

# Health Consultation

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## Public Comment Version

Evaluation of Per- and Polyfluoroalkyl Substances (PFAS) in Private Wells  
near the Saint Gobain Performance Plastics Site in Southern New Hampshire

SAINT GOBAIN PERFORMANCE PLASTICS

MERRIMACK, NEW HAMPSHIRE

EPA FACILITY ID: NHD982746778

**DECEMBER 15, 2021**

**PUBLIC COMMENT PERIOD ENDS: MARCH 1, 2022**

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES  
Agency for Toxic Substances and Disease Registry  
Office of Community Health and Hazard Assessment  
Atlanta, Georgia 30333

## **Health Consultation: A Note of Explanation**

An ATSDR health consultation is a verbal or written response from ATSDR to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

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## HEALTH CONSULTATION

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Prepared by the  
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Agency for Toxic Substances and Disease Registry  
Office of Community Health and Hazard Assessment  
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## Summary

### Introduction

The Agency for Toxic Substances and Disease Registry (ATSDR) evaluates community exposures and makes recommendations to prevent harmful exposures to hazardous substances in the environment. This report evaluates past and current exposures to per- and polyfluoroalkyl substances (PFAS) in private drinking water wells in five towns near the Saint-Gobain Performance Plastics facility in Merrimack, New Hampshire.

The Saint-Gobain facility's processes used several PFAS, including perfluorooctanoic acid (PFOA). In 2016, PFOA was found in groundwater near the site. Since then, the New Hampshire Department of Environmental Services (NH DES) has led sampling of public water systems and private wells in five towns surrounding the Saint-Gobain facility: Merrimack, Litchfield, Londonderry, Bedford, and Manchester. Since the discovery of the contamination, state and local officials have taken several actions to reduce exposures, including treating public water supplies and providing alternate or treated water to affected private well owners.

NH DES and the New Hampshire Department of Health and Human Services (NH DHHS) asked ATSDR to do this evaluation. ATSDR staff have been working with the state since 2016 to provide health information to the public regarding PFAS exposure. The state provided ATSDR data from public and private water supplies in 2019. ATSDR will release a separate report evaluating data from public water supplies in the area.

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### Conclusions of ATSDR's Evaluation

ATSDR estimated exposure to PFAS and the resulting potential risk of harmful health effects from drinking well water for over 2,700 private wells in the area. We reached the following general conclusions.

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#### Conclusion 1

**Before actions began in 2016 to reduce exposures, drinking private well water contaminated with PFAS could have increased the risk for harmful health effects for some community members.**

#### Basis for Conclusion

- Most of the private wells evaluated in the five towns of Merrimack, Litchfield, Londonderry, Bedford, and Manchester were contaminated with PFAS. PFOA was

detected most frequently and at the highest concentrations. Based on ATSDR's evaluation of both individual PFAS and PFAS mixture effects detailed in this report, more than 230 out of 2,745 wells had PFAS at levels that could harm infants or young children, and about 9% of those wells had levels that could harm all age groups. Developmental effects are the most likely possible health effects from exposure, and the risk of developmental effects would increase as PFAS levels and exposure increased. Immune or liver effects would also be possible from exposure to the highest PFAS levels. Other sources of PFAS exposure (such as from food or consumer products) could increase the risk of harmful health effects beyond the risk from the drinking water exposures alone.

- The remaining wells, with lower or no detections of PFAS, are not expected to have harmed health. However, this conclusion is uncertain. Many wells were sampled only once, and the actual PFAS levels could have fluctuated over time. Also, knowledge about health effects of the PFAS evaluated is still evolving, and many wells contained other PFAS which have not been studied enough to evaluate the potential for health effects.
- The increased risk of developing cancer from exposure to PFAS in the area is uncertain. There is suggestive evidence that both PFOA and PFOS are carcinogenic, but the science on PFOA, PFOS, and other PFAS is too limited at this time to quantify risk.

### **Next Steps**

- Private well owners who had potentially harmful exposures in the past should discuss their exposure with their health care provider and consider taking steps to reduce other potential PFAS exposures, such as those from consumer products containing PFAS.
- Residents should reduce exposure from background sources of PFAS by avoiding or limiting the use of products containing PFAS. Examples of products that may contain PFAS include food packing materials, stain resistant carpets, water resistant clothing, cleaning products, and some cosmetics.
- ATSDR recommends nursing mothers continue to breastfeed and contact their healthcare providers with specific concerns. ATSDR is available to consult with healthcare providers as needed. To help protect formula-fed infants from potential exposure, caregivers should use pre-mixed formula or reconstitute dry formula with water sources not containing PFAS.

**Conclusion 2**

**Currently, harmful exposures to PFAS in private wells have been minimized by providing alternate water and taking other actions. People who continue to drink contaminated, untreated private well water may still have an increased risk for harmful health effects.**

**Basis for Conclusion**

- Since 2016, bottled water has been provided to residents whose private wells were affected by PFAS. More than 750 private wells in the area have been switched to treated public water or equipped with point-of-entry treatment systems which are regularly tested for treatment effectiveness. Some private wells with low levels of PFAS, or wells with no detections, may remain in use. Based on the current science, harmful health effects are unlikely if PFAS concentrations in those wells remain low. Residents drinking from private wells that were never tested, or who were offered but declined alternate water, may experience harmful health effects if they drink water with high PFAS concentrations.

**Next Steps**

- Residents using point-of-entry treatment systems to remove PFAS from private well water should have the systems maintained and checked periodically to ensure removal effectiveness.
- Residents continuing to drink from private wells should monitor their well water quality and should work with local authorities to take appropriate action to remove harmful contaminants, if needed.
- ATSDR will work with NH DES and NH DHHS to identify any private wells with PFAS levels of concern that have not been addressed through previous actions.

ATSDR is available to discuss individual results with private well owners and will continue to be available, upon request, to answer other public health questions related to the site.

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## ATSDR and the topic of this report

The Agency for Toxic Substances and Disease Registry's (ATSDR) mission is to serve the public through responsive public health actions; to promote healthy and safe environments; and prevent harmful human exposures. This health consultation provides an evaluation of the public health implications of past and current exposures to per- and polyfluoroalkyl substances (PFAS) in private drinking water wells in five towns near the Saint-Gobain Performance Plastics facility in Merrimack, New Hampshire. The report includes recommendations to protect public health. ATSDR worked with the New Hampshire Department of Health and Human Services (NH DHHS) and the New Hampshire Department of Environmental Services (NH DES) in preparing this report.

## Background and brief history of the site

The Saint-Gobain Performance Plastics facility in Merrimack, New Hampshire produces specialty coated fabrics and films for a range of industrial applications, such as heavy-duty roofing fabrics and hazardous materials-resistant clothing. In 2001, Saint-Gobain took over the operations of the Chemical Performance Fabrics (ChemFab) company, which had operated since the late 1980s. The manufacturing process uses several PFAS, including perfluorooctanoic acid (PFOA). Figure 1 shows the facility's location and the area evaluated in this health consultation.

In 2016, after PFOA groundwater contamination had been discovered at similar facilities in the U.S., the Merrimack Saint-Gobain facility voluntarily conducted water testing and identified PFOA present in public water-supplied tap water at the plant. Since 2016, NH DES has led efforts to conduct sampling of public water systems and private wells in five towns surrounding the Saint-Gobain facility: Merrimack, Litchfield, Londonderry, Bedford, and Manchester. Saint-Gobain is responsible for sampling within an "Outer Boundary" determined by a 2018 consent decree with NH DES [1].<sup>1</sup> NH DES reviews data from areas outside the Outer Boundary and may conduct additional sampling, if needed [2].

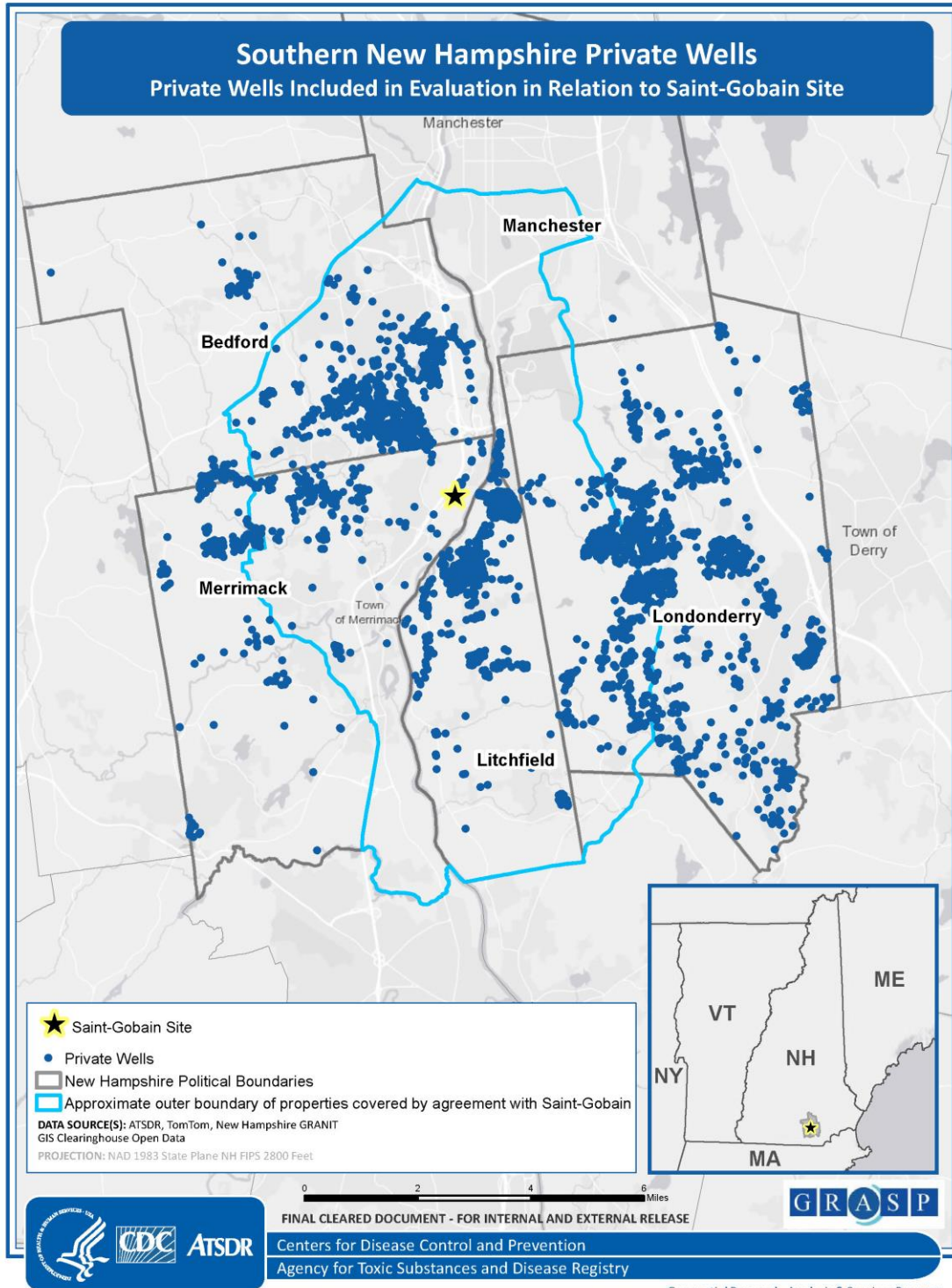
Since the discovery of the contamination, local actions have reduced exposures to PFAS in drinking water. Public water supplies within the Outer Boundary are treated to remove PFAS. People using private wells found to exceed state health-based drinking water standards<sup>2</sup> were offered bottled water, connection to the public water supply, or installation of a treatment system to remove PFAS. Hundreds of properties supplied with water from private wells have been connected to local municipal water systems or provided treatment systems [1,2,4].

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<sup>1</sup> The Outer Boundary as indicated on Figure 1 includes a portion of the town of Hudson, NH. At the time of the original request to ATSDR, no samples had been collected from private wells in Hudson, and it is not included in the evaluation. The general conclusions and recommendations in this report would apply to private wells in other towns, depending on the PFAS levels found.

<sup>2</sup> From 2016 until 2020, New Hampshire's drinking water standard was 0.07 micrograms per liter for PFOA, PFOS, or a combination of the two chemicals. This value is identical to the U.S. Environmental Protection Agency's (EPA's) lifetime health advisory for PFOA and/or PFOS [3]. In September 2019, the state adopted new rules with lower limits for PFOA, PFOS, PFHxS, and PFNA; these rules were enacted via legislation in July 2020.

Figure 1. Location of the Saint-Gobain facility in relationship to the five surrounding towns for which private well data were evaluated



## How ATSDR became involved

In 2016, NH DHHS and NH DES requested assistance from ATSDR in helping assess and respond to potential health impacts from exposure to PFAS in drinking water in the area surrounding the facility [5]. Since then, ATSDR staff have been working with the state to provide health information to the public regarding PFAS exposure. The state also requested ATSDR develop health consultation reports evaluating data from public and private water supplies; these data have recently become available to ATSDR. ATSDR will produce two health consultation reports. This report focuses on private well data. A separate report will evaluate data from public water supplies in the area.

## Focus of this report

This health consultation focuses on evaluating the potential impacts of exposure to PFAS in drinking water from private wells in five New Hampshire towns surrounding the Saint-Gobain site: Merrimack, Litchfield, Londonderry, Bedford, and Manchester. NH DES requested private well data from the five towns be included in the private well evaluation [6]. There are other potential sources of PFAS in this general area, and PFAS detected in private wells may not originate from the Saint-Gobain facility. This health consultation makes no attempt to attribute PFAS contamination to the facility or any other source.

For evaluating PFAS exposures from private wells, ATSDR considered only the drinking (ingestion) exposure route and did not include breathing (inhalation) or skin contact (dermal) contributions to exposure. PFAS do not easily evaporate from water during bathing and showering, and absorption of PFAS through skin is slow or limited [7]. Therefore, inhalation or skin exposures from private well water will be negligible compared to ingestion exposures.

The data ATSDR obtained from NH DES included PFAS sample results only. We did not have data on any other types of potential contamination in the wells such as chemicals other than PFAS, biological contamination, or other physical indicators that may affect the suitability of the water for human consumption.

NH DES asked ATSDR to comment on potential health effects resulting from drinking water exposures to PFAS. No data describing PFAS levels in other environmental media besides drinking water near the site were available to us. This document does not include consideration of any other potential past, present, or future exposures, including

- Inhalation exposure to PFAS released into the air from the facility;
- Direct contact or incidental ingestion exposure to PFAS in soil, surface water, or sediment;
- Indirect ingestion of PFAS in biota (fish, shellfish, or plants) that may have bioaccumulated PFAS from their local environment; or
- Exposure to PFAS from consumer products in the home or community.

More details on ATSDR's analysis of exposures possible at this site can be found in Appendix B.

## Stepwise discussion of ATSDR's evaluation of private well data

### Environmental sampling data handling

In October 2019, NH DES provided ATSDR with private well PFAS sampling results extracted from the database of PFAS sampling results reported to the state [8]. In April 2021, NH DES provided an updated spreadsheet containing newer results and additional PFAS component results [9]. The complete results comprise over 4,000 private well sample results from almost 2,750 different addresses in five towns. The spreadsheet included results collected between March 2016 and April 2021 and contained 56 different PFAS results fields. Different laboratories and sampling events analyzed different PFAS and often used different reporting conventions for results.

