

AR226-3953

Taft/

Taft Stettinius & Hollister LLP

425 Walnut Street, Suite 1800 / Cincinnati, OH 45202-3957 / Tel: 513.381.2838 / Fax: 513.381.0205 / www.taftlaw.com
Cincinnati / Cleveland / Columbus / Dayton / Indianapolis / Northern Kentucky / Phoenix / Beijing

ROBERT A. BILOTT
513-357-9638
bilott@taftlaw.com

August 31, 2010

EPA Docket Center, MC 2822T
U.S. Environmental Protection Agency
EPA West, Room 3334
1200 Pennsylvania Avenue, NW
Washington, D.C. 20460-0001

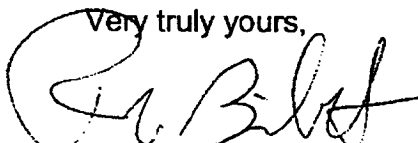
Re: Submission to IRIS and AR-226 Database For PFOA/PFOS: EPA-HQ-
ORD-2003-0016

To IRIS Database for PFOA/PFOS:

In response to the Notice issued by USEPA on February 23, 2006, regarding USEPA's efforts to consider perfluorooctanoic acid ("PFOA") and perfluorooctane sulfonate ("PFOS") within the Integrated Risk Information System ("IRIS"), 71 Fed. Reg. 9333-9336 (Feb. 23, 2006), we are submitting the following additional information to USEPA for inclusion in that review, and for inclusion in the AR-226 database:

1. Minnesota Department of Health, "Public Health Assessment: Perfluorochemical Contamination in Southern Washington County, Northern Dakota County, and Southeast Ramsey County, Minnesota," (public comment release draft) (August 25, 2010); and
2. Kraugerud, M., et al, "Pefluorinated Compounds Differentially Affect Steroidogenesis in the Human Adrenocortical Carcinoma (H295R) *inVitro* Cell Assay," (doctoral thesis manuscript) (August 2010).

Very truly yours,



Robert A. Bilott

RAB:mdm
Enclosure

CONTAINS NO CBI

August 31, 2010
Page 2

cc: Gloria Post (NJDEP)(w/ encl.) (via U.S. Mail)
Helen Goeden (MDH)(w/ encl.) (via U.S. Mail)
Lora Werner (ATSDR)(w/ encl.) (via U.S. Mail)

Perfluorinated compounds differentially affect steroidogenesis in the human adrenocortical carcinoma (H295R) *in vitro* cell assay.

Marianne Kraugerud^{a*}, Karin E. Zimmer^b, Erik Ropstad^a, Steven Verhaegen^a

^aDepartment of Production Animal Sciences, Norwegian School of Veterinary Science, Postboks 8146 Dep, 0033 Oslo, Norway

^bDepartment of Basic Sciences and Aquatic Medicine, Norwegian School of Veterinary Science, Postboks 8146 Dep, 0033 Oslo, Norway

*All correspondence to:

Marianne Kraugerud

Phone: +47 22597049

Fax: +4722597081

Email: Marianne.Kraugerud@veths.no

Key words: perfluorooctane sulfonate; perfluorooctanoic acid; perfluorononanoic acid; steroidogenesis; endocrine disruption; H295R

Acknowledgements

This work was supported by the Research Council of Norway (158849/I10 and 175098/V40).

The authors would like to thank Ellen Dahl, Kristine von Krogh and Camilla Almås for excellent technical assistance. S. V. would like to thank Dr. Christine Nelleman and Dr. Anders Elleby Engel-Kofoed at the National Food Institute, Denmark, for the chance of visiting their lab and for assisting us in establishing and standardizing the H295R steroidogenesis assay.

Abstract

Perfluorinated compounds (PFCs) are man-made chemicals which are present throughout the ecosystem, raising concerns about potential harmful effects of PFCs on humans and the environment. In order to investigate the effects of PFCs on steroid hormone production, human adrenocortical H295R cells were exposed to three PFCs including perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA) and perfluorononanoic acid (PFNA) at six different concentrations (6 nM to 600 μ M) for 48 hours. Oestradiol, progesterone, testosterone and cortisol were measured in the cell medium using radioimmunoassays. In addition, gene expression of 14 genes encoding proteins involved in steroidogenesis was measured using qRT-PCR. Exposure to PFOS resulted in an increase in oestradiol, progesterone and testosterone secretion at 600 μ M. Furthermore, testosterone was elevated at 6 μ M and 600 nM of PFOA and reduced at 60 μ M PFNA. Gene expression of *CYP11A* was decreased by all exposure doses of PFOA, whereas *HMGR* was decreased by 60 nM PFNA. We conclude that all PFCs tested are capable of altering steroidogenesis in the H295R *in vitro* model. The greatest effects were seen with PFOS exposure at the highest dose tested. The alterations in hormone secretion caused by PFOS appeared to be mediated by mechanisms other than changes in gene expression of steroidogenic enzymes.

Introduction

Perfluorinated compounds (PFCs) are man-made chemicals used as ingredients in a wide variety of applications including stain repellents and lubricants as well as in the production of fluoropolymers used in non-stick cookware. Due to their widespread use, PFCs are now detectable throughout all compartments of the ecosystem including air, water, sediments, animals and humans (Fromme et al., 2009; Nakata et al., 2006; Yamashita et al., 2005) even in remote places such as the arctic (AMAP, 2009). Recently, much attention has been directed towards the potential harmful effects on humans and the environment associated with the long-term exposure to PFCs. As a result, the United Nations Environment Programme (UNEP) made the decision to list the PFC perfluorooctane sulfonate (PFOS) and its salts as persistent organic pollutants (POPs) at the Stockholm Convention in Geneva, May, 2009.

PFCs consist of a fully fluorinated straight carbon backbone of typically 4-14 carbons attached to a charged functional moiety, giving rise to a large variety of molecules (Figure 1). Due to the stability of the C-F bond, PFCs are slowly degradable, and human half-lives as long as 5.4 years and 3.8 years for PFOS and perfluorooctanoic acid (PFOA), two of the most abundant PFCs, have been reported (Olsen et al., 2007).

Although the evidence of adverse effects of PFCs in human epidemiological studies has been a subject of debate, some studies have found that increasing human plasma PFCs are correlated with a lower birth weight (Apelberg et al., 2007; Fei et al., 2008). Furthermore, studies on laboratory animals suggest that PFCs increases the risk of stillbirth, decreases foetal weight and causes developmental defects (Case et al., 2001; Lau et al., 2003; Thibodeaux et al., 2003). This is of great concern, particularly considering that clearance rates of PFCs are much higher in some of the laboratory species than in humans. For example, in female rats half-lives of 0.08 and 2.44 days for PFOA and perfluorononanoic acid (PFNA), respectively, have been reported (Ohmori et al., 2003).

It is also becoming increasingly recognized that PFCs can act as endocrine disruptors. Several *in vivo* studies typically report decreased plasma levels of testosterone and the thyroid hormone thyroxine, in addition to an increase in oestradiol and cortisol following exposure to PFCs including PFOS and PFOA (Biegel et al., 1995; Biegel et al., 2001; Lau et al., 2003; Oakes et al., 2004; Thibodeaux et al., 2003). Studies have also identified a weak carcinogenic

effect of PFOA, manifested by an increased incidence of hepatic and Leydig cell adenomas in rats (Biegel et al., 1995; Biegel et al., 2001). Whereas the hepatic tumour induction is mediated through peroxisome proliferator receptor (PPAR) binding, it is postulated that the neoplastic transformation of Leydig cells are caused, at least partially, by elevated oestradiol levels (Biegel et al., 2001). This is of great concern due to the possible link between oestrogenic effects of environmental pollution and the increased prevalence of human testicular cancer (Skakkebaek et al., 2001).

To date, little research has focussed on the steroidogenesis pathway as a potential target for PFCs. Furthermore, previous studies have invariably been carried out on tissues and cell cultures of animal origin. Considering the great differences in response to PFCs and their elimination between some species, it is evident that inter-species extrapolation of these results would be inaccurate.

The human adrenocortical cell line, H295R, shows characteristics of an undifferentiated foetal adrenal cortex and is capable of full steroidogenesis (Gazdar et al., 1990; Skakkebaek et al., 2001). Furthermore, it has been employed as an *in vitro* model for studying endocrine disruptors and it is currently being evaluated by the US-EPA and OECD as a Tier 1 screening assay for effects on steroidogenesis.

The aim of this study was to investigate how three structurally different PFCs commonly found in environmental samples affect steroidogenesis in the human H295R *in vitro* cell model. The compounds include the eight carbon chain molecules PFOS and PFOA in addition to the nine carbon chain molecule PFNA.

Materials and methods

Chemicals

Tetrabutylammonium heptadecafluorooctanesulfonate, perfluorooctanoic acid and perfluorononanoic acid were purchased in powder form from Sigma-Aldrich (St Louis, MO,

USA) with a purity of >95%, >90% and >97% respectively. Chemicals were dissolved in dimethyl sulfoxide (DMSO) to 600 mM stock solutions and stored in aliquots at -20°C.

H295R cell culture

The H295R cell line was obtained from the American Type Culture Collection (ATCC CRL-2128, ATCC, Manassas, VA, USA) and cultured in 75cm² flasks in Dulbecco's modified Eagle medium/HamF12 (DMEM/F12) containing HEPES buffer, L-glutamine and pyridoxine HCl (Gibco, Invitrogen, Paisley, UK). The medium was further supplemented with 1% ITS + premix and 2.5% NuSerum (BD Biosciences, Bedford, MA). Cells were incubated at 37°C with 5% CO₂ in a humidified atmosphere. Medium was changed every 2-3 days and cells passaged at approximately 80% confluence by a brief exposure to 0.25% trypsin/0.53 mM EDTA (Gibco, Invitrogen, Paisley, UK) followed by centrifugation and reseeded. The cells were used between passages 5-13.

Exposure studies

For experiments, cells were seeded at 3×10^5 cells/well in 24-well cell culture plates (Falcon, Franklin Lakes, NJ, USA). Cells were incubated 24 hours prior to exposure to the compounds.

H295R cells were exposed for 48 hours to six concentrations (6 nM, 60 nM, 600nM, 6 µM, 60 µM or 600 µM) of PFOS, PFOA or PFNA of in triplicates. Each plate also contained triplicate wells of 600 µM tetrabutylammoniumchloride dissolved in DMSO (PFOS) or 0.1% DMSO (PFOA and PFNA) as solvent control and 10 µM forskolin as positive control. Forskolin stimulates cyclic AMP (cAMP) production in H295R cells and has similar effects to adrenocorticotrophic hormone (ACTH), the physiological stimulant of adrenocortical steroidogenesis (Gracia et al., 2006). The experiment was conducted independently three times for hormone analysis and cell viability assay, and five times for qRT-PCR.

Cell viability assay

Cell viability was estimated using Alamar blue assay. At the end of exposure, medium was collected and stored at -20°C for subsequent hormone analysis. Each well received 1 ml fresh medium containing 10% AlamarBlue (Invitrogen, Carlsbad, CA). Plates were incubated for a

further 3 hours, then a 100 μ l sample from each well was transferred in duplicates to a fresh 96-well ELISA plate (Falcon, Franklin Lakes, NJ, USA) and read in a Victor3TM spectrophotometer (Perkin Elmer, Shelton, USA) at 570 nm and 600 nm. Viability was expressed as percentage of solvent control.

Radioimmunoassay

Oestradiol-17 β , testosterone and cortisol secreted in the cell culture medium were measured using Coat-a-count radioimmunoassay (RIA) kits (Siemens, CA, LA, USA). Progesterone was measured using Spectria Progesterone RIA kit (Orion Diagnostica, Espoo, Finland). All kits were used according to manufacturer's instructions. The only modification was the use of fresh standard solutions prepared in medium from same batch as the cell cultures, rather than the supplied standards. The limits of detection were 20 pg/ml, 0.8 ng/ml, 0.10 ng/ml and 3.0 ng/ml and inter-assay coefficients of variation were 6.3%, 9.5%, 8.85% and 8.55% for oestradiol, progesterone, testosterone and cortisol respectively.

RNA preparation

Total RNA was isolated from cell culture plates using Qiagen RNeasy mini-kit (Qiagen, Crawley, UK) according to manufacturer's recommendations. Lysis buffer was added to wells and cells detached by scraping the bottom of the well with the pipette tip. Prior to RNA extraction, cell lysates were then centrifuged through Qiagen shredder spin columns (Qiagen, Crawley, UK). Samples were treated with DNase (RNase-Free DNase Set, Qiagen, Crawley, UK) on-column for 15 min at room temperature. After isolation, samples were eluted in RNase-free H₂O and stored at -70°C. RNA concentration and quality was determined using NanoDrop (Thermo-Scientific, Waltham, MA, USA) and Bioanalyzer (Agilent Technologies, Santa Clara, CA, USA) respectively.

Quantitative RT-PCR

GeNorm human detection kit and software (PrimerDesign Ltd, Southampton, UK) was used to predict the most stable reference genes. Out of the five genes tested (*B2M*, *β -actin*, *YWHAZ*, *ATP5B*, *GAPDH*), *YWHAZ* and *ATP5B* were the most stable were thus selected as reference genes in this study.

Primer sequences for *CYP11A*, *CYP11B2*, *CYP17*, *CYP19*, *CYP21*, *3 β HSD2*, *17 β HSD1*, *17 β HSD4*, *StAR* and *HMGR* were obtained from (Hilscherova et al., 2004). Primers for *CYP11B1* were designed by PrimerDesign Ltd (Southampton, UK). Primer sequences for *SF-1*, *DAX-1* and *ACTH-R* were designed in house using PrimerExpress version 1.5 (Applied Biosystems, Foster City, CA, USA) (Table 1). Specificities for all primer pairs were checked using nucleotideBLAST and primerBLAST (<http://blast.ncbi.nlm.nih.gov/Blast.cgi>). All primers were used in a working concentration of 200 nM. The primers were tested with regard to annealing temperature. PCR products were analysed on an ethidium bromide agarose gel to ensure the expected product was formed and to check for potential primer artefacts.

Synthesis of cDNA and quantitative PCR was performed using Superscript Platinum III Two-Step qRT-PCR with SYBR Green (Invitrogen, Paisley, UK) according to manufacturer's instructions. The cDNA synthesis was performed using a Peltier Thermal Cycler-225 (MJResearch, Waltham, MA, USA) and qRT-PCR was carried out using a DNA Engine Thermal Cycler with Chromo 4 Real-Time Detector (MJResearch, Waltham, MA, USA) and its software, Opticon Monitor 3, (Bio-Rad Laboratories, Hercules, CA, USA). In the reverse transcription reaction (cDNA synthesis), RNA samples were split into technical triplicates. A negative control that had not been subjected to reverse transcriptase enzyme was added per sample to ensure no DNA contamination of the RNA sample. Furthermore, a negative control where no template was added prior to cDNA synthesis was included for each primer pair to check for primer dimer formations and contamination. The amount of cDNA added to the qPCR reaction was 10 ng calculated from the original RNA concentration. Cycling conditions were 50°C, 2 min (UDG incubation), 95°C, 2 min (enzyme activation), followed by 40 cycles of 95°C in 15 sec, 62°C in 30 sec and 72°C in 30 sec. At the end of each run a melting curve from 65 – 90°C, read for 1 sec every 0.3°C, was included to monitor potential formation of primer dimers or products from genomic DNA and other DNA contamination. Negative controls without template and without RT enzyme were also monitored for any products formed during the qPCR reaction.

qPCR data analysis

Data was transferred from Opticon Monitor 3 software into an Excel spreadsheet for further processing. The ΔC_t was calculated from the difference in expression between the gene of

interest and the mean expression of the two reference genes. The $\Delta\Delta C_t$ was calculated from the difference in ΔC_t between cells exposed to solvent control and the chemical of interest. Fold change was calculated using $2^{-\Delta\Delta C_t}$.

Statistical analysis

Statistical analysis was performed using JMP software (SAS Institute Inc, Cary, NC, USA). The distribution of data was tested using the Shapiro-Wilk test. Oestradiol data was not normally distributed and was therefore log transformed prior to analysis. Cortisol, progesterone and testosterone had a satisfactory fit to the normal distribution and were used without log transformation. Differences in hormone secretion between solvent control and exposed cells were evaluated using Student's t-test. Dose-response relationships were assessed in General Linear Models (GLM) where measured hormone concentrations were included as dependent variables and experiment ($n = 3$) and dose of the relevant test compounds were entered as continuous variables. The hormone data from the highest exposure doses of PFOA and PFNA (600 μ M) were excluded from statistical analyses due to cytotoxic effects on the cell viability assay. Differences in gene expression were evaluated by the Student's t-test by comparing the log2 transformed $2^{-\Delta\Delta C_t}$ value of each exposure to solvent control. P values < 0.05 were considered statistically significant.

Results

Cell viability assay

Cell viability as assessed by Alamar blue conversion indicated a viability of $> 90\%$ in all tested doses of PFOS. Exposure to PFOA or PFNA at 600 μ M resulted in a dramatic decrease in viability, whereas cells exposed to the other doses of these two compounds had a viability of $> 90\%$ (Figure 2).

Hormone analyses

All three test compounds affected hormone production in H295R cells (Figure 3). Oestradiol levels followed a significantly positive dose-response relationship when cells were exposed to

PFOS. In addition, oestradiol was significantly elevated with the highest test dose (600 μ M) of PFOS. PFNA exposure between 0 and 60 μ M also resulted in a significantly positive dose-response relationship on oestradiol. At 60 μ M exposure, PFNA gave a numerically higher, though not statistically significant, concentration of oestradiol. Cells treated with PFOA did not show any significant differences in oestradiol secretion to cells treated with solvent control.

A positive dose-response relationship between progesterone and PFOS exposure was identified. Furthermore, the highest dose of PFOS (600 μ M) resulted in a significant increase in progesterone compared to solvent control. Although PFOA and PFNA exposure also resulted in a positive dose-response relationship on progesterone, no significant differences were seen when each dose was compared to the solvent control.

Testosterone levels were influenced by all three compounds. Cells exposed to PFOS showed a significantly positive relationship between testosterone concentration and increasing level of exposure. When comparing each dose to control, exposure to PFOS only increased testosterone in the highest concentration of the test compound, whereas testosterone was elevated by 6 μ M and 600 nM of PFOA. In contrast, PFNA exposure led to a significant negative dose-response relationship on testosterone. Additionally, the highest non cytotoxic dose of PFNA (60 μ M) resulted in a significant reduction of testosterone.

Cortisol was not significantly altered by exposure to PFOS and PFOA, although a negative dose-response relationship was identified with exposure to PFNA.

Gene expression

Only PFOA and PFNA induced changes in gene expression in the current study (Figure 4). All three test doses of PFOA decreased CYP11A levels. The other two test compounds did not affect mRNA levels of CYP11A. PFNA, however, significantly reduced HMGR expression, although only at 60 μ M. The expression of the other genes tested was not significantly altered by exposure to the test compounds.

Discussion

In the current study we demonstrated that PFOS, PFOA and PFNA have the capability to modulate steroidogenesis in the human adrenocortical H295R cell assay.

Whereas an increase in oestradiol was observed with the highest test dose (600 μ M) of PFOS, the PFOA and PFNA exposure at the same concentration gave a sharp decrease of all hormones measured due to cytotoxicity. At the second highest dose level tested (60 μ M) the oestradiol level appeared increased with PFNA exposure, however this was not statistically significant. Oestradiol was not altered by PFOA. In contrast, some previous *in vitro* studies on rat Leydig cells have reported a characteristic increase in oestradiol secretion induced by 10 μ M to 500 μ M PFOA (Biegel et al., 1995; Liu et al., 1996). In addition, elevated plasma oestradiol levels following PFOA exposure in rats has been detected (Cook et al., 1992). Previous investigations of the ability of other fluorochemicals, such as PFOS, to elevate oestradiol is limited to one *in vivo* report where PFOS increases serum oestradiol secretion in fathead minnows (*Pimephales promelas*), however the response appeared not to be dose-dependent and the results are therefore difficult to interpret (Oakes et al., 2005). Our findings, however, suggest oestradiol secretion can also be increased by PFOS. A possible explanation for the lack of response in oestradiol production with PFOA exposure in the current study could be the narrow response range limited by cytotoxicity in this particular *in vitro* model.

Interestingly, we observed no significant changes in mRNA levels of *CYP 19*, the gene encoding the P450 aromatase enzyme responsible for conversion of testosterone to oestradiol, with exposure to any of the test chemicals. Hence, the PFOS-induced increase in oestradiol levels could be a result of a different mechanism than control of gene expression.

In the current study, the exposure compounds affected medium testosterone concentration in different patterns. Whereas PFNA decreased testosterone, PFOS and PFOA exposure led to an increase in testosterone. Previous studies on rats have demonstrated a non significant negative dose response relationship between PFOA and testosterone, which became significant when rats were challenged by hCG (Biegel et al., 2001). A similar response was seen by exposure to 500 or 50 μ M PFOA in Leydig cells where testosterone levels were normal in the absence of hCG and dramatically reduced when hCG was present (Biegel et al., 1995; Liu et al., 1996). Although immature rainbow trout (*Oncorhynchus mykiss*) had an

elevated plasma testosterone following PFOS exposure, this was not seen in other fish species (Oakes et al., 2005). The increase in testosterone *in vitro* with the highest dose of PFOS observed in this study has, to the best of our knowledge, not been previously reported.

