IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF SOUTH CAROLINA
CHARLESTON DIVISION

IN RE: AQUEOUS FILM-FORMING FOAMS PRODUCTS LIABILITY LITIGATION	MDL No. 2:18-mn-2873-RMG **PLAINTIFFS' MOTION TO COMPEL DISCOVERY FROM DEFENDANT 3M COMPANY** **This Document relates to ALL CASES**

NOW COME Plaintiffs, by and through the Plaintiffs Executive Committee ("PEC"), and move this Honorable Court, pursuant to Rules 26, 34 and 37 of the Federal Rules of Civil Procedure and Local Civil Rules 7.04 and 37.01, for an order compelling Defendant 3M Company to produce the custodial file of former 3M Chief Executive Officer ("CEO") and Chairman of the Board, Lewis Lehr.

Mr. Lehr was CEO of 3M from 1979-1986 and member of its board of directors from 1974-1991, a time when 3M was aware of the potential for litigation involving widespread environmental contamination from its PFAS chemicals. Indeed, at that time, 3M was aware that a PFAS chemical it manufactured was in the blood of non-occupationally exposed people across the United States. Despite this knowledge, 3M, through its executives, including Mr. Lehr, undertook multiple efforts to conceal these facts and ultimately delayed for decades outside scientific inquiry[1] as well as litigation.

[1] *See generally* Grandjean, P. *Delayed discovery, dissemination, and decisions on intervention in environmental health: a case study on immunotoxicity of perfluorinated alkylate substances.* ENVIRON HEALTH 17, 62 (2018), attached to the Declaration of Michael A. London ("London Decl.") as Exhibit A.

It is against this backdrop that Plaintiffs file this motion relating to Mr. Lehr's custodial file. As a 3M executive, Mr. Lehr played a central role in business decisions related to investigating and reporting potential effects associated with 3M's fluorochemical products. It is undisputed that 3M has not produced Mr. Lehr's custodial file despite repeated requests, including protracted meet and confers. As a result, Plaintiffs seek an order from the Court requiring production of Mr. Lehr's custodial file.

Procedural Background

In this MDL, 3M has agreed to produce the custodial files of numerous individuals who were involved in the company's fluorochemical business. As the PEC has reviewed 3M's ongoing productions in this litigation, it has identified additional individuals who it believes may possess relevant information and has requested production of those individuals' custodial files. In October 2021, the PEC identified Lewis Lehr as an additional custodian and requested that 3M produce his custodial file. 3M agreed to conduct a reasonable search of Mr. Lehr's documents.[2] After almost a month without any updates on 3M's search and production of Mr. Lehr's custodial file, the PEC requested a date by which the PEC could expect to receive the completed production.[3] Two days later, 3M responded that it "has not identified a custodial file, nor documents associated with a custodial file (whether hard copy or electronic), for Lewis Lehr whose employment tenure at 3M ended in 1986, aside from a single nonresponsive lab notebook."[4] 3M reiterated its findings, or lack thereof, during a subsequent telephonic meet and confer on November 30, 2021, explaining that while certain documents produced in this litigation mention Mr. Lehr, 3M was unable to

[2] *See* Email correspondence dated Oct. 20, 2021 and Oct. 25, 2021, attached to the London Decl. as Exhibit B, at pp. 2-3.

[3] *Id.* at p. 1.

[4] *Id.*

identify a custodial file with respect to the agreed-upon search terms because of the timeframe of Mr. Lehr's employment with 3M.

Through its review, however, the PEC has identified 283 documents in 3M's productions referencing Mr. Lehr, many of which appear to have been created while Mr. Lehr was a 3M employee, which are highly relevant to the core issues in this case. As a result, the PEC requested that 3M search its records once again to confirm the existence of Mr. Lehr's custodial file.[5] During the Parties' second telephonic meet and confer on this topic on December 16, 2021, 3M indicated that after a subsequent search, it had still only identified Mr. Lehr's technical lab notebook.

Despite the Parties' extensive meet and confer efforts, 3M has failed to produce these documents or otherwise provide a reasonable explanation as to why some historical documents from the same timeframe were preserved, while the custodial file of Mr. Lehr was not.

Factual Background

In order to appreciate the relevance and importance of Mr. Lehr's custodial file, the following factual and historical context is important.

On May 15, 1998, 3M notified the Environmental Protection Agency ("EPA") that it had determined that its proprietary chemical, perfluorooctane sulfonate ("PFOS"), of which 3M was the primary U.S. manufacturer, was widespread in the environment and present in the blood of virtually every man, woman and child.[6] Shortly after this discovery,[7] 3M discontinued

[5] *See* Letter from David Hoyle to Daniel L. Ring, dated Dec. 7, 2021, attached to the London Decl. as Exhibit C.

[6] *See* 3M_BELL02796621, attached to the London Decl. as Exhibit D.

[7] On May 4, 2000, 3M provided the EPA with copies of 149 internal 3M studies dating from 1975 which filled "several boxes." *See* Document #448, attached to the London Decl. as Exhibit E, at #448.2. These internal 3M studies addressed the physical and chemical properties, environmental fate and transport, environmental monitoring, ecotoxicity, acute toxicity, genotoxicity, repeated-dose toxicity, pharmacokinetics, teratology, medical surveillance, and epidemiology of PFOS. Subjects of these toxicity studies included rats, monkeys, rabbits, albatross, goats, fish, bald eagles,

manufacturing this family of chemicals, which was responsible for more than $300 million in annual sales.[8] Similarly, the EPA effectively banned others from making or importing these same chemicals stating that "these chemical substances may be hazardous to human health and the environment."[9] In statements to the press, 3M claimed they were only able to make this discovery due to recent advancements in analytical techniques, and further claimed that the discovery of PFOS in the blood of the general population in the late 1990s was "a complete surprise."[10] Contrary to this carefully crafted narrative, the truth was that 3M possessed this knowledge for more than *20 years*, and had spent two decades actively hiding, distracting or misleading those outside of 3M about this important public health matter.

In the summer of 1975, Dr. Warren Guy, a toxicologist and professor at the University of Florida, called 3M's corporate headquarters concerning research he and Dr. Donald Taves, a toxicologist and professor at the University of Rochester, were going to present at a symposium organized by the American Chemical Society ("ACS"). Dr. Guy and Dr. Taves had discovered the presence of an unidentified organic fluorine chemical compound in human blood obtained from blood banks in five U.S. cities. According to an internal 3M memorandum documenting these phone calls, Dr. Guy called 3M to see if it knew of the "possible sources" as Dr. Guy correctly "got the information that 3M's fluorocarbon carboxylic acids are used as surfactants and wanted

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mice, guinea pigs, quail, mallard ducks, mink, river otters, and oysters. Twelve days later, and following "negotiations" with the EPA, 3M announced that it was "voluntarily" phasing out production of PFOS. *See* 3M_BELL00848209, attached to the London Decl. as Exhibit F, at 3M_BELL00848209; *see also* 3M_AFFF_MDL00207575, attached to the London Decl. as Exhibit G.

[8] *See* Olsen Deposition Exhibit LP193, attached to the London Decl. as Exhibit H, at p. 3.

[9] https://www.federalregister.gov/documents/2002/12/09/02-31011/perfluoroalkyl-sulfonates-significant-new-use-rule.

[10] *See* Exhibit H, Olsen Deposition Exhibit LP193, at p. 3.

to know if they were present in 'Scotchgard' or other items in general use by the public." Incredulously, 3M chose to "plead ignorance" and instead "adopted a position of scientific curiosity and desire to assist in any way possible..."[11]

In another phone call, Dr. Taves specifically asked 3M if the "fluorochemical they have found in human blood is either a derivative of a perfluorocarboxylic acid or a perfluorosulphonic acid," and whether "the fluorochemical found in the blood might be coming from [3M's] paper or paperboard" products.[12] On August 26, 1975, Dr. Guy and Dr. Taves presented their research at an ACS symposium in Chicago. Shortly thereafter, on October 16, 1975, Dr. Guy sent 3M a copy of the paper presented in Chicago.[13] Dr. Guy asked in return that: "[i]f you or other interested parties at 3M have any ideas on how we can better characterize these fluorocompounds please let either Dr. Taves or me know."[14]

The manuscript detailed the methods used by Drs. Guy and Taves to conclude that organic fluorine had been found on average at 30 parts per billion in the blood of the general population.[15] Dr. Guy and Dr. Taves stated that based on their analysis, "the fluorine containing part of the compounds in the isolate (from human plasma) resemble perfluorooctanoic acid ("PFOA")."[16] The manuscript further posited: "These findings suggest that there is widespread contamination of human tissues with trace amounts of organic fluorocompounds derived from commercial products. All available information on this subject is in accordance with this

[11] *See* 3M_AFFF_MDL00419718, attached to the London Decl. as Exhibit I.

[12] *See* 3M_BELL00054741, attached to the London Decl. as Exhibit J, at 3M_BELL00054741.

[13] *See* 3MA00257421, attached to the London Decl. as Exhibit K.

[14] *Id*. at 3MA00257421.

[15] *Id*. at 3MA00257423.

