the science in the forefront to ensure technically defensible standards are developed and are appropriate for the long term. The Agency should consider interim, conditional, or similar alternatives to prematurely establishing formal standards given the inadequate technical information and scientific consensus regarding PFAS health risks. A reasonable approach for the Agency is to consider reevaluation of PFAS health effects on an annual basis following review of new studies and other scientific developments.

Basis for Standards Development

Overall, more information is needed to document the technical basis for each of the proposed standards. The procedures described in the regulations are not sufficient to understand the basis of the proposed standards. The toxicity value and any other values that the Illinois EPA used to calculate the standards are still being revised and subject to further change and review. As an example "The research conducted to date reveals possible (emphasis added) links between human exposures to PFAS and adverse health outcomes.", and "While knowledge about the potential health effects of PFAS has grown, many questions remain unanswered" (https://www.niehs.nih.gov/health/topics/agents/pfc/index.cfm, accessed 6/14/2021)

Unified Guidance

Reference to the Unified Guidance is a good addition to Part 620 as long as it allows the regulated entity to apply any of the statistical methods that are applicable within the guidance document. The application of the Unified Guidance procedures should be synchronized with other Illinois regulatory programs such as Part 811 for solid waste landfills and Part 724 for RCRA facilities.

BY ELECTRONIC MAIL

June 25, 2021

Illinois Environmental Protection Agency
Bureau of Water
1021 North Grand Avenue
Springfield, IL 62794

Re: Proposed amendments to 35 Ill. Adm. Code 620; groundwater quality

To Whom It May Concern:

The American Chemistry Council provides the enclosed comments on the proposal to establish groundwater quality standards for five perfluoroalkyl substances, to lower the standard for 1.4-dioxane, and to revise the human non-threshold toxicity advisory concentration (HNTAC) for substances suspected of increasing cancer risk through a mutagenic mode of action. As detailed in the enclosure --

- The US Environmental Protection Agency (USEPA) has established lifetime health advisories for perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS) and recently announced that it will develop national drinking water standards for these two substances. The USEPA's health advisories are the appropriate basis for the state groundwater standards until national standards are developed.
- The minimal risk levels developed by the Agency for Toxic Substances and Disease Registry (ATSDR) for perfluorohexane sulfonic acid (PFHxS) and perfluorononanoic acid (PFNA) are intended as "screening levels" and are not an appropriate basis for state groundwater quality standards.
- The available scientific evidence provides strong support for a threshold mode of action for the occurrence of tumors in laboratory animals exposed to 1,4-dioxane. That is the conclusion of authoritative bodies around the world, including Health Canada and the World Health Organization who recommend a drinking water level of 0.050 mg/L.
- The oral slope factor (SFo) used in calculating the human non-threshold advisory concentration (HNTAC) is based on a default linear, low-dose extrapolation assuming a mutagenic mode of action which includes a significant level of conservatism. There is no reason to include age-adjusted water intake factors to account for increased cancer risk from childhood exposure for substances suspected of being mutagenic carcinogens unless information exists for the specific substance to indicate early life sensitivity.
- The Agency has not provided evidence that many of the substances listed in Appendix E act by the same mode of action as specified in the regulation. The additivity of potential health effects of these substances should not be considered unless a common mode of action can be established using standard assessment frameworks.

ACC urges IEPA to revise its proposal to address the issues described in the enclosed comments. Please feel free to contact me at srisotto@americanchemistry.com or at (202) 249-6727 if you have questions about the comments or wish to discuss them further.

Sincerely,

Steve Risotto

Stephen P. Risotto
Senior Director

Enclosures

Comments of the American Chemistry Council
on Draft Proposed Amendments to
35 Ill. Adm. Code 620 – Groundwater Quality

Introduction

Illinois EPA is proposing significant changes to its groundwater quality regulation that would establish new standards for several perfluoroalkyl substances (PFAS), revise the existing standard for 1,4-dioxane, apply an age-dependent adjustment factor in calculating advisory concentrations for substances considered to be mutagenic carcinogens, and identify a significant of substances as similar acting for purposes of assessing the toxicity of mixtures of mixtures of substances. As described below, the standards for four PFAS and 1,4-dioxane are not based on the best available science. In addition the updated approach to mutagenic carcinogens fails to consider of the data that may be available for a particular substance. Moreover, the proposal for defining similar acting substances does not provide evidence to establish a common mode of action as required.

Perfluorooctanoic Acid (PFOA)

The proposed groundwater quality standard for perfluorooctanoic acid (PFOA) is based on an assessment by California's Office of Environmental Health Hazard Assessment (OEHHA)[1] of the results of a chronic bioassay conducted by the National Toxicology Program (NTP).[2] While NTP reported increased incidence of hepatocellular and pancreatic tumors in male rats exposed to PFOA in their diet, reports of unanticipated toxicity in the study and elevated preneoplastic lesions in the control group raise concerns about the findings.

As IEPA is no doubt aware, the US Environmental Protection Agency (USEPA) has developed a lifetime health advisory (LHA) of 70 parts per trillion (ppt) for PFOA based on a thorough review of the available scientific information for the substance and the application of standard scientific methods.[3] In March of this year,[4] USEPA announced its intent to develop a national drinking water standard for PFOA that will consider information published since the LHA was established, including the NTP bioassay results. Pending the outcome of the USEPA's review, the LHA can serve as a health protective basis for the groundwater quality standard.

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1 OEHHA. Notification Level recommendations – perfluorooctanoic acid and perfluorooctane sulfonate in drinking water. California Environmental Protection Agency (August 2019).

2 NTP. Technical report on the toxicology and carcinogenesis studies of perfluorooctanoic acid administered in feed to Sprague-Dawley rats. Technical Report 598. Department of Health and Human Services. Research Triangle Park, North Carolina (2019).

3 USEPA. Drinking water health advisory for perfluorooctanoic acid (PFOA). EPA 822-R-16-005. Office of Water (May 2016).

4 86 *Federal Register* 12272 (March 3, 2021). https://www.govinfo.gov/content/pkg/FR-2021-03-03/pdf/2021-04184.pdf

The association with liver tumors reported by NTP is not supported by the available epidemiological evidence from occupational and general population studies. Human evidence for other tumor types, including pancreatic tumors, is conflicting and a recent comprehensive evaluation of the epidemiology suggests that reported associations are likely the result of chance, confounding, and/or bias. Laboratory studies in rats exposed to PFOA have reported a "tumor triad" – liver, testis, and pancreatic tumors – consistent with evidence for other substances known to activate the peroxisome proliferator-activated receptor α (PPAR α) in rodents with uncertain relevance to human health risk assessment.

Results of the NTP Bioassay

In the NTP study that is the basis for the proposed groundwater quality standard, NTP reported an increased incidence of liver adenomas and pancreatic acinar cell (PAC) adenomas in male Sprague-Dawley rats exposed to PFOA in the diet. In the study, male rats were exposed postweaning to 0, 20, 40, and 80 parts per million (ppm), equivalent to 0, 1.0, 2.2, and 4.6 milligrams per kilogram, or mg/kg, per day, while females were exposed to 0, 300, and 1000 ppm (0, 18.2, and 63.4 mg/kg per day).[5] The male rat portion of the study was repeated using significantly lower exposures after "unanticipated toxicity" was observed in male rats exposed to 150 and 300 ppm after 16 weeks. In light of the fact that male SD rats tolerated doses as high as 300 ppm in a previous chronic studies (described below), the reports of unanticipated toxicity at comparable levels in the male rats in the NTP study raise concern about the overall confidence in the study.[6]

In the NTP study statistically significant increases in hepatocellular adenomas were reported among the male rats exposed to the two highest doses (2.2 and 4.6 mg/kg per day). Hepatocellular carcinomas were increased at the highest dose (4.6 mg/kg per day), but the increase was not statistically significant. The study also reported significant increases in hepatocyte cytoplasmic alteration and hypertrophy in the males in all exposure groups. Significant increases were also observed in single cell hepatocyte death, necrosis, mixed cell foci, inflammation, cystic degeneration, and bile duct hyperplasia.

An increase in PAC adenomas was statistically significant in male rats in all exposure groups, but not in the female groups.[7] PAC adenocarcinomas were also increased in the males, but the increase was not statistically significant. The study also noted a significant increase in PAC hyperplasia - a potentially preneoplastic lesion - in all the male groups, including the control group in which hyperplasia was reported in 36 percent of the animals. The high background

[5] The study included groups of animals exposed to PFOA perinatally and postweaning to assess the potential impact of gestational and lactational exposure but reported very few significant differences between the response in animals exposed postweaning only to those with both perinatal and postweaning exposure.

[6] In addition, survival rates among the female animals were quite low – ranging from 46 percent in the control group to between 46 and 64 percent in the exposure groups.

[7] A non-significant increase of combined PAC adenomas and carcinomas was observed in females at the highest dose. Unlike in the males, acinus hyperplasia was not reported in the females.

rate for preneoplastic lesions observed in this study is consistent with the historical sensitivity of the Sprague-Dawley rats compared to other rat stains – and more significantly when compared to humans.

Epidemiology

Occupational studies examining cancer mortality have been conducted among workers occupationally exposed to PFOA in Minnesota and West Virginia focusing on kidney, bladder, liver, pancreatic, testicular, prostate, thyroid, and breast cancers. Two studies of communities exposed to PFOA in drinking water also are available. The results from these studies are conflicting and interpretation is limited by the small number of observed deaths and incident cases.

Raleigh et al. (2014) updated a study of cancer mortality among 4,668 PFOA workers in Minnesota followed through 2008.[8] Exposure estimates for inhalation exposures were calculated from work history records and industrial hygiene monitoring data; notably serum levels were not reported. The analysis reported no association between PFOA exposure and mortality from any cancer type. A slight elevation of bladder and pancreatic cancer incidence was reported although the confidence intervals were quite large; no association with kidney cancer incidence and PFOA exposure was reported.[9] The mean age of the workers was 29 years at the start of employment and 63 years at the end of follow-up.

Steenland and Woskie (2012) updated a cohort mortality study of 5,791 workers in West Virginia who had worked in a manufacturing facility using PFOA for at least 1 year between 1948 and 2002.[10] Mean duration of employment was 19 years. Exposure quartiles were assessed by estimated cumulative annual serum levels based on blood samples taken from 1,308 workers and time spent in various job categories. Referent groups included both nonexposed workers in the same region and the U.S. population. Overall, the mean cumulative exposure among the workers was 7.8 ppm-years and the estimated average annual serum level was 0.35 milligrams per liter (mg/L).[11] The authors reported a significant positive trend for kidney cancer incidence among workers in the highest exposure quartile, while no association was reported between PFOA exposure and liver, pancreatic, testicular, or bladder cancer incidence.

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8 Raleigh KK *et al.* Mortality and cancer incidence in ammonium perfluorooctanoate production workers. *Occup Environ Med* 71(7):500-506 (2014). http://dx.doi.org/10.1136/oemed-2014-102109

9 The authors report that the study had limited power to evaluate exposure response for testicular, bladder, liver, and pancreatic cancers.

10 Steenland K and Woskie S. Cohort mortality study of workers exposed to perfluorooctanoic acid. *Am J Epidemiol* 176(10):909–917 (2012). https://doi.org/10.1093/aje/kws171

11 For comparison, the mean serum level of PFOA in the 2016 biomonitoring survey conducted by the Center for Disease Control and Prevention was 0.0016 mg/L. https://www.cdc.gov/exposurereport/index.html

Liver cancer mortality was elevated in a small observational study of 642 male employees who had worked at least 6 months before 2009 for a factory producing PFOA and other chemicals.[12] Confounding factors were not well controlled. Serum levels in 120 workers were used to predict PFOA concentrations of each individual; serum concentrations ranged from 19 to 91,900 nanograms per milliliter (ng/mL). A statistically significant increase for mortality of liver cancer and liver cirrhosis was reported in the highest cumulative internal dose group when compared to the regional populations and workers of a nearby factory

Two studies involving communities in West Virginia and Ohio affected by contaminated drinking water (the C8 Health Project) reported a positive association between blood levels of PFOA and kidney and testicular cancers. Vieira *et al.* (2013) investigated incidences of 18 cancer types among residents supplied by six public water districts in Ohio and West Virginia contaminated with PFOA.[13] The analysis included over 25,000 cancer cases. Exposure levels and serum PFOA concentrations were estimated based on residence at time of diagnosis. Exposures were categorized as very high, high, medium, low, or unexposed based on PFOA serum concentrations.

Among all cancer endpoints, the odds ratio for testicular cancer was elevated in one of the two areas with the highest concentration of PFOA in drinking water. There was no statistically significant increase in the odds ratio for testicular cancer in the total exposed population, however, or in the other districts, or in the other estimated dose-level categories. Kidney cancer incidence was increased significantly in one district with the two highest levels of individual exposure. Despite the large overall sample size, the authors noted that their analysis was limited by small numbers of individual cancers in the high-exposure groups. Moreover, there was little consistency across exposure categories, with no evidence of a dose response.

Barry *et al.* (2012) conducted an analysis of cancer incidence among 32,254 individuals in the same geographic area as Vieira *et al.*, including 3,713 workers with occupational exposure to PFOA.[14] Cumulative PFOA serum concentrations were estimated based on historical regional monitoring data and individual residential histories. Based on measurements taken in 2005-2006, mean serum concentrations were 0.024 mg/L for community residents and 0.113 mg/L for workers. A total of 2,500 cancers were validated through a cancer registry or medical records. The authors reported that PFOA exposure was positively associated with kidney and testicular cancer across the exposure quartiles within the population, although the incidence of either tumor type was not elevated when compared to the US population.

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[12] Girardi P and Merler E. A mortality study on male subjects exposed to polyfluoroalkyl acids with high internal dose of perfluorooctanoic acid. Env Research 179(Part A):108743 (2019). https://doi.org/10.1016/j.envres.2019.108743

[13] Vieira VM *et al.* Perfluorooctanoic acid exposure and cancer outcomes in a contaminated community: a geographic analysis. *Environ Health Persp* 121(3):318-323 (2013). https://doi.org/10.1289/ehp.1205829

[14] Barry V *et al.* Perfluorooctanoic acid (PFOA) exposures and incident cancers among adults living near a chemical plant. *Environ Health Persp* 121(11-12): 1313-1318 (2013). https://doi.org/10.1289/ehp.1306615

Two additional population studies did not report an association of liver or pancreatic cancer and PFOA exposure. A study of 57,000 individuals with no previous cancer diagnosis enrolled in a prospective cohort during 1993-97 reported no association between liver and pancreatic cancer and elevated levels of PFOA; kidney and testicular cancer information was not presented.[15] PFOA concentrations were based on a single measure of plasma level taken at recruitment. A study of residents exposed to contaminated drinking water near a PFAS manufacturing facility in the Veneto Region of Italy with exposure to multiple PFAS, reported no increase in mortality caused by kidney, pancreatic, liver, or testicular cancer. [16] Some excess kidney cancer mortality was reported among women.

A review of the epidemiological evidence for cancer from 18 studies of occupational and general population exposure to PFOA reported a lack of concordance between community exposures and occupational exposures one or two magnitudes higher than those for the general population.[17] The authors evaluated the studies based on the study design, subjects, exposure assessment, outcome assessment, control for confounding, and sources of bias using Bradford Hill guidelines and concluded that the discrepant findings across the study populations were likely due to chance, confounding, and/or bias. A more recent review of the evidence by the epidemiologists involved in the C8 study concluded that the evidence for an association between PFOA exposure and kidney and testicular cancer was suggestive overall, there was little evidence for an association with liver or pancreatic cancer.[18]

The relevance of the liver tumor data from the NTP study is further called into question by recent clinical data reported by Convertino *et al.* (2018).[19] In a study of a sensitive subpopulation of cancer patients with normal liver function exposed to weekly PFOA doses as high as 1,200 mgs (about 16 mg/kg per day), Convertino *et al.* reported no differences in clinical

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15 Eriksen KT *et al.* Perfluorooctanoate and perfluorooctanesulfonate plasma levels and risk of cancer in the general Danish population. *J Natl Cancer Inst* 101:605–609 (2009). https://doi.org/10.1093/jnci/djp041

16 Mastrantonio M *et al.* Drinking water contamination from perfluoroalkyl substances (PFAS): an ecological mortality study in the Veneto Region. Italy. *Eur J Public Health* 28(1):180–185 (2018). https://doi.org/10.1093/eurpub/ckx066

17 Chang ET *et al.* A critical review of perfluorooctanoate and prefluorooctanesulfonate exposure and cancer risk in humans. *Crit Rev in Toxicol* 44(51):1–81 (2014). https://doi.org/10.3109/10408444.2014.905767

18 Steenland K *et al.* Review: evolution of evidence on PFOA and heath following the assessments of the C8 Science Panel. *Environ Intl* 145: 106125 (2020). https://doi.org/10.1016/j.envint.2020.106125

19 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

hepatic measures.[20] Similarly a study of PFOA production workers reported no abnormal liver function, hypolipidemia, or cholestasis.[21]

Animal Bioassays

In addition to the NTP study, two chronic bioassays have been conducted in rats exposed to PFOA through diet. Although the results are not consistent, one or both studies have reported liver, LC, or PAC tumors.[22]

Butenhoff *et al.* (2012), reporting on a previously conducted study of male and female Sprague-Dawley (SD) rats exposed to dietary levels of 30 and 300 ppm of PFOA (approximately 1.5 and 15 mg/kg per day), observed a dose-dependent increase in LC adenomas that was statistically significant at the highest dose.[23] Elevated incidence of hepatic and PAC lesions were also reported in males at 300 ppm, but the authors did not report increases in hepatic or PAC tumors despite exposures that were three times higher than those used in the NTP study.

A subsequent single-dose, dietary study with male CD rats reported LC adenomas, as well as liver and PAC adenomas and combined pancreatic adenomas and carcinomas at 300 ppm (13.6 mg/kg per day).[24] Increased incidences of LC and PAC hyperplasia were also observed. Hepatic ß-oxidation activity was significantly elevated, but cell proliferation in the liver was not.

Relevance of the Animal Data

A significant amount of genotoxicity and mechanistic data are available to assist in evaluating the results of the epidemiology and animal bioassay results described above. Multiple *in vivo* and *in vitro* assays provide clear evidence that PFOA is not mutagenic and may only cause genotoxicity at toxic concentrations. Consequently, it is generally agreed that PFOA causes tumors in laboratory animals via a non-genotoxic or epigenetic mechanism.[25]

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[20] Clinical measurements included triglycerides, urea, glucose, AST, GGT, alkaline phosphatase, total bilirubin, fibrinogen, PTT and aPTT.

[21] Olsen GW *et al.* Plasma cholecystokinin and hepatic enzymes, cholesterol and lipoproteins in ammonium perfluorooctanoate production workers. *Drug Chem Toxicol* 23(4):603–20 (2000). https://doi.org/10.1081/DCT-100101973

[22] The incidence of testicular (Leydig cell, or LC) adenomas was not reported in the NTP bioassay.

[23] Butenhoff JL *et al.* Chronic dietary toxicity and carcinogenicity study with ammonium perfluorooctanoate in Sprague-Dawley rats. *Toxicol* 298(1–3): 1–13 (2012). Target doses for the study were 0, 1.3, and 14.2 mg/kg body weight per day in males and 0, 1.6, and 16.1 mg/kg per day in females. https://doi.org/10.1016/j.tox.2012.04.001

[24] Biegel LB *et al.* Mechanisms of extrahepatic tumor induction by peroxisome proliferators in male CD rats. *Toxicol Sci* 60(1): 44–45 (2001). https://doi.org/10.1093/toxsci/60.1.44

[25] US Environmental Protection Agency (USEPA). Health Effects Support Document for Perfluorooctanoic Acid (PFOA). EPA 822-R-16-003. Office of Water. Washington, DC. (May 2016).

The tumor types that have been reported consistently in rats exposed to PFOA – liver, LC, and PAC – have been observed with other substances that are PPARα agonists. Because of key toxicodynamic and biological differences in responses between rodents and humans, PPARα activators are considered unlikely to induce tumors in humans. For liver tumors, this conclusion is based on minimal or no effects observed on growth pathways, hepatocellular proliferation and liver tumors in humans and/or species (*e.g.*, hamsters, guinea pigs and *Cynomolgous* monkeys) where PPARα expression is more similar to humans.

Several key studies provide support for the key events in the proposed PPARα-activated mode of action (MOA) for rat liver tumors (Table 1) and confirm that the MOA has little relevance to humans. These data are summarized by Klaunig *et al.* (2012) –

> Analysis of gene expression changes elicited following short-term administration of PFOA demonstrated the up regulation of genes characteristic of PPARα activation, including genes involved in fatty acid homeostasis/peroxisomal proliferation as well as those related to cell cycle. In addition, PFOA has been shown to induce peroxisome proliferation in mouse and rat liver and causes hepatomegaly in mice and rats. While the liver growth caused by PFOA was predominantly attributed to a hypertrophic response, an increase in DNA synthesis following PFOA exposure was observed and predominated in the periportal regions of the liver lobule. Thus, the effect of PFOA on induction of cell cycle gene expression and the increase in DNA synthesis provide evidence in support of both key events 2 and 3 in the proposed MOA for liver tumor induction by PFOA. Empirical evidence also exists in support of the clonal expansion of preneoplastic hepatic lesions by PPARα activators (Step 4). Using an initiation-promotion protocol for induction of liver tumors in Wistar rats, PFOA was shown to increase the incidence of hepatocellular carcinomas in rat liver (33% in PFOA exposed rats vs. 0% in controls).[26]

Klaunig *et al.* also note that the key events in Table 1 appear in a temporal sequence and demonstrate dose-related effects further strengthening the evidence for the PPARα-agonist MOA. Although there are indications that PFOA may also act through PPARα-independent mechanisms[27] in rodents, differences in binding affinity between the rodent and human receptors suggest that it is also unlikely that PFOA induces cancers in humans through the other mechanisms that have been suggested.[28] In evaluating their results, Convertino *et al.*

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[26] Klaunig JE *et al.* Mode of action analysis of perfluorooctanoic acid (PFOA) tumorigenicity and human relevance. *Reprod Toxicol* 33:410-418 (2012). https://doi.org/10.1016/j.reprotox.2011.10.014

[27] Activation of the constitutive activated receptor (CAR) and pregnane X receptor (PXR) by PFOA have been suggested in animal studies.

[28] Hall AP *et al.* Liver Hypertrophy: A Review of Adaptive (Adverse and Non-Adverse) Changes-Conclusions from the 3rd International ESTP Expert Workshop. *Toxicol Pathol* 40:971-994 (2012). https://doi.org/10.1177%2F0192623312448935

concluded that the disparity between animal and human liver endpoint studies, emphasizing a lack of risk of hepatomegaly, fatty liver, or cirrhosis, are likely due to MOA differences. Increased liver weight due to hepatocellular hypertrophy can often be an adaptive (protective) response in animals due to up-regulation of detoxification enzymes, leading toxicologists to revisit the relevance key liver endpoint studies in animals.[29]

Table 1. PPARα Mode of Action for PFOA-Induced Liver Tumors in Rats (from Klaunig *et al.* 2012)

	Key Event	Support	Key Reference[30]
1	Activation of the PPARα receptor	✓	Maloney & Waxman 1999; Vanden Heuvel *et al.* 2006
2	Induction of cell growth gene expression in the liver	✓	Martin *et al.* 2007; Kennedy *et al.* 2004
3	Cell proliferation	✓	Biegel *et al.* 2001; Martin *et al.* 2007; Thottassery *et al.* 1992
4	Selective clonal expansion of preneoplastic hepatic foci	✓	Abdellatif *et al.* 1990
5	Liver neoplasms	✓	Biegel *et al.* 2001

For the induction of rat PAC tumors by PFOA, the available mechanistic data are less robust, but also point to the importance of PPARα activation in the liver. Several factors may contribute to the development of PAC hypertrophy, hyperplasia, and adenomas in the rat, such as testosterone and estradiol levels, growth factor expression (cholecystokinin, or CCK), growth factor receptor overexpression (CCKA receptor),and high fat diet (Klaunig *et al.*).[31] Studies with the compound Wyeth 14,643, a well-studied and potent peroxisome proliferator in rodents, suggest that peroxisome proliferation induces PAC tumors by an indirect mechanism. In this study PPARα activation in the liver caused by exposure to Wyeth triggered reduced bile flow and/or changes in bile composition that produced an increase in CCK levels secondary to hepatic cholestasis.[32] As CCK has been shown to act as a growth factor for PACs in rats, a sustained increase in CCK levels would explain the increase in PAC proliferation observed following PFOA exposure and is likely therefore a preneoplastic lesion.

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[29] 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). https://doi.org/10.1016/j.tox.2011.06.012

[30] Complete citations are provided in Klaunig *et al.* 2012.

[31] Differences in the diets used in the Butenhoff *et al.* and Biegel *et al.* studies have been suggested as the likely reason for the quantitative difference in the PAC lesions observed in the two studies (USEPA 2016).

[32] Obourn JD *et al.* Mechanisms for the pancreatic oncogenic effects of the peroxisome proliferatorWyeth-14,643. *Toxicol Appl Pharmacol* 145:425–36 (1997). https://doi.org/10.1006/taap.1997.8210

As with PPARα, expression of CCK receptors in humans is much lower as compared to rodents, and the available non-human primate and human data suggest that the CCK pathway is not relevant to human cancer risk. A study with Cynomolgus monkeys exposed to PFOA did not demonstrate an effect on CCK levels or evidence of hepatic cholestasis.[33] Olsen et al reported a statistically significant negative (inverse) association between mean CCK levels and serum PFOA levels among PFOA production workers, even after adjusting for potential confounders.[34]

Perfluorooctane Sulfonic Acid (PFOS)

As is the case for PFOA, USEPA has developed an LHA for perfluorooctane sulfonic acid (PFOS),[35] based on a review of the available science and the application of standard scientific principles, and has indicated that it will develop a national drinking water standard for the substance. The LHA is based on the same animal study used by the Agency for Toxic Substances and Disease Registry (ATSDR) [36] which is the basis for the proposed groundwater quality standard for PFOS - a two-generation study by Luebker *et al.* (2005) reporting delayed eye opening and decreased pup weight in rats.[37] In its analysis, however, ATSDR ignored the conclusions of the authors regarding the relevant dose resulting in the adverse effects and inappropriately applied an additional uncertainty factor as described below. As a result, the proposed standard should be based on the analysis conducted by USEPA in developing the LHA, rather than that conducted by ATSDR.

In the case of pup weight, Luebker *et al.* noted the decreases observed in the second generation (F2) offspring at 0.4 mg/kg per day were transient, disappearing by the end of lactation. Reduced body weights were not reported in the F1 pups from the 0.4 mg/kg dose group. For both F1 and F2 offspring, body weight was reduced in the 1.6 mg/kg group. As a result, the authors identified 0.4 mg/kg as a no-observed-adverse-effect level (NOAEL) and 1.6 mg/kg as a lowest-observable-adverse-effect level (LOAEL). ATSDR, in contrast, inappropriately considered the LOAEL to be 0.4 mg/kg without explanation.

Similarly, Luebker *et al.* conclude that the slight delay in eye opening observed in the F1 pups from the 0.4 mg/kg dose group should not be considered an adverse effect and identified 0.4 mg/kg as the NOAEL. This finding is consistent with the results from the other studies in rats

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[33] Butenhoff J *et al.* Toxicity of ammonium perfluorooctanoate in male cynomolgus monkeys after oral dosing for 6 months. *Toxicol Sci* 69(1):244–57 (2002). https://doi.org/10.1093/toxsci/69.1.244

[34] Olsen GW *et al.* Plasma cholecystokinin and hepatic enzymes, cholesterol and lipoproteins in ammonium perfluorooctanoate production workers. *Drug Chem Toxicol* 23(4):603–20 (2000). https://doi.org/10.1081/DCT-100101973

[35] USEPA. Drinking water health advisory for perfluorooctane sulfonic acid (PFOA). EPA 822-R-16-004. Office of Water (May 2016).

[36] ATSDR. Toxicological profile for perfluoroalkyls. Department of Health and Human Services (May 2021).

[37] Luebker DJ *et al.* Two-generation reproduction and cross-foster studies of perfluorooctanesulfonate (PFOS) in rats. *Toxicol* 215(1-2):126-148 (2005). https://doi.org/10.1016/j.tox.2005.07.018

and mice referenced in the ATSDR Toxicological Profile which report NOAELs of 1.0 mg/kg or more. The decision to consider 0.4 mg/kg as a LOAEL, rather than NOAEL, has a significant impact on the ATSDR calculation and the proposed standards.

In its analysis ATSDR also applies a modifying uncertainty factor of 10 for PFOS based on a concern that "immunotoxicity may be a more sensitive endpoint of PFOS toxicity than developmental toxicity." While ATSDR provides no guidance on how to apply a modifying factor based on data base uncertainty, EPA's guidance explains that a database uncertainty factor (UF_D) is applied when reproductive and developmental toxicity studies are missing since they have been found to provide useful information for establishing the lowest no adverse effect level.[38] The EPA guidance notes that, for a reference dose (RfD) 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.[39] In deciding whether to apply an UFD, EPA advises that the assessor should consider both the data lacking and the data available for particular organ systems as well as life stages.

In the case of PFOS, the reproductive and development data base is robust and does not suggest the need to account for an incomplete characterization of toxicity. Similarly, the potential immunotoxic effects of PFOS have been studied in both laboratory animals and humans. The results of these studies are inconsistent and both EPA[40] and Health Canada[41] have questioned the relevance of immune system effects observed in mice and the small antibody variations seen in epidemiology studies to adverse health effects in humans. It is inappropriate, therefore, to conclude that immunotoxic effects represent a more sensitive health effect to justify the inclusion of a modifying factor of 10.

In developing the proposed groundwater standard for PFOS, IEPA assumes a relative source contribution (RSC) of 20 percent, despite the fact that PFOS use has decreased substantially.[42] 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 PFOS have declined drastically. According to data collected by the Center for Disease Control and

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[38] Dourson ML *et al.* (1996) Evolution of science-based uncertainty factors in noncancer risk assessment. *Regul Toxicol Pharmacol* 24:108–120 (1996). https://doi.org/10.1006/rtph.1996.0116

[39] EPA 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

[40] EPA. Health effects support document for perfluorooctane sulfonate (PFOS). EPA 822-R-16-002 (May 2016). https://www.epa.gov/sites/production/files/2016-05/documents/pfos_hesd_final_508.pdf

[41] Health Canada. Guidelines for Canadian Drinking Water Quality: Guideline Technical Document – Perfluorooctance Sulfinate (PFOS). Ottawa (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

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

Prevention (CDC), mean serum levels of PFOS declined by 85 percent in the US population between 1999 and 2016.[43] (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.

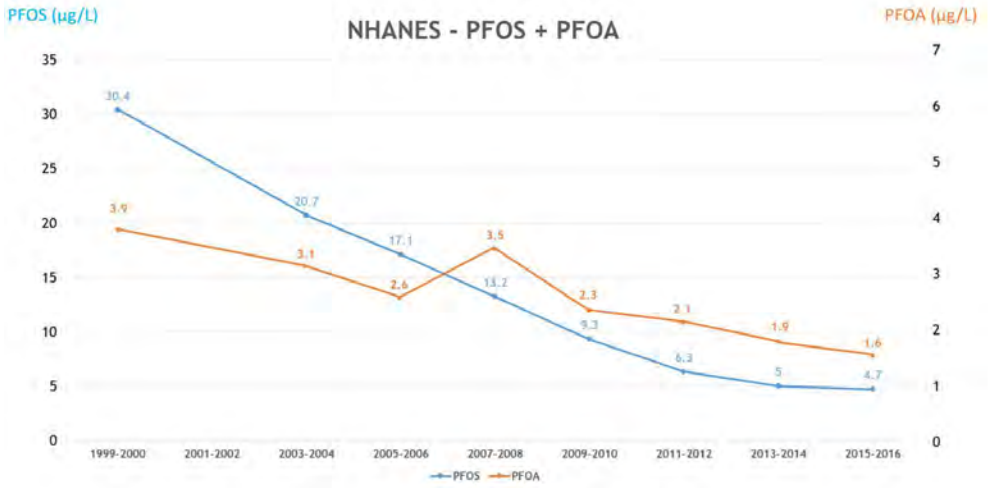

Figure 1. Serum levels of PFOA and PFOS, 1999-2016.[44]

Perfluorohexane Sulfonic Acid (PFHxS)

Very few studies exist that can be used as a basis for calculating a groundwater quality standard for perfluorohexane sulfonic acid **(**PFHxS). The available information report liver and thyroid effects in laboratory animals. The increases in liver weight and hepatocellular hypertrophy that have been reported, however, appear related to PPARα activity which ATSDR notes is a mechanism that "cannot be reliably extrapolated to humans" in the absence of other degenerative lesions.[45] ATSDR derived its minimum risk level (MRL), which is the basis for the proposed IEPA standard, from thyroid follicular cell damage reported by Butenhoff *et al.* 2009, despite the fact that the authors noted that the observed changes in rats "are consistent with the known effects of inducers of microsomal enzymes where the hepatocellular hypertrophy results in a compensatory hypertrophy and hyperplasia of the thyroid." While ATSDR acknowledged the questions regarding the relevance of the thyroid alterations reported by Butenhoff *et al.* to humans, including the significant differences in thyroid function between

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[43] CDC. Fourth national report on human exposure to environmental chemicals, updated tables (March 2021). https://www.cdc.gov/exposurereport/index.html

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

[45] ATSDR 2021, at A-72.

rodents and humans[46], it nevertheless selected thyroid as the basis for its MRL in the absence of other data.