To organize, tabulate, and summarize these data for our public health evaluation, ATSDR performed the following actions on the data provided by NH DES.

- As a fundamental assumption, ATSDR assumed that all sample results associated with a particular address described water from a single private well. We believe this (one well per address) to be largely correct; however, we could not verify it in all instances because some sample descriptors in the very large database were vague, incomplete, or inconsistent. This assumption is appropriate for our goal of gaining a general evaluation of the implications of PFAS in private wells in the area.
- ATSDR manually corrected address spacing issues and standardized abbreviations for street names to allow correct sorting of results by address in the database.
- Some laboratories reported certain PFAS using different conventions (some reported them as acids, and others reported the same substance as a dissociated salt, or anion). For sulfonate anion/sulfonic acid pairs, either reporting convention would result in a value that is practically equivalent (differing by the weight of a single hydrogen atom). For these PFAS, ATSDR considered values by either convention as equivalent. This practice is consistent with technical guidance developed by the Interstate Technical and Regulatory Council (ITRC) [10].
- ATSDR dropped from consideration PFAS listed in the database which were not analyzed, or which had no detections<sup>3</sup>.
- The above considerations reduced the number of PFAS to be evaluated to 25.
- About 11% of the results included field replicates or samples from more than one location (tap, outside spigot, etc.) at the same address on the same date. ATSDR followed standard practice and averaged results of field replicate samples. Also, as stated above, ATSDR considered all samples from a particular address to represent a single private well, and (if sampled on a single day) considered them as replicates. ATSDR applied a single latitude-longitude to all replicate samples from the address, since we only needed a general sense of the location of the private well for our evaluation.

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<sup>3</sup> PFAS listed in the database which contained no analysis results were 1-Propene-1,1,2,3,3,3-hexafluoro-, dimer; perfluorobutylsulfonamide; and perfluorohexanesulfonamide.. PFAS which were reported as analyzed at least once but had no detections reported in any well include 11-chloroeicosafuoro-3-oxaundecane-1-sulfonic acid; 9-chlorohexadecafluoro-3-oxanone-1-sulfonic acid; PFODA; PFHpS Sulfonate; Perfluoro-3-methoxypropanoic acid; Perfluoro(4-methoxybutanoic) acid; PFHxDA; ETFOSE; PFDoDS; PFNS; 10:2 FTSA; EtFOSA; MeFOSA; MeFOSE; GenX (Acid or Salt); DONA; and ADONA. Please see Appendix A for full compound names, chemical formulae, and Chemical Abstract Services Registry numbers.

- About 18% of the private wells had results from multiple sampling dates. For these wells, ATSDR selected the highest concentration of each PFAS detected for screening, preparing summary tables, and estimating exposure dose. The highest concentration is used because exposures to PFAS may have harmful health effects over relatively short periods of exposure (weeks to months); using a long-term average concentration could underestimate potentially harmful exposures.

The resulting dataset included results for up to 25 PFAS detected in water from 2,745 different private wells in the five-town area of Merrimack, Litchfield, Londonderry, Bedford, and Manchester.

### **Determining the timeframe of potential exposure**

Production at the facility currently operating as Saint-Gobain began in 1986. We do not have any historical data showing levels of PFAS in groundwater or private wells. We assume past exposure to PFAS from private wells could have occurred continuously, beginning a short time after the facility began operating. This is a conservative assumption made because of the lack of historical data showing when the wells were actually contaminated. The levels and composition of PFAS in groundwater and private wells likely varied over the years and could be higher or lower than those measured in recent sampling. The available data from 2016 to 2021 best represent more recent exposures.

After discovery of the PFAS contamination, Saint-Gobain and local authorities acted to protect people whose drinking water contained PFAS. Affected neighborhoods were eligible for free bottled water, and over 750 affected homes were offered connection to treated municipal water or their wells fitted with point-of-entry treatment systems. Water line extension projects were completed in 2020. For residents who are now drinking treated water, PFAS exposure may have occurred in the past, but exposure to PFAS above screening levels from drinking water is no longer occurring.

Some private well owners may have declined well water testing, and some who were eligible for connection to municipal water or a point-of-entry treatment system declined the offer. Ongoing testing has identified additional affected private wells in the area.<sup>4</sup> These residents may have ongoing exposure to PFAS as well as past exposures. Current or future residents who drink from an untreated, contaminated private well will continue to be exposed.

### **Screening and summarizing the data**

The next step in ATSDR's evaluation process is to screen the well water contaminant data against health-based, chemical-specific comparison values (CVs). This step allows ATSDR to focus attention on wells and contaminants of most potential concern by eliminating from further consideration those that are unlikely to result in harmful exposures. The CVs used in this report are concentrations of chemicals in drinking water below which no harmful health effects are expected to occur, even with continual exposure of small children and infants. CVs are not regulatory clean up values, and concentrations higher than the corresponding CV do not necessarily result in harm. ATSDR evaluates contaminants detected at concentrations above a

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<sup>4</sup> Other sources beside Saint-Gobain exist in the area; ATSDR makes no source attribution in this report.

CV further in ATSDR's process. ATSDR has derived CVs called environmental media evaluation guides, or EMEGs, for four PFAS: PFOA, PFOS, PFHxS, and PFNA.

As of the date of this report, ATSDR has not derived CVs for other PFAS. However, several U.S. states and international organizations have developed health-based drinking water guidance or screening values for other PFAS.<sup>5</sup> ATSDR considered these values, when available, while evaluating PFAS for which no ATSDR CV was available. These substances are discussed qualitatively later in this report, in the section entitled "Other PFAS present in wells" beginning on page 16. Some state health-based values for other PFAS may not be included on Table 1 for various reasons (for example, if they were extrapolated from studies on a different PFAS or were not specifically developed for drinking water). Those PFAS without CVs that were detected frequently and at levels higher than the lowest PFAS CV available (0.014 g/L for PFOS) were retained and evaluated qualitatively later in the report.

Table 1 summarizes the detections and compares the highest concentrations of each PFAS detected with its corresponding CV, if available. PFAS are listed in the table in order of decreasing frequency of detection; PFOA was detected most frequently, in 91% of the wells tested. Of the PFAS with CVs available,

- PFOA and PFOS were detected above their corresponding CVs the most frequently (in 30% and 3% of the private wells, respectively);
- PFHxS and PFNA were detected above their corresponding CVs in only one well each; and
- PFBA and PFBS were not detected above health-based state screening values in any well.

ATSDR has not derived or fully reviewed other states' substance-specific, health-based CVs at this time. For this evaluation, we discuss results and possible health effects for all PFAS detected frequently and at higher levels than the lowest PFAS CV available.

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<sup>5</sup> Standards and guidance values for PFAS are changing rapidly; values in this report are from the Interstate Technology and Regulatory Council's (ITRC's) May 2021 tables [11].

**Table 1. Summary of PFAS detected in private well sampling near the Saint-Gobain Merrimack, NH facility, 2016-2021 listed in order of decreasing frequency of detection – see Appendix A for full compound names and chemical information**

PFAS*	# of wells with detections / # tested (%)	Maximum <sup>†</sup> concentration (µg/L)	PFAS-specific comparison value (CV) <sup>‡</sup> (µg/L)	CV source <sup>‡</sup>	# / % of wells with results above CV
PFOA	2,498 / 2,745 (91%)	1.6	0.021	ATSDR intermediate child EMEG <sup>‡</sup>	825 / 30%
PFHxA <sup>€</sup>	1,905 / 2,509 (76%)	0.42	none	No CV available <sup>§</sup>	n/a
PFPeA <sup>€</sup>	1,682 / 2,494 (67%)	0.23	none	No CV available <sup>§</sup>	n/a
PFHpA <sup>€</sup>	1,819 / 2,740 (66%)	0.42	none	No CV available <sup>§</sup>	n/a
PFBS	1,578 / 2,739 (58%)	0.14	1	Michigan screening level	0 / 0%
PFOS	1,445 / 2,745 (53%)	0.12	0.014	ATSDR intermediate child EMEG <sup>‡</sup>	71 / 3%
PFHxS	1,424 / 2,742 (52%)	0.24	0.14	ATSDR intermediate child EMEG <sup>‡</sup>	1 / 0%
PFBA	1,180 / 2,455 (48%)	0.14	7	Minnesota chronic noncancer health risk limit	0 / 0%
PFPeS	176 / 839 (21%)	0.012	none	No CV available <sup>§</sup>	n/a
PFNA	288 / 2,742 (11%)	0.085	0.021	ATSDR intermediate child EMEG <sup>‡</sup>	1 / 0%
4:2 FTSA	42 / 831 (5%)	0.0035	none	No CV available <sup>§</sup>	n/a
6:2 FTSA <sup>€</sup>	51 / 1,750 (3%)	0.57	none	No CV available <sup>§</sup>	n/a
PFDA	57 / 2,429 (2%)	0.0058	none	No CV available <sup>§</sup>	n/a
PFHpS	37 / 1,750 (2%)	0.0094	none	No CV available <sup>§</sup>	n/a
FOSA <sup>€</sup>	18 / 1,170 (2%)	0.059	none	No CV available <sup>§</sup>	n/a
PFTeDA	25 / 1,890 (1%)	0.0040	none	No CV available <sup>§</sup>	n/a
Perfluoro-3,6-dioxaheptanoic acid	7 / 615 (1%)	0.00098	none	No CV available <sup>§</sup>	n/a
PFTrDA	12 / 1,890 (0.6%)	0.0065	none	No CV available <sup>§</sup>	n/a
EtFOSAA	5 / 994 (0.5%)	0.0028	none	No CV available <sup>§</sup>	n/a
Perfluoro(2-ethoxyethane)sulfonic acid	2 / 615 (0.3%)	0.00067	none	No CV available <sup>§</sup>	n/a
PFDoDA	7 / 2,429 (0.3%)	0.0074	none	No CV available <sup>§</sup>	n/a
PFUnDA	7 / 2,429 (0.3%)	0.0048	none	No CV available <sup>§</sup>	n/a
8:2 FTSA <sup>€</sup>	4 / 1,750 (0.2%)	0.044	none	No CV available <sup>§</sup>	n/a

PFAS*	# of wells with detections / # tested (%)	Maximum <sup>†</sup> concentration (µg/L)	PFAS-specific comparison value (CV) <sup>‡</sup> (µg/L)	CV source <sup>‡</sup>	# / % of wells with results above CV
MeFOSAA	2 / 1,055 (0.2%)	0.0017	none	No CV available <sup>§</sup>	n/a
PFDS	1 / 1,287 (0.1%)	0.002	none	No CV available <sup>§</sup>	n/a

# - number

µg/L – micrograms per liter

CV – comparison value

n/a – not applicable

\*See Appendix A for full compound names and chemical information.

Shaded cells indicate PFAS that exceeded the corresponding comparison value.

<sup>†</sup>Field replicates collected on the same sample date were averaged to obtain a single result. ATSDR considered all samples from a particular address to represent a single private well, and (if sampled on a single day) considered them as replicates. Thus, maximum concentration refers to the highest concentration representing any of the 2,745 private wells and could itself be an average of more than one result collected from that well on a single day.

<sup>‡</sup>EMEG = Environmental Media Evaluation Guide (developed from ATSDR intermediate minimal risk level). If no ATSDR CV is available, CVs from other sources may be used. As new studies become available, CVs can change. Please see Appendix B for more information about comparison values used in this evaluation.

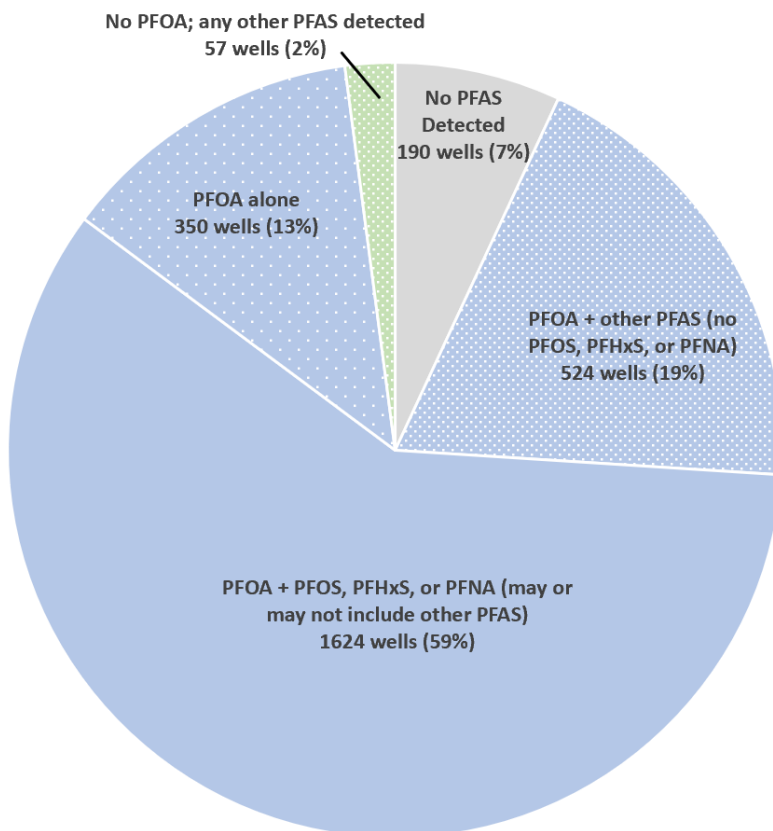
<sup>§</sup>No substance-specific, health-based drinking water screening value was identified. Those substances detected more frequently and at concentrations higher than the lowest PFAS CV available (0.014 µg/L for PFOS) are evaluated qualitatively.

<sup>¶</sup>Substance evaluated qualitatively.



The PFAS detected in private wells varied in composition as well as concentration. As Figure 2 illustrates, PFOA was the most frequently detected PFAS in the private wells. However, in over three-fourths of the wells, one or more other PFAS were also detected. Private wells with multiple PFAS detected contained between 2 and 13 different PFAS.

**Figure 2. Frequency of PFAS Detection in Private Wells Near the Saint-Gobain Site in Merrimack, New Hampshire**



PFOA was the most frequently detected PFAS in 2,745 private wells in the five towns surrounding the Saint-Gobain site in Merrimack, New Hampshire. Many wells showed detections of other PFAS.

Because multiple PFAS were present in many wells, we considered the potential for health effects from exposure to mixtures as well as individual PFAS. Further details are presented below.

**Estimating PFAS exposure doses; comparison with health guidelines**

The next step of ATSDR’s process is to estimate exposure doses for each contaminant. Exposure dose is the amount of contaminant that could get in a person’s body for a specified situation. The estimated dose is expressed on a body weight basis (in amount of contaminant per kilogram of body weight per day) to allow comparison with relevant health guidelines presented in the same units.