[16] *Id.* at 3MA00257430.

interpretation."[17] Included within this manuscript was the below spectra, based upon nuclear magnetic resonance (NMR) analysis, of the organic fluorine found by Drs. Guy and Taves in blood bank blood[18]:

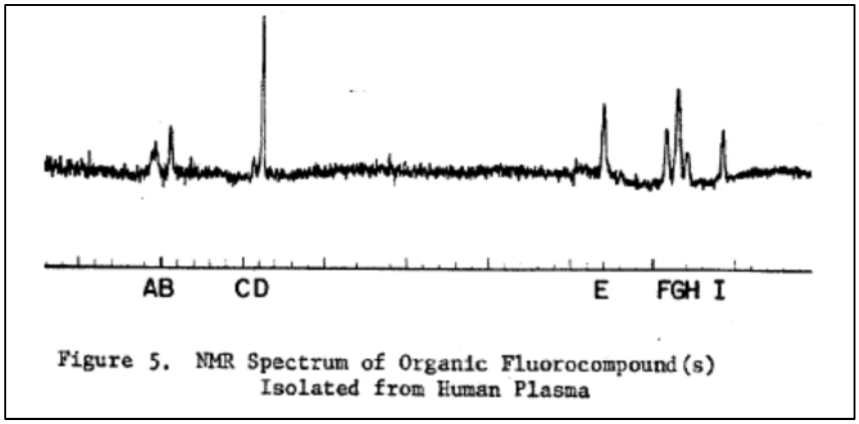

Figure 5. NMR Spectrum of Organic Fluorocompound(s) Isolated from Human Plasma

In response to the manuscript authored by Drs. Guy and Taves, 3M's Commercial Chemicals Division Laboratory submitted samples of ten different "perfluorocarboxylic and perfluorosulfonic acid derivatives [made by 3M] to Central Research Analytical for 19F NMR analysis in an attempt to identify the material found by Guy and Taves in human blood."[19] On November 6, 1975, 3M scientist Richard Newmark, of the Central Analytical Laboratory ("CAL"), authored a report which compared the ten chemical compounds made by 3M to the chemical discovered by Drs. Guy and Taves.[20] Dr. Newmark concluded that the chemical spectrum presented by Drs. Guy and Taves "resembled most closely" PFOS, the PFAS compound manufactured exclusively by 3M and not PFOA.[21] Despite this conclusion, 3M never provided Dr. Guy or Dr. Taves with this information. Instead, an internal 3M timeline indicates that,

[17] *Id.*

[18] *Id.* at 3MA00257442.

[19] *See* 3M_BELL00054589, attached to London Decl. as Exhibit L.

[20] *See* 3MA00967400, attached to the London Decl. as Exhibit M.

[21] *Id.*

"[a]ccording to Richard Newmark, 3M lawyers urge CAL not to release the true identity (PFOS) of the [organic fluorine] compound."[22] Without this information, Drs. Guy and Taves published their manuscript in the *Proceedings of the American Chemical Society* documenting their continued search for the source of organic fluorine in the blood bank blood – a source identified and kept secret by 3M.

In light of this startling discovery, namely that their proprietary chemical was found in the blood of the general population, 3M attempted to identify which specific 3M products could be responsible for the presence of PFOS in the blood of the general population. While 3M manufactured pure PFOS for use as an industrial surfactant, it represented a tiny fraction of their PFAS production and surfactants were not believed to be in routine contact with the public at large.[23] However, two of 3M's chemically-related (PFOS precursor) products were in contact with the general public – Scotchgard stain repellant and Scotchban food packing paper. In 1977, 3M fed Scotchban and Scotchgard to rats and mice and discovered that both 3M products metabolize to PFOS in the blood.[24] The study author told his colleagues that this was a "significant finding," and concluded that "the public health issue [is] simply one of frequency and type of exposure to 3M products."[25]

Internal 3M documents indicate that Drs. Guy and Taves' finding of PFOS in the blood of the general population was the initiating "event" that led to toxicology studies conducted on rats,

[22] *See* 3M_BELL00054594, attached to the London Decl. as Exhibit N.

[23] As of 1980 3M manufactured 16,000 pounds a year of pure PFOS while manufacturing several million pounds of PFOS precursors.

[24] *See* Exhibit L, 3M_BELL00054589.

[25] *See* 3MA10035579, attached to the London Decl. as Exhibit O, at 3MA10035580.

mice and monkeys.[26] These studies, which were concluded in 1979, reported that "[PFOS] was the most toxic of the three compounds studied and certainly more toxic than anticipated."[27] The 90-day monkey study, for example, reported "GI tract toxicity, lipid depletion of adrenals, atrophy of pancreatic exocrine cells and serous alveolar cells of the salivary glands."[28]In total, 20 of the 28 rhesus monkeys in the study died as a result of their exposure to PFOS.[29]

3M CEO and Chairman of the Board, Lewis Lehr

News that a proprietary chemical made exclusively by 3M was toxic and present in the blood of the general population attracted the attention of 3M's highest-ranking executives, including Mr. Lehr. The incomplete production of Mr. Lehr's file clearly demonstrates his active involvement in this burgeoning crisis within 3M. For example, a May 26, 1978 memo states, "[t]his will confirm arrangements made for a meeting at 9:30am July 12 in Mr. Lehr's conference room […] on the subject of fluorochemicals in blood."[30] Importantly, it was Mr. Lehr who decided to seek advice from experts outside of 3M to determine whether 3M was required to inform the EPA of their discovery:

> This meeting is being called to consider the use of an outside consultant to review our results to date in the fluorochemicals in blood program. Mr. Lehr has specifically requested that an outside consultant review our results and render an independent opinion as to whether we are correct in our assumption that we do not have a reportable situation under Section 8(e) of the Toxic Substances Act.[31]

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[26] *See* 3M_AFFF_MDL00080683, attached to the London Decl. as Exhibit P, at 3M_AFFF_MDL00080700.

[27] 3M_AFFF_MDL02174949, attached to the London Decl. as Exhibit Q, at 3M_AFFF_MDL02174949.

[28] *See* 3M_AFFF_MDL00080683, Ex. P, at 3M_AFFF_MDL0080705.

[29] *Id*. at 3M_AFFF_MDL0080704-05.

[30] 3M_AFFF_MDL02342766, attached to the London Decl. as Exhibit R.

[31] 3M_AFFF_MDL02342749, attached to the London Decl. as Exhibit S.

In response to Mr. Lehr's directive, 3M sought consultation from two outside scientists: Dr. Jerry R. Mitchell, M.D., Ph.D., a Professor of Chemical Toxicology at the Baylor College of Medicine and Harold C. Hodge, Ph.D., a Professor of Pharmacology and Oral Biology at the University of California San Francisco who was the first president of the Society of Toxicology.

On April 12, 1979, nine 3M employees, including Les Krogh, a 3M Division Vice President, as well as the company's Medical Director, Dr. Frank Ubel, traveled on a 3M Company Gulfstream II jet to San Francisco to meet with Dr. Hodge.[32] After presenting data from the 90-day toxicology studies performed on rats, mice, and monkeys, Dr. Hodge encouraged 3M to perform metabolism studies on its Scotchban paper packaging product, stating this was of the "utmost importance," and to then determine if those metabolites are present in the blood of the general population. Dr. Hodge's initial assessment was that: "we could have a serious problem."[33] Most troubling, and quite revealing as to 3M's state of mind at this time, is the fact that this stark assessment by Dr. Hodge was deleted from the draft meeting minutes prepared by 3M and thus do not appear in the official report from this meeting.[34]

Similarly, one day after the meeting with Dr. Hodge, the same nine 3M employees traveled on a 3M Company plane to Houston, Texas, to meet with another external expert, Dr. Jerry R. Mitchell.[35] 3M representatives presented the same toxicology data they presented to Dr. Hodge the prior day. Like Dr. Hodge, Dr. Mitchell provided 3M with a stark assessment of the data. Dr. Mitchell told 3M that "some of the symptoms in animals from these 90-day studies are similar to

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[32] *See* 3M_AFFF_MDL00435666, attached to London Decl. as Exhibit T, at 3M_AFFF_MDL00435666; 3M_BELL03185972, attached to London Decl. as Exhibit U.

[33] *See* 3MA00967775, attached to London Decl. as Exhibit V, at 3MA00967780.

[34] *See* 3M_AFFF_MDL00647433, attached to London Decl. as Exhibit W.