Perfluorinated compounds have previously been suggested to interfere with fatty acid metabolism and lipid transport in the livers of rare minnows (*Gobiocypris rarus*) (Wei et al., 2008) and appear to inhibit the transport of cholesterol into the mitochondria in rat Leydig cell cultures (Boujrad et al., 2000). However, such effects would lead to a reduced steroidogenesis due to a lack of substrate and could therefore not explain the elevated testosterone observed in the current study. A significantly elevated progesterone, in addition to oestradiol and testosterone suggest that PFOS could affect the steroid synthesis upstream of progesterone in our study.

In the current study, no significant effect on cortisol production was observed by any of the three test compounds. Little research has been attributed to the effect of PFCs on cortisol production. Two studies in which mice were exposed to PFNA, reported increased plasma cortisol levels (Fang et al., 2008; Fang et al., 2009). In one of these studies, an elevated level of ACTH was also found, suggesting that PFCs may affect cortisol production upstream of the adrenal gland in the hypothalamic-pituitary-adrenal axis. This could explain the lack of effects on cortisol synthesis observed in our study.

In summary, PFCs including PFOS, PFOA and PFNA are capable of modulating steroid secretion in human adrenocortical H295R cells. Oestradiol, progesterone and testosterone was elevated by the highest dose (600 μ M) of PFOS. PFOA and PFNA did not significantly alter oestradiol and progesterone secretion; however, testosterone was increased by PFOA and decreased by PFNA. Only PFOA and PFNA induced changes in gene expression resulting from up regulation CYP11A and HMGR respectively. We therefore propose that the changes in hormone secretion by PFOS are mediated by mechanisms other than modulation of gene expression of the steroidogenic enzymes investigated.

Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

References

- AMAP, 2009. Arctic Pollution 2009. Arctic Monitoring and Assessment Programme, Oslo. xi+83pp.
- Apelberg, B.J., Witter, F.R., Herbstman, J.B., Calafat, A.M., Halden, R.U., Needham, L.L., Goldman, L.R., 2007. Cord serum concentrations of perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA) in relation to weight and size at birth. *Environ. Health Perspect.* 115, 1670-1676.
- Biegel, L.B., Hurtt, M.E., Frame, S.R., O'Connor, J.C., Cook, J.C., 2001. Mechanisms of extrahepatic tumor induction by peroxisome proliferators in male CD rats. *Toxicol. Sci.* 60, 44-55.
- Biegel, L.B., Liu, R.C., Hurtt, M.E., Cook, J.C., 1995. Effects of ammonium perfluorooctanoate on Leydig cell function: in vitro, in vivo, and ex vivo studies. *Toxicol. Appl. Pharmacol.* 134, 18-25.
- Boujrad, N., Vidic, B., Gazouli, M., Culty, M., Papadopoulos, V., 2000. The peroxisome proliferator perfluorodecanoic acid inhibits the peripheral-type benzodiazepine receptor (PBR) expression and hormone-stimulated mitochondrial cholesterol transport and steroid formation in Leydig cells. *Endocrinology* 141, 3137-3148.
- Case, M.T., York, R.G., Christian, M.S., 2001. Rat and rabbit oral developmental toxicology studies with two perfluorinated compounds. *Int. J. Toxicol.* 20, 101-109.
- Cook, J.C., Murray, S.M., Frame, S.R., Hurtt, M.E., 1992. Induction of Leydig cell adenomas by ammonium perfluorooctanoate: a possible endocrine-related mechanism. *Toxicol. Appl. Pharmacol.* 113, 209-217.
- Fang, X., Feng, Y., Shi, Z., Dai, J., 2009. Alterations of cytokines and MAPK signaling pathways are related to the immunotoxic effect of perfluorononanoic acid. *Toxicol. Sci.* 108, 367-376.
- Fang, X., Zhang, L., Feng, Y., Zhao, Y., Dai, J., 2008. Immunotoxic effects of perfluorononanoic acid on BALB/c mice. *Toxicol. Sci.* 105, 312-321.
- Fei, C., McLaughlin, J.K., Tarone, R.E., Olsen, J., 2008. Fetal growth indicators and perfluorinated chemicals: a study in the Danish National Birth Cohort. *Am. J. Epidemiol.* 168, 66-72.
- Fromme, H., Tittlemier, S.A., Volkel, W., Wilhelm, M., Twardella, D., 2009. Perfluorinated compounds--exposure assessment for the general population in Western countries. *Int. J. Hyg. Environ. Health* 212, 239-270.
- Gazdar, A.F., Oie, H.K., Shackleton, C.H., Chen, T.R., Triche, T.J., Myers, C.E., Chrousos, G.P., Brennan, M.F., Stein, C.A., La Rocca, R.V., 1990. Establishment and characterization of a human adrenocortical carcinoma cell line that expresses multiple pathways of steroid biosynthesis. *Cancer Res.* 50, 5488-5496.

- Gracia, T., Hilscherova, K., Jones, P.D., Newsted, J.L., Zhang, X., Hecker, M., Higley, E.B., Sanderson, J.T., Yu, R.M., Wu, R.S., Giesy, J.P., 2006. The H295R system for evaluation of endocrine-disrupting effects. *Ecotoxicol. Environ. Saf* 65, 293-305.
- Hilscherova, K., Jones, P.D., Gracia, T., Newsted, J.L., Zhang, X., Sanderson, J.T., Yu, R.M., Wu, R.S., Giesy, J.P., 2004. Assessment of the effects of chemicals on the expression of ten steroidogenic genes in the H295R cell line using real-time PCR. *Toxicol. Sci.* 81, 78-89.
- Lau, C., Thibodeaux, J.R., Hanson, R.G., Rogers, J.M., Grey, B.E., Stanton, M.E., Butenhoff, J.L., Stevenson, L.A., 2003. Exposure to perfluorooctane sulfonate during pregnancy in rat and mouse. II: postnatal evaluation. *Toxicol. Sci.* 74, 382-392.
- Liu, R.C., Hahn, C., Hurtt, M.E., 1996. The direct effect of hepatic peroxisome proliferators on rat Leydig cell function in vitro. *Fundam. Appl. Toxicol.* 30, 102-108.
- Nakata, H., Kannan, K., Nasu, T., Cho, H.S., Sinclair, E., Takemurai, A., 2006. Perfluorinated contaminants in sediments and aquatic organisms collected from shallow water and tidal flat areas of the Ariake Sea, Japan: environmental fate of perfluorooctane sulfonate in aquatic ecosystems. *Environ. Sci. Technol.* 40, 4916-4921.
- Oakes, K.D., Sibley, P.K., Martin, J.W., MacLean, D.D., Solomon, K.R., Mabury, S.A., Van Der Kraak, G.J., 2005. Short-term exposures of fish to perfluorooctane sulfonate: acute effects on fatty acyl-coa oxidase activity, oxidative stress, and circulating sex steroids. *Environ. Toxicol. Chem.* 24, 1172-1181.
- Oakes, K.D., Sibley, P.K., Solomon, K.R., Mabury, S.A., Van Der Kraak, G.J., 2004. Impact of perfluorooctanoic acid on fathead minnow (*Pimephales promelas*) fatty acyl-CoA oxidase activity, circulating steroids, and reproduction in outdoor microcosms. *Environ. Toxicol. Chem.* 23, 1912-1919.
- Ohmori, K., Kudo, N., Katayama, K., Kawashima, Y., 2003. Comparison of the toxicokinetics between perfluorocarboxylic acids with different carbon chain length. *Toxicology* 184, 135-140.
- Olsen, G.W., Burris, J.M., Ehresman, D.J., Froehlich, J.W., Seacat, A.M., Butenhoff, J.L., Zobel, L.R., 2007. Half-life of serum elimination of perfluorooctanesulfonate, perfluorohexanesulfonate, and perfluorooctanoate in retired fluorochemical production workers. *Environ. Health Perspect.* 115, 1298-1305.
- Skakkeback, N.E., Rajpert-De, M.E., Main, K.M., 2001. Testicular dysgenesis syndrome: an increasingly common developmental disorder with environmental aspects. *Hum. Reprod.* 16, 972-978.
- Thibodeaux, J.R., Hanson, R.G., Rogers, J.M., Grey, B.E., Barbee, B.D., Richards, J.H., Butenhoff, J.L., Stevenson, L.A., Lau, C., 2003. Exposure to perfluorooctane sulfonate during pregnancy in rat and mouse. I: maternal and prenatal evaluations. *Toxicol. Sci.* 74, 369-381.
- Wei, Y., Liu, Y., Wang, J., Tao, Y., Dai, J., 2008. Toxicogenomic analysis of the hepatic effects of perfluorooctanoic acid on rare minnows (*Gobiocypris rarus*). *Toxicol. Appl. Pharmacol.* 226, 285-297.

Yamashita, N., Kannan, K., Taniyasu, S., Horii, Y., Petrick, G., Gamo, T., 2005. A global survey of perfluorinated acids in oceans. *Mar. Pollut. Bull.* 51, 658-668.

Figure 1: Skeletal formulas of the PFOS (A), PFOA (B) and PFNA (C) molecules.

Figure 2: Mean (SE) cell viability of H295R cells assessed with Alamar blue assay for each concentration of PFOS, PFOA and PFNA expressed as % of solvent control.

Figure 3: Mean (SE) concentrations of steroid hormones measured in medium of H295R cells after 48 hour exposure to six concentrations of PFCs. * denotes means significantly different from solvent control ($P < 0.05$) † denotes cell viability $< 90\%$.

Figure 4: Mean (SE) fold change in gene expression of CYP11A and HMGR following 48 hour exposure to three concentrations of PFCs. * denotes significantly different from solvent control ($P < 0.05$).

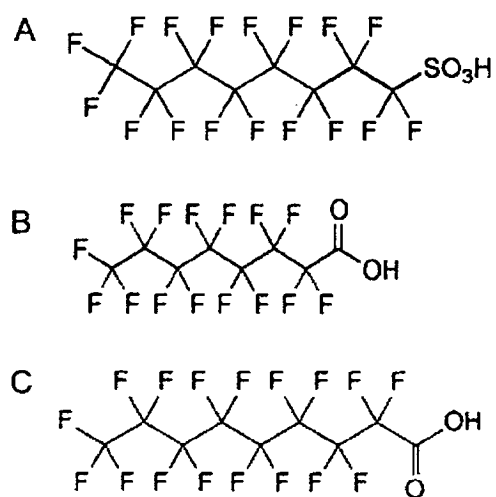
Figure 1.

Figure 2.

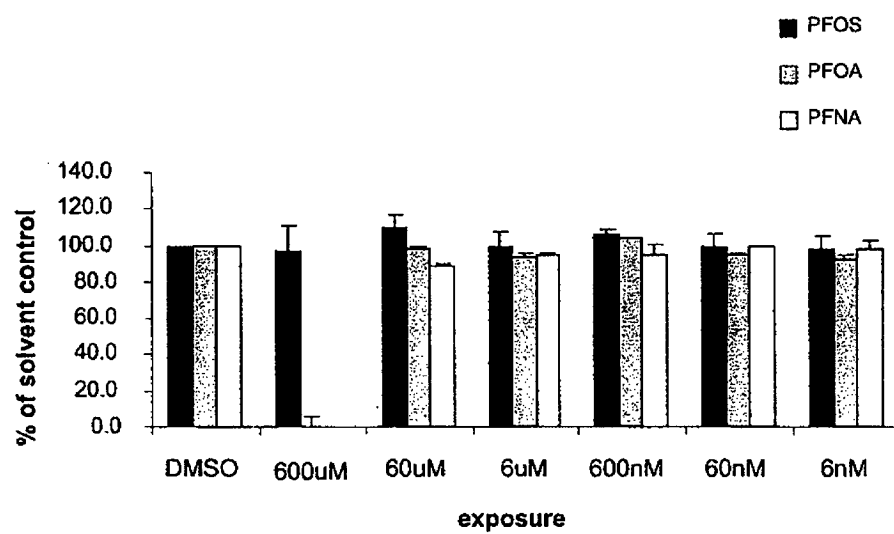
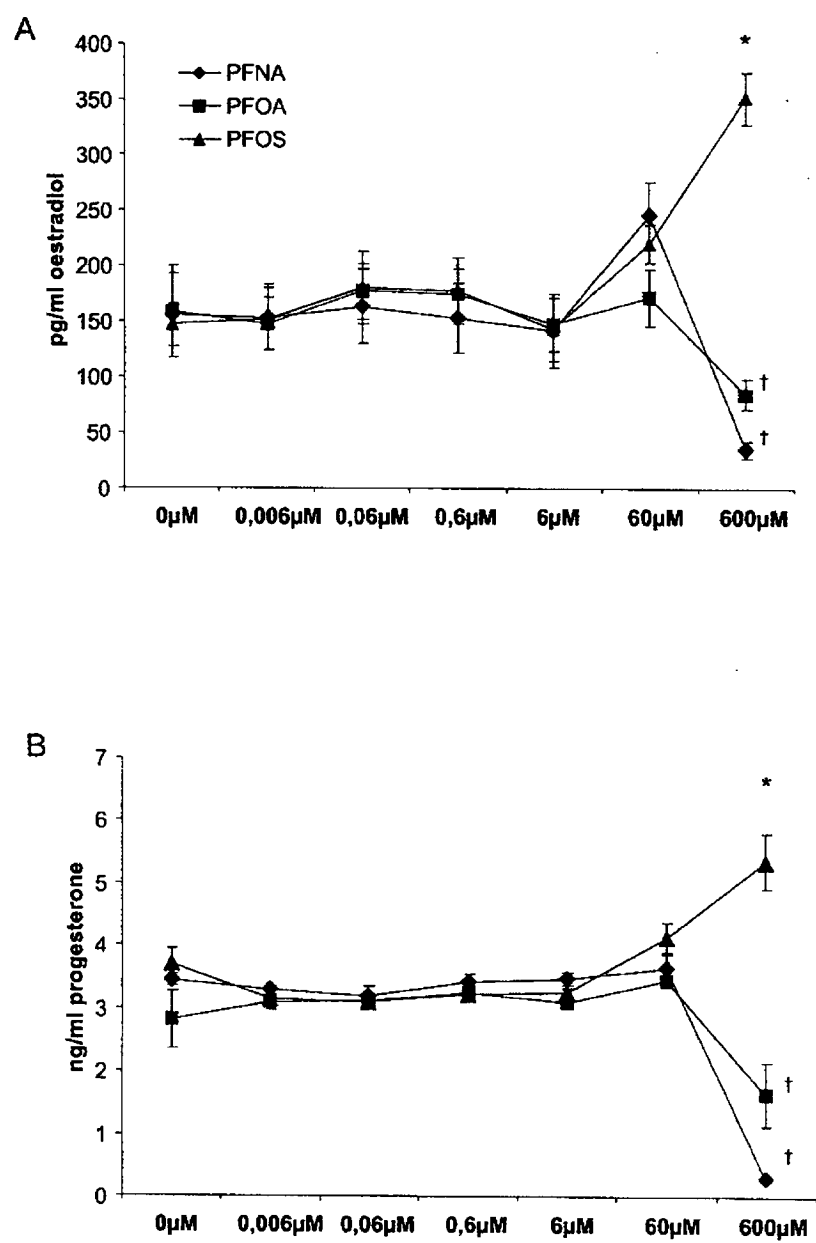


Figure 3.



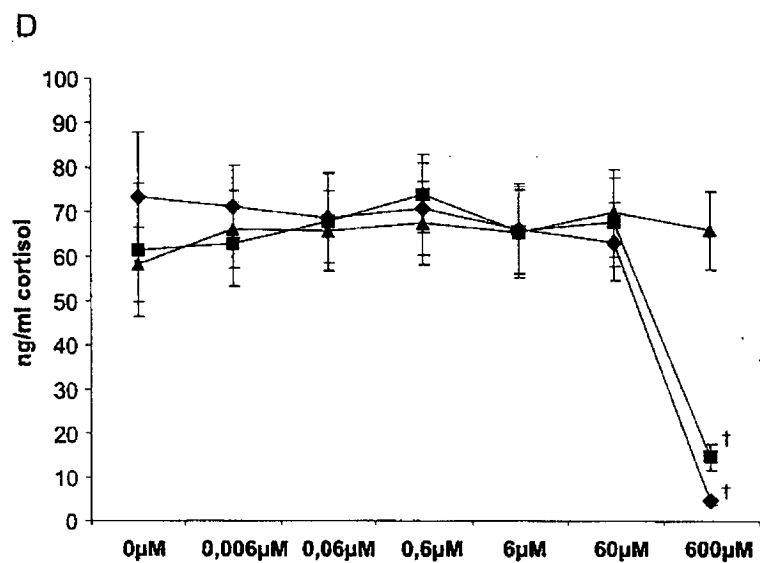
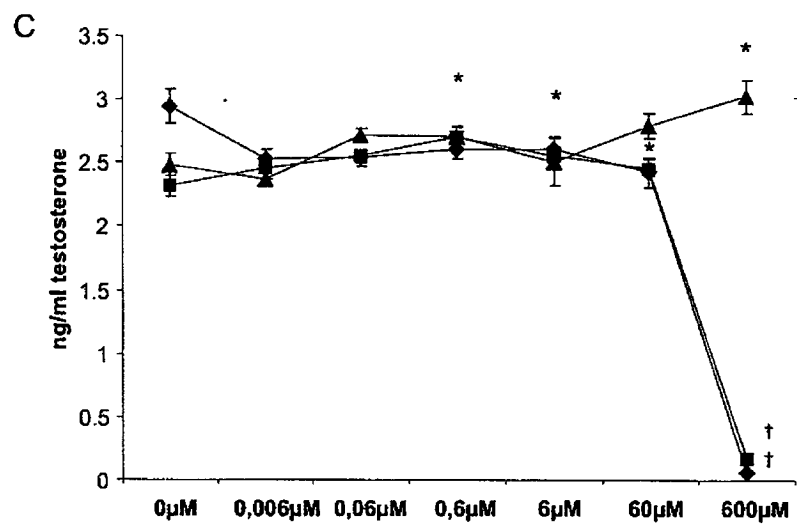


Figure 4.

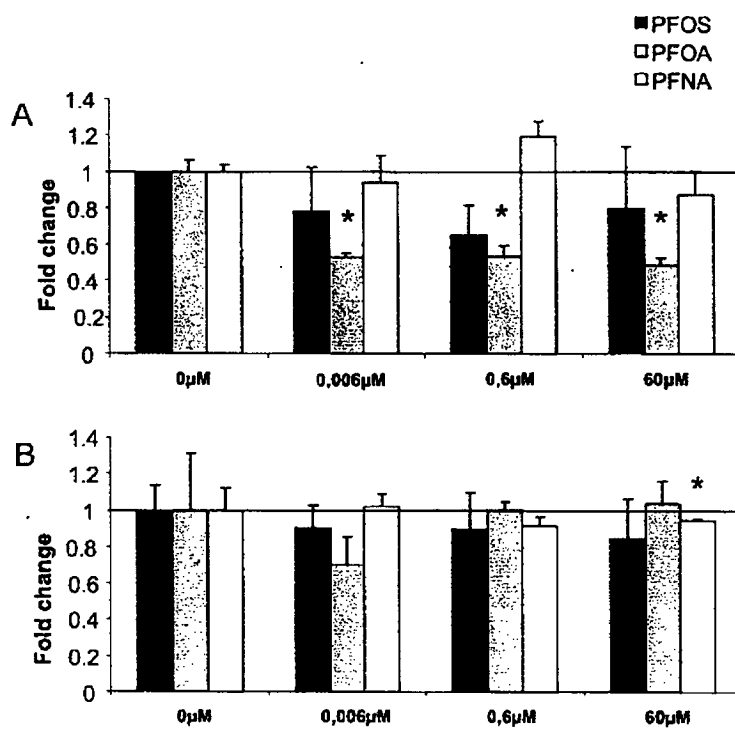


Table 1: Primer sequences used for quantitative RT-PCR. Primer sequences for *CYP11A*, *CYP11B2*, *CYP17*, *CYP19*, *CYP21*, *3 β HSD2*, *17 β HSD1*, *17 β HSD4*, *StAR* and *HMGR* are given in Hilscherova et al. (2004). *CYP11B1* was designed by PrimerDesign Ltd.