Appendix B details how we estimated exposure doses in this report. We used ATSDR standard guidance to estimate exposure doses for age groups ranging from birth through adulthood and who consumed water at rates ranging from typical (i.e., average) to high-end (i.e., 95<sup>th</sup> percentile) for each age group [12,13]. We assumed daily consumption of water containing the highest contaminant concentration measured in each well. We estimated exposure doses on a well-by-well basis. Further details, example calculations, and a summary of results are in Appendix B.

Health guidelines used in this report are ATSDR *minimal risk levels* (MRLs) or *reference doses* developed by other organizations. MRLs and reference doses represent a dose of a single contaminant that is unlikely to result in harmful health effects, to even the most sensitive groups, over the timeframe of exposure. Doses less than the MRL or reference dose are unlikely to result in harmful noncancer effects, while higher doses are evaluated more thoroughly to determine whether harmful health effects are possible.

ATSDR has derived intermediate oral MRLs for four PFAS: PFOA, PFOS, PFHxS, and PFNA [7]. These oral MRLs are based on different studies in which animals were exposed to the substance for between 2 weeks and one year—considered an intermediate duration. ATSDR uses these intermediate oral MRLs to evaluate chronic exposures lasting longer than one year, as well [7]. State health agencies have developed chronic reference doses for two other PFAS: PFBA and PFBS [14,15]. A summary of the derivation of these health guidelines is included in Appendix B.

For the drinking water exposures evaluated in this report, the highest estimated doses are for children from birth to one year old who drink high-end amounts of water (that is, more water than 95% of their age group). Table 2 presents the highest exposure doses estimated for those PFAS with health guidelines available. Because the drinking water CVs used for screening earlier in this report are developed from health guidelines using assumptions for this same sensitive group (children from birth to one year old who drink high-end amounts of water), the summary in Table 2 shows similar results as in Table 1. Calculating the doses is needed, however, for further evaluation of the potential exposures for all age groups and drinking water consumption patterns, as will be discussed later.

As shown in Table 2, hundreds of private wells had estimated doses of PFOA higher than the MRL. A smaller number of wells had estimated PFOS doses exceeding its MRL, and very few of the wells had any PFHxS, PFNA, PFBA, or PFBS doses exceeding their respective health guidelines.

The next section discusses general health implications of PFAS exposure and how ATSDR uses information from human epidemiology and animal toxicology studies in evaluating impacts from PFAS exposures on community's health. Immediately following this section, we discuss implications from exposures to individual PFAS detected in the private wells at this site. In the "PFAS mixtures evaluation" section beginning on page 18, we consider the possibility for health effects from exposures to mixtures of PFAS in private wells.

**Table 2. Summary of highest estimated doses of PFAS (compared to health guidelines) for people drinking from private wells near the Saint-Gobain Merrimack, NH facility**

PFAS*	Highest estimated dose in any well <sup>†</sup> , µg/kg/day	Health guideline, µg/kg/day	Health guideline	# of wells with dose exceeding health guideline <sup>‡</sup>
PFOA	0.230	0.003	ATSDR intermediate MRL	825
PFOS	0.017	0.002	ATSDR intermediate MRL	71
PFHxS	0.034	0.020	ATSDR intermediate MRL	1
PFNA	0.012	0.003	ATSDR intermediate MRL	1
PFBA	0.020	2.9	Minnesota chronic reference dose [14]	0
PFBS	0.020	2.3	Michigan oral reference dose [15]	0

µg/kg/day = micrograms per kilogram body weight per day

MRL = minimal risk level

# - number

\*See Appendix A for full compound names and chemical information.

<sup>†</sup>Highest dose is for children from birth up to one year old who drink high-end (95<sup>th</sup> percentile) amounts of water every day. Doses are generally lower for those who drink less water or who weigh more and thus have a lower dose per body weight. See Appendix B for assumptions and a more detailed summary.

<sup>‡</sup>highest estimated dose (birth up to one-year-old age group with high-end drinking water consumption) exceeds MRL.

### Noncancer health effects from exposure to PFAS

Numerous human epidemiology studies have examined associations between various harmful health effects and serum levels of PFAS in exposed workers, residents exposed to high levels of PFAS released by facilities, and people exposed to background levels of PFAS. The weight of evidence suggests links between PFAS exposure and several harmful health effects in humans, including increased cholesterol levels, changes in liver enzymes, decreased vaccine response in children, increased risk of high blood pressure or pre-eclampsia in pregnant women, and small decreases in infant birth weight [7,16,17].

The human epidemiology studies are valuable in identifying potential hazards associated with PFAS exposure; however, most of them were not designed to show causality, and there were some inconsistencies in findings across the studies. In addition, most studies did not adequately characterize the environmental exposure levels and routes of exposure that produced the observed effects, and most studies involved potential exposures to multiple PFAS at once [7,16]. For these reasons, ATSDR relies on experimental toxicology studies on animals, which have greater ability to control and measure exposures and examine specific biological mechanisms, as the primary basis for evaluating health risks related to PFAS exposure. This introduces uncertainty, because humans and other species process PFAS differently. Rather than using simple dose extrapolation, the nominal doses to which animals are exposed should be converted, whenever possible, to *human equivalent doses* to relate animal toxicity data to possible effects in humans.

The primary noncancer effects observed in toxicological studies on animals exposed to PFAS include developmental toxicity, immune toxicity, and liver toxicity [7,18,19].<sup>6</sup> Other effects, typically observed at higher doses, include weight loss and changes in the microscopic structure of reproductive tissues or the thyroid gland. Not all of these effects were seen across all PFAS tested, and effect levels varied. However, in general, the sensitive targets of toxicity identified in laboratory animals are similar to those observed in human epidemiology studies [7].

### **Individual PFAS exposure evaluation**

#### **PFOA**

PFOA was present in over 90% of the private wells; concentrations ranged from non-detect to 1.6 µg/L. Drinking the most-highly contaminated water would result in doses ranging from about 0.05 to 0.20 µg/kg/day for various age groups with high-end water consumption rates. These doses greatly exceed the corresponding MRL for PFOA of 0.003 µg/kg/day. Age groups with typical water consumption rather than high consumption would have doses about a third to a half as high, but still exceeding the MRL for the highest PFOA concentrations. The toxicology literature has identified several potential health effects from PFOA exposures. A brief summary of the PFOA-specific developmental, immune, and liver effects considered the primary effects observed in animals exposed to PFAS is presented below.

- **Developmental effects.** Skeletal changes and increased activity levels were observed in offspring of mice fed PFOA during pregnancy [7,20,21]. These effects occurred at a human equivalent dose of 0.82 µg/kg/day. The study showing skeletal changes is the basis for ATSDR's intermediate MRL.
- **Immune effects.** A lowered antibody response to applied antigens was observed in mice exposed to PFOA in drinking water [22,23]. This effect occurred at a human equivalent dose of 3.3 µg/kg/day.
- **Liver effects.** Studies on monkeys and rodents have reported signs of liver damage following exposure to PFOA [24–28]. Not all rodent liver effects are considered relevant to humans, and not all studies contain enough information to calculate human equivalent doses [7]. The lowest-effect human equivalent doses for liver effects that could be calculated and appear to be relevant to humans range from about 4 to 20 µg/kg/day in rodent and monkey studies [26–28].

Other sensitive effects, such as changes in mammary gland development observed in mice exposed to low levels of PFOA, have been observed [29]. The biological significance of the finding is uncertain (the changes did not appear to harm milk production or survival of the offspring), and ATSDR has not evaluated the quantitative potential for such effects [7].

The estimated doses for the highest PFOA concentrations approach effect levels determined in toxicology studies. Drinking from the wells with the highest PFOA concentrations could increase

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<sup>6</sup> Not all liver effects observed in rodent studies are considered relevant for humans. ATSDR generally uses the criteria published by Hall et al. in 2012, which is based on an expert panel workshop convened by the European Society of Toxicological Pathology, to discern human toxicological relevance of liver effects observed in rodent studies [19].

the risk of developmental, immune, or liver effects in all age groups. An increased risk of developmental effects might be observed at doses greater than about 0.01  $\mu\text{g}/\text{kg}/\text{day}$ . For the most sensitive group (children between birth and one year old with high-end water consumption), this dose would correspond to drinking private well water containing greater than approximately 0.07  $\mu\text{g}/\text{L}$  of PFOA. As PFOA concentrations increase, the risk of developmental, immune, or liver effects increases. Approximately 200 of the properties with private wells had PFOA concentrations at or above this level. In addition, mixtures effects may have contributed to risk: most of the wells with PFOA had other PFAS detected as well.

Many homes with private wells, including all those with PFOA (or PFOA plus PFOS) concentrations greater than 0.07  $\mu\text{g}/\text{L}$ , have been provided alternate water and connection to a public water source or point-of-entry treatment systems. These actions would have halted known current harmful exposures; however, harmful exposures likely occurred in the past. Harmful exposures could still occur to residents who declined to be added to public water or a treatment system and are still drinking from an untreated contaminated well. Also, any wells that were not tested may have PFOA present at levels of concern. Homeowners using private wells should monitor their well water quality and work with local authorities to take appropriate action to remove harmful contaminants, if needed.

#### *PFOS*

PFOS was present in about 53% of the private wells and at concentrations ranging from non-detect to 0.12  $\mu\text{g}/\text{L}$ . Drinking the most-highly contaminated water would result in doses ranging from about 0.004 to 0.02  $\mu\text{g}/\text{kg}/\text{day}$  for various age groups with high-end water consumption rates. These doses exceed the corresponding MRL for PFOS of 0.002  $\mu\text{g}/\text{kg}/\text{day}$ . Age groups with typical water consumption rather than high consumption would have doses about a third to a half as high, but still exceeding the MRL for some age groups at the highest PFOS concentrations. The toxicology literature has identified several potential health effects from PFOS exposures. A brief summary of the PFOS-specific developmental, immune, and liver effects considered the primary effects observed in animals exposed to PFAS is presented below.

- **Developmental effects.** Offspring of rats exposed to PFOS by gavage before mating, during gestation, and after giving birth showed delays in eye opening and a transient decrease in body weight [30,7]. These effects, which are the basis for ATSDR's intermediate MRL, occurred at a human equivalent dose of 2.1  $\mu\text{g}/\text{kg}/\text{day}$ .
- **Immune effects.** Mice exposed to PFOS by gavage at a human equivalent dose of 0.031  $\mu\text{g}/\text{kg}/\text{day}$  showed decreased resistance to influenza A virus infection [31]. In two reports from another study, mice exposed to a human equivalent dose of 0.41  $\mu\text{g}/\text{kg}/\text{day}$  of PFOS by gavage had a decreased immune response to sheep red blood cells [32,33]. ATSDR believes that the immune effect level of concern from PFOS exposures lies somewhere between the human equivalent effect levels of these two studies.
- **Liver effects.** Monkeys exposed to PFOS were found to have increased liver weights and other hepatic changes at a human equivalent dose of 10  $\mu\text{g}/\text{kg}/\text{day}$  [34].

Other sensitive effects, such as changes in glucose metabolism in mice fed a high-fat diet [35] or changes in levels of estradiol, a female reproductive hormone, in male monkeys [34], have been observed upon exposure of animals to low levels of PFOS. The biological significance of these changes is uncertain, and ATSDR has not evaluated the quantitative potential for such effects [7].

The estimated doses for the highest PFOS concentration are well below effect levels for developmental and liver effects; however, they approach possible effect levels for immune effects. The exact PFOS concentration that may increase the risk of immune effects is uncertain. However, it is likely that drinking from the wells with the highest PFOS concentrations increased the risk of immune effects in all age groups. In addition, mixtures effects may have contributed to risk: for almost all PFOS detections in private wells, PFOA or other PFAS were detected as well.

Many homes with private wells, including all those with PFOS (or PFOA plus PFOS) concentrations greater than 0.07 µg/L, have been provided alternate water and connection to a public water source or point-of-entry treatment systems. These actions would have halted known current harmful exposures; however, harmful exposures likely occurred in the past. Harmful exposures could still occur to residents who declined to be added to public water or a treatment system and are still drinking from an untreated contaminated well. Also, any wells that were not tested may have PFOS present at levels of concern. Homeowners using private wells should monitor their well water quality and work with local authorities to take appropriate action to remove harmful contaminants, if needed.

#### *PFHxS*

PFHxS was present in about 52% of the private wells and at concentrations ranging from non-detect to 0.24 µg/L. Drinking the most-highly contaminated water would result in doses ranging from about 0.008 to 0.03 µg/kg/day for various age groups with high-end water consumption rates. These doses approach and exceed the corresponding MRL for PFHxS of 0.02 µg/kg/day. Age groups with typical water consumption rather than high consumption would have doses about a third to a half as high – doses that all fall below the MRL. The toxicology literature has identified health effects from PFHxS exposures, including

- **Thyroid effects.** Thyroid changes were observed in adult male rats exposed to PFHxS at a lowest-effect level corresponding to a human equivalent dose of 7.3 µg/kg/day [7,36,37]. This finding is the basis for ATSDR's intermediate MRL for PFHxS.

Few of the limited studies on PFHxS have shown an association between PFHxS exposure and developmental, immune, or liver effects considered the primary effects observed in animals exposed to PFAS. No developmental or reproductive effects were reported at any dose tested in the rat study that observed thyroid changes [7,36,37]. A few epidemiological studies have suggested that PFHxS exposure is associated with immune-related effects; however, findings are complicated by co-exposures of study subjects to additional PFAS, particularly PFOA and PFOS, and no toxicological studies on immune effects of PFHxS have been identified [7,38].

Finally, PFHxS exposure has been shown to cause liver effects in rats and mice, but ATSDR determined that the liver effects were not relevant to human exposure [7].

The estimated doses for the highest PFHxS concentration are orders of magnitude below the effect level for thyroid effects determined in toxicology studies. Exposure to PFHxS alone in private well water is unlikely to increase the risk of either thyroid effects or developmental, immune, or liver effects. Because PFHxS was almost always detected with one or more other PFAS, we evaluated the potential for PFHxS exposure to contribute to mixture effects.

Many homes with private wells, including those with the highest concentrations of PFHxS, have been provided alternate water and connection to a public water source or point-of-entry treatment systems. These actions have minimized known current exposures. Homeowners using private wells should monitor their well water quality and work with local authorities to take appropriate action to remove harmful contaminants, if needed.

#### *PFNA*

PFNA was present in about 10% of private wells and at concentrations ranging from non-detect to 0.085 µg/L. Drinking the most-highly contaminated water would result in doses ranging from about 0.003 to 0.01 µg/kg/day for various age groups with high-end water consumption rates. These doses approach and exceed the corresponding MRL for PFNA of 0.003 µg/kg/day. Age groups with typical water consumption rather than high consumption would have doses about a third to a half as high – only exceeding the MRL for the youngest age group. The toxicology literature has identified several potential health effects from PFNA exposures. A brief summary of the PFNA-specific developmental, immune, and liver effects considered the primary effects observed in animals exposed to PFAS is presented below.