[35] *See* 3M_BELL01440050, attached to London Decl. as Exhibit X.

those observed with carcinogens."[36] Incredulously, this statement by Dr. Mitchell was also removed from the final draft of the meeting minutes.[37] This statement by Dr. Mitchell takes on additional significance when one considers another element that was discussed in their meeting: legal liability. At the conclusion of the meeting, Dr. Mitchell prepared a slide to summarize the meeting. In his slide summary, Dr. Mitchell identified "[p]ublic health" and "[e]nvironmental (fish, fowl, etc.)" as categories of "People at Risk." Further, under a heading titled "Legal Issues," Dr. Mitchell enumerated both "Human Injury" as well as "TSCA Sec. 8(e)."[38]

The Toxic Substances Control Act ("TSCA")[39] was passed by Congress in 1976 to protect the public from chemicals that present an "unreasonable risk of injury to health or the environment." One of the primary ways that TSCA implements this mandate is a requirement under section 8(e) that a chemical manufacturer must "immediately inform" the EPA of "information which reasonably supports the conclusion that such substance or mixture presents a substantial risk of injury to health or the environment." "Substantial Risk" has been defined by the EPA as "any non-trivial adverse effect, heretofore unknown to the administrator, associated with a chemical known to have bioaccumulated to a pronounced degree or to be widespread in the environment."[40] Indeed, 3M invoked TSCA 8(e) when it finally reported the widespread presence of PFOS in the blood of the general population to the EPA in 1998.[41] As the evidence and record

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[36] *See* 3MA10034826, attached to London Decl. as Exhibit Y, at 3MA10034828.

[37] *See* 3M_BELL01440050, Ex. X.

[38] *See* 3MA10034826, Ex. Y, at 3MA10034829.

[39] As the Court may recall from prior Joint Status Reports, the PEC has had to threaten 3M with motion practice in order to obtain documents from 3M related to TSCA.

[40] *See* 3M_AFFF_MDL00410829, attached to London Decl. as Exhibit Z, at 3M_AFFF_MDL00410830.

[41] *See* 3M_BELL02796621, Ex. D.

is clear, this is almost twenty years after Mr. Lehr consulted with colleagues within 3M and consultants external to 3M, and Mr. Lehr made the decision to not inform the EPA and instead to keep this important matter of public health a secret.[42]

As set forth above, multiple documents reveal that as early as 1979, 3M was on notice of a profound threat to public health posed by their products. Accordingly, 3M actively contemplated litigation and regulatory implications resulting from its knowledge of this significant threat to public health, which, Plaintiffs submit, triggered its duty to preserve documentary and related evidence. However, rather than preserve evidence as a result of a clear contemplation of anticipated litigation, 3M appears to have actively attempted to re-write its history and/or purge incriminating evidence from its files.

It is against this factual backdrop[43] that the instant motion is before the Court.

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[42] *See* August 19, 2021, Deposition Transcript of Fed. R. Civ. 30(b)(6) witness John Gerber, at 90:23-91:25, attached to the London Decl. as Exhibit AA.

> Q: True or false: By 1980, 3M was in possession of information that PFOS was a bioaccumluative compound, that it was widespread in the blood of the general population, and that it killed rhesus monkeys
> that were exposed to it. […]
>
> A: Based on my review of the documents, **3M had all of – had those pieces of information** […] but […] all of that information needs to be put together and judgment applied to making a TSCA 8(e) reporting
> decision.
>
> Q: Right. And 3M did that. 3M had all of that information and **decided not to disclose** it at that time in 1980, right?
>
> A: **Yes**. I've reviewed documents that – you know, after the – those studies were conducted, that information was reviewed against EPA's reporting criteria, and the company made the determination that the information was not substantial risk information under TSCA 8(e).

[43] Parallel to 3M's internal studies on the toxicity and metabolism of their products that they knew were in the blood of the general population, 3M made efforts to conceal this very information from the public. For example, E. I. du Pont de Nemours ("DuPont") was a major customer of 3M's

Legal Standard

Rule 26 of the Federal Rules of Civil Procedure provides that a party may "obtain discovery regarding any non-privileged matter that is relevant to any party's claim or defense and proportional to the needs of the case…." Fed. R. Civ. P. 26(b)(1). "[T]he discovery rules are given 'a broad and liberal treatment.'" *Harry v. Pilgrim's Pride Corp.*, No. 3:12-cv-2268-CMC-SVH, 2013 U.S. Dist. LEXIS 22700, at *2 (D.S.C. Feb. 20, 2013) (quoting *Nat'l Union Fire Ins. Co. of Pittsburgh, P.A. v. Murray Sheet Metal Co. Inc.*, 967 F.2d 980, 983 (4th Cir. 1992)). The scope of discovery permitted by Rule 26 is designed to provide a party with information reasonably necessary to afford a fair opportunity to develop its case. *Nat'l Union*, 967 F.2d at 983; *Ashmore v. Owens*, 2017 U.S. Dist. LEXIS 105543, at *2-3 (D.S.C. June 2, 2017). 3M, as the party resisting discovery, "bears the burden of persuading the court that the requested information is outside the scope of discovery." *Ashmore*, 8:15-cv-02373-JMC, 2017 U.S. Dist. LEXIS 105543, at *3. 3M cannot meet its burden.

As this Court has previously recognized, the Rule 26 standard "shifts heavily toward production" in this MDL, where there are more than 100 consolidated cases, important claims, and

PFOA – the chemical mis-identified by Drs. Guy and Taves as the mystery organic fluorine found in sera. According to a memo from a 1978 meeting between DuPont and 3M, after DuPont asked 3M about the findings of Drs. Guy and Taves, and whether or not they agreed that PFOA was the mystery compound in the blood of the general population, "they were told that we disagreed, but were given no further clarification." *See* 3M_AFFF_MDL00419874, attached to London Decl. as Exhibit BB. Even worse, in 1980, 3M published in the peer-reviewed literature that the mystery chemical observed by Guy and Taves was not a man-made chemical at all but was instead a naturally occurring substance. *See* Belisle, J. *Organic Fluorine in Human Serum: Natural Versus Industrial Sources*. Science, Vol. 212, 26 June 1981, attached to London Decl. as Exhibit CC.

significant amounts in controversy.[44] As the Court noted on April 3, 2020, "discovery in an MDL is robust."[45]

Argument

I. 3M had an obligation to preserve these documents.

3M had a duty in 1979 to preserve the custodial file of Lewis Lehr as it knew or reasonably should have known that these documents were relevant to future litigation. *Silvestri v. General Motors Corp.*, 271 F.3d 583, 590 (4th Cir. 2001). "The duty to preserve material evidence arises not only during litigation but also extends to that period before the litigation when a party reasonably should know that the evidence may be relevant to anticipated litigation." *Id*. at 591; *see also Struthers Patent Corp. Nestle Co.*, 558 F. Supp. 747, 765-66 (D.N.J. 1981) (holding the destruction of documents which a party knew or should have known would be relevant to a future lawsuit is sanctionable); *Cecil Corley Motor Co. v. Gen. Motors Corp.*, 380 F. Supp. 819, 859 (M.D. Tenn. 1974) ("When a litigant destroys, removes, or withholds records or documents while litigation is pending, or even while litigation is being contemplated, the strongest inferences may be drawn against that party which the opposing evidence in the record permits.")

While it is difficult to articulate a precise date when 3M was on notice of future litigation involving PFAS—because this notice goes back nearly as long as 3M has manufactured PFAS—certainly the requirement for notice was met by November 6, 1975, the date 3M scientist Dr. Newmark determined that a 3M proprietary chemical was in the blood of non-occupationally exposed Americans. Such a duty is only strengthened by the contemporaneous efforts by 3M attorneys and others within 3M to prevent the release of 3M's internal knowledge that its chemical

[44] *See* December 13, 2019 CMC Transcript at 6:7-15.

[45] *See* April 3, 2020, CMC Transcript at 32:19-20.

compound PFOS was in the blood of non-occupationally exposed Americans. Similarly, in a document which has a "date received" stamp of August 15, 1980 by "3M Toxicology," 3M prepared 12 pages of "Some Probable Questions on 3M Fluorochemicals with Suggested Answers."[46] Section K of that document addressed "Legal Issues" as set forth below:

> K-1 What liability is 3M willing to accept for any ill effect that we, the customers, determine now or in the future?
>
> K-2 Does 3M face any law suits as a result of causing elevated levels of fluorine in anybody's blood?[47]

Notably, in 1980, 3M left the "suggested answers" for these specific "probable questions" concerning "legal issues" unanswered.[48]

In other words, by the time that 3M was certainly well aware of potential health effects (it was likely aware of these for at least 10 years), possible lawsuits, and potential environmental exposure, 3M *knew* that its chemical compound PFOS was in the blood of non-occupationally exposed Americans. As previously discussed, Mr. Lehr held a powerful position in the company at the time and played a key role in discussions related to what information 3M knew about fluorochemicals in blood versus what it would share with the industry, the public, and government agencies. Thus, there can be no question that 3M was under an obligation at that time to preserve his custodial file.

II. 3M has produced documents from the same time period when Mr. Lehr was a 3M executive.

[46] *See* 3M_BELL01939676, attached to London Decl. as Exhibit DD.

[47] *Id*. at 3M_BELL01939688.

[48] *Id*.

As of February 14, 2022, 3M has produced 756,797 documents in this litigation, approximately 29,209 of which were created during Mr. Lehr's tenure as CEO from 1979-1980 and CEO and Chairman of the Board from 1980-1986. The fact that 3M has produced other historical documents from this timeframe strongly suggests that the requested documents were maintained by 3M at least at some point in time. For example, 3M has produced seemingly irrelevant documentation authorizing the use of 3M's corporate airplane to facilitate its employees' meetings in 1979 with outside consultants, and yet claims that it did not preserve the files of its former CEO and Chairman of the Board because his employment with 3M ended in 1986.