Gene abbreviation	Gene name	Gene product abbreviation	Gene product	Forward primer	Reverse primer
<i>NR5A1</i>	Nuclear receptor subfamily 5 group A, member 1	SF-1	Steroidogenic factor -1	TGGAGCTGACCACAGTGGC	GGAACTTCAAATCCAGGCTGAA
<i>NR0B1</i>	Nuclear receptor subfamily 0 group B, member 1	DAX-1	Dosage-sensitive sex reversal, adrenal hypoplasia critical region, on chromosome X, gene 1	CTCTTTAACCCGGACGTGCC	GGTGGCTCATCTCGGTGTGT
<i>ACTHR</i>	Adrenocorticotrophic hormone receptor	ACTHR	Adrenocorticotrophic hormone receptor	GGAGGCAAAACACCACCTCATT	CACTCTTCCATTGCTGGCATCT
<i>ATP5B</i>	ATP synthase subunit beta, mitochondrial		ATP synthase subunit beta, mitochondrial	ACCATCAAAGGATTCCAGCA	GCTTTTGCCACAGCTTCTTC
<i>YWHAZ</i>	Tyrosine 3-monooxygenase/tryptophan 5-monooxygenase activation protein, zeta polypeptide	14-3-3 protein zeta/delta	Tyrosine 3-monooxygenase/tryptophan 5-monooxygenase activation protein, zeta polypeptide	CCCAATGCTTCACAAGCAGAG	CCTTCTTGTCATCACCAGCG

Public Health Assessment

Public Comment Release

**PERFLOUROCHEMICAL CONTAMINATION IN SOUTHERN
WASHINGTON COUNTY, NORTHERN DAKOTA COUNTY, AND
SOUTHEAST RAMSEY COUNTY, MINNESOTA**

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

EPA FACILITY ID: MND980679385

**Prepared by
Minnesota Department of Health**

August 25, 2010

COMMENT PERIOD ENDS: October 12, 2010

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

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

This Public Health Assessment-Public Comment Release was prepared by ATSDR pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) section 104 (i)(6) (42 U.S.C. 9604 (i)(6)), and in accordance with our implementing regulations (42 C.F.R. Part 90). In preparing this document, ATSDR's Cooperative Agreement Partner has collected relevant health data, environmental data, and community health concerns from the Environmental Protection Agency (EPA), state and local health and environmental agencies, the community, and potentially responsible parties, where appropriate. This document represents the agency's best efforts, based on currently available information, to fulfill the statutory criteria set out in CERCLA section 104 (i)(6) within a limited time frame. To the extent possible, it presents an assessment of potential risks to human health. Actions authorized by CERCLA section 104 (i)(11), or otherwise authorized by CERCLA, may be undertaken to prevent or mitigate human exposure or risks to human health. In addition, ATSDR's Cooperative Agreement Partner will utilize this document to determine if follow-up health actions are appropriate at this time.

This document has previously been provided to EPA and the affected state in an initial release, as required by CERCLA section 104 (i) (6) (H) for their information and review. Where necessary, it has been revised in response to comments or additional relevant information provided by them to ATSDR's Cooperative Agreement Partner. This revised document has now been released for a 30-day public comment period. Subsequent to the public comment period, ATSDR's Cooperative Agreement Partner will address all public comments and revise or append the document as appropriate. The public health assessment will then be reissued. This will conclude the public health assessment process for this site, unless additional information is obtained by ATSDR's Cooperative Agreement Partner which, in the agency's opinion, indicates a need to revise or append the conclusions previously issued.

Use of trade names is for identification only and does not constitute endorsement by the U.S. Department of Health and Human Services.

Please address comments regarding this report to:

Agency for Toxic Substances and Disease Registry
Attn: Records Center
1600 Clifton Road, N.E., MS F-09
Atlanta, Georgia 30333

You May Contact ATSDR Toll Free at
1-800-CDC-INFO or
Visit our Home Page at: <http://www.atsdr.cdc.gov>

Perfluorochemical Contamination in Southern Washington County,
Northern Dakota County and Southeastern Ramsey County, Minnesota

Public Comment

PUBLIC HEALTH ASSESSMENT

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

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

EPA FACILITY ID: MND980679385

Prepared by:

Minnesota Department of Health
Under Cooperative Agreement with the
U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry

This information is distributed by the Agency for Toxic Substances and Disease Registry for public comment under applicable information quality guidelines. It does not represent and should not be construed to represent final agency conclusions or recommendations.

FOREWORD

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

- *Evaluating exposure:* MDH scientists begin by reviewing available information about environmental conditions at the site. The first task is to find out how much contamination is present, where it is found on the site, and how people might be exposed to it. Usually, MDH does not collect its own environmental sampling data (although this case is an exception). Rather, MDH relies on information provided by the Minnesota Pollution Control Agency (MPCA), the Minnesota Department of Agriculture (MDA), the US Environmental Protection Agency (EPA), private businesses, and the general public.
- *Evaluating health effects:* If there is evidence that people are being exposed—or could be exposed—to environmental contaminants, MDH scientists will take steps to determine whether that exposure could be harmful to human health. MDH's report focuses on public health—that is, the health impact on the community as a whole. The report is based on existing scientific information.
- *Developing recommendations:* In this report, MDH outlines conclusions regarding any potential health threat posed by a site and offers recommendations for reducing or eliminating human exposure to pollutants. The role of MDH is primarily advisory. For that reason, the evaluation report will typically recommend actions to be taken by other agencies—including EPA and MPCA. If, however, an immediate health threat exists, MDH will issue a public health advisory to warn people of the danger and will work to resolve the problem.
- *Soliciting community input:* The evaluation process is interactive. MDH starts by soliciting and evaluating information from various government agencies, the individuals or organizations responsible for the site, and community members living near the site. Any conclusions about the site are shared with the individuals, groups, and organizations that provided the information. Once an evaluation report has been prepared, MDH seeks feedback from the public. *If you have questions or comments about this report, we encourage you to contact us.*

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

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

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

Table of Contents

FOREWORD	2
Table of Contents	3
Summary	4
Introduction	5
Background	7
3M-Woodbury Disposal Site Description and History	7
Geology / Hydrogeology	8
PFC Analysis	11
Evaluation of PFCs in Drinking Water	11
Community Well Monitoring	12
Private Well Sampling	14
PFC-Related Investigations at the 3M-Woodbury Disposal Site	15
MPCA – 3M Consent Order for PFC Disposal Sites	17
Remedial Options for the 3M-Woodbury Disposal Site	18
Site Visits	19
Demographics, Land Use, and Natural Resources	20
General Regional Issues	21
Community Concerns	21
Evaluation of Environmental Fate and Exposure Pathways	21
Introduction	21
Environmental Fate	22
Evaluation of Impacts on Groundwater	23
Exposure through Private Wells	27
Exposure through Public Water Supplies	28
Exposure through other Pathways	29
Public Health Implications of PFC Exposure	29
Summary of Toxicological Information	30
Summary of Human Exposure Information	32
Discussion of the Public Health Implications of PFC Exposure	34
Health Outcome Data Review	35
Child Health Considerations	36
Conclusions	36
Recommendations	37
Public Health Action Plan	37
References	38
Preparers of Report	47
CERTIFICATION	48
Glossary	49

Appendices

Appendix 1: Figures

Appendix 2: Tables

Summary

INTRODUCTION	The Minnesota Department of Health's (MDH) mission is to protect, maintain, and improve the health of all Minnesotans. For communities affected by perfluorochemicals (PFCs) in their drinking water, MDH's goal is to protect people's health by providing health information the community needs to take actions to protect their health. MDH also monitors public water supplies for PFCs, and advises the MPCA on actions that can be taken to protect public health. PFC-containing wastes were disposed of by 3M in land disposal sites in Oakdale, Lake Elmo, and Woodbury, Minnesota. PFCs were released to the groundwater from these sites, possibly shortly after the disposal occurred, resulting in contamination of nearby drinking water wells. There were also possible air emissions during the handling, disposal, or burning of waste, and people could have come into direct contact with the waste. PFCs continue to be detected in public and private wells across a wide area of south Washington County, and in parts of northern Dakota County and southeastern Ramsey County.
OVERVIEW	MDH reached two important conclusions in this Public Health Assessment.
CONCLUSION 1	MDH cannot conclude whether drinking or breathing PFCs in water or air or contact with PFC-containing wastes in the past harmed people's health.
BASIS FOR DECISION	A pilot biomonitoring study conducted in residents of Oakdale, Lake Elmo, and Cottage Grove indicated levels of PFCs in blood above national averages. While available evidence suggests that these PFC levels are unlikely to cause adverse health effects, there is no information available regarding PFC levels in resident's blood or in drinking water in the past.
NEXT STEPS	• MDH should consider additional biomonitoring studies to evaluate PFC levels in area residents exposed to PFOA and PFOS in drinking water.
CONCLUSION 2	MDH concludes that currently, drinking water from public or private wells that contain PFCs is not expected to harm people's health.
BASIS FOR DECISION	Current exposures to PFCs are below health-based exposure limits because bottled water or whole-house activated carbon filters have been provided at 29 homes that were issued a drinking water well advisory by MDH. No one is drinking water that has PFCs at levels above health concern. Remediation actions to address PFCs at the waste disposal sites are in the early implementation stages by 3M and the MPCA.

NEXT STEPS

-
- 3M should continue to follow the requirements of the Consent Order to implement the selected remediation options for soil and groundwater at the 3M-Woodbury Disposal Site.
 - People should avoid trespassing on the 3M-Woodbury Disposal Site, especially during remediation activities.
 - Extensions of the Cottage Grove municipal water supply to serve areas where private wells contain levels of PFCs in excess of MDH HRLs or HBVs should be considered.
 - Monitoring of selected private wells in the affected area should continue under agreed upon sampling plans to track changes in the plume and monitor for changes in concentration in individual wells.
 - 3M should ensure the monitoring network at the disposal site provides adequate information regarding water quality in the "high transmissivity zone" of the Prairie du Chien aquifer.
-

FOR MORE INFORMATION

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

Introduction

The 3M Company (3M; formerly Minnesota Mining and Manufacturing Company) began research and development of perfluorochemicals (PFCs) at its Cottage Grove, Minnesota facility in southern Washington County, Minnesota in the late 1940s; with commercial production of various PFC compounds occurring from the early 1950s until 2002. Production of one PFC, perfluorobutanoic acid (PFBA) ceased in 1998. The production or use of other PFC-related compounds in research or pilot scale projects continues today.

MDH prepared a Health Consultation focusing on PFC releases at the Cottage Grove facility (MDH 2005). Wastes from the electrofluorochemical PFC production process, including production wastes and wastewater treatment plant sludge, were disposed of at the Cottage Grove facility and several known disposal sites identified by 3M in Washington County (Weston 2005). The names of these facilities, the types of wastes disposed of, and the estimated time of the disposal were formally provided to the Minnesota Pollution Control Agency (MPCA) by 3M in June of 2005 and are listed below:

Disposal Facilities in Washington County that Received 3M PFC Wastes

Disposal Facility	Waste Disposed	Estimated Dates
3M-Oakdale Disposal Site	Liquid and solid industrial waste	1956 – 1960
3M-Woodbury Disposal Site	Liquid and solid industrial waste	1960 – 1966
Washington County Landfill, Lake Elmo	Wastewater treatment plant sludge, incinerator scrubber sludge and ash, iron oxide sludge	1971 – 1974
3M-Cottage Grove Facility	Industrial wastes, ash, sludge	1950s – 1970s
Pigs Eye Dump, St. Paul*	Incinerator scrubber sludge and ash (also received municipal waste water treatment plant incinerator ash which may have contained PFCs)	1971

*This site is actually in Ramsey Co., near the border with Washington Co.

The general locations of the above disposal sites, along with the 3M Cottage Grove facility are shown in Figure 1 (all figures can be found in Appendix 1). A separate report was issued in August 2008 evaluating PFC waste disposal at the 3M-Oakdale Disposal Site and the Washington County Landfill in Lake Elmo and its potential impact on those communities (MDH 2008). An updated report on the 3M-Cottage Grove facility and PFC impacts to the Mississippi River is planned. The Pigs Eye Dump is included in the list because it was identified by 3M as a disposal site that received PFC-containing wastes; PFCs have been detected in groundwater and surface water at the site. It is not included in the Consent Order agreement between 3M and the MPCA (see below).

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

PFCs unique physical and chemical properties allow them to move easily through the environment (EPA 2002, OECD 2002, ATSDR 2009). As a result, they have been found globally at low levels. Some PFCs are bio-accumulative (i.e., build up in living organisms) and one PFC, perfluorooctane sulfonate (PFOS) has been detected in the blood and tissues of humans and animals from virtually all parts of the world. It should be noted that while the use of PFCs has been restricted in the United States by the EPA to certain products for which there is no adequate substitute, they are still manufactured and used in other countries around the world, including Italy, Russia, China, Japan, and Korea.

Toxicological research on PFCs is ongoing in government, industry and academia. Published studies show that animal exposure to PFCs at high concentrations adversely

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

PFCs disposed of at the sites identified above have impacted soil, groundwater, surface water, sediments, biota, and nearby drinking water wells, both public and private. MDH has prepared this report in response to requests from the MPCA, Washington County, local communities, and citizens to:

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

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

Background

3M-Woodbury Disposal Site Description and History

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

From 1960 to 1966, private contractors operated the site for a short time, and then sold it to 3M who used the site to dispose of liquid and solid industrial wastes (solvents, tapes, plastics, and resins) generated at their Cottage Grove facility. The wastes were buried in trenches. In addition, municipal wastes from the cities of Woodbury and Cottage Grove were disposed of in two separate areas of the site (approximately five acres total) from 1964-1966 (Weston 2008a).

Groundwater contamination at the site was first detected in 1966 when volatile organic compounds (VOCs, mostly solvents) were found in groundwater monitoring wells on the site and a private well located immediately west of the site. A groundwater extraction

system was completed at the site by 1973 and has operated continuously since. The extracted groundwater is pumped via a pipeline to the 3M Cottage Grove manufacturing plant, where it is used as cooling or process water and then discharged to the Mississippi River.

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

In 1992, 3M entered the site into the MPCA's Voluntary Investigation and Cleanup (VIC) program, under which additional investigation and response actions were taken to further address contamination at the site. In 1996, 3M backfilled open areas and regraded the site, placed a cap consisting of a minimum of 24 inches of clean soil over the former disposal areas, and filed an institutional control on the property deed to restrict future land use at the property.

Geology / Hydrogeology

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

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

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

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

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

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

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

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

Groundwater in the St. Peter migrates primarily through the pore spaces between the sand grains, although fractures and solution cavities are present in the St. Peter, particularly near the buried bedrock valleys (Alexander 2007; Runkel et al, 2007). Such solution

cavities may create pathways through which groundwater and contaminants migrate more quickly than is typically observed in the St. Peter.

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

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

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

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

PFC Analysis

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

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

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

Water samples are collected in clean 250 milliliter polyethylene bottles. Care is taken to avoid the use of products that could contain PFCs during sampling. The analysis is conducted using a combined high-pressure liquid chromatography tandem mass spectrometry (LC/MS/MS) method, using radio-labeled PFOA and PFOS standards. Each sample is spiked in the lab with a known quantity of labeled standard. The recovery rate of the added standard must be within $\pm 30\%$ of the known labeled standard concentration added to the sample to meet quality control requirements. In September 2007, the MDH Public Health Laboratory issued new formal reporting levels for the seven PFCs of 0.3 parts per billion (ppb), or 300 parts per trillion (ppt) in water (P. Swedenborg, personal communication, 2007). PFCs detected at concentrations between 50 and 300 ppt are reported as estimated, or "J" flagged values.

Evaluation of PFCs in Drinking Water

MDH has established Health Risk Limits (HRLs) in Minnesota Rules of 0.3 ppb for both PFOS and PFOA. In February 2008, MDH established a Health Based Value (HBV) for PFBA of 7 ppb based in part on toxicological and pharmacokinetic studies completed in late 2007 by EPA and 3M. A HBV is a criterion that is established using the same risk assessment procedures and policies used for HRLs, but that has not yet been promulgated through rulemaking. Information on MDH HRLs and HBVs, including the specific methodology, exposure assumptions, and references, is available at <http://www.health.state.mn.us/divs/eh/risk/guidance/gw/index.html>.

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

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

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

Community Well Monitoring

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

In mid-2006, the MDH Public Health Laboratory expanded the list of PFCs for analysis and lowered the analytical detection limits as described previously. Low levels of PFBA (0.1 to 0.3 ppb) were detected in several Woodbury community wells during routine sampling. No other PFCs were detected. By fall of 2006, it was found that low levels (0.1

to 0.5 ppb) of PFBA were present in all 16 Woodbury community wells. Although the presence of PFBA in those wells appears to be the result of PFCs moving away from the 3M waste disposal sites in Oakdale and Lake Elmo (MDH 2008), the city of Cottage Grove requested that their community wells be re-tested for PFCs using the expanded PFC list. In December 2006, PFBA was detected in all of the Cottage Grove municipal wells at concentrations ranging from 0.3 to 1.5 ppb. The detection of PFBA at concentrations higher than those found in the Woodbury city wells suggested that the PFCs in the Cottage Grove city wells were from another source located in or near Cottage Grove, rather than the 3M-Oakdale Disposal Site and Washington County Landfill.

The detections of PFBA in community wells in Woodbury and Cottage Grove triggered sampling of other community wells in south Washington and northern Dakota Counties to determine if they had been impacted by PFCs, beginning in January 2007. This sampling eventually included Woodbury, Cottage Grove, Newport, St. Paul Park, Hastings and South St. Paul; PFBA was detected in some or all of the community wells in each of these cities. Low levels (0.44 ppb on average) of PFBA were also found in a community water supply well serving a housing development in Cottage Grove known as Eagles Watch, in the eastern part of the city. Sampling was conducted on a monthly basis in 2007 to determine if the levels of PFBA were changing. Sampling frequency changed from monthly to quarterly in 2008 when it became apparent that the levels were stable or even declining slightly.

The average and range of concentrations of PFBA detected in the wells serving each community's water supply system from January 2007 through September 2009 are shown in the table below.

Community Well PFBA Data, Jan. 2007 – Sept. 2009

City	No. of Comm. Wells	No. of Comm. Wells w/PFBA	Range (ppb)	Avg. of all Detects (ppb)
Cottage Grove	11	11	0.30 – 1.78	1.00
Hastings	5	5	0.12 – 0.67	0.26
Newport	2	2	0.17 – 0.69	0.42
St. Paul Park	3	3	0.96 – 2.30	1.36
South St. Paul	5	3	0.08 – 0.37	0.20
Woodbury	17	17	0.07 – 0.55	0.27

PFBA is the only PFC that has been detected in community wells in Newport and South St. Paul. Low levels of PFBS (up to 0.32 ppb) and PFHxS (up to 0.16 ppb) have been consistently detected in three Cottage Grove community wells; trace amounts of PFPeA and PFHxA (less than 100 ppt) have also been intermittently detected in various Cottage Grove community wells (see Table 4, Appendix 2). PFOA has been intermittently detected at approximately 50 ppt in one Hastings well and one St. Paul Park well. In Woodbury, PFHxS has been intermittently detected at approximately 50 ppt in one well, and PFOA was detected once at 50 ppt in one community well.

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

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

Private Well Sampling

In 2005, as a result of being informed by 3M that PFC containing wastes may have been disposed of at the 3M-Woodbury Disposal Site, 3M's consultant, Weston Solutions, Inc. (Weston) collected groundwater samples from monitoring wells at the site (Weston, 2007a). PFOA, PFOS, PFBS and PFHxS were reported in monitoring wells on the landfill property in both the Prairie du Chien and Jordan aquifers and the water discharged from the pump-out wells at the site (Table 1, Appendix 2). The samples were not analyzed for PFBA.

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

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

Given the scope of the potentially affected area, a private well sampling program was developed to provide rapid information regarding how far and how deep the PFC contamination had spread and where the highest concentrations were located. The state's County Well Index (CWI) was used to identify wells with geologic information in recorded driller's logs. Wells drawing water from each of the drinking water aquifers

used in the investigation area were selected to provide geographic coverage of all of the neighborhoods in the potentially affected communities. More intensive sampling (i.e. sampling a higher density of wells) was undertaken in the following areas: 1) closest to and downgradient of the disposal site, 2) where PFBA concentrations were highest, 3) where the bedrock geology is complicated by faults and/or bedrock valleys, and 4) where spatial trends in PFBA concentrations were unpredictable. Sampling proceeded in an iterative fashion – with each round of sampling results informing decisions about the next round of samples. This allowed the MPCA and MDH to refine the sampling plan to identify and focus on areas of greatest potential health concern. By the end of 2008, water samples had been collected from over 900 residential and 56 business wells in the affected communities. A general summary of sampling results is provided in Table 2 (in Appendix 2).

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

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

PFC-Related Investigations at the 3M-Woodbury Disposal Site

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

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

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

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

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

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

In 2006-2007, twenty additional monitoring wells were installed to provide additional information about PFC distribution at the site and to monitor for any movement of contaminants away from the site (Weston, 2007a). Sampling has shown that PFCs are present in multiple monitoring wells at the site, primarily downgradient of the waste (Table 3, Appendix 2). The highest levels have consistently been found in monitoring well MW-2, near the Northeast Disposal Area (Figure 3). PFC concentrations at the site

have been stable over the limited sampling period, and generally low concentrations are detected in wells near the boundaries of the 3M property. However, only one of the monitoring wells completed in the Prairie du Chien (well MW-S06PC) actually intersects the "high transmissivity zone" identified by Tipping, et al (2006), and it is located upgradient of the waste disposal areas. 3M's consultant identified this zone as likely carrying the majority of flow in the Prairie du Chien (Weston, 2008a). The absence of monitoring wells intercepting this zone means it is possible that a critical pathway for PFC migration may be missed by the monitoring network.