- **Developmental effects.** Offspring of mice exposed to PFNA by gavage during gestation showed decreased body weight gain, transient changes in liver weight, and delays in postnatal development (eye opening, signs of male and female puberty) [39,7]. These effects, which are the basis for ATSDR's intermediate MRL for PFNA, occurred at a human equivalent dose of 1.7 µg/kg/day.
- **Immune effects.** The toxicological literature on immune effects of PFNA is limited. Acute duration exposures to PFNA caused changes in the thymus or spleen (considered to be immune-related effects) in rat and mouse studies [7]. However, no longer-duration immune studies are available, and it is not known whether these changes would occur or be relevant to immune function in humans exposed for longer periods
- **Liver effects.** PFNA exposure resulted in transient increased liver weights in pregnant mice and their offspring [39,7]. The observed liver changes do not appear to be relevant to humans.

The estimated doses for the highest PFNA concentration are orders of magnitude below the effect levels for developmental effects determined in toxicology studies. Exposure to PFNA in private well water is unlikely to increase the risk of developmental, immune, or liver effects.

Because PFNA was almost always detected with one or more other PFAS, we evaluated the potential for PFNA exposure to contribute to mixture effects.

Many homes with private wells have been provided alternate water and connection to a public water source or point-of-entry treatment systems. These actions have minimized known current exposures. Homeowners using private wells should monitor their well water quality and work with local authorities to take appropriate action to remove harmful contaminants, if needed.

#### *PFBA*

Although PFBA concentrations in well water did not exceed screening levels, the following gives a brief discussion of what is known about PFBA's health effects, so all PFAS detected in private wells in this community are included. PFBA was detected in about 48% of 2,455 private wells analyzed; almost all detections were in wells that also had detections of PFOA, PFOS, PFHxS, or PFNA. The highest concentration detected was 0.12 µg/L. The state of Minnesota derived a chronic noncancer health risk limit of 7 µg/L for PFBA based on a study that showed developmental delays and liver, blood, and thyroid changes in offspring of rats fed PFBA at a human equivalent dose of 860 µg/kg/day [14]. By applying uncertainty factors to this dose, the state derived an oral chronic reference dose of 2.9 µg/kg/day. The highest concentration of PFBA detected in the private wells would result in a maximum dose to the most sensitive age group (birth up to one year old) of 0.02 µg/kg/day. Exposure to the levels of PFBA detected would be unlikely to result in harmful health effects. However, PFBA shares potential developmental and liver endpoints with the other PFAS evaluated, and thus may contribute to possible health effects. The extent of the possible contribution is unknown.

#### *PFBS*

Although PFBS concentrations in well water did not exceed screening levels, the following gives a brief discussion of what is known about PFBS's health effects, so all PFAS detected in private wells in this community are included. PFBS was detected in about 58% of 2,739 private wells analyzed; almost all of the detections occurred in wells that also had detections of PFOA, PFOS, PFHxS, or PFNA. The highest concentration detected was 0.14 µg/L. The state of Michigan derived a chronic health-based screening value of 1 µg/L based on a different study that showed adverse kidney effects in rats fed PFBS at a human equivalent dose of 225 µg/kg/day [15]. By applying uncertainty factors to this dose, the state derived a reference dose of 0.23 µg/kg/day. The highest concentration of PFBS detected in the private wells would result in a maximum dose to the most sensitive age group (birth up to one year old) of 0.02 µg/kg/day. Exposure to the levels of PFBS detected would be unlikely to result in harmful health effects.

#### *Other PFAS present in wells*

As discussed above, other PFAS for which no CVs were available were detected in some private wells. Not enough is currently known about health effects of other PFAS to allow a quantitative evaluation of their contribution, if any, to harmful health effects. Some of these PFAS were detected infrequently at low levels (below the lowest PFAS CV available, 0.014 µg/L for PFOS)



and are not discussed further.<sup>7</sup> The below discussion provides additional qualitative information about the other PFAS most commonly detected in the private wells. Full compound names can be found in Appendix A.

PFHxA was detected in about 76% of 2,509 private wells analyzed; all but 9 of the detections were in wells that also had detections of PFOA, PFOS, PFHxS, or PFNA. The highest concentration detected was 0.42 µg/L. At this time, too few studies have been conducted on PFHxA to be able to evaluate possible health effects from PFHxA ingestion. The chemical structure of this PFAS (a short carboxylic acid chain of fewer than eight carbon atoms) suggests possibly faster elimination from the human body and lower potential for bioaccumulation compared to other PFAS [7]. However, given the lack of information, ATSDR cannot make definitive health conclusions regarding PFHxA exposure at this time.

PFHpA was detected in about 66% of 2,740 private wells analyzed; all detections were in wells that also had detections of PFOA, PFOS, PFHxS, or PFNA. The highest concentration detected was 0.42 µg/L. At this time, too few studies have been conducted on PFHpA to be able to evaluate possible health effects from its ingestion. The chemical structure of this PFAS (a short carboxylic acid chain of fewer than eight carbon atoms) suggests possibly faster elimination from the human body and lower potential for bioaccumulation compared to other PFAS [7]. However, given the lack of information, ATSDR cannot make definitive health conclusions regarding PFHpA exposure at this time.

PFPeA was detected in about 67% of 2,494 private wells analyzed; all detections were in wells that also had detections of PFOA, PFOS, PFHxS, or PFNA. The highest concentration detected was 0.23 µg/L. At this time, too few studies have been conducted on PFPeA to be able to evaluate possible health effects from its ingestion. The chemical structure of this PFAS (a short carboxylic acid chain of fewer than eight carbon atoms) suggests possibly faster elimination from the human body and lower potential for bioaccumulation compared to other PFAS [7]. However, given the lack of information, ATSDR cannot make definitive health conclusions regarding PFPeA exposure at this time.

6:2 FTSA was detected in about 3% of 1,750 private wells analyzed; all detections were in wells that also had detections of PFOA, PFOS, PFHxS, or PFNA. The highest concentration detected was 0.57 µg/L. At this time, too few studies have been conducted on 6:2 FTSA to be able to evaluate possible health effects from its ingestion [7]. Given the lack of information, ATSDR cannot make definitive health conclusions regarding 6:2 FTSA exposure at this time.

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<sup>7</sup> These other PFAS are PFTeDA (detected in 25 wells at a maximum concentration of 0.0040 µg/L); PFTrDA (detected in 12 wells at a maximum concentration of 0.0065 µg/L); PFDoDA (detected in 7 wells at a maximum concentration of 0.0074 µg/L); PFUnDA (detected in 7 wells at a maximum concentration of 0.0048 µg/L); PFDA (detected in 57 wells at a maximum concentration of 0.0058 µg/L); PFDS (detected in 1 well at a concentration of 0.002 µg/L); PFHpS (detected in 37 wells at a maximum concentration of 0.0094 µg/L); PFPeS (detected in 176 wells at a maximum concentration of 0.012 µg/L); EtFOSAA (detected in 5 wells at a maximum concentration of 0.0028 µg/L); MeFOSAA (detected in 2 wells at a maximum concentration of 0.0017 µg/L); Perfluoro(2-ethoxyethane)sulfonic acid (detected in 2 wells at a maximum concentration of 0.00067 µg/L); Perfluoro-3,6-dioxaheptanoic acid (detected in 7 wells at a maximum concentration of 0.00098 µg/L); and 4:2 FTSA (detected in 42 wells at a maximum concentration of 0.0035 µg/L).

8:2 FTSA was detected in less than 1% of 1,750 private wells analyzed; all detections were in wells that also had detections of PFOA, PFOS, PFHxS, or PFNA. The highest concentration detected was 0.044 µg/L. At this time, too few studies have been conducted on 8:2 FTSA to be able to evaluate possible health effects from its ingestion [7]. Given the lack of information, ATSDR cannot make definitive health conclusions regarding 8:2 FTSA exposure at this time.

FOSA was detected in about 2% of 1,170 private wells analyzed; all detections were in wells that also had detections of PFOA, PFOS, PFHxS, or PFNA. The highest concentration detected was 0.059 µg/L. At this time, too few studies have been conducted on FOSA to be able to evaluate possible health effects from its ingestion [7]. Given the lack of information, ATSDR cannot make definitive health conclusions regarding FOSA exposure at this time.

### ***PFAS mixtures evaluation***

Many wells contained detections of multiple PFAS. For mixtures, ATSDR recommends a tiered approach to determine whether further evaluation of mixture effects is necessary [40]:

- In Tier 1, a hazard quotient is calculated for each of the identified contaminants. The hazard quotient is the ratio of the estimated dose of a contaminant and its corresponding noncancer or cancer-based health guideline. For the PFAS assessed in this report, we can only evaluate mixtures using noncancer health guidelines.<sup>8</sup> Mixtures of contaminants with hazard quotients greater than 0.1 are carried forward for Tier 2 analysis.

Table 3 shows that for the private wells evaluated in this report, PFOA, PFOS, PFHxS, and PFNA all had hazard quotients greater than 0.1 in some wells. PFBA and PFBS had hazard quotients lower than 0.1 in all wells and are not carried forward to Tier 2. This Tier 1 analysis identified four PFAS in 1,101 wells to be included in the Tier 2 analysis.<sup>9</sup> See Appendix B for further details.

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<sup>8</sup> Intermediate MRLs based on noncancer effects are available for PFOA, PFOS, PFHxS, and PFNA. State reference doses based on noncancer effects are available for PFBA and PFBS. No official cancer slope factors exist for PFAS at the time of this report. Potential cancer effects are discussed later in this report.

<sup>9</sup> Of the 1,644 wells not included in further mixtures analysis, we note that 1,004 of them included detections of other PFAS for which no health guidelines exist. ATSDR cannot evaluate the potential mixture effects of these other PFAS.

**Table 3. Tier 1 (hazard quotient) analysis of PFAS in private wells near the Saint-Gobain Merrimack, NH facility**

PFAS	Highest estimated hazard quotient in any well†	Number of wells with hazard quotient $\geq 0.1$ ‡	Number of those wells at least one other PFAS with hazard quotient $\geq 0.1$ ‡	Include PFAS in Tier 2 mixtures evaluation?
PFOA	76	2,362	1,101	Yes
PFOS	9	1,097	1,088	Yes
PFHxS	2	34	33	Yes
PFNA	4	24	24	Yes
PFBA	0.007	0	Not applicable	No
PFBS	0.09	0	Not applicable	No

†Hazard quotient is the highest dose (for children from birth up to one year old with high-end water consumption) divided by the minimal risk level or reference dose listed in Table 2. Individual contaminants with hazard quotient greater than one are evaluated further, and those wells with more than one PFAS hazard quotient greater than 0.1 are included in the Tier 2 mixtures evaluation.

‡Numbers of wells are not additive, since some wells contained multiple PFAS.

- In Tier 2, for multi-component mixtures, all hazard quotients (regardless of the target organ) are summed to obtain a hazard index. Mixtures with a hazard index greater than 1 are carried forward to Tier 3 analysis. Tier 2 analysis assumes that doses are additive. Of the 1,101 properties with private wells evaluated in Tier 2, 670 of them had a hazard index greater than 1 and were evaluated further.
- Tier 3 analysis is a detailed analysis of potential mixture effects, considering, for example, shared target toxicities of each mixture component, sensitive subpopulations, or more refined estimates of potential exposure to the mixture.

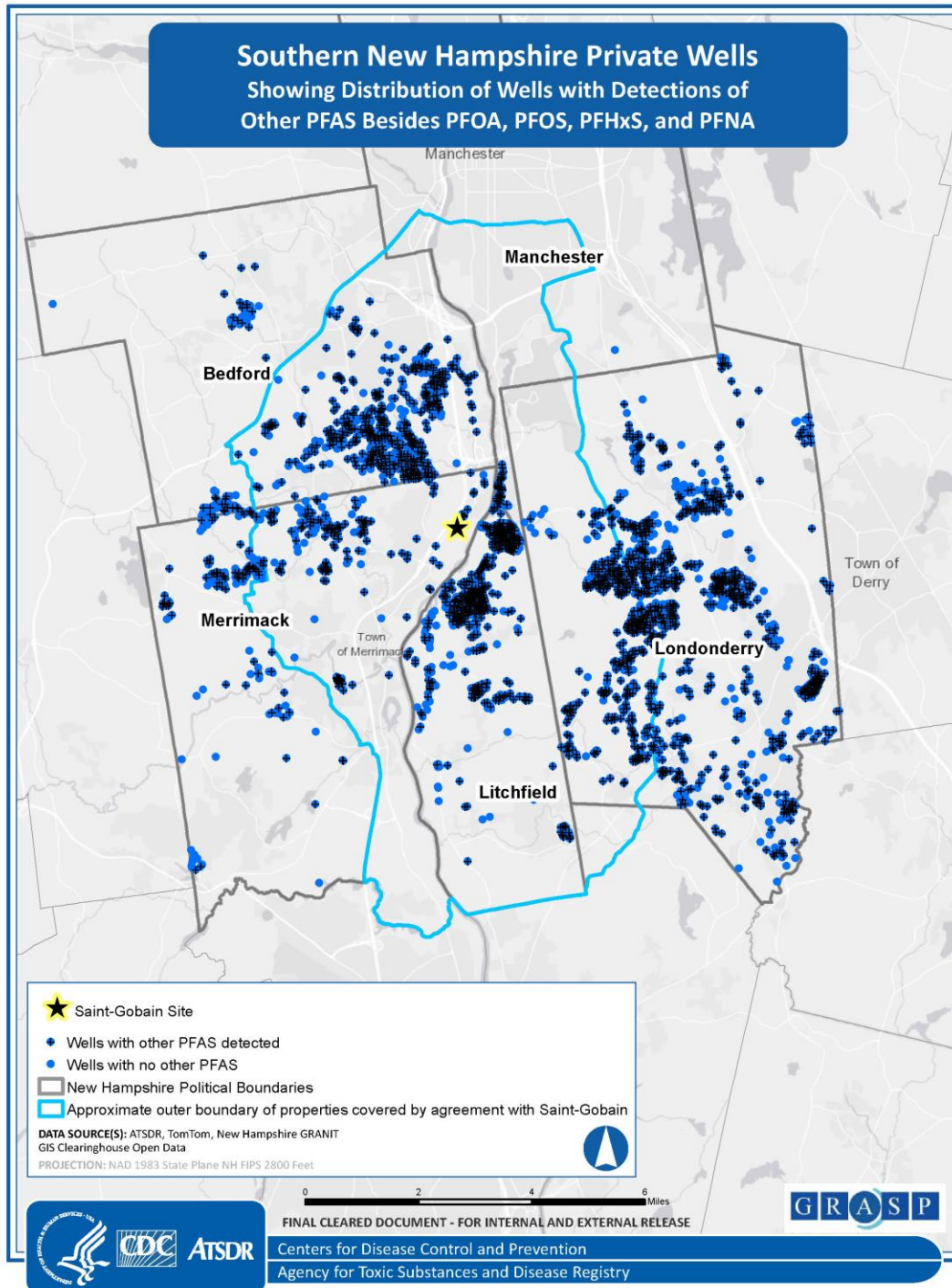
Discussion of our findings from the mixtures analysis follows. More information about ATSDR's mixtures evaluation process is in Appendix B, beginning on page B-7.