Since ATSDR completed its analysis, the National Toxicology Program (NTP) has released the results of a 28-day study in rats that adds additional uncertainty to the relevance of the thyroid effects. Consistent with the earlier studies, NTP reported liver weight increases and decreases in thyroid hormones (T3, free and total T4) in rats exposed to PFHxS, along with a significant increase in PPARα activity.[47] Despite the decrease in hormone levels in a dose-response manner, the NTP study did not observe a consistent increase in thyroid stimulating hormone (TSH), as would be expected, nor were any histopathological changes (hyperplasia/ hypertrophy) observed in the thyroid gland. In reviewing these findings, the NTP report explains that "[t]he reason for a lack of TSH response in the face of substantially low thyroid hormone concentrations in these sulfonate studies is not clear and not consistent with a disruption in the hypothalamic-pituitary-thyroid axis." NTP further hypothesizes that the observed decrease in total T4 and T3 may be "related to activation of PPARα and constitutive androstane receptor (CAR) resulting in an increase in thyroxine-UDP glucuronosyltransferase and accelerated degradation of thyroxine by the liver."

Given the likelihood that both the available hepatic and thyroid effects data from studies of laboratory animal exposed to PFHxS are associated with PPARα in the liver which, as noted by ATSDR, cannot be reliably extrapolated to humans, IEPA should withdraw the proposed standard for PFHxS until more robust data are available.

In addition to the uncertainty in the endpoint used as a basis for the proposal for PFHXs, IEPA overestimates exposure to PFHxS from sources other than drinking water. As is the case for PFOS, CDC data indicate that serum levels of PFHxS have declined since 2000 consistent with the phase out of manufacture of the substance. As a result, those states that have evaluated PFHxS exposure have used a relative source contribution of 0.5, rather than the default of 0.2.

Perfluorononanoic Acid (PFNA)

As is the case with other PFAS, the liver appears to be the major organ of toxicity for PFNA. Consistent with the evidence for PFHxS, animals exposed to PFNA exhibited a significant increase in PPARα suggesting that the hepatic effects are a rodent-specific phenomenon. Deceases in thyroid hormones also have been consistently reported in the animal studies, with

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[46] Capen CC *et al.* Species differences in thyroid, kidney, and urinary bladder carcinogenesis. *IARC Scientific Publications* 147:1-14 (1999). https://publications.iarc.fr/Book-And-Report-Series/Iarc-Scientific-Publications/Species-Differences-In-Thyroid-Kidney-And-Urinary-Bladder-Carcinogenesis-1999

[47] NTP. Technical report on the toxicity studies of perfluoroalkyl sulfonates (perfluorobutane sulfonic acid, perfluorohexane sulfonate potassium salt, and perfluorooctane sulfonic acid) administered by gavage to Sprague Dawley (HSD:Sprague Dawley SD) Rats. NTP Tox 96. US Department of Health and Human Services (August 2019). https://ntp.niehs.nih.gov/publications/reports/tox/000s/tox096/index.html

no resulting increase in TSH, suggesting that the thyroid effects may be related to PPARα activity in the liver and of questionable relevance to humans.

The MRL developed by ATSDR, which is the basis for the proposed groundwater standard, is based on developmental effects reported by Das *et al.* 2015 who reported decreased body weight and developmental delays in the offspring of female mice exposed during gestation. The doses at which these developmental effects were observed also resulted in maternal effects, however. More significantly Wolf et al. (2010) did not find alterations in body weight or postnatal development in the offspring of PPARα knockout mice dams exposed to 2 mg/kg per day. This finding supports the conclusion that the developmental effects noted in rodents are dependent on PPARα and not relevant to humans.

The 2019 NTP 28-day study also included exposure to up to 2.5 mg/kg per day of PFNA in males (6.25 mg/kg per day in females) and measured the PFNA serum levels in the animals.[48] As with PFHxS, the hepatic and thyroid effects were accompanied by a significant increase in PPARα and CAR activity and suggest that these effects may not be relevant to humans. Among the other effects reported, NTP observed decreases in absolute and relative spleen and thymus weights in males exposed to 1.25 mg/kg per day and reduced testosterone levels and testis damage in male rats exposed to 2.5 mg/kg per day. No thymus weight or reproductive effects were reported in the female rats.

In addition to the uncertainty in the endpoint used as a basis for the proposal for PFNA, IEPA overestimates exposure to PFNA from sources other than drinking water. As is the case for other legacy PFAS, CDC data indicate that serum levels of PFNA have declined since 2000 consistent with the phase out of manufacture of the substance. As a result, those states that have evaluated PFHxS exposure have used a relative source contribution of 0.5, rather than the default of 0.2.

In its analysis ATSDR also applies a modifying uncertainty factor of 10 for PFNA based on the lack of a comprehensive study of reproductive effects and a general concern about sensitivity to immune function for other PFAS. As noted earlier EPA's guidance indicates that a database uncertainty factor (UF_D) is applied when reproductive and developmental toxicity studies are missing since they have been found to provide useful information for establishing the lowest no adverse effect level.[49] The EPA guidance notes that, for an RfD 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.[50] Since the reproductive data base for PFNA is lacking, a UF_D of 3 is appropriate. Although reports of reduced spleen and thymus weight may suggest effects on the immune system, the doses at which the effects have been

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[48] The NTP study included a higher dose group for either sex – 5 mg/kg for males and 12.5 mg/kg for females – but serum levels for animal in these groups was not reported.

[49] Dourson ML *et al.* (1996)

[50] EPA Risk Assessment Forum 2002.

observed are comparable to those for other effects and do not suggest a greater sensitivity of the immune system.

1,4-Dioxane

IEPA is proposing to lower the groundwater quality standard for 1,4-dioxane from 0.0077 to 0.00078 mg/L based on USEPA's 2013 toxicity assessment from the Integrated Risk Information System (IRIS).[51] The EPA IRIS analysis notes that, while there is substantial evidence that 1,4-dioxane causes cancer in laboratory animal via a threshold MOA, the supporting data is not sufficiently robust. Consequently, the IRIS assessment defaults to a mutagenic MOA in characterizing risk from 1,4-dioxane exposure. Since 2013 a significant amount of information has become available that supporting a threshold for carcinogenic response in animals exposed to 1,4-dioxane.

Based on the currently available evidence, the mutagenic MOA is inappropriate primarily because 1,4-dioxane is not genotoxic. This conclusion is based on extensive testing with *in vitro* assay systems with prokaryotic organisms, non-mammalian eukaryotic organisms, mammalian cells, *in vivo* genotoxicity assays, and most recently toxicogenomics analysis. In addition, there is ample evidence that the development of tumors only occurs when dosing exceeds the threshold of metabolic saturation. USEPA acknowledged this and included the threshold MOA in its assessment as noted in **Figure 2.** Metabolism studies confirm that, while the substance is readily metabolized and quickly eliminated from the body, the metabolic pathway becomes saturated at higher exposure levels of 1,4-dioxane. Moreover, available evidence demonstrates that toxicity occurs only after the clearance pathway becomes saturated and the parent compound accumulates in the blood. Thus, there is ample evidence to support a threshold MOA when assessing risks from exposure to 1,4-dioxane.

Although 1,4-dioxane has been reported to evoke multiple tumors in animal bioassays, the increased tumor incidences tend to occur at the highest dose only, and all are consistent with a threshold-based, non-mutagenic mode of action. Chronic and subchronic studies in laboratory animals exposed to levels above metabolic saturation have consistently demonstrated a threshold response to tumor formation from 1,4-dioxane exposure. This has been recognized by authoritative bodies worldwide who have applied a threshold assumption when characterizing risk of the substance. As a result, the World Health Organization (WHO)[52] and Health Canada[53] have developed a recommendation of 0.050 mg/L in drinking water.

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[51] USEPA. Toxicological review of 1,4-Dioxane (with inhalation update) (CAS No. 123-91-1) in support of summary information on the Integrated Risk Information System (IRIS.) EPA-635/R-11/003-F. Washington, DC (2013).

[52] WHO. 1,4-Dioxane in Drinking Water. Background document for development of WHO Guideline for Drinking Water Quality. WHO/SDE/WSH/05.08/120 (2005).

[53] Health Canada. (2021). Guidelines for Canadian drinking water quality. Guideline technical document - 1,4-Dioxane.Ottawa, Ontario. https://www.canada.ca/en/healthcanada/services/publications/healthy-living/guidelines-canadian-drinking-water-qualityguideline-1-4-dioxane.html

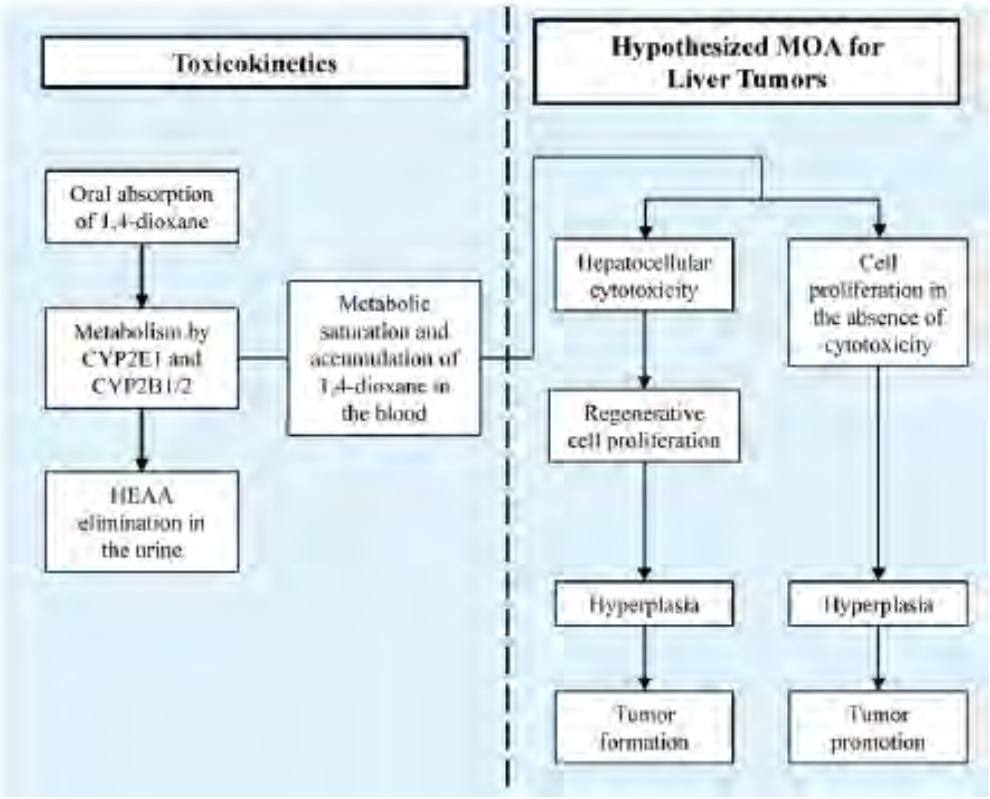

Figure 2. Identification of key events in liver tumor formation following exposure to 1,4-dioxane[54]

Results of Recent 90-Day Mode-of-Action Study

A recently competed 90-day study significantly adds to our understanding of 1,4-dioxane toxicokinetics by demonstrating a clear threshold for adverse effects in the liver of female mice. The results of this study are consistent with previous animal evidence that the metabolism of 1,4-dioxane shifts from linear, first-order metabolism to a zero-order kinetics with increasing exposures resulting in metabolic saturation. Once saturated, increased exposures result in a disproportional increase in circulating levels of 1,4-dioxane.

This study was designed to examine biological responses at specified, interim time points within the overall 90-day exposure period. Groups of ten female B6D2F1 mice were given drinking water at concentrations of 0, 40, 200, 600, 2000 or 6000 ppm 1,4-dioxane for a duration of 7, 28, or 90 days. The targeted dose levels were 0, 10, 50, 150, 500, and 1500 mg/kg per day. The results of the study have been reported in two separate publications which are enclosed with this submission.

[54] USEPA IRIS 2013, at 95.

When administered via the drinking water, the results of the 90-day study indicate a clear time- and dose-dependent threshold for hepatic effects.[55] The molecular and apical treatment-induced biological changes correlate with increased quantifiable concentrations of 1,4-dioxane in the blood and a potential shift in metabolism over time. After 7 days of treatment, liver weight increased in the 6000-ppm group correlated to increased centrilobular vacuolation characteristic of glycogen storage. After 28 and/or 90 days, liver weight increases in the 6000-ppm groups correlated with histopathological findings of increased centrilobular vacuolation, hypertrophy, and apoptosis. Notably, the magnitude of hepatocellular proliferative induction (~5-fold) at the highest dose is comparable to other mitogenic, non-genotoxic hepatocarcinogens.[56,57] Furthermore, under these experimental conditions, the inhibition of apoptosis MoA (*i.e.*, as measured by a decrease in the basal rate of caspase-positive staining) can be ruled-out as a significant contributing factor in 1,4-dioxane-mediated murine hepatocarcinogenesis.

Collectively, these data indicate that after administration at metabolically saturating doses of 1,4-dioxane, a direct mitogenic response is triggered in the liver of female mice. This mitogenic response occurs relatively early and likely adds to the regenerative repair that is suggested with the slight increase in single cell necrosis (apoptosis) seen in this study as well as in the chronic 2-year findings where more regenerative repair has been reported. Although these responses are small, they are happening in a target organ (liver) in a mouse strain that is highly susceptible to the induction of liver cancer. Most importantly, there is a clear threshold for all of these effects, supporting a threshold for the eventual induction of liver cancer.

The results of the transcriptomics analysis reported in the second publication from this 90-day study demonstrate an increase in signals consistent with xenobiotic metabolism, a subtle, yet significant, dose- and time-responsive increase in mitotic cell cycle and cellular proliferation, and a decrease in complement cascade processes and lipid metabolism.[58] The signals for proliferative response only occur at exposures of 2000 ppm or greater, while those related to xenobiotic metabolism occur as low as 600 ppm. There was no evidence of activation of DNA damage response and/or repair mechanisms at any of the concentrations and time points evaluated. Importantly, and consistent with all other findings, there were no significant changes in signaling pathways/gene sets at the transcriptomic level at drinking water concentrations below 600 ppm.

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[55] Lafranconi M *et al.* 2021. See enclosed publication.

[56] Geter DR *et al.* Dose-response modeling of early molecular and cellular key events in the CAR-mediated hepatocarcinogenesis pathway. *Toxicol Sci* 138(2):425-45 (2014). https://doi.org/10.1093/toxsci/kfu014

[57] LaRocca JL *et al.* Integration of novel approaches demonstrates simultaneous metabolic inactivation and CAR-mediated hepatocarcinogenesis of a nitrification inhibitor. *Toxicol Reports* 4:586-597 (2017). https://doi.org/10.1016/j.toxrep.2017.10.007

[58] Chappell GA *et al.* 2021. See enclosed publication.

The study adds significantly to our understanding of 1,4-dioxane toxicokinetics by demonstrating a clear threshold for any effect in the liver at a genomic level. Furthermore, the results of the study are consistent with those in the 13-week drinking water study reported by Kano *et al.* (2008) in which BDF1 mice were exposed to up to 25,000 ppm of 1,4-dioxane in drinking water.[59] The doses required to cause liver effects in the two 13-week study are considerably higher than those reported to cause liver tumors in female mice in the 2-year bioassay on which USEPA's IRIS assessment is based[60] which has caused some to question the significance of these tumors. In considering the results of the bioassay by Kano *et al.*, for example, Health Canada concluded that --

> The absence of non-cancer histopathological changes and the concomitant increase in liver enzymes in the [Kano *et al.* bioassay] despite the presence of both endpoints in the subchronic studies from the same group . . . lend credence to the uncertainty surrounding the development of tumors at this low dose.[61]

Given the clear evidence for a threshold MOA for cancer in animals exposed to 1,4-dioxane, application of approach to calculating a human threshold toxicant advisory concentration (HTTAC) is more appropriate for developing the groundwater standard for this substance.

Proposed Model for Carcinogens that Operate a Mutagenic Mode of Action

As part of the current rulemaking, IEPA is proposing to revise the model for calculating the human non-threshold advisory concentration (HNTAC) for carcinogens that operate by a mutagenic MOA to account for the possibility of increased risks from childhood exposure. The current cancer assessment methodology protects both adults and children and additional default assumptions and safety factors are not warranted because there is inadequate scientific evidence that the current methods are not suitably health protective. With respect to approaches for assessing the contribution of early life exposures to lifetime theoretical cancer risk, there is compelling and robust scientific evidence that mechanisms of carcinogenicity which operate in adults also operate in children, and that to the extent children may be more, less, or equally sensitive to some substances, current cancer assessment methodology is sufficiently conservative to protect children.

The hypothesis that exposure to carcinogens early in life leads to increased probability of tumor development, compared to exposure commencing later in life is not supported when a weight of evidence evaluation is conducted. Specifically, USEPA's analysis which is the basis of the

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[59] Kano H *et al.* Thirteen-week oral toxicity of 1,4-dioxane in rats and mice. *J Toxicol Sci* 33:141-153 (2008). https://doi.org/10.2131/jts.33.141

[60] Kano H *et al.* Carcinogenicity studies of 1,4-dioxane administered in drinking-water to rats and mice for 2 years. *Food Chem Toxicol* 47: 2776-2784 (2009). https://doi.org/10.1016/j.fct.2009.08.012

[61] Health Canada 2021.

IEPA proposal, is based on a on a very limited set of lab animal studies and substances: data sets from 4 chemicals from repeat exposure studies, data sets from 3 chemicals from lifetime exposure studies and data sets from 42 chemicals from acute exposure studies.[62] The analysis found the following –

- For the repeat exposure data sets, 45 ratios of susceptibility were analyzed by EPA and **58% demonstrated equal or less sensitivity** of the early life exposure period compared to exposure later in life.
- For the lifetime exposure data sets, 6 ratios of susceptibility were analyzed by EPA and **33% demonstrated equal or less sensitivity** of the early life exposure period compared to exposure later in life.
- For the acute exposure data sets, 515 ratios of susceptibility were analyzed by EPA and **45% demonstrated equal or less sensitivity** of the early life exposure period compared to exposure later in life

Combining all the datasets included in the analysis indicates that nearly half showed an equal or lower sensitivity of the early life exposure period.

In a separate analysis USEPA's Science Advisory Panel (SAP) reviewed data from 69 carcinogenicity bioassays, 40 of which contained combined perinatal and adult exposure and 12 of which contained a neonatal exposure component.[63] Although the majority of the studies were not designed to answer the question of relative susceptibility, the SAP noted that "combined perinatal and adult exposure slightly increases the incidence of a given type of tumor." Importantly, the SAP also noted "it is not known if this reflects the effect of an increased length of exposure or a heightened sensitivity of the young animal to the carcinogenic effects of the chemical."

Other studies have examined childhood sensitivity to both carcinogenic and noncarcinogenic substances. For example, Charnley and Putzrath (2001) provides a detailed discussion of how childhood risk may be greatly overestimated for carcinogens that must be metabolized by cytochrome P450 enzymes to become biologically active.[64] In general, Charnley and Putzrath observed that it is "difficult to make generalizations about the effect of age on susceptibility to chemical carcinogens. Age can affect metabolism, cell proliferation rates, and hormone levels, for example, which can in turn affect tumor incidence, latency, and tumor type, as can myriad other interactions that are genetically, behaviorally, and environmentally determined." Hatten examined studies that included prenatal exposures and concluded that fetal animals are often

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62 https://pubmed.ncbi.nlm.nih.gov/16140616/

63 U.S. EPA. Scientific Advisory Panel (SAP) September 1997 meeting session 2: A proposed OPP policy on determining the need for in-utero/perinatal carcinogenicity testing on a pesticide. Office of Pesticide Programs. Washington, DC (1997).

64 Charnley G and Putzrath RM. Children's health, susceptibility, and regulatory approaches to reducing risks from chemical carcinogens. *Environ Health Perspect* 109(2):187-192 (2001). https://doi.org/10.1289/ehp.01109187

not more susceptible to carcinogens.[65] He noted that only one of the substances he examined (ethylnitrosourea) demonstrated prenatal carcinogenicity "as it does not require enzymatic activation."

Data on early life sensitivity from the pharmacology literature also are available. For example, Crom reported that "anticancer drugs provide a useful model for assessing important differences in pharmacokinetic disposition between children and adults, particularly with regard to environmental exposure to toxins."[66] He observed that for many anticancer drugs the maximum tolerated dose in children is greater than for adults. This may be due to faster clearance of some compounds, decreased sensitivity to the toxic effect, or both. Differences in clearance may be the result of greater organ reserve (i.e., better kidney and liver function) in children, compared to adults who have been exposed to other drugs or toxins that may damage these organs. In general, however, children are often able to tolerate higher dosages than adults. Crom concludes "children have unique physiologic and pathologic characteristics that distinguish them from adults, but in general, drug disposition is more variable than in adults, and, on average, children have faster clearances of many drugs and can tolerate larger dosages (based on body size) than adults."

As a result of the significant uncertainty as to early life susceptibility, it is not appropriate to apply the updated model for mutagens unless IEPA can confirm that an assumption of such susceptibility is appropriate for the specific substance. Consistent with the USEPA's approach and recommendations, any assessment of cancer susceptibility will begin with a critical analysis of the available information. Chemical-specific data relating to MOA (e.g., toxicokinetic or toxicodynamic information) may suggest that even though a compound has a mutagenic MOA, higher cancer risks may not result. Such data should be considered before applying the age-dependent adjustment factors. Moreover, regarding a determination of the MOA, the Texas Council on Environmental Quality notes -

> The determination that a chemical carcinogen is capable of producing mutation is not sufficient to conclude that it causes specific tumors by a mutagenic MOA or that mutation is the only key event in the pathway to tumor induction. For a chemical to act by a mutagenic MOA, either the chemical or its direct metabolite is the agent inducing the mutations that initiate cancer. This is contrasted with a MOA wherein mutagenicity occurs as an indirect effect of another key event in carcinogenesis occurring later in the process.[67]

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[65] Hatten DG. In Utero Phase Carcinogenicity Testing. *Intl J Toxicol* 17:337-353 (1998). https://doi.org/10.1080/109158198226611

[66] Crom WR 1994. Pharmacokinetics in the child. *Environ Health Perspect* 102 Suppl 11:111–117 (1994). https://doi.org/10.1289/ehp.94102s11111

[67] TCEQ. Guidelines to Develop Toxicity Factors. RG-442. Toxicity Division (2015). https://www.tceq.texas.gov/assets/public/comm_exec/pubs/rg/rg-442.pdf

Appendix E: Proposal for Similar Acting Substances

The proposed Appendix E lists numerous substances that it proposes should be considered similar acting in various organ systems and, for which it assumes additivity of health effects and for which the dose-addition model described in Appendix B would be applied. However, while the proposed Appendix provides information on the target organ/system, it fails to identify a "common mode of toxic action" by which the substances cause an effect in the organ - as required in Appendix C. In fact, IL EPA has not identified the MOA for any of the substances included in Appendix E much less established a common MOA for multiple substances. The additivity of potential health effects of these substances should not be considered unless and until a common MOA can be established through an established framework. Such frameworks exist for both cancer[68] and non-cancer[69] MOAs.

The proposal for identifying similar acting substances also inappropriately seeks to apply the advisory concentration (threshold or non-threshold) to organs/systems for which the reference dose of slope factor do not apply. Since the advisory concentrations are based on the most sensitive effect that has been observed, applying that same level of toxicity to organs for which effects occur at higher does or that are clearly established for a substance significantly overstates the toxicity of the mixture. For example, as proposed Appendix E would include PFHxS in estimates of mixture toxicity for five organs or systems – circulatory, immune, developmental, liver, and thyroid - despite the fact that the advisory concentration for the substance is based on effects in only one (thyroid). IEPA has provided no data to support an association between circulatory, developmental, and immune effects and PFHxS exposure. Moreover, ATSDR, the source for the proposed HTTAC, has concluded that the liver effects observed in animals are not relevant to humans.

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[68] Boobis AR *et al.* IPCS Framework for analyzing the relevance of a cancer mode of action for humans. *Crit Rev Toxicol* 36(10):781-92 (2006). https://doi.org/10.1080/10408440600977677

[69] Boobis AR *et al.* IPCS Framework for analyzing the relevance of a noncancer mode of action for humans. *Crit Rev Toxicol* 38(2):87-96. https://doi.org/10.1080/10408440701749421

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A 90-day drinking water study in mice to characterize early events in the cancer mode of action of 1,4-dioxane

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ABSTRACT

Studies demonstrate that with sufficient dose and duration, 1,4-dioxane (1,4-DX) induces liver tumors in laboratory rodent models. The available evidence aligns with a threshold-dependent, tumor promotion mode of action (MOA). The MOA and key events (KE) in rats are well developed but less so in the mouse. Therefore, we conducted a 90-day drinking water study in female mice to evaluate early KE at 7, 28, and 90 days. Female B6D2F1/Crl mice consumed drinking water containing 0, 40, 200, 600, 2000 or 6000 ppm 1,4-DX. 1,4-DX was detected in blood at 90-days of exposure to 6000 ppm, but not in the other exposure groups, indicating a metabolic clearance threshold between 2000 and 6000. Early events identified in this study include glycogen-like vacuolization, centrilobular hypertrophy, centrilobular GST-P staining, apoptosis, and pan-lobular increase in cell proliferation observed after 90-days of exposure to 6000 ppm 1,4-DX. There was minimal evidence of hepatotoxicity over the duration of this study. These findings demonstrate a previously unreported direct mitogenic response following exposures exceeding the metabolic clearance threshold of 1,4-DX. Collectively, the information generated in this study supports a threshold MOA for the development of liver tumors in mice after exposure to 1,4-DX.

1. Introduction

Lifetime inhalation or oral exposure to 1,4-dioxane (1,4-DX) causes liver and other organ tumors in laboratory animals (Argus et al., 1973; Argus MF, Arcos JC, 1965; International Center for Medical Research et al., 1988; Kano et al., 2009; Kasai et al., 2009; Kociba et al., 1974; NCI, 1978). Tumor development in these studies generally occurs only at or near the maximum tolerated dose.

Currently available information from both chronic and sub-chronic rodent studies by various routes of administration is consistent with a threshold regenerative hyperplasia Mode of Action (MOA) as proposed by Dourson et al. (2014, 2017). While there is abundant information for characterizing the MOA for tumor development in rats, the evidence in mice is less developed. Earlier Japanese and NCI cancer bioassays, and their sub-chronic companion studies, provided only limited details concerning KE in the mouse model (Kano et al., 2009, 2008; NCI, 1978). In two recent analyses of the rodent liver tumor evidence, the 2-year NCI cancer bioassay in mice was re-evaluated with updated pathology standards to better characterize both the tumor and non-tumor lesions (Dourson et al., 2014). While this effort greatly expanded our understanding of the MOA for 1,4-DX in mice, there were still information gaps for characterizing early events in the development of hepatic tumors in mice exposed to 1,4-DX.

In this publication we present evaluations of clinical chemistry, biochemical and histological hepatic effects in female B6D2F1/Crl mice after exposure to 1,4-DX in drinking water for 7, 28, and 90 days. In addition, we related these findings to blood concentrations of 1,4-DX and its primary metabolite, hydroxyethoxy acetic acid (HEAA).

2. Materials and methods

This study was conducted in compliance with Good Laboratory Practice Standards and designed to generate information that would be relevant for interpreting the results from previous studies with 1,4-DX.

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Female B6D2F1 mice were selected to match as closely as possible the mouse strain used by Kano et al. (Kano et al, 2008, 2009). Drinking water concentrations were selected to reproduce the critical outcomes from previous studies, such as metabolic saturation, cytotoxicity, cell proliferation, apoptosis, and GSTP expression.

2.1. Chemicals

1,4-DX was obtained from Sigma-Aldrich (Lot SHBJ7415), St. Louis MO and was determined to be 99.98% pure. Reagents for BrdU (BD Biosciences:BD Pharmigen™ BrdU In-Situ detection Kit BD Biosciences, San Diego, CA; #551321) were obtained from Dako, (Carpenteria, CA). Caspase-3 reagents and antibodies were obtained from Dako (Carpenteria, CA and Biocare Medical (Concord, CA). GST-P (placental) (+ reagents and antibodies were obtained from Dako (Carpenteria, CA), Biocare Medical (Concord, CA), Biogenex, Fremont, CA), and Vector Labs (Burlingame, CA). A Provantis data collection system (Instem PLC, UK) was used to record information from the study.

2.2. Animals

Female B6D2F1/Crl mice, between the ages of 5 and 8 weeks old, were obtained from Charles River Laboratories, Inc. (Raleigh, NC). Initiation of treatment groups (i.e., 7-, 28-, or 90-day duration) were staggered to more closely align animal age at necropsy and to minimize growth-related hepatocellular proliferation. Animal care was in full accordance with applicable animal welfare standards including the U.S. Department of Agriculture's Animal Welfare Act (9) Code of Federal Regulations (CFR) Parts 1, 2 and 3, National Research Council Guide for the Care and Use of Laboratory Animals. Washington, DC (NRC, 2011), and the American Veterinary Medical Association (AVMA) Guidelines for the Euthanasia of Animals (AVMA, 2013).

Mice were implanted with transponders (BioMedic Data Systems, Seaford, DE) and acclimated for one week prior to continuous exposure to 1,4-DX in drinking water during which time they were pair-housed and provided with a Shephard Shack for enrichment purposes. The mice were fed LabDiet Certified Rodent Diet #5002 (PMI Nutrition International, St. Louis MO) ad libitum. All study animals were implanted with mini-osmotic pumps model 2ML1 (Alzet Corporation, Palo Alto, CA) eight days prior to scheduled necropsy for BrdU delivery. After pump implantation animals were housed individually.

2.3. Route of administration and exposure levels

Six groups of animals were treated with 1,4-DX in drinking water at 0 (control), 40, 200, 600, 2000 and 6000 ppm to achieve targeted dose levels of 0, 10, 50, 150, 500 and 1500 mg/kg/day. Final estimates of doses delivered were calculated utilizing concentrations of 1,4-DX in the drinking water, average water consumption, and body weights for each group.

2.4. Study design

Ten mice per exposure group were treated for 7, 28, or 90 days. At 7, 28, and 90 days of exposure, gross pathology, liver weights, histopathology and biomarkers were determined in all 10 animals per group at each time point. In addition, blood levels of 1,4-DX, and HEAA were assessed in five animals from each exposure group.

2.5. Liver histopathology, biomarkers, and microscopic evaluations

At 7, 28, and 90 days of exposure, non-fasted mice were anesthetized with isoflurane and CO_2, blood was collected and the mice were euthanized by decapitation. After weighing the liver, cross sections of the liver through the middle of the left lateral lobe, middle of the right medial lobe, and through the right lateral lobe were taken and preserved in neutral, phosphate-buffered 10% formalin. These liver sections were used for histopathological examination. The formalin fixed liver was processed for light microscopy which includes histochemical (hematoxylin and eosin; H&E) and immunohistochemical (BrdU)-labeled cells, caspase-3, and placental glutathione S-transferase (GST-P) staining. Further information on the biomarker assessments is provided in supplemental information.

2.6. 1,4-DX and HEAA analysis of whole blood

Blood samples were collected via the retro-orbital sinus from five non-fasted mice/dose/exposure duration at necropsy following anesthesia at the time of terminal sacrifice. Each blood sample was collected into pre-weighed vials containing methanol and 1% formic acid and stored at −80 °C until analyzed by via GC/MS methods. The limit of quantification in blood was determined to be 0.05 μg/mL for HEAA and 0.2 μg/mL for 1,4-DX.

3. Results

The approximate doses of 1,4-DX estimated for each exposure group were 0, 7.2 (±0.624), 37.3 (±2.59), 116 (±10.2), 364 (±27.0) and 979 (±83.9) mg/kg/day for animals consuming drinking water containing 0, 40, 200, 600, 2,000, and 6000 ppm 1,4-DX, respectively. Values are means for each group (± Standard Deviation).

There were no treatment related effects in clinical signs, body weights, or clinical chemistry parameters in any of the 7-, 28-, or 90-day 1,4-DX treated groups compared to their respective controls. There were no early deaths; all animals survived to scheduled necropsy.

During the 7-day treatment period, animals exposed to 6000 ppm 1,4-DX had a slight transient decrease in water consumption from test days 1–4 (14%), but this was not statistically different from test day 4–8, when compared to their respective control group. There were no treatment-related differences in water consumption in any 1,4-DX treated animals during the 28-day treatment period when compared to their respective control group. During most intervals in the 90-day treatment period, animals exposed to 6000 ppm 1,4-DX had treatment-related decreases in water consumption ranging from 12 to 29% compared to their respective control. There were no differences in feed consumption.

3.1. Liver weights

After 7, 28, and 90 days there was a modest increase in relative liver weights of 8.7%, 10.7% and 8.9%, respectively, of animals exposed to 6000 ppm 1,4-DX with sporadic increases in relative liver weights in the 2000 ppm exposure group. No changes in relative liver weights were observed in groups exposed to 1,4-DX at less than 2000 ppm – see Supplemental Information Table 3

3.2. Microscopic observations

Histopathological (H&E) findings are briefly summarized in Table 1. After 7 days of exposure, minimal to mild vacuolation consistent with glycogen deposition was observed in the centrilobular regions of the liver in animals exposed to drinking water concentrations of 600 ppm and higher. By day 28, the centrilobular vacuolation was largely resolved.