Given the context surrounding 3M's investigations of fluorochemicals in blood and the potential impact those findings would have had on 3M's business when Mr. Lehr was CEO of the company, Plaintiffs are entitled to know what information was or was not available to him. The Federal Rules of Civil Procedure and the principles articulated by this Court concerning discovery in this MDL require nothing less than the immediate production of his custodial file.

Conclusion

For the reasons discussed above, Plaintiffs respectfully request that this Honorable Court enter an order compelling Defendant 3M Company to produce the custodial file of former 3M CEO and Chairman of the Board, Lewis Lehr, as well as such other and further relief that this Court deems appropriate.

Dated: February 15, 2022

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CERTIFICATE OF SERVICE

I hereby certify that a true and correct copy of the foregoing was electronically filed with this Court's CM/ECF on this 15th day February, 2022 and was thus served electronically upon counsel of record.

/s/ Fred Thompson, III

UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF SOUTH CAROLINA
CHARLESTON DIVISION

IN RE: AQUEOUS FILM-FORMING FOAMS PRODUCTS LIABILITY LITIGATION	MDL No. 2:18-mn-2873-RMG This Document relates to: ALL CASES

DECLARATION OF MICHAEL A. LONOND IN SUPPORT OF PLAINTIFFS' MOTION TO COMPEL DISCOVERY FROM DEFENDANT 3M COMPANY

I, Michael A. London, hereby declare as follows:

1. I am a member of the law firm Douglas & London and Co-Lead Counsel of the Plaintiffs' Executive Committee. I submit this declaration in support of Plaintiffs' Motion to Compel Discovery from Defendant 3M Company ("3M").

2. Attached hereto as Exhibit A is a true and correct copy of Grandjean, P. *Delayed discovery, dissemination, and decisions on intervention in environmental health: a case study on immunotoxicity of perfluorinated alkylate substances.* ENVIRON HEALTH 17, 62 (2018).

3. Attached hereto as Exhibit B is a true and correct copy of the email correspondence dated Oct. 20, 2021 and Oct. 25, 2021.

4. Attached hereto as Exhibit C is a true and correct copy of a letter from David Hoyle to Daniel L. Ring, dated Dec. 7, 2021.

5. Attached hereto as Exhibit D is a true and correct copy of the document bearing bates number 3M_BELL02796621.

6. Attached hereto as Exhibit E is a true and correct copy of a document bearing document identification number #448.

7. Attached hereto as Exhibit F is a true and correct copy of the document bearing

bates number 3M_BELL00848209.

8. Attached hereto as Exhibit G is a true and correct copy is a true and correct copy of the document bearing bates number 3M_AFFF_MDL00207575.

9. Attached hereto as Exhibit H is a true and correct copy is a true and correct copy of the Olsen Deposition Exhibit LP193.

10. Attached hereto as Exhibit I is a true and correct copy of the document bearing bates number 3M_AFFF_MDL00419718.

11. Attached hereto as Exhibit J is a true and correct copy of the document bearing bates number 3M_BELL00054741.

12. Attached hereto as Exhibit K is a true and correct copy of the document bearing bates number 3MA0025742.

13. Attached hereto as Exhibit L is a true and correct copy of the document bearing bates number 3M_BELL00054589.

14. Attached hereto as Exhibit M is a true and correct copy of the document bearing bates number 3MA00967400.

15. Attached hereto as Exhibit N is a true and correct copy of the document bearing bates number 3M_BELL00054594.

16. Attached hereto as Exhibit O is a true and correct copy of the document bearing bates number 3MA10035579.

17. Attached hereto as Exhibit P is a true and correct copy of the document bearing bates number 3M_AFFF_MDL00080683.

18. Attached hereto as Exhibit Q is a true and correct copy of a document bearing bates number 3M_AFFF_MDL02174949.

19. Attached hereto as Exhibit R is a true and correct copy of the document bearing bates number 3M_AFFF_MDL02342766.

20. Attached hereto as Exhibit S is a true and correct copy of the document bearing bates number 3M_AFFF_MDL02342749.

21. Attached hereto as Exhibit T are true and correct copy of the document bearing bates number 3M_AFFF_MDL00435666.

22. Attached hereto as Exhibit U is a true and correct copy of the document bearing bates number 3M_BELL03185972.

23. Attached hereto as Exhibit V is a true and correct copy of the document bearing bates number 3MA00967775.

24. Attached hereto as Exhibit W is a true and correct copy of the document bearing bates number 3M_AFFF_MDL00647433.

25. Attached hereto as Exhibit X is a true and correct copy of the document bearing bates number 3M_BELL01440050.

26. Attached hereto as Exhibit Y is a true and correct copy of the document bearing bates number 3MA10034826.

27. Attached hereto as Exhibit Z is a true and correct copy of the document bearing bates number 3M_AFFF_MDL00410829.

28. Attached hereto as Exhibit AA is a true and correct copy of the relevant portions of the deposition transcript of 3M Fed. R. Civ. 30(b)(6) witness John Gerber.

29. Attached hereto as Exhibit BB is a true and correct copy of the document bearing bates number 3M_AFFF_MDL00419874.

30. Attached hereto as Exhibit CC is a true and correct copy of the document titled

Organic Fluorine in Human Serum: Natural Versus Industrial Sources. Science, Vol. 212, 26 June 1981.

31. Attached hereto as Exhibit DD is a true and correct copy of the document bearing bates number 3M_BELL01939676.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on February 15, 2022 in New York, New York.

s/ Michael A. London
Michael A. London

EXHIBIT A

Grandjean *Environmental Health* (2018) 17:62
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Environmental Health

EDITORIAL Open Access

Delayed discovery, dissemination, and decisions on intervention in environmental health: a case study on immunotoxicity of perfluorinated alkylate substances

Philippe Grandjean[1,2]

Abstract

Identification and characterization of environmental hazards that impact human health must rely on the best possible science to inform and inspire appropriate public health intervention. The perfluorinated alkylate substances (PFASs) are persistent emerging pollutants that are now being recognized as important human health hazards. Although the PFASs have been produced for over 60 years, academic research on environmental health aspects has appeared only in the most recent 10 years or so. In the meantime, these persistent chemicals accumulated in the global environment. Some early studies e.g., on population exposures and toxicity, were not released to the public until after year 2000. Still, the first PFAS risk assessments ignored these reports and relied on scant journal publications. The first guidelines and legal limits for PFAS exposure, e.g., from drinking water, were proposed 10 years ago. They have decreased substantially since then, but remain higher than suggested by data on human adverse effects, especially on the immune system, that occur at background exposure levels. By now, the best-known PFASs are being phased out, and related PFASs are being introduced as substitutes. Given the substantial delays in discovery of PFAS toxicity, in dissemination of findings, and in regulatory decisions, PFAS substitutes and other persistent industrial chemicals should be subjected to prior scrutiny before widespread usage.

Late emergence of early evidence

Industrial chemicals are often regarded inert or safe, unless proven otherwise, i.e., the so-called "untested chemicals assumption," although this belief is of course not logical [1, 2]. A high-priority group of environmental chemicals, the perfluorinated alkylate substances (PFASs), constitute a clear example how narrow reliance on published toxicity studies can be misleading and result in insufficient and delayed protection of public health [3]. New insight on PFAS immunotoxicity shows that the path from discovery of toxicity to decisions on intervention can be stalled for decades (Table 1).

After the beginning of commercial PFAS production in the 1950s, a brief review article from 1980 [4] for the first time mentioned industry-sponsored studies, some of which were carried out in monkeys. Perfluorooctanoic acid (PFOA) showed specific toxicity to the reticuloendothelial system (i.e. immune system) [5]. In this 90-day study, compound-related microscopic lesions were seen in bone marrow, spleen and lymph nodes, thus clearly suggesting immunotoxicity, although functional tests were not carried out. A parallel study on perfluorooctanoic sulfonic acid (PFOS), also from 1978, was aborted due to mortality of the monkeys at all doses (the lowest being 10 mg/kg/day) [6]. These two internal reports were eventually shared with the U.S. Environmental Protection Agency (EPA) in 2000 [7] and then became accessible to the public.

A medical thesis from 1992 mentioned the evidence from the monkey study and noted: "No follow-up studies of these observations have been reported" [8]. The thesis analyzed clinical examination data from PFOA production workers and found clear associations between increased PFAS concentrations in the blood and decreased

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Table 1 Time course of important developments regarding PFAS exposure and health risks [5, 6, 8, 10, 11, 13, 15, 16, 28, 31, 32, 44, 50]

Year	Exposure evidence	Reference
1968	Organic fluoride compounds discovered in human blood	[11]
1976	Organofluorines determined in blood from production workers	[10]
1981	PFOA found in umbilical cord blood when female worker gives birth	[13]
1993	Transfer of PFOS into milk observed in goats	[10]
1998	PFOS found in blood from the general population	[10]
2003	PFAS in blood from Red Cross blood donors	[16]
2004	PFAS detected in human milk	[15]
2014	Breastfeeding shown to be major source of PFAS exposure in infants	[31]
	Immunotoxicity	
1978	Immunotoxicity and other adverse effects in monkeys exposed to PFOA, and mortality in monkeys exposed to PFOS	[5, 6]
1992	Leukocyte cell count changes in PFOA production workers	[8]
2008	Mouse study shows immunotoxicity at serum PFAS concentrations similar to elevated human exposures	[50]
2012	Immunotoxicity reported in PFAS-exposed children	[28]
2013	Benchmark Dose calculations suggest that guidelines are far from protective	[44]
2017	PFAS exposure during infancy associated with subsequent immune deficiency	[32]

Unpublished information is shaded

leukocyte counts. The results were not reported in a scientific journal. However, in connection with a recent law suit, a draft manuscript on this study has been released ("Peripheral blood lymphocyte count in men occupationally exposed to perfluorooctanoic acid" [9]). The draft concluded: "PFOA is associated with alterations in peripheral blood lymphocyte numbers in PFOA production workers, suggesting that cell-mediated immunity may be affected by PFOA". Other company materials outlined in an expert report include the comment "We're working with [the author] regarding some of the wording" [10]. Evidently, an agreement was not reached, and the findings were not published.

Human exposure to organofluorine compounds was discovered as early as 1968 [11] and was later confirmed in a more extensive study [12]. However, the exact identity and the sources were unknown at the time. Soon thereafter, PFASs were identified in blood from production workers, and in 1981 also in umbilical cord blood at a female worker's childbirth [13]. Although the latter finding signified placental passage and prenatal PFAS exposure, this observation was not revealed until 20 years later, after which it was soon confirmed in a larger study [14]. Of additional public health significance, an unpublished study on goats from 1993 showed that PFOS was transferred into milk [10], and this pathway was verified in humans, again many years later [15].

New insight into a hidden hazard

By about 2000, the widespread occurrence and persistence of PFASs in the environment became known [7], as reflected also by the presence of PFASs in serum samples from blood banks [16]. Only after this time, and especially during the most recent 10 years, did the scientific literature on PFASs expand (Fig. 1) [17]. Immune system deficits in PFOA-exposed mice were at first observed in studies of peroxisome proliferator activation [18]. Later, experimental studies of PFOS showed reductions in lymphoid cell numbers and de novo antibody synthesis [19], and a study in mice from 2009 demonstrated that PFOS exposure reduced the survival after influenza A infection [20]. Then followed in vitro evidence of adverse effects in human white blood cells [21]. Although the 1978 monkey study [5] could have been obtained from the U.S. EPA, none of these studies referred to these original findings.

Important evidence emerged after the discovery of PFAS contamination in the Mid-Ohio River Valley and

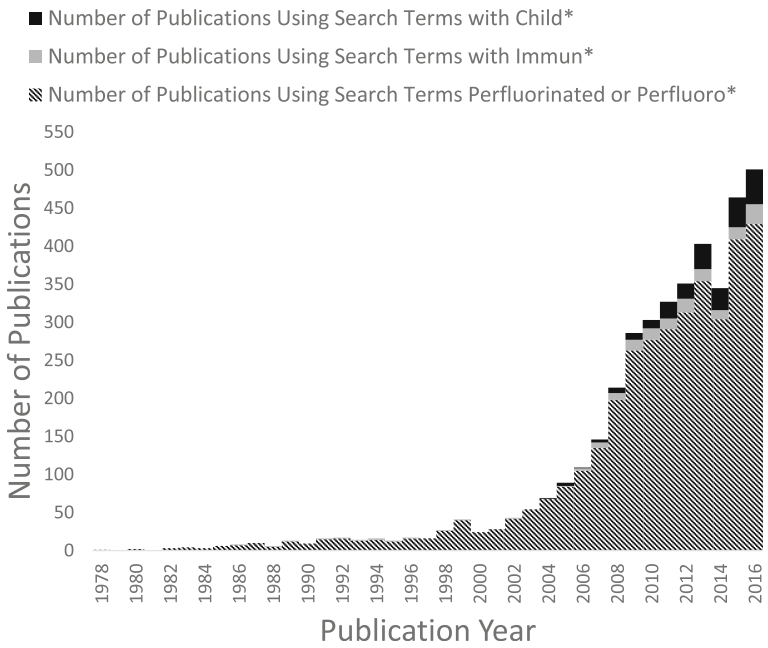

Fig. 1 Number of publications on PFASs over time, according to the Web of Science database (between 1978 and 2017), using the search terms "perfluorinated or perfluoro"* and restricting to environmental sciences, toxicology, or public, environmental, and occupational health categories. This search was further refined using the search terms "immun*" and "child*"

the court-mandated health examinations [22]. In regard to immunotoxicity, an interim report showed that increased PFOA exposure was associated with changes in serum concentrations of immunoglobulins [23]. A more focused study determined antibody responses to flu vaccination [24]. Elevated serum-PFOA concentrations were associated with a reduced antibody titer rise, particularly to an A influenza virus strain, with an increased risk of not attaining the antibody level needed to provide long-term protection. A later study on 12 adult volunteers with background exposures showed that two of the subjects failed to respond to a tetanus-diphtheria booster and that the steepness of the antibody responses was negatively associated with the serum-PFAS concentrations [25]. Cross-sectional data have also suggested lower vaccination antibody concentrations at elevated background PFAS exposures [26].

The first prospective study assessing children's antibody responses to routine childhood immunizations reported in 2012 that a doubling in exposure to PFOS and PFOA was associated with an overall decrease by up to 50% in the specific vaccine antibody concentration [27, 28]. When mutually adjusted, the regression coefficients for PFOA and PFOS changed only little [27]. Booster vaccine responses in children at age 5 years were lower at elevated serum-PFAS concentrations [28, 29]. A smaller Norwegian study of about 50 children aged 3 years also showed tendencies toward lower vaccination antibody concentrations at higher exposures during pregnancy [30]. As PFASs are now known to be transferred to the infant via human milk [31], it seems likely that PFAS exposures in early infancy represent a particular hazard to the adaptive immune system [32]. If true, the routine modeling of lifetime exposures for risk assessment is inappropriate, as it ignores the presence of vulnerable time windows.

PFAS exposure can also impact the body's ability to fight off common infections, such as colds and gastroenteritis, as seen in the Norwegian study [30]. A larger, prospective study in Denmark found that increased maternal serum concentrations of PFOA and PFOS were significantly associated with a higher frequency of fever and symptoms in the children [33], in agreement with a subsequent study from Japan that relied on retrospective assessment of the disease incidence [34]. In contrast, a substudy from the Danish National Birth Cohort examined the hospitalization rates for a variety of infections, such as airway infection, middle ear infection, and appendicitis, through to age 11 years and showed no association with PFOS and PFOA in early pregnancy serum from the mother [35]. However, a recent report from the project team raised doubt about the validity of the PFAS analyses [36].

Delayed interventions

Despite the support from both experimental and epidemiological data [37], most regulatory risk assessments of PFASs have focused on other target organs and have

emphasized toxicity testing in rodents [4]. The first opinion from the European Food Safety Authority (EFSA) in 2009 [38] listed a single report on immunotoxicity under "Other endpoints". That same year, the EPA issued provisional health advisories and concluded that "epidemiological studies of exposure to PFOA and adverse health outcomes in humans are inconclusive at present" [39]. Neither report referred to the 1978 monkey study that had become available in 2000. Early and more recent guidelines and recommended limits for PFOS and PFOA are shown in Table 2.

The EPA prepared more detailed risk assessment reports for PFOA and PFOS in 2014 [40, 41]. These drafts conclude that the two major PFASs exhibit immunotoxicity in experimental models and that the epidemiological evidence is additive, although mixed exposures complicate the attribution of effects to specific PFASs. A similar conclusion was reached by an ATSDR ToxProfile on the perfluoroalkyls in 2015 [42]. The coverage of human immunotoxicity was very brief, and no mention of this potential was made in the sections on public health implications. Although the monkey studies were cited, the risk assessment reports did not refer to the 1992 study of exposure-associated immune cell abnormalities in workers.

More recently, the National Toxicology Program (NTP) in 2016 reviewed the immunotoxicity information on PFOS and PFOA and concluded that both are "presumed" to constitute immune hazards to humans [37]. The term "presumed" is the strongest below "known" in the NTP vernacular. Both PFASs suppress the antibody response in animal studies, while the evidence in humans is "moderate", as all studies are observational (not experimental) and refer to mixed PFAS exposures. The revised ATSDR ToxProfile [43] just released concluded that decreased antibody response to vaccines is a potential outcome from exposure to all five PFASs commonly found in human blood samples. However, ATSDR stopped short of using epidemiology evidence for derivation of exposure limits.

Table 2 Guideline values expressed in terms of acceptable concentrations of PFOS and PFOA in drinking water (ng/L),[a] as compared with the estimated limit based on benchmark dose calculations for immunotoxicity in children [44]

Authority	Year	PFOS	PFOA
Australia	2016	70	560
Canada	2016	600	200
U.S. EPA	2009	200	400
	2016	70	70
ATSDR	2015	70	100
	2018	7	11
Minnesota	2008	300	300
	2017	27	35
New Jersey	2007	-	40
	2017	13	14
EFSA	2009	70	700
	2018	6.5	3
BMDL-based	2013	< 1	< 1

[a]Estimated from total intake limits, assuming 20% exposure contribution from water (rounded values)

Regulatory agencies frequently use benchmark dose calculations as a basis for generating exposure limits [38]. This approach relies on fitting a dose-response function to the data, and the benchmark dose (BMD) is defined as the dose that leads to a specific loss (or degree of abnormality) known as the benchmark response (BMR) in the outcome variable. The lower one-sided 95% confidence limit of the BMD is the benchmark dose level (BMDL), which is used as the point of departure for calculation of exposure limits. Relying on the vaccine antibody responses, BMDLs for PFOS and PFOA were calculated in 2013 to be about 1 μg/L serum [44], i.e., levels that are exceeded by a majority of the general population [45]. However, at first, these results were disregarded because of the absence of an unexposed control group [42], a condition that would be impossible to meet. Another concern was the high correlation between exposure components, such as PFOA and PFOS [40, 41, 43]. Still, mutual adjustment is possible and shows clear negative impacts of both of these major PFASs on immune system responses [27], and other calculations show virtually unchanged BMDLs for PFOA and PFOS after such adjustment [46].

In an updated opinion on PFOS and PFOA [47], EFSA used separate BMD calculations for several outcomes in humans, including immunotoxicity, relying on summary data in deciles or quartiles. For the vaccine response data [28], EFSA assumed that all subjects in the lowest decile exposure group had the same exposure, and the BMDs were similar to the average serum concentration in that group. For this reason, EFSA's calculated BMDs are several fold higher than the ones obtained from the continuous dose-effect relationship [44]. Still, the new tolerable intake limits are substantially lower than other published guidelines (Table 2), though quite similar to the Minimal Risk Levels developed by ATSDR [43].

The "untested chemicals assumption", as highlighted by the National Research Council [1] has clearly been inappropriately relied upon in past risk assessments of PFASs, and these substances must now be added to the list of environmental hazards [48] where standard risk assessment has failed. As a major reason, early evidence on PFAS toxicity was kept secret for 20 years or more, and even after its release, it was apparently overlooked. A related reason is the absence of academic PFAS

research on the immune system and other sensitive target organs until about 10 years ago. Further, regulatory agencies relied on experimental toxicity studies and disregarded emerging epidemiological evidence. As a result, even some of the current guidelines are orders of magnitude above exposure levels at which associations with adverse effects have been reported.

The PFASs therefore constitute an unfortunate example that risk assessment may be inappropriate to assess human health risks from chemical exposures when crucial documentation has not yet been published. Recognizing the weaknesses of conventional risk assessment, scientists from the U.S. EPA recently recommended to consider the full range of available data and to include health endpoints that reflect the range of subtle effects and morbidities in humans [48]. The present summary of delayed discovery, dissemination and decision-making on the PFASs indicates that a more comprehensive assessment of adverse health risks is urgently needed and that PFAS substitutes, as well as other persistent industrial chemicals, should not be considered innocuous in the absence of relevant documentation [49].

Conclusions

Early research on environmental PFAS exposures and their health implications became available at a substantial delay and was not taken into account in initial regulatory decisions on exposure abatement. Only in the last 10 years or so has environmental health research focused on the PFASs and revealed important human health risks, e.g., to the immune system. Although guideline values for PFASs in drinking water have decreased over time, they remain too high to protect against such toxicity. While the most commonly used PFASs will remain in the environment for many years, new PFAS substitutes are being introduced, although little information on adverse health risks is available. Given the serious delays in the discovery of PFAS toxicity, their persistence in the environment, and their public health impact, PFAS substitutes and other persistent industrial chemicals should be subjected to prior research scrutiny before widespread usage.

Abbreviations

BMD: Benchmark dose; BMDL: Benchmark dose level; BMR: Benchmark response; EFSA: European Food Safety Authority; EPA: Environmental Protection Agency; NTP: National Toxicology Program; PFAS: Perfluorinated alkylate substance; PFOA: Perfluorooctanoic acid; PFOS: Perfluorooctanoic sulfonic acid

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Authors' Contributions

The author read and approved the final manuscript.

Competing interests

The author is an editor-in-chief of Environmental Health. The author recently served as a health expert for the State of Minnesota in a lawsuit against a PFAS-producing company.

Publisher's Note

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EXHIBIT B

Lara J. Say

From: Faucette, Joshua <JFaucette@mayerbrown.com>
Sent: Wednesday, November 24, 2021 5:56 PM
To: Stephanie Biehl; Ring, Daniel L.
Cc: Fonseca, Rebecca; Hoyle, David
Subject: RE: AFFF MDL | 3M Custodian Request - Lewis Lehr [MB-AME.FID2338174]

CAUTION:EXTERNAL

Stephanie – After reasonable investigation, 3M has not identified a custodial file, nor documents associated with a custodial file (whether hard copy or electronic), for Lewis Lehr whose employment tenure at 3M ended in 1986, aside from a single nonresponsive lab notebook. As you are aware, some documents referring to Mr. Lehr are in 3M's production, but we have not identified a further collection of documents associated with Mr. Lehr as a document custodian to review for responsive documents.

Regards,

- Josh.

Joshua A. Faucette
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Mayer Brown LLP
71 S. Wacker Drive
Chicago, Illinois 60606
T : 312 701 8780
Email Address: jfaucette@mayerbrown.com
LinkedIn | Twitter
mayerbrown.com

From: Stephanie Biehl <stephanie@sheredling.com>
Sent: Monday, November 22, 2021 1:23 PM
To: Faucette, Joshua <JFaucette@mayerbrown.com>; Ring, Daniel L. <DRing@mayerbrown.com>
Cc: Fonseca, Rebecca <rfonseca@motleyrice.com>; dhoyle@motleyrice.com
Subject: RE: AFFF MDL | 3M Custodian Request - Lewis Lehr [MB-AME.FID2338174]

****EXTERNAL SENDER****

Hi Josh,

Hope you had a nice weekend. I'm just following up on when we can expect to receive the completed Lewis Lehr production(s).

Thanks very much,
Steph

Stephanie Biehl

SHER EDLING LLP
100 Montgomery St., Ste. 1410
San Francisco CA 94104

628.231.2507 | sheredling.com

From: Stephanie Biehl
Sent: Tuesday, October 26, 2021 6:33 AM
To: Faucette, Joshua <JFaucette@mayerbrown.com>; Ring, Daniel L. <DRing@mayerbrown.com>
Cc: Fonseca, Rebecca <rfonseca@motleyrice.com>; dhoyle@motleyrice.com
Subject: RE: AFFF MDL | 3M Custodian Request - Lewis Lehr [MB-AME.FID2338174]

Thank you, Josh. If you have a ballpark for when the documents might be produced, we'd appreciate that once the searches are run.

Thanks again,
Steph

Stephanie Biehl

SHER EDLING LLP
100 Montgomery St., Ste. 1410
San Francisco CA 94104
628.231.2507 | sheredling.com

From: Faucette, Joshua <JFaucette@mayerbrown.com>
Sent: Monday, October 25, 2021 10:40 PM
To: Stephanie Biehl <stephanie@sheredling.com>; Ring, Daniel L. <DRing@mayerbrown.com>
Cc: Fonseca, Rebecca <rfonseca@motleyrice.com>; dhoyle@motleyrice.com
Subject: RE: AFFF MDL | 3M Custodian Request - Lewis Lehr [MB-AME.FID2338174]

Stephanie – 3M agrees to the PEC's request regarding Lewis Lehr, and will conduct a reasonable search in accordance with our agreements regarding document discovery of 3M in this MDL, including but not limited to our understandings with the PEC regarding the multiple rounds of agreed search terms for purposes of document discovery.

Regards,

- Josh.

Joshua A. Faucette
Associate – Litigation & Dispute Resolution
Mayer Brown LLP
71 S. Wacker Drive

Chicago, Illinois 60606
T : 312 701 8780
Email Address: jfaucette@mayerbrown.com
LinkedIn | Twitter
mayerbrown.com

From: Stephanie Biehl <stephanie@sheredling.com>
Sent: Wednesday, October 20, 2021 10:18 PM
To: Ring, Daniel L. <DRing@mayerbrown.com>; Faucette, Joshua <JFaucette@mayerbrown.com>
Cc: Fonseca, Rebecca <rfonseca@motleyrice.com>; dhoyle@motleyrice.com
Subject: AFFF MDL | 3M Custodian Request - Lewis Lehr

****EXTERNAL SENDER****

Hi Dan and Josh,

Hope you both are well. We write briefly to request that 3M add Lewis Lehr as a custodian and produce Mr. Lehr's documents accordingly. Of course, we are happy to discuss further via telephone if needed, but please let us know if 3M will agree to this request by Monday, October 25, 2021.

Thank you,
Steph

Stephanie Biehl

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San Francisco CA 94104
628.231.2507 | sheredling.com

EXHIBIT C

MotleyRice LLC
ATTORNEYS AT LAW
www.motleyrice.com

"I will stand for my client's rights.
I am a trial lawyer."
–Ron Motley (1944–2013)

28 Bridgeside Blvd.
Mt. Pleasant, SC 29464
o. 843.216.9000 **f.** 843.216.9450

T. David Hoyle
Licensed in DC, FL, GA, SC
direct: 843.216.9136
dhoyle@motleyrice.com

December 7, 2021

BY EMAIL: DRing@mayerbrown.com
Daniel L. Ring, Esquire
Mayer Brown LLP
71 South Wacker Drive
Chicago, IL 60606

Re: In re: Aqueous Film-Forming Foam Products Liability Litig., MDL No. 2873 (D.S.C.) – Custodial File of Lewis Lehr

Dear Dan:

The PEC is in receipt of and thanks you for 3M's email dated November 24, 2021, informing the PEC that 3M has not identified a custodial file, nor documents associated with a custodial file, for former CEO and COB, Lewis Lehr. The PEC finds this quite shocking.

Mr. Lehr was CEO from 1979-1980 and Chairman of the Board and CEO from 1980-1986, a pivotal time in the company's history related to fluorochemicals. Based on our review of documents produced by 3M to date, it appears that Mr. Lehr played a central role in 3M's business decisions with respect to investigating and reporting potential effects associated with its fluorochemical products.[1]

Indeed, documents produced in this litigation indicate that Mr. Lehr was actively involved in discussions concerning organic fluorine found in blood and investigation of waste disposal activities at 3M's fluorochemical manufacturing plants. According to minutes from a meeting that occurred on July 12, 1978, Mr. Lehr was one of six members of 3M management who met to discuss investigations of fluorochemicals in blood and the methods for communicating their findings to the industry, the public, and appropriate government agencies. *See* 3M_AFFF_MDL00435681. Among those present at the meeting were other key decision-makers at the time, including individuals responsible for the groups and divisions within which 3M's AFFF business operated and who were tasked with reporting to the Corporate Responsibility Committee regarding 3M's fluorochemicals in blood program. Ultimately, this small group of 3M leaders determined there was "no need" to report their findings to the EPA under Section 8(e) of the Toxic Substances Act and instead decided that publishing 3M's studies in the scientific literature would be "the preferred approach." *Id.*

[1] As acknowledged in your November 24th email, several documents referring to Mr. Lehr have been produced in this litigation. In fact, Mr. Lehr's name appears in 283 documents produced by 3M in this matter to date.

MotleyRice LLC
ATTORNEYS AT LAW

In addition, Mr. Lehr proposed possible solutions for decreasing exposure to fluorochemicals at 3M's manufacturing plants, apparently expressing dissatisfaction with the levels of organic fluorine found in employees' blood and suggesting the use of self-contained suits to decrease employees' exposure to fluorochemicals. *See* 3M_AFFF_MDL00417206. Mr. Lehr was also involved in and regularly updated on developments related to 3M's investigations of industrial waste disposal at its Oakdale, MN site, including public relations communications plans. *See e.g.*, 3M_AFFF_MDL00417249, 3M_AFFF_MDL00417252, 3M_AFFF_MDL00417391.

Furthermore, while it is our understanding that Mr. Lehr's employment with 3M ended in 1986, numerous historical documents have been produced in this litigation from custodial files of individuals who were employed during the same time frame when Mr. Lehr was a 3M employee. For example, approximately 114 documents have been produced from the custodial file of Hugh Bryce who retired from 3M in 1983. The PEC is puzzled by this discrepancy.

In light of Mr. Lehr's evident involvement in business decisions related to 3M's fluorochemical products, the PEC believes documents from Mr. Lehr's custodial file are central to the core issues in this case. Therefore, the PEC requests that 3M once again search its records to confirm the existence of the custodial file for Lewis Lehr by 12 pm EST on Wednesday, December 15, 2021.

As always, the PEC is willing to meet and confer further concerning these issues in order to avoid unnecessary motions practice.

Thank you for your attention to this matter and your courtesy.

With kind regards, I remain,

Sincerely yours,

T. David Hoyle

cc: Michael A. London, Esquire
Paul J. Napoli, Esquire
Scott Summy, Esquire
Fred Thompson, III, Esquire
Stephanie Biehl, Esquire
Rebecca Fonseca, Esquire
Ned McWilliams, Esquire

EXHIBIT D

3M General Offices

3M Center
St. Paul, MN 55144-1000
612 733 1110

8EHQ-0598-373

3M May 15, 1998

Containing No CBI

8EHQ-90-373

BY CERTIFIED MAIL

Document Processing Center (7407)
Attn: Section 8(e) Coordinator
Office Of Toxic Substances
United States Environmental Protection Agency
401 M Street, Southwest
Washington, D. C. 20460

Re: **TSCA Section 8(e) -- Perfluorooctane Sulfonate -- Docket Numbers 8EHQ-1180-373; 8EHQ-1180-374; 8EHQ-0381-0394**

Dear Sir/Madam:

With this letter, 3M Company is submitting information to the EPA Administrator pursuant to Section 8(e) of the Toxic Substances Control Act ("TSCA"). As detailed below, this information relates to fluorochemicals -- specifically, perfluorooctane sulfonate ("PFOS") [CAS No. 2795-39-3] -- and consists of analysis of blood sera samples showing PFOS at very low (*i.e.*, parts per billion ("ppb")) levels. The presence of organic fluorochemicals in the blood of the general population and subpopulations, such as workers, has been known dating back to the 1970's,[1] and 3M's epidemiological study of its own workers indicates no adverse effects at parts per million levels. 3M does not believe that any reasonable basis exists to conclude that PFOS "presents a substantial risk of injury to health or the environment." Nevertheless, as a precautionary measure, 3M is submitting this information to the TSCA Section 8(e) docket at this time.

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1 See, e.g., Taves, D.R.; *Comparison of "Organic" Fluoride in Human and Nonhuman Serums*, 50 J. Dent. Res. 783 (1971); Guy, W.S., et al.; *Biochemistry Involving Carbon Fluorine Bonds*, American Chemical Society, 117-34 (1976); Ubel, F.A.,et al.; *Health Status Of Plant Workers Exposed to Fluorochemicals -- A Preliminary Report*, 4 American Indus. Hygiene J. 584 (Aug. 1980). 3M has submitted PFOS-related medical surveillance and epidemiological information on its own production workers as well as animal toxicology data previously to the TSCA Section 8(e) docket. See Docket Numbers 8EHQ-1180-373; 8EHQ-1180-374; 8EHQ-0381-0394

In the process of validating analytical methodology for measuring PFOS, a product of the electrochemical fluorination process, an outside laboratory detected PFOS at ppb levels in blood samples from individuals with no known occupational exposure to fluorochemicals. Subsequent analyses of commercially pooled sera from human blood bank samples in different regions of the United States measured PFOS levels between 9 ppb and 56 ppb.[2] Analyses of limited historical blood samples from 1969 and 1976 showed mean PFOS levels of 28 ppb and 33 ppb, respectively. Analyses of limited animal sera samples found comparable PFOS levels. 3M also has conducted qualitative *in vitro* and *in vivo* metabolism studies, which suggest the possibility that non-occupational presence of PFOS could result from the metabolic conversion of other fluorochemicals to PFOS.

3M would welcome the opportunity to discuss our findings and our plans. We are sending a copy of this letter to Charles Auer, Director of the Chemical Control Division, and will be contacting him shortly to arrange a meeting for this purpose. In the meantime, please do not hesitate to contact William Weppner at (612) 733-6374 with any questions

Sincerely,

Dr. Charles Reich
Group Vice President
Chemical Markets Group

---

2 3M also analyzed these sera samples for another fluorochemical -- perfluorooctanoate ("PFOA") [CAS No. 3825-26-1] -- but detected the presence of PFOA at quantifiable levels of 12 and 22 ppb in only two of the samples.

EXHIBIT E

CD No. 1

3M General Offices

3M Center
St. Paul, MN 55144-1000
612 733 1110

3M

May 4, 2000

VIA FEDERAL EXPRESS

Dr. Charles Auer
Director
Chemical Control Division
Office Of Pollution Prevention And Toxics
United States Environmental Protection Agency
401 M Street, Southwest
Room 403 East Tower (Mail Code 7405)
Washington, D. C. 20460

Re: **Information On Perfluorooctane Sulfonates**

Dear Charlie:

Pursuant to our recent communications, 3M is enclosing additional information on perfluorooctane sulfonates. The enclosed information supplements information submitted to you previously under cover of our April 21, 2000 letter. Again, we are providing this information on a voluntary basis as part of our continuing discussions with EPA regarding fluorochemistry.

The enclosed information covers perfluorooctane sulfonates, including CAS numbers 1763-23-1 (acid); 29081-56-9 (ammonium salt); 70225-14-8 (DEA salt); 2795-39-3 (potassium-salt); 29457-72-5 (lithium salt). It consists of the following:

⇒ Copies of post-1975 studies and certain other information relating to the following environmental science areas: (i) physical and chemical properties; (ii) environmental fate and transport; (iii) environmental monitoring; and (iv) ecotoxicity. For each study, 3M has prepared a summary in the HPV "robust summary" format. An executive summary also has been included for each area.

⇒ Copies of post-1975 studies and certain other information relating to the following health effects areas: (i) acute toxicity; (ii) genotoxicity; (iii) repeated-dose toxicity;

(iv) pharmacokinetics; (v) teratology; and (vi) medical surveillance and epidemiology. 3M has included a detailed index of this information.

⇒ A list of all studies in progress and planned studies, along with study protocols or study plans, where available. With regard to the health effects area, this list supplements the list provided under cover of our April 21, 2000 letter to you.

⇒ A bibliography of pre-1976 studies in the environmental science and health effects areas on perfluorooctane sulfonates.

⇒ A bibliography of acute toxicity studies on perfluorooctane sulfonates, except that we are providing copies of key acute studies (with reference to the HPV guidance).

⇒ A bibliography of published studies on the perfluorooctane sulfonates in 3M's possession.

⇒ An index of submissions made by 3M to the TSCA Section 8(e) docket. This index has been subdivided by EPA docket number. Rather than attempt to segment the index for perfluorooctane sulfonates only, we have included other fluorochemical submissions on the index, as several of 3M's submissions have dealt with multiple fluorochemicals.

3M is continuing our file review and will supplement the enclosed information as appropriate. As you review this information, we ask that you bear several points in mind:

⇒ The enclosed information spans several boxes. We have organized the information in each box with labeled file folders and indices to aid EPA's review. To ensure that you and your staff are able to access the most pertinent information, we also are attaching to this cover letter the executive summaries for the environmental science areas and the indices covering studies and other information.

⇒ In some cases, the enclosed information reflects recent developments that may supplement studies and other information previously provided to you. As just one example, 3M's previously submitted document entitled "Fluorochemical Use, Distribution And Release Overview" (5/26/99) contains a qualitative assessment based on the assumption that all other fluorochemicals could breakdown to perfluorooctane sulfonates. Another document submitted by 3M entitled "Sulfonated Perfluorochemicals in the Environment: Sources, Dispersion, Fate and Effects" also provided estimates of potential exposure and waste generation based on such an assumption. Recent information in the environmental fate and transport area suggests, however, that this assumption may reflect an unrealistic

"worst case" which significantly overstates exposure potential to perfluorooctane sulfonates. In particular, this information (which is enclosed) indicates that perfluorooctane sulfonates may not be a degradation product of many fluorochemicals and that high molecular weight fluorochemical polymers and fluorochemical phosphate esters are relatively stable in the environment.

⇒ The enclosed information includes some studies and other information on mixtures containing perfluorooctane sulfonates. As we have discussed, 3M will be providing you with further information on other fluorochemicals within the next several weeks. We will include additional studies and other information on mixtures containing perfluorooctane sulfonates at that time.

⇒ 3M has not provided you with all analytical chemistry reports on perfluorooctane sulfonates. Rather, we have enclosed certain analytical chemistry reports which may prove useful to EPA in interpreting certain studies; understanding the details of analytical chemistry methods; or verifying human and biomonitoring data.

⇒ 3M is continuing its work to refine the analytical characterization of the perfluorooctane sulfonates test material being used for our current study program. We will keep you informed of any pertinent developments.

⇒ Finally, please note that some of this information qualifies as confidential business information (CBI); CBI information has been placed in a separate, labeled envelope. Also, incorrectly applied legends relating to legal privileges and proprietary protections have been removed from certain documents.

3M looks forward to discussing the enclosed information with you and other EPA staff. In the meantime, please do not hesitate to contact me with any questions.

Very truly yours,

William A. Weppner

William A. Weppner, Ph.D
Director of Environmental, Health, Safety
And Regulatory Affairs
Specialty Materials Markets
3M
Bldg 236-1B-10
3M Center
St. Paul, MN 55144
E-mail: waweppner@mmm.com

Enclosures

Attachment to Letter to C. Auer Dated May 4, 2000: Environmental Studies on Perfluorooctanesulfonates (Post-1975)

Physical/chemical Properties

Title	Laboratory or Author	Completion Date	Type
Determination of the Melting Point/Melting Range of PFOS; Boiling Point (Not Conducted)	Wildlife International, Ltd.	2/24/99	Robust Summaries, Final Report, Protocol
Determination of the Vapor Pressure of PFOS Using the Spinning Rotor Guage Method	Wildlife International, Ltd.	5/5/99	Robust Summary, Final Report, Protocol
PFOS: Determination of the *n*-Octanol/Water Partition Coefficient by the Shake Flask Method - A Non-GLP Feasibility Study in Support of Wildlife International, Ltd. Project Number: 454C-108	Wildlife International, Ltd.	2/11/00	Robust Summary, Feasibility Study
Testing Results: Air-Water Partition Coefficient (K_{AW}) for PFOS	3M/Wildlife International, Ltd., U of Trent	3/19/99	Robust Summary, Letter Report
Determination of the Water Solubility of PFOS by the Shake Flask Method	Wildlife International, Ltd.	4/26/00	Robust Summary, Final Report, Protocol
Technical Report. Solubility Measurements on FC-95	3M Env. Lab	2/6/81	Brief Robust Summary, critique from Endwin Tucker (3/1/93), Final Report
Solubility Estimate of FC-95 by use of Xertex TOC Analyzer	Xertex, 3M Env. Lab	6/29/82	Brief robust summary, letter report

Environmental Fate and Transport

Title	Laboratory or Author	Completion Date	Type
Adsorption of FC 95 and FC 143 on Soil (Note: the 3M Env. Lab summary is titled: Summary of the Soil Adsorption study of the Potassium Salt of Perfluorooctanesulfonic acid, 7/22/98)	3M Env. Lab	2/27/78	Robust Summary, 3M Env. Lab Summary, Comments from Stephen A. Boyd from MSU, Final Report
FC-95/Photolysis Study Using Simulated Sunlight. (Note: the 3M Env. Lab summary is titled: summary of Photolysis Study Using Simulated Sunlight on the Potassium Salt of Perfluorooctanesulfonic acid)	3M Env. Lab	1/9/79	Robust Summary, 3M Env. Lab Summary, Final Report
Biodegradation Studies of Fluorocarbons (8/12/76) report and Biodegradation Studies of Fluorocarbons - III (7/19/78) report. (Note: both reports summarized with one robust summary)	3M Env. Lab	8/12/1976, 7/19/78	Brief Robust Summary, 2 Final Reports
BOD/COD results for FC-94-X (Li salt of PFOS)	Pace Analytical	3/30/94	Computer-generated Summary of Testing Results
BOD/COD results for FC-99 (DEA salt of PFOS)	3M Env. Lab	6/8/79	Robust Summary and final Reports
Transport between environmental compartments (fugacity modeling) included in letter from Don Mackay on the air/water partitioning coefficient calculations	DMER	No date	Robust Summary, letter report
Analysis for fluorochemicals in Bluegill fish.	3M Env. Lab	5/17/79	Robust Summary, Technical Report

Ecotoxicity Elements

Title	Laboratory or Author	Completion Date	Type
PFOS: A 96-Hour Static Acute Toxicity Test with the Fathead Minnow (*Pimephales promelas*)	Wildlife International, Ltd.	4/26/00	Robust Summary, Final Report, Protocol
PFOS: A 96-Hour Toxicity Test with the Freshwater Alga (*Selenastrum capricornutum*)	Wildlife International, Ltd.	4/26/00	Robust Summary, Final Report, Protocol
PFOS: A 48-Hour Static Acute Toxicity Test with the Cladoceran (*Daphnia magna*)	Wildlife International, Ltd.	4/26/00	Robust Summary, Final Report, Protocol
PFOS: A 96-Hour Shell Deposition Test with the Eastern Oyster (*Crassostrea virginica*)	Wildlife International, Ltd.	4/26/00	Robust Summary, Final Report, Protocol
PFOS: A 96-Hour Static Acute Toxicity Test with the Freshwater Mussel (*Unio complamatus*)	Wildlife International, Ltd.	4/26/00	Robust Summary, Final Report, Protocol
PFOS: An Activated Sludge, Respiration Inhibition Test	Wildlife International, Ltd.	4/28/00	Robust Summary, Final Report, Protocol
PFOS: A 96-Hour Static Acute Toxicity Test with the Saltwater Mysid (*Mysidopsis bahia*)	Wildlife International, Ltd.	4/26/00	Robust Summary, Final Report, Protocol
PFOS: An Early Life-Stage Toxicity Test with the Fathead Minnow (*Pimephales promelas*)	Wildlife International, Ltd.	4/26/00	Robust Summary, Final Report, Protocol
PFOS: A Semi-Static Life-Cycle Toxicity Test with the Cladoceran ((*Daphnia magna*)	Wildlife International, Ltd.	4/26/00	Robust Summary, Final Report, Protocol
PFOS: A Flow-through Life-Cycle Toxicity Test with the Saltwater Mysid (*Mysidopsis bahia*)	Wildlife International, Ltd.	4/26/00	Robust Summary, Final Report, Protocol
PFOS: A Dietary LC50 Study with the Mallard	Wildlife International, Ltd.	4/26/00	Robust Summary, Final Report, Protocol
PFOS: A Dietary LC50 Study with the Northern Bobwhite	Wildlