

Health Consultation

3M CHEMOLITE

PERFLUOROCHEMICAL RELEASES AT THE
3M – COTTAGE GROVE FACILITY

CITY OF COTTAGE GROVE, WASHINGTON COUNTY, MINNESOTA

EPA FACILITY ID: MND006172969

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U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation
Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

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

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

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FOREWORD

This document summarizes public health concerns at a hazardous waste site in Minnesota. It is based on 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. Rather, MDH relies on information provided by the Minnesota Pollution Control Agency (MPCA), the U.S. Environmental Protection Agency (EPA), and other government agencies, private businesses, and the general public.
- *Evaluating health effects:* If there is evidence that people are being exposed—or could be exposed—to hazardous substances, 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 the evaluation report, MDH outlines its conclusions regarding any potential health threat posed by a site and offers recommendations for reducing or eliminating human exposure to contaminants. The role of MDH in dealing with hazardous waste sites 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 cleaning up 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.*

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Summary

3M produced perfluorochemicals (PFCs) at their Cottage Grove facility from the late 1940s until 2002 (on a pilot scale or in full production), using an electrofluorochemical process. PFC products were produced, handled, used or packaged at several locations at the site. During production, air emissions of PFCs occurred, and may have extended off the site property. Wastes from the PFC production process were disposed in an on-site pit, and possibly in off-site locations as well. Wastewater treatment plant effluent containing PFCs was discharged to the adjacent Mississippi River for decades, and sludge from the wastewater treatment plant and ponds that contained PFCs were also disposed on site. Fire-fighting foams containing PFCs were also used at a fire-training area on the west side of the site.

The results of limited environmental monitoring to date indicate that groundwater beneath the site is contaminated with perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA), in some locations at levels significantly in excess of the MDH Health-Based Values (HBVs) for groundwater. The full extent of the groundwater contamination has not been identified. Much of the contaminated groundwater is contained and collected by an extensive system of production wells, and is processed through the site wastewater treatment plant. The plant has not historically been able to remove the PFCs from the effluent. However, the recent (2004) addition of a large granular activated carbon treatment system has effectively eliminated PFC discharges to the Mississippi River. An area of shallow groundwater contamination (in the D1 Area) is not captured by the production wells, and likely discharges to the Mississippi River. The effects of past discharges to the Mississippi River on surface water, sediments, or biota have not been determined. Low levels of PFCs may also be discharged to the river by the adjacent Eagles Point wastewater treatment plant.

Soil data for PFCs were not available for the site. Because of their physical properties, PFCs may move easily with infiltrating water through some soil types, resulting in groundwater contamination. The limited number of studies regarding PFC migration suggest that PFCs are capable of entering groundwater from source areas (such as fire-training sites) and moving long distances. Analysis of water samples for PFOS and PFOA from four private wells located just to the east of the facility did not show the presence of either chemical. However, the wells are completed a significant distance below ground, and are in a side-gradient direction in terms of groundwater flow. The absence of PFCs in these wells does not rule out the possibility of PFC contamination in groundwater on or off of the site as a result of aerial deposition of PFCs and subsequent infiltration into groundwater. This transport mechanism is thought to have occurred (and is being investigated) at other PFC facilities in the US.

Workers at the site have been exposed to PFCs through their work activities and through the facility's water supply. 3M has monitored workers at the facility for the presence of PFCs in their blood since the 1970s. Studies of PFC concentrations in blood serum have shown concentrations of PFOA of up to 115 parts per million (ppm). Epidemiological studies of workers at Cottage Grove have shown little apparent impact of PFC exposure on worker mortality. Epidemiological data for these chemicals is lacking for the general population.

Studies of PFCs in blood samples from the general population have shown that PFCs are ubiquitous in human blood, at concentrations much lower than seen in PFC production workers, and are not age-dependent. The estimated half-lives of PFOS and PFOA in humans is on the order of several years. The source of exposure to PFCs in the general population is unclear, but is likely through a number of pathways including food, water, use of consumer products, or other environmental pathways. PFCs have also been found in the blood and tissues of various species of wildlife from around the world. The highest concentrations have been observed in bald eagles and mink in the Midwestern U.S. PFOS has been shown to bioconcentrate in fish.

Toxicological research on PFCs is ongoing. Animal exposure to PFCs at high concentrations can have adverse effects on the liver and other organs, and has caused the death of test animals (cynomolgus monkeys) for reasons that are not entirely clear. Exposure to high concentrations of PFOA over long durations has been shown to cause cancer in some test animals, although again the mechanisms are not clear. Developmental effects have also been observed in the offspring of pregnant rats exposed to PFCs.

The potential impacts on public health from perfluorochemical releases from the 3M - Cottage Grove facility cannot be fully assessed by MDH at this time, because there are not sufficient environmental data available regarding PFC impacts from the facility in soil, groundwater, surface water, sediments, and biota. For this reason, MDH has recommended that additional investigation take place. Understanding the contribution of individual sources of PFCs to the environment is important, given the lack of information available about how the general population is exposed to PFCs, the long half-life of PFCs in humans, and their potential for toxicity based on animal studies. MDH will continue to work with the MPCA and 3M to investigate and assess PFC releases from the 3M - Cottage Grove facility.

1. Purpose

The manufacture and disposal of PFCs at the site has resulted in documented contamination of groundwater at the site. Potential contamination of soil, and of surface water and sediments in the adjacent Mississippi River remains to be investigated. Wastewaters containing PFCs were discharged to the river. PFCs have been detected in multiple on-site monitoring and production wells, and in the water supply system serving the facility. The Minnesota Pollution Control Agency (MPCA) Superfund Program has been overseeing site investigation and cleanup activities; because of other contamination issues the site was originally added to the Permanent List of Priorities, the state Superfund List, in 1985. 3M has been conducting various investigations and response actions under a consent order with the MPCA since that time. The MPCA staff requested that MDH review site documents prepared to date, the results of environmental monitoring conducted at the site, and information available on the toxicity of PFCs and their behavior in the environment in order to develop conclusions and recommendations regarding potential public health impacts from the site.

II. Background and History

The 3M Company (formerly Minnesota Mining and Manufacturing Company) operates a facility on approximately 865 acres in the city of Cottage Grove, Minnesota. The facility has been in operation since 1947. The southeastern portion of the property has been used for a variety of industrial operations such as the manufacture of adhesive products, industrial polymers, and reflective road sign materials, and for research and development of similar products (Barr 1991). The facility also includes a permitted hazardous waste incinerator used to treat wastes generated at this and other 3M facilities. The remainder of the property has been used for recreation and farming, or simply left in a natural state. The site has been known variously as the 3M Cottage Grove Center and the 3M Chemolite Center. The location of the site is shown in Figure 1, and the site layout is shown on a recent aerial photo in Figure 2. Note that 3M uses a numbering system for the various buildings at the facility; the building numbers for the areas discussed in this report are shown in Figure 2.

Perfluorochemicals (PFCs), primarily perfluorooctanoic acid (PFOA) and one of its salts, ammonium perfluorooctanoate (APFO), as well as lesser amounts of related PFC products derived from perfluorooctanesulfonyl fluoride (POSF) have been manufactured at the site since approximately 1947 through an electrochemical fluorination process known as the Simons process (Abe and Nagase, 1982; Gilliland and Mandel 1993; Olsen et al 1998; 3M 1999a; Alexander 2001; OECD 2002). 3M voluntarily ceased production of PFCs at the site in 2002 (ERG 2004; 3M 2000a). Perfluorochemicals are a class of organic chemicals in which fluorine atoms completely replace the hydrogen atoms that are typically attached to organic hydrocarbon molecules (3M 2001a). Because of the very high strength of the carbon-fluorine bond, PFCs are inherently stable, nonreactive, and resistant to degradation (3M 1999a). The PFCs manufactured by 3M at the site were used in a variety of commercial and industrial products by 3M and other companies, including stain repellents (such as ScotchgardTM), surfactants, fire retardants and fire-fighting foams, and other chemical products.

The POSF production process through electrochemical fluorination yields about 35%-40% POSF, along with a mixture of byproducts and waste products of variable composition (3M 1999a; 3M 2000b). PFOA and its salts are typically produced in a similar fashion through a batch process (3M 2000c; EPA 2002). Volatile wastes and byproducts were vented to the atmosphere, and some byproducts were re-used in the manufacturing process. Waste tars from the PFC production process were at times disposed in an on-site pit, or later incinerated. Wastewaters containing PFCs from operations at the site have been discharged to the Mississippi River. Although wastewater from the site is routed through an on-site wastewater treatment plant prior to discharge to the river, many PFCs are resistant to treatment because of their chemical stability. One of the byproducts of the production of POSF is perfluorooctane sulfonate (PFOS), which is usually resistant to further degradation in the environment. It can also be produced by the subsequent chemical or enzymatic hydrolysis of POSF. 3M estimated that during POSF production at their Decatur, Alabama production plant, approximately 90% of the wastes generated were in the form of solid wastes (incinerated or disposed in landfills), 9% of the wastes were discharged as wastewater, and 1% in the form of air emissions (3M 2003b).

Geology/Hydrogeology

The 3M Cottage Grove facility is underlain by fill materials and unconsolidated glacial deposits of sand, gravelly sand, and gravel terrace deposits associated with the adjacent Mississippi River. The thickness of these deposits ranges from approximately 20 feet in the northwest to more than 200 feet at the southern end of the site and in the stream-cut ravines along the eastern and western borders of the property. In the ravines, the upper bedrock formations have been partially or completely removed by erosion.

Beneath the glacial deposits, the first bedrock formation is the Prairie du Chien Formation, composed of dolomite (a magnesium-rich form of limestone). The upper portion of the Prairie du Chien has abundant solution cavities, but the lower portion tends to be more massive. Beneath the site, the bedrock has been uplifted on a series of faults and only the lower, more massive portion of the Prairie du Chien is present. The Prairie du Chien Formation overlies the Jordan Sandstone. Groundwater generally does not flow readily from the more massive, basal Prairie du Chien into the Jordan Sandstone, except where there are fractures or solution cavities. Beneath the Jordan Sandstone, the shaley St. Lawrence formation acts as a “confining layer” that inhibits the downward migration of groundwater to the underlying Franconia Sandstone. The cross-section in Figure 3 illustrates the geology underlying the site.

Two deeply incised glacial river valleys run from north to south along the eastern and western edges of the site. Intermittent streams run through the valleys. Erosion along these stream channels has created steep ravines on the southeast and southwest sides of the facility. A recent study by Mossler (2003) indicates the presence of a series of faults oriented northeast-southwest in this portion of Washington County, with associated minor faults oriented northwest-southeast. A pair of intersecting faults is reportedly present beneath the site (see Figure 4). Analysis of these fault systems by Barr Engineering (Barr, 2003) suggests there may be up to 50 feet of vertical off-set on the faults on the 3M property (see Figure 5). The northwest-southeast trending fault on the site appears to control the location of the stream valley in the southeastern portion of the site, where the ravine turns abruptly southeast before discharging to the Mississippi River.

The surface of the ground water, or water table, ranges from 60 to 100 feet below the ground surface and generally follows the surface topography. The water table is found in the glacial deposits near the river, in the Jordan Sandstone near the river bluffs, and in the Prairie du Chien Formation further from the river. The groundwater in the various formations is interconnected and is essentially one unit. The normal groundwater flow direction (i.e. when not influenced by pumping wells on-site) is towards the Mississippi River. Groundwater modeling by Barr (2003) suggests the faults and fractures in this area may have some influence on the pathways groundwater follows as it migrates toward the river. The model did not specifically evaluate faults on the 3M property, however it would be expected that once groundwater enters the faults, it likely flows parallel to the fault trace.

However, groundwater flow beneath the site is heavily influenced by the facility’s high-capacity production wells. Some of these wells reportedly have been in operation since the 1940s. In 2002, 3M reported pumping just over one billion gallons of water from the production wells on

the property (DNR 2003). In previous years, an even larger amount of water was pumped from the aquifers beneath the site.

In fact, most of the groundwater from beneath the majority of the 865-acre site, and especially from the developed portion of the site, appears to be captured by the action of the production wells as shown in Figure 6 (ERG 2004). This figure is from a past groundwater model for the site developed for 3M. The source of the model, and the data and assumptions upon which it was created, were not available at the time this document was written. For example, it is not known whether the model incorporated pumping effects from the numerous nearby residential wells or included geologic features such as the intersecting faults recently identified in this area. These factors may affect the pathway of groundwater flow, but it is likely that most ground water that migrates beneath the site is captured by the facility production wells and only a small portion may discharge to the Mississippi River. One exception appears to be the southeastern-most portion of the site, where Sites D1 and D2 are located (see below). This area does not appear to be within the capture zone of the production wells and the groundwater beneath this area likely discharges to the Mississippi River.

As shown on Figure 7, there are approximately 100 private water supply wells located within one mile of the 3M property boundary. Most private and public wells in the area for which there is geologic information available are completed in the Jordan Sandstone. Because groundwater flows primarily to the south-southeast toward the Mississippi River, it appears that no private or public water supply wells on the north side of the river are located in areas downgradient of contaminant source areas.

Superfund Site History

The investigation and remediation activities conducted at the site under the MPCA Superfund program have generally centered around ten waste disposal areas originally identified by the MPCA and 3M (MPCA 1998). These activities, which were conducted under a consent order signed between the MPCA and 3M in 1985, were not focused on PFCs. The MPCA and 3M are currently negotiating an addendum to the existing consent order that will focus on the investigation of PFCs in all media at the site (David Douglas, MPCA, personal communication, 2004). The existence of the waste disposal areas (not all of which were related to PFC manufacture) was a primary reason the site was added to the state Superfund list. The locations of the waste disposal areas are shown in Figure 8. They are as follows:

Site D1: Hydrofluoric Acid Neutralization Pit

This site was used to neutralize hydrofluoric acid tars (containing unspecified fluorochemical by-products and hydrofluoric acid) with lime. Neutralization was thought to have been done in a concrete pit or vault, but this has not been confirmed. The tar materials in the pit were never directly sampled and analyzed for PFCs; instead hydrofluoric acid tars from the PFC production process were analyzed to determine if they were a hazardous waste as defined under federal and state regulations. Trace concentrations of metals were identified in the tars, but the neutralized tar material itself was considered to be non-hazardous. Although PFCs were detected in the

groundwater in this area, site D1 has not been fully characterized as to the magnitude or extent of PFC contamination.

Site D2: Sludge Disposal Site

This area was used for the disposal of sediments and sludge dredged from on-site wastewater treatment ponds, and may be up to four acres in size. Laboratory analysis of samples of the sludge material found elevated concentrations of numerous fluorinated compounds (including likely by-products of the PFC production process). Samples of the sludge and soil beneath the sludge showed lower levels of several volatile organic compounds (VOCs). This area has not been characterized with respect to PFCs.

Site D3: Ash Disposal Area

The location of this site was investigated using ground-penetrating radar, and no evidence of waste materials was found.

Site D4: Phenolic Waste Pit

This site was used for the disposal of a small process wastewater stream from Building 7 for a period of three years. The wastewater stream contained phenol and possibly formaldehyde. Part of Building 26 was built on top of this site, limiting infiltration of water through the former pit. While it was never formally investigated, it was believed that some biodegradation of the wastes would have occurred, and the construction of the building over the site would serve as an effective cap preventing human contact or migration of any remaining contaminants.

Site D5: Solids Burn Pit Area

This area is a concrete pit approximately 10 feet deep and 350 feet in diameter. 3M used the pit to burn off-spec products such as glass, tape, rubber, adhesives, rags, paper, wood, fiberglass, oily sludge material, plastics, and resins. The burning was occasionally fueled with waste solvents. The area has since been covered with several feet of fill. Soil borings drilled in this area found the presence of wastes, sludge, ash and cinders. Low levels of VOCs, such as toluene, ethylbenzene, trichloroethylene, and methylene chloride were detected in several soil samples collected from the borings; the area was subsequently given regulatory closure by the MPCA.

Site D6: Active Ash Disposal Area

This area is an inactive, MPCA-permitted waste disposal area for boiler ash and incinerator residues. Due to its permitted status, it has not been investigated under the Superfund program.

Site D7: Pit Burning Area

The history of this area is unclear. Three borings advanced in this area did not encounter waste materials, and soil sample analysis did not detect the presence of heavy metals or VOCs.

Site D8: Waste Disposal Area

This area is located along the bluff leading down to the Mississippi River. A variety of construction debris and other waste materials, including numerous drums, were disposed here by

dumping them over the edge of the bluff. An Interim Remedial Action (IRM) at Site D8 involved the removal of approximately 200 drums and drum carcasses (rotted drums). The drums were disposed in the on-site 3M incinerator. A composite sample of the soil beneath the drums showed no VOCs, but polychlorinated biphenyls (PCBs) were found at elevated levels. A composite sample of the waste materials in the drums contained various VOCs and PCBs. It is not known if samples were analyzed for fluorochemicals. Due to the extreme topography of Site D8, not all of the wastes were removed, and the site was covered and replanted.

Boiler Ash Fill Area:

This area is located on the western edge of the facility, in an area that is used for training on-site fire fighting personnel and for testing fire-fighting products (see Figure 8). Boiler ash from a coal-fired boiler, used to produce steam for heat and various industrial processes, was used for fill in this area (Barr 1991). Soil borings drilled in this area showed the boiler ash fill to be approximately one foot in thickness, and the volume was estimated at 850 cubic yards. Some of the boiler ash was exposed at the ground surface. Laboratory analysis of a sample of surface water that had pooled in the area showed elevated levels of metals, including antimony, arsenic, nickel, and vanadium. The ash was determined to be non-hazardous, and the area was covered with clean fill and vegetated.

Acrylic Acid Release Area:

In October of 1973, 3M discovered that approximately 17,000 gallons of acrylic acid had been released from an underground storage tank (UST) located adjacent to Building 7 (Barr 1991). The UST that was the source of the release was abandoned in 1986. This area was investigated by 3M, and no further action was required by the MPCA because the acrylic acid was thought to have degraded naturally.

Areas of PFC Production and Use

As stated previously, PFC production began (on a pilot scale) at the site around 1947; full-scale commercial PFOA production reportedly began in 1976. POSF-derived chemical production began in the 1960s. The main area for PFC production, storage, and testing was centered around Buildings 7, 15, 16, and 25, which are shown in Figure 2 (ERG 2004). The production of PFCs was phased out at the end of 2002. Wastes from the PFC production process were disposed in Site D1 and possibly Site D8. Wastewaters containing PFCs were routed through the on-site wastewater treatment plant before discharge to the river. Sludge from the wastewater treatment plant was disposed at one time in Site D2. PFC containing fire fighting chemicals were also tested on the west side of the facility in the area of Building 43.

In 2001, the chemical sewer lines running from various chemical production areas of the site to the wastewater treatment plant were upgraded and replaced (ERG 2003). Excavation of the old sewer pipes at the northeast corner of Building 15 (the PFC production plant) revealed that the pipes were corroded and had leaked. The soils in the base and sidewalls of the excavation had a strong phenolic odor. A composite sample of the sidewall soil showed low levels of metals, VOCs (trichloroethene (TCE) and 1,1-dichloroethane) and phenolic compounds. 3M also removed a portion of the interior floor from the northeast corner of Building 15 in response to

concerns over possible damage to the building foundation from releases of hydrofluoric acid. Soil samples were collected from borings placed around the interior trench. Analysis of the soil samples showed very low levels of metals, and one semi-volatile compound (butyl benzyl phthalate). The activities related to the sewer replacement in and around Building 15 indicate that releases to the soil (and possibly groundwater) of chemicals used in the PFC production process did occur while the PFC production plant was in operation. No analyses for PFCs themselves were conducted, however, so it is not clear if PFCs are present in soil and groundwater at Building 15.

In 1991, an air dispersion model was developed for VOC and inorganic emissions at the 3M - Cottage Grove facility (Pace 1991). The emission points modeled included two 48-foot stacks at Building 15, the PFC production plant, where hydrogen fluoride emissions occurred. The emission rate used in the model was 0.38 pounds of hydrogen fluoride per hour of operation, with a stack exit velocity of 1,440 feet per minute at ambient (70° F) temperature. The horizontal extent and the estimated concentrations of hydrogen fluoride (both the 1986 annual average and the second highest 24-hour average in micrograms per cubic meter) predicted by the model are shown in Figures 9a and 9b. The results of the air dispersion model indicate that hydrogen fluoride emissions extended off-site in 1986.

The PFC production process would also have resulted in the release of some PFCs to the atmosphere, as mentioned previously. 3M estimated that 1,950 pounds of PFOA compounds were released to the air from vent stacks at the Cottage Grove facility in 1997, and that the releases occurred between 100 to 200 days per year (3M 2000c). Presumably, at least some of the PFOA compound releases to the air were from Building 15, or nearby buildings where PFCs were produced, handled, or used. Fugitive emissions of PFOA (both vapor and particulate) were also likely from the various operations, such as drum loading, reactor sampling, and drying operations (3M 2000c). The physical properties of PFOA and other PFCs are different from hydrogen fluoride, and their behavior once released to the air are likely to differ as a result. However, the air dispersion model results for hydrogen fluoride emissions shown in Figures 9a and 9b suggest that PFOA emissions (both particulate and vapor phase) from the Building 15 area may also have extended off the site property. Deposition of PFOA to the soil from these emissions may also have occurred.

On-Site Groundwater Monitoring and Use

Since investigation activities at the site began, at least 21 permanent monitoring wells have been installed at and around the site to evaluate groundwater quality (Figure 10). The monitoring well identifiers, unique well numbers, depth, and general locations are as follows:

Well ID	Unique Well Number	Depth (feet)	Monitoring Well General Location
MW-1	233567	200	Northern site boundary
MW-2	233568	192	East side of site
MW-3	233569	210	Center of site

MW-4	233570	200	South-central area of site
MW-5	233571	210	Northwest corner of site
MW-6	233572	219	West-central area of site
MW-7	233573	146	Northeast corner of site
MW-8	233574	173	Southwest area of site
MW-9	233575	104	West-central area of site
MW-10	233554	237	Southeast area of site
MW-11	233950	200	Southeast area of site
MW-12	233951	126	Southern edge of site
MW-13	233952	126	Southeast area of site, near ponds
MW-14	421705	60	D8 Area, southern edge of site, near PW-6
MW-15	431237	186	Southern edge of site
MW-16	431238	140	Southern edge of site
MW-17	570322	112	West-central area of site
MW-18	570323	91	West-central area of site
MW-19	612713	62	West-central area of site
MW-101	680685	100	D1 Area, southeast corner of site
MW-102	680686	96	D1 Area, southeast corner of site

Since the facility opened in 1947, 3M has installed eight production wells to serve the facility's potable water supply and to provide water for various industrial operations (Figure 10). They are as follows:

Well ID	Unique Well Number	Date Completed	Depth (feet)	Casing Diameter (inches)
PW-1	231867	1947	205	20
PW-2	231868	1948	202	20
PW-3	231869	1956	224	16
PW-4	231870	1958	275	16
PW-5	231871	c. 1960s	113	24
PW-6	229117	1970	143	24
PW-7	233576	c. 1980s	200	Unk.
PW-8	424131	1986	208	8

Four of the eight production wells (PW-2 to PW-5) serve the potable water distribution system, while two wells are used on a periodic basis for fire suppression (PW-1) and to supply non-contact cooling water to the 3M waste incinerator (PW-6; ERG 2004). Of the remaining two wells, PW-7 is used occasionally at the 3M on-site trap range and PW-8 supplies the guard shack.

In the past, low levels of VOCs including TCE, 1,1,2-trichloroethane, 1,1-dichloroethane, ethylbenzene, toluene, and methylene chloride have been detected in various monitoring and production wells at the southern end of the facility, specifically MW-4, MW-14, PW-5, and PW-

6, which are located in the vicinity of D8. Levels of these VOCs have only occasionally exceeded health-based drinking water criteria in the individual monitoring wells, and the distribution of the contaminants suggests that the sources of the VOC contamination are localized and not extensive (ERG 2001a, ERG 2001b). The concentrations of individual VOCs in PW-5, one of the wells that are used for the drinking water supply, have recently been less than approximately one microgram per liter ($\mu\text{g/L}$). Such concentrations do not exceed applicable regulatory or health-based standards for a water supply system. The system is regulated and monitored by MDH as a public water supply. Exposure to VOCs in groundwater at the site does not appear to be a human health concern at this time.

PFC Monitoring at the Site

3M has been monitoring groundwater, production wells, the water distribution system, and wastewater treatment plant effluent for PFCs (primarily PFOS and PFOA) for a number of years. Data from monitoring wells, production wells, and the water distribution system are shown in Table 1, while effluent data from the wastewater treatment plant are presented in Table 2. The majority of the data is for PFOA alone, because it has been the focus of investigation activities at the site being conducted by 3M under a voluntary agreement with EPA (3M 2001b). Some samples were analyzed for PFOS, PFOA, and the 4-, 6-, and 7-carbon perfluorosulfonates and other acids. The 4-, 5-, and 6-carbon PFCs were likely found in the groundwater because they are present in the PFC wastes that were disposed in several areas of the site. Much of the data were collected only in 2001, so information on long-term trends in the PFC concentrations in groundwater is not yet available.