Minimal to mild centrilobular hypertrophy, appearing as granular eosinophilic cytoplasm, appeared after 7 days of exposure. At 90-days of exposure, there was increased severity of eosinophilic, slightly granular cytoplasmic hypertrophy in the livers of mice exposed to 6000 ppm 1,4-DX – see Fig. 1. Single cell necrosis (interpreted as apoptosis) was increased at 6000 ppm with all mice showing evidence of minimal or mild single-cell necrosis at 90 days of exposure. No evidence of single cell necrosis was observed in mice exposed at or below 600 ppm 1,4-DX

Table 1
Incidence of liver histopathology findings.

Finding: Observation	1,4-DX Concentration (ppm)					
	0	40	200	600	2000	6000
Day 7 Number of Mice Examined	10	10	10	10	10	10
Centrilobular Vacuolation Minimal	0	0	0	2	6	0
Mild	0	0	0	0	4	10
Day 28 Number of Mice Examined	10	10	10	10	10	10
Centrilobular Vacuolation Minimal	0	0	0	3	6	1
Mild	0	0	0	0	3	9
Centrilobular Hypertrophy Minimal	0	0	0	0	0	6
Mild	0	0	0	0	0	4
Centrilobular Apoptosis Minimal	0	0	0	0	0	7
Mild	0	0	0	0	0	1
Day 90 Number of Mice Examined	10	10	10	10	10	10
Centrilobular Vacuolation Minimal	0	0	0	0	8	1
Mild	0	0	0	0	1	1
Centrilobular Hypertrophy Minimal	0	0	0	0	1	0
Mild	0	0	0	0	0	1
Moderate	0	0	0	0	0	9
Centrilobular Apoptosis Minimal	0	0	0	0	1	6
Mild	0	0	0	0	0	4

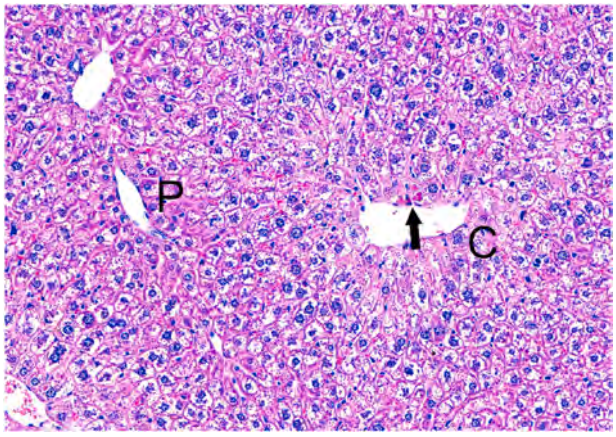

Fig. 1. Liver, 90-day, 6000 ppm. Moderate centrilobular hepatocyte hypertrophy and minimal vacuolation. There is also mild centrilobular hepatocyte apoptosis (arrow); note densely eosinophilic condensed cell bodies and lack of inflammation. "P" denotes Periportal while "C" marks the Centrilobular region. See supplemental information for additional photomicrographs of liver sections from this study.

(see Fig. 2).

3.3Biomarkers of Liver response

There were no consistent, treatment-related changes in hepatocellular proliferation in any dose group at 7 or 28 days. There was a treatment-related increase in hepatocellular proliferation as measured by BrdU incorporation at 90-days in animals exposed to 6000 ppm 1,4-DX. The BrdU incorporation was pan-lobular with a 4.3% and 20.8% labeling index in control and 6000 ppm group after 90 days of exposure. This increase in BrdU incorporation corresponds with the increase in relative liver weights as well as blood levels of 1,4-DX.

Consistent with the histopathological assessment, the 28-day and 90-day animals exposed to 6000 ppm 1,4-DX had statistically significant increases in apoptosis as measured by Caspase-3 positive cells compared to controls (0.08 and 0.46 for 28-day animals, and 0.04 and 1.08 for 90-day animals per 10,000 cells in control and high dose, respectively). There were no treatment-related differences in any other treatment group of the 28-day or 90-day treated animals when compared to their respective controls – see Supplemental Information Table 4.

GST-P+ labeling area was evaluated in the animals exposed to 6000 ppm 1,4-DX for 90 days. Earlier time points and exposures were not evaluated based on reports from prior 90-day studies (Kasai et al., 2008). GST-P+ captures possible altered hepatic foci but instead of focal collections of cells representing the clonal expansion of pre-neoplastic hepatocytes, an enhanced centrilobular staining of zone 3 hepatocytes was observed following exposures to 1,4-DX. Quantitative morphometry was not done, but visual inspection revealed that the GST-P+ centriblobular expression was greater in the 6000 ppm 1,4-DX treated group than the controls, as evidenced by a larger number of stained hepatocytes radiating away from the central vein (Fig. 3).

3.4. Blood concentrations of 1,4-DX and HEAA

Blood levels of HEAA exhibited linear, dose-proportional concentrations across all dose groups at all treatment durations. There was only sporadic detection of 1,4-DX in animals from the lower exposure groups (<2000 ppm) demonstrating that at these lower levels of exposure, metabolism of 1,4-DX was complete. Blood levels of 1,4-DX showed an abrupt increase in animals exposed to 6000 ppm 1,4-DX for 90-days. The appearance of 1,4-DX was biphasic, increasing in greater proportion relative to the exposure) at 6000 ppm after 90 days of exposure. This pattern is consistent with saturation of metabolic clearance pathways of 1,4-DX after prolonged exposures between 2000 ppm (approximately 400 mg/kg/day) and 6000 ppm (approximately 1000 mg/kg/day) 1,4-DX (Fig. 4).

4. Discussion

Identifying the MOA and its KE framework is an important element in modeling the cancer risk from rodent carcinogenicity data (Simon

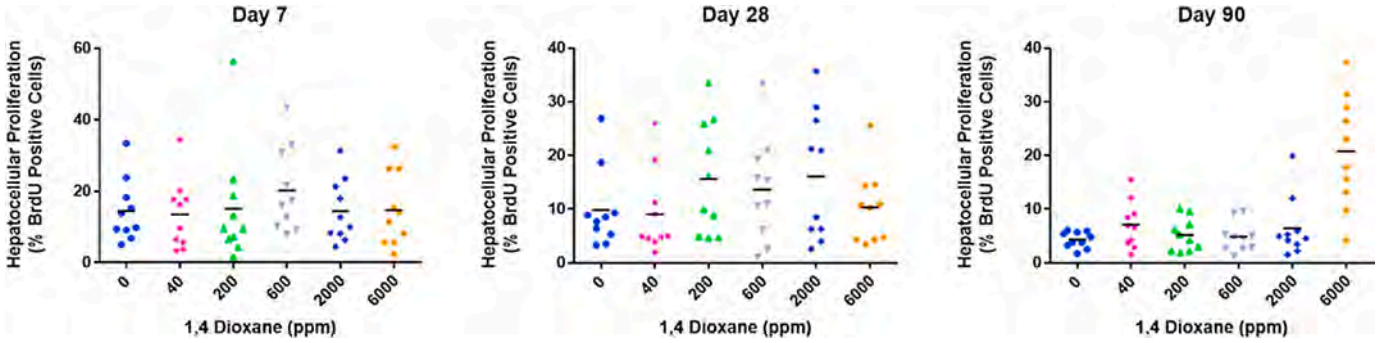

Fig. 2. Individual BrDU values after 7, 28, and 90 days of exposure to 1,4-DX in drinking water. Bars indicate means.

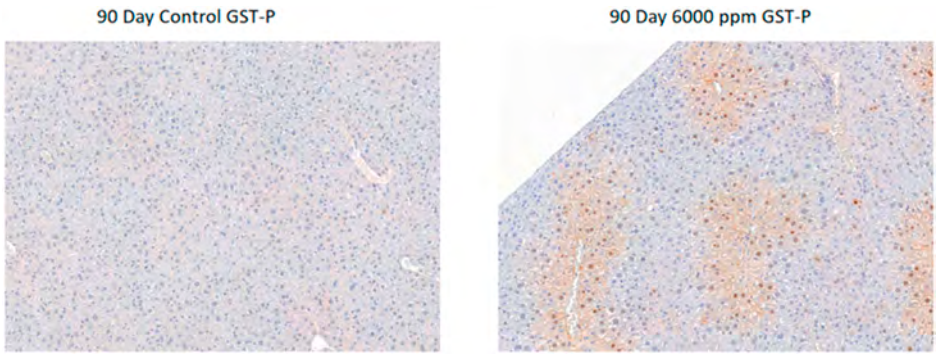

Fig. 3. Hepatic GST-P + Staining after 90 days of exposure to 1,4-DX in drinking water.

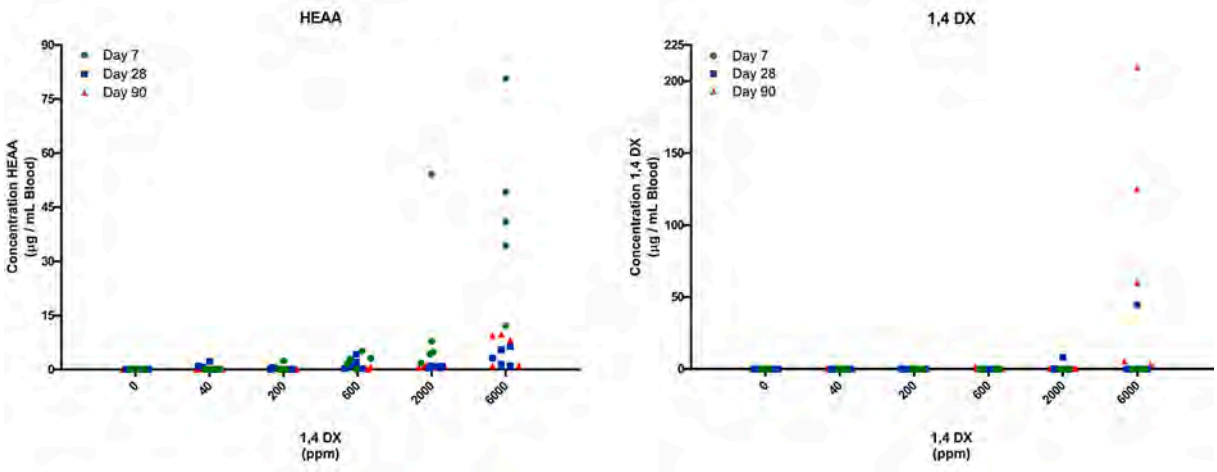

Fig. 4. Blood concentrations of HEAA metabolite and 1,4-DX after 7, 28, and 90 days of exposure to 1,4-DX in drinking water.

et al., 2014; US EPA, 2005a). For most carcinogens that are not mutagens, a threshold MOA establishes safe exposure level below which there is no adverse outcome (Bevan and Harrison, 2017; US EPA, 2005a). However, to support a threshold effect under the US EPA's 2005 Cancer Risk Assessment Guidelines (US EPA, 2005a), the MOA must be established. The elimination of a mutagenic MOA does not default to a threshold approach. To date, there are only a few chemicals with carcinogenic potential assessed in EPA's IRIS, database, (e.g., chloroform, perchlorate, and 2-butoxyethanol) that have met the standard for a threshold MOA (US EPA, 2010a; 2005, 2001). This study establishes that histological and biomarker responses are linked to saturation metabolisms of 1,4-DX thus providing additional KE information supporting a threshold approach for 1,4-DX cancer risk assessments.

Under the conditions of this study, there appeared to be two phases of response of female B6D2F1/Crl mice to 1,4-DX exposure from drinking water. The early phase consisted of increased liver weights, interpreted as hepatic hypertrophy, and transient increases in hepatic glycogen content. Both of these responses were adaptive and observed in the first 28-days of exposure to 1,4-DX. The late phase responses included a mitogenic response of hepatocellular proliferation, an increase in single-cell necrosis (or apoptosis) and a loss of stored glycogen. The late phase responses appeared between 28 days and 90-days of exposure to 1,4-DX and correlated with the appearance of 1,4-DX in the blood.

The mitogenic response was characterized by a pronounced increase in lobule-wide BrdU incorporation in the 6000 ppm exposure group after 90-days of exposure. Accompanying the onset of high dose centrilobular single cell apoptosis and cell proliferation in the late-phase response was a loss of glycogen-like vacuolation and increased centrilobular staining for GST-P. A similar pattern of hepatocyte proliferation was reported in rats (Goldsworthy et al., 1991). After two weeks of continuous administration of drinking water containing 1% (10,000 ppm) 1,4-DX there was a doubling of the labeling index of 3H thymidine incorporation in the rat liver. However, administration of approximately 1000 mg/kg in a single bolus dose by oral gavage did not increase the labelling index in rats at either 24 or 48 h after administration.

Notably, the late phase responses in this current study occurred at exposures that exceeded the metabolic clearance threshold although some mild changes in the liver were seen at lower exposures. The results from this study indicate there is a mitogenic response which appears to be a KE in the mouse liver tumor MOA. This mitogenic response precedes the later-developing cytotoxicity observed in longer-term studies in mice (Dourson et al., 2014, 2017).

4.1. *Blood concentrations of 1,4-DX and HEAA*

The dose dependent increase in the appearance of blood levels of HEAA is consistent with current understanding of its metabolism in rodents and humans. In both rodents and humans, 1,4-DX is metabolized by cytochrome P-450 (primarily Cyp2b1/2 and Cyp2e1) to HEAA in a linear, first-order process (Nannelli et al., 2005; Sweeney et al., 2008; Young et al, 1977, 1978). This kinetic pattern has been demonstrated directly by monitoring plasma levels after intravenous administration of 1,4-DX, indirectly from studies monitoring the elimination of HEAA in the urine, and from studies with rodent and human hepatocytes. This metabolic transformation is responsible for the rapid clearance of 1, 4-DX and elimination in the urine. However, higher levels of exposure saturate the biotransformation of 1,4-DX which transitions to zero-order

kinetics resulting in the appearance of circulating levels of parent 1,4-DX (Sweeney et al., 2008; Young et al., 1978).

The results from this current study demonstrate a biphasic kinetic profile and saturation. Blood levels of HEAA decline with an associated appearance of measurable levels of 1,4-DX after 90-days of exposure. This late onset of measurable levels of 1,4-DX in the presence of falling HEAA concentrations directly correlates with the appearance of late KE (apoptosis and increased DNA synthesis) observed at 90-days. Average blood levels of 1,4-DX were less than 1 μg/mL in mice exposed for 90 days in drinking water at 2000 ppm 1,4-DX (limit of detection 0.2 μg/mL) but increased to an average blood concentration of 81 μg/mL in mice exposed to 6000 ppm 1,4-DX. At the same time, the ratio of 1,4-DX to HEAA in blood increased from 0.38 in the 2000 ppm group to 13.7 in the 6000 ppm group. The increase in circulating levels of 1,4-DX and the role in hepatic injury, including development of liver tumors after a lifetime of exposure in rats and mice, has been well documented (ATSDR, 2012; Dourson et al., 2014, 2017; US EPA, 2019; 2013).

There is good evidence that metabolism of 1,4-DX does not generate reactive intermediates capable of causing cytotoxicity. Investigations into the formation of reactive intermediates have failed to generate evidence of DNA reactivity and repair, protein binding, or enhancement of cytotoxicity after induction of xenobiotic biotransformation (Goldsworthy et al., 1991; Stott et al., 1981; Woo et al., 1977). Although the specific molecular initiating event causing toxicity from 1,4-DX exposure is unknown, the available evidence points to the accumulation of parent 1,4-DX as the toxic species. The toxicologically-relevant events observed in this study, cell proliferation and apoptosis, correlated with the appearance of circulating blood levels of 1,4-DX.

In this study, the threshold for metabolic saturation was between 2000 and 6000 ppm of 1,4-DX in drinking water which is equivalent to approximately 400 and 1000 mg/kg/day respectively, after 90-days of exposure to 1,4-DX. Sweeney et al. (2008) estimated a metabolic saturation threshold in male B6C3F1 mice of approximately 200 mg/kg/day after a single oral gavage. The difference in the threshold estimate from this current study may be related to the strain and sex differences between studies. Female mice have a pronounced enhancement of expression of mRNA from the Cyp 2 b subfamily compared to males with some isoforms expressed more than 100-fold in female mouse liver compared to males (Renaud et al., 2011). Other sub-families of Cyp also show higher expression in females. These differences could account for the increased capacity for biotransformation of 1,4-DX and the increased metabolic threshold observed in this study. In addition, the method of dosing may have influenced the observed metabolic threshold in this study compared to estimates from previous studies. Sweeney et al. dosed 1,4-DX in a single bolus oral dose. In this current study 1,4-DX was administered ad libitum in drinking water.

Blood samples obtained at day 7 consistently show higher concentrations of HEAA than either the 28 or 90-day samples. The declining HEAA concentrations with later time points could indicate a shift in metabolic capability between 7 and 28 days of exposure favoring a competing metabolic pathway, such as conjugation, resulting in lower total HEAA blood levels at the later time points. Studies by Woo et al. (1977) demonstrated that 1,4-DX may induce its own metabolism via mixed function oxidases. However, the 1,4-DX-induced changes in metabolism may be more complex than simple induction of one system and with different time courses for reaching steady-state in the presence of 1,4-DX.

4.2. Cytotoxicity

There were no consistent statistical or treatment-related differences in the serum liver enzymes measured in the blood in any of the 7-, 28-, or 90-day 1,4-DX-treated animals when compared to their respective controls. This is consistent with results from previous 90-day studies in mice (Kano et al., 2008; Kasai et al., 2008). In both of these studies, there were significant increases in circulating levels of ALT and AST in the highest exposure groups but no changes in groups exposed to 1,4-DX at exposure levels comparable to the exposures used in this current study.

Likewise, there were no histopathological findings to indicate cytotoxicity in livers from animals exposed to less than 2000 ppm at any time point. There was some minimal to mild centrilobular vacuolation which appeared at 7 and 28 days from exposures of 600 ppm and greater. This vacuolation was judged to be an increase in glycogen and considered an adaptive response. At 90-days of exposure, the vacuolation resolved in the 600 ppm 1,4-DX exposure group and only appeared in the higher dose groups. This is similar to findings from previous studies in which glycogen storage was reduced after exposure to 1,4-DX (Dourson et al., 2014; Stott et al., 1981). It is difficult to determine the significance of these observations but the role of glycogen storage modulation has shown to be relevant in the progression of hepatic tumors (Bannasch et al., 1997; Nayak et al., 1996).

There was also a time and concentration dependent increase in single-cell necrosis in liver sections from mice exposed to 2000 and 6000 ppm 1,4-DX for 28 and 90 days in this study. Similar findings of single-cell necrosis at higher doses were reported in other mouse studies (Kano et al., 2008; NCI, 1978). Single-cell necrosis is generally interpreted as an indication of apoptosis (Elmore et al., 2016). The apoptosis interpretation is also supported by the increase in caspase-3 positive hepatocytes noted in livers from mice exposure to 6000 ppm 1,4-DX for 28 and 90 days. Premature loss of hepatocytes due to 1,4-DX-triggered apoptosis could contribute to a regenerative response as evidenced by increased BrdU (discussed below) and the slight hepatic hypertrophy that was observed.

The findings from this study reveal no evidence of cytotoxicity below 2000 ppm and only limited evidence of hepatic injury based on the increase in apoptosis at higher exposure levels. This is in contrast to the observations from the two-year NCI study (1978) where evidence of cytotoxicity was observed from clinical chemistry and histologic pathology (Dourson et al., 2014, 2017). The difference between the findings from this current study, and those obtained from chronic mouse studies is likely due to the time course of exposure. It appears the development of cytotoxicity requires exposures greater than the 90 days employed in this study.

4.3. Biomarkers

In this study we did not observe the emergence of pre-neoplastic foci including basophilic, eosinophilic, clear cell or mixed cell foci, or a clear expression of GST-P foci as was observed in chronic studies of rats (Dourson et al., 2014; Kasai et al., 2009). GST-P foci have been used as a pre-neoplastic biomarker in rats (Satoh et al., 1985) but the absence of foci in this study is not surprising. Mice express high levels of GST-P constitutively in the liver which can mask the appearance of foci (Hayes and Pulford, 1995).

4.4. Mode of Action

Establishing the MOA is important in determining the appropriate model for evaluating cancer risk (US EPA, 2005a). The current prevailing MOA for 1,4-DX is the regenerative hyperplasia model (Dourson et al., 2014, 2017). Evidence supporting this model was largely derived from chronic rodent bioassays and shorter-term studies (primarily 90-day studies) with limited information available to characterize early events in the MOA. In this study we have attempted to characterize the earlier time course of events involved in the induction of mouse liver tumors.

The results from this study provide further evidence for the metabolic saturation of clearance pathways as a KE leading to accumulation of systemic 1,4-DX. There was a time- and dose-dependent threshold for this saturation and the development of the subsequent KE. In the cancer studies with 1,4-DX, exposures above the metabolic threshold led to the development of hepatic tumors (Kano et al., 2009; Kociba et al., 1974;

NCI, 1978). This relationship has been described previously from data generated in both rats and mice (Dourson et al., 2017). Importantly, the mitogenic stimulation observed in this study, approximately a five-fold increase in liver proliferation (labeling index) in the 6000 ppm exposure group after 90 days, occurs prior to the development of cytotoxicity and the regenerative repair that is a cornerstone of the regenerative hyperplasia MOA. In this study, 1,4-DX exposure stimulated hepatic proliferation as a result of an apparent direct mitogenic response which is recognized as a carcinogenic MOA (US EPA, 2005a). We note that magnitude of the proliferative response observed is comparable to other mitogenic, non-genotoxic hepatocarcinogens (Geter et al., 2014; LaRocca et al., 2017).

While the essentiality of the metabolic clearance threshold relationship for subsequent tumor development has been demonstrated in numerous rodent studies, we recognize that in one study (Kano et al., 2009), hepatic tumors in female mice have been reported at exposures predicted to be below the estimated metabolic saturation. In the Kano bioassay study, there was a significant increase in combined hepatocellular adenomas and carcinomas in female mice exposed to 500 ppm (approximately 66 mg/kg/d) 1,4-DX in drinking water for two years. The estimated metabolic threshold for mice is 200 mg/kg/day (Sweeney et al., 2008). However, this was determined in male mice from a different mouse strain (B6C3F1). It is possible that the metabolic threshold in female mice is lower than that the male mice used by Sweeney et al. However, it is unlikely to account for the three-fold difference between the dose leading to tumor formation in the low dose females from the Kano bioassay study and the dose estimated to achieve metabolic saturation in either female or male mice. Thus, the observations of tumors in female mice in the Kano et al. studies at doses below presumed metabolic saturation is inconsistent with the weight of evidence from other rodent cancer bioassays or with information generated in this 90-day study.

The weight of evidence supports the conclusion that 1,4-DX is not likely to be genotoxic (ATSDR, 2012; US EPA, 2019, 2010). Numerous in vitro and in vivo studies have reported no genotoxicity with only sporadic reports of genotoxicity observed in rats exposed to 1,4-DX (Morita and Hayashi, 1998; Roy et al., 2005) and more recently (Gi et al., 2018; Itoh and Hattori, 2019; Totsuka et al., 2020). 1,4-DX-induced cytotoxicity has already been associated with weak genotoxicity outcomes before, but this was not considered relevant MOA for 1,4-DX-induced tumorigenesis (IRIS, 2013). The positive findings from in vivo studies occurred at doses that exceed the threshold for metabolic clearance and lend further support to the threshold nature of the tumor response to 1,4-DX.

Receptor mediated MOAs, such as the peroxisome proliferator-activated receptor -alpha (PPARα) and the constitutive androstane receptor (CAR), can also play a role in the developments of tumors in rodents exposed to non-genotoxic carcinogens (Elcombe et al., 2014; Klaunig et al., 2003). However, the pattern of responses in rodents resulting from 1,4-DX exposure do not completely align with the MOA. Peroxisome proliferation is a key observation observed from PPARa activity and CAR activation generally leads to inhibition of apoptosis (Felter et al., 2018). Neither of these are observed in 1,4-DX exposed rodent liver. Furthermore, whole transcriptome analyses of mRNA of liver tissues from our 90 mouse study shows no evidence of PAPRα or CAR activity (Chappell et al. manuscript in preparation). Specifically, there was no change in the expression of individual CYP-encoding genes that are considered markers of activation of such nuclear receptors, nor enrichment of gene-level changes in the signaling pathways relevant to these nuclear receptors.

The observations from this current study support the regenerative hyperplasia model with one important additional modification – inclusion of an early onset, direct mitogenic stimulus occurring prior to the development of cytotoxicity, necrosis and the regenerative processes as described in previous MOA rodent hepatic tumor models (Dourson et al., 2017) and depicted in Fig. 5. This mitogenic response occurs early and likely adds to the regenerative repair that is suggested from the increase in single cell necrosis (apoptosis) seen in this study. Although these responses are small, they occur in a target organ (liver) in a mouse strain that is highly susceptible to the induction of liver cancer (Holsapple et al., 2006; Katagiri et al., 1998; Yamate et al., 1990). Importantly, there is a clear threshold of these effects which only occur at exposures that exceed the metabolic clearance threshold and only after 90-days of exposure.

The mitogenic event is presented in red to indicate the new finding from this study. All other events have been reported previously.

5. Conclusion

When 1,4-DX was administered via the drinking water to female B6D2F1/Crl mice for up to 90 days, there was a strong time- and exposure-dependent threshold for hepatic effects. These effects progressed from an early phase of adaptive effects to a late phase of adverse effects. The molecular and apical treatment-induced biological changes correlated with increased quantifiable concentrations of 1,4-DX in the blood. Within the first 90 days of drinking water exposure to 1,4-DX the

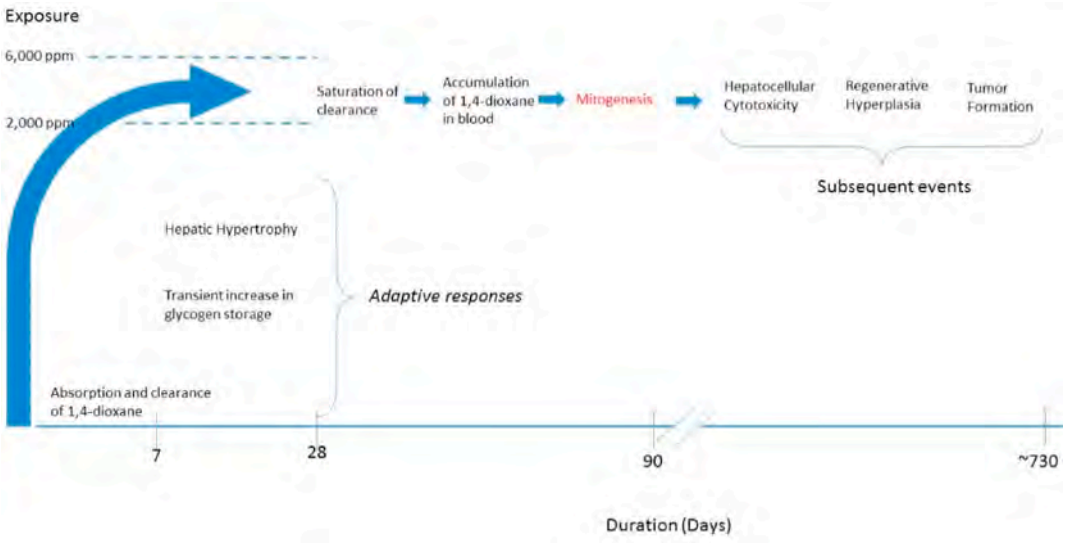

Fig. 5. Updated MOA of 1,4-DX induced development of hepatic tumors.

absence of evidence of significant hepatic cytotoxicity and the increase in cell proliferation indicate that a cytotoxicity/regenerative MOA alone does not account for the subsequent sequence of events leading to tumor formation. Collectively, these data indicate that after 90 days of exposure, at metabolically saturating doses of 1,4-DX, a mitogenic response is triggered in the liver of a sensitive strain of female mice that precedes the development of cytotoxicity and regenerative hyperplasia, ultimately leading to tumor development. This mitogenic response may be considered a KE in support of the threshold MOA for development of liver tumors in female mice after exposure to 1,4-DX.

The findings from this study extend the understanding of the MOA for 1,4-DX-induced hepatic tumors in mice. This is important in that the MOA of an environmental agent is key to the appropriate application of the most up-to-date cancer risk assessment approaches (Boobis et al., 2006; Cohen et al., 2019; Holsapple et al., 2006; Wolf et al., 2019).

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.yrtph.2020.104819.

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Transcriptomic analyses of livers from mice exposed to 1,4-dioxane for up to 90 days to assess potential mode(s) of action underlying liver tumor development

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ABSTRACT

1,4-Dioxane is a volatile organic compound with industrial and commercial applications as a solvent and in the manufacture of other chemicals. 1,4-Dioxane has been demonstrated to induce liver tumors in chronic rodent bioassays conducted at very high doses. The available evidence for 1,4-dioxane-induced liver tumors in rodents aligns with a threshold-dependent mode of action (MOA), with the underlying mechanism being less clear in the mouse than in rats. To gain a better understanding of the underlying molecular mechanisms related to liver tumor development in mice orally exposed to 1,4-dioxane, transcriptomics analysis was conducted on liver tissue collected from a 90-day drinking water study in female B6D2F$_1$/Crl mice (Lafranconi et al., 2020). Using tissue samples from female mice exposed to 1,4-dioxane in the drinking water at concentrations of 0, 40, 200, 600, 2,000 or 6,000 ppm for 7, 28, and 90 days, transcriptomic analyses demonstrate minimal treatment effects on global gene expression at concentrations below 600 ppm. At higher concentrations, genes involved in phase II metabolism and mitotic cell cycle checkpoints were significantly upregulated. There was an overall lack of enrichment of genes related to DNA damage response. The increase in mitotic signaling is most prevalent in the livers of mice exposed to 1,4-dioxane at the highest concentrations for 90 days. This finding aligns with phenotypic changes reported by Lafranconi et al. (2020) after 90-days of exposure to 6,000 ppm 1,4-dioxane in the same tissues. The transcriptomics analysis further supports overarching study findings demonstrating a non-mutagenic, threshold-based, mitogenic MOA for 1,4-dioxane-induced liver tumors.

1. Introduction

1,4-Dioxane is a volatile organic compound currently used in industrial processes as a solvent, in the manufacture of other chemicals, and as a laboratory reagent (ATSDR, 2012). Chronic exposure to high levels of 1,4-dioxane via the inhalation or oral routes has been observed to cause liver tumors in laboratory rodents (Argus et al., 1973; Argus MF, Arcos JC, 1965; International Center for Medical Research. et al., 1988; Kano et al., 2009; Kasai et al., 2009; Kociba et al., 1974; NCI, 1978). In recent years, investigators have put forth a hypothesized MOA for 1,4-dioxane-induced mouse liver tumors, with hepatic cytotoxicity and subsequent regenerative hyperplasia proposed as key events (KE) for tumor development, subsequent to metabolic saturation and consequential accumulation of the parent compound in the blood (Dourson et al., 2014, 2017). However, questions remain as to whether there is sufficient information to understand early events in the development of hepatic tumors in mice exposed to 1,4-dioxane, and to support an initiating event of hepatotoxicity.

To further investigate the MOA related to 1,4-dioxane hepatocarcinogenicity in rodents, specifically mice, female B6D2F$_1$/Crl mice were exposed to 0, 40, 200, 600, 2,000 or 6,000 ppm (approximately 0, 7.2, 37.3, 116, 364, and 979 mg/kg bw/day) 1,4-dioxane in drinking water for 7, 28, or 90 days (Lafranconi et al., 2020). The B6D2F$_1$ mouse, which has been shown to be particularly susceptible to the development of liver tumors (Yamate et al., 1990; Katagiri et al., 1998), was selected to match as closely as possible the strain used in a previous study that demonstrated increased liver tumors at 66 mg/kg bw/day 1,4-dioxane (Kano et al., 2009). In the in-life portion of the present study reported by Lafranconi et al. (2020), the threshold for metabolic clearance was determined to be between 2000 and 6000 ppm, with pathological changes observed in the liver only after

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90 days of exposure to the highest concentration (6000 ppm). The liver pathology was characterized as glycogen-like vacuolation, centrilobular hypertrophy, increased centrilobular GST-P staining, apoptosis, and a pan-lobular increase in cell proliferation (Lafranconi et al., 2020). These findings were concluded to demonstrate an early mitogenic response to 1,4-dioxane following sub-chronic (90 days) exposure to concentrations that exceeded the metabolic clearance threshold (Lafranconi et al., 2020). Mitogenesis is well-recognized as a nongenotoxic MOA for cancer (Cohen and Ellwein, 1990; U.S. EPA, 2005) with species differences (Elcombe et al., 2014), and is especially relevant to liver tumors in sensitive strains of mice (Maronpot, 2009).

