BOARD OF WATER COMMISSIONERS

MINUTES FOR THE MEETING OF
September 22, 2020

The statutory requirements as to notice having been complied with, the meeting was convened at 5:07 PM.

Members Present: Chairman Joshua M. Fox, Commissioner Robert H. Sheldon and Commissioner Robert E. Boyd.

Members Absent: None

Others Present: Treasurer Thomas S. Travers, Office Manager/Assistant Treasurer Karen A. Moretti, Executive Director Vincent J. Roy, and via Zoom; Blake Martin, Senior Associate of Weston and Sampson Engineers.

Commissioner Sheldon called the meeting to order.

1. REVIEW MEETING MINUTES

The Commissioners approved the Minutes for the Meeting of September 8, 2020 as amended. The motion for approval was made, duly seconded, and approved by unanimous roll call:

Joshua A. Fox, Chairman
Robert H. Sheldon, Commissioner
Robert E. Boyd Jr., Commissioner

2. EMERGING CONTAMINANTS RESPONSE PLANNING REPORT/Q&A W/ BLAKE MARTIN:

The attached report was presented by Blake Martin of Weston & Sampson Engineers on the Emerging Contaminant Response Planning Report that addresses potential District approaches to PFAS contamination. Commissioner Sheldon asked Mr. Martin if it was possible for something in the treatment plant process to cause an increase in PFAS levels. Mr. Martin stated that he was not aware of anything at this time, but he recommended that the District continue to monitor and beware of rising trends in PFAS analytical results. Mr. Roy informed the Board that the District had not been awarded funding from a recent PFAS Treatment Grant submittal. Mr. Roy emphasized that the District would resubmit a new application when new funding becomes available. Mr. Martin recommended that District staff schedule a site visit at a recently constructed PFAS treatment facility located in Devens, Massachusetts.

3. REVIEW DIRECTORS SEPTEMBER 4TH REPORT

Director Roy presented his bi-weekly report to the Board and addressed questions regarding several topics:

Proposed Eversource/DCR Project:
Chairman Fox asked Mr. Roy if had new information regarding the project to which Mr. Roy stated that he had not yet spoken with District Counsel, but would this coming week.

2018 JCB Backhoe:
Director Roy informed the Commissioners that the 2018 JCB Backhoe had been experiencing on-going hydraulic problems when operating the backhoe portion of the machine. He stated that since December 2018 the machine had been serviced several times to fix the problem. Mr. Roy stated that in an attempt to resolve the issue he had organized several meetings, site visits and phone conversations with the dealer, Northland JCB. The dealer agreed to reimburse the District the entire purchase price ($122,000) for the backhoe and apply it toward the purchase price ($132,000) of a new 2021 JCB Backhoe. The Commissioners agreed to authorize Director Roy to move forward with the transaction. Treasurer Travers requested that Mr. Roy consult with District Auditor, Richard Hingston of Guisti and Hingston CPA so that proper accounting procedures for the transaction are followed.

4. SUDBURY CROSSING SHOPPING CENTER – PRESENTATION FOR PROPOSASED WASTEWATER SYSTEM:

Mr. Roy reported to the Commissioners that he had conducted reference checks of three separate Nitrix wastewater treatment systems and that all facilities performed as designed, meeting regulatory requirements. Mr. Roy also stated that he discussed the permitting process with MassDEP officials from both the Western and Northeast Regional offices. Both offices reported that the Nitrix system ranks high as an innovative alternative to conventional activated sludge systems. Mr. Roy stated that MassDEP Environmental Analyst Marybeth Chubbs informed him that she was working closely with the Town's Health Department regarding the approval process and that she would need an

official letter from the District on their recommendation of the proposed system. Chairman Fox motioned to authorize Mr. Roy to make recommendations to the Health Department in support of the proposed project, subject to MassDEP approval, appropriate scrutiny, and a monitoring well. The motion was seconded by Commissioner Sheldon and approved by unanimous roll call:

Joshua A. Fox, Chairman
Robert H. Sheldon, Commissioner
Robert E. Boyd Jr., Commissioner

Chairman Fox moved to conclude the open meeting and to move into executive session for the purpose of discussing deployment strategies with respect to cyber security and to not return to open meeting. The chair confirmed that having discussion in an open meeting may have a detrimental effect on infrastructure security pursuant to General Laws, Chapter 30A, §21(a) (exception 4). The motion was seconded by Commissioner Sheldon and approved by unanimous roll call:

Joshua A. Fox, Chairman
Robert H. Sheldon, Commissioner
Robert E. Boyd Jr., Commissioner

There being no further discussions the meeting adjourned at 6:45 PM.

55 Walkers Brook Drive, Suite 100, Reading, MA 01867
Tel: 978.532.1900

September 8, 2020

Mr. Vincent Roy
Executive Director
Sudbury Water District
P.O. Box 111
Sudbury, MA 01776

Re: Emerging Contaminants Response Planning

Dear Mr. Roy:

Increasing awareness and increasingly restrictive environmental regulation have brought emerging contaminants to the forefront of concern for many water systems. Although numerous water quality requirements are placed on water utilities, an area of uncertainty exists regarding current and future emerging contaminants. Most recently this discussion has focused on a class of over 4,000 chemicals called Per and Poly-fluoroalkyl substances (PFAS), described as the "forever" chemicals, due to their stability and persistence once introduced into the natural environment. This class of chemicals has a history of an evolving and rapidly changing landscape relative to drinking water guidelines and standards. Thus, planning for water utilities requires an understanding of risk and cost. The Sudbury Water District, in a commendable proactive measure, has undertaken this planning effort to determine potential current and future actions to minimize costs while maintaining the safe production capacity of the water system.

The State of Massachusetts has recently closed the public comment period (February 28, 2020) for draft regulations establishing a final regulatory limit or Maximum Containment Level (MCL) under the Massachusetts Drinking Water Program, Massachusetts Department of Environmental Protection. This report provides both a background and understanding of PFAS impacts, and it develops an assessment of potential impacts to the Sudbury Water District's system in the context of historical changes and proposed regulatory efforts. The following sections present preliminary assessments for Sudbury's sources of supply, estimated infrastructure needs to manage any potential future impact, and recommended actions to minimize disruption of source production in the District's water system.

Background on Emerging Contaminants

Under the Safe Drinking Water Act, the US EPA undertakes an ongoing evaluation of water quality in potable drinking water sources in the United States. Part of this process is to ascertain the presence and impacts of certain chemical compounds and assess their health effects. In general, US EPA's efforts result in policies, guidelines, standards, and regulations. Although complex and often confusing, the intent is to eliminate undue exposure to harmful chemicals based on sound science and economic feasibility for managing or treating these chemicals.

This ongoing process has resulted in three rounds of testing for lists of chemical compounds designated under the Unregulated Contaminant Monitoring Rule (UCMR). The third round of national testing (including the Sudbury Water District) was completed in 2014 and is commonly referred to as the UCMR3 list. While systems over 10,000 people served were tested for over 600 compounds, excessive focus has been placed on two compounds, Perfluorooctanoic Acid (PFOA), and Perfluorooctane Sulfonate (PFOS), both of which are within the PFAS class of chemicals.

The UCMR3 testing indicated that, of the drinking water supplies tested, approximately 2% had PFOS and PFOA total concentrations above 20 parts per trillion (ppt) or nanograms per liter (ng/l). At the time close to the testing, US EPA had set a health advisory level of 400 and 200 ppt, respectively, for these two compounds. Subsequent evaluation of these compounds led to a more conservative Lifetime Health Advisory Level of 70 ppt for the combined concentrations of PFOS and PFOA. It should be noted that the Lifetime Health Advisory Level, while defining a maximum recommended exposure concentration, does not constitute a regulatory value or enforcement standard.

Analysis of the District's water in 2014 under UCMR3 failed to show the presence of these compounds in source water for Sudbury within the analytical detection limits at that time.

US EPA has not yet set a drinking water standard, often referred to as a Maximum Contaminant Level (MCL), for PFAS compounds in drinking water (either in total, as individual compounds, or combinations of individual compounds). The Lifetime Health Advisory Level left state regulators in a quandary regarding all PFAS compounds, not just PFOS and PFOA. Without a promulgated federal standard, states were left to develop their own toxicological assessments and determine guidelines, standards, or advisories on their own.

In addition to the chaotic scramble by each state, laboratory methods, equipment and comparative lab standards were not thoroughly developed and continue to evolve to this day, with lower and lower achievable detection limits. Much of this information and the rapidly changing approaches by each state to PFAS compounds are tracked by the Interstate Technology and Regulatory Council (ITRC) (www.itrcweb.org), a national non-profit organization. Rhode Island, Texas, New Hampshire, New Jersey, and some other states continue to set lower and lower standards for individual PFAS compounds, or combined concentrations of more than one compound. Massachusetts has embarked on the process to set a State MCL under the State Drinking Water Standards. The State's efforts are discussed below.

Introduction to PFAS for Sudbury

Of the current list of emerging contaminants, PFAS have received an excessive amount of press coverage and have been the subject of alarming regulatory changes. With laboratory reporting limits in the parts per trillion, drinking water regulators appear to be lowering drinking water standards in many states across America. Although epidemiological data for many, if not all the chemicals (some 4,000 chemical compounds designated as PFAS) is relatively incomplete, there is evidence that some PFAS compounds may bioaccumulate in the human body. Currently, two advisory levels frame the discussion for drinking water utilities in Massachusetts. They are:

1. US EPA has set a Lifetime Health Advisory Level (HA) of 70 ppt for the sum of PFOA and PFOS, two of the many PFAS compounds.
2. Massachusetts DEP has adopted groundwater standards to guide site clean-up activities under the Bureau of Waste Site Cleanup. This GW-1 category standard is 20 ppt for the sum of six PFAS compounds

While the Bureau of Waste Site Cleanup efforts impact contaminated land areas under the Massachusetts Contingency Plan, and are not the same as drinking water standards, the Waste Site Cleanup standards reflect the potential for future drinking water standards. Comment letters under the current regulatory review from Massachusetts Water Works Association (MWWA) indicate the vagaries of lab testing results, and many of the issues related to setting potable drinking water standards (i.e. treatment issues, costs, incomplete scientific evidence of harm, etc.). (See MWWA letter Attachment A.) Further, comments from risk assessors and toxicologists such as Green Associates (see Attachment B Couch/Green comments on ATSDR Toxicological Profile) indicate that unwarranted safety factors may be applied, not based on science, but rather as a reflection of public fear. The low laboratory detection limits, variability of results, and the widespread use of PFAS compounds in so many commercial products result in their ubiquitous presence in our built and natural environments.