The well monitoring results indicate that PFOS and PFOA are present in groundwater at the site in the D1 area (MW-101 and MW-102) and the D8 area (MW-14, PW-5 and PW-6). Levels of PFOS and/or PFOA exceed the MDH Health Based Values (HBVs) for PFOS and PFOA in these wells, sometimes by a factor of 100 or more. The HBVs represent a level of a contaminant in drinking water that MDH considers to be safe for human consumption over a lifetime. The HBVs were developed by MDH based on review of available toxicological information as of November 2002; neither the values themselves nor the toxicological inputs were derived by the EPA. The HBV for PFOS is $1 \mu\text{g/L}$; the HBV for PFOA is $7 \mu\text{g/L}$. The derivation of the MDH HBVs for these two compounds and their toxicological basis can be found in Appendix 1. MDH has not developed HBVs for the other perfluorosulfonates and acids, mainly because of a lack of available toxicological information.

Detectable levels of PFCs (in some cases slightly above the HBVs) were also found in: MW-4 at the southern end of the facility on top of the bluff; PW-2 at the northern end of the facility; PW-4 northwest of the main facility; and in the water distribution system itself. The sample from the distribution system was collected from the cafeteria in Building 116. A very low level of PFOA (less than one $\mu\text{g/L}$) was also found in MW-7 northeast (and upgradient hydrogeologically) from the main facility. Note that PFC data are not available for all of the monitoring and production wells at the site. 3M has proposed to collect a coordinated round of groundwater sampling from all of the available monitoring and production wells at the site. This would be very helpful in characterizing PFC contamination in groundwater across the facility. The available data

indicate, however, that groundwater in several areas of the site has been affected by past PFC production or disposal practices. This in turn is affecting the water wells serving the facility.

A groundwater model developed by 3M and found in the site files suggests that much of the contaminated groundwater is likely captured by the pumping action of the production wells at the site, as shown in Figure 6, with the exception of the D1 and D2 areas, located just southeast of the area shown in the figure. The PFCs detected in the water distribution system lends support to this conclusion, although it is not possible to evaluate the validity of this model because the underlying data and assumptions used in its construction were not available at the time this document was written. Contaminated groundwater in the D1 Area most likely discharges to the Mississippi River, either directly or possibly via the intermittent stream in the ravine immediately north of the D1 Area.

The facility's water distribution system is used for potable water, and for various industrial processes. Bottled water has been provided to employees for drinking water for some time, however, and a GAC treatment system has been installed in the main cafeteria in building 116 to treat water used in food preparation and cleanup. Wastewater from these uses is collected in various sewer systems (see below) for treatment in the on-site wastewater treatment plant. The wastewater therefore contains PFCs from the groundwater contamination, and any PFCs picked up during the use of the water for production or other purposes throughout the plant.

Under its federal National Pollutant Discharge Elimination System (NPDES) permit (MN000149) the effluent from the wastewater treatment plant is monitored before discharge to the Mississippi River. Since 2000, 3M has regularly collected 24-hour composite samples of the treated effluent for analysis for PFOA. Limited data is available back to 1996 (see Table 2). Levels of PFOA have generally declined since 1996, with an overall high of 1,991µg/L detected in early 2000. With the phase out of PFOA production in late 2002, effluent concentrations of PFOA and PFOS should continue to drop. 3M also installed a large granular activated carbon (GAC) treatment plant at the site to remove organic contaminants (including PFCs) from the wastewater treatment plant effluent before discharge to the Mississippi River. It should also be noted that the effluent from a regional wastewater treatment plant (the Eagles Point plant, operated by the Metropolitan Council) located essentially within the grounds of the 3M - Cottage Grove facility (see Figure 2) may also contain low levels of PFCs as has been found in limited studies at other wastewater treatment plants in the U.S. (see page 23).

For most chemicals, aerial deposition of a contaminant is not typically a pathway for groundwater contamination. However, given the physical properties and environmental behavior of PFOA and other PFCs it is possible. Air emissions of PFOA and/or other PFCs at production facilities in West Virginia and Alabama are suspected to have contributed to PFOA/PFC contamination of soil and groundwater from those facilities, in addition to other releases (West Virginia Dept. of Environment Protection 2003; Daikin 2004).

Because of the potential for past air emissions (and deposition) of PFOA to have extended off site, in December 2003 MDH staff collected water samples from four private residential wells

located just east of the 3M - Cottage Grove Facility for analysis for PFOA and PFOS by the MDH laboratory. The locations of the four residential wells are shown in Figure 11, along with the approximate extent of air emissions of hydrofluoric acid predicted by the 1991 air model. All four wells are relatively deep (approximately 220 feet below grade). The results of the PFOA/PFOS analysis showed no detections of PFOA or PFOS above the laboratory detection limits of 1.0 µg/L and 0.5 µg/L, respectively, in any of the four wells. However, the absence of detectable PFOA or PFOS in the four deep wells sampled does not resolve the question of whether surface deposition and subsequent infiltration has occurred. The MDH laboratory does not have the ability to analyze for the 4-, 5-, or 6- carbon PFCs at this time.

Site Visit

On October 14, 2003 MDH staff visited the 3M - Cottage Grove facility, along with representatives of the MPCA Superfund program. MPCA staff arranged the site visit for the purpose of becoming acquainted with the facility layout and areas of the facility where perfluorochemicals (PFCs) were manufactured and used, and where PFC wastes were disposed. 3M facility and corporate staff conducted the site visit, along with their lead environmental consultant for the facility (ERG).

The 865-acre site is located just south of the intersection of US Highway 61 and Washington County Road 19 in Cottage Grove, Minnesota, on the Mississippi River. Because the site is a chemical plant, it is a secure facility with a full perimeter fence and controlled entry. The facility is used for chemical manufacture, testing, product development, and for the incineration of hazardous chemical wastes. To the east of the facility are a golf course and residential development (River Oaks). To the south are the Burlington Northern Santa Fe Railroad main line and the Mississippi River. To the west are a regional wastewater treatment plant (the Eagles Point plant, operated by the Metropolitan Council), agricultural and rural residential land. To the north are US 61, scattered homes, and a regional park.

The site visit focused on the following areas because of their association with PFC manufacture, use, treatment, or disposal at the site: the fire training area, production wells PW-5 and PW-6, the PFC production area, the D1 land disposal area, the wastewater treatment plant outfall at the Mississippi River, and the wastewater treatment plant area.

Fire Training Area:

The fire training area is located below the main facility, near Building 43 (Figure 2). Facility employees are trained here in fire fighting through various mock situations, such as a chemical spill, a fire in a laboratory vent hood, or a leaking pipeline. Part of this area is underlain by a gravel-covered concrete pad with drains leading to a lined holding pond. The area appears to have been upgraded relatively recently. 3M staff indicated that the area was used for facility staff training purposes and to test fire suppressants containing PFCs (ERG 2004).

Production Wells PW-5 and PW-6:

These two production wells are located on the southern edge of the facility, close to the Mississippi River (see Figure 10). PW-5 feeds into the facility water distribution system, while

PW-6 is only used for non-potable cooling water for the incinerator. Both PW-5 and PW-6 have detectable levels of PFCs, and a monitoring well located adjacent to PW-6 (MW-14) has elevated levels of PFCs. A disposal site (D8) was located on the hillside just above PW-6 and MW-14. Apparently, construction debris and drums of waste materials were removed from this location during the mid-1980s. The wastes had reportedly been dumped over the edge of the hillside and buried sometime in the past. The main wastes identified at D8 were volatile organic compounds (VOCs); it is not known if PFC wastes were present as well. 3M has agreed to complete additional PFC monitoring in the area of PW-5 and PW-6.

PFC Production Area:

PFCs were produced in Building 15 for many years; the plant has now been shut down and is to be decommissioned. PFCs were used in the production of other compounds in Buildings 7, 16, and 25. These buildings are shown in Figure 2. Wastes from these processes were discharged to buried sewer lines that ran to the on-site wastewater treatment plant. These buried sewer lines have since been replaced with an upgraded system that is contained within a concrete trench open to the ground surface. There are numerous stacks and vents in the PFC production and use areas, and 3M staff confirmed that there were air emissions of chemicals (permitted by the MPCA) from these stacks.

D1 Area:

This area was used in the past for the disposal of PFC production wastes. It is located on the top of a narrow peninsula of land that extends southeast from the rest of the facility (Figure 8). From the top of the peninsula, the land drops off sharply to the south towards the railroad and Mississippi River. To the north the land drops towards a ravine, through which the wastewater treatment plant outfall stream runs. Another disposal site, D2, is located just west of D1. Two monitoring wells (MW-101 and MW-102) flank the D1 disposal site. These wells have shown the highest levels of PFOS so far detected at the site, and are located slightly downhill from the D1 area. According to 3M's consultant, no seeps or springs have been observed around the base of the peninsula or in the stream.

Wastewater Treatment Plant Outfall:

The output of the wastewater treatment plant is piped to an intermittent stream that runs through a ravine along the eastern edge of the facility. The output enters the stream through a pipe after exiting the smallest (and last) treatment pond just west of the D2 and D1 land disposal areas. There is a permanent effluent monitoring point there as well. Stormwater is also discharged at this point when necessary. The stream enters a small pond just north of the railroad tracks, passes under the railroad track bridge, and enters the Mississippi River, as shown in Figure 2. The stream is very clear, and vegetation and small fish could be readily observed in it.

Wastewater Treatment Plant:

The current wastewater treatment plant consists of various settling basins, biologic treatment vessels, and filters to handle four of the five waste streams at the facility (sanitary, organic wastes, inorganic wastes, and the incinerator process wastewater). Stormwater is not usually routed through the treatment plant, but can be in the event of a spill or accidental release. At the

end of the treatment process the wastewater is piped into a series of holding ponds before discharge to the river, as described above.

3M has constructed a large granular activated carbon (GAC) treatment plant to augment its wastewater treatment operations and remove PFCs from the wastewater treatment plant effluent. The efficiency of the GAC for removing the PFCs from the waste stream has so far been in excess of 99%. The GAC treatment plant consists of 18 large GAC treatment vessels that are the final treatment step for the combined waste streams from the sanitary sewer, organic wastes, and inorganic wastes. A subset of the treatment vessels will be used specifically for treating the incinerator wastewater stream. The existing treatment ponds are to be abandoned and filled, with the exception of the largest one, which has been refurbished with a synthetic liner and will be used as a backup storage pond when needed.

Off-site Water Use and Sampling

As noted in the “Geology/Hydrology” section, there are approximately 100 private and commercial wells located within one mile of the 3M property boundary. In addition, the well field for the City of Cottage Grove is located approximately 1.5 miles northwest of the 3M property, and the well field for the City of Hastings is located approximately the same distance to the southeast, across the Mississippi River. Water samples from four residential wells located immediately east of the site were analyzed for PFOS and PFOA by the MDH lab (see Figure 11). Neither compound was detected in the sampled wells.

Public Comments

On June 24, 2004 a draft version of this document was released for public comment. Comments were received from EPA, MPCA, Washington County, the City of Cottage Grove, and 3M. The comments are attached as Appendix 2.

EPA provided several general comments, as well as suggestions as to specific language to describe EPA’s ongoing work with the perfluorochemical industry to investigate the sources, fate, and transport of PFCs in the environment. EPA did not review the document for toxicological accuracy, in part because a draft risk assessment for PFOA is not due until late 2004. The specific comments and suggestions made by EPA were incorporated into the document. The MPCA comments were mostly general in nature; MDH staff addressed them by clarifying the text in several places described in the comments, including the description of past investigations at the site and the status of the consent order between the MPCA and 3M.

Washington County’s comments generally were in the form of recommendations to 3M for further investigation or disclosure of information relative to releases of PFCs and VOCs at the site. As the county’s comments appeared to reinforce MDHs own recommendations and statements made in the text of the document, no further changes were made to the document itself as a result. The City of Cottage Grove comment letter simply expressed support for the recommendations made in the draft Health Consultation, including the need for continued monitoring.

3M submitted extensive comments on each section of the document. General comments from 3M on the conclusions and recommendations did not result in significant changes by MDH. Specific comments regarding the text of the document were helpful in that they clarified certain historical facts regarding PFC production and disposal at the site; changes were made to reflect these comments. 3M provided missing information relative to certain monitoring and production wells at the site. 3M also provided several useful toxicological references that were not available at the time the document was first written, and these references have been included. Specific comments on toxicological issues were addressed with the major exception of the comments on the potential for developmental effects from exposure to PFOS (see 3M comments, page 12 in Appendix 2). This comment was apparently in response to MDH's statement on page 20 of this document that MDH may consider developmental effects when reviewing the current HBV for PFOS. Because MDH is not currently reviewing the PFOS HBV, this comment was not addressed.

III. Discussion

Perfluorochemicals (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; $C_8F_{17}SO_2F$) have been manufactured or used at the site since 1947. 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. These chemicals are used by 3M and other companies around the world in the production of stain repellents, lubricants, fire retardants and suppressants, and pesticides, and as industrial surfactants and emulsifiers.

The chemical structures of PFOA and PFOS make them extremely resistant to breakdown. As a result, they are persistent once released to the environment. On the basis of its physical properties, PFOS is essentially non-volatile, and would not be expected to evaporate from water (OECD 2002). If discharged to air (such as during production of POSF) it will rapidly deposit to soil and due to its low sorption tendency, once in soil it tends to remain there with the major loss due to run-off to surface water (DMER 1999). Infiltration of water could also carry it into the subsurface or into groundwater, however. In soil-water mixtures, PFOS has a strong tendency to remain in water due to its solubility (typically 80% in water and 20% in soil). PFOS does not easily adsorb to sediments, and is expected to be mobile in water at equilibrium (3M 2003b).

PFOA is slightly more volatile than PFOS, although it still has a very low volatility and vapor pressure (EPA 2002). PFOA is very soluble and completely disassociates in water; in aqueous solution it may loosely collect at the air/water interface and partition between them (3M 2003a).

In limited studies, PFOA has shown a high mobility in some soil types (EPA 2002). In an attempt to estimate the potential for long-range transport of PFOA released to the air, Franklin (2002; unpublished report on EPA's PFOA web site) stated that PFOA emitted to the air is likely to undergo dry or wet deposition within a few days, but could under certain conditions travel a distance of up to 800 kilometers from the source.

In a study of PFCs in groundwater at a former military fire-training site in Michigan, Moody et al (2003) found PFOS concentrations up to 120 µg/L and PFOA as high as 105 µg/L near the original concrete pad used for the training. Concentrations of PFOS and PFOA in excess of the MDH HBVs were found in groundwater as far away as 500 meters from the pad. The facility was used for fire-training from 1952 until the early 1990s, and fire fighting foams containing PFCs were routinely used in training exercises. The results of the study indicate that PFCs in aqueous solution are easily capable of migrating into groundwater. They can travel extended distances with little or no retardation of the contaminants through adsorption to the aquifer substrate, and can persist for years after they were used at the ground surface. The 3M site contains a similar fire-training area where fire fighting foams containing PFCs were reported to have been used (ERG 2004). While the site studied by Moody et. al. has some similarities to the 3M fire-training site, actual site characteristics will determine the potential for PFCs to enter the groundwater and migrate away from the site. This has not yet been evaluated at the 3M fire-training site.

Because of the recent widespread interest in PFC compounds such as PFOS and PFOA, a great deal of toxicological, epidemiological, and environmental monitoring information has been published in government and industry reports and in peer-reviewed literature. Much of this research has been funded or conducted by 3M. Most recently, an analysis of the potential risk to the general population from exposure PFOA was published by Butenhoff et al (2004), and 3M has produced an updated environmental and health assessment of PFOS (3M 2003b). The following represents a brief summary of available information.

Summary of Toxicological Information

Animal studies have shown that 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). PFOS is also well absorbed orally, but is not absorbed well through inhalation or dermal contact (OECD 2002). In the past, workers at the 3M - Cottage Grove facility were occupationally exposed to PFOA, and it is believed that dermal absorption of PFOA was significant (EPA 2002). Once absorbed, APFO disassociates to the PFOA anion. Both PFOA and PFOS are distributed and found mainly in the blood serum, liver and kidney (EPA 2002; Kudo and Kawashima 2003; OECD 2002). PFOA and PFOS are not metabolized, and are excreted in the urine and feces at different rates in various test animal species and humans. There also appear to be significant gender differences in excretion rates for PFOA in rats, but these differences have not generally been observed in higher animals and humans. The estimated half-life of PFOA in animals ranges from four hours in female rats and nine days in male rats to hundreds of hours in dogs (Kudo and Kawahima 2003). 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). In a limited study of retired 3M workers, the mean serum half-life of PFOA was estimated to be 4.37 years, and the mean half-life of PFOS was estimated at 8.67 years (EPA 2002; OECD 2002).

Exposure to high levels of PFOA and PFOS is acutely toxic in test animals (Kudo and

Kawashima 2003; OECD 2002). 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). 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). The effects on the liver may be more severe in aged rats (Badr and Birnbaum, 2004). Exposure to PFOA in rats results in a phenomenon in the liver known as peroxisome proliferation. This phenomenon is limited to rats and similar test animals, and is not observed in primates (or humans). Some of the adverse liver effects observed in rats (such as an increase in liver weight) that are in part attributed to peroxisome proliferation may not be seen in higher animals. Adverse liver effects in higher animals are likely the result of a different mode of action.

A 90-day study of relatively high-dose oral PFOA exposure in rhesus monkeys resulted in adverse effects on the adrenal glands, bone marrow, spleen, lymphatic system, and death in some animals (EPA 2002). A six-month study of oral PFOA exposure in male cynomolgus monkeys exposed to different doses of APFO showed toxicity (primarily to the liver) at even the lowest doses studied. Extreme toxicity was observed at the highest exposure level, prompting a modification of the dosage to prevent the death of the test animals (Butenhoff et al 2002). Even with the dosage adjustment, one test animal at the highest dose became extremely ill and had to be sacrificed. A similar condition developed in one of the lowest dose group animals. The toxicological mechanism for the apparent extreme adverse reaction in these two animals is unknown. A steady-state concentration of PFOA in the serum was reached within four to six weeks after dosing began; mean serum PFOA concentrations ranged from 77 parts per million (ppm) in the low dose group to 158 ppm in the high dose group (Butenhoff et al 2002). This study did demonstrate that the dose-response characteristics of APFO in this species of monkey are very steep – indicating that a small increase in dose can be associated with a significant increase in the number or severity of adverse effects.

Exposure studies of PFOS in rats have also demonstrated effects on the liver, weight loss, and death, with a steep dose-response curve for mortality observed (OECD 2002). In studies of PFOS exposure in rhesus monkeys, adverse effects included anorexia, convulsions, a marked decrease in serum cholesterol, and adrenal effects. Similar effects were observed in studies of cynomolgus monkeys. The adverse effects were no longer observed after a 52-week recovery period, and in fact some recovery was noted much earlier.

Some long-term animal studies suggest that exposure to PFOA (and possibly PFOS) could increase the risk of cancer of the liver, pancreas, and testes (Kudo and Kawashima 2003, EPA 2002, OECD 2002). The mechanism of potential carcinogenesis is unclear, but evidence suggests that the cancers are the result of tumor 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 seen in higher animals or 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 relatively low doses of PFOS and APFO (EPA 2002, OECD 2002). These studies demonstrate that exposure to APFO/PFOA and PFOS can result in adverse effects on the offspring of rats exposed while pregnant.

At the request of the MPCA, in November 2002 MDH developed Health-Based Values (HBVs) for drinking water for PFOS and PFOA of 1 ppb and 7 ppb, respectively, based on existing toxicological information (liver toxicity; see Appendix 1). The HBVs represent a level of a contaminant in drinking water that MDH considers to be safe for human consumption over a lifetime. The HBV documentation in Appendix 1 states that reproductive and developmental effects occur at levels higher than doses associated with liver toxicity. However, recent studies on PFOS (Thibodeaux et al 2003; Lau et al 2003) suggest that developmental effects may also be of concern. These recent studies may lead MDH to examine developmental toxicity as a possible basis for the PFOS HBV, which could result in a different HBV for PFOS. MDH is awaiting further information or guidance from EPA before initiating a review of the HBVs for PFOS and PFOA. Note that MDH is also in the process of revising all HRLs to more directly account for childhood exposures, and this change could result in the lowering of all HBVs by a factor of three or four (see Appendix 1).

Also at the request of the MPCA, MDH staff developed interim Soil Reference Values (SRVs) for both PFOS and PFOA of 40 ppm and 200 ppm, respectively. The SRVs are soil evaluation criteria for protection of people from direct contact with contaminated soil through ingestion, skin contact, and inhalation of vapors and/or contaminated soil particles. Soil concentrations at or below the SRV are considered to be safe.

In summary, human exposure to PFOS and PFOA lead to the buildup of these chemicals in the body. Studies in test animals show that exposure to high concentrations for sufficient time periods may cause adverse effects on the liver and other organs. Developmental effects have also been observed in the offspring of rats exposed to PFOS and APFO. Exposure to PFOA may be associated with an increased risk of certain types of cancer in some test animals.

Summary of Epidemiological Data

The 3M Company has conducted a medical monitoring program of employees engaged in the manufacture of perfluorochemicals since at least the 1970s. The company initially measured total serum organic fluorine. In the mid-1990s, the company began measuring serum PFOA and PFOS when such analyses became available (Olsen et al 1998; Olsen et al 2003a; Olsen et al 2003b). A study of 3M employees at its Decatur, Alabama PFC manufacturing facilities showed a mean serum PFOS concentration of 1.32 parts per million (range 0.06 to 10.06 ppm) and a mean serum PFOA concentration of 1.78 ppm (range 0.04 to 12.70 ppm) in 263 employees. The mean concentrations in employees at 3M's Antwerp, Belgium facility were approximately 50% less (Olsen et al 2003b). There was no association between serum PFOS and PFOA concentrations and decreased serum cholesterol (or other common biological parameters) observed in this group of employees such as has been observed in animal studies. Exposure to PFOS and PFOA has been shown in test animals (including primates) to interfere with cholesterol metabolism and alter (usually lower) serum lipid and cholesterol concentrations.

A separate study of reproductive hormones in male 3M employees occupationally exposed to PFOA at the Cottage Grove facility showed no significant linear association between serum PFOA concentration and the measured hormones, although mean concentrations of one hormone (estradiol) were 10% higher in those employees (five in all) with a serum PFOA concentration above 30 ppm (Olsen et al 1998). This association was confounded by a high body mass index in the five employees, however. Serum PFOA concentrations in this study ranged from 0 to 115 ppm for the Cottage Grove workers. The higher serum PFOA concentrations observed in some workers in this study suggests that occupational exposures to PFOA at the Cottage Grove facility were higher than at the Decatur and Antwerp facilities, and/or that the exposures were of a longer duration. No association between serum estradiol and serum PFOA levels was observed for workers in 3Ms Decatur and Antwerp facilities.

Mortality of employees at the Cottage Grove facility has also been the subject of several epidemiological studies (Gilliland and Mandel 1993; Alexander 2001). In the earlier study, Gilliland and Mandel (1993) reported that the overall standardized mortality ratios (SMR) for 2,788 male and 749 female employees who worked at the facility for at least six months between 1947 and 1983 were 0.77 and 0.75, respectively (a value significantly below the expected rates). The SMR represents the ratio of the observed deaths in a study population over the expected deaths in a study population based on death rates in a non-exposed population of similar characteristics. This phenomenon, where the overall SMR is significantly below the expected rate for a similar, non-exposed population, is sometimes referred to as the “healthy-worker effect” in occupational studies. The study findings did show that male employees who worked in the PFOA production area for greater than 10 years had a 3.3-fold increase in mortality from prostate cancer. However, the low number of prostate cancers (four) in this group makes the findings tentative, and a later study by the same lead author (Olsen et al 1998) reported that only one of the four cases of prostate cancer occurred in a worker directly engaged in PFOA production. A separate study of workers at the 3M Decatur, Alabama facility who were primarily exposed to POSF/PFOS also showed an overall low SMR for all causes of death, but a higher than average risk of death from bladder cancer. This was due to three cases observed, again meaning that the findings may not be repeatable (Alexander et al 2003). There is no current toxicological evidence that suggests that the bladder is a critical target organ of PFOS (3M 2003b).

In a later study at Cottage Grove, Alexander (2001) looked at the mortality of 3,992 workers employed at the facility for at least one year prior to the end of 1997. The cohort was divided into three exposure groups based on their work history: definite PFOA exposure, probable PFOA exposure, and no PFOA exposure. It should be noted that, given the past exposure by workers to PFC contamination in the facility water supply, there may have been some exposure to PFOA even in the “no PFOA exposure” group. The results of this study showed that the overall SMR for all causes of death (0.85) for the workers was again well below the expected rate. No increase in prostate cancer was observed in this later study, but deaths from cerebrovascular disease were elevated in the definite PFOA exposure group. Once again, the low number of cases of cerebrovascular disease in this group (five) makes the findings tentative and difficult to interpret. Taken together, the results of these studies (three different findings of slightly elevated

disease - different in each study - based on small numbers of cases) do not represent epidemiological findings of significance.

PFOS, PFOA, and other perfluorochemicals have been detected in human blood serum from adults and children in the general population at levels from 1/100 to 1/1000 of those seen in workers (Olsen et al 2003c, Olsen et al 2003d, 3M 2001c). In a study of 645 adult donor serum samples from six Red Cross donation centers across the U.S., PFOS concentrations ranged from <4.1 ppb (the limit of detection) to 1,656 ppb. No substantial differences in PFOS concentrations in serum were observed with age of the donor. Serum PFOA concentrations ranged from <1.9 ppb to 52.3 ppb. A preliminary study of sera from 599 children ages 2-12 years from 23 different states showed PFOS concentrations ranging from 6.7 to 515 ppb, and PFOA concentrations ranging from <1.92 to 56.1 ppb. A study of elderly people in the Seattle area showed similar PFOS and PFOA serum concentrations compared to the rest of the population that has been studied so far (Olsen et al 2004). The source(s) of exposure to PFOS, PFOA, and other perfluorochemicals in the general population is unclear, but could include consumer products, environmental exposures, or other occupational exposures (3M 1999c). Analysis of blood samples collected in the early 1950s from army recruits show no PFOS (3M 1999c). Both PFOS and PFOA have been detected in samples of dust collected from household vacuum cleaner bags in Japan, indicating the indoor environment is a potential source of exposure (Moriwaki et al 2003).