As transcriptomic data can provide additional and/or supporting information regarding underlying mechanisms of effects associated with specific exposure scenarios (Gao et al., 2015; Dean et al., 2017; Joseph, 2017; Mulas et al., 2017), and can potentially be integrated into mode of action (MOA) analysis and human health risk or hazard assessments (Chepelev et al., 2015; Moffat et al., 2015; Johnson et al., 2020; LaRocca et al., 2020), whole transcriptome analyses were conducted on liver tissues from the 90-day drinking water study (7, 28, or 90 days of exposure). Transcriptomic signatures can also demonstrate adaptive, transient, and/or beneficial reactive responses to exposure. Considering the existing 1,4-dioxane evidence base, we hypothesized that genes related to xenobiotic metabolism, cell death, and cell proliferation would be altered by 1,4-dioxane exposure. Further, we sought to identify any additional molecular signaling alterations related to the liver effects seen in 1,4-dioxane-exposed mice. To address the question of genotoxicity, the presence of mRNA-level responses that may indicate enrichment of DNA damage and/or response pathways was specifically investigated. Gene set enrichment analysis and dose–response modeling were conducted to understand alterations in biological and disease processes across treatment groups. The transcriptomic signatures in the livers of exposed mice were also considered in relation to phenotypic data (i.e., apical endpoints) as determined by histopathological and immunohistochemical analyses of sections from the same liver tissue blocks, which demonstrated a significant increase in single-cell apoptosis and proliferation after 90 days of exposure, and an overall lack of significant treatment effect in the liver at concentrations of 1,4-dioxane below 6000 ppm (Lafranconi et al., 2020). The transcriptomic alterations were considered together with the phenotypic data reported by Lafranconi et al, (2020) to inform the MOA underlying the liver effects observed in female B6C2F$_1$/Crl mice. This information is important for understanding the relevance of the findings and dose–response observed in sensitive strains of mice for assessing human health risks where potential exposure occurs with much lower dosages, such as via ingestion of contaminated drinking water.

2. Materials and methods

2.1. Animal husbandry and exposure conditions

The in-life study method details are described in Lafranconi et al. (2020). Briefly, the subchronic toxicity of 1,4-dioxane was evaluated in a 90-day study in female B6D2F$_1$(BDF1)/Crl mice (Charles River Laboratories, Inc. [Raleigh, NC] aged between 5 and 8 weeks) exposed continuously to 0, 40, 200, 600, 2000, or 6000 ppm 1,4-dioxane in drinking water for 7, 28, or 90 days. The targeted mg/kg/day dose levels were 0, 10, 50, 150, 500, and 1500 mg/kg/day. The mouse strain and route of exposure (drinking water) were chosen to enable comparison to the results of the cancer bioassay findings reported by Kano et al. (Kano et al., 2009). Daily dosages at various time points were estimated using drinking water concentrations, body weights and average water consumption per group. Female mice were fed *ad libitum* LabDiet Certified Rodent Diet #5002 (PMI Nutrition International; St. Louis, MO). Animal care followed applicable animal welfare standards including the U.S. Department of Agriculture's Animal Welfare Act (9) Code of Federal Regulations (CFR) Parts 1, 2 and 3, National Research Council Guide for the Care and Use of Laboratory Animals. Washington, DC (NRC, 2011), and the American Veterinary Medical Association (AVMA) Guidelines for the Euthanasia of Animals (AVMA, 2013). Non-fasted mice were euthanized by CO_2 anesthesia and decapitation at 7, 28, and 90 days of exposure. Liver tissues were fixed in neutral, phosphate-buffered 10% formalin and embedded in paraffin.

2.2. RNA sequencing

Formalin-fixed paraffin embedded (FFPE) liver samples from each mouse (n = 5 per treatment group; i.e., each duration and concentration) were microtomed to obtain a single 4–6 μm liver section mounted on a glass slide (uncovered), yielding a total of 90 samples for RNA sequencing. Slides were shipped to BioSpyder Technologies (Carlsbad, CA) where the unstained liver sections were evenly scraped from the slides and processed according to the TempO-Seq protocol, as previously described (Yeakley et al., 2017). DNA libraries created from each liver sample were sequenced using a HiSeq 2500 Ultra-High-Throughput Sequencing System (Illumina, San Diego, CA). RNA sequencing data are publicly available at NCBI's Gene Expression Omnibus[1] (GEO series accession number GSE154899).

2.2.1. Data processing and analysis

Sequencing data were analyzed using multiple packages in the R software environment, version 4.0.2 (cran.r-project.org/). The number of sequenced reads per probe were extracted from the sequencing output files; a traditional alignment step was not required because TempO-Seq uses gene-specific probe sequences. The DESeq2 R package (version 1.28.1) (Love et al., 2014) was used to normalize data to account for sample-to-sample variation in sequencing depth. Samples with below-optimal sequencing depth or low representation of expressed genes were not included in the comparative analysis. This was characterized by a total number of sequence reads >2 standard deviations below the mean sequenced reads per sample (5,635,830 and 8,839,173 across two sequencing runs), or a total number of genes sequenced >2 standard deviations below the mean number of genes sequenced per sample (16,132 and 17,350 across two sequencing runs). Application of these criteria resulted in the removal of five samples from the total 90 samples that were sequenced. Removal of low-count probes was not conducted because it is not necessary when using the DESeq2 package, owing to the application of shrunken fold-changes and independent filtering to stabilize low-count probes (Love et al., 2014).

2.2.2. Identification of differentially expressed genes

Significant differentially expressed genes (DEGs) were identified for each concentration of 1,4-dioxane within DESeq2 based upon estimated variance-mean dependence in the TempO-Seq count and a model using the negative binomial distribution. DEGs for each concentration compared to controls within the same timepoint were determined using a Wald statistical test and betaPrior set to "false" within DESeq2. Genes were considered to be significant DEGs if one of their corresponding probes had a false discovery rate (FDR) <10% following adjustment for multiple testing using the Benjamini and Hochberg (BH) procedure (Love et al., 2014).

2.2.3. Biological pathway enrichment analysis across concentrations of 1,4-dioxane

Biological pathways associated with gene expression profiles were

[1] https://www.ncbi.nlm.nih.gov/geo/

identified by pathway enrichment analysis. Mouse gene identifiers were converted to human identifiers using the biomaRt R package (v2.44.1) based on the Ensembl genome database (http://uswest.ensemble.org/index.html). The gene expression data were then queried for enrichment among gene sets in collections available in the Molecular Signatures Database (MSigDB) (http://software.broadinstitute.org/gsea/msigdb/index.jsp). The Canonical Pathways sub-collections were used (c2.cp.v6.2), which include gene sets from the following pathway databases: BioCarta online maps of metabolic and signaling pathways (BIOCARTA) (Nishimura, 2001), the Kyoto Encyclopedia of Genes and Genomes (KEGG) (Ogata et al., 1999), the Pathway Interaction Database (PID) (Schaefer et al., 2009), and the Reactome database of reactions, pathways, and biological processes (REACTOME) (Croft et al., 2011).

Enrichment of gene sets and pathways was determined using two methods: the gene set enrichment analysis (GSEA) statistical method and a hypergeometric test. The GSEA method follows the analysis platform made available by the Broad Institute (http://software.broadinstitute.org/gsea/index.jsp); the second employed a simpler hypergeometric test (Falcon and Gentleman, 2008). The GSEA method (Subramanian et al., 2005) determines whether sets of genes (e.g., the members of a molecular signaling pathway) are significantly concordant between various defined groups (in the case presented herein, different doses and timepoints) based on a ranking metric (in this case, the Wald statistic for expression differences between the 1,4-dioxane concentrations and control mice). The GSEA method was applied within Platform for Integrative Analysis of Omics data (PIANO) R package (v2.4.0) (Väremo et al., 2013), with geneSetStat = "gsea" and significance calculated using permutation-based nominal P values based on weighted Kolmogorov-Smirnov test enrichment scores, adjusted for multiple hypothesis testing by calculating FDRs using the BH method (Subramanian et al., 2005). The second method, a hypergeometric test, considers only significant DEGs (i.e., FDR $<10\%$ by DESeq2 analysis) for overrepresentation among genes sets listed in the Canonical pathways sub-collections using the Fisher combined probability test function in the PIANO R package (using "runGSAhyper"). No fold-change criteria were set. For both analyses, a minimum of 5 and a maximum of 500 genes was set for the gene set size (number of member genes represented in the dataset tested, i.e., the results of the sequencing experiment presented herein) criteria for inclusion in the analysis. Gene sets with an FDR $<10\%$ were considered to be significantly enriched.

2.2.4. Investigation of DNA damage response

To further investigate enrichment of gene sets relevant torel DNA damage response and/or repair, a collection of gene sets was curated by searching through all gene sets in the MSigDB collections (v6.2) using key words related to DNA damage response. A total of 89 gene sets that are related to DNA damage and/or response were identified and then tested for enrichment among significant DEGs (i.e., FDR $<10\%$ by DESeq2 analysis) using a hypergeometric test for overrepresentation, using all genes among these 89 gene sets as the background (i.e., the gene "universe"). No fold-change criteria were set for the DEGs tested for enrichment. This targeted approach was conducted separately from the gene set enrichment analysis using the broader Canonical Pathways gene sets as a means to specifically evaluate enrichment of DNA damage-related gene sets. Some overlap exists in the gene sets from the Canonical Pathways collection and the curated list of DNA damage-specific list of 89 gene sets. A minimum of 5 and a maximum of 500 genes was set for the gene set size (number of member genes represented in the dataset tested, i.e., the results of the sequencing experiment presented herein) criteria for inclusion in the analysis. Gene sets with an FDR $< 10\%$ were considered to be significantly enriched.

Additionally, high-throughput screening (HTS) data available via the US EPA's ToxCast downloadable data (invitroDBv3.2 database summary files[2]) were reviewed for 1,4-dioxane in a battery of nine assays (plus relevant viability or baseline assays) that are related DNA damage/repair (Hsieh et al., 2019).

2.3. Benchmark dose analysis

Dose-response modeling was conducted in using BMDExpress software (v2.2) (Phillips et al., 2019) using normalized expression data from DESeq2 without transformation. A Williams trend test (P value cutoff = 0.05) was employed to identify genes perturbed by 1,4-dioxane exposure. Fold-change filters and correction for multiple tests were not applied. Benchmark dose (BMD) analysis was conducted with linear, power, hill, 2° and 3° polynomial, and exponential models 2 to 5. The models were run assuming constant variance and a benchmark response (BMR) of 1 standard deviation. Functional classification of dose-responsive genes (genes with BMD P < 0.1) was conducted using the Gene Ontology (GO) and REACTOME gene sets available within BMDExpress. Genes were filtered from the analysis according to the default parameters within BMDExpress, as follows: genes with BMD/BMDL > 20, BMDU/BMDL > 40, BMDs above the highest dose (6000 ppm)), and/or genes with a BMD $>$ 10-fold below the lowest positive dose were removed from functional classification analysis. No filters for minimum or maximum number of genes per gene set were used. Benchmark doses for the gene sets were also estimated. Additional parameters for the BMD modeling and pathway analyses can be found in Supplemental Materials.

3. Results

3.1. In-life summary

The results of the in-life portion of the study are described in Lafranconi et al. (2020). Briefly, there was no treatment-related effect on clinical signs, clinical chemistry parameters, body weights, or survival at any dose or timepoint in 1,4-dioxane-treated groups compared to controls. Liver weights were slightly increased in the 6000 ppm group at all timepoints; no changes were observed at lower concentrations. Histopathological analysis revealed minimal to mild vacuolation consistent with glycogen deposition in the centrilobular regions of the liver in animals exposed to 1,4-dioxane at concentrations ≥600 ppm after 7 days of exposure, which was nearly completely resolved by day 28. Minimal to mild centrilobular hypertrophy and centrilobular apoptosis was evident in the 2000 ppm and 6000 ppm groups at 28 and 90 days of exposure. There were no consistent treatment-related changes in hepatocellular proliferation in any dose group at 7 or 28 days according to immunohistochemical staining for bromodeoxyuridine (BrdU) incorporation, while there was a treatment-related increase at 90-days in the 6000 ppm group. The increase in BrdU incorporation corresponded with increased relative liver weights and blood levels of 1,4-dioxane (Lafranconi et al., 2020).

3.2. Transcriptomic changes associated with exposure to 1,4-dioxane

RNA sequencing was performed on liver samples to examine exposure effects of 1,4-dioxane on the hepatic gene expression of female mice compared to time-matched control mice. All sample libraries passed quality control measures necessary to be sequenced. As already noted in the *Materials and Methods*, following sequencings, five samples were removed from the analysis due to low sequencing depth or low gene diversity, from the 200 and 600 ppm groups across all three timepoints. There was an overall lack of transcriptomic response in the 40 and 200 ppm concentration groups, with the number of DEGs for

[2] https://www.epa.gov/chemical-research/exploring-toxcast-data-downloadable-data (downloaded August 16th, 2019)

these two concentrations across the three timepoints ranging from 0 to 22 (Table 1, Supplemental Table S1). At 600 ppm, an increase in the transcriptomic response was observed at both the 7 and 90 day timepoints, but not at 28 days. At 6000 ppm, the number of DEGs exhibited a similar pattern as the 600 ppm concentration, with a higher response at 7 and 90 days relative to 28 days (Table 1, Fig. 1). Approximately half of the DEGs for the 600 ppm group were also differentially expressed in the 6000 ppm group at 90 days, while at 7 days there was less overlap in the same genes being differentially expressed in the 600 vs. the 6000 ppm groups. At 2000 ppm, the transcriptomic response was similar across timepoints. The lower number of DEGs at 2000 ppm compared to 600 ppm at days 7 and 90 was an unexpected finding, and is without evidence of spurious origin. The overall relatively low number of DEGs across all experimental groups and timepoints likely contributed to this variability. The virtual lack of transcriptomic response following exposure to 1,4-dioxane concentrations below 600 ppm at all timepoints supports a conclusion that there is a threshold concentration for hepatic transcriptomic response to 1,4-dioxane in female mice somewhere in the range of 200 to 600 ppm. It is noted that the lowest dose tested in the Kano et al. (2009) bioassay was 500 ppm and the lowest dose in the NCI (1978) drinking water bioassay in mice was 5000 ppm (NCI, 1978; Kano et al., 2009).

3.3. Gene set enrichment analysis

3.3.1. Analysis by dose group relative to time-matched controls

The results of both gene set enrichment analysis methods were evaluated to further understand 1,4-dioxane treatment-related effects. Although the top-most significantly enriched pathways were similar across the two methods used for pathway enrichment analysis (Supplemental Tables S2 and S3), the hypergeometric method was determined to be less informative for the objective of the present study due to the minimal changes in gene expression at the lower concentrations (40–200 ppm), resulting in a complete lack of gene set enrichment at those concentrations. Thus, the results discussed below focus on the pre-ranked GSEA method for enrichment analysis. Due to the minimal treatment effect of 1,4-dioxane at any dose or timepoint on gene expression changes, liberal criteria were applied to identify DEGs and enriched signaling pathways. The full set of results for the hypergeometric test can be found in Supplemental Table S3. Pathway enrichment analysis of significantly differentially expressed genes at 40 ppm and 200 ppm 1,4-dioxane yielded very few significantly enriched gene sets/pathways using the pre-ranked GSEA test (Table 2, Supplemental Table S2).

The decreased regulation of complement and coagulation cascades, mitochondrial β-oxidation and several other fatty acid metabolism pathways observed in the 600 ppm group is consistent with other transcriptomic analyses of liver tissues from primate and mouse studies in which animals were treated with nuclear receptor agonists that induce mitosis and DNA synthesis (e.g., fibrates (Cariello et al., 2005; Lu et al., 2011; de la Rosa Rodriguez et al., 2018) (Table 2).

At the 2000 and 6000 ppm concentrations, significant enrichment of xenobiotic metabolism pathways was evident, which increased in significance with increasing time and dose. Examples of enriched up-regulated gene sets associated with phase II metabolism, specific to glutathione conjugation, include KEGG “glutathione metabolism” and REACTOME “glutathione conjugation” (Table 2). The enrichment of these gene sets was driven by altered genes that encode glutathione transferase isoforms. Additionally, similar to the pathway alterations observed at 600 ppm, complement and coagulation cascade pathways were enriched in the negative direction (down-regulated) at 2000 and 6000 ppm 1,4-dioxane due to decreased expression of genes encoding proteolytic subunits in the complement system and gene members of the serpin family (serine protease inhibitors) relative to controls (Table 2). The complement cascade is a part of the innate immune system and deficiency of certain serpins (e.g., Serpina1) has been associated with liver damage (Law et al., 2006), which is consistent with the reported increase in cell death in the highest 1,4-dioxane dose groups (Lafranconi et al., 2020). Down-regulation of extracellular matrix regulators is also related to the loss of genes related to clotting factors. The significant decrease in expression of lipid metabolism-related gene sets in the 2000 and 6000 ppm may be related to liver injury in these high 1,4-dioxane dose-groups following saturation of metabolism and accumulation of the parent compound (as described in Lafranconi et al., 2020).

At the 90-day timepoint, mitotic cell cycle and DNA synthesis pathways were significantly enriched in the 6000 ppm treatment group: aurora B kinase signaling, mitotic phase transition and checkpoint signaling, and general cell cycle (e.g., Reactome “Cell cycle, mitotic”) (Fig. 2, Table 2), indicating a mitogenic proliferative response that was not observed at earlier time points. Enriched tubulin folding pathways share many of the same gene members as the cell cycle gene sets (Fig. 2). This is consistent with biomarkers of proliferative liver response in the same tissues, as reported in Lafranconi et al. (2020). Specifically, a treatment-related pan-lobular increase in hepatocellular proliferation was observed at 90-days in animals exposed to 6,000 ppm, as measured by BrdU incorporation. The increase in BrdU incorporation corresponded with an increase in relative liver weight as well as blood levels of 1,4-dioxane. There were no consistent, treatment-related changes in hepatocellular proliferation at 7 or 28 days in any dose group (Lafranconi et al., 2020).

3.3.2. Targeted analysis of DNA damage response

According to the targeted analysis (hypergeometric test for over-representation) of changes in expression in genes included in a curated list of 89 gene sets related to DNA damage response and repair (Supplemental Table S4), there was no enrichment. A hypergeometric test was necessary for this assessment, due to the nature of the evaluation using a focused list of gene sets (Supplemental Table S5). Additionally, 1,4-dioxane was inactive in the battery of HTS assays used to identify compounds with genotoxic potential (Supplemental Table S6). It should be noted that challenges exist in testing volatile chemicals (such as 1,4-dioxane) in HTS assays, as these *in vitro* assays involve the use of open vessels with incubations carried out at temperatures ranging from 4 °C to 37 °C. In such conditions, a substance with a high vapor pressure can potentially volatilize during the course of the assay, thereby influencing the concentration of the test substance in the system. While 1,4-dioxane has a molecular weight less than 140 g/mol (88.11 g/mol), which indicates volatility, its vapor pressure and log octanol/water partition coefficients (38.1 mmHg and −0.27, respectively) are within suitable boundaries for ToxCast/Tox21 assays (Tice et al., 2013; Richard et al., 2016). Overall, these results are consistent with other findings indicating that 1,4-dioxane does not cause direct DNA damage in the liver *in vivo* in mice, nor does it cause changes in *in vitro* assays designed to detect DNA damaging agents (as reviewed in (EPA, 2010; ATSDR, 2012)). These findings support a non-mutagenic MOA.

3.3.3. Benchmark dose modeling

The dose–response for individual genes were analyzed using BMD modeling, and functional characterization of the dose-responsive genes was analyzed and visualized. The BMD results confirmed pathway enrichment results obtained for single dose groups. For example, similar to what was found at 90 days in the ≥600 ppm 1,4-dioxane groups, the REACTOME gene sets “glutathione conjugation” and “Phase II – Conjugation of compounds” were significantly enriched at 90 days with median BMD_{1SD} values of 1548 and 1652 ppm, respectively, and median BMDLs of 1236 and 1251 ppm, respectively (Table 3, Fig. 3). In addition, “innate immune system”, a part of the complement and coagulation cascade pathway, was also significantly enriched in the negative direction with a median $BMD_{1SD} > 3200$ ppm. Cell cycle and mitosis gene sets (median $BMD_{1SD} > 3400$ ppm or

Table 1
Number of differentially expressed genes for each dose and length of exposure compared to time-matched control groups (shown as total DEG (Up-regulated [↑], Down-regulated [↓])). Full DESeq2 results can be found in Supplemental Table S1.

Exposure Duration (days)	1,4-Dioxane Concentration (ppm)				
	40	200	600	2000	6000
7	0 (0)	2 (↑0, ↓2)	411 (↑165, ↓246)	20 (↑6, ↓14)	415 (↑180, ↓235)
28	1 (↑0, ↓1)	1 (↑0, ↓1)	1 (↑0, ↓1)	49 (↑21, ↓28)	232 (↑87, ↓145)
90	5 (↑1, ↓4)	22 (↑11, ↓11)	323 (↑165, ↓158)	33 (↑25, ↓8)	727 (↑352, ↓375)

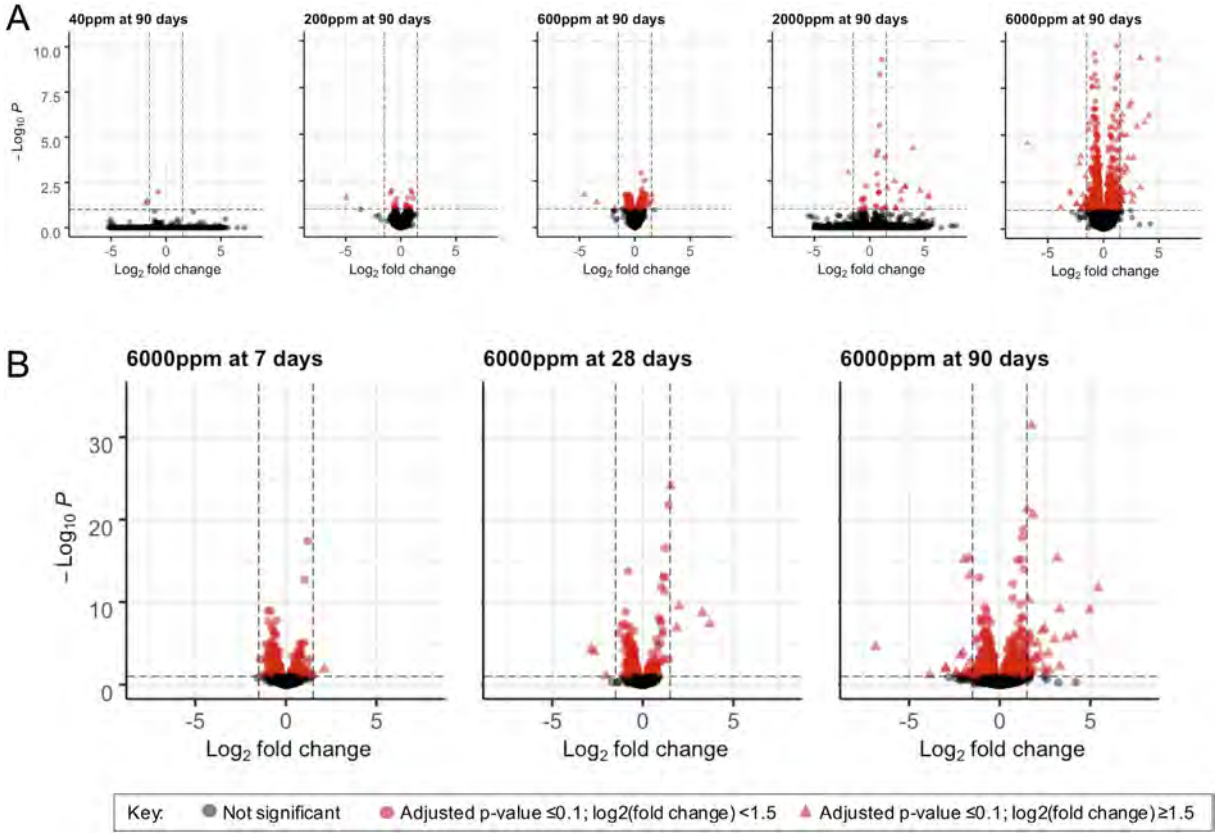

Fig. 1. Volcano plots showing differentially expressed genes across concentrations at the 90-day timepoint (A), and across all three timepoints at the 6000 ppm concentration (B). Red points represent probes with an adjusted p-value ≤ 0.1; circles represent probes within a log2 (fold change) < 1.5, and red triangles represent probes with a log2 (fold change) ≥ 1.5. A: y-axis is scaled for all plots from 0 to 10 (resulting in some points cut off the plot for the 6000 ppm concentration). B: y-axis for all plots is scaled from 0 to 35. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

higher, BMDLs > 2200 ppm or higher) along with a single "DNA repair" gene set (median BMD_{1SD} > 4000 ppm) were enriched among dose-responsive genes at 90 days. Individual genes within the "DNA repair" gene set that were identified as dose-responsive are mainly histone encoding genes and DNA polymerase genes involved in DNA synthesis (Supplemental Table S7). An exception is the DNA repair gene *Rad51*, which was found to have a significant dose-responsive trend via BMDExpress (p = 0.0217 by Williams Trent Test) at 90 days. However, this gene had generally low expression in all treatment groups and was not significantly differentially expressed at any individual dose at any timepoint relative to time-respective controls according to DESeq2 analysis (adjusted p-value ≥ 0.1 for all probes for all concentrations and timepoints; Supplemental Table S1).

Similar to the gene sets that were determined as significantly enriched at 90 days according to the GSEA analysis at individual doses/timepoints, phase II metabolism, cell cycle and mitosis gene sets were up-regulated with comparable BMD values at 28 days (Fig. 3). The "DNA Repair" gene set was not significantly enriched according to BMD modeling at 28 days. Moreover, "fatty acid metabolism" and "immune system" were down-regulated at 28 days (median BMD_{1SD} 3385 and 3367 ppm, respectively) but with less statistical significance compared to the results at 90 days (i.e., lower Fisher's test p-values). Among the dose-responsive genes at 7 days, phase II metabolism gene sets were enriched, with much higher median BMD_{1SD} values than those determined at 28 and 90 days. Although the gene ontology used in the BMDExpress software (REACTOME) was not an exact match to that of the GSEA analysis, the REACTOME gene sets were included in both analyses. Further, many similar gene sets exist across different ontologies, enabling a reasonable comparison of biological signals within the two analyses.

At 7 days, the top-most enriched gene sets among dose-responsive genes were related to signal transduction and were down-regulated, with median BMDs > 2000 ppm. This may represent an early stress response and/or cytotoxicity. "Glucuronidation" and "Phase II – Conjugation of compounds" were enriched, and up-regulated (median BMDs 1092 and 2301 ppm, respectively). The "DNA repair" gene set

Table 2
Top most significantly enriched pathways for each treatment group according to the GSEA method (Subramanian et al., 2005). The top five most significantly enriched pathways for each direction of change are shown in the table; in cases where five gene sets were not significantly enriched, only those with an adjusted p-value < 0.1 are shown. Full results are presented in Supplemental Table S2.

1,4-Dioxane (ppm)	Duration (days)	Overall Direction	Gene set	Adjusted p-value
40	7	Up	None	NA
		Down	None	NA
	28	Up	REACTOME DEGRADATION OF THE EXTRACELLULAR MATRIX	0.023524
			REACTOME HS GAG DEGRADATION	0.094038
		Down	None	NA
	90	Up	None	0.000913
		Down	REACTOME SRP DEPENDENT COTRANSLATIONAL PROTEIN TARGETING TO MEMBRANE	0.0015971
			REACTOME UNFOLDED PROTEIN RESPONSE	0.0019783
			KEGG TERPENOID BACKBONE BIOSYNTHESIS	0.0024729
			REACTOME TRANSLATION	0.0031942
			REACTOME CHOLESTEROL BIOSYNTHESIS	0.0032972
200	7	Up	REACTOME TRANSLATION	< 0.0001
			REACTOME PEPTIDE CHAIN ELONGATION	< 0.0001
			REACTOME 3 UTR MEDIATED TRANSLATIONAL REGULATION	< 0.0001
			REACTOME SRP DEPENDENT COTRANSLATIONAL PROTEIN TARGETING TO MEMBRANE	< 0.0001
			KEGG RIBOSOME	< 0.0001
		Down	KEGG BIOSYNTHESIS OF UNSATURATED FATTY ACIDS	0.019492
			KEGG PEROXISOME	0.021777
			REACTOME SULFUR AMINO ACID METABOLISM	0.022062
			KEGG ARGININE AND PROLINE METABOLISM	0.028247
			REACTOME PYRUVATE METABOLISM AND CITRIC ACID TCA CYCLE	0.029404
	28	Up	None	NA
		Down	None	NA
	90	Up	None	NA
		Down	None	NA
600	7	Up	REACTOME 3 UTR MEDIATED TRANSLATIONAL REGULATION	< 0.0001
			REACTOME PEPTIDE CHAIN ELONGATION	< 0.0001
			REACTOME NONSENSE MEDIATED DECAY ENHANCED BY THE EXON JUNCTION COMPLEX	< 0.0001
			KEGG RIBOSOME	< 0.0001
			REACTOME INFLUENZA VIRAL RNA TRANSCRIPTION AND REPLICATION	< 0.0001
		Down	KEGG PROPANOATE METABOLISM	< 0.0001
			REACTOME FORMATION OF FIBRIN CLOT CLOTTING CASCADE	< 0.0001
			REACTOME COMMON PATHWAY	< 0.0001
			KEGG FATTY ACID METABOLISM	< 0.0001
			KEGG BUTANOATE METABOLISM	< 0.0001
	28	Up	None	NA
		Down	REACTOME TRNA AMINOACYLATION	0.066645
			PID PLK1 PATHWAY	0.098824
	90	Up	None	
		Down	REACTOME HEPARAN SULFATE HEPARIN HS GAG METABOLISM	0.026099
			REACTOME A TETRASACCHARIDE LINKER SEQUENCE IS REQUIRED FOR GAG SYNTHESIS	0.036539
			REACTOME HS GAG BIOSYNTHESIS	0.095041
2000	7	Up	REACTOME INFLUENZA VIRAL RNA TRANSCRIPTION AND REPLICATION	< 0.0001
			REACTOME INFLUENZA LIFE CYCLE	< 0.0001
			REACTOME PEPTIDE CHAIN ELONGATION	< 0.0001
			REACTOME TRANSLATION	< 0.0001
			REACTOME SRP DEPENDENT COTRANSLATIONAL PROTEIN TARGETING TO MEMBRANE	< 0.0001
		Down	KEGG VALINE LEUCINE AND ISOLEUCINE DEGRADATION	< 0.0001
			KEGG TRYPTOPHAN METABOLISM	< 0.0001
			KEGG PPAR SIGNALING PATHWAY	< 0.0001
			KEGG FATTY ACID METABOLISM	< 0.0001
			KEGG PROPANOATE METABOLISM	< 0.0001
	28	Up	REACTOME PEPTIDE CHAIN ELONGATION	< 0.0001
			KEGG RIBOSOME	0.0021614
			REACTOME INFLUENZA VIRAL RNA TRANSCRIPTION AND REPLICATION	0.0023879
			REACTOME 3 UTR MEDIATED TRANSLATIONAL REGULATION	0.012228
			REACTOME CELL DEATH SIGNALLING VIA NRAGE NRIF AND NADE	0.014006
		Down	KEGG BIOSYNTHESIS OF UNSATURATED FATTY ACIDS	< 0.0001
			REACTOME FATTY ACYL COA BIOSYNTHESIS	0.020023
			REACTOME POST TRANSLATIONAL PROTEIN MODIFICATION	0.021946
			KEGG STEROID HORMONE BIOSYNTHESIS	0.022519
			REACTOME METABOLISM OF AMINO ACIDS AND DERIVATIVES	0.023419
	90	Up	REACTOME FORMATION OF TUBULIN FOLDING INTERMEDIATES BY CCT TRIC	0.045617
			BIOCARTA P53 PATHWAY	0.059268
			SIG REGULATION OF THE ACTIN CYTOSKELETON BY RHO GTPASES	0.068946
			REACTOME GLUTATHIONE CONJUGATION	0.079748
			REACTOME POST CHAPERONIN TUBULIN FOLDING PATHWAY	0.085827
		Down	REACTOME DEGRADATION OF THE EXTRACELLULAR MATRIX	< 0.0001
			BIOCARTA INTRINSIC PATHWAY	< 0.0001
			KEGG COMPLEMENT AND COAGULATION CASCADES	0.0053303
			REACTOME LIPID DIGESTION MOBILIZATION AND TRANSPORT	0.015464

(*continued on next page*)

Table 2 (*continued*)

1,4-Dioxane (ppm)	Duration (days)	Overall Direction	Gene set	Adjusted p-value
			REACTOME FORMATION OF FIBRIN CLOT CLOTTING CASCADE	0.016814
6000	7	Up	BIOCARTA EIF PATHWAY	< 0.0001
			KEGG RIBOSOME	< 0.0001
			REACTOME PEPTIDE CHAIN ELONGATION	< 0.0001
			REACTOME INFLUENZA VIRAL RNA TRANSCRIPTION AND REPLICATION	< 0.0001
			REACTOME INFLUENZA LIFE CYCLE	< 0.0001
		Down	KEGG COMPLEMENT AND COAGULATION CASCADES	< 0.0001
			BIOCARTA COMP PATHWAY	< 0.0001
			NABA ECM REGULATORS	0.0010723
			REACTOME FORMATION OF FIBRIN CLOT CLOTTING CASCADE	0.0012178
			BIOCARTA CLASSIC PATHWAY	0.0013393
	28	Up	REACTOME GLUTATHIONE CONJUGATION	0.05646
		Down	KEGG COMPLEMENT AND COAGULATION CASCADES	< 0.0001
			KEGG ARGININE AND PROLINE METABOLISM	0.0027586
			NABA ECM REGULATORS	0.0036022
			BIOCARTA INTRINSIC PATHWAY	0.0036782
			REACTOME FORMATION OF FIBRIN CLOT CLOTTING CASCADE	0.004578
	90	Up	PID AURORA B PATHWAY	0.00041562
			REACTOME FORMATION OF TUBULIN FOLDING INTERMEDIATES BY CCT TRIC	0.00083123
			REACTOME GLUTATHIONE CONJUGATION	0.017352
			REACTOME POST CHAPERONIN TUBULIN FOLDING PATHWAY	0.026444
			REACTOME PREFOLDIN MEDIATED TRANSFER OF SUBSTRATE TO CCT TRIC	0.043203
		Down	PID HNF3A PATHWAY	< 0.0001
			KEGG PANTOTHENATE AND COA BIOSYNTHESIS	< 0.0001
			REACTOME LIPID DIGESTION MOBILIZATION AND TRANSPORT	0.0014751

that was enriched at 90 days was also enriched at 7 days (BMD median of 3506 ppm). The histone and ubiquination genes underlying the enrichment of this gene set are involved in DNA synthesis and potentially cell proliferation.

