# Public Health Assessment

**Final Release**

**PERFLUOROCHEMICAL CONTAMINATION IN SOUTHERN WASHINGTON COUNTY, NORTHERN DAKOTA COUNTY, AND SOUTHEASTERN RAMSEY COUNTY, MINNESOTA**

**(Including: Woodbury, Cottage Grove, Newport, St. Paul Park, Afton, Grey Cloud Island Township, Denmark Township, South St. Paul, Hastings and Maplewood)**

EPA FACILITY ID: MN980679385

**Prepared by the**
**Minnesota Department of Health**

JANUARY 5, 2012

Prepared under a Cooperative Agreement with the
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation
Atlanta, Georgia 30333

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

This Public Health Assessment was prepared by ATSDR's Cooperative Agreement Partner pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) section 104 (i)(6) (42 U.S.C. 9604 (i)(6)), and in accordance with our implementing regulations (42 C.F.R. Part 90). In preparing this document, ATSDR's Cooperative Agreement Partner has collected relevant health data, environmental data, and community health concerns from the Environmental Protection Agency (EPA), state and local health and environmental agencies, the community, and potentially responsible parties, where appropriate.

In addition, this document has previously been provided to EPA and the affected states in an initial release, as required by CERCLA section 104 (i)(6)(H) for their information and review. The revised document was released for a 30-day public comment period. Subsequent to the public comment period, ATSDR's Cooperative Agreement Partner addressed all public comments and revised or appended the document as appropriate. The public health assessment has now been reissued. This concludes the public health assessment process for this site, unless additional information is obtained by ATSDR's Cooperative Agreement Partner which, in the agency's opinion, indicates a need to revise or append the conclusions previously issued.



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# PUBLIC HEALTH ASSESSMENT

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## FOREWORD

This document summarizes public health concerns related to a waste disposal site in Minnesota, and is a formal site evaluation prepared by the Minnesota Department of Health (MDH). For a formal site evaluation, a number of steps are necessary:

- *Evaluating exposure:* MDH scientists begin by reviewing available information about environmental conditions at the site. The first task is to find out how much contamination is present, where it is found on the site, and how people might be exposed to it. Usually, MDH does not collect its own environmental sampling data (although this case is an exception). Rather, MDH relies on information provided by the Minnesota Pollution Control Agency (MPCA), the Minnesota Department of Agriculture (MDA), the US Environmental Protection Agency (EPA), private businesses, and the general public.

- *Evaluating health effects:* If there is evidence that people are being exposed—or could be exposed—to environmental contaminants, MDH scientists will take steps to determine whether that exposure could be harmful to human health. MDH's report focuses on public health— that is, the health impact on the community as a whole. The report is based on existing scientific information.

- *Developing recommendations:* In this report, MDH outlines conclusions regarding any potential health threat posed by a site and offers recommendations for reducing or eliminating human exposure to pollutants. The role of MDH is primarily advisory. For that reason, the evaluation report will typically recommend actions to be taken by other agencies—including EPA and MPCA. If, however, an immediate health threat exists, MDH will issue a public health advisory to warn people of the danger and will work to resolve the problem.

- *Soliciting community input*: The evaluation process is interactive. MDH starts by soliciting and evaluating information from various government agencies, the individuals or organizations responsible for the site, and community members living near the site. Any conclusions about the site are shared with the individuals, groups, and organizations that provided the information. Once an evaluation report has been prepared, MDH seeks feedback from the public. *If you have questions or comments about this report, we encourage you to contact us.*


*Please write to:* Community Relations Coordinator<br>
Site Assessment and Consultation Unit<br>
Minnesota Department of Health<br>
625 North Robert Street / P.O. Box 64975<br>
St. Paul, MN 55164-0975

*OR call us at:* (651) 201-4897 *or* 1-800-657-3908<br>
(toll free call - press "4" on your touch tone phone)

*On the web:* http://www.health.state.mn.us/divs/eh/hazardous/index.html

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## Summary

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| **INTRODUCTION** | The Minnesota Department of Health's (MDH) mission is to protect, maintain, and improve the health of all Minnesotans.<br><br>For communities affected by perfluorochemicals (PFCs) in their drinking water, MDH's goal is to protect people's health by providing health information the community needs to take actions to protect their health. MDH also monitors public water supplies for PFCs, and advises the MPCA on actions that can be taken to protect public health.<br><br>PFC-containing wastes were disposed of by 3M Corporation (3M) in land disposal sites in Oakdale, Lake Elmo, and Woodbury, Minnesota and at their manufacturing facility in Cottage Grove, Minnesota. This report focuses on the Woodbury disposal site and surrounding affected communities south of Interstate 94, where PFCs from all four sites have contributed to groundwater contamination.<br><br>In the past, people may have been exposed to air emissions during the handling, disposal, or burning of waste at the disposal sites. People also may have come into direct contact with the waste or contaminated soils if they entered the sites. However, these exposure pathways appear to have been addressed by site cleanup activities.<br><br>PFCs were released to the groundwater from the disposal sites, resulting in contamination of nearby public and private drinking water wells. PFCs continue to be detected in public and private wells across a wide area of south Washington County, and in parts of northern Dakota County and southeastern Ramsey County. Twenty-four private wells in the area covered by this report have PFC concentrations that exceed MDH health-based drinking water exposure limits and those homeowners have been provided treatment or bottled water to reduce exposure. PFCs in all other wells, public and private, are below MDH exposure limits. |
| **OVERVIEW** | MDH reached three important conclusions in this Public Health Assessment. |
| **CONCLUSION 1** | MDH cannot conclude whether drinking or breathing PFCs in water or air or contact with PFC-containing wastes in the past harmed people's health. |
| **BASIS FOR DECISION** | No information is available regarding past levels of PFCs in the water or air of the affected communities. Similarly, no information is available regarding what, if any, PFC waste people may have encountered if they entered the 3M-Woodbury Disposal Site in the past. As a result, it is not possible to determine the level of PFCs to which people may have been exposed in the past. Biomonitoring studies of residents in Oakdale, Lake Elmo, and Cottage Grove indicated that levels of PFCs in their blood are above national averages, but have fallen due to the |

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|---|---|
| | provision of treated drinking water to residents previously exposed to PFCs above Minnesota standards. While available evidence suggests that the measured PFC levels are unlikely to cause adverse health effects, there is no information available regarding past PFC levels in resident's blood and whether those past levels would have resulted in adverse health effects. |
| **NEXT STEPS** | Although nothing can be done to alter past exposures, MDH will continue to provide health information regarding PFCs to the affected communities, as it becomes available. |
| **CONCLUSION 2** | MDH concludes that currently, drinking water from public or private wells that contain PFCs is not expected to harm people's health. |
| **BASIS FOR DECISION** | Current exposures to PFCs are below health-based exposure limits because bottled water or whole-house activated carbon filters have been provided at 24 homes that were issued a drinking water well advisory by MDH, although such filters are not considered by MDH to be the best, long-term solution if other sources of water are available. No one currently is drinking water that has PFCs at levels above MDH health-based exposure limits.<br><br>Remediation actions to address PFCs at the waste disposal sites are currently being implemented by 3M and the MPCA. |
| **NEXT STEPS** | • 3M should continue to follow the requirements of the Consent Order to implement the selected remediation options for soil and groundwater at the 3M-Woodbury Disposal Site.<br>• Although site cleanup actions have reduced or eliminated PFC contamination in surface soils at the 3M-Woodbury Disposal Site, people should avoid trespassing on the property.<br>• 3M should improve and maintain the existing fencing of the site, particularly along the south boundary, to prevent trespassing.<br>• Extensions of the Cottage Grove municipal water supply to serve areas where private wells contain levels of PFCs in excess of MDH HRLs should be considered.<br>• Monitoring of selected private wells in the affected area should continue, under MDH and MPCA approved sampling plans, as needed to track changes in the plume and monitor for changes in concentration in individual wells.<br>• MPCA should ensure that there is an adequate monitoring well network at the disposal site to provide sufficient information regarding water quality in the "high transmissivity zone" of the Prairie du Chien aquifer.<br>• |
| **CONCLUSION 3** | Treatment of city and private wells that exceed MDH HRLs or cumulative drinking water guidelines has reduced or eliminated PFC exposure for the users of those wells and resulted in decreased average concentrations of PFCs in the blood of those users. |

**BASIS FOR CONCLUSION**

Monitoring of treated water at the City of Oakdale and in private residences where GAC filter systems were installed demonstrate removal of PFCs to below laboratory reporting limits. Biomonitoring of PFCs in the blood of Oakdale, Lake Elmo, and Cottage Grove residents previously exposed to PFCs through their drinking water, but now drinking treated city or private well water, demonstrated significant reductions in blood levels of PFCs between 2008 and 2010.

**NEXT STEPS**

- The MPCA will continue to monitor and maintain whole-house granular activated carbon (GAC) filter systems in homes where the well water exceeds MDH HRLs or cumulative drinking water guidelines.
- MDH will continue to work with the City of Oakdale and 3M to ensure proper treatment of city wells that exceed MDH HRLs.
- MDH will consult with its Scientific Advisory Board regarding possible future biomonitoring in the affected communities, if funding is available.

**FOR MORE INFORMATION**

If you have concerns about your health, you should contact your health care provider. You may also call MDH at 651-201-4897 or 1-800-657-3908 (press #4) and ask for information on PFCs. You may also visit our PFC Web site at http://www.health.state.mn.us/divs/eh/hazardous/topics/pfcs/index.html

## Introduction

The 3M Company (3M; formerly Minnesota Mining and Manufacturing Company) began research and development of perfluorochemicals (PFCs) at its Cottage Grove, Minnesota facility in southern Washington County, Minnesota in the late 1940s. Commercial production of eight-carbon PFC compounds, including perfluorooctanoic acid (PFOA) and precursors for perfluoro-octane sulfonate (PFOS) and PFOA, occurred from the early 1950s until 2002. Production of the four-carbon compound, perfluorobutanoic acid (PFBA) ceased in 1998. 3M currently produces perfluorobutane sulfonate (PFBS)-based products which are substitutes for the earlier eight-carbon PFCs. PFBS is also a four-carbon compound. In addition, 3M continues to use and/or produce one- to three-carbon perfluoroalkyl substances at the Cottage Grove facility (3M, 2010a).

MDH prepared a Health Consultation focusing on PFC releases at the Cottage Grove facility (MDH, 2005). Until the 1970's, wastes from the facility, including electrofluorochemical PFC production process wastes such as production wastes and wastewater treatment plant sludge, were disposed of at the Cottage Grove facility and several known disposal sites identified by 3M in Washington County (Weston, 2005). In the early 1970's, 3M built an on-site incinerator where wastes were processed; since the mid-1970's the incinerator ash and scrubber sludge have been disposed off-site in an industrial waste landfill. The types of wastes disposed of at these sites and the estimated time of the disposal are listed below:

**Disposal Facilities in Washington County that Received 3M Wastes**

| **Disposal Facility** | **Waste Disposed** | **Estimated Dates** |
|---|---|---|
| 3M-Oakdale Disposal Site | Liquid and solid industrial waste | 1956 – 1960 |
| 3M-Woodbury Disposal Site | Liquid and solid industrial waste | 1960 – 1966 |
| Washington County Landfill, Lake Elmo | Wastewater treatment plant sludge, incinerator scrubber sludge and ash, iron oxide sludge | 1971 – 1974 |
| 3M-Cottage Grove Facility | Industrial wastes, ash, sludge | 1950s – 1970s |

The general locations of the above disposal sites, along with the 3M Cottage Grove facility are shown in Figure 1 (all figures can be found in Appendix 1). PFCs disposed of at the sites identified in the table above have impacted soil, groundwater, surface water, sediments, biota, and nearby drinking water wells, both public and private, in large areas of the affected communities covered by this report.

Figure 1 also includes the Pigs Eye Dump site in St. Paul. 3M reported sending incinerator ash to this site in 1971, although the more likely source for the PFCs are the municipal incinerator ash and solid waste disposed of at the dump. It is not included in the Consent Order agreement between 3M and the MPCA (see below), but is included in the figure because high levels of PFCs have been detected in groundwater and surface water at the site and it is located within the area covered by this report. However, PFCs at that site have not affected nearby drinking water wells.

Although the 3M-Woodbury Disposal Site is not on the National Priorities List (NPL), MDH has prepared this report in response to requests from the MPCA, Washington County, local communities, and citizens to:

- summarize current conditions in southern Washington County, northern Dakota County, and southeastern Ramsey County relative to the PFC contamination;
- evaluate the potential health risks associated with the use of drinking water impacted by PFCs, especially in public water supplies;
- provide the public with an opportunity to comment on the public health actions taken to date; and
- provide recommendations to protect public health in the future.

MDH has consulted with staff from the U.S. Environmental Protection Agency (EPA), MPCA, Washington County, the cities of Woodbury, Cottage Grove, Hastings, St. Paul Park, and other local governments in the affected area, community members, and 3M to gather information for this report.

MDH prepared a separate report focusing on the PFC releases at the 3M-Oakdale Disposal Site and Washington County Landfill and the affected surrounding communities north of Interstate 94 (I-94). Investigations in those communities revealed that PFBA from the two sites "co-mingled" in places to create a large plume of groundwater contamination that extended to, and likely beyond, I-94 (MDH, 2008a). This report evaluates PFC contamination in the affected communities south of I-94; while it focuses mainly on the 3M-Woodbury Disposal Site, this report also evaluates the overall PFC impacts to these communities, including those from the PFC plumes emerging from the Oakdale and Lake Elmo disposal sites and migrating south of I-94.

Perfluorochemicals, broadly speaking, are a class of organic chemicals in which fluorine atoms completely replace the hydrogen atoms that are typically attached to the carbon 'backbone' of organic hydrocarbon molecules. The PFCs that are the subject of this report are characterized by such a perfluorinated carbon chain with a functional end group (Figure 2). Because of the very high strength of the carbon-fluorine bond, PFCs are inherently stable, nonreactive, and resistant to degradation (3M, 1999a). PFCs made by 3M at its Cottage Grove facility were used in the manufacture of a variety of commercial and industrial products by 3M and other companies, including fabric coatings (such as Scotchgard™), surfactants, non-stick products (including Teflon™), fire-fighting foams, film coatings, and other products.

The unique physical and chemical properties of many PFCs allow them to move easily through the environment (EPA, 2002; OECD, 2002; ATSDR, 2009). As a result, they have been found globally at low levels. Some PFCs are bio-accumulative (i.e., build up in living organisms) and one PFC, perfluorooctane sulfonate (PFOS) has been detected in the blood and tissues of humans and animals from virtually all parts of the world. It should be noted that while the use of PFCs has been restricted in the United States by the

EPA to certain products for which there is no adequate substitute, they are still manufactured and used in other countries around the world, including Italy, Russia, China, Japan, and Korea.

Toxicological research on PFCs is ongoing in government, industry and academia. Published studies show that animal exposure to PFCs at high concentrations adversely affects the liver and other organs (ATSDR, 2009). The mechanisms of toxicity are not entirely clear; one likely major mechanism involves effects on certain enzymes regulating metabolic pathways in the liver. Exposure to high concentrations of one PFC, perfluorooctanoic acid (PFOA) over long durations has been shown to cause tumors in some test animals, although the specific mechanisms are not clear and the relevance to humans may be low. Developmental effects have also been observed in the offspring of pregnant rats and mice exposed to high doses of PFOA and PFOS.

## Background

3M-Woodbury Disposal Site Description and History

The 656-acre 3M property that contains the 40-acre 3M-Woodbury Disposal Site straddles the border of Woodbury and Cottage Grove (see Figure 3). The actual waste disposal areas are located in Woodbury and consist of two areas referred to as the Main Disposal Area (approximately ten acres) and the Northeast Disposal Area (approximately five acres; Weston, 2007a).

Prior to 1960, industrial waste generated at the 3M manufacturing plants located in Cottage Grove and downtown St. Paul were hauled and disposed of by a private waste hauler at a property located in Oakdale (now referred to as the 3M-Oakdale Disposal Site). Following the owner’s death in 1959, 3M contracted with St. Paul Terminal Warehouse, who continued to haul and dispose of material on the 3M-Oakdale Disposal site property.

In 1960, St. Paul Terminal Warehouse purchase 240 acres of farmland in Woodbury for use as a waste disposal site. In 1960, 3M signed a contract with St. Paul Terminal Warehouse for disposal of wastes at the new Woodbury site. In 1961, 3M purchased the land and continued to use the site to dispose of liquid and solid industrial wastes (solvents, tapes, plastics, and resins) generated at their Cottage Grove and downtown St. Paul facilities.

3M is believed to be the only company to have used the Woodbury site for industrial waste disposal. The wastes were buried in clay-lined trenches. In addition, municipal wastes from the cities of Woodbury and Cottage Grove were disposed of in two separate areas of the site (approximately five acres total) from 1964-1966 (Weston, 2008a).

Groundwater contamination at the site was first detected in 1966 when volatile organic compounds (VOCs, mostly solvents) were found in groundwater monitoring wells on the site and a private well located immediately west of the site. The first wells for a groundwater extraction (barrier well) system were installed by 3M in 1967 and by 1973 the system consisted of four wells which have operated continuously since. The extracted groundwater is pumped via a pipeline to the 3M Cottage Grove manufacturing plant, where it is used as cooling or process water and then discharged to the Mississippi River under an NPDES permit issued by the MPCA.

Additional cleanup measures were taken at the site to consolidate and burn wastes, with the goal of reducing sources of VOC contamination to the groundwater. Approximately 200,000 cubic yards of wastes were excavated from the Main Disposal Area and burned on-site in February of 1968. The remaining ash and waste were consolidated and re-buried in the Main Disposal Area trenches.

In 1992, 3M entered the site into the MPCA's Voluntary Investigation and Cleanup (VIC) program, under which additional investigation and response actions were taken to further address contamination at the site. A 1994 report of that work noted that fluorochemical wastes had been disposed of and were present in soil and soil gases at the site, but analytical methods were not sensitive enough at that time to detect PFCs in the groundwater (3M, 1994). In 1996, 3M backfilled open areas and regraded the site, placed a cap consisting of a minimum of 24 inches of clean soil over the former disposal areas, and filed an institutional control on the property deed to restrict future land use at the property.

<u>Geology / Hydrogeology</u>

The geology of the region where the 3M-Woodbury Disposal Site is located consists of glacial drift and alluvial sediments (stratified sand, silt, and clay deposited by glaciers and rivers, respectively) overlying a thick sequence of Paleozoic sedimentary rock formations made up of sandstone, limestone, dolomite, and shale. These, in turn, overlay pre-Cambrian volcanic rock formations composed primarily of basalt. The bedrock formations tilt and thicken slightly to the south and west, forming the eastern rim of a large geologic structure known as the Twin Cities Basin. Figure 4 shows the sequence of bedrock units in south Washington County. The geology of this area has been extensively studied by the Minnesota Geological Survey and others (Tipping et al., 2006; Runkel et al., 2003; MGS, 1990).

Before the glacial drift and alluvial sediments were deposited, streams eroded deep valleys into the surface of the bedrock. In some places the valleys cut down to the Jordan Sandstone. The valleys were later filled with glacial and alluvial sediments, leaving little or no evidence at the surface of their presence below, except for a series of elongated lakes in Lake Elmo and a deep ravine in southern Cottage Grove. Figure 1 shows the location of a major bedrock valley that extends from Lake Elmo south to the Mississippi River valley. The bedrock valleys in south Washington County provide pathways that allow contaminants in the groundwater to enter deeper aquifers more rapidly than would be the case if the bedrock layers were intact.

The bedrock valley shown in Figure 1 is present beneath the west side of the site. As the valley was eroded, successively deeper bedrock formations were exposed from east to west. As a result, the uppermost bedrock layer in the northeastern portion of the site is the Platteville Limestone, while in the center of the site it is the St. Peter Sandstone, and on the west side it is the Prairie du Chien Group dolomite and Jordan Sandstone (see Figure 5).

The bedrock in southern Washington County also has been altered by major faults (fractures in the rock along which movement has occurred, see Figure 1). In some places, bedrock units on either side of such faults have been displaced vertically as much as 150 feet. This means that in some parts of the investigation area, one geologic formation may be in direct contact with another (see Figure 4). This may allow groundwater and contaminants to move between aquifers that might not otherwise be connected. It also means that two wells of similar depth and separated by a distance of only a few hundred feet may draw water from completely different formations.

Regional groundwater flow in the area of the site is further complicated by the presence of two major regional groundwater discharge features, the Mississippi and St. Croix Rivers. In general, groundwater in the east half of Washington County flows toward and discharges to the St. Croix River, while groundwater in the west half of the county flows toward and discharges to the Mississippi River. The zone where this divergence of the groundwater flow occurs is often referred to as a groundwater divide (Figure 1). While similar in concept to the better known "continental divide" (that separates rivers that flow east to the Atlantic Ocean from those that flow west to the Pacific Ocean), a groundwater divide is less fixed and may shift its location as a result in changes in climate and pumping of groundwater. As a result, the location of the actual groundwater divide is approximate, will change over time, and may be slightly different in each aquifer.

In southern Washington County, the groundwater divide is located somewhere near or under the east side of the 3M-Woodbury Disposal Site (Kanivetsky and Cleland, 1990). This means that groundwater contaminants beneath eastern portions of the site could move east-southeast toward the St. Croix River while those beneath western portions of the site would more likely move west-southwest toward the Mississippi River. Additionally, in Denmark Township and southeast Cottage Grove, near where the two rivers converge, the regional groundwater flow direction "fans out." The result is that contaminants released to the groundwater in this area could potentially affect a larger area than is typically seen at most sites.

The type of geologic units beneath the disposal site also affects how groundwater and contaminants move. The sand and gravel deposits in the buried bedrock valley, and the Platteville, St. Peter and Prairie du Chien formations beneath the site are highly permeable, allowing groundwater to easily move downward through pore spaces between sand grains and along fractures.

There are four major drinking water aquifers in the investigation area, that are from shallowest to deepest: St. Peter Sandstone, Prairie du Chien Group, Jordan Sandstone, and Franconia Sandstone (Figure 4). State well records also indicate there are some wells using the overlying sand and gravel deposits, but this appears to be rare. All of the municipal water supply wells in the affected communities draw water from the Jordan Sandstone.

Groundwater in the St. Peter migrates primarily through the pore spaces between the sand grains, although fractures and solution cavities are present in the St. Peter, particularly near the buried bedrock valleys (Alexander, 2007; Runkel et al., 2007). Such solution cavities may create pathways through which groundwater and contaminants migrate more quickly than is typically observed in the St. Peter.

Groundwater flow in the Prairie du Chien dolomite is heavily influenced by fractures (cracks and voids) in the formation. The Prairie du Chien is actually considered a “group” composed of two separate dolomite formations referred to as the Shakopee and the Oneota members of the Prairie du Chien Group. For general purposes, this report will consider the Prairie du Chien Group as a single unit. However, it is useful to note that although the rock itself in the lower Oneota formation tends to be more massive (i.e. denser, with little pore space) than the sandier overlying Shakopee formation, the Oneota tends to have more solution cavities. As a result, the Oneota provides the higher yield of water to wells (Lindholm et al., 1974). Hydraulic conductivity is a measure of how much water can pass through an aquifer, and depends not only on the amount of pore space that water can pass through (the aquifer’s porosity), but how well connected those pore spaces are to one another (the aquifer’s permeability). The hydraulic conductivity of similar fractured bedrock groundwater systems in southeast Minnesota has been shown to sometimes exceed several thousand feet per day (Runkel et al., 2007). Tipping et al. (2006) identified a zone – referred to as the “high transmissivity zone” - near the contact of the Shakopee and Oneota members of the Prairie du Chien which has densely spaced fractures - this create a horizon where groundwater flow rates are very high. In fact, pumping of wells that pull water from that horizon can even result in upward flow of groundwater from the underlying Jordan aquifer (Tipping et al., 2006).

Below the Prairie du Chien is the Jordan Sandstone. Although the lower Oneota formation of the Prairie du Chien group may limit downward migration of groundwater from the Prairie du Chien to the Jordan (Tipping et al., 2006), pumping wells in the Jordan can cause groundwater to move downward from the Prairie du Chien into the Jordan. Preliminary modeling of groundwater flow by MDH suggests that groundwater flow from the Prairie du Chien to the Jordan may be occurring primarily in the areas immediately around municipal wells as a result of the high pumping rates of those wells (A. Djerrari, MDH, personal communication, 2007).

Beneath the Jordan Sandstone is the St. Lawrence formation, composed of dolomite and siltstone. This formation is not considered an aquifer but rather a “confining unit” because it has low vertical permeability, so groundwater generally does not move downward through it. This means that in most areas, the St. Lawrence “protects” the aquifers beneath it from downward migration of contaminants. Below the St. Lawrence formation, in descending order, are the Franconia, Ironton, and Galesville sandstone aquifers (which are often considered to be one single aquifer), the Eau Claire confining unit, and the Mount Simon sandstone aquifer.

Under natural conditions, the top of the water table at the 3M-Woodbury Disposal Site is located approximately 80-120 feet below the ground surface. The large volumes of water (an average of 4.6 million gallons per day) being removed by the groundwater containment system at the site has lowered the water table, especially near the pump-out wells, so that the depth to the top of the water table is now between 80-140 feet below ground.

PFC Analysis

In late 2003, the MDH Public Health Laboratory developed the capability to analyze water samples for two PFCs, perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA). These two PFCs have been the focus of the majority of the scientific research on perfluorochemicals. PFOA and PFOS accumulate in humans and other species (EPA 2002, OECD 2002). PFOS, but not PFOA, has been shown to bioaccumulate in fish. Both have been found to be widespread in the environment. PFOA was produced at the 3M-Cottage Grove plant on a large scale; some PFOS production or use also reportedly occurred, although most of the eight-carbon fluorosulfonate production involved chemical precursors to PFOS (MDH, 2005). Eight-carbon fluorocarbon production at the site was phased out in 2002.

In the spring of 2006, the MDH Public Health Laboratory expanded their PFC method to include a total of seven PFCs. This was done in response to a request from the MPCA in late 2005 following the detection of other PFCs in soil and water samples collected by the MPCA at the former Washington County Sanitary Landfill and analyzed by a laboratory in British Columbia, Canada (Axys Analytical Services). The seven PFCs currently being analyzed in water by MDH are:

- PFBA : Perfluorobutanoic acid
- PFPeA : Perfluoropentanoic acid
- PFHxA : Perfluorohexanoic acid
- PFOA : Perfluorooctanoic acid
- PFBS : Perfluorobutane sulfonate
- PFHxS : Perfluorohexane sulfonate
- PFOS : Perfluorooctane sulfonate

Water samples are collected in clean 250 milliliter polyethylene bottles. Care is taken to avoid the use of products that could contain PFCs during sampling. The analysis is conducted using a combined high-pressure liquid chromatography tandem mass spectrometry (LC/MS/MS) method, using radio-labeled PFOA and PFOS standards. Each sample is spiked in the lab with a known quantity of labeled standard. The recovery rate of the added standard must be within ± 30% of the known labeled standard concentration added to the sample to meet quality control requirements. In September 2007, the MDH Public Health Laboratory (PHL) issued new, lower reporting levels for the seven PFCs of 0.3 parts per billion (ppb), or 300 parts per trillion (ppt) in water (P. Swedenborg, PHL, personal communication, 2007). The “reporting level” is the concentration at or above which the laboratory can consistently detect and quantify PFCs with a high level of certainty. PFCs detected at concentrations between 50 and 300 ppt are reported as estimated, or "J" flagged values, which have slightly less certainty than those at or above the reporting level. The MDH PHL has the capability to detect and quantify PFC concentrations at levels below 50 ppt, but with even less certainty in the quantification.

Evaluation of PFCs in Drinking Water

MDH has established Health Risk Limits (HRLs) in Minnesota Rules of 0.3 ppb for both PFOS and PFOA. The HRL represents the level of a contaminant in groundwater that MDH considers safe for daily human consumption over a lifetime. The HRLs are protective for adults, children, and developing fetuses, and in the case of PFCs are based on non-cancer effects. In February 2008, MDH established a Health Based Value (HBV) for PFBA of 7 ppb, based, in part, on toxicological and pharmacokinetic studies completed in late 2007 by EPA and 3M. A HBV is a criterion that is established using the same risk assessment procedures and policies used for HRLs, but that has not yet been promulgated through rulemaking. MDH develops HBVs when Minnesota agencies need guidance for chemicals that do not have HRLs. MDH may also calculate HBVs to update an existing value if there is significant new scientific information for the chemical and/or to apply new risk assessment methods. HBVs may be used by the public, state and local risk managers, and other stakeholders to assist in evaluating potential health risks to humans from exposures to a chemical. In general, MDH anticipates that HBVs for water will become HRLs at the time that MDH next amends the Health Risk Limits for Groundwater rule. Information on MDH HRLs and HBVs, including the specific methodology, exposure assumptions, and references, is available at http://www.health.state.mn.us/divs/eh/risk/guidance/gw/index.html.

Because specific information on PFBA toxicity needed to develop a HBV was lacking, MDH could not establish a HBV for PFBA before February 2008 (see http://www.health.state.mn.us/divs/eh/risk/guidance/gw/pfba.pdf). Therefore, as a cautious public health approach, prior to the issuance of the HBV for PFBA MDH used a level of 1 ppb as a "point of departure" for offering advice to private well owners about reducing exposure to PFBA. In other words, MDH was confident that exposure to PFBA in drinking water at levels below 1 ppb was unlikely to be of health concern. Because MDH could not quantify the potential health risk at levels above 1 ppb, advice was provided to those private well owners (or community water supply customers) on how to reduce their exposure if they chose.

The available scientific information for the four remaining PFCs that MDH currently analyzes for is more limited than the information available for PFOS, PFOA, or PFBA. Based on their chemical characteristics, it is anticipated that research will show that PFPeA and PFHxA are generally less toxic than PFOA and, like PFBA, have a short half-life. PFBS and PFHxS have been studied more extensively. PFHxS in particular is known to have a long half-life in humans (see below). MDH has reviewed the available toxicological information on PFBS and PFHxS, and recently established an HBV for PFBS of 7 ppb (see http://www.health.state.mn.us/divs/eh/risk/guidance/gw/pfbs.pdf). MDH staff determined that there was insufficient information to establish an HBV for PFHxS.

HRLs and HBVs are used by MDH to determine if a drinking water well advisory is warranted for an individual well. The MPCA uses MDH advisories to take actions to protect public health from long-term exposure to PFCs, such as providing bottled water or individual water treatment. In cases where a combination of PFOS, PFOA, and PFBA (or other chemicals with similar toxicological endpoints) are present, but do not exceed their individual HRLs or HBV, MDH calculates a Hazard Index to account for possible effects of exposure to more than one PFC at a time. The Hazard Index is the sum of the ratios of the concentrations of PFOS and PFOA over their individual HRLs, and PFBA over its HBV. If the Hazard Index exceeds a value of one, a drinking water well advisory is issued.

Municipal and Non-municipal Community Well Monitoring[1]

In late 2004, after releases of PFCs were documented at the 3M-Cottage Grove facility (later described in MDH 2005), 3M collected samples from municipal wells in Cottage Grove and Hastings for analysis for PFOS and PFOA. The samples, collected under the supervision of MDH and city staff, were sent to 3M's contract laboratory, Exygen Research (now MPI) in State College, Pennsylvania, for analysis. Neither PFOS nor PFOA were detected in the eleven Cottage Grove municipal wells. A trace of PFOA (defined as between 25 and 50 ppt) was detected in one of five Hastings municipal wells.

In mid-2006, the MDH Public Health Laboratory expanded the list of PFCs for analysis and lowered the analytical detection limits as described previously. Low levels of PFBA (0.1 to 0.3 ppb) were detected in several Woodbury municipal wells during routine sampling. No other PFCs were detected. By fall of 2006, it was found that low levels (0.1 to 0.5 ppb) of PFBA were present in all 16 Woodbury municipal wells. The presence of PFBA in those wells appeared to be the result of PFCs moving away from the disposal sites used by 3M in Oakdale and Lake Elmo (MDH, 2008). The city of Cottage Grove requested that their municipal wells be re-tested for PFCs using the expanded PFC list. In December 2006, PFBA was detected in all of the Cottage Grove municipal wells at concentrations ranging from 0.3 to 1.5 ppb. The detection of PFBA at concentrations higher than those found in the Woodbury municipal wells suggested that the PFCs in the Cottage Grove city wells were from another source, such as the 3M-Woodbury Disposal Site, located in or near Cottage Grove, rather than the 3M-Oakdale Disposal Site and Washington County Landfill.

The detections of PFBA in municipal wells in Woodbury and Cottage Grove triggered sampling of other municipal and community wells in south Washington and northern Dakota Counties to determine if they had been impacted by PFCs, beginning in January 2007. This sampling eventually included Woodbury, Cottage Grove, Newport, St. Paul Park, Hastings and South St. Paul; PFBA was detected in some or all of the municipal wells in each of these cities. Low levels (0.44 ppb on average) of PFBA were also found in a non-municipal community water supply well serving a housing development in

[1] Community wells are those that serve at least 25 people or 15 service connections. Municipal community wells are owned and operated by incorporated municipal governments; non-municipal community wells may be owned and operated by a homeowners association, manufactured mobile home park, etc.

Cottage Grove known as Eagles Watch, in the eastern part of the city. Sampling was conducted on a monthly basis in 2007 to determine if the levels of PFBA were changing. Sampling frequency changed from monthly to quarterly in 2008 when it became apparent that the levels were stable or even declining slightly.

The median, average and range of concentrations of PFBA detected in the affected wells serving each community's water supply system from June 2006 through December 2010 are shown in the table on page 15.

PFBA is the only PFC that has been detected in municipal wells in Newport and South St. Paul. Low levels of PFBS (up to 0.32 ppb) and PFHxS (up to 0.16 ppb) have been consistently detected in three Cottage Grove municipal wells; trace amounts of PFPeA and PFHxA (less than 0.1 ppb) have also been intermittently detected in various Cottage Grove municipal wells (see Table 4, Appendix 2). PFOA has been intermittently detected at approximately 0.05 ppb in one Hastings municipal well and one St. Paul Park municipal well. In Woodbury, PFHxS has been intermittently detected at approximately 0.05 ppb in one well, and PFOA was detected once at 0.05 ppb in one municipal well.

**Municipal Well PFBA Data, June 2006 – Dec. 2010**

| City | No. of Comm. Wells | No. of Comm. Wells w/PFBA | Range of PFBA (ppb) | Median of all Results (ppb) |
|---|---|---|---|---|
| Cottage Grove | 11 | 11 | 0.30 – 1.79 | 0.98 |
| Hastings | 5 | 5 | ND – 0.76 | 0.21 |
| Newport | 2 | 2 | 0.15 – 0.69 | 0.37 |
| St. Paul Park | 3 | 3 | 0.90 – 2.30 | 1.18 |
| So. St. Paul | 5 | 3 | ND – 0.37 | 0.05 |
| Woodbury | 17 | 17 | ND – 0.55 | 0.25 |
| NOTES: PFBA = perfluorobutanoic acid<br>ppb = parts per billion<br>ND = not detected above the method detection limit | | | | |

Levels of PFBA appear to be stable or declining slightly in many of the municipal and non-municipal community wells that have been regularly sampled by MDH since early 2007. As an example, Figure 6 is a graph of the concentration of PFBA in the 11 Cottage Grove municipal wells from January 2007 to September 2009. The reasons for a decline are unclear, but could reflect 1) an actual decline in the levels of PFBA in the Jordan aquifer; 2) movement of the contamination plume through increased pumping of community wells to meet demand; or 3) improvements in the accuracy of the analytical method over time. Continued data collection should help determine if the decline is real, and shed some light as to its causes.

MDH has also sampled several dozen non-community public wells located at churches, businesses, parks, and other locations throughout southern Washington and northern Dakota Counties. Results from these wells showed either no or low levels of PFBA.

Private Well Sampling

In June 2005, following detection of PFCs in monitoring wells at the 3M-Woodbury Disposal Site (Weston, 2007a), the MPCA and MDH collected water samples from 15 private wells near the site. These wells were selected as being representative of the Quaternary sand and gravel deposits above the bedrock and the three bedrock aquifers (St. Peter, Prairie du Chien, and Jordan) in use near the disposal site. At that time, PFOS and PFOA were the only PFCs for which analytical methods had been developed by the MDH Public Health Laboratory. Neither PFOA nor PFOS were detected.

In late 2006 - early 2007, the detection of PFBA in Woodbury, Cottage Grove, and several other city wells led to sampling of residential and non-community public wells (such as churches, businesses, etc.). Sampling of these wells indicated the area of PFBA contamination also included the communities of Grey Cloud Island Township, Denmark Township, the western edge of Afton, and the southernmost portion of Maplewood. The total area of PFBA contamination (including the areas affected by the Oakdale and Lake Elmo sites) encompasses over 100 square miles. Residents within this area (and in all of Washington County) rely entirely on groundwater as the source of their drinking water. Most residents are connected to city water, but it is estimated there over 4,000 private wells in these communities.

Given the scope of the potentially affected area, a private well sampling program was developed to provide rapid information regarding how far and how deep the PFC contamination had spread and where the highest concentrations were located. The state's County Well Index (CWI) was used to identify wells with geologic information in recorded driller's logs. Wells drawing water from each of the drinking water aquifers used in the investigation area were selected to provide geographic coverage of all of the neighborhoods in the potentially affected communities. More intensive sampling (i.e. sampling a higher density of wells) was undertaken in the following areas: 1) closest to and downgradient of the disposal site, 2) where PFBA concentrations were highest, 3) where the bedrock geology is complicated by faults and/or bedrock valleys, and 4) where spatial trends in PFBA concentrations were unpredictable. Sampling proceeded in an iterative fashion – with each round of sampling results informing decisions about the next round of samples. This allowed the MPCA and MDH to refine the sampling plan to identify and focus on areas of greatest potential health concern. By the end of 2008, water samples had been collected from over 900 residential and 56 business wells in the affected communities. A general summary of sampling results is provided in Table 2 (in Appendix 2).

The most commonly detected contaminant in private and business wells in the investigation area is PFBA. PFPeA is also frequently detected, but is typically found only in wells with PFBA concentrations of 1 ppb or greater. Most of the wells sampled were completed in the Prairie du Chien, Jordan, and Franconia aquifers, or had no record to indicate the aquifer in which they were completed. Figures 7 – 10 show the distribution of PFBA in the four major drinking water aquifers (St. Peter, Prairie du Chien, Jordan, and Franconia).

Other PFCs have been detected at the 3M-Woodbury Disposal Site and in two isolated areas of Cottage Grove. The first area is located south and west of Highway 10, primarily in the Langdon and River Acres neighborhoods, where multiple PFCs have been detected at low concentrations in some private wells. The second area is the main Cottage Grove city well field, where trace levels of PFHxS, PFBS, PFPeA, and PFHxA have been detected in several community wells. Further information on these detections is provided below.

PFC-Related Investigations at the 3M-Woodbury Disposal Site
To better define residual PFC levels in soil and waste materials at the site and provide some clues as to the source(s) of PFCs in groundwater, in April 2007 3M's consultant, Weston, installed a series of soil borings in the former Northeast Disposal Area (Weston, 2008a). Available historic information for the site suggested the presence of two trenches separated by soil mounds.

A total of 14 soil borings were drilled using direct-push drilling technology, generally to a depth of ten feet. Selected borings within the trench features were advanced to the bedrock, approximately 20 feet deep. Soil samples were collected continuously to characterize the soil cover (1.5 to 6.5 feet) and backfill material (1.5 to 17 feet) thickness and depth, as well as other descriptive information. Soils were also screened for organic vapors and pH. Selected soil samples were collected based on visual appearance and sent to the 3M Environmental Laboratory for PFC analysis. Selected samples were also analyzed for VOCs.

The soil boring locations and results for PFOS, PFOA, and PFBA in the Northeast Disposal Area are presented in Figure 11. PFC concentrations were generally higher in the backfill material in the former trench areas than in the mound areas. PFOA was detected in each of the soil samples collected in the former trench areas, at levels that range from 1,220 ppb to 26,100 ppb. Lower levels of PFBA were detected in 20 of 21 soil samples collected in the former Northeast Disposal Area. PFBA levels in the former trench areas ranged from 15 to 175 ppb. PFOS was detected in 11 of the 12 samples collected from backfill material in the former trenches. Detected concentrations ranged from 2,800 to 19,300 ppb. Other PFCs were infrequently detected; most notable was PFHxS in 11 soil samples at levels that ranged from 283 to 10,100 ppb. Petroleum related and chlorinated VOCs were also commonly detected in soil samples.

In the fall of 2007, soil borings were installed to further assess the material remaining in the disposal trenches at the former Main Disposal Area and Municipal Fill Areas for PFCs. Eighteen direct-push borings were completed within the eight former Main Disposal Area trenches (A-H) and two former Municipal Fill Areas (I, J). With approval from the MPCA, soil samples from this area were analyzed for a smaller list of five PFCs (PFOA, PFOS, PFHxS, PFBA and PFBS). Two soil samples were collected and analyzed for the five PFCs from each soil boring, usually one sample from the bottom of the disposal trench and a second sample from approximately five feet below the bottom of the trench. The soils borings ranged in depth from eight to 28 feet. The soil boring locations and PFC results for these areas are shown in Figure 12.

PFCs were detected at each of the soil boring locations within the former Main Disposal Area. PFC concentrations in the samples collected from beneath the fill/waste material were either non-detect or were less than that of the samples collected from the fill/waste material. The highest PFC concentration (PFOA at a concentration of 3,020,000 ppb) was detected at boring GPA04 at a depth of 16.5 feet. PFOS was detected at each of the 14 borings in the Main Disposal Area, at concentrations that ranged from 197 ppb to 17,600 ppb. PFBA was detected at lower concentrations compared to PFOA and PFOS, ranging from 1 to 225 ppb. Lower levels of PFCs were found in the soil samples collected from the four borings drilled in the former Municipal Fill Area.

In 2008, the MPCA requested that 3M collect a water sample from a small pond on the site known as Gables Lake. This pond is located approximately 2,000 feet south of the former Main Disposal Area, as shown in Figure 3. A sample collected one foot below the surface of the lake contained 0.103 ppb PFBA, 0.0913 ppb PFOA, and 0.0373 ppb PFOS (Weston, 2009).

In 2006-2007, twenty additional monitoring wells were installed to provide additional information about PFC distribution at the site and to monitor for any movement of contaminants away from the site (Weston, 2007a). Sampling has shown that PFCs are present in multiple monitoring wells at the site, primarily downgradient of the waste (Table 3, Appendix 2). The highest levels have consistently been found in monitoring well MW-2, near the Northeast Disposal Area (Figure 3). PFC concentrations at the site have been stable over the limited sampling period, and generally low concentrations are detected in wells near the boundaries of the 3M property. However, only three of the monitoring wells completed in the Prairie du Chien (wells MW-S06PC, MW-4L and MW-5) actually intersect the "high transmissivity zone" (HTZ) identified by Tipping, et al. (2006), which 3M's consultant identified as likely carrying the majority of flow in the Prairie du Chien (Weston, 2008a). MW-S06PC is located upgradient of the waste disposal areas and wells MW-4L and MW-5 are located west and west-southwest of the disposal areas, respectively (Figure 3). As discussed later in this report, although the regional groundwater flow direction is to the south-southwest, the highest PFC concentrations in groundwater are located south of the site in the area of the buried bedrock valley. The absence of monitoring wells intercepting the HTZ south of the site in the area where highest PFBA concentrations have been detected, and to the southwest (i.e. downgradient) of the high PFBA concentrations in the bedrock valley, means it is possible that a critical pathway for PFC migration may be missed by the monitoring network.

A groundwater remediation system is in operation at the 3M-Woodbury Disposal site. The system was originally installed to control migration of VOCs. The groundwater remediation system includes four gradient control/recovery wells. The system pumps an average of 4.6 million gallons of water per day from the wells. The water is discharged through a six mile long pipeline to the 3M-Cottage Grove facility where it is primarily used for non-contact cooling water prior to discharge to the Mississippi River under a permit issued by the MPCA. A hydraulic conductivity zone analysis was conducted by

Weston (2007b) which concluded the gradient control wells are fully containing the contamination on-site. This is consistent with previous evaluations of the gradient control system. However, possible gaps in the monitoring network, as described above, make it impossible to verify this.

3M proposed reducing the pumping rate at the disposal site, first by 25 percent and then, if no evidence is observed of contaminant migration from the site, by 50 percent (3M, 2010b). In their approval letter (MPCA, 2010a), the MPCA stated that groundwater data generated during the initial (25%) pumping rate reduction will be reviewed to determine what conditions, such as placement of additional monitoring wells that intercept the HTZ, might be necessary before any further pumping reductions might be allowed.

To determine if potential leakage from the pipeline carrying the pumped-out water from the site could be contributing to groundwater contamination in South Washington County, the MPCA requested that 3M conduct a thorough evaluation of the integrity of the line (Weston, 2008a). This was done over a period of several weeks during April and May 2007. The evaluation was done using direct sensing technology wherever possible (i.e. it was not done by measuring water flow into and out of the pipeline). No leaks were detected in the conveyance pipeline. There were two pipeline segments, located in Cottage Grove Ravine Park, where the evaluation was difficult because the segments were too long and inaccessible. Evaluating shorter pipeline sections in this area was not feasible due to the difficult physical setting (steep elevations, wooded areas, asphalt surfaces, etc.) and/or the pipeline depth (over 10 feet deep in some areas). 3M indicated that this portion of the pipeline was more recently installed (in 2003) and that it was intact at that time.

3M has also indicated that there are pressure monitoring points where the four groundwater extraction wells enter the pipeline, and where the line ends at the 3M Cottage Grove facility (3M, 2008). 3M has stated that any significant leaks from the line would be quickly detected and reported, and that no such leaks have been reported recently.

<u>MPCA – 3M Consent Order for PFC Disposal Sites</u>
At the MPCA April 24, 2007 Citizens' Board meeting, the Board was asked to approve a series of enforcement actions under the state Superfund law to ensure 3M undertook response actions to address the PFC contamination from three known PFC disposal sites: the 3M-Cottage Grove facility, the 3M-Woodbury Disposal Site, and the 3M-Oakdale Disposal Site (MPCA, 2007c). The former Washington County Landfill was not included because under the MPCA Closed Landfill Program, the MPCA has assumed responsibility for the site.

Instead of approving the enforcement actions, the Citizens' Board directed MPCA staff to negotiate a Consent Order with 3M on PFC contamination in Minnesota (MPCA, 2007d). A Consent Order is a site cleanup agreement between the MPCA and a responsible party. The Board directed staff to address seven concerns with regards to the disposal sites and proposed actions in the Order, as follows:

1. A rigorous, robust cleanup plan for the disposal sites.
2. Recognition of the MPCA's jurisdiction.
3. Municipal and private drinking water supplies addressed.
4. Address future actions on PFBA.
5. Address additional studies on health and environmental effects.
6. Address cooperation from 3M on sharing research and information.
7. Preserve the MPCA's right to take action in the future.

The MPCA and 3M negotiated the Order, and presented it to the MPCA Citizens' Board for approval at its May 22, 2007 meeting. The Citizens' Board unanimously approved the Consent Order with 3M. The consent order can be accessed on the MPCA web site at: http://www.pca.state.mn.us/cleanup/pfc/index.html (MPCA, 2007e):

In the Consent Order, 3M agreed to contribute up to $8 million to remediate the former Washington County Landfill. 3M is also obligated under the Consent Order to provide alternate sources of drinking water in the case where PFC levels in a public or private well exceed MDH health-based exposure limits. Also included in the Consent Order is an agreement that the MPCA does not waive its right to pursue any natural-resource damage claims related to releases of PFCs from the sites. 3M provides regular updates to the MPCA and MDH on various activities under the Consent Order; the MPCA Board is updated on a quarterly basis by MPCA and MDH staff on 3M's progress under the Consent Order.

Remedial Options for the 3M-Woodbury Disposal Site

Based on data collected during previous investigations conducted at the site, and the PFC-related investigations described above, 3M has evaluated various response action alternatives for the site (Weston, 2008a). The alternatives evaluated by 3M and Weston, both sitewide and for specific contaminated media, include:

- Sitewide Alternative 1 – No action.
- Sitewide Alternative 2 – Institutional controls, access restriction and groundwater monitoring.
- Groundwater Alternative 1 – Continued groundwater recovery with GAC treatment, as necessary, to meet appropriate discharge criteria.
- Soil Alternative 1 – Excavation of the former Northeast Disposal Area trenches; disposal at an existing off-site landfill.
- Soil Alternative 2 – Excavation of the former Northeast Disposal Area trenches; disposal at an existing off-site landfill; and 4 feet (minimum) cover over selected areas in the former Main Disposal Area.
- Soil Alternative 3 – Excavation of the former Northeast Disposal Area trenches and selective removal of soil which exceeds Industrial SRVs from trenches in the former Main Disposal Area; disposal at an existing off-site landfill.

Each of the response action alternatives was evaluated against the primary goal of protecting the public health and the environment. The proposed response actions also were evaluated in light of the requirements agreed to by 3M in the Consent Order. Finally, the response action alternatives were evaluated using the following "balancing criteria:" 1) long-term effectiveness; 2) implementability; 3) short-term risks; and 4) total costs. A 'no action' alternative is evaluated at every site. Based on these evaluations, 3M proposed the following recommended response action alternatives for the site:

- Institutional controls, access restriction, and groundwater monitoring (Sitewide Alternative SW-2), and
- Continued groundwater recovery with GAC treatment as necessary (Groundwater Alternative GW-1).

3M also stated that any of the three soil alternatives were acceptable, but left the final decision to the MPCA. The MPCA selected Soil Alternative 3, which included the most extensive soil and waste excavation, and documented their decision in a Minnesota Decision Document for the site dated December 22, 2008. After further analysis, MPCA determined that by slightly reducing the mass of PFCs removed from the site, the cleanup could be completed much sooner and at less cost to the environment in terms of fuel use, truck mileage, and landfill space. This "modified Soil Alternative 3" is in fact the plan being implemented at the site by 3M. Additional details on the cleanup of the site, including various steps taken to prepare the site for cleanup can be found on the MPCA's web site at http://www.pca.state.mn.us/cleanup/pfc/pfcsites.html.

3M began this phase of soil cleanup work at the site in the summer of 2009. Approximately 24,900 cubic yards of soil and waste were excavated from the main and northeast disposal areas and transported to the SKB Landfill in Rosemount, Minnesota. The soil and waste were placed in a dedicated cell constructed specifically to receive the soil and waste from the Woodbury and Oakdale sites, and waste that was buried at the 3M-Cottage Grove facility. An additional 2,750 cubic yards of excavated soil and waste from the main disposal area were transported for out-of-state disposal at a permitted hazardous waste facility due to the presence of other regulated contaminants.

Site Visits

MDH staff has conducted several visits to the 3M-Woodbury Disposal Site and its vicinity during the past three years to conduct private well searches, collect well water samples, and attend local government and public meetings.

The 3M-Woodbury Disposal Site is partially fenced, and access from County Road 19 is limited by several locked gates. "No Trespassing" signs are also prominently placed along the fence and gates. However, MDH staff were shown by neighbors where the fencing has fallen into disrepair along the south border of the property; foot trails onto the property and information from nearby residents indicate at least some trespassing does occur.