Based on animal studies and available human epidemiological data for PFOA concentrations in blood serum, in a preliminary report in 2003 the EPA calculated a margin of exposure (MOE) range for PFOA for women of childbearing age and children of between 66 and 9,125 (EPA 2003). The MOE describes the relative difference between current measured human PFOA serum levels and serum levels determined in animal studies to be associated with adverse developmental effects. There are numerous uncertainties in such calculations as a result of intra- and interspecies differences, dose metrics used, and the choice of the animal model; EPA advises that they must be interpreted cautiously. The preliminary EPA report also may have seriously underestimated the serum PFOA concentrations in the rat study used to derive the MOE, making the low end of the MOE range too low. In a recent evaluation of the risk of PFOA exposure to the general population, a 3M scientist (Butenhoff et al 2004) calculated a MOE of between 1600 and 8900 for various toxicological endpoints, with a mean of 2100 based on the mean serum PFOA concentration in general population data. For PFOS, 3M has calculated a MOE range for non-occupationally exposed people of 310 to 1550 based on PFOS serum levels measured in the human population (3M 2003b). While the 3M MOE calculations suggest that the health risks to the general population from exposure to PFOA and PFOS are low, again there are inherent uncertainties in such calculations.

Summary of Environmental Data

PFOS has been detected in the plasma and tissues of wildlife from across the globe, including seals, otters, dolphins, aquatic birds, bald eagles, polar bears, freshwater and saltwater fish, and reptiles (Giesy and Kannan 2001). The results of this study show that PFOS is widely distributed in the global environment. Levels of PFOS were higher in fish-eating and predatory

animals than in their typical prey, indicating that PFOS may bioaccumulate as it moves up the food chain. Bald eagles from the Midwestern U.S. showed the highest levels of PFOS in plasma (up to 2,570 nanograms per milliliter), and mink from the Midwestern U.S. showed the highest levels in tissue (in liver; up to 3,680 nanograms per gram). Concentrations of other PFCs in wildlife samples, such as PFOA, are typically approximately ten times lower and are much less widely distributed (Giesy et al 2001).

Broader studies have found detectable levels of PFOS in surface waters, fish and bird blood and livers, and human blood collected in Japan, with the highest levels observed in the waters and fish from heavily industrialized Tokyo Bay (Taniyasu et al 2003). A decreasing gradient of PFOS levels in aquatic invertebrates and two species of fish in an estuary and the North Sea was observed with distance from the port of Antwerp, Belgium (Van de Vijver et al 2003; Hoff et al. 2003). 3M operated a PFC manufacturing plant in Antwerp.

Estimated bioconcentration factors for PFOS in fish range from 200 to 1,124 in bluegills and carp (OECD 2002). Studies of APFO and PFOA have estimated that bioconcentration factors are quite low (1.8 in fathead minnows). Therefore, in contrast to PFOS, PFOA does not bioconcentrate through the food chain (EPA 2002).

In the United States, 3M researchers conducted a study of PFOA and PFOS levels in the Tennessee River both upstream and downstream of its facility in Decatur, Alabama (Hansen et al 2002). Analysis of 40 water samples showed that low levels of PFOS were present throughout the 80-mile section of the river studied. Concentrations increased from an average of 32 +/- 11 parts per trillion (ppt) upstream of the PFC manufacturing facility in Decatur to an average of 114 +/- 19 ppt downstream. Concentrations of PFOA were below the laboratory detection limits (25 ppt) upstream of the Decatur facility, but averaged 394 +/- 128 ppt downstream of the facility. The relatively consistent concentrations of PFOS and PFOA found in the Tennessee River suggest that there are no significant removal mechanisms (such as volatilization or adsorption to sediment) affecting their presence in the water. Boulanger et. al. (2004) studied PFOS and PFOA concentrations in sixteen water samples collected from Lake Erie and Lake Ontario. PFOS concentrations ranged from 21 – 70 ppt (mean 43 +/- 18 ppt) in the two lakes, while PFOA concentrations ranged from 27 – 50 ppt (mean 39 +/- 9 ppt). These concentrations were higher than those observed in the Tennessee River upstream of the 3M facility in Decatur. Ongoing studies (coordinated mainly by 3M) are designed to determine PFC concentrations in drinking water, food products, sediments, wastewater treatment plant effluent, sewage sludge, and landfill leachate in a number of cities across the U.S. (Battelle 2000; OECD 2002, EPA 2002). Four cities where PFCs are manufactured or used (supply cities), and two control cities were initially targeted. PFOS concentrations in wastewater treatment plant effluent ranged from 0.041 to 5.29 ppb while PFOA concentrations ranged from 0.040 ppb to 2.42 ppb. In dried treatment plant sludge the PFOS concentrations ranged from 0.2 ppb to 3,120 ppb and PFOA concentrations were from non-detect to 244 ppb. Drinking water samples showed maximum PFOS and PFOA concentrations of 0.063 ppb and 0.029 ppb, respectively; landfill leachate ranged from non-detect to 53.1 ppb for PFOS and non-detect to 48.1 ppb for PFOA. Surface waters ranged from non-detect to 0.138 ppb for PFOS and from non-detect to 0.083 ppb for

PFOA; sediments ranged from non-detect to 1.13 ppb for PFOS and from non-detect to 1.75 ppb for PFOA. Data from the control cities were generally at the lower end of these ranges, with a few exceptions. More than 200 food product samples (green beans, apples, pork, milk, chicken, eggs, bread, fish, and ground beef) were also collected. PFOS was only detected in five samples, (one ground beef and four milk samples), at a maximum concentration of 0.852 nanograms per gram (ng/g). Only one of the four milk samples was from a control city, with the remainder from supply cities. PFOA was detected at concentrations up to 2.35 ng/g in two ground beef samples from control cities, two bread samples (from one control and supply cities), two apple samples (supply cities), and one green bean sample from a supply city.

The available data regarding the presence of PFCs in the environment suggest that they are widespread. Humans may be exposed to PFCs through numerous pathways and common activities – the exact routes and exposure concentrations are not currently known.

Planned Actions

ERG, on behalf of 3M, has proposed a workplan conducting a facility-wide investigation of PFC releases at the site (ERG 2004). The purpose of the workplan is to:

- Define the extent and magnitude of on-site contamination resulting from the past site waste disposal practices of PFCs;
- Define the hydrology and geology of the site and the potential routes of exposure; and
- Provide information and data needed for consideration of response actions.

The workplan involves the collection of historical information on PFC production, use, and disposal, including releases to the environment, summarizing all available information regarding groundwater monitoring and production wells on the site. It also involves preparation of a groundwater flow model, and collection of groundwater samples for PFC analysis from all wells on the site. A further step will be to collect groundwater samples near the Mississippi River using push-probes in locations where PFCs were used or disposed, and finally preparation of a summary report.

EPA's Office of Pollution Prevention and Toxics, through an enforceable consent agreement (ECA) process undertaken with various manufacturers and users of PFCs (including 3M) and other interested parties, has been studying the extent, distribution, and fate of PFCs (primarily PFOA) in the environment associated with the manufacture, use, or disposal of PFCs or PFC containing products. All documents related to this undertaking are posted and available on an EPA web site (www.epa.gov/edocket/) under docket number OPPT-2003-0012.

In this ECA process, EPA identified several needs for monitoring information, including monitoring in the vicinity of facilities currently manufacturing, processing, and using various PFCs. Three companies – 3M, Dyneon (a 3M company), and DuPont – participating in this process have indicated a willingness to enter into Memoranda of Understanding (MOU) with the EPA for monitoring on and around their respective fluoropolymer manufacturing facilities located in Decatur, Alabama and Washington, West Virginia. 3M/Dyneon and EPA executed an MOU on October 25, 2004. A fourth company, Daikin America, is undertaking an independent,

voluntary monitoring program at its fluoropolymer manufacturing facility, which is co-located with the 3M/Dyneon plant in Decatur, Alabama. The 3M - Cottage Grove facility has not been included in this effort to date because it is no longer producing PFOA on a commercial basis (M.F. Dominiak, EPA, personal communication, 2004). The phased-approach monitoring plan for the 3M/Dyneon plant in Decatur, Alabama involves the following (in no particular order; Weston 2004):

- Monitoring of groundwater wells and plant effluent (on and off-site);
- Monitoring of surface water, sediments, aquatic organisms and fish in the adjacent Tennessee River;
- Air dispersion modeling of PFC emissions;
- Soil sampling (on and off-site);
- A well survey in the area of the plant;
- Sampling of the Decatur water supply and wastewater treatment plant effluent;
- Sampling of terrestrial vegetation and vertebrates (on and off-site); and
- Monitoring of aquatic avian biota (on and off-site).

The 3M/Dyneon MOU with EPA, as well as the full text of the monitoring plan are located on the EPA web site (www.epa.gov/edocket/) under docket number OPPT-2004-0112. Some of the proposed monitoring has already been conducted, with other work proposed for 2004 and 2005. The results of the studies will be provided to EPA when completed. Similar monitoring (including air monitoring for PFCs) has been proposed for other PFC manufacturing sites. The proposed scope of this monitoring plan is broader than the scope proposed by ERG for the 3M - Cottage Grove facility. Due to business data privacy concerns, the relative sizes of the two facilities in terms of the production quantities of PFCs are not available from 3M. However, there are many apparent similarities in terms of overall PFC production, site layout, past on-site waste disposal, discharge of PFC containing wastes to a major waterway (the Tennessee River in Decatur and the Mississippi River in Cottage Grove), and the length of time PFCs were produced (40+ years at Decatur and as many as 50 years at Cottage Grove). Based on these factors, a similar, phased scope of investigative work for the 3M - Cottage Grove site may be needed to properly assess the potential impact of decades of PFC production and waste disposal. Some aspects of the Decatur workplan may not be applicable to the Cottage Grove facility. The MPCA has also stated that PFC production wastes from the Cottage Grove facility may have been disposed at other known 3M waste disposal sites in the Twin Cities area (MPCA 2004). If so, there is a potential for PFCs to have affected various media (soil, groundwater, or surface water) in these locations as well.

Child Health Considerations

ATSDR and MDH recognize that the unique vulnerabilities of infants and children make them of special concern to communities faced with contamination of their water, soil, air, or food. Children are at greater risk than adults from certain kinds of exposures to hazardous substances at waste disposal sites. They are more likely to be exposed because they play outdoors and they often bring food into contaminated areas. They are smaller than adults, which means they

breathe dust, soil, and heavy vapors close to the ground. Children also weigh less, resulting in 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 management decisions, housing decisions, and access to medical care.

Because the site is a secure chemical production and waste disposal facility, children are very unlikely to have been exposed to PFCs at the site itself. There are currently no data available to determine if children could have been exposed to PFCs off of the site property. If air emissions of PFCs extended off the site property, children who may have been living in areas beyond the site boundaries could have been exposed while production was occurring, or could be exposed through other environmental media. PFCs have been detected in blood samples of children from at least 23 different states.

IV. Conclusions

The potential impacts on public health from perfluorochemical releases at the 3M - Cottage Grove facility cannot be fully assessed by MDH at this time, because there are not sufficient environmental data available regarding PFC impacts from the facility in soil, groundwater, surface water, sediments, and biota. At this time perfluorochemical releases from the site represent an indeterminate public health hazard. There is a lack of information about how the general population is exposed to PFCs. PFCs have a long half-life in humans and animal studies indicate a potential for toxicity to the liver and effects on reproduction and development.

V. Recommendations

1. Consideration should be given to developing and implementing a scope of investigation work that is generally similar to that developed by 3M for the Decatur, Alabama facility under their proposed voluntary agreement with the EPA (see pages 23-24). Some aspects of the Decatur workplan may not be applicable to the Cottage Grove facility, so a phased approach is recommended. The data from such an investigation are needed to understand the extent of PFC contamination from the facility in all media, and to assess its potential impact on public health.
2. 3M should continue to take action to ensure that workers at the Cottage Grove facility are not exposed to PFCs through the facility water supply at concentrations in excess of the MDH HBVs (currently being implemented by 3M).
3. While releases of PFCs to the Mississippi River are now being generally prevented by the installation of GAC treatment, 3M should continue to identify and reduce (or eliminate where possible) any other potential ongoing discharges of PFOS and PFOA to the environment from the facility.
4. Information should be gathered by 3M regarding any off-site locations where PFC processing wastes from the site were disposed in the past, and appropriate steps should be taken to investigate possible PFC releases from those locations.

VI. Public Health Action Plan

MDH's Public Health Action Plan for the site consists of continued consultation with MPCA staff on the investigation of PFC releases at the site, distribution of this report, possible additional private well sampling, and participation in any planned public outreach activities.

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VII. References

- 3M 1999a. The Science of Organic Fluorochemistry. 3M Company, St. Paul, Minnesota. February 5, 1999.
- 3M 1999b. Fluorochemical Use, Distribution, and Release Overview. 3M Company, St. Paul, Minnesota. May 26, 1999.
- 3M 1999c. Perfluorooctane Sulfonate: Current Summary of Human Sera, Health, and Toxicology Data. 3M Company, St. Paul, Minnesota. January 21, 1999.
- 3M 2000a. Correspondence from William A. Weppner, 3M Company, to Charles Auer, US Environmental Protection Agency. June 16, 2000.
- 3M 2000b. Voluntary Use and Exposure Information Profile – Perfluorooctane Sulfonic Acid and Various Salt Forms. 3M, St. Paul, Minnesota. April 27, 2000.
- 3M 2000c. Voluntary Use and Exposure Information Profile – Perfluorooctanoic Acid and Salts. 3M, St. Paul, Minnesota. June 8, 2000.
- 3M 2001a. Sulfonated Perfluorochemicals in the Environment: Sources, Dispersion, Fate and Effects. 3M, St. Paul, Minnesota. March 1, 2001.
- 3M 2001b. Correspondence from Michael A. Santoro, 3M Company to the US EPA Office of Pollution Prevention and Toxics. August 1, 2003.
- 3M 2001c. Identification of fluorochemicals in sera of children in the United States. Interim report, 3M Company Medical Department, St. Paul, Minnesota. June 25, 2001.
- 3M 2003a. 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 2003b. Environmental and Health Assessment of Perfluorooctane Sulfonate Acid and its Salts. 3M Company, St. Paul, Minnesota. August 20, 2003.
- Abe, T. and Nagase, S. 1982. Electrochemical fluorination (Simons Process) as a route to perfluorinated organic compounds of industrial interest. In: Banks, R.E., editor. Preparation, properties, and industrial applications of organofluorine compounds. New York: John Wiley & Sons; 1982. p. 19-43.
- Alexander, B.H. 2001. Mortality Study of Workers Employed at the 3M Cottage Grove Facility. (unpublished).
- Alexander, B.H., Olsen, G.W., Burris, J.M., Mandel, J.H., and Mandel, J.H. 2003. Mortality of

employees of a perfluorooctanesulphonyl fluoride manufacturing plant. *Occupational and Environmental Medicine* 60: 722-729.

Badr, M.Z., and Birnbaum, L.S. 2004. Enhanced potential for oxidative stress in livers of senescent rats by the peroxisome proliferator-activated receptor alpha agonist perfluorooctanoic acid. *Mechanisms of Ageing and Development* 125: 69-75.

Barr 1991. Remedial Investigation/Detailed Analysis Report – Boiler Ash/Acrylic Acid Release Areas, 3M Chemolite Center. Barr Engineering Company, August 1991.

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

Battelle 2000. Design and Structure of Multi-City Study. Battelle Memorial Institute, Columbus, Ohio. May 1, 2000.

Boulanger, B., Vargo, J., Schnoor, J.L., and Hornbuckle, K.C. 2004. Detection of perfluorooctane surfactants in Great Lakes water. *Environmental Science and Technology* 38: 4064-4070.

Butenhoff, J., Costa, G., Elcombe, C., Farrar, D., Hansen, K., Iwai, H., Jung, R., Kennedy, G., 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., Gaylor, D.W., Moore, J.A., Olsen, G.W., Rodricks, J., Mandel, J.H., and Zobel, L.R. 2004. Characterization of risk for general population exposure to perfluorooctanoate. *Regulatory Toxicology and Pharmacology* 39: 363-380.

Daikin 2004. Letter to Charles M. Auer, U.S. EPA, from Satoshi Doi, Daikin America, Inc. January 28, 2004.

DMER 1999. Letter to Dr. Rich Purdy, 3M and Dr. Joyce Cooper, Battelle. D. Mackay Environmental Research, Ltd., Peterborough, Ontario, Canada. April, 1999.

DNR 2003. Division of Waters Appropriations Permit Data. Found online at: http://www.dnr.state.mn.us/waters/watermgmt_section/appropriations/wateruse.html. Minnesota Department of Natural Resources, St. Paul, Minnesota.

ERG 2001a. Correspondence from Paul Book, Environmental Resource Group, LLC, to Mark Rys, MPCA. January 30, 2001.

ERG 2001b. Correspondence from Paul Book, Environmental Resource Group, LLC, to Dave Douglas, MPCA. July 17, 2001.

ERG 2003. 3M Cottage Grove Chemical Sewer Replacement Environmental Oversight Report. Environmental Resource Group, LLC. September 9, 2003.

ERG 2004. Facility-wide Fluorocarbon Investigation Work Plan – 3M Cottage Grove. Environmental Resource Group, LLC. January 27, 2004.

EPA 2002. Revised Draft Hazard Assessment of Perflouroctanoic 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 Perflouroctanoic Acid and its Salts. U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics. April 10, 2003.

Franklin, J. 2002. Screening Assessment of the Potential for Long-Range Atmospheric Transport of Perfluorooctanoic Acid. James Franklin, Solvay Research & Technology. March 24, 2002. Accessed at www.epa.gov/edocket/, OPPT-2003-0012-0183.

Giesy, J.P., and Kannan, K. 2001. Global distribution of perfluorooctane sulfonate in wildlife. *Environmental Science and Technology* 35: 1339-1342.

Giesy, J.P., Kannan, K., and Jones, P.D. 2001. Global biomonitoring of perflourinated organics. *The Scientific World* 1: 627-629.

Gilliland, F.D. and Mandel, J.S. 1993. Mortality among employees of a perfluorooctanoic acid production plant. *Journal of Occupational Medicine* 35: 950-954.

Hansen, K.J., Johnson, H.O., Eldridge, J.S., Butenhoff, J.L., and Dick, L.A. 2002. Quantitative characterization of trace levels of PFOS and PFOA in the Tennessee River. *Environmental Science and Technology* 36: 1681-1685.

Haughom, B. and Spydevold, O. 1992. The mechanism underlying the hypolipemic effect of perfluorooctanoic acid (PFOA), perfluorooctane sulphonic acid (PFOSA) and clofibrac acid. *Biochimica et Biophysica Acta* 1128: 65-72.

Hoff, P.T., Van de Vijver, K., Van Dongen, W., Esmans, E.L., Blust, R., and De Coen, W.M. 2003. Perfluorooctane sulfonic acid in bib (*Trisopterus luscus*) and plaice (*Pleuronectes platessa*) from the western scheldt and the Belgian North Sea: distribution and biochemical effects. *Environmental Toxicology and Chemistry* 22: 608-614.

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.
- Kudo, N. and Kawashima, Y. 2003. Toxicity and toxicokinetics of perfluorooctanoic acid in humans and animals. *The Journal of Toxicological Sciences* 28: 49-57.
- Lau, C., Thibodeaux, J.R., Hanson, R.G., Rogers, J.M., Grey, B.E., Stanton, M.E., Butenhoff, J.L., and Stevenson, L.A. 2003. Exposure to perfluorooctane sulfonate during pregnancy in rat and mouse. II: postnatal evaluation. *Toxicological Sciences* 74: 382-392.
- MPCA 1998. Site Evaluation Checklist & Summary. Eric Porcher, Minnesota Pollution Control Agency, St. Paul, Minnesota. February 9, 1998.
- MPCA 2004. 3M Cottage Grove (Chemolite) Site and 3M Fluorochemical Disposal. Memorandum from David Douglas to Bruce Brott. Minnesota Pollution Control Agency, St. Paul, Minnesota. February 5, 2004.
- Moody, C.A., Hebert, G.N., Strauss, S.H., and Field, J.A. 2003. Occurrence and persistence of perfluorooctanesulfonate and other perfluorinated surfactants in groundwater at a fire-training area at Wurtsmith Air Force Base, Michigan, USA. *Journal of Environmental Monitoring* 5: 341-345.
- Moriwaki, H., Takata, Y., and Arakawa, R. 2003. Concentrations of perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) in vacuum cleaner dust collected in Japanese homes. *Journal of Environmental Monitoring* 5: 753-757.
- Mossler, J.H. 2003. Generalized structure of the Jordan Sandstone. Minnesota Geological Survey, unpublished manuscript map.
- OECD 2002. Hazard Assessment of Perfluorooctane Sulfonate (PFOS) and its Salts. Organization for Economic Cooperation and Development. November 21, 2002.
- Olsen, G.W., Gilliland, F.D., Burlew, M.M., Burris, J.M., Mandel, J.S., and Mandel, J.H. 1998. An epidemiologic investigation of reproductive hormones in men with occupational exposure to perfluorooctanoic acid. *Journal of Occupational Environmental Medicine* 40: 614-622.
- Olsen, G.W., Logan, P.W., Hansen, K.J., Simpson, C.A., Burris, J.M., Burlew, M.M., Vorarath, P.P., Venkateswarlu, P., Schumpert, J.C., and Mandel, J.H. 2003a. An occupational exposure assessment of a perfluorooctanesulfonyl fluoride production site: biomonitoring. *Journal of the American Industrial Hygiene Association* 64: 651-659.

Olsen, G.W., Burris, J.M., Burlew, M.M., and Mandel, J.H. 2003b. Epidemiologic assessment of worker serum perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA) concentrations and medical surveillance examinations. *Journal of Occupation Environmental Medicine* 45: 260-270.

Olsen, G.W., Hansen, K.J., Stevenson, L.A., Burris, J.M., and Mandel, J.H. 2003c. Human donor liver and serum concentrations of perfluorooctanesulfonate and other perfluorochemicals. *Environmental Science and Technology* 37: 888-891.

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. 2003d. 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., Larson, E.B., van Belle, G., Lundberg, J.K., Hansen, K.J., Burris, J.M., Mandel, J.H., and Zobel, L.R. 2004. Serum concentrations of perfluorooctanesulfonate and other fluorochemicals in an elderly population from Seattle, Washington. *Chemosphere* 54: 1599-1611.

Pace 1991. 3M Company Chemolite Center Site: Air Dispersion Modeling Report – VOC and Inorganic Emissions. Pace, Incorporated. May, 1991.

Taniyasu, S., Kannan, K., Horii, Y., Hanari, N., and Yamashita, N. 2003. A survey of perfluorooctane sulfonate and related perfluorinated organic compounds in water, fish, birds, and humans from Japan. *Environmental Science and Technology* 37: 2634-2639.

Thibodeaux, J.R., Hanson, R.G., Rogers, J.M., Grey, B.E., Barbee, B.D., Richards, J.H., Butenhoff, J.L., Stevenson, L.A., and Lau, C. 2003. Exposure to perfluorooctane sulfonate during pregnancy in rat and mouse. I: maternal and prenatal evaluations. *Toxicological Sciences* 74: 382-392.

Van de Vijver, K.I., Hoff, P.T., Van Dongen, W., Esmans, E.L., Blust, R., and De Coen, W.M. 2003. Exposure patterns of perfluorooctane sulfonate in aquatic invertebrates from the western scheldt estuary and the southern North Sea. *Environmental Toxicology and Chemistry* 22: 2037-2041.

West Virginia Dept. of Environmental Protection 2003. Ammonium Perfluorooctanoate (C-8) Groundwater Investigation Steering Team Report. August, 2003.

Weston 2004. Phase 2 Workplan for Sampling Environmental Media for PFOA at the 3M Decatur, Al Plant. Weston Solutions, Inc., West Chester, PA. October 2004.

CERTIFICATION

This 3M - Cottage Grove Health Consultation was prepared by the Minnesota Department of Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the health consultation was begun.



Jeff Kellam
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ATSDR

The Division of Health Assessment and Consultation, ATSDR, has reviewed this public health consultation and concurs with the findings.



Roberta Erlwein
Chief, Cooperative Agreement Team, SSAB, DHAC, ATSDR

for RE

Table 1: PFC Well Monitoring Results
3M Cottage Grove Facility
(ug/L)

Sample Point	Date Sampled	PFOS (C8)	Perfluoroheptane -sulfonate (C7)	Perfluorohexane -sulfonate (C6)	Perfluorobutane -sulfonate (C4)	PFOA (C8)	Perfluoroheptanoic Acid (C7)	Perfluorohexanoic Acid (C6)
MW-4	10/31/2001	5		7.3	5.5	6.33		
	11/12/2001					5.29		
	6/5/2003					10.2		
MW-7	6/5/2003	ND		ND	ND	0.314		
MW-14	9/7/2001					846		
	10/31/2001					798		
	11/12/2001					825		
PZ-14	9/7/2001	0.68		1.9	1.02	6.41		
	10/31/2001					5.37		
	11/12/2001					4.63		
	6/5/2003					4.81		
MW-101	12/3/2002	346	33.5	1893	37.4	174	206	181
	4/1/2003	275	47.4	2917	32.8			
	5/19/2003	211	43.7	1227	39.1			
	6/5/2003	224		1123	19.3	136		
MW-102	12/3/2002	371	2.3	76.2	16	366	25.2	66
	4/1/2003	76.3	2.4	36.4	21.2			
	5/19/2003	101	3.1	63.7	33.5			
PW-2	6/10/2001					0.51		
	6/28/2001					5.4		
	9/7/2001					0.39		
	10/31/2001					2.3		
	11/12/2001					0.61		
PW-3	6/10/2001					0.57		
	6/28/2001					0.77		
	9/7/2001					0.72		
	10/31/2001					0.56		
	11/12/2001					0.72		

Table 1: PFC Well Monitoring Results
3M Cottage Grove Facility
(ug/L)

Sample Point	Date Sampled	PFOS (C8)	Perfluoroheptane -sulfonate (C7)	Perfluorohexane -sulfonate (C6)	Perfluorobutane -sulfonate (C4)	PFOA (C8)	Perfluoroheptanoic Acid (C7)	Perfluorohexanoic Acid (C6)
PW-4	6/10/2001					1.15		
	6/28/2001					0.95		
	9/7/2001					0.81		
	10/31/2001					10.5		
	11/12/2001					0.79		
PW-5	6/10/2001					48.2		
	6/28/2001					28.5		
	9/7/2001					28.3		
	10/31/2001					40		
	11/12/2001					21.8		
PW-6	7/11/2001					135		
	9/7/2001							
	10/31/2001					12.3		
	11/12/2001					92.9		
Water Distribution System (Building 116)	7/11/2001					1.19		
	10/31/2001					40.1		
	11/12/2001					13.1		
	6/5/2003	9.4		1.8	1.7	27.9		
MDH HBV*		1	NE	NE	NE	7	NE	NE

*Health-Based Value, MDH 2002

Bold indicates exceedance of HBV

ND = Not Detected

NE = No HBV Established

Table 2: PFC Effluent Monitoring Results
3M Cottage Grove Facility
(ug/L)

Date Sampled		PFOS (C8)	Perfluoroheptane-sulfonate (C7)	Perfluorohexane-sulfonate (C6)	Perfluorobutane-sulfonate (C4)	PFOA (C8)	Perfluoroheptanoic Acid (C7)	Perfluorohexanoic Acid (C6)	Perfluorobutyric Acid (C4)
1/1996 - 10/2001	Max	384	12	12	138	267	19	32	643
	Avg	262	NA	11	64	216	14	29	346
	# of Samples	3	3	3	3	3	3	3	3
	# of Detects	3	1	3	3	3	3	3	3
1/2000 - 3/2000	Avg of 8 Data Points					1991			
9/2000 - 10/2000	Avg of 3 Data Points					216			
12/2002						180			
1/2003	Avg of 2					79			
2/2003	Avg of 2					79.4			
2/2003	Avg of 2					74.5			
4/2003	Avg of 2					110.5			
5/2003	Avg of 2					98			
6/2003	Avg of 2					17.7			

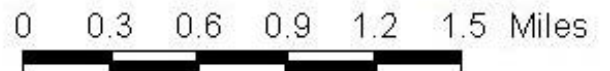
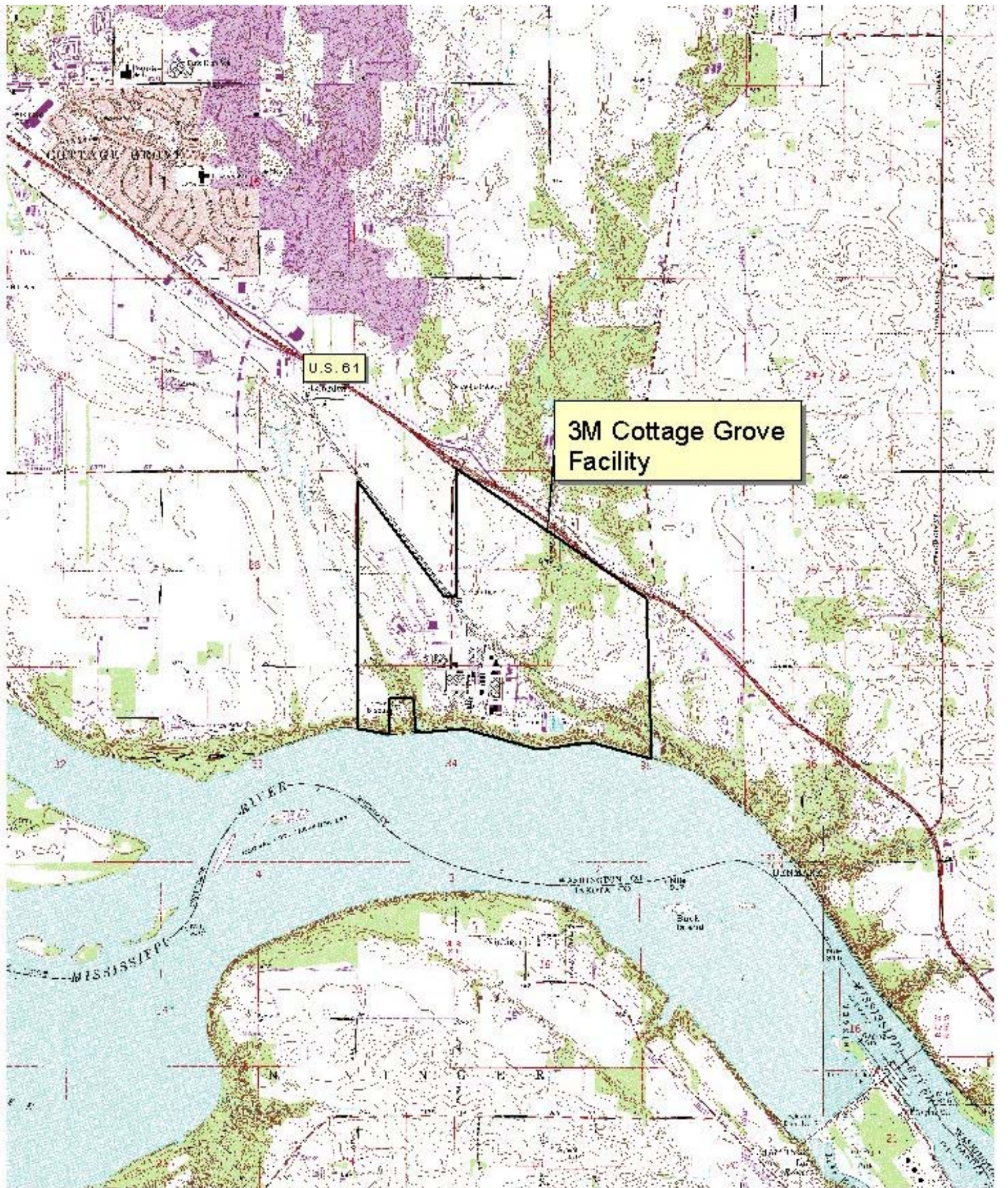


Figure 1: Site Location

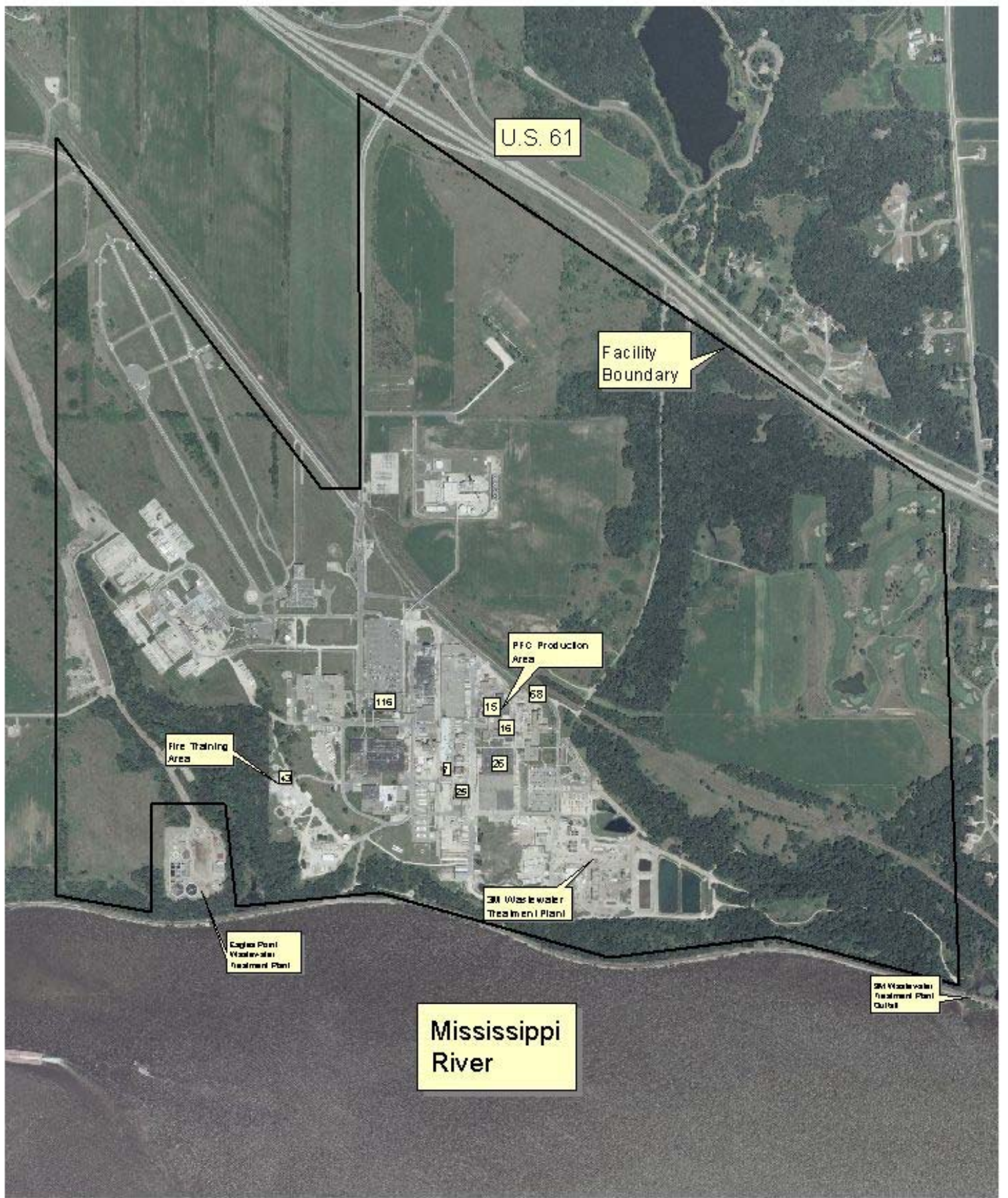


Figure 2: Site Features

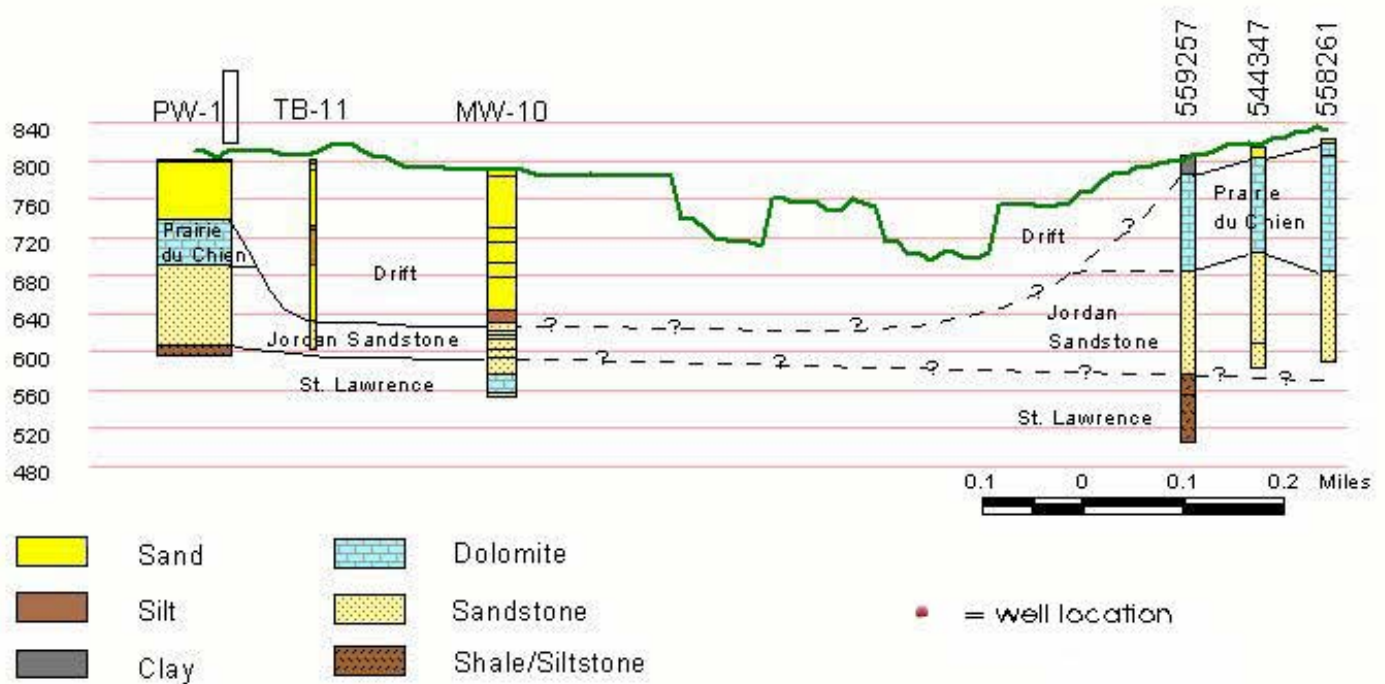
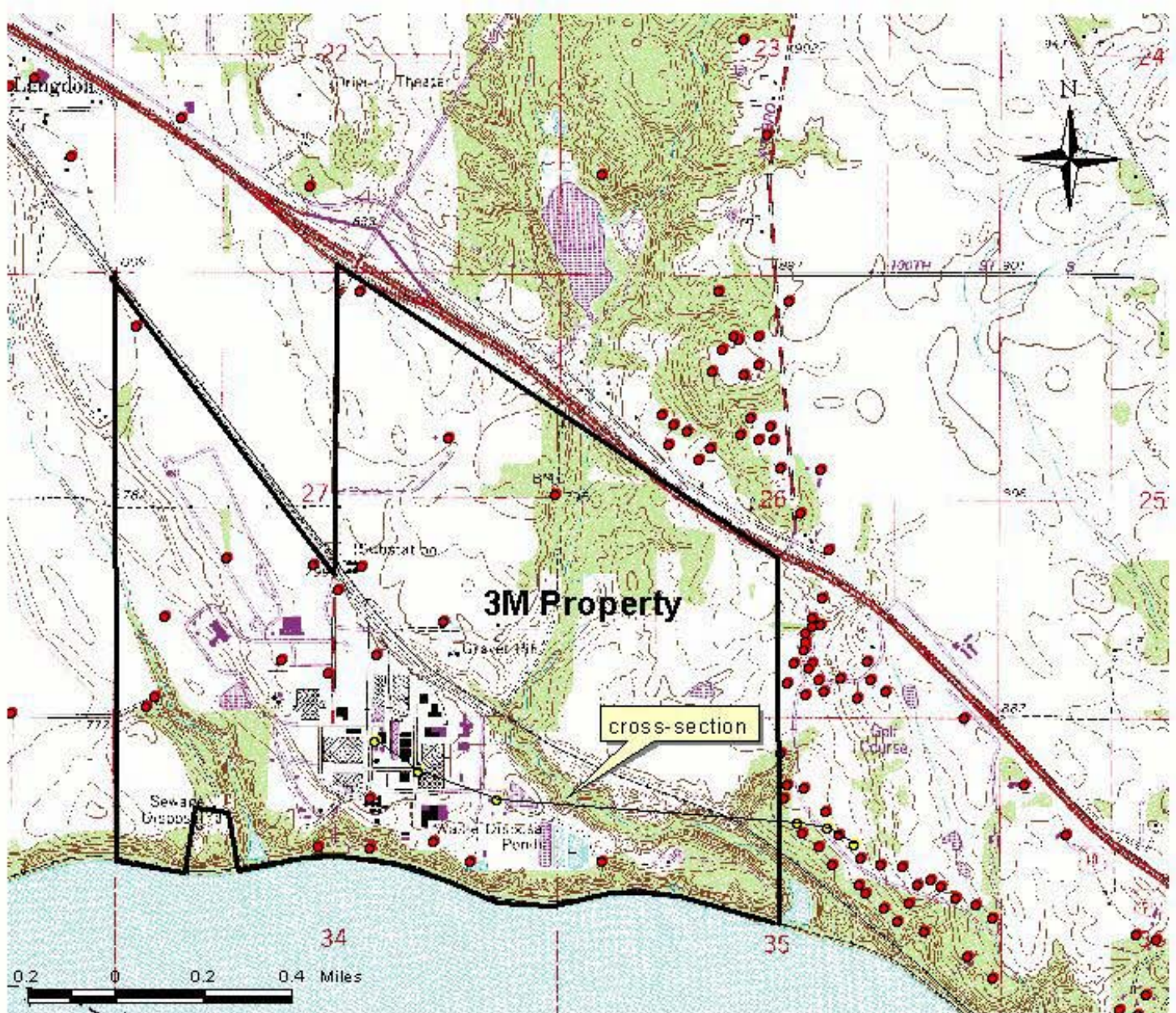


Figure 3: Site Geologic Cross-section

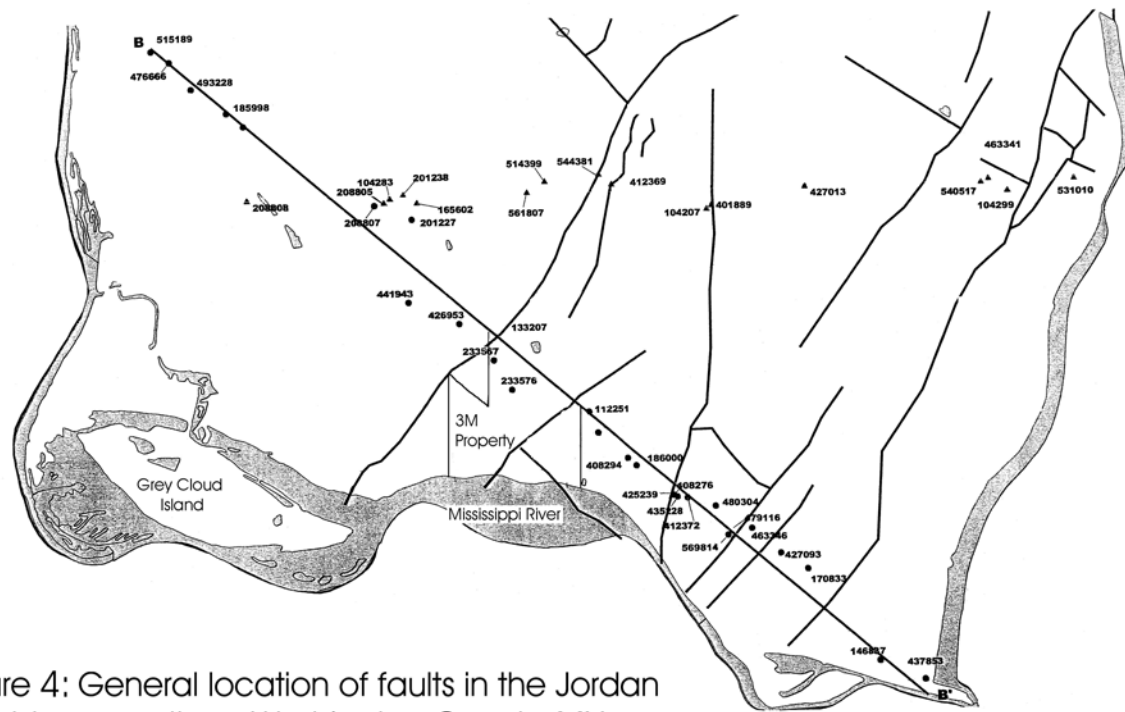
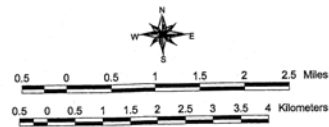


Figure 4: General location of faults in the Jordan Sandstone, southern Washington County, MN
 Cross-section B-B' located approximately along Hwy. 61



Modified from Mossler, 2003



Figure 5: Cross-section showing faults in bedrock
 Cross-section location shown on Figure 4

111419 Minnesota Unique Well Number
 ↓↑ Indicates Relative Movement Along Fault

NOTES: 1. Fault Locations Provided by Minnesota Geological Survey
 2. Fault Orientations are Assumed to be Vertical or Nearly So
 3. Well Logs Obtained from Minnesota Geological Survey's County Well Index

Modified from Barr Engineering, 2003

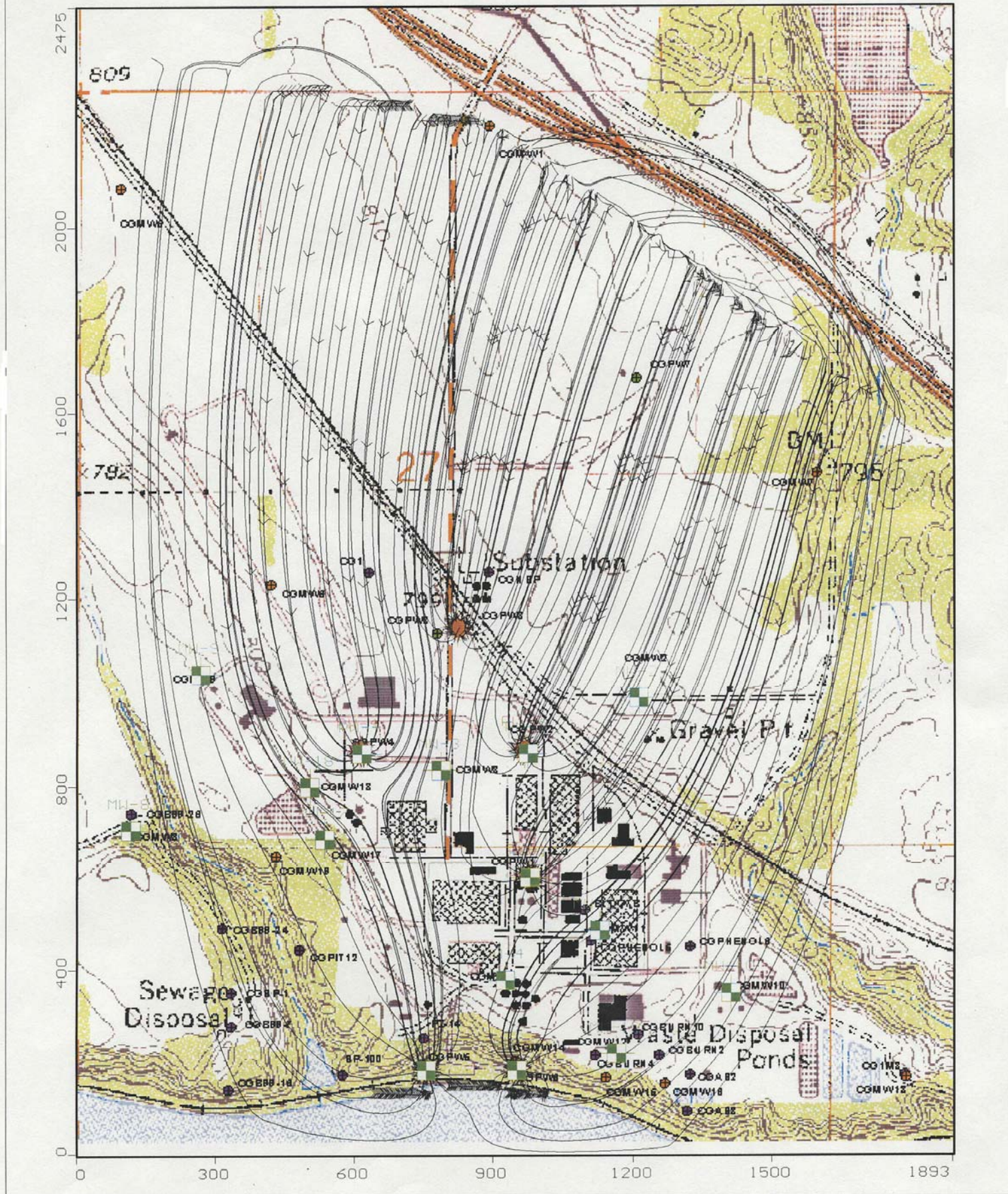


Figure 6: Groundwater Flow Model Results

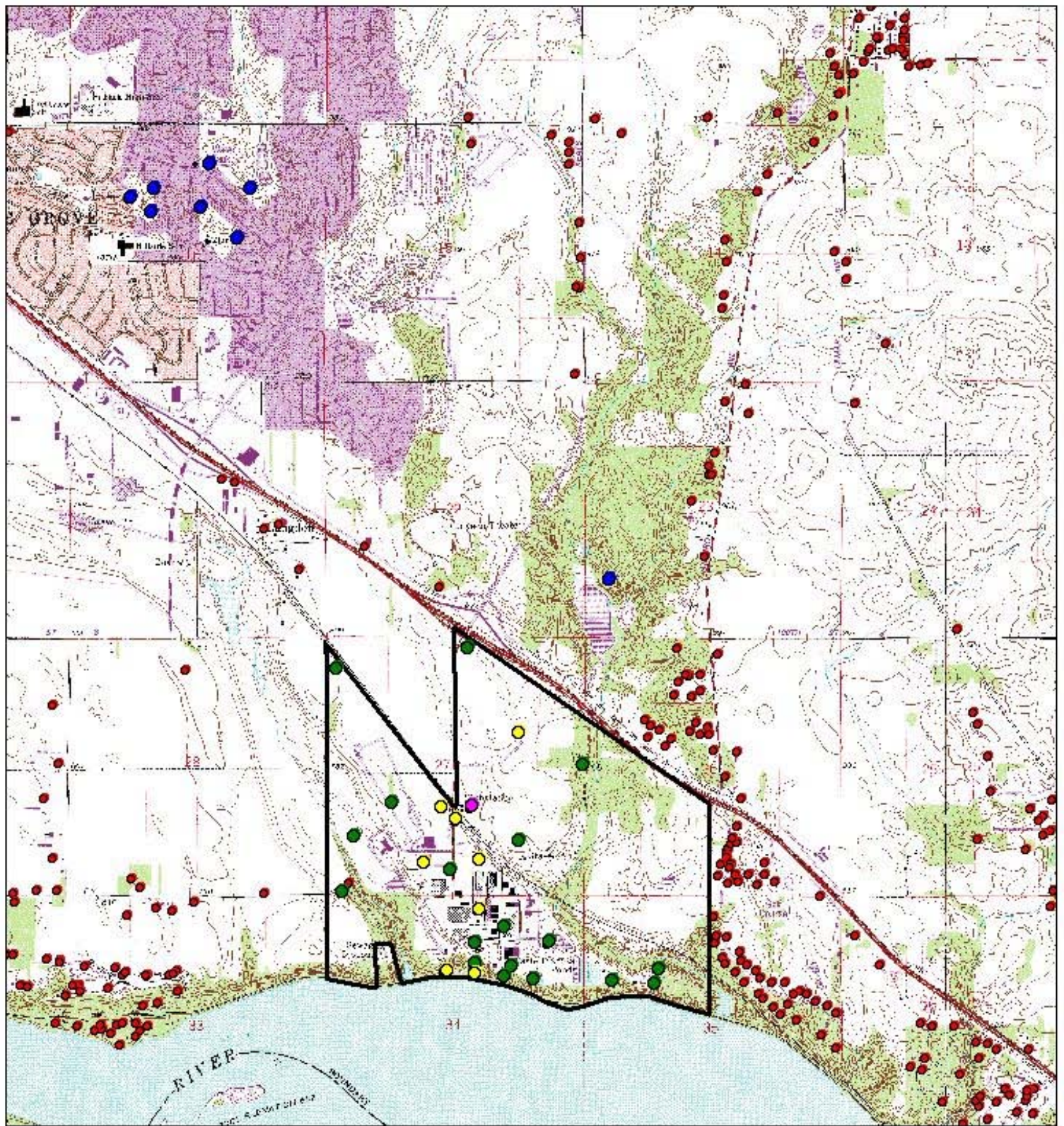
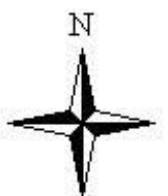


Figure 7: Location of Water Supply Wells Near 3M Site

LEGEND

- Public Water Supply Well
- Private Well (Residential or Commercial)
- 3M Monitoring Well
- 3M Production Well
- NSP (aka Xcel Energy) Well
- Border of 3M Property



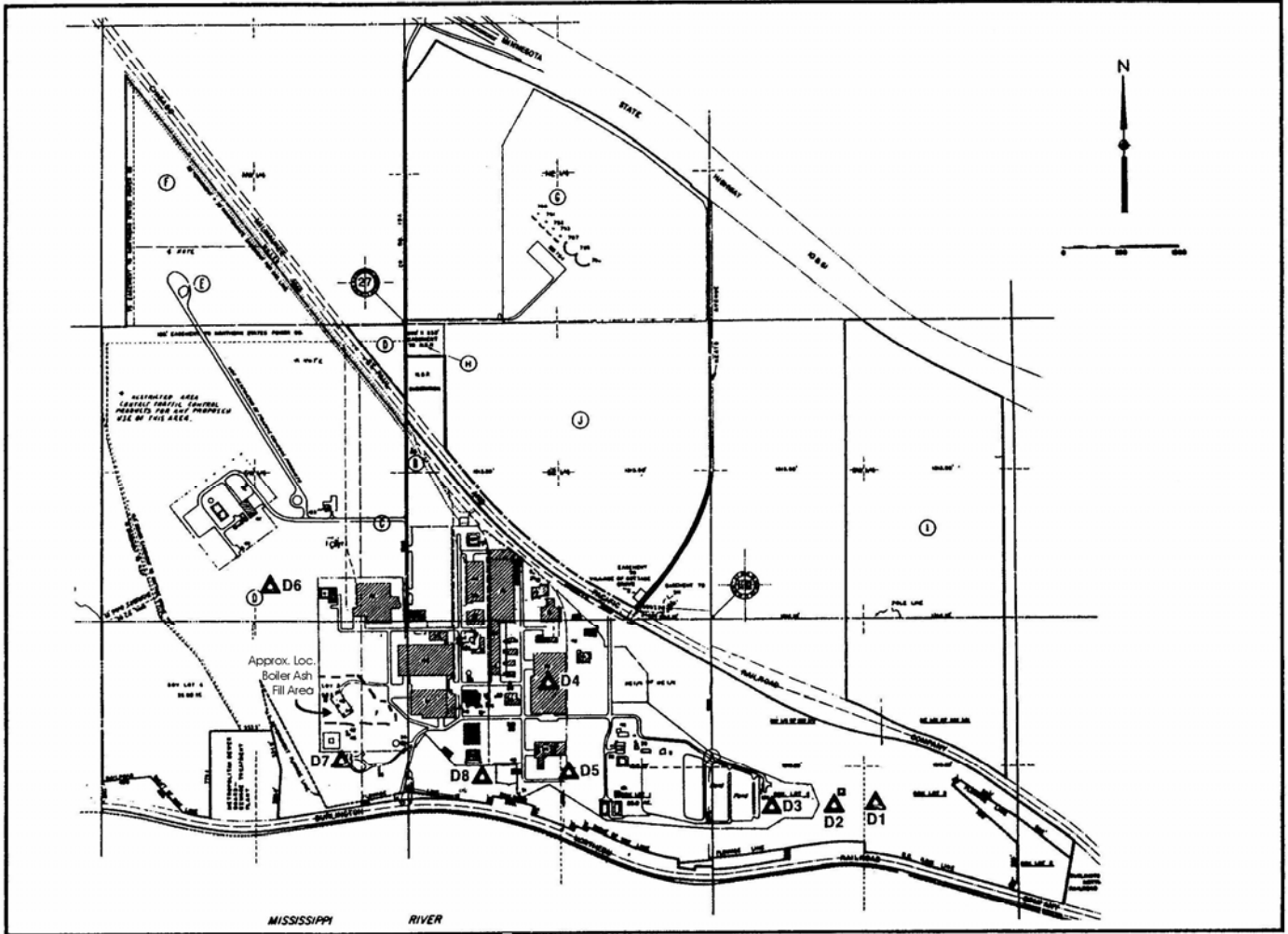


FIGURE 8: LOCATIONS OF KNOWN WASTE DISPOSAL AREAS AT THE 3M CHEMOLITE CENTER

Source: Barr 1991

Figure 9a
3M CHEMOLITE BLDG 15
1986 Annual Average
HF Concentration ($\mu\text{g}/\text{m}^3$)
Urban Mode, Elevated Terrain

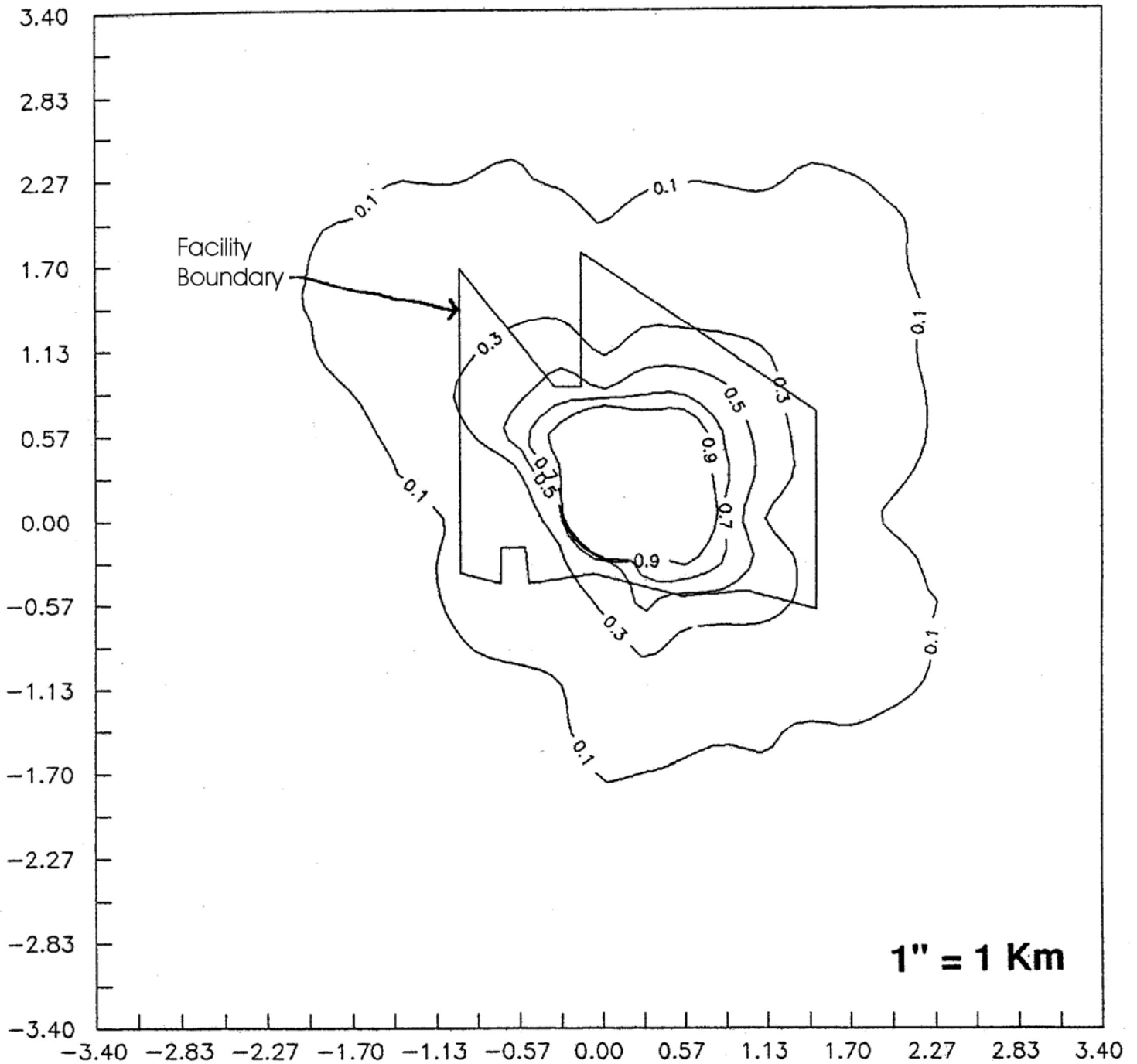
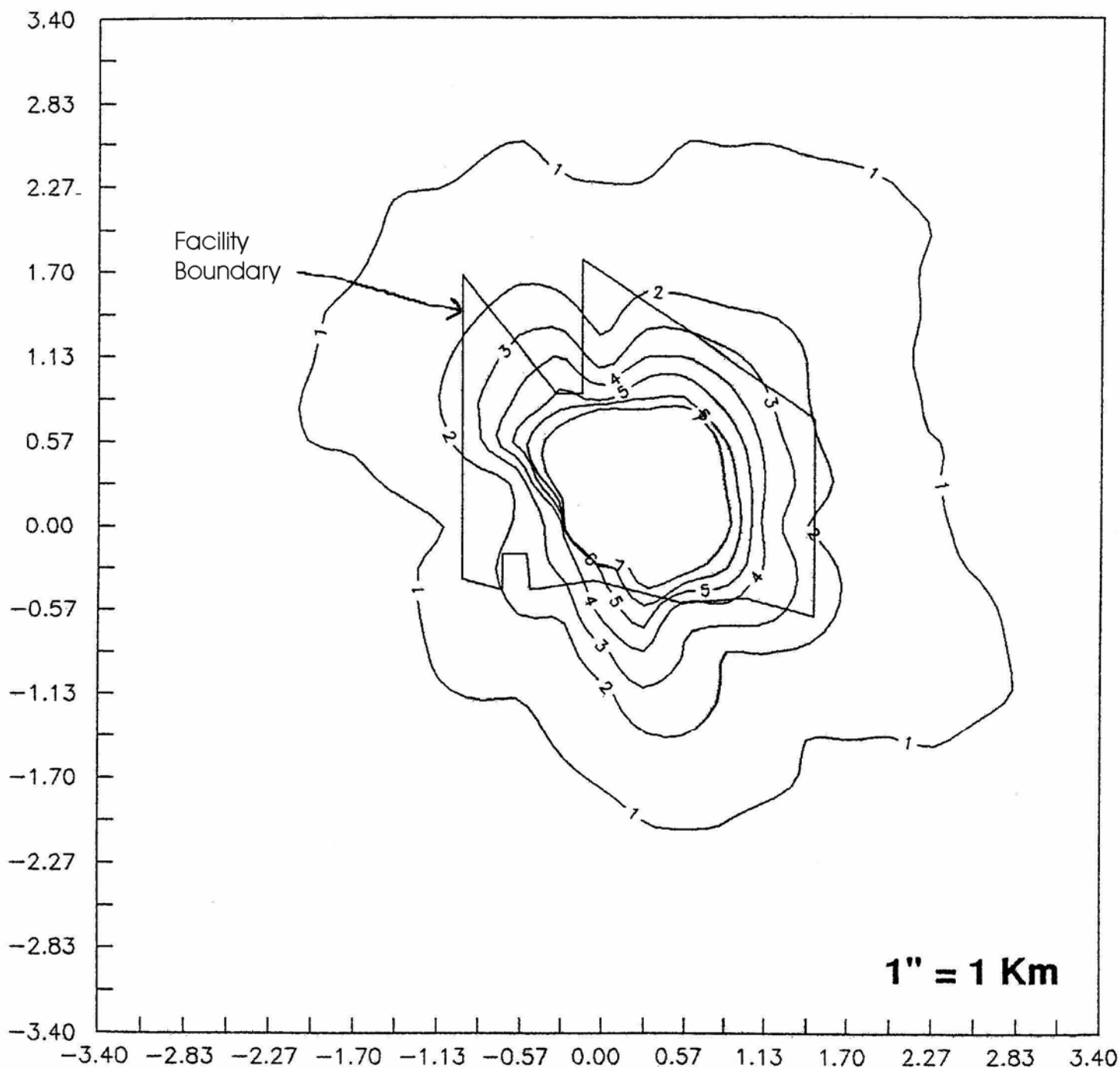


Figure 9b
3M CHEMOLITE BLDG 15
1986 2nd Highest 24-Hour Average
HF Concentration ($\mu\text{g}/\text{m}^3$)
Urban Mode, Elevated Terrain



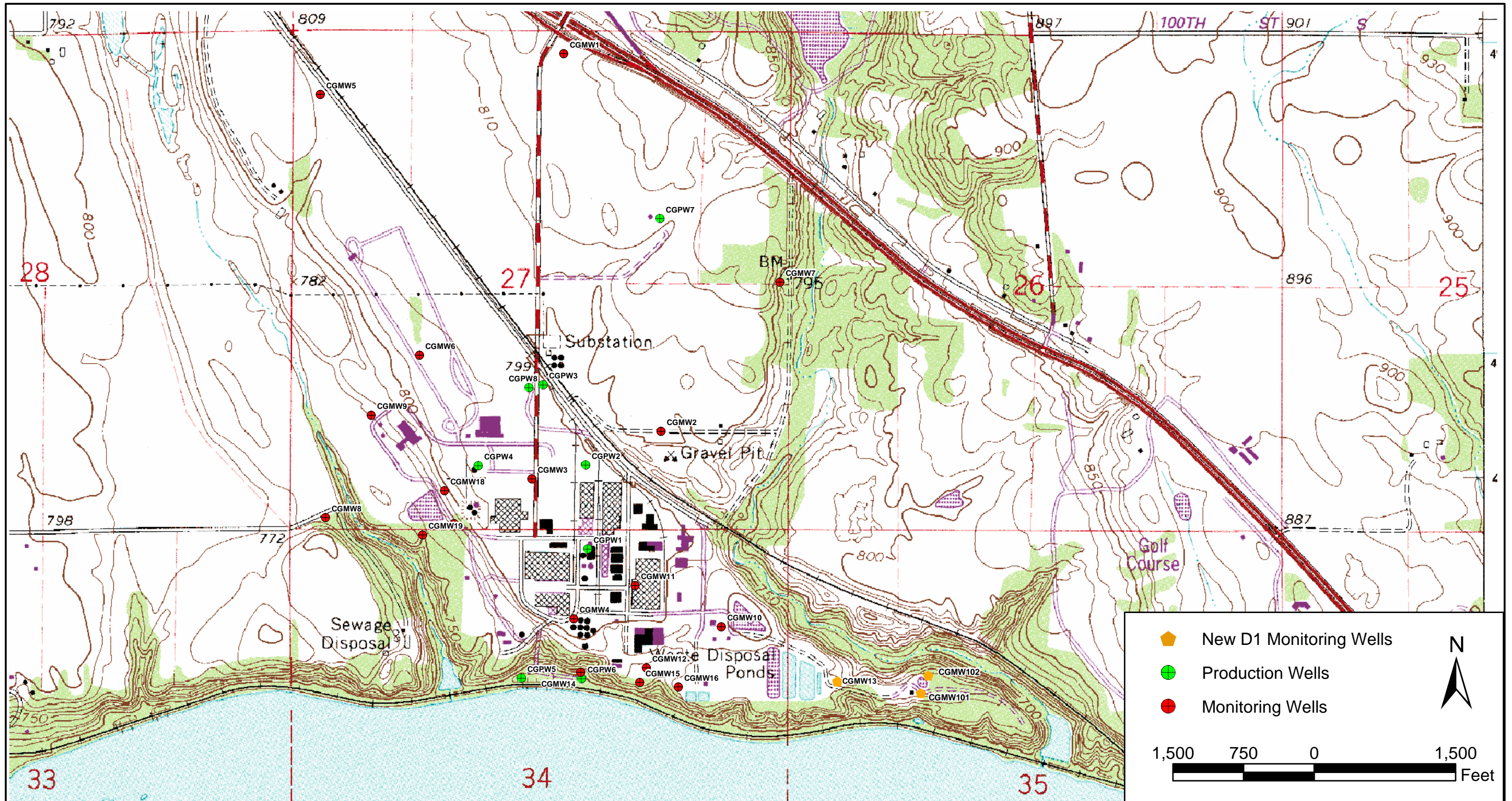


Figure10
Well Locations
 3M - Cottage Grove Facility

Source: ERG 2004

DATE: 7/2/02
 REVISED: 09/24/2003
 SCALE: 1:12,000
 DRAWN BY: M. Bless
 H:\Clients\3M_CottageGrove\MMM2002-110\2002\ArcGIS\CG_Well.ocs

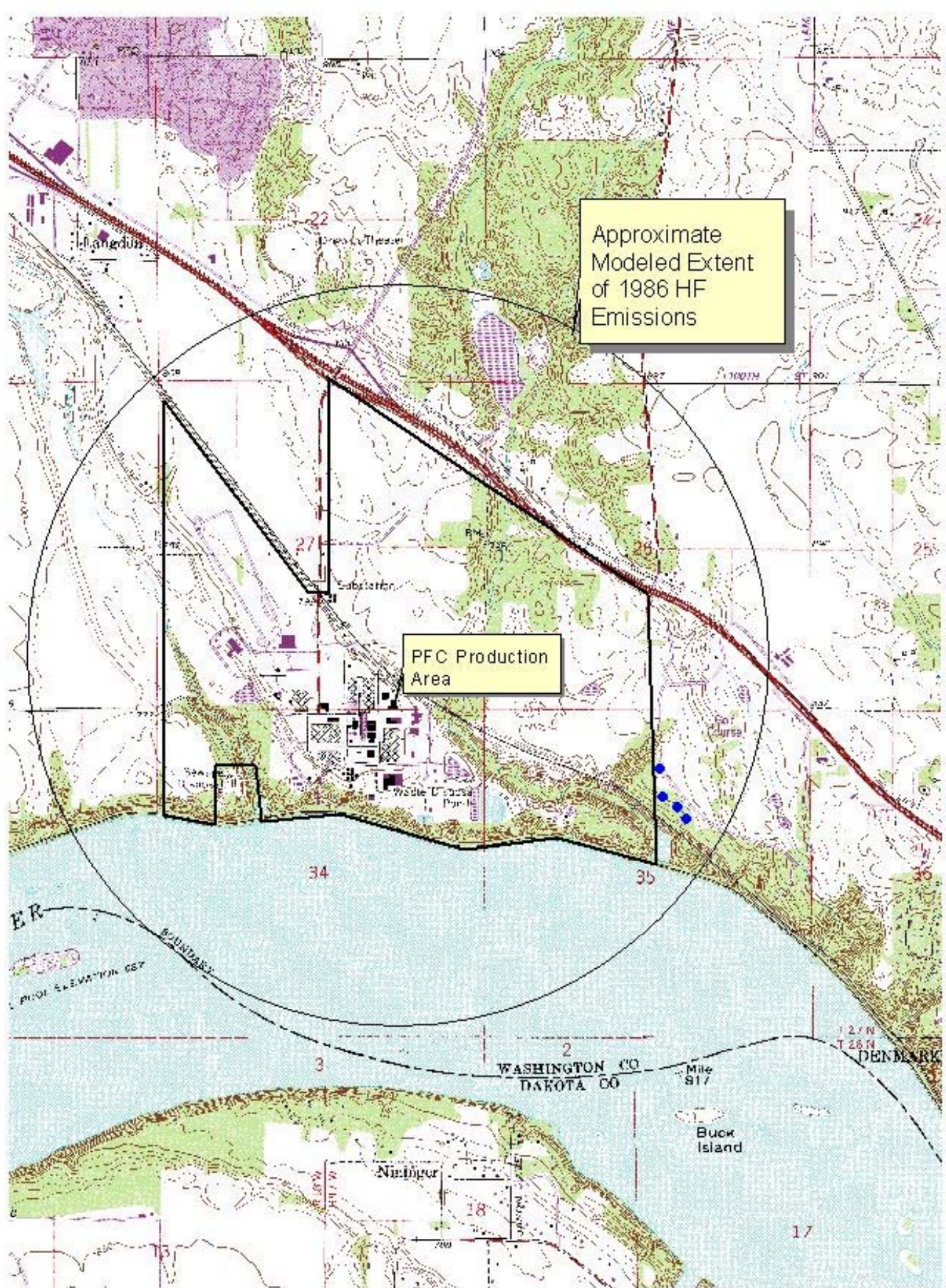


Fig. 11: Private Well Sample Locations



● = Private Well Sample, 2003

0 0.10.20.30.40.5 Miles



Appendix 1

Derivation of MDH Health-Based Values and
Soil Reference Values for PFOS and PFOA

Memo



Date: November 20, 2002
To: Douglas Wetzstein
Dave Douglas
From: Helen Goeden, Health Risk Assessment Unit
Phone: (651) 215-0874
Subject: Response to Request for Health Based Values and interim Soil Reference Values

This memorandum is in response to a request by the Minnesota Pollution Control Agency (08/21/02) for Health Based Values (HBVs) and interim Soil Reference Values (SRVs) for perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS).

There is limited published information on the toxicity of PFOA and PFOS. The MDH relied heavily on readily available toxicity summary information provided by 3M, EPA and the West Virginia Department of Environmental Protection. After reviewing this information the MDH modified the RfD and RfC values proposed by 3M.

Health Based Values (HBVs)

<u>Chemical</u>	<u>CAS #</u>	<u>Endpoint</u>	<u>RfD</u> (mg/kg/d)	<u>HBV</u> µg/L
PFOA	3825-26-1	Liver	0.001	7
PFOS	2795-39-3/ 1763-23-1	Liver	0.0002	1

Soil Reference Values (SRVs)

<u>Chemical</u>	<u>CAS#</u>	<u>Endpoint</u>	<u>RfD</u> (mg/kg/d)	<u>RfC</u> (mg/m ³)	<u>Residential</u> <u>SRV (mg/kg)</u>	<u>Industrial</u> <u>SRV (mg/kg)</u>
PFOA	3825-26-1	Liver	0.001	2E-5	30	200
PFOS	2795-39-3/ 1763-23-1	Liver	0.0002	2E-5	6	40

Toxicity Value Sources: See Attachment II.

Based on information currently available we feel that the above values will provide an adequate level of protection from exposure to PFOA and PFOS in drinking water and direct exposure to PFOA or PFOS in soil; however, there is a degree of uncertainty associated with the HBVs and SRVs, and they should be considered provisional. The above criteria do not address impacts to groundwater as a result of soil leaching, food chain impacts or ecological impacts.

Please note that carcinogenicity studies in the rat have shown PFOA and PFOS to be potentially carcinogenic. However, at this time the available data are not sufficient to determine relevance to humans or for development of cancer potency values.

The data utilized in the derivation of the HBVs is provided in Attachment I. Standard assumptions of a 70 kilogram person with a drinking water ingestion rate of 2 liters per day, and a relative source contribution of 20 percent were used to calculate these values.

MDH is in the process of revising its Health Risk Limits for groundwater rule. The MDH is likely to recommend that the standard assumptions of 70 kilograms and 2 liters/day be replaced by a body weight and an intake rate more appropriate for children. If this recommendation is accepted and promulgated as rule, HBVs would likely decrease by a factor of 3 to 4.

The data utilized in the derivation of the SRVs is provided in Attachment II. The default exposure scenarios and target risk values presented in the MPCA's Draft Guidelines for the Soil-Human Health Pathway, Technical Support Document (Working Draft, January 1999) were utilized to calculate these values.

The MDH's authority to promulgate health risk limits under the Groundwater Protection Act is limited to situations where degradation has already occurred. Similarly, the HBVs and SRVs provided are intended to serve as interim advice issued for specific sites where a contaminant has been detected. As such, neither the HBVs nor SRVs are developed for the purpose of providing an upper limit for degradation.

cc: Larry Gust, MDH
Anne Kukowski, MDH
Jim Kelly, MDH
Gerry Smith, MDH
Shelley Burman, MPCA
Luke Charpentier, MPCA
Mary Dymond, MPCA
Laura Solem, MPCA
Michael Santoro, 3M
John Butenhoff, 3M

ATTACHMENT I

DATA FOR DERIVATION OF GROUND WATER HEALTH BASED VALUE (HBV)

Compound Name: **Perfluorooctanoate (PFOA)**
CAS #: **3825-26-1** (Oct. 16, 2002 personal communication with Dr. John Butenhoff, 3M)

LOAEL (*ingestion*): **3 mg/kg/day**
Uncertainty Factor: **3000** (3 - interspecies; 10 - intraspecies; 10 subchronic-to-chronic; 10 LOAEL-to-NOAEL)

Modifying Factor: **1**
RfD*: **0.001 mg/kg/day**

Health effect: **Liver**

Relative Source Contribution (RSC): **20%**

Oral Slope Factor: **NA**
Applied Risk Level: **NA**

$$\begin{aligned} \text{HBV} &= \frac{(\text{RfD, mg/kg/d}) (\text{RSC}) (1000 \text{ } \mu\text{g/mg})}{\text{Intake Rate (2 L per day/70 kg)}} \\ &= \frac{(0.001 \text{ mg/kg/d}) (0.2) (1000 \text{ } \mu\text{g/mg})}{0.029 \text{ L/kg/d}} = 7 \text{ } \mu\text{g/L} \end{aligned}$$

Data Sources:

1. EPA Revised Draft Hazard Assessment of Perfluorooctanoic Acid and Its Salts (Nov 4, 2002);
2. EPA Draft Hazard Assessment of Perfluorooctanoic Acid and Its Salts (Feb 2002);
3. 3M Lifetime Drinking Water Health Advisory for Perfluorooctane sulfonate (April 2002);
4. 3M Soil Screening Guidelines for PFOS (May 2002);
5. Subchronic Toxicity Studies on Perfluorooctanesulfonate Potassium Salt in Cynomolgus Monkeys. Seacat et al., Toxicological Sciences 68:249-264, 2002; and
6. 3M Soil Screening Guidelines for PFOA (March 2002).

* Carcinogenicity studies in the rat have shown PFOA to be carcinogenic. However, at this time the available data are not sufficient for a quantitative assessment. Reproductive and developmental effects, based on studies in rats and rabbits, occur at levels higher than doses causing liver toxicity. However, due to rapid elimination in female rats (serum half-life of 1 day) it is unclear to what degree the fetuses and neonates were exposed. Ovarian tubular hyperplasia has also been observed in female rats at doses as low as 1.6 mg/kg/d (note: a NOAEL was not determined for this effect since effects were observed at the lowest dose evaluated). Women do not appear to have the same active secretory mechanism that exists in the female rat.

Compound Name: **Perfluorooctanesulfonate (PFOS)**
CAS #: **2795-39-3 (potassium salt)**
1763-23-1 (free salt)
(Oct. 16, 2002 personal communication with Dr. John Butenhoff, 3M)

LOAEL (*ingestion*): **0.15 mg/kg/day**
Uncertainty Factor: **1000** (3 - interspecies; 10 - intraspecies; 10 subchronic-to-chronic; 3 LOAEL-to-NOAEL)

Modifying Factor: **1**
RfD*: **0.0002 mg/kg/day**

Health effect: **Liver**

Relative Source Contribution (RSC): **20%**

Oral Slope Factor: **NA**
Applied Risk Level: **NA**

$$\begin{aligned} \text{HBV} &= \frac{(\text{RfD, mg/kg/d}) (\text{RSC}) (1000 \mu\text{g/mg})}{\text{Intake Rate (2 L per day/70 kg)}} \\ &= \frac{(0.0002 \text{ mg/kg/d}) (0.2) (1000 \mu\text{g/mg})}{0.029 \text{ L/kg/d}} = \mathbf{1 \mu\text{g/L}} \end{aligned}$$

Data Sources:

- 1) EPA Hazard Assessment and Biomonitoring Data on Perfluorooctane Sulfonate – PFOS (July 2000);
- 2) 3M Lifetime Drinking Water Health Advisory for Perfluorooctane sulfonate (April 2002);
- 3) 3M Soil Screening Guidelines for PFOS (May 2002);
- 4) Subchronic Toxicity Studies on Perfluorooctanesulfonate Potassium Salt in Cynomolgus Monkeys. Seacat et al., *Toxicological Sciences* 68:249-264, 2002; and
- 5) 3M Comments on Interspecies Uncertainty in Risk Assessment for PFOS.

*Carcinogenicity studies in the rat have shown PFOS to be carcinogenic. However, at this time the available data are not sufficient for a quantitative assessment. Reproductive and developmental effects, based on studies in rats and rabbits, occur at levels higher than doses causing liver toxicity.

Date (Prepared or Modified): November 14, 2002
Prepared by: H. Goeden

ATTACHMENT II

DATA FOR DERIVATION OF SOIL REFERENCE VALUE (SRV)

Compound Name:	Perfluorooctanoate (PFOA)
CAS #:	3825-26-1 (Oct. 16, 2002 personal communication with Dr. John Butenhoff, 3M)
LOAEL (<i>ingestion</i>):	3 mg/kg/day
Uncertainty Factor:	3000 (3 - interspecies; 10 - intraspecies; 10 subchronic-to-chronic; 10 LOAEL-to-NOAEL)
Modifying Factor:	1
RfD*:	0.001 mg/kg/day
RfC**:	2E-5 mg/m³
Dermal Absorption:	10% (MPCA Default for organic compounds)
Health effect:	Liver
Hazard Quotient:	0.2 (MPCA target risk value)
Oral Slope Factor:	NA
Inhalation Unit Risk:	NA
Residential SRV:	30 mg/kg
Industrial SRV:	200 mg/kg

Data Sources:

- 1) EPA Revised Draft Hazard Assessment of Perfluorooctanoic Acid and Its Salts (Nov 4, 2002);
- 2) EPA Draft Hazard Assessment of Perfluorooctanoic Acid and Its Salts (Feb 2002);
- 3) 3M Lifetime Drinking Water Health Advisory for Perfluorooctane sulfonate (April 2002);
- 4) 3M Soil Screening Guidelines for PFOS (May 2002);
- 5) Subchronic Toxicity Studies on Perfluorooctanesulfonate Potassium Salt in Cynomolgus Monkeys. Seacat et al., *Toxicological Sciences* 68:249-264, 2002; and
- 6) 3M Soil Screening Guidelines for PFOA (March 2002).

* Carcinogenicity studies in the rat have shown PFOA to be carcinogenic. However, at this time the available data are not sufficient for a quantitative assessment. Reproductive and developmental effects, based on studies in rats and rabbits, occur at levels higher than doses causing liver toxicity. However, due to rapid elimination in female rats (serum half-life of 1 day) it is unclear to what degree the fetuses and neonates were exposed. Ovarian tubular hyperplasia has also been observed in female rats at doses as low as 1.6 mg/kg/d (note: a NOAEL was not determined for this effect since effects were observed at the lowest dose evaluated). Women do not appear to have the same active secretory mechanism that exists in the female rat.

** There is insufficient information on the toxicological effects of PFOA following inhalation exposure. PFOA is not considered to be a volatile chemical and therefore the inhalation exposure pathway is anticipated to be a minor pathway. 3M has suggested a RfC of 2E-5 mg/m³ based on a generic exposure guideline for chemicals found to be carcinogenic in animals but with unknown relevance to humans. The CATT report generated a RfC of 1.1E-3 mg/m³. In the absence of information the provisional RfC suggested by 3M will be utilized for the development of an interim Soil Reference Value.

Compound Name: **Perfluorooctanesulfonate (PFOS)**
CAS #: **2795-39-3 (potassium salt)**
1763-23-1 (free salt)
(Oct. 16, 2002 personal communication with Dr. John Butenhoff, 3M)

LOAEL (*ingestion*): **0.15 mg/kg/day**
Uncertainty Factor: **1000** (3 - interspecies; 10 - intraspecies; 10 subchronic-to-chronic; 3 LOAEL-to-NOAEL)
Modifying Factor: **1**
RfD*: **0.0002 mg/kg/day**

RfC**:

2E-5 mg/m³

Dermal Absorption: **10% (MPCA Default for organic compounds)**

Health effect: **Liver**

Hazard Quotient: **0.2 (MPCA target risk value)**

Oral Slope Factor: **NA**

Inhalation Unit Risk: **NA**

Residential SRV: **6 mg/kg**

Industrial SRV: **40 mg/kg**

Data Sources:

Data Sources:

- 1) EPA Hazard Assessment and Biomonitoring Data on Perfluorooctane Sulfonate – PFOS (July 2000);
- 2) 3M Lifetime Drinking Water Health Advisory for Perfluorooctane sulfonate (April 2002);
- 3) 3M Soil Screening Guidelines for PFOS (May 2002);
- 4) Subchronic Toxicity Studies on Perfluorooctanesulfonate Potassium Salt in Cynomolgus Monkeys. Seacat et al., *Toxicological Sciences* 68:249-264, 2002; and
- 5) 3M Comments on Interspecies Uncertainty in Risk Assessment for PFOS.

*Carcinogenicity studies in the rat have shown PFOS to be carcinogenic. However, at this time the available data are not sufficient for a quantitative assessment. Reproductive and developmental effects, based on studies in rats and rabbits, occur at levels higher than doses causing liver toxicity.

**There is insufficient information on the toxicological effects of PFOS following inhalation exposure. PFOS is not considered to be a volatile chemical and therefore the inhalation exposure pathway is anticipated to be a minor pathway. 3M suggested a RfCs of 2E-4 and 2E-5 mg/m³ for PFOS and PFOA, respectively. The value for PFOA was based on a generic exposure guideline for chemicals found to be carcinogenic in animals but with unknown relevance to humans. PFOS appears to be carcinogenic in rats but it is not clear whether suggested mechanism of action is relevant to humans. In the absence of information the provisional RfC for PFOA (2E-5 mg/m³) suggested by 3M will be utilized for the development of an interim Soil Reference Value for PFOS as well.

Date (Prepared or Modified): November 14, 2002

Prepared by: H. Goeden

Appendix 2

Public Comments Received



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C., 20460

OFFICE OF
PREVENTION, PESTICIDES AND
TOXIC SUBSTANCES

September 1, 2004

VIA E-MAIL

Mr. James Kelly
Environmental Health Division
Minnesota Department of Health
james.kelly@health.state.mn.us

Dear Mr. Kelly:

Thank you for the opportunity to provide comments on the draft Public Health Consultation for the 3M Cottage Grove Facility. The Agency has a number of general comments, and also some specific points. General comments are addressed first.

General Comments

EPA has not reviewed the document for toxicological accuracy, but has several overall comments. First, EPA understands the need for toxicological values to quantitatively assess potential health risks of perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS). However, use of the terms "RfD" and "RfC" throughout the documents attached in Appendix 1 implies that these are EPA-derived values, and have been subjected to the vigorous peer review that EPA requires prior to their release on the Integrated Risk Information System (IRIS). The values presented by the Minnesota Department of Health (MDH) are not EPA-calculated values. EPA therefore requests that MDH call these values something other than RfDs or RfCs, or at a minimum make clear that these proposed values were not derived by EPA nor produced through the IRIS process.

Second, MDH cites EPA draft documents (2000-2002) and a preliminary EPA risk assessment on PFOA (2003) as sources for their analyses. MDH should be aware that there have been rapid advances in the pharmacokinetics and toxicology of PFOA and PFOS. In addition, there have been recent analyses of the mode of action of the liver toxicity and tumor findings in rodents, and the possible relevance of this mode of action for humans. This new information may have implications for the quantitative analysis conducted by MDH.

Finally, EPA is in the process of finishing a draft risk assessment of PFOA that will be reviewed by its Science Advisory Board (SAB) in late 2004. This draft risk assessment will become available to the public when it is submitted to the SAB, and will be posted on both the SAB website (www.epa.gov/sab/) and on the EPA's PFOA webpage (www.epa.gov/oppt/pfoa/). IRIS assessments of PFOA and PFOS are also being prepared, but will not be complete until after the PFOA SAB review.

Specific Comments

Page 3:

The summary and the later detailed section both note that 3M had phased out the production of perfluorochemicals (PFCs) at the Cottage Grove site. What is not clear is whether any other PFC-related activities still continue at the facility, such as handling, use, processing, or packaging, and whether there may still be releases of PFCs associated with those activities at the facility. Additional clarity would be useful.

Page 3 and Page 16:

The document presents assumptions about the behavior of PFCs in air and soil based on the structure of the chemicals and very limited data. Clearly identifying the assumptions made and the limitations of the available data would help to prevent the reader from ascribing certainty to these assumptions.

The Agency would also be very interested in reviewing the unpublished report from Franklin (2002) cited on page 16, concerning estimating the potential for long-range transport of PFOA released to air.

Page 4 and Page 21:

The summary references one of the ranges of margins of exposure calculated in the EPA's preliminary risk assessment on PFOA based on developmental effects data in animal studies and measured human PFOA serum levels. If this range is used, it should be specifically identified as a preliminary figure, and the caveats on the use of the range described in the assessment document need to accompany the range.

Page 13:

In the discussion of air emissions, deposition to soil, and sampling off-site wells, it should be noted that the absence of detection of PFOA or PFOS in the four deep wells sampled does not resolve the question of whether surface deposition has occurred.

Page 22:

The paragraph at the bottom of the page incorrectly characterizes the EPA's ongoing enforceable consent agreement (ECA) process. The Agency does not have an ECA with manufacturers at this time for the information MDH has described. To more accurately capture the PFOA ECA process, the Agency would suggest the following changes to the existing language, shown in redline for additions and ~~strikeout~~ for deletions:

The U.S. EPA's Office of Pollution Prevention and Toxics, through an enforceable consent agreement (ECA) process undertaken with various manufacturers and users of PFCs (including 3M) and other interested parties, has been studying the extent, distribution, and fate of PFCs (primarily PFOA) in the environment associated with the manufacture, use, or disposal of PFCs or PFC containing products. All documents related to this undertaking are posted and available on an EPA web site (www.epa.gov/edocket/) (~~http~~ ~~://cascade.epa.gov/RightSite/dk_public_home.htm~~) under docket number OPPT-2003-0012.

In this ECA process, EPA identified several needs for monitoring information, including monitoring in the vicinity of facilities currently manufacturing, processing, and using various PFCs. Three companies – 3M, Dyneon (a 3M company), and DuPont – participating in this process have indicated a willingness to enter into Memoranda of Understanding (MOUs) with the Agency for monitoring on and around their respective fluoropolymer manufacturing facilities located in Decatur, Alabama and Washington, West Virginia. These MOUs are currently under negotiation. A fourth company, Daikin America, is undertaking an independent, voluntary monitoring program at its fluoropolymer manufacturing facility, which is co-located with the 3M/Dyneon plant in Decatur, Alabama. Under the ECA monitoring plans have been developed to assess the impact of previous PFC operations, waste disposal practices, and PFC manufacturing at several locations where PFCs have been or will continue to be produced. These sites include the 3M plant in Decatur, Alabama, and two facilities co-located at the Decatur site (Dyneon (a 3M company) and Daikin America), as well as DuPont's large facility in Parkersburg, West Virginia. The 3M Cottage Grove facility has not been included in this effort to date because it is no longer producing PFOA on a commercial basis (M.F. Dominiak, U.S. EPA, personal communication, 2004). The phased approach monitoring plan proposed by 3M for the 3M/Dyneon plant in Decatur, Alabama involves the following (in no particular order; Weston 2004):

Page 24:

Section V., item 1., should be corrected to note that the MOU for voluntary monitoring at the 3M/Dyneon facility in Decatur, Alabama is still under development. The current sentence should be amended as follows:

Consideration should be given to developing and implementing (using a phased approach if necessary) a scope of investigation work similar to that developed by 3M for the Decatur, Alabama facility under their proposed voluntary agreement with the U.S. EPA (see pages ~~20-21~~22-23).

The Agency has not commented on the toxicological accuracy of the report or on the hazard and risk conclusions drawn by the MDH because EPA's own risk assessment activities on the PFCs are still in progress.

However, EPA concurs with MDH that additional monitoring information concerning the Cottage Grove facility would be valuable in helping to understand the sources, pathways of exposure, and behavior of PFCs in the environment.

If you have any questions concerning these comments, please contact Mary Dominiak of my staff by email at dominiak.mary@epa.gov, or by telephone at 202-564-8104.

Sincerely,

/s/

Charles M. Auer, Director
Office of Pollution Prevention and Toxics

Office Memorandum

DATE: August 12 , 2004

TO: Jim Kelly, Minnesota Department of Health

FROM: David Douglas, Project Manager
Superfund Unit 2/Superfund Section
Superfund Section
Majors and Remediation Division

PHONE: 296-7818

SUBJECT: 3M Chemolite/Health Consultation

This memorandum is written in response to the Public Comment Release draft of the Health Consultation for the 3M Cottage Grove Facility, dated June 24, 2004. Thank you for considering Minnesota Pollution Control Agency (MPCA staff) comments to the previous draft of this document. The following are additional MPCA staff comments to the June 24th draft or clarifications of previous MPCA staff comments.

Summary, page 3, first paragraph

From previous 3M briefings to MPCA and MDH staff, it is the MPCA staff's understanding that 3M continues to manufacture and/or test eight-carbon perfluorochemical (PFC) Scotchguard fire-fighting foam at the facility. If MDH has not verified the status of this situation, the MPCA staff suggests that the MDH request that 3M identify the chemical formula of the fire-fighting foam tested at the facility and its status regarding manufacture and testing at the facility.

Summary, page 4, last paragraph, last sentence

The MPCA staff understands that this statement is related to classifications for evaluating risk as specified by the Agency for Toxic Substances and Disease Registry (ATSDR). However, as cited in Appendix 1, the MDH has developed Health-Based Values and Soil Reference Values for PFOS and PFOA. 3M as found PFOS and PFOA in some pumpout wells, some of which have been used as facility drinking water wells (see Table 1) and in ground water near Site D1 at levels that exceed their respective HBVs. It is the MPCA staff's understanding from 3M briefings that 3M employees have consumed facility drinking water exceeding their respective HBVs. As a result, for some time, 3M has provided bottled drinking water to its facility employees. The MPCA staff has classified PFOS and PFOA as MERLA hazardous substances and considers ingestion of these chemicals at levels above their respective HBVs to represent unacceptable risks. In this context, and for the record, the MPCA staff is concerned that these actual human exposures from contaminated facility drinking water represent unacceptable human exposures to these PFCs and that these exposures do not represent an "indeterminate public health hazard."

Superfund Site History, page 7

The MPCA staff requests that narrative be added here or elsewhere in the document (if this is not the appropriate place) that captures the following:

- the remedial investigation and remedial actions cited in this section did not focus on PFCs in any medium;



- a consent order addendum is being negotiated to modify the scope of the remedial investigation and remedial actions to focus on PFCs in all media at the facility and in all media where PFCs were or could have been released;
- these sites are related to the old consent order which merely refers to the disposal of “neutralized hydrofluoric tars;” and
- analytical methods to distinguish individual PFCs were not available at the time that the consent order was executed.

Site D4: Phenolic Waste Pit, page 8

The MPCA staff had previously commented on the possibility of PFC vapor intrusion in Building 26. It does not appear that MDH addressed this comment in the document. If MDH believes that vapor intrusion of this building is not an issue (MDH notes that the volatility of PFOS is “essentially non-volatile” in the first paragraph of Section III. Discussion), then the MPCA staff recommends that this reasoning be articulated in the document.

Areas of PFC Production and Use, page 10, first complete paragraph

Does MDH believe the release of PFCs to the atmosphere represents a threat to public health?

PFC Monitoring at the Site, page 13, first complete paragraph

Don Kriens of the MPCA staff has been contacted about the possibility of PFCs being in the effluent of Metropolitan Council’s Eagle Point Waste Water Treatment Facility. The MPCA staff will keep MDH informed about the outcome of any efforts to determine if PFCs are in this facility’s effluent.

Please call me at (651) 296-7818 if you have any questions concerning this memorandum.



**Department of Public
Health and Environment**

Mary L. McGlothlin
Director

Lowell Johnson
Deputy Director

August 17, 2004

James Kelly
Minnesota Department of Health
Site Assessment and Consultation Unit
121 East 7th Pl STE 220
PO Box 64975
Saint Paul MN 55164-0975

RE: Health Consultation - 3M Cottage Grove Facility (aka 3M Chemolite)

Thank you for the opportunity to comment on the Health Consultation for the 3M Cottage Grove Site, prepared by the Site Assessment and Consultation Unit of the Minnesota Department of Health.

Prior to finalizing the County's comments, Mary McGlothlin and I met with Fred Luden, 3M Director of Operations and Michael Santoro, 3M Director of Environmental, Health, Safety and Regulatory Affairs.

The majority of the County's comments relate to the release of Perfluorochemicals (PFCs). Comment 6 and comment 8 also address volatile organic chemicals (VOCs).

Our comments are as follows:

1. 3M should model the historical air emissions of PFCs to accurately determine possible contamination off-site (last modeled in 1991). Based on results from the air emission model, the soil and groundwater in these off-site areas should be tested for possible contamination.
2. 3M should identify the extent of contamination in groundwater from other releases on the property, including the accidental release from Bldg 15, discovered during sewer pipe replacement, and from the various dump sites. 3M should install barrier and/or source pump out wells to prevent contamination from moving off-site.
3. 3M should install additional monitoring wells to fully characterize the extent and magnitude of contamination, including monitoring wells in the plume. If additional monitoring wells are already in existence, their location, depth and PFC levels should be noted in the Health Consultation.
4. 3M should develop a water model to integrate groundwater and surface water flow, incorporating the findings of Mossler (2003) and Barr Engineering (2003) referenced in the Health Assessment. According to the Health Assessment, the source of the current 3M model is unknown, and the data and assumptions upon which it was created are also not known.

5. 3M should gain a better understanding of the fate of PFCs discharged to the Mississippi River, including bioaccumulation and/or biomagnification in fish, persistence in bottom sediments, etc.
6. In addition to PFCs, there are a number of releases of VOCs referenced in the document. The impact of these releases should be fully characterized by 3M.
7. 3M should coordinate a round of groundwater sampling of all monitoring wells and production wells to better understand the extent of groundwater contamination and extent of PFC exposure from ingestion of drinking water to workers.
8. After treatment ponds are abandoned, 3M should test the pond sediment for VOCs and PFCs, and remove any contaminated soil.
9. The location of other disposal sites should be disclosed by 3M. The sites identified by 3M should be assessed for impact to the environment. (e.g. PFCs are found in groundwater samples in the Lake Jane Landfill area)
10. Concentrations of PFOs and PFOAs are significantly above the Minnesota Department of Health health based values (HBVs). The County is concerned about long term health effects to 3M employees and the fate of the PFCs in the various media (air, water, soil, biota, humans).
11. Based on the abbreviated summary of toxicological and epidemiological studies in the Health Consultation, it appears there are a number of possible health outcomes, including cancer, death, reproductive and developmental effects, interference with cholesterol metabolism, etc. Workers have historically been exposed both on the job and by ingesting contaminated drinking water. 3M should ensure that all workers are drinking water free of PFCs and VOCs.

If you have any questions regarding the above comments, please contact me at 651-430-6703.

Sincerely,

Cindy Weckwerth, REHS, MS
Program Manager

C: Myra Peterson, County Commissioner
Jim Schug, County Administrator
Mary McGlothlin, Department Director
Fred Luden, Director, 3M
Michael Santoro, Director, 3M Environment, Health, Safety and Regulatory
Affairs



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August 16, 2004

Mr. James Kelly
Site Assessment and Consultation Unit
Minnesota Department of Health
121 East Seventh Place
P.O. Box 64975
St. Paul, MN 55164-0975

RE: Public Health Consultation – 3M Cottage Grove Facility

Dear Mr. Kelly:

The City of Cottage Grove has reviewed the public health study of perfluorochemicals (PFCs) at the 3M Cottage Grove Facility. We understand that monitoring the impacts of PFCs and other substances present at the site is the responsibility of the Minnesota Pollution Control Agency and the Minnesota Department of Health.

The study indicates that there are no known immediate health risks for the larger community from past discharges at the Cottage Grove facility. This includes no known contamination of wells in the area surrounding the 3M facility. The City does support the recommendations included in the report, particularly the need for continued monitoring of potential health impacts from PFCs at the site.

Thank you for the opportunity to comment on the report. We would appreciate being notified of the results of future studies on the 3M Cottage Grove Facility.

Sincerely,

A handwritten signature in black ink that reads "Howard Blin".

Howard Blin
Community Development Director

cc: Mayor and City Council
Ryan Schroeder, City Administrator

EQUAL OPPORTUNITY EMPLOYER

August 20, 2004



Mr. James Kelly
Site Assessment and Consultation Unit
Environmental Health Division
Minnesota Department of Health

Via E-Mail: james.kelly@health.state.mn.us

Re: 3M Cottage Grove, MN Consultation

Dear Mr. Kelly:

3M appreciates the opportunity to comment on the Minnesota Health Department's draft consultation report. As you know, 3M has been working and continues to work actively with the Minnesota Pollution Control Agency (MPCA) and Health Department to address issues at the Cottage Grove site. We have carefully reviewed the draft consultation report. We very much appreciate the Department's efforts to understand the extensive database on fluorochemicals, and would like to 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 as possible. Once you have had an opportunity to review these comments, 3M would like to meet with you and your colleagues in order to respond to any questions you may have

COMMENTS ON THE CONCLUSIONS

The stated conclusions of the draft consultation report suggest there is a "lack of available information" in a number of areas. We believe this is an overly broad statement which fails to take into account the totality of the scientific information regarding fluorochemicals.

- Although the document states that it addresses only the Cottage Grove site, we are concerned by the sweeping statements in the conclusions on page 24 regarding a lack of understanding of fluorochemical toxicity and general population exposure. Exposures to the general population have been characterized, and the use of serum concentration data to reflect exposure from all pathways reduces the uncertainty typically found in exposure assessment. 3M has monitored its workers -- the most highly exposed population -- for over 25 years, and found no causal relationship between fluorochemical exposure and adverse clinical findings, despite serum concentrations two to three or more orders of magnitude above the general population. The epidemiologic data do not suggest any adverse effects on the general population from fluorochemicals. The toxicological

database on PFOA and PFOS is comprehensive, and forms the basis of robust, independently-reviewed risk assessments for both PFOA and PFOS. With respect to PFOA, see Butenhoff et al., "Characterization of Risk for General Population Exposure to Perfluorooctanoate," *Regulatory Toxicology & Pharmacology* 39:363-380 (2004), and for PFOS, see "Environmental and Health Assessment for Perfluorooctane Sulfonic Acid and its Salts," August 20, 2003 (3M, 2003). As reported there, margins of exposure for the 95th percentile general population serum levels of both PFOA and PFOS are substantial. These risk assessments provide a science-based analysis of all the data, and should provide a level of assurance as to the lack of potential impact on the general population.

- The conclusions on page 24 further state that there are limited environmental data available and thus the potential impact on public health from releases at the Cottage Grove facility cannot be assessed at this time. The statement that "the site currently represents an indeterminate public health hazard" is overly broad given the data available.
 - Data can always be said to be limited, but 3M has obtained substantial information about the geology and hydrogeology at the site and the effectiveness of the on-site well pumping system to control off-site movement of groundwater, and considerable data on the presence of PFOS and PFOA at the site and the physical and chemical characteristics of these substances. This information has been shared with MPCA.
 - There is no evidence of fluorochemicals in nearby offsite wells, and 3M has for decades operated production wells which create a cone of depression for groundwater emanating from the developed portion of the property. At this time, there is no indication that groundwater migration from the plant is a completed exposure pathway.
 - Furthermore, the production of PFOS- and PFOA-related substances was discontinued as of December 2002, thus reducing releases from the production processes. The activated carbon treatment system for plant wastewater discharges mentioned in the draft consultation report is fully operational.

COMMENTS ON THE RECOMMENDATIONS

The draft consultation report recommends a number of steps that 3M has already initiated.

- While a significant body of data has already been submitted to the MPCA, 3M has agreed to obtain additional data at the site. 3M supports a phased approach to investigation at the site, and last fall submitted to MPCA an aggressive timeline for the investigation of fluorochemicals at the site -- including the coordinated groundwater sampling the draft report recommends. While we do not believe the approach will mirror precisely the

activities at 3M's Decatur site (given the different current and historical operations, physical settings, remediation activities, and different regulatory contexts), 3M is committed to further investigation and to appropriate actions

- With regard to the recommendation that 3M should take action to ensure that Cottage Grove workers are not exposed to fluorochemicals via the water supply at the facility in excess of Health-Based Values,¹ the document should acknowledge that 3M has already taken steps to provide bottled drinking water to workers. Contrary to the statement on page 13, bottled water is used for drinking water and cooking, and the plant is in the process of installing a treatment system for water used in cooking, so that the kitchen need not rely on bottled water.
- Similarly, 3M will continue to take steps to identify and as appropriate reduce any potential ongoing discharges from the facility, and requests that the document acknowledge that 3M is already actively engaged in such efforts. The Granular Activated Carbon system referenced on page 13 is fully installed, not merely in the process of being installed, and has shown good removal efficiency (>99%).
- As to the fourth recommendation, to gather information regarding off-site waste disposal locations, 3M supports such a recommendation in the context of the phased investigation. A review of 3M's files with respect to off-site disposal is already underway. The phased approach will address on-site media and then off-site media with confirmed pathways

In sum, 3M brought the fluorochemical issues to MPCA's attention, has provided extensive information, instigated appropriate steps, and proposed and initiated further investigation. 3M will continue to work actively with MPCA and the Health Department.

COMMENTS ON THE TEXT

Summary

Apart from these concerns with the report's conclusions, we have a number of concerns regarding the specifics of the document, which we will address in detail below. To summarize our key specific comments:

- The draft should refrain from speculation or from vague qualitative characterizations such as references to "high levels." Reference to air dispersion modeling for an entirely different chemical is speculative, as it may not be applicable. Similarly, reference to groundwater migration at a fire-training site in Michigan may not be pertinent to hydrogeologic conditions at other locations. Reference to potential

¹ 3M has previously provided the Department of Health with input regarding the conservatism inherent in the Health-Based Values.

exposure to children is also speculative, particularly absent evidence of any completed exposure pathways.

- It would be useful to clarify that not all of the areas described in connection with the 865-acre site are relevant to fluorochemicals. We suggest some specific changes in the description of certain areas of the site and of site geology.
- Several recent reviews provide summaries of environmental, toxicologic and/or epidemiologic data on PFOS and PFOA. We suggest these reviews be referenced
- More recent or detailed information on the half-life of PFOS in animals that differs from the information cited is available. In addition, the description of the chronic studies confuses PFOS and PFOA data.
- The draft report speculates that Minnesota may lower its Health Based Values for PFOS in light of recently published data in Thibodeaux, et al. (2003) and Lau, et al. (2003). We review that new data and explain why it should not result in more stringent health-based levels than the current Minnesota calculation.
- The reference to a possible effect on estradiol in workers is unfounded. We review the data in the cited study and other pertinent studies that were not cited, and explain why we believe the statement is inappropriate. Similarly, we explain why the reference to prostate cancer in the Gilliland and Mandel mortality study is not appropriate unless accompanied by a full explanation that subsequent data do not support an association of PFOA with prostate cancer mortality.
- We provide references for updated information on general population serum levels of PFOS and PFOA. The difference in mean serum levels between the general population and workers engaged in either PFOS or PFOA fluorochemical production is about two orders of magnitude for PFOS and three or more orders of magnitude for PFOA, not one order of magnitude as indicated in the draft report.
- The draft report cites “margins of exposure” -- comparing human general population exposure to benchmark levels from the developmental study of PFOA in rats -- that are taken from a *preliminary* draft EPA document that has since been revised. We explain why the cited margins of exposure are simply incorrect in light of the unique pharmacokinetics affecting the excretion of PFOA in female rats. In the recently published risk assessment for PFOA (Butenhoff, et al. 2004), the authors report margins of exposure for the 95th percentile general population exposure of 2100 for post-natal effects -- a substantial margin of safety.

We elaborate on these and other comments below.

Discussion of Specific Comments

Chemical Terminology

The draft consultation report refers to “PFCs” to encompass fluorochemicals such as PFOA and PFOS. While such an abbreviation seems logical, it could cause confusion, as the term perfluorochemicals also encompasses perfluorinated inerts (fully fluorinated carbon chains that lack a functional end group) that are sometimes called “perfluorocarbons” and abbreviated as PFCs. This is an entirely different category of chemicals from the perfluoroalkylacids formerly produced at the Cottage Grove facility. Accordingly, we suggest reference simply to “fluorochemicals” rather than use of the PFC acronym throughout the document.

Characterizations

In a number of places throughout the document, the text refers to “high” or “significant” levels without appropriate context. (See, e.g., page 3 referring to high levels in groundwater; page 12 referring to high levels and significantly impacted groundwater; page 14 referring to relatively high levels.) These are relative terms. Their import is unclear, and any suggestion of unacceptably high levels is inappropriate in this context. We suggest the document refrain from vague or speculative qualitative characterizations.

Similarly, the document suggests there may be an issue with regard to fluorochemical discharges from the Eagles Point wastewater treatment plant, but provides no foundation for this comment.

The document also includes what appears to be a boilerplate section suggesting children “*could* have been exposed to PFCs from air emissions while PFC production was occurring, and *could* continue to be exposed to soil contaminated from the deposition of PFCs. . . .” (Emphasis added.) It further suggests “[c]hildren *may* also be exposed to PFCs from the site through contaminated surface waters or sediments” (Emphasis added.) If exposure pathways are identified, they will be evaluated and addressed as appropriate. However, absent some indication that there are such completed exposure pathways, such speculation serves no purpose, and should be deleted from the document.

Site Description

The 3M Cottage Grove Facility occupies approximately 865 acres of property in Cottage Grove, Minnesota. Generally, only the southeastern portion of this property has been utilized for manufacturing and development of 3M products. The remaining portion has been used for recreation and farming, or has remained as natural habitat.

As the draft acknowledges, 3M has been cooperating with the MPCA since at least 1985 to investigate and address various areas at the site.² Moreover, 3M has cooperated with the State since the 1960s to permit and address other environmental activities at the site. Thus, a great deal of information is available regarding various areas of the site.

Much of the discussion of the areas addressed under the site remediation activities are not relevant to fluorochemicals (e.g., areas related to an acrylic acid release). While we appreciate the Department's desire to include some background descriptive information given the extent of investigation available, it would be useful to clarify that only some of the areas described in the draft consultation report relate to fluorochemicals. Moreover, as the document indicates, the volatile organic compounds ("VOCs") at the site which have been the focus of a great deal of the investigative and remedial activities to date do not pose a human health concern.

Geology

The comment on page 6 that there are abundant solution cavities in the dolomite geology is unfounded. The dolomite is described as being uplifted, with only the lower portion remaining beneath the site, and the lower portion is acknowledged to be massive, with few solution features. Thus, the probability of solution features beneath the site is low.

The fault line referenced on page 6 is at the outer edge of the cone of groundwater depression, and thus should have little effect on the performance of the site production wells. We have confirmed with the author of the report cited on page 6 that the fault should have minimal influence on the cone of depression. We therefore suggest revising the discussion on page 6.

Six high-capacity pumping wells (installed during the period 1947 to 1970) supply water for manufacturing operations at the site. In general, the pumping of groundwater for on-site use locally alters the north-to-south regional flow direction by inducing inward gradients toward the pumping wells at the Cottage Grove facility. Although historical water level data indicates a natural hydraulic gradient toward the river, pumping of the wells (which started in 1947) has created a cone of depression in the ground water beneath the developed portions of the site. The cone of depression effectively limits movement of ground water from these developed areas to the adjacent river.

² On May 30, 1985, 3M and MPCA entered into a Consent Order to investigate and remediate locations on site utilized formerly for waste disposal. Between 1987 and 2003, numerous monitoring wells and soil borings were installed to evaluate the site and to verify the MPCA approved response actions were effective. All response actions required by the MPCA in the Consent Order were satisfactorily completed as documented in the MPCA's Site Summary Web Page (<http://www.pca.state.mn.us/programs/pubs/plp-2001.pdf>), page 48.

Disposal Areas

The document (pages 3, 5) refers to disposal of fluorochemicals in an on-site “dump.” Later the document clarifies that the disposal location (Area D1) is believed to have been a lined vault, and the materials placed in it were semisolid tars that were neutralized and were not hazardous waste. MPCA approved the closure and management of this disposal site. This should be clarified in the summary as well, and the term “dump” avoided.

In addition, the document refers to both D1 and D2 areas as not having been fully characterized. Both areas have been previously deemed appropriately closed by MPCA, and a source area groundwater investigation for PFOA and PFOS has been completed in the D1 area to the satisfaction of MPCA.

The document on page 8 states that Area D5 showed low levels of VOCs. This is misleading without also pointing out that the area was given closure by MPCA with the acknowledgement that the VOCs were appropriately managed.

Page 8 says Area D6 “was once an active, MPCA-permitted waste disposal area . . .” It still is a permitted waste disposal area, although now inactive.

With regard to Area D8, it is important to note that construction debris was also disposed of in this area; it is inaccurate to suggest this was simply a drum disposal area.

In discussing the chemical sewer lines in the fluorochemical Production Area on page 9, the draft report notes that the previous sewer pipes had been leaking. This statement should be accompanied by information that there are no data suggesting any potential impact to groundwater.

Fire Training Area

Language on page 14 may give the appearance of contradictory information regarding use of the fire training area. The description of testing of fire suppressants at the fire training area in the ERG Work plan (2004) refers to dual uses of these materials at this location. The fire suppressants were used for both fire training exercises for the facility Emergency Management Team and for meeting test requirements established by the Navy to certify the product. 3M received permission annually from the state, starting in the late 1960s, to conduct these operations at the fire training area.

The discussion of the Moody et al. paper regarding groundwater contamination and migration at a military fire-training area in Michigan (pages 16-17 of the draft report) should not be generalized to the Cottage Grove site absent evidence that hydrogeologic and other conditions are comparable. The report should be clear that the migration observed in that study was under the conditions of that particular site.

Air Modeling

While we appreciate that the air dispersion modeling related to hydrogen fluoride (HF) is cited because it is available, we believe the document should caution that HF has very different physical and chemical properties from PFOS, PFOA and related and fluorochemicals, and that the emissions sources and concentrations differed. Accordingly, the predicted HF dispersion may not be concurrent with fluorochemical deposition

At the top of page 15, please clarify the last line to state that the stacks mentioned there were permitted

Well Testing

In addition to the private wells tested by MDH, 3M has also tested an irrigation well on the far northwest portion of 3M's property for fluorochemicals. No fluorochemical compounds were detected.

In the table at the top of page 11 describing on-site monitoring wells, the depths of MW 14, 15, 18 and 19, respectively are 60, 186, 91 and 62 feet. The missing or corrected unique well numbers are 421705, 431237, 570323 and 612713. MW-17 is omitted and the depth and unique well number are 112 feet and 570322, respectively. For the paragraph beneath the table on page 11, PW-7 is used occasionally at the 3M on-site trap range, and PW-8 supplies the guard shack.

In the fourth paragraph on page 12, PW-4 is in the northwest, not the northeast portion of the facility.

This paragraph recommends a coordinated round of groundwater sampling from all of the available wells to characterize fluorochemical levels. 3M agrees, and last year submitted a Work Plan to undertake such sampling. We request that the document acknowledge that 3M has already offered such a proposal.

Pages 7 and 13 indicate that the source of the model and assumptions underlying the groundwater modeling are unknown. While they may have been unknown to the Department of Health, that information has been provided to MPCA and can be made available to the Department if that would be helpful.

The suggestion that groundwater from the D1 area may discharge to the river via the intermittent stream is unfounded. Groundwater flow in the D1 area was triangulated in the investigation report for that area. There is no evidence that flow moves from the D1 area toward the intermittent stream.

Page 14, in the discussion of PW-5 and PW-6, should include reference to the fact that 3M agreed to complete additional monitoring of fluorochemicals in the area. Based on previous response actions at the D8 area related to VOC's, MPCA agreed no further monitoring for VOCs need be completed.

Chemistry, Physical/Chemical Properties

Page 16 explains that PFOS can be produced by the hydrolysis of POSF and other long-chain PFC compounds. Longer chain fluorochemicals do not degrade to shorter chain fluorochemicals (e.g., perfluorodecane sulfonate does not degrade to perfluorooctane sulfonate). Only the POSF-derived substances degrade to PFOS. The reference should be to POSF-derived compounds and not to other compounds.

Page 12, at the end of the second full paragraph, refers to “perfluorooctanesulfonates and acids.” As perfluorooctanesulfonate is an acid, the reference should be to “perfluorooctanesulfonates and *other* acids.”

Page 16 says that PFOS discharged to air will rapidly deposit to soil. We are not aware of data to support this statement. Moreover, the vapor pressure of PFOS is reported as 3.31×10^{-4} Pa @ 20 °C.

Toxicological Information

We appreciate that two pages of summary cannot do justice to the extensive toxicological database on fluorochemical substances. However, it would be helpful to cite more recent reviews, including Butenhoff et al., “Characterization of Risk for General Population Exposure to Perfluorooctanoate,” *Regulatory Toxicology & Pharmacology* 39:363-380 (2004), providing a review and risk assessment of PFOA, and Kennedy et al., 2004, reviewing PFOA toxicology. For PFOS, more information is available in 3M’s “Environmental and Health Assessment for Perfluorooctane Sulfonic Acid and its Salts,” August 20, 2003 (3M, 2003).

The draft consultation report on pages 4 and 21 states that PFOS is “more toxic” than PFOA. While the effects of PFOS in two-generation rat reproductive studies produce a greater incidence of effects, the calculated benchmark doses for serum levels of the two compounds that cause effects in rats and monkeys are similar.³ No-effect levels in repeat-dose studies are also similar. Thus, this statement should be deleted.

Half-Life in Serum

The half-life figures presented on page 17 correctly note that the estimated half-life of PFOA varies widely in different species. However, the differences among species in the half-life of PFOS are not so great as suggested.

³ Compare the benchmark doses (serum concentrations) presented for PFOS in 3M (2003), of 26 to 92 ppm for various endpoints, to the benchmark doses for PFOA presented in Butenhoff, et al. (2004), ranging from 23 ppm for liver weight increases, 29 ppm for post-natal effects in rats, to 125 for Leydig cell tumors in rats.

The draft consultation report relies on an OECD document on PFOS indicating a half-life of 7.5 days in rats and 200 days in cynomolgus monkeys. The serum elimination half-life in rats of 7.5 days apparently is taken from Johnson et al. (1979a); however, that observation represents redistribution as opposed to excretion, and the half-life of elimination in rats is substantially longer than this, in the range of 100-120 days.⁴

The 2002 OECD document correctly cited a half-life of approximately 200 days in cynomolgus monkeys in a sub-chronic study (Seacat et al., 2002). However, a recent, single intravenous dose pharmacokinetic study in male and female cynomolgus monkeys (Noker and Gorman, 2003) found a mean half-life of 132 days in males (range 122-146) and 110 days in females (range 88-138). This study is more comparable to the single-dose study in rats. Thus, there is no large difference between rats and monkeys in elimination half-life. The half-life of elimination in the rat is in the range of 100-120 days, and the half-life observed in monkeys in a comparable single-dose intravenous pharmacokinetic study ranged from 88 to 146 days with means of 110 and 132 days, in females and males respectively.

Effects in Animal Studies

The draft report on pages 17 and 18 refers to adverse liver effects in rats

- In the case of PFOS, liver effects are predominantly adaptive except at doses that produce mortality, and thus are not an appropriate endpoint to represent toxicity and adverse health effects in risk assessment.⁵

⁴ A pharmacokinetic study by the same authors demonstrated that the whole-body elimination half-life of PFOS in male rats is greater than 89 days following an intravenous dose (Johnson et al., 1979b). In that study, 42% of the radiolabel was excreted in urine and feces by day 89 post-dose. Based on this observation, the elimination half-life in the rat must be greater than 89 days, and is likely to be in the range of 100 to 120 days. Evidence from serum PFOS concentration data obtained at four and 14 weeks in a dietary chronic toxicity and cancer study in rats (Seacat et al., 2003) also support a longer half-life in rats. In repeat-dose pharmacokinetics, steady state is usually reached after approximately five half lives, and thereafter, serum concentrations would not be expected to increase significantly. If the elimination half-life were 7.5 days, the rats would be nearing steady-state serum PFOS concentrations in 5-6 weeks (37.5 days). However, in the chronic study, serum concentrations continued to increase substantially between weeks four and 14 in a linear fashion, indicating that the half-life is significantly longer than 7.5 days.

⁵ The hepatocellular hypertrophy observed at lower doses in PFOS-exposed animals is actually an adaptive response rather than an adverse effect. The hypertrophy was minimal to mild, and was reversible on cessation of dosing. Male rats with hypertrophy actually had a statistically significant increase in life span over controls. More serious liver pathology representing possible liver damage (e.g., necrosis and hyperplasia) was not a treatment-related finding in the 104-week chronic dietary study. Hyperplasia of liver cells was not observed in sub-chronic studies with PFOS, and hepatocellular necrosis was observed only in one sub-chronic study at doses that produced lethality. Serum clinical chemistry results from studies in rats, monkeys, and human workers do not indicate cellular toxicity in the liver.

- For PFOA, the liver is a primary target organ for both short-term and chronic effects of PFOA in rats (Griffith & Long, 1980; Olson & Anderssen, 1983; Kennedy, 1985; Pastoor *et al.*, 1987) and cynomolgus monkeys (Butenhoff *et al.*, 2002). The increased liver weight does not appear to be a result of hepatocellular hyperplasia (no increase in nuclear DNA) and has been variously attributed to increases in peroxisomes, endoplasmic reticulum and mitochondria (Ikeda *et al.*, 1985; Pastoor *et al.*, 1987; Butenhoff *et al.*, 2002; Berthiaume & Wallace, 2002; Biegel *et al.*, 2001). PFOA has been shown to activate the PPAR α receptor (Maloney & Waxman, 1999). Higher doses lead to liver degeneration and necrosis and the appearance in the serum of enzymes reflecting liver damage.

On page 18, the second full paragraph states that adverse effects in PFOS-exposed cynomolgus monkeys were not observed after a 52-week recovery period. In fact, clinical chemistry values generally had recovered within two months, and histological values showed recovery at the first examination at six months of recovery. (Seacat *et al.*, 2002)

The paragraph on page 18 regarding cancer risk confuses PFOA and PFOS. The two compounds produce different results in cancer bioassays.

- Chronic dietary exposure of rats to PFOS caused a low-level increase in hepatocellular adenoma (benign liver tumors) at the highest dose tested (20 ppm in diet). The hepatocellular tumors are likely the result of a non-genotoxic mechanism. PFOS has been shown to be a peroxisome proliferator. (Berthiaume and Wallace (2003); Ikeda *et al.* (1987) Sohlenius *et al.* (1992); Case *et al.* (2001); Seacat *et al.* (2002); Thomford (2002)) Given the rather weak response in terms of benign hepatocellular adenoma, taken together with the demonstrated lack of genotoxicity of PFOS, PFOS should not present a risk of cancer to humans at the levels of exposure that have been determined. Tumor incidence was reduced (statistically significantly in males) when dosing was suspended at one year. The tumor-incidence dose-response curve suggests a non-linear, threshold relationship between dose and increased lifetime risk of excess liver tumors. An increase in thyroid follicular cell adenoma in the high-dose recovery males is likely unrelated to treatment since this finding was not observed in males or females in the high-dose group or in recovery group females, and no other evidence of thyroid involvement was seen in the study.
- The oncogenicity of PFOA has been investigated in two separate two-year feeding studies in rats. PFOA was found to increase the incidence of three tumor types, liver, Leydig cell, and pancreatic acinar cell tumors.⁶ (Riker, 1983, and Biegel *et al.* 2001) These tumors are frequently observed in rats treated with peroxisome proliferators. It is generally recognized that rats have a heightened response to peroxisome proliferators relative to other species,

⁶ An apparent increase in mammary fibroadenomas, seen in the PFOA-treated female rats, was the result of an unusually low incidence of fibroadenomas in this particular control group. The incidence of mammary tumors in all test groups was within the range expected for this strain of rat based on historical control data

including humans. The human significance of these three tumor types is not clear. These tumors are rare in humans and excesses have not been observed in exposed workers. Available data for humans who have had long-term treatment with hypolipidemic drugs (which are potent peroxisome proliferators in rats) show no increase in these three cancers.

Developmental Effects

Page 19 of the draft consultation report suggests that recent studies on PFOS by Thibodeaux et al. (2003) and Lau et al. (2003) may lead MDH to consider revising the Health-Based Values to different, and likely lower, values based on developmental effects. A review of those papers suggests this is incorrect, and the speculation should be withdrawn.

The literature on the effects of PFOS includes teratology studies (which examine structural defects at the time pups are born) and reproductive and developmental studies (which examine reproductive function, which is not affected, and effects on postnatal rat pups). These studies have been conducted by outside laboratories for 3M, and by EPA researchers Lau, Thibodeaux, et al. The teratology studies are generally unremarkable. The effect of concern for human risk assessment is the postnatal developmental effects of PFOS on rat pups at experimental doses. (Lau et al. 2004.)

Teratology Studies

In a recent review paper, Lau et al. (2004), characterized the PFOS teratology studies as follows:

“Teratological studies have been conducted in rat, rabbit, and mouse with PFOS (potassium and lithium salts) (Case et al., 2001b; Christian et al., 1999a; Gortner, 1980; Henwood et al., 1994; Thibodeaux et al., 2003; Wetzel, 1983). The findings are in agreement between laboratories and across species examined, and are generally unremarkable when maternal effects are taken into consideration.” (Emphasis added.)

This summary in Lau et al. (2004) encompassed the paper by Thibodeaux, et al. (2003), referenced in the draft consultation report, on which Lau was the senior (last) author. Thibodeaux et al. reported on maternal and developmental evaluations in rat and mice exposed to PFOS. (A companion paper discussed below, Lau et al. (2003), addressed the more important postnatal findings.) Thibodeaux et al. (2003) found that mice are generally less sensitive than rats to the postnatal effects of PFOS. Birth defects were observed primarily at the highest dose levels. However, the authors note “profound deficits in maternal weight gain” in the PFOS-exposed rats and maternal toxicity in the mice as well. The conclusion in Thibodeaux et al. (2003) states:

“In summary, exposure to PFOS during pregnancy led to significant physiological alterations in the rat and mouse that are

indicative of maternal toxicity, as well as to anatomical defects observed in the fetuses at term at high dosages. These adverse outcomes are dose-dependent and can be correlated with body burden of the fluorochemical. Generally, the mouse appeared to be a less sensitive species than the rat in regard to the PFOS-induced toxicity.”

The NOEL for cleft palate was 5 mg/kg/day in rats and 10 mg/kg/day in mice. The paper indicates increased sternal defects were seen in rats at 2 mg/kg/day and 10 mg/kg/day doses, but not at doses of 3 or 5 mg/kg/day. In mice, sternal defects had a NOEL of 1 mg/kg/day; they were increased at 2 mg/kg/day. (Tables 1 and 2 in Thibodeaux et al. 2003.) These values are all well above the 0.15 mg/kg/day NOEL value from the PFOS monkey study used to derive the current Minnesota HBVs.

Given the unremarkable nature of the structural abnormalities and the observed maternal toxicity, and the occurrence of postnatal effects at generally lower doses than the structural abnormalities, human risk assessment should be based on the values for post-natal effects rather than teratogenic endpoints.

Reproductive and Postnatal Effects

Lau et al. (2003) reported on the postnatal evaluation of the same animals studied by Thibodeaux, et al. (2003) in a companion publication. Neonatal mortality occurred at lower doses than birth defects. The NOAEL for effects on the rat pups was 1 mg/kg/day concentration (Table 2 in Lau et al. 2003) The LBMD₅ values for survival at postpartum day 8 in rats was 0.58 mg/kg/day, and at postpartum day 6 in mice was 3.88 mg/kg/day. Both the NOAEL and the benchmark dose values are higher than the 0.15 mg/kg/day dose used in the Minnesota HBVs.

Similarly, the benchmark doses for postnatal effects in the 3M one- and two-generation studies of PFOS calculated in 3M (2003) are higher than the value used in deriving Minnesota's current HBVs. Benchmark doses (specifically, the lower 95% confidence limits of the benchmark dose for a 5% change) for various effects from 3M's PFOS reproduction studies are shown in the table below.

Lower 95% CL of the Benchmark Dose and Benchmark Internal Concentration for Developmental Effects at 5% Benchmark Response Level

Study	Endpoint	LBMD ₅ (mg/kg/day)	LBMIC ₅ (ug/mL)
2-Gen Repro/Dev	F ₁ Pup Weight Gain (LD21) ^a	0.34	26
2-Gen Repro/Dev	F ₁ Pup Weight Gain (LD21) ^b	0.34	36
2-Gen Repro/Dev	F ₁ Litter Size (LD4) ^a	0.39	30
2-Gen Repro/Dev	F ₁ Litter Size (LD4) ^b	0.39	39
1-Gen Repro/Dev	F ₁ Litter Size (LD5) ^a	0.83	71
2-Gen Repro/Dev	F ₁ Pup Mortality (LD4) ^a	0.84	71
1-Gen Repro/Dev	F ₁ Pup Mortality (LD5) ^a	0.83	83
2-Gen Repro/Dev	F ₁ Pup Mortality (LD4) ^b	0.84	84

^a Based on serum samples taken on GD 21

^b Based on serum samples taken on GD 0.

(Beginning of gestation values are appropriate for comparison to measured human concentrations.)

Thus, the most stringent benchmark dose (lower confidence limit on the benchmark dose for a 5% incidence) for these various endpoints from the 3M PFOS reproductive studies is approximately 0.34 mg/kg/day dose. This is higher than the current Minnesota HBV based on a dose level of 0.15 mg/kg/day from the cynomolgus monkey study.

Thus, the values used for HBVs would *not* be more stringent if based upon the developmental studies. This speculation should be deleted from the draft consultation report.

The draft also suggests on page 19 that the HBVs may be decreased to account for childhood exposures. In the case of PFOS and PFOA, developmental studies are available, and thus the HBVs can directly address potential effects on children without having to apply a default safety factor.

Epidemiologic Information

Worker Monitoring

3M has conducted medical surveillance of fluorochemical production workers for over 25 years. A battery of clinical tests (including lipids, hematological parameters, enzymes and 11 different hormone assays) showed no pattern of association between these measurements and PFOS or PFOA levels in workers.

The reference on page 3 to “possible effect on levels of one hormone” is misleading. Page 19 elaborates, citing a *Journal of Occupational and Environmental Medicine* publication (Olsen et al. 1998a) of a study of reproductive hormones in Cottage Grove workers in 1993 and 1995 that found elevated estradiol concentrations in five workers with PFOA serum concentrations above 30 ppm in the 1995 medical surveillance.

- The draft consultation report omits the rest of the sentence from the study, which states that: “A 10% increase in mean estradiol level was observed among employees who had the highest levels of serum PFOA, although this association was confounded by body mass index.” (Study abstract, emphasis added.) Body mass is a known confounder for estradiol. All five employees with PFOA levels above 30 ppm had Body Mass Indexes (BMI) of 28 or more. Id. at 617. Taking into account this potential confounding, there was no pattern of association between PFOA and estradiol levels
- The scatterplots on page 616 of the Olsen et al. 1998a paper present a clear visual representation that estradiol does not vary with increasing PFOA exposure.
 - As noted on page 617, “Simple linear regression of the natural log of [estradiol] with PFOA, treated as a continuous variable, resulted in no statistically significant coefficients”
 - The text there further states that “linear and nonlinear relationships, taking into account potential confounders (especially age and BMI) as well as other covariates that may be on the biologic pathway of effect, resulted in no significant associations with PFOA except for 17-HP in the 1995 analysis.”

Accordingly, we do not believe it is appropriate to suggest an effect on estradiol from PFOA given the lack of findings in either linear or nonlinear models.

- The referenced 1998 publication presents hormone data from medical surveillance at Cottage Grove in 1993 and 1995. In addition, hormone levels in workers at 3M’s Decatur, Alabama and Antwerp, Belgium fluorochemical production plants were tested in 1995 and 1997, and although the workers’ levels of PFOA were lower than at Cottage Grove, there was no association between their PFOA levels and estradiol. The published paper addressing the Decatur and Antwerp surveillance (Olsen et al. 1998b) does not address the findings on PFOA and hormones, but the data are discussed in the full study report.⁷ With respect to PFOA, the report states:

“PFOA production workers in Cottage Grove with serum levels up to 30 ppm appeared not to have altered serum estradiol levels [Olsen et al., 1998]. . . . We did not observe any significant positive association between estradiol and serum PFOA levels in these Antwerp and Decatur employees.” (p. 30)

⁷ The Decatur and Antwerp surveillance focused on PFOS and clinical chemistries. (Olsen et al. 1998b) A statistically significant quadratic model was fit between PFOS and estradiol; however, residual diagnostics showed this model was highly influenced by one specific employee whose serum PFOS concentrations was 12.8 ppm, the highest measured in the study, with an estradiol value of 92 pg/dl. This employee was also obese (BMI = 33), an important confounder (Olsen et al. 1998b)

In sum, the draft consultation report's reference to estradiol levels in five Cottage Grove workers in one year's medical surveillance does not provide a complete and accurate review of the body of information presented in the referenced publication, nor the overall body of data available on this issue. The epidemiologic evidence does not indicate that PFOA affects estradiol at the concentrations measured in Cottage Grove workers.

Mortality Studies

On page 20, the draft consultation report concludes that the findings of the mortality studies "do not represent epidemiological findings of significance." Yet, the summary on pages 3-4 says that the epidemiologic data are inconclusive. We suggest the language from the text also be used in the summary.

The draft consultation report (pages 19-20) discusses the original mortality study of Cottage Grove workers by Gilliland and Mandel (1993) and also the subsequent study by Alexander (2001), which used an improved job-calendar-year exposure matrix. Although the draft consultation cites finding in the Gilliland and Mandel (1993) study of a 3-fold excess of prostate cancer among workers with more than ten years employment, this association was not confirmed in the updated Alexander study. If the earlier finding is going to be included in the consultation report, then the report needs to provide some additional detail.

The Gilliland and Mandel study used duration of employment in the Chemical Division at Cottage Grove (or lack thereof) as a surrogate for PFOA exposure. As noted in the draft consultation report, there were four prostate cancer deaths observed in Chemical Division workers. Subsequent research has shown that only one of these employees worked in the PFOA production building. (Olsen 1998a, p. 615.) Additional data have shown that employment duration is not a good surrogate for serum PFOA concentrations among employees in the Cottage Grove Chemical Division (Olsen et al. 2003a). Thus, the association reported in the original mortality study between *duration of employment in the Chemical Division* and prostate cancer mortality is very difficult to interpret. The original authors themselves caution against over-interpretation of the findings.

In the updated study by Alexander (2001), prostate cancer mortality was not significantly associated with definite or probable PFOA exposure categories. Furthermore, in a recently published review of the toxicology of PFOA (Kennedy et al. 2004), the updated mortality data on prostate cancer are further presented and do not show an association with duration of employment in an external analysis among those with definite or probable exposure to PFOA (observed/expected in parentheses): 0-<1 year (0/0.1); 1-<5 (2/1.4); 5-<10 (0/0.8); and ≥ 10 (4/2.9). Thus, we caution against citation to the Gilliland and Mandel (1993) study results without full elaboration of subsequent findings.

The draft report notes a finding of excess cerebrovascular disease in Alexander (2001). Alexander (2001) considered this finding difficult to interpret and was unable to consider it a causal association at this time.

General Population Exposure

Page 4 states that general population levels of fluorochemical substances are about ten-fold less than levels in workers. This is incorrect.

- Olsen et al (2003a) reported the median serum concentrations of PFOA from surveillance in 2000 of the Cottage Grove workforce who have worked only in the PFOA production area to be approximately 5 ppm; the mean concentration was 18.4 ppm (95% CI 6.7-30.1). Antwerp and Decatur workers' serum PFOA and PFOS concentrations averaged between 1 and 2 ppm. (Olsen et al., 1999; Olsen et al. 2003c).
- The general population has average levels of 0.005 ppm PFOA and 0.040 ppm PFOS. (Olsen et al. 2003b; Olsen et al. 2004a; Olsen et al. 2004b.⁸)

Thus, the difference in mean serum levels between the general population and workers engaged in either PFOS or PFOA fluorochemical production is about two orders of magnitude for PFOS and three or more orders of magnitude for PFOA.

Margins of Exposure

The draft consultation report (pages 4, 21) cites margins of exposure for childbearing women and attributes these to EPA. The information comes from an April 2003 "*preliminary draft*" EPA document for PFOA (USEPA 2003). A year later, in a March 29, 2004 Federal Register notice, EPA indicated that it had completed its *draft* PFOA risk assessment and would submit it to review by a Science Advisory Board. 69 Fed. Reg. 16249. EPA has not yet released that draft, nor has it yet convened the Science Advisory Board to review the draft. Accordingly, citation of the obsolete preliminary draft is inappropriate.

The preliminary EPA draft reflected a misunderstanding of the pharmacokinetics of PFOA in rats. The EPA preliminary draft presented margins of exposure using blood levels from female rats without adjusting for their rapid clearance of PFOA, and thus underestimated rat serum levels and the attendant margin of exposure.⁹

⁸ These papers characterize serum levels in the U.S. population of adults, children and the elderly. The three studies showed consistent results, with little variation by age or gender. For additional references on general population concentrations, see Hansen et al. (2001); Kannan et al. (2004 in press); Kuklennyik et al. (2004) and 3M (2003).

⁹ The *preliminary draft* risk assessment document calculated an estimated range for margins of exposure (MOE) between human serum concentrations of PFOA and the serum concentrations that might be found in weanling rats that experienced developmental effects in a two-generation reproductive study. Weanling rat serum concentrations of PFOA were estimated from adult levels that were measured 24 hours after dosing. Use of the adult F₀ female serum concentration from a sample obtained 24 hours after the last dose is a gross underestimate of the values likely to exist in weanling rats given that female rats excrete virtually all PFOA within 24 hours. Use of the area-under-the-curve approach to provide an average serum concentration corrects for this pharmacokinetic issue.

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Butenhoff et al. (2004), have presented a risk assessment for PFOA that takes into account the complex pharmacokinetics of PFOA (using an area-under-the-curve approach to calculate average female rat serum levels). The authors report margins of exposure for the mean serum PFOA concentration (0.01 ppm) estimated to be the 95th percentile of general population exposure to be between 1600 and 8900 for various endpoints. The margin of exposure for post-natal effects for the mean serum concentration estimated for the 95th percentile general population is 2100. (Table 10 in Butenhoff et al. 2004.)

The PFOA margins of exposure reported in the draft consultation report are inaccurate and should not be used. The suggestion that the margin of exposure is 66 is scientifically unsound and misleading.

Environmental Data

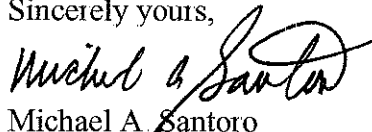
Page 21 of the draft report gives a BCF for PFOS in bluegills of 4013, citing the OECD document. However, the OECD document makes clear this value is for the non-edible portion of the fish only. The edible portion (BCF 1124) would be relevant for human health assessment, and the whole fish value (BCF 2796) would be relevant for ecological risk assessment. We do not understand why a BCF for non-edible portions of the fish would be the relevant value to mention.

* * *

3M appreciates the Department's efforts in providing this consultation, and we hope the foregoing comments are helpful in improving the scientific accuracy of the consultation report. If you deem it appropriate, we would appreciate your forwarding a copy of these comments to interested parties such as ATSDR and local authorities.

3M would be pleased to provide any additional information that would be helpful to the Department.

Sincerely yours,



Michael A. Santoro
Director, Environmental Health, Safety and
Regulatory Affairs

cc: Dave Douglas, MPCA
Cindy Weckwerth, Washington Co.

References:

- 3M (2003). Environmental and Health Assessment of Perfluorooctane Sulfonic Acid and Its Salts USEPA Public Docket AR-226.
- Alexander, B.H. (2001). "Mortality study of workers employed at the 3M Cottage Grove facility." Final Report. Division of Environmental and Occupational Health, School of Public Health, University of Minnesota, April 26, 2001. EPA docket AR226-1030a018.
- Berthiaume, J. and Wallace, K. (2002). Perfluorooctanoate, perfluorooctane sulfonate, and N-ethyl-perfluorooctanesulfonamido ethanol; peroxisome proliferation and mitochondrial biogenesis. *Toxicology Letters*, 129: 23-32.
- Biegel, L.B., Hurtt, M.E., Frame, S.R., O'Connor, J.C., and Cook, J.C. (2001) Mechanisms of extrahepatic tumor induction by peroxisome proliferators in male CD rats. *Toxicol. Sci.*, 60: 44-55.
- Butenhoff, J.L., D.W. Gaylor, J.A. Moore, G.W. Olsen, J. Rodricks, J.H. Mandel, L. R. Zobel (2004). "Characterization of risk for general population exposure to perfluorooctanoate." *Regulatory Toxicology and Pharmacology* 39:363-380
- Butenhoff, J., Costa, G., Elcombe, C., Farrar, D., Hansen, K., Iwai, H., Jung, R., Kennedy, G., Lieder, P., Olsen, G., and Thomford, P. (2002). Toxicity of ammonium perfluorooctanoate (PFOA) in male cynomolgus monkeys after oral dosing for six months *Toxicol. Sci.*, 69: 244-257
- Case, M.T., York, R.G. and Butenhoff, J.L. (2001). Oral (gavage) cross-fostering study of potassium perfluorooctane sulfonate (PFOS) in rats. *Toxicol. Sci.* 60 (S-1), 1055.
- Gilliland, F.D. & Mandel, J.S. (1993) *Mortality Among Employees of a Perfluorooctanoic Acid Production Plant. JOM* 35: 950-954.
- Griffith, F.D. and Long, J.E. (1980). Animal toxicity studies with ammonium perfluorooctanoate. *Am. Ind. Hyg. Assoc. J.*, 41: 576-583.
- Hansen KJ, Clement LA, Ellefson ME, Johnson HL (2001). Compound-specific quantitative characterization of organic fluorochemicals in biological matrices. *Environ Sci Technol* 35: 766-770.
- Ikeda, T., Fukuda, K., Mori, I., Enomoto, M., Komai, T. and Suga, T. (1987). Induction of cytochrome P-450 and peroxisome proliferation in rat liver by perfluorinated octanesulfonic acid. In: *Peroxisomes in Biology and Medicine*, H.D. Fahimi and H. Sies, Eds, Springer Verlag, New York, 304-208.

- Johnson, J.D. and Ober, R.E. (1979a). Absorption of FC-95-¹⁴C in rats after a single oral dose. Project No 8900310200, Riker Laboratories, Inc., St. Paul, MN. (US EPA Docket No. 8(e)HQ-1180-00374)
- Johnson, J.D., Gibson, S.J., and Ober, R.E. (1979b). Extent and route of excretion and tissue distribution of total carbon-14 in rats after a single i.v. dose of FC-95-¹⁴C. Project No. 8900310200, Riker Laboratories, Inc., St. Paul, MN. (US EPA Docket No. 8(e)HQ-1180-00374)
- Kannan K, Corsolini S, Falandysz J, Fillman G, Kumar KS, Loganathan B, Mohd MA, Olivero J, van Woue N, Yang JH, Aldous KM (2004). Perfluorooctanesulfonate and related fluorochemicals in human blood from several countries. *Environ Sci Technol* (in press).
- 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(4):351-384.
- Kennedy, G.L., Jr. (1985). Dermal toxicity of ammonium perfluorooctanoate. *Toxicol. Appl. Pharmacol.* 81: 348-355.
- Kuklenyik Z, Reich JA, Tully JS, Needham LL, Calafat AM. (2004). Automated solid-phase extraction and measurement of perfluorinated organic acids and amides in human serum and milk. *Environ Sci Technol* 38: 3698-3704
- Lau, C., Butenhoff, J.L., and Rogers, J.M. (2004). The developmental toxicity of perfluoroalkyl acids and their derivatives. *Toxicol. Appl. Pharmacol.* (in press, available on web site).
- Lau, C., Thibodeaux, J.R., Hanson, R.G., Rogers, J.M., Grey, B.E., Stanton, M.E., Butenhoff, J.L. and Stevenson, L.A. (2003). Exposure to Perfluorooctane Sulfonate During Pregnancy in Rat and Mouse. II. Postnatal Evaluation. *Toxicol. Sci.* 74: 382-392
- Maloney, E.D. and Waxman, D.J. (1999). "Trans-Activation of PPAR alpha and PPAR gamma by structurally diverse environmental chemicals. *Toxicol. Appl. Pharm.*, 161:209-18.
- Noker, P.E. and Gorman, G.S. (2003). A pharmacokinetic study of potassium perfluorooctanesulfonate in the cynomolgus monkey. Southern Research Institute. unpublished report. Available on USEPA Administrative Record 226.
- Olsen, G.W., Church, T.R., Larson, E.B., van Belle, G., Lundberg, J.K., Hansen, K.J., Burris, J.M., Mandel, J.H., Zobel, L.R. (2004a). "Serum concentrations of perfluorooctanesulfonate and other fluorochemicals in an elderly population from Seattle, Washington." *Chemosphere* 54(11):1599-1611.

- Olsen, G W , T. R., Miller, K. Hansen, J. Burris, J. Butenhoff, J. Mandel, L. Zobel (2004b). Quantitative Evaluation of Perfluorooctanesulfonate (PFOS) and Other Fluorochemicals in the Serum of Children. *J. Children's Health* 2(1): 53-76.
- Olsen, G W., Butenhoff, J L., Mandel, J H (2003a) "Assessment of lipid, hepatic and thyroid function in relation to an occupational biologic limit value for perfluorooctanoate." St. Paul(MN):3M Company. June 9, 2003 EPA docket AR-226-1351.
- Olsen, G W , Church, T. R., Miller, J P., Burris, J. M., Lundberg, J. K., Herron, R. M., Medhdizadehkashi, Z., Nobiletti, J. B , O'Neill, E. M , Mandel, J. H., Zobel, L. R (2003b). "Perfluorooctanesulfonate and other fluorochemicals in the serum of American Red Cross adult blood donors." *Environ. Health Perspect.*, 111(16):1892-1901 [Erratum in *Environ. Health Perspect.*, 111(16):1900].
- Olsen, G W , Burris, J.M., Burlew, M.M., Mandel, J H. (2003c). Epidemiologic assessment of worker serum perfluorooctanesulfoante (PFOS) and perfluorooctanoate (PFOA) concentrations and medical surveillance examinations. *J Occup. Env Med.* 45:260-270
- Olsen, G.W., Burris, J.M., Burlew, M M., and Mandel, JH. (2000). Plasma cholecystokinin and hepatic enzymes, cholesterol and lipoproteins in ammonium perfluorooctanoate production workers. *Drug Chem. Toxicol.* 23:603-620.
- Olsen, G.W , Burris, J M., Mandel, J H , Zobel, L R. (1999). Serum perfluorooctane sulfonate and hepatic and lipid clinical chemistry tests in fluorochemical production employees. *J Occup. Env. Med.* 41:799-806.
- Olsen, G.W., et al. (1998a). An Epidemiologic Investigation of Reproductive Hormones in Men with Occupational Exposure to Perfluorooctanoic Acid. *J. Occup. Env. Med.* 40:614-619.
- Olsen, G.W , Burris, J.M., Mandel, J.H., Zobel, L.R. (1998b). "An Epidemiologic Investigation of Clinical Chemistries, Hematology and Hormones in Relation to Serum Levels of Perfluorooctane Sulfonate in Male Fluorochemical Production Employees". EPA Docket AR226.0030.
- Olson, C T. and Anderson, M E. (1983). The acute toxicity of perfluorooctanoic and perfluorodecanoic acids in male rats and effects on tissue fatty acids. *Toxicol. Appl. Pharmacol.*, 70: 362-372
- Pastoor, T.P., Lee, K P., Perri, M A., and Gillies, P J. (1987). Biochemical and morphological studies of ammonium perfluorooctanoate-induced hepatomegaly and peroxisome proliferation. *Exp. Mol. Pathol.* , 47: 98-109.
- Riker (1983). Two year oral (diet) toxicity/carcinogenicity study of fluorochemical FC-143 in rats. Riker Laboratories, Inc., Experiment No. 0281CR0012, May 1983.
- Seacat, A M , Thomford, P J., Hansen, K J., Clemen, L.A., Eldridge, S.R., Elcombe, C.R., and Butenhoff, J L. (2003). Sub-chronic dietary toxicity of potassium perfluorooctanesulfonate in rats. *Toxicology* 183: 117-131.

- Seacat, A.M., Thomford, P.J., Hansen, K.J., Olsen, G.W., Case, M.T., and Butenhoff, J.L. (2002) subchronic toxicity studies on perfluorooctanesulfonate potassium salt in Cynomolgus monkeys. *Toxicol. Sci.* 68: 249-264
- Sohlenius, A. K., Eriksson, A. M., Hogstrom, C., Kimland, M. and DePierre, J. W. (1993) Perfluorooctane sulfonic acid is a potent inducer of peroxisomal fatty acid beta-oxidation and other activities known to be affected by peroxisome proliferators in mouse liver. *Pharmacol. Toxicol.* 72: 90-3.
- Thibodeaux, J. R., Hanson, R. G., Rogers, J. M., Grey, B. E., Barbee, B. D., Richards, J. H., Butenhoff, J. L., Stevenson, L. A., and Lau, C. (2003). Exposure to perfluorooctane sulfonate during pregnancy in rat and mouse. I: Maternal and prenatal evaluations. *Toxicol. Sci.* 74: 369-381.
- Thomford, P. J., Seacat, A. M. and Butenhoff, J. L. (2002). Terminal observations in Sprague-Dawley rats after lifetime dietary exposure to N-ethylperfluorooctanesulfonamide ethanol. *Toxicol. Sci.* 66 (S-1), 185-186 (Abstract ID: 907).
- USEPA, "Preliminary Risk Assessment of the Developmental Toxicity associated with exposure to Perfluorooctanoic Acid and its Salts," April 14, 2003