Nonetheless, MassDEP sent letters to municipal water supply systems indicating that the Office of Research Standards and Guidelines (ORSG) had changed the guidelines for drinking water to 70 ppt for the sum of the following five compounds of concern:

- PFOA – Perfluoroctanoic Acid
- PFOS – Perfluoroctane Sulfonic Acid
- PFHxS – Perflurohexane Sulfonic Acid
- PFNA – Perfluorononanoic Acid
- PFHpA – Perfluoroheptanoic Acid

In December 2019, MassDEP proposed revisions to drinking water regulations to establish an MCL of 20 ppt for six compounds. This proposed MCL added the compound Perfluorodecanoic Acid (PFDA). In January 2020, an updated ORSG was established at 20 ppt for the six compounds (see ORSG upadate, Attachment C). While establishing an enforceable guideline, MassDEP has not yet promulgated an MCL or modified current drinking water regulations.

The key to understanding the impacts to water systems is that with laboratory analysis now able to report concentrations down to 2 ppt, trace levels of 6 compounds can quickly add or sum to a value exceeding 20 ppt.

The following feasibility analysis discusses the groundwater sources in Sudbury with respect to actions and feasible options that Sudbury could undertake should PFAS levels exceed standards in any source of supply. This memorandum starts with a background explaining the context of feasible options. It is helpful to categorize response planning into three general categories or scenarios. Proposed regulatory limits by DEP are consistent with the following general categories:

1. Detection of PFAS compounds between 2 (laboratory reporting limit) and 10 ppt.
2. Detection of PFAS compounds between 10 and 20 ppt.
3. Detection of PFAS compounds at 20 ppt and above.

All detections discussed assume cumulative concentrations for the 6 compounds. Detections below 2 ppt will not be considered as actionable levels. Although no current regulation requires action for levels above10 but below 20, MassDEP has often recommended monitoring and contingency planning. In many instances, utilities are being asked to determine the potential threats and/or sources of PFAS for their water supply. Proposed MCL's of 20 ppt, along with the ORSG values require mitigation actions to remove the source, provide alternate drinking water sources or treat the

affected drinking water source. The following discussion focuses on planning for third scenario where PFAS levels rise above 20 ppt in any given groundwater source.

Treatment Options and Response Actions

Drinking water treatment for PFAS in groundwater sources is generally limited to pressure filtration using granular activated carbon (GAC) or ion exchange resin. Membrane treatment using nanofiltration can also be employed but remains prohibitively expensive and does not solve the problem of how to dispose of volumes of backwash waste with high concentrations of accumulated PFAS. Advances in membrane filtration are occurring quickly but may be several years away before they are cost effective. Pressure filtration using GAC filters is usually accomplished with two vessels in series (a lead and lag configuration). This leaves the second vessel as a polishing vessel allowing the first vessel to be monitored for break-through of contaminants. Pressure vessels are most commonly found in 10 and 12-foot diameters. Based on a GAC contact time of 10 minutes, filters generally satisfy 450 and 600 gallons per minute (gpm) flow rates respectively. Applications of ion exchange resin media result in a highly varied vessel size and configuration throughout the industry. Due to the shorter required media contact times, vessels can be designed with a smaller footprint. Recent designs for ion exchange have shown a 4-foot diameter filter, 8 feet tall, can treat 200 gpm flows with shorter media contact time ranging between 2.5 and 5 minutes.

In addition, other actions must also be considered dependent on levels and exposure. These actions include:

- Public notification and outreach
- Source monitoring and forensic analysis within a watershed or aquifer
- Alternative source development
- Point of Use (POU) or Point of Entry (POE) treatment, and
- Supply of bottled water to end users.

Of these actions, the supply of bottled water and the installation of POU or POE systems are impractical for the number of domestic and commercial users supplied by the Sudbury Water District. Furthermore, some brands of bottled water have been found to contain PFAS compounds, therefore potentially not necessarily an improvement over a public water system containing PFAS compounds. Public notification, source monitoring and investigatory work, and identification of alternative sources (either alternate groundwater supplies or interconnections) are discussed in various scenarios below.

The Sudbury Water District's two major aquifers provide some degree of flexibility. The impacts of a contaminant on any source must be viewed through the lenses of system demand. In a low demand period, the loss of one source may not be considered an impact to system reliability.

However, during a high demand period the loss of one source or multiple sources could be catastrophic. In discussing both response actions and treatment options, consideration was given to:

- Source Capacity
- Site Construction issues
- General access, including emergency response issues and infrastructure upgrades
- Current or planned treatment systems already in place

The second general area that impacts treatment selection is general water chemistry. As treatment technology and methods (including media types and even membranes) advance, selection will be tailored to optimizing performance given the "type" or characteristics of that source's water quality beyond PFAS compounds. Specifically, current filtration media (either GAC or resins) are impacted by the presence of metals, with iron (Fe) and manganese (Mn) being prevalent throughout New England and Sudbury. In addition, the following constituents must be known prior to final process selection. These include:

- Total Organic Compounds (TOC)
- Chloride (CL)
- Sulfates (SO4)
- Nitrates, Nitrites (NO4, NO3)

Thus, when considering site constraints, capacity, and water quality, feasible options are relegated to the following broader categories with an associated defining description.

- Seasonal Treatment: This option is relegated to lower yield sources and /or poor current water quality. These types of sources are likely to be used only for summer, peak system demands.
- Emergency Treatment: This option is best suited for key sources in the system which are needed to maintain production capacity. Providing emergency treatment is also ideal if there are expected technology advances or longer-term infrastructure is planned, such as a new plant or a combination of the source with another source.
- Permanent Treatment: This option is discussed for sources that are high yield and/or may require PFAS removal and another treatment process. Source waters with multiple water quality impairments require optimization of processes, thus emergency treatment must be followed by pilot testing and design of long-term permanent solutions. Permanent treatment is important if alternative source development costs are excessive.

Treatment strategies for PFAS compounds are generally constrained by three variables: flow, water quality and infrastructure/space limitations. Each of these issues is discussed in greater detail below. While flow issues include individual source yield, pump capacities, and permitted withdrawals, a framework for treatment selection can be developed to incorporate all three issues. Water quality parameters affecting PFAS treatment include metals, chlorides, sulfate, and nitrate. In addition, dissolved solids and turbidity can be compounding factors. The reaction of various filter media to secondary drinking water constraints will dictate selection and performance of the media. Fortunately, the District's existing treatment facilities at its two plants would be located prior to PFAS treatment and therefore remove almost all constituents that could adversely affect performance of PFAS removal. The final variable deals with existing infrastructure design and available space at the current facilities. Further discussion of flow, water quality and infrastructure constraints is provided below.

Well Yield / Source Capacity

A review of the Annual Statistical Reports (ASR) from 2017, 2018 and 2019 was undertaken to characterize reliable capacity. The intent of the evaluation was to understand the difference in peak uses and average day demands. In addition, upper flow boundaries can be established by evaluating historical use, pump capacity and withdrawal limits under the Water Management Act.

Review of recent Annual Statistical Reports (ASR) indicates Sudbury Water District sources of supply produce roughly 500 to 606 million gallons per year (or 1.4 to 1.7 million gallons per day), and peak raw water production from these sources occurred in 2018 at 3.46 million gallons per day (MGD), which includes water used within the treatment system, primarily for backwashing. Maximum finished water production (water that flows to the distribution system) was 3.41 MGD for this period with a more recent peak daily production volume of 3.62 million gallons (occurred 6/18/2020, personal Communique with the Executive Director of the Sudbury Water District). Peak source production and peak finished water production may not occur on the same day due to available storage tank capacity of 6.3 million gallons. Given variable accuracy in meter readings, average day demands ranged from 1.4 to 1.66 MGD, which is well below the previous WMA permitted withdrawal of 2.08 MGD and the revised WMA permit value of 1.77 MGD. For planning purposes related to PFAS treatment, it is clear that the District must protect average day demands of 1.4 to 1.7 MGD while designing flexible PFAS treatment systems and/or operational changes to satisfy or reduce peak day demands of up to 3.5 MGD.

Because the yield comes from multiple sources a treatment strategy for the presence of PFAS generally requires either treating the average day demand or designing for peak well production capacity.

The presence of two treatment plants allows us to divide Sudbury's system into three categories: Raymond Road Water Treatment Plant, East Street Water Treatment Plan, and directly connected well sources. For the last three years the wells routed through the Raymond Road WTP included 2A, 7 and 9. Annually these wells produce approximately 1 to 1.1 MGD. ASR data indicated no production capacity until rehabilitation efforts at Well 4 were completed late in the summer of 2019. This well is treated locally with withdrawal rates at approximately 0.5 MGD. The wells contributing to the East Street WTP, 10, 3A and 8A, produce approximately 0.6 MGD on average, with well 3A contributing only minor amounts annually. Well 5 showed little to no use due to elevated levels of iron. Well 6 discharges directly into the distribution system with possible future plans allowing it to be routed through the Raymond Road WTP if necessary. Thus, the dominant production is gained from sources that pass through the Raymond Road WTP.

Further dissection of the individual well yields indicates that peak daily use for any individual well feeding the Raymond Road WTP was approximately 0.65 MGD, with the exception of Well 7, which is used more sparingly. Combining peak production rates provides a suggested design capacity of roughly 2.6 MGD for all sources at the Raymond Road WTP, as opposed to 0.65 MGD for any given source.

In contrast to that, the East Street WTP Wells 10, 3A and 8A have individual peak daily production closer to a range of 0.50 to 0.55 MGD. Combined peak capacity for Wells 10 and 8A is approximately 1.1 MGD but increases to 1.7 MGD if Well 3A is added for short durations. Limited use of Well 3A suggests its capacity should not be considered in this study. These treatment flow rates are important in sizing the PFAS treatment process for both individual wells and combined source flow rates.

The review of yearly use and operational data suggest that Well 5 remains only for emergency use. PFAS concentrations could be monitored at this well but planning for treatment of PFAS is not warranted given its limited benefit to the system. Well 3A appears to be the only other well that could

be considered for additional use once the planned SCADA system improvements are put into place. Currently being pumped at between 8 and 10 million gallons per year, this well is used sparingly and represents only a minor portion of the East Street WTP Well group. Thus, the planning for PFAS treatment at all other wells is dependent on individual source water quality. Where source water is free from elevated Fe, Mn or sulfates, nitrates and chlorides, treatment for PFAS prior to the current WTF's could be viable, Water quality impacts are discussed in the next section and emergency and permanent treatment scenarios are discussed in the final section.

Water Quality

In addition to PFAS concentrations, the general water chemistry plays an important role in the selection of treatment options. As stated previously, GAC and ion exchange are the favored treatment systems for PFAS at this time, but their performance is affected by the concentrations of other compounds and elements within the water entering PFAS treatment. The water quality evaluation for the District's sources focuses on chemistry affecting treatment. Although the discussion is theoretical, it is based on potential future PFAS concentrations rising above the proposed MCL at a groundwater source.

