Appendix 2

Public Comments Received

City of
Cottage Grove
Minnesota

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August 16, 2004

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

RE: Public Health Consultation – 3M Cottage Grove Facility

Dear Mr. Kelly:

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

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

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

Sincerely,

Howard Blin

Howard Blin
Community Development Director

cc: Mayor and City Council
Ryan Schroeder, City Administrator

DEPARTMENT: POLLUTION CONTROL AGENCY

STATE OF MINNESOTA

Office Memorandum

DATE: August 12 , 2004

TO: Jim Kelly, Minnesota Department of Health

FROM: David Douglas, Project Manager
Superfund Unit 2/Superfund Section
Superfund Section
Majors and Remediation Division
PHONE: 296-7818

SUBJECT: 3M Chemolite/Health Consultation

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

Summary, page 3, first paragraph

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

Summary, page 4, last paragraph, last sentence

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

Superfund Site History, page 7

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

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

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

Site D4: Phenolic Waste Pit, page 8

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

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

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

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

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

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

Washington County

Department of Public Health and Environment

Mary L. McGlothlin
Director

Lowell Johnson
Deputy Director

August 17, 2004

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

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

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

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

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

Our comments are as follows:

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

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

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

Sincerely,

Cindy Weckwerth, REHS, MS
Program Manager

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

3M General Offices

3M Center
St Paul, MN 55144-1000
651 733 1110

August 20, 2004

3M

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

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

Re: 3M Cottage Grove, MN Consultation

Dear Mr. Kelly:

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

COMMENTS ON THE CONCLUSIONS

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

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

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

- The conclusions on page 24 further state that there are limited environmental data available and thus the potential impact on public health from releases at the Cottage Grove facility cannot be assessed at this time. The statement that "the site currently represents an indeterminate public health hazard" is overly broad given the data available.

    - Data can always be said to be limited, but 3M has obtained substantial information about the geology and hydrogeology at the site and the effectiveness of the on-site well pumping system to control off-site movement of groundwater, and considerable data on the presence of PFOS and PFOA at the site and the physical and chemical characteristics of these substances. This information has been shared with MPCA.

    - There is no evidence of fluorochemicals in nearby offsite wells, and 3M has for decades operated production wells which create a cone of depression for groundwater emanating from the developed portion of the property. At this time, there is no indication that groundwater migration from the plant is a completed exposure pathway.

    - Furthermore, the production of PFOS- and PFOA-related substances was discontinued as of December 2002, thus reducing releases from the production processes. The activated carbon treatment system for plant wastewater discharges mentioned in the draft consultation report is fully operational.

COMMENTS ON THE RECOMMENDATIONS

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

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

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

- With regard to the recommendation that 3M should take action to ensure that Cottage Grove workers are not exposed to fluorochemicals via the water supply at the facility in excess of Health-Based Values,[1] the document should acknowledge that 3M has already taken steps to provide bottled drinking water to workers. Contrary to the statement on page 13, bottled water is used for drinking water and cooking, and the plant is in the process of installing a treatment system for water used in cooking, so that the kitchen need not rely on bottled water.

- Similarly, 3M will continue to take steps to identify and as appropriate reduce any potential ongoing discharges from the facility, and requests that the document acknowledge that 3M is already actively engaged in such efforts. The Granular Activated Carbon system referenced on page 13 is fully installed, not merely in the process of being installed, and has shown good removal efficiency (>99%).

- As to the fourth recommendation, to gather information regarding off-site waste disposal locations, 3M supports such a recommendation in the context of the phased investigation. A review of 3M's files with respect to off-site disposal is already underway. The phased approach will address on-site media and then off-site media with confirmed pathways

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

COMMENTS ON THE TEXT

Summary

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

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

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

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

- It would be useful to clarify that not all of the areas described in connection with the 865-acre site are relevant to fluorochemicals. We suggest some specific changes in the description of certain areas of the site and of site geology.

- Several recent reviews provide summaries of environmental, toxicologic and/or epidemiologic data on PFOS and PFOA. We suggest these reviews be referenced

- More recent or detailed information on the half-life of PFOS in animals that differs from the information cited is available. In addition, the description of the chronic studies confuses PFOS and PFOA data.

- The draft report speculates that Minnesota may lower its Health Based Values for PFOS in light of recently published data in Thibodeaux, et al. (2003) and Lau, et al. (2003). We review that new data and explain why it should not result in more stringent health-based levels than the current Minnesota calculation.

- The reference to a possible effect on estradiol in workers is unfounded. We review the data in the cited study and other pertinent studies that were not cited, and explain why we believe the statement is inappropriate. Similarly, we explain why the reference to prostate cancer in the Gilliland and Mandel mortality study is not appropriate unless accompanied by a full explanation that subsequent data do not support an association of PFOA with prostate cancer mortality.

- We provide references for updated information on general population serum levels of PFOS and PFOA. The difference in mean serum levels between the general population and workers engaged in either PFOS or PFOA fluorochemical production is about <u>two orders of magnitude</u> for PFOS and <u>three or more orders of magnitude</u> for PFOA, not one order of magnitude as indicated in the draft report.

- The draft report cites "margins of exposure" -- comparing human general population exposure to benchmark levels from the developmental study of PFOA in rats -- that are taken from a *preliminary* draft EPA document that has since been revised. We explain why the cited margins of exposure are simply <u>incorrect</u> in light of the unique pharmacokinetics affecting the excretion of PFOA in female rats. In the recently published risk assessment for PFOA (Butenhoff, et al. 2004), the authors report margins of exposure for the 95th percentile general population exposure of 2100 for post-natal effects -- a substantial margin of safety.