Overall, BMD analysis confirmed the increase in phase II xenobiotic metabolism and a decrease in complement cascade and lipid metabolism pathways that was observed via analysis at each individual dose, as well as a significant increase mitotic cell cycle and cellular proliferation at concentrations above 2000 ppm at 90 days (Fig. 3). The BMD_{1SD} and BMDLs were well above 600 ppm for some pathways that were significant at 600 ppm according to gene set enrichment analysis comparing each dose relative to the controls. This may be explained by the fact that BMD modeling analysis accounts for variability across the whole experiment, as well as the general dose–response curve information, as opposed to specifically comparing one dose group to the time-matched controls.

4. Discussion

Mechanistic data provide important information for human health risk assessment, in particular with respect to providing an understanding of the underlying mode/mechanisms of an adverse outcome. Such mechanistic data can inform the MOA of a chemical via the identification of specific key molecular or cellular events. Specifically, transcriptomic analysis can contribute to understanding drug- or chemical-induced liver toxicity by identifying biomarkers of effect or exposure, expression signatures, and/or changes in signaling (Merrick and Bruno, 2004; Cui and Paules, 2010). The identification of a MOA for a carcinogen is important for the selection of the risk assessment approach under current regulatory paradigms. Specifically, a mutagenic vs. a non-mutagenic MOA have historically been subject to linear low-dose extrapolation vs. a threshold approach, respectively, for risk assessment (U.S. EPA, 2005). In the case of 1,4-dioxane, several groups, including regulatory agencies, have applied a threshold approach (NICNAS, 1998; TNO/RIVM, 1999; Stickney et al., 2003; Health Canada, 2005), while others have applied a non-threshold approach (OEHHA, 2002; U.S. EPA, 2013). Previously, a MOA for rodent liver tumors was hypothesized that included metabolic saturation followed by cytotoxicity-induced regenerative repair (Dourson et al., 2014, 2017). Biomarker analyses and histological examinations were conducted on the same liver tissues discussed herein, and reported by Lafranconi et al (2020). Collectively, these analyses demonstrated saturated metabolism of 1,4-dioxane in mice, as well as increased proliferation following 90 days of oral exposure via drinking water, at concentrations ≥2000 ppm. These results provide additional mechanistic information for 1,4-dioxane, informing potential key events in a MOA for liver cancer in a sensitive strain of mouse. The transcriptomic information also adds insights as to molecular events that explain these biomarker and histopathology findings. Moreover, the transcriptomic analyses serve to identify potential key events for further examination that were not visible with the more conventional histopathological observations.

As described in the *Materials and Methods*, gene expression data from the livers of 1,4-dioxane-exposed mice were analyzed for individual gene changes, gene set enrichment using two different statistical methods, and BMD modeling for individual genes, as well as functional classification of dose-responsive genes. The results demonstrate a generally low response to 1,4-dioxane in the livers of mice and the mRNA level. Overall, gene set enrichment demonstrated up-regulation of phase II metabolism in a dose–response manner. After 90 days of exposure, an increase in cell cycle signaling was evident in the highest concentration treatment group. Changes in individual genes that did not converge into gene set enrichment, and a general loss of signal transduction at the pathway level at the 7-day timepoint likely represents a non-specific adaptive and/or general stress response. Such changes were mitigated after 28 days of exposure, potentially related to the up-regulation of Phase II metabolism and, thus, detoxification. Importantly, transcriptomic profiling conducted to specifically query the enrichment of DNA damage response gene sets demonstrated a lack of DNA damage response at the mRNA level. The few enriched gene sets related to up-regulation of DNA damage response at 6000 ppm (i.e., p53 signaling pathways) according to the more lenient GSEA enrichment analysis and BMD functional classification analysis may be related to apical endpoints reported in Lafranconi et al. (2020): up-regulation of signaling pathways for cell cycle are potentially related to the reported increased BrdU labeling, and enrichment of cell death signaling potentially related to the increase in apoptosis as evi-

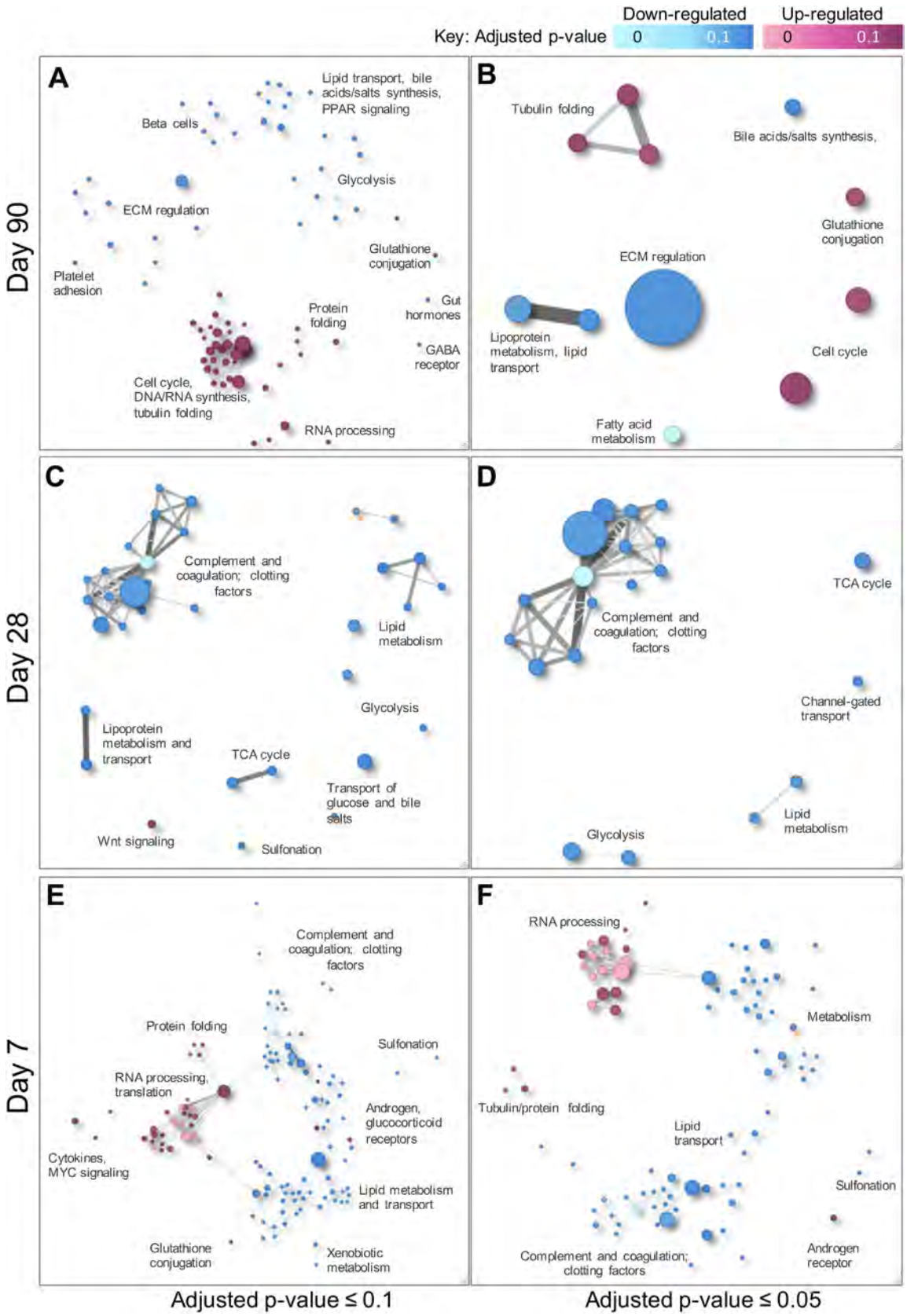

Fig. 2. Network plots showing enriched gene sets at 6000 ppm relative to controls. (A/C/E: adjusted p-value ≤ 0.1, B/D/F: adjust p-value ≤ 0.05). Node size is scaled on number of member genes within the gene set, and node color is scaled according to significance (lighter blue/pink node color indicates more highly significant relevant to darker blue/pink node color). Nodes are spatially organized according to likeness, according to common individual genes within the gene sets. Lines connecting nodes represents common members, with thickness of the line scaled according to number of common gene members. Color of the nodes represents statistical significance as noted in the color bar key. For visualization, general descriptive categories are denoted for gene sets with common genes and, thus, similar functionality, as opposed to listing all actual gene set names. Select individual gene set of highest statistically significant enrichment are shown. Full results for all dose groups and timepoints are in Supplemental Table S2. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Table 3
BMD modeling results for select up-regulated enriched gene sets related to xenobiotic metabolism and cell cycle. Full results are presented in Supplemental Table S7.

Gene set	Exposure Duration (days)	Median BMD_{1SD} (ppm)	Median BMDL (ppm)	Fisher's exact two-tail test p-value
Glutathione Conjugation	7	2305	1819	$7.46x10^{-4}$
	28	1682	1399	$6.75x10^{-5}$
	90	1548	1236	$6.16x10^{-4}$
Phase II - Conjugation of compounds	7	2301	1696	$4.11x10^{-5}$
	28	1903	1401	$9.28x10^{-9}$
	90	1652	1251	$3.84x10^{-2}$
Cell Cycle	7	3521	2333	NS
	28	5455	2523	NS
	90	3874	2243	$4.54x10^{-3}$
Cell Cycle Checkpoints	7	3628	2428	NS
	28	5455	2849	NS
	90	3474	2265	$6.56x10^{-2}$
Cell Cycle, Mitotic	7	3521	2333	NS
	28	3639	2523	$2.56x10^{-2}$
	90	3414	2242	$9.44x10^{-3}$

NS, not significant for enrichment among dose-responsive genes.

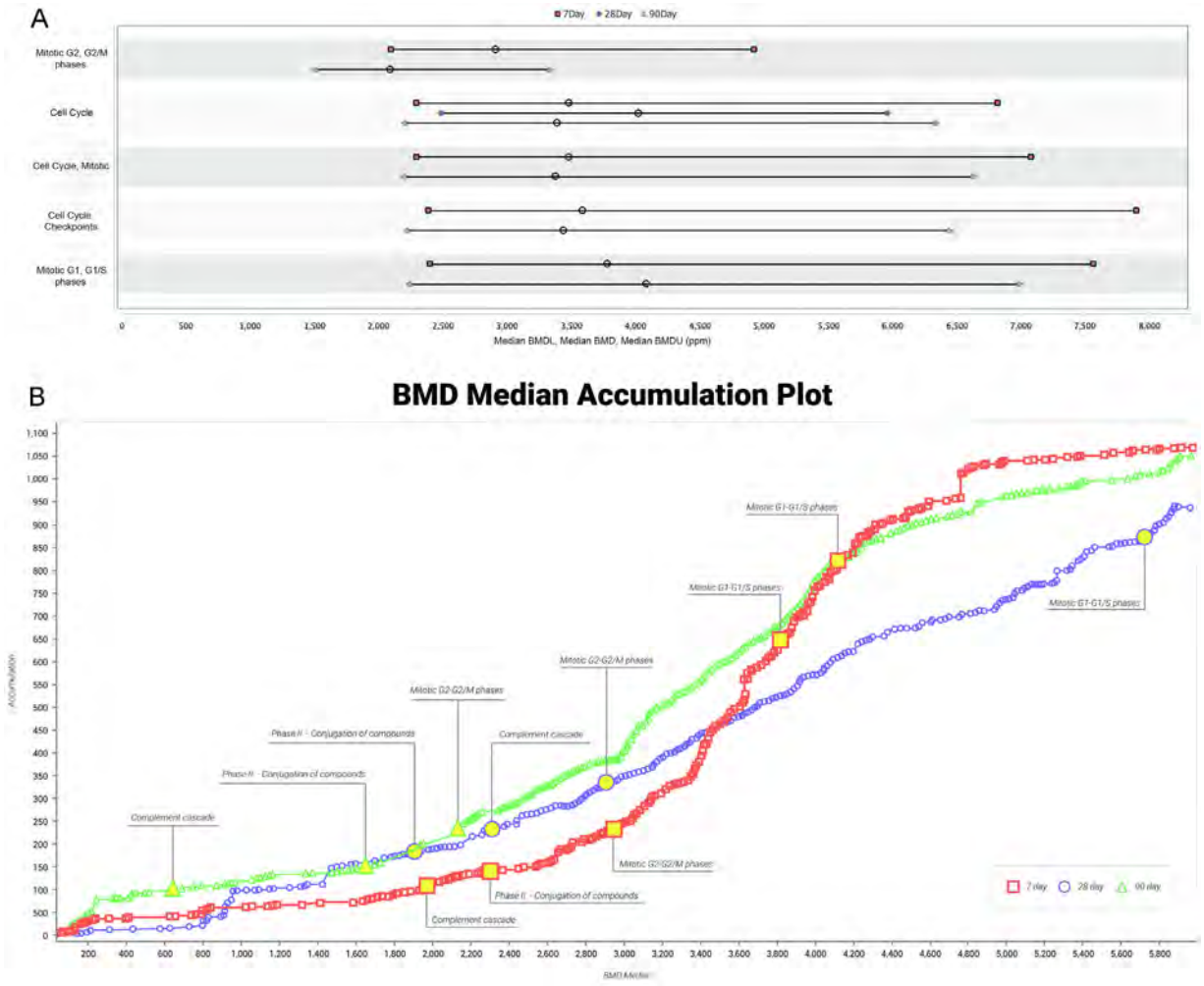

Fig. 3. BMDExpress analysis visualizations. A: Range plots for selected gene sets related to cell cycle. Data are shown for gene sets/timepoints with significant enrichment. B: Accumulation plot for all three timepoints, with select gene sets discussed herein annotated by text.

denced by Caspase 3 staining. While p53 signaling is known to be activated by DNA damage, it also can be activated by non-genotoxicants (Catizone et al., 2019). The individual genes driving the enrichment of p53-relevant pathways in the GSEA and BMDExpress analyses were regulators of apoptosis (e.g., *Bax*) and cytokines, without alteration to DNA repair enzymes nor the p53 gene itself. No individual genes for DNA damage repair enzymes were differentially expressed compared to controls at any dose or timepoint according to the DESeq2 analysis. This indicated that changes in cell cycle occurred in the high concentration group independent of DNA damage. This finding is corroborated by an overall negative profile for 1,4-dioxane in a set of HTS assays within the ToxCast/Tox21 database that are indicators of

DNA damage and/or repair (Hsieh et al., 2019). This finding aligns with the proposed MOA for 1,4-dioxane rodent hepatotoxicity involving cytotoxicity and subsequent regenerative hyperplasia (Dourson et al., 2017), as well as with the mitogenic response reported for the same liver tissues evaluated herein (Lafranconi et al., 2020)

Transcriptomics data can provide important information for proposing potential key events for an alternative MOA or supporting existing key events in established MOAs. Signaling on the molecular level can demonstrate or inform underlying mechanisms of toxicity. Transcriptomic responses following relatively short exposures that are transient may represent a non-specific adaptive and/or stress response (Dean et al., 2017). For example, after 7 days of exposure to 1,4-dioxane in the present study, there were many more DEGs than following 28 days of exposure at the 600 and 6000 ppm concentrations. However, there were very few enriched gene sets at the 7-day timepoint, for any exposure concentration. This indicates that the altered genes are not members of a cohesive signaling pathway and may represent a transient response to the exposure scenario. After 28 days of exposure, the majority of the DEGs at the 7-day timepoint had returned to levels similar to the time-matched controls for the 600 and 6000 ppm groups. While the 2000 ppm group had overall more DEGs at 28 days compared to either 7 and 90 days, most of the DEGs at 7 days were not differentially expressed at 28 days. Following a sub-chronic exposure duration of 90 days, transcriptomic response was increased at the 600 and 6000 ppm concentrations. A 28-day "sub-acute" timepoint has been used to identify liver chemical carcinogenicity signatures in experimental animals (Waters et al., 2003), while 90-day exposures have been suggested to accentuate gene expression changes related to the carcinogenic activity of chemicals (Auerbach et al., 2010). Notably, transcriptomic analysis in target tissue following exposure durations of 14 days or less in *in vivo* models has been shown to be predictive of non-DNA-reactive mechanisms in hepatic tumors (Fielden et al., 2007). In the present study, the transcriptomic profiles at three different exposure durations were absent of a gene expression signal for DNA damage response or repair.

In addition to the pathway level enrichment of phase II metabolism and an increase in mitotic cell cycle at high concentrations and later timepoints, reduced expression of genes involved in coagulation and complement cascade, as well as extra-cellular matrix regulation, was a significant and transient signal in the present study; this signal was normalized at 90 days at all concentrations except for 6000 ppm. Although the significance of this finding is not fully known, downregulation of coagulation cascade proteins in the livers of mice with hyperplasia-mediated liver regeneration has been previously demonstrated (Tatsumi et al., 2009).

Although alterations to nuclear receptors involved in xenobiotic metabolism represents a known molecular initiating event for some cases of chemically-induced hepatotoxicity and/or hepatocarcinogenicity, in particular those with increased proliferation, the only general nuclear receptor gene set included in the analysis presented herein ("BIOCARTA_NUCLEARRS_PATHWAY") was not significantly enriched. Thus, individual CYP-encoding genes that are considered indicators of several common nuclear receptors known to play a role in rodent liver pathogenesis (aryl hydrocarbon receptor [AhR], constitutive androstane receptor [CAR], peroxisome proliferator-activated receptor [PPAR], and pregnane X receptor [PXR]) were reviewed for treatment effect. The CYP-encoding genes were not differentially expressed in any dose group or timepoint, with the exception of the PXR-related *Cyp3a11* (human homolog *CYP3A4,* Li et al., 2009), which was significantly up-regulated at 90 days in the 600 and 6000 ppm dose groups (Supplemental Table S1). The biological plausibility that PXR may be affected by 1,4-dioxane in mouse livers is supported by the fact that PXR regulates phase II conjugating enzymes. However; *Cyp3a11* was only significantly up-regulated at the highest dose at 90 days, while phase II metabolism pathways were up-regulated at early timepoints as well as at the 600 ppm concentration, indicating that the two expression changes may not be dependent upon one another. PXR, among other xenobiotic-metabolizing nuclear receptors, is known to be differentially expressed across species, leading to species-specific liver effects in rodents (Luisier et al., 2014; Yamada et al., 2015). While this result suggests the possibility that 1,4-dioxane exposure affects the PXR, further investigation beyond *Cyp3a11* mRNA level is necessary to confirm such a molecular event.

It should be noted that in the present study, due to the minimal treatment effect of 1,4-dioxane on gene expression at any dose or timepoint, liberal criteria were applied to identify DEGs and enriched signaling pathways. For example, no fold-change criterion was set for the identification of DEGs, and the use of the full complement of genes ranked by the Wald statistic for gene set enrichment rather than filtered by a significance cut-off was the approach emphasized herein (GSEA method as opposed to the hypergeometric test, with the exception of the DNA damage response analysis). These liberal criteria enabled identification of minimally altered genes and signaling networks and demonstrated that changes to signaling pathways were limited. Trends in changes to signaling pathways related to mechanisms of hepatoxicity and/or carcinogenesis were subtle and specific to high dose groups. This highlights the overall low effect of 1,4-dioxane on gene expression in the livers of mice, particularly at concentrations below 600 ppm. The results indicate that the threshold concentration for hepatic transcriptomic response to 1,4-dioxane in female mice, whether it be transient and/or adaptive or related to pathology, exists somewhere in the range of 600–2000 ppm.

In summary, the transcriptomic response in livers of mice exposed to 1,4-dioxane in a drinking water study demonstrates minimal treatment effects on global gene expression at concentrations below 600 ppm, with an increase in phase II metabolism and cellular cycle signaling in the absence of a significant increase in DNA damage response signaling at the mRNA level at 600 ppm and above. These findings align with the phenotypic findings of histopathological and biochemical analysis of the same liver tissues, and support the non-mutagenic, threshold-based mitogenic MOA for mouse liver tumors proposed by Lafranconi et al. (2020) based on all the study findings.

CRediT authorship contribution statement

G.A. Chappell: Conceptualization, Formal analysis, Investigation, Methodology, Project administration, Visualization, Writing - original draft, Writing - review & editing. **M.M. Heintz:** Formal analysis, Writing - original draft. **L.C. Haws:** Funding acquisition, Supervision, Project administration, Writing - review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.crtox.2021.01.003.

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From: Bailey, Sabrina <Sabrina.Bailey@Illinois.gov>
Sent: Wednesday, June 9, 2021 8:33:46 AM
To: Terranova, Sara <Sara.Terranova@Illinois.gov>; Brown, Michael L. <Michael.L.Brown@Illinois.gov>; Dunaway, Lynn <LYNN.DUNAWAY@Illinois.gov>; Frost, Brad <Brad.Frost@Illinois.gov>; Lieberoff, Barb <Barb.Lieberoff@Illinois.gov>; Wake, Elizabeth <Elizabeth.Wake@Illinois.gov>; Guy, Jeff <Jeff.Guy@Illinois.gov>; Nifong, Heather <Heather.Nifong@Illinois.gov>; Diers, Stefanie <Stefanie.Diers@Illinois.gov>; Sofat, Sanjay <Sanjay.Sofat@Illinois.gov>; Ankney, Clayton <Clayton.Ankney@Illinois.gov>; Martin, Lauren <Lauren.Martin2@Illinois.gov>; Hawbaker, Carol <Carol.Hawbaker@Illinois.gov>; Woods, Teschlyn <Teschlyn.Woods@Illinois.gov>; Irlam, Justin <Justin.Irlam@Illinois.gov>; Shaw, Melinda <Melinda.Shaw@illinois.gov>; Wilson, Nicole <Nicole.Wilson@Illinois.gov>; Dunn, Greg <Greg.Dunn@Illinois.gov>; Summers, Michael <Michael.Summers@Illinois.gov>
Subject: Re: 620 Questions and Comments 6/9/21

Good Morning All,
Below are comments from Illinois American Water.

From Rachel Bretz, Director of Water Quality and Environmental Compliance
Organization: Illinois American Water
Comment:

- included PFAS (PFBS, PFHxS, PFNA, PFOA, PFOS) in both Class I and II groundwater limits
- Levels are slightly different than the drinking water HALs they established (Table below)

Acronym		**Health-Based Guidance Level**	Groundwater Quality Standard **Proposed**
		(ng/L)	**(ng/L)**
Perfluorobutanesulfonic acid	PFBS	2,100*	1200
Perfluorohexanesulfonic acid	PFHxS	140	77
Perflurooctanesulfonic acid	PFOS	14	7.7
Perfluorooctanoic acid	PFOA	2	2
Perfluorohexanoic acid	PFHxA	560,000	NONE
PFNA (perfluorononanoic acid)	PFNA	NONE	12

Sabrina Bailey, PhD
Office of Community Relations
Illinois EPA
(847) 294-4394
Sabrina.Bailey@illinois.gov

From: Bailey, Sabrina
Sent: Wednesday, May 26, 2021 11:35 AM
To: Terranova, Sara <Sara.Terranova@Illinois.gov>; Brown, Michael L. <Michael.L.Brown@Illinois.gov>; Dunaway, Lynn <LYNN.DUNAWAY@Illinois.gov>; Frost, Brad <Brad.Frost@Illinois.gov>; Lieberoff, Barb <Barb.Lieberoff@Illinois.gov>; Wake, Elizabeth <Elizabeth.Wake@Illinois.gov>; Guy, Jeff <Jeff.Guy@Illinois.gov>; Nifong, Heather <Heather.Nifong@Illinois.gov>; Diers, Stefanie <Stefanie.Diers@Illinois.gov>; Sofat, Sanjay <Sanjay.Sofat@Illinois.gov>; Ankney, Clayton <Clayton.Ankney@Illinois.gov>; Martin, Lauren <Lauren.Martin2@Illinois.gov>; Hawbaker, Carol <Carol.Hawbaker@Illinois.gov>; Woods, Teschlyn <Teschlyn.Woods@Illinois.gov>; Irlam, Justin <Justin.Irlam@Illinois.gov>; Shaw, Melinda <Melinda.Shaw@illinois.gov>; Wilson, Nicole <Nicole.Wilson@Illinois.gov>; Dunn, Greg <Greg.Dunn@Illinois.gov>; Summers, Michael <Michael.Summers@Illinois.gov>
Subject: 620 Questions and Comments

Good Morning All,
Attached are comments and questions concerning 620 proposed changes. I will send a daily update of the comments in word, and they will be added to an excel spreadsheet that will be updated weekly and shared.

Sabrina Bailey, PhD
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(847) 294-4394
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ANDREWS ENGINEERING
3300 Ginger Creek Drive | 217.787.2334
Springfield, IL 62711

February 28, 2020

Sara Terranova
Assistant Counsel
Division of Legal Counsel
Illinois Environmental Protection Agency
1021 North Grand Avenue East, P.O. Box 19276
Springfield, Il 62794-9276

email:sara.terranova@illinois.gov

re: Comments for 35 Ill. Adm. Code 620 Proposed Revisions

Dear Ms. Terranova:

On behalf of Republic Services, submitted herein are comments pertaining to the proposed revisions to 35 Ill. Adm. Code 620.

Should you have any questions or require additional information, please contact Eric Ballenger at 224-970-1128 or me at 217-787-2334. Thank you.

Sincerely,

Brad J. Hunsberger

Brad J. Hunsberger, LPG
Vice President

BJH:bjh

w/attachments

cc: Peggy Macenas (NWRA) – email
Kenn Liss (Andrews Engineering) - email

PROPOSED REVISIONS TO 35 IAC PART 620

AFFECTS TO THE GROUNDWATER IMPACT ASSESSMENT

The purpose of the Groundwater Impact Assessment is to provide an integrated evaluation of the acceptability of the physical setting and design of the landfill units through contaminant transport modeling. The impacts of leachate seepage from the unit must be addressed (i.e. modeled) in a systematic fashion using the techniques described in 35 IAC 811.317 and 812.316 [Appendix C to LPC-PA2]. The statutory requirements for the GIA are provided in 35 IAC 811.317 for a waste disposal facility complying with the regulations of 35 IAC Part 812 - Subpart C, and Part 814 - Subpart C.

The proposed revisions to the regulations of 35 IAC Part 620 will have a significant effect to the results of the Groundwater Impact Assessment (GIA) process for solid waste disposal units; specifically the proposed addition of Section 620.410(d)(3). The proposed addition states:

1) The concentrations of the following constituents must not be exceeded in Class I groundwater at both the individual standards and a combined standard of 0.000021 mg/L.

CAS No.	*Constituent*	*Standard (mg/L)*
335-67-1	*Perfluorooctanoic Acid (PFOA)*	*0.000021*
1763-23-1	*Perfluorooctane Sulfonic Acid (PFOS)*	*0.000014*

The extremely low proposed standards and relatively non-attenuative properties of the PFOA and PFOS constituents make for a worst-case scenario with respect to an acceptable GIA. The GIA is conducted for all new waste units and is evaluated at least once every five years (35 IAC 813.304) pursuant to the permit renewal process contained in 35 IAC Part 813, Subpart C for existing units. Therefore, all 38 active landfill facilities (2018 Illinois Landfill Disposal Capacity Report) will be economically impacted by this rulemaking.

The parameters listed in 35 IAC 620.410 automatically become part of the GIA process as those are referenced in (at a minimum):

Section 811.315(e)(1)(G)(i) – background concentrations must be established for "*Any constituent for which there is a standard at 35 Ill. Adm. Code 620 established by the Board and which is expected to appear in the leachate, and"*

Section 811.317(a)(2) – "*The concentration of constituents in the leachate shall be determined from actual leachate samples from the waste or similar waste, or laboratory derived extracts."* This regulation infers the 620 parameters via Section 811.315(e)(1)(G)(i).

Section 811.317(a)(3) – *"A contaminant transport model meeting the standards of subsection (c) shall be utilized to estimate the concentrations of the leachate constituents over time and space. The Agency must review a groundwater*

contaminant transport model for acceptance in accordance with 35 Ill. Adm. Code 813.111."

Section 811.320(a)(3(B) – Applicable Groundwater Quality Standards – For the purposes of this Part: "*"Board established standard" is the concentration of a constituent adopted by the Board as a groundwater quality standard adopted by the Board pursuant to Section 14.4 of the Act or Section 8 of the Illinois Groundwater Protection Act."*

There are multiple complexities within the GIA process that arise as part of the subject proposed rule revisions. Those are discussed individually below:

1. Establishment of AGQSs

The GIA through contaminant transport modeling provides predicted model concentrations that are compared to AGQS values derived pursuant to Section 811.320(d). If all predicted model concentrations fall below the AGQS, the GIA is deemed acceptable. However, derivation of accurate AGQSs for PFOA and PFOS constituents will be difficult at best, and may be suspect due to many factors.

Establishment of background concentrations require at least four quarters of good data (the timing and number of sampling intervals may be altered if approved by the Illinois EPA). Good data is dependent upon sampling and testing methods, as well as a monitor well network free of PFOA and PFOS constituents. Sampling methods have to some extent been established. However, many laboratory testing methods are in draft stages and are specific to clean water, not for samples that may contain turbidity, or with respect to leachate - probable matrix interference issues.

Cross contamination from the wells is also a potential due to well construction methods. Illinois EPA documentation (Appendix C to LPC-PA2 (Instructions for the Groundwater Protection Evaluation for Putrescible and Chemical Waste Landfills)) specifically recommended well materials that are known PFAS sources. Section IV.B of Appendix C states:

The application must provide detailed documentation of the monitoring well and piezometer construction. Casing and screen material must be inert to avoid contributing contamination or causing interference with the analysis of the water sample. Teflon, Stainless Steel 316, and Stainless Steel 304 are recommended as durable, corrosion- resistant materials. Since plastic (PVC) may have a significant effect on the ability to obtain a "representative" sample, the Agency only allows the use of plastic casing for piezometers or through the unsaturated zone for wells.

Entire monitor well networks contain pumps with Teflon bladders, gaskets, discharge tubing, and Teflon-coated wire, all in direct contact with the groundwater samples (potential for direct cross contamination). In addition, Teflon seals or tape were commonly used on the threads of the well screens and casings. Packaging for well materials may have contained PFAS, including bags and containers for sand (screen sand pack) and bentonite, cross contaminating the well unaffiliated with the waste unit. Also, the Illinois EPA requires that potable water be used in construction of the wells. Most water supplies for well installation and equipment decontamination are obtained from city supply lines or bulk stations that may contain PFAS compounds. Potable water sources will need to be located that can be certified free of PFAS,

otherwise, any well installed may be cross contaminated by the potable water supply. The source of low level PFAS concentrations may never be identified with the potential for cross contamination from numerous sources. It is unreasonable to assume that all wells will need to be replaced that show low level PFAS contamination because of potential cross contamination when the well was installed pursuant to IEPA guidelines. For the installation of new wells, testing may be necessary throughout each phase of installation. This would include the potable water supply, the drilling contractor equipment (including water tanks, lines, hoses, and pumps), and well materials.

Upon approval and implementation of the proposed rules, it will likely be difficult to identify the source of PFOA and PFOS constituents if detected in any well, upgradient or downgradient. More time and effort will be spent trying to validate the data such that it is useable and meaningful. Alternate sources will be evaluated as part of this process, which will require significant additional time. If the AGQS values are suspect, the GIA process may be of little to no use for the PFOA and PFOS constituents.

2. Source Concentration

The source concentration is probably the single most important model input parameter. A high source concentration for particularly sensitive parameters (largely non-attenuative) such as ammonia, chloride, or boron, normally result in initial failure of the GIA baseline model. Pursuant to Section 811.317(a)(2), leachate samples from the applicable waste units will require analyses for PFOA and PFOS constituents once the rule revisions are approved. The constituents will be utilized as source concentrations for the contaminant transport model, resulting in a predicted model concentration used to determine if the GIA is acceptable.

Analyses of the subject parameters in the leachate will be difficult due to probable matrix interference. This will likely increase the Practical Quantitation Limit (PQL), which can artificially increase the source concentration resulting in a higher predicted model concentration and likely resulting in failing model results. Laboratory analytical methods have not been advanced sufficiently to provide accurate results from a leachate matrix.

The source concentration must be accurate. Similar cross contamination issues described above apply to obtaining a representative leachate sample. The leachate collection system within a modern waste unit consists of collection and conveyance lines, sealing materials, and numerous pump systems that can contribute PFOA and PFOS constituents to the leachate samples. Detection of low level concentrations in the leachate will be suspect and the source concentration likely inaccurate. Cross contamination of PFAS may be sufficient to cause failure of the GIA, or failure of the original assumptions of the GIA in the case of a permit renewal application.

The Illinois EPA Bureau of Land should revise the guidance document (LPC-PA2) or create a new document to standardize sample retrieval and testing methods for leachate.

3. Potential Design Changes

Each operational landfill and many closed waste units (35 IAC Part 814, Subpart C) maintain approved GIAs. Pursuant to 35 IAC 813.304, the GIA must be re-evaluated at least every five years (permit renewal process) or sooner if changes to the facility or its operations would

result in an increased probability of exceeding a groundwater quality standard beyond the zone of attenuation.