3M reports that they have regular security patrols at and around the site. 3M allows some of the land to be used for agricultural purposes, and employee recreational activity clubs also use portions of the site, but these areas are not located near the former disposal sites.

Demographics, Land Use, and Natural Resources

Nearly 150,000 people live in areas of Washington and Dakota Counties where measurable levels of PFBA have been detected in community water supplies or private wells. The estimated 2008 populations for the affected cities and townships are (Minnesota Department of Administration, 2009):

- Woodbury: 58,430
- Cottage Grove: 34,017
- St. Paul Park: 5,293
- Newport: 3,542
- Afton: 2,899
- Denmark Township: 1,726
- Grey Cloud Island Township: 362
- Hastings: 22,488
- South St. Paul: 20,250

These cities and townships represent a variety of land uses, from a typical suburban mix of compact residential areas, light commercial districts, and retail areas, to mainly rural residential and agricultural areas. The area has experienced significant population growth and development in the last ten to twenty years. In the larger cities, the majority of the population is served by community water supplies, but more rural areas rely on private wells for drinking water, and will for the foreseeable future.

The area is home to numerous parks and recreational areas, including Afton State Park, St. Croix Bluffs and Cottage Grove Ravine Regional Parks, Point Douglas Park, and the Carpenter Nature Center. MDH has collected water samples from drinking water wells within the parks to look for the presence of PFCs. Low levels of PFBA were detected in two wells serving the Carpenter Nature Center (0.9 and 1.8 ppb), one well at Cottage Grove Ravine Regional Park (0.5 ppb), and the well serving Point Douglas Park (0.1 ppb). No PFCs were detected in wells at Afton State Park, or St. Croix Bluffs Regional Park. Both of these parks are on the eastern border of Washington County, along the St. Croix River.

In August 2007 MDH issued a press release announcing revised fish consumption advice for several lakes in the Twin Cities metro area, including Ravine Lake, which is located within Cottage Grove Ravine Regional Park. The press release was issued due to the detection of PFOS in fish tissue samples collected by the MPCA from several lakes at levels high enough to warrant revised fish consumption advice for certain fish species. In the case of Ravine Lake, fish consumption advice was issued recommending no more than one meal per week of black crappie and largemouth bass. Additional data collected by the MPCA later in 2007 resulted in new fish consumption advice being issued for one other lake in south Washington County, Powers Lake in Woodbury, based on PFOS levels in fish. For specific guidance, please refer to MDH's Fish Consumption Advice web site at http://www.health.state.mn.us/divs/eh/fish/index.html. The specific source of the PFOS detected in fish in these lakes is not clear.

General Regional Issues

This region of the eastern Twin Cities metropolitan area will likely continue to experience substantial population growth in the coming years, although development has slowed recently due to the economic downturn in 2008. Because continued growth may present a strain on area resources such as water supplies, the need for expansion of water supply systems has been evaluated by the cities and new community supply wells will be needed. The widespread PFC contamination in the aquifers typically used for municipal water supplies has complicated this process. Currently, Woodbury and Cottage Grove are in the process of siting or constructing new community wells to meet projected demand.

Community Concerns

MDH staff have had numerous contacts with citizens living in the affected areas of Washington, Dakota, and Ramsey Counties who have expressed concern about PFCs in their private well or the community water supply. Community meetings were held by MDH and MPCA in Hastings, Woodbury, Cottage Grove, St. Paul Park, and South St. Paul in February 2007, and again in February 2008. A total of approximately 400 people attended these meetings. MDH has also attended many other local meetings in the two cites, and responded to hundreds of phone calls and e-mails.

Some residents have expressed concern about the following: that cancer or other disease rates in the area seem higher than normal, the health implications for children who may have been exposed to contaminated water (both before and after birth), the health of domestic animals that may be drinking contaminated water, and the potential for uptake of PFCs by plants irrigated with contaminated water. Residents also had questions about multiple exposure pathways to PFCs, and the lack of health-based exposure limits for some PFCs in water. MDH has made every effort to address these health issues where possible, including an analysis of cancer rates that is described later in this report. MDH has produced multiple information sheets for area residents, regularly updated its web site on PFCs (www.health.state.mn.us/divs/eh/hazardous/topics/pfcs/index.html), and created an e-mail distribution list (1,330 subscribers as of September 2009) to notify interested residents and local officials of new information. The cities (and Washington County) have also provided updates for local residents in their city newsletters, annual water quality reports, and web sites. There have also been numerous stories in state, Twin Cities, and local media.

A draft version of this Public Health Assessment report was released for public comment on August 25, 2010. MDH received written comments from State Senator Katie Sieben, the MPCA, and 3M (Appendix 3). Sen. Sieben's comments primarily focused on the unknowns regarding past PFC exposure levels, possible long-term health effects, and the need for continued monitoring of water quality and public health in the affected communities (Sieben, 2010). The MPCA comments focused primarily on differences in interpretation regarding the adequacy of the monitoring well network at the 3M-Woodbury Disposal Site and the source PFCs (other than PFBA) in the Langdon-River

Acres neighborhood in south Cottage Grove (MPCA, 2010b). These comments were addressed in the "PFC-Related Investigations at the 3M-Woodbury Disposal Site" and "Evaluation of Impacts to Groundwater" sections of this document. 3M's comments addressed a number of areas, but primarily focused on: differences in hydrogeologic interpretations, the adequacy of the monitoring well network, 3M's PFC waste disposal and reporting history, PFC toxicology and the potential for human health effects associated with PFC releases from the 3M-Woodbury Disposal Site, and the recommendation to extend city water to the most contaminated neighborhoods (3M, 2010a). These comments were addressed, as appropriate, throughout the document, but primarily in the "PFC-Related Investigations at the 3M-Woodbury Disposal Site," "Evaluation of Impacts to Groundwater," "Exposure Through Private Wells, " and "Public Health Implications of PFC Exposure" sections of this document.

## Evaluation of Environmental Fate and Exposure Pathways

Introduction

PFCs, primarily perfluorooctanoic acid (PFOA; $C_8F_{15}O_2H$) and one of its salts, ammonium perfluorooctanoate (APFO; $C_8F_{15}O_2NH_4$), as well as lesser amounts of other PFCs such as perfluorooctanesulfonyl fluoride (POSF; $C_8F_{17}SO_2F$) and perfluorobutanoic acid (PFBA, $C_4F_7O_2H$) were manufactured by 3M at their Cottage Grove facility (formerly known as Chemolite) from the early 1950s until 2002. Production of the four-carbon compound, perfluorobutanoic acid (PFBA) ceased in 1998. 3M currently produces perfluorobutane sulfonate (PFBS)-based products which are substitutes for the earlier eight-carbon PFCs. PFBS is also a four-carbon compound. In addition, 3M continues to use and/or produce one- to three-carbon perfluoroalkyl substances at the Cottage Grove facility (3M, 2010a). One of the byproducts of the production of POSF is perfluorooctane sulfonate (PFOS; $C_8F_{17}SO_3^-$), which can also be produced by the subsequent chemical or enzymatic hydrolysis of POSF. The chemical structures of PFOA, PFOS, and PFBA are shown in Figure 2.

In 2000, 3M announced it was voluntarily phasing out production of all of its eight-carbon PFCs, including PFOS and products which could degrade or metabolize to PFOS. 3M ceased production of PFOA, PFOS and precursor materials by the end of 2002. In its reformulated stain repellent and other commercial products such as Scotchgard™, 3M used a chemistry based on the four carbon sulfonic acid, PFBS, instead of the eight carbon PFOS (Brezinski 2003) and such production continues today. EPA has prohibited the production or import of PFOS and PFOS precursors, except for certain critical use exemptions where no alternatives were available and the use involves very low volumes and low exposure risk. There are still some commercial uses of PFOS in specialty products (primarily in the semi-conductor, metal plating, and aviation industries).

3M ceased production of PFBA in 1998. To the knowledge of MDH, there is currently no commercial production of PFBA in the U.S., but some PFBA is reportedly imported for commercial applications and for use in analytical laboratories, which may also use other four-carbon compounds that break down to PFBA in the environment. Certain fluorochemicals which break down to PFBA in the environment have also been used in pesticides, although it is not clear whether this use continues today.

Environmental Fate

The carbon-fluorine bond is a high-energy bond, one of the strongest known among organic molecules. As a result, the chemical structures of the PFCs described above make them extremely resistant to natural breakdown, and they are persistent once released to the environment. Their structure also makes them excellent surfactants. The word surfactant is an acronym for 'surface active agent' - a molecule that lowers surface tension in a liquid. Surfactant molecules contain both a hydrophobic ('water-hating') and hydrophilic ('water-loving') component, making them semi-soluble in both organic and aqueous solvents. Surfactants are the active ingredients in soaps and detergents, where the hydrophobic component sticks to grease and dirt while the hydrophilic section sticks to water, helping to remove dirt from skin and hair and stains from fabric. These same properties can also be used to essentially help make materials resistant to water and stains, one of the primary markets for these chemicals. Information on the physical properties of PFCs that would make them potentially useful in industrial applications was published by 3M scientists in technical journals as far back as the early 1950s (Kauck and Diesslin, 1951; Reid et al., 1955).

On the basis of its physical properties, PFOS is essentially non-volatile, and would not be expected to evaporate from water (OECD, 2002). In soil-water mixtures, PFOS has a strong tendency to remain in water due to its solubility (typically 80% remains in water and 20% in soil). PFOS does not easily adsorb to sediments, and is expected to be mobile in water at equilibrium (3M, 2003a).

PFOA is slightly more volatile than PFOS, although it also has a very low volatility and vapor pressure (EPA, 2002). PFOA salts are very soluble and completely disassociate in water; in aqueous solution PFOA may loosely collect at the air/water interface and partition between them (3M, 2003b). In published studies and reports, PFOA has shown a high mobility in some soil types (EPA, 2002). In a study of the sorption potential for various PFCs in sediments, Higgins and Luthy (2006) found that the carbon chain length had a major effect on sorption potential – the longer the chain the more likely adsorption would occur, and that perfluorosulfonates (i.e. PFOS) tended to bind more readily to sediment than perfluorocarboxylates (i.e. PFOA). Other environmental conditions that could affect adsorption include organic carbon content of the sediment, pH, and dissolved calcium. Other studies have shown generally similar results, and adsorption behavior in soils is likely to be very similar to that observed in sediments (Prevedouros et al., 2006). A review of the bioaccumulation potential of a variety of PFCs by Conder et al. (2008) found similar results, in that PFOS and longer chain perfluorocarboxylates (greater than eight carbons) had a greater potential to accumulate in living organisms.

The vapor pressure and water solubility of PFBA are similar to PFOA (Kwan, 2001). PFBA is very soluble in water, and appears to travel easily with groundwater. A number of fluorinated compounds (fluorinated benzoates, not perfluorinated chemicals) are in fact used as tracers in groundwater flow studies due to their environmental persistence and negligible adsorption to soil and aquifer materials (Flury and Wai, 2003; Shapiro, 2008). The study of sediment adsorption of selected PFCs by Higgins and Luthy (2006), which unfortunately did not include PFBA, nonetheless supports the notion that PFBA may be even more mobile than PFOA or PFOS in the environment because it is a perfluorocarboxylate with a short carbon chain length.

Evaluation of Impacts on Groundwater

The information obtained from investigation and remedial activities at the disposal sites, surface water sampling, and sampling of private, municipal, and non-community wells has been used to evaluate the magnitude, extent, and possible migration history of the PFC contamination in south Washington County. PFBA has been detected in all four of the major drinking water aquifers in the investigation area, with the most widespread contamination documented in the Prairie du Chien and Jordan aquifers (these are also the aquifers most widely used in this area and so have the most wells to be sampled). The extent of contamination in the St. Peter and overlying Quaternary sands and gravels is poorly understood, due to the few number of wells present in those aquifers. Contamination in the Franconia is generally low in concentration and appears to be localized near major bedrock faults that have brought the Franconia into contact with the Jordan (Figure 4), allowing PFCs in the Jordan to migrate into the Franconia.

An area of contaminated groundwater is often referred to as a "plume" (like a plume of smoke). "Typical" contaminant plumes have their highest concentrations near the source area, decrease in concentration with distance from that source, and are roughly elliptical in shape with the axis of the plume roughly in line with the direction groundwater is flowing.

In contrast, the PFBA plume in the investigation area is quite complex (see Figures 8 and 9). The highest concentrations of PFBA are detected south of the site and appear to coincide with the buried bedrock valley that underlies the western edge of the site and trends south through Cottage Grove Ravine Park to the Mississippi River (Figures 8 and 9). Although the regional groundwater flow direction in the area of the site is to the southwest, bedrock features, such as faults and buried valleys, and even smaller scale features such as solution cavities within the bedrock, may exert significant control over groundwater and contaminant migration on a local scale.

Similarly, a patch of slightly higher PFBA concentrations near the border of Cottage Grove, St. Paul Park, and Newport (shown in yellow, just southeast of the intersection of Hwy 61 and I-494) appears to follow the outline of the bedrock valley in that area (see Figure 8). An arm of that valley appears to trend back toward the site, but an absence of wells in this area limits our ability to definitely trace the zone of higher PFBA concentrations back to the site.

Another unusual area within the plume is the seemingly isolated area of PFBA present in the Jordan aquifer near the St. Croix River in the southeastern portion of the investigation area (Figure 9). This area is bounded by a series of major bedrock faults and the top of the Jordan east of this faulted zone is over 200 feet lower in elevation than the top of the

Jordan to the west of the zone. The Jordan on the east side of this heavily faulted zone is essentially at the same elevation as the Franconia aquifer on the west side. As shown in Figure 10, PFBA is present in the Franconia west of the faulted zone and at concentrations similar to those found in the Jordan east of the fault. In other words, the PFBA plume appears to be migrating from the Jordan into the Franconia across a fault near the ravine, traveling through the Franconia as the groundwater moves to the east-southeast, and then crosses another fault and back into the Jordan near the St. Croix River, as illustrated in Figure 4.

PFBA has also dispersed south from the two 3M waste disposal sites in Lake Elmo and Oakdale (Figure 1; MDH, 2008a), further complicating our understanding of the PFBA distribution in the area south of I-94. Groundwater modeling (Barr, 2005a) and measured PFBA concentration trends downgradient of those two disposal sites suggest they may be the source of the PFBA detected in parts of Maplewood, north Woodbury, and Afton, and may also contribute some of the PFBA contamination in the other affected communities.

There are also areas with unusual PFBA distribution patterns that, at present, cannot be attributed to bedrock structures or migration from the northern disposal sites. The most striking of these is the "finger" of elevated PFBA in the Prairie du Chien aquifer extending north of the 3M-Woodbury Disposal Site (Figure 8). Although it appears to follow the same northeast-southwest orientation of the faults in the area, no fault has been identified in this portion of the investigation area. The mechanism that created this "finger" of PFBA has not been determined.

Although PFBA is the most widely detected PFC in Washington County, multiple PFCs were detected at the disposal site and the 3M Cottage Grove facility, and are also found in private wells in several isolated areas of Cottage Grove (Figure 13). The largest area is located south and west of Highway 61, primarily in the Langdon and River Acres neighborhoods, where non-PFBA PFCs (PFOA, PFOS, PFHxS, PFBS, PFPeA, and PFHxA) have been detected in some private wells, generally at low concentrations (see table below). Although Figure 13 shows three distinct areas with multiple PFCs south of Highway 61, there are few wells between these areas so currently it is not possible to determine whether they are hydraulically connected or separate plumes. The second area is the main Cottage Grove city well field, where trace levels of PFHxS, PFBS, PFPeA, and PFHxA have been detected in several of the wells (Table 4, Appendix 2). A trace level of PFHxA (0.06 ppb) has also been detected in one well near Tower Road, but this result has not been confirmed.

**Maximum Concentrations of non-PFBA PFCs in Langdon/River Acres Area Private Well Water**

| PFOA | PFOS | PFPeA | PFHxA | PFBS | PFHxS |
|---|---|---|---|---|---|
| 1.4 | 0.9 | 0.3 | 0.16 | 0.12 | 0.15 |
| NOTES: All concentrations in ppb<br>PFC = perfluorinated chemicals | | | | | |

The presence of multiple PFCs, other than PFBA and PFPeA, in these two areas is unusual because these compounds are not usually detected in wells between the disposal site and the two areas described (Figure 14), although the relatively lower density of wells in this area means the data are somewhat limited. MPCA considered whether the multiple PFCs detected in these areas may be related to fire-fighting activities. Many fire-fighting foams used to fight chemical fires contain PFCs. The Cottage Grove Fire Department originally reported to the MPCA that it used small amounts of such foam in their training activities, which occur near the main city well field (Delta, 2008). However, the department later stated that although they use PFC-bearing foams to fight fires, they do not use them for training purposes.

Another possible link to use of fire-fighting chemicals in the areas south of Highway 61 was considered. In late 2002, PFC-bearing foams were used to extinguish a major industrial fire immediately south of Highway 61 and the foams reportedly discharged to a wetland area immediately southwest of the Langdon neighborhood and upgradient of the River Acres neighborhood. However, the MPCA analyzed environmental data collected near the site of the fire as well as from other fire-fighting foam sites, and has concluded that the fire was not a major contributor to groundwater contamination in the area. Furthermore, the MPCA has determined that the source of the PFC contamination in the area is the 3M Woodbury Disposal Site (MPCA, 2010b; see comment letter in Appendix 3). It is the MPCA's responsibility to determine contamination sources, with MDH advice (MDH/MPCA Memorandum of Agreement to Address Groundwater Contamination Situations, June 2010, revised November 2010). As noted elsewhere, public health is being protected by provision of whole-house Granulated Activated Carbon (GAC) filters to homes served by wells with PFC contaminant levels above HRLs.

On-going monitoring of private wells continues to track PFC concentrations in each of the affected drinking water aquifers across the investigation area over time. As with the initial sampling effort, more wells are sampled in areas which have: higher PFC levels, more complex geology, and/or unpredictable trends in PFC concentrations. In areas where concentrations are lower and the distribution patterns are predictable, "sentry wells" are sampled regularly to provide representative results for each of the aquifers in use in that area. These wells act as early warning "sentries" for any unusual results that might warrant sampling of additional wells in the area. The sampling schedule, both in terms of frequency and number of wells, is revised regularly as more data is collected and the behavior of the PFC plume in the affected communities is better understood. The number of wells sampled and the sampling frequency in most areas has decreased over time because the PFC concentrations are low and stable throughout most of the affected areas and so do not warrant frequent monitoring.

Sampling of the monitoring wells at the disposal site also indicate stable concentrations and appear to indicate that, as a result of the groundwater capture system in operation there, PFCs are not migrating off-site. However, as noted above, the absence of monitoring wells screened across the "high transmissivity zone" in the Prairie du Chien south of the site (i.e. the direction in which the highest PFBA concentrations have been observed) and southwest (i.e. downgradient) of the bedrock valley, may represent a gap in the integrity of the monitoring network. In 2010, the MPCA approved a request for 3M to reduce the number of wells and the volume of water being pumped at the site, on the condition that additional monitoring occur to ensure the reduce pumping rates did not result in movement of contaminants from the site. MPCA also noted that additional monitoring wells may be required if monitoring indicates they are needed (MPCA, 2010a). 3M has yet to reduce pumping rates.

The groundwater monitoring results indicate PFC concentrations in all of the aquifers are stable. This is consistent with findings in Lake Elmo and Oakdale (MDH, 2008a) and suggests that the size, shape, and concentrations of the PFC plumes in the individual aquifers may have reached a state of equilibrium, so that even though PFCs continue to migrate with the groundwater, their concentrations remain generally stable. However, groundwater systems are inherently dynamic and conditions may change over time, which is why sampling of private wells continues to track any changes that may occur.

Based on our understanding of the way PFCs move in the groundwater environment and the high flow velocities known to exist in the investigation area, it is likely the PFCs from the disposal site migrated rapidly through the affected communities relatively soon after the wastes were disposed and what is present today are remnants of contaminants slowly washing out of the aquifers. This slow washing out of contaminants is sometimes referred to as “secondary flow” or “matrix diffusion”, and occurs because there are major (i.e. larger, faster) flow paths and minor (i.e. smaller, slower) flow paths within the aquifer (Figure 15). The majority of the contaminants flow quickly through the major flow paths in an initial “pulse”, rapidly establishing the contaminant plume, while contaminants that moved into the minor flow paths slowly release over time at more dilute concentrations. If this is what happened in the investigation area, earlier concentrations in the aquifers may have been higher than what is currently being detected. However, due to the high level of uncertainty involved in such calculations, MDH has not attempted to calculate how high those concentrations might have been or how long the higher concentrations may have lasted.

The persistence of PFCs in the environment means that ultimately the PFCs in the groundwater in Washington County will discharge to the Mississippi and St. Croix Rivers. However, the low PFC concentrations in the groundwater (except at the 3M-Cottage Grove facility) and the large volume of flow in the rivers will likely dilute this discharge to low concentrations. Samples of water from the Mississippi River were collected as part of the investigation of the waste disposal areas at the 3M Cottage Grove Plant (MDH, 2005; Weston, 2008c). While elevated concentrations of PFCs were detected in samples near and immediately downstream of the plant, no PFCs were detected in water samples collected upstream of the plant. Low levels of PFOS (0.289 – 1.34 ppb) were detected in sediment samples collected upstream of the plant. Although PFOS is found in the groundwater at the 3M-Woodbury site, it is not present in the groundwater in most of the areas covered by this report with the exception of the trace levels detected in 23 wells in the anomalous area south of Hwy 61 and much higher concentrations in the groundwater at the 3M-Cottage Grove facility. The distribution of

PFOS in the groundwater and the proximity of the contaminated sediments to the 3M-Cottage Grove facility suggest the source of the PFOS in the contaminated sediments is the facility via discharge of waste water and contaminated groundwater.

Exposure through Private Wells

PFCs can affect human health only if the chemicals move from the environment and come into contact with or accumulate in a person's body. The movement of PFCs (or other contaminants) from the environment into a person's body is called an exposure pathway.

An environmental exposure pathway contains five parts: (1) a source of contamination, (2) contaminant transport through an environmental material (i.e., soil, air, water, or food), (3) a point of exposure, (4) a route of human exposure, and (5) a receptor population. An exposure pathway is considered *complete* if evidence exists that all five of these elements are or have been present in a community or a given situation. More simply stated an exposure pathway is considered complete when people are likely to be exposed to the chemical of concern. A pathway is considered a *potential* exposure pathway if at least one of the elements is missing but could be found at some point. An *incomplete* pathway is when at least one element is missing and will never be present.

A completed environmental exposure pathway to PFCs from the disposal of PFC-containing wastes in disposal sites in Washington County exists through the consumption of PFC contaminated drinking water.

Samples have been collected from over 900 private wells and 56 business wells in Woodbury, Cottage Grove, and other communities in south Washington County (south of I-94). PFCs have been detected in 745 private and business wells. No wells, public or private, south of I-94 have been found to contain PFBA at concentrations above the Health Based Value (HBV) of 7 ppb for PFBA. The highest PFBA concentration detected was 5.5 ppb, but the majority of wells sampled had PFBA concentrations below 1 ppb.

As of late 2010, 34 private well owners in Cottage Grove and Grey Cloud Island had received a drinking water well advisory from MDH due to the presence of PFCs. All of these wells are located in neighborhoods south of U.S. Highway 61, in the area of anomalous PFC detections, as discussed above. Nine of these private wells have PFOA at concentrations above the HRL of 0.3 ppb; one of which also has PFOS at concentrations above the HRL of 0.3 ppb. Sixteen of the wells have concentrations of PFCs that exceed a Hazard Index value of one based on multiple PFCs.

The remaining ten wells have PFC levels that are below current health advisory levels. The advisories for these ten wells, where multiple PFCs are present, were issued in 2007 when the health risks of PFBA were poorly understood and a HBV had not yet been set for it. The drinking water well advisories have remained in effect due to uncertainties over groundwater flow.

Where drinking water well advisories have been issued by MDH, exposures have been reduced to acceptable levels by the provision of bottled water and/or GAC filters by the MPCA. It is also possible that the use of owner-installed filter systems may have reduced past exposure to PFCs in drinking water. During the course of the investigation, it was found that a number of private well owners who subsequently received a drinking water well advisory due to PFCs had previously installed reverse osmosis water filters to remove nitrate, a common groundwater contaminant in the area (Barr, 2005b; MDH, 2002). Although not related to the PFC contamination, the high nitrate levels generally occur in the same places as higher PFC concentrations as their movement in the groundwater is controlled by the same bedrock features and groundwater flow that control the movement of the PFCs (Barr, 2003). Reverse osmosis filters, if maintained properly, are also very effective at removing PFCs according to a study conducted by MDH in 2008 (MDH, 2008b). The MDH study also documented which types of point-of-use carbon filters are effective at removing PFCs from drinking water.

Using adsorption factors developed by 3M for a similar GAC system installed at their Cottage Grove plant, the predicted breakthrough time for each filter can be calculated based on the influent concentration and an assumed water use rate of 300 gallons per day. MDH and MPCA staff use a tracking system to monitor water use at each home, and have collected multiple samples to monitor system performance. At average water use, the filters are predicted to last, in some cases, for years before maintenance is needed. The MPCA has determined that the filters will be changed according to a predictable, conservative schedule in order to ensure they operate effectively.

In 2008, MDH conducted a biomonitoring study of PFCs in the blood serum of Oakdale city water users and private well users in Lake Elmo and Cottage Grove, whose wells exceeded Minnesota drinking water standards (MDH, 2009). The study found 100 percent of the exposed individuals had PFOA, PFOS and PFHxS in their blood serum, the average concentrations of which exceeded the averages for the general US population. A 2010 follow-up biomonitoring study was conducted with the same individuals to determine if removal of PFCs from the city and private well water (as a result installing GAC filters) had any effect on their blood serum PFC levels. Average PFOA, PFOS, and PFHxS blood serum concentrations decreased by 21%, 26% and 13%, respectively, indicating that GAC treatment of the water had effectively reduced PFC exposures and body burdens for the users of the treated water (MDH, 2011).

Even though GAC filters are very effective in removing organic contaminants such as PFCs, MDH does not consider them to be the best long-term solution in areas where alternate water supplies are available. If not properly maintained, GAC filters are a friendly environment for bacterial growth. As noted above, nitrate is commonly found in the groundwater in south Washington County. Nitrate in standing water that remains in a GAC filter over long periods of time (such as an extended vacation when the house is unoccupied) may be converted by bacteria to nitrite at potentially unsafe levels (MDH 2007d). As a result, GAC filters require regular maintenance, which the MPCA is providing, but this is not an ideal solution for numerous individual households.

The length of time local residents were exposed to PFCs through their drinking water is unknown. As discussed above, exposures could have started soon after PFCs wastes were placed in the disposal sites, given the mobility of PFCs in the environment. It is also possible that the levels in the groundwater were higher than currently detected, although MDH has not attempted to determine what the concentrations may have been in the past or how long those higher concentrations may have lasted, due to the high degree of uncertainty involved in such calculations. Continued routine monitoring of select private wells with levels of PFCs below the health based drinking water standards will ensure that, if levels of PFCs rise, future exposure to levels above health-based exposure limits will be brief.

Exposure through Public Water Supplies

The municipal and community wells in the affected area were sampled for PFCs by MDH on a monthly basis through 2007, and are now sampled on a quarterly basis, and the results of monitoring are reported to the city staff. While not required under the federal Safe Drinking Water Act, some cities report PFC results in their annual "Consumer Confidence Report" to their water customers. MDH will continue to monitor the wells, but to date very little change (other than perhaps a slight declining trend) has been observed in PFC levels in the municipal and community wells.

No individual municipal or community well in the affected communities covered by this report have exceeded current MDH health-based exposure limits for PFCs. The city of Oakdale has several community wells and the city of Lake Elmo has one non-operational well which have exceeded the MDH drinking water standards. This is described in a previous MDH report (MDH, 2008a).

Exposure through other Pathways

The use of water contaminated with low levels of PFCs for bathing, showering, or other incidental uses is unlikely to contribute appreciably to overall exposure. Ingestion of the contaminated water is by far the predominant exposure pathway. Use of PFC contaminated water for canning or cooking purposes may also contribute to exposure, as reported by Emmett et al. (2006a) and Holzer et al. (2008). Irrigation of plants with PFC contaminated water may possibly lead to some uptake of PFCs by the plants, also contributing to overall exposure. MDH is currently examining the potential for exposure to PFCs by analyzing soil and produce samples collected at properties in Oakdale, Lake Elmo, and Cottage Grove that have or had a PFC-contaminated water supply (see http://www.health.state.mn.us/divs/eh/hazardous/topics/pfcs/pihgs.html).

So-called "market basket" surveys of food products occasionally show low levels of PFCs. In a study conducted in the United Kingdom, PFOS was found at low concentrations in potatoes, some canned vegetables, eggs, and in the sugars and preserves food groups, while PFOA was detected only in potatoes (UK Food Standards Agency, 2006). A similar study in Spain found low levels of PFOS in fish, dairy products, and meat, while PFOA was only detected in milk (Ericson et al., 2008). A recent study of food items in Canada also found low levels of PFOS and PFOA in some food products, including beef, fish, and microwave popcorn (Tittlemier et al., 2007). It is less likely that PFBA would be taken up by plants, but data are not available.

The consumption of fish from local lakes or rivers where PFOS has been detected in fish populations represents a completed exposure pathway, although the source of the PFOS may not be in all cases from land disposal of PFC-containing wastes.

The specific sources of exposure to PFOS, PFOA, and other perfluorochemicals in the general population are unclear, but could include consumer products, environmental exposures, or other occupational exposures (Butenhoff et al., 2006). Both PFOS and PFOA have been detected in samples of household dust collected from vacuum cleaner bags in Japan (Moriwaki et al., 2003), Canada (Kubwabo et al., 2005), and the U.S. (Strynar and Lindstrom, 2008), indicating the indoor environment is one potential source of exposure. Low ppt levels of PFOS have also been detected in rainwater collected in Winnipeg, Canada (Loewen et al., 2005). It should be noted that since 2000, levels of PFOS and PFOA in blood serum have decreased (Olsen, et al., 2008).

Small amounts of unbound fluorotelomer alcohols that can break down to PFOA (or other PFCs depending on their specific composition; also referred to as PFC precursors) have also been found in consumer and industrial products (Joyce et al., 2006). Release of telomer alcohols, and subsequent degradation in the environment or by organisms, could also be a source of human exposure to PFCs.

## Public Health Implications of PFC Exposure

This section briefly summarizes information reviewed on the toxicity of PFCs to animals and humans for this report, and summarize the public health implications of exposure to PFCs through drinking water in affected communities in south Washington County, Minnesota.

Note: ATSDR published a draft "Toxicological Profile for Perfluoroalkyl Compounds" for public review and comment in May 2009 (available at http://www.atsdr.cdc.gov/toxprofiles/tp200.html#bookmark04). The Toxicological Profile summarizes the available scientific literature on the toxicology of PFCs , as does a summary report by Lau et al. (2007). Both of these references provide a more detailed summary of the scientific literature on PFCs than is possible or appropriate for this report.

### Summary of Toxicological Information

PFOS is well absorbed orally, but is not absorbed well through inhalation or dermal contact (OECD, 2002). Half-lives of PFOS have been estimated at over 100 days in rats in a single-dose study, and 200 days in a sub-chronic dosing study in cynomolgus monkeys (OECD, 2002). Animal studies have shown that, in their pure form, PFOA and APFO (its ammonium salt) are easily absorbed through ingestion, inhalation, and dermal contact (EPA 2002; Kennedy 1985; Kennedy et al., 1986; Kudo and Kawashima, 2003) although there may be differences between animals and humans and this pathway may not be relevant for environmental (i.e. lower concentration) exposures. The estimated half-life of PFOA in animals ranges from four hours in female rats and nine days in male rats to 20 days in male cynomolgus monkeys (Kudo and Kawashima, 2003; Butenhoff et al., 2004). The mean blood serum half-life of PFOA in humans was estimated to be 3.8 years in a published study of 26 retired 3M workers, and the mean serum half-life of PFOS was estimated at 5.4 years (Olsen et al., 2007a). The mean serum half-life of PFHxS has been reported as 8.5 years. This indicates that some PFCs are retained in the human body for a much longer period than in mice, rats, or monkeys, and that carbon chain length is not necessarily directly related to half-life in humans. PFCs are not metabolized, and are excreted in the urine and feces at different rates in various test animal species and humans.

Exposure to high levels of PFOA, PFOS, and PFBA is acutely toxic in test animals (Kudo and Kawashima, 2003; OECD, 2002; Takagi et al., 1991). Chronic or sub-chronic exposure to lower doses of PFOA in rats typically results in reductions in body weight and weight gain, and in liver effects such as an increase in liver weight and alterations in lipid metabolism (Kudo and Kawashima, 2003). Immune system effects have also been reported in mice exposed to high doses of PFOA (DeWitt et al., 2008). The liver appears to be the primary target organ of PFOA toxicity in rats, although effects on the kidneys, pancreas, testes, and ovaries have also been observed (EPA, 2002). Exposure to PFOA (and other PFCs) in rats results in a process in the liver known as activation of the peroxisome proliferator activated receptor alpha (PPARα). This phenomenon is considered to be limited to rats and similar test animals, and is not observed in primates. Some of the adverse liver effects observed in rats such as an increase in liver weight are in part attributed to peroxisome proliferation. Adverse liver effects in primates are likely the result of a different mode of action.

Chronic exposure to PFOS at high doses has been shown to result in liver toxicity and mortality, with a steep dose-response curve for mortality in rats and primates (OECD 2002). Indications of toxicity observed in 90-day rat studies include increases in liver enzymes and other adverse liver effects, gastrointestinal effects, blood abnormalities, weight loss, convulsions, and death. Immunotoxicity has also been reported in studies conducted in mice at relatively low doses (Peden-Adams et al., 2008). 3M has suggested that these effects have not been reproduced by other researchers, and may be related to liver toxicity (3M, 2010a).

Some long-term animal studies suggest that exposure to PFOA could increase the risk of tumors of the liver, pancreas, and testes (Kudo and Kawashima, 2003; EPA, 2002). The mechanism of potential tumor formation is unclear, but evidence suggests that the tumors are the result of promotion (via oxidative stress, cell death, or hormone-mediated mechanisms) and not from direct damage to the genetic material within cells (genotoxicity). The tumors observed in rats may be a result of peroxisome proliferation, and may not be of relevance in humans (Kennedy et al., 2004).

Various reproductive studies of rats followed for two generations showed postnatal deaths and other developmental effects in offspring of female rats exposed to PFOS and APFO (EPA, 2002; OECD, 2002). These studies demonstrate that exposure to APFO/PFOA and PFOS in sufficient doses can result in adverse effects on the offspring of rats exposed while pregnant.

PFBA has not been studied as extensively as PFOA or PFOS, and until 2008 MDH lacked necessary information to derive a HBV for it. Like other PFCs, PFBA has been demonstrated to cause peroxisome proliferation in the livers of rats exposed through their diet or by intraperitoneal injection (Ikeda et al., 1985; Takagi et al., 1991). The effects of treatment with PFBA were less severe than was observed with PFOA in these two studies. Similar effects have been seen in mouse studies (Permadi et al., 1992). In a similar study comparing the effects of PFOA and PFBA on rat livers, Just et al. (1989) found that the effects of treatment with PFBA were similar to that of PFOA for some parameters measured in the study.

A key question MDH considered in the development of the 2008 HBV for PFBA was its half-life in animals and humans. Chang et al. (2008) summarized data from a study of the pharmacokinetics of PFBA in several animal species. The study showed that PFBA was eliminated quickly through urine in male and female rats, with a half-life of approximately 8 hours in male rats and less than two hours in female rats. The half-life in monkeys was less than two days. In study published in 2008, the mean half-life of PFBA in four male employees at the 3M-Cottage Grove plant and seven male and three female employees at the 3M-Cordova, Illinois plant was calculated to be 72 hours in males and 87 hours in females (Chang et al., 2008).

A 28-day oral toxicity study of PFBA in rats (Lieder et al., 2007) showed that male rats exposed to PFBA had increased liver weights and decreased cholesterol, and other minor effects that went away once the exposure was stopped. In this study, some rats were also exposed separately to PFOA as a positive control. The main differences between male rats given PFBA and PFOA were that PFBA treated rats did not have lower body weights, but did have lower cholesterol. PFOA exposed rats did have a reduction in body weight, exhibited less physical activity and overall health, and had slight reductions in parameters related to red blood cells.

The findings of a developmental study of PFBA in mice conducted at the EPA laboratory in North Carolina was also reviewed by MDH (Das et al., 2007). In the study, exposure to PFBA by pregnant mice did not appear to significantly affect maternal weight gain or fertility. Some developmental delays were observed in the offspring of the mice, and developmental effects were considered a co-critical effect along with liver, blood and thyroid effects in establishing the HBV for PFBA.

No animal studies regarding exposure to multiple PFCs at the same time have been located in the scientific literature.

The current MDH HRLs for PFOS and PFOA are based on toxicological studies conducted on cynomolgus monkeys. In the case of PFOS, the key study (Thomford, 2002) was used by MDH to derive a toxicity value (known as a reference dose, or RfD) of 0.00008 milligrams PFOS per kilogram of body weight perday (mg/kg-d). The RfD included a human equivalent dose adjustment to account for the long half-life in humans (5.4 years), as well as a total uncertainty factor of 30 (a factor of 10 to account for variability in sensitivity between humans and a factor of 3 to account for uncertainty regarding sensitivity between laboratory animals and humans). The critical effects used to determine the RfD were a decrease in serum high-density lipoprotein (i.e., HDL) and changes in thyroid hormones.

For PFOA, the key study (Butenhoff et al., 2002) was used to derive an RfD of 0.000077 mg/kg-d. The RfD for PFOA included a human equivalent dose adjustment to account for the long half-life in humans (3.8 years), as well as a total uncertainty factor of 30. The critical effect used to determine the RfD was an increase in relative liver weight.

The 2008 HBV for PFBA is based on toxicological studies conducted on rats. Several different HBVs based on different exposure periods (short-term, sub-chronic, and chronic) were derived based on more recent MDH practices. The lowest value, which in the case of PFBA is the short-term value, became the final HBV for all three exposure periods. For this value, a short-term RfD of 0.0038 mg/kg-d was derived from the key study (Butenhoff, 2007), which included a 'dose metric adjustment' of eight due to the much shorter mean half-life of PFBA in humans (3 days) versus rats (9.22 hours). The total uncertainty factor was 100 (to account for variability in sensitivity between humans, uncertainty regarding sensitivity between laboratory animals and humans, and database insufficiencies).

Summary of Human Exposure Information

PFCs, primarily PFOS and PFOA, have been detected in the blood of U.S. citizens in multiple studies (Olsen et al., 2003, 2004a, and 2004b). PFCs have also been shown to cross the placenta. In a study of fifteen pairs of maternal and cord blood samples in Japan, Inoue et al. (2004) detected PFOS in the cord blood samples at approximately one-third the concentration in maternal blood. PFOA was detected in maternal blood, but not in cord blood. A similar study of 11 paired maternal and cord blood samples collected in Germany showed PFOS in cord blood at approximately 60% of the maternal blood concentration (Midasch et al..,2007). This study did detect low levels of PFOA (median of 3.4 ppb) in cord blood samples, slightly above that found in the maternal blood samples. A larger study conducted in the city of Baltimore measured ten PFCs in the cord serum of 299 newborns (Apelberg et al., 2007a). PFOS and PFOA were detected in nearly all of the samples, at a geometric mean level of 4.9 and 1.6 ppb, respectively. Other PFCs were detected much less frequently, and at lower levels. In a follow-up study (Apelberg, 2007b), a small (sub-clinical) negative association between both PFOA and PFOS cord serum concentration and birth weight and size was observed. A similar study conducted in Denmark showed an inverse correlation between maternal serum PFOA levels and birth weight, but no statistical association with PFOS (Fei et al., 2007). These (and other) studies appear to offer inconsistent findings of a potential reduction in birth weight associated with increased PFOA and PFOS levels in serum (Steenland et al., 2010).

The most comprehensive data on PFC levels in the blood of the U.S. population has been produced by the Centers for Disease Control and Prevention's (CDC) National Health and Nutrition Examination Survey (NHANES) as reported by Calafat et al. (2007a and 2007b). Blood serum data (PFOS, PFOA, PFHxS) have been reported for several thousand people, age 12 and above, for the survey years 1999-2000, and 2003-2004, as shown in the table below.

**Geometric mean data for PFCs, U.S. population, in ppb**

| **Survey Year** | **PFOS** | **PFOA** | **PFHxS** |
|---|---|---|---|
| 1999-2000 | 30.4 | 5.2 | 2.1 |
| 2003-2004 | 20.7 | 3.9 | 1.9 |
| NOTES: PFCs = perfluorinated chemicals<br>ppb = parts per billion | | | |

The data suggest that PFC levels in the blood serum of the general population are declining, perhaps due to a reduction in the use of PFCs in consumer products. Many other PFCs were included in the NHANES sample analysis, but data are not presented here because the PFCs were below detection limits, or only sporadically detected. PFBA was not included in the NHANES analyses. Other studies of serum levels in Red Cross adult blood donors have shown similar declines in PFC concentrations (Olsen et al., 2008).

Information on PFC levels in Washington County residents exposed to PFCs through drinking water has been collected through a biomonitoring pilot program created and funded by the Minnesota Legislature in 2007 (MDH, 2009). For the pilot study, health scientists interviewed and obtained blood samples from 196 randomly selected adult participants in order to measure the levels of seven PFCs in their blood. One half of the participants' homes were served by private wells in Lake Elmo and Cottage Grove and the other half were served by the Oakdale municipal water system. The private wells had to have at least trace levels (0.1 ppb or greater) of PFOA or PFOS for the residents to be eligible for the study.

PFOS, PFOA, and PFHxS were detected in all 196 participants of the study. The geometric mean results for the 196 participants were as follows:

**Geometric mean data for PFCs, MDH Biomonitoring Pilot Study, in ppb**

| **Survey Year** | **PFOS** | **PFOA** | **PFHxS** |
|---|---|---|---|
| 2008-2009 | 35.9 | 15.4 | 8.4 |
| NOTES: PFCs = perfluorinated chemicals<br>ppb = parts per billion | | | |

PFBA was detected in 55 (28%) of the 196 serum samples collected from the project population (PFBA level of detection (LOD) was 0.1 ppb), with a maximum detected value of 8.5 ppb. PFBS was detected in 5 (3%) of the 196 serum samples collected from the population (PFBS LOD was 0.1 ppb). With so many of the samples measuring below the level of detection, an accurate geometric mean or other measure of central tendency for PFBS could not be calculated. PFPeA and PFHxA were not detected in any of the 196 serum samples collected.

Overall, the pilot biomonitoring showed that the levels of three PFCs were higher in east metro residents than in the U.S. population as a whole. There was little difference between the Oakdale and Lake Elmo/Cottage Grove participants. Individual results were mailed to those participants who indicated they wished to receive them. Participants also received other helpful information about national findings for PFCs in people's blood, PFCs in general, and ways to reduce exposure to PFCs. MDH has initiated a follow-up biomonitoring study to determine if blood levels of PFCs have fallen, now that exposures through drinking water in these populations have been reduced or ended for the most part.

The largest investigation of human exposure to PFOA is taking place in the Ohio-West Virginia area, and grew out of a court settlement of a class action lawsuit against the DuPont Corporation in 2005. This investigation is known as the C8 Health Project, and information on it can be found on the project's web site, http://www.hsc.wvu.edu/som/cmed/c8/. The project has enrolled 69,030 people who may have been exposed to PFOA through drinking water. The participants have been tested for PFOA (and some other PFC) exposure through analysis of blood samples. The project will also involve ten separate studies to help determine whether PFOA exposure is associated with any observable human health effects. Eight of the studies will focus on diseases such as cancer, heart disease, stroke, diabetes, immune function, liver and hormone disorders, and birth outcomes. Two studies will look at exposure to PFOA and its half-life in the general population. The studies are estimated to be complete in several years, although some preliminary results have been posted on the study's web site. Details of the studies and results can be found on the C8 Science Panel web site at http://www.c8sciencepanel.org/index.html. Recently, a summary of the epidemiological studies conducted to date was published by the C8 Health Project directors (Steenland et al., 2010). Associations have been observed between PFOA levels in blood and other parameters such as uric acid in adults, serum cholesterol levels, and delayed onset of puberty. Firm conclusions regarding cause and effect have not yet been drawn, however, and any associations must be interpreted with caution.

The State of West Virginia examined cancer rates in three counties near the West Virginia DuPont PFOA plant (Colsher et al., 2005). The study found that some tumors that may be associated with PFOA exposure in animal studies were elevated in some parts of the study area, but that the reported cancers could not be directly related to PFOA exposure through drinking water. Also, other chemical exposures were known to exist in the area.

Discussion of the Public Health Implications of PFC Exposure

Blood levels of PFCs observed in the east metro area are well below levels of departure in animals (23 parts per million (ppm) for PFOA, 35 ppm for PFOS) used to calculate MDH HRLs (MDH, 2007a and 2007b). The level of departure is the serum level in an animal at which critical adverse health effects are observed. Blood levels are also well below levels measured in 3M-Cottage Grove plant workers (PFOA, geometric mean 850 ppb; PFOS, geometric mean 440 ppb; 3M, 2003c). Past studies of 3M workers have not observed reproducible or consistent health effects (i.e. Alexander et al., 2003); a more recent study by Lundin et al (2009) did suggest positive associations between PFOA exposure and prostate cancer, cerebrovascular disease, and diabetes in some 3M-Cottage Grove workers. MDH is closely following the ongoing 3M workforce studies, as well as the West Virginia study, which will provide more definitive information about the health implications of PFC exposure in the east metro area.

On January 8, 2009, the U.S. EPA's Office of Water issued Provisional Health Advisories for PFOS and PFOA of 0.2 and 0.4 ppb, respectively (EPA, 2009). This action was taken in response to a situation in Alabama where it was found that sludge from a wastewater treatment plant that received PFC contaminated industrial wastewater had been land-applied. The sludge is believed to be responsible for low levels of PFOA found in nearby community water systems.

The 2009 EPA Provisional Health Advisory values are very close to MDH's HRLs for PFOS and PFOA, but were calculated in a slightly different way. MDH has determined that if the EPA values were applied in situations where drinking water is contaminated with PFOS or PFOA, no additional well advisories or other health-protection actions would be necessary.

MDH's health-based exposure limits are protective for all segments of the population, including vulnerable sub-populations. Nevertheless, those who may be especially concerned with their continued exposure to low levels of PFCs through drinking water (even at levels below MDH HRLs or HBVs), such as pregnant women or parents with infants, can take additional steps to reduce exposure by using bottled water for drinking, cooking, or making formula, or by using point of use filters to treat water used for these purposes. MDH recently completed a study of the effectiveness of point of use water treatment devices for PFCs which demonstrated that reverse-osmosis and activated carbon filters work well under both laboratory and real-world conditions (MDH, 2008b).

Health Outcome Data Review

On June 7, 2007 the Minnesota Cancer Surveillance System (MCSS), located within the Chronic Disease and Environmental Epidemiology Section of MDH issued a report presenting detailed profiles of cancer rates among residents of Dakota and Washington Counties (MDH, 2007c). Using MCSS data for the 15-year period 1988-2002, county-wide cancer rates for all cancers combined and for each of about 25 of the most frequent types of cancer, including liver and thyroid cancer were examined. In addition, analyses were also conducted to examine incidence rates for 16 selected cancers for specific communities, by zip code, within each county. For that analysis, data from the years

1996-2004 were used, largely due to population growth in some communities and limitations on community census data.

The report (which can be accessed at the MDH web site, www.health.state.mn.us/) found that overall cancer rates in Washington and Dakota counties are very similar to the rest of the state, or slightly lower. In addition, the rates and types of cancers that occurred within specific communities in the two counties were generally similar with other communities in the Twin Cities metropolitan area.

Analyses of community cancer rates are rarely useful for evaluating potential cancer risks from low levels of environmental pollutants. Nevertheless, such data can be helpful in addressing public concerns over cancer rates in a county or a community. The reader is referred to the full report for a more detailed description of the benefits and limitations of the analysis.

## Child Health Considerations

MDH recognizes that the unique vulnerabilities of infants and children are of special concern to communities faced with contamination of their water, soil, air, or food. Children are at a greater risk than adults are from certain kinds of exposures to environmental contaminants at waste disposal sites. They are more likely to be exposed because they often play outdoors and bring food into contaminated areas. Children are smaller than adults and so receive higher doses of chemical exposure per body weight. The developing body systems of children can sustain permanent damage if toxic exposures occur during critical growth stages. Most importantly, children depend completely on adults for risk-identification and risk-management decisions, housing decisions, and for access to medical care.

Many children have been exposed to low levels of PFCs in contaminated drinking water in the affected communities described in this report. MDH health-based exposure limits are calculated with protection of children's health in mind. As stated previously, those who may be especially concerned with children's exposure to low levels of PFCs through drinking water, such as pregnant women or parents with infants, can take additional steps to reduce exposure by using bottled water for drinking, cooking, or making formula, or by using point of use filters to treat water used for these purposes.

## Conclusions

PFC-containing wastes were disposed of by 3M in land disposal sites in Oakdale, Lake Elmo, Woodbury, and Cottage Grove, Minnesota. PFCs were released to groundwater from these sites, possibly shortly after the disposal occurred, resulting in contamination of nearby drinking water wells. The levels of PFCs in drinking water in the past are unknown and in the past exposure could have occurred through drinking water, possible air emissions during the handling, disposal, or burning of waste or direct contact with the waste. A pilot biomonitoring study conducted in Oakdale, Lake Elmo, and Cottage Grove indicated levels of PFCs in resident's blood that are above national averages. While available evidence suggests that these PFC levels are unlikely to cause adverse health effects, there is no information available regarding PFC levels in resident's blood or in drinking water in the past. MDH cannot conclude whether drinking PFCs in water, breathing them in the air, or coming into direct contact with PFC-containing wastes in the past harmed people's health.

Currently, PFCs have been detected in public and private wells across a wide area of south Washington County, and in parts of northern Dakota County and southeastern Ramsey County. In the areas covered by this report, the PFC concentrations are below levels of public health concern in all of the public wells and the vast majority of private wells. Exposure to PFCs at levels above health concern in 24 private wells in south Cottage Grove is currently being addressed by the use of bottled water or whole-house activated carbon filters. Biomonitoring studies indicate these measures have resulted in decreased average blood serum levels of PFOS, PFOA, and PFHxS for these water users. However, such filters require continued monitoring and maintenance to ensure they function properly. For those reasons, filter systems are not considered by MDH to be the best, long-term solution if other sources of water are available. MDH concludes that currently, the levels of PFCs found in drinking water from public or private wells in the area covered by this report are not expected to harm people's health; new data will be evaluated as it becomes available. Remediation actions to address PFCs at the waste disposal sites are underway by 3M and the MPCA.

## Recommendations

1. 3M should continue to follow the requirements of the Consent Order to implement the selected remediation options for soil and groundwater at the 3M-Woodbury Disposal Site.
2. Although remedial actions at the site appear to have eliminated surface soil as an exposure pathway, as a precaution people should avoid trespassing on the 3M-Woodbury Disposal Site and 3M should improve the fencing of the site, particularly along the south boundary, to prevent trespassing.
3. Extension of the Cottage Grove municipal water supply to serve areas where private wells contain levels of PFCs in excess of MDH HRLs or HBVs should be considered.
4. Monitoring of selected private wells in the affected area should continue under agreed upon sampling plans to track changes in the plume and monitor for changes in concentration in individual wells. If the plume remains stable, the frequency and number of wells monitored may be reduced while still providing assurance that public health is protected.
5. MPCA should continue to monitor and maintain whole-house GAC filter systems (or provide bottled water) in homes where the well water exceeds Minnesota drinking water standards.
6. MDH will continue to work with the City of Oakdale and 3M to ensure proper treatment of city wells that exceed Minnesota drinking water standards.

7. As part of the proposed reduction in groundwater pumping at the site, MPCA should evaluate the monitoring data to determine whether the monitoring well network at the 3M-Woodbury Disposal Site provides adequate information regarding water quality in the “high transmissivity zone” of the Prairie du Chien aquifer.

## Public Health Action Plan

The MDH Public Health Action Plan for the site includes the following: 1) distribution of this public health assessment (and/or an information sheet summarizing the information contained in this public health assessment) to area residents; 2) continued consultation with the MPCA, 3M, Washington County, and the affected communities on implementing investigation and response-action activities and the recommendations provided in the *Recommendations* section of this document; 3) continued outreach to private-well owners; 4) continued monitoring of public water supplies; 5) organization and participation in public meetings and meetings with local government officials as needed, 6) evaluation of other potential pathways for environmental exposure to PFCs, and additional biomonitoring studies to evaluate PFC levels in area residents exposed to PFOA and PFOS in drinking water.

## References


3M, 2003a. Environmental and Health Assessment of Perfluorooctane Sulfonate Acid and its Salts. 3M Company, St. Paul, Minnesota. August 20, 2003.

3M, 2003b. Letter from Michael A. Santoro, 3M, and George H. Millet, 3M, to the U.S. EPA Office of Pollution Prevention and Toxics. August 1, 2003.

3M, 2003c. Assessment of Lipid, Hepatic, and Thyroid Function in Relation to an Occupational Biologic Limit Value for Perfluorooctanoate. Medical Department, 3M Company, St. Paul, Minnesota 55144. June 9, 2003.

3M, 2008. Letter from Katie Winogrodzki, 3M, to Gerald Stahnke, MPCA. September 19, 2008.

3M, 2010a. Comments of 3M Company on Minnesota Department of Health's Draft Public Health Assessment on Perfluorochemicals in Southern Washington County, Northern Dakota County, and Southeast Ramsey County, Minnesota. October 12, 2010.

3M, 2010b. 3M Woodbury Site: Request to Reduce Barrier Well Pumping Rates. Letter to Mr. Douglas Wetzstein, MPCA, dated March 16, 2010 from Ms. Katie Winogrodzki, 3M.

Alexander, B.H., Olsen, G.W., Burris, J.M., Mandel, J.H., and Mandel, J.S. Mortality of employees of a perfluorooctanesulphonyl fluoride manufacturing facility. Occupational and Environmental Medicine 60: 722–729.

Alexander, C., 2007. Fractured sandstone karst aquifers, the St. Peter, Jordan and Hinckley Formations: Examples for Askov, Woodbury, Rochester and Elsewhere. Presented at the Minnesota Groundwater Association spring conference, April 19, 2007.

Apelberg, B.J., Goldman, L.R., Calafat, A.M., Herbstman, J.B., Kuklenyik, Z., Heidler, J., Needham, L.L., Halden, R.U., and Witter, F.R., 2007a. Determinants of fetal exposure to polyfluoroalkyl compounds in Baltimore, Maryland. Environmental Science and Technology 41: 3891-3897.

Apelberg, B.J., Witter, F.R., Herbstman, J.B., Calafat, A.M., Halden, R.U., Needham, L.L., and Goldman, L.R., 2007b. Cord serum concentrations of perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA) in relation to weight and size at birth. Environmental Health Perspectives 115: 1670-1676.

ATSDR, 2009. Draft Toxicological Profile for Perfluoroalkyl Acids. Agency for Toxic Substances and Disease Registry, Atlanta GA. May 2009.

Barr, 2003. Cottage Grove Area Nitrate Study Report. Prepared by Barr Engineering for Washington County. October 2003.

Barr, 2005. Former Oakdale Disposal Site/Washington County Landfill Groundwater Modeling. Prepared by Barr Engineering for the Minnesota Pollution Control Agency. November 2005.

Barr, 2005b. Integrating Groundwater & Surface Water Management – Southern Washington County. A joint report by Barr Engineering Co., Washington Conservation District, and Washington County Department of Public Health & Environment. August 2005.

Brezinski, D., 2003. Laying the Foundation for New Technologies: 3M Creates a new building block for its fluorosurfactants. Paint & Coatings Industry, January 2003.

Butenhoff, J., Costa G, Elcombe C, Farrar D, Hansen K, Iwai H, Jung R, Kennedy G Jr, Lieder P, Olsen G, and Thomford P., 2002. Toxicity of Ammonium Perfluorooctanoate in Male Cynomolgus Monkeys After Oral Dosing for 6 Months. Toxicological Sciences 69: 244-257.

Butenhoff J.L., Kennedy G.L., Hinderliter P.M., Lieder P.H., Jung R., Hansen K.J., Gorman G.S., Noker P.E., and Thomford P.J., 2004. Pharmacokinetics of perfluorooctanoate in cynomolgus monkeys. Toxicological Sciences 82: 394-406.

Butenhoff, J.L., Olsen, G.W., and Pfahles-Hutchens, A., 2006. The applicability of biomonitoring data for perfluorooctanesulfonate to the environmental public health continuum. Environmental Health Perspectives 114: 1776-1782.

Butenhoff, JL., 2007. E-mail correspondence conveying benchmark dose calculations conducted by 3M for liver weight and cholesterol – 28 day PFBA study. February 6, 2007.

Calafat, A.M., Kuklenyik, Z., Reidy, J.A., Caudill, S., Tully, J.S., and Needham, L.L., 2007a. Serum concentrations of 11 polyfluoroalkyl compounds in the U.S. population: data from the National Health and Nutrition Examination Survey (NHANES) 1999-2000. Environmental Science and Technology 41: 2237-2242.

Calafat, A.M., Wong, L-Y., Kuklenyik, Z., Reidy, J.A., and Needham, L.L., 2007b. Polyfluoroalkyl chemicals in the U.S. population: data from the National Health and Nutrition Examination Survey (NHANES) 2003-2004 and comparisons to NHANES 1999-2000. Environmental Health Perspectives 115: 1596-1602.

Chang, S., Hart, J., Ehresman, D., Das, K., Lau, C., Noker, P., Gorman, G., Tan, Y., and Butenhoff, J., 2007. The pharmacokinetics of perfluorobutyrate (PFBA) in rats, mice, and monkeys. The Toxicologist, Supplement to Toxicological Sciences 96: abstract 937.

Chang, S., Das, K., Ehresman, D.J., Ellefson, M.E., Gorman, G.S., Hart, J.A., Nokers, P.E., Tan, Y.M., Lieder, P.H., Lau, C., Olsen, G.W., and Butenhoff, J.L., 2008. Comparative pharmacokinetics of perfluorobutyrate in rats, mice, monkeys, and humans and relevance to human exposure via drinking water. Toxicological Sciences 104: 40-53.

Colsher, P., Ducatman, A., and Haddy, L., 2005. Examination of community concerns about water contamination with perfluorooctanoic acid and related chemicals using population-based cancer registry data. West Virginia Department of Health and Human Services, Charleston, W.V.

Conder, J.M., Hoke, R.A., De Wolf, W., Russell, M.H., and Buck, R.C., 2008. Are PFCAs bioaccumulative? A critical review and comparison with regulatory criteria and persistent lipophilic compounds. Environmental Science and Technology 42: 995-1003.

Das, K.P., Grey, B., Butenhoff, J., Tanaka, S., Ehresman, D., Zehr, D., Wood, C., and Lau, C., 2007. Effects of perfluorobutyrate exposure in mice during pregnancy. Toxicological Sciences, advance published May 28, 2008.

De Silva, A.O., and Mabury, S.A., 2006. Isomer distribution of perfluorocarboxylates in human blood: potential correlation to source. Environmental Science and Technology 40: 2903-2909.

DeWitt, J.C., Copeland, C.B., Strynar, M.J., and Luebke, R.W., 2008. Perfluorooctanoic acid-induced immunomodulation in adult C57BL/6J or C57BL/6N female mice. Environmental Health Perspectives 116: 644-650.

Delta, 2008. Perfluorocarbon (PFC)-Containing Firefighting Foams and Their Use in Firefighting Training in Minnesota. Prepared for Minnesota Pollution Control Agency. June 30, 2008.

Emmett, E.A., Shofer, F.S., Zhang, H., Freeman, D., Desai, C., and Shaw, L.M., 2006a. Community exposure to perfluorooctanoate: relationships between serum concentrations and exposure sources. Journal of Occupational and Environmental Medicine 48: 759-770.

Emmett, E.A., Zhang, H., Shofer, F.S., Freeman, D., Rodway, N.V., Desai, C., and Shaw, L.M., 2006b. Community exposure to perfluorooctanoate: relationships between serum levels and certain health parameters. Journal of Occupational and Environmental Medicine 48: 771-779.

EPA, 2002. Revised Draft Hazard Assessment of Perflourooctanoic Acid and its Salts. U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics. November 4, 2002.

EPA, 2003. Preliminary Risk Assessment of the Developmental Toxicity Associated with Exposure to Perflourooctanoic Acid and its Salts. U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics. April 10, 2003.

EPA, 2006. 2010/15 PFOA Stewardship Program. U.S. Environmental Protection Agency, Washington, D.C. Accessed May 23, 2007 at http://www.epa.gov/opptintr/pfoa/pubs/pfoastewardship.htm

EPA, 2009. Provisional Health Advisories for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS). U.S. Environmental Protection Agency, Office of Water, Washington, D.C. January 8, 2009.

Ericson, I., Marti-Cid, R., Nadal, M., Van Bavel, B., Lindstrom, G., and Domingo, J.L., 2008. Human exposure to perfluorinated chemicals through the diet: intake of perfluorinated compounds in foods from the Catalan (Spain) market. Journal of Agricultural and Food Chemistry 56: 1787-1794.

Fei, C., McLaughlin, J.K., Tarone, R.E., and Olsen, J., 2007. Perfluorinated chemicals and fetal growth: a study within the Danish national birth cohort. Environmental Health Perspectives 115: 1677-1682.

Flury, M. and Wai, N.N., 2003. Dyes as tracers for vadose zone hydrology. Review of Geophysics 41: 1002-1005.

Higgins, C.P., and Luthy R.G., 2006. Sorption of perfluorinated surfactants on sediments. Environmental Science and Technology 40: 7251-7256.

Holzer, J., Midasch, O., Rauchfuss, K., Kraft, M., Reupert, R., Angerer, J., Kleeschulte, P., Marschall, N., and Wilhelm, M., 2008. Biomonitoring of perfluorinated compounds in children and adults exposed to perfluorooctanoate-contaminated drinking water. Environmental Health Perspectives 116: 651-657.

Ikeda, T., Aiba, K., Fukuda, K., and Tanaka, M., 1985. The induction of peroxisome proliferation in rat liver by perfluorinated fatty acids, metabolically inert derivatives of fatty acids. Journal of Biochemistry 98: 475-482.

Inoue, K., Okada, F., Ito, R., Kato, S., Sasaki, S., Nakajima, S., Uno, A., Saijo, Y., Sata, F., Yoshimura, Y., Kishi, R., and Nakazawa, H., 2004. Perfluorooctane sulfonate (PFOS) and related perfluorinated compounds in human maternal and cord blood samples: assessment of PFOA exposure in susceptible population during pregnancy. Environmental Health Perspectives 112: 1204-1207.

Joyce, M., Dinglasan-Panlilio, A., and Mabury, S.A., 2006. Significant residual fluorinated alcohols present in various fluorinated materials. Environmental Science and Technology 40: 1447-1453.

Just, W.W., Gorgas, K., Hartl, F.U., Heinemann, P., Salzer, M., and Schmissek, H., 1989. Biochemical effects and zonal heterogeneity of peroxisome proliferation induced by perfluorocarboxylic acids in rat liver. Hepatology 9: 570-581.

Kanivetsky R. and Cleland, J. M., 1990. Hydrogeology, in: Geologic Atlas of Washington County, Minnesota. Minnesota Geological Survey; County Atlas Series, Atlas C-5, Plate 5.

Kärrman, A., Ericson, I., van Bavel, B., Darnerud, P.O., Aune, M., Glynn, A., Lignell, S., and Lindstrom, G., 2007. Exposure of perfluorinated chemicals through lactation: levels in matched human milk and serum and a temporal trend, 1996-2004, in Sweden. Environmental Health Perspectives 115: 226-230.

Kauck, E.A. and Diesslin, A.R., 1951. Some properties of perfluorocarboxylic acids. Industrial and Engineering Chemistry 43: 2332-2334.

Kennedy, G.L., 1985. Dermal toxicity of ammonium perfluorooctanoate. Toxicology and Applied Pharmacology 81: 348-355.

Kennedy, G.L., Hall, G.T., Britelli, M.R., Barnes, J.R., and Chen, H.C., 1986. Inhalation toxicity of ammonium perfluorooctanoate. Food Chemistry and Toxicology 24: 1325-1329.

Kennedy, G.L., Butenhoff, J.L., Olsen, G.W., O'Connor, J.C., Seacat, A.M., Perkins, R.G., Biegel, L.B., Murphy, S.R., and Farrar, D.G., 2004. The toxicology of perfluorooctanoate. Critical Reviews in Toxicology 34: 351-384.

Kubwabo, C., Stewart, B., Zhu, J., and Marro, L., 2005. Occurrence of perfluorosulfonates and other perfluorochemicals in dust from selected homes in the city of Ottawa, Canada. Journal of Environmental Monitoring 7: 1074-1078.

Kudo, N. and Kawashima, Y., 2003. Toxicity and toxicokinetics of perfluorooctanoic acid in humans and animals. The Journal of Toxicological Sciences 28: 49-57.

Kwan, W.C., 2001. Physical Property Determination of Perfluorinated Surfactants (doctoral thesis). University of Toronto, Graduate Department of Chemistry.

Lau, C., Anitole, K., Hodes, C., Lai, D., Pfahles-Hutchens, A., and Seed, J., 2007. Perfluoroalkyl acids: a review of monitoring and toxicological findings. Toxicological Sciences 99: 366-394.

Lieder, P.H., Tanaka, S., Ehresman, D.J., Roy, R.R., Otterdijk, F., and Butenhoff, J.L., 2007. A 28-day oral (gavage) toxicity study of ammonium perfluorobutyrate (APFB). . The Toxicologist, Supplement to Toxicological Sciences 96: abstract 931.

Lindholm, G.F., Helgesen, J.O., Broussard, W.L., and Farrell, D.F., 1974. Water Resources of the Lower St. Croix River Watershed, East-Central Minnesota. Hydrologic Investigations Atlas HA-490; U.S. Geological Survey, Reston, VA.

Loewen, M., Halldorson, T., Wang, F., and Tomy, G., 2005. Fluorotelomer carboxylic acids and PFOS in rainwater from an urban center in Canada. Environmental Science and Technology 39: 2944-2951.

Lundin, J.I., Alexander, B.H., Olsen, G.W., and Church, T. R., 2009. Ammonium perfluorooctanoate production and occupational mortality. Epidemiology 20: 921-928.

MDH, 2002. Nitrate exposure and infant risk study (NEXIR). Accessed online at http://www.health.state.mn.us/divs/eh/hazardous/topics/nexirsummary2003.pdf .

MDH, 2005. Health Consultation on Perfluorochemical Releases at the 3M Cottage Grove Facility. Minnesota Department of Health, St. Paul, Minnesota, February 18, 2005.

MDH, 2007a. Health Based Values for Perfluorooctanoic Acid. Memorandum from Helen Goeden, Health Risk Assessment Unit to John Stine, Environmental Health Division Director. Minnesota Department of Health, St. Paul, Minnesota, February 26, 2007.

MDH, 2007b. Health Based Values for Perfluorooctane Sulfonate. Memorandum from Helen Goeden, Health Risk Assessment Unit to John Stine, Environmental Health Division Director. Minnesota Department of Health, St. Paul, Minnesota, February 26, 2007.

MDH, 2007c. Cancer Incidence in Dakota and Washington Counties. MCSS Epidemiology Report 2007:1. Minnesota Cancer Surveillance System, Chronic Disease and Environmental Epidemiology Section, Minnesota Department of Health, St. Paul, Minnesota. June 7, 2007.

MDH, 2007d. Health Consultation on Nitrite Generation in a Granular Activated Carbon Filter – Baytown Township Groundwater Contamination Site, Washington County, Minnesota, EPA Facility ID: MND982425209. Minnesota Department of Health, St. Paul, Minnesota, November 9, 2007.

MDH, 2008a. Public Health Assessment on Perfluorochemical Contamination in Lake Elmo and Oakdale, Washington County, Minnesota. Minnesota Department of Health, St. Paul, Minnesota, August 29, 2008.

MDH, 2008b. Performance Evaluation, Removal of Perfluorochemicals (PFCs) with Point-of-Use (POU) Water Treatment Devices. Minnesota Department of Health, St. Paul, Minnesota, May 1, 2008.

MDH, 2009. East Metro Perfluorochemical Biomonitoring Pilot Project Report. Minnesota Department of Health, St. Paul, Minnesota, July 21, 2009. Online at: http://www.health.state.mn.us/divs/hpcd/tracking/biomonitoring/projects/pfcfinalrpt2009.pdf

MDH, 2011. East Metro PFC Biomonitoring Follow-up Project: December 2011 Report to the Community. Minnesota Department of Health, St. Paul, Minnesota. Online at: www.health.state.mn.us/divs/hpcd/tracking/biomonitoring/projects/pfcfollowuprpt2011.pdf

MGS, 1990. Geologic Atlas of Washington County, Minnesota. County Atlas Series, Atlas C-5. Minnesota Geological Survey, University of Minnesota. 7 plates.

Midasch, O., Drexler, H., Hart, N., Beckmann, M.W., and Angerer, J., 2007. Transplacental exposure of neonates to perfluorooctanesulfonate and perfluorooctanoate: a pilot study. International Archives of Occupational and Environmental Health, DOI 10.1007/s00420-006-0165-9.

Minnesota Department of Administration, 2009. Annual estimates of city and township population, households and persons per household, 2000 to 2008. Minnesota State Demographic Center. Accessed September 1, 2009 online at http://www.demography.state.mn.us/

Moriwaki, H., Takata, Y., and Arakawa, R., 2003. Concentrations of perlfuorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) in vacuum cleaner dust collected in Japanese homes. Journal of Environmental Monitoring 5: 753-757.

MPCA, 2007a. Soil Reference Values (SRVs) for PFOA and PFOS. Memorandum from Emily Hansen, MPH to Kathryn Sather, May 8, 2007.

MPCA, 2007b. Surface Water Quality Criteria for Perfluorooctane Sulfonic Acid and Perfluorooctanoic Acid. August 2007. Accessed online at: http://www.pca.state.mn.us/hot/pfc.html#pfos

MPCA, 2007c. Request for Issuance of a Request for Response Action (RFRA) to the 3M Company: For the Release and Threatened Release of Hazardous Substances from the 3M Chemolite Disposal Site, 3M Oakdale Disposal Site, and 3M Woodbury Site. Minnesota Pollution Control Agency, St. Paul, Minnesota. April 13, 2007. Accessed online at http://www.pca.state.mn.us/index.php/view-document.html?gid=10835

MPCA, 2007d. Proposed Consent Order on PFCs: A Summary of the Negotiations. Minnesota Pollution Control Agency, St. Paul, Minnesota. Accessed online at http://www.pca.state.mn.us/publications/pfc-consentorderfactsheet.pdf.

MPCA, 2007e. Settlement Agreement and Consent Order with 3M. Minnesota Pollution Control Agency, St. Paul, Minnesota. Accessed online at http://www.pca.state.mn.us/publications/pfc-3mchemolite-consent.pdf

MPCA, 2010a. 3M Woodbury Disposal Site – Request to Reduce Barrier Well Pumping. Letter from Ms. Kathryn Sather, MPCA, dated June 30, 2010, to Mr. Gary Hohenstein, 3M.

MPCA, 2010b. MPCA Comments on MDH Draft Public Health Assessment – 3M Woodbury Disposal Site. December 6, 2010.

OECD, 2002. Hazard Assessment of Perfluorooctane Sulfonate (PFOS) and its Salts. Organization for Economic Cooperation and Development. November 21, 2002.

Ohmori, K., Kudo, N., Katayama, K., and Kawashima, Y., 2003. Comparison of the toxicokinetics between perfluorocarboxylic acids with different carbon chain length. Toxicology 84: 135-140.

Olsen, G.W., Church, T.R., Miller, J.P., Burris, J.M., Hansen, K.J., Lundberg, J.K., Armitage, J.B., Herron, R.M., Medhdizadehkashi, Z., Nobiletti, J.B., O'Neill, E.M., Mandel, J.H., and Zobel, L.R., 2003. Perfluorooctanesulfonate and other fluorochemicals in the serum of American Red Cross adult blood donors. Environmental Health Perspectives 111: 1892-1901.

Olsen, G.W., Church, T.R., Hansen, K.J., Burris, J.M., Butenhoff, J.L., Mandel, J.H., and Zobel, L.R., 2004a. Quantitative evaluation of perfluorooctanesulfonate (PFOS) and other fluorochemicals in the serum of children. Journal of Children's Health 2: 53-76.

Olsen, G.W., Church, T.R., Larson, E.B., van Belle, G., Lundberg, J.K., Hansen, K.J., Burris, J.M., Mandel, J.H., and Zobel, L.R., 2004b. Serum concentrations of perfluorooctanesulfonate and other fluorochemicals in an elderly population from Seattle, Washington. Chemosphere 54: 1599-1611.

Olsen, G.W., Burris, J.M., Ehresman, D.J., Froehlich, J.W., Seacat, A.M., Butenhoff, J.L., and Zobel, L.R., 2007a. Half-life of serum elimination of perfluorooctanesulfonate, perfluorohexanesulfonate, and perfluorooctanoate in retired fluorochemical production workers. Environmental Health Perspectives 115: 1298-1305.

Olsen, G.W., Mair, D.C., Reagan, W.K., Ellefson, M.E., Ehresman, D.J., Butenhoff, J.L., and Zobel, L., 2007b. Preliminary evidence of a decline in perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA) concentrations in American Red Cross blood donors. Chemosphere 68: 105-111.

Olsen, G.W., and Zobel, L.R., 2007. Assessment of lipid, hepatic, and thyroid parameters with serum perfluorooctanoate (PFOA) concentrations in fluorochemical production workers. International Archives of Occupational and Environmental Health 81: 231-246.

Olsen, G.W., Mair, D.C., Church, T.R., Ellefson, M.E., Reagen, W.K., Boyd, T.M., Herron, R.M., Medhdizadehkashi, Z., Nobiletti, J.B., Rios, J.A., Butenhoff, J.L., and Zobel, L., 2008. Decline in perfluorooctanesulfonate (PFOS) and other perfluoroalkyl chemicals in American Red Cross adult blood donors, 2000-2006. Environmental Science and Technology 42: 4989-4995.

Peden-Adams, M.M., Keller, J.M., EuDaly, J.G., Berger, J., Gilkeson, G.S., and Keil, D.E., 2008. Suppression of humoral immunity in mice following exposure to perfluorooctane sulfonate (PFOS). Toxicological Sciences, advance published March 20, 2008.

Permadi, H., Lundgren, B., Andersson, K., and DePierre, J.W., 1992. Effects of perfluoro fatty acids on xenobiotic-metabolizing enzymes, enzymes which detoxify reactive forms of oxygen and lipid peroxidation in mouse liver. Biochemical Pharmacology 44: 1183-1191.

Prevedouros, K., Cousins, I.T., Buck, R.C., and Korzeniowski, S.H., 2006. Sources, fate and transport of perfluorocarboxylates. Environmental Science and Technology 40: 32-44.

Reid, T.S., Codding, D.W., and Bovey, F.A., 1955. Vinyl esters of perfluoro acids. Journal of Polymer Science 18: 417-421.

Runkel, A.C., Tipping, R.G., Alexander, E.C., Jr., Green, J.A., Mossler, J.H., and Alexander, S.C., 2003, Hydrogeology of the Paleozoic bedrock in southeastern Minnesota: Minnesota Geological Survey Report of Investigations 61, 105 p., 2 pls.

Runkel, A.C., Mossler. J.H., and Tipping, R.G., 2007. The Lake Elmo downhole logging project: hydrostratigraphic characterization of fractured bedrock at a perfluorochemical contamination site. Minnesota Geological Survey, St. Paul, Minnesota. November 1, 2007.

Shapiro, A.M., 2008. Groundwater Flow and Chemical Transport in Fractured Rocks; USGS webinar, November 18, 2008.

Sieben, K., 2010. Letter from State Senator Katie Sieben to the Site Assessment and Consultation Unit, Minnesota Department of Health. September 29, 2010.

So, M.K., Tamashita, N., Taniyasu, S., Jiang, Q., Giesy, J.P., Chen, K., and Lam, P.K.S., 2006. Health risks in infants associated with exposure to perfluorinated compounds in human breast milk from Zhoushan, China. Environmental Science and Technology 40: 2924-2929.

Steenland, K., Fletcher, T., and Savitz, D.A., 2010. Epidemiologic evidence on the health effects of perfluorooctanoic acid (PFOA). Environmental Health Perspectives 118: 1100 – 1108.

Strynar, M.J. and Lindstrom, A.B., 2008. Perfluorinated compounds in house dust from Ohio and North Carolina, USA. Environmental Science and Technology 42: 3751-3756.

STS, 2007. Surface Water Quality Criterion for Perfluorooctanoic Acid. Prepared by STS Consultants, Inc. for the Minnesota Pollution Control Agency. August 2007. Accessed online at: http://www.pca.state.mn.us/hot/pfc.html#pfos

Takagi, A., Sai, K., Umemura, T., Hasegawa, R., and Kurokawa, Y., 1991. Short-term exposure to the peroxisome proliferators, perfluorooctanoic acid and perfluorodecanoic acid, causes significant increase of 8-hydroxydeoxyguanosine in liver DNA of rats. Cancer Letters 57: 55-60.

Tao, L., Kannan, K., Wong, C.M., Arcaro, K.F., and Butenhoff, J.L., 2008. Perfluorinated compounds in human milk from Massachusetts, U.S.A. Environmental Science and Technology 42: 3096-3101.

Thomford, P., 2002 Final Report: 104 Week Dietary Chronic Toxicity and Carcinogenicity Study with Perfluorooctane Sulfonic Acid Potassium Salt (PFOS: T-6295) in Rats. (Abstract only).
Tipping, RG., Runkel, A.C., Alexander, E.C., Jr., Alexander, S.C., and Green, J.A. (2006) Evidence for hydraulic heterogeneity and anisotropy in the mostly carbonate Prairie du Chien Group, southeastern Minnesota, USA. Sedimentary Geology, 184:305-330.

Tittlemier, S.A., Pepper, K., Seymour, C., Moisey, J., Bronson, R., Cao, X.L., and Dabeka, R.W., 2007. Dietary exposure of Canadians to perfluorinated carboxylates and perfluorooctane sulfonate via consumption of meat, fish, fast foods, and food items prepared in their packaging. Journal of Agricultural and Food Chemistry 55: 3203-3210.

UK Food Standards Agency, 2006. Flourinated chemicals: UK dietary intakes. Food Standards Agency, Chemical Safety Division. London, United Kingdom, November 2006.

Weston, 2005. Fluorochemical (FC) Site Related Environmental Assessment Program, 3M Cottage Grove, Minnesota Facility. Weston Solutions, Inc., West Chester, Pennsylvania, July 2005.

Westo,n 2007a. Fluorochemical (FC) Assessment Work Plan for the 3M Woodbury Site. Weston Solutions, Inc., West Chester, Pennsylvania, February 2007.

Weston, 2007b. Hydraulic Evaluation of the Barrier Well Recovery System – Former 3M Woodbury Disposal Site, Woodbury, MN. Weston Solutions, Inc., West Chester, Pennsylvania, September, 2007.

Weston, 2008a. Remedial Investigation / Feasibility Study, 3M Woodbury Disposal Site. Weston Solutions, Inc., West Chester, Pennsylvania, February 2008.

Weston, 2008b. Addendum 2 to the Feasibility Study for the Woodbury Site. Weston Solutions, Inc., West Chester, Pennsylvania, July 2008.

Weston, 2008c. Feasibility Study – Cottage Grove Site. Weston Solutions, Inc., West Chester, Pennsylvania, March 2008.

Weston, 2009. Gables Lake Surface Water Sampling Report, 3M Woodbury, MN Site. Weston Solutions, Inc., West Chester, Pennsylvania, July 2009.

## REPORT PREPARATION

This Public Health Assessment was prepared by the Minnesota Department of Health under a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with the approved agency methods, policies, procedures existing at the date of publication. Editorial review was completed by the cooperative agreement partner. ATSDR has reviewed this document and concurs with its findings based on the information presented.

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## Glossary

**Acute**
Occurring over a short time [compare with chronic].

**Acute exposure**
Contact with a substance that occurs once or for only a short time (up to 14 days) [compare with intermediate duration exposure and chronic exposure].

**Adverse health effect**
A change in body function or cell structure that might lead to disease or health problems.

**Analyte**
A substance measured in the laboratory. A chemical for which a sample (such as water, air, or blood) is tested in a laboratory. For example, if the analyte is mercury, the laboratory test will determine the amount of mercury in the sample.

**Aquifer**
A geologic unit (sediments, rock) in which the pore spaces are fully saturated with groundwater and that can yield water in usable quantities for springs or wells.

**Bedrock valley**
A valley eroded into the bedrock by the action of streams, glaciers, wind, etc. If the valley is subsequently filled with sediment (sand, gravel, silt, or clay), it is referred to as a buried valley or buried bedrock valley.

**Biomonitoring**
Measuring chemical substances in biologic materials (such as blood, hair, urine, or breath) to determine whether exposure has occurred. A blood test for lead is an example of biologic monitoring.

**Biota**
Plants and animals in an environment. Some of these plants and animals might be sources of food, clothing, or medicines for people.

**Buried valley**
A bedrock valley that has been filled with sediment. See "bedrock valley".

**Cancer**
Any one of a group of diseases that occur when cells in the body become abnormal and grow or multiply out of control.

**Cancer risk**
A theoretical risk for getting cancer if exposed to a substance every day for 70 years (a lifetime exposure). The true risk might be lower.

**CERCLA** [see Comprehensive Environmental Response, Compensation, and Liability Act of 1980].

**Chronic**
Occurring over a long time [compare with acute].

**Chronic exposure**
Contact with a substance that occurs over a long time (more than 1 year) [compare with acute exposure and intermediate duration exposure].

**Completed exposure pathway** [see exposure pathway].

**Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA)**
CERCLA, also known as Superfund, is the federal law that concerns the removal or cleanup of hazardous substances in the environment and at hazardous waste sites. ATSDR, which was created by CERCLA, is responsible for assessing health issues and supporting public health activities related to hazardous waste sites or other environmental releases of hazardous substances. This law was later amended by the Superfund Amendments and Reauthorization Act (SARA).

**Concentration**
The amount of a substance present in a certain amount of soil, water, air, food, blood, hair, urine, breath, or any other media.

**Contaminant**
A substance that is either present in an environment where it does not belong or is present at levels that might cause harmful (adverse) health effects.

**Dermal**
Referring to the skin. For example, dermal absorption means passing through the skin.

**Dermal contact**
Contact with (touching) the skin [see route of exposure].

**Detection limit**
The lowest concentration of a chemical that can reliably be distinguished from a zero concentration.

**Dose** (for chemicals that are not radioactive)
The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An "exposure dose" is how much of a substance is encountered in

the environment. An "absorbed dose" is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.

**Dose-response relationship**
The relationship between the amount of exposure [dose] to a substance and the resulting changes in body function or health (response).

**Downgradient**
A location "downstream" relative to groundwater flow directions, or the direction to which groundwater is flowing.

**Environmental media**
Soil, water, air, biota (plants and animals), or any other parts of the environment that can contain contaminants.

**Environmental media and transport mechanism**
Environmental media include water, air, soil, and biota (plants and animals). Transport mechanisms move contaminants from the source to points where human exposure can occur. The environmental media and transport mechanism is the second part of an exposure pathway.

**EPA**
United States Environmental Protection Agency.

**Epidemiology**
The study of the distribution and determinants of disease or health status in a population; the study of the occurrence and causes of health effects in humans.

**Exposure**
Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be short-term [acute], of intermediate duration, or long-term [chronic].

**Exposure investigation**
The collection and analysis of site-specific information and biologic tests (when appropriate) to determine whether people have been exposed to chemical substances.

**Exposure pathway**
The route a substance takes from its source (where it began) to its endpoint (where it ends), and how people can come into contact with (or get exposed to) it. An exposure pathway has five parts: a source of contamination (such as an abandoned business); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing, or touching), and a receptor population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.

**Fault**
In geology, a fault is a planar rock feature which shows evidence of relative movement.

**Feasibility study**
A study to determine the best way to clean up environmental contamination. A number of factors are considered, including health risk, costs, and what methods will work well.

**Groundwater**
Water beneath the earth's surface in the spaces between soil particles and between rock surfaces [compare with surface water].

**Groundwater Divide**
The boundary between to groundwater "basins" where the water on one side flows toward one basin and the water on the other side flows to the other. This is similar in concept to a watershed divide or the continental divide for surface waters.

**Half-life (t½)**
The time it takes for half the original amount of a substance to disappear. In the environment, the half-life is the time it takes for half the original amount of a substance to disappear when it is changed to another chemical by bacteria, fungi, sunlight, or other chemical processes. In the human body, the half-life is the time it takes for half the original amount of the substance to disappear, either by being changed to another substance or by leaving the body. In the case of radioactive material, the half-life is the amount of time necessary for one-half the initial number of radioactive atoms to change or transform into another atom (that is normally not radioactive). After two half-lives, 25% of the original number of radioactive atoms remain.

**Hazard**
A source of potential harm from past, current, or future exposures.

**Health consultation**
A review of available information or collection of new data to respond to a specific health question or request for information about a potential environmental hazard. Health consultations are focused on a specific exposure issue. Health consultations are therefore more limited than a public health assessment, which reviews the exposure potential of each pathway and chemical [compare with public health assessment].

**Health education**
Programs designed with a community to help it know about health risks and how to reduce these risks.

**Health Base Value (HBV)**
An MDH criteria, a HBV is the concentration of a contaminant in water that is considered safe for people if they drink water daily for a lifetime. HBVs have not undergone the state's rule-making process.

**Health Risk Limit (HRL)**
An MDH standard, a HRL is the concentration of a contaminant in water that is considered safe for people if they drink water daily for a lifetime.

**Incidence**
The number of new cases of disease in a defined population over a specific time period [contrast with prevalence].

**Ingestion**
The act of swallowing something through eating, drinking, or mouthing objects. A chemical substance can enter the body this way [see route of exposure].

**Inhalation**
The act of breathing. A chemical substance can enter the body this way [see route of exposure].

**MDH**
The Minnesota Department of Health.

**Medical monitoring**
A set of medical tests and physical exams specifically designed to evaluate whether an individual's exposure could negatively affect that person's health.

**Metabolism**
The conversion or breakdown of a substance from one form to another by a living organism.

**mg/kg**
Milligram per kilogram.

**Migration**
Moving from one location to another.

**Minimal risk level (MRL)**
An ATSDR estimate of daily human exposure to a environmental contaminant at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), noncancerous effects. MRLs are calculated for a route of exposure (inhalation or oral) over a specified time period (acute, intermediate, or chronic). MRLs should not be used as predictors of harmful (adverse) health effects [see reference dose].

**Mortality**
Death. Usually the cause (a specific disease, a condition, or an injury) is stated.

**MPCA**
The Minnesota Pollution Control Agency.

**National Priorities List for Uncontrolled Hazardous Waste Sites (National Priorities List or NPL)**
EPA's list of the most serious uncontrolled or abandoned hazardous waste sites in the United States. The NPL is updated on a regular basis.

**NPL** [see National Priorities List for Uncontrolled Hazardous Waste Sites]

**PFC**
Perfluorochemical, a family of fully fluorinated hydrocarbons.

**Plume**
A volume of a substance that moves from its source to places farther away from the source. Plumes can be described by the volume of air or water they occupy and the direction they move. For example, a plume can be a column of smoke from a chimney or a substance moving with groundwater.

**Point of exposure**
The place where someone can come into contact with a substance present in the environment [see exposure pathway].

**Population**
A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).

**ppb**
Parts per billion.

**ppm**
Parts per million.

**ppt**
Parts per trillion.

**Public comment period**
An opportunity for the public to comment on agency findings or proposed activities contained in draft reports or documents. The public comment period is a limited time period during which comments will be accepted.

**Public health action**
A list of steps to protect public health.

**Public health advisory**
A statement made by ATSDR to EPA or a state regulatory agency that a release of hazardous substances poses an immediate threat to human health. The advisory includes recommended measures to reduce exposure and reduce the threat to human health.

**Public health assessment (PHA)**
An ATSDR document that examines environmental contaminants, health outcomes, and community concerns at a waste site to determine whether people could be harmed from coming into contact with those contaminants. The PHA also lists actions that need to be taken to protect public health [compare with health consultation].

**Public health hazard**
A category used in ATSDR's public health assessments for sites that pose a public health hazard because of long-term exposures (greater than 1 year) to sufficiently high levels of hazardous substances or radionuclides that could result in harmful health effects.

**Public meeting**
A public forum with community members for communication about a site.

**Receptor population**
People who could come into contact with environmental contaminants [see exposure pathway].

**Reference dose (RfD)**
An EPA estimate, with uncertainty or safety factors built in, of the daily lifetime dose of a substance that is unlikely to cause harm in humans.

**Registry**
A systematic collection of information on persons exposed to a specific substance or having specific diseases [see exposure registry and disease registry].

**Remedial investigation**
The CERCLA process of determining the type and extent of hazardous material contamination at a site.

**RfD** [see reference dose]

**Risk**
The probability that something will cause injury or harm.

**Route of exposure**
The way people come into contact with a hazardous substance or environmental contaminant. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].

**Sample**
A portion or piece of a whole. A selected subset of a population or subset of whatever is being studied. For example, in a study of people the sample is a number of people chosen from a larger population [see population]. An environmental sample (for example, a small amount of soil or water) might be collected to measure contamination in the environment at a specific location.

**Saturated thickness**
The vertical thickness of an aquifer in which all of the available pore space is filled with water.

**Solvent**
A liquid capable of dissolving or dispersing another substance (for example, acetone or mineral spirits).

**Source of contamination**
The place where an environmental contaminant comes from, such as a landfill, waste pond, incinerator, storage tank, or drum. A source of contamination is the first part of an exposure pathway.

**Special Well Construction Area (SWCA)**
Minnesota Statutes, section 103I, subdivision 5, clause 7, grants the commissioner of health the authority to establish standards for the construction, maintenance, sealing, and water quality monitoring of wells in areas of known or suspected contamination. Minnesota Rules, part 4725.3650, detail the requirements for construction, repair, or sealing within a designated SWCA, including plan review and approval, water quality monitoring, and other measures to protect public health and prevent degradation of groundwater.

**Stakeholder**
A person, group, or community who has an interest in activities at a waste site.

**Superfund** [see Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) and Superfund Amendments and Reauthorization Act (SARA)

**Superfund Amendments and Reauthorization Act (SARA)**
In 1986, SARA amended the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) and expanded the health-related responsibilities of ATSDR. CERCLA and SARA direct ATSDR to look into the health effects from substance exposures at hazardous waste sites and to perform activities including health education, health studies, surveillance, health consultations, and toxicological profiles.

**Surface water**
Water on the surface of the earth, such as in lakes, rivers, streams, ponds, and springs [compare with groundwater].

**Survey**
A systematic collection of information or data. A survey can be conducted to collect information from a group of people or from the environment. Surveys of a group of people can be conducted by telephone, by mail, or in person. Some surveys are done by interviewing a group of people [see prevalence survey].

**Toxicological profile**
An ATSDR document that examines, summarizes, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.

**Toxicology**
The study of the harmful effects of substances on humans or animals.

**Transmissivity**
In hydraulics, this is a measure of the rate at which water moves through an aquifer or a defined portion of an aquifer.

**Tumor**
An abnormal mass of tissue that results from excessive cell division that is uncontrolled and progressive. Tumors perform no useful body function. Tumors can be either benign (not cancer) or malignant (cancer).

**Uncertainty factor**
Mathematical adjustments for reasons of safety when knowledge is incomplete. For example, factors used in the calculation of doses that are not harmful (adverse) to people. These factors are applied to the lowest-observed-adverse-effect-level (LOAEL) or the no-observed-adverse-effect-level (NOAEL) to derive a minimal risk level (MRL). Uncertainty factors are used to account for variations in people's sensitivity, for differences between animals and humans, and for differences between a LOAEL and a NOAEL. Scientists use uncertainty factors when they have some, but not all, the information from animal or human studies to decide whether an exposure will cause harm to people [also sometimes called a safety factor].

**Upgradient**
A location "upstream" relative to groundwater flow directions, or the direction from which groundwater is flowing.

**Volatile organic compounds (VOCs)**
Organic compounds that evaporate readily into the air. VOCs include substances such as benzene, toluene, methylene chloride, and TCE.

**Water table**
The subsurface layer below which all available pore space is completely saturated with groundwater.

Other glossaries and dictionaries:

U.S. Environmental Protection Agency (http://www.epa.gov/OCEPAterms/)

National Center for Environmental Health/Agency for Toxic Substances and Disease Registry (CDC) (http://www.cdc.gov/nceh/dls/report/glossary.htm)
http://www.cdc.gov/exposurereport/

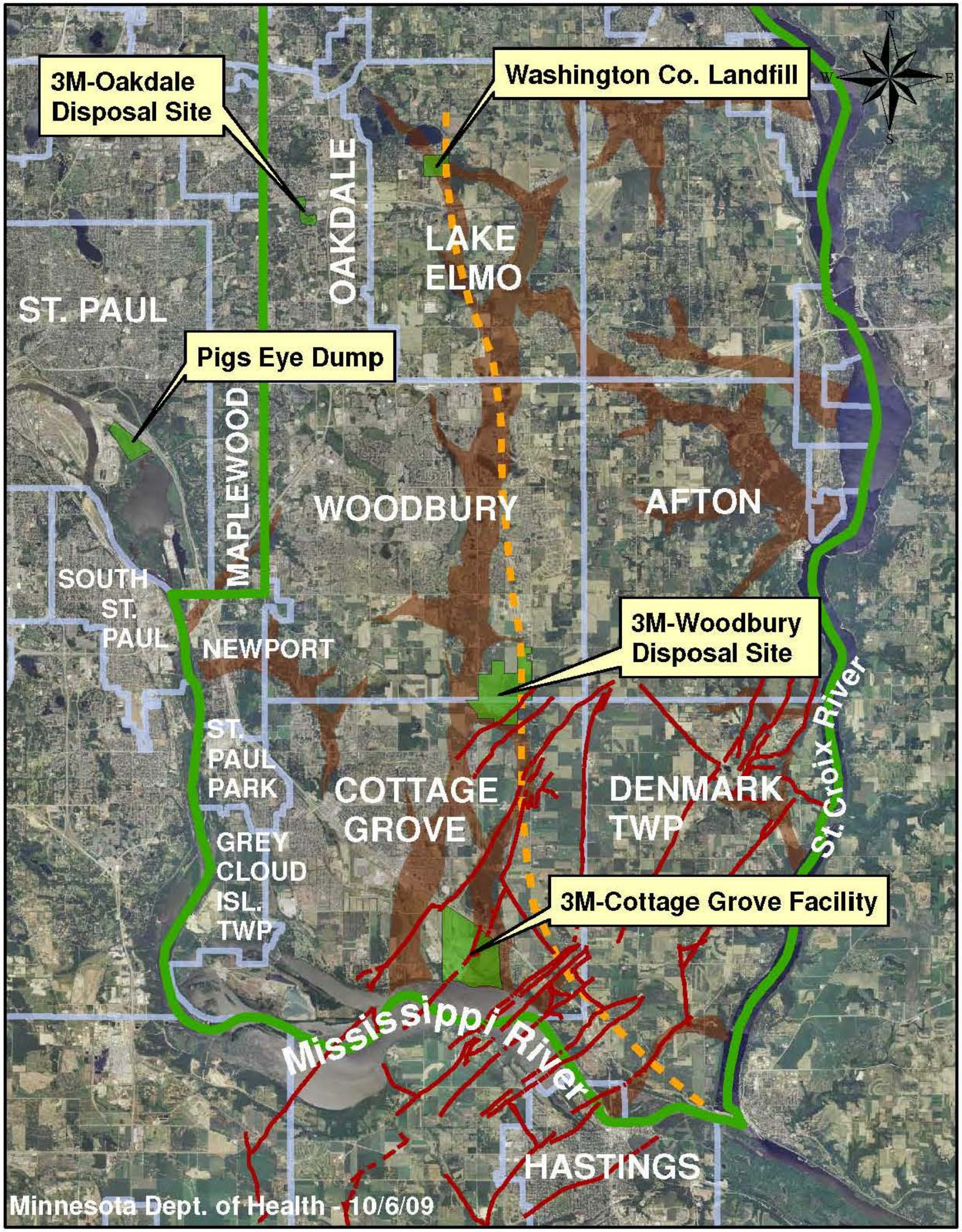
National Library of Medicine (NIH)
(http://www.nlm.nih.gov/medlineplus/mplusdictionary.html)
http://www.nlm.nih.gov/medlineplus/mplusdictionary.html

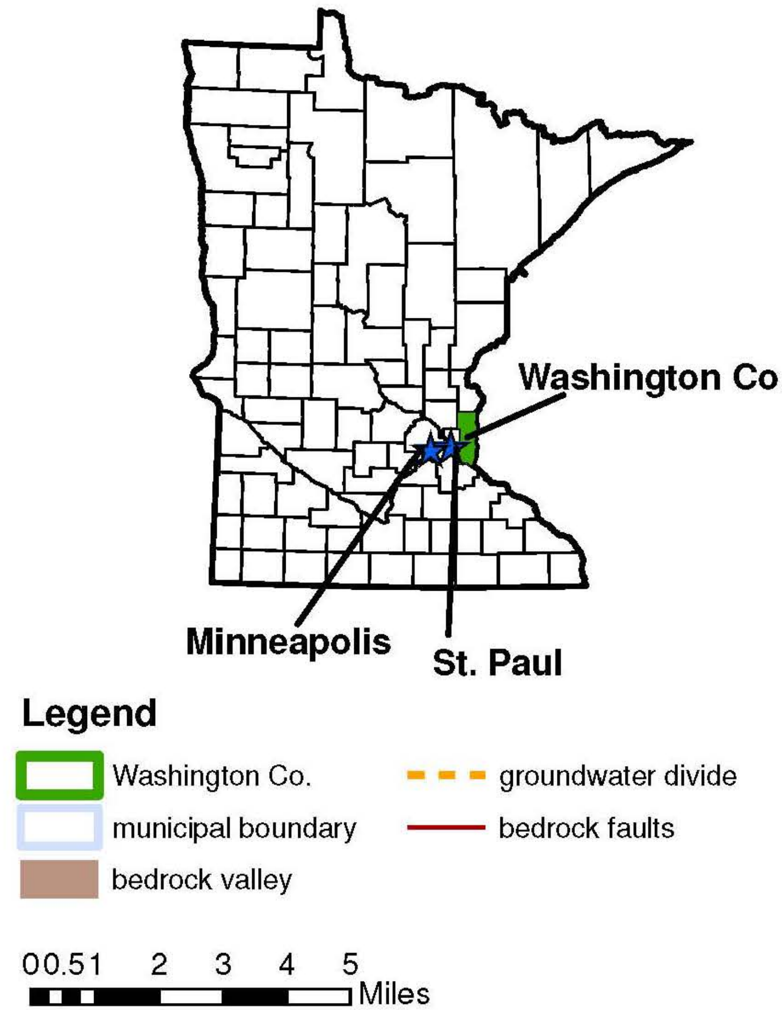
For more information on the work of ATSDR, please contact:

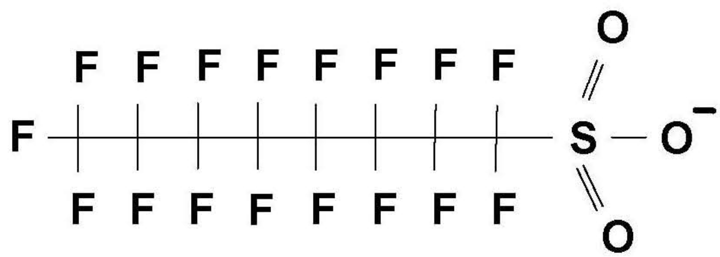
Office of Communications
National Center for Environmental Health/Agency for Toxic Substances and Disease Registry
1600 Clifton Road, N.E. (MS E-29)
Atlanta, GA 30333
Telephone: (404) 498-0080

# Appendix 1: Figures

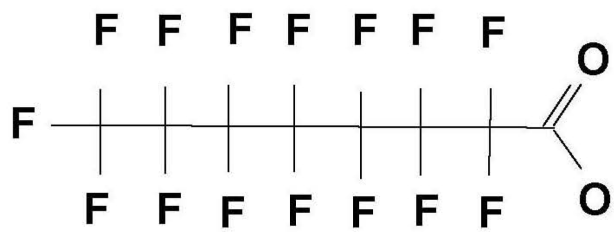
# Figure 1 - Location of 3M Sites and Major GeologicFeatures in Washington Co., MN



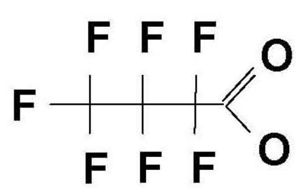





Perfluoro-octane sulfonate (PFOS)

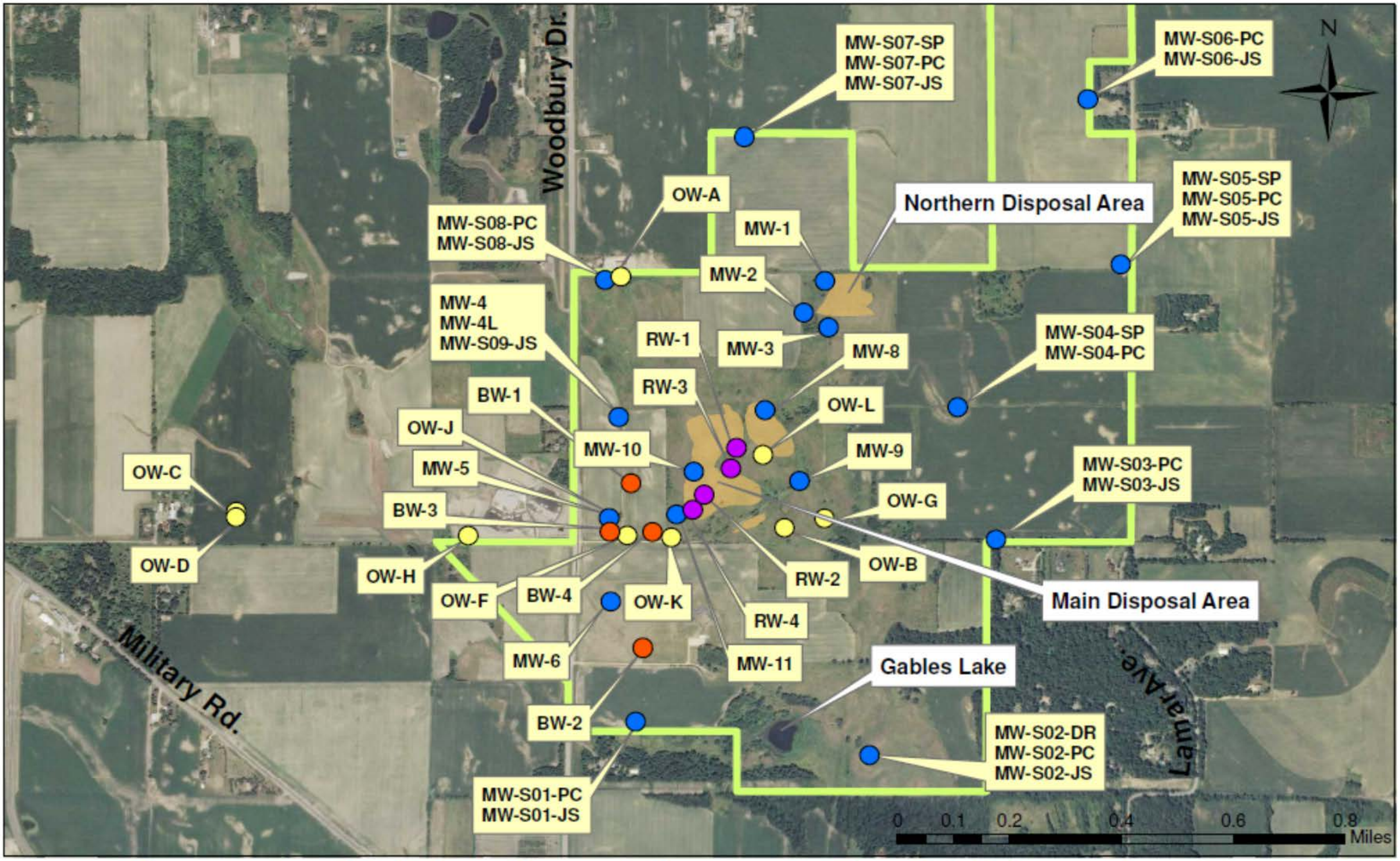


Perfluorooctanoic acid (PFOA)



Perfluorobutanoic acid (PFBA)

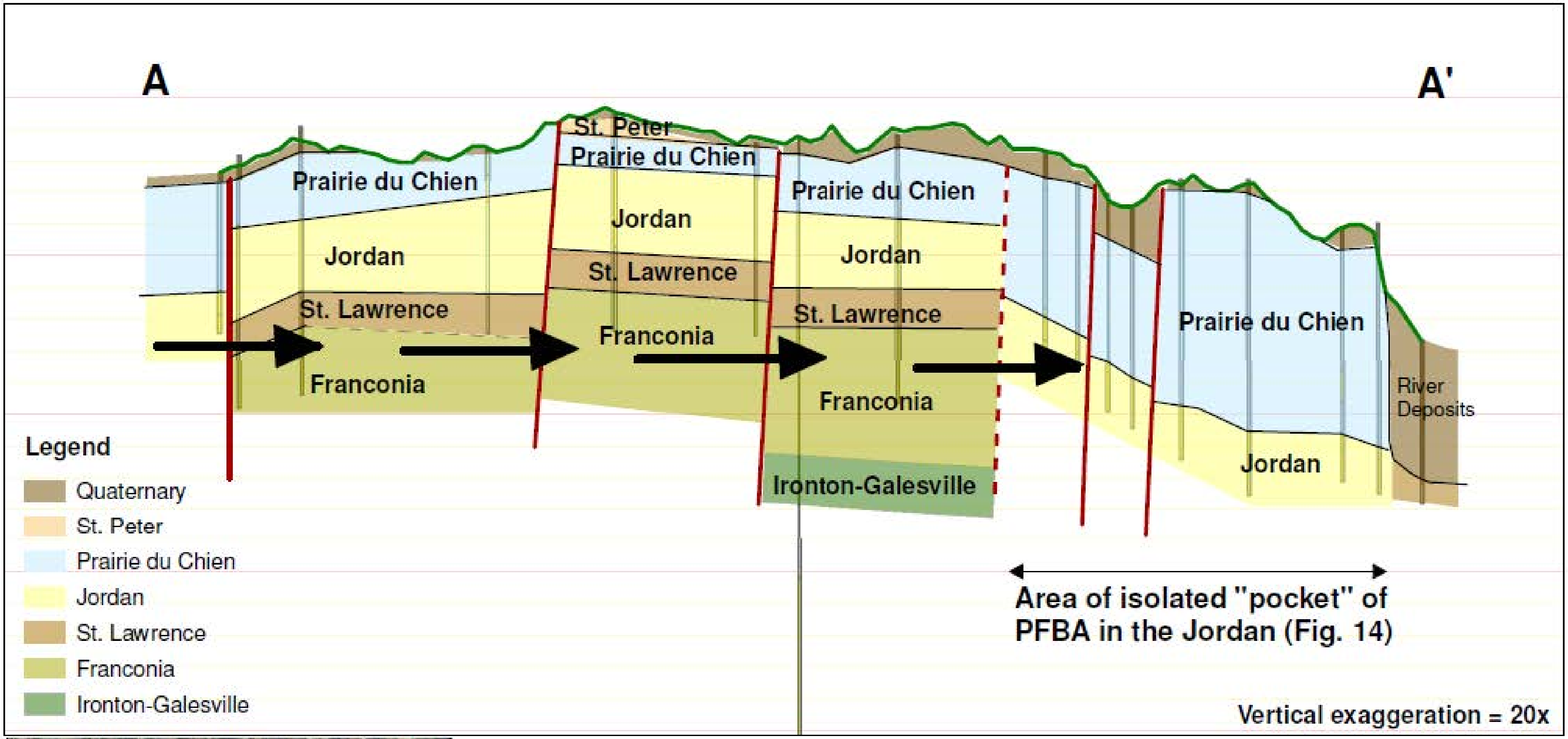
**Figure 2: Chemical structures of the major PFCs detected in the groundwater of southern Washington County, Minnesota.**

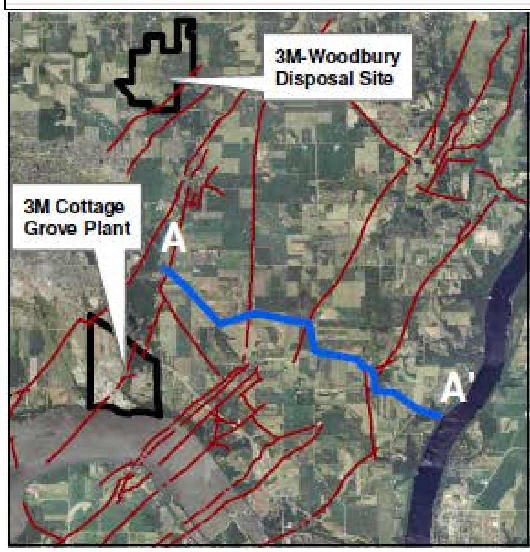



# Figure 3 - 3M-Woodbury Disposal Site Well Location Map

- Monitoring well
- Recovery well
- Observation well
- Barrier well
- 3M-Woodbury Property Boundary
- Approx. location of PFC disposal areas

Minnesota Dept. of Health - 10/6/09

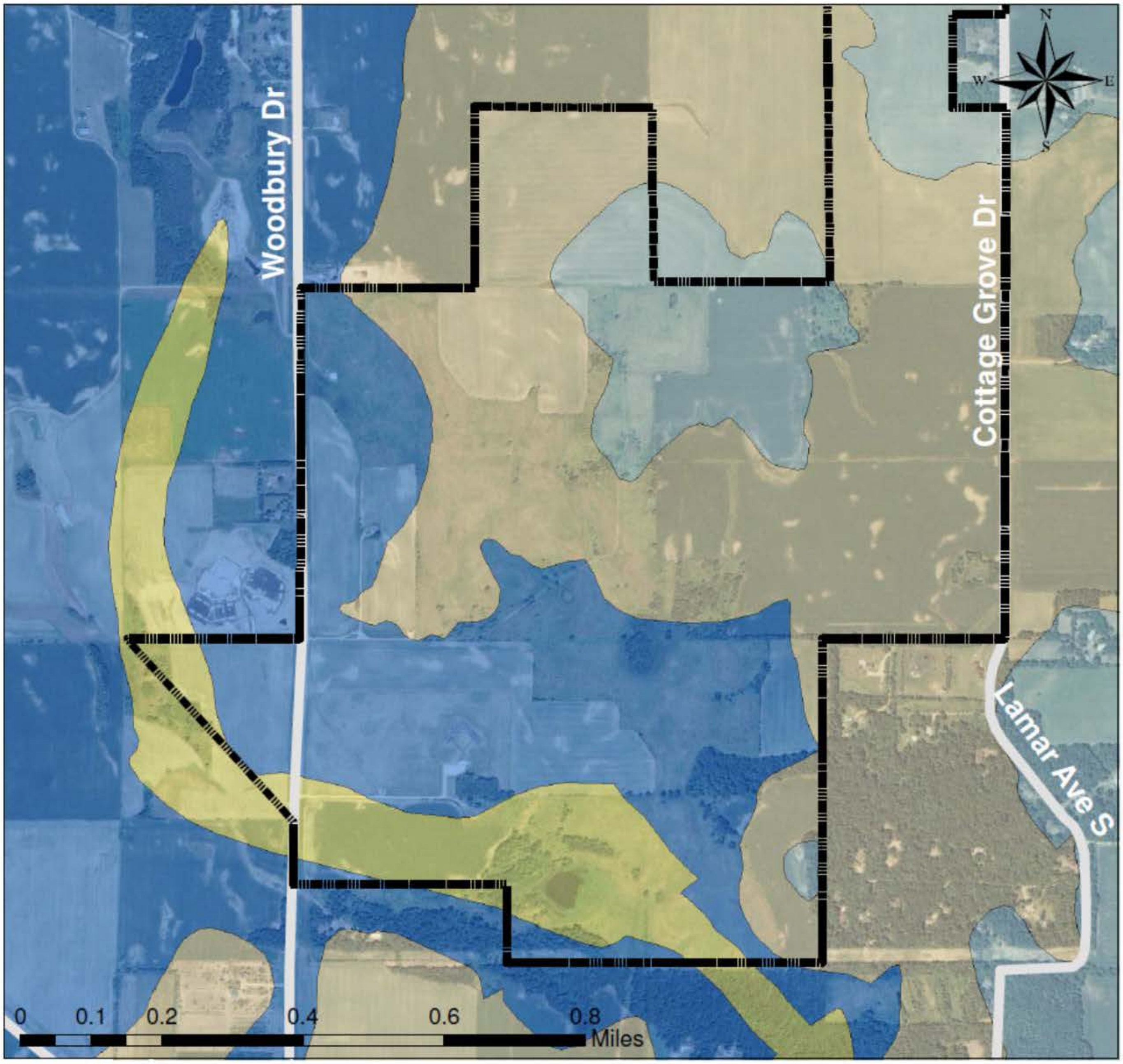



## Figure 4 - Cross-Section Across Highly Faulted Zone

**This figure shows a cross-sectional view of the bedrock along a transect (blue line in map) from Cottage Grove near Ravine Park and trending east-southeast to the St. Croix River. Many bedrock faults (shown in red) have disturbed the geology in this area, bringing different bedrock units into contact with one another. This may have allowed PFBA to move from one aquifer to another, creating what appear to be isolated "pockets" of contamination when viewed on a map (such as Fig. 9). The arrows on the cross-section view show a hypothetical pathway that PFBA may have followed that would allow the contaminant to migrate from the Jordan into the Franconia and back into the Jordan again.**

Prepared by MDH - 8/26/09




# Figure 5 - 3M-Woodbury Disposal Site Bedrock

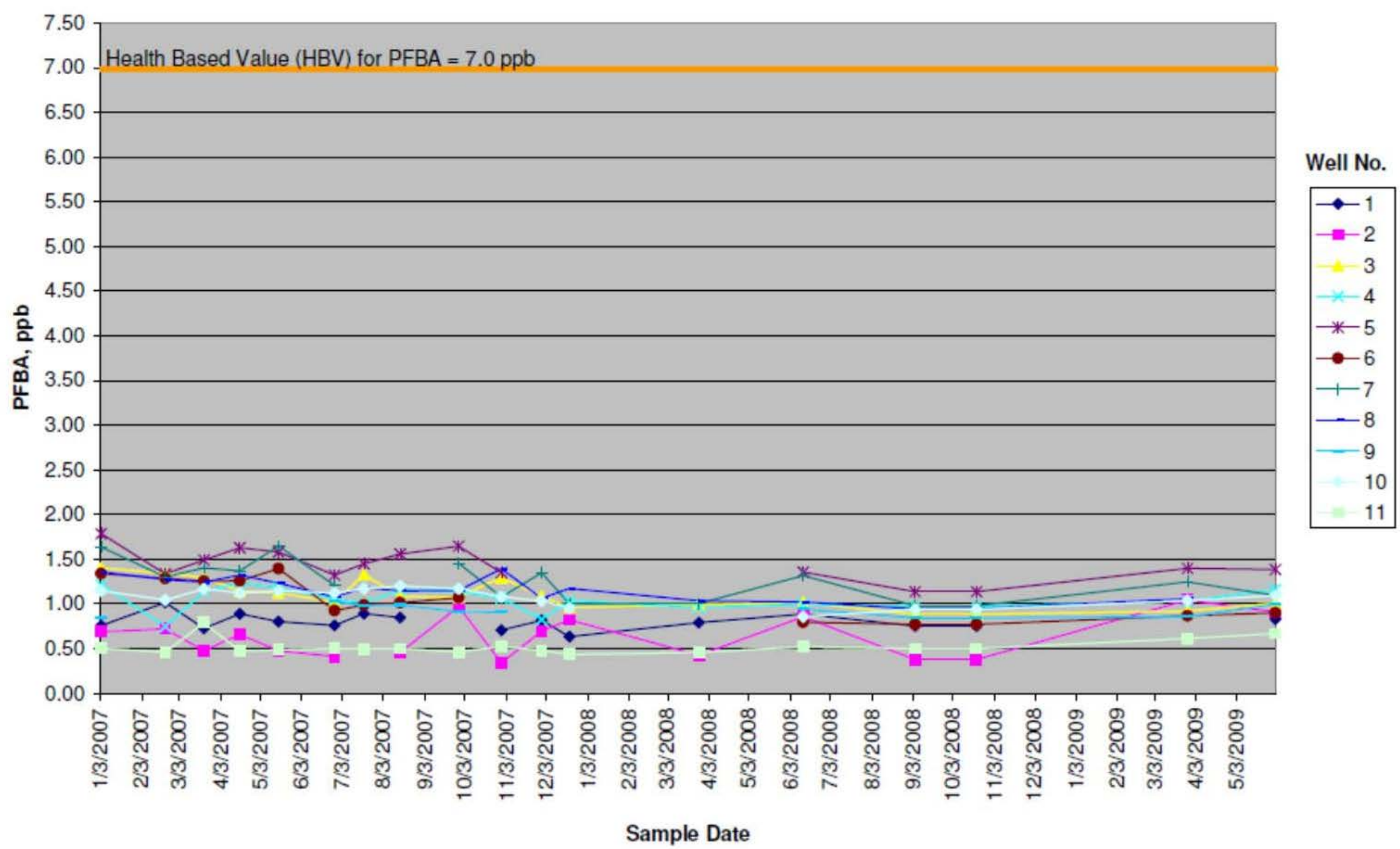
**Legend**

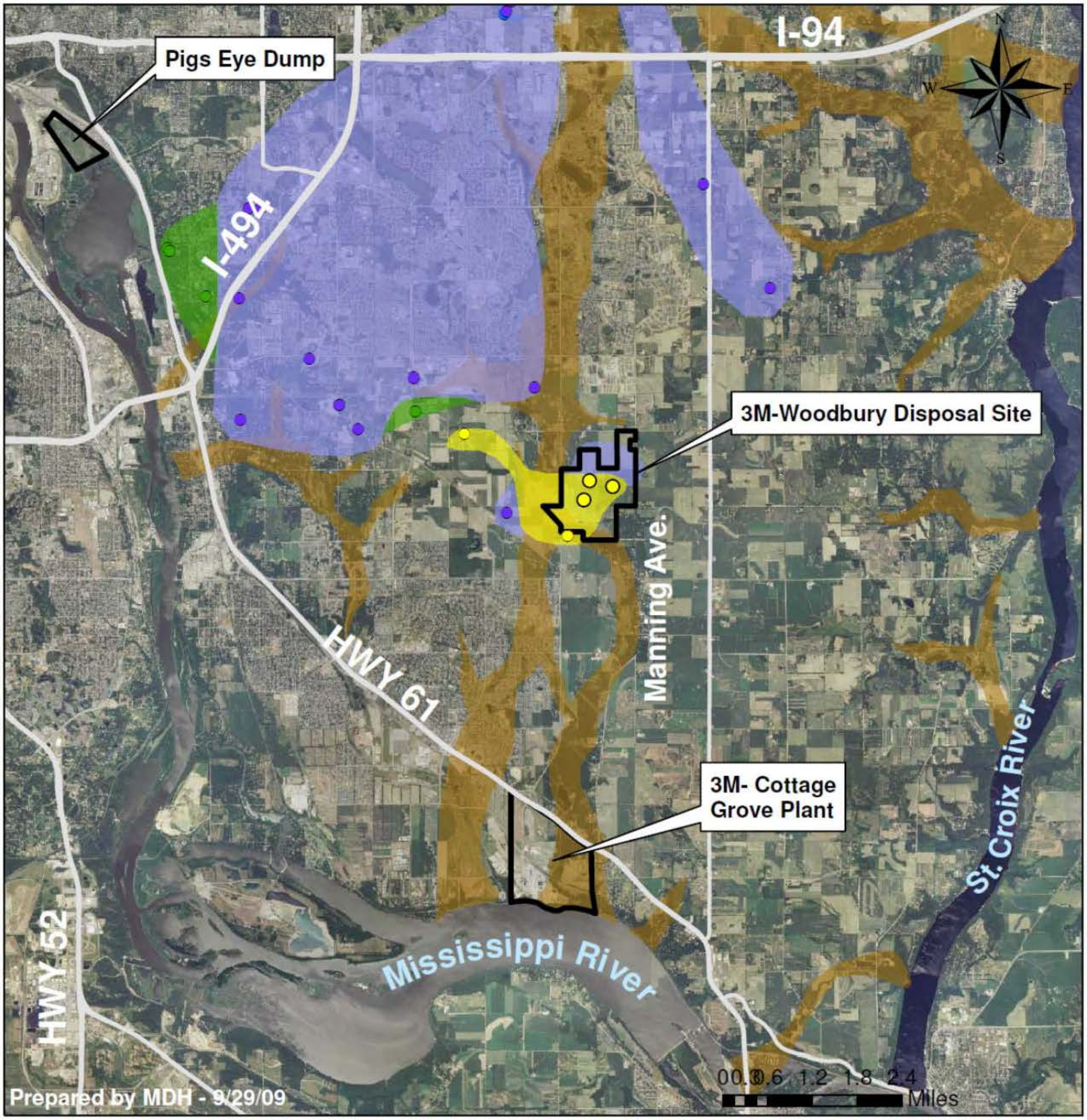
- 3M-Woodbury Disposal Site
- Platteville-Glenwood
- St. Peter
- Prairie du Chien
- Jordan

**This figure shows the uppermost bedrock units that underlie the 3M-Woodbury Disposal Site (i.e. the first bedrock unit beneath the unconsolidated soil, sand, and clay near the surface). Note that from northeast to southwest across the site, successively deeper units are the "top" bedrock layer. The areas where the Prairie du Chien and Jordan are shown coincides with a major buried bedrock valley.**

**Prepared by MDH - 8/5/09**

## Figure 6: PFBA Levels in Cottage Grove Municipal Wells, 2007-2009



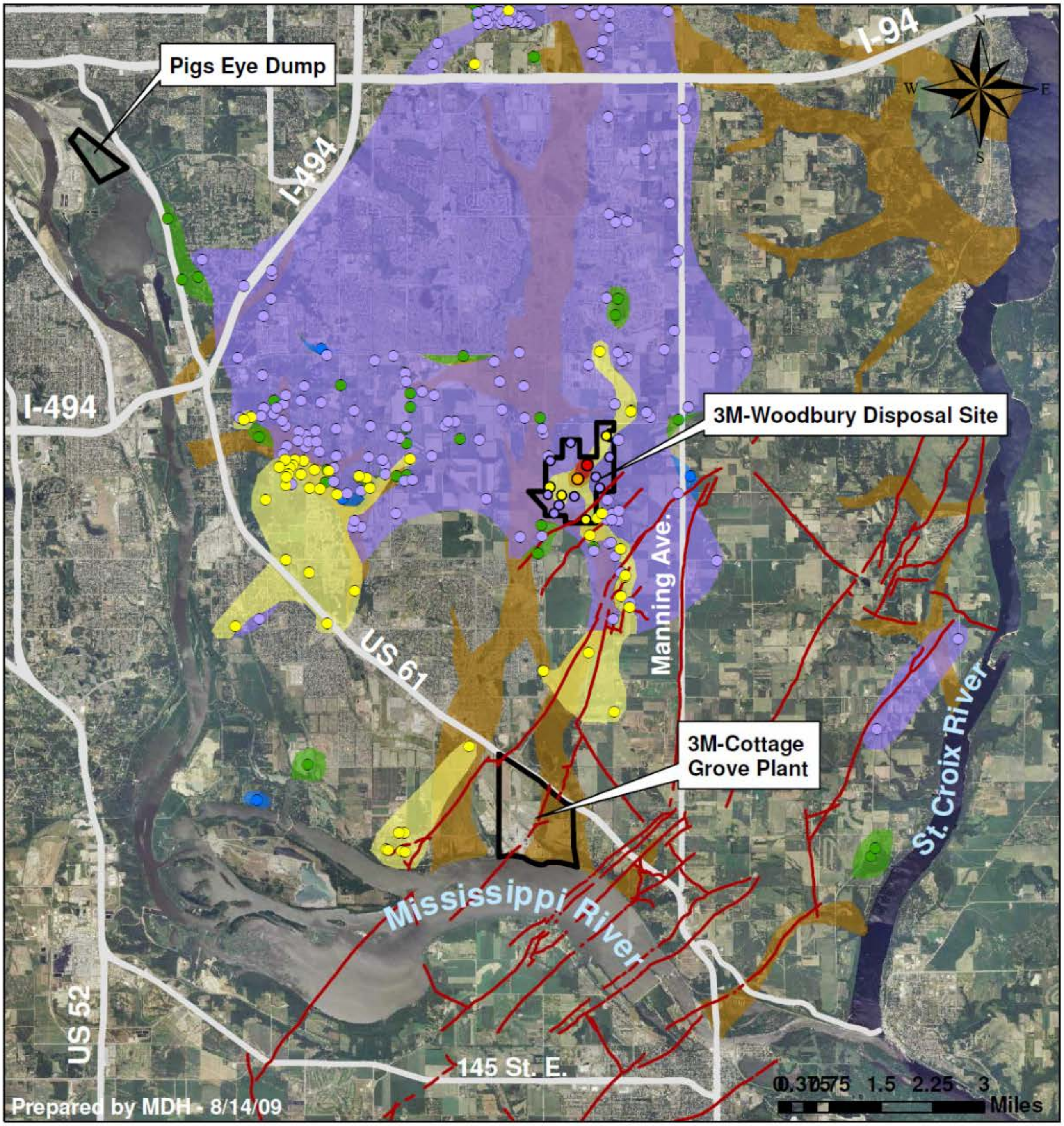



# Figure 7 - PFBA in the St. Peter

**Legend**

| | |
|---|---|
| PFBA > 7 ppb | PFBA = 0.1 - 0.99 ppb |
| PFBA = 3.5 - 7 ppb | PFBA < 0.1 ppb |
| PFBA = 1 - 3.49 ppb | PFBA not detected |
| bedrock valley | sampled well |

**Note: in areas with no color coding, there were no wells in this aquifer to sample and insufficient information to fill in the PFBA concentrations, or the St. Peter is not present in this area.**

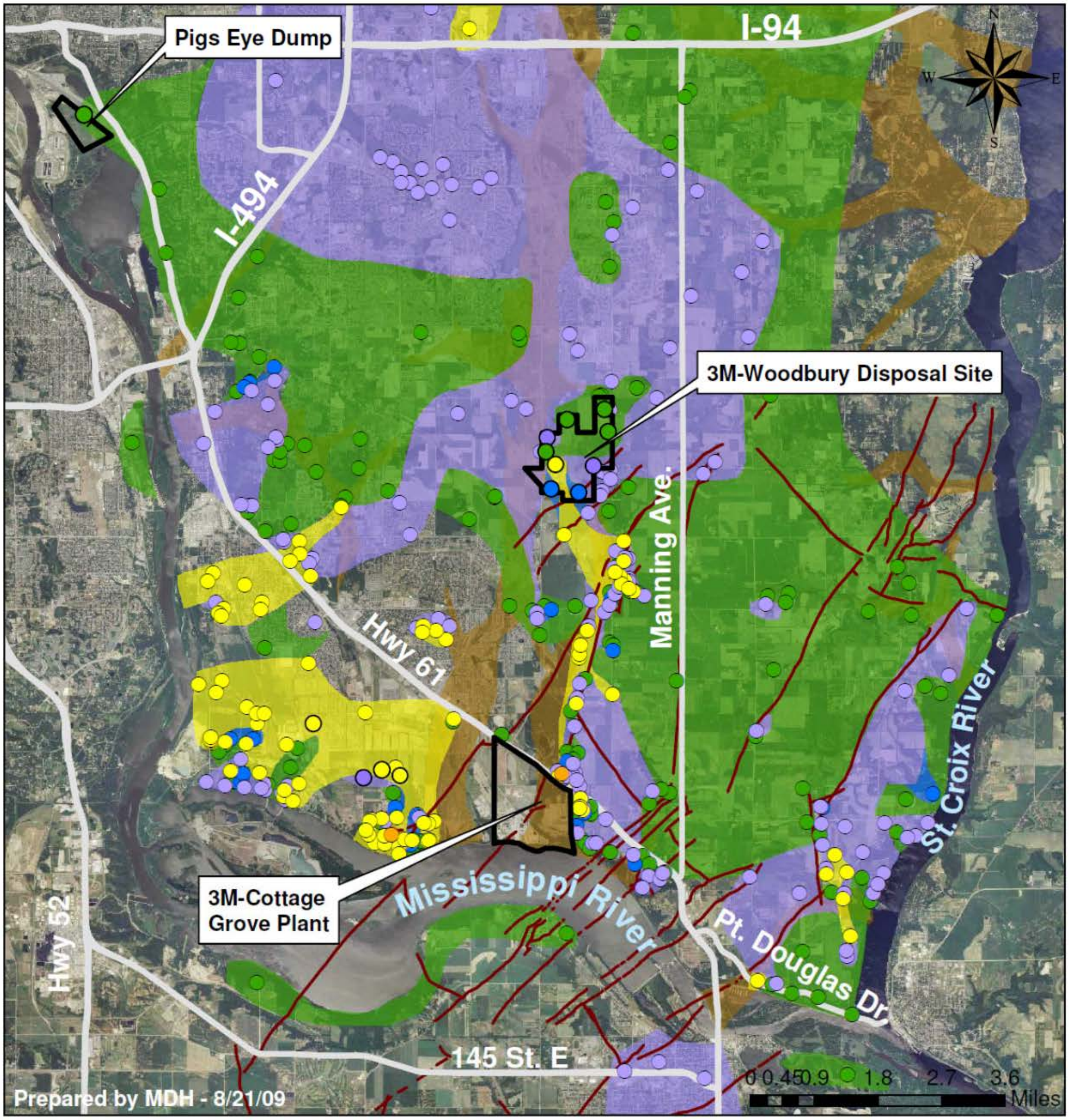



# Figure 8: PFBA in the Prairie du Chien

**Legend**

| | |
|---|---|
| PFBA > 7 ppb | PFBA = 0.1 - 0.99 ppb |
| PFBA = 3.5 - 7 ppb | PFBA < 0.1 ppb |
| PFBA = 1 - 3.49 ppb | PFBA not detected |
| bedrock valley | fault |
| **sampled well** | |

**Note: in areas with no color coding, there were no wells in this aquifer to sample and insufficient information to fill in the PFBA concentrations, or the Prairie du Chien is not present in this area.**

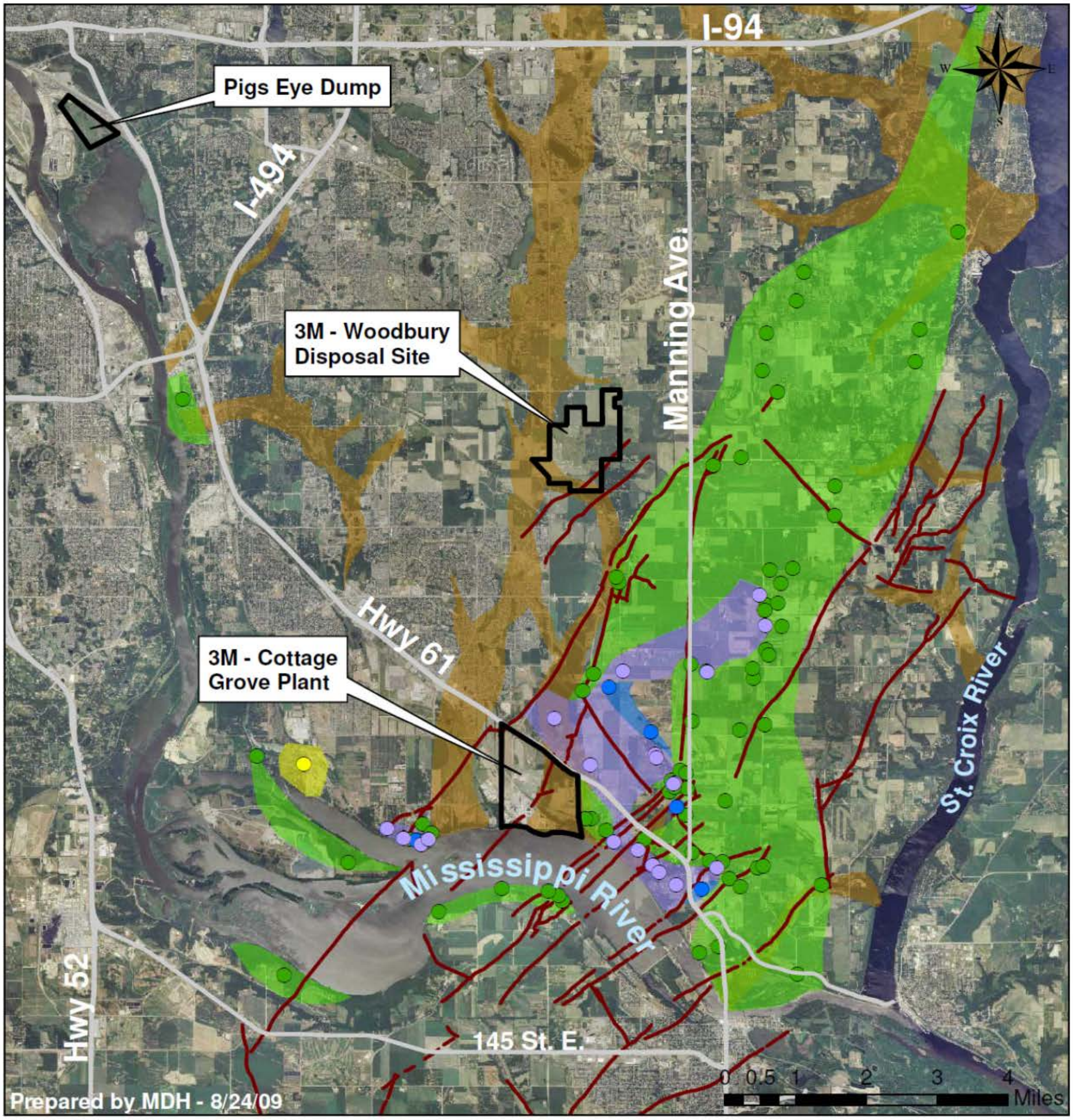



# Figure 9: PFBA in the Jordan

**Legend**

- PFBA = 3.5 - 6.9 ppb
- PFBA = 1 - 3.49 ppb
- PFBA = 0.1 -0.99 ppb
- PFBA < 0.1 ppb
- PFBA not detected
- Bedrock valley
- Fault
- sampled well

**Note: in areas with no color coding, there were no wells in this aquifer to sample and insufficient information to fill in the PFBA concentrations.**

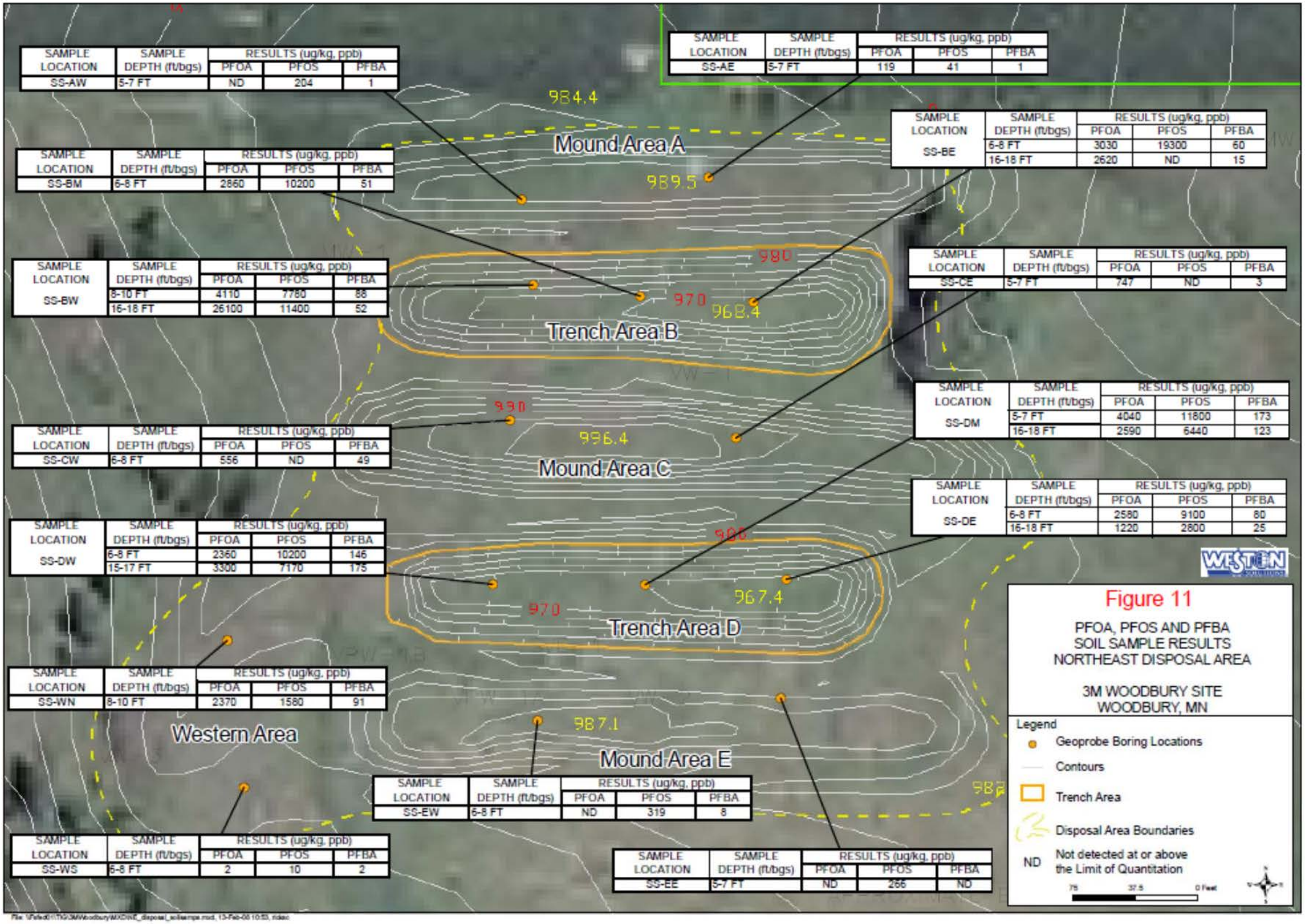



# Figure 10: PFBA in the Franconia

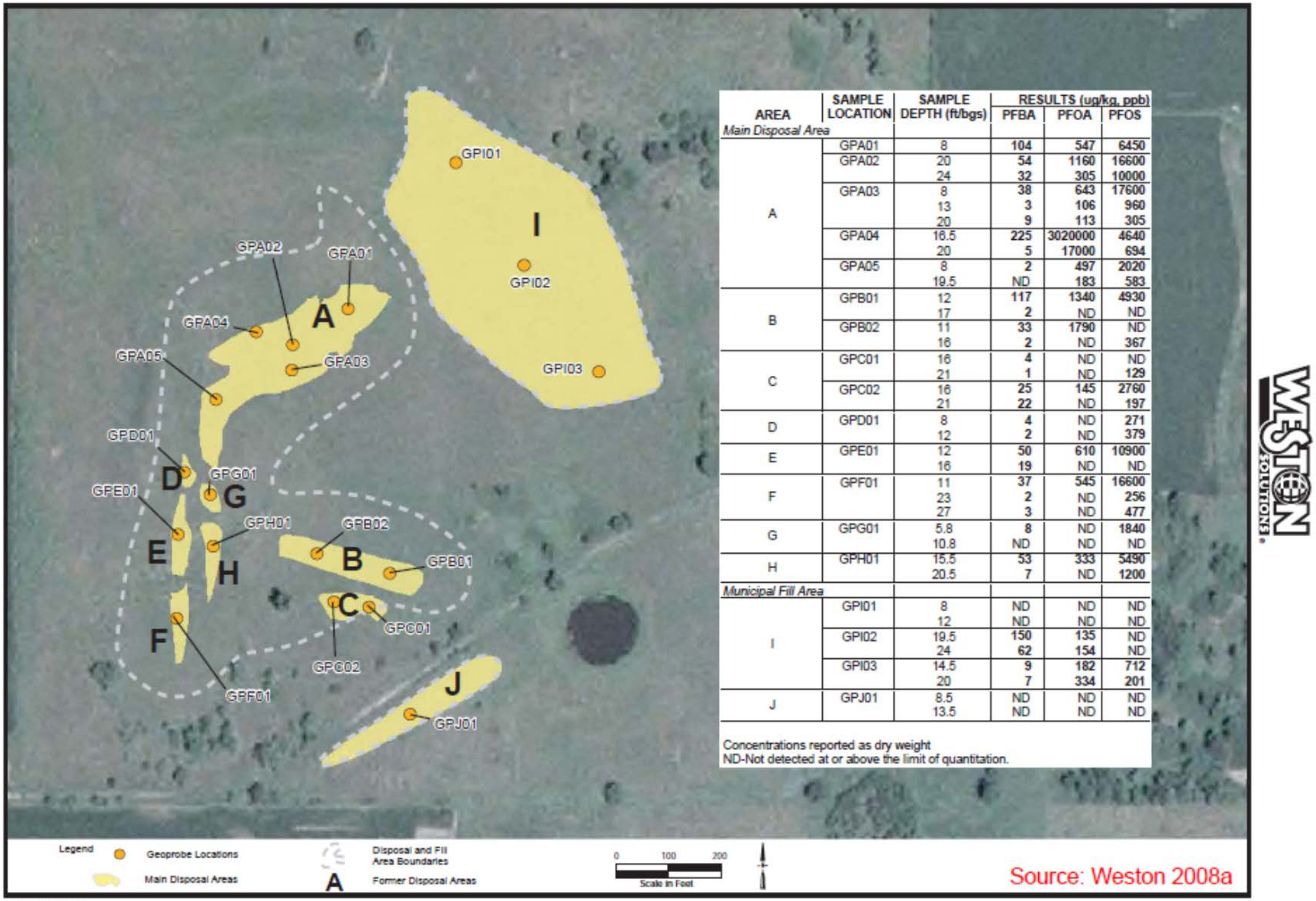
**Legend**

PFBA = 1 - 3.49 ppb
PFBA = 0.1 - 0.99 ppb
PFBA < 0.1 ppb
PFBA not detected
bedrock valley
faults

**Note: in areas with no color coding, there were no wells in this aquifer to sample and insufficient information to fill in the PFBA concentrations.**




Source: Weston 2008a

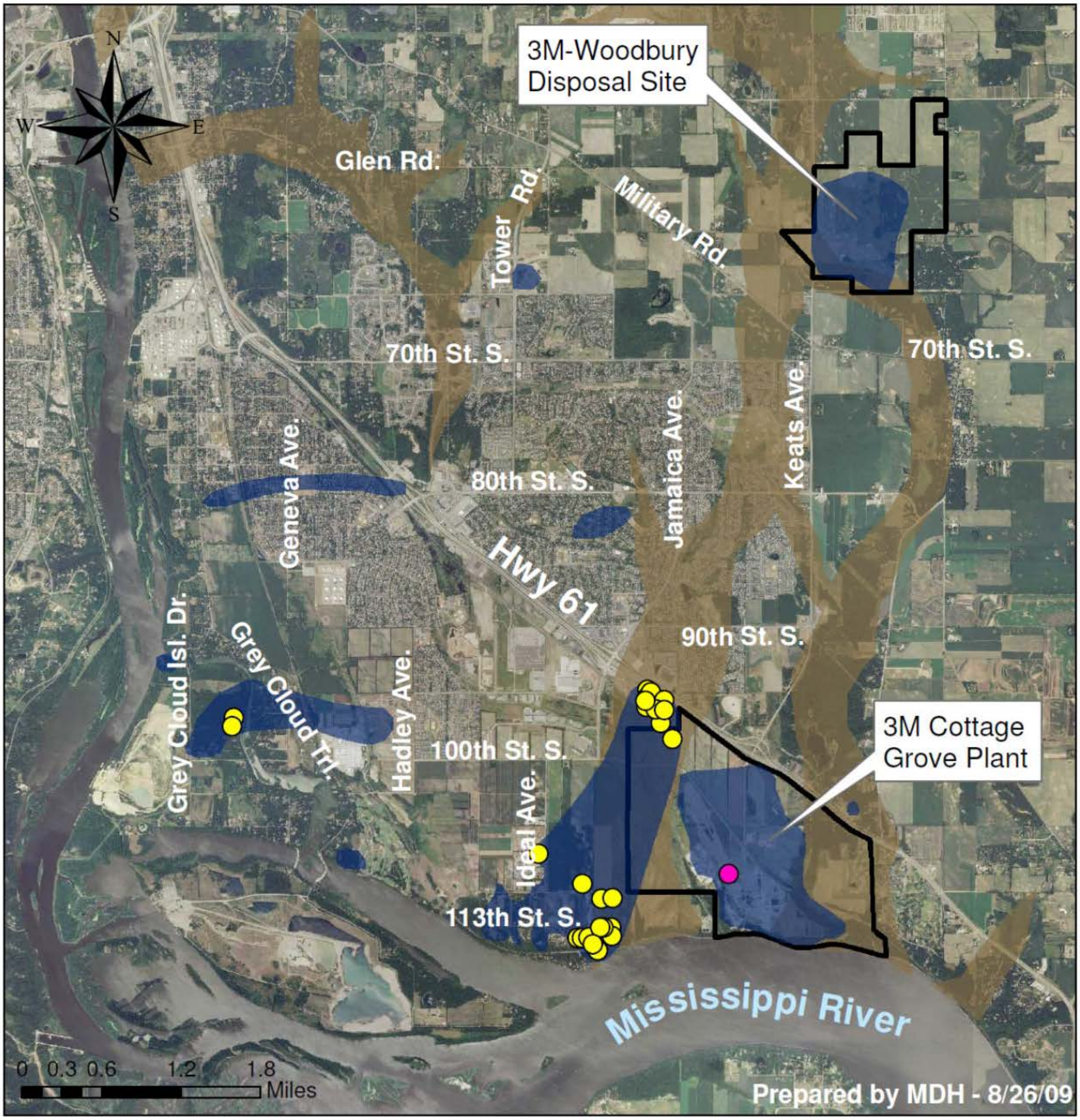



| AREA | SAMPLE LOCATION | SAMPLE DEPTH (ft/bgs) | RESULTS (ug/kg, ppb) PFBA | PFOA | PFOS |
|---|---|---|---|---|---|
| *Main Disposal Area* | | | | | |
| A | GPA01 | 8 | 104 | 547 | 6450 |
| | GPA02 | 20 | 54 | 1160 | 16600 |
| | | 24 | 32 | 305 | 10000 |
| | GPA03 | 8 | 38 | 643 | 17600 |
| | | 13 | 3 | 106 | 960 |
| | | 20 | 9 | 113 | 305 |
| | GPA04 | 16.5 | 225 | 3020000 | 4640 |
| | | 20 | 5 | 17000 | 694 |
| | GPA05 | 8 | 2 | 497 | 2020 |
| | | 19.5 | ND | 183 | 583 |
| B | GPB01 | 12 | 117 | 1340 | 4930 |
| | | 17 | 2 | ND | ND |
| | GPB02 | 11 | 33 | 1790 | ND |
| | | 16 | 2 | ND | 367 |
| C | GPC01 | 16 | 4 | ND | ND |
| | | 21 | 1 | ND | 129 |
| | GPC02 | 16 | 25 | 145 | 2760 |
| | | 21 | 22 | ND | 197 |
| D | GPD01 | 8 | 4 | ND | 271 |
| | | 12 | 2 | ND | 379 |
| E | GPE01 | 12 | 50 | 610 | 10900 |
| | | 16 | 19 | ND | ND |
| F | GPF01 | 11 | 37 | 545 | 16600 |
| | | 23 | 2 | ND | 256 |
| | | 27 | 3 | ND | 477 |
| G | GPG01 | 5.8 | 8 | ND | 1840 |
| | | 10.8 | ND | ND | ND |
| H | GPH01 | 15.5 | 53 | 333 | 5490 |
| | | 20.5 | 7 | ND | 1200 |
| *Municipal Fill Area* | | | | | |
| I | GPI01 | 8 | ND | ND | ND |
| | | 12 | ND | ND | ND |
| | GPI02 | 19.5 | 150 | 135 | ND |
| | | 24 | 62 | 154 | ND |
| | GPI03 | 14.5 | 9 | 182 | 712 |
| | | 20 | 7 | 334 | 201 |
| J | GPJ01 | 8.5 | ND | ND | ND |
| | | 13.5 | ND | ND | ND |

Concentrations reported as dry weight
ND-Not detected at or above the limit of quantitation.

07P-1561-3e

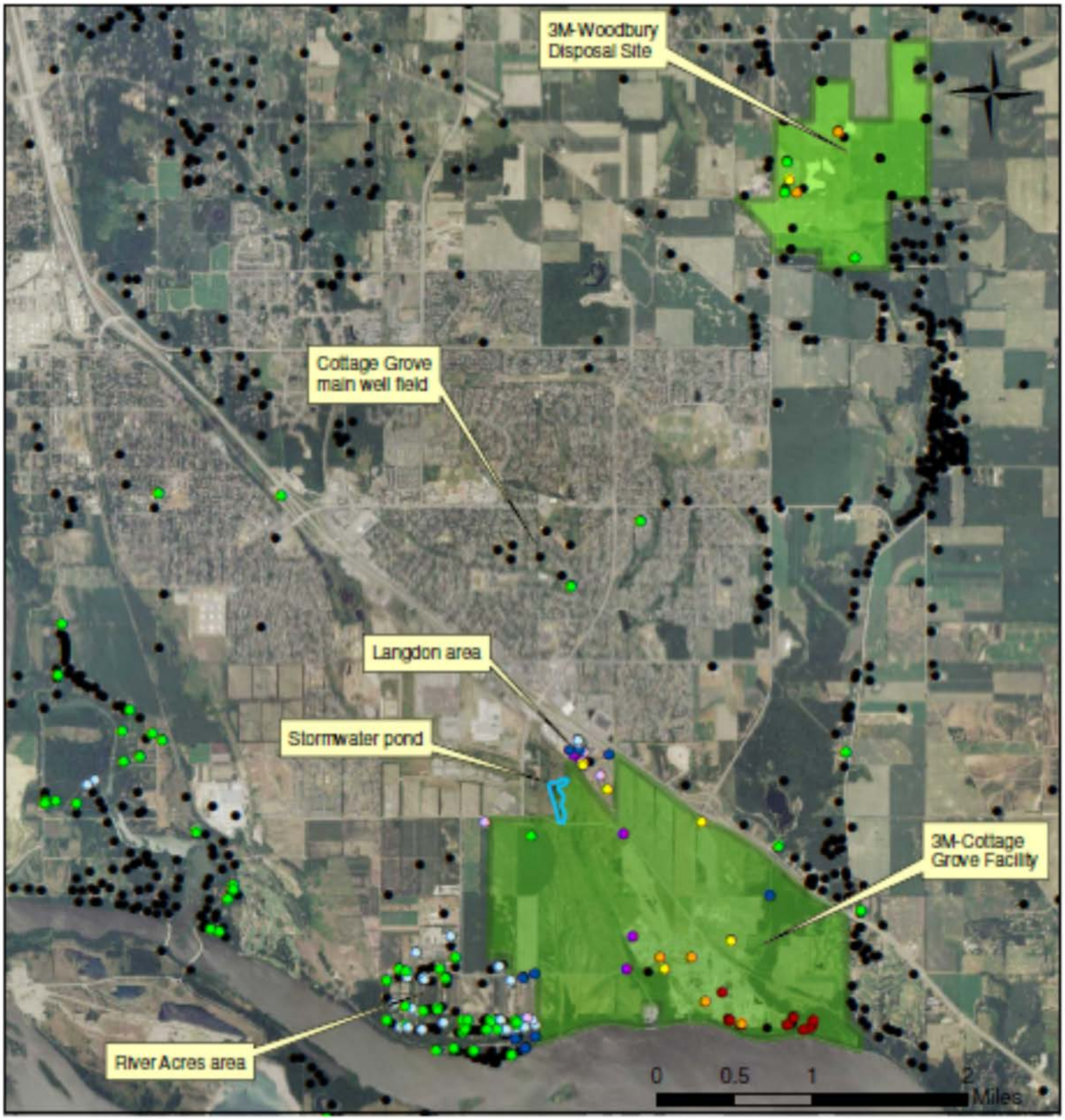
Figure 12 **SOIL SAMPLE RESULTS FOR FORMER MAIN DISPOSAL AREA AND MUNICIPAL FILL AREAS**
**3M WOODBURY SITE**
**WOODBURY, MN**




## Figure 13 - Areas Where Multiple PFCs Were Detected

### Legend

- Area with multiple PFCs
- Bedrock valley
- Private well with drinking water advisory
- Non-community public well with PFC levels that require treatment

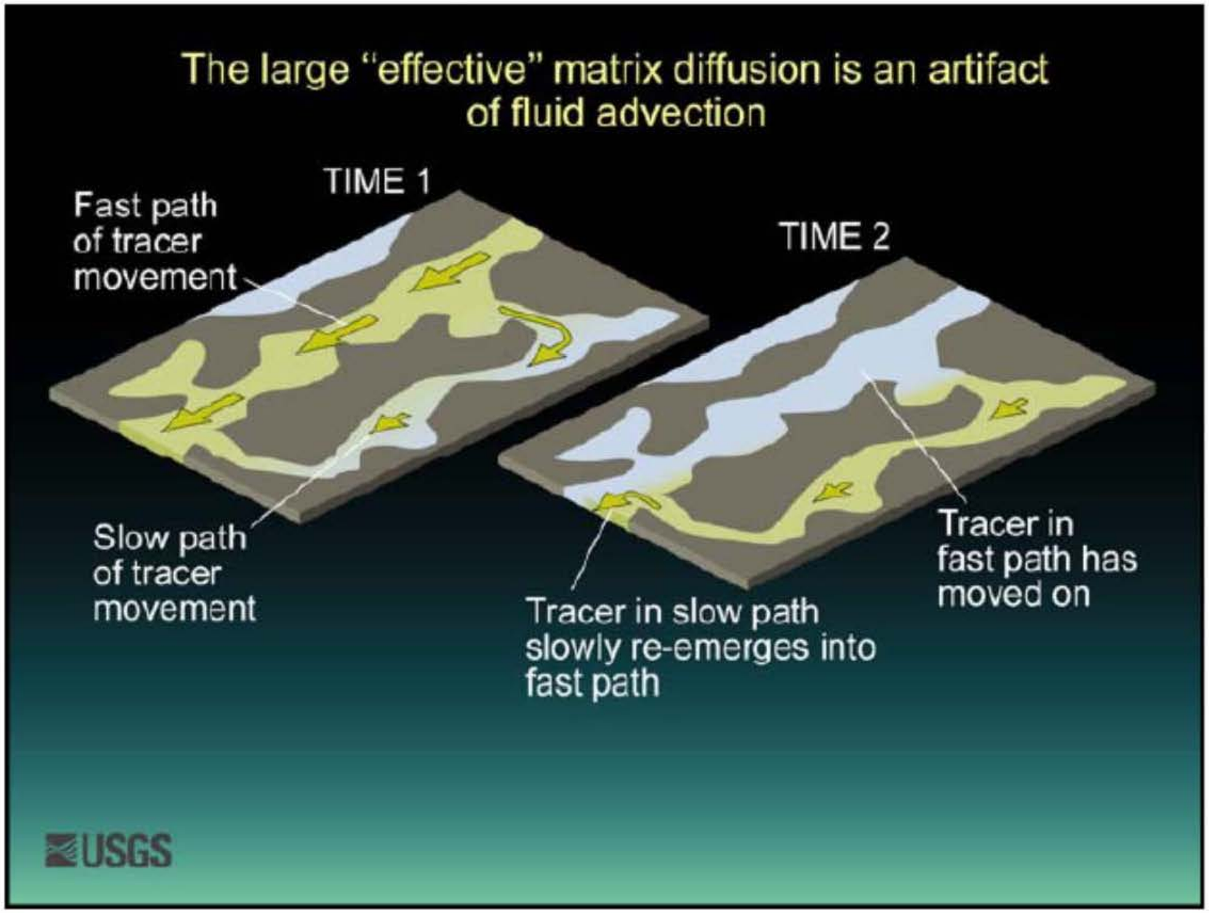



# Figure 14: PFOA Detections in Cottage Grove

**PFOA Concentration (ppb)**

- 0.02 - 0.10
- 0.11 - 0.20
- 0.21 - 0.30
- 0.31 - 0.50
- 0.51 - 1.00
- 1.01 - 2.00
- 2.01 - 10.00
- 10.01 - 1000.00
- Well sampled, no PFOA

# Figure 15: Secondary Flow Paths in Fractured Rock




This figure from a tracer study performed by Shapiro (2008) illustrates the concept of primary and secondary flow paths in fractured rock, like the Prairie du Chien in Washington County. The majority of contaminants in the aquifer quickly move through the primary (or "fast") flowpath, creating the first "pulse" of contaminant migration (shown as "Time 1"). This is followed by slower movement of the remaining contaminants through the secondary (or "slow") flowpaths, resulting in a more dilute but longer lasting plume (shown as "Time 2").

# Appendix 2: Tables

## Table 1: Initial 3M-Woodbury Disposal Site Monitoring Well Data[1], 2005 – 2006 (in ppb)

| Sample Location [2] | Aquifer [3] | PFOS | PFOA | PFBA | PFBS | PFHxS |
|---|---|---|---|---|---|---|
| B1 | CJDN | 0.056 – 0.069 | **2.26 – 2.33** | 1.66 | 1.83 – 3.47 | 2.31 – 2.61 |
| B2 | OPDC | ND [6] | ND | 0.476 | ND | ND |
| B3 | OPDC | 0.095 - 0.109 | 0.153 – 0.159 | 0.724 | 0.337 – 0.478 | 1.03 – 1.20 |
| B4 | OPDC | **1.83 – 2.29** | **2.78 – 3.12** | 1.31 | 5.72 – 11.0 | 19.7 – 23.3 |
| MW-2 | OPDC | NA [7] | NA | **118** | NA | NA |
| MW-3 | OSTP | NA | NA | 3.25 | NA | NA |
| MW-5 | OPDC | NA | NA | 1.7 | NA | NA |
| MW-7 | OSTP | NA | NA | 1.72 | NA | NA |
| MW-8 | OPDC | NA | NA | 5.09 | NA | NA |
| MW-11 | OPDC | NA | NA | 0.883 | NA | NA |
| CWM [4] | --- | **0.916 – 1.23** | **1.96 – 2.18** | 1.29 | 3.51 – 7.26 | 10.3 – 11.6 |
| CWD [5] | --- | **1.28 – 1.38** | **2.61 – 3.22** | NA | 3.40 – 7.34 | 7.76 – 9.61 |

Notes:
Values shown in boldface type exceed MDH drinking water criteria. No criteria exist for PFBS or PFHxS.
ppb = parts per billion
PFOS = perfluoro-octane sulfonate
PFOA = perfluoro-octanoic acid
PFBA = perfluorobutanoic acid
PFBS = perfluorobutane sulfonate
PFHxS = perfluorohexane sulfonate
[1] Data are from Weston (2007a). Results are average values calculated from two samples from same well collected on same date
[2] Well locations are shown in Figure 4; B = barrier well; MW = monitoring well
[3] Aquifer abbreviations: CJDN = Jordan, OPDC = Prairie du Chien, OSTP = St. Peter
[4] Combined discharge from Woodbury pumping wells
[5] Non-contact process water discharge from retention pond at 3M-Chemolite Plant
[6] ND = not detected above the method detection limit
[7] NA = not analyzed

## Table 2: General Results of 2007 Private and Business Well Sampling, results in ppb

| Aquifer | No. of Wells Sampled | No. of Wells w/ PFCs Detected | Max. PFOA (% wells) [1] | Max. PFOS (% wells) | Max. PFBA(% wells) | Max. PFPeA (% wells) | Max. PFHxA (% wells) | Max. PFBS (% wells) | Max. PFHxS (% wells) |
|---|---|---|---|---|---|---|---|---|---|
| Quaternary | 12 | 9 | ND | ND | 2.8 (75%) | 0.132 (17%) | ND | ND | ND |
| St. Peter | 15 | 11 | ND | ND | 2.5 (73%) | 0.091 (7%) | ND | ND | ND |
| Prairie du Chien | 173 | 163 | 0.926 (4%) | ND | 3.3 (94%) | 0.205 (11%) | 0.218 (5%) | ND | ND |
| Prairie du Chien – Jordan | 5 | 3 | ND | ND | 2.3 (60%) | 0.104 (20%) | 0.053 (20%) | ND | ND |
| Jordan | 307 | 217 | 0.3 (7%) | 0.1086 (1%) | 4.0 (71%) | 0.3 (14%) | 0.125 (6%) | 0.076 (1%) | 0.147 (1%) |
| Franconia | 88 | 10 | ND | ND | 1.608 (30%) | 0.108 (2%) | ND | ND | ND |
| Unknown | 339 | 319 | 1.378 (18%) | 0.943 (1%) | 5.6 (94%) | 0.42 (51%) | 0.141 (22%) | 0.118 (2%) | 0.1645 (2%) |
| Multiple | 21 | 13 | ND | ND | 3.031 (62%) | 0.172 (24%) | 0.068 (14%) | 0.1 (5%) | ND |

[1] First number is maximum concentration of the compound detected in any well in the designated aquifer, in parts per billion (ppb). Number in parentheses is the percent of wells in that aquifer in which the compound was detected at any concentration.
[2] ND = not detected above the method detection limit in any well in this aquifer
ppb = parts per billion
PFOS = perfluoro-octane sulfonate; PFOA = perfluoro-octanoic acid; PFBA = perfluorobutanoic acid; PFPeA = perfluoropentanoic acid; PFHxA = perfluorohexanoic acid; PFBS = perfluorobutane sulfonate; PFHxS = perfluorohexane sulfonate

## Table 3: 3M-Woodbury Disposal Site Groundwater, 2007-2008 (in ppb)

| Well | Date | PFBA | PFPeA | PFHxA | PFHpA | PFOA | PFBS | PFHxS | PFOS |
|---|---|---|---|---|---|---|---|---|---|
| B-1 | June 2007 | **1.59** | **0.487** | **0.974** | **0.143** | **1.44** | **1.73** | **1.52** | ND |
| | March 2008 | **1.79** | **0.509** | **0.847** | **0.128** | **1.2** | **1.75** | **1.39** | **0.041** |
| B-2 | June 2007 | **0.471** | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | **0.536** | ND | ND | ND | ND | ND | ND | ND |
| B-3 | June 2007 | **0.728** | **0.074** | **0.119** | ND | **0.207** | **0.362** | **1.29** | **0.171** |
| | March 2008 | **0.769** | **0.0672** | **0.0946** | ND | **0.0202** | **0.429** | **1.43** | **0.156** |
| B-4 | June 2007 | **1.5** | **0.406** | **0.96** | **0.342** | **2.44** | **3.48** | **11.5** | **1.78** |
| | March 2008 | **1.69** | **0.447** | **0.837** | **0.388** | **2.94** | **3.84** | **13.9** | **3.06** |
| MW-2 | June 2007 | **126** | **9.31** | **13** | NR | **4.92** | **14.4** | **4.65** | ND |
| | March 2008 | **71.5** | **9.14** | **8.14** | **1.51** | **3.47** | **9.15** | **2.06** | **0.0361** |
| MW-4 | June 2007 | **0.809** | **0.062** | **0.0537** | **0.0357** | **0.0401** | **0.216** | **0.433** | **0.172** |
| | March 2008 | **0.873** | **0.0586** | **0.0317** | ND | **0.0269** | **0.124** | **0.267** | **0.104** |
| MW-G | June 2007 | **0.127** | ND | ND | ND | ND | ND | ND | **0.114** |
| | March 2008 | **0.201** | ND | ND | ND | ND | ND | ND | ND |
| MW-H | June 2007 | ND | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | ND | ND | ND | ND | ND | ND | ND | ND |
| S01JS | June 2007 | ND | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | **0.0395** | ND | NR | ND | ND | ND | ND | ND |
| S01PC | June 2007 | **0.85** | **0.0347** | **0.0367** | ND | ND | ND | ND | ND |
| | March 2008 | **0.942** | **0.0398** | **0.12** | ND | ND | ND | ND | ND |
| S02DR | June 2007 | **0.594** | **0.0252** | ND | ND | **0.033** | ND | **0.0373** | ND |
| | March 2008 | **0.647** | ND | ND | ND | **0.0326** | ND | **0.0499** | ND |
| S02JS | June 2007 | ND | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | **0.0360** | ND | ND | ND | ND | ND | ND | ND |
| S02PC | June 2007 | **1.69** | **0.0573** | **0.0255** | ND | **0.0286** | ND | **0.0526** | ND |
| | March 2008 | **1.2** | **0.0384** | **0.0403** | ND | ND | ND | **0.0435** | ND |
| S03JS | June 2007 | **0.272** | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | **0.233** | ND | ND | ND | ND | ND | ND | ND |

## Table 3 (continued): 3M-Woodbury Disposal Site Groundwater, 2007-2008 (in ppb)

| Well | Date | PFBA | PFPeA | PFHxA | PFHpA | PFOA | PFBS | PFHxS | PFOS |
|---|---|---|---|---|---|---|---|---|---|
| S03PC | June 2007 | **0.71** | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | **0.67** | ND | ND | ND | ND | ND | ND | ND |
| S04PC | June 2007 | **0.335** | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | **0.643** | ND | **0.0418** | ND | ND | ND | ND | ND |
| S04SP | June 2007 | **1.14** | **0.0557** | ND | ND | ND | ND | ND | ND |
| | March 2008 | **1.58** | **0.041** | **0.0357** | ND | ND | ND | ND | ND |
| S05JS | June 2007 | ND | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | ND | ND | ND | ND | ND | ND | ND | ND |
| S05PC | June 2007 | **0.393** | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | **0.55** | ND | ND | ND | ND | ND | ND | ND |
| S05SP | June 2007 | **0.438** | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | **0.917** | **0.0308** | ND | ND | ND | ND | ND | ND |
| S06JS | June 2007 | ND | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | ND | ND | ND | ND | ND | ND | ND | ND |
| S06PC | June 2007 | **1.1** | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | **0.868** | ND | ND | ND | ND | ND | ND | ND |
| S07JS | June 2007 | ND | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | ND | ND | ND | ND | ND | ND | ND | ND |
| S07PC | June 2007 | **0.984** | **0.0317** | ND | ND | ND | ND | ND | ND |
| | March 2008 | **0.756** | ND | ND | ND | ND | ND | ND | ND |
| S07SP | June 2007 | **0.58** | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | **0.839** | ND | ND | ND | ND | ND | ND | ND |
| S08JS | June 2007 | **0.353** | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | **0.309** | ND | ND | ND | ND | ND | ND | ND |
| S08PC | June 2007 | **0.122** | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | **0.100** | ND | ND | ND | ND | ND | ND | ND |
| S09JS | June 2007 | ND | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | ND | ND | ND | ND | ND | ND | ND | ND |

Data from: Weston (2007b) and Weston (2008b) ppb = parts per billion ND = not detected NR = not reportable

## Table 4: Range of PFC Concentrations in Cottage Grove City Wells (in ppb)

| Well No. | PFBA | PFPeA | PFHxA | PFOA | PFBS | PFHxS | PFOS |
|---|---|---|---|---|---|---|---|
| **1** | 0.6 – 1.02 | ND | ND | ND | ND | ND | ND |
| **2** | 0.38 – 1.0 | ND | ND | ND | ND | ND | ND |
| **3** | 0.89 – 1.4 | ND – 0.07 | ND | ND | 0.1 – 0.25 | ND – 0.14 | ND |
| **4** | 0.75 – 1.22 | ND – 0.07 | ND – 0.56[a] | ND | 0.1 – 0.32 | ND – 0.21 | ND |
| **5** | 1.14 – 1.79 | ND – 0.07 | ND | ND | ND | ND | ND |
| **6** | 0.77 – 1.34 | ND | ND | ND | ND – 0.17 | ND – 0.07 | ND |
| **7** | 0.98 – 1.74 | ND – 0.06[b] | ND – 0.05[a] | ND | ND – 0.16[b] | ND | ND |
| **8** | 0.95 – 1.36 | ND – 0.07 | ND – 0.06[a] | ND | ND – 0.09[a] | ND – 0.14[a] | ND |
| **9** | 0.83 – 1.02 | ND | ND | ND | ND | ND | ND |
| **10** | 0.94 – 1.23 | ND | ND | ND | ND | ND | ND |
| **11** | 0.3 – 0.6 | ND | ND | ND | ND | ND | ND |

Notes:

PFC = perfluorinated chemicals

ND = not detected above the method detection limit

ppb = parts per billion

a – This compound detected in only one sample from this well since November 2006

b – This compound detected in only two samples from this well since November 2006

# Appendix 3: Public Comments

A draft version of this Public Health Assessment report was released for public comment on August 25, 2010. MDH received written comments from State Senator Katie Sieben, the Minnesota Pollution Control Agency (MPCA), and 3M Corporation (3M). The texts of the comments are included in this appendix.

Sen. Sieben's comments primarily focused on the unknowns regarding past PFC exposure levels, possible long-term health effects, and the need for continued monitoring of water quality and public health in the affected communities (Sieben, 2010).

The MPCA comments focused primarily on differences in interpretation regarding the adequacy of the monitoring well network at the 3M-Woodbury Disposal Site and the source PFCs (other than PFBA) in the Langdon-River Acres neighborhood in south Cottage Grove (MPCA, 2010b). These comments were addressed in the "PFC-Related Investigations at the 3M-Woodbury Disposal Site" and "Evaluation of Impacts to Groundwater" sections of this document. Specifically, the MPCA comments were addressed in the following locations:

1. **Potential Source of PFCs Near Langdon Neighborhood** – these comments were addressed on page 28 and by the addition of Figure 14.
2. **Adequacy of 3M's Monitoring Well Network at the 3M Woodbury Disposal Site** – these comments were addressed on pages 18, 29, and 41.

3M's comments addressed a number of areas, but primarily focused on: differences in hydrogeologic interpretations, the adequacy of the monitoring well network, 3M's PFC waste disposal and reporting history, PFC toxicology and the potential for human health effects associated with PFC releases from the 3M-Woodbury Disposal Site, and the recommendation to extend city water to the most contaminated neighborhoods (3M, 2010a). These comments were addressed, as appropriate, throughout the document, but primarily in the "PFC-Related Investigations at the 3M-Woodbury Disposal Site," "Evaluation of Impacts to Groundwater," "Exposure Through Private Wells, " and "Public Health Implications of PFC Exposure" sections of this document. Specifically, the 3M comments were addressed in the following locations:

1. **Summary section, Introduction heading** – these comments were addressed on page 4. Reference to possible exposure to air emissions were retained in the introduction as a possible exposure pathway since dust and ash (generated during waste burning) could have contained PFCs.
2. **Summary section, Conclusions 1 and 2** – 3M's comments are more narrative than editorial and largely go beyond the scope of the summary section, MDH believes the caveats included in Conclusion 1 are sufficient to explain the uncertainties associated with past exposures.
3. **Summary section, Next Steps** – these comments were addressed, where appropriate, on page 5.
4. **Introduction Section** – the suggested historical information has been incorporated on page 6; other comments have been addressed, as appropriate, on pages 6-8.
5. **Background Section, Site Description and History** – these comments were addressed, where appropriate, on pages 8-11. MDH does not agree with 3M's hydrogeologic

interpretations, as well sampling data clearly demonstrates the contaminant migration pathways described in this section. In addition, a recent minor reduction in pumping rates at the site has resulted in migration of PFCs to not only the Prairie du Chien, but also Jordan aquifer in downgradient monitoring wells, contradicting 3M's assertion regarding potential for contaminant migration into that aquifer.

6. **Background Section, PFC Analysis** – this comment was address on page 11.
7. **Background Section, PFCS in Drinking Water** – the suggested clarification regarding hazard index calculations has been addressed on page 12.
8. **Community Well Monitoring** – the suggested clarifications were made on pages 12 and 13 and figure 6; MDH notes that despite 3M's assertion regarding the data shown in the table on page 13, there is almost no difference between the average and median data.
9. **Private Well Sampling** – these comments and suggested clarifications were addressed on pages 14 – 17; MDH disagrees with 3M's hydrogeologic interpretation and assessment of the adequacy of the monitoring well network.
10. **MPCA- 3M Consent Order for PFC Disposal Sites –** the suggested clarifications have been made on pages 17 – 18.
11. **Remedial Options for the 3M**-Woodbury Disposal Site – the suggested clarifications were made on page 19.
12. **General Regional Issues** – MDH and the cities of Woodbury, Cottage Grove, Lake Elmo, and Oakdale have expended significant resources evaluating the citing of new city wells to avoid the PFC plumes, or minimize the levels of PFCs in the new wells. This additional effort would not have been necessary except for the presence of the PFC contamination.
13. **Community Concerns** – 3M's comments are not accepted, as the purpose of this section is to identify community concerns, which are addressed by the document as a whole.
14. **Evaluation of Environmental Fate and Exposure Pathways, Introduction** – these comments were addressed, as appropriate, on pages 21-23.
15. **Evaluation of Impacts on Groundwater** – these comments were addressed, where appropriate, on pages 23-26; however, MDH does not agree with much of 3M's hydrogeologic interpretation. As 3M notes in several places in their overall comments, the groundwater extraction system has been operating for over 40 years and presumed to be capturing all of the groundwater at the site, yet the PFC groundwater distribution documented by the sampling of hundreds of wells clearly demonstrates PFCs have migrated away from the site and do not follow a simple "regional groundwater flow direction" pathway, but instead are strongly controlled by small- and large-scale structures in the bedrock. In addition, a recent slight reduction in pumping rates at the site, which 3M's model showed would result in no loss of PFC containment was followed quite rapidly by detection of PFCs in monitoring wells not previously contaminated, suggesting that there are significant flaws in 3M's groundwater model and hydrogeologic understanding of the site.
16. **Exposure Through Private Wells** – these comments were addressed, where appropriate, on pages 26-29. MDH notes that as several additional well advisories have been issued since the original draft of this report were written, it is appropriate to retain language regarding the possibility of increased PFC levels in private wells.
17. **Exposure Through Other Pathways** – these comments were addressed, where appropriate, on page 29.

18. **Public Health Implications of PFC Exposure** – 3M is correct in pointing out that this document only summarizes a small portion of the scientific literature on PFCs, as is appropriate for a document of this nature. The reader is referred to the ATSDR draft Toxicological Profile and the review paper by Lau et al (2007) for a more thorough review. The remaining comments were addressed, where appropriate, in the corresponding sections of the document. 3M's comments are also attached below if the reader wishes to peruse them.
19. **Summary of Human Exposure Information** – A review of all available studies on PFCs is beyond the scope of the document. Modifications where appropriate were made in the corresponding sections to address 3M's comments.
20. **Child Health Considerations** – the suggested clarification has been included on page 36.
21. **Conclusions** – MDH does not agree with the suggested changes in 3M's comments. MDH cannot determine whether 3M's past disposal practices were legal. It is appropriate to discuss all possible exposure pathways – airborne dust and ash were almost certainly generated at the site in the past and were very likely contaminated with PFCs, posing a possible exposure pathway albeit one that cannot be evaluated. MDH staff observed evidence of trespass on the 3M property, so direct contact with contaminated soil (and in the past, PFC waste) was a potential exposure pathway. The comparison of 3M's workers (a small population of primarily healthy, male adults) to the general population is not appropriate. Without adequate information regarding past exposures, MDH cannot make any conclusions regarding possible health effects of those past exposures.
22. **Recommendations** – the language regarding the trespassing issue has been modified. MDH does not agree with 3M's comments regarding the other recommendations. MDH has long stated its preference for municipal water over individual filter systems, where feasible, to address drinking water contamination. MDH does not agree with 3M's hydrogeologic interpretations or assessment of the adequacy of the monitoring well network.

**KATIE SIEBEN**
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Email: sen.katie.sieben@senate.mn

**Senate**

**State of Minnesota**

September 29, 2010

Site Assessment and Consultation Unit
Minnesota Department of Health
P.O. Box 64975
625 Robert Street North
St. Paul, MN 55164-0975

To Whom It May Concern:

I am writing in response to the publication of "Perfluorochemical Contamination in Southern Washington County, Northern Dakota County, and Southeast Ramsey County, Minnesota." I appreciate the opportunity for public comment.

I strongly support and appreciate the work and research that has been done to evaluate the health effects of PFC contamination and expect that same dedication to continue into the future.

It is clear to me after reading the report, that the health effects are currently unknown. The report cites several studies taking place now and expected to be completed in the next few years. I expect MDH to monitor these studies and share the results with Minnesotans as soon as they're available.

I also think it is important to differentiate between unknown and an absence of health effects. I was disappointed to see local media coverage of the report stating that low PFC levels in the Eastern metro are unlikely to result in adverse health effects. Not only do we not know that to be true, we also don't know the previous levels of PFCs in the water. The report states that both, "The length of time local residents were exposed to PFCs through their drinking water is unknown." And, "It is also possible that the levels in the groundwater were higher than currently detected." It is important that we stay vigilant and continue to keep the health of Minnesotans forefront in our minds.

Thank you, again, for the opportunity to comment. I look forward to continuing to work with MDH on this important issue.

Sincerely,

*Katie Sieben*

Katie Sieben
State Senator

December 6, 2010

**MPCA COMMENTS ON MDH DRAFT PUBLIC HEALTH ASSESSMENT**
**3M WOODBURY DISPOSAL SITE**

At a meeting with MPCA Superfund staff and management on December 2, 2010, Rita Messing, Supervisor of the MDH Site Assessment and Consultation Unit, requested a clarification of several MPCA comments regarding the MDH Draft Public Health Assessment (PHA) - Perfluorochemical Contamination in Southern Washington County, Northern Dakota County and Southeast Ramsey County, Minnesota. The following clarification is a discussion/elaboration of several MPCA comments that MPCA submitted during the official public comment period that ended on October 12, 2010.

The Draft PHA focused on the 3M Woodbury Disposal site (Site). MPCA's comments on the PHA, dated October 12, 2010, included brief responses to two issues that were discussed by MDH: (1) A potential source of PFC contamination near the Langdon neighborhood in Cottage Grove; and (2) The adequacy of 3M's monitoring well network at the Site.

After receiving MPCA's October 12, 2010 comments, MDH staff prepared a technical summary of the two issues, and submitted it to MPCA. This document is MPCA's response to the MDH technical summary package, and constitutes the MPCA position on the two issues.

**Potential Source of PFCs Near Langdon Neighborhood**

In December 2002, a large fire occurred at Up North Plastics, a manufacturing facility located approximately one-half mile west of the Langdon Neighborhood. The fire required the application of approximately 4000 gallons of Class B (PFC-containing) firefighting foam in order to extinguish it.

Approximately 20 fire departments responded to the Up North Plastics fire. Records indicate that most of the foam used at the fire was manufactured by 3M, and based on 3M Material Safety Data Sheets, the foam contained approximately 0.5-1.5% PFOSs (See MSDS). Documentation showed that the foam ran southward off the Up North property and into a storm drain, a ditch and a pond south of the facility (Figure).

As part of the recent awareness regarding PFCs in groundwater in the eastern metropolitan area, local legislators and other officials expressed concerns that the Up North fire could have caused ground water and/or well contamination in the area. In response to requests from local officials, the MPCA ordered one of its multi-site contractors to conduct a limited investigation, and collect soil, surface water and sediment samples in the vicinity of Up North Plastics (Figure).

The Up North data show that PFCs were detected in all three media, but at concentrations below corresponding health-based standards (Tables, email). PFOS and PFHxS were the PFCs with the highest concentrations in soil and sediment, while the pond water samples were highest in PFBA and PFOA. MPCA investigations at other known firefighting foam sites have shown that PFOS and PFHxS are often major components in contaminated groundwater (Table – e.g. WAFTA site).

As part of the limited Up North investigation, MPCA's contractor also sampled four irrigation wells located southwest (downgradient) from the Up North property (Figure). Water samples from these irrigation wells contained detections of several PFCs (mainly PFBA and PFOA) that were below health-based standards (Tables, email). PFBA and PFOA concentrations in these well samples are consistent with the regional distribution of PFCs in groundwater.

Based on the limited investigation near the Up North property, and on data from other firefighting foam sites (e.g., Minneapolis-St. Paul Airport), MPCA determined that the Class B foam used in the Up North fire did not appear to be a major contributor to PFC groundwater contamination in the area. Furthermore, the PFC-contaminated wells in the Langdon neighborhood do not appear to be impacted by PFCs from the Up North firefighting foam. The fact that the fire was a one-time event, the low concentrations or lack of PFOS and PFHxS in the Langdon neighborhood wells, and the upgradient location of the wells relative to the Class B foam release from the Up North fire are all evidence that the Up North fire was not a likely source for PFC contamination in these wells. Similarly, the Up North fire was not a likely source for the observed PFC contamination in the River Acres neighborhood wells.

3M submittals to the MPCA provided quite a bit of information about PFC waste disposal at the Site. MPCA files indicate that from 1960 to 1963, 3M disposed approximately 16,000 gallons of hydrofluoric acid sludge and tars into the northeast disposal area at the Site. The sludge and tars were dumped in limestone-lined (i.e., native bedrock) or limestone slurry filled trenches. 3M records show that the PFC-containing acid tar waste was placed into the NE Disposal Area as a bulk liquid. However, an investigation 40 years later found that most of the acid sludge and tar had migrated from the trenches and out into the aquifers and may have moved away from the Site before the barrier wells were installed. MPCA files indicate that high concentrations of PFCs were found in contaminated soil at the main disposal area. Groundwater data show that at least eight different PFCs, including PFOS and PFHxS, have been detected at the Site (see Table 3 from PHA).

The extent of PFC groundwater contamination downgradient (i.e., south and slightly west) from the Woodbury Disposal site is displayed in part in monitoring wells at the 3M Cottage Grove facility. Specifically, two monitoring wells (MW-1 and MW-5) located on the north side of the facility, and therefore upgradient of known disposal areas at the Cottage Grove facility, exhibit elevated PFOA and PFOS concentrations (Figure). The presence of PFC contamination in these monitoring wells is consistent with the interpretation that the Woodbury Disposal site is the source for regional PFC groundwater contamination.

The PFC waste disposal practices at the Site, the prevailing groundwater flow directions and the presence of bedrock valleys and bedrock fractures, and the regional distribution of PFC groundwater contamination all support the conceptual model that the 3M Woodbury Disposal site is the major source of PFC contamination in the Langdon and River Acres neighborhoods. The relatively small number of wells situated along the bedrock valley north of the Langdon and River Acres neighborhoods in comparison to the relatively large number of private wells in these two neighborhoods may bias the data, and may also skew the interpretation of the data. Overall, based on the available information, the MPCA has determined that the 3M Woodbury Disposal site is the predominant source for the PFC contamination in the Langdon and River Acres wells.

**Adequacy of 3M's Monitoring Well Network at the 3M Woodbury Disposal Site**

MPCA's comments on the PHA included a comment intended to address concerns that only one on-site monitoring well (MW-S06-PC) intersects the high transmissivity zone (HTZ) in the Prairie du Chien aquifer. Based on 3M's Feasibility Study and other submittals, two other monitoring wells (MW-4L, and MW-5) are open to the HTZ. In addition, barrier wells B-3 and B-4 are open to the HTZ, while barrier well B-1 may intersect the HTZ (Figures – cross-sections).

Overall, MPCA acknowledges the PHA's inference that 3M's current monitoring well network configuration is not optimal for evaluating the HTZ in the area downgradient from the disposal areas. As a consequence, one of MPCA's comments on the PHA requested specific recommendations from MDH for improving the HTZ monitoring.

MPCA's June 30, 2010 letter response to 3M's Request to Reduce Barrier Well Pumping, states that MPCA and MDH will review groundwater data during the initial (25%) barrier well pumping reduction at the Site to determine what conditions, such as the placement of additional monitoring wells, might be necessary for any further pumping reductions. Thus, data generated during the barrier well pumping reduction monitoring will help MPCA/MDH determine if 3M will be required to improve the HTZ monitoring capabilities of the monitoring well network.

From MPCA's perspective, MDH's technical summary contains several relatively minor areas of differing interpretations regarding the existing monitoring well network. Specifically, MPCA does not share MDH's characterization of the relative location and available data for MW-4L and MW-5. However, these are relatively minor differences, and can be resolved through further communication between MDH and MPCA as additional data becomes available from the project.

MATERIAL SAFETY DATA SHEET
3M
3M Center
St. Paul, Minnesota
55144-1000
1-800-364-3577 or (651) 737-6501 (24 hours)



DIVISION: 3M SPECIALTY MATERIALS
TRADE NAME:
FC-602 LIGHT WATER(TM) ATC PLUS(TM) AR-AFFF 3% OR 6%
ID NUMBER/U.P.C.:

| | | | |
|---|---|---|---|
| 98-0211-4114-2 | 00-51135-02487-4 | 98-0211-4115-9 | 00-51135-02488-1 |
| 98-0211-7291-5 | 00-51135-10672-3 | CF-1206-0960-1 | - - - |
| CF-1206-0961-9 | - - - | CF-1206-0965-0 | - - - |
| CG-7900-7591-7 | - - - | GF-1000-0045-4 | - - - |
| XF-0008-0077-1 | - - - | XF-0008-0078-9 | - - - |
| XF-0008-0100-1 | - - - | ZF-0002-0089-7 | - - - |
| ZF-0002-0178-8 | - - - | ZF-0002-0199-4 | - - - |
| ZF-0002-0318-0 | - - - | ZF-0002-0319-8 | - - - |
| ZF-0002-0425-3 | - - - | ZF-0002-0438-6 | - - - |
| ZF-0002-0508-6 | - - - | ZF-0002-0859-3 | - - - |
| ZF-0002-1008-6 | - - - | | |

ISSUED: December 17, 1999
SUPERSEDES: April 08, 1999
DOCUMENT: 10-4146-6

| 1. INGREDIENT | C.A.S. NO. | PERCENT |
|---|---|---|
| WATER.................................... | 7732-18-5 | 58 - 64 |
| ETHYLENE GLYCOL.......................... | 107-21-1 | 20 |
| DIETHYLENE GLYCOL BUTYL ETHER............ | 112-34-5 | 12 |
| AMPHOTERIC FLUOROALKYLAMIDE DERIVATIVES(2) +(5133P)............... | TradeSecret | 1 - 5 |
| MAGNESIUM SULFATE........................ | 7487-88-9 | 1 |
| ALKYL SULFATE SALTS(2) +(5156P, 5147P).. | TradeSecret | 1 - 5 |
| PERFLUOROALKYL SULFONATE SALTS(5) +(5145P)............................. | TradeSecret | 0.5 - 1.5 |
| THICKENERS + (5126P, 5122P).............. | TradeSecret | 0.5 - 1.5 |
| RESIDUAL ORGANIC FLUOROCHEMICALS......... | Mixture | Unknown |

The components of this product are in compliance with the chemical notification requirements of TSCA. All applicable chemical ingredients in this material are listed on the European Inventory of Existing Chemical Substances (EINECS), or are exempt polymers whose monomers are listed on EINECS.

This product contains the following toxic chemical or chemicals subject to

Abbreviations: N/D - Not Determined N/A - Not Applicable CA - Approximately


◎ **Soil or sediment sampling location**

□ **Surface water sampling location**

**Scale 1 inch = 350 feet**

↑ **North**

| **UTM Coordinates** | | | |
|---|---|---|---|
| **Soil-1** | Zone 15 505204E 4961673N | **Sed-1** | Zone 15 505323E 4961591N |
| **Soil-2** | Zone 15 505184E 4961714N | **Sed-2** | Zone 15 505332E 4961534N |
| **Soil-3** | Zone 15 505047E 4961821N | **Sed-3** | Zone 15 505356E 4961598N |
| **Soil-4** | Zone 15 505224E 4961655N | **Sed-4** | Zone 15 505403E 4961317N |
| **Soil-5** | Zone 15 505331E 4961638N | **SW-1** | Zone 15 505323E 4961591N |
| | | **SW-2** | Zone 15 505403E 4961317N |

**WCEC**
ENVIRONMENTAL CONSULTANTS

**Figure 2: PFC Sampling Locations**
**Fire Fighting Foam Investigation, Cottage Grove, MN**
**MPCA Work Order No.: SFWC1004**
**WCEC Project No.: 08-6484-30**

| CLIENT ID | Soil 1 | Duplicate SED | Soil 2 | Soil 3 | Soil 4 |
|---|---|---|---|---|---|
| AXYS ID | L13143-1 | L13143-10 | L13143-2 | L13143-3 (A) | L13143-4 |
| WORKGROUP | WG29635 | WG29635 | WG29635 | WG29635 | WG29635 |
| Sample Size | 5.14 g (dry) | 4.89 g (dry) | 5.09 g (dry) | 4.95 g (dry) | 5.19 g (dry) |
| UNITS | ng/g (dry weight basis) | ng/g (dry weight basis) | ng/g (dry weight basis) | ng/g (dry weight basis) | ng/g (dry weight basis) |
| PFBA | 2.45 | 1.25 | 0.985 | 0.203 | < 0.0964 |
| PFPeA | 0.419 | < 0.102 | < 0.0982 | < 0.101 | < 0.0964 |
| PFHxA | 0.682 | < 0.102 | 0.205 | < 0.101 | 0.233 |
| PFHpA | 0.189 | < 0.102 | 0.115 | < 0.101 | < 0.0964 |
| PFOA | 1.18 | 0.726 | 0.381 | < 0.101 | 0.172 |
| PFNA | 0.342 | < 0.102 | < 0.0982 | < 0.101 | < 0.0964 |
| PFDA | 0.642 | < 0.102 | < 0.0982 | < 0.101 | 0.097 |
| PFUnA | 2.46 | < 0.102 | 0.341 | < 0.101 | 1.88 |
| PFDoA | 1.27 | < 0.102 | 0.343 | < 0.101 | < 0.0964 |
| PFBS | 0.296 | < 0.204 | < 0.196 | < 0.202 | < 0.193 |
| PFHxS | 20.6 | < 0.204 | 2.07 | < 0.202 | 3.91 |
| PFOS | 258 | 1.67 | 59.1 | < 0.202 | 355 |
| PFOSA | 8.91 | < 0.102 | 2.99 | < 0.101 | 16.5 |
| % Moisture | 66.8 | 46 | 32.4 | 24.3 | 20.7 |

| Soil 5 | SED 1 | SED 2 | SED 3 | SED 4 | Lab Blank |
|---|---|---|---|---|---|
| L13143-5 | L13143-6 | L13143-7 | L13143-8 | L13143-9 | WG29635-101 |
| WG29635 | WG29635 | WG29635 | WG29635 | WG29635 | WG29635 |
| 5.19 g (dry) | 5.18 g (dry) | 4.56 g (dry) | 4.83 g (dry) | 4.20 g (dry) | 5.00 g |
| ng/g (dry weight basis) | ng/g (dry weight basis) | ng/g (dry weight basis) | ng/g (dry weight basis) | ng/g (dry weight basis) | ng/g |
| 3.82 | 0.659 | 3.37 | 14.2 | 2.35 | < 0.100 |
| 0.628 | < 0.0965 | 0.195 | 1.94 | 0.265 | < 0.100 |
| 0.477 | < 0.0965 | 0.19 | 1.32 | 0.143 | < 0.100 |
| 0.266 | < 0.0965 | < 0.110 | 0.608 | < 0.119 | < 0.100 |
| 8.29 | 0.406 | 0.957 | 14.6 | 1.49 | < 0.100 |
| < 0.0964 | < 0.0965 | 0.113 | < 0.104 | < 0.119 | < 0.100 |
| < 0.0964 | < 0.0965 | < 0.110 | < 0.104 | 0.331 | < 0.100 |
| 0.122 | < 0.0965 | 0.165 | < 0.104 | 0.657 | < 0.100 |
| 0.128 | < 0.0965 | 0.713 | 0.188 | 1.24 | < 0.100 |
| 0.199 | < 0.193 | 0.284 | < 0.207 | < 0.238 | < 0.200 |
| 0.712 | < 0.193 | 1.65 | 0.764 | 0.596 | < 0.200 |
| 7.48 | 1.15 | 104 | 16.3 | 13.6 | < 0.200 |
| 0.428 | < 0.0965 | 0.782 | < 0.104 | 0.325 | < 0.100 |
| 63.1 | 36.2 | 77.3 | 65.5 | 79 | |

| Spiked Matrix WG29635-102 WG29635 | Soil 3 (MS) WG29635-103 WG29635 4.86 g (dry) | Soil 3 (MS) WG29635-103 WG29635 4.86 g (dry) | Soil 3 (MSD) WG29635-104 WG29635 4.93 g (dry) | Soil 3 (MSD) WG29635-104 WG29635 4.93 g (dry) |
|---|---|---|---|---|
| % Recov | ng/g (dry weight basis) | % Recov | ng/g (dry weight basis) | % Recov |
| 119 | 54.3 | 105 | 51.3 | 101 |
| 105 | 51.2 | 99.5 | 50.2 | 98.9 |
| 111 | 49.9 | 97.1 | 50.3 | 99.2 |
| 102 | 54.9 | 107 | 52.8 | 104 |
| 125 | 52 | 101 | 52.7 | 104 |
| 114 | 50.3 | 97.7 | 55.1 | 109 |
| 110 | 50.7 | 98.6 | 50.8 | 100 |
| 89.7 | 40.5 | 78.7 | 44 | 86.6 |
| 114 | 44.4 | 86.4 | 42.8 | 84.3 |
| 139 | 101 | 97.9 | 114 | 112 |
| 129 | 106 | 103 | 116 | 115 |
| 109 | 96.3 | 93.6 | 105 | 103 |
| 130 | 47.8 | 92.8 | 53.5 | 105 |
| | 25.7 | 25.7 | 24.7 | 24.7 |

| CLIENT ID | Field Blank | SW 1 | SW 2 | Duplicate water | Lab Blank | Spiked Matrix | SW 1 (MS) | SW 1 (MS) |
|---|---|---|---|---|---|---|---|---|
| AXYS ID | L13142-2 | L13142-3 (A) | L13142-4 | L13142-5 | WG29657-101 | WG29657-102 | WG29657-103 | WG29657-103 |
| WORKGROUP | WG29657 | WG29657 | WG29657 | WG29657 | WG29657 | WG29657 | WG29657 | WG29657 |
| Sample Size | 0.242 L | 0.496 L | 0.495 L | 0.501 L | 0.500 L | | 0.495 L | 0.495 L |
| UNITS | ng/L | ng/L | ng/L | ng/L | ng/L | % Recov | ng/L | % Recov |
| PFBA | < 5.16 | 1230 | 436 | 572 | < 2.50 | 93.8 | 1660 | 84 |
| PFPeA | < 5.16 | 64.3 | 36.1 | 39.4 | < 2.50 | 87.8 | 517 | 89.6 |
| PFHxA | < 5.16 | 34.5 | 26.9 | 28.1 | < 2.50 | 90.9 | 533 | 98.6 |
| PFHpA | < 5.16 | 12 | 9.43 | 9.92 | < 2.50 | 84 | 546 | 106 |
| PFOA | < 5.16 | 242 | 78.3 | 87.5 | < 2.50 | 91.6 | 723 | 95.2 |
| PFNA | < 5.16 | < 2.52 | 3.37 | < 2.50 | < 2.50 | 94.3 | 493 | 97.5 |
| PFDA | < 5.16 | < 2.52 | < 2.53 | < 2.50 | < 2.50 | 102 | 519 | 103 |
| PFUnA | < 5.16 | < 2.52 | < 2.53 | < 2.50 | < 2.50 | 90.5 | 418 | 82.7 |
| PFDoA | < 5.16 | < 2.52 | < 2.53 | < 2.50 | < 2.50 | 97.8 | 546 | 108 |
| PFBS | < 10.3 | 20.7 | 9.42 | 10.3 | < 5.00 | 87.9 | 1040 | 101 |
| PFHxS | < 10.3 | 32.4 | 7.4 | 10.8 | < 5.00 | 95.1 | 1150 | 111 |
| PFOS | < 10.3 | < 5.04 | < 5.06 | 7.64 | < 5.00 | 97.3 | 1050 | 104 |
| PFOSA | < 5.16 | < 2.52 | < 2.53 | < 2.50 | < 2.50 | 105 | 602 | 119 |

| | |
|---|---|
| SW 1 (MSD) | SW 1 (MSD) |
| WG29657-104 | WG29657-104 |
| WG29657 | WG29657 |
| 0.496 L | 0.496 L |
| ng/L | % Recov |
| 1650 | 83.3 |
| 506 | 87.8 |
| 537 | 99.8 |
| 554 | 108 |
| 721 | 95.2 |
| 487 | 96.7 |
| 504 | 100 |
| 427 | 84.8 |
| 575 | 114 |
| 970 | 94.2 |
| 980 | 94 |
| 970 | 96.2 |
| 540 | 107 |

**Campbell, Fred (MPCA)**

From: Fellows, Nile (MPCA)
Sent: Thursday, November 04, 2010 11:00 AM
To: Krueger, Gary (MPCA); Campbell, Fred (MPCA)
Subject: FW: RE: Water samples submitted on 7/29/09

-----Original Message-----
From: pcarter@wcec.com [mailto:pcarter@wcec.com]
Sent: Tuesday, September 22, 2009 11:31 AM
To: Nile.Fellows@state.mn.us
Subject: Fwd: RE: Water samples submitted on 7/29/09

----- Forwarded message from "Virginia Yingling (MDH)" <Virginia.Yingling@state.mn.us> -----
Date: Mon, 14 Sep 2009 12:58:28 -0500
From: "Virginia Yingling (MDH)" <Virginia.Yingling@state.mn.us>
Reply-To: "Virginia Yingling (MDH)" <Virginia.Yingling@state.mn.us>
Subject: RE: Water samples submitted on 7/29/09
To: "pcarter@wcec.com" <pcarter@wcec.com>, "Jill Korinek (MDH)" <Jill.Korinek@state.mn.us>

Hi Paul:

The results are listed below. However, the Smallidge well that you sampled, I believe, was their irrigation well (UN 224647), not their residential well, which was sealed this spring (UN 257609). The last well listed in your email (egdillams, 56709)was a duplicate for Smallidge.

Smallidge: PFOA = 0.05 ppb, PFBA = 1 ppb, PFPeA = 0.05 ppb

107045 Zyweic IRR-1: PFBA = 1.2 ppb, PFPeA = 0.05 ppb

457148 " IRR-2: PFBA = 0.4 ppb

151779 " IRR-3: PFOA = 0.06 ppb, PFBA = 2.1 ppb, PFPeA = 0.1 ppb, PFHxA = 0.06 ppb

56709? Egdillams: PFOA = 0.06 ppb, PFBA = 1.1 ppb

Ginny

-----Original Message-----
From: pcarter@wcec.com [mailto:pcarter@wcec.com]
Sent: Monday, September 14, 2009 10:27 AM
To: Jill Korinek (MDH); Virginia Yingling (MDH)
Subject: Water samples submitted on 7/29/09

Ginny/Jill, we collected water samples from 4 irrigation wells near the Langdon, C.Grove, neighborhood on 7/29/09. I am writing a report for the Up North

Plastics/2002 fire foam job we completed for Nile Fellows. Do we have any results for those samples? The ID/Sample Source are:

257609 Smallidge
107045 Zyweic IRR-1
457148 " IRR-2
151779 " IRR-3
56709? egdillams

Thanks for any help

Paul Carter, Sr. Project Manager, PG
West Central Environmental Consultants, Inc.
7871 Hickory St. NE, Fridley, MN 55432
phone: 763-571-4944
fax: 763-571-2267
email: pcarter@wcec.com

!DSPAM:10,4aae8489176222958492736!

----- End forwarded message -----

Paul Carter, Sr. Project Manager, PG
West Central Environmental Consultants, Inc.
7871 Hickory St. NE, Fridley, MN 55432
phone: 763-571-4944
fax: 763-571-2267
email: pcarter@wcec.com

## TABLE 5

Groundwater and Surface Water Analytical Results, PFCs
Minnesota Fire Foam Training and Discharge Sites
Delta Project No. 1936[illegible]DEL08

| | | | Perfluorobutanoic acid (PFBA) | Perfluoro-n-pentanoic acid (PFPeA) | Perfluorohexanoic acid (PFHxA) | Perfluoroheptanoic acid (PFHpA) | Perfluorooctanoic acid (PFOA) | Perfluorononanoic acid (PFNA) | Perfluorodecanoic acid (PFDA) | Perfluoroundecanoic acid (PFUnA) | Perfluorododecanoic acid (PFDoA) | Perfluorobutane sulfonate (PFBS) | Perfluorohexane sulfonate (PFHxS) | Perfluorooctane sulfonate (PFOS) | Perfluorooctane sulfonamide (PFOSA) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | #Perfluorinated Carbon Chains: | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 4 | 6 | 8 | 8 |
| | | Health-Based Limits: | 7000 | ND | ND | ND | 300 | ND | ND | ND | ND | 7000 | RAA | 300 | ND |
| WAFTA, St. Bonifacius | | | | | | | | | | | | | | | |
| Training Frequency: | unknown | | | | | | | | | | | | | | |
| Last Training Event: | [illegible] | | | | | | | | | | | | | | |
| Foam Usage per Training Event: | unknown | | | | | | | | | | | | | | |
| Foam Brand: | unknown | | | | | | | | | | | | | | |
| Sample ID | Sample Date | Laboratory | | | | | | | | | | | | | |
| WAFTA SG-2 | 5/11/2006 | MDH | < 1000 | < 1000 | < 1000 | NA | 1000 | NA | NA | NA | NA | < 500 | 200 | < 500 | NA |
| WAFTA SG-4 | 5/11/2006 | MDH | 980 | 2200 | 2300 | NA | 2100 | NA | NA | NA | NA | < 500 | 2100 | 2300 | NA |
| WAFTA MW-1 | 5/11/2006 | MDH | < 1000 | < 1000 | 300 | NA | 3400 | NA | NA | NA | NA | < 500 | 200 | < 500 | NA |
| WAFTA MW-2 | 5/11/2006 | MDH | 3400 | 8500 | 7500 | NA | 7500 | NA | NA | NA | NA | 400 | 9800 | 5000 | NA |
| WAFTA MW-3 | 5/10/2006 | MDH | < 1000 | < 1000 | 380 | NA | < 1000 | NA | NA | NA | NA | 200 | 5100 | 23000 | NA |
| WAFTA MW-4 | 5/10/2006 | MDH | 5900 | 42000 | 20000 | NA | 43000 | NA | NA | NA | NA | 1900 | 42000 | 110000 | NA |
| WAFTA MW-4 | 5/10/2006 | Koppen | 54100 | 65300 | 41400 | NA | 41100 | NA | NA | NA | NA | 1820 | 43400 | 114000 | NA |
| WAFTA MW-5 | 5/10/2006 | MDH | < 1000 | 260 | 360 | NA | 780 | NA | NA | NA | NA | < 500 | 700 | 2100 | NA |
| WAFTA MW-5 | 5/10/2006 | Koppen | < 1000 | < 1000 | < 1000 | NA | < 1000 | NA | NA | NA | NA | < 1000 | < 1000 | 1400 | NA |
| WAFTA MW-7 | 5/11/2006 | MDH | 1200 | 3400 | 3400 | NA | 9000 | NA | NA | NA | NA | 200 | 2300 | 3800 | NA |
| WAFTA MW-8 | 5/10/2006 | MDH | 90 | 480 | 380 | NA | 180 | NA | NA | NA | NA | < 500 | < 500 | 1300 | NA |
| WAFTA MW-8 | 5/10/2006 | Koppen | < 1000 | < 1000 | < 1000 | NA | < 1000 | NA | NA | NA | NA | < 1000 | < 1000 | < 1000 | NA |
| WAFTA MW-9 | 5/11/2006 | MDH | < 1000 | < 1000 | < 1000 | NA | < 1000 | NA | NA | NA | NA | < 500 | < 500 | < 500 | NA |
| WAFTA MW-10 | 5/10/2006 | MDH | 780 | 3300 | 3000 | NA | 2300 | NA | NA | NA | NA | 800 | 13000 | 27000 | NA |
| WAFTA MW-10 | 5/10/2006 | Koppen | < 1000 | 3300 | 3300 | NA | 2270 | NA | NA | NA | NA | < 1000 | 11600 | 18400 | NA |
| WAFTA MW-11 | 5/10/2006 | MDH | < 1000 | < 1000 | < 1000 | NA | < 1000 | NA | NA | NA | NA | < 500 | < 500 | < 500 | NA |
| WAFTA MW-11 | 5/10/2006 | Koppen | < 1000 | < 1000 | < 1000 | NA | < 1000 | NA | NA | NA | NA | < 1000 | < 1000 | < 1000 | NA |
| WAFTA MW-12 | 5/11/2006 | MDH | < 1000 | < 1000 | < 1000 | NA | < 1000 | NA | NA | NA | NA | < 500 | < 500 | < 500 | NA |
| WAFTA MW-13 | 5/10/2006 | MDH | < 1000 | < 1000 | < 1000 | NA | < 1000 | NA | NA | NA | NA | < 500 | 300 | < 500 | NA |

TABLE 5

Groundwater and Surface Water Analytical Results, PFCs

Minnesota Fire Foam Training and Discharge Sites

Delta Project No. [illegible]DEL08

| | | | Perfluorobutanoic acid (PFBA) | Perfluoro-n-pentanoic acid (PFPeA) | Perfluorohexanoic acid (PFHxA) | Perfluoroheptanoic acid (PFHpA) | Perfluorooctanoic acid (PFOA) | Perfluorononanoic acid (PFNA) | Perfluorodecanoic acid (PFDA) | Perfluoroundecanoic acid (PFUnA) | Perfluorododecanoic acid (PFDoA) | Perfluorobutane sulfonate (PFBS) | Perfluorohexane sulfonate (PFHxS) | Perfluorooctane sulfonate (PFOS) | Perfluorooctane sulfonylamide (PFOSA) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | #Perfluorinated Carbon Chains: | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 4 | 6 | 8 | 8 |
| | | Health Based Limits: | 7000(1) | ND | ND | ND | 300(2) | ND | ND | ND | ND | 7000(2) | RAA(4) | 300(3) | ND |
| Lp North Plastics Fire | | | | | | | | | | | | | | | |
| Date of Foam Discharge: | December 2002 | | | | | | | | | | | | | | |
| Foam Usage: | 4,000 gallons or more | | | | | | | | | | | | | | |
| Foam Brand: | unknown | | | | | | | | | | | | | | |
| Sample ID | Sample Date | Laboratory | | | | | | | | | | | | | |
| Lp North Plastics SW-1 | 1/15/2008 | Axys | 1230 | 64.2 | 34.8 | 12 | 242 | < 2.52 | < 2.52 | < 2.52 | < 2.52 | 28.7 | 30.4 | < 5.04 | < 2.52 |
| Lp North Plastics SW-2 | 1/15/2008 | Axys | 436 | 36.1 | 26.8 | 8.43 | 78.3 | 3.37 | < 2.83 | < 2.83 | < 2.83 | 8.42 | 7.4 | < 5.66 | < 2.83 |
| Lp North Plastics SW Dup | 1/15/2008 | Axys | 573 | 39.4 | 28.1 | 9.92 | 67.5 | < 2.50 | < 2.50 | < 2.50 | < 2.50 | 18.3 | 14.8 | 7.64 | < 2.50 |
| Lp North Plastics Zywiec Irrigation Well 1 | 7/20/2006 | MDH | 1342.3 | 51.4 | [illegible] | NA | [illegible] | NA | NA | NA | NA | 0 | 0 | 0 | NA |
| Lp North Plastics Zywiec Irrigation Well 2 | 7/20/2006 | MDH | 447 | [illegible] | [illegible] | NA | [illegible] | NA | NA | NA | NA | 0 | 0 | 0 | NA |
| Lp North Plastics Zywiec Irrigation Well 3 | 7/20/2006 | MDH | 2153.6 | 166.2 | 61 | NA | 56 | NA | NA | NA | NA | 0 | 0 | 0 | NA |
| Lp North Plastics Strawbridge | 7/20/2006 | MDH | 1946.1 | 51.6 | [illegible] | NA | 61.3 | NA | NA | NA | NA | 0 | 0 | 0 | NA |

Notes:

All results and standards are in nanograms per liter (ng/L), which is equivalent to parts per trillion.

Axys: Axys Analytical Services LTD

MPI: MPI Research

MDH: Minnesota Department of Health Environmental Laboratory

Exygen: Exygen Research

**Bolded** type indicates detection above the laboratory method detection limit.

Highlighted concentrations exceed the HBV or HRL.

(T) Last training event prior to sampling, dates are approximate

(1) Health-Based Value (HBV) for chronic exposure defined by the Minnesota Department of Health.

(2) Health Risk Limit (HRL) for drinking water defined by the Minnesota Department of Health.

(4) Risk Assessment Advise (RAA) set by the Minnesota Department of Health for PFHxS does not specify numeric values.

ND: No health-based limit defined.

(5) Manually Calculated Result is 18.9

(6) Manually Calculated Result is 17.1

(7) Manually Calculated Result is 23.3

(8) Manually Calculated Result is 21.7

(J) Analyte positively identified, result is below reporting limit and is estimated.

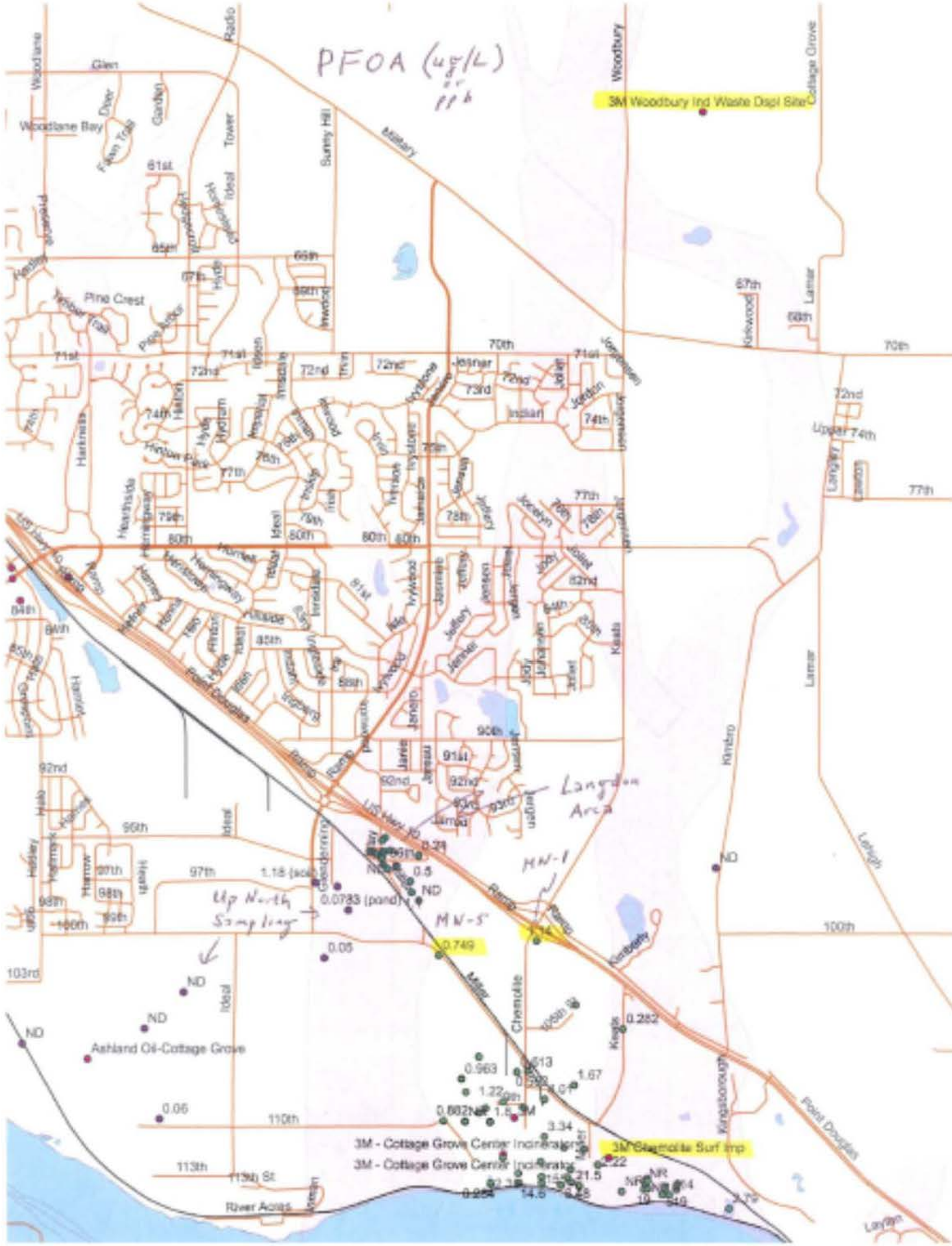
*Sample collected upgradient of fire foam training or discharge area, intended to act as "background" sample.

NA: Not analyzed

DELTA

**Table 3: 3M-Woodbury Disposal Site Groundwater, 2007-2008 (in µg/L)**

| Well | Date | PFBA | PFPeA | PFHxA | PFHpA | PFOA | PFBS | PFHxS | PFOS |
|---|---|---|---|---|---|---|---|---|---|
| B-1 | June 2007 | 1.59 | 0.487 | 0.974 | 0.143 | 1.44 | 1.73 | 1.52 | ND |
| | March 2008 | 1.79 | 0.509 | 0.847 | 0.128 | 1.2 | 1.75 | 1.39 | 0.041 |
| B-2 | June 2007 | 0.471 | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | 0.536 | ND | ND | ND | ND | ND | ND | ND |
| B-3 | June 2007 | 0.728 | 0.074 | 0.119 | ND | 0.207 | 0.362 | 1.29 | 0.171 |
| | March 2008 | 0.769 | 0.0672 | 0.0946 | ND | 0.0202 | 0.429 | 1.43 | 0.156 |
| B-4 | June 2007 | 1.5 | 0.406 | 0.96 | 0.342 | 2.44 | 3.48 | 11.5 | 1.78 |
| | March 2008 | 1.69 | 0.447 | 0.837 | 0.388 | 2.94 | 3.84 | 13.9 | 3.06 |
| MW-2 | June 2007 | 126 | 9.31 | 13 | NR | 4.92 | 14.4 | 4.65 | ND |
| | March 2008 | 71.5 | 9.14 | 8.14 | 1.51 | 3.47 | 9.15 | 2.06 | 0.0361 |
| MW-4 | June 2007 | 0.809 | 0.062 | 0.0537 | 0.0357 | 0.0401 | 0.216 | 0.433 | 0.172 |
| | March 2008 | 0.873 | 0.0586 | 0.0317 | ND | 0.0269 | 0.124 | 0.267 | 0.104 |
| MW-G | June 2007 | 0.127 | ND | ND | ND | ND | ND | ND | 0.114 |
| | March 2008 | 0.201 | ND | ND | ND | ND | ND | ND | ND |
| MW-H | June 2007 | ND | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | ND | ND | ND | ND | ND | ND | ND | ND |
| S01JS | June 2007 | ND | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | 0.0395 | ND | NR | ND | ND | ND | ND | ND |
| S01PC | June 2007 | 0.85 | 0.0347 | 0.0367 | ND | ND | ND | ND | ND |
| | March 2008 | 0.942 | 0.0398 | 0.12 | ND | ND | ND | ND | ND |
| S02DR | June 2007 | 0.594 | 0.0252 | ND | ND | 0.033 | ND | 0.0373 | ND |
| | March 2008 | 0.647 | ND | ND | ND | 0.0326 | ND | 0.0499 | ND |
| S02JS | June 2007 | ND | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | 0.0360 | ND | ND | ND | ND | ND | ND | ND |
| S02PC | June 2007 | 1.69 | 0.0573 | 0.0255 | ND | 0.0286 | ND | 0.0526 | ND |
| | March 2008 | 1.2 | 0.0384 | 0.0403 | ND | ND | ND | 0.0435 | ND |
| S03JS | June 2007 | 0.272 | ND | ND | ND | ND | ND | ND | ND |
| | March 2008 | 0.233 | ND | ND | ND | ND | ND | ND | ND |


PFOA (ug/L)
3M Woodbury Ind Waste Displ Site
Langdon Area
MW-1
MW-5
Up North Sampling
0.749
0.05
0.06
0.282
1.67
0.963
3.34
21.5
14.5
ND
Ashland Oil-Cottage Grove
3M - Cottage Grove Center Incinerators
3M - Cottage Grove Center Incinerator
3M Chemolite Surf Imp
Woodbury
Cottage Grove
Military
Radio
Glen
Woodlane
Woodlane Bay
Sunny Hill
Tower
Ideal
Pine Crest
70th
72nd
77th
80th
84th
86th
90th
92nd
95th
97th
100th
103rd
110th
113th
113th St
River Acres
Jamaica
Kimbro
Lamar
Leigh
Point Douglas
Kingsborough
Chemolite
US Hwy 10
Lehigh

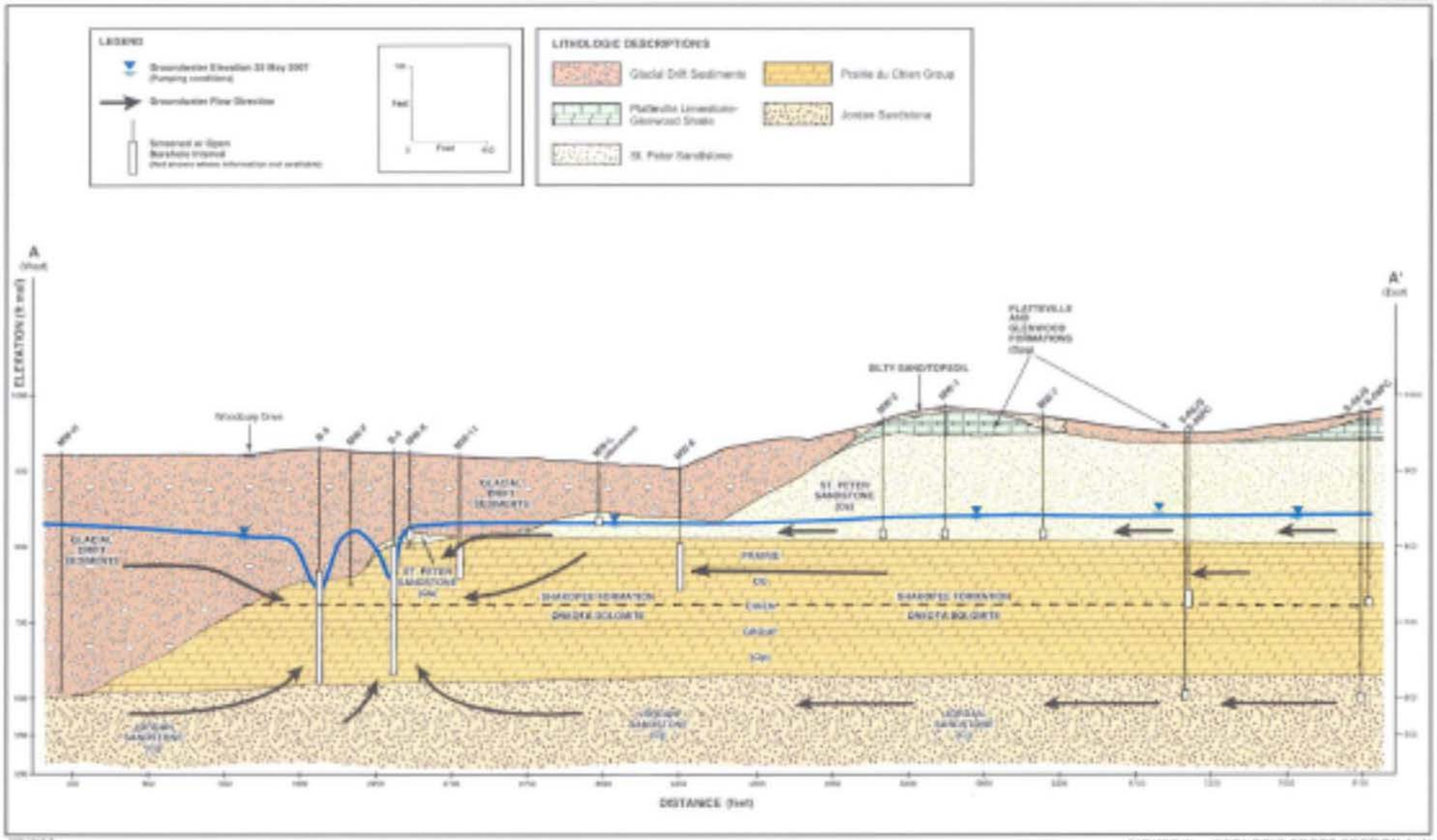
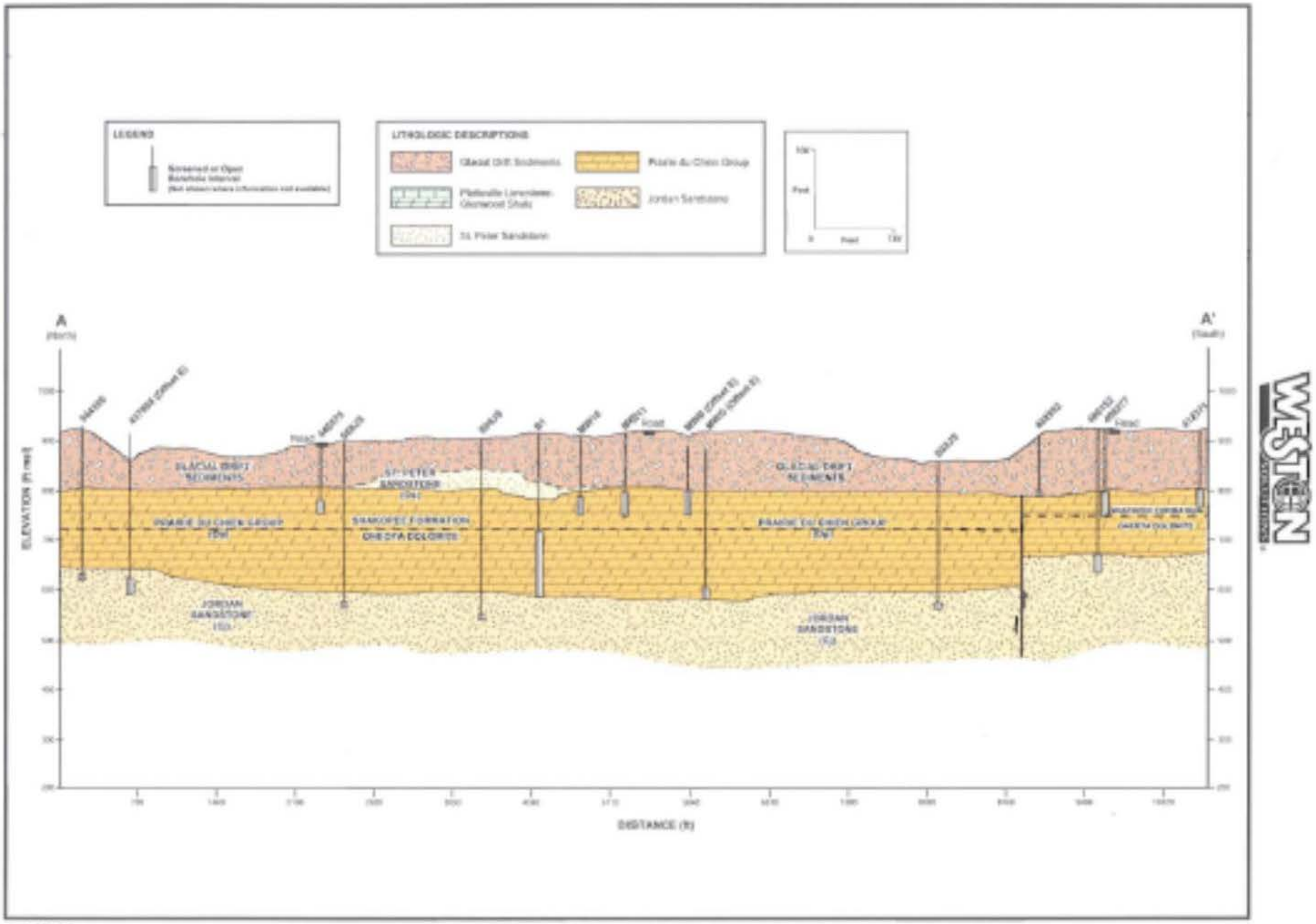
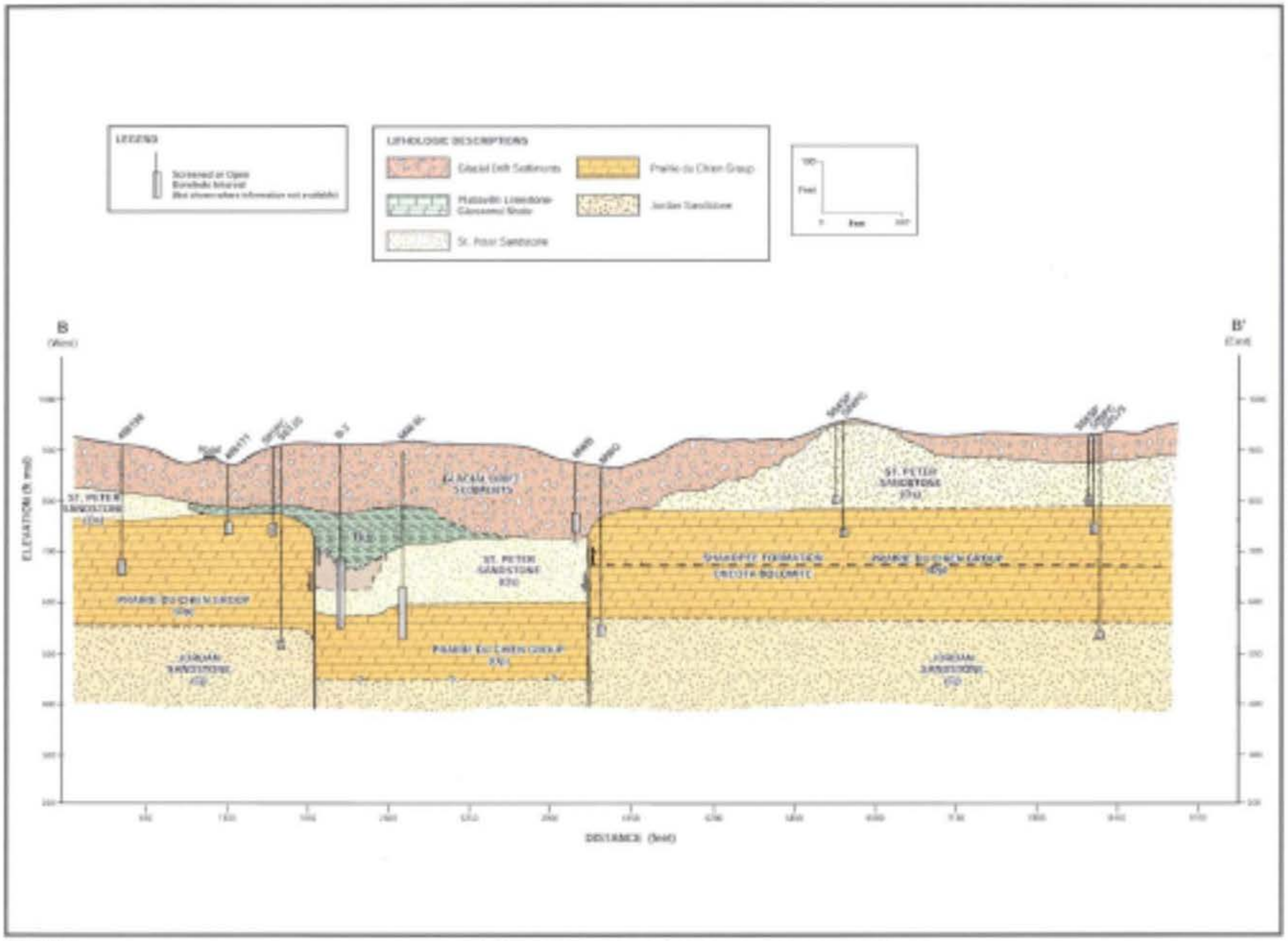



FIGURE 3 GEOLOGIC CROSS SECTION A-A'
3M WOODBURY SITE
WOODBURY, MN




07P-1041-5

**FIGURE 4-2 GEOLOGIC CROSS SECTION A-A'**
**3M WOODBURY SITE**
**WOODBURY, MN**




07P-1041-6

**FIGURE 4-3 GEOLOGIC CROSS SECTION B-B'**
**3M WOODBURY SITE**
**WOODBURY, MN**

# COMMENTS OF 3M COMPANY

# ON MINNESOTA DEPARTMENT OF HEALTH'S

# DRAFT PUBLIC HEALTH ASSESSMENT ON PERFLUOROCHEMICALS IN SOUTHERN WASHINGTON COUNTY, NORTHERN DAKOTA COUNTY, AND SOUTHEAST RAMSEY COUNTY, MINNESOTA

**October 12, 2010**

3M Company appreciates the opportunity to comment on the draft Public Health Assessment dated August 25, 2010, prepared by the Minnesota Department of Health ("MDH") for the Agency for Toxic Substances and Disease Registry ("ATSDR"). The draft document addresses the presence of perfluorochemicals ("PFCs") in Southern Washington County, Northern Dakota County, and Southeast Ramsey County, Minnesota. We appreciate the efforts of both MDH and ATSDR in preparing this draft document.

In these comments, 3M attempts to point out additional information that MDH may wish to consider. We respectfully offer the following comments on the conclusions, recommendations and text of the draft report in an effort to assist you in making the document as accurate and useful as possible. Our comments below are directed to specific pages, with the titles corresponding to the titles in the draft assessment. As authors or sponsor of many of the studies discussed in the draft assessment, we would be pleased to answer any questions or to provide any additional information that would be helpful.

By way of preface, we note that although the draft assessment states that it was prepared pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act ("CERCLA") and its implementing regulations, PFCs are not hazardous substances under CERCLA or its Minnesota counterpart, the Minnesota Environmental Response and Liability Act ("MERLA"). 3M recognizes, however, that ATSDR and MDH nonetheless have authority to issue health assessments under CERCLA, and we appreciate the agencies' efforts to communicate accurate information to the public.

## COMMENTS

### Comments on the "Summary" Section

#### "Introduction" Heading

MDH states on page 4 that its goal is "to protect people's health by providing health information the community needs to take actions to protect their health." Consistent with MDH's expressed goal, the Public Health Assessment should clarify that the presence of PFCs in drinking water at trace levels does not equate to harm.

MDH should also use the Introduction to consolidate details about PFC findings that are currently spread throughout the draft assessment. For example, the summary states PFCs "continue to be detected in public and private wells," but does not explain that:

- Wherever monitoring of a drinking water supply has indicated PFC levels above established standards, advisories have been issued and alternatives have been offered to ensure safe supplies of water.

- MDH's HRLs and HBVs are based not on any health effects observed in humans, but on laboratory animal data with ample safety factors.

- Blood levels of PFOS and PFOA in local residents exposed via drinking water are in the parts per billion. 3M's chemical production workers have had parts per million levels in their blood; they have been studied for over thirty years, and taken together the studies do not show that this exposure has caused adverse health effects. Even if certain PFCs persist in the body, as the Centers for Disease Control state, "[t]he measurement of an environmental chemical in a person's blood or urine is an indication of exposure; it does not by itself mean that the chemical causes disease or an adverse effect." *Fourth National Report on Human Exposure to Environmental Chemicals*, p. 1 (2009). Adverse effects do not occur in laboratory animals until the animals are exposed to parts per million of PFOS or PFOA -- levels significantly higher than the levels found in people exposed via drinking water in Minnesota.

Finally, 3M questions why MDH states in the summary that there were possible air emissions during the handling, disposal, or burning of PFC-containing wastes at disposal sites and that people could have come into direct contact with the waste. These theoretical possibilities lack support, and their inclusion in the summary does not seem warranted.

**"Conclusion 1," "Basis For Decision," and "Next Steps"**

MDH concludes that current levels in local residents' blood are unlikely to cause adverse health effects, but that there is insufficient information to determine whether historical levels of PFCs in local water, air, or wastes harmed people's health. The conclusion with respect to possible past harm in unnecessarily speculative.

As MDH observed in its *Statement of Need and Reasonableness (SONAR) In the Matter of the Proposed Rules Relating to Health Risk Limits for Groundwater* (Aug. 2010, at p. 33), "a contaminant concentration above an HRL, without consideration of other information, may not necessarily indicate a public health problem." Thus, even if past levels exceeded HRLs, that does not imply that there was ever a risk to health.

While it is true the precise historical levels of PFC exposure are not known, there is important context missing from the conclusion in the draft document. It is known that 3M's chemical production workers had PFC blood serum levels one to two (or sometimes three) orders of magnitude higher than recent levels in persons who have consumed PFCs in drinking water in Minnesota. It is unlikely that exposure via water, air or waste (if any), would have led to higher blood levels than the exposure of the chemical production workers working directly with PFC materials for many years. It is also known that serum levels in the Minnesota population are lower than the levels found in a community in Parkersburg, West Virginia where PFOA was present in drinking water.

Moreover, as ATSDR has stated, "it is difficult to envision a health condition that could be attributed solely to exposure to perfluoroalkyls." *Draft Toxicological Profile for Perfluoroalkyls*, p. 207 (2009). 3M has monitored the health of its chemical production workers for thirty years. The large body of information about PFC health effects includes mortality studies in multiple cohorts, and other studies of potential health effects in workers and the general population - and the totality of the evidence does not show that PFCs have ever caused any health effect in humans. There is also a robust toxicological database, which has clearly shown the range of serum levels at which various endpoints seen in laboratory animals begin to occur. Pharmacokinetic and mode of action data are also available from the toxicological studies. These studies, along with the ability to compare biomonitoring data for humans versus the levels that produce effects in laboratory animals, provide an ample basis for MDH to conclude that historical exposure to PFCs in Minnesota water, air or waste (if any) is unlikely to have harmed local residents' health.

MDH recommends further biomonitoring. It would be useful to explain that there is an unmistakable downward trend in the levels of PFCs found in the U.S. general population since 3M's phaseout of production of these materials. For example, as detailed later in these comments, Centers for Disease Control ("CDC") data indicate mean blood levels of PFOS in the general population have declined by more than 50% since their peak, and $95^{th}$ percentile values have declined even more. PFOA levels have also declined, although less so given continued production and use by others.

**"Conclusion 2," "Basis for Decision," "Next Steps"**

3M agrees with Conclusion 2 (*i.e.*, that drinking water from public or private wells that contain PFCs is not expected to harm people's health). As MDH notes, no one is drinking water that has PFCs at a level posing a health concern. However, for the public to appreciate the meaning of this conclusion, it is important to explain in the document that MDH's health-based limits (HRLs or HBVs) are set many times below the levels at which effects begin to occur in laboratory animal studies. The public may not appreciate that MDH's reference to levels posing a "health concern" actually refers to values that are based on laboratory animal studies and which then incorporate substantial safety factors to account for any uncertainty and the possibility of multiple routes of exposure:

- The HBV for PFBA includes an uncertainty factor of 100 and dose metric of 8; as a result, it is set at 800 times below the level at which effects began to occur in laboratory animal studies.

- The HRL for PFOS is set using an uncertainty factor of 100 and a dose metric of 20, i.e., at a level 2,000 times below the level at which effects begin to occur in laboratory animal studies.

- For PFOA, the HRL is set using an uncertainty factor of 300 and a dose metric of 70, putting it at a level 21,000 times below the level at which effects begin to occur in laboratory animal studies.

- For all of the above standards, the "relative source contribution" is set at 20%. This means that only 20% of the allowable exposure is being allocated to the drinking water pathway. Since there are few, if any, other significant pathways for these constituents today, this also represents a very conservative approach.

These calculations are explained later in the draft assessment, but it is important to provide necessary context in the summary to explain that the levels of "concern" already incorporate a substantial margin of protection.

On page 5, under the recommended Next Steps, the second bullet point recommends that people avoid trespassing on the 3M-Woodbury Disposal Site. It is inappropriate to cast this as a recommendation without acknowledging that the site has long been fenced and posted with No Trespassing signs. It would be appropriate to recommend that people should obey 3M's posted No Trespassing signs. Or, the bullet point could be revised to state "Consistent with 3M's long-standing recommendations, people should continue to avoid trespassing on the 3M-Woodbury Disposal Site."

Also on page 5, 3M questions why MDH identifies as a next step considering extending the Cottage Grove municipal water supply to serve areas where private wells contain levels of PFCs in excess of HRLs or HBVs. On page 28, MDH notes that in the few locations where well advisories have been issued, filters have been used that are effective at removing PFCs from drinking water.

MDH's final recommendation is that 3M should ensure that the monitoring network at the Woodbury disposal site provides adequate information on water quality in the high transmissivity zone of the Prairie du Chien aquifer. The monitoring well network at the site is in fact sufficient to monitor this zone. Indeed, the data demonstrate that the barrier well network is effectively capturing the PFC-containing groundwater at the site, and in particular is effective in capturing the groundwater from the high transmissivity zone.

As discussed in the 2007 and 2008 hydraulic evaluation reports for the Woodbury Site, a zone near the contact between the upper Shakopee Formation and lower Oneota Dolomite of the Prairie du Chien Group is likely providing the highest volume of groundwater to the barrier well network. As noted in the draft assessment document, this contact between the two formations is described as the "high transmissivity zone" within the Prairie du Chien Group. At the Woodbury Site, the three highest producing barrier wells (B-1, B-3, and B-4) are all screened across the high transmissivity zone within the Prairie du Chien formation. The average pumping rate for these three wells is 2,950 gallons per minute, which accounts for 96% percent of the total average pumping rate of the entire extraction well system. Thus, the overwhelming majority of groundwater being pumped at the Site is being extracted from the high transmissivity zone.

Several site groundwater monitoring wells were constructed to monitor groundwater conditions within the high transmissivity zone. The MDH statement that only one well (MW-S06PC) monitors this zone is incorrect. There are two additional monitoring wells (MW-4L and MW-5) that monitor this zone, and three of the four barrier wells (B-1, B-3 and B-4) intersect the high transmissivity zone and are also monitored.

Groundwater data were collected during the two hydraulic evaluations performed at the Site in May 2007 and May 2008 to assess the impact of pumping the barrier wells on water levels within the high transmissivity zone. The following highlight key findings from this work:

- Monitoring well MW-5 in the high transmissivity zone is located near barrier wells B-3 and B-4. A decrease of approximately 36.8 feet was measured in the water level in monitoring well MW-5 between periods of non-pumping and pumping of the barrier well network in May 2008.

- In addition, a drawdown of approximately 22.6 feet was measured in barrier well B-3 in the high transmissivity zone after barrier wells B-4 and B-1 had been pumping for two and one hours, respectively. (Nearby monitoring wells that were installed to monitor water levels in the glacial drift, upper Prairie du Chien, and Jordan Sandstone formations showed that water levels in these wells declined approximately 11.7, 11.5, and 5.1 feet, respectively, in response to the operation of the full barrier well network.)

- A similar response in water levels was observed in the water level data collected from monitoring wells MW-4, MW-4L and S-09JS located to the northwest of barrier well B-1. Monitoring well MW-4 is completed within the shallow Prairie du Chien formation, monitoring well MW-4L is completed within the Prairie du Chien formation across or near the high transmissivity zone, and S-09JS is screened within the Jordan Sandstone formation. Groundwater data collected while the barrier well network is operating indicates that the largest decline in water level was in monitoring well MW-4L. In addition, the groundwater elevation of monitoring well MW-4L is consistently lower than the water level in the adjacent shallow (MW-4) and deeper (S-09JS) monitor wells during the operation of the barrier well network. These data confirm that the vertical hydraulic gradient is toward the high transmissivity zone while the barrier well network is in operation.

The water level data collected from monitoring wells MW-5 and MW-4L provide ample demonstration that the effect of pumping of the barrier well network is greatest within the high transmissivity zone. The decline of water levels within the high transmissivity zone in the vicinity of the barrier well network indicates that a significant cone of depression is established in this unit. In addition, the vertical hydraulic gradients across the area suggest that groundwater flow from the overlying and underlying water-bearing units is toward the high transmissivity zone and the barrier well network. This induces groundwater beneath the former disposal areas to flow toward the barrier well network. The effectiveness of the barrier well network has been confirmed by repeated sampling by multiple consultants (including Liesch, Conestoga Rovers, Barr Engineering and Weston) as well as by MPCA.

Based on the above information, 3M believes the final "Next Step" listed in the draft assessment should be removed since the issue has been addressed.

## Comments on the "Introduction" Section

The first paragraph of the Introduction should be revised to clarify 3M's historical and current PFC production. Both here and elsewhere, for purposes of clarity, we suggest dividing the description to address eight-carbon PFCs including PFOA or PFOS and PFOS precursors separately from PFBA and PFBS, which are four-carbon compounds, each with a different history:

- In the 1950s, 3M began commercial production of eight-carbon PFCs. 3M was the only U.S. producer of PFOS-related products, although there were other producers around the world. 3M phased out the production of the eight-carbon products, including PFOA and PFOS-related products, from 2000-2002. Others continue to make or import PFOA-related products in the U.S.

- PFBA, a four-carbon material, was produced by 3M for use in photographic film. 3M produced PFBA at Cottage Grove until 1998. There are many other sources of PFBA.

- Today, for 3M, PFBS serves as the substitute foundational chemistry for many of the eight-carbon materials that are no longer manufactured. The only PFCs 3M currently produces at its Cottage Grove plant are PFBS-related products. (The Cottage Grove plant may also make or handle one- to three-carbon perfluoroalkyl substances, which are generally not referred to as PFCs.)

Thus, the text should not imply that 3M produces or uses eight-carbon PFCs today.

MDH also states on page 5 that 3M "formally provided" information to MPCA in June 2005 about five facilities that received PFC wastes between 1956-1971: the Oakdale Disposal Site, the Woodbury Disposal Site, the Washington County Landfill, the 3M Cottage Grove Facility, and the Pigs Eye disposal site. First, it should be noted that all of the referenced sites were used for legal disposal of wastes. We suggest that MDH reference MPCA's statement with respect to legality of the disposal: MPCA, *3M Oakdale Disposal Site: Proposed Cleanup Plan for PFCs* (May 2008), found at http://www.pca.state.mn.us/index.php/waste/waste-and-cleanup/cleanup-programs-and-topics/topics/perfluorochemicals-pfc/perfluorochemical-pfc-waste-sites.html. Second, the table is incorrectly titled, indicating that the listed wastes in the table on page 6 of the draft document are PFC wastes. The Weston 2005 document to which MDH refers does not indicate that these are all PFC-containing wastes.

Third, as currently written, the draft assessment could be read to suggest MPCA was not aware 3M disposed of PFC wastes at these sites until that information was "formally provided" in June 2005. That is not the case. 3M has a long history of providing MDH, MPCA, and others information about its production of PFCs, its use of sites to dispose waste generated from PFC production, and its research on PFCs. In 2007, 3M provided MPCA with a detailed chronology and collection of documents showing many of these historical communications. We highlight below some of these communications, in support of our request that MDH refrain from any implication in the draft assessment that the State was not aware of 3M's perfluorochemical production and waste disposal.

3M's communications with Minnesota agencies about PFC production date back more than fifty years. In 1958, MDH visited the Cottage Grove plant, performed its own water discharge sampling, and issued a report noting that "[s]pecific limitations have not been recommended for the discharge of fluorides, but the Company was asked to include this determination in the monthly limit." (MDH 1958.) In 1971, MPCA issued 3M an operating permit for its "fluorination cells." (MPCA 1971.) In 1974, MPCA issued 3M an "Installation Permit for Fluorination of Organic Compounds" that noted "[3M's] Production Equipment are three (3) process reaction vessels used to fluorinate organic compounds." (MPCA 1974.) Two years later, 3M told MPCA that "[f]luorides can reasonably be expected in the effluent as a constituent of the manufacturing process." (Susag 1976.) And in 1985, 3M submitted an air permit application to MPCA "for a new fluorochemical distillation system." The application specifically identified expected emission rates for both inert fluorochemicals and PFOA. (Baltutis 1985.) Various subsequent air permit documents also described the fluorochemical production process.

In 1980, Minnesota OSHA inspected the Cottage Grove plant's perfluorochemical production operations and conferred with 3M's Medical Department regarding perfluorochemical issues. (Riehle 1980; Curti 1980.) In connection with this work in 1980, Minnesota OSHA consulted with MDH regarding the toxicity information on these compounds. (Garry 1980.)

3M also communicated with Minnesota regulators about its use of area disposal sites early and often. For example, 3M remediated the Woodbury site in the *1960s* under State supervision. (3M 1961.) In August of 1980, an EPA contractor provided MPCA with a draft report about 3M's use of the Woodbury site which expressly stated "[t]oward the end of use of the disposal pit facility, 3M had begun to make fluorocarbons. Concentrated wastes from fluorocarbon production went into the Platefill [sic - Platteville] limestone pits as well as the various acids. However, this practice was not long lasting." (Neely 1980, at p. 8-9.) The report went on to state: "Based upon analytical results, both 3M and the Minnesota Pollution Control Agency appear to be satisfied that the barrier wells have effectively prevented migration of the contaminants from the Woodbury site." (*Id*. at pp. 8-1 - 8-2.) The report also noted: "When the problem of ground water contamination first appeared in connection with the Woodbury 3M facility, 3M immediately took responsible actions to mitigate and correct the problem. Previous practices were stopped, an investigation was undertaken to determine the extent of the problem, and corrective actions were initiated. . . . In general, the Minnesota Pollution Control Agency has been pleased with the efforts and prompt action of 3M and believe that the barrier well system is an appropriate corrective action." (*Id*. at p. 8-21.)

In 1994, 3M provided MPCA with an Investigation Report for the Woodbury site. (Ashenmacher 1994.) The report expressly stated that "[i]n the 1960s, 3M disposed of industrial waste at a 200 acre site in Woodbury, MN" and that "In addition to target list VOC contaminants, on-site techniques also identified two other types of contaminants found at the site: fluorochemicals and [a heptane hydrocarbon mixture]." The report provided MPCA with analytical data showing that fluorochemicals, including PFOA, PFOS, and PFHS, were present in soil and soil gases at the site, although they were not detected in groundwater with the analytical techniques available at the time (detection limit 25 ppb). In short, it would be misleading to imply that MPCA learned of 3M's disposal of waste at the Woodbury site in June of 2005. Moreover, this report provides important information about historical levels in groundwater, as neither PFOS nor PFOA was detected in site groundwater with a detection limit of 25 parts per billion using then-available technology.

Communications about disposal at the Oakdale site and at the Cottage Grove plant likewise date back many years. In 1981, MPCA told 3M it was investigating a disposal site at the Cottage Grove plant used by 3M for waste disposal, potentially including "drums containing . . . fluorocarbons" disposed of between 1950 and 1955. (MPCA 1981.) The letter asks 3M to provide information about what specific 3M wastes were disposed of at the site. It also asks for information on other sites used by 3M, but indicates the request excludes the Oakdale and Woodbury sites because they were already known to MPCA. 3M complied with MPCA's request. (Susag 1981.) Over the next few years, 3M generated reports about the Cottage Grove site that were shared with MPCA as part of the development and implementation of remediation plans. Those reports included, among other items, a 1981 work plan (Santoro 1981), 1982 site characterization reports on the investigations of waste disposal areas (Pilney 1982), a 1983 hydrogeological investigation report that states that ten fluorinated compounds, including PFOA, were identified in sludge disposal areas (Pilney 1983), and a 1986 remedial investigation report that again included analytical results for PFOA found in sludge samples (Weston 1986), along with a feasibility study addressing remediation options. Also in 1986, MPCA approved 3M's response actions at the Cottage Grove facility. (MPCA 1986.) The MPCA decision document identifies the HF tar neutralization pit, the wastewater sludge disposal area, and other areas at the plant site as areas of known or alleged disposal but states they are not a source of release to ground water.

All of these communications and others have been provided to the State and are summarized in the chronology 3M provided to MPCA in 2007. As evidenced there, not only has 3M been cooperating with both MDH and MPCA about these sites for decades, but 3M and others have published numerous articles about PFCs in the open scientific literature over the past 50 years. As of May 2009, 3M had published 51 articles on the toxicology of PFCs, 26 articles on the environmental behavior of PFCs, 19 articles on studies of PFC workers, 18 articles on studies of PFCs in the general population, and 16 analytical chemistry articles on PFCs. 3M also has a long history of submitting its studies to agency public dockets; in addition to submissions to EPA under the Toxic Substances Control Act, 3M has submitted over 1500 studies to U.S. EPA's AR-226 public docket. Any suggestion in the draft MDH document that information about PFCs was not available lacks factual basis.

On page 6, 3M questions why the description of waste disposed at the Pigs Eye site speculates that the site received municipal waste water treatment plant incinerator ash that "may" have contained PFCs. This statement is unsupported conjecture. Incineration is an effective method for destroying PFCs. (University of Dayton 2003.)

The definition of "Perfluorochemicals" in the second paragraph of page 6 is overly broad in defining PFCs as organic hydrocarbon molecules in which all hydrogens are replaced by fluorine. That definition encompasses the inert perfluorinated carbon chains, such as perfluorobutane, perfluorohexane, perfluorooctane, etc. The perfluorochemicals at issue are the functionalized compounds shown in MDH's Figure 2, containing the perfluorinated carbon chain and a functionalized end group.

Furthermore, two of the three structures shown in Figure 2 are incorrectly drawn for the names given. Perfluorooctanoic acid and perfluorobutyric acid would have acid hydrogens;

otherwise, their names should be changed to perfluorooctanoate and perfluorobutyrate with negative ion signs associated with the single-bonded oxygens.

The third paragraph indicates "unique physical and chemical properties" for PFCs that "allow them to move easily through the environment." This is an overgeneralization. In fact, the physical and chemical properties of PFCs vary widely. The toxicity profile is different for each PFC, and their movement through the environment also differs among the different members of the class of PFCs. We suggest that MDH refrain from generalizations and avoid any suggestion that PFCs can simply be grouped together in terms of their characteristics, properties, etc.

On page 7, the discussion of tumors in animals exposed to high levels of PFOA should address the fact that the tumors observed in the PFOA studies were benign. There is no evidence of an increase in malignant tumors in animals. This discussion should also note that none of the tumor types seen in the animals have been observed in the human studies, including those with high exposure under occupational settings.

Also at page 7, regarding the discussion of developmental effects observed in the offspring of pregnant rats and mice, additional human data have now been reported in the literature, and the available data are summarized in a review paper, Olsen et al., *Perfluoroalkyl chemicals and human fetal development: An epidemiologic review with clinical and toxicological perspectives*, Reproductive Toxicology 27:212-230 (2009). The table presented there shows that the reported associations in human studies are inconsistent. The draft document should direct the reader to the published review paper, as it is much more comprehensive than what is presented within this draft document.

## Comments on the "Background" Section

### "3M-Woodbury Disposal Site Description and History"

The description states on pages 7-8 that a groundwater extraction system was completed at the Woodbury site by 1973 and has operated continuously since then. Indeed, well construction began in 1967. This discussion should also state that 3M took responsibility for constructing and operating the groundwater extraction system, as the draft assessment does not address that issue.

At the top of page 8, the reference to 3M's discharge of cooling or process water to the Mississippi River should note those discharges were (and are) authorized by an NPDES permit issued by MPCA under the Clean Water Act.

On page 8, the draft assessment correctly notes 3M performed additional investigation and response action when it entered the Woodbury site into the MPCA's Voluntary Investigation and Cleanup (VIC) Program in 1992. This discussion should also note that 3M submitted this testing data to MPCA. As set forth above, 3M provided MPCA with an Investigation Report for the Woodbury site in February 1994. The report notified MPCA that fluorochemicals, including but not limited to PFOS, PFHS, and PFOA, were present in soil and soil gases at the Woodbury site, but the analytical technology at the time was not able to detect PFCs in groundwater.

The third paragraph on page 9 addresses a groundwater divide near the Woodbury Site. All historical groundwater elevation data collected at the Woodbury Site indicates that the groundwater divide described there is located east of the 3M-Woodbury Site, and there is no data to support the statement that contaminants beneath eastern portions of the site could move east-southeast. Groundwater elevation data collected during brief periods that the barrier well network has not been pumping indicates groundwater flows to the south or southwest.

On page 9, the last sentence in the third full paragraph states groundwater from the Woodbury site "could potentially affect a larger area than is typically seen at most sites." 3M believes there are two problems with this sentence. First, it is speculation. Second, groundwater hydraulic controls have been in place for more than 40 years, and 3M is continuing remediation work today; accordingly, there is no basis to suggest that larger areas could potentially be affected in the future.

3M agrees with the assertion in the second full paragraph on page 10 that pumping in the Jordan Sandstone could cause groundwater to move downward from the Prairie du Chien into the Jordan. However, hydraulic data collected at the Site indicate that the pumping of the barrier well network at the Woodbury Site causes groundwater to flow upward from the Jordan Sandstone to the "high transmissivity zone" within the Prairie du Chien. The flow of groundwater upward from the Jordan to the Prairie du Chien has also been observed in monitoring wells that are open to both the "high transmissivity zone" of the Prairie du Chien and the Jordan. This is well documented in *Evidence for hydraulic heterogeneity and anisotropy in the mostly carbonate Prairie du Chien Group, southeastern Minnesota, USA (Tipping et al., 2006).* This paper also documents the fact that the lower Oneota formation of the Prairie du Chien Group appears to act as a confining unit. This also restricts the potential migration of shallow groundwater in the Prairie du Chien Group to the underlying Jordan Sandstone.

**"PFC Analysis"**

On page 11, the draft assessment states that MDH "developed a method" to analyze water samples for PFOS and PFOA in 2003. The analytical method was not original to MDH. It may be more accurate to say that MDH developed the capability of analyzing for PFCs. 3M contributed significantly to MDH's efforts, as well as working on development of ever-improving analytical methods for decades.

**"Evaluation of PFCs in Drinking Water"**

Page 11 discusses HRLs and HBVs. As MDH notes, an HBV is a criterion that, unlike a HRL, has not been promulgated through rulemaking. Although not raised in the draft assessment, 3M does want to address a statement MDH recently made about the relationship between a HBV and a HRL. At a May 19, 2010 public meeting presentation discussing draft amendments to the HRLs, MDH stated: "A newer HBV or RAA [Risk Assessment Advice] takes precedence over an older HRL, even though the HRL is promulgated and the HBV or RAA is not. . . . *It is always the more recent value that takes precedence***."** (Id., available at http://www.health.state.mn.us/divs/eh/risk/rules/water/mtngpresentation.pdf, at Slide 68, emphasis in original.) We find this assertion surprising, and legally unsupportable.

Minnesota Statutes Section 103H.201, subd.(2)(a) provides that "Health risk limits shall be adopted by rule." The Minnesota Administrative Procedures Act ("MAPA") defines a "Rule" as "every agency statement of general applicability and future effect, including amendments, suspensions, and repeals of rules, adopted to implement or make specific the law enforced or administered by that agency or to govern its organization or procedure." Minn. Stat. § 14.02 Subd. 4 (2009). The Minnesota Supreme Court has made clear that all types of rules are subject to the rulemaking requirements of MAPA. *Cable Communications Board v. Nor-West Cable Communications Partnership*, 356 N.W.2d 658, 667 (Minn. 1984). We do not understand how MDH can supersede or amend a validly promulgated binding regulation by a subsequently-issued agency statement that has not been adopted as a regulation. As the Minnesota Supreme Court has made clear in *Cable Communications Board, supra,* if an agency's new policy is not consistent with its current rules, the court will invalidate the agency action unless MAPA procedures for rulemaking were followed. *Id*. at 667-668. Thus, MDH is required under MAPA to amend its HRLs by rulemaking, not by announcing a new policy setting a different standard.

On page 12, in the third paragraph, MDH describes the use of its Hazard Index calculation if multiple substances are present in drinking water. MDH should clarify that the Hazard Index applies *if and only if* the substances in question have the same endpoints identified in MDH's regulations. It is not toxicologically valid to use a Hazard Index approach unless the endpoints at issue are the same. Nor do the MDH regulations allow the use of the Hazard Index approach unless MDH has specified the same endpoints for the substances. *See* Minn. Administrative Rules Section 4717.7880.

**"Community Well Monitoring"**

The discussion of community well monitoring beginning on page 12 states that MDH activity at the Cottage Grove site did not begin until "late 2004, after releases of PFCs were documented at the 3M-Cottage Grove facility." While it is correct that in 2004 3M voluntarily undertook an investigation of its plant site for the presence of PFCs, it is not correct to imply that 2004 saw the first activity related to investigation or remediation of PFCs. As detailed above, in the early 1980s, 3M and MPCA investigated disposal activities at the Cottage Grove site. A 1983 investigation report to MPCA specifically reported the presence of PFOA in a sludge disposal area at the plant site. Those investigations resulted in the entry of a consent order between 3M and MPCA in 1985 and various remediation activities at the time. 3M then undertook its own investigation in 2004, and worked with MDH to sample nearby water supplies.

In the first paragraph on page 13, the assessment refers to "3M waste disposal sites in Oakdale and Lake Elmo." 3M does not operate disposal sites. The reference to Lake Elmo presumably is to the Washington County Landfill. MPCA has responsibility for the Washington County Landfill under the MPCA Closed Landfill Program. The sentence should refer to disposal sites that had been used by 3M. References throughout the assessment to Lake Elmo are unclear and should refer to the Washington County landfill.

A table on page 13 presents the PFBA analytical results for various community wells. The table provides a "Range," but the range given is only for the detections. A number of wells did not have detectable or quantifiable PFCs. Thus, the range should be from "Not detected" to the highest value. The table also presents the average of "all Detects." This skews the data by not including the Non-Detects and J-flag (not quantifiable) values. We suggest using median values in the table. Alternatively, if MDH is to provide mean values, then the non-detects and J-qualified values need to be included in the calculation (perhaps using a standard convention of half the detection limit for the non-detect values), in order to more accurately depict the data set.

On page 14, MDH directs readers to Figure 6, a graph illustrating the concentration of PFBA in Cottage Grove community wells sampled from January 2007 to September 2009. As currently graphed, the y-axis on Figure 6 ranges from 0 to 2 ppb, and the highest concentration detected in any Cottage Grove well is 1.8 ppb. The y-axis should be revised to range from 0 to more than 7 ppb, in order to show the HBV for PFBA. We suggest a horizontal line be drawn across the graph at 7 ppb to illustrate that all detections of PFBA in Cottage Grove wells have been below the HBV. This would provide important context for the values shown on the graph.

**"Private Well Sampling"**

The discussion of private well sampling on page 14 states that 3M's consultant, Weston Solutions, Inc., conducted testing at the Woodbury disposal site in 2005 "as a result of being informed by 3M that PFC containing wastes may have been disposed of at the 3M-Woodbury site." It is misleading to suggest 3M first did testing at the site in 2005. To do so fails to take into account 3M's 1994 testing at the Woodbury site specifically for PFCs and submission of those results to MPCA. We suggest MDH simply state that 3M, which had previously investigated and remediated the site, again sampled for PFCs at the site in 2005 using modern analytical technology.

On page 16, the fourth paragraph cites a 2008 MDH site assessment document on Oakdale and Lake Elmo for the proposition that PFCs migrate from groundwater to surface water. Pages 31-33 of the 2008 assessment describe the migration of contaminants from Raleigh Creek to groundwater. That document does not identify migration *from* groundwater *to* surface water. In addition, the current draft document sets out PFC concentrations in samples taken from surface water in Gables Lake near the main disposal area at the Woodbury site, and the levels there are quite low. Gables Lake is dry during periods of low precipitation and any water present in the Lake is not a result of groundwater discharge. The suggestion that groundwater and surface water are connected in this area of the Site property is incorrect.

On page 17, the last sentence in each of the first two paragraphs suggests there may be a gap in the current monitoring network at the Woodbury site. The hydraulic evaluations that have been performed at the Site indicate an inward hydraulic gradient toward the barrier well network in all water-bearing units beneath the Site. As discussed above, this is especially true in the "high transmissivity zone" where the greatest response to pumping was observed during the operation of the barrier well network. There are no critical gaps in the monitoring network.

Also on page 17, the assessment correctly notes a groundwater remediation system is in operation at the Woodbury site. It would be useful to note that the remediation system has been in place for more than 40 years.

**"MPCA - 3M Consent Order for PFC Disposal Sites"**

On page 17, the draft assessment states a Citizens' Board meeting was held in April 2007 "to compel 3M to respond to PFC contamination from three known PFC disposal sites: the 3M-Cottage Grove facility, the 3M-Woodbury Disposal Site, and the 3M-Oakdale Disposal site." The use of the phrase "compel 3M to respond" mischaracterizes both 3M's historical efforts at those sites and the purpose of the meeting.

As discussed above, 3M had already performed significant remediation activities at each of the sites long before the April 2007 meeting. 3M had also built a GAC treatment system for two of the City of Oakdale's municipal wells (which became operational in October 2006), and provided Lake Elmo a grant in the amount of $3.3 million to connect over 200 homes to municipal water. Also prior to the April 2007 Board meeting, 3M had provided MPCA with a written offer to enter into a binding agreement for further activities at each of the disposal sites, and in fact was already proceeding with work under MPCA's supervision. The April 2007 meeting was not held because MPCA needed to issue an order in order to compel 3M to take action. The meeting addressed whether to formalize the enforceability of 3M's ongoing voluntary activities by issuing Requests for Response Action under the MERLA statute. 3M disputes that the MERLA statute applies. The Board decided not to issue the requests under MERLA, but rather directed MPCA to enter into a Settlement Agreement and Consent Order (Consent Order) with 3M. The draft document should be revised to accurately reflect these facts.

Also in the discussion of the Consent Order, the draft document states on page 18 that 3M agreed to contribute up to $8 million to remediate the Washington County Landfill. 3M has made that contribution. The Washington County landfill was by law the responsibility of MPCA, and MPCA has characterized 3M's contribution as a "gift" to assist the State in addressing this site.

In the middle of page 18, the draft assessment states that natural resource damage claims related to releases of PFCs are "allowed under state and federal law." This statement is a legal conclusion, and an incorrect one with respect to PFCs. A public health assessment should refrain from including legal conclusions, and this statement should be removed from the final assessment document.

**"Remedial Options for the 3M-Woodbury Disposal Site"**

On page 19, the discussion of remedial options states that "[a]fter a cost-benefit analysis, it was determined that by slightly reducing the mass of PFCs removed from the site, the cleanup could be completed much sooner and at less cost to the environment in terms of fuel use, truck mileage, and landfill space." This sentence should be revised by replacing "it was determined" with "MPCA determined" to clarify that MPCA made the determination reflected in the MPCA Decision Document discussed there.

In the following paragraph, MDH states that "3M began cleanup work at the site in the summer of 2009." As discussed above, 3M performed significant work at the Woodbury site long before 2009. To accurately reflect 3M's prior work at the site, this sentence should be revised to state that "3M began *this phase of* clean up work at the site in the summer of 2009."

**"General Regional Issues"**

On page 21, in discussing the expansion of water supply systems to accommodate population growth, 3M questions why MDH states that "The widespread PFC contamination in the aquifers typically used for municipal water supplies has complicated this process." 3M disagrees. The water is safe, and treatment has been provided in the few localized areas where exceedances of standards have been detected. 3M is not aware of any problems in locating new supply wells.

**"Community Concerns"**

The discussion of community concerns on page 21 says residents have expressed concern that cancer rates in the area seem higher than normal. As MDH notes on page 35, the agency's own study issued in 2007 found that overall cancer rates in Washington and Dakota counties are very similar to or slightly lower than in the rest of the state. The discussion of cancer rates as a community concern should be revised to incorporate either a summary of these findings or, at a minimum, a cross-reference to the discussion of the study's findings that appears later in the document.

**Comments on "Evaluation of Environmental Fate and Exposure Pathways" Section**

**"Introduction"**

On pages 21-22, the discussion of 3M's historical and current PFC manufacturing should be revised. As written, the assessment incorrectly states 3M ceased production of all PFCs in 2002. As set forth above, 3M continues, with EPA's consent, to manufacture PFBS-related and other short-chain materials today.

The first full paragraph on page 22 does not take into account that while the electrochemical fluorination (ECF) process was unique to 3M in the United States, it has been used by a number of other manufacturers around the world. EPA still allows imports of PFOS-related products for certain critical uses in the United States.

The draft document suggests that PFCs in the environment from historical waste disposal necessarily came from the ECF process. However, telomer-based processes have been used to produce straight-chain molecules historically, not just following 3M's phaseout. Thus, apart from any 3M manufacturing waste, PFCs in landfills could also be telomer-derived products from consumer or industrial use unrelated to 3M.

3M does agree that the distinction between branched and linear PFCs may provide some useful information. This distinction between materials from different processes affects the chemical nature of the material produced and appears to have a significant effect on toxicity (i.e, with materials from the telomer process which has never been used by 3M appearing to have greater toxicity).[1]

On page 22, the final paragraph of the Introduction section discusses PFOS, PFOA, PFBS and PFBA. For clarity, these concepts should be separated into separate paragraphs. 3M suggests including the following clarifications:

PFOS Phase-Out and Issuance of Significant New Use Rules. In 2000, 3M announced it was voluntarily phasing out production of all of its eight-carbon PFCs, including PFOS and products that could degrade or metabolize to PFOS. 3M ceased manufacturing both PFOS and precursor materials by the end of 2002. After 3M ceased the manufacture of PFOS, U.S. EPA promulgated federal regulations that prevent other manufacturers (as well as 3M) from manufacturing or importing PFOS or PFOS precursors without EPA permission, subject to certain critical use exceptions with limited exposure potential approved by EPA. *See* 40 Code of Federal Regulations § 721.9582, listing several hundred PFOS precursors that cannot be manufactured or imported without EPA permission, and the permissible uses approved by EPA. *See also* http://www.epa.gov/oppt/pfoa/pubs/pfas.html (EPA's rules "allowed the continuation of a few specifically limited, highly technical uses of these chemicals for which no alternatives were available, and which were characterized by very low volume, low exposure, and low releases. Any other uses of these chemicals would require prior notice to and review by the Agency.")

PFBS Substitute Products. The draft assessment correctly indicates that 3M's current stain repellant and other products are based on the four-carbon PFBS chemistry rather than on PFOS-related chemistry.

EPA's PFOA Stewardship Program. As written, the draft assessment incorrectly indicates 3M "remained in the PFOA industry" as of January 2006. As set forth above, 3M announced its decision to phase out production of all eight-carbon PFCs, including PFOA, in 2000, and ceased its manufacture of PFOA by the end of 2002. 3M participated in EPA's product stewardship program because its subsidiary Dyneon still ***used*** a small amount of PFOA at the time that EPA program commenced to make specialty fluoroelastomers at 3M's Decatur, Alabama facility and in Germany. (PFOA was an emulsifier in the process, not an ingredient in the final products.) Dyneon's use of PFOA in the Decatur facility ceased at the end of 2004. As the draft assessment notes, 3M/Dyneon have already met EPA's 2010 and 2015 goals.

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[1] See, e.g., Martin, JW et al., "Metabolic products and pathways of fluorotelomer alcohols in isolated rat hepatocytes," Chem.-Biol. Interact. 155:165-180 (2005) (transient metabolites in the conversion of fluorotelomers to perfluorinated carboxylic acids include hydrofluoric acid as well as fluorinated aldehydes and unsaturated fluorinated aldehydes); Phillips, MM et al., "Fluorotelomer Acids are More Toxic than Perfluorinated Acids," Environ. Sci. Technol. 41:7159-63 (2007) (telomers' toxicity thresholds in *Daphnia magna*, *Chironomous tentans*, and *Lemna gibba* are "up to 10,000 times smaller" than their perfluorocarboxylic acid degradation products); Dr. Scott A. Mabury's presentation entitled "Origin and Environmental Fate of Polyfluorinated Materials," Joint Midwest SETAC and Northland SOT Meeting, Duluth, MN, March 31 – April 2, 2008 (Dr. Mabury has presented an overview of these data to MPCA)**;** Loveless, et al., "Comparative responses of rats and mice exposed to linear/branched, linear, or branched ammonium perfluorooctanoate," Toxicology 220: 203–217 (2006).

Other Sources of PFBA. 3M ceased its manufacture of PFBA in 1998 for business reasons; 3M sold PFBA to a customer who used it for photographic film. MDH states that to its knowledge, there is no current commercial production of PFBA in the United States. However, there remain some important commercial applications for PFBA. Eastman Kodak imports hundreds of pounds of PFBA per year from a Japanese company, F-Tron (according to U.S. Customs databases), presumably for use in photographic film. U.S. Customs databases also indicate that PFBA is listed as a constituent of computer parts that are imported by Maersk from Japan. These databases also list one large import of PFBA by Iljin Industries, which supplies automotive parts.

In addition to applications in photographic film, computer parts, and automotive parts, PFBA is a valuable chemical for analytical laboratories. Suppliers such as Sigma-Aldrich, Alfa-Aesar, and PIERCE supply PFBA (often denoted as "heptafluorobutyric acid" or "HFBA"). Analytical laboratories often use PFBA as an ion-pair reagent for HPLC, in protein sequencing, as a protein or peptide solubilizing agent, in amino acid analysis, and in the detection and quantification of environmental or occupational chemicals. *See, e.g.,* http://www.piercenet.com/Products/Browse.cfm?fldID=02040610 (identifying HFBA as an **"***ion pair reagent for the reverse-phase HPLC separation of proteins and peptides").*

Analytical laboratories also use other four-carbon compounds that break down to PFBA in the environment, such as heptafluorobutyryl imidazole ("HFBI") and heptafluorobutyric acid anhydride ("HFAA"). These chemicals are often used to detect or quantify chemicals via gas chromatography that would otherwise be difficult to detect. For example, HFBI and HFAA are used to detect drugs of abuse such as cocaine or methamphetamines in blood, urine, or hair samples. These chemicals are indispensable for government agencies such as the Drug Enforcement Agency.

ATSDR's own analytical methods require PFBA or HFBI to detect the following compounds: sulfur mustard, benzidine, methylenedianiline, nitro- and dinitrophenols, MBOCA, and ethylene oxide. MDH and ATSDR may wish to check whether local law enforcement or private laboratories use PFBA-related materials.

Besides PFBA, HFBI, and HFAA, there are other products that may break down to PFBA in the environment. For example, EPA has approved the use of certain fluorochemicals as pesticide inerts. One compound in particular, "mono- and bis-(1-H, 1-H, 2-H, 2-H, perfluoroalkyl) phosphates in the C6-C12 range," could degrade to PFBA. This chemical was approved by EPA for a food tolerance exemption on October 24, 1984 (see 49 Fed. Reg. 42758). EPA revoked the tolerance on August 9, 2006, noting that two companies, Bayer Crop Sciences and Mason Chemical Company, protested the revocation (see 71 Fed. Reg. 45408). It is unclear whether these companies continue to manufacture or use PFCs in their pesticidal formulations; nevertheless, the companies conceded that PFCs (including PFBA-precursors) were an important constituent of pesticides before August 2006.

The draft assessment should also note that researchers are continuing to identify potential additional sources of PFCs. In a study published in 2007, researchers observed biotransformation of polyfluoroalkyl phosphates (PAPs) - substances used in pesticides and food packaging - into perfluorinated carboxylic acids in a rat model. (D'eon and Mabury 2007.) The researchers found that cleavage of the phosphate ester linkage of the PAPs studied resulted in the release of a fluorotelomer alcohol (FTOH) that subsequently degraded to PFOA. This year, researchers published a study demonstrating microbial biodegradation of PAPs into carboxylate PFCs ("PFCAs"), again via release of an intermediate fluorotelomer alcohol. (Lee et al. 2010.) The researchers suggest microbial biodegradation of PAPs at wastewater treatment plants may be a source of PFCs to the environment, including PFBA. The researchers concluded that depending on the amount of sludge treated at a wastewater treatment plant, microbial degradation of PAPs could account for a significant portion of PFCA production.

**"Evaluation of Impacts on Groundwater"**

In the last paragraph at the bottom of page 23, the draft assessment states that the highest concentrations of PFBA appear to follow the buried bedrock valley that underlies the western edge of the site and trends south. The hydraulic evaluations performed at the Woodbury Site in May 2007 and 2008 clearly demonstrate that the barrier well network is in hydraulic communication with monitoring wells completed within the sediments in the buried bedrock valley. The direction of groundwater flow is from the buried bedrock valley to the west toward the depression in the groundwater surface induced by the barrier well network. The barrier well network is located between the former disposal areas on Site and the buried bedrock valley. The unconsolidated sediments within the buried bedrock valley are lower in permeability than the Prairie du Chien and Jordan aquifers beneath the Site. Therefore, the suggestion that the bedrock valley may be acting as a conduit for contaminants in groundwater in the Site vicinity is not valid.

On page 24, the third full paragraph offers speculation that PFBA in east Woodbury and west Afton migrated from the Washington County Landfill, and that PFBA in south Maplewood and northern Woodbury likely migrated from the Oakdale Landfill. This discussion, even qualified by "likely," is more definitive than existing knowledge permits. It is not possible to draw firm conclusions with respect to sources of PFBA in local groundwater.

At the bottom of page 24 and the top of page 25, 3M questions why the assessment speculates about a "finger" of PFBA in the Prairie du Chien aquifer. This discussion is conjecture which should not be included in the final assessment.

Also on page 25, the assessment states, "Some specialized types of fire-fighting foams contain PFCs." Virtually all aqueous film-forming foams (AFFF) contain fluorochemical surfactants. Although 3M has ceased manufacturing its Lightwater® brand AFFF, other products on the market contain or can degrade to PFCs.

Page 26 points to an absence of monitoring wells in the high transmissivity zone at the Woodbury disposal site as a possible important gap in the integrity of the monitoring network. As explained above with respect to MDH's draft recommendations, there is no such gap. There are multiple monitoring wells in the high transmissivity zone, and the data clearly demonstrate the effectiveness of the barrier well pumping system in controlling flow from the high transmissivity zone.

As discussed in detail in earlier sections of the MDH document, a bedrock valley exists to the west and south of the Site. Within this bedrock valley, the entire thickness of the Prairie du Chien Group has been eroded and the sub cropping bedrock within the center of the bedrock valley is the Jordan Sandstone. Since the "high transmissivity zone" is eroded in the center of the bedrock valley, groundwater within that zone and other water-bearing units that are intersected by the bedrock valley would theoretically flow into the unconsolidated sediments above the Jordan Sandstone. In the areas where the high transmissivity zone is eroded or absent, it is not possible to install wells within the "high transmissivity zone" in the Prairie du Chien Group. Monitoring wells were installed within, or adjacent to, the bedrock valley to monitor groundwater quality and hydraulic conditions.

As shown in Figure 1 in the draft assessment, monitoring well MW-H is installed within the bedrock valley in an area that is likely hydraulically down gradient of the Site under non-pumping conditions (southwest of the barrier well network). Also as depicted in Figure 1, there are additional site monitoring wells S-01JS and S-01PC on the opposite side of the bedrock valley as interpreted by the Minnesota Geologic Survey (MGS) map presented (Mossler, 2006). Finally, monitoring wells S-02DR, S-02PC, and S-02JS are located within the bedrock valley as depicted in the MGS map. These are additional down gradient (under non-pumping conditions) monitoring wells. These further points again lead us to disagree with MDH's assertion that there is an important gap in the integrity of the monitoring network.

**"Exposure Through Private Wells"**

On page 27, the draft assessment states that an environmental exposure pathway is complete if evidence shows the parts of a pathway have been *or will be* present and that "[m]ore simply stated an exposure pathway is considered complete when people *are likely* exposed to the chemical of concern." (emphasis added). 3M questions the logic of describing an exposure pathway as complete even if people have not in fact been exposed to the chemical of concern. In contrast, MDH on page 54 provides a standard definition of exposure pathway which states that "[w]hen all five parts *are present*, the exposure pathway is termed a completed exposure pathway." (emphasis added). The definition of an environmental exposure pathway on page 27 should be revised to be consistent with the definition on page 54.

Also on page 27, the discussion of PFBA findings notes that there have been no exceedances of the HBV south of I-94. The discussion should also note the levels of PFBA detected were *far* below the HBV, which itself incorporates a margin of safety based on extrapolation from laboratory rat studies.

The draft assessment, at the bottom of page 27, states that well advisories remain in effect for seven wells where PFCs are below current health advisory levels due to "potential changes in the PFC plumes." 3M questions why well advisories remain in effect for these specific wells when well advisories do not remain in effect for other wells where PFCs are below health advisory levels. "Potential changes" in the future should not be reason for action now. These wells have already been monitored on multiple occasions over a several-year period.

On page 28, the discussion of routine monitoring going forward "will ensure that if levels of PFCs rise, future exposure to levels about health-based exposure limits will be brief." It is highly unlikely that levels of PFCs will rise in the future. 3M believes this sentence can be omitted from the final assessment or revised to state that a rise of PFC levels is highly unlikely and not anticipated. As MDH acknowledges at the top of page 14 of the draft assessment, "Levels of PFBA appear to be stable or declining slightly in many of the community wells that have been regularly sampled by MDH since early 2007."

**"Exposure through Other Pathways"**

On page 29, the draft assessment incorrectly states that fluorotelomer alcohols can break down to PFOS. Although fluorotelomer alcohols can degrade to PFOA or higher or lower homolog carboxylates, they do not degrade to PFOS or any sulfonate compound. (There are fluorotelomer sulfonic acids, for example 8:2 fluorotelomer sulfonic acid, $C_8F_{17}CH_2CH_2SO_3H$, but these do not degrade to PFOS.)

Also on page 29, the discussion of exposure should note that the presence of perfluoroalkyls (PFOS and PFOA) has declined substantially since 2000. In comparing the geometric mean PFOS and PFOA concentrations in 600 individual American Red Cross adult blood donor samples obtained in 2006 each from six regional donation centers (100 samples per center) to the geometric means from the same approximate number of samples from the same regional centers obtained in 2000-2001, Olsen et al. found approximately 60% and 25% declines in the geometric means for PFOS and PFOA, respectively. *See* Olsen et al., *Decline in Perfluorooctanesulfonate and Other Perfluoroalkyl Chemicals in American Red Cross Blood Donors, 2000-2006*, Environ. Sci. Technol. 42:4989-95 (2008).

Data for PFOS-related compounds from these studies appear in Figures 1 and 2 below. Figure 1 demonstrates trends in the geometric mean for PFOS and precursors from 1999-2006. Figure 2 shows the decline in the 95$^{th}$ percentile levels. Collectively, these data show an unmistakable downward trend in serum/plasma PFOS concentrations in the United States general population since production ceased in the United States.

Concentrations of PFOS precursors have also decreased. As examples, we have included data in Figures 1 and 2 for two precursors, N-methyl perfluorooctane sulfonamidoacetate ("Me-PFOSA-AcOH") ($C_8F_{17}SO_2N(CH_3)CH_2CO^-$) and N-ethyl perfluorooctane sulfonamidoacetate ("Et-PFOSA-AcOH") ($C_8F_{17}SO_2N(CH_2CH_3)CH_2COO^-$).

**Figure 1: Trends in the Geometric Mean for PFOS and Precursors, 1999 - 2006**

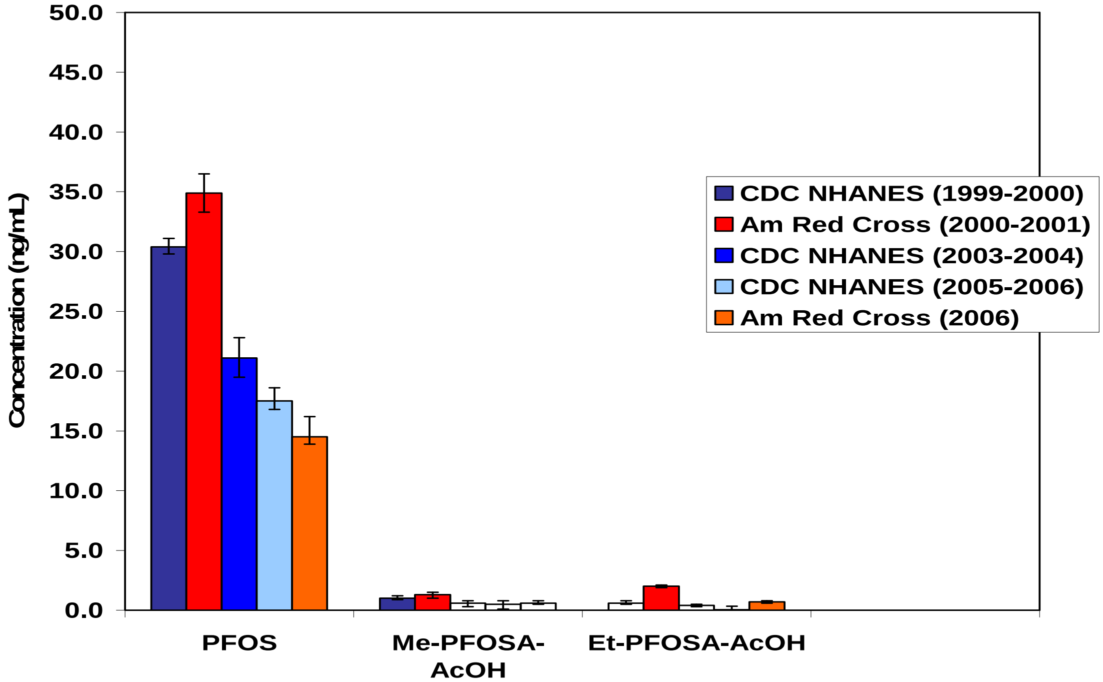
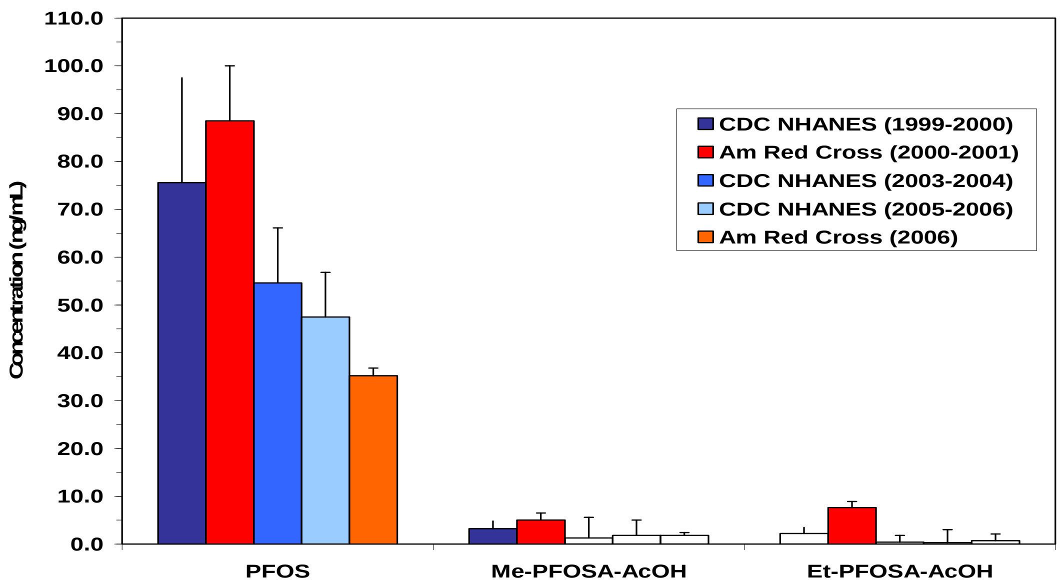



**Figure 2. Trends in the 95th Percentile of Distribution for PFOS and Precursors, 1999 - 2006.**




Serum levels of PFOA in the general population showed a decline following 3M's phaseout, but that decline has tapered off due to the continued production and use by others in the U.S. of PFOA or its precursors. According to CDC data, PFOA geometric mean concentrations declined between 1999-2000 (geometric mean = 5.2 ng/mL, 95% CI 4.7 – 5.7) and 2003-2004 (geometric mean = 3.9 ng/mL, 95% CI 3.7 – 4.3) (Calafat et al. 2007). However, they did not decline further when measured in 2005-2006 (geometric mean = 3.9 ng/mL, 95% CI 3.5 – 4.4). (See latest CDC data at http://www.cdc.gov/exposurereport/data_tables/LBXPFOA_DataTables.html). The lack of continued declining trend for PFOA suggests ongoing sources of biotransformation of fluorotoelomer alcohols and polyfluoroalkyl phosphate surfactants (PAPS) (D'Eon and Mabury 2007).

## Comments on "Public Health Implications of PFC Exposure" Section

This part of the document states, "This section will briefly summarize current information on the toxicity of PFCs to animals and humans . . . ." However, the few epidemiologic articles cited represent less than 5 percent of the published epidemiologic literature pertaining to PFOS and PFOA. These citations therefore do not represent the current literature. 3M will summarize below some of the current literature, which supports MDH's conclusion with respect to the lack of health effects from PFC exposures in Minnesota.

### "Summary of Toxicological Information" Section

The discussion of toxicological information as a whole would benefit by comparing the levels seen in animal studies to the levels observed in humans. As set forth above, the HBV for PFBA includes an uncertainty factor of 100 and dose metric of 8; as a result, the HBV is set at 800 times below the level of possible concern based on animal studies. MDH's values for PFOA and PFOS are also conservative.

In the second paragraph on page 30, the statement is made that "Animal studies have shown that PFOA and APFO (its ammonium salt) are easily absorbed through … dermal contact…" This statement does not consider a published paper by Fasano et al. (2005) which reports dermal permeability coefficients of $3.25 \pm 1.51 \times 10^{-5}$ cm/h for rats versus $9.49 \pm 2.86 \times 10^{-7}$ cm/h for humans. In a 48-h period, only $0.048 \pm 0.01\%$ of applied APFO had penetrated the human skin, and rat skin was over 30-fold more permeable than human skin. Therefore, the statement that APFO is easily absorbed via skin is not supportable in the case of human skin. The paragraph should be modified accordingly.

In the third paragraph on page 30, there appears to be overreliance on older secondary sources in discussing modes of action. In the case of PFOA, it has been clearly demonstrated that liver responses in rodent species are the result of activation of the nuclear receptor peroxisome proliferator activated receptor α (PPARα), with a lesser role for activation of the nuclear receptors, constitutive androstane receptor (CAR) and pregnane X receptor (PXR). (*See e.g.,* Elcombe et al. 2010; Klaunig et al. 2003; Rosen et al. 2009.)

For some time, it has been known that human liver is refractory or less responsive to many of the downstream events of PPARα activation. (*See* Klaunig et al. 2003; USEPA 2003). It has also been demonstrated recently that activation of the human form of PPARα does not support the hepatocellular proliferative response observed with the rodent forms of the receptor. (*See* Gonzalez and Shah 2008.) This latter observations was also verified with APFO

(Nakamura et al. 2009). Liver hypertrophy in primates exposed to PFOA may be the result, at least in part, of increased mitochondrial mass (*See* Walters et al. 2009; Butenhoff et al. 2002.)

The paragraph beginning at the bottom of page 30 suffers from overreliance on the 2002 OECD "Hazard Assessment of Perfluorooctane Sulfonate (PFOS) and its Salts." MDH's document appears to rely primarily on older citations derived from this review, despite the enormous amount of literature developed since 2002. For example, it is curious that the paragraph focuses on a 1978 rat 90-day study at obviously lethal doses of PFOS, yet gives scant attention to a modern six-month oral toxicity study with potassium PFOS in cynomolgus monkeys (Seacat et al. 2002), as well as Health Canada's published 28-day rat study (Curran et al. 2008) and 3M's published 90-day dietary study in rats (Seacat et al. 2003).

It is similarly curious that the discussion of immunotoxicity is limited to one recent study in mice (Peden-Adams et al. 2008), but does not consider multiple other available studies. Citing only the 2008 immunotoxicity study by Peden-Adams et al. does not provide a balanced view of the published immunotoxicology literature for PFOS, which includes one study in rats and eight studies in mice (see the table below). Considering the weight of evidence with regard to immunotoxicology, the Peden-Adams study becomes an outlier. This is particularly notable considering the thorough dietary study by Qazi et al. (2010a) in the same strain of mice used by Peden-Adams et al., though at much higher doses. In Qazi et al. 2010a, there was no effect on splenic and thymic weights, *in vivo* SRBC-specific IgM and IgG, total plasma cells in spleen and thymus, total circulating plasma leukocytes, PFC assay, and hemoagglutination assay, at a serum concentration of 11 ug/mL [ppm] in mice.

The immunotoxicological discussion should be revised. Briefly, the papers on PFOS immune effects in laboratory rodents have shown suppression of adaptive immunity in mice and enhancement of innate immunity in mice. Both of these outcomes were attenuated by knocking out PPARα, and the studies showed potential species, strain, and route differences. The effects noted may be secondary to other changes, for example, PPARα-mediated liver effects. The following chart describes the eight studies of potential PFOS immunotoxicity and underscores the differences noted above.

# Immune Effect Studies on PFOS

| Study | Species strain | Sex | Duration | Admin. | Dose µg/g diet | Dose mg/kg body weight | Serum PFOS µg/mL (ppm) | Outcomes | NOAEL mg/kg | NOAEL µg/mL |
|---|---|---|---|---|---|---|---|---|---|---|
| Lefebvre (2008) | Rat/SD | M & F | 28 d | Diet | 2 - 100 | 0.14 - 7.58 | 0.95 - 43 | Changes in immune parameters did not manifest as functional alterations in response to immune challenge with Keyhole Limpet Hemocyanin and may be secondary to hepatic-mediated effects | <0.15 liver weight; | <1.5 liver weight; |
| Kiel (2008) | Mouse B6C3F1 | Pups | GD 1 - 17 | Gavage | | 0.1 - 5.0 | | Decreased NK-cell activity, IgM production, and lymphocyte subpopulations in offspring at 8 weeks postnatal. Liver weight increased in males at 4 weeks. | 0.1 | |
| Peden-Adams (2008) | Mouse B6C3F1 | M & F | 28 d | Gavage | | 0.000166 - 0.166 | 0.018 - 0.666 | Increased NK-cell activity and plasma lysozyme activity; changes in thymic and/or splenic lymphocyte subpopulations; decreased SRBC-specific IgM at lowest dose. | 0.000166 | 0.018 |
| Qazi (2009a) | Mouse C57BL/6 | M | 10 d | Diet | 10 & 200 | 1.6 - 32 | | Lymphopenia; decreased macrophages in bone marrow but not spleen and peritoneum; increased release of TNFα and IL-6 from bone marrow and peritoneal macrophages (not splenic macrophages); enhanced innate immunity. | 1.6 | |
| Qazi (2009b) | Mouse C57BL/6; Sv/129 | M | 10d | Diet | 10 - 200 | 1.6 - 32 | 51 - 340 | Liver hypertrophy as most sensitive endpoint. Decreased cellularity of thymus and spleen. Histological alterations in thymus. PPARα plays a role, although extent of this remains to be determined. | <1.6 liver; 8 immune | <51 liver; 97 immune |
| Zheng (2009) | Mouse C57BL/6 | M | 7 d | Gavage | | 5 - 40 | 110 - 338 | Stress (increased corticosterone) at ≥20. Decreased body weight, spleen weight, thymus weight, splenic and thymic cellularity, B-cell proliferation, and NK activity at ≥20. Increased liver weight at ≥5. Decreased SRBC-specific IgM and proliferation of T cells in spleen at ≥5. | <5 | <110 |
| Dong (2009) | Mouse C57BL/6 | M | 60 d | Gavage | | 0.0083 - 2.08 | 0.674 - 121 | Decreased Body weight, thymus weight, spleen weight, splenic and thymic cellularity at ≥0.42. Decreased kidney weight, increased corticosterone (stress), NK-cell activity, and decreased T-cell proliferation in spleen at ≥0.83. Increased liver weight and decreased SRBC-specific IgM at ≥0.083 (7.1 ppm in serum) | 0.0083 | 0.674 |
| Qazi (2010a) | Mouse B6C3F1 | M | 28 d | Diet | 1.56 | 0.25 | 11 | Increased liver weight and decreased body weight at 0.25 (11 ppm in serum). No effect on splenic and thymic weights, in vivo SRBC-specific IgM and IgG, total plasma cells in spleen and thymus, total circulating plasma leukocytes, PFC assay, and hemoagglutination assay. | <0.25 LW & BW; 0.25 immune | <11 LW & BW; 11 immune |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Qazi (2010b) | Mouse C57BL/6 | M | 10 d | Diet | 50 | 8 | | Reduced in serum cholesterol and triglycerides, moderately increased serum alkaline phosphatase and hepatomegaly, with no effect on other immune organs. Enhanced numbers of hepatic erythrocyte progenitor cells. Attenuated hepatic levels of tumor necrosis factor-alpha (TNF-α), interferon-gamma (IFN-γ) and interleukin-4 (IL-4). Increased hepatic erythropoietin. | <8 | |

The paragraph at the top of page 31 incorrectly cites OECD 2002 in referring to "long-term animals studies" with PFOA. OECD 2002 is a hazard assessment of PFOS and its salts. Biegel et al. (2001) is the study in which the incidence of benign tumors of the liver, acinar pancreas, and testicular Leydig cells was elevated by dietary exposure of male rats to ammonium PFOA, and should be cited. In addition, Kennedy et al. (2004) provide a detailed discussion of the two-year dietary study of ammonium PFOA in male and female rats conducted by Sibinski and colleagues at Riker Pharmaceuticals.

Further in the same paragraph, there is discussion on the mechanism of "potential carcinogenesis." The reference should be to "potential tumorigenesis" in rats. The mechanism of formation of the *benign* tumors observed in the two chronic dietary bioassays of ammonium PFOA in rats has been studied and discussed in terms of relevance to humans (Biegel et al. 2001; Klaunig et al. 2003; Nakamura et al. 2009; Elcombe et al. 2010; Rosen et al. 2009). These citations provide a firm basis for evaluating PFOA's mode of action and human relevance.

The second paragraph on page 31 begins: "Various reproductive studies of rats followed for two generations showed postnatal deaths and other developmental effects in offspring of female rats exposed to relatively low doses of PFOS and APFO. . . . " There is one two-generation study in rats for each of PFOS (Luebker et al. 2005a) and APFO (Butenhoff et al. 2004). The paragraph fails to mention additional one-generation reproduction studies for PFOS in rats (Luebker et al. 2005a,b) or the several developmental studies for PFOS and APFO. Again, the citations are out-of-date. More recent reviews are available (Lau et al. 2004; Lau et al. 2007; Andersen et al. 2008) as well as the primary publications. Rather than just referring to "relatively low doses" of PFOS and APFO, an explanation of how the doses employed relate to body burden as evidenced by serum concentration should be provided. The reference to low doses in laboratory animal studies has the potential to create significant confusion for the public, who might equate the reference to vastly lower environmental exposures.

In the fourth paragraph on page 31, the citation to Chang et al. (2007), which was an abstract to a poster presentation, should be replaced with Chang et al. (2008), the published manuscript.

The fifth paragraph on page 31 discussing toxicity studies using PFBA in rats should discuss the serum concentrations of PFBA and PFOA associated with the reported effects, as well as the doses employed. The paragraph also needs to address the 90-day study of PFBA, not just the 28-day study.

On page 32 at the top, the draft assessment indicates that developmental delays in offspring of pregnant female mice dosed with PFBA were considered co-critical effects along with liver, blood and thyroid effects. The specific developmental delays, liver, blood, and thyroid effects have not been detailed.

- Based on studies supported by 3M, we believe the hepatic effects observed on repeat oral dosing with PFBA represent normal, non-adverse adaptive and reversible responses (USEPA 2002) mediated by activation of the nuclear receptors PPARα and CAR (at higher doses). With regard to the human/rodent differences in response to PFBA-mediated PPARα activation, two publications support this point (Bjork and Wallace 2009; Foreman et al. 2009).

- The loss of thyroxine from serum of rats given PFBA has been studied (Chang et al. 2009) and shown to result from both displacement of loosely bound thyroxine by PFBA and induction of thyroid hormone metabolism. Quantitative histomorphological changes in the thyroid were not noted when the thyroids were examined using advance histomorphological techniques. Further evidence for lack of thyroid stress was obtained through measurement of the pituitary TSH in serum, which did not increase on dosing with PFBA. Moreover, differences between humans and rats in response to agents that activate PPARα as well as in the manner in which thyroid hormone is carried in serum would suggest that the effects observed with PFBA in rats would be much less likely to occur in humans (Curran 1991; Capen 1997).

- The hematological changes observed on repeat dosing of rats with PFBA were small in magnitude and not of clinical relevance.

The paragraph on page 32 beginning, "The 2008 HBV for PFBA…" indicates that the use of a "dose metric adjustment" of 8 for PFBA is "much smaller" (than the dose metric adjustments for PFOS and PFOA) due to PFBA's "much shorter mean half-life in humans (3 days) versus rats (9.22 hours)." While MDH's use of a dose metric adjustment in the cases of PFOA and PFOS is based on the relative differences of 70 and 20 times, respectively, between the human and monkey serum elimination half lives, the use of and need for such an adjustment factor for PFBA is less compelling considering that the relative difference between the human and rat elimination half lives is less than 10 and within the range of the standard compound default uncertainty factors used in risk assessment. Is it MDH's intention to apply such dose metric adjustments on a routine basis in developing HBVs and HRLs? We are not aware of any other instance in which MDH has applied the dose metric approach in deriving HBVs and HRLs. Given PFBA's relatively short elimination half-life, it seems inappropriate to treat PFBA differently from other compounds with a comparably short half-life.

We note that MDH has referred to an obsolete calculation when describing the Reference Dose for PFOA on page 32.

## "Summary of Human Exposure Information" Section

### Exposure Levels

The discussion of blood levels on pages 32-34 should incorporate the research by Olsen et al. (2008) discussed above that found, based on 2006 samples collected by the American Red Cross, approximately 60% and 25% declines in the geometric means for PFOS and PFOA, respectively, compared to 2000 data. Because those findings are based on samples collected in 2006, current serum levels are expected to be even lower for PFOS. Whether there is a continued decline in PFOA concentrations in the general population remains to be determined as multiple sources of direct and indirect exposure remain for PFOA.

The second paragraph on page 34 discusses the ongoing work of the C8 Science Panel in the Parkersburg, West Virginia area communities where PFOA was found in drinking water. MDH should note that exposure levels in Parkersburg were higher than in Minnesota. In Parkersburg, the mean PFOA serum concentration was 82 ng/mL (Steenland 2010), compared to the mean serum value of 15 ng/mL MDH's biomonitoring study in Minnesota. This discussion should also direct readers to the review article by Steenland et al. (2010), which provides a much more comprehensive discussion of the C8 Health Project than can be provided in the assessment.

### West Virginia Studies

With respect to results of the Science Panel work, the draft assessment document cites only a website videotape of a graduate student's presentation (Frisbee 2008) to suggest the existence of several associations based on preliminary results. It is inappropriate to cite preliminary, unadjusted data, as opposed to those published studies that are now available. *See, e.g.,* Frisbee, et al., *Perfluorooctanoic acid, perfluorooctanesulfonate, and serum lipids in children and adolescents: results from the C8 Health Project*, Arch. Pediatr. Adolesc. Med., 164(9):860-869 (2010). Moreover, the graduate student presentation that is referenced is misleading. Ms. Frisbee presented univariate (unadjusted, *i.e.*, "crude") analyses, and she does not indicate the normal reference ranges on her graphs, which would reveal that each and every mean value for each decile and their 95% confidence limits were within <u>normal</u> ranges. Because of the confusion generated by Ms. Frisbee's presentation, the C8 Science Panel issued a press release clarifying that these preliminary data "do not represent a thorough data analysis and therefore, we do not believe they provide valid information regarding the presence or absence of association between C8 exposure and health outcomes." May 16, 2008 C8 Science Panel Press Release, *available at* http://www.c8sciencepanel.org/index.html. MDH should therefore delete the discussion of Frisbee's preliminary report.

The third paragraph on page 34 cites an unpublished report (Colsher et al. 2005) regarding prostate cancer incidence in the Parkersburg, West Virginia area communities. If the assessment is to cite the unpublished West Virginia report, the same paragraph should acknowledge MDH's own review of cancer rate date for the relevant communities in Minnesota. As reported in the draft assessment (page 35), prostate cancer incidence was not found to be above the expected rates in either Washington or Dakota counties, or by selected zip codes within each county.

**Reproductive and Developmental Epidemiology**

The bottom of page 32 cites the research by Apelberg et al. (2007), who reported a small (subclinical) negative association between both maternal serum PFOS and PFOS cord serum concentration and birth weight. The draft also mentions an inverse correlation between maternal serum PFOA levels and birth weight reported by Fei et al. (2007). However, the draft assessment does not acknowledge that Fei et al. did not find a statistical association between decreased birth weight and maternal serum PFOS levels, in contrast to Apelberg et al. If the assessment is going to mention these studies, it is important to point out that they do not report consistent findings.

Moreover, the study by Apelberg et al. (2007; see also Apelberg 2006) on birth weight and PFOA and PFOS umbilical cord concentrations prompted a series of research papers regarding human developmental outcomes and, subsequently, reproductive parameters. As is often a trend in the epidemiology literature, the initial published papers of a topic are suggestive of associations. However, it is only through a series of research studies that an understanding of the weight of the evidence emerges. In this regard, 20 papers have been published pertaining to human reproductive and developmental outcomes related to perfluorochemical exposures.

We briefly note the reproductive and developmental studies for MDH's benefit here. This expanded review of the current epidemiologic literature continues to support the conclusion offered by in the draft assessment that drinking water from public or private wells that contained PFCs (at the levels reported in Washington and Dakota counties) is not expected to harm people's health.

Table 1 summarizes statistically significant associations reported in the developmental epidemiological studies. A much more detailed tabular review is offered in the review by Olsen et al., *Perfluoroalkyl chemicals and human fetal development: An epidemiologic review with clinical and toxicological perspectives*, Reproductive Toxicology 27:212-230 (2009).

## Table 1.
## Summary of Statistically Significant Human Development Associations from Published Epidemiology Studies of PFOA and PFOS

| Endpoint | Olsen | Grice | Inoue | Apelberg | Fei | Monroy | Washino | Hamm | Nolan | Stein |
|---|---|---|---|---|---|---|---|---|---|---|
| Gestational Age | | | | | | | | | | |
| PFOA | | | | N.S. | N.S. | N.S. | | N.S. | N.S. | N.S. |
| PFOS | | N.S. | | N.S. | N.S. | N.S. | | N.S. | | N.S. |
| Birth Weight | | | | | | | | | | |
| PFOA | | | | N.S. | **S.S.** | N.S. | N.S. | N.S. | N.S. | N.S. |
| PFOS | | N.S. | N.S. | N.S. | N.S. | N.S. | **S.S.** $^{females}$ | N.S. | | **S.S.** |
| Birth Length | | | | | | | | | | |
| PFOA | | | | N.S. | **S.S.** | | N.S. | | | |
| PFOS | | | | N.S. | N.S. | | N.S. | | | |
| Head Circum | | | | | | | | | | |
| PFOA | | | | **S.S.** | N.S. | | N.S. | | | |
| PFOS | | | | **S.S.** | N.S. | | N.S. | | | |
| Abdominal/chest | | | | | | | | | | |
| PFOA | | | | | **S.S.** | | N.S. | | | |
| PFOS | | | | | N.S. | | N.S. | | | |
| Apgar Score | | | | | | | | | | |
| PFOA | | | | | N.S. | | | | | |
| PFOS | | | | | N.S. | | | | | |
| Ponderal Index | | | | | | | | | | |
| PFOA | | | | **S.S.** | N.S. | | | | | |
| PFOS | | | | **S.S.** | N.S. | | | | | |
| Placental Weight | | | | | | | | | | |
| PFOA | | | | | N.S. | | | | | |
| PFOS | | | | | N.S. | | | | | |
| Miscarrriage | | | | | | | | | | |
| PFOA | | | | | | | | | | N.S. |
| PFOS | N.S. | N.S. | | | | | | | | N.S. |
| Birth Defects | | | | | | | | | | |
| PFOA | | | | | | | | | N.S. | N.S. |
| PFOS | N.S. | | | | | | | | | N.S. |
| Preeclampsia | | | | | | | | | | |
| PFOA | | | | | | | | | | N.S. |
| PFOS | | | | | | | | | | **S.S.** |
| Developmental Milestones | | | | | | | | | | |
| PFOA | | | | | N.S. | | | | | |
| PFOS | | | | | N.S. | | | | | |

N.S. = not a statistically significant result ($p \geq 0.05$); **S.S**. = statistically significant result ($p < 0.05$)

The majority of developmental studies have focused on gestational age and birth weight related to maternal serum PFOA and PFOS concentrations. Several investigators have more than one publication from the same set of serum samples (e.g., Fei et al.).

- Most studies examined maternal 1st, 2nd, or 3rd trimester blood PFOA or PFOS concentrations originating from general populations.

- Two investigators (Nolan et al. 2009 from the University of Pennsylvania; 2010; Stein et al. 2009 for the Science Panel) examined Parkersburg area communities whose drinking water contained PFOA. Nolan et al. (2009) reported birth certificate data in relation to public drinking water sources. Stein et al. (2009) examined individual serum PFOA concentrations, obtained from a cross-sectional survey, in relation to self-reported pregnancy outcomes reported within the previous 5 years for continuous residents of the area. Therefore, neither Nolan et al. (2009; 2010) nor Stein et al. (2009) directly analyzed maternal serum concentrations during pregnancy, unlike others (Inoue et al. 2004; Apelberg et al. 2007; Fei et al. 2007; Monroy et al. 2008; Washino et al. 2009; Hamm et al. 2009). However, Nolan et al. and Stein et al. targeted communities that had considerably higher serum PFOA concentrations.

Of all outcomes studied, gestational age and birth weight predominate in the literature.

- Statistically significant associations have not been reported with gestational age and PFOA.

- Fei et al. (2007) reported birth weight was negatively associated with measured PFOA concentrations in both umbilical cord and maternal serum concentrations at general population levels of exposure. The Fei et al. results were statistically significant ($p < 0.05$). The birth weight data from Apelberg et al. approached statistical significance. Neither Apelberg et al. nor Fei et al. reported statistically significant findings with birth weight and PFOS, although the Apelberg et al. data suggested such an association. Neither study, however, showed statistically significant associations with *low* birth weight (based on a standard definition of < 2500 g).

- Subsequent investigations of maternal serum PFOA and PFOS concentrations have not supported statistically significant negative associations with birth weight (Monroy et al. 2008; Washino et al. 2009; Hamm et al. 2009), although Washino et al. observed a nonsignificant negative trend with PFOA.

- Despite their targeted community's much greater exposures to PFOA, neither Nolan et al. (2009, 2010) nor Stein et al. (2010) observed gestational age or birth weight to be significantly negatively associated with PFOA. Stein did report a statistically significant negative association with birth weight and PFOS. In a letter to the editor, Fei and J. Olsen (2009) questioned whether the reference group used by Stein et al was overly broad, but Stein and Savitz (2009) observed similar findings with the alternatives suggested.

- In an occupational study with even much higher serum concentrations of PFOS (Olsen et al. 2003), Grice et al. (2007) reported no statistically significant associations between various categories of cumulative exposure to PFOS and birth weight. The PFOS exposure in that study was also highly correlated with PFOA (Olsen et al. 2003).

In conclusion, the initial observations suggesting small decreases in mean birth weight were associated with PFOA (Apelberg et al. 2007; Fei et al. 2007), have not been consistently replicated by others. Furthermore, these small changes in birth weight likely have minimal clinical relevance (Savitz et al. 2007; Steenland et al. 2010). Additionally, the varying study results may be disparate because of the different covariates included in the statistical models by the investigators (Olsen et al. 2009).

In addition, time to pregnancy has been evaluated in several studies.

- Fei et al. (2009) reported women with higher PFOA and PFOS concentrations had a longer time to pregnancy (TTP) that indicated infertility. Time to pregnancy is defined as the amount of time it takes to become pregnant when pregnancy is planned. A period of > 12 months was defined as being infertile. Fei et al. (2007a) previously acknowledged several points about their database, including: 1) the lowest exposure quartile differed from the upper three for several of these parameters; 2) parity was the covariate that appeared to most markedly change regression coefficients; and 3) PFOA and PFOS levels were higher in nulliparous women than multiparous women. Despite these concerns, they never showed data stratified by parity in any of their studies except for breast feeding (Fei et al. 2010). Their odds ratios have been adjusted for parity, without interaction terms, and this may have masked important effects that could be seen with stratified odds ratios. Olsen et al. (2009) alluded to this in their critique of Fei et al.'s time to pregnancy results.

- Given the reasonable assumption that perfluorochemical levels will be lower after a pregnancy, Olsen et al. (2010) suggested a longer interval between births would result in more time for a woman to absorb PFOA and other perfluorochemicals that would replace the loss incurred through placental transfer to the fetus. Women who begin with comparable PFOA and PFOS concentrations and equal parity may have different concentrations at their next birth based on the time between births. All else being equal, those women with longer TTP will have longer intervals of time between births and so may have higher perfluorochemical levels prior to the next pregnancy. This would result in longer TTP measurements associated with PFC levels, but the direction of the causality would be backwards; it would be the longer time between births (including the TTP) that resulted in higher PFC concentrations.

- This issue has been initially addressed via a preliminary analysis of the Norwegian Mother and Child cohort study (Whitworth et al. 2010). From a random sample of 425 cases of TTP (> 12 months) and 499 controls (TTP ≤ 12 months), adjusted (maternal age and BMI) odds ratios were estimated for PFOA quartiles stratified by parity. Median PFOA concentration was 2.3 ng/mL (interquartile range 1.73 – 3.0 ng/mL). This is consistent with levels found in the general population. Overall, there was no association

Some studies also evaluated semen quality:

- Joensen et al. (2009) conducted a cross-sectional analysis of frozen (-20°C), archived (5 years) serum samples collected from 105 Danish male military recruits and measured the concentrations of nine perfluorochemicals, including PFOA and PFOS, as well as several sex hormones. They also evaluated semen parameters that were originally measured at the date of examination. Focusing their analyses on PFOA (median 4.9 ng/mL), Joensen et al. did not observe statistically significant associations with testosterone, estradiol, sex hormone binding globulin, luteinizing hormone (LH), follicle stimulating hormone (FSH), or inhibin B. All regression coefficients were not statistically significant, but they were negative in direction for PFOA with semen volume, sperm concentration, count, motility, and morphology. Joensen et al. concluded that PFOA levels, as well as other perfluorochemicals they studied, on an aggregate basis, may contribute to unexplained low semen quality seen in the general population, but cautioned that their results were preliminary.

- To further investigate the question of reduced semen quality, Raymer et al. (2010) examined a total of 256 men who were recruited from those who presented with their partners to the Duke University fertility clinic for an infertility assessment. Blood and semen were collected and analyzed for PFOA (and PFOS), FSH, LH, prolactin, estradiol, T3, T4, and free and total testosterone. Logistic and linear modeling were performed with semen profile measurements as outcomes and PFOS, PFOA, and PFOS*PFOA interaction in semen and plasma as explanatory variables, controlling for age and duration of abstinence. Plasma PFOA was positively associated with concentrations of LH in plasma. When evaluating the semen categorical parameters as a function of perfluorinated compound concentration, the odds ratio of "abnormal" viscosity in semen was significantly different from the null hypothesis as a function of PFOA concentration in semen. In the regression models for the continuous semen profile variables (adjusted for age and duration of abstinence), only semen pH with the PFOS*PFOA interaction term was statistically significant. All other semen profile variables, including sperm concentration and the swim-up variable, an important indicator of in-vitro fertilization success probability, were not associated with semen or plasma PFOA (or PFOS) concentrations.

- Another epidemiologic study (only an abstract available) also reported no association between semen quality and perfluorochemical concentrations that were measured in both serum and semen (Dahl et al. 2010). A total of 37 men participated who had a mean serum concentration of 21.3 ng/mL. Four subjects had detectable levels of PFOA in

Other epidemiologic studies have looked at development in children:

- The Fei et al. studies described above, using the same 1400 maternal serum samples, also investigated associations with maternally reported developmental milestones in infancy (Fei et al. 2010a) and risk for hospitalization for infectious diseases in early childhood (Fei et al. 2010b). Fei et al. did not observe adverse associations for either of these endpoints with the measured maternal first trimester serum concentrations of PFOA or PFOS. For developmental milestones, mothers were questioned about motor and mental development of the infants. Information on hospitalizations for infections were obtained by linkage to the Danish National Hospital Discharge Register. According to the authors, after stratifying by the child's age at infection, the 1st trimester maternal serum concentrations of PFOA or PFOS were associated with lower risks of hospitalization during the first year of life but with no consistent dose-response pattern. After the first year, there were no apparent patterns of risk for hospitalization for either PFOA or PFOS.

- Christensen et al. (2010) conducted a nested case-control study of girls who reported menarche before the age of 11.5 years compared to a random sample. Exposure was considered to be their mothers' pregnancy serum perfluorochemical concentrations. The authors concluded that gestational PFC exposure during pregnancy did not appear to be associated with age at menarche in this cohort.

- The C8 Science Panel has recently issued a two-page report on its website regarding patterns of age of puberty among children aged 8 – 18 at the time of the C8 Health Project survey (2005-2006) with corresponding serum measurements of PFOA and PFOS. In other words, this is another cross-sectional investigation based on the C8 Health Project survey. According to this report, higher exposure to either PFOA or PFOS was associated with reduced odds of having reached puberty in girls. For boys, an association was seen for PFOS but not PFOA. The C8 Science Panel exercised caution in the interpretation of these results because of the inability to address temporality and the fact that menarche was self-reported. As stated by the C8 Science Panel, "it may be that growth changes associated with puberty lead to changes in PFOA and PFOS blood levels, rather than these compounds having any effect on age at puberty."

- In another cross-sectional study of NHANES data, Hoffman et al. (2010) reported a statistically significant association between exposure to polyfluoroalkyl chemicals and attention deficit hyperactivity disorder (ADHD) in children aged 12 – 15 years. Of the 571 children with measured serum concentrations, 48 were reported by their parents to have been diagnosed with ADHD. The adjusted odds ratio for PFOS for a 1 ng/mL (part per billion) increase in serum PFOS was 1.03 (95a% CI 1.01 – 1.05). However, such an extremely narrow (precise) confidence interval, given only 48 cases, suggests some type of statistical error in the investigators' data analyses. Similar findings were observed for PFOA.

In short, there is a large body of information pertaining to developmental outcomes published in the peer-reviewed scientific literature which is not cited in the draft assessment. Many of these studies have examined the same cross-sectional data collected during either NHANES or the C8 Health Project. Reference to the two review papers (Olsen et al. 2009; Steenland et al. 2010) would be an efficient way to summarize much of the extensive literature, which is not adequately represented by reference to the two studies mentioned in the draft.

**"Public Health Implications" Section**

The last paragraph on page 34 mentions the publication by Lundin et al. (2009) that reported positive statistical associations between PFOA and mortality from prostate cancer, cerebrovascular disease, and diabetes among 3M workers at the Cottage Grove plant. However, this paragraph fails to discuss the reservations Lundin et al. had regarding these data and the statistically significant *decreased* risks for prostate cancer and cerebrovascular disease among the lowest exposed workers that contributed to these statistical associations.

Here again, we provide a brief description of the epidemiologic literature for MDH's benefit. Lundin et al. did not observe exposure to ammonium perfluorooctanoate (APFO) to be associated with liver or pancreatic cancer mortality. This is important observation given the fact these tumors were reported in the PFOA toxicological research (Lau et al. 2007). For prostate cancer, Lundin et al. reported SMRs (95% CI) with no, probable, and definite PFOA exposure strata at 0.4 (0.1 – 0.9), 0.9 (0.4 – 1.8), and 2.1 (0.4 – 6.1), respectively, none of them statistically significant. Because of the statistically significantly low SMR for prostate cancer in the 'no' exposed strata, any internal referent analyses would then magnify the associations seen. Thus, Lundin et al. reported hazard ratios of 1.0 (referent), 3.0 (0.9 – 9.7), and 6.6 (1.1 – 37.7) for nonexposed, moderately exposed, and highly exposed APFO workers, respectively, when using the internal referent population. The same phenomenon was observed for deaths from cerebrovascular disease where the stratum specific SMRs were 0.5 (0.3 – 0.8), 0.7 (0.4 – 1.1), and 1.6 (0.5 – 3.7), respectively (again not statistically significant). Lundin et al. cautioned that "while an internal referent population may provide a more valid comparison, the interpretation of this internal analysis should consider the stratum specific prostate cancer and cerebrovascular disease SMRs. The SMRs for the exposed categories were modestly above unity, while the nonexposed members of the cohort were markedly below." In sum, it is important to interpreting the study to understand that the reported increases were based on comparisons of the higher-dose groups to the lowest exposed group, which had a deficit for these diseases.

Additional insights regarding prostate caner risk and PFOA can be gained by examining a DuPont cohort of workers who may have had occupational exposure to APFO that was used as a processing aid in the emulsification of fluoropolymers (Leonard et al. 2008). Unfortunately, this particular study did not conduct PFOA-specific analyses. In this cohort of 6,027 men and women who had worked at the DuPont plant between 1948 and 2002, there were 12 deaths from prostate cancer compared to 18.4 expected (SMR 0.65, 95% CI 0.34 – 1.14) and 35 observed deaths from cerebrovascular disease compared to 40.6 expected (SMR 0.86, 95% CI 0.60 – 1.20). Expected deaths were based on a DuPont worker reference population.

Among an occupational cohort that had potential exposure to PFOS, as well as PFOA, Alexander et al. (2003) reported no deaths from prostate cancer at the 3M Company's Decatur, Alabama manufacturing facility. An additional analysis of self-reported prostate cancer at this site did not reveal any statistically significant associations with PFOS (Grice et al. 2007). An unexpected increased mortality risk for bladder cancer was observed (Alexander et al. 2003) but this was not confirmed with a subsequent incidence study of bladder cancer within the same workforce (Alexander and Olsen et al. 2007). The draft assessment mentions the mortality study (Alexander et al. 2003), but fails to reference the follow-up incidence study. Because bladder cancer generally is not fatal, the incidence data are more meaningful and should be addressed.

Another major epidemiologic study not cited in the draft Assessment was a Danish case-cohort study of 772 prostate cancer cases diagnosed between 1993 and 1997 (Eriksen et al. 2009). These investigators did not observe statistically significant trends for p adjusted incidence rate ratios for prostate cancer and increasing quartiles of PFOA or PFOS serum concentrations. Nor were there significant increased trends for adjusted incidence rate ratios of bladder cancer (n = 312 cases), pancreatic cancer (n = 128 cases), or liver cancer (n = 67 cases). Average serum PFOA and PFOS concentrations approximated those of the general U.S. population prior to the 3M phase-out of its production of perfluorooctanyl chemistry (i.e., approximately 5 ng/mL PFOA and 30 ng/mL PFOS).

In both the Lundin et al. (2009) and Leonard et al. (2008) studies of 3M and DuPont workers, there were more observed deaths from diabetes than expected. Compared to their least exposed 3M workers (Hazard Ratio = 1.0), Lundin et al. reported an HR of 3.7 for moderately exposed APFO production employee (95% CI 1.4 – 10.0) but no deaths were observed among the highest exposed workers. In their DuPont cohort, Leonard et al. reported a total of 22 deaths from diabetes compared to 11.2 expected (SMR 1.97, 95% CI 1.23 – 2.98).

The associations regarding diabetes in the occupational cohorts prompted the C8 Science Panel to conduct a cross-sectional analysis of type II diabetes in their Ohio-West Virginia community study (MacNeil et al. 2009). As part of this study, MacNeil et al. medically validated the self-reported diabetes cases. Adjusted for confounders, MacNeil et al. observed a decreased risk for diabetes with increased serum concentrations of PFOA or PFOS. Nor was fasting serum glucose associated with PFOA. In their analyses of NHANES data, Nelson et al. (2009) did not find an association with insulin resistance and serum concentrations of PFOA or PFOS. On the other hand, using the same NHANES database, Li et al. (2009a) did associate PFOS with increased blood insulin, insulin resistance and β-cell function.

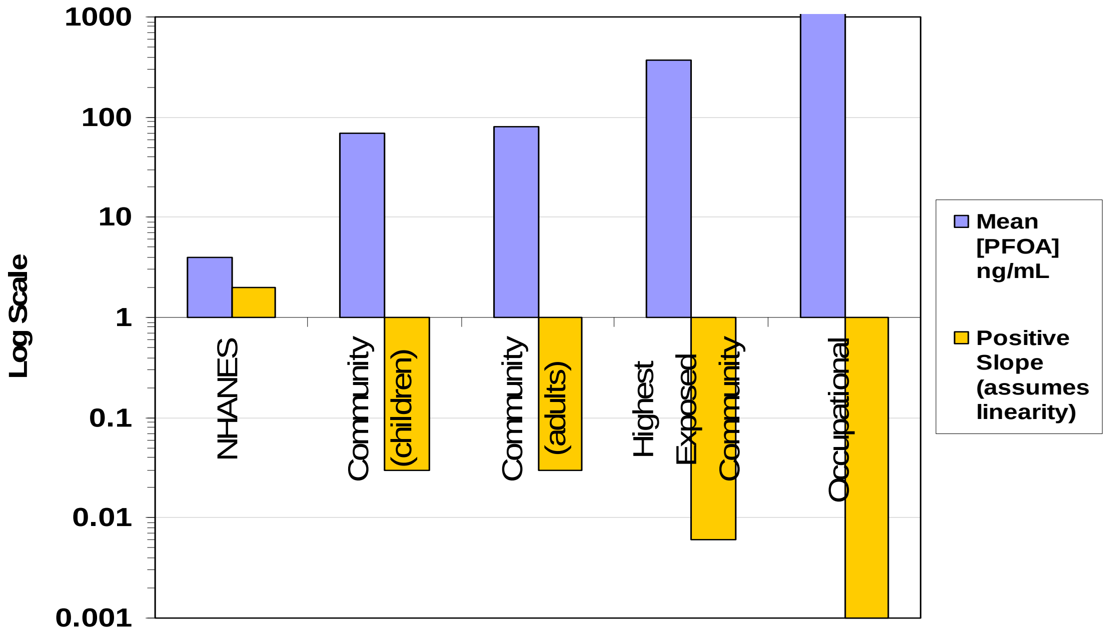
As the NHANES database has been used by multiple investigators, a few words of caution are necessary. First, it bears repeating that all investigations using NHANES data are cross-sectional. Temporality can not be addressed. Several statistical associations have been reported with PFOA and PFOS including total and LDL cholesterol (Nelson et al. 2009), thyroid hormone (Melzer et al. 2010), chronic obstructive lung disease (negative association (Melzer et al. 2010), insulin resistance (Li et al. 2009a) or not (Nelson et al. 2009), increased hepatic enzymes (Lin et al. 2009b), and ADHD (Hoffman et al. 2010). However, the magnitude of change for some of the clinical chemistries with these statistical associations has been small and of questionable clinical relevance. Furthermore, these associations have either not been observed, or the magnitude of change was even smaller yet, for much higher exposed occupational PFOA and/or PFOS populations for cholesterol (Olsen and Zobel 2007; Olsen et al. 2010; Sakr et al. 2007a; 2007b), thyroid hormones (Costa et al. 2007; Olsen et al. 2003; Olsen and Zobel 2007), and hepatic enzymes (Costa et al. 2007; Olsen et al. 2003; 2010; Olsen and Zobel 2007; Sakr et al. 2007a; 2007b). Nor have these associations been reported among the highest exposed community to the contaminated PFOA drinking water in the Ohio-West Virginia area (Emmet et al. 2006). Thus, the NHANES data should be viewed cautiously.

Finally, we note that the brief discussion of epidemiology in the draft assessment does not address cholesterol and perfluorochemicals:

- Based on toxicological evidence, PFOS exposure at sufficiently high dosages resulted in hypolipidemia (decreased cholesterol) in rats, mice, and primates treated in the laboratory with PFOS. The mode of action (i.e., PPARα with possible other nuclear receptors CAR and PXR) is largely understood (Lau et al. 2007). Hypolipidemia was observed in rats administered the ammonium salt of PFOA but neither hypo or hyperlipidemia was reported in a 6-month primate feeding study of PFOA (Butenhoff et al. 2002).

- Contrary to the toxicological evidence, several epidemiologic studies have reported positive associations between non-HDL cholesterol and PFOS and/or PFOA serum concentrations, but the magnitude of these associations decreased as serum concentrations increased. This is illustrated in Figure 3, modified from a table presented in the Steenland et al. (2010) review paper. As seen in Figure 3, as PFOA concentrations increased from 5 ng/mL in the NHANES general population study (Nelson et al. 2009) to approximately 1000 ng/mL in the occupational studies, the slope of the cholesterol linear trend goes in the opposite direction from +2 in the NHANES study to +0.001 in the occupational studies. In other words, as the "dose" (PFOA concentration) increases 3 orders of magnitude, the "effect" (serum cholesterol) decreases 3 orders of magnitude. This does not support a dose-response relationship and suggests non-causal hypotheses. Furthermore, most of these investigations were cross-sectional in design which prevents any assessment of temporality.

**Figure 3. Mean PFOA Concentration (ng/mL) and Slope of Linear Relationship with Cholesterol in Epidemiology Research Studies. Adapted from Table 1 in Steenland et al. (2010a)**



- Two of the studies in Figure 3 pertain to children and adult populations in the Parkersburg communities exposed to PFOA through drinking water (Frisbee et al. 2010; Steenland et al. 2009). In both of these studies there is a striking nonlinear association at less than 25 ng/mL PFOA. There was a minimum increase in cholesterol associated with PFOA above 25 ng/mL. This suggests a possible saturation point in an underlying physiologic condition (Frisbee et al. 2010). Possible reasons may include saturated absorption or binding. To add to the confusion, the two most recently published cross-sectional investigations have not observed any association between total cholesterol (and LDL) and perfluorochemicals among Inuit (Château-Degat et al. 2010) and female adolescent populations (Pinney et al. 2010).

- Although there have been several noteworthy occupational cross-sectional studies published (Olsen et al. 2000; 2003; 2007;Costa et al. 2007; Sakr et al. 2007a), there have been four longitudinal analyses reported (Olsen et al. 2003; 2010; Costa et al. 2007; Sakr et al. 2007b). In the largest of these four studies (Sakr et al. 2007b), a longitudinal follow-up of a cohort of 454 DuPont workers, Sakr et al. reported a 1,000 ng/mL increase in PFOA was significantly associated with a 1.06 mg/dL increase in total cholesterol but was not associated with changes in triglycerides or other lipoproteins after adjusting for potential confounders.

- In an as-yet unpublished study of 3M contract workers who were involved in the demolition and disposal of the building that housed the manufacture of PFOA at the Cottage Grove plant (Olsen et al. 2010), 45 workers' cholesterol levels were essentially unchanged through the course of this work even though they experienced an average of 133 ng/mL increase in serum FPOA concentrations. Their initial average PFOA concentration was 23 ng/mL, approximately the same concentration at which an association was reported to begin in the Frisbee and Steenland Parkersburg studies. It is at this level where the suggestion of a saturated physiologic response might occur. [Note: MDH received this 3M study report in August 2010.]

Although the explanation of the inconsistent positive cholesterol associations will likely remain unanswered for a period of time, two published occupational cohort mortality studies of 3M and DuPont workers have not shown any consistent associations between deaths from heart disease and increasing cumulative weighted exposure categories of PFOA (Lundin et al. 2009; Sakr et al. 2009). Refined exposure matrices should improve the precision of these occupational studies (Raleigh et al. 2010) as well as those conducted in community settings (Shin et al. 2010).

Again, this overview of the current epidemiologic literature supports the conclusion set forth in the draft assessment document that "drinking water from public or private wells that contain PFCs is not expected to harm people's health."

**"Public Health Implications of PFC Exposure" Section**

As the assessment notes on page 34, blood levels of PFCs observed in the East Metro area are well below points of departure in animals used to calculate MDH's HRLs. This discussion should further elaborate that in the animal studies where MDH says there are health effects, those levels were measured in parts per million. In contrast, human serum levels in the East Metro area are measured in parts per billion, roughly 1,000 times lower than levels observed in the animal studies.

On page 35, the draft assessment incorrectly states that sludge from a wastewater treatment plant in Alabama is believed to be responsible for low levels of PFOA found in nearby community water systems. No community water supply wells in the Decatur, Alabama area have had levels of PFOA or PFOS in excess of EPA's Provisional Health Advisory levels.

**Comments on "Child Health Considerations" Section**

On page 36, the discussion of child health considerations definitively states that children have been exposed to low levels of PFCs in drinking water. 3M believes it would be appropriate to revise that statement to note that children *may* have been exposed to low levels of PFCs in drinking water. Although it is plausible that some children have been exposed, it is likely that others have not.

**Comments on "Conclusions" Section**

In the first sentence of the Conclusions on page 36, 3M requests that MDH indicate that 3M *legally* disposed of wastes at Oakdale, Washington County and Woodbury landfills. *See* MPCA, *3M Oakdale Disposal Site: Proposed Cleanup Plan for PFCs* (May 2008), http://www.pca.state.mn.us/index.php/waste/waste-and-cleanup/cleanup-programs-and-topics/topics/perfluorochemicals-pfc/perfluorochemical-pfc-waste-sites.html.

Again, we question whether air emissions or direct contact with waste warrant discussion in the conclusion section.

3M suggests the conclusions also discuss, as set forth above, that there are numerous other sources of PFBA.

3M disagrees with the conclusion that there is insufficient evidence to conclude whether drinking or breathing PFCs in water or air or contact with PFC-containing wastes in the past harmed people's health. Even if past levels in drinking water exceeded HRLs or HBVs, that does not imply that there was ever a risk to health given the large margins of safety incorporated in the HRLs or HBVs. While precise historical levels of PFC exposure are not known, it is known that 3M's chemical production workers had PFC blood serum levels one to two (or sometimes three) orders of magnitude higher than recent levels in persons who have consumed PFCs in drinking water in Minnesota. It is unlikely that exposure via water, or air or waste if any, would have led to higher blood levels than the exposure of the chemical production workers working directly with PFC materials for many years. It is also known that serum levels in the Minnesota population are lower than the levels found in a community in Parkersburg, West Virginia where PFOA was present in drinking water. Moreover, as ATSDR has stated, "it is difficult to envision a health condition that could be attributed solely to exposure to perfluoroalkyls." *Draft Toxicological Profile for Perfluoroalkyls*, p. 207 (2009). As set forth throughout these comments, the large body of epidemiological studies showing no human health effects provide ample basis for MDH to conclude that historical levels of PFCs in Minnesota water or air likely have not harmed local residents' health.

## Comments on "Recommendations" Section

As noted above, MDH's second recommendation - that people avoid trespassing on the 3M-Woodbury Disposal Site - has long been recommended by 3M, and the site is fenced and posted with No Trespassing signs. 3M suggests that recommendation be revised to state "Consistent with 3M's longstanding recommendations, people should continue to avoid trespassing on the 3M-Woodbury Disposal Site."

Also as noted above, 3M questions why MDH recommends considering extending the Cottage Grove municipal water supply to serve areas where private wells contain levels of PFCs in excess of HRLs or HBVs. Elsewhere in the assessment, MDH notes that in the few locations where well advisories have been issued, filters have been used that are effective at removing PFCs from drinking water.

MDH should remove the recommendation regarding the need for monitoring groundwater in the high transmissivity zone at the Woodbury site. Monitoring has been conducted and demonstrates the effectiveness of the containment system.

## References

3M 1963. Report to Woodbury Town Board.

Alexander, BH, Olsen GW, Burris JM, Mandel JH, Mandel JS. 2003. Mortality of employees of a perfluorooctanesulfonyl fluoride manufacturing facility. Occup Environ Med 60:722-729.

Alexander BH, Olsen, GW. 2007. Bladder cancer in perfluorooctanesulfonyl fluoride manufacturing workers. Ann Epidemiol 17: 471-478.

Andersen ME, Butenhoff JL, Chang SC, Farrar DG, Kennedy GL Jr, Lau C, Olsen GW, Seed J, Wallace KB. 2008. Perfluoroalkyl acids and related chemistries--toxicokinetics and modes of action. Toxicol Sci 102:3-14.

Apelberg BJ. 2006. Fetal exposure to perfluorinated compounds: distributions and determinants of exposure and relationships with weight and size at birth. Doctoral dissertation. Baltimore, MD:Johns Hopkins University. August 2006.

Apelberg BJ, Whitter FR, Herbstman JB, Calafat AM, Halden RU, Needham LL, Goldman LR. 2007. Cord serum concentrations of perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA) in relation to weight and size at birth. Environ Health Perspect 115:1670-1676.

Ashenmacher, T. 1994. Letter from Thomas Ashenmacher of 3M to Kenneth Haberman of MPCA transmitting the Investigation Report for the Woodbury Site (Feb. 25, 1994).

Baltutis, TF 1985. Letter from Thomas F. Baltutis of 3M to J. Liefert of MPCA (June 10, 1985).

Biegel LB, Hurtt ME, Frame SR, O'Connor JC, Cook JC. 2001. Mechanisms of extrahepatic tumor induction by peroxisome proliferators in male CD rats. Toxicol Sci 60:44-55.

Bjork JA and Wallace KB. 2009. Structure-activity relationships and human relevance for perfluoroalkyl acid-induced transcriptional activation of peroxisome proliferation in liver cell cultures. Toxicol Sci 111:89-99.

Butenhoff J, Costa G, Elcombe C, Farrar D, Hansen K, Iwai H, Jung R, Kennedy G Jr, Lieder P, Olsen G, Thomford P. 2002. Toxicity of ammonium perfluorooctanoate in male cynomolgus monkeys after oral dosing for 6 months. Toxicol Sci 69:244-257.

Butenhoff JL, Kennedy GL Jr, Frame SR, O'Connor JC, York RG. 2004. The reproductive toxicology of ammonium perfluorooctanoate (APFO) in the rat. Toxicology 196:95-116.

Capen, CC. 1997. Mechanistic data and risk assessment of selected toxic end points of the thyroid gland. Toxicol Pathol 25:39-48.

Chang SC, Das K, Ehresman DJ, Ellefson ME, Gorman GS, Hart JA, Noker PE, Tan YM, Lieder PH, Lau C, Olsen GW, Butenhoff JL. 2008. Comparative pharmacokinetics of perfluorobutyrate in rats, mice, monkeys, and humans and relevance to human exposure via drinking water. Toxicl Sci 104:40-53.

Chang S, Bjork J, Wallace K, Butenhoff J. 2009. Effects of perfluorobutyrate on thyroid hormone status in rats (abstract). Repro Toxicol 27:417.

Chateau-Degat, ML, Pereg D, Dallaire, R, Ayotte P, Dery S, Dewailly E. 2010 Effects of perfluorooctanesulfonate exposure on plasma lipid levels in the Inuit population of Nunavik (Northern Quebec). Environ Res 110: 710-717.

Christiansen, KY, Maisonet, M, Rubin C, Holmes A, Calafat AM, Kato K, Flanders WD, Heron J, McGeehin MA, Marcus, M. 2010. Exposure to polyfluoroalkyl chemicals during pregnancy is not associated with offspring age at menarche in a contemporary British cohort. Environ Int 2010. doi:10.1016/j.envint.2010.08.007.

Costa G, Sartori S, Consonni D. 2009. Thirty years of medical surveillance in perfluorooctanoic acid production workers. J Occup Environ Med 51:364-372.

Curran PG and DeGroot LJ. 1991. The effect of hepatic enzyme-inducing drugs on thyroid hormones and the thyroid gland. Endocr Rev 12:135-150.

Curran I, Hierlihy SL, Liston V, Pantazopoulos P, Nunnikhoven A, Tittlemier S, Barker M, Trick K, Bondy G. 2008. Altered fatty acid homeostasis and related toxicologic sequelae in rats exposed to dietary potassium perfluorooctanesulfonate (PFOS). J Toxicol Environ Health A 71:1526-1541.

Curti, B. 1980. Memorandum from 3M Industrial Relations Manager Bernie Curti describing Minnesota OSHA inspection (Apr. 4, 1998).

Dahl SK, Robins JC, Williams DB, Kubatova A, Wu DH, Thomas MA. 2010 Perfluorooctanoic acid, an environmental toxin, confers no adverse effect on semen parameters (abstract P-466). Fertil Steril. S227

D'eon and Mabury. 2007. Production of Perfluorinated Carboxylic Acids (PFCAs) from the Biotransformation of Polyfluoroalkyl Phosphate Surfactants (PAPS): Exploring Routes of Human Contamination, Environ. Sci. Technol. (41) 4799-4805.

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

Elcombe CR, Elcombe BM, Foster JR, Farrar DG, Jung R, Chang SC, Kennedy GL, Butenhoff JL. 2010. Hepatocellular hypertrophy and cell proliferation in Sprague-Dawley rats following dietary exposure to ammonium perfluorooctanoate occurs through increased activation of the xenosensor nuclear receptors PPARalpha and CAR/PXR. Arch Toxicol 84:787-798.

Emmett EA, Zhang H, Shofer FS. Community exposure to perfluorooctanoate: relationships between serum levels and certain health parameters. J Occup Environ Med 48:771-779.

Eriksen KT, Sörensen M, McLaughlin JK, Lipworth L, Tjönneland A, Overvad K, Raaschou-Nielsen O. 2009. Perfluorooctanoate and perfluorooctanesulfonate plasma levels and risk of cancer in the general Danish population. J Natl Cancer Inst 101:605-609.

Fasano WJ, Kennedy GL, Szostek B, Farrar DG, Ward RJ, Haroun L, Hinderliter PM. 2005. Penetration of ammonium perfluorooctanoate through rat and human skin in vitro. Drug Chem Toxicol 28:79-90.

Fei C, McLaughlin JK, Tarone RE, Olsen J. 2007. Perfluorinated chemicals and fetal growth: a study within the Danish National Birth Cohort. Environ Health Perspect 115:1677-1682.

Fei C, McLaughlin JK, Tarone RE, Olsen J. 2008a. Fetal growth indicators and perfluorinated chemicals: a study in the Danish National Birth Cohort. Am J Epidemiol 168:66-72.

Fei C, McLaughlin JK, Lipworth L, Olsen J. 2008b. Prenatal exposure to PFOA and PFOS and maternally reported developmental milestones in infancy. Environ Health Perspect 116:1391-1395.

Fei C, McLaughlin JK, Lipworth L, Olsen J. 2009. Maternal levels of perfluorinated chemicals and subfecundity. Human Repro 24:1200-1205.

Fei C, Olsen J. 2009. Letter to the Editor. Am J Epidemiol doi:10.1093/aje/kwp384

Fei C, McLaughlin JK, Lipworth L, Olsen J. 2010a. Maternal concentrations of perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA) and duration of breastfeeding. Scand J Work Environ Health 36:413-421.

Fei C, McLaughlin JK, Lipworth L, Olsen J. 2010b. Prenatal exposure to PFOA and PFOS and risk of hospitalization for infectious diseases in early childhood. Environ Res doi:10.1016/j.envres.2010.08.004.

Foreman JE, Chang SC, Ehresman DJ, Butenhoff JL, Anderson CR, Palkar PS, Kang BH, Gonzalez FJ, Peters JM. 2009. Differential hepatic effects of perfluorobutyrate (PFBA) mediated by mouse and human PPARa. Toxicological Sciences 110:204-211.

Frisbee SJ, Shankar A, Knox SS, Steenland K, Savitz DA, Fletcher T, Ducatman AM. 2010. Perfluorooctanoic acid, perfluorooctanesulfonate and serum lipids in children and adolescents. Arch Pediatr Adolesc Med 164:860-869.

Garry, VF 1980. Memorandum from Vincent F. Garry of MDH to Steve Mackey of Minnesota OSHA describing Chemolite Plant Investigation (May 15, 1980).

Gonzalez FJ, Shah YM. 2008. PPARalpha: mechanism of species differences and hepatocarcinogenesis of peroxisome proliferators. Toxicology 246:2-8.

Grice MM, Alexander BH, Hoffbeck R, Kampa DM. 2007. Self-reported medical conditions in perfluorooctanesulfonyl fluoride manufacturing workers. J Occup Environ Med 49:722-729.

Hamm MP, Cherry NM, Chan E, Martin JW, Gurstyn I. 2009. Maternal exposure to perfluorinated acids and fetal growth. J Exp Sci Environ Epi doi:10.1038/jes.2009.57. Online 28 October 2009.

Hoffman K, Webster TF, Weisskopf, MG, Weinberg J, Vieria VM. Exposure to polyfluoroalkyl chemicals and attention deficit hyperactivity disorder in U.S. children aged 12-15 years. Environ Health Perspect doi:10.1289/ehp.1001898. Online 15 June 2010.

Inoue K, Okada F, Ito R, Kato S, Sasaki S, Nakajima S, Uno A, Saijo Y, Sata F, Yoshimura Y, Kishi R, Nakazawa H. 2004. Perfluorooctane sulfonate (PFOS) and related perfluorinated compounds in human maternal cord blood samples: assessment of PFOS exposure in a susceptible population during pregnancy. Environ Health Perspect 112:1204-1207.

Joensen UN, Bossi R, Leffers H, Astrup A, Skakkebaek NE, Jørgensen N. 2009. Do perfluoroalkyl compounds impair human semen quality? Environ Health Perspect 117:923-927.

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

Klaunig JE, Babich MA, Baetcke KP, Cook JC, Corton JC, David RM, DeLuca JG, Lai DY, McKee RH, Peters JM, Roberts RA, Fenner-Crisp PA. 2003. PPARalpha agonist-induced rodent tumors: modes of action and human relevance. Crit Rev Toxicol 33:655-780.

Lau C, Butenhoff JL, Rogers JM. 2004. The developmental toxicity of perfluoroalkyl acids and their derivatives. Toxicol Appl Pharmacol 198:231-241.

Lau C, Anitole K, Hodes C, Lai D, Pfahles-Hutchens A, Seed J. 2007. Perfluoroalkyl acids: a review of monitoring and toxicological findings. Toxicol Sci 99:366-394.

Lee et al., 2010. Biodegradation of Polyfluroalkyl Phosphates as a Source of Perfluorinated Acids to the Environment, Environ. Sci. Technol. (44): 3305-3310.

Lefebvre DE, Curran I, Armstrong C, Coady L, Parenteau M, Liston V, Barker M, Aziz S, Rutherford K, Bellon-Gagnon P, Shenton J, Mehta R, Bondy G. 2008. Immunomodulatory Effects of Dietary Potassium Perfluorooctane Sulfonate (PFOS) Exposure in Adult Sprague-Dawley Rats. J Toxicol Environ Health A 71:1516-1525.

Leonard RC, Kreckman KH, Sark CJ, Symons JM. 2008. Retrospective cohort mortality study of workers in a polymer production plant including a reference population of regional workers. Ann Epidemiol 18:15-22.

Lin CY, Lin YC, Chen PC, Lin LY. 2009a. Association among serum perfluoroalkyl chemicals, glucose homeostasis, and metabolic syndrome in adolescents and adults. Diabetes Care 32:702-707.

Lin CY, Lin YC, Chiang CK, Wang WJ, Su YN, Hung KY, Chen PC. 2009b. Investigation of the associations between low-dose serum perfluorinated chemicals and liver enzymes in US adults. Am J Gastroenterol 105:1354-1363.

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

Luebker DJ, York RG, Hansen KJ, Moore JA, Butenhoff JL. 2005. Neonatal mortality from in utero exposure to perfluorooctanesulfonate (PFOS) in Sprague-Dawley rats: dose-response, and biochemical and pharmacokinetic parameters. Toxicology 215:149-169.

Lundin JI, Alexander BH, Olsen GW, Church TR. 2009. Ammonium perfluorooctanoate production and occupational mortality. Epidemiology 20: 921-928.

MacNeil J, Steenland NK, Shankar A, Ducatman A. 2009. A cross-sectional analysis of type II diabetes in a community with exposure to perfluorooctanoic acid (PFOA). Environ Res 109:997-1003.

MDH 1958. Division of Environmental Sanitation Section of Water Pollution Control, Report on Investigation of Waste Disposal at Minnesota Mining and Manufacturing Company Chemolite Plant, Washington County (Sept. 22-26, 1958).

Monroy R, Morrison K, Teo K, Atkinson S, Kubwabo C, Steward B, Foster WG. 2008. Serum levels of perfluoroalkyl compounds in human maternal and umbilical cord blood samples. Environ Res 108:56-62.

MPCA 1971. Operating Permit for Air Pollution Control Equipment on Production and Processing Equipment # 23-71-O-4 (Mar. 10, 1971).

MPCA 1974. Installation Permit for Fluorination of Organic Compounds # 32A-74-I-32 (May 1, 1974).

MPCA 1981. Letter from Michael B. Ayers of MPCA to Russ Susag of 3M (May 4, 1981).

MPCA, 1986. Minnesota Enforcement Decision Document for 3M Chemolite Site (Aug. 7, 1986).

Nakamura T, Ito Y, Yanagiba Y, Ramdhan DH, Kono Y, Naito H, Hayashi Y, Li Y, Aoyama T, Gonzalez FJ, Nakajima T. 2009. Microgram-order ammonium perfluorooctanoate may activate mouse peroxisome proliferator-activated receptor α, but not human PPARα. Toxicology 265:27-33.

Neely NS 1980. Letter from Nancy S. Neely of SCS Engineers to Russ Susag of 3M transmitting report and noting a copy was also sent to Gary Kimball at MPCA (Aug. 11, 1980).
Nolan LA, Nolan HM, Schofer FS, Rodway NV, Emmett EA. 2009. The relationship between birth weight, gestational age and perfluorooctanoic acid (PFOA)-contaminated public drinking water. Repro Toxicol 27:231-238.

Nolan LA, Nolan JM, Shofer FS, Rodway NV, Emmett EA. 2010. Congenital anomalies, labor/delivery complications, maternal risk factors and their relationship with perfluorooctanoic acid (PFOA)-contaminated public drinking water. Repro Toxicol 29:147-155.

Olsen GW, Logan PW, Hansen KJ, Simpson CA, Burris JM, Burlew MM, Vorarath PP, Pothapragada V, Schumpert JC, Mandel JH. 2003. An occupational exposure assessment of a perfluorooctanesulfonyl fluoride production site: biomonitoring. AIHAJ 64:651-659.

Olsen GW, Burris JM, Burlew MM, Mandel JH. 2003. Epidemiologic assessment of worker perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA) concentrations and medical surveillance examinations. J Occup Environ Med 45: 260-270.

Olsen GW, Burlew MM, Marshall JC, Burris JM, Mandel JH. 2004. Analysis of episodes of care in a perfluorooctanesulfonyl fluoride production facility. J Occup Environ Med 46:837-846.

Olsen GW, Zobel LR. 2007. Assessment of lipid, hepatic, and thyroid parameters with serum perfluorooctanoate (PFOA) concentrations in fluorochemical production workers. Int Arch Occup Environ Health 81:231-246.

Olsen GW, Butenhoff JL, Zobel LR. 2009. Perfluoroalkyl chemicals and human fetal development: An epidemiologic review with clinical and toxicological perspectives. Repro Toxicol 27:212-230.

Olsen GW, Bigson BA, Ehresman DJ, Madsen DC. 2010. Biomonitoring assessment of the 3M Cottage Grove Building 15 demolition and disposal project. Phase I. August 20, 2010. St. Paul, MN:3M company. August 20, 2010.

Peden-Adams MM, Keller JM, Eudaly JG, Berger J, Gilkeson GS, Keil DE. 2008. Suppression of humoral immunity in mice following exposure to perfluorooctane sulfonate. Toxicol Sci 104:144-154.

Pilney J. 1982. Letter from John Pilney of 3M to Michael B. Ayers of MPCA transmitting Final Report (Phase I) and Site Characterization Work Plan (Phase II) of the Investigation of Waste Disposal Areas at the 3M Chemolite Plant (Mar. 18, 1982).

Pilney J. 1983. Letter from John Pilney of 3M to Michael B. Ayers of MPCA transmitting hydrogeological investigation report (July 13, 1983).
Pinney SM, Biro FM, Herrick R, Yaghjyan L, Calafat A, Kato K, Succop P, Brown MK, Hernick A, Bornschein R. 2010. Exposure to perfluorooctanoic acid (PFOA) and plasma lipid concentrations in young girls. Abstract P54. U.S. EPA PFAA Days III. Research Triangle Park, NC

Qazi MR, Bogdanska J, Butenhoff J, Nelson BD, DePierre JW, Abedi-Valugerdi M. 2009a. High-dose, short-term exposure of mice to perfluorooctanesulfonate (PFOS) or perfluorooctanoate (PFOA) affects the number of circulating neutrophils differently, but enhances the inflammatory responses of macrophages to lipopolysaccharide (LPS) in a similar fashion. Toxicology 262:207-214.

Qazi MR, Xia Z, Bogdanska J, Chang SC, Ehresman DJ, Butenhoff JL, Nelson BD, Depierre JW, Abedi-Valugerdi M. 2009b. The atrophy and changes in the cellular compositions of the thymus and spleen observed in mice subjected to short-term exposure to perfluorooctanesulfonate are high-dose phenomena mediated in part by peroxisome proliferator-activated receptor-alpha (PPARalpha). Toxicology 260, 68-76.

Qazi MR, Nelson BD, DePierre JW, Abedi-Valugerdi M. 2010a. 28-day dietary exposure of mice to a low total dose (7 mg/kg) of perfluorooctanesulfonate (PFOS) alters neither the cellular compositions of the thymus and spleen nor humoral immune responses: Does the route of administration play a pivotal role in PFOS-induced immunotoxicity? Toxicology 267:132-139.

Qazi MR, Abedi MR, Nelson BD, Depierre JW, Abedi-Valugerdi M. 2010b. Dietary exposure to perfluorooctanoate or perfluorooctane sulfonate induces hypertrophy in centrilobular hepatocytes and alters the hepatic immune status in mice. Int Immunopharmacol [Epub ahead of print].

Raleigh KK, Alexander BH, Ramachanddran G, Morey SZ, Logan P, Olsen G. 2010. Occupational exposure to ammonium perfluorooctanoate: exploring two exposure models to account for workers' exposure over time. Abstract P53. U.S. EPA PFAA Days III. Research Triangle Park, NC.

Raymer JA, Michael LC, Studabaker WS, Olsen GW, Sloa CS, Wilcosky W, Walmer D. 2010a. Concentrations of perfluorooctanesulfonate and perfluorooctanoic acid in human males and their associations with semen parameters. Abstract P56. U.S. EPA PFAA Days III. Research Triangle Park, NC.

Riehle, PF 1980a, 1980b. Memoranda from 3M Cottage Grove Plant Manager P.F. Riehle to R.H. Ahlness, et al. describing Minnesota OSHA inspection (Mar. 24, 1980 and Apr. 4, 1980).

Rosen MB, Lau C, Corton JC. 2009. Does exposure to perfluoroalkyl acids present a risk to human health? Toxicol Sci 111:1-3.

Sakr CJ, Leonard RC. Kreckmann KH, Slade MD, Cullen MR. 2007. Longitudinal study of serum lipids and liver enzymes in workers with occupational exposure to ammonium perfluorooctanoate. J Occup Environ Med 49:872-879.

Sakr CJ, Kreckmann KH, Green JW, Gillies PJ, Reynolds JL, Leonard RC. 2007. Cross-sectional study of lipids and liver enzymes related to a serum biomarker of exposure (ammonium perfluorooctanoate or APFO) as part of a general health survey in a cohort of occupationally exposed workers. J Occup Environ Med 49:1086-1096.

Sakr CJ, Symons JM, Kreckmann KH, Leonard RC. Ischaemic heart disease mortality study among workers with occupational exposure to ammonium perfluorooctanoate. Occup Environ Med 66:699-703.

Santoro, MA. 1981. Letter from Mike Santoro of 3M to Michael B. Ayers of MPCA transmitting work plan (Nov. 17, 1981).

Savitz DA. 2007. Biomarkers of perfluorinated chemicals and birth weight. Environ Health Perspect 115:A528-529.

Seacat AM, Thomford PJ, Hansen KJ, Clemen LA, Eldridge SR, Elcombe CR, Butenhoff JL. 2003. Sub-chronic dietary toxicity of potassium perfluorooctanesulfonate in rats. Toxicology 183:117-131. [Erratum in Toxicology (2003) 183:117-131.]

Seacat AM, Thomford PJ, Hansen KJ, Olsen GW, Case MT, Butenhoff JL. 2002. Subchronic toxicity studies on perfluorooctanesulfonate potassium salt in cynomolgus monkeys. Toxicol Sci 68:249-264.

Shin HM, Vieria V, Ryan PB, Etwiler R, Sanders B, Steenland K, Bartell S. 2010. Retrospective exposure estimation for perfluorooctanoic acid (PFOA) for participants in the C8 Health Project. Abstract P47. U.S. EPA PFAA Days III. Research Triangle Park, NC
Steenland K, Tinker S, Frisbee S, Ducatman A, Vaccarino V. 2009. Association of perfluorooctanoic acid and perfluorooctane sulfonate with serum lipids among adults living near a chemical plant. Am J Epidemiol 170:1268-1278.

Steenland K, Fletcher T, Savitz DA. 2010a. Epidemiologic evidence on the health effects of perfluorooctanoic acid (PFOA). Environ Health Perspect 118:1100-1108.

Steenland K, Tinker S, Shankar A, Ducatman A. 2010b. Association of perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) with uric acid among adults with elevated community exposure to PFOA. Environ Health Perspect 118:229-233.

Stein CR, Savitz DA, Dougan M. 2009. Serum levels of perfluorooctanoic acid and perfluorooctane sulfonate and pregnancy outcome. Am J Epidemiol 170:837-846.

Stein CR, Savitz DA. 2009. The Authors Reply. Am J Epidemiol doi:10.1093/aje/kwp385.

Susag R. 1976. Letter from Russ Susag of 3M to Richard Svanda of MPCA (Nov. 26, 1976).

Susag R. 1981. Letter from Russ Susag of 3M to Louis Breinhurst of MPCA (May 29, 1981).

University of Dayton Research Institute 2003. Incineration Study: Final Report, Laboratory-Scale Thermal Degradation of Perfluoro-Octanyl Sulfonate and Related Precursors. EPA Docket AR-226-1366, 1367

USEPA. 2002. Hepatocellular Hypertrophy. HED ToxSAC 2002 HED Guidance Doc G2002.01.

USEPA. 2003. Proposed OPPTS science policy: PPARa-mediated hepatocarcinogenesis in rodents and relevance to human health risk assessments. USEPA, Washington, D.C., pp. 1-39.

Walters MW, Bjork JA, Wallace KB. 2009. Perfluorooctanoic acid stimulated mitochondrial biogenesis and gene transcription in rats. Toxicology 264:10-15.

Washino N, Saijo Y, Sasaki S, Kato S, Ban S, Konishi K, Nakata A, Iwasaki Y, Saito K, Nakazawa H, Kishi R. 2009. Correlations between prenatal exposure to perfluorinated chemicals and reduced fetal growth. Environ Health Perspect 117:660-667.

Weston 1986. Final Remedial Investigation Report for the 3M Chemolite Center, submitted to MPCA (1986).

Whitworth KW, Haug LS, Baird DD, Becher G, Hoppin JA, Eggesbo M, Skjaerven R, Thomsen C, Longnecker MP. 2010. Preliminary results on subfecundity and plasma concentrations of perfluorinated compounds during pregnancy in the Norwegian Mother and Child Cohort study, 2002-2004. Abstract P60. U.S. EPA PFAA Days III. Research Triangle Park, NC.

Zheng L, Dong GH, Jin YH, He QC. 2009. Immunotoxic changes associated with a 7-day oral exposure to perfluorooctanesulfonate (PFOS) in adult male C57BL/6 mice. Arch Toxicol 83:679-68