Of the 1,101 private wells with potential mixture effects, 670 wells had a hazard index greater than 1 and were evaluated further. Toxicological literature suggests that PFOA, PFOS, PFHxS, and PFNA may share sensitive endpoints including developmental, immunological, and liver effects. ATSDR evaluated potential health implications from exposure to mixtures of these four PFAS by adding the estimated doses of PFOA, PFOS, PFHxS, and PFNA for each well. Because PFOA is the main PFAS driving public health concern at this site, we compared the summed dose to PFOA effect levels for harmful health effects.

This comparison identified 34 additional wells of potential concern that were not identified through the individual PFAS analysis. Exposure to contaminants from these wells could increase the risk of developmental effects, with increasing risk and additional risk of immune and liver effects as overall doses increase. In addition, most of the wells assessed for mixture effects contained PFOS, which could contribute to immune effects at lower concentrations than those observed for PFOA alone.

The presence of other PFAS (besides those that could be evaluated quantitatively) lends uncertainty to the health evaluation. Figure 3 shows the wells in the area, indicating those with detection of other PFAS not included in the mixtures evaluation due to a lack of toxicological information. Most of these other PFAS were detected along with PFOA, PFOS, PFHxS, and PFNA in private wells, and scientists do not know how their presence may affect health implications of exposure to the mixture. Also, many of the wells' analyses only included a limited number of the other PFAS, so it is possible that some wells contained other PFAS which were not analyzed. ATSDR recommends all private wells remaining in use in the area be tested regularly for a full range of PFAS and other contaminants.

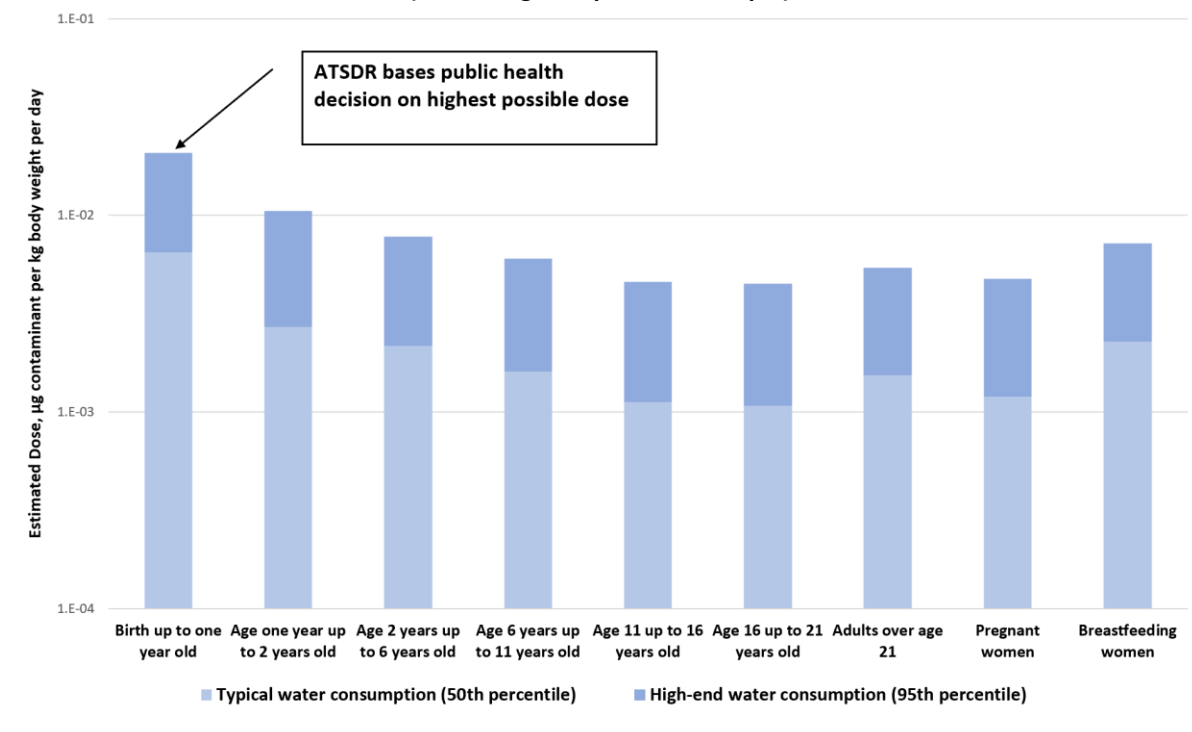
Figure 3. Distribution of private wells near the Saint-Gobain site in Merrimack, NH with other PFAS that could not be evaluated quantitatively through mixtures framework



**Summary - noncancer health effects**

ATSDR makes its public health recommendations to protect the most highly exposed and sensitive group—in this case, children between birth and one year of age who drink higher amounts of water from their private well. Other groups and those who consume more typical amounts of water will have less exposure, and thus less risk. The potential health implications from drinking from a particular private well depend not only on the levels of PFAS present, but also on the age of the child or adult drinking and how much water they drink. This concept is depicted in Figure 4, which shows how the dose calculated for one contaminant concentration varies for different age groups consuming different amounts of water every day.

**Figure 4. How age group and water consumption affect estimated dose at a set contaminant concentration (0.1 micrograms per liter example)**

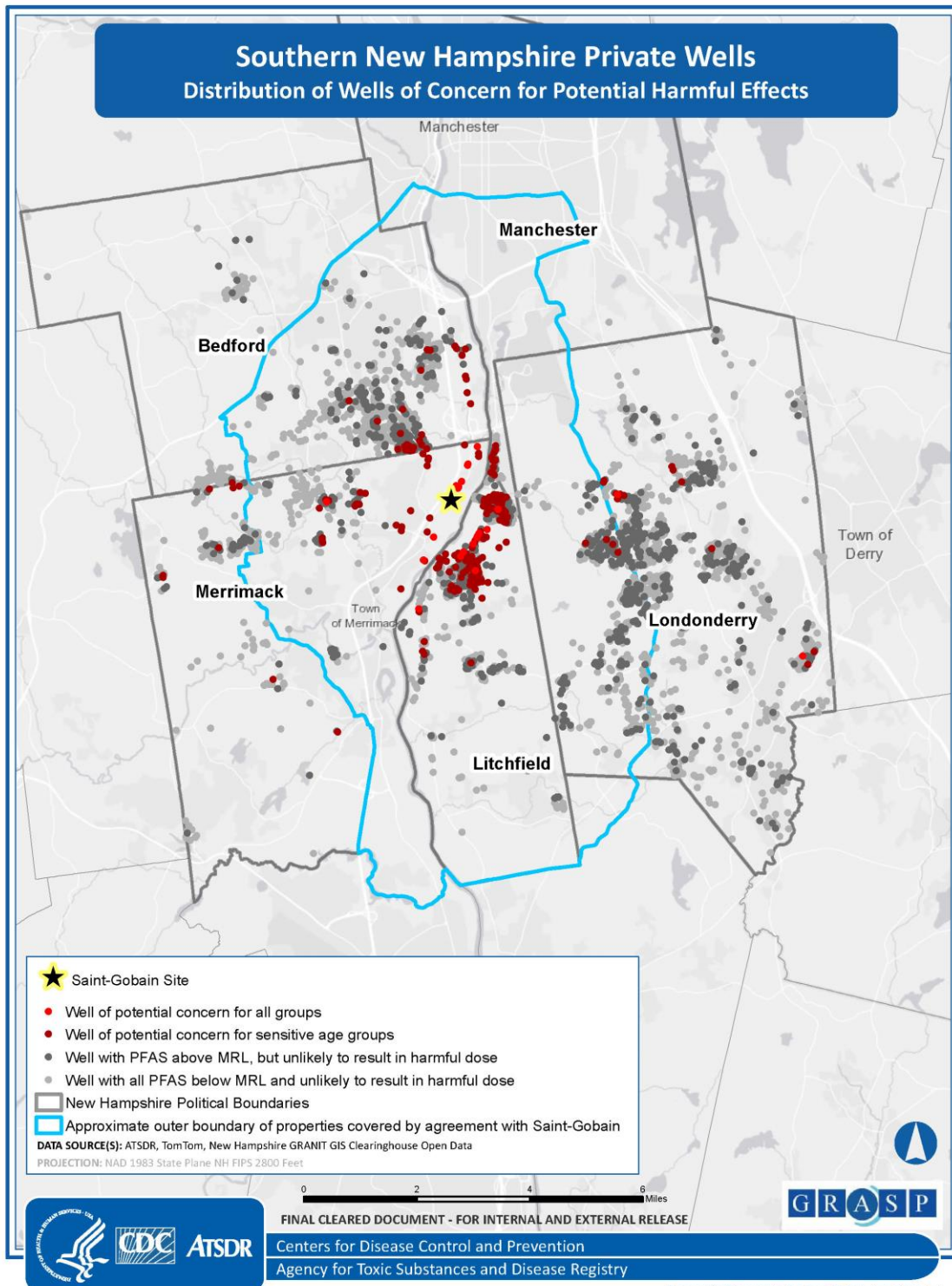


Based on the most sensitive age group drinking a high-end amount of water every day, past consumption from 237 private wells in the five towns evaluated could have increased the risk for harmful health effects. About 9% (22 wells) of those wells had PFAS concentrations high enough to increase risk for all age groups. Developmental effects would likely be the most sensitive effect, with the possibility of immune or liver effects increasing as contaminant levels increased.

Figure 5 shows the spatial distribution of private wells included in this evaluation and those that could have resulted in harmful health effects for past exposures.<sup>10</sup> Private wells with PFAS

<sup>10</sup> As stated earlier, affected private wells in the area have been connected to a treated public water source or provided point-of-entry treatment systems, so harmful exposures should no longer be occurring. Homeowners still using private wells in the area should have their water tested and use an alternate drinking water source if PFAS contamination is found.

Figure 5. Distribution of private wells of concern for potential harmful health effects near the Saint-Gobain site in Merrimack, NH





concentrations that could have posed a risk of harmful health effects to sensitive age groups are shown in dark red, and those with higher PFAS concentrations that could have posed a risk to all age groups are shown in brighter red. Wells of concern are present in each of the five towns included in the evaluation; most appear to be concentrated in the areas closer to the Saint-Gobain facility.

### **Cancer effects from exposure to PFOA and other PFAS**

Analysis of human epidemiological studies on PFOA exposure suggest that PFOA exposure is associated with some types of cancer, including kidney, testicular, and prostate cancers; a causal relationship has not been proven [7,41–44]. Animal studies have shown some evidence that PFOA might cause several cancers, including liver, testicular, kidney, forestomach, thyroid, and pancreatic cancers [7,45,46]. Although we do not know if cancer at these sites in animals results from a mode of action that is relevant to humans, an association between PFOA exposure and kidney and testicular cancers have been shown in both human and animal studies.

Based on available information, EPA has concluded that there is suggestive evidence of carcinogenic potential of PFOA in humans [43]. The International Agency for Research on Cancer (IARC) has classified PFOA as possibly carcinogenic to humans based on limited evidence in humans, including a positive association observed for cancers of the testis and kidney, and on limited evidence in experimental animals [44].

For PFOS, EPA has concluded that there is suggestive evidence of its carcinogenic potential based on limited evidence of liver cancer in rats [47]. Little to no information is currently available on the carcinogenicity of PFNA, PFHxS, or other PFAS.

Currently, ATSDR cannot estimate a quantitative cancer risk for PFOA, PFOS, or other PFAS. At this time, carcinogenic potential for most PFAS has not been fully assessed, and the science is too limited to quantify risk.<sup>11</sup> The increased risk of developing cancer from exposure to PFAS in the area is uncertain.

Concerns about cancer risks led the NH DHHS to review cancer incidence in the town of Merrimack in a 2018 report [49]. Between 2004 and 2014<sup>12</sup> in Merrimack, there were no statistical differences between observed diagnoses of any type of cancer and the number expected based on New Hampshire standard cancer incidence rates.

The NH DHHS cancer review covered a different population than evaluated in this report. The cancer review included all residents of Merrimack, not only private well users, and it did not include any residents of Litchfield, Londonderry, Bedford, or Manchester.

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<sup>11</sup> In 2016, EPA used data from a rat study of PFOA exposure and testicular cancer to calculate a provisional PFOA oral cancer slope factor of  $7 \times 10^{-5}$  per  $\mu\text{g}/\text{kg}/\text{day}$  [43]. However, this was not an official oral cancer slope factor. Findings of more recent studies suggest that the provisional cancer slope factor is no longer appropriate for estimating PFOA cancer risk, but as of this date an alternative factor has not been developed.

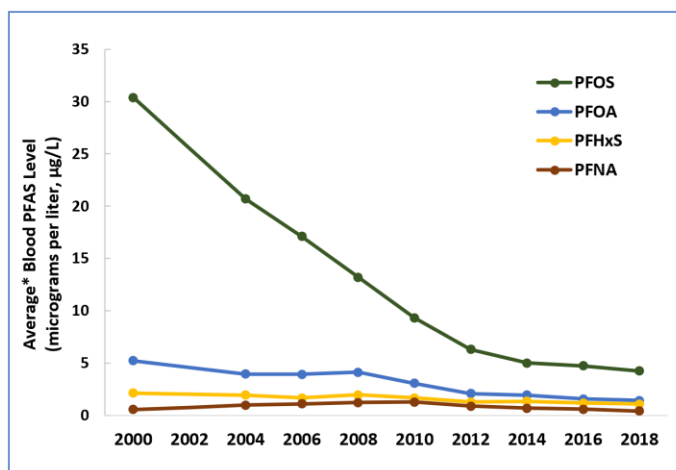
<sup>12</sup> Data for lung and bronchus and prostate cancer were only available through 2013.



**PFAS levels in blood: U.S. population versus private well users near this site**

PFAS are retained in the human body and can be measured in a person’s blood serum. Since 1999, the National Health and Nutrition Examination Survey (NHANES) has measured blood PFAS as part of its program to evaluate the health and nutrition of adults and children in the United States [50]. As shown in Figure 6, NHANES data has shown a steady decline in serum PFOS and PFOA levels since 2002, when these substances began being phased out of production and use.

**Figure 6. U.S. population's average\* blood serum PFAS levels over time, as measured through the National Health and Nutrition Examination Survey**



\*Average = geometric mean

Data Sources: NHANES fourth report [50];

[https://www.cdc.gov/exposurereport/pfas\\_early\\_release.html](https://www.cdc.gov/exposurereport/pfas_early_release.html).

Although science does not yet allow us to tell what levels of PFAS in serum can cause harmful health effects, blood serum PFAS levels can be useful to compare against population averages to determine if unusual exposures may have occurred.

Responding to the PFAS contamination found in private wells around the Saint-Gobain Performance Plastics facility, NH DHHS expanded an existing blood testing program to include the southern New Hampshire area. In 2016 and 2017, the state measured PFAS in the blood of 219 private drinking water well users. NH DHHS reported the findings in 2018 [51,52]. The people who participated in this blood testing may not be representative of all users of the private wells evaluated in this report. However, this testing provided relevant data on possible exposures. We provide a brief summary of the findings, compared against NHANES data from roughly the same timeframe, below.

The NH DHHS reports state that PFOA, PFOS, and PFHxS were detected in over 95% of the people tested; these are the only PFAS for which results were discussed. As summarized in Table 4, PFOA levels in private well users’ blood appeared to be significantly elevated compared to the general U.S. population. Almost half of private well users from the NH DHHS testing had PFOA blood levels that would place them in the top 5% of exposure measured in the general U.S.

population. In contrast, PFOS and PFHxS blood serum concentrations were similar to general population levels.