The GIA of record for any facility was completed utilizing site specific data (hydrogeologic and leachate analyses) or as otherwise approved by the Illinois EPA as being representative of the facility setting. In many cases, the initial baseline model runs for the new waste units were borderline or even failed. To address those, design changes were incorporated to include thicker liner systems, revision the slope and leachate collection system to reduce the leachate head (seepage rate), revision to the liner system placement within the hydrogeologic setting (relocate the liner elevations to provide additional in-situ low hydraulic conductivity deposits between the liner invert and uppermost aquifer), and/or revision to the final cover system design to decrease the precipitation infiltration into the waste unit. The model also incorporated partitioning coefficients for specific surrogate groups which aided in reduction of the predicted model concentrations for typically problematic constituents, resulting in an acceptable model.

Regulatory constraints and guidance for the contaminant transport models have been largely consistent since the mid to late 1990s. Design and cell construction have been permitted for all active facilities, as well as final closure for many waste units. The final cover systems were designed based on HELP modeling which was used to determine seepage rate for the input to the contaminant transport model.

The addition of PFOA and PFOS constituents through the 35 IAC 620 rule revisions has the potential to cause failure of many permitted GIAs which are acceptable under the current requirements. It would have been possible during the initial design stage to address results of the PFOA and PFOS constituents through design changes. However, the potential for design changes to existing waste units are very limited, with only the final cover system realistically remaining for redesign to lower infiltration to the waste unit during post closure, thus possibly reducing the leachate head on the liner system.

Design changes for future cells (already permitted) yet to be constructed may be necessary if the results of the contaminant transport model fail due to the addition of the PFOA and PFOS constituents. However, this will be highly dependent upon the geologic setting and may be restricted by the local siting resolution pursuant to Section 39.2 of the Act. If the Illinois EPA is to go forward with the revisions as proposed, a mechanism needs to be created allowing existing facilities a way to address GIA failures without automatically reverting to a contingent remediation program.

4. Appropriate Contaminant Transport Models

The GIA is a determination of the time and distance dependent potential impact of a landfill unit on local groundwater chemistry. The GIA is based on a site-specific solute transport model of the actual design, site-specific hydrogeology, and conservative performance standards for the liner system, leachate management system and final cover system. The GIA is considered acceptable if the groundwater contaminant transport model predicts that the concentrations of all leachate constituents outside of the zone of attenuation are less than the Applicable Groundwater Quality Standards (AGQS) of 35 IAC 811.320 within 100 years of closure of the unit.

Typically contaminant transport models associated with the GIA have been generally simplistic, being one- and/or two-dimensional, such as POLLUTE and MIGRATE. The conceptual model assumes:

- all geologic units and soil liners are homogeneous and isotropic with respect to all lithologic and hydrologic parameters,
- that all layers are laterally extensive and the thickness of each layer is uniform,
- all layers are fully saturated,
- the external stresses on the system are constant through time,
- the source concentration is constant over the entire modeling period, and
- baseline surrogates were prepared in which no retardation or decay occurs.

Allowing the use of more reasonable model parameters would help reduce the model prediction factor and increase the probability of an acceptable model. The model input parameters are typically the most conservative across the board. When combined with conservative parameters for use in the HELP modeling, the end result is an ultraconservative model where surrogate groups are often needed to achieve an acceptable model. This would be a policy change for the Bureau of Land, not a regulatory change.

Under fully saturated conditions (bottom of the liner system to the bottom of the upper most aquifer), the models utilized for the approved GIAs are likely adequate for evaluation of the PFOA and PFOS constituents. However, settings where unsaturated conditions exist or a vadose zone exists beneath the liner system, a more complex model would better simulate transport of the PFOA and PFOS constituents as transport through such deposits are significantly less. Recent studies have shown PFOA and PFOS constituents are substantially retained in unsaturated deposits via solid phase adsorption, and also at the air-water interface. Differing models may simulate this characteristic better than the typical one- and two-dimensional models used for previous GIAs. Most contaminant transport models are incapable of working with the small-scale changes for these parameters that are seen within many geologic materials. The introduction of other contaminant transport models to deal specifically with the PFOA and PFOS constituents will be costly and time consuming not only for the facility but for review purposes by the Bureau of Land's Permit Section.

5. Bureau of Land Guidance

Even though the proposed 620 rule changes are being driven by the Bureau of Water, ramifications to the Bureau of Land programs are paramount. Prior to sending the proposed rule changes to the Illinois Pollution Control Board, the Bureau of Land should vet the potential ramifications to the regulations of 35 IAC Parts 811-815. The Bureau of Land should then provide a draft update to Appendix C to LPC-PA2 (Instructions for the Groundwater Protection Evaluation for Putrescible and Chemical Waste Landfills) for review and comment by the waste disposal industry. The Illinois EPA has provided two revisions to Appendix C based on what was learned over time during the permitting process. It is reasonable to expect the Bureau of Land should do the same with respect to implications to existing solid waste disposal facilities for revision of the 620 rules. Topics that should be addressed include but are not limited to:

a. Legacy impacts (cross contamination) to groundwater quality – what if the wells already exhibit PFOA and PFOS concentrations in excess of the proposed standards
    i. Well construction issues
    ii. Pump materials
    iii. Impacts to AGQS determination
b. Sampling protocols for groundwater and leachate
c. Laboratory analyses test methodology and limitations – how can "draft" methods be placed into a state regulations
d. GIA – It is a tool
    i. Computer models – provide insight on potential other models for use
    ii. Input parameters
        - Use of more realistic values versus overly conservative values
        - Use of averages or statistical derivations, not the maximum or minimum
        - Update Attachment 1 to Appendix C to include PFOS and PFOA constituents
    iii. Surrogate Modeling for PFOA and PFOS constituents
        - Retardation allowances
        - Sensitivity analyses constraints
e. Use of contingent remediation programs to address predicted exceedences
f. Permitted Contingent Remediation Plans – will all of these need to be re-evaluated with the inclusion of PFOA and PFOS constituents
g. Impacts to permitted waste units in corrective action (35 IAC 807 and 814 Subpart C and D)
h. Impacts to permitted waste units conducting corrective action pursuant to consent orders and/or in conjunction with the US EPA or other entities
i. Sites finishing post closure care (the Affidavit for Certification of Completion of Post-Closure Care has been submitted) – will PFOA and PFOS constituents require analyses prior to release
j. Reasonable dates and timelines for implementation
k. Regulatory exclusion – if the proposed rules are passed, while a facility evaluates its water and leachate quality, the Illinois EPA must provide temporary exclusion from Section 18 of the Act, or others that may apply

Once a new standard is promulgated in Part 620, it is then incorporated into the relevant programs administered by the Bureau of Land. As described above, the process to evaluate potential contaminants are imposed through permits issued by the Bureau of Land. The mere detection of the PFOA and PFOS constituents at a landfill monitor well requires the owner/operator to disprove the potential of a release to the environment. Considering the current body of scientific knowledge, facilities will likely be thrust into the environmental investigation process. That process leads to corrective action. No economic impact study has been conducted to evaluate the cost or the value of expending resources on this path.

The next public meeting should include members from the Bureau of Land prepared to discuss implications to the existing permitted landfill facilities. These issues should be considered prior to submittal of the proposed rule revisions to the Illinois Pollution Control Board for approval.

Illinois Association of Wastewater Agencies
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February 28, 2020

Stephanie Flowers, Part 620
Illinois Environmental Protection Agency
1021 North Grand Ave. East
P.O. Box 19276
Springfield, IL 62794-9276

RE: Comments on Proposed Changes to 35 Ill. Admin. Code 620: Groundwater Quality

Dear Ms. Flowers:

The Illinois Association of Wastewater Agencies (IAWA) is pleased to provide comments on the *Proposed Changes to the Language of 35 Ill. Admin. Code 620: Groundwater Quality*, as provided through a public outreach letter dated December 24, 2019 and a Public Hearing on the proposed changes IEPA conducted on February 13, 2020. The IAWA specific interest is on Item 1) a. regarding the proposed addition of standards for perflouoroalkyl substances (PFAS). The proposed changes include standards for five PFAS compounds for Class I (potable resource) and Class II (general resource) groundwater: Perfluorooctanoic Acid (PFOA), Perfluorooctane Sulfonic Acid (PFOS), Perfluorobutane Sulfonic Acid (PFBS), Perfluorohexane Sulfonic Acid (PFHxS), and Perfluorononanoic Acid (PFNA). The IAWA specific comments are as follows:

1. **The distribution of the notice of the proposed changes was too limited**

   The IEPA distributed the notice to a limited list of stakeholders. Apparently, none of the wastewater treatment utilities that are members of IAWA were included in this list of stakeholders. Therefore, IAWA or its members received the notice only about halfway through the comment period. We would like the agency to include IAWA on future notifications list pertaining to any regulatory changes related to PFAS.

2. **The IEPA should wait until toxicological data becomes available through the sources identified as highest priority**

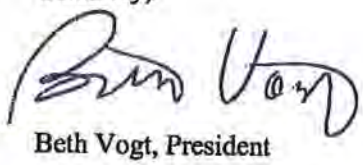
   At the February 13, 2020 Public Hearing on the proposed standards, IEPA clarified that the Agency's methodology follows the approach outlined in Part 620 Subpart F, Appendix A and the guidance outlined in USEPA OSWER Directive 9285.7-53, dated December 5, 2003 on hierarchy for the selection of toxicity values. In this Directive, the USEPA's Integrated Risk Information System (IRIS) is listed as the highest priority for toxicity values. However,

IEPA indicated that toxicity values used to develop the draft standards for PFOA, PFOS, PFNA and PFHxS were derived from Agency for Toxic Substances and Disease Registry (ATSDR) draft toxicological profiles for these compounds. We note that the draft ATSDR profiles were submitted to the Office of Management and Budget for review and is not yet finalized. However, the ATSDR profile is listed as a low priority source (Tier 3) in the USEPA OSWER Directive 9285.7-53 hierarchy guidance. In addition, IEPA also indicated that the values for PFBS were derived from USEPA's Provisional Peer Reviewed Toxicity Values (PPRTV), which is identified as a Tier 3 source.

At the February 13, 2020 Hearing, in response to a question about the implementation of the proposed rule changes, IEPA indicated that the Agency has no schedule for implementation of the proposed standards. Since there is no current schedule for implementation of the proposed standards, it is prudent that IEPA wait until USEPA develop Maximum Contaminant Level Goals for these compounds or until further reviews are completed and the toxicity values for the compounds are available in the IRIS database (highest priority source). The Agency's approach of proposing new standards for PFAS compounds in advance of USEPA's standards or higher priority toxicological values unnecessarily adds to the wide variation of PFAS standards that are being established by different states throughout the US. In addition, although IEPA indicated that it has the option to revise the standards when new information becomes available, this premature action without the need for urgency will cause IEPA to use additional resources unnecessarily, thus contributing to inefficiencies in developing regulations.

Based on the inconsistency outlined above, IAWA contends that the approach used to develop the draft standards for PFAS compounds is unreasonable. The IAWA recommends that the Agency wait until more thoroughly reviewed higher priority toxicological data become available such that groundwater quality standards for PFAS compounds are developed using technically sound data and in concert with USEPA established procedures. This will avoid unreasonable impacts on the wastewater utilities since these new standards might be also used as a basis for establishing future wastewater treatment plant effluent discharge limits for PFAS compounds and land application rates of biosolids in Illinois.

Sincerely,



Beth Vogt, President

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February 28, 2020

Via Email: sara.terranova@illinois.gov

Ms. Sara G. Terranova
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Illinois Environmental Protection Agency
1021 North Grand Avenue East
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Springfield, IL 62794-9276

Re: Comments Regarding Amendments To 35 Ill.Adm.Code 620: Groundwater Quality

Dear Ms. Terranova:

This firm represents the Illinois Chapter of The National Waste & Recycling Association (NWRA). As you know, NWRA submitted comments to the Illinois Environmental Protection Agency on the above-referenced amendments on February 10, 2020. In addition, several members of NWRA attended the stakeholder session on February 13, 2020.

The following members of NWRA have prepared supplemental comments, which are attached:

- PDC Technical Services, Inc.
- Andrews Engineering (on behalf of Republic Services)
- Millennium Waste Incorporated
- Waste Management

Please be advised that the membership of the Illinois Chapter of NWRA endorses and adopts the comments that accompany this letter.

If you have any questions regarding this submission, please contact me.

Sincerely,

James M. Morphew

James M. Morphew
JMM/jf
Attachments

4727166 2/28/2020

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February 27, 2020

Sara Terranova, Part 620
Illinois Environmental Protection Agency
1021 North Grand Avenue East
P.O. Box 19276
Springfield, IL 62794-9276

Dear Sara:

We are pleased to submit our enclosed comments regarding the proposed changes to the language of 35 Ill. Adm. Code 620: Groundwater Quality. If you have any questions regarding our comments please feel free to contact us. We appreciate the Agency allowing us the opportunity to comment on the proposed changes and we look forward to the next step in the rulemaking process.

Sincerely,

George L. Armstrong P.E.
Vice President—Engineering and Consulting Services

Charles Hostetler, Ph.D.
Director of Environmental Services

Enclosure: PDC Technical Services, Inc. Comments

PDC Technical Services, Inc.'s comments on the draft Part 620, dated 12-19-2019, are as follows:

Section 620.410 QWQS for Class I: Potable Resource Groundwater and Section 620.420 GWQS for Class II: General Resource Groundwater

1. Proposed PFAS standards will affect existing practices and procedures used by the solid waste industry

   In the state of Illinois, Municipal Solid Waste Landfills (i.e. landfills regulated under 35 IAC 811) must complete a Groundwater Impact Assessment (GIA) prior to initial permitting to demonstrate that the landfill will have no effect on groundwater quality for a period extending 100 years following landfill closure. The IEPA requires that the GIA models used to permit landfills assume that the landfill has a defective liner system. A key component of GIAs is the concentration of each constituent in leachate. The initial GIA is based on assumed concentrations of a long list of pollutants in leachate. Actual leachate concentrations are reviewed as part of each 5-year permit renewal application and, if they are greater than assumed in the initial GIA, additional modeling or computations are required. It has been reported that PFAS in landfill leachate have been detected at levels greater than 3,500 ppt (Lang, et al. 2017). Considering the proposed PFAS standards, and the anticipated high concentrations of PFAS in leachate, until the PFAS compound fate and transport mechanisms are better understood, we have concern whether or not any landfill GIA would pass under the assumption of a defective liner system, or if the typical models used for GIAs are stable to the proposed concentrations. Further, it is reasonable to assume high laboratory reporting limits of PFAS in leachate due to analytical (matrix) interferences. Industry practice is to assume that leachate parameters that were not detected are present at the reporting limit. Will GIA models demonstrate satisfactory results if the high reporting limits are used, or only at relatively low PFAS concentrations?

   Illinois is the only state in the United States which requires a GIA in a landfill's siting, initial operating permit, and permit renewal application processes. Therefore, the concerns described above are unique to the solid waste industry in the state. It is acknowledged that the proposed PFAS standards are not as conservative as those promulgated by some state agencies (e.g., Michigan, New Jersey); however, it is unclear if the agency accounted for the state-specific requirements and the implementability of the proposed standards for the solid waste industry.

Older, closed sanitary landfills are regulated under 35 IAC 807. Many of these landfills are owned by municipalities, and are nearing the end of their post-closure care period. Prior to being released from post-closure care, the IEPA Bureau of Land requires that the groundwater monitoring wells be sampled and analyzed for all constituents for which a groundwater quality standard has been established at 35 IAC 620. Considering the very low concentration standards that are being proposed and the ubiquitous nature of PFAS compounds, detection of PFAS at concentrations greater than the 620 standards will likely result in significant additional costs to these legacy landfill owners, even after years of satisfactory groundwater monitoring results.

2. There is significant uncertainty associated with the environmental health risks associated with PFAS compounds and, in particular Acceptable Daily Exposure (ADE) values used in calculating the Human Threshold Toxicant Advisory Concentration (HTTAC), as described in 35 Ill. Adm. Code 620, Appendix A.

The Centers for Disease Control and Prevention states "The human health effects from exposure to low environmental levels of PFOA are unknown....More research is needed to assess the human health effects of exposure to PFOA" (https://www.cdc.gov/biomonitoring/PFOA_FactSheet.html, downloaded 2/14/2020). Similarly, the National Institute of Environmental Health Sciences states "More research is needed to fully understand all sources of exposure, and if and how they cause health problems", "The research conducted to date reveals possible (emphasis added) links between human exposures to PFAS and adverse health outcomes.", and "While knowledge about the potential health effects of PFAS has grown, many questions remain unanswered" (https://www.niehs.nih.gov/health/topics/agents/pfc/index.cfm, accessed February 19, 2020).

The available research regarding exposure to PFAS has created a large disparity in the federal and state advisory levels promulgated by governing agencies. The variation is largely related to the different definitions of critical health effects and relative scarcity of human and mammalian studies. Human health studies are largely limited to populations of individuals who 1) have been occupationally exposed during the production or use of PFAS, 2) live in a community with high levels of PFAS measured in drinking water, or 3) have been exposed to background levels of PFAS. Mammalian studies are limited due to the difficulty of extrapolating results from a small animal population provided a controlled exposure dose to the human population in an uncontrolled environment. Further, the mammalian studies which have been conducted have not consistently defined the same critical health effects, making it more difficult to accurately determine an ADE value.The uncertainty associated with ADE values can dramatically shift groundwater standards. IEPA should review the endpoints of reference doses and critical health effects in available literature to determine the magnitude of differences between ADE values.

3. PFAS cleanup objectives are not provided as part of the proposed standards

IEPA does not provide cleanup objectives with the proposed standards. In an instance where PFAS compounds are detected in groundwater at a landfill, what are the expectations for corrective action? Will acceptable background concentrations be considered if PFAS is detectable in upgradient locations?

4. There has been an apparent lack of due process in the establishment of the proposed groundwater standards

It is not immediately apparent if peer reviews have been conducted on the proposed groundwater standards. If not, it should be considered imperative that IEPA conduct a peer review of their proposed standards to ensure that the Agency's standard development procedure is consistent with other regulatory agencies.

5. IEPA should consider the ubiquitous extent of PFAS compounds in groundwater and complete a statewide groundwater survey prior to promulgating regulatory standards

Considering the apparent extent of PFAS in the environment, it is possible that non-attributable concentrations of PFAS compounds will be detected in groundwater upgradient and downgradient of landfills. The state of Illinois is currently conducting a statewide survey of drinking water systems. It is reasonable to conduct a statewide groundwater survey, similar in scope, to determine areas of known PFAS contamination. Development of such a database would provide data to support background analyses and support alternate source demonstrations in scenarios where detectable PFAS concentrations are not attributable to the landfill. At a minimum, IEPA should clarify how the presence of PFAS will be handled in situations not commonly associated with landfill operations (i.e., detectable concentrations in upgradient monitoring wells).

6. There is currently a lack of established analytical methods for more complex leachate, soil and groundwater matrices.

Sampling and laboratory analysis methods have not been established for more complex leachate, soil and groundwater matrices. Laboratories must rely on their own modified analytical methods for analyzing these matrices. Modified methods vary from laboratory to laboratory. As of the date of this submittal, the solid waste industry is waiting on SW-486 method 8328 and Office of Water method 1600 to be issued by the USEPA. Inconsistent results between laboratories could result in analytical results that are not reproducible or defensible.

7. Drinking water standards should be promulgated before groundwater standards.

The purpose of the Class I (Potable Resource) Groundwater Standards is to protect drinking water supplies. The IEPA should not propose Class I Groundwater Standards until after drinking water standards are established. Additionally, the proposed Class I Groundwater Standards are based on concentrations in water that is consumed, and does not factor the probability of whether or not groundwater classified as Class I at any one location will ever be consumed as drinking water, nor does it factor contaminant fate and transport mechanisms. This is overly conservative considering that the vast majority of groundwater that is classified as Class I will never be used for drinking water.

8. Class II Groundwater is not used as a source of drinking water.

The proposed Class II (General Resource) Groundwater Standards are identical to the proposed Class I (Potable Resource) Groundwater Standards. Class II Groundwater is generally not suitable for a drinking water supply. Class II Groundwater Standards should not be based on direct consumption, but rather should be based on protecting other drinking water supplies considering location and fate and transport mechanisms.

9. All regulated landfills in the State must either routinely test for all parameters for which 620 standards are established, or will be required to test for them prior to ending post-closure care.

   Considering the apparent ubiquitous extent of PFAS compounds, it is probable that PFAS compounds will be detected in groundwater upgradient and downgradient of landfills. Because it is a VOC, landfill gas could not automatically be ruled out as a contributor of PFAS in groundwater upgradient of a landfill. As a result, it could be a very expensive and lengthy process to demonstrate that the landfill is not the source of PFAS compounds in groundwater that will never be ingested.

10. IEPA prescribed groundwater monitoring device construction and practices may not be compatible with obtaining representative groundwater quality data consistent with the proposed standards.

    Many of the dedicated groundwater monitoring well sampling bailers and pumps that are currently in use were likely manufactured with PFAS-containing compounds, specifically Teflon. In some cases, Teflon well casing might also have been used, and/or other well construction materials might have inadvertently contained PFAS compounds. It is possible that PFAS compounds from this equipment could have leached into groundwater making it difficult to distinguish the source of extremely low concentrations of PFAS compounds. Additionally, it would be very costly to replace all dedicated sampling pumps, and possibly groundwater monitoring wells themselves, using equipment and supplies that can be certified free of PFAS compounds.

11. PFAS-containing waste acceptance criteria are little understood.

    A better understanding of which wastestreams exhibit high concentrations of PFAS compounds (e.g. remediation wastes, municipal and industrial wastewater sludges, etc.) is needed before imposition of the groundwater standards. Unduly stringent groundwater standards could create an inappropriate lack of disposal capacity for such wastes.

ANDREWS ENGINEERING

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February 28, 2020

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re: Comments for 35 Ill. Adm. Code 620 Proposed Revisions

Dear Ms. Terranova:

On behalf of Republic Services, submitted herein are comments pertaining to the proposed revisions to 35 Ill. Adm. Code 620.

Should you have any questions or require additional information, please contact Eric Ballenger at 224-970-1128 or me at 217-787-2334. Thank you.

Sincerely,

Brad J. Hunsberger

Brad J. Hunsberger, LPG
Vice President

BJH:bjh

w/attachments

cc: Peggy Macenas (NWRA) – email
Kenn Liss (Andrews Engineering) - email

PROPOSED REVISIONS TO 35 IAC PART 620

AFFECTS TO THE GROUNDWATER IMPACT ASSESSMENT

The purpose of the Groundwater Impact Assessment is to provide an integrated evaluation of the acceptability of the physical setting and design of the landfill units through contaminant transport modeling. The impacts of leachate seepage from the unit must be addressed (i.e. modeled) in a systematic fashion using the techniques described in 35 IAC 811.317 and 812.316 [Appendix C to LPC-PA2]. The statutory requirements for the GIA are provided in 35 IAC 811.317 for a waste disposal facility complying with the regulations of 35 IAC Part 812 - Subpart C, and Part 814 - Subpart C.

The proposed revisions to the regulations of 35 IAC Part 620 will have a significant effect to the results of the Groundwater Impact Assessment (GIA) process for solid waste disposal units; specifically the proposed addition of Section 620.410(d)(3). The proposed addition states:

> *1) The concentrations of the following constituents must not be exceeded in Class I groundwater at both the individual standards and a combined standard of 0.000021 mg/L.*
>
> | *CAS No.* | *Constituent* | *Standard (mg/L)* |
> |---|---|---|
> | *335-67-1* | *Perfluorooctanoic Acid (PFOA)* | *0.000021* |
> | *1763-23-1* | *Perfluorooctane Sulfonic Acid (PFOS)* | *0.000014* |

The extremely low proposed standards and relatively non-attenuative properties of the PFOA and PFOS constituents make for a worst-case scenario with respect to an acceptable GIA. The GIA is conducted for all new waste units and is evaluated at least once every five years (35 IAC 813.304) pursuant to the permit renewal process contained in 35 IAC Part 813, Subpart C for existing units. Therefore, all 38 active landfill facilities (2018 Illinois Landfill Disposal Capacity Report) will be economically impacted by this rulemaking.

The parameters listed in 35 IAC 620.410 automatically become part of the GIA process as those are referenced in (at a minimum):

Section 811.315(e)(1)(G)(i) – background concentrations must be established for "*Any constituent for which there is a standard at 35 Ill. Adm. Code 620 established by the Board and which is expected to appear in the leachate, and*"

Section 811.317(a)(2) – "*The concentration of constituents in the leachate shall be determined from actual leachate samples from the waste or similar waste, or laboratory derived extracts.*" This regulation infers the 620 parameters via Section 811.315(e)(1)(G)(i).

Section 811.317(a)(3) – *"A contaminant transport model meeting the standards of subsection (c) shall be utilized to estimate the concentrations of the leachate constituents over time and space. The Agency must review a groundwater*

contaminant transport model for acceptance in accordance with 35 Ill. Adm. Code 813.111."

Section 811.320(a)(3(B) – Applicable Groundwater Quality Standards – For the purposes of this Part: ""*Board established standard" is the concentration of a constituent adopted by the Board as a groundwater quality standard adopted by the Board pursuant to Section 14.4 of the Act or Section 8 of the Illinois Groundwater Protection Act."*

There are multiple complexities within the GIA process that arise as part of the subject proposed rule revisions. Those are discussed individually below:

1. Establishment of AGQSs

The GIA through contaminant transport modeling provides predicted model concentrations that are compared to AGQS values derived pursuant to Section 811.320(d). If all predicted model concentrations fall below the AGQS, the GIA is deemed acceptable. However, derivation of accurate AGQSs for PFOA and PFOS constituents will be difficult at best, and may be suspect due to many factors.

Establishment of background concentrations require at least four quarters of good data (the timing and number of sampling intervals may be altered if approved by the Illinois EPA). Good data is dependent upon sampling and testing methods, as well as a monitor well network free of PFOA and PFOS constituents. Sampling methods have to some extent been established. However, many laboratory testing methods are in draft stages and are specific to clean water, not for samples that may contain turbidity, or with respect to leachate - probable matrix interference issues.

Cross contamination from the wells is also a potential due to well construction methods. Illinois EPA documentation (Appendix C to LPC-PA2 (Instructions for the Groundwater Protection Evaluation for Putrescible and Chemical Waste Landfills)) specifically recommended well materials that are known PFAS sources. Section IV.B of Appendix C states:

The application must provide detailed documentation of the monitoring well and piezometer construction. Casing and screen material must be inert to avoid contributing contamination or causing interference with the analysis of the water sample. Teflon, Stainless Steel 316, and Stainless Steel 304 are recommended as durable, corrosion- resistant materials. Since plastic (PVC) may have a significant effect on the ability to obtain a "representative" sample, the Agency only allows the use of plastic casing for piezometers or through the unsaturated zone for wells.

Entire monitor well networks contain pumps with Teflon bladders, gaskets, discharge tubing, and Teflon-coated wire, all in direct contact with the groundwater samples (potential for direct cross contamination). In addition, Teflon seals or tape were commonly used on the threads of the well screens and casings. Packaging for well materials may have contained PFAS, including bags and containers for sand (screen sand pack) and bentonite, cross contaminating the well unaffiliated with the waste unit. Also, the Illinois EPA requires that potable water be used in construction of the wells. Most water supplies for well installation and equipment decontamination are obtained from city supply lines or bulk stations that may contain PFAS compounds. Potable water sources will need to be located that can be certified free of PFAS,

otherwise, any well installed may be cross contaminated by the potable water supply. The source of low level PFAS concentrations may never be identified with the potential for cross contamination from numerous sources. It is unreasonable to assume that all wells will need to be replaced that show low level PFAS contamination because of potential cross contamination when the well was installed pursuant to IEPA guidelines. For the installation of new wells, testing may be necessary throughout each phase of installation. This would include the potable water supply, the drilling contractor equipment (including water tanks, lines, hoses, and pumps), and well materials.

Upon approval and implementation of the proposed rules, it will likely be difficult to identify the source of PFOA and PFOS constituents if detected in any well, upgradient or downgradient. More time and effort will be spent trying to validate the data such that it is useable and meaningful. Alternate sources will be evaluated as part of this process, which will require significant additional time. If the AGQS values are suspect, the GIA process may be of little to no use for the PFOA and PFOS constituents.

2. Source Concentration

The source concentration is probably the single most important model input parameter. A high source concentration for particularly sensitive parameters (largely non-attenuative) such as ammonia, chloride, or boron, normally result in initial failure of the GIA baseline model. Pursuant to Section 811.317(a)(2), leachate samples from the applicable waste units will require analyses for PFOA and PFOS constituents once the rule revisions are approved. The constituents will be utilized as source concentrations for the contaminant transport model, resulting in a predicted model concentration used to determine if the GIA is acceptable.

Analyses of the subject parameters in the leachate will be difficult due to probable matrix interference. This will likely increase the Practical Quantitation Limit (PQL), which can artificially increase the source concentration resulting in a higher predicted model concentration and likely resulting in failing model results. Laboratory analytical methods have not been advanced sufficiently to provide accurate results from a leachate matrix.

The source concentration must be accurate. Similar cross contamination issues described above apply to obtaining a representative leachate sample. The leachate collection system within a modern waste unit consists of collection and conveyance lines, sealing materials, and numerous pump systems that can contribute PFOA and PFOS constituents to the leachate samples. Detection of low level concentrations in the leachate will be suspect and the source concentration likely inaccurate. Cross contamination of PFAS may be sufficient to cause failure of the GIA, or failure of the original assumptions of the GIA in the case of a permit renewal application.

The Illinois EPA Bureau of Land should revise the guidance document (LPC-PA2) or create a new document to standardize sample retrieval and testing methods for leachate.

3. Potential Design Changes

Each operational landfill and many closed waste units (35 IAC Part 814, Subpart C) maintain approved GIAs. Pursuant to 35 IAC 813.304, the GIA must be re-evaluated at least every five years (permit renewal process) or sooner if changes to the facility or its operations would

result in an increased probability of exceeding a groundwater quality standard beyond the zone of attenuation.

The GIA of record for any facility was completed utilizing site specific data (hydrogeologic and leachate analyses) or as otherwise approved by the Illinois EPA as being representative of the facility setting. In many cases, the initial baseline model runs for the new waste units were borderline or even failed. To address those, design changes were incorporated to include thicker liner systems, revision the slope and leachate collection system to reduce the leachate head (seepage rate), revision to the liner system placement within the hydrogeologic setting (relocate the liner elevations to provide additional in-situ low hydraulic conductivity deposits between the liner invert and uppermost aquifer), and/or revision to the final cover system design to decrease the precipitation infiltration into the waste unit. The model also incorporated partitioning coefficients for specific surrogate groups which aided in reduction of the predicted model concentrations for typically problematic constituents, resulting in an acceptable model.

Regulatory constraints and guidance for the contaminant transport models have been largely consistent since the mid to late 1990s. Design and cell construction have been permitted for all active facilities, as well as final closure for many waste units. The final cover systems were designed based on HELP modeling which was used to determine seepage rate for the input to the contaminant transport model.

The addition of PFOA and PFOS constituents through the 35 IAC 620 rule revisions has the potential to cause failure of many permitted GIAs which are acceptable under the current requirements. It would have been possible during the initial design stage to address results of the PFOA and PFOS constituents through design changes. However, the potential for design changes to existing waste units are very limited, with only the final cover system realistically remaining for redesign to lower infiltration to the waste unit during post closure, thus possibly reducing the leachate head on the liner system.

Design changes for future cells (already permitted) yet to be constructed may be necessary if the results of the contaminant transport model fail due to the addition of the PFOA and PFOS constituents. However, this will be highly dependent upon the geologic setting and may be restricted by the local siting resolution pursuant to Section 39.2 of the Act. If the Illinois EPA is to go forward with the revisions as proposed, a mechanism needs to be created allowing existing facilities a way to address GIA failures without automatically reverting to a contingent remediation program.

4. Appropriate Contaminant Transport Models

The GIA is a determination of the time and distance dependent potential impact of a landfill unit on local groundwater chemistry. The GIA is based on a site-specific solute transport model of the actual design, site-specific hydrogeology, and conservative performance standards for the liner system, leachate management system and final cover system. The GIA is considered acceptable if the groundwater contaminant transport model predicts that the concentrations of all leachate constituents outside of the zone of attenuation are less than the Applicable Groundwater Quality Standards (AGQS) of 35 IAC 811.320 within 100 years of closure of the unit.

Typically contaminant transport models associated with the GIA have been generally simplistic, being one- and/or two-dimensional, such as POLLUTE and MIGRATE. The conceptual model assumes:

- all geologic units and soil liners are homogeneous and isotropic with respect to all lithologic and hydrologic parameters,
- that all layers are laterally extensive and the thickness of each layer is uniform,
- all layers are fully saturated,
- the external stresses on the system are constant through time,
- the source concentration is constant over the entire modeling period, and
- baseline surrogates were prepared in which no retardation or decay occurs.

Allowing the use of more reasonable model parameters would help reduce the model prediction factor and increase the probability of an acceptable model. The model input parameters are typically the most conservative across the board. When combined with conservative parameters for use in the HELP modeling, the end result is an ultraconservative model where surrogate groups are often needed to achieve an acceptable model. This would be a policy change for the Bureau of Land, not a regulatory change.