A groundwater remediation system is in operation at the 3M-Woodbury Disposal site. The system was originally installed to control migration of VOCs. The groundwater remediation system includes four gradient control/recovery wells. The system pumps an average of 4.6 million gallons of water per day from the wells. The water is discharged through a six mile long pipeline to the 3M-Cottage Grove facility where it is primarily used for non-contact cooling water prior to discharge to the Mississippi River under a permit issued by the MPCA. A hydraulic conductivity zone analysis was conducted by Weston (2007b) which concluded the gradient control wells are fully containing the contamination on-site. This is consistent with previous evaluations of the gradient control system. However, critical gaps in the monitoring network, as described above, make it impossible to verify this.

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

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

MPCA – 3M Consent Order for PFC Disposal Sites

At the MPCA April 24, 2007 Citizens' Board meeting, the Board was asked to approve a series of enforcement actions under the state Superfund law to compel 3M to respond to PFC contamination from three known PFC disposal sites: the 3M-Cottage Grove facility, the 3M-Woodbury Disposal Site, and the 3M-Oakdale Disposal Site. The former

Washington County Landfill was not included because under the MPCA Closed Landfill Program, the MPCA has assumed responsibility for the site.

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

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

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

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

Remedial Options for the 3M-Woodbury Disposal Site

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

- Sitewide Alternative 1 – No action.
- Sitewide Alternative 2 – Institutional controls, access restriction and groundwater monitoring.
- Groundwater Alternative 1 – Continued groundwater recovery with GAC treatment, as necessary, to meet appropriate discharge criteria.
- Soil Alternative 1 – Excavation of the former Northeast Disposal Area trenches; disposal at an existing off-site landfill.

- Soil Alternative 2 – Excavation of the former Northeast Disposal Area trenches; disposal at an existing off-site landfill; and 4 feet (minimum) cover over selected areas in the former Main Disposal Area.
- Soil Alternative 3 – Excavation of the former Northeast Disposal Area trenches and selective removal of soil which exceeds Industrial SRVs from trenches in the former Main Disposal Area; disposal at an existing off-site landfill.

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

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

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

3M began cleanup work at the site in the summer of 2009. To date, 19,100 tons of excavated soil and waste has been transported to the SKB Landfill in Rosemount, Minnesota, where a specially constructed, dedicated cell has been constructed to receive the soil and waste from the Woodbury and Oakdale sites, and waste that was buried at the 3M-Cottage Grove facility. An additional 4,100 tons of excavated soil and waste has been transported for out of state disposal at a permitted hazardous waste facility due to the presence of other regulated contaminants.

Site Visits

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

The 3M-Woodbury Disposal Site is partially fenced, and access from County Road 19 is limited by several locked gates. “No Trespassing” signs are also prominently placed

along the fence and gates. 3M has regular security patrols at and around the site. 3M allows some of the land to be used for agricultural purposes, and employee recreational activity clubs also use portions of the site.

Demographics, Land Use, and Natural Resources

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

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

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

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

In August 2007 MDH issued a press release announcing revised fish consumption advice for several lakes in the Twin Cities metro area, including Ravine Lake, which is located within Cottage Grove Ravine Regional Park. The press release was issued due to the detection of PFOS in fish tissue samples collected by the MPCA from several lakes at levels high enough to warrant revised fish consumption advice for certain fish species. In the case of Ravine Lake, fish consumption advice was issued recommending no more than one meal per week of black crappie and largemouth bass. Additional data collected by the MPCA later in 2007 resulted in new fish consumption advice being issued for one other lake in south Washington County, Powers Lake in Woodbury, based on PFOS

levels in fish. For specific guidance, please refer to MDH's Fish Consumption Advice web site at <http://www.health.state.mn.us/divs/eh/fish/index.html>. The specific source of the PFOS detected in fish in these lakes is not clear.

General Regional Issues

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

Community Concerns

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

Some residents have expressed concern about the following: that cancer or other disease rates in the area seem higher than normal, the health implications for children who may have been exposed to contaminated water (both before and after birth), and the health of domestic animals that may be drinking contaminated water. Residents also had questions about multiple exposure pathways to PFCs, and the lack of health-based exposure limits for some PFCs in water. MDH has made every effort to address these health issues where possible. MDH has produced multiple information sheets for area residents, regularly updated its web site on PFCs

(www.health.state.mn.us/divs/eh/hazardous/topics/pfcs/index.html), and created an e-mail distribution list (1,330 subscribers as of September 2009) to notify interested residents and local officials of new information. The cities (and Washington County) have also provided updates for local residents in their city newsletters, annual water quality reports, and web sites. There have also been numerous stories in state, Twin Cities, and local media.

Evaluation of Environmental Fate and Exposure Pathways

Introduction

PFCs, primarily perfluorooctanoic acid (PFOA; $C_8F_{15}O_2H$) and one of its salts, ammonium perfluorooctanoate (APFO; $C_8F_{15}O_2NH_4$), as well as lesser amounts of other PFCs such as perfluorooctanesulfonyl fluoride (POSF; $C_8F_{17}SO_2F$) and perfluorobutanoic acid (PFBA, $C_4F_7O_2H$) were manufactured by 3M at their Cottage Grove facility

(formerly known as Chemolite) from the early 1950s until 2002. PFBA production reportedly ceased in 1998. One of the byproducts of the production of POSF is perfluorooctane sulfonate (PFOS; $C_8F_{17}SO_3^-$), which can also be produced by the subsequent chemical or enzymatic hydrolysis of POSF. The chemical structures of PFOA, PFOS, and PFBA are shown in Figure 2.

PFCs were produced at the 3M Cottage Grove facility using an electrochemical fluorination process, a batch process where hydrogen fluoride was added to organic molecules and electricity was applied to facilitate the complete replacement of hydrogen atoms with fluorine. A unique aspect of this process was the production of both straight-chain and branched isomers (Prevedouros et al. 2006). The other main PFC production process (and the one currently in use by the remaining PFC manufacturers) is a fluorotelomer process and produces only straight-chain molecules. This fact could help distinguish PFCs found in the environment or in living things as having originated from historical PFC manufacture and waste disposal (such as from the various disposal sites historically used by 3M) as opposed to the modern use of PFCs in commercial products (De Silva and Mabury 2006).

In January 2006, the administrator of the EPA initiated the PFOA Stewardship Program, in which the eight major companies who remained in the PFOA industry (including 3M, through its subsidiary Dyneon) committed voluntarily to reduce facility emissions and product content of PFOA and related chemicals on a global basis by 95 percent no later than 2010, and to work toward eliminating emissions and product content of these chemicals by 2015 (EPA 2006). 3M/Dyneon has already met the 2010 goal. There are still some commercial uses of PFOS in specialty products (primarily in the semiconductor, metal plating, and aviation industries). To the knowledge of MDH, there is currently no commercial production of PFBA in the U.S., but some PFBA is reportedly imported for commercial applications and for use in analytical laboratories. In its reformulated stain repellent and other commercial products such as Scotchgard™, 3M used a chemistry based on the four carbon sulfonic acid PFBS, instead of the eight carbon PFOS (Brezinski 2003).

Environmental Fate

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

published by 3M scientists in technical journals as far back as the early 1950s (Kauck and Diesslin 1951; Reid et al. 1955).

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

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

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

Evaluation of Impacts on Groundwater

The information obtained from investigation and remedial activities at the disposal sites, surface water sampling, and sampling of private, municipal, and non-community wells has been used to evaluate the magnitude, extent, and possible migration history of the PFC contamination in south Washington County. The highest concentrations of PFBA are detected south of the site and appear to follow the buried bedrock valley that underlies the western edge of the site and trends south through Cottage Grove Ravine Park to the Mississippi River. PFBA has been detected in all four of the major drinking water aquifers in the investigation area, with the most widespread contamination documented in the Prairie du Chien and Jordan aquifers (these are also the aquifers most widely used in this area). The extent of contamination in the St. Peter (and overlying Quaternary sands and gravels) is poorly understood, due to the few number of wells

present in those aquifers. Contamination in the Franconia is generally low in concentration and appears to be localized near major bedrock faults that have brought the Franconia into contact with the Jordan (Figure 4), allowing PFCs in the Jordan to migrate into the Franconia.

An area of contaminated groundwater is often referred to as a "plume" (like a plume of smoke). "Typical" contaminant plumes have their highest concentrations near the source area, decrease in concentration with distance from that source, and are roughly elliptical in shape with the axis of the plume aligned roughly with the direction groundwater is flowing. In contrast, the PFBA plume in the investigation area is quite complex (see Figures 8 and 9). While concentrations are high near the source area, there are scattered patches of higher concentrations that don't appear to connect back to the source area. In part, this appears to be due to the influence of bedrock structures, such as buried valleys and faults. In both Figures 8 and 9, a zone of higher PFBA concentrations trends southward from the 3M-Woodbury Disposal Site and appears to follow the course of the bedrock valley in that area. Similarly, a patch of higher PFBA concentrations near the border of Cottage Grove, St. Paul Park, and Newport (shown in yellow, just southeast of the intersection of Hwy 61 and I-494) appears to follow the outline of the bedrock valley in that area (see Figure 8). An arm of that valley appears to trend back toward the site, but an absence of wells in this area limits our ability to definitely trace the zone of higher PFBA concentrations back to the site.

Another unusual area within the plume is the seemingly isolated area of PFBA present in the Jordan aquifer near the St. Croix River in the southeastern portion of the investigation area (Figure 9). However, this area is bounded by a series of major bedrock faults and the top of the Jordan east of this faulted zone is over 200 feet lower in elevation than the top of the Jordan to the west of the zone. The Jordan on the east side of this heavily faulted zone is essentially at the same elevation as the Franconia aquifer on the west side. As shown in Figure 10, PFBA is present in the Franconia west of the faulted zone and at concentrations similar to those found in the Jordan east of the fault. In other words, the PFBA plume appears to be migrating from the Jordan into the Franconia across a fault near the ravine, traveling through the Franconia as the groundwater moves to the east-southeast, and then crosses another fault and back into the Jordan near the St. Croix River, as illustrated in Figure 4.

PFBA is also moving south from the two 3M waste disposal sites in Lake Elmo and Oakdale (Figure 1), further complicating our understanding of the PFBA distribution. It is likely that the PFBA detected in east Woodbury and west Afton actually has migrated southeast from the Washington County Landfill. Similarly, the PFBA detected in south Maplewood and northern Woodbury likely migrated south-southwest from the 3M-Oakdale Disposal site. Elsewhere in the investigation area, it is difficult to determine how much, if any, of the PFBA detected has been contributed from these two northern sites.

There are also areas with unusual PFBA distribution patterns that, at present, cannot be attributed to bedrock structures or migration from the northern disposal sites. The most striking of these is the "finger" of elevated PFBA in the Prairie du Chien aquifer

extending north of the 3M-Woodbury Disposal Site (Figure 8). Although it appears to follow the same northeast-southwest trend of the faults in the area, no fault has been identified in this portion of the investigation area. One possibility is that several large capacity wells (city and irrigation wells) located north of this "finger" of PFBA, may be drawing some of the contamination in this direction. Pump tests at the site suggest the high volumes of water being removed by the groundwater capture system would prevent this from occurring, and water level measurements do not indicate influence by off-site wells affecting this portion of the site. It has been suggested this "finger" may be a remnant of a plume that was established before the pump-out system began operation. However, the high capacity wells north of the site were constructed after 1990 and the pump-out system began operation in the early 1970s. If this "finger" of PFBA was established before groundwater extraction began at the site, the mechanism permitting the PFBA to migrate against the regional groundwater flow is unknown.

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

Maximum Concentrations of non-PFBA PFCs in Langdon/River Acres Area

PFOA	PFOS	PFPeA	PFHxA	PFBS	PFHxS
1.4	0.9	0.3	0.16	0.12	0.15

All concentrations in µg/L

The presence of multiple PFCs, other than PFBA and PFPeA, in these two areas is unusual because none of those compounds are detected in wells between the disposal site and the two areas described (Figure 13). While the actual source is as yet unknown, it is possible that the multiple PFCs detected in these areas may be related to fire-fighting activities. Some specialized types of fire-fighting foams contain PFCs. The Cottage Grove Fire Department originally reported to the MPCA that it used small amounts of such foam in their training activities, which occur near the main city well field (Delta, 2008). However, the department later stated that although they use PFC-bearing foams, they do not use them for training purposes. As a result, the source of the multiple PFCs in the area of the city well field remains uncertain. The possible link to fire-fighting chemicals in the areas south of Highway 61 appears to be stronger. In 2002, PFC-bearing foams were used to extinguish a major industrial fire immediately south of Highway 61 and the foams reportedly discharged to a surface water infiltration pond immediately

southwest of the Langdon neighborhood and upgradient of the River Acres neighborhood. The MPCA is currently investigating what, if any, role fire-fighting foams may have played in creating the anomalous PFC signature in this area.

On-going monitoring of private wells continues to track PFC concentrations in each of the affected drinking water aquifers across the investigation area over time. As with the initial sampling effort, more wells are sampled in areas with higher PFC levels, where the geology is complex, and/or the PFC concentration spatial trends are unpredictable. In areas where concentrations are lower and the distribution patterns are predictable, "sentry wells" selected to provide representative results for each of the aquifers in use in that area and to act as an early warning "sentry" for any unusual results that might warrant sampling of additional wells in the area. The sampling schedule, both in terms of frequency and number of wells, is adjusted as more data is collected and the behavior of the PFC plume in the affected communities is better understood.

Sampling of the monitoring wells at the disposal site also indicate stable concentrations and appear to indicate that PFCs are not migrating off-site, as a result of the groundwater capture system in operation there. However, as noted above, the absence of downgradient monitoring wells screened across the "high transmissivity zone" in the Prairie du Chien, may represent an important gap in the integrity of the monitoring network.

The monitoring results indicate PFC concentrations in all of the aquifers are quite stable. This is consistent with findings in Lake Elmo and Oakdale (MDH 2008a) and suggests that the size, shape, and concentrations of the PFC plumes in the individual aquifers may have reached a state of equilibrium (i.e. are not changing). Based on our understanding of the way PFCs move in the groundwater environment and the high flow velocities known to exist in the investigation area, it is likely the PFCs from the disposal site migrated rapidly through the affected communities relatively soon after the wastes were disposed and what is present today are remnants of contaminants slowly washing out of the aquifers. This slow washing out of contaminants is sometimes referred to as "secondary flow" or "matrix diffusion", and occurs because there are major (i.e. larger, faster) flow paths and minor (i.e. smaller, slower) flow paths within the aquifer (Figure 14). The majority of the contaminants flow quickly through the major flow paths in an initial "pulse", rapidly establishing the contaminant plume, while contaminants that have moved into the minor flow paths slowly release over time at more dilute concentrations. If this is what happened in the investigation area, earlier concentrations in the aquifers may have been higher than what is currently being detected. However, it is impossible to calculate how high those concentrations might have been or how long the higher concentrations may have lasted.

The persistence of PFCs in the environment means that ultimately the PFCs in the groundwater in Washington County will discharge to the Mississippi and St. Croix Rivers. However, the low concentrations in the groundwater and the large volume of flow in the rivers will likely dilute this discharge to extremely low concentrations. Samples of water from the Mississippi River were collected as part of the investigation of

the waste disposal areas at the 3M Cottage Grove Plant (MDH 2005; Weston 2008c). While elevated concentrations of PFCs were detected in samples near and immediately downstream of the plant, no PFCs were detected in water samples collected upstream of the plant. Low levels of PFOS (0.289 – 1.34 ppb) were detected in sediment samples collected upstream of the plant. PFOS represents a very minor component of the PFC plumes in south Washington County and it is unlikely that discharge of those plumes contributed significantly to the PFOS detected in the sediment samples.

Exposure through Private Wells

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

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

A completed environmental exposure pathway to PFCs from the disposal of PFC-containing wastes in disposal sites in Washington County exists primarily from consuming PFC contaminated water. The consumption of fish from local lakes or rivers where PFOS has been detected in fish populations also represents a completed exposure pathway, although the source of the PFOS may not be in all cases from land disposal of PFC-containing wastes.

Samples have been collected from over 900 private wells and 56 business wells in Woodbury, Cottage Grove, and other communities in south Washington County (south of I-94). PFCs have been detected in 745 private and business wells. No wells, public or private, south of I-94 have been found to contain PFBA at concentrations above the Health Based Value (HBV) of 7.0 ppb for PFBA.

As of late 2009, 29 private well owners in Cottage Grove and Grey Cloud Island had received a drinking water well advisory from MDH due to the presence of PFCs. Nine of these private wells have PFOA at concentrations above the HRL of 0.3 ppb; one also has PFOS at concentrations above the HRL of 0.3 ppb. In 12 of these wells, concentrations of PFCs exceed a Hazard Index value of one based on multiple PFCs.

At the other seven wells, levels of PFCs are below current health advisory levels but the drinking water well advisories remain in effect due to uncertainties over long-term concentration trends and potential changes in the PFC plumes that could result from site remediation activities. All of these wells are located in neighborhoods south of U.S.

Highway 61, in the area of anomalous PFC detections, as discussed above. One of the wells was sealed by the MPCA, at the owner's request.

Where drinking water well advisories have been issued by MDH, exposures have been reduced to acceptable levels by the provision of bottled water and/or GAC filters by the MPCA. It is also possible that the use of owner-installed filter systems may have reduced past exposure to PFCs in drinking water. During the course of the investigation, it was found that a number of private well owners who subsequently received a drinking water well advisory due to PFCs had previously installed reverse osmosis water filters to remove nitrate, a common groundwater contaminant in the area. Reverse osmosis filters, if maintained properly, are also very effective at removing PFCs according to a study conducted by MDH in 2008 (MDH 2008b). The MDH study also documented which types of point-of-use carbon filters are effective at removing PFCs from drinking water.

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

The length of time local residents were exposed to PFCs through their drinking water is unknown. As discussed above, exposures could have started soon after PFCs wastes were placed in the disposal sites, given the mobility of PFCs in the environment. It is also possible that the levels in the groundwater were higher than currently detected, although it is impossible to determine what the concentrations may have been in the past or how long those higher concentrations may have lasted. Continued routine monitoring of select private wells with levels of PFCs below HRLs or the PFBA HBV will ensure that if levels of PFCs rise, future exposure to levels above health-based exposure limits will be brief.

Exposure through Public Water Supplies

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

No individual community wells in the affected area have exceeded current MDH health-based exposure limits for PFCs, except in the City of Oakdale. This is described in a previous MDH report (MDH 2008a).

Exposure through other Pathways

The use of water contaminated with low levels of PFCs for bathing, showering, or other incidental uses is unlikely to contribute appreciably to overall exposure. Ingestion of the contaminated water is by far the predominant exposure pathway. Use of PFC contaminated water for canning or cooking purposes may also contribute to exposure, as reported by Emmett et al. (2006a) and Holzer et al (2008). Irrigation of plants with PFC contaminated water may possibly lead to some uptake of PFCs by the plants, also contributing to overall exposure. So-called “market basket” studies of food products occasionally show low levels of PFCs. In a study conducted in the United Kingdom, PFOS was found at low concentrations in potatoes, some canned vegetables, eggs, and in the sugars and preserves food groups, while PFOA was detected only in potatoes (UK Food Standards Agency 2006). A similar study in Spain found low levels of PFOS in fish, dairy products, and meat, while PFOA was only detected in milk (Ericson et al 2008). A recent study of food items in Canada also found low levels of PFOS and PFOA in some food products, including beef, fish, and microwave popcorn (Tittlemier et al. 2007). It is less likely that PFBA would be taken up by plants, but data are not available.

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

Small amounts of unbound fluorotelomer alcohols that can break down to PFOS or PFOA (or other PFCs depending on their specific composition; also referred to as PFC precursors) have also been found in consumer and industrial products (Joyce et al. 2006). Release of telomer alcohols, and subsequent degradation in the environment or by organisms, could also be a source of human exposure to PFCs. One recent study estimated that, for some individuals, PFC precursor-based exposure could contribute up to 60-80% of total PFOS and 28-55% of total PFOA exposure (Vestergren et al 2008).

Public Health Implications of PFC Exposure

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

Note: ATSDR published a draft “Toxicological Profile for Perfluoroalkyl Compounds” for public review and comment in May 2009 (available at <http://www.atsdr.cdc.gov/toxprofiles/tp200.html#bookmark04>). The Toxicological Profile reviews much of the information below, as does a report by Lau et al (2007).

Summary of Toxicological Information

MDH has previously summarized the available toxicological information on PFOS and PFOA in its Health Consultation on the 3M-Cottage Grove Facility (MDH 2005), and more recently in the Public Health Assessment regarding PFC contamination in Lake Elmo and Oakdale published in August 2008 (MDH 2008a). This section will only briefly summarize that information and will also describe available information on the toxicity of PFBA.

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

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

Chronic exposure to PFOS at high doses results in liver toxicity and mortality, with a steep dose-response curve for mortality in rats and primates (OECD 2002). Indications of toxicity observed in 90-day rat studies include increases in liver enzymes and other adverse liver effects, gastrointestinal effects, blood abnormalities, weight loss, convulsions, and death. Immunotoxicity has also been reported in studies conducted in mice at relatively low doses (Peden-Adams et al. 2008).

Some long-term animal studies suggest that exposure to PFOA could increase the risk of tumors of the liver, pancreas, and testes (Kudo and Kawashima 2003, EPA 2002, OECD 2002). The mechanism of potential carcinogenesis is unclear, but evidence suggests that the tumors 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 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.

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

A key question MDH considered in the development of the 2008 HBV for PFBA was its half-life in animals and humans. Chang et al. (2007) summarized data from a study of the pharmacokinetics of PFBA in several animal species. The study showed that PFBA was eliminated quickly through urine in male and female rats, with a half-life of approximately 8 hours in male rats and less than two hours in female rats. The half-life in monkeys was less than two days. In study published in 2008, the mean half-life of PFBA in four male employees at the 3M-Cottage Grove plant and seven male and three female employees at the 3M-Cordova, Illinois plant was calculated to be 72 hours in males and 87 hours in females (Chang et al. 2008).

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

The findings of a developmental study of PFBA in mice conducted at the EPA laboratory in North Carolina was also reviewed by MDH (Das et al. 2007). In the study, exposure to PFBA by pregnant mice did not appear to significantly affect maternal weight gain or

fertility. Some developmental delays were observed in the offspring of the mice, and developmental effects were considered a co-critical effect along with liver, blood and thyroid effects in establishing the HBV for PFBA.

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

The current MDH HRLs for PFOS and PFOA are based on toxicological studies conducted on Cynomolgus monkeys. In the case of PFOS, the key study was used to derive a toxicity value (known as a reference dose, or RfD) of 0.000075 milligrams per kilogram-day (mg/kg-d). The RfD included a 'dose metric adjustment' of 20 to account for the large difference in half-life between Cynomolgus monkeys (110-132 days) and humans (5.4 years), as well as a total uncertainty factor (used to account for various uncertainties in applying animal studies to humans, among other factors) of 100. The critical effects used to determine the RfD were a decrease in serum high-density lipoprotein (i.e. "good" cholesterol) and thyroid hormones. For PFOA, the key study was used to derive an RfD of 0.00014 mg/kg-d. The RfD for PFOA included a 'dose metric adjustment' of 70 to account for the even larger relative difference in half-life between Cynomolgus monkeys (20 days) and humans (3.8 years), as well as a total uncertainty factor of 300. The critical effect used to determine the RfD was an increase in relative liver weight.

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

Summary of Human Exposure Information

PFCs, primarily PFOS and PFOA, have been detected in the blood of U.S. citizens in multiple studies (Olsen et al 2003, 2004a, and 2004b). PFCs have also been shown to cross the placenta. In a study of fifteen pairs of maternal and cord blood samples in Japan, Inoue et al. (2004) detected PFOS in the cord blood samples at approximately one-third the concentration in maternal blood. PFOA was detected in maternal blood, but not in cord blood. A similar study of 11 paired maternal and cord blood samples collected in Germany showed PFOS in cord blood at approximately 60% of the maternal blood concentration (Midasch et al. 2007). This study did detect low levels of PFOA (median of 3.4 µg/L) in cord blood samples, slightly above that found in the maternal blood samples. A larger study conducted in the city of Baltimore measured ten PFCs in the cord serum of 299 newborns (Apelberg et al. 2007a). PFOS and PFOA were detected in nearly all of the samples, at a geometric mean level of 4.9 and 1.6 ppb, respectively. Other PFCs were detected much less frequently, and at lower levels. In a follow-up study (Apelberg 2007b), a small (sub-clinical) negative association between both PFOA and PFOS cord

serum concentration and birth weight and size was observed. A similar study conducted in Denmark showed an inverse correlation between maternal serum PFOA levels and birth weight (Fei et al. 2007).

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

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

Survey Year	PFOS	PFOA	PFHxS
1999-2000	30.4	5.2	2.1
2003-2004	20.7	3.9	1.9

The data suggest that PFC levels in the blood serum of the general population are declining, perhaps due to a reduction in the use of PFCs in consumer products. Many other PFCs were included in the NHANES sample analysis, but data are not presented here because the PFCs were below detection limits, or only sporadically detected. PFBA was not included in the NHANES analyses.

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

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

Survey Year	PFOS	PFOA	PFHxS
2008-2009	35.9	15.4	8.4

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

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

The largest investigation of human exposure to PFOA is taking place in the Ohio-West Virginia area, and grew out of a court settlement of a class action lawsuit against the DuPont Corporation in 2005. This investigation is known as the C8 Health Project, and information on it can be found on the project's web site, <http://www.hsc.wvu.edu/som/cmed/c8/>. The project has enrolled 69,030 people who may have been exposed to PFOA through drinking water. The participants have been tested for PFOA (and some other PFC) exposure through analysis of blood samples. The project will also involve ten separate studies to help determine whether PFOA exposure is associated with any observable human health effects. Eight of the studies will focus on diseases such as cancer, heart disease, stroke, diabetes, immune function, liver and hormone disorders, and birth outcomes. Two studies will look at exposure to PFOA and its half-life in the general population. The studies are estimated to be complete in several years, although some preliminary results have been posted on the study's web site. Details of the studies and results can be found on the C8 Science Panel web site at <http://www.c8sciencepanel.org/index.html>. Selected preliminary results from the project were described in 2008 in a presentation at the West Virginia University School of Medicine (Frisbee 2008). The preliminary results described possible associations between PFOA exposure and indicators of inflammation, immune, liver, and thyroid function, and cholesterol that are consistent with some animal studies.

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

Discussion of the Public Health Implications of PFC Exposure

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

workers. MDH is closely following the ongoing 3M workforce studies, as well as the West Virginia study, which will provide more definitive information about the health implications of PFC exposure in the east metro area.

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

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

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

Health Outcome Data Review

On June 7, 2007 the Minnesota Cancer Surveillance System (MCSS), located within the Chronic Disease and Environmental Epidemiology Section of MDH issued a report presenting detailed profiles of cancer rates among residents of Dakota and Washington Counties (MDH 2007c). Using MCSS data for the 15-year period 1988-2002, county-wide cancer rates for all cancers combined and for each of about 25 of the most frequent types of cancer, including liver and thyroid cancer were examined. In addition, analyses were also conducted to examine incidence rates for 16 selected cancers for specific communities, by zip code, within each county. For that analysis, data from the years 1996-2004 were used, largely due to population growth in some communities and limitations on community census data.

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

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

Child Health Considerations

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

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

Conclusions

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

Currently, PFCs have been detected in public and private wells across a wide area of south Washington County, and in parts of northern Dakota County and southeastern Ramsey County. Exposure to PFCs at levels above health concern is currently being addressed by the use of bottled water or whole-house activated carbon filters at homes that have been issued a drinking water well advisory by MDH. MDH concludes that currently, drinking water from public or private wells that contain PFCs is not expected to harm people's health; new data will be evaluated as it becomes available. Remediation actions to address PFCs at the waste disposal sites are underway by 3M and the MPCA.

Recommendations

1. 3M should continue to follow the requirements of the Consent Order to implement the selected remediation options for soil and groundwater at the 3M-Woodbury Disposal Site.
2. People should avoid trespassing on the 3M-Woodbury Disposal Site, especially during remediation activities.
3. Extensions of the Cottage Grove municipal water supply to serve areas where private wells contain levels of PFCs in excess of MDH HRLs or HBVs should be considered.
4. Monitoring of selected private wells in the affected area should continue under agreed upon sampling plans to track changes in the plume and monitor for changes in concentration in individual wells. If the plume remains stable, the frequency and number of wells monitored may be reduced while still providing assurance that public health is protected.
5. 3M should ensure the monitoring network at the disposal site provides adequate information regarding water quality in the "high transmissivity zone" of the Prairie du Chien aquifer.

Public Health Action Plan

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

References

- 3M 2003a. Environmental and Health Assessment of Perfluorooctane Sulfonate Acid and its Salts. 3M Company, St. Paul, Minnesota. August 20, 2003.
- 3M 2003b. Letter from Michael A. Santoro, 3M, and George H. Millet, 3M, to the U.S. EPA Office of Pollution Prevention and Toxics. August 1, 2003.
- 3M 2003c. Assessment of Lipid, Hepatic, and Thyroid Function in Relation to an Occupational Biologic Limit Value for Perfluorooctanoate. Medical Department, 3M Company, St. Paul, Minnesota 55144. June 9, 2003.
- 3M 2008. Letter from Katie Winogrodzki, 3M, to Gerald Stahnke, MPCA. September 19, 2008.
- Alexander, B.H., Olsen, G.W., Burris, J.M., Mandel, J.H., and Mandel, J.S. Mortality of employees of a perfluorooctanesulphonyl fluoride manufacturing facility. *Occupational and Environmental Medicine* 60: 722-729.
- Alexander, C. 2007. Fractured sandstone karst aquifers, the St. Peter, Jordan and Hinckley Formations: Examples for Askov, Woodbury, Rochester and Elsewhere. Presented at the Minnesota Groundwater Association spring conference, April 19, 2007.
- Apelberg, B.J., Goldman, L.R., Calafat, A.M., Herbstman, J.B., Kuklennyik, Z., Heidler, J., Needham, L.L., Halden, R.U., and Witter, F.R. 2007a. Determinants of fetal exposure to polyfluoroalkyl compounds in Baltimore, Maryland. *Environmental Science and Technology* 41: 3891-3897.
- Apelberg, B.J., Witter, F.R., Herbstman, J.B., Calafat, A.M., Halden, R.U., Needham, L.L., and Goldman, L.R., 2007b. Cord serum concentrations of perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA) in relation to weight and size at birth. *Environmental Health Perspectives* 115: 1670-1676.
- ATSDR 2009. Draft Toxicological Profile for Perfluoroalkyl Acids. Agency for Toxic Substances and Disease Registry, Atlanta GA. May 2009.
- Brezinski, D. 2003. Laying the Foundation for New Technologies: 3M Creates a new building block for its fluorosurfactants. *Paint & Coatings Industry*, January 2003.
- Butenhoff J.L., Kennedy G.L., Hinderliter P.M., Lieder P.H., Jung R., Hansen K.J., Gorman G.S., Noker P.E., Thomford P.J. 2004. Pharmacokinetics of perfluorooctanoate in cynomolgus monkeys. *Toxicological Sciences* 82: 394-406.
- Butenhoff, J.L., Olsen, G.W., and Pfahles-Hutchens, A. 2006. The applicability of biomonitoring data for perfluorooctanesulfonate to the environmental public health continuum. *Environmental Health Perspectives* 114: 1776-1782.

- Calafat, A.M., Kuklenyik, Z., Reidy, J.A., Caudill, S., Tully, J.S., and Needham, L.L. 2007a. Serum concentrations of 11 polyfluoroalkyl compounds in the U.S. population: data from the National Health and Nutrition Examination Survey (NHANES) 1999-2000. *Environmental Science and Technology* 41: 2237-2242.
- Calafat, A.M., Wong, L.-Y., Kuklenyik, Z., Reidy, J.A., and Needham, L.L. 2007b. Polyfluoroalkyl chemicals in the U.S. population: data from the National Health and Nutrition Examination Survey (NHANES) 2003-2004 and comparisons to NHANES 1999-2000. *Environmental Health Perspectives* 115: 1596-1602.
- Chang, S., Hart, J., Ehresman, D., Das, K., Lau, C., Noker, P., Gorman, G., Tan, Y., and Butenhoff, J. 2007. The pharmacokinetics of perfluorobutyrate (PFBA) in rats, mice, and monkeys. *The Toxicologist, Supplement to Toxicological Sciences* 96: abstract 937.
- Chang, S., Das, K., Ehresman, D.J., Ellefson, M.E., Gorman, G.S., Hart, J.A., Nokers, P.E., Tan, Y.M., Lieder, P.H., Lau, C., Olsen, G.W., and Butenhoff, J.L. 2008. Comparative pharmacokinetics of perfluorobutyrate in rats, mice, monkeys, and humans and relevance to human exposure via drinking water. *Toxicological Sciences* 104: 40-53.
- Colsher, P., Ducatman, A., and Haddy, L. Examination of community concerns about water contamination with perfluorooctanoic acid and related chemicals using population-based cancer registry data. West Virginia Department of Health and Human Services, Charleston, W.V. 2005.
- Conder, J.M., Hoke, R.A., De Wolf, W., Russell, M.H., and Buck, R.C. 2008. Are PFCAs bioaccumulative? A critical review and comparison with regulatory criteria and persistent lipophilic compounds. *Environmental Science and Technology* 42: 995-1003.
- Das, K.P., Grey, B., Butenhoff, J., Tanaka, S., Ehresman, D., Zehr, D., Wood, C., and Lau, C. 2007. Effects of perfluorobutyrate exposure in mice during pregnancy. *Toxicological Sciences*, advance published May 28, 2008.
- De Silva, A.O., and Mabury, S.A. 2006. Isomer distribution of perfluorocarboxylates in human blood: potential correlation to source. *Environmental Science and Technology* 40: 2903-2909.
- DeWitt, J.C., Copeland, C.B., Strynar, M.J., and Luebke, R.W. 2008. Perfluorooctanoic acid-induced immunomodulation in adult C57BL/6J or C57BL/6N female mice. *Environmental Health Perspectives* 116: 644-650.
- Delta (2008) Perfluorocarbon (PFC)-Containing Firefighting Foams and Their Use in Firefighting Training in Minnesota. Prepared for Minnesota Pollution Control Agency. June 30, 2008.

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

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

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

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

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

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

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

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

Frisbee, S.J. 2008. The C8 Health Project: how a class action lawsuit can interact with public health – history of events. Presentation at the West Virginia University School of Medicine, May 7, 2008. Accessed online at: <http://www.hsc.wvu.edu/som/cmed/ophp/pdfs/Public%20Health%20Grand%20Rounds%2005-07-2008%20-%20C8%20Health%20Project.pdf>

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

- Holzer, J., Midasch, O., Rauchfuss, K., Kraft, M., Reupert, R., Angerer, J., Kleeschulte, P., Marschall, N., and Wilhelm, M. 2008. Biomonitoring of perfluorinated compounds in children and adults exposed to perfluorooctanoate-contaminated drinking water. *Environmental Health Perspectives* 116: 651-657.
- Ikeda, T., Aiba, K., Fukuda, K., and Tanaka, M. 1985. The induction of peroxisome proliferation in rat liver by perfluorinated fatty acids, metabolically inert derivatives of fatty acids. *Journal of Biochemistry* 98: 475-482.
- Inoue, K., Okada, F., Ito, R., Kato, S., Sasaki, S., Nakajima, S., Uno, A., Saijo, Y., Sata, F., Yoshimura, Y., Kishi, R., and Nakazawa, H. 2004. Perfluorooctane sulfonate (PFOS) and related perfluorinated compounds in human maternal and cord blood samples: assessment of PFOA exposure in susceptible population during pregnancy. *Environmental Health Perspectives* 112: 1204-1207.
- Joyce, M., Dinglasan-Panlilio, A., and Mabury, S.A. 2006. Significant residual fluorinated alcohols present in various fluorinated materials. *Environmental Science and Technology* 40: 1447-1453.
- Just, W.W., Gorgas, K., Hartl, F.U., Heinemann, P., Salzer, M., and Schmissek, H. 1989. Biochemical effects and zonal heterogeneity of peroxisome proliferation induced by perfluorocarboxylic acids in rat liver. *Hepatology* 9: 570-581.
- Kanivetsky R. and Cleland, J. M. 1990. Hydrogeology, in: *Geologic Atlas of Washington County, Minnesota*. Minnesota Geological Survey; County Atlas Series, Atlas C-5, Plate 5.
- Kärman, A., Ericson, I., van Bavel, B., Darnerud, P.O., Aune, M., Glynn, A., Lignell, S., and Lindstrom, G. 2007. Exposure of perfluorinated chemicals through lactation: levels in matched human milk and serum and a temporal trend, 1996-2004, in Sweden. *Environmental Health Perspectives* 115: 226-230.
- Kauck, E.A. and Diesslin, A.R. 1951. Some properties of perfluorocarboxylic acids. *Industrial and Engineering Chemistry* 43: 2332-2334.
- Kennedy, G.L. 1985. Dermal toxicity of ammonium perfluorooctanoate. *Toxicology and Applied Pharmacology* 81: 348-355.
- Kennedy, G.L., Hall, G.T., Britelli, M.R., Barnes, J.R., and Chen, H.C. 1986. Inhalation toxicity of ammonium perfluorooctanoate. *Food Chemistry and Toxicology* 24: 1325-1329.
- Kennedy, G.L., Butenhoff, J.L., Olsen, G.W., O'Connor, J.C., Seacat, A.M., Perkins, R.G., Biegel, L.B., Murphy, S.R., and Farrar, D.G. 2004. The toxicology of perfluorooctanoate. *Critical Reviews in Toxicology* 34: 351-384.

- Kubwabo, C., Stewart, B., Zhu, J., and Marro, L. 2005. Occurrence of perfluorosulfonates and other perfluorochemicals in dust from selected homes in the city of Ottawa, Canada. *Journal of Environmental Monitoring* 7: 1074-1078.
- Kudo, N. and Kawashima, Y. 2003. Toxicity and toxicokinetics of perfluorooctanoic acid in humans and animals. *The Journal of Toxicological Sciences* 28: 49-57.
- Kwan, W.C. 2001. Physical Property Determination of Perfluorinated Surfactants (doctoral thesis). University of Toronto, Graduate Department of Chemistry.
- Lau, C., Anitole, K., Hodes, C., Lai, D., Pfahles-Hutchens, A., and Seed, J. 2007. Perfluoroalkyl acids: a review of monitoring and toxicological findings. *Toxicological Sciences* 99: 366-394.
- Lieder, P.H., Tanaka, S., Ehresman, D.J., Roy, R.R., Otterdijk, F., and Butenhoff, J.L. 2007. A 28-day oral (gavage) toxicity study of ammonium perfluorobutyrate (APFB). *The Toxicologist, Supplement to Toxicological Sciences* 96: abstract 931.
- Lindholm, G.F., Helgesen, J.O., Broussard, W.L., and Farrell, D.F. 1974. Water Resources of the Lower St. Croix River Watershed, East-Central Minnesota. *Hydrologic Investigations Atlas HA-490*; U.S. Geological Survey, Reston, VA.
- Loewen, M., Halldorson, T., Wang, F., and Tomy, G. 2005. Fluorotelomer carboxylic acids and PFOS in rainwater from an urban center in Canada. *Environmental Science and Technology* 39: 2944-2951.
- Lundin, J.I., Alexander, B.H., Olsen, G.W., and Church, T. R. 2009. Ammonium perfluorooctanoate production and occupational mortality. *Epidemiology* 20: 921-928.
- MDH 2005. Health Consultation on Perfluorochemical Releases at the 3M Cottage Grove Facility. Minnesota Department of Health, St. Paul, Minnesota, February 18, 2005.
- MDH 2007a. Health Based Values for Perfluorooctanoic Acid. Memorandum from Helen Goeden, Health Risk Assessment Unit to John Stine, Environmental Health Division Director. Minnesota Department of Health, St. Paul, Minnesota, February 26, 2007.
- MDH 2007b. Health Based Values for Perfluorooctane Sulfonate. Memorandum from Helen Goeden, Health Risk Assessment Unit to John Stine, Environmental Health Division Director. Minnesota Department of Health, St. Paul, Minnesota, February 26, 2007.
- MDH 2007c. Cancer Incidence in Dakota and Washington Counties. MCSS Epidemiology Report 2007:1. Minnesota Cancer Surveillance System, Chronic Disease

and Environmental Epidemiology Section, Minnesota Department of Health, St. Paul, Minnesota. June 7, 2007.

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

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

MDH 2009. East Metro Perfluorochemical Biomonitoring Pilot Project Report. Minnesota Department of Health, St. Paul, Minnesota, July 21, 2009.

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

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

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

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

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

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

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

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

- OECD 2002. Hazard Assessment of Perfluorooctane Sulfonate (PFOS) and its Salts. Organization for Economic Cooperation and Development. November 21, 2002.
- Ohmori, K., Kudo, N., Katayama, K., and Kawashima, Y. 2003. Comparison of the toxicokinetics between perfluorocarboxylic acids with different carbon chain length. *Toxicology* 84: 135-140.
- Olsen, G.W., Church, T.R., Miller, J.P., Burris, J.M., Hansen, K.J., Lundberg, J.K., Armitage, J.B., Herron, R.M., Medhdizadehkashi, Z., Nobiletti, J.B., O'Neill, E.M., Mandel, J.H., and Zobel, L.R. 2003. Perfluorooctanesulfonate and other fluorochemicals in the serum of American Red Cross adult blood donors. *Environmental Health Perspectives* 111: 1892-1901.
- Olsen, G.W., Church, T.R., Hansen, K.J., Burris, J.M., Butenhoff, J.L., Mandel, J.H., and Zobel, L.R. 2004a. Quantitative evaluation of perfluorooctanesulfonate (PFOS) and other fluorochemicals in the serum of children. *Journal of Children's Health* 2: 53-76.
- Olsen, G.W., Church, T.R., Larson, E.B., van Belle, G., Lundberg, J.K., Hansen, K.J., Burris, J.M., Mandel, J.H., and Zobel, L.R. 2004b. Serum concentrations of perfluorooctanesulfonate and other fluorochemicals in an elderly population from Seattle, Washington. *Chemosphere* 54: 1599-1611.
- Olsen, G.W., Burris, J.M., Ehresman, D.J., Froehlich, J.W., Seacat, A.M., Butenhoff, J.L., and Zobel, L.R. 2007a. Half-life of serum elimination of perfluorooctanesulfonate, perfluorohexanesulfonate, and perfluorooctanoate in retired fluorochemical production workers. *Environmental Health Perspectives* 115: 1298-1305.
- Olsen, G.W., Mair, D.C., Reagan, W.K., Ellefson, M.E., Ehresman, D.J., Butenhoff, J.L., and Zobel, L. 2007b. Preliminary evidence of a decline in perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA) concentrations in American Red Cross blood donors. *Chemosphere* 68: 105-111.
- Olsen, G.W., and Zobel, L.R. 2007. Assessment of lipid, hepatic, and thyroid parameters with serum perfluorooctanoate (PFOA) concentrations in fluorochemical production workers. *International Archives of Occupational and Environmental Health* 81: 231-246.
- Peden-Adams, M.M., Keller, J.M., EuDaly, J.G., Berger, J., Gilkeson, G.S., and Keil, D.E. 2008. Suppression of humoral immunity in mice following exposure to perfluorooctane sulfonate (PFOS). *Toxicological Sciences*, advance published March 20, 2008.
- Permadi, H., Lundgren, B., Andersson, K., and DePierre, J.W. 1992. Effects of perfluoro fatty acids on xenobiotic-metabolizing enzymes, enzymes which detoxify reactive forms of oxygen and lipid peroxidation in mouse liver. *Biochemical Pharmacology* 44: 1183-1191.