Because PFOA was the predominant contaminant in private well water, these blood results support the general conclusion that private drinking well water contamination could have led to elevated exposures, consistent with the findings of the evaluation in this health consultation. These findings, however, can't be used to directly predict a person's PFAS blood level from their well water PFAS concentration, or vice versa. ATSDR did not access the original data from the blood testing and has not examined the relationship between serum PFAS results and PFAS concentration in private wells used.<sup>13</sup> The people who participated in the New Hampshire blood testing may not be representative of all private well users in the area. In addition, a person's blood PFAS level could include exposures from various sources, including other environmental media, food, or consumer products, in addition to well water.

**Table 4. ATSDR summary of NH private well users' 2016-2017 PFAS blood testing results compared to U.S. population data**

PFAS	NH private well users geometric mean serum concentration for 2016-17 sampling in µg/L	General U.S. population geometric mean serum concentration for survey years 2015-16, 2017-18 in µg/L	NH private well users 95 <sup>th</sup> percentile* serum concentration for 2016-17 sampling in µg/L	General U.S. population 95 <sup>th</sup> percentile* serum concentration for survey years 2015-16, 2017-18 in µg/L	Approximate % of NH private well users that exceeded the U.S. general population 95 <sup>th</sup> percentile*
PFOA	4.4	1.6, 1.4	26.6	4.2, 3.8	37-64%
PFOS	5.4	4.7, 4.3	16.4	18.3, 14.6	1-9%
PFHxS	1.3	1.2, 1.1	3.4	4.9, 3.7	1-6%

\*The 95<sup>th</sup> percentile is that blood serum concentration that 95% of the results fell below. Measured values within a given population would be expected to exceed the 95<sup>th</sup> percentile only about 5% of the time, on average.

Data sources: NH WISDOM website [52], [https://www.cdc.gov/exposurereport/pfas\\_early\\_release.html](https://www.cdc.gov/exposurereport/pfas_early_release.html)

General U.S. population statistics from values reported for 2015-16 and for 2017-18 survey years reported at [https://www.cdc.gov/exposurereport/pfas\\_early\\_release.html](https://www.cdc.gov/exposurereport/pfas_early_release.html), rounded to one decimal.

### Health considerations for susceptible populations

ATSDR is committed to considering potential health effects of exposure to all groups, including those that might be unusually susceptible to environmental contamination. Pregnant women, the developing fetus, infants, children, and people of all age groups with certain pre-existing conditions might be unusually vulnerable to harmful health effects from PFAS exposure.

<sup>13</sup> The NH DHHS summary report for the southern New Hampshire private well users states, “Individuals with higher concentrations of PFOA in their private well water have higher blood PFOA levels” [51]. ATSDR did not examine the raw data to verify this statement.

- Pregnant women exposed to excessive PFAS levels could have an increased risk of high blood pressure or pre-eclampsia. High PFOA or PFOS levels in pregnant women's blood serum were associated with decreases in their babies' birth weights, but the changes were small and may not be clinically relevant.
- Infants may be exposed to PFAS through their mother's milk. Developmental effects are the most sensitive effect resulting from any early life exposure. However, breastfeeding provides many health and nutritional benefits to a child, including reduced risk of ear and respiratory infections, asthma, obesity, and sudden infant death syndrome. Breastfeeding can also help lower a mother's risk of high blood pressure, type 2 diabetes, and ovarian and breast cancer [53]. In general, CDC and the American Academy of Pediatrics recommend breastfeeding, despite the presence of chemical toxicants [54,55]. A woman's decision to breastfeed is a personal choice, made after consideration of many different factors specific to the mother and child, and best made in consultation with her healthcare provider. ATSDR has developed information to guide doctors in this decision-making process (See <https://atsdr.cdc.gov/pfas/docs/clinical-guidance-12-20-2019.pdf>). Women who plan to breastfeed should reduce their potential exposures to toxic substances as much as possible.
- Infants may also be exposed to PFAS through formula made with contaminated water. In addition to exposure from water, infants could have additional exposure, such as from hand-to-mouth behavior after contacting carpets or other household items previously treated with PFAS. In this report, ATSDR based its public health decisions on infants, which would have the highest dose because of their higher water intake and smaller body weight compared to other age groups. Children exposed to contaminated water also have a greater dose of PFAS compared to adults because of higher contaminant intakes in proportion to body size, and they may also be exposed to PFAS from hand-to-mouth behavior. Developmental effects would be the most sensitive adverse health effect resulting from early life exposure. PFAS exposure may also decrease children's antibody responses to childhood vaccines.
- People of all age groups with certain pre-existing conditions could be more susceptible to harm from PFAS exposures. For example, exposure to certain PFAS could increase cholesterol levels in some people. A greater health impact could result if the person exposed already has high cholesterol or other risk factors for cardiovascular disease. Similarly, PFAS exposure could disproportionately affect people who already have compromised immune system or liver function or who have high blood pressure. More research is needed to understand how exposure to PFAS might affect people with pre-existing risk factors for cardiovascular and other diseases.

## Conclusions

**Before actions began in 2016 to reduce exposures, drinking private well water contaminated with PFAS could have increased the risk for harmful health effects for some community members.**

Most of the private wells evaluated in the five towns of Merrimack, Litchfield, Londonderry, Bedford, and Manchester were contaminated with PFAS. PFOA was detected most frequently and at the highest concentrations. Based on ATSDR's evaluation of both individual PFAS and PFAS mixture effects detailed in this report, more than 230 out of 2,745 wells had PFAS at levels that could harm infants or young children, and about 9% of those wells had levels that could harm all age groups. Developmental effects are the most likely possible health effects from exposure, and the risk of developmental effects would increase as PFAS levels and exposure increased. Immune or liver effects would also be possible from exposure to the highest PFAS levels. Other sources of PFAS exposure (such as from food or consumer products) could increase the risk of harmful health effects beyond the risk from the drinking water exposures alone.

The remaining wells, with lower or no detections of PFAS, are not expected to have harmed health. However, this conclusion is uncertain. Many wells were sampled only once, and the actual PFAS levels could have fluctuated over time. Also, knowledge about health effects of the PFAS evaluated is still evolving, and many wells contained other PFAS which have not been studied enough to evaluate the potential for health effects.

The increased risk of developing cancer from exposure to PFAS in the area is uncertain. There is suggestive evidence that both PFOA and PFOS are carcinogenic, but the science on PFOA, PFOS, and other PFAS is too limited at this time to quantify risk.

**Currently, harmful exposures to PFAS in private wells have been minimized by providing alternate water and taking other actions. People who continue to drink contaminated, untreated private well water may still have an increased risk for harmful health effects.**

Since 2016, bottled water has been provided to residents whose private wells were affected by PFAS. More than 750 private wells in the area have been switched to treated public water or equipped with point-of-entry treatment systems which are regularly tested for treatment effectiveness. Some private wells with low levels of PFAS, or wells with no detections, may remain in use. Based on the current science, harmful health effects are unlikely if PFAS concentrations in those wells remain low. Residents drinking from private wells that were never tested, or who were offered but declined alternate water, may experience harmful health effects if they drink water with high PFAS concentrations.

## Recommendations

- Private well owners who had potentially harmful exposures in the past should discuss their exposure with their health care provider and consider taking steps to reduce other potential PFAS exposures, such as those from consumer products containing PFAS.
- Residents should reduce exposure from background sources of PFAS by avoiding or limiting the use of products containing PFAS. Examples of products that may contain PFAS include food packing materials, stain resistant carpets, water resistant clothing, cleaning products, and some cosmetics.
- ATSDR recommends nursing mothers continue to breastfeed and contact their healthcare providers with specific concerns. ATSDR is available to consult with healthcare providers as needed. To help protect formula-fed infants from potential exposure, caregivers should use pre-mixed formula or reconstitute dry formula with water sources not containing PFAS.
- Residents using point-of-entry treatment systems to remove PFAS from private well water should have the systems maintained and checked periodically to ensure removal effectiveness.
- Residents continuing to drink from private wells should monitor their well water quality and should work with local authorities to take appropriate action to remove harmful contaminants, if needed.
- ATSDR will work with NH DES and NH DHHS to identify any private wells with PFAS levels of concern that have not been addressed through previous actions.

ATSDR is available to discuss individual results with private well owners and will continue to be available, upon request, to answer other public health questions related to the site.

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## Appendix A. Full names and chemical information for PFAS in report

**Table A1. Full names, chemical formulae, and Chemical Abstract Services Registry numbers for compounds included in the data provided to ATSDR by NH DES (listed in order of increasing total number of carbon atoms, detected substances shown in bold)**

PFAS Abbreviation	Full Name	Chemical Formula	Chemical Abstract Services Registry Number
<b>PFBA</b>	Perfluorobutyric acid	C <sub>3</sub> F <sub>7</sub> COOH	375-22-4
<b>PFBS</b>	Perfluorobutane sulfonate anion Perfluorobutane sulfonic acid	C <sub>4</sub> F <sub>9</sub> SO <sub>3</sub> <sup>-</sup> C <sub>4</sub> F <sub>9</sub> SO <sub>3</sub> H	45187-15-3 375-73-5 or 59933-66-3
<b>PFPeA</b>	Perfluoropentanoic acid	C <sub>4</sub> F <sub>9</sub> COOH	2706-90-3
Perfluoro-3-methoxypropanoic acid	Perfluoro-3-methoxypropanoic acid	C <sub>4</sub> HF <sub>7</sub> O <sub>3</sub>	377-73-1
<b>Perfluoro(2-ethoxyethane)sulfonic acid</b>	Perfluoro(2-ethoxyethane)sulfonic acid	C <sub>4</sub> HF <sub>9</sub> O <sub>4</sub> S	113507-82-7
Perfluorobutylsulfonamide	Perfluorobutylsulfonamide	C <sub>4</sub> H <sub>2</sub> F <sub>9</sub> NO <sub>2</sub> S	30334-69-1
<b>PFPeS</b>	Perfluoropentane sulfonic acid	C <sub>5</sub> F <sub>11</sub> SO <sub>3</sub> H	2706-91-4
<b>PFHxA</b>	Perfluorohexanoic acid	C <sub>5</sub> F <sub>11</sub> COOH	307-24-4
Perfluoro(4-methoxybutanoic) acid	Perfluoro(4-methoxybutanoic) acid	C <sub>5</sub> HF <sub>9</sub> O <sub>3</sub>	863090-89-5
<b>Perfluoro-3,6-dioxaheptanoic acid</b>	Perfluoro-3,6-dioxaheptanoic acid	C <sub>5</sub> HF <sub>9</sub> O <sub>4</sub>	151772-58-6
<b>PFHxS</b>	Perfluorohexane sulfonate anion Perfluorohexane sulfonic acid	C <sub>6</sub> F <sub>13</sub> SO <sub>3</sub> <sup>-</sup> C <sub>6</sub> F <sub>13</sub> SO <sub>3</sub> H	108427-53-8 355-46-4
<b>4:2 FTSA</b>	4:2 Fluorotelomer sulfonate anion 4:2 Fluorotelomer sulfonic acid	C <sub>4</sub> F <sub>9</sub> CH <sub>2</sub> CH <sub>2</sub> SO <sub>3</sub> <sup>-</sup> C <sub>4</sub> F <sub>9</sub> CH <sub>2</sub> CH <sub>2</sub> SO <sub>3</sub> H	414911-30-1 757124-72-4
GenX	Hexafluoropropylene oxide dimer acid Ammonium salt form	C <sub>6</sub> HF <sub>11</sub> O <sub>3</sub> C <sub>6</sub> H <sub>4</sub> F <sub>11</sub> NO <sub>3</sub>	13252-13-6 62037-80-3
<b>PFHpA</b>	Perfluoroheptanoic acid	C <sub>6</sub> F <sub>13</sub> COOH	375-85-9
1-Propene, 1,1,2,3,3,3-hexafluoro-, dimer	1-Propene, 1,1,2,3,3,3-hexafluoro-, dimer	C <sub>6</sub> F <sub>12</sub>	13429-24-8
Perfluorohexanesulfonamide	Perfluorohexanesulfonamide	C <sub>6</sub> H <sub>2</sub> F <sub>13</sub> NO <sub>2</sub> S	41997-13-1
<b>PFHpS</b>	Perfluoroheptane sulfonate anion Perfluoroheptane sulfonic acid	C <sub>7</sub> F <sub>15</sub> SO <sub>3</sub> <sup>-</sup> C <sub>7</sub> F <sub>15</sub> SO <sub>3</sub> H	146689-46-5 375-92-8
DONA / ADONA	4,8-Dioxa-3H-perfluorononanoic acid (DONA) Ammonium salt form (ADONA)	C <sub>7</sub> H <sub>2</sub> F <sub>12</sub> O <sub>4</sub> C <sub>7</sub> H <sub>5</sub> F <sub>12</sub> NO <sub>4</sub>	919005-14-4 958445-44-8

PFAS Abbreviation	Full Name	Chemical Formula	Chemical Abstract Services Registry Number
PFOA	Perfluorooctanoic acid	C <sub>7</sub> F <sub>15</sub> COOH	335-67-1
PFOS	Perfluorooctane sulfonate anion Perfluorooctane sulfonic acid	C <sub>8</sub> F <sub>17</sub> SO <sub>3</sub> <sup>-</sup> C <sub>8</sub> F <sub>17</sub> SO <sub>3</sub> H	45298-90-6 1763-23-1
6:2 FTSA	6:2 Fluorotelomer sulfonate anion 6:2 Fluorotelomer sulfonic acid	C <sub>6</sub> F <sub>13</sub> CH <sub>2</sub> CH <sub>2</sub> SO <sub>3</sub> <sup>-</sup> C <sub>6</sub> F <sub>13</sub> CH <sub>2</sub> CH <sub>2</sub> SO <sub>3</sub> H	425670-75-3 27619-97-2
FOSA	Perfluorooctane sulfonamide	C <sub>8</sub> F <sub>17</sub> SO <sub>2</sub> NH <sub>2</sub>	754-91-6
9-Chlorohexadecafluoro-3-oxanone-1-sulfonic acid	9-Chlorohexadecafluoro-3-oxanone-1-sulfonic acid	C <sub>8</sub> HCIF <sub>16</sub> O <sub>4</sub> S	756426-58-1
PFNA	Perfluorononanoic acid	C <sub>8</sub> F <sub>17</sub> COOH	375-95-1
PFNS	Perfluorononane sulfonic acid	C <sub>9</sub> F <sub>19</sub> SO <sub>3</sub> H	474511-07-4 or 68259-12-1
MeFOSA	N-Methyl perfluorooctane sulfonamide	C <sub>8</sub> F <sub>17</sub> SO <sub>2</sub> NH(CH <sub>3</sub> )	31506-32-8
PFDA	Perfluorodecanoic acid	C <sub>9</sub> F <sub>19</sub> COOH	335-76-2
PFDS	Perfluorodecane sulfonate anion Perfluorodecane sulfonic acid	C <sub>10</sub> F <sub>21</sub> SO <sub>3</sub> <sup>-</sup> C <sub>10</sub> F <sub>21</sub> SO <sub>3</sub> H	126105-34-8 335-77-3
8:2 FTSA	8:2 Fluorotelomer sulfonate anion 8:2 Fluorotelomer sulfonic acid	C <sub>8</sub> F <sub>17</sub> CH <sub>2</sub> CH <sub>2</sub> SO <sub>3</sub> <sup>-</sup> C <sub>8</sub> F <sub>17</sub> CH <sub>2</sub> CH <sub>2</sub> SO <sub>3</sub> H	481071-78-7 39108-34-4
EtFOSA	N-Ethyl perfluorooctane sulfonamide	C <sub>8</sub> F <sub>17</sub> SO <sub>2</sub> NH(C <sub>2</sub> H <sub>5</sub> ) (sulfluramid)	4151-50-2
11-Chloroeicosafluoro-3-oxaundecane-1-sulfonic acid	11-Chloroeicosafluoro-3-oxaundecane-1-sulfonic acid	C <sub>10</sub> HCIF <sub>20</sub> O <sub>4</sub> S	763051-92-9
PFUnDA	Perfluoroundecanoic acid	C <sub>10</sub> F <sub>21</sub> COOH	2058-94-8
MeFOSE	N-Methyl perfluorooctane sulfonamidoethanol	C <sub>8</sub> F <sub>17</sub> SO <sub>2</sub> N(CH <sub>3</sub> )CH <sub>2</sub> CH <sub>2</sub> OH	24448-09-7
MeFOSAA	N-Methyl perfluorooctane sulfonamidoacetic acid	C <sub>8</sub> F <sub>17</sub> SO <sub>2</sub> N(CH <sub>3</sub> )CH <sub>2</sub> COOH	2355-31-9
PFDoDA	Perfluorododecanoic acid	C <sub>11</sub> F <sub>23</sub> COOH	307-55-1
PFDoDS	Perfluorododecane sulfonic acid	C <sub>12</sub> F <sub>25</sub> SO <sub>3</sub> H	79780-39-5
10:2 FTSA	10:2 Fluorotelomer sulfonic acid	C <sub>10</sub> F <sub>21</sub> CH <sub>2</sub> CH <sub>2</sub> SO <sub>3</sub> H	120226-60-0
ETFOSE	N-Ethyl perfluorooctane sulfonamidoethanol	C <sub>8</sub> F <sub>17</sub> SO <sub>2</sub> N(C <sub>2</sub> H <sub>5</sub> )CH <sub>2</sub> CH <sub>2</sub> OH	1691-99-2

<b>PFAS Abbreviation</b>	<b>Full Name</b>	<b>Chemical Formula</b>	<b>Chemical Abstract Services Registry Number</b>
<b>EtFOSAA</b>	N-Ethyl perfluorooctane sulfonamidoacetic acid	$C_8F_{17}SO_2N(C_2H_5)CH_2COOH$	2991-50-6
<b>PFTrDA</b>	Perfluorotridecanoic acid	$C_{12}F_{25}COOH$	72629-94-8
<b>PFTeDA</b>	Perfluorotetradecanoic acid	$C_{13}F_{27}COOH$	376-06-7
PFHxDA	Perfluorohexadecanoic acid	$C_{15}F_{31}COOH$	67905-19-5
PFODA	Perfluorooctadecanoic acid	$C_{17}F_{35}COOH$	16517-11-6

**Bold abbreviations** indicate the substance was analyzed and detected at least once in private well testing provided to ATSDR. Data from [56,57,7]

## Appendix B. ATSDR evaluation process and details

### Exposure pathway analysis

For contaminant exposure to occur to a person, there must be an uninterrupted chain whereby the chemical moves from its source to the person's body, where harmful effect might occur. ATSDR terms this chain an *exposure pathway*. Exposure pathways consist of five elements: a contamination *source*; *transport* of the contaminant through an environmental medium like air, soil, or water; an *exposure point* where people can come in contact with the contaminant; an *exposure route* whereby the contaminant can be taken into the body; and an *exposed population* of people actually coming in contact with site contaminants [58]. ATSDR evaluates each of these five elements to determine whether exposure is occurring to community members living near a site. Exposure may occur through multiple different pathways. If exposure through a particular pathway is determined to have occurred, it does not necessarily mean that harmful health effects will occur. A chemical's ability to harm health depends on many factors, including how much of the chemical is present, how long and how often a person is exposed to the chemical, and how toxic the chemical is. Further evaluation of the specific exposure occurring is needed to determine whether the exposure could cause harmful health effects.

The private well pathway evaluated in this health consultation consists of the following five elements:

- **Source** – Releases of PFAS from the Saint-Gobain site into the air<sup>14</sup>
- **Transport** – PFAS dispersing in air, settling onto the ground, and washing down into underlying groundwater used for drinking water for private wells
- **Exposure Point** – Drinking water taps of people living in the area using private wells
- **Exposure Route** – Ingestion of drinking water provided by private wells
- **Exposed Population** – People living or working in the area who drink or drank water from private wells

This exposure pathway is considered

- **Complete for past exposures** because the presence of PFAS used in the Saint-Gobain processes was confirmed in many wells, and people used these wells as a drinking water source
- **Incomplete for current exposures** for people who are no longer drinking from private wells or if their water is treated to remove PFAS
- **Complete for current exposures** for people who continue to drink from private wells with any detection of PFAS

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<sup>14</sup> Although PFAS contamination of private wells in the area is believed to primarily originate from air emissions from operations of the Saint-Gobain facility, the groundwater data which we used for estimating PFAS exposure may have included detections of PFAS chemicals from other sources in the area, such as landfills, fire training facilities, or other unknown sources. Our conclusions and recommendations are general and based on exposure as described by private well data; we make no attempt to attribute measured contaminants to the site or to other sources.

- **Potential for current exposures** for area residents whose well water has never been tested

No data describing PFAS levels in other environmental media besides drinking water near the site were available to us. Therefore, we did not consider any other potential past, present, or future exposure pathways in this report, including

- Inhalation exposure to PFAS released into the air from the facility;
- Direct contact or incidental ingestion exposure to PFAS in soil, surface water, or sediment;
- Indirect ingestion of PFAS in biota (fish, shellfish, or plants) that may have bioaccumulated PFAS from their local environment; or
- Exposure to PFAS from consumer products in the home or community.

### Contaminant screening

In evaluating chemical contaminant data, ATSDR used comparison values (CVs) to prioritize which chemicals or which exposure points (for example, which private wells) are of most potential concern. The health-based CVs used in this report are contaminant concentrations in drinking water that are not expected to result in harmful health effects, even to a small child drinking the water every day. Exceeding a CV does not mean that health effects will occur, just that more evaluation is needed.

ATSDR develops CVs for many substances; different CVs may be developed based on noncancer or cancer health effects. In the absence of ATSDR-derived CVs, state or other agency-developed screening values may be used.

In this report, ATSDR used the following CVs for PFAS:

*Environmental Media Evaluation Guides (EMEGs)* for PFOA, PFOS, PFHxS, and PFNA; derived from the ATSDR intermediate minimal risk levels (MRLs) for these PFAS and representing estimated contaminant concentrations in drinking water that are unlikely to cause noncancer health effects.

*Michigan chronic health-based screening level* for PFBS, based on a reference dose derived by the state and considered safe for chronic exposures.

*Minnesota chronic noncancer health risk limit* for PFBA, based on a reference dose estimated by the state and considered safe for chronic exposures.

The screening of PFAS at this site is presented in the body of the report in Table 1. PFOA, PFOS, PFHxS, and PFNA exceeded their respective CV in at least one private well.

### Estimating exposure

The potential for harmful health effects from drinking water with PFAS contamination is evaluated further by estimating the *exposure dose*, or the amount of contaminant that gets into a person's body. The exposure dose is expressed as micrograms of contaminant per kilogram of body weight of the person exposed, per day ( $\mu\text{g}/\text{kg}/\text{day}$ ), and accounts for differing water consumption and different body weights of various age groups in the exposed population.



The exposure dose associated with drinking water with a particular concentration of a PFAS is given by the following equation:

$$\text{Dose } (\mu\text{g/kg/day}) = \text{PFAS concentration } (\mu\text{g/L}) \times \text{consumption (L/day)} \div \text{body weight (kg)}$$

ATSDR used standard guidance to determine drinking water consumption and body weight used in this equation to estimate exposure doses to various age groups; these assumptions are presented in Table B1 [12,13]. We used the highest concentration of each PFAS in each private well with the assumptions in Table B1 to estimate exposure doses. For example, a child less than one year old with high-end consumption of drinking water containing the highest concentration of PFOA (1.6  $\mu\text{g/L}$ ) every day will receive a PFOA dose of:

$$\text{PFOA dose} = 1.6 \mu\text{g/L} \times 1.113 \text{ L/day} \div 7.8 \text{ kg} = 0.23 \mu\text{g/kg/day}$$

**Table B 1. Assumed body weights and drinking water consumption for private well users in five towns near the Saint-Gobain facility in Merrimack, New Hampshire [9,10]**

Group	Body weight in kilograms	High-end (95 <sup>th</sup> percentile) ingestion of drinking water in liters per day	Typical (average) ingestion of drinking water in liters per day
Children from birth up to 1 year old	7.8	1.113	0.504
Children from 1 year old up to age 2	11.4	0.893	0.308
Children from 2 years old up to age 6	17.4	0.977	0.376
Children from 6 years old up to age 11	31.8	1.404	0.511
Children from 11 years old up to age 16	56.8	1.976	0.637
Children from 16 years old up to age 21	71.6	2.444	0.77
Adults 21 years old or more	80	3.092	1.227
Pregnant women	73	2.589	0.872
Lactating women	73	3.588	1.665

### Evaluating noncancer health effects

The calculated exposure doses are then compared to an appropriate health guideline for that chemical. Health guideline values are considered safe doses; that is, health effects are unlikely below this level. The health guideline value is based on valid toxicological studies for a chemical, with appropriate safety factors built in to account for human variation, animal-to-human differences, and/or the use of the lowest study doses that resulted in harmful health effects (rather than the highest dose that did not result in harmful health effects).

Health guidelines used in this report include ATSDR minimal risk levels (MRLs) for four PFAS (PFOA, PFOS, PFHxS, and PFNA) and state-derived reference doses for two other PFAS (PFBA and PFBS). A description of the derivation of these health guidelines from toxicological studies is presented below.

#### **PFOA**

ATSDR derived an intermediate oral MRL of 0.003  $\mu\text{g/kg/day}$  for PFOA based on a developmental study that observed various endpoints in offspring of pregnant mice fed a diet containing PFOA during pregnancy [7,21]. Physical development of the offspring

was measured at 15 or 17 months by examining body weight and bone structure of sacrificed mice [21]. The study found prenatal exposure to a human equivalent dose as low as 0.82 µg/kg/day was associated with skeletal changes (altered long bone structure and decreased bone density) when compared with offspring from untreated mice. ATSDR used this dose with uncertainty factors of 10 (for use of a lower effect level), 3 (for extrapolation from animals to humans with dosimetric adjustments), and 10 (for human variability) to derive the intermediate MRL.

#### **PFOS**

ATSDR derived an intermediate oral MRL of 0.002 µg/kg/day for PFOS based on a developmental study that examined groups of rats exposed to PFOS by gavage before mating, during gestation, and after giving birth [30,7]. At the lowest effect level (a human equivalent dose of 2.1 µg/kg/day), offspring of the rats showed delays in eye opening and a transient decrease in body weight. ATSDR used the human equivalent dose at which none of the developmental changes occurred, 0.515 µg/kg/day, with uncertainty factors of 3 (for extrapolation from animals to humans with dosimetric adjustments) and 10 (for human variability) and a modifying factor of 10 to derive the intermediate MRL.

The modifying factor was included because of concerns that PFOS immunotoxicity may be a more sensitive endpoint than developmental toxicity. Studies on PFOS immune toxicity lacked pharmacokinetic modeling information needed to develop an MRL directly; however, they showed effects on the immune system at serum PFOS concentrations about 10-fold lower than those in the developmental study used as the basis for the MRL.

#### **PFHxS**

ATSDR derived an intermediate oral MRL of 0.02 µg/kg/day for PFHxS based on an intermediate-duration study that exposed male and female rats to PFHxS by gavage before, during, and after mating; adult rats and the offspring were examined for numerous development and reproductive endpoints [7,36,37]. No developmental or reproductive effects were reported at any dose tested, but thyroid changes (specifically, follicular cells damage) was observed in adult male rats at a lowest-effect level corresponding to a human equivalent dose of 7.3 µg/kg/day [7,36,37]. ATSDR used the human equivalent dose at which no harmful changes occurred, 4.7 µg/kg/day, with uncertainty factors of 3 (for extrapolation from animals to humans with dosimetric adjustments) and 10 (for human variability) and a modifying factor of 10 (for database limitations) to derive the intermediate MRL.

#### **PFNA**

ATSDR derived an intermediate oral MRL of 0.003 µg/kg/day for PFNA based on a developmental study in which pregnant mice were exposed to PFNA by gavage during gestation [39,7]. Offspring showed decreased body weight gain, transient changes in liver weight, and statistically significant delays in postnatal development (eye opening, signs of male and female puberty) at a lowest-effect dose corresponding to a human equivalent dose of 1.7 µg/kg/day. ATSDR used the human equivalent dose at which none of the developmental changes occurred, 1 µg/kg/day, with uncertainty factors of 3 (for

extrapolation from animals to humans with dosimetric adjustments) and 10 (for human variability) and a modifying factor of 10 (for database limitations) to derive the intermediate MRL.

**PFBA**

The state of Minnesota derived an oral chronic reference dose of 2.9 µg/kg/day based on a study that showed developmental delays and liver, blood, and thyroid changes in offspring of rats fed PFBA at a human equivalent dose of 860 µg/kg/day [14]. The state applied uncertainty factors of 3 (for interspecies differences), 10 (for intraspecies variability), and 10 (for database uncertainty) to this dose to derive the oral chronic reference dose.

**PFBS**

The state of Michigan derived a reference dose of 0.23 µg/kg/day based on a different study that showed adverse kidney effects in rats fed PFBS at a human equivalent dose of 225 µg/kg/day [15]. The state applied uncertainty factors of 3 (for toxicodynamic differences in rats and humans), 3 (for database gap since no developmental study has been conducted), 10 (human to human variability), and 10 (for less than chronic duration) to this dose to derive the oral chronic reference dose.

If the estimated exposure dose for a chemical is less than the health guideline value, then the exposure is unlikely to cause a noncancer health effect in that specific situation. If the exposure dose for a chemical is greater than the health guideline, then the exposure dose is compared to known toxicological values for that chemical and is discussed in more detail in the evaluation report. These toxicological values are doses derived from human and animal studies summarized in the ATSDR Toxicological Profiles, reports included in EPA's Integrated Risk Information System, and in current scientific literature. A direct comparison of site-specific exposure and doses to study-derived exposures and doses that cause adverse health effects is the basis for deciding whether health effects are likely or not.