We elaborate on these and other comments below.

Discussion of Specific Comments

Chemical Terminology

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

Characterizations

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

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

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

Site Description

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

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

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

Geology

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

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

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

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

Disposal Areas

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

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

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

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

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

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

Fire Training Area

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

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

Air Modeling

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

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

Well Testing

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

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

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

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

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

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

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

Chemistry, Physical/Chemical Properties

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

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

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

Toxicological Information

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

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

Half-Life in Serum

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

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

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

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

Effects in Animal Studies

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

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

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

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

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

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

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

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

- The oncogenicity of PFOA has been investigated in two separate two-year feeding studies in rats. PFOA was found to increase the incidence of three tumor types, liver, Leydig cell, and pancreatic acinar cell tumors.[6] (Riker, 1983, and Biegel et al. 2001) These tumors are frequently observed in rats treated with peroxisome proliferators. It is generally recognized that rats have a heightened response to peroxisome proliferators relative to other species,

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

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

Developmental Effects

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

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

Teratology Studies

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

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

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

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

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

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

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

Reproductive and Postnatal Effects

Lau et al. (2003) reported on the postnatal evaluation of the same animals studied by Thibodeaux, et al. (2003) in a companion publication. Neonatal mortality occurred at lower doses than birth defects. The NOAEL for effects on the rat pups was 1 mg/kg/day concentration (Table 2 in Lau et al. 2003) The $LBMD_5$ values for survival at postpartum day 8 in rats was 0.58 mg/kg/day, and at postpartum day 6 in mice was 3.88 mg/kg/day. Both the NOAEL and the benchmark dose values are higher than the 0 15 mg/kg/day dose used in the Minnesota HBVs.

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

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

Study	Endpoint	$LBMD_5$ (mg/kg/day)	$LBMIC_5$ (ug/mL)
2-Gen Repro/Dev	F_1 Pup Weight Gain (LD21) [a]	0.34	26
2-Gen Repro/Dev	F_1 Pup Weight Gain (LD21) [b]	0.34	36
2-Gen Repro/Dev	F_1 Litter Size (LD4) [a]	0.39	30
2-Gen Repro/Dev	F_1 Litter Size (LD4) [b]	0.39	39
1-Gen Repro/Dev	F_1 Litter Size (LD5) [a]	0.83	71
2-Gen Repro/Dev	F_1 Pup Mortality (LD4) [a]	0.84	71
1-Gen Repro/Dev	F_1 Pup Mortality (LD5) [a]	0.83	83
2-Gen Repro/Dev	F_1 Pup Mortality (LD4) [b]	0.84	84

[a] Based on serum samples taken on GD 21
[b] Based on serum samples taken on GD 0
(Beginning of gestation values are appropriate for comparison to measured human concentrations.)

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

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

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

Epidemiologic Information

Worker Monitoring

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

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

- The draft consultation report omits the rest of the sentence from the study, which states that: "A 10% increase in mean estradiol level was observed among employees who had the highest levels of serum PFOA, although this association was confounded by body mass index." (Study abstract, emphasis added.) Body mass is a known confounder for estradiol. All five employees with PFOA levels above 30 ppm had Body Mass Indexes (BMI) of 28 or more. Id. at 617. Taking into account this potential confounding, there was no pattern of association between PFOA and estradiol levels

- The scatterplots on page 616 of the Olsen et al. 1998a paper present a clear visual representation that estradiol does not vary with increasing PFOA exposure.

    - As noted on page 617, "Simple linear regression of the natural log of [estradiol] with PFOA, treated as a continuous variable, resulted in no statistically significant coefficients . . ."

    - The text there further states that "linear and nonlinear relationships, taking into account potential confounders (especially age and BMI) as well as other covariates that may be on the biologic pathway of effect, resulted in no significant associations with PFOA except for 17-HP in the 1995 analysis."

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

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

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

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

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

Mortality Studies

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

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

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

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

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

General Population Exposure

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

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

- The general population has average levels of 0.005 ppm PFOA and 0.040 ppm PFOS. (Olsen et al. 2003b; Olsen et al 2004a; Olsen et al. 2004b.[8])

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

Margins of Exposure

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

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

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

[9] The *preliminary draft* risk assessment document calculated an estimated range for margins of exposure (MOE) between human serum concentrations of PFOA and the serum concentrations that might be found in weanling rats that experienced developmental effects in a two-generation reproductive study. Weanling rat serum concentrations of PFOA were estimated from adult levels that were measured 24 hours after dosing. Use of the adult F_0 female serum concentration from a sample obtained 24 hours after the last dose is a gross underestimate of the values likely to exist in weanling rats given that female rats excrete virtually all PFOA within 24 hours. Use of the area-under-the-curve approach to provide an average serum concentration corrects for this pharmacokinetic issue.

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

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

Environmental Data

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

* * *

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

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

Sincerely yours,

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

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

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USEPA, "Preliminary Risk Assessment of the Developmental Toxicity associated with exposure to Perfluorooctanoic Acid and its Salts," April 14, 2003

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

OFFICE OF
PREVENTION, PESTICIDES AND
TOXIC SUBSTANCES

September 1, 2004

VIA E-MAIL

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

Dear Mr. Kelly:

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

General Comments

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

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

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

Specific Comments

Page 3:

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

Page 3 and Page 16:

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

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

Page 4 and Page 21:

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

Page 13:

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

Page 22:

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

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

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

Page 24:

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

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

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

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

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

Sincerely,

/s/

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