Under fully saturated conditions (bottom of the liner system to the bottom of the upper most aquifer), the models utilized for the approved GIAs are likely adequate for evaluation of the PFOA and PFOS constituents. However, settings where unsaturated conditions exist or a vadose zone exists beneath the liner system, a more complex model would better simulate transport of the PFOA and PFOS constituents as transport through such deposits are significantly less. Recent studies have shown PFOA and PFOS constituents are substantially retained in unsaturated deposits via solid phase adsorption, and also at the air-water interface. Differing models may simulate this characteristic better than the typical one- and two-dimensional models used for previous GIAs. Most contaminant transport models are incapable of working with the small-scale changes for these parameters that are seen within many geologic materials. The introduction of other contaminant transport models to deal specifically with the PFOA and PFOS constituents will be costly and time consuming not only for the facility but for review purposes by the Bureau of Land's Permit Section.

5. Bureau of Land Guidance

Even though the proposed 620 rule changes are being driven by the Bureau of Water, ramifications to the Bureau of Land programs are paramount. Prior to sending the proposed rule changes to the Illinois Pollution Control Board, the Bureau of Land should vet the potential ramifications to the regulations of 35 IAC Parts 811-815. The Bureau of Land should then provide a draft update to Appendix C to LPC-PA2 (Instructions for the Groundwater Protection Evaluation for Putrescible and Chemical Waste Landfills) for review and comment by the waste disposal industry. The Illinois EPA has provided two revisions to Appendix C based on what was learned over time during the permitting process. It is reasonable to expect the Bureau of Land should do the same with respect to implications to existing solid waste disposal facilities for revision of the 620 rules. Topics that should be addressed include but are not limited to:

a. Legacy impacts (cross contamination) to groundwater quality – what if the wells already exhibit PFOA and PFOS concentrations in excess of the proposed standards
    i. Well construction issues
    ii. Pump materials
    iii. Impacts to AGQS determination
b. Sampling protocols for groundwater and leachate
c. Laboratory analyses test methodology and limitations – how can "draft" methods be placed into a state regulations
d. GIA – It is a tool
    i. Computer models – provide insight on potential other models for use
    ii. Input parameters
        - Use of more realistic values versus overly conservative values
        - Use of averages or statistical derivations, not the maximum or minimum
        - Update Attachment 1 to Appendix C to include PFOS and PFOA constituents
    iii. Surrogate Modeling for PFOA and PFOS constituents
        - Retardation allowances
        - Sensitivity analyses constraints
e. Use of contingent remediation programs to address predicted exceedences
f. Permitted Contingent Remediation Plans – will all of these need to be re-evaluated with the inclusion of PFOA and PFOS constituents
g. Impacts to permitted waste units in corrective action (35 IAC 807 and 814 Subpart C and D)
h. Impacts to permitted waste units conducting corrective action pursuant to consent orders and/or in conjunction with the US EPA or other entities
i. Sites finishing post closure care (the Affidavit for Certification of Completion of Post-Closure Care has been submitted) – will PFOA and PFOS constituents require analyses prior to release
j. Reasonable dates and timelines for implementation
k. Regulatory exclusion – if the proposed rules are passed, while a facility evaluates its water and leachate quality, the Illinois EPA must provide temporary exclusion from Section 18 of the Act, or others that may apply

Once a new standard is promulgated in Part 620, it is then incorporated into the relevant programs administered by the Bureau of Land. As described above, the process to evaluate potential contaminants are imposed through permits issued by the Bureau of Land. The mere detection of the PFOA and PFOS constituents at a landfill monitor well requires the owner/operator to disprove the potential of a release to the environment. Considering the current body of scientific knowledge, facilities will likely be thrust into the environmental investigation process. That process leads to corrective action. No economic impact study has been conducted to evaluate the cost or the value of expending resources on this path.

The next public meeting should include members from the Bureau of Land prepared to discuss implications to the existing permitted landfill facilities. These issues should be considered prior to submittal of the proposed rule revisions to the Illinois Pollution Control Board for approval.

MWI
Millennium Waste Incorporated

February 27, 2020

VIA e-mail: sara.terranova@illinois.gov
Ms. Sara G. Terranova
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Division of Legal Counsel
Illinois Environmental Protection Agency
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RE: Comments Regarding Proposed Amendments to 35 Ill. Adm. Code 620: Groundwater Quality

Dear Ms. Terranova:

We are the owner of Quad Cities Landfill in Milan, IL and are compelled to submit the comments below regarding proposed changes to the groundwater quality rules in 35 Ill. Adm. Code 620.

Background

Several Per- and Poly-Fluoroalkyl Substances (PFAS) compounds [man-made hydrophobic chemicals] are being proposed as additions to the potable (Class I) and general resource (Class II) groundwater quality lists. Specifically, the following compounds and groundwater standards are being proposed:

- Perfluorobutane Sulfonic Acid (PFBS) 140,000 ng/L (0.14 mg/L)
- Perfluorohexane Sulfonic Acid (PFHxS) 140 ng/L
- Perfluorononanoic Acid (PFNA) 21 ng/L
- Perfluorooctanoic Acid (PFOA) 21 ng/L
- Perfluorooctane Sulfonic Acid (PFOS) 14 ng/L

The amendments propose both individual and combined values for PFOA (21 ng/L) and PFOS (14 ng/L), which combined are not to exceed 21 ng/L.

Documentation suggests the range of PFOA + PFOS concentrations in landfills generally vary from 500 to 5,000 ng/L depending on the facility's acceptance of industrial waste or biosolids from wastewater treatment plants (WWTP). Continued acceptance of biosolids from WWTP will progressively concentrate PFAS compound mass.

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Concerns for Current Compliance

There are several concerns for active solid waste landfills that are currently regulated under 35 IAC Part 811 that should be accounted for if the new drinking water standards are adopted.

Groundwater Monitoring

One of the biggest concerns is the effect of adding PFAS compounds to the groundwater monitoring lists and the interferences (false positives) that will occur from sampling from the existing groundwater monitoring systems. Landfills regulated under 35 IAC 811 have established leak detection monitoring systems. Detection monitoring systems are based on conservative constituents (e.g., chloride) that are even more mobile that PFAS compounds; additional monitoring wells will not be required. However, many (if not most) active landfills have dedicated submersible sampling pumps that are permanently installed in the observation wells that make up the monitoring network of upgradient and downgradient wells. The Sampling and Analysis Plans (SAP) for permitted landfills have IEPA approval regulated under 35 IAC 811.318. Unfortunately, countless landfills (such as Quad Cities Landfill IV) have existing leak detection monitoring systems in place that are not suitable for sampling of PFAS compounds since the dedicated sampling tubing are lined with Teflon™ for its hydrophobic properties to prevent adsorption of constituents during sampling. Teflon is specifically identified as one of three materials approved for use (along with Stainless Steel 304 & 316) as durable, corrosion-resistant material allowed by IEPA for water sampling as outlined in IEPA's *Instructions for the Groundwater Protection Evaluation for Putrescible and Chemical Waste Landfills* [Appendix C to LPC-PA2].

Entire monitoring well networks may contain pumps with Teflon bladders, gaskets, discharge tubing, and Teflon-coated wire, all in direct contact with the groundwater samples. Teflon tape is commonly used on the threads of pumps and possibly at joints of well screens and casings. Therefore, the dedicated monitoring wells and tubing of solid waste facilities may be subject to significant burden of demonstration that alternate sources are the cause for false positive results.

If PFAS sampling is limited in its adoption to solid waste facilities, such as a single sampling event confirming detects less than drinking water standards (similar to the addition of new volatile organic compounds to the 620 standards), temporary removal of the sampling pumps, followed by redevelopment of the monitoring wells prior to PFAS sampling may be a work around. However, these procedures would be substantially burdensome if they had to continue long-term.

Groundwater impact Assessment (GIA)

The requirements of 35 IAC 811.317 is a unique permitting element to the Solid Waste Regulations in Illinois. Groundwater contaminant transport (GCT) modeling results must demonstrate predicted concentrations of all constituents in leachate outside the zone of attenuation are less than applicable groundwater standards within 100 years of closure of the

unit. Addition of PFAS compounds to the Part 620.410 and 620.420 groundwater standards and their subsequent addition to GIA's will result in countless landfills having GCT models that will no longer meet the requirements of 811.819(b) and be out of compliance. There are several reasons for the concerns with the GIA modeling that are outlined below:

- Leachate Source Characterization – This is a concern that is similar to the groundwater monitoring network, in that leachate collection and distribution components may contain PFAS compounds (including Teflon-bearing plumber's tape, as well as other gaskets, washers, and o-rings within leachate pumps, values, and tubing). This equipment is not readily replacable. Biased high results from system components in leachate would have direct effect on the GIA since these are required as conservative source concentrations in the GCT modeling.

  Should characterization of PFAS in leachate be required for the GIA, a reasonable alternative would be an allowance of average PFAS concentrations. Currently, leachate concentrations are required to be at least in the upper 95% confidence interval of detected concentrations. Since leachate concentrations will likely be biased high due to PFAS compounds being present in leachate collection and conveyance components, allowance of an averaged source concentration is appropriate.

- Transport and Fate Properties – Components for fate and transport of chemical-specific groundwater modeling of the PFAS compounds are uncertain. Preliminary data indicate that these compounds are known to be soluble, very stable, and non-volatile. PFAS compounds that are most commonly detected in the environment typically have competing tendencies of the head and the tail. The tail is hydrophobic (tends to repel water), whereas the head groups are polar and hydrophilic (tend to mix with water). The variations in tail lengths lead to a wide distribution in the environment (https://pfas-1.itrcweb.org/).

  Given heterogeneous subsurface environments, other geochemical factors such as pH, and presence of polyvalent cations, multiple partitioning mechanisms should be considered when characterizing PFAS fate and transport (Guelfo and Higgins 2013; McKenzie et al. 2016; Brusseau 2018). This statement suggests that accurate (or average) site conditions be considered in GCT modeling versus the most conservative assumptions that are currently required for GIA inputs. For example, at relevant environmental pH values, some PFAS constituents are typically present as organic anions and therefore tend to associate with the organic carbon fraction that may be present in the subsurface. Instead of calculating migration with accurate (average) organic content values, an overly conservative input of the lower 95% confidence interval is currently required for GCT modeling. This requirement could be relaxed for PFAS compounds so that average site conditions are represented for complicated PFAS migration processes and recent uncertainties in GCT results.

  Organic carbon-water partition coefficients (Koc values) are being established for many commonly detected PFAS compounds that are often detected at release sites (https://pfas-1.itrcweb.org/). However, diffusivity properties of PFOS compounds are still in development. PFOS diffusion in groundwater appears not to have been a priority in initial

migration studies since diffusion rates are significantly slower relative to advection processes. However, in composite or clay-lined landfills in clay-rich subsurface environments that are common in Illinois, knowledge of diffusion rates is required. Thus, implementation of GCT modeling requirements that will be triggered by new 35 IAC 620 groundwater standards is worrisome for solid waste owners and operators.

Alternatives to the current configuration of the solid waste regulations have been added to the discussions above. Additional alternatives for the IEPA to consider are reducing PFAS constituents pending investigations and elimination for the GIA requirement.

- Consider eliminating or reducing requirements for certain PFAS constituents that may be detected as false-positives as a result of cross-contamination from existing (and permitted) groundwater monitoring systems and/or approved standard landfill design guidelines.
- Consider eliminating the GIA. The requirement of a GCT model is unique to Illinois and is not necessary if minimum design considerations are met. The GIA serves no material practical purpose for the construction of landfills. It is well demonstrated that the standard Subtitle D landfill design has served to provide environmental protection. Regarding landfills, the GIA serves no material benefit to environmental projection. It is time to eliminate the GIA.

We are grateful for the opportunity to submit these comments. Please do not hesitate to contact me if you have any questions or comments.

Sincerely,

Millennium Waste Incorporated

Dominic J Remmes, PE
Region Engineer

WASTE MANAGEMENT

Comments on 35 Ill. Adm. Code 620 Proposed Amendments
February 28, 2020

A. Detection and Quantification

The proposed Part 620 amendments include many standards listed at levels that will be difficult for commercial laboratories to quantify. Any proposed standards must consider a commercial laboratory's ability to quantify and report at these levels (i.e., at the practical quantification limit - PQL). The purpose of the PQL is to adjudicate between a health-based level and a laboratory's ability to quantify at that level. Any proposed standard must consider the commercial laboratory's capability to quantify at that level to avoid falsely reporting a standards exceedance when it does not exist. The following points are critical for IEPA to address prior to finalizing proposed standards in Part 620.

1. **Section 620 [All Subsections] - The use of PQLs in setting numeric standards needs to be retained or added:** Setting of numeric standards must consider analytical capability and variability to represent legal and attainable regulatory limits. Fundamental to any regulatory establishment of numeric standards is the evaluation and adjustment of health-based 'goals', where needed, to create standards that analytical technology can reliably quantify. It is this 'adjudication' of the environmental 'goal' to what is practically achievable that provides the technical foundation for the Agency to regulate, and for regulated parties to comply with regulation. Where a numeric standard is set without this adjudication, the establishment of a standard is arbitrary and capricious. A direct example of federal adjudication to analytical technology's limitations is MCLGs being adjusted to MCLs. [See Fed. Reg. 54, #97, May 22, 1989 page 22100 for a discussion of MCLs and the use of PQLs].

   For RCRA landfill groundwater regulations at the federal level, the PQL model is defined in regulation and is used for this adjudication. Although the current IEPA Title 35 Part 620 includes a definition for PQL, how it is used in the rule is unclear. The seeming removal of PQL from the Agency 's numeric standard setting process ignores the vital role of the PQL in establishing 'reasonableness' in setting regulatory levels. In this scenario, regulated parties must use analytical technology to measure and compare to a regulatory standard, which is inconsistent with RCRA and environmental regulation in general.

2. **IEPA needs to address how PQLs are utilized in the setting of standards:** and consider the analytical minimum capabilities (sensitivity) and variability (precision and accuracy) of the commercial laboratories in making regulatory decisions. Setting and using PQLs and assuring the upper end of the uncertainty bounds at the health-based numeric value when less than PQL are needed. Use of the PQL will prevent arbitrary and discriminatory enforcement of standards for those who apply them.

3. **A clear definition of the PQL is needed in Part 620:** USEPA initially defined PQL at 50 Fed. Reg. 46906 (Nov. 13, 1985) under the Clean Drinking Water Act. PQL is defined as *"the lowest level achievable by good laboratories within specified limits during routine operating conditions."* 620.110, Definitions, defines PQL as *"the lowest concentration or level that can be reliably measured within specified limits of precision and accuracy during routine laboratory operating conditions in accordance with "Test Methods for Evaluating Solid Wastes, Physical/Chemical Methods", EPA Publication No. SW-846, incorporated by reference at Section 620.125."* In 40 CFR

Part 257.23.g.5 (page 16 of 81), the PQL is *"the lowest concentration level that can be reliably achieved within specified limits of precision and accuracy during routine laboratory operating conditions that are available to the facility."*

Most importantly, the Statistical Analysis of Groundwater Monitoring Data at RCRA Facilities (Unified Guidance - 2009) which is referenced in Part 620 states on p. 2-7 that *"Any practical quantification limit (pql) approved by the Regional Administrator under §264.97(h) [or §258.53(g)] that is used in the statistical method shall be the lowest concentration level that can be reliably achieved within specified limits of precision and accuracy during routine laboratory operating conditions available to the facility."* It is recommended that the Unified Guidance definition be incorporated into Part 620.

While these definitions may vary somewhat, the key elements are:

- PQLs must be established using laboratories available to the regulated facilities
- Routine operating conditions must be in place at the time the data are being developed for the determination of the PQLs.
- Specified limits of (aka known and controlled) precision and accuracy requires the Agency to select precision and accuracy values. These values represent the allowable Relative Measurement Error (RME) and, thus, significant digits (or parts thereof) required before quantitation is established.
- Marginal changes to the definition do not change the science or the Agency responsibility to select criteria.

The application of statistics also requires that other requirements be specified and met such as sample size, normality and confidence.

B. Addition of 5 PFAS Compounds (PFOA, PFOS, PFBS, PFHxS, and PFNA)

The IEPA should address and resolve key scientific uncertainties before developing standards for PFAS. Existing literature demonstrates significant scientific uncertainty where standards are being developed with insufficient technical knowledge. Developing standards for PFAS at this time will lead to flawed rulemaking and will impose unwarranted, unfair, and oppressive legal, economic, and operational burdens on the regulated community. Standards developed would be scientifically unsound, fundamentally unfair, and create confusion and unintended societal consequences in the future.

1. **The accelerated pace to established PFAS standards does not allow the time needed to adequately assess the potential toxicity of a given compound:** let alone to develop MCLs that consider economic and technological factors. By way of comparison, and focusing solely on the toxicity component alone, USEPA has been assessing the potential toxicity of dioxin and furans – a group of merely 210 compounds, a much smaller group than the 4,000 unique PFAS compounds – since 1985. USEPA's assessment of dioxin-like compounds has been reviewed by USEPA Science Advisory Boards on four separate occasions, has been reviewed by the National Academy of Sciences, and has undergone multiple rounds of public comment. It took USEPA more than 20

years to reach consensus on the noncancer effects of dioxin and furans, and, even after such technical scrutiny, USEPA still has not reached a consensus on dioxin's cancer potency. This is for a group of chemicals for which the mode of toxic action and the relative potency among congeners is well-developed. None of these conditions hold for PFAS, and yet IEPA wants to establish standards for five PFAS when significant data gaps exist. The current science does not support standards establishment. Standards should not be developed until much greater scientific certainty and technical understanding is gained.

2. **The development of PFAS standards should start with properly assessing human health risks** not with assuming that the science is settled. Over 2,000 studies have been conducted on either PFOA or PFOS in laboratory animals, including mice, rats, and primates, plus over 400 human epidemiological studies have been published on PFAS, primarily on PFOA and PFOS. Although many independent studies have been performed, results are inconsistent and scientific consensus is lacking on what the data mean regarding human health risks and PFAS toxicity. It is imperative to keep the state of the science in the forefront to ensure technically defensible standards are developed and are appropriate for the long term.

3. **IEPA should consider interim, conditional, or similar alternatives to prematurely establishing formal standards** given the inadequate technical information and scientific consensus regarding PFAS health risks. For example, delaying rulemaking until adequate technical information is available by performing state-funded studies and reviewing ongoing studies being performed worldwide to fill data gaps would not only be procedurally justified but would be a more responsible use of funding. A reasonable approach for IEPA is to consider reevaluation of PFAS health effects on an annual basis following review of new studies and other scientific developments.

C. Concerns with Approach in Development of PFAS Groundwater Quality Standards

It is clear from the literature that too little is known currently to derive reliable standards for PFAS. Standards for PFAS based on the current state of knowledge are driven by uncertainty that needs to be responsibly addressed within the scientific community. If standards development is going to move forward, the process needs to be more rigorous and transparent so that better values can be supported. It is again emphasized that IEPA should consider interim, conditional, or similar alternatives to prematurely establishing formal standards.

1. **The state of knowledge on PFAS toxicology is still developing and many uncertainties exist. IEPA should refrain from developing PFAS groundwater standards until these scientific uncertainties are better explored.** Discrepancies exist between effects observed in rodent toxicology studies and human epidemiology studies. Furthermore, the adversity of many effects observed in human epidemiology studies is an area of active debate. Given the uncertainty in underlying data, there is wide variation in guideline/regulatory values that have been developed by different organizations.

Comments on 35 Ill. Adm. Code 620 Proposed Amendments
February 28, 2020

2. **Use of ATSDR's draft MRLs results in overly conservative groundwater quality standards that are driven by uncertainty. If IEPA moves forward with developing groundwater quality standards, they should consider using different Tier III toxicity values.** In developing their draft minimum risk levels (MRLs), ATSDR applied dosimetric adjustment factors of 10,000 (PFOA), 14,400 (PFOS), 15,500 (PFHxS), and 6,500 (PFNA) to account for differences between humans and rodents. Additionally, a combined uncertainty factor of 300 was applied to each PFAS such that the total adjustment factors used were 3,000,000 for PFOA, 4,300,000 for PFOS, 4,650,000 for PFHxS, and 1,950,000 for PFNA. Application of such large adjustment factors is excessive and is the main driver of IEPA's proposed low groundwater quality standards. As outlined further below, the chronic oral reference doses (RfDs) developed by USEPA Office of Water in 2016 for PFOA and PFOS should be used by IEPA to develop groundwater quality standards.
3. **If IEPA moves forward with developing groundwater quality standards for PFAS, they need to document which Tier III toxicity values they considered and how they determined which values were the most appropriate.** ATSDR's PFAS MRLs are still in draft format. During the stakeholder meeting on February 13, 2020, a number of scientific and technical issues were raised regarding some of ATSDR's draft MRLs. As discussed further below, more scientifically supportable Tier III toxicity values are available and should be considered by IEPA if they move forward with developing groundwater quality standards for these compounds.

4. **If IEPA moves forward with developing groundwater quality standards for PFOS, USEPA's chronic oral reference dose should be used.** ATSDR developed an intermediate-duration MRL of 2 ng/kg-day for PFOS based upon results from a two-generation reproductive toxicity study (Luebker et al. 2005), which is the same key study underlying the chronic oral RfD (20 ng/kg-day) developed by USEPA Office of Water in 2016. Both USEPA and ATSDR used similar approaches to develop their toxicity values. Both USEPA and ATSDR applied UFs of 10 to account for human variability and 3 to account for interspecies variability; however, ATSDR applied an additional modifying factor (MF) of 10 to their PFOS MRL to account for uncertainty that immunotoxicity may be a more sensitive endpoint than developmental toxicity. Application of an additional MF of 10 is excessive when a total adjustment factor of 430,000 had already been applied. Furthermore, USEPA considered immunotoxicity endpoints in their PFOS assessment and determined that due to uncertainties related to mode of action and the level, duration, and time of exposure it is not appropriate to quantitatively assess immunotoxicity[1].

5. **ATSDR used low validity studies to develop their PFOA MRL. If IEPA moves forward with developing groundwater quality standards for PFOA, US EPA's chronic oral reference dose should be used.** ATSDR developed the PFOA MRL using results from two developmental toxicity studies (Koskela et al. 2016; Onishchenko et al. 2011). Both studies utilized a single dose level (i.e., 0.3 mg/kg-day) and had a small sample size (i.e., n=5-10 animals per dose). According to 35

[1] See Section 8.3 (Consideration of Immunotoxicity) in Drinking Water Health Advisory for Perfluorooctane Sulfonate (PFOS). EPA Document number 822-R-16-004.

Ill. Adm. Code 620 App. A, the studies utilized by ATSDR are low validity studies. To avoid developing groundwater quality standards based on low validity studies, IEPA should use the chronic oral RfD developed by USEPA Office of Water in 2016. USEPA's PFOA RfD is the basis of USEPA's 2016 drinking water Health Advisory Level[2] and USEPA's 2019 interim recommendations for addressing PFOA contaminated groundwater[3]. Use of USEPA's PFOA RfD would align IEPA with current federal practices. Use of USEPA's RfD for PFOA and supports a groundwater quality standard of 140 ng/L for PFOA.

6. **USEPA's draft chronic oral RfD for PFBS may be the most appropriate toxicity value for developing a groundwater quality standard.** Currently, the proposed groundwater quality standard of 140,000 ng/L PFBS is based upon USEPA's 2014 provisional peer-reviewed toxicity value of 0.02 mg/kg-day PFBS. However, in November 2018 USEPA's Office of Research and Development issued a draft chronic oral RfD of 0.01 mg/kg-day PFBS[4]. Although this value is not yet finalized, it received favorable peer-reviews and it represents the best available science for PFBS. IEPA should consider using USEPA's 2018 draft RfD for PFBS, which supports a groundwater quality standard of 70,000 ng/L PFBS.

7. **Use of a default relative source contribution (RSC) value of 20% is not appropriate for developing groundwater quality standards for all PFAS.** The use of the default RSC of 20% likely overestimates the contribution of diet and other non-drinking water sources in situations where exposure to elevated PFAS in drinking water occurs. For example, based on chemical-specific data, the New Jersey Drinking Water Quality Institute determined that a RSC of 50% was most appropriate for developing a drinking water MCL for PFNA. Use of a RSC of 50% for PFNA would increase the proposed drinking water standard from 21 ng/L to 53 ng/L. IEPA should evaluate the relative source contributions for each of the PFAS for which it develops a groundwater quality standard. Chemical-specific data is available for some of the PFAS for which IL EPA has proposed groundwater quality standards.

D. Basis for Standards Development

Overall, more information is needed to document the technical basis for each of the proposed standards. The procedures described in the regulations, including Appendices A and B of Title 35, are not sufficient to understand the basis of the proposed standards. It is not possible to track the basis of all of the numbers simply by following what is documented in the subject Appendices of the regulations.

1. **We recommend that Illinois document the toxicity value and any other values used to calculate the standards** and provide that supporting information in the regulation so that the technical

[2] https://hero.epa.gov/hero/index.cfm/reference/details/reference_id/3603279
[3] https://www.epa.gov/pfas/interim-recommendations-addressing-groundwater-contaminated-pfoa-and-pfos
[4] PFBA Draft Toxicity Assessment. Available from: https://www.epa.gov/pfas/genx-and-pfbs-draft-toxicity-assessments

basis for each standard is transparent. For example, if the standard is based on the MCL or MCLG, IEPA should document the value and its source. If the value is calculated using a toxicity criterion and a relative source contribution (RSC) factor, IEPA should document the toxicity value used in the calculation, the source of that value, and the assumed RSC. IEPA also should provide the rationale for and basis of any differences between Class I and Class II standards. Without this information, the technical soundness of the criteria cannot be fully evaluated.

For example:

- IEPA states that MCLs/MCLGs are used as the basis for Class I standards, when available. Yet that is not the case for the proposed Class I standards for fluoride, lead, selenium, and copper, which differ from (and are lower than) the MCLs. The rationale for those differences should be clearly documented.

- IEPA states that it uses USEPA noncancer reference doses and a default RSC of 0.2 to calculate Class I standards when MCLs/MCLGs are not available. Yet, a simple comparison of the proposed Class I standards to risk screening levels (RSLs) developed by USEPA for drinking water exposures shows that has not been done consistently. Given the differences in the ingestion rate and body weight assumed by USEPA and IEPA and the additional application of an RSC term by IEPA, the proposed Class I standards would be lower than USEPA's ingestion only RSLs, however this is not the case for several noncarcinogens (e.g., 1,3,5-trinitrobenzene and HMX). The basis for these differences should be documented.

- IEPA should document the sources of and the values used for the cancer slope factors (CSFs) included in the calculations for potential carcinogenic chemicals. The information provided in the Appendices does not identify the preferred source for CSFs used for deriving Class I criteria for carcinogens. A simple comparison of the proposed Class I standards to USEPA's drinking water RSL (ingestion only) values for potential carcinogens indicates that IEPA either did not use USEPA CSFs or used some other undefined factors in their calculation (e.g., Class 1 criteria for carcinogenic PAHs are more than 3-fold higher than EPA's RSL for ingestion only).

- IEPA should provide an explanation of how the endpoint of concern (i.e., cancer vs. noncancer) is selected when deriving Class I criteria. One would assume that when toxicity criteria are available from USEPA for cancer and noncancer endpoints that the more sensitive endpoint would be selected as the basis for calculating a Class I criteria. This does not appear to be the case in all instances. For example, the Class I criteria for 2,4,6-Trinitrotoluene appears to be based on the noncancer endpoint, however a Tier 1 EPA CSF for this compound is available and results in a lower risk-based level (as indicated by USEPA's RSL for this compound).

2. **IEPA should provide a chemical-specific analysis to support its use of an RSC of 0.2** in calculation of Class I values. Appendix A states that a default relative source contribution (RSC) of 20% should

be used in calculating Class I criteria for non-carcinogens, unless chemical-specific data are available. The RSC is based on the assumption that only 20% of a person's daily exposure to a chemical is via drinking water, and that the rest is from other sources, such as diet. There is no evidence that this assumption is valid for any of the regulated chemicals, and it is likely that a chemical-by-chemical analysis would document that it is invalid in many instances. For example, many chemicals are unlikely to be present in the diet, which is an important source of non-drinking water exposure factored into the RSC. In these cases, drinking water exposures could represent up to 100% of the exposures, and the calculated Tier 1 standards could be 5 times higher. A reconsideration of the use of a default RSC of 0.2 is warranted.

3. **IEPA should provide an explanation of how Class II (for General Resource groundwater) criteria** are derived. No explanation is provided, nor does there appear to be consistency in the criteria proposed. For some chemicals with an MCL available the MCL appears to be adopted for the Class II criteria, whereas for others it is not. Some Class II criteria look to be set equal to the Class I criteria, whereas others are lower, and still others are higher.

E. Background Update/Unified Guidance

Reference to the Unified Guidance (2009) is a good addition to the rule as long as it allows the regulated entity to apply any of the methods that are applicable within the guidance document.

1. **Using the Unified Guidance (2009) should be strongly advocated by IEPA** as its use will provide statistical flexibility and more up to date statistical approaches to be followed both when developing and updating background at a facility and developing the most appropriate statistical limits to satisfy permit conditions. Finally, the application of the Unified Guidance (2009) procedures should be synchronized with other State regulatory programs such as Rule 811 to be as effective as possible to all the regulated community.

F. References

Koskela, A., Finnilä, M.A., Korkalainen, M., Spulber, S., Koponen, J., Håkansson, H., Tuukkanen, J. and Viluksela, M., 2016. Effects of developmental exposure to perfluorooctanoic acid (PFOA) on long bone morphology and bone cell differentiation. *Toxicology and applied pharmacology*, *301*, pp.14-21.

Luebker, D.J., Case, M.T., York, R.G., Moore, J.A., Hansen, K.J. and Butenhoff, J.L., 2005. Two-generation reproduction and cross-foster studies of perfluorooctanesulfonate (PFOS) in rats. *Toxicology*, *215*(1-2), pp.126-148.

Onishchenko, N., Fischer, C., Ibrahim, W.N.W., Negri, S., Spulber, S., Cottica, D. and Ceccatelli, S., 2011. Prenatal exposure to PFOS or PFOA alters motor function in mice in a sex-related manner. *Neurotoxicity research*, *19*(3), pp.452-461.

From: Hawbaker, Carol
To: Guy, Jeff
Subject: FW: docs
Date: Thursday, May 13, 2021 9:16:15 AM
Attachments: Draft Proposed 35 Ill. Adm. Code 620 (6).pdf
Draft Proposed 35 Ill. Adm. Code 620 (5).pdf
Draft Proposed 35 Ill. Adm. Code 620 (4).pdf
Draft Proposed 35 Ill. Adm. Code 620 (3).pdf
Draft Proposed 35 Ill. Adm. Code 620 (2).pdf
Draft Proposed 35 Ill. Adm. Code 620 (1).pdf
PN Screen Capture 620 210512.PNG

Carol

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From: Frost, Brad <Brad.Frost@Illinois.gov>
Sent: Wednesday, May 12, 2021 3:59 PM
To: Diers, Stefanie <Stefanie.Diers@Illinois.gov>; Terranova, Sara <Sara.Terranova@Illinois.gov>; LeCrone, Darin <Darin.LeCrone@Illinois.gov>; Bailey, Sabrina <Sabrina.Bailey@Illinois.gov>; Dunaway, Lynn <LYNN.DUNAWAY@Illinois.gov>; Summers, Michael <Michael.Summers@Illinois.gov>; Brown, Michael L. <Michael.L.Brown@Illinois.gov>; Martin, Lauren <Lauren.Martin2@Illinois.gov>; Ankney, Clayton <Clayton.Ankney@Illinois.gov>; Hawbaker, Carol <Carol.Hawbaker@Illinois.gov>
Subject: RE: docs

e-mails sent. Sara attached are pdfs of the e-mails and a screen capture of the posted public notice for your record.

From: Frost, Brad
Sent: Wednesday, May 12, 2021 3:32 PM
To: Diers, Stefanie <Stefanie.Diers@Illinois.gov>; Terranova, Sara <Sara.Terranova@Illinois.gov>;

LeCrone, Darin <Darin.LeCrone@Illinois.gov>; Bailey, Sabrina <Sabrina.Bailey@Illinois.gov>; Dunaway, Lynn <LYNN.DUNAWAY@Illinois.gov>; Summers, Michael <Michael.Summers@Illinois.gov>; Brown, Michael L. <Michael.L.Brown@Illinois.gov>; Martin, Lauren <Lauren.Martin2@Illinois.gov>; Ankney, Clayton <Clayton.Ankney@illinois.gov>; Hawbaker, Carol <Carol.Hawbaker@Illinois.gov>
Subject: RE: docs

Pages are live. E-mails to be sent shortly
https://www2.illinois.gov/epa/about-us/rules-regs/water/Pages/620-Groundwater-Quality.aspx

https://www2.illinois.gov/epa/public-notices/Pages/general-notices.aspx

https://www2.illinois.gov/epa/about-us/rules-regs/Pages/new-and-proposed-rules.aspx

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From: Frost, Brad
Sent: Wednesday, May 12, 2021 12:14 PM
To: Diers, Stefanie <Stefanie.Diers@Illinois.gov>; Terranova, Sara <Sara.Terranova@Illinois.gov>; LeCrone, Darin <Darin.LeCrone@Illinois.gov>; Bailey, Sabrina <Sabrina.Bailey@Illinois.gov>; Dunaway, Lynn <LYNN.DUNAWAY@Illinois.gov>; Summers, Michael <Michael.Summers@Illinois.gov>; Brown, Michael L. <Michael.L.Brown@Illinois.gov>; Martin, Lauren <Lauren.Martin2@Illinois.gov>; Ankney, Clayton <Clayton.Ankney@illinois.gov>; Hawbaker, Carol <Carol.Hawbaker@Illinois.gov>
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For review

From: Frost, Brad
Sent: Wednesday, May 12, 2021 11:30 AM
To: Diers, Stefanie <Stefanie.Diers@Illinois.gov>; Terranova, Sara <Sara.Terranova@Illinois.gov>; LeCrone, Darin <Darin.LeCrone@Illinois.gov>; Bailey, Sabrina <Sabrina.Bailey@Illinois.gov>; Dunaway, Lynn <LYNN.DUNAWAY@Illinois.gov>; Summers, Michael <Michael.Summers@Illinois.gov>; Brown, Michael L. <Michael.L.Brown@Illinois.gov>; Martin, Lauren <Lauren.Martin2@Illinois.gov>; Ankney, Clayton <Clayton.Ankney@illinois.gov>; Hawbaker, Carol <Carol.Hawbaker@Illinois.gov>
Subject: docs

e-mail language

Subject: Draft Proposed 35 IAC 620

The Illinois EPA is proposing changes to 35 IAC 620. Prior to filing with the Illinois Pollution Control Board, the Illinois EPA will accept comments on the draft proposed rules. Written comments must be received by the Illinois EPA by June 25, 2021. Comments must be submitted to EPA.620.rulemaking@illinois.gov. Additionally, the Illinois EPA will host a virtual public meeting to review the proposed changes and answer questions concerning the proposal. The meeting will be

held at 1:00 pm on May 26, 2021. See the 620 website for additional details about the rulemaking and how to participate during the comment period.

From: Frost, Brad
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Bcc: "info@yourwaterwellandpump.com"; "cpiusweibel@hotmail.com"; EXT Rios, Monica; "WalkerD@mwrd.org"; "deborah.williams@cwlp.com"; "jmore@schiffhardin.com"; "sbonebrake@schiffhardin.com"; "aantoniolli@schiffhardin.com"; "Cynthia.Vodopivec@vistraenergy.com"; "Cynthia.Vodopivec@vistraenergy.com"; "rgranholm@schiffhardin.com"; "victor.modeer@vistraenergy.com"; "susana.hildebrand@vistraenergy.com"; "phil.morris@vistraenergy.com"; "Rcipriano@schiffhardin.com"; "msmallwood@ameren.com"; "jmartin@heplerbroom.com"; "czeman@ppi.coop"; "David.bacher@nrg.com"; "Nathan.rozic@nrg.com"; "Sharene.shealey@nrg.com"; "Walter.stone@nrg.com"; "kg@nijmanfranzetti.com"; "sf@nijmanfranzetti.com"; "va@nijmanfranzetti.com"; "Elizabeth.quirk-hendry@nrgenergy.com"; "Keith.schmidt@nrgenergy.com"; "joseph.kotas@nrg.com"; EXT Gulley, Donald; "wwatson@sipower.org"; "iergstaff@ierg.org"; "kthompson@ierg.org"; "Jmontgomery@schiffhardin.com"; "mbilut@mwe.com"; "PJ.Becker@cwlp.com"; "tedhartke@hartke.pro"; "twubker@commercialliabilitypartners.com"; "Pharvey@Geosyntec.com"; "sharene.shealey@nrg.com"; "ron@atonenv.com"
Subject: Draft Proposed 35 Ill. Adm. Code 620
Date: Wednesday, May 12, 2021 3:41:00 PM
Attachments: Factsheet 620.pdf
Notice 620.pdf

The Illinois EPA is proposing changes to 35 Ill. Adm. Code 620. Prior to filing with the Illinois Pollution Control Board, the Illinois EPA will accept comments on the draft proposed rules. Written comments must be received by the Illinois EPA by June 25, 2021. Comments must be submitted to EPA.620.rulemaking@illinois.gov.

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See the 620 website for additional details about the rulemaking and how to participate during the comment period.

Attached to this e-mail are the public notice and factsheet.

From:	Frost, Brad
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Subject:	Draft Proposed 35 Ill. Adm. Code 620
Date:	Wednesday, May 12, 2021 3:41:00 PM
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From:	Frost, Brad
To:	EPA.620.rulemaking
Bcc:	ssylvester@atg.state.il.us; aarmstrong@atg.state.il.us; KPamenter@atg.state.il.us; Lohrenz, Eric; Mool, Bob; Mauer, Paul; San Diego, Nick; newman.christopher@epa.gov; eaj2@illinois.edu; services@staterepcarolammons.com; mooney.susan@epa.gov; jackson.mary@epa.gov; staniec.carol@epa.gov; setnicar.mary@epa.gov; cunningham.michael@epa.gov; dufficy.craig@epa.gov; gonzalez.maria@epa.gov; triantafillou.kathy@epa.gov; moody.jonathan@epa.gov; cecala.ian@epa.gov; celeste.laurel@epa.gov; kenney.thomas@epa.gov; ireland.scott@epa.gov; faison.george@epa.gov; behan.frank@epa.gov; news@wmbd.com; dzalaznik@pjstar.com; newsguy102@yahoo.com; jellybelly7615@gmail.com; evans.larry9@gmail.com; business@offrte29.net; janpred65@gmail.com; alice_vernon@me.com; dnny9743@yahoo.com; vrancuret@gmail.com; gschindler@pdclab.com; chostetler@pdcarea.com; opltocc@ezl.com; bscrawford76@charter.net; stonesoup@gmx.com; al3206@sbcglobal.net; cromanus1249@yahoo.com; kayahaus@yahoo.com; Kenpage1@comcast.net; jahchoices@gmail.com; rachael@labrysweb.com; rongtrimmer@gmail.com; jestpr@aol.com; rcjuras@yahoo.com; rlocke@illinois.edu
Subject:	Draft Proposed 35 Ill. Adm. Code 620
Date:	Wednesday, May 12, 2021 3:41:00 PM
Attachments:	Notice 620.pdf Factsheet 620.pdf

The Illinois EPA is proposing changes to 35 Ill. Adm. Code 620. Prior to filing with the Illinois Pollution Control Board, the Illinois EPA will accept comments on the draft proposed rules. Written comments must be received by the Illinois EPA by June 25, 2021. Comments must be submitted to EPA.620.rulemaking@illinois.gov.

Additionally, the Illinois EPA will host a virtual public meeting to review the proposed changes and answer questions concerning the proposal. The meeting will be held at 1:00 pm on May 26, 2021. The public notice contains additional details about the comment period and meeting.

See the 620 website for additional details about the rulemaking and how to participate during the comment period.

Attached to this e-mail are the public notice and factsheet.

From:	Frost, Brad
To:	EPA.620.rulemaking
Bcc:	epfume@comcast.net; ejhubertz@wustl.edu; jane@rebuildswi.org; willcountyenvironmentalnetwork@hotmail.com; willccare@gmail.com; imoreno@nrdc.org; williamrable@charter.net; planetlvr22@gmail.com; darleneschambers@gmail.com; aeowyn1@gmail.com; jrosko@wm.com; libby.reuter@charter.net; bobpashos@gmail.com; markscoats@yahoo.com; dien.bruce@gmail.com; c_budzinski@hotmail.com; dvdgpittman@gmail.com; gerryt1@comcast.net; attorney@johntbrady.com; luner@sbcglobal.net; blb2409@yahoo.com; ldisbrow@wm.com; pate82000@yahoo.com; patriciawagner1963@gmail.com; piperpetrocelli@gmail.com; uscrows@gmail.com; sher2dear@gmail.com; Harleyflight@att.net; aliceenglebretsen@gmail.com; aliciahenry228@gmail.com; services@staterepcarolammons.com; skinnera@danville118.org; bergeron.ann@gmail.com; chrismain1219@gmail.com; cwbullard3@comcast.net; conniejcunningham@gmail.com; dottydetorres@gmail.com; lohrbergj@gmail.com; jefffran4@aol.com; joeandsherry64@yahoo.com; jmhurd56@gmail.com; kggdiver@gmail.com; lois2@comcast.net; greenchi.yes@gmail.com; mcraig5774@gmail.com; ntgoodall@gmail.com; rhondaselvis@gmail.com; soellis72@gmail.com; steve.ogle@comcast.net; stuartnlevy@gmail.com; sjmisner@msn.com; suzanne56smith@gmail.com; thawisher@gmail.com; farmertom79@gmail.com; anthony.c.heath@gmail.com; vincekoers@aol.com; eaglewayne25@aol.com; david.main@carle.org; nireo72@hotmail.com; jpseymour1954@gmail.com; dylanjblke1@gmail.com; chynedog@hotmail.com; colinmarcbyers1@gmail.com; Peggy@faithinplace.org; guadalupe080400@gmail.com; hmania5787@gmail.com; business@offrte29.net; carolyntrimble1@gmail.com; germainelight53@gmail.com; cindy@faithinplace.org; Kara.S.Coats@usace.army.mil
Subject:	Draft Proposed 35 Ill. Adm. Code 620
Date:	Wednesday, May 12, 2021 3:46:00 PM
Attachments:	Notice 620.pdf Factsheet 620.pdf

The Illinois EPA is proposing changes to 35 Ill. Adm. Code 620. Prior to filing with the Illinois Pollution Control Board, the Illinois EPA will accept comments on the draft proposed rules. Written comments must be received by the Illinois EPA by June 25, 2021. Comments must be submitted to EPA.620.rulemaking@illinois.gov.

Additionally, the Illinois EPA will host a virtual public meeting to review the proposed changes and answer questions concerning the proposal. The meeting will be held at 1:00 pm on May 26, 2021. The public notice contains additional details about the comment period and meeting.

See the 620 website for additional details about the rulemaking and how to participate during the comment period.

Attached to this e-mail are the public notice and factsheet.

Electronic Filing: Received, Clerk's Office 3/08/2022

From:	Frost, Brad
To:	EPA.620.rulemaking
Bcc:	Cathleen.m.collins.civ@mail.mi; thecomptons311@comcast.net; rkohlhase@fw.com; dunmire@ilrwa.org; cgrieves@baxterwoodman.com; jacobsen_K@cityofelgin.org; Ted.Meckes@cwlp.com; JDonahue@northparkwater.org; elvfam@wowway.com; bmartin2@ameren.com; amessina@heplerbroom.com; kellyspivey@springnet1.com; Jmartin2@mmm.com; EXT Griffith, Donovan; jmore@schiffhardin.com; maureen.sullivan18.civ@mail.mil; president@illinoisfirefighters.org; jmnorman@htc.net; Ettinger.Albert@gmail.com; cindy.skrukrud@sierraclub.org; EXT Morphew, James; colleen@ilenviro.org; weibel@isgs.illinois.edu; SHKuykendall@mchenrycountyil.gov; LLurkins@ilfb.org; jeanp@ifca.com; j.agnoletti@bacog.org; EXT Darin, Jack
Subject:	Draft Proposed 35 Ill. Adm. Code 620
Date:	Wednesday, May 12, 2021 3:50:00 PM
Attachments:	Notice 620.pdf Factsheet 620.pdf

The Illinois EPA is proposing changes to 35 Ill. Adm. Code 620. Prior to filing with the Illinois Pollution Control Board, the Illinois EPA will accept comments on the draft proposed rules. Written comments must be received by the Illinois EPA by June 25, 2021. Comments must be submitted to EPA.620.rulemaking@illinois.gov.

Additionally, the Illinois EPA will host a virtual public meeting to review the proposed changes and answer questions concerning the proposal. The meeting will be held at 1:00 pm on May 26, 2021. The public notice contains additional details about the comment period and meeting.

See the 620 website for additional details about the rulemaking and how to participate during the comment period.

Attached to this e-mail are the public notice and factsheet.

From:	Frost, Brad
To:	EPA.620.rulemaking
Bcc:	james.r.hartman2@usace.army.mil; robert.dalzell.1@us.af.mil; mahalingam.ravichandran@us.af.mil; laurie.mitchell@us.af.mil; aubrey.m.higginbotham.mil@mail.mi; Dan.Petersen@erm.com; David.Klatt@jacobs.com; Denice.Nelson@erm.com; Elsie.Millano@erm.com; Jean.oliva@TRCcompanies.com; jleed@leedenvironmental.com; JVarsho@Geosyntec.com; GrabsJC@cdmsmith.com; Marcus.Byker@obg.com; narendra.prasad@wecenergygroup.com; Patrick.dunne@stantec.com; Patrick.Kenny@wecenergygroup.com; Susan.Smith@agrati.com; thomas.mroz@valero.com; Thomas.Hahne@tetratech.com; Henry.Stremlau@chevron.com; KPhillips@ene.com; Joseph.a.abel@exxonmobil.com; Wilmer.Reyes@cbs.com; Ray.Mastrolonardo@tetratech.com; Chit.Christian@tetratech.com; MONIQUE.M.LARRIVA@leidos.com; Richard.A.Kennard@usace.army.mil
Subject:	Draft Proposed 35 Ill. Adm. Code 620
Date:	Wednesday, May 12, 2021 3:51:00 PM
Attachments:	Notice 620.pdf Factsheet 620.pdf

The Illinois EPA is proposing changes to 35 Ill. Adm. Code 620. Prior to filing with the Illinois Pollution Control Board, the Illinois EPA will accept comments on the draft proposed rules. Written comments must be received by the Illinois EPA by June 25, 2021. Comments must be submitted to EPA.620.rulemaking@illinois.gov.

Additionally, the Illinois EPA will host a virtual public meeting to review the proposed changes and answer questions concerning the proposal. The meeting will be held at 1:00 pm on May 26, 2021. The public notice contains additional details about the comment period and meeting.

See the 620 website for additional details about the rulemaking and how to participate during the comment period.

Attached to this e-mail are the public notice and factsheet.

View up to date information on Illinois' Covid-19 vaccine plan and vaccination eligibility from the State of Illinois Coronavirus Response Site

Illinois Environmental Protection Agency

Public Hearings And Other Notices

Public Hearings and Other Notices

Notices published prior to January 1, 2016 are available on the archive pages by year. See the Section Navigation Menu at the right.

Search:

Subject	Hearing Date	Available Documents	Comment Period Ends	Posted
MMAC Measurement Subcommittee	May 14, 2021	Meeting Notice		May 12, 2021
Environmental Protection Trust Fund Committee Meeting	June 10, 2021	Meeting Notice		May 12, 2021
Draft Proposed 35 Ill. Adm. Code 620	May 26, 2021	Comment Period and Meeting Notice Factsheet	June 25, 2021	May 12, 2021
MMAC Infrastructure Subcommittee	May 6, 2021	Meeting Notice		May 3, 2021
Materials Management Advisory Committee	April 27, 2021	Meeting Notice/Agenda/Minutes		April 26, 2021

ILLINOIS EPA PUBLIC NOTICES
General Public Notices
Bureau of Air Public Notices
NPDES Permit and Hearing Notices
NPDES Settlement Public Notices
401 Water Quality Certifications and Notices
Non-coal Mines Notifications
Public Water Supplies Notifications
Pesticide Application Notifications
Domestic Lagoon Wastewater Facilities
Hydrostatic Testing of Pipelines and Tanks
2015

From:	Frost, Brad
To:	Bailey, Sabrina; Guy, Jeff; Terranova, Sara
Subject:	FW: Draft Proposed 35 Ill. Adm. Code 620
Date:	Monday, May 17, 2021 10:48:20 AM
Attachments:	Factsheet 620.pdf Notice 620.pdf

Please add to the mailing list

From: Alec Messina <amessina@ilchamber.org>
Sent: Monday, May 17, 2021 10:40 AM
To: Frost, Brad <Brad.Frost@Illinois.gov>
Subject: [External] Fw: Draft Proposed 35 Ill. Adm. Code 620

Morning, Brad! Can you add me to this email list moving forward?

Thanks

Alec

From: Alec Davis <adavis@ierg.org>
Sent: Monday, May 17, 2021 10:36 AM
To: Alec Messina <amessina@ilchamber.org>
Subject: FW: Draft Proposed 35 Ill. Adm. Code 620

External Email

Alec M. Davis
Executive Director
Illinois Environmental Regulatory Group
215 E. Adams St.
Springfield, IL 62701
217-522-5512 x 289
www.ierg.org

From: Frost, Brad <Brad.Frost@Illinois.gov>
Sent: Wednesday, May 12, 2021 3:42 PM
To: EPA.620.rulemaking <EPA.620.rulemaking@Illinois.gov>
Subject: Draft Proposed 35 Ill. Adm. Code 620

The Illinois EPA is proposing changes to 35 Ill. Adm. Code 620. Prior to filing with the Illinois Pollution Control Board, the Illinois EPA will accept comments on the draft proposed rules. Written comments must be received by the Illinois EPA by June 25, 2021. Comments must be submitted to EPA.620.rulemaking@illinois.gov.

Additionally, the Illinois EPA will host a virtual public meeting to review the proposed changes and answer questions concerning the proposal. The meeting will be held at 1:00 pm on May 26, 2021. The public notice contains additional details about the comment period and meeting.

See the 620 website for additional details about the rulemaking and how to participate during the comment period.

Attached to this e-mail are the public notice and factsheet.

ILLINOIS ENVIRONMENTAL PROTECTION AGENCY
1021 NORTH GRAND AVENUE EAST, P.O. BOX 19276, SPRINGFIELD, ILLINOIS 62794-9276 • (217) 782-3397
JB PRITZKER, GOVERNOR JOHN J. KIM, DIRECTOR

35 Ill. Adm. Code 620; Groundwater Quality
Pre-Filing Public Comment Period
Factsheet and Overview of Proposed Changes

Draft Proposed Rules

The Illinois EPA is proposing draft language to update 35 Ill. Adm. Code 620. The proposed updates include nine new chemicals, three new atrazine metabolites, and procedures for selecting toxicity values consistent with current federal guidance. Definitions are updated and references are consistent with those criteria and practices as incorporated. Site specific groundwater standards for designated Class III Special Resource Groundwater are also added. Exposure factors are updated, and the Human Non-Threshold Toxicant Advisory Concentration model is updated. Tables for similar-acting constituents are added. Finally, this proposal includes groundwater quality standards for five Per- and Polyfluoroalkyl Substances (PFAS): perfluorooctanoic acid (PFOA), perfluorooctanesulfonic acid (PFOS), perfluorononanoic acid (PFNA), perfluorohexanesulfonic acid (PFHxS), and perfluorobutanesulfonic acid (PFBS).

A summary of the key provisions is below. More information concerning the draft proposed rule may be found at
https://www2.illinois.gov/epa/about-us/rules-regs/water/Pages/620-Groundwater-Quality.aspx

Public Comment

Prior to submitting proposed rules to the Illinois Pollution Control Board for review and final adoption, the Illinois EPA is entertaining public comment on draft proposed rules. The Illinois EPA will accept written public comment until **June 25, 2021**. Comments should be submitted to **EPA.620.rulemaking@illinois.gov**

All comments, including proposed alternative language, received by Illinois EPA will be considered prior to the Agency filing the proposed rule with Illinois Pollution Control Board. Questions about the process or rulemaking should be submitted to the e-mail address above.

Public Meeting

The Illinois EPA will host a virtual public meeting to review the proposed changes and answer questions concerning the proposal. **The meeting will be held at 1:00 pm on May 26, 2021.**
The meeting link is:
https://illinois.webex.com/illinois/j.php?MTID=m19e9dc943bb9f835453fc6b6e8823826
Computer and telephone connection instructions are provided at the bottom of this Notice. If you have questions about connecting to the meeting, contact Jeff Guy at (217) 785-8724 or by submitting an e-mail to **EPA.620.rulemaking@illinois.gov.**

Key Provisions

1. Updates the methodology located in Appendix A for developing oral reference doses (RfDs), when a verified RfD is not available. The updated methodology is the method used by U.S. EPA Integrated Risk Information System (IRIS), the Tier 1 source for selecting toxicity criteria.
2. Provides the hierarchy for selecting a verified RfD from various sources. The hierarchy is in Appendix A.
3. Updates the Exposure Factors used in the Human Threshold Toxicant Advisory Concentration (HTTAC) equation and the Human Non-Threshold Toxicant Advisory Concentration (HNTAC) equations for both carcinogens and mutagens to be consistent with the U.S. EPA Exposure Factors Handbook (2011) and U.S. EPA Regional Screening Level calculator. Updates the exposure population from an average adult to a child ages 0-6 years for the HTTAC equation.
4. Updates Class I groundwater quality standards in tables at Part 620.410, based on updates to toxicity values, exposure factors and other methodologies.
5. Updates Class II groundwater quality standards in tables at Part 620.420, based on updates to Class I groundwater quality standards and updates to treatment factors, based on updates to dimensionless Henry's Law Constants when calculated at 20 ^{o}C and organic carbon partition coefficients.
6. Establishes groundwater quality standards for nine new chemicals, adds three metabolites as a mixture to atrazine, and moves atrazine and its metabolites tables to Part 620.410(c)(2) and Part 620.420(c)(2) for complex mixtures. Combines Radium 226 and 228 to form CASRN 7440-14-4: Radium (combined 226+228), updates the Class I groundwater quality standard for radium (combined 226+228) to an updated standard of 5 pCi/L, equal to the U.S. EPA Drinking Water MCL, and adds a Class II groundwater quality standard for radium (combined 226+228) at Part 620.420(a)(2). Establishes a Class II groundwater quality standard for silver and adds it to the table at Part 620.420(a)(2).
7. Updates constituent tables to include Chemical Abstract Services Registry Numbers (CASRNs) as additional identifiers for the constituents.
8. Adds footnotes to tables identifying the sources or methods for determining the groundwater quality standards.
9. Removes the explosive constituents at Parts 620.410(c) and 620.420(c); integrates the constituents into Parts 620.410(b) and 620.420(b).
10. Adds Appendix E, providing tables for similar-acting non-carcinogenic constituents by health effect (Table A) and similar-acting carcinogen constituents by cancer effect (Table B).
11. Updates the names of eleven constituents.
12. Adds carcinogen designations for four existing chemicals and one new chemical.
13. Adds mutagen designations for eleven chemicals.
14. Updates toxicity values for the constituents whose groundwater quality standards are based on the Human Threshold Toxicant Advisory Concentration (HTTAC) equation for noncarcinogens or the Human Nonthreshold Toxicant Advisory Concentration (HNTAC) equation for carcinogens.

A detailed list of Key Provisions can be found at
https://www2.illinois.gov/epa/about-us/rules-regs/water/Pages/620-Groundwater-Quality.aspx

Proposed Changes to 620 Sub Part A-C

Sub Part	Section	Proposed Changes
Part A	**620.110**	Adds definition of "Chemical Abstract Service Registry Numbers (CASRN)", "Lowest Concentration Minimum Reporting Level", and "Mutagen". Updates definition of "Carcinogen" to be consistent with updates to terminology used by U.S. EPA Integrated Risk Information System, and definition of "Detection" to language currently used in test methods. Removes the definition of "Practical Quantitation Level".
	620.125	Updates CFR references to most recent iteration of the code. Adds Illinois EPA "Integrated Water Quality Report and Section 303(d) List" and National Academy of Science "Water Quality Criteria" (1973) to incorporated references and updates several test methods. Adds references from the U.S. EPA Office of Research and Development, National Center for Environmental Assessment, and reference from U.S. EPA Office of Resource Conservation and Recovery. Updated for groundwater guidance from USEPA 2017.
Part B	**620.210**	Removes permeameter as an acceptable means to determine hydraulic conductivity. Adds the wellhead protection area of a community water supply well or well field as a specific area to which Class I groundwater quality standards are applicable.
	620.250	Lists a standard set of documentation that must be included with all groundwater management zone applications.
Part C	**620.302**	Adds to the list of examples of persons who do groundwater monitoring.
	620.310	Updates table at Part 620.310(a)(3)(A)(i) to include CASRN for each constituent; and removes para-dichlorobenzene and ethylbenzene from the table due to their updated carcinogen classification and the Board Note for 620.310(a)(3)(A). Adds a table at Part 620.310(a)(3)(A)(ii) depicting the constituents in the subsection; and removes *gamma*-HCH (*gamma*-hexachlorocyclohexane, lindane) and isopropylbenzene (cumene) due to their updated carcinogen classification and the Board Note for 620.310(a)(3)(A). Amends Board Note for 620.310(a)(3)(A) to revised outdated language.

Proposed Changes to 620 Sub Part D-F

Sub Part	Section	Proposed Changes
Part D	**620.410**	Adds Class I groundwater quality standards for nine new chemicals. Updates constituent tables to add CASRN for each constituent. Adds footnotes detailing the sources of the standards. Updates Class I groundwater quality standards as applicable. Removes explosive constituents table at 620.410(c) and integrates the constituents into table at 620.410(b). Moves atrazine from 620.410(b) to the complex chemical mixtures tables at 620.410(c) with the addition of atrazine metabolites.
	620.420	Adds Class II groundwater quality standards for nine new chemicals and two chemicals listed in 620.410 without prior Class II groundwater quality standards. Updates constituent tables to add a CASRN for each constituent, and update Class II groundwater quality standards as applicable. Adds footnotes detailing the sources of the standards. Removes explosive constituents table at 620.420(c) and integrates the constituents into table at 620.420(b). Moves atrazine from 620.420(b) to the complex chemical mixtures tables at 620.420(c) with the addition of atrazine metabolites.
	620.430	Establishes site specific Class III groundwater quality standards for chloride and pH at four dedicated nature preserves, which are caves, pursuant to 620.230(b). Establishes site specific Class III groundwater quality standards for chloride at two dedicated nature preserves, which are wetlands, pursuant to 620.230(b).
	620.440	Updates names of explosive constituents.
	620.450	Updates names of explosive constituents.
Part E	**620.510**	Requires that the 2009 Unified Guidance be used to determine background groundwater quality unless other methods are specified by regulation. Replaces the use of the PQL with the LLOQ, LCMRL or MDL, as appropriate to the nature of the chemical.
Part F	**620.601**	(b)-Updates code reference to 604.200.
	620.605	(b)(1) Designates the more stringent toxicity value of the (Human Threshold Toxicant Advisory Concentration (HTTAC) or Human Nonthreshold Toxicant Advisory Concentration (HNTAC) as the guidance value in the absence of a Maximum Contaminant Level (MCL) or Maximum Contaminant Level Goal (MCLG).
		(b)(2) Removes the Human Nonthreshold Toxicant Advisory Concentration (HNTAC) language and equation and relocates it to Appendix A.

Proposed Changes to 620 Appendices

Appendix	Section	Proposed Changes
A	**(a)**	Updates exposure factors representative of a child for the HTTAC model, which is consistent with Illinois Administrative Code Part 742 and U.S. EPA Regional Screening Levels (per capita daily water consumption = 0.78 liters per day, assumed average weight of a child 0-6 years of age = 15 kg).
	(b)(2)	Incorporates U.S. EPA's hierarchy of toxicity sources from "*Tier 3 Toxicity Value White Paper*", dated May 16, 2013, by U.S. EPA Office of Solid Waste and Emergency Response Human Health Regional Risk Assessors Forum (OSWER) for determining an appropriate verified oral reference dose.
	(b)(3)	Revises methodology used to calculate guidance values when a verified oral reference dose is not available to make language consistent with U.S. EPA Reference Dose Guidance.
	(b)(4)	Clarifies usage of uncertainty factors.
	(c)(1)	Adds equation for calculating HNTAC guidance level for chemicals designated as mutagens.
	(c)(2)	Updates equation for calculating HNTAC guidance levels for chemicals designated as carcinogens that are not designated as mutagens.
B	**(c)**	Removes language specific to mixtures of ortho-dichlorobenzene and para-dichlorobenzene, and 1,1-dichloroethane and 1,1,1-trichloroethane, and adds reference to Appendix E.
E		Provides tables of similar acting non-carcinogenic and carcinogenic constituents.

Illinois Environmental Protection Agency
Notice of Comment Period and Public Meeting
35 Ill. Adm. Code 620; Groundwater Quality

The Illinois EPA is proposing to update 35 Ill. Adm. Code 620: Groundwater Quality. The rules are the state standards that set acceptable levels for various pollutants in groundwater. Prior to submitting proposed rules to the Illinois Pollution Control Board for review and final adoption, the Illinois EPA is soliciting public comment on draft proposed rules.

The Illinois EPA will accept written public comment until **June 25, 2021**. Comments should be submitted to **EPA.620.rulemaking@illinois.gov.** All comments, including proposed alternative language, received by Illinois EPA will be considered prior to the Agency filing the proposed rule with Illinois Pollution Control Board. Questions about the process or rulemaking should be submitted to the e-mail address above.

The Illinois EPA will host a virtual public meeting to review the proposed changes and answer questions concerning the proposal. **The meeting will be held at 1:00 pm on May 26, 2021.**
The meeting link is:
https://illinois.webex.com/illinois/j.php?MTID=m19e9dc943bb9f835453fc6b6e8823826
Computer and telephone connection instructions are provided at the bottom of this Notice. If you have questions about connecting to the meeting, contact Jeff Guy at (217) 785-8724 or by submitting an e-mail to **EPA.620.rulemaking@illinois.gov.**

The proposed updates include nine new chemicals, three new atrazine metabolites, and procedures for selecting toxicity values consistent with current federal guidance. Definitions are updated and references are consistent with those criteria and practices as incorporated. Site specific groundwater standards for designated Class III Special Resource Groundwater are also added. Exposure factors are updated, and the Human Non-Threshold Toxicant Advisory Concentration model is updated. Tables for similar-acting constituents are added. Finally, this proposal includes groundwater quality standards for five Per- and Polyfluoroalkyl Substances (PFAS): perfluorooctanoic acid (PFOA), perfluorooctanesulfonic acid (PFOS), perfluorononanoic acid (PFNA), perfluorohexanesulfonic acid (PFHxS), and perfluorobutanesulfonic acid (PFBS).

More information concerning the draft proposed rule may be found at
https://www2.illinois.gov/epa/about-us/rules-regs/water/Pages/620-Groundwater-Quality.aspx

Meeting Connection Instructions

Cisco Webex Meeting Information
Date: Wednesday, May 26, 2021
Time: 1:00 p.m. CT
Meeting Number: 177 758 5798
Meeting Password: E2TePWPcg25

Connect by Computer

1. Select this link, which will direct you to the Webex webpage for the meeting:

https://illinois.webex.com/illinois/j.php?MTID=m19e9dc943bb9f835453fc6b6e8823826

2. Enter your information (name and address) and select “Join Now”. You may be prompted for a Meeting Number or Meeting Password, above.

3. An audio connection is required. The best connection option is “Call Me” (from the “Select Audio Connection” drop down, select “Call Me”). Input or select your telephone number.

Connect by Dial-in Phone

1. Call +1-312-535-8110

2. You will be prompted to enter the access code or meeting number. Enter the Meeting Number, above, and select the # sign.

Tips

- Find a quiet location with a power source for your device.
- Close all background applications or browser sessions.
- Reduce distractions and practice good meeting etiquette.
- Non-smartphone cellular (mobile) phones or landlines provide an audio-only experience.
- Smartphone, iPad or Tablets use the Webex mobile application.

From: Guy, Jeff
To: Terranova, Sara
Cc: Ankney, Clayton
Subject: RE: 620 outreach prep
Date: Thursday, May 13, 2021 3:52:00 PM
Attachments: image001.png

Yes, do you want to set up a Webex in mid-morning?

Jeffrey J. Guy
Illinois EPA
Office of Community Relations
(217) 785-8724
Jeff.Guy@illinois.gov

From: Terranova, Sara <Sara.Terranova@Illinois.gov>
Sent: Thursday, May 13, 2021 3:47 PM
To: Guy, Jeff <Jeff.Guy@Illinois.gov>
Cc: Ankney, Clayton <Clayton.Ankney@Illinois.gov>
Subject: RE: 620 outreach prep

Do you have time tomorrow?

From: Guy, Jeff <Jeff.Guy@Illinois.gov>
Sent: Thursday, May 13, 2021 3:05 PM
To: Terranova, Sara <Sara.Terranova@Illinois.gov>
Cc: Ankney, Clayton <Clayton.Ankney@Illinois.gov>
Subject: RE: 620 outreach prep

Hi Sara,

I agree it would be a good idea to go over a few things in advance of the call next week to discuss general logistics.

Jeffrey J. Guy
Illinois EPA
Office of Community Relations
(217) 785-8724
Jeff.Guy@illinois.gov

From: Terranova, Sara <Sara.Terranova@Illinois.gov>
Sent: Thursday, May 13, 2021 2:31 PM
To: Guy, Jeff <Jeff.Guy@Illinois.gov>
Cc: Ankney, Clayton <Clayton.Ankney@Illinois.gov>
Subject: RE: 620 outreach prep

Hi Jeff. Do we need to meet earlier than next Wednesday in order to confirm logistics for the public meeting on the 26th? If I need to be doing anything in the meantime – please just let me know!

Thanks,
Sara

From: Guy, Jeff <Jeff.Guy@Illinois.gov>
Sent: Thursday, May 13, 2021 9:12 AM
To: Terranova, Sara <Sara.Terranova@Illinois.gov>
Subject: RE: 620 outreach prep

Thank you.

Jeffrey J. Guy
Illinois EPA
Office of Community Relations
(217) 785-8724
Jeff.Guy@illinois.gov

From: Terranova, Sara <Sara.Terranova@Illinois.gov>
Sent: Wednesday, May 12, 2021 2:57 PM
To: Guy, Jeff <Jeff.Guy@Illinois.gov>
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Subject: 620 outreach prep

Hi Jeff. I just wanted to touch base with you regarding the outreach meeting on Wednesday, May 26, 2021 at 1pm. I plan to set up at least one pre-meeting with the workgroup to discuss the logistics of the meeting, who will be required to be present (versus calling in), what room we have reserved, the format of questions/comments, and any early comments we might receive. If you need anything from me in the meantime or have any additional thoughts on the process, please feel free to reach out! And welcome to the workgroup!

Thanks,
Sara

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