For every PFAS with an available health guideline, ATSDR calculated doses for exposure to the highest concentration measured in any private well. The doses for various age groups with high-end or typical water consumption, compared against the appropriate health guideline, are presented in Table B2.

**Table B 2. Doses for age groups with different water consumption exposed to the highest concentrations measured in private wells compared to corresponding health guidelines**

Age group	Dose for exposure to 1.6 µg/L PFOA, µg/kg/day (high-end / typical consumption)	Dose for exposure to 0.12 µg/L PFOS, µg/kg/day (high-end / typical consumption)	Dose for exposure to 0.24 µg/L PFHxS, µg/kg/day (high-end / typical consumption)	Dose for exposure to 0.085 µg/L PFNA, µg/kg/day (high-end / typical consumption)	Dose for exposure to 0.14 µg/L PFBA, µg/kg/day (high-end / typical consumption)	Dose for exposure to 0.14 µg/L PFBS, µg/kg/day (high-end / typical consumption)
Children birth up to 1 year old	<b>0.2 / 0.1</b>	<b>0.02 / 0.008</b>	<b>0.03 / 0.02</b>	<b>0.01 / 0.005</b>	0.02 / 0.009	0.02 / 0.009
Children 1 year old up to age 2	<b>0.1 / 0.04</b>	<b>0.009 / 0.003</b>	0.02 / 0.006	<b>0.007 / 0.002</b>	0.011 / 0.004	0.011 / 0.004
Children 2 years old up to age 6	<b>0.09 / 0.04</b>	<b>0.007 / 0.003</b>	0.01 / 0.005	<b>0.005 / 0.002</b>	0.008 / 0.003	0.008 / 0.003
Children 6 years old up to age 11	<b>0.07 / 0.03</b>	<b>0.005 / 0.002</b>	0.01 / 0.004	<b>0.004 / 0.001</b>	0.006 / 0.002	0.006 / 0.002
Children 11 years old up to age 16	<b>0.06 / 0.02</b>	<b>0.004 / 0.001</b>	0.008 / 0.003	0.003 / 0.001	0.005 / 0.002	0.005 / 0.002
Children 16 years old up to age 21	<b>0.06 / 0.02</b>	<b>0.004 / 0.001</b>	0.008 / 0.003	0.003 / 0.0009	0.005 / 0.002	0.005 / 0.002
Adults 21 years old or more	<b>0.06 / 0.03</b>	<b>0.005 / 0.002</b>	0.009 / 0.004	<b>0.003 / 0.001</b>	0.005 / 0.002	0.005 / 0.002
Pregnant women	<b>0.06 / 0.02</b>	<b>0.004 / 0.001</b>	0.009 / 0.003	<b>0.003 / 0.001</b>	0.005 / 0.002	0.005 / 0.002
Lactating women	<b>0.08 / 0.04</b>	<b>0.006 / 0.003</b>	0.01 / 0.005	<b>0.004 / 0.002</b>	0.007 / 0.003	0.007 / 0.003
<i>Lowest health guideline in µg/kg/day</i>	<i>0.003</i>	<i>0.002</i>	<i>0.02</i>	<i>0.003</i>	<i>2.9</i>	<i>0.23</i>

µg/L = micrograms per liter      µg/kg/day = micrograms per kilogram per day      See Appendix A for full compound names and chemical information.  
 Health guideline is the intermediate MRL for PFOA, PFOS, PFHxS, and PFNS; it is the lowest state reference dose for PFBA and PFBS.  
 Doses are rounded to one significant figure. **Doses greater than or equal to the corresponding MRL are shown in bold** (due to rounding, some unbold values appear equal to the MRL).

We calculated a dose for each PFAS measured, for each separate private well included in the dataset. Because of the large number of private wells (almost 2,750) and to protect personally identifying information, these calculations and individual results are not presented in this report.

The individual PFAS exposure doses estimated using the above process were evaluated by comparing them with effect levels observed in animal toxicological studies on the corresponding PFAS. This evaluation is detailed in the body of the report. In addition, because many wells contained detections of more than one PFAS, we conducted additional evaluation of the potential for mixture effects.

### Evaluating PFAS mixtures

Many wells contained detections of multiple PFAS. For mixtures, ATSDR recommends a tiered approach to determine whether further evaluation of mixture effects is necessary [40]. The three tiers as applied in this site-specific evaluation are described below.

#### *Determine which wells could exhibit mixture effects (mixtures framework Tier 1)*

In Tier 1, a *hazard quotient* is defined for each contaminant as the estimated dose divided by a noncancer or cancer-based health guideline. For the PFAS assessed in this report, only noncancer health guidelines are available.<sup>15</sup> For each PFAS “*i*”, the hazard quotient is given by the following equation:

$$\text{Hazard Quotient}_{\text{PFAS } i} = \text{Estimated dose}_{\text{PFAS } i} (\mu\text{g/kg/day}) \div \text{Health guideline}_{\text{PFAS } i} (\mu\text{g/kg/day}),$$

where the health guideline is the contaminant-specific minimal risk level or reference dose.

Mixtures containing more than one component with a hazard quotient greater than 0.1 are carried forward for Tier 2 analysis.

Table B3 summarizes the Tier 1 analysis for the private wells near the Saint-Gobain Merrimack facility. For each PFAS, the table lists the highest estimated dose (the dose to children from birth to one year old drinking high-end amounts of water every day from the private well with the highest concentration of the contaminant measured), health guideline, highest estimated hazard quotient, number of wells with a hazard quotient greater than 0.1 for that PFAS, number of those wells that had a second PFAS component with a hazard quotient greater than 0.1, and whether the PFAS should be included in additional, Tier 2 analysis. Due to a lack of health guideline values, we could not calculate hazard quotients for all PFAS.

For the private wells evaluated in this report, PFOA, PFOS, PFHxS, and PFNA all had hazard quotients greater than 0.1 in some wells. PFBA and PFBS had hazard quotients lower than 0.1 in all wells and are not carried forward to Tier 2. This Tier 1 analysis identified four PFAS in 284 private wells to be included in the Tier 2 analysis.<sup>16</sup>

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<sup>15</sup> Intermediate MRLs based on noncancer effects are available for PFOA, PFOS, PFHxS, and PFNA. State reference doses based on noncancer effects are available for PFBA and PFBS. No official cancer slope factor for PFOA or any other PFAS exists at the time of this report. Potential cancer effects for PFOA are discussed later in this report based on a provisional oral cancer slope factor available now.

<sup>16</sup> Of the 1,099 wells not included in further mixtures analysis, we note that 484 of them included detections of other PFAS for which no health guidelines exist. ATSDR cannot evaluate the potential mixture effects of these other PFAS.

**Table B 3. Tier 1 mixtures analysis summary for private wells near the Saint-Gobain Merrimack, New Hampshire facility**

PFAS	Highest estimated dose in µg/kg/day*	PFAS-specific health guideline in µg/kg/day**	Health guideline source	Corresponding highest hazard quotient (HQ)	# of wells with HQ ≥0.1‡	# of those wells at least one other PFAS with HQ ≥0.1‡	Include PFAS in Tier 2 mixtures evaluation?
PFOA	0.23	0.003	ATSDR MRL	76	2,362	1,101	Yes
PFOS	0.017	0.002	ATSDR MRL	9	1,097	1,088	Yes
PFHxS	0.034	0.02	ATSDR MRL	2	34	33	Yes
PFNA	0.012	0.003	ATSDR MRL	4	24	24	Yes
PFBA	0.02	2.9	Minnesota chronic RfD	0.007	0	Not applicable	No
PFBS	0.02	0.23	Michigan RfD	0.09	0	Not applicable	No

MRL = intermediate minimal risk level

RfD = reference dose

\*Highest dose represents a small child with high-end water consumption drinking water with the highest concentration of each PFAS measured in any well.

\*\*No health guidelines were available for other PFAS listed in Table 1. These substances were not included in any further mixtures analysis.

‡Numbers of wells are not additive, since some wells contained multiple PFAS.

*Determine hazard index for wells with mixtures (Mixtures framework Tier 2)*

For the PFAS and wells carried forward to Tier 2, the next step is to calculate a *hazard index* for each well’s PFAS mixture and preliminarily evaluate the potential for noncancer effects from the mixture.

The hazard index, which assumes dose additivity, is the sum of the respective hazard quotients for the well, given in this case as:

$$\text{Hazard Index} = \text{Hazard quotient (HQ)}_{\text{PFOA}} + \text{HQ}_{\text{PFOS}} + \text{HQ}_{\text{PFHxS}} + \text{HQ}_{\text{PFNA}}$$

where the subscripts indicate which PFAS the hazard quotient is calculated for. Mixtures with a hazard index greater than 1 are carried forward to Tier 3 analysis.

Figure B1 illustrates Tier 1 and Tier 2 mixtures analysis using selected de-identified private well results from this site. ATSDR evaluated 284 of the private wells using hazard indices described in Tier 2. Of these well, 206 had a hazard index greater than 1 and were included in Tier 3’s further evaluation.

Figure B 1. Selected data from private wells from New Hampshire database, illustrating Tier 1 and Tier 2 mixtures evaluation

Well Index #	Concentration, microgram per L (ug/L)				Dose for a 0-1 year old, 95%ile consumption, ug/kg/day					Hazard Quotient (based on 0-1 year old with high-end consumption)					Hazard Index	
	PFOA	PFOS	PFHXS	PFNA	Tier 1				Combined Dose	PFOA	PFOS	PFHXS	PFNA	+to be evaluated for mixture effects	Hazard Index - Sum of HQs for PFOA, PFOS, PFHXS, PFNA	
					PFOA	PFOS	PFHXS	PFNA								
<b>WELLS ANALYZED FOR MIXTURES EFFECTS (MORE THAN ONE HQ GREATER THAN OR EQUAL TO 0.1)</b>																
1	1.6	0	0.021	0	2E-01	0E+00	3E-03	0E+00	2.E-01		76.10	0.00	0.15	0.00	+	76.3
18	0.29	0	0.092	0	4E-02	0E+00	1E-02	0E+00	5.E-02		13.79	0.00	0.66	0.00	+	14.4
20	0.27	0.076	0.072	0	4E-02	1E-02	1E-02	0E+00	6.E-02		12.84	5.42	0.51	0.00	+	18.8
66	0.13	0.0034	0.0088	0	2E-02	5E-04	1E-03	0E+00	2.E-02		6.18	0.24	0.06	0.00	+	6.5
67	0.13	0	0.016	0	2E-02	0E+00	2E-03	0E+00	2.E-02		6.18	0.00	0.11	0.00	+	6.3
75	0.124	0.01	0.004	0	2E-02	1E-03	6E-04	0E+00	2.E-02		5.90	0.71	0.03	0.00	+	6.6
126	0.096	0.007	0.004	0.002	1E-02	1E-03	6E-04	3E-04	2.E-02		4.57	0.50	0.03	0.10	+	5.2
131	0.095	0.019	0.0043	0.012	1E-02	3E-03	6E-04	2E-03	2.E-02		4.52	1.36	0.03	0.57	+	6.5
1009	0.0178	0.0029	0.0014	0	3E-03				1.E-03		0.85	0.21	0.01	0.00	+	1.1
1024	0.017	0.0093	0.0026	0	2E-03				1.E-03		0.81	0.66	0.02	0.00	+	1.5
1062	0.017	0.0016	0.0035	0	2E-03				1.E-03		0.81	0.11	0.02	0.00	+	0.95
1073	0.0169	0.0066	0.004	0	2E-03				1.E-03		0.80	0.47	0.03	0.00	+	1.3
1075	0.0168	0.0024	0	0	2E-03	3E-04	0E+00	0E+00	3.E-03		0.80	0.17	0.00	0.00	+	0.97
1528	0.0108	0.0087	0.0021	0	2E-03	1E-03	3E-04	0E+00	3.E-03		0.51	0.62	0.01	0.00	+	1.2
2016	0.0056	0.0021	0.0016	0	8E-04	3E-04	2E-04	0E+00	1.E-03		0.27	0.15	0.01	0.00	+	0.4
2025	0.0055	0.012	0	0	8E-04	2E-03	0E+00	0E+00	2.E-03		0.26	0.86	0.00	0.00	+	1.1
2347	0.0023	0.0029	0.0025	0	3E-04	4E-04	4E-04	0E+00	1.E-03		0.11	0.21	0.02	0.00	+	0.3
<b>WELLS NOT ANALYZED FOR MIXTURES EFFECTS (ONLY ONE HQ GREATER THAN OR EQUAL TO 0.1)</b>																
2	1.5	0	0.0072	0	2E-01	0E+00	1E-03	0E+00	2.E-01		71.35	0.00	0.05	0.00		
184	0.075	0	0	0	1E-02	0E+00	0E+00	0E+00	1.E-02		3.57	0.00	0.00	0.00		
223	0.064	0	0	0	9E-03	0E					3.04	0.00	0.00	0.00		
228	0.062	0	0	0	9E-03	0E					2.95	0.00	0.00	0.00		
2677	0	0.0061	0	0	0E+00	9E					0.00	0.44	0.00	0.00		
2613	0	0.0042	0.0027	0	0E+00	6E					0.00	0.30	0.02	0.00		
2649	0	0	0.0257	0	0E+00	0E+00	4E-03	0E+00	4.E-03		0.00	0.00	0.18	0.00		
2499	0	0	0.0003	0	0E+00	0E+00	5E-05	0E+00	5.E-05		0.00	0.00	0.00	0.00		
2500	0	0	0	0	0E+00	0E+00	0E+00	0E+00	0.E+00		0.00	0.00	0.00	0.00		

**Tier 1**

**Tier 2**

More than one HQ greater than or equal to 0.1 --> do Tier 2 mixture analysis

Hazard index greater than or equal to 1 --> do Tier 3 mixture analysis

Only one or fewer HQ greater than or equal to 0.1 --> mixture effects unlikely

*Refined evaluation of potential effects considering target organs and other factors (Mixtures framework Tier 3)*

Tier 3 analysis is a detailed analysis of potential mixture effects, considering, for example, target toxicities of each mixture component, sensitive subpopulations, or more refined estimates of potential exposure to the mixture. The text of this report describes that the PFAS in these mixtures may target similar organ systems and may all potentially contribute to development, immune, or liver effects. For further evaluation, ATSDR used the combined dose of all four PFAS included in the mixtures evaluation to determine the potential for harmful health effects. Because PFOA is the main contaminant at this site, we relied primarily on toxicological information for PFOA to determine whether effects from the mixtures were likely.

**Evaluating cancer health effects**

In general, the estimated added lifetime risk of developing cancer from an oral exposure to a carcinogenic contaminant is calculated by multiplying the site-specific estimated exposure dose, averaged over a lifetime, by an appropriate cancer slope factor. ATSDR uses this quantitative risk estimate as part of a weight-of-evidence approach to decide whether exposures to cancer-causing contaminants are of concern. ATSDR describes estimated increased cancer risk qualitatively and in terms of background rates of cancer occurring in the U.S. population.

At this time, there are no appropriate cancer slope factors for any PFAS to allow a quantitative estimate of increased cancer risk from exposure to PFAS. ATSDR has discussed cancer risk associated with PFAS qualitatively in the body of the report, beginning on page 24.