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

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

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

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

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

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

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

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

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

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

Tipping, R.G., Runkel, A.C., Alexander, E.C., Jr., Alexander, S.C., and Green, J.A. (2006) Evidence for hydraulic heterogeneity and anisotropy in the mostly carbonate Prairie du Chien Group, southeastern Minnesota, USA. *Sedimentary Geology*, 184:305-330.

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

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

Vestergren, R., Cousins, I.T., Trudel, D., Wormuth, M., and Scheringer, M. 2008. Estimating the contribution of precursor compounds in consumer exposure to PFOS and PFOA. *Chemosphere* 73: 1617-1624.

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

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

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

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

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

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

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

Preparers of Report

James Kelly, M.S.
Health Assessor
Site Assessment and Consultation Unit
Minnesota Department of Health
Telephone: (651) 201-4910
James.Kelly@state.mn.us

Virginia Yingling, M.S.
Hydrogeologist
Site Assessment and Consultation Unit
Minnesota Department of Health
Telephone: (651) 201-4930
Virginia.Yingling@state.mn.us

CERTIFICATION

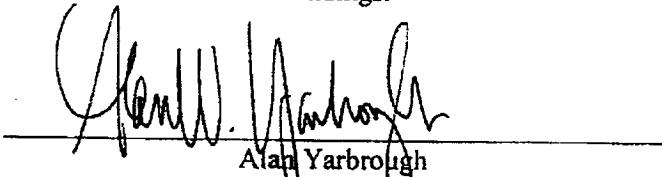
This Public Health Assessment was prepared by the Minnesota Department of Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the Public Health Assessment was begun. Editorial review was completed by the Cooperative Agreement partner.



Trent LeCoultré

Technical Project Officer, SPS, SSAB, DHAC, ATSDR

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



Alan Yarbrough

Chief, State Program Section, SSAB, DHAC, ATSDR

Glossary

Absorption

The process of taking in. For a person or an animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs.

Acute

Occurring over a short time [compare with chronic].

Acute exposure

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

Additive effect

A biologic response to exposure to multiple substances that equals the sum of responses of all the individual substances added together [compare with antagonistic effect and synergistic effect].

Adverse health effect

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

Ambient

Surrounding (for example, ambient air).

Analyte

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

Analytic epidemiologic study

A study that evaluates the association between exposure to hazardous substances and disease by testing scientific hypotheses.

Antagonistic effect

A biologic response to exposure to multiple substances that is less than would be expected if the known effects of the individual substances were added together [compare with additive effect and synergistic effect].

Aquifer

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

Background level

An average or expected amount of a substance or radioactive material in a specific environment, or typical amounts of substances that occur naturally in an environment.

Bedrock valley

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

Biodegradation

Decomposition or breakdown of a substance through the action of microorganisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).

Biologic indicators of exposure study

A study that uses (a) biomedical testing or (b) the measurement of a substance [an analyte], its metabolite, or another marker of exposure in human body fluids or tissues to confirm human exposure to a chemical substance [also see exposure investigation].

Biologic monitoring

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

Biologic uptake

The transfer of substances from the environment to plants, animals, and humans.

Biomedical testing

Testing of persons to find out whether a change in a body function might have occurred because of exposure to a chemical substance.

Biota

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

Body burden

The total amount of a substance in the body. Some substances build up in the body because they are stored in fat or bone or because they leave the body very slowly.

Buried valley

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

Cancer

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

Cancer risk

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

Carcinogen

A substance that causes cancer.

Case study

A medical or epidemiologic evaluation of one person or a small group of people to gather information about specific health conditions and past exposures.

Case-control study

A study that compares exposures of people who have a disease or condition (cases) with people who do not have the disease or condition (controls). Exposures that are more common among the cases may be considered as possible risk factors for the disease.

CAS registry number

A unique number assigned to a substance or mixture by the American Chemical Society Abstracts Service.

Central nervous system

The part of the nervous system that consists of the brain and the spinal cord.

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

Chronic

Occurring over a long time [compare with acute].

Chronic exposure

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

Cluster investigation

A review of an unusual number, real or perceived, of health events (for example, reports of cancer) grouped together in time and location. Cluster investigations are designed to confirm case reports; determine whether they represent an unusual disease occurrence; and, if possible, explore possible causes and contributing environmental factors.

Community Assistance Panel (CAP)

A group of people from a community and from health and environmental agencies who work with ATSDR to resolve issues and problems related to hazardous substances in the community. CAP members work with ATSDR to gather and review community health concerns, provide information on how people might have been or might now be exposed to hazardous substances, and inform ATSDR on ways to involve the community in its activities.

Comparison value (CV)

Calculated concentration of a substance in air, water, food, or soil that is unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level

during the public health assessment process. Substances found in amounts greater than their CVs might be selected for further evaluation in the public health assessment process.

Completed exposure pathway [see exposure pathway].

Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA)

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

Concentration

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

Contaminant

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

Delayed health effect

A disease or an injury that happens as a result of exposures that might have occurred in the past.

Dermal

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

Dermal contact

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

Descriptive epidemiology

The study of the amount and distribution of a disease in a specified population by person, place, and time.

Detection limit

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

Disease prevention

Measures used to prevent a disease or reduce its severity.

Disease registry

A system of ongoing registration of all cases of a particular disease or health condition in a defined population.

Dose (for chemicals that are not radioactive)

The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An "exposure dose" is how much of a substance is encountered in the environment. An "absorbed dose" is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.

Dose (for radioactive chemicals)

The radiation dose is the amount of energy from radiation that is actually absorbed by the body. This is not the same as measurements of the amount of radiation in the environment.

Dose-response relationship

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

Downgradient

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

Environmental media

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

Environmental media and transport mechanism

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

EPA

United States Environmental Protection Agency.

Epidemiologic surveillance [see Public health surveillance].

Epidemiology

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

Exposure

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

Exposure assessment

The process of finding out how people come into contact with a chemical substance or environmental contaminant, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.

Exposure-dose reconstruction

A method of estimating the amount of people's past exposure to environmental contaminants. Computer and approximation methods are used when past information is limited, not available, or missing.

Exposure investigation

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

Exposure pathway

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

Exposure registry

A system of ongoing followup of people who have had documented environmental exposures.

Fault

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

Feasibility study

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

Gaining stream

A stream into which groundwater enters through the stream banks and streambed.
Compare to "losing stream".

Geographic information system (GIS)

A mapping system that uses computers to collect, store, manipulate, analyze, and display data. For example, GIS can show the concentration of a contaminant within a community in relation to points of reference such as streets and homes.

Grand rounds

Training sessions for physicians and other health care providers about health topics.

Groundwater

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

Groundwater Divide

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

Half-life ($t_{1/2}$)

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

Hazard

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

Hazardous Substance Release and Health Effects Database (HazDat)

The scientific and administrative database system developed by ATSDR to manage data collection, retrieval, and analysis of site-specific information on hazardous substances, community health concerns, and public health activities.

Health consultation

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

Health education

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

Health investigation

The collection and evaluation of information about the health of community residents. This information is used to describe or count the occurrence of a disease, symptom, or

clinical measure and to evaluate the possible association between the occurrence and exposure to chemical substances.

Health promotion

The process of enabling people to increase control over, and to improve, their health.

Health Base Value (HBV)

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

Health Risk Limit (HRL)

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

Health statistics review

The analysis of existing health information (i.e., from death certificates, birth defects registries, and cancer registries) to determine if there is excess disease in a specific population, geographic area, and time period. A health statistics review is a descriptive epidemiologic study.

Indeterminate public health hazard

The category used in ATSDR's public health assessment documents when a professional judgment about the level of health hazard cannot be made because information critical to such a decision is lacking.

Incidence

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

Ingestion

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

Inhalation

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

Intermediate duration exposure

Contact with a substance that occurs for more than 14 days and less than a year [compare with acute exposure and chronic exposure].

In vitro

In an artificial environment outside a living organism or body. For example, some toxicity testing is done on cell cultures or slices of tissue grown in the laboratory, rather than on a living animal [compare with in vivo].

In vivo

Within a living organism or body. For example, some toxicity testing is done on whole animals, such as rats or mice [compare with in vitro].

Losing stream

A stream in which the surface water infiltrates through the stream banks and streambed, down into the groundwater. Such streams are often intermittent and may appear to be dry for much of the summer, although water is still migrating within the streambed sediments. Compare to "gaining stream".

Lowest-observed-adverse-effect level (LOAEL)

The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.

MDH

The Minnesota Department of Health.

Medical monitoring

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

Metabolism

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

Metabolite

Any product of metabolism.

mg/kg

Milligram per kilogram.

mg/cm²

Milligram per square centimeter (of a surface).

mg/m³

Milligram per cubic meter; a measure of the concentration of a chemical in a known volume (a cubic meter) of air, soil, or water.

Migration

Moving from one location to another.

Minimal risk level (MRL)

An ATSDR estimate of daily human exposure to a environmental contaminant at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), noncancerous effects. MRLs are calculated for a route of exposure (inhalation or oral)

over a specified time period (acute, intermediate, or chronic). MRLs should not be used as predictors of harmful (adverse) health effects [see reference dose].

Morbidity

State of being ill or diseased. Morbidity is the occurrence of a disease or condition that alters health and quality of life.

Mortality

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

MPCA

The Minnesota Pollution Control Agency.

Mutagen

A substance that causes mutations (genetic damage).

Mutation

A change (damage) to the DNA, genes, or chromosomes of living organisms.

National Priorities List for Uncontrolled Hazardous Waste Sites (National Priorities List or NPL)

EPA's list of the most serious uncontrolled or abandoned hazardous waste sites in the United States. The NPL is updated on a regular basis.

National Toxicology Program (NTP)

Part of the Department of Health and Human Services. NTP develops and carries out tests to predict whether a chemical will cause harm to humans.

No apparent public health hazard

A category used in ATSDR's public health assessments for sites where human exposure to contaminated media might be occurring, might have occurred in the past, or might occur in the future, but where the exposure is not expected to cause any harmful health effects.

No-observed-adverse-effect level (NOAEL)

The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.

No public health hazard

A category used in ATSDR's public health assessment documents for sites where people have never and will never come into contact with harmful amounts of site-related substances.

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

Physiologically based pharmacokinetic model (PBPK model)

A computer model that describes what happens to a chemical in the body. This model describes how the chemical gets into the body, where it goes in the body, how it is changed by the body, and how it leaves the body.

Pica

A craving to eat nonfood items, such as dirt, paint chips, and clay. Some children exhibit pica-related behavior.

PFC

Perfluorochemical, a family of fully fluorinated hydrocarbons.

PLP

Permanent List of Priorities, the Minnesota state Superfund list

Plume

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

Point of exposure

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

Population

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

Potentially responsible party (PRP)

A company, government, or person legally responsible for cleaning up the pollution at a hazardous waste site under Superfund. There may be more than one PRP for a particular site.

ppb

Parts per billion.

ppm

Parts per million.

ppt

Parts per trillion.

Prevalence

The number of existing disease cases in a defined population during a specific time period [contrast with incidence].

Prevalence survey

The measure of the current level of disease(s) or symptoms and exposures through a questionnaire that collects self-reported information from a defined population.

Prevention

Actions that reduce exposure or other risks, keep people from getting sick, or keep disease from getting worse.

Public availability session

An informal, drop-by meeting at which community members can meet one-on-one with ATSDR staff members to discuss health and site-related concerns.

Public comment period

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

Public health action

A list of steps to protect public health.

Public health advisory

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

Public health assessment (PHA)

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

Public health hazard

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

Public health hazard categories

Public health hazard categories are statements about whether people could be harmed by conditions present at the site in the past, present, or future. One or more hazard categories might be appropriate for each site. The five public health hazard categories are no public health hazard, no apparent public health hazard, indeterminate public health hazard, public health hazard, and urgent public health hazard.

Public health statement

The first chapter of an ATSDR toxicological profile. The public health statement is a summary written in words that are easy to understand. The public health statement

explains how people might be exposed to a specific substance and describes the known health effects of that substance.

Public health surveillance

The ongoing, systematic collection, analysis, and interpretation of health data. This activity also involves timely dissemination of the data and use for public health programs.

Public meeting

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

RCRA [see Resource Conservation and Recovery Act (1976, 1984)]

Receptor population

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

Reference dose (RfD)

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

Registry

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

Remedial investigation

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

Resource Conservation and Recovery Act (1976, 1984) (RCRA)

This Act regulates management and disposal of hazardous wastes currently generated, treated, stored, disposed of, or distributed.

RfD [see reference dose]

Risk

The probability that something will cause injury or harm.

Risk reduction

Actions that can decrease the likelihood that individuals, groups, or communities will experience disease or other health conditions.

Risk communication

The exchange of information to increase understanding of health risks.

Route of exposure

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

Safety factor [see uncertainty factor]

SARA [see Superfund Amendments and Reauthorization Act]

Sample

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

Sample size

The number of units chosen from a population or an environment.

Saturated thickness

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

Solvent

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

Source of contamination

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

Special populations

People who might be more sensitive or susceptible to exposure to environmental contaminants because of factors such as age, occupation, sex, or behaviors (for example, cigarette smoking). Children, pregnant women, and older people are often considered special populations.

Special Well Construction Area (SWCA)

Minnesota Statutes, section 103I, subdivision 5, clause 7, grants the commissioner of health the authority to establish standards for the construction, maintenance, sealing, and water quality monitoring of wells in areas of known or suspected contamination.

Minnesota Rules, part 4725.3650, detail the requirements for construction, repair, or sealing within a designated SWCA, including plan review and approval, water quality monitoring, and other measures to protect public health and prevent degradation of groundwater.

Stakeholder

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

Statistics

A branch of mathematics that deals with collecting, reviewing, summarizing, and interpreting data or information. Statistics are used to determine whether differences between study groups are meaningful.

Substance

A chemical.

Substance-specific applied research

A program of research designed to fill important data needs for specific hazardous substances identified in ATSDR's toxicological profiles. Filling these data needs would allow more accurate assessment of human risks from specific substances contaminating the environment. This research might include human studies or laboratory experiments to determine health effects resulting from exposure to a given hazardous substance.

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

Superfund Amendments and Reauthorization Act (SARA)

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

Surface water

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

Survey

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

Synergistic effect

A biologic response to multiple substances where one substance worsens the effect of another substance. The combined effect of the substances acting together is greater than the sum of the effects of the substances acting by themselves [see additive effect and antagonistic effect].

Teratogen

A substance that causes defects in development between conception and birth. A teratogen is a substance that causes a structural or functional birth defect.

Toxic agent

Chemical or physical (for example, radiation, heat, cold, microwaves) agents that, under certain circumstances of exposure, can cause harmful effects to living organisms.

Toxicological profile

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

Toxicology

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

Transmissivity

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

Tumor

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

Uncertainty factor

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

Upgradient

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

Urgent public health hazard

A category used in ATSDR's public health assessments for sites where short-term exposures (less than 1 year) to hazardous substances or conditions could result in harmful health effects that require rapid intervention.

Volatile organic compounds (VOCs)

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

Water table

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

Other glossaries and dictionaries:

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

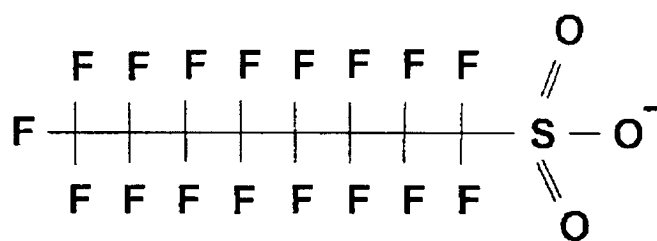
National Center for Environmental Health/Agency for Toxic Substances and Disease Registry (CDC) (<http://www.cdc.gov/nceh/dls/report/glossary.htm>)
<http://www.cdc.gov/exposurereport/>

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

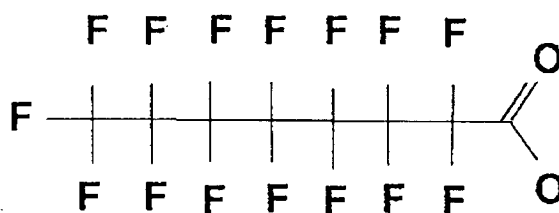
For more information on the work of ATSDR, please contact:

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

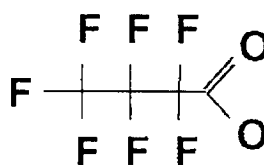
Appendix 1: Figures



Perfluoro-octane sulfonate (PFOS)



Perfluorooctanoic acid (PFOA)



Perfluorobutanoic acid (PFBA)

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

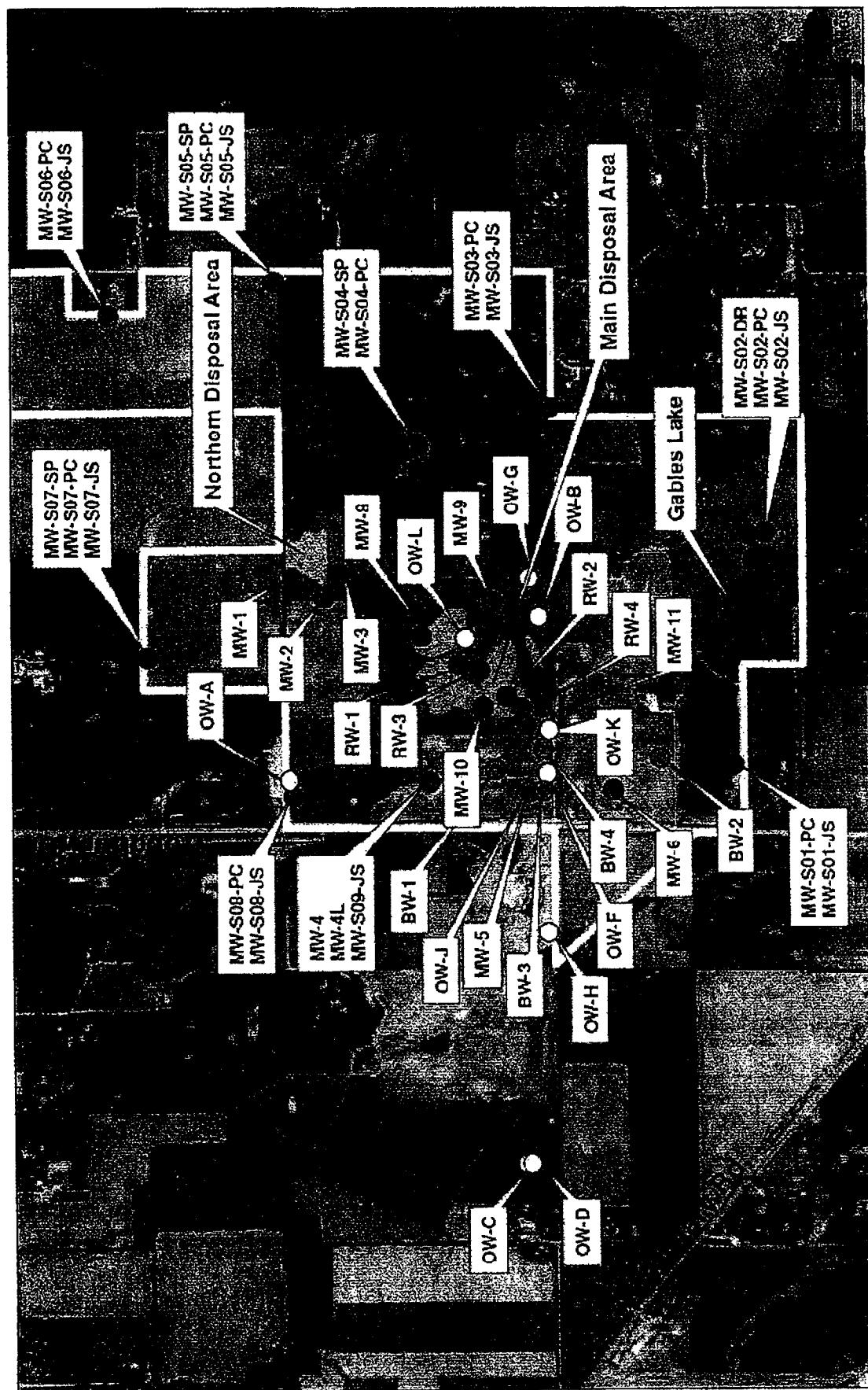


Figure 3 - 3M-Woodbury Disposal Site Well Location Map

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

Minnesota Dept. of Health - 10/6/09

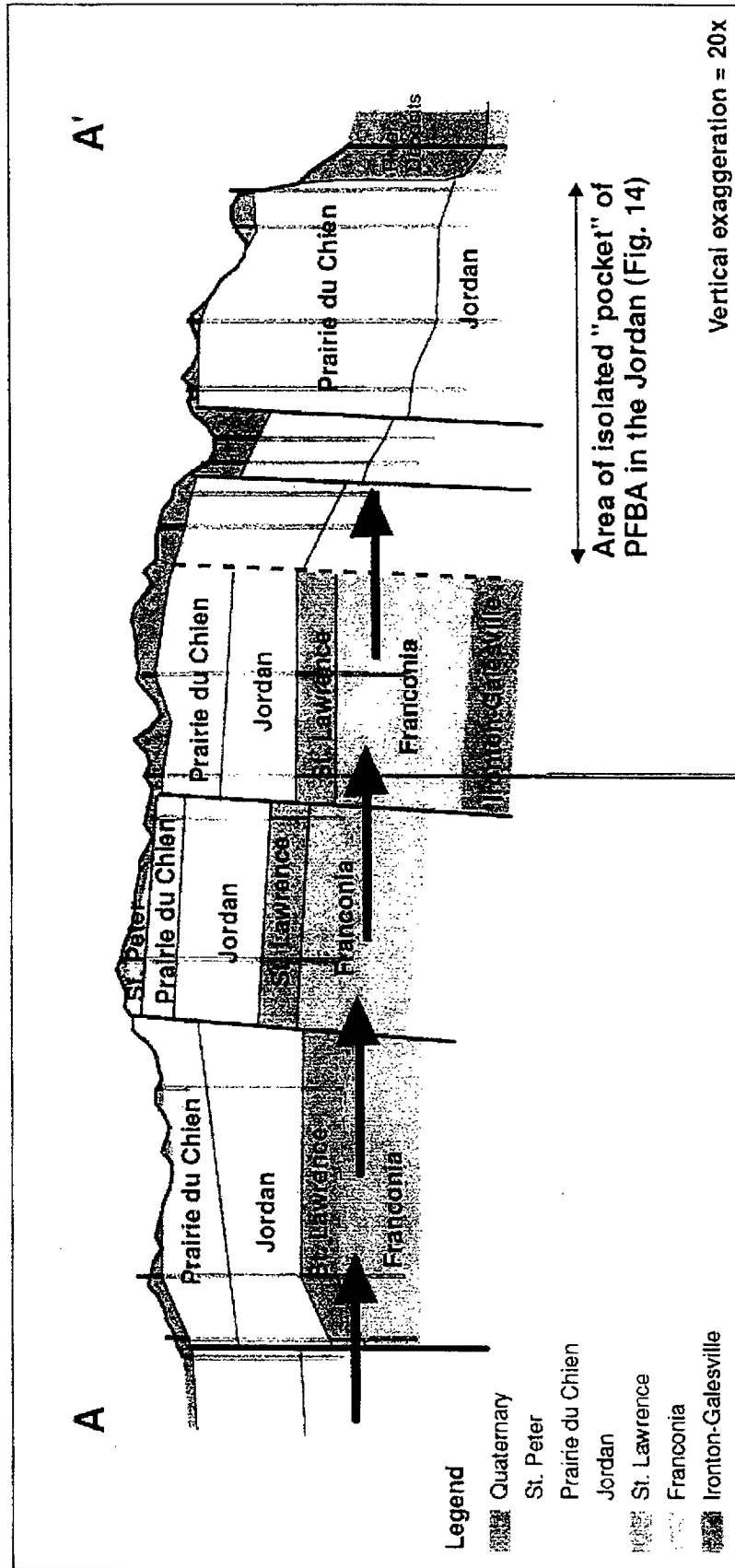


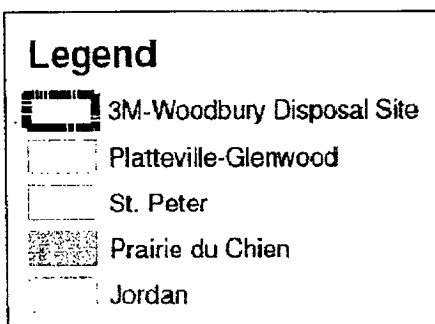
Figure 4 - Cross-Section Across Highly Faulted Zone

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

Prepared by MDH - 8/26/09



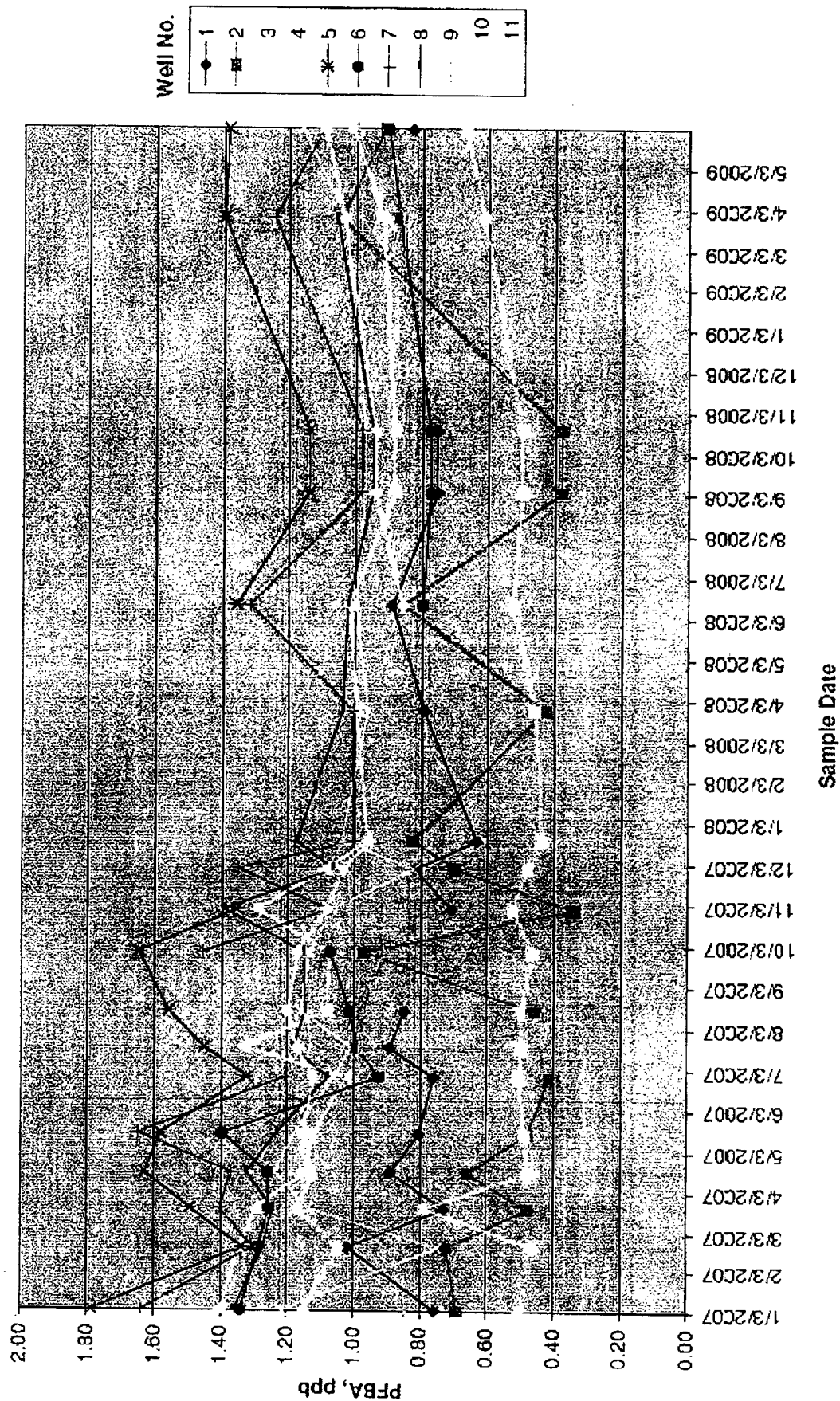
Figure 5 - 3M-Woodbury Disposal Site Bedrock



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

Prepared by MDH - 8/5/09

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



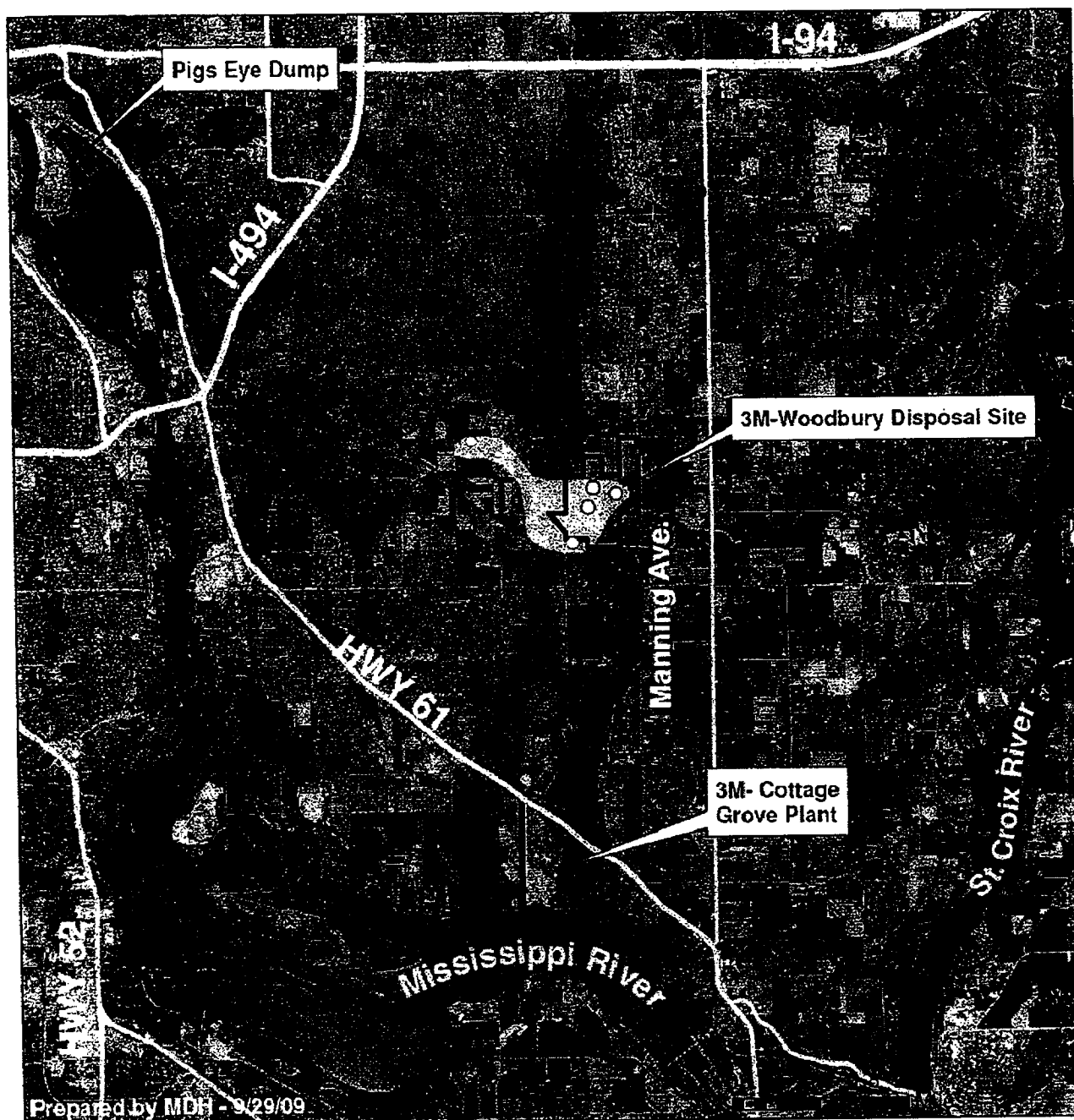


Figure 7 - PFBA in the St. Peter

Legend

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

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

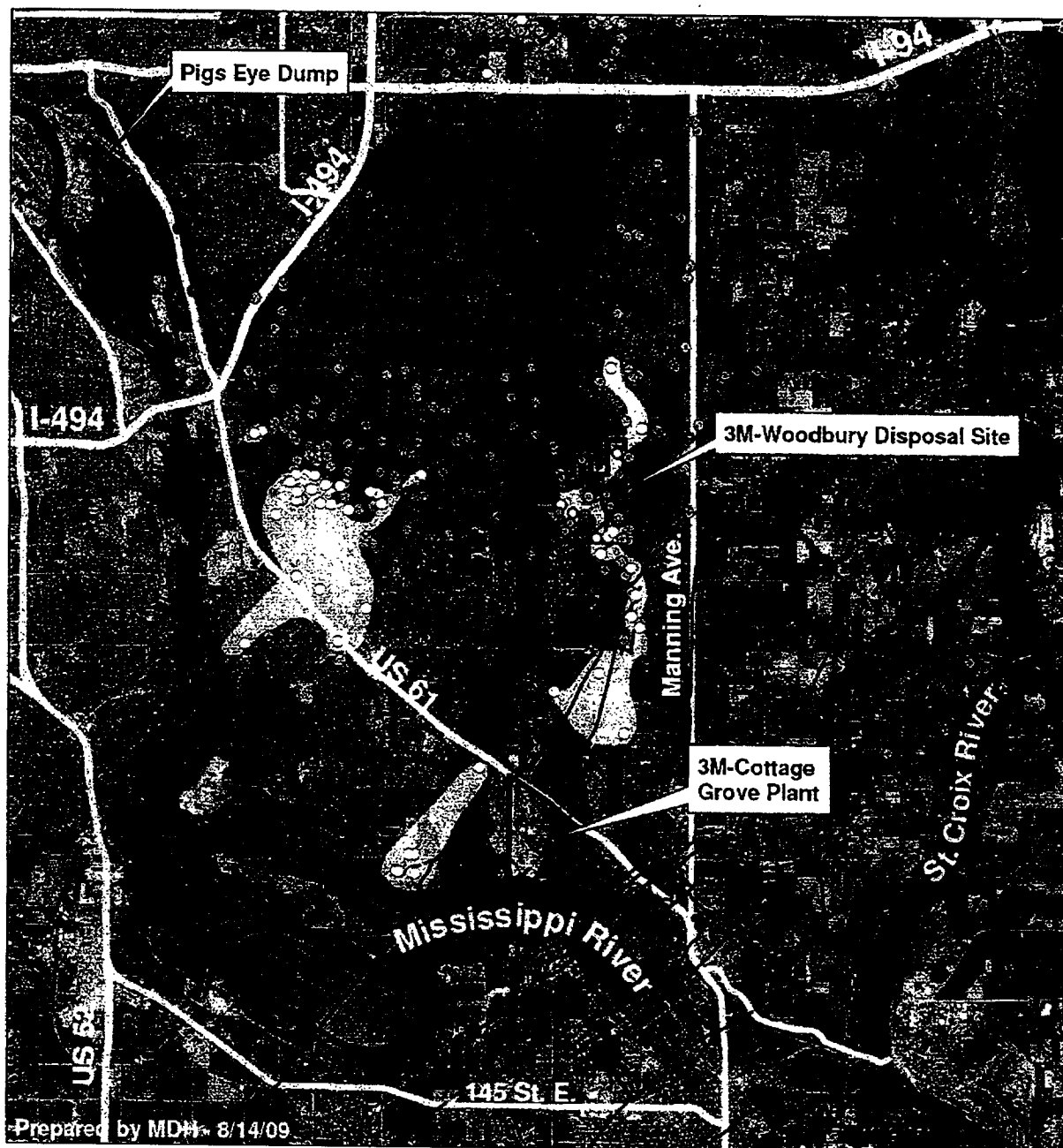


Figure 8: PFBA in the Prairie du Chien

Legend

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

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

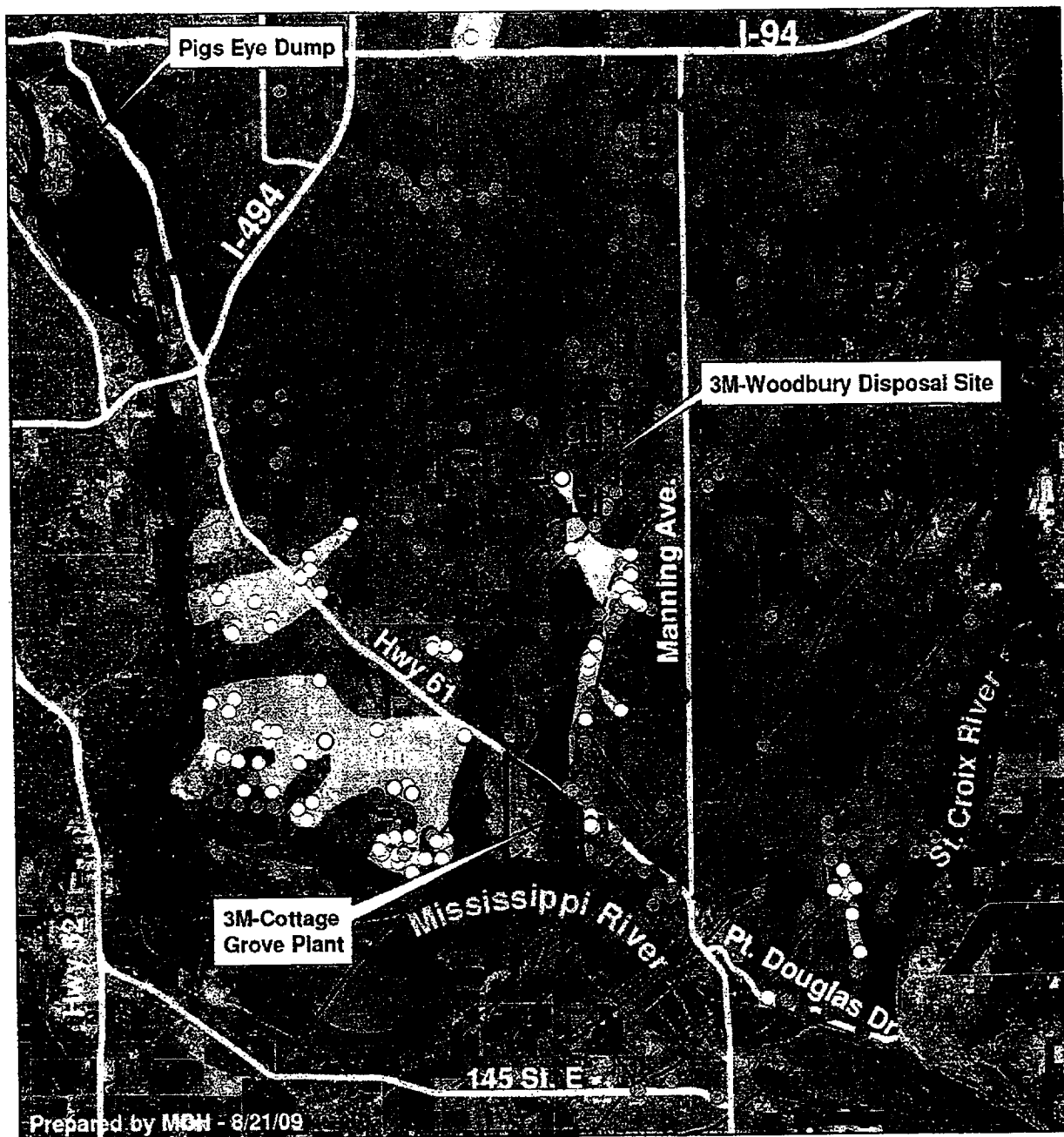
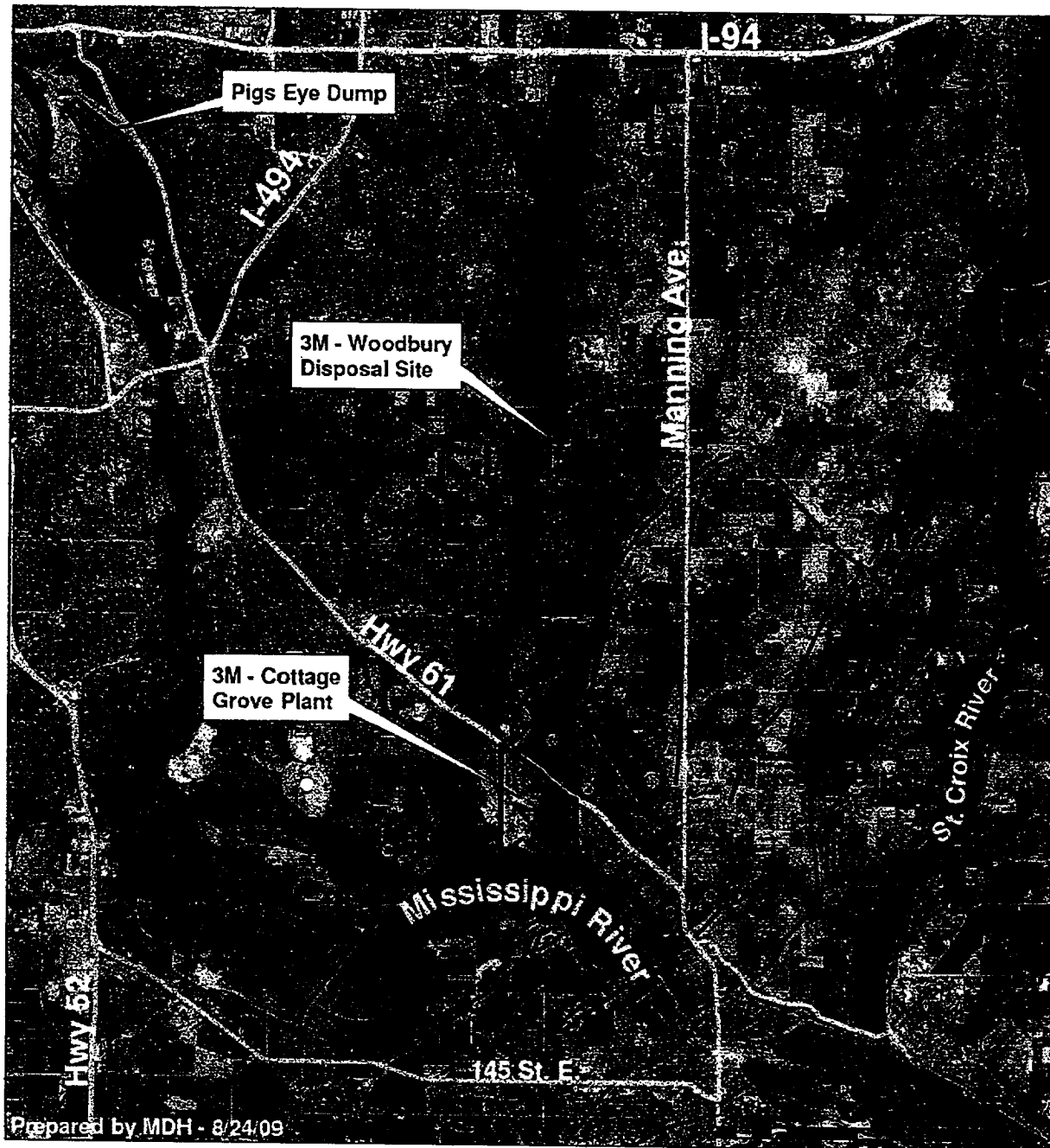


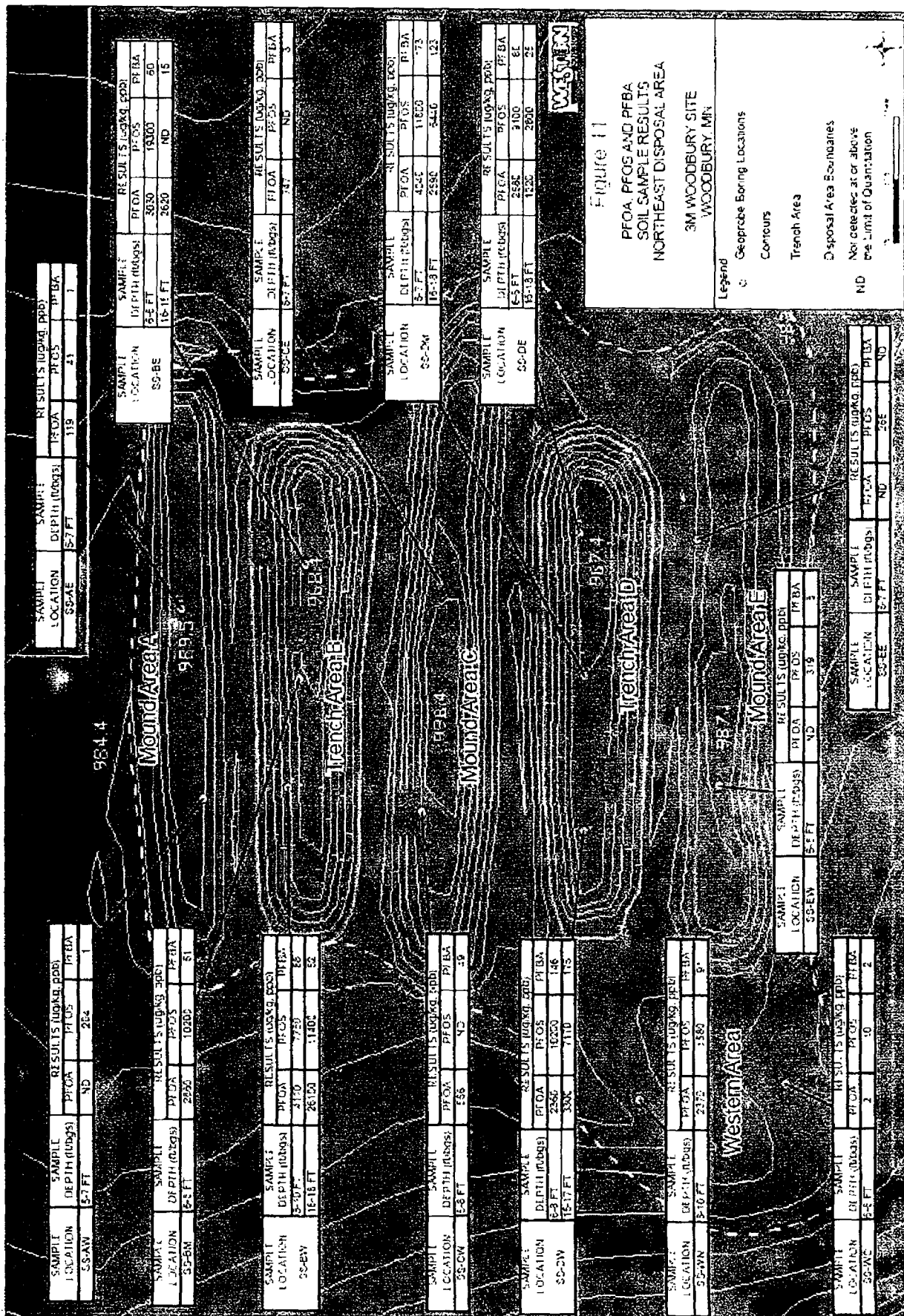
Figure 9: PFBA in the Jordan

Legend

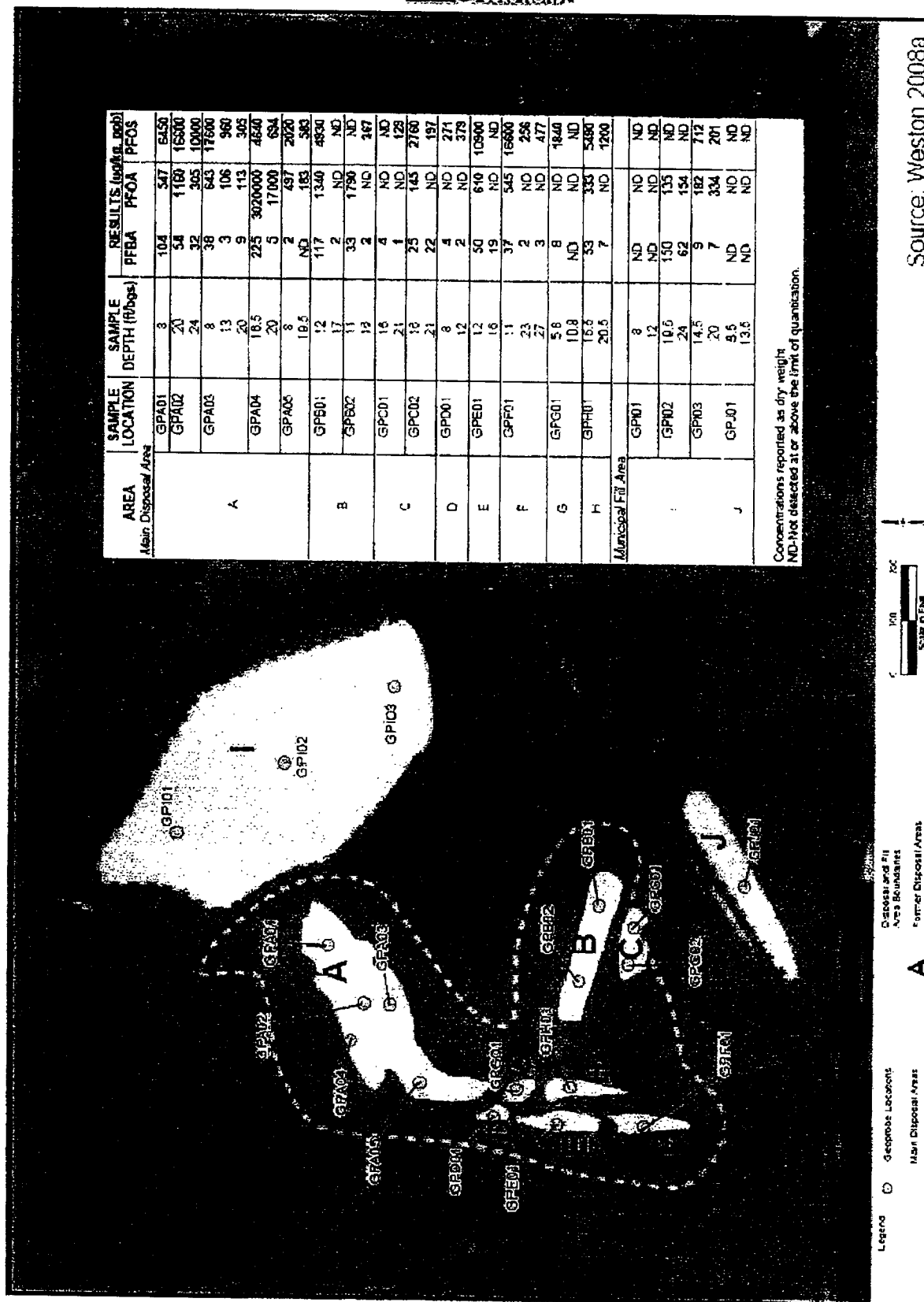
PFBA = 3.5 - 6.9 ppb	PFBA = 0.1 - 0.99 ppb	PFBA not detected	— Fault
PFBA = 1 - 3.49 ppb	PFBA < 0.1 ppb	Bedrock valley	○ sampled well

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





Source: Weston 2008a



AREA	LOCATION	SAMPLE DEPTH (ft)	RESULTS (ppm, lbs)		
			PFBA	PFOS	PFOS
A	GP101	3	104	547	6450
	GP102	20	54	1160	16500
	GP103	24	32	305	10000
	GP104	8	38	643	17600
B	GP105	13	3	106	960
	GP106	20	9	113	305
	GP107	16.5	235	3020000	4640
	GP108	20	5	17000	684
C	GP109	8	2	497	2020
	GP110	18.5	ND	183	583
	GP111	12	117	1340	4830
	GP112	17	2	ND	ND
D	GP113	11	33	1750	ND
	GP114	12	2	ND	247
	GP115	15	4	ND	ND
	GP116	21	1	ND	128
E	GP117	16	25	145	2760
	GP118	21	22	ND	197
	GP119	8	4	ND	271
	GP120	12	2	ND	378
F	GP121	12	50	610	10500
	GP122	16	19	ND	ND
	GP123	11	37	545	16000
	GP124	23	2	ND	258
G	GP125	27	3	ND	477
	GP126	5.8	8	ND	1840
	GP127	10.8	ND	ND	ND
	GP128	15.8	53	335	5480
H	GP129	20.5	7	ND	1200
	GP130				
	GP131				
	GP132				
J	GP133	8	ND	ND	ND
	GP134	12	ND	ND	ND
	GP135	10.5	130	135	ND
	GP136	24	62	154	ND
Municipal Fill Area	GP137	14.5	9	182	713
	GP138	20	7	334	201
	GP139	9.5	ND	ND	ND
	GP140	13.5	ND	ND	ND

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

Source: Weston 2008a

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

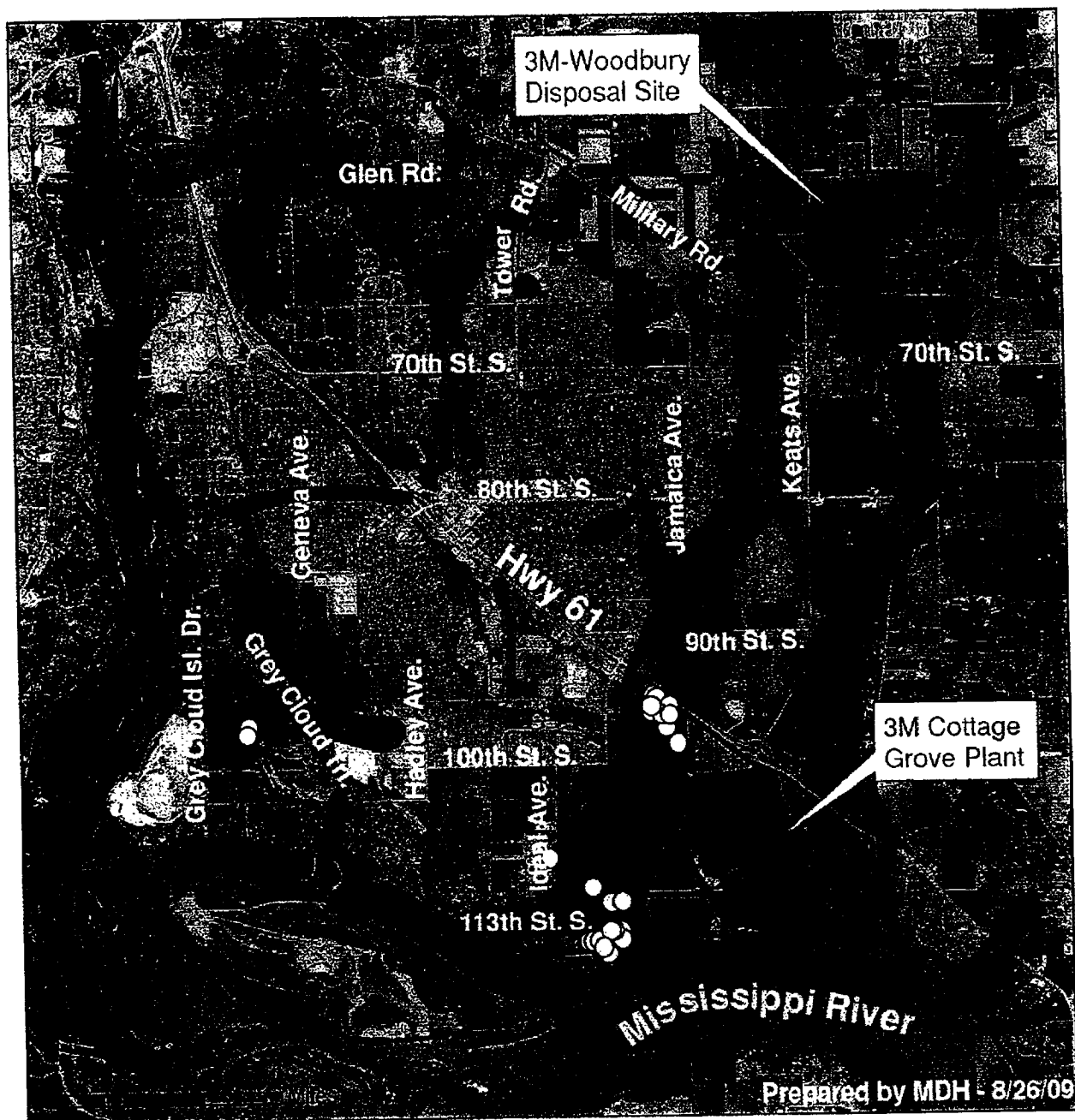
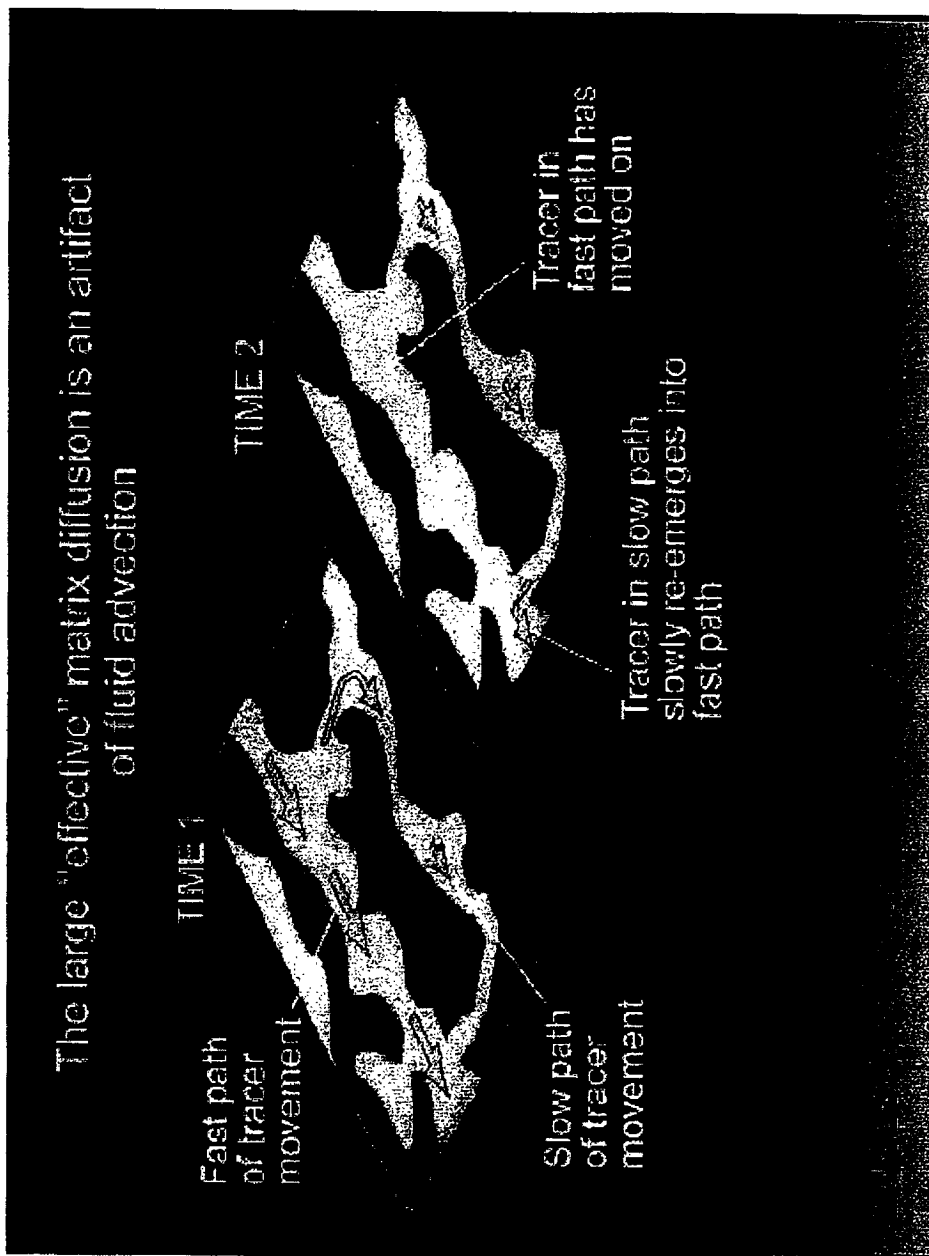


Figure 13 - Areas Where Multiple PFCs Were Detected

Legend

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

Figure 14: Secondary Flow Paths in Fractured Rock



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

Appendix 2: Tables

Table 1: Initial 3M-Woodbury Disposal Site Monitoring Well Data¹, 2005 – 2006 (in µg/L)

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

Notes:

Values shown in boldface type exceed MDH drinking water criteria. No criteria exist for PFBS or PFHxS.

¹ Data are from Weston (2007a). Results are average values calculated from two samples from same well collected on same date

² Well locations are shown in Figure 4

³ Aquifer abbreviations: CJDN = Jordan, OPDC = Prairie du Chien, OSTP = St. Peter

⁴ Combined discharge from Woodbury pumping wells

⁵ Non-contact process water discharge from retention pond at 3M-Chemolite Plant

⁶ ND = not detected

⁷ NA = not analyzed

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

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

¹ First number is maximum concentration of the compound detected in any well in the designated aquifer, in parts per billion (ppb).

Number in parentheses is the percent of wells in that aquifer in which the compound was detected at any concentration.

² ND = not detected in any well in this aquifer

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

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

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

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

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

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

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

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

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