The Raymond Road WTP sources include Wells 2A, 7, and 9. Metals concentrations in Wells 2A, 7 and 9 are too high for PFAS treatment at the well head. Wells 4 and 6 have acceptably low concentrations of total iron and manganese, indicating PFAS removal could be accomplished at the well head. Water quality at Well 6 reveals slightly elevated total dissolved solids and chloride levels of approximately 110 to 120 mg/l. Well 4 does not appear affected by sodium and chloride levels but nitrate concentrations as high as 4.l mg/l have been identified in 2017. Recent pilot testing at sites in Massachusetts and New Hampshire indicates that resin media removal efficiencies can be impacted by chloride levels at or above 100 mg/l. Similarly nitrates and sulfates may reduce ion exchange resin performance. However, GAC is not affected by those concentrations of chlorides, nitrates, or sulfates. Unless pilot testing demonstrates that ion exchange would be a successful treatment option, GAC filtration would be the preferred filtration method should Well 4 or 6 be impacted by elevated PFAS levels. Emergency treatment and permanent treatment could be achieved for Wells 4 and 6 using very similar designs. This scenario is discussed in the next section.

The remainder of the wells feeding the Raymond Road WTP would require PFAS treatment placed after the water treatment plant. Assuming the combined flows of Wells 2A, 7 and 9 were to exceed state standards for PFAS, design flow rates for treatment would need to be 1.8 to 2.0 MGD. Because treated water typically has chloride concentrations of approximately 120 mg/l, ion exchange resins would be expected to be impaired, making GAC filtration the most cost-effective treatment option for the Raymond Road WTP sources.

The East Street WTP well sources include Wells 3A, 8A and 10. Improvements to Well 3 are expected to allow increased use and flow capacity. If the combined flows of Well 8A and Well 10 and Well 3 exceed state standards for PFAS, the PFAS treatment system would need to be based on a design flow rate of approximately 1.4 MGD. Current chloride concentrations in the plant effluent are about 91 mg/l which is low enough that ion exchange resins would be expected to work effectively. That means that the East Street WTP could be followed by PFAS treatment using either GAC media or resin media. Treatment scenarios are discussed in the next section.

Treatment Scenarios

Review of available data suggests the following with respect to a hypothetical increase in PFAS above 20 mg/l.

- Wells 4 and 6 water quality suggests direct source treatment with GAC would be viable for both emergency and permanent treatment
- Raymond Road WTP sources (Wells 2A, 7, and 9) would require metals removal prior to PFAS removal. Only Well 3 has iron and manganese concentrations low enough to consider isolated well head treatment. Elevated chlorides favor GAC as a preferred pressure filter media.
- At the East Street WTP, Wells 8A and 10 could accommodate GAC or resin filtration or a hybrid system using both in series. Only Well 3 has iron and manganese concentrations low enough to consider isolated well head treatment. Table I reflects this treatment scenario summary.

Table I

Option	Treatment	PFAS 20-40 ppt	PFAS 400–2000 ppt	PFAS >2000	Comments
Well 3A	Remove from system until upgrades complete	GAC	GAC	GAC/Resin	Flows may be separated at well head or at the Plant
Well 6	Emergency	Temp GAC	Temp GAC	Temp GAC	At wellhead or plant with historic piping connections
Well 4 and 6	Permanent	GAC	GAC	GAC/Resin	At wellhead or plant with historic piping connections
Wells 2A, 7, 9	Emergency-Permanent	GAC	GAC	GAC	Post Greensand removal for Fe and Mn
Wells 8A, 10	Emergency-Permanent	GAC	GAC/Resin	Resin	Post Greensand removal for Fe and Mn

Well 4 or 6 Treatment Scenario

If elevated PFAS levels occur at Well 4 or 6, pressure filtration using GAC filtration should be installed. Flow rates of up to 0.8 MGD suggest the use of 12-foot diameter filters with approximately 30,000 lbs. of carbon. An emergency system would likely consist of a single vessel placed at or near the well station. Alternatively, yard piping at the Raymond Road WTP could be modified and a filter could be placed to the rear of the plant and dedicated to PFAS treatment for Wells 4 and/or 6. Emergency systems can be housed in temporary structures to protect the system from freezing. A permanent system would require housing the filter in an enclosed structure. Because Wells 4 and 6 represent only a portion of the flow for the overall system, advocating for a single filter assembly for both emergency and long-term treatment with appropriate monitoring is warranted. These well sources could be temporarily shut down should maintenance be required or media change-out.

Anticipated costs for either Well 4 or Well 6 PFAS treatment with temporary housing would include the following:

Filter and Media	$225,000
Yard piping Modifications	50,000
Valving/SCADA/Electrical	25,000
Temporary Enclosure with treatment	68,000
Concrete Pad	25,000
Engineering	35,000
Start-up	25,000
Total	**$453,000**

Adding a permanent structure or facility at the individual well heads would increase the costs by approximately 200,000 to 250,000 dollars. Costs are presented per well.

Well 2A, 7 and 9 (Raymond Road WPT) Treatment Scenario

The District's system is highly dependent on the water from these wells. A scenario resulting in elevated levels of PFAS in these wells represents a significant impact to the system, because no other single set of sources produces the amount of water that these wells produce in combination. Emergency treatment for a portion or all of the capacity at the Raymond Road WTP is possible. However, MassDEP has required that permanent solutions with adequate filter redundancy be developed as soon as possible following an exceedance of the proposed MCL. Due to the limited space within the current facility, an external building or extension to the existing building would be necessary. For PFAS concentrations under 400 ppt, GAC has proven to be cost effective. Flows would need to be routed through a primary filter and a lag filter. Flows in the range of 1200 – 1400 gpm (up to 2 MGD) would require 12-foot diameter filters with a calculated empty bed contact time of 10 minutes, and each filter would require 40,000 pounds of carbon.

An alternate design would include primary GAC filters followed by ion exchange resin-filled pressure vessels. Resin media by Purolite, Calgon and Evoqua have shown great promise in Portsmouth, NH, and Devens, MA. These ion exchange vessels can be a smaller diameter and height, reducing overall plant footprint. When placed after iron removal and GAC, resin media should be unaffected by other

water quality parameters, with the exception of chlorides. Should the concentrations of PFAS exceed 400 ppt, extensive pilot testing is recommended prior to final treatment selection to minimize long-term operational costs.

Budget costs for PFAS removal at the Raymond Road WTP would be as follows:

2 pairs GAC vessels (4 total)	$1,100,000
Process Piping	250,000
Yard Piping	125,000
Controls, SCADA	60,000
Building	500,000
Electrical, HVAC	125,000
Engineering	235,000
Subtotal	**$2,395,000**
Total with contingency 20%	**$2,874,000**

Well 10 and 8A (East Street WTP) Treatment Scenario

The PFAS treatment scenario for the East St. Wells 10 and 8A is similar to Raymond Rd. yet on a reduced scale. Assuming Well 3A is not required or that its use would only occur if 8A or 10 were shutdown, design flows would be approximately 1.1 MGD. With the added flow from Well 3/3A, design flows would be 1.4 MGD. Although this rate is at the upper range for the standard 12-foot diameter filters, average flow rates would fall below this value. As is the case at the Raymond Road WTP, limited plant space dictates the placement of PFAS pressure filtration in a new structure. Oxidants used in the removal of iron and manganese would require neutralization if resin vessels were to be considered. Assuming concentrations below 400 ppt, GAC is recommended. When compared to the Raymond Road WTP full scale design, the reduced building footprint and a single pair of 12-foot diameter GAC filters should result in a 30-40% reduction in capital costs placing the facility costs at approximately 1.7 to 2.0 million dollars.

Budget costs for PFAS removal at the East Street WTP would be as follows:

1 pair GAC vessels (2 total)	$590,000
Process Piping	180,000
Yard Piping	100,000
Controls, SCADA	60,000
Building	400,000
Electrical, HVAC	100,000
Engineering	200,000
Subtotal	**$1,650,000**
Total with contingency 20%	**$1,980,000**

Conclusions and Recommendations

Detection of low levels of PFAS concentrations found in the Sudbury Water District wells indicates that there is no immediate need for installation of water treatment facilities to remove PFAS. However, the

presence of PFAS suggests a proactive plan be enacted to prepare the District in case concentrations increase. With that plan in place, the District will be able to respond quickly to the need for treatment, minimizing costs while maintaining production of reliable and safe drinking water. Production capacities suggest the focus of such a plan should be the Raymond Road WTP and the wells which feed it. A parallel, secondary effort can be prepared for the East Street WTP wells. While Well 4 or 6 could be treated with an emergency single filter should PFAS levels become unacceptable at those locations, the remaining wells require PFAS removal following the greensand filtration system at either of the existing WTF's. The following are specific recommended courses of action to prepare for the potential of elevated PFAS concentrations in the water supply:

1. Sudbury Water District should initiate investigative efforts for the source of PFAS and develop a potential monitoring system with appropriately placed sentry wells. This process should be initiated with the approval of MassDEP and the Bureau of Waste Site Cleanup in order to take advantage of any available funding.
2. For the wells at the Raymond Road WTP, develop preliminary/conceptual design plans for yard piping, controls, and housing of the filters. This design effort would evaluate existing piping layouts and evaluate well pump/system hydraulics.
3. For the wells at the East Street WTP, develop preliminary/conceptual design plans for yard piping, controls, and housing of the filters. This design effort would evaluate existing piping layouts and evaluate well pump/system hydraulics.
4. Sudbury Water District should apply for current design funding under the MassDEP program for PFAS Contamination. Although PFAS levels have not exceeded the proposed MCL of 20 ppt for 6 compounds, a grant application will indicate a proactive stance and provide justifications for any future funding requests.

We look forward to discussing the findings of this report in further detail with you. Please do not hesitate to contact me with questions.

Sincerely,
WESTON & SAMPSON ENGINEERS, INC.

Blake A. Martin
Vice President Water Resources

\\wse03.local\WSE\Projects\MA\Sudbury MA\PFAS Contingency Plan\Report\Letter Report 09082020.docx

Attachment A
MWWA Comment Letter

Commonwealth of Massachusetts
Executive Office of Energy & Environmental Affairs

Department of Environmental Protection
One Winter Street Boston, MA 02108 • 617-292-5500

Charles D. Baker
Governor

Karyn E. Polito
Lieutenant Governor

Matthew A. Beaton
Secretary

Martin Suuberg
Commissioner

Date: April 17, 2019

Dear Public Water Supplier:

This notice provides important updated information regarding MassDEP's efforts to address Per- and Polyfluoroalkyl Substances (PFAS) and provides guidance on health protective limits for these chemicals in drinking water.

MassDEP PFAS Updated Information

Per- and polyfluoroalkyl substances (PFAS) are a family of chemicals used since the 1950s to manufacture stain-resistant, water-resistant, and non-stick products. PFAS are widely used fire-fighting foams and in common consumer products as coatings, on food packaging, outdoor clothing, carpets, leather goods, ski and snowboard waxes, and more. PFAS in drinking water is an important emerging issue nationwide.

The United States Environmental Protection Agency (EPA) in 2016 published a drinking water Health Advisory Level for two of the PFAS compounds (Perfluorooctanesulfonic acid, PFOS, and Perfluoroocatanoic acid, PFOA) combined at 0.070 micrograms per liter (ug/L) or 70 parts per trillion (ppt). In June 2018, MassDEP issued an Office of Research and Standards guideline (ORSG) for drinking water of 70 ppt for PFOA, PFOS, PFNA (Perfluorononanoic acid), PFHxS (Perfluorohexanesulfonic acid) and PFHpA (Perfluoroheptanoic acid) combined. The three additional PFAS were included because they share very similar chemical structures and the available data indicates they are likely to exhibit similar toxicities. The ORSG was established to be protective against adverse health effects for all people consuming the water for a lifetime and is also applicable to shorter-term exposures of weeks to months during pregnancy and breast-feeding.

Based on the current ORSG, MassDEP has recommended that:

1) consumers in sensitive subgroups (pregnant women, nursing mothers and infants) not consume water when the level of the five PFAS substances, individually or in combination, is above 70 ppt; and,

2) public water suppliers take steps expeditiously to lower levels of the five PFAS, individually or in combination, to below 70 ppt for all consumers.

As part of the agency's efforts to address PFAS compounds, MassDEP has continued to review the current scientific information, studies and assessments on PFAS. Some of these include the federal Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profile for Perfluoroalkyls; EPA Health Effects Support and Drinking Water Health Advisory documents for PFOA and PFOS; the National Toxicology Program (NTP) Monograph, Immunotoxicity Associated with Exposure to PFOA; the December 2018 European Food Safety Authority publication on risks attributable to PFOS and PFOA in food; and recent published research and various assessments by agencies in other states.

Based on this evaluation, MassDEP is undertaking the following actions:

1) MassDEP is proposing draft amendments to the Massachusetts hazardous waste cleanup regulations (the Massachusetts Contingency Plan or "MCP"), that include groundwater and soil cleanup standards. Consistent with the proposed ORSG level described below, the proposed Method 1 GW-1 Standard – applicable to groundwater protected for its current and/or future use as drinking water – is 20 ppt for the 5 compounds noted above plus Perfluorodecanoic acid (PFDA)(six total).
2) MassDEP's Office of Research and Standards is convening its Health Effects Advisory Committee to provide scientific input on the technical basis of the proposed MCP standards and their implication regarding a potential revised ORSG with a limit of 20 ppt for the sum of the 6 PFAS compounds.
3) MassDEP also recently initiated the process to develop a drinking water standard for public drinking water systems, known as a Maximum Contaminant Level (MCL), for the six PFAS compounds. This forthcoming MCL will be promulgated under the Commonwealth's standard regulation development process, including a public comment period. MassDEP seeks to align the forthcoming MCL with the MCP GW-1 standard and any revised ORSG. In April 2019, the agency launched s stakeholder process to obtain input into the MCL development. Information on this MCL development effort, including information on upcoming stakeholder meetings, can be found at https://www.mass.gov/lists/development-of-a-pfas-drinking-water-standard-mcl.

MassDEP is accepting comment on the draft MCP regulations until July 19, 2019, including the proposed PFAS cleanup standards. Any comments received on the proposed MCP GW-1 standard will also be considered by the Department in the revision of the ORSG and the forthcoming MCL. MassDEP will keep all PWS informed on PFAS MCL development, including opportunity for public comment.

PFAS has multi-media impacts and MassDEP has initiated several actions across different media programs. These actions are described on our webpage at https://www.mass.gov/info-details/per-and-polyfluoroalkyl-substances-pfas and include the following:

- Requiring testing of drinking water sources that are proximal to known or potential sources of PFAS. As of today, MassDEP has been working with 13 of 1,729 public water systems to address PFAS related issues, and all systems with PFAS detects are shown on the webpage and all PFAS data is available on the data portal at https://eeaonline.eea.state.ma.us/portal#!/home .
- Testing new sources of drinking water during the source approval process.
- Approving laboratories for PFAS analysis.
- Conducting a fire-fighting foam [Aqueous Film-Forming Foam (AFFF)] collection and disposal program. As of April 2019, MassDEP and the Mass Dept. of Fire Services has collected and disposed of 149,000 pounds of pre-2003 foam.
- Initiated a program for Massachusetts permitted bottled water companies for voluntarily report PFAS testing of bottled water. See the current list of Massachusetts permitted Bottled Water companies that have voluntarily provided MassDEP with their results for posting at bottled water companies.
- Requiring the testing of residuals for PFAS. As of January 2019, MassDEP is requiring PFAS sampling & analysis for new and renewed land application approvals for residuals.

What should you do with this updated information?

If you have not been contacted by MassDEP's Drinking Water Program with specific instructions for your system, you do not have to take any action at this time. However, we encourage you to learn about PFAS, and to follow and engage in the PFAS MCL development process. See https://www.mass.gov/lists/development-of-a-pfas-drinking-water-standard-mcl for information on the PFAS MCL process. If you have been contacted by MassDEP's Drinking Water Program with specific requirements or recommendations for your system, follow the instructions provided.

For more information about PFAS in drinking water see:

- MassDEP webpage https://www.mass.gov/info-details/per-and-polyfluoroalkyl-substances-pfas
- MassDEP Fact Sheet – PFAS in Drinking Water: Questions and Answers for Consumers https://www.mass.gov/doc/massdep-fact-sheet-pfas-in-drinking-water-questions-and-answers-for-consumers
- USEPA's Drinking Water Health Advisories can be found at: https://www.epa.gov/ground-water-and-drinking-water/drinking-water-health-advisories-pfoa-and-pfos
- The Centers for Disease Control and Prevention's Public Health Statement for PFOS and PFOA can be found at: https://www.atsdr.cdc.gov/pfas/index.html
- For additional information on possible health effects, you may contact the Massachusetts Department Environmental Protection, Office of Research and Standards, at 617-556-1165.

We encourage you to read the MassDEP Factsheets: "PFAS in Drinking Water: Questions and Answers for Consumers" located at https://www.mass.gov/doc/massdep-fact-sheet-pfas-in-drinking-water-questions-and-answers-for-consumers and the attached "Per- and Polyfluoroalkyl Substances (PFAS) in Public Drinking Water Supplies - Questions and Answers for Public Water Suppliers". If you have any additional questions about PFAS in drinking water, such as whether you should test your water, please contact your MassDEP Drinking Water Program contacts below.

Region	Name	Phone #	Email
Western	Catherine Wanat	413-755-2216	Catherine.wanat@mass.gov
Central	Robert Bostwick	508-849-4036	Robert.Bostwick@mass.gov
Northeast	Amy LaPusata	978-694-3291	Amy.lapusata@mass.gov
Southeast	William Schwartz	508-946-2818	William.schwartz@mass.gov
Boston: Program.director-dwp@mass.gov or Margaret Finn 617-292-5746			

As we continue to work to address PFAS issues and develop a PFAS MCL, we will keep you informed.

Sincerely,

Yvette DePeiza
Program Director
Drinking Water Program
MassDEP/BWR

Attachment: MassDEP Fact Sheet "PFAS in Public Drinking Water Supplies, Questions and Answers for Public Water Suppliers"

ecc: MassDEP/DWP Regional Chief, MassDEP/ORS- Mark Smith, MassDEP/DWP Program Director
MDPH – Jana Ferguson, Jan Sullivan
Local Board of Health
Certified operator

Attachment B
Crouch/Green Comments on ATSDR Toxicological Profile

**Comments on Massachusetts Department of Environmental Protection's (DEP's)
groundwater and soil standards
for perfluoroalkyl substances (PFAS)
in the Department's proposed 2019 amendments
to the Massachusetts Contingency Plan**

Laura C. Green, Ph.D., D.A.B.T. and Edmund A.C. Crouch, Ph.D.
July 19, 2019

Introduction and Overview

The Massachusetts Department of Environmental Protection (MassDEP, 2019) proposes new standards for the sum of six perfluoroalkyl substances (PFAS):

- perfluorooactanoic acid (PFOA),
- perfluoroheptanoic acid (PFHpA),
- perfluorononanoic acid (PFNA),
- perfluorodecanoic acid (PFDA),
- perfluorooctane sulfonic acid (PFOS), and
- perfluorohexane sulfonic acid (PFHxS).

Unfortunately, MassDEP's proposed PFAS standards are not based on current evidence, but could and should be revised. Among other issues, MassDEP's currently proposed standards:

- Are not based on any reliable evidence of adverse effects in humans;
- Are instead based almost entirely on only two studies in rodents:
  - One study of PFOA in laboratory mice (Lau et al., 2006), in which minor, transient, developmental effects were reported; and
  - One study of PFOS in laboratory rats (Luebker et al., 2005) that reported "delayed eye opening" and reduced birth weights in neonates;
- Do not reflect well-established, marked differences in sensitivities to PFOA and other PFAS between and among laboratory rats, mice, monkeys, and humans;
- Ignore reliable, relevant evidence from controlled studies of PFOA and PFOS in laboratory monkeys; and
- Fail to account for recent, relevant, clinical and epidemiological studies of PFOA.

With regard to the first point, it remains the case that epidemiologic and/or clinical evidence has so far failed to establish that any PFAS harms human health at or near environmental exposure-levels (ATSDR, 2018). MassDEP should make this clear, but currently it does not.

Green Toxicology LLC www.GreenToxicology.com Green@GreenToxicology.com Crouch@GreenToxicology.com

High-level, experimental exposures to some PFAS do harm the health of laboratory animals, and it is entirely appropriate to base health-protective guidelines on exposure-response data derived from laboratory animal studies (in the absence of, or in addition to, usable exposure-response data from studies of humans).

Ideally, health-based guidelines and standards should be based on controlled studies of (i) humans, (ii) monkeys, and/or (iii) other laboratory mammals known to mimic humans with regard to relevant biological responses. Unfortunately, the two studies on which MassDEP rely are in none of these three categories.

In what follows, we present constructive criticisms of MassDEP's approach, and offer alternate bases for regulation. In particular, we show that the results from studies of PFOA and PFOS in laboratory monkeys can, and should, be used to derive highly protective, evidence-based "reference doses" (essentially, acceptable daily intakes), which in turn should be used to fashion regulations intended to protect public health, with an ample margin of safety.

The evidence-based, highly conservative, reference doses that we derive herein are 89 ng PFOA per kg body weight per day and 240 ng PFOS/kg-day. We also note that reference doses for other PFAS should be based on chemical-specific evidence.

Health-risks from PFOA

Based on minor, transient, developmental effects in CD-1 mice exposed to high doses of PFOA (Lau et al., 2006), U.S. EPA, California EPA, and others (Goeden et al., 2019) assume that this PFAS poses a risk of developmental toxicity to humans. And MassDEP, by extension, assumes the same for all of the six PFAS that it proposes to regulate, despite zero such evidence for at least four of these PFAS (all but PFOS, about which more below).

As it happens, the fundamental uncertainties in this assumption render these CD-1 mouse bioassay results entirely unsuitable for purposes of assessing risks to human health — even from exposures to PFOA, let alone from exposures to the other five PFAS of interest to MassDEP. Why did MassDEP rely on this single study in CD-1 mice, when, as explained below, controlled, reliable, and relevant studies of PFOA in monkeys have been peer-reviewed, published (Butenhoff et al., 2002, 2004a, and 2004b), and serve as much better predictors of effects in humans?[1]

[1] One answer is that MassDEP decided to simply accept U.S. EPA's (2016) reference dose at face value; despite the facts that EPA's derivation of its PFOA reference dose has not been peer-reviewed and has not been relied upon by EPA for standard-setting. Moreover, environmental guidelines and standards for PFOA, as established by various regulatory expert-groups internationally, *vary by 750-fold* (Dourson et al., 2019): this alone is indication that various analysts' assumptions and subjective judgments — rather than a set of objective, verifiable, unambiguous, health-effects data — are what drive these disparate, bottom-line numbers for "acceptable" exposures to PFOA.

Green Toxicology LLC www.GreenToxicology.com Green@GreenToxicology.com Crouch@GreenToxicology.com

The developmental (and many other) effects of PFOA in mice are mediated via the cell-nuclear hormone receptor, peroxisome proliferator-activated receptor alpha (PPARα; Abbott et al., 2012; Albrecht et al., 2013).[2] However, the activity-levels, structures, and functions of PPARα vary substantially among rodent-species and other animal-species; and, importantly, vary substantially between laboratory, "wild-type" mice (such as CD-1 mice) and humans (Bell et al., 1998; Corton et al., 2018). Abundant evidence indicates that rats and mice are highly susceptible to the effects (both adverse and beneficial) of chemicals (both endogenous and exogenous) that act via PPARα, while humans and other mammals — including guinea pigs, hamsters, rabbits, and monkeys — are relatively resistant to these effects (Klaunig et al., 2003 and 2012; Hoivik et al., 2004; Corton et al., 2018).

In addition to mice, laboratory rabbits have been used to assess the developmental effects of PFOA (Gortner et al., 1982). As just noted, rabbits can serve as faithful models for humans with regard to the actions of peroxisome proliferators on PPARα (Staels & Auwerx, 1998). In the relevant study, pregnant New Zealand White/Minikin rabbits were dosed with the ammonium salt of PFOA at 0, 1.5, 5, and 50 mg/kg-day on gestational days 6 through 18 (Gortner et al., 1982). The highest dose-rate, as expected, caused significant, temporary weight loss in the pregnant rabbits; but their fetuses at gestational day 29 showed zero indications of reproductive toxicity, embryotoxicity, or gross, skeletal, or internal malformations, or any other adverse effects, in *any* PFOA dose-group, including the highest.

MassDEP currently takes no notice of this important study. U.S. EPA also did not even mention this rabbit bioassay in its assessment of PFOA (U.S.EPA, 2016), which is surprising, since the study-report is included in EPA's Administrative Record.

Standard regulatory guidance (and common sense) dictates that when extrapolating results from developmental studies, health risk-assessors should rely on laboratory animal-species *that best mimic humans with regard to relevant biological mechanisms*. *Per* U.S. FDA (2017):

---

[2] PPARs are present in all animal-species, although with different forms in different species. As explained by Hall et al. (2012):

> PPARs regulate lipid and cholesterol metabolism through induction of (peroxisome proliferator response element (PPRE)) containing target genes resulting in increased beta-oxidation of fatty acids (Xu, Li, and Kong 2005). Natural ligands for PPARα include saturated and unsaturated fatty acids, eicosinoids, and linoleic acid metabolites. However, a diverse range of xenobiotics from many classes and structures are also able to activate PPARα such as the fibrate hypolipidaemic agents (clofibrate, fenofibrate, gemfibrozil amongst others), methaphenilene, thromboxane synthetase inhibitors, dehydroepiandosterone, non-steroidal anti-oestrogens, ibuprofen, Wy-14,643, diphenyl ether herbicides, and phenoxy herbicides (Greaves 2007).

Green Toxicology LLC www.GreenToxicology.com Green@GreenToxicology.com Crouch@GreenToxicology.com

> The rabbit has proven to be useful in identifying human teratogens that have not been detected in rodents; and the rabbit is routinely used as the non-rodent species based on the extensive historical background data, availability of animals, and practicality.

Importantly, the epidemiology on PFOA does not indicate that this chemical harms human development. As noted by ATSDR (2018):

> . . . most [epidemiological] studies found *no association* between maternal serum PFOA levels and the risk of low birth weight infants (typically defined as <2,500 g) . . . or found a *decreased* risk of low birth weight infants . . . [*emphasis* added]

And summarizing the literature on infant birth-weights *in the normal range*, ATSDR (2018) notes that although three sets of studies on women exposed to background concentrations *did* report inverse associations between maternal serum PFOA and birth weight, another *twelve similar studies found no such associations.*

Thus, although the CD-1 mouse data on the biological and toxicological effects of PFOA are of little-to-no relevance with regard to effects of PFOA on humans, more reliable and relevant data on the biological and toxicological effects of PFOA have been generated in laboratory monkeys (Butenhoff et al., 2002,[3] 2004a, and 2004b); and these primate data, combined with information from studies in humans, can be used to generate estimates of risks to human health from PFOA. We do so as follows.

Butenhoff and co-workers (2002, 2004a, and 2004b) examined the effects of the ammonium salt of PFOA (APFO) in male cynomolgus monkeys, during and after oral dosing for 6 months. The dose-rates were 3, 10, and 30 mg of APFO/kg body weight/day, although because the monkeys in the high dose-rate reduced their food intake and failed to gain weight, this highest dose-rate was reduced 20 mg/kg-day.

Doses of 30 and/or 20 mg/kg-day were plainly toxic, with evidence of liver injury in the highest dosed monkeys, but doses of 10 mg/kg-day and 3 mg/kg-day were not: no histopathologic evidence of liver injury was observed in monkeys in these middle and low dose-groups, and concentrations of liver enzymes in their blood-sera were normal.

All doses of APFO did increase the relative weights of the monkeys' livers, due to proliferation of liver mitochondria. This effect was expected, since statin drugs and other peroxisome proliferators (which act like PFOA in the liver) also cause increased biosynthesis of mitochondria. Although this is clearly a chemically-induced (and drug-induced) effect, it is not

[3] Individual animal data for this study are available in Thomford (2001) and 3M Environmental Laboratory (2001).

Green Toxicology LLC www.GreenToxicology.com Green@GreenToxicology.com Crouch@GreenToxicology.com

clear that it is an *adverse* effect, as opposed to merely an adaptive effect (Berthiaume and Wallace, 2002; Butenhoff et al., 2002; Hall et al., 2012; Convertino et al, 2018).

Nonetheless, the authors (Butenhoff et al., 2004b) erred on the side of safety by using the relative increase in liver weight (expressed as the ratio of animals' liver weight to brain weight) to derive a benchmark concentration (BMC) for PFOA that could be used for purposes of human health risk assessment.

Their BMC analysis used mean values by dose group of concentration and liver-to-brain weight ratio, and omitted the high-dose group. However, there is substantial intraspecies variation in concentrations at fixed dose rates; for example, the two animals in the high dose group differed by almost a factor of 3 in their plasma concentrations of PFOA (averaged over weeks 20 to 26, as used by Butenhoff et al., 2004b; see Butenhoff et al., 2004a or 3M Environmental Laboratory, 2001 for individual animal concentrations in this experiment). The same sort of variation in the ratio of plasma concentration to dose can be expected in humans, since the weight-specific volume of distribution is unlikely to vary substantially between individuals while the half-life varies substantially, as seen in a cohort in Sweden and in the C8 study (Li et al., 2017, 2018).

A BMC analysis using individual animal data is sensitive to inclusion/exclusion of the monkey with highest concentration or inclusion/exclusion of the high dose animals (**Figure 1**, **Table 1**).

Green Toxicology LLC www.GreenToxicology.com Green@GreenToxicology.com Crouch@GreenToxicology.com

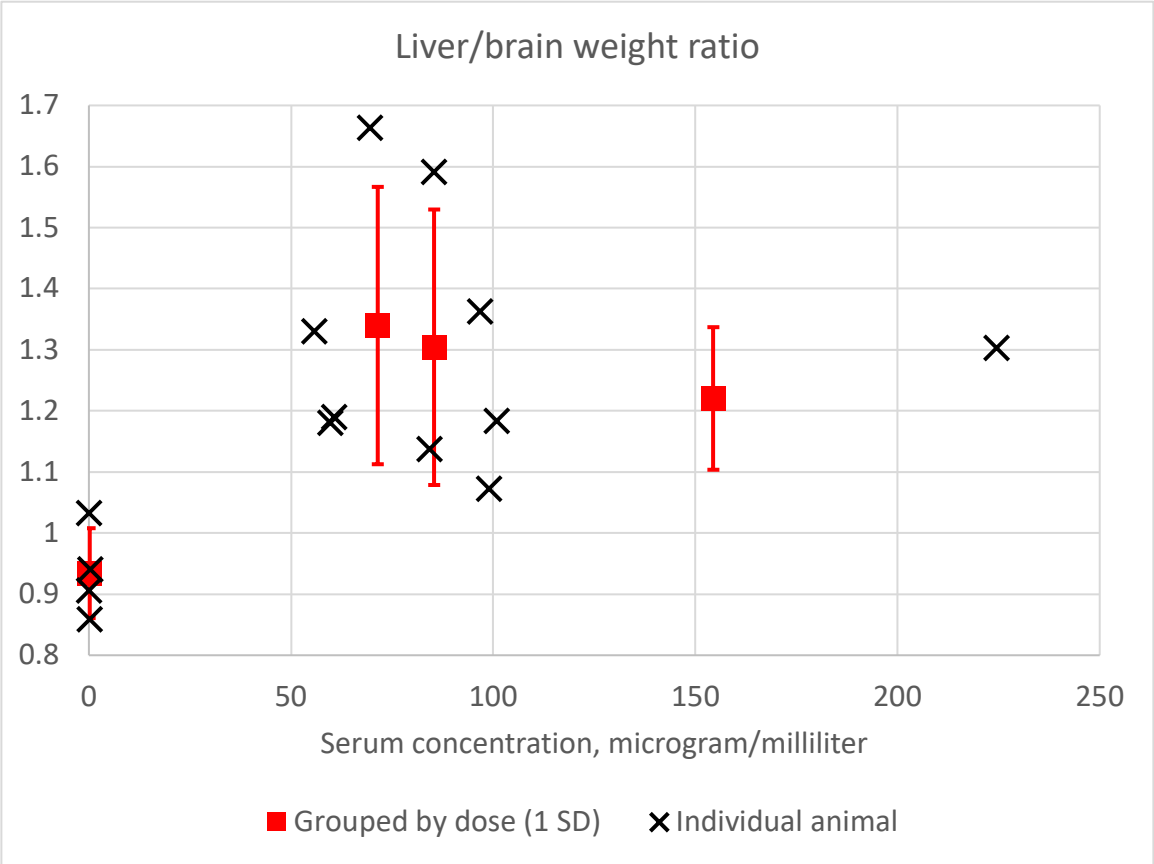

Figure 1 Liver/brain weight ratio in Butenhoff et al. (2002)

	BMCLo	BMC	BMCHi
Grouped, all doses	45.0	79.7	343.9
Grouped, omit high dose	22.6	35.5	79.8
Individual, all animals	57.5	113.2	3099.8
Individual, omit high concentration	29.9	52.4	205.1
Individual, omit high dose	28.3	49.1	178.4

Table 1 BMC estimates (serum concentrations, μg/ml) using liver/brain weight (95% confidence limits, 1 SD, linear model, constant variance)

In fact, in this experiment, the liver/bodyweight ratio provides a more sensitive endpoint (**Figure 2**, **Table 2**). The BMCLo obtained using the individual animal data is the most appropriate for cross-species extrapolation using serum concentration as the relevant metric, so we use that as the point of departure (POD).

Green Toxicology LLC
www.GreenToxicology.com
Green@GreenToxicology.com
Crouch@GreenToxicology.com

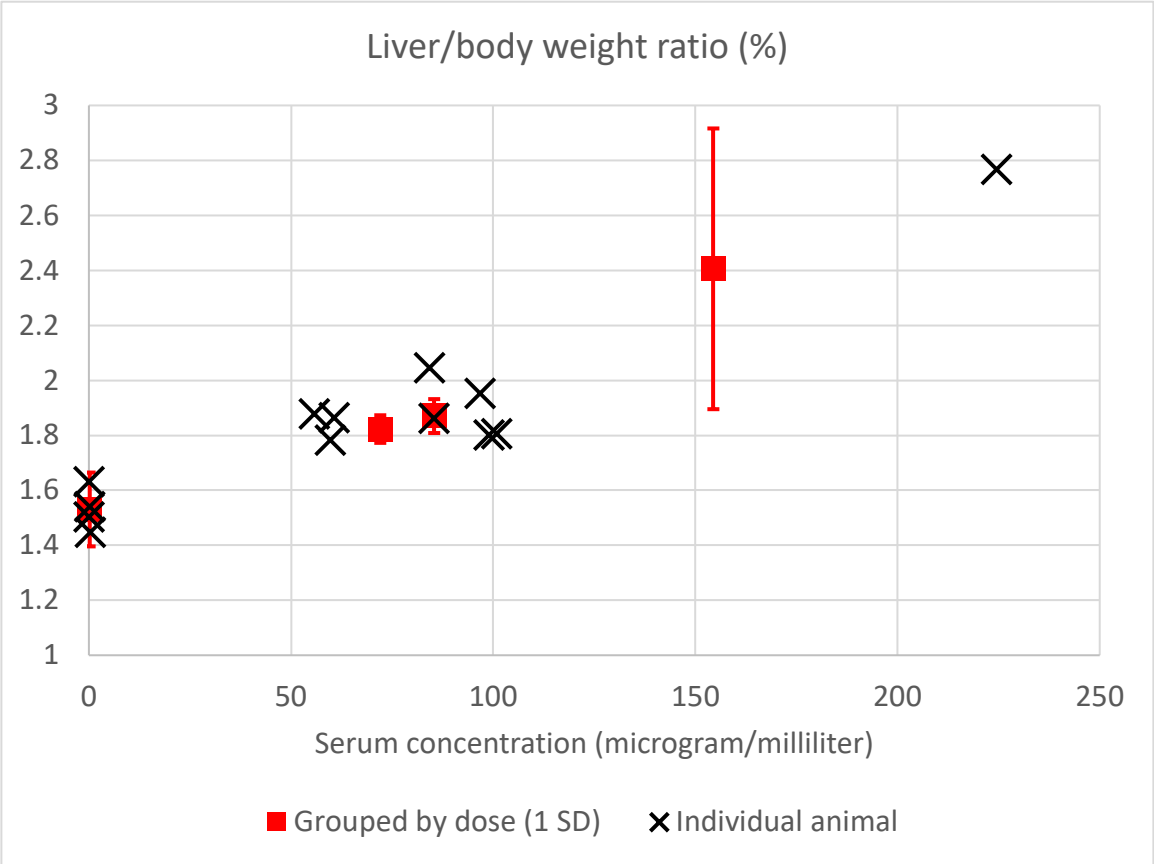

Figure 2 Liver/bodyweight ratio in Butenhoff et al. (2002).

	BMCLo	BMC	BMCHi
Grouped, all doses	26.0	50.9	88.5
Individual, all animals	19.0	32.5	57.4

Table 2 BMC estimates (serum concentrations, μg/ml) using liver/body weight ratio (95% confidence limits, 1 SD, restricted power model, constant variance)

Extrapolating this POD to humans using an interspecies factor of 3 and an intraspecies factor of 10 (compared with the 3-fold difference from 5th to 95th percentile expected solely from the variation in half-lives, Li et al., 2017, 2018), leads to a human plasma concentration of 633 ng/ml. The potential effects of PFOA exposure are seen with short induction times, so no factor is required for extrapolation from subchronic to chronic exposure. Assuming a distribution volume of 0.2 L/kg (ATSDR 2018, Table A-4) and a median half-life of 2.7 years for humans (Li et al., 2017, 2018) gives a reference dose of 89 ng/kg-day.

This primate results-based, reference dose is highly conservative, since, as noted, it assumes that liver weight gain in PFOA-exposed monkeys, in the absence of any indication of liver damage, is an adverse, as opposed to simply adaptive, effect.

Of course, risk assessment is intended to err on the side of safety, so this conservatism is, we believe, appropriate. We recommend that MassDEP consider using this more reliable and relevant value for PFOA as it continues to refine its approach for the regulation of this chemical.

We would add that we think it quite important for risk assessors to communicate that chemicals, such as PFOA, with very small reference doses based on laboratory animal study-results (with multiple safety factors applied) are *not necessarily* highly toxic to humans. Indeed, analysts should make plain that PFAS are *categorically* different from chemicals such as arsenic, lead, mercury, benzene, 2,3,7,8-TCDD, and a multitude of other environmental contaminants for which adverse effects in humans have long been well-established.

As it happens, PFOA has been found to combat certain tumor-types, and has actually, perhaps surprisingly, been administered at extremely large dose-rates — up to 1.2 grams per patient per week, which is about 2,300,000 ng PFOA/kg-day! — to cancer patients in a phase I trial (Convertino et al., 2018). The resulting blood-serum concentrations of PFOA in these phase I study patients were, as noted by Convertino et al. (2018) "the highest ever reported in humans." Yet their serum liver enzyme levels remained normal, and there was otherwise no indication of organ toxicity.[4]

Health-risks from PFOS

Next, PFOS has been studied in laboratory rats, rabbits and monkeys (Case et al., 2001; Seacat et al., 2002; Chang et al., 2012 and 2017); and here again the monkey data can be used to estimate risks to human health.

In developmental toxicity studies in both rabbits and rats (Case et al., 2001), the highest dose rates of PFOS caused frank maternal toxicity, which in turn led to some fetal losses and reversible, delayed ossification. However, per the study-authors, "detailed external gross, soft tissue, and skeletal fetal examinations failed to reveal any compound-related malformations in either species," giving a NOEL for developmental toxicity of 1 mg/kg-d. Moreover, "[t]he

[4] Interestingly, at these high doses, the apparent half-life of PFOA in these patients was on the order of only weeks (Dourson et al., 2019) — substantially lower than the median half-life of 2.7 years that has been derived from people exposed only environmentally (via contaminated drinking water), who have vastly lower plasma concentrations of PFOA.

Green Toxicology LLC www.GreenToxicology.com Green@GreenToxicology.com Crouch@GreenToxicology.com

finding that PFOS was not a selective developmental toxicant to rabbit fetuses concurs with results of previously conducted rat developmental toxicology studies."

Chang et al. (2017) dosed male and female cynomolgus monkeys with one, two, or three doses of PFOS at various times during a 422 day experiment, examining clinical chemistry parameters and measuring serum PFOS concentrations. PFOS serum concentrations at the highest extreme reached values close to those demonstrating overtly toxic effects in an earlier bioassay (Seacat et al., 2002): nonetheless, all clinical chemistry parameters remained within normal biological limits during the experiment. As expected, serum concentrations of two exposure-markers, total thyroxine (TT4) and high density lipoprotein (HDL), did decrease with PFOS treatment, although these varied only within the normal range. Moreover, again as expected, the PFOS-associated decreases in serum TT4 (due presumably to competitive binding) were not accompanied by alterations in serum concentrations of thyroid stimulating hormone (TSH), thus indicating no toxicologically significant effect of PFOS on thyroid function (Chang et al., 2017).

A benchmark concentration (BMC) analysis using individual animal data, based on the conservative assumption that the slight decrements in serum HDL were adverse, yielded a BMCLo (1 SD) of 74,259 and 76,373 ng/ml for males and females respectively. Once again, as in the case of PFOA, evaluation using individual animal data is essential since standard analyses (not shown) based on the published grouped data provide substantially different results (both higher and lower, depending on the assumptions made), presumably because of the large variation in serum concentration to dose ratios.

Extrapolating an average point of departure of 75,300 ng/ml to humans, using an interspecies factor of 3 and an intraspecies factor of 10 (again, larger than the expected major component of such intraspecies factor, the dose-to-serum concentration ratio, which is approximately a factor of 3 between 5th and 95th percentiles, Li et al., 2017, 2018), leads to a human plasma concentration of 2,510 ng/ml. All potential effects of PFOS exposure in animal models are seen with short induction times, so no factor is required for extrapolation from subchronic to chronic exposure. Assuming a distribution volume of 0.2 L/kg (ATSDR 2018, Table A-4) and a human half-life of 3.4 years (Li et al., 2017, 2018) gives a reference dose for PFOS of 280 ng/kg-day.

We recommend that MassDEP consider using this more reliable and relevant value for PFOS as it continues to refine its approach for the regulation of this chemical. MassDEP should also note that this most sensitive effect — a slight reduction in serum HDL — was, as noted by the study-authors, *of no significance to the health of the test-animals*. Indeed, serum lipid levels decreased overall with PFOS-exposure, and this is not adverse.

Risks from other PFAS

In deriving its proposed PFAS standards, MassDEP applies an extra safety factor of 4 (further reducing U.S. EPA's reference doses for PFOA and PFOS from 20 ng/kg-day to 5 ng/kg-day), to account for what DEP claims is the possibility that all six PFAS could harm people's immune

Green Toxicology LLC www.GreenToxicology.com Green@GreenToxicology.com Crouch@GreenToxicology.com

systems at or near these miniscule dose-rates. This factor of 4 is entirely arbitrary, and is not justified by MassDEP by any holistic analysis of the weight of scientific evidence. We would note that such an holistic analysis has been peer-reviewed and published (Chang et al., 2016), and it concludes:

> With few, often methodologically limited studies of any particular health condition, generally inconsistent results, and an inability to exclude confounding, bias, or chance as an explanation for observed associations, the available epidemiologic evidence is insufficient to reach a conclusion about a causal relationship between exposure to PFOA and PFOS and any immune related health condition in humans. When interpreting such studies, an immunodeficiency should not be presumed to exist when there is no evidence of a clinical abnormality.

We would also note that the two rodent bioassays on which U.S. EPA's reference doses for PFOA and PFOS are based reported no effects on the exposed animals' immune systems.

More generally, ATSDR (2018) has extensively reviewed studies of immune system effects for several of the PFAS of interest: the Agency finds no compelling evidence that PFAS-exposure compromises people's immune systems.

With regard to PFOA, ATSDR (2018) notes that "no consistent associations" have been "found between serum PFOA and disease resistance, as measured by episodes of the common cold, cough, fever, or hospitalization for infectious disease."

With regard to PFOS, ATSDR (2018) notes, "Mixed results have been observed in studies evaluating infectious disease resistance. Similarly, inconsistent results have been examined in studies evaluating associations between serum PFOS and hypersensitivity outcomes, such as asthma; no associations were found for eczema, dermatitis, food allergies/sensitizations."

With regard to PFHxS, ATSDR (2018) notes, "In general, the available studies do not suggest an association between serum PFHxS and decreased infectious disease resistance."

And with regard to PFNA, ATSDR (2018) notes, "Most studies examining a possible association between serum PFNA levels and immunosuppression have not found associations."

We would add that MassDEP should regulate each individual PFAS based on the chemical, biological, and toxicological evidence for that specific PFAS — rather than simply, and counterfactually, assuming that all six PFAS (i) act identically and (ii) pose identical risks to public health. Clearly, they do not.

Green Toxicology LLC www.GreenToxicology.com Green@GreenToxicology.com Crouch@GreenToxicology.com

Concluding remarks

Assessing risks to public health from PFAS is not straight-forward, and there is no one best approach. Nonetheless, we believe that MassDEP can and will improve upon its draft assessment.

The currently proposed PFAS regulations are both inordinately stringent and unusually poorly justified. We believe that when MassDEP takes the time it needs to evaluate the relevant scientific evidence, from studies in humans and non-human primates alike, the Department will conclude that these six PFAS do not pose the extreme health-threat implied by the currently proposed standards.

Acknowledgements

We received no funding for these comments, received no input from any interested parties with regard to these comments, and have no conflict of interest.

References

Note: Copies of the EPA Administrative Record AR-226 may be requested on CD-ROM from the EPA Docket Office by calling 202-566-0280 or sending an email request to: oppt.ncic@epa.gov.

3M Medical Department. 26-Week Capsule Toxicity Study with Ammonium Perfluorooctanoate (APFO/POAA) in Cynomolgus Monkeys — Determination of the Presence and Concentration of Perfluorooctanoate Fluorochemical in Liver, Serum, Urine and Feces Samples. 3M Environmental Laboratory, Building 2-3E-09, 935 Bush Avenue, St. Paul, MN 55106; and Centre Analytical Laboratories, Inc., 3048 Research Drive, Stage College, PA 16801. Study T-6889.3, FACT TOX-026, LAN-U2782. June 11, 2001. EPA Administrative Record AR226-1052a.

Abbott BD, Wood CR, Watkins AM, Tatum-Gibbs K, Das KP, Lau C. Effects of perfluorooctanoic acid (PFOA) on expression of peroxisome proliferator-activated receptors (PPAR) and nuclear receptor-regulated genes in fetal and postnatal CD-1 mouse tissues. Reproductive Toxicology. 2012 Jul 1;33(4):491–505.

Agency for Toxic Substances and Disease Registry (ATSDR). June 2018. Toxicological Profile for Perfluoroalkyls. Draft for Public Comment. U.S. Department of Health and Human Services.

Albrecht PP, Torsell NE, Krishnan P, Ehresman DJ, Frame SR, Chang SC, Butenhoff JL, Kennedy GL, Gonzalez FJ, Peters JM. A species difference in the peroxisome proliferator-activated receptor α-dependent response to the developmental effects of perfluorooctanoic acid. Toxicological Sciences. 2013;131(2):568–582.

Green Toxicology LLC www.GreenToxicology.com Green@GreenToxicology.com Crouch@GreenToxicology.com

Bell AR, Savory R, Horley NJ, Choudhury AI, Dickins M, Tim JB, Salter AM, Bell DR. Molecular basis of non-responsiveness to peroxisome proliferators: the guinea- pig PPARα is functional and mediates peroxisome proliferator-induced hypolipidaemia. Biochemical Journal. 1998 Jun 15;332(3):689–693.

Berthiaume J, Wallace KB. Perfluorooctanoate, perflourooctanesulfonate, and N-ethyl perfluorooctanesulfonamido ethanol; peroxisome proliferation and mitochondrial biogenesis. Toxicol Lett. 2002 Mar 24;129(1-2):23-32.

Butenhoff J, Costa G, Elcombe C, Farrar D, Hansen K, Iwai H, Jung R, Kennedy Jr G, Lieder P, Olsen G, Thomford P. Toxicity of ammonium perfluorooctanoate in male cynomolgus monkeys after oral dosing for 6 months. Toxicological sciences. 2002 Sep 1;69(1):244–257.

Butenhoff JL, Gaylor DW, Moore JA, Olsen GW, Rodricks J, Mandel JH, Zobel LR. Characterization of risk for general population exposure to perfluorooctanoate. Regulatory Toxicology and Pharmacology. 2004b Jun 30;39(3):363–380.

Butenhoff JL, Kennedy Jr GL, Hinderliter PM, Lieder PH, Jung R, Hansen KJ, Gorman GS, Noker PE, Thomford PJ. Pharmacokinetics of perfluorooctanoate in cynomolgus monkeys. Toxicological Sciences. 2004a Oct 6;82(2):394–406.

California EPA. 2016. Notice of Intent to List: perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS). Available at https://oehha.ca.gov/media/downloads/crnr/noilpfoapfos.pdf

Case MT, York RG, Christian MS. 2001. Rat and rabbit oral developmental toxicology studies with two perfluorinated compounds. Int J Toxicol 20(2):101–109. (Full reports of the rabbit study are in the EPA Administrative Record AR226-0949 and AR226-1030A003).

Chang ET, Adami HO, Boffetta P, Wedner HJ, Mandel JS. A critical review of perfluorooctanoate and perfluorooctanesulfonate exposure and immunological health conditions in humans. Crit Rev Toxicol. 2016;46(4):279-331.

Chang S, Allen BC, Andres KL, Ehresman DJ, Falvo R, Provencher A, Olsen GW, Butenhoff JL. Evaluation of serum lipid, thyroid, and hepatic clinical chemistries in association with serum perfluorooctanesulfonate (PFOS) in cynomolgus monkeys after oral dosing with potassium PFOS. Toxicological Sciences. 2017 Jan 23;156(2):387–401.

Chang SC, Noker PE, Gorman GS, Gibson SJ, Hart JA, Ehresman DJ, Butenhoff JL. Comparative pharmacokinetics of perfluorooctanesulfonate (PFOS) in rats, mice, and monkeys. Reproductive Toxicology. 2012 Jul 1;33(4):428–440.

Convertino M, Church TR, Olsen GW, Liu Y, Doyle E, Elcombe CR, Barnett AL, Samuel LM, MacPherson IR, Evans TR. Stochastic Pharmacokinetic-Pharmacodynamic Modeling for Assessing the Systemic Health Risk of Perfluorooctanoate (PFOA). Toxicological Sciences. 2018 Feb 16;163(1):293–306.

Corton JC, Anderson SP, Stauber A. Central role of peroxisome proliferator–activated receptors in the actions of peroxisome proliferators. Annual Review of Pharmacology and Toxicology. 2000 Apr;40(1):491–451.

Dourson, ML, Gadagbui, B, Onyema, C., McGinnis, PM, York, RG. Data Derived Extrapolation Factors for Developmental Toxicity: Preliminary Research Case Study with Perfluorooctanoate. In press. Available at https://www.tera.org/about/DDEFforPFOADevelopmentalToxicity7-3-19.pdf

Goeden HM, Greene CW, Jacobus JA. A transgenerational toxicokinetic model and its use in derivation of Minnesota PFOA water guidance. J Expo Sci Environ Epidemiol. 2019 Mar;29(2):183–195.

Gortner, E.G., Lamprecht, E.G., Case, M.T. 1982. Oral Teratology Study of T-3141CoC in Rabbits. Safety Evaluation Laboratory, Riker Laboratories, Inc., St. Paul, Minnesota. EPA Administrative Record AR226-0465.

Hall AP, Elcombe CR, Foster JR, Harada T, Kaufmann W, Knippel A, Küttler K, Malarkey DE, Maronpot RR, Nishikawa A, Nolte T. Liver hypertrophy: a review of adaptive (adverse and non-adverse) changes—conclusions from the 3rd International ESTP Expert Workshop. Toxicologic Pathology. 2012 Oct;40(7):971–994.

Hoivik DJ, Qualls Jr CW, Mirabile RC, Cariello NF, Kimbrough CL, Colton HM, Anderson SP, Santostefano MJ, Morgan RJ, Dahl RR, Brown AR. Fibrates induce hepatic peroxisome and mitochondrial proliferation without overt evidence of cellular proliferation and oxidative stress in cynomolgus monkeys. Carcinogenesis. 2004 Sep 1;25(9):1757–1769.

Klaunig JE, Babich MA, Baetcke KP, Cook JC, Corton JC, David RM, DeLuca JG, Lai DY, McKee RH, Peters JM, Roberts RA. PPARα agonist-induced rodent tumors: modes of action and human relevance. Critical reviews in toxicology. 2003 Jan 1;33(6):655–780.

Klaunig JE, Hocevar BA, Kamendulis LM. Mode of action analysis of perfluorooctanoic acid (PFOA) tumorigenicity and human relevance. Reproductive toxicology. 2012 Jul 31;33(4):410–418.

Lau C, Thibodeaux JR, Hanson RG, Narotsky MG, Rogers JM, Lindstrom AB, Strynar MJ. Effects of perfluorooctanoic acid exposure during pregnancy in the mouse. Toxicological Sciences. 2006 Apr;90(2):510–518.

Li Y, Mucs D, Scott K, Lindh C, Tallving P, Fletcher T, Jakobsson K. 2017b. Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water. Report No 2:2017, Occupational & Environmental Medicine in Gothenburg, The Sahlgrenska Academy Institute of Medicine, Goteborgs Universitet. http://hdl.handle.net/2077/52661.

Li Y, Fletcher T, Mucs D, Scott K, Lindh CH, Tallving P, Jakobsson K. Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water. Occup Environ Med. 2018;75(1):46–51.

Luebker DJ, Case MT, York RG, Moore JA, Hansen KJ, Butenhoff JL. Two-generation reproduction and cross-foster studies of perfluorooctanesulfonate (PFOS) in rats. Toxicology. 2005 Nov 5;215(1–2):126–148.

MassDEP, 2019. 2019 Proposed MCP revisions. https://www.mass.gov/lists/2019-proposed-mcp-revisions.

Seacat AM, Thomford PJ, Hansen KJ, Olsen GW, Case MT, Butenhoff JL. Subchronic toxicity studies on perfluorooctanesulfonate potassium salt in Cynomolgus monkeys. Toxicological Sciences. 2002 Jul 1;68(1):249–264. (Full report in the EPA Administrative Record AR226-1051a.)

Staels, B., Auwerx, J. 1998. Regulation of apo A-1 gene expression by fibrates. Atherosclerosis 137 Suppl. S19-S23.

Thomford PJ. 26-Week Capsule Toxicity Study with Ammonium Perfluorooctanoate (APFO) in Cynomolgus Monkeys. Covance Laboratories Inc., 3301 Kinsman Boulevard, Madison, Wisconsin 5304-2594. Covance 6329-321. Sponsored by APME Ad-Hoc APFO Toxicology Working Group. Sponsor Study 3M T-6889.3. December 18, 2001. EPA Administrative Record AR226-1052a. (Note: on the material supplied by EPA, the 26-week study file is incorrectly labeled as the 4-week study, and vice versa.)

U.S. EPA, 2016. Health effects support document for perfluorooctanoic acid (PFOA). EPA 822-R-16-003, May 2016.

U.S. FDA, 2017. Detection of Toxicity to Reproduction for Human Pharmaceuticals. Draft ICH Harmonised Guideline. International Council for Harmonisation of Technical Requirements for Pharmaceuticals for Human Use.

Green Toxicology LLC www.GreenToxicology.com Green@GreenToxicology.com Crouch@GreenToxicology.com

Attachment C
ORSG Update for PFAS Compounds

Documentation for Updated Office of Research and Standards Guidelines (ORSG) for Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water

PFAS included[1]:	CASRN:	**Update: January 2020**
Perfluorooctane Sulfonic Acid (PFOS)	1763-23-1	
Perfluorooctanoic Acid (PFOA)	335-67-1	
Perfluorohexane Sulfonic Acid (PFHxS)	355-46-4	
Perfluorononanoic Acid (PFNA)	375-95-1	
Perfluoroheptanoic Acid (PFHpA)	375-85-9	
Perfluorodecanoic Acid (PFDA)	335-76-2	

[1]The compounds and associated CAS registry numbers (CASRN) listed refer to the acid form of these PFAS compounds. The information presented in this document and the ORSG are also applicable to the respective anionic forms of these compounds. These anions may form salts with any of a number of cations resulting in a variety of possible chemical species, each having a unique CASRN.

Updated ORSG: 0.000020 mg/L (20 ppt). When all or some of the listed compounds occur together in drinking water, the detected concentrations for these PFAS should be summed and compared to 0.000020 mg/L. This value is also applicable to the individual compounds.

Federal Regulatory Limit: The US EPA has not published an MCL for any of these PFAS.

Basis for Criteria - Non-Cancer Health Risk:

In consideration of recent PFAS assessments by other organizations and states, and new data, MassDEP ORS reassessed the toxicity values and ORSGs for the PFAS compounds in the noted subgroup. This reassessment reflects public comments received on the draft Massachusetts Contingency Plan (MCP) PFAS standards issued for public comment on April 19, 2019, as well as technical input from the MassDEP Health Effects Advisory Committee. Detailed supporting information can be found in the Technical Support Document Per- and Polyfluoroalkyl Substances (PFAS): An Updated Subgroup Approach to Groundwater and Drinking Water Values (the TSD) (https://www.mass.gov/info-details/per-and-polyfluoroalkyl-substances-pfas#development-of-a-pfas-drinking-water-standard-(mcl)-).

In summary, MassDEP ORS concluded that the toxicity value (RfD) for compounds in this subgroup of longer-chain PFAS should be adjusted downward from that used in the 2018 ORSG derivation, to 5×10^{-6} milligrams per kilogram body weight per day (mg/kg-day). The revised MassDEP RfD value results from the application of an additional uncertainty factor (UF) of $10^{1/2}$ in the RfD derivations for PFOA and PFOS as detailed in the TSD. This was done to account for considerable and convincing evidence associating exposures to these compounds with adverse responses in laboratory animals at levels of exposure lower than those used to derive the previous RfD. ORS also concluded that one additional compound, PFDA, should be included in the subgroup, based on structural and toxicological similarity. As discussed in the

TSD the revised MassDEP ORS RfD is applied to the noted subgroup of six closely-related PFAS. Based on their close structural similarities, similar toxicities and long half-lives, MassDEP ORS also concluded that it is appropriate to treat these six compounds additively.

Application of the revised RfD, using the same water ingestion rate and body weight parameters for a lactating woman (i.e., a water ingestion rate of 54 mL/kg-day) and relative source contribution factor of 0.2 previously applied, results in an ORSG of 20 ppt.

Cancer Risk:

MassDEP ORS also considered the potential carcinogenicity of these compounds. A study of people exposed to PFOA and other PFAS concluded that the data supported a probable link between exposure and cancers of the kidney and testes (Barry et al. 2013). No potency estimates were derived. Animal bioassay data from the NTP (NTP 2019c) reported elevated pancreatic and liver tumor rates following high dose exposures to PFOA. Although NTP has issued summary data tables for this study, a final report has not been issued and, as of June 29, 2019, no agency had established drinking water values based on this data. The cancer data is concerning to MassDEP, because some carcinogens can present a degree of risk at any exposure level. To account for this potential risk, MCL goals (MCLGs) of zero have been established by US EPA for some chemicals and may ultimately be warranted for certain PFAS. MCLGs are guidance values rather than standards and are levels of a contaminant in drinking water at or below which there is no known or expected risk to health. At this time, however, the level of cancer risk posed by these compounds is unclear.

Though data on carcinogenicity are not available for PFHxS, PFNA, PFHpA and PFDA, given the similarities in structure and toxicity of these PFAS to PFOA and PFOS, the potential for the carcinogenicity of these other PFAS cannot be ruled out.

MassDEP ORS will follow and assess research in this area to determine if future revisions to the drinking water values are needed.

Class: Organic

Analytical Information:

Analytical Methods: US EPA Method 537 (US EPA, 2009)
US EPA Method 537.1 (US EPA, 2018)

Laboratories should achieve a MRL of 2 ppt.

Analytical methods may have been updated since this guidance value was last revised. Updated analytical methods for drinking water may be found at https://www.epa.gov/measurements-modeling/collection-methods#2

Other Information:

Information and data (e.g. Health Advisories (HAs), RfDs, cancer assessments or cancer potency factors (CPFs) referenced in this document pertain to the derivation of the current guidance value and may not reflect the most current information. Updated information may be available from the following sources:

HAs – The US EPA provides guidance for shorter-term exposures for chemicals based on their non-cancer effects. More current HAs may be found at https://www.epa.gov/sites/production/files/2018-03/documents/dwtable2018.pdf.

RfDs, cancer assessments and CPFs – For specific information pertaining to derivation of drinking water criteria, consult the Federal Register notice that announces the availability of the most current guidance for that chemical. In addition, information on other current RfDs and CPFs as well as cancer assessments for specific chemicals may be found in the US EPA Integrated Risk Information System (IRIS) at https://www.epa.gov/iris. Please note that the information in IRIS may differ from that used in the derivation process as published in the Federal Register notice.

References:

Lau, C., J.R. Thibodeaux, R.G. Hanson, M.G. Narotsky, J.M. Rogers, A.B. Lindstrom, and M.J. Strynar (2006). Effects of perfluorooctanoic acid exposure during pregnancy in the mouse. *Toxicological Sciences* 90:510–518.

Luebker, D.J., R.G. York, K.J. Hansen, J.A. Moore, and J.L. Butenhoff (2005a). Neonatal mortality from in utero exposure to perfluorooctanesulfonate (PFOS) in Sprague-Dawley rats: Dose-response and biochemical and pharmacokinetic parameters. *Toxicology* 215:149–169.

Luebker, D.J., M.T. Case, R.G. York, J.A. Moore, K.J. Hansen, and J.L. Butenhoff (2005b). Two-generation reproduction and cross-foster studies of perfluorooctanesulfonate (PFOS) in rats. *Toxicology* 215:126–148.

MassDEP (2019). Massachusetts Department of Environmental Protection (MassDEP). Technical Support Document - Per- and Polyfluoroalkyl Substances (PFAS): An Updated Subgroup Approach to Groundwater and Drinking Water Values. Office of Research and Standards.

US EPA (2005). United States Environmental Protection Agency. Guidelines for Carcinogen Risk Assessment. Risk Assessment Forum. EPA/630/P-03/001B.

US EPA (2009). United States Environmental Protection Agency. Method 537, Determination of Selected Perfluorinated Alkyl Acids in Drinking Water By Solid Phase Extraction and Liquid Chromatography/Tandem Mass Spectrometry (LC/MS/MS) Version 1. US EPA, Office of Research and Development, National Exposure Research Laboratory. EPA/600/R-08/092.

US EPA. (2016a). United States Environmental Protection Agency. Drinking Water Health

Advisory for Perfluorooctaanoic Acid (PFOA) and Health Effects Support Document for Perfluorooctaanoic Acid (PFOA). US EPA Office of Water. EPA 822-R-16-005

US EPA. (2016b). United States Environmental Protection Agency. Drinking Water Health Advisory for Perfluorooctane Sulfonate (PFOS) and Health Effects Support Document for Perfluorooctane Sulfonate (PFOS). US EPA Office of Water. EPA 822-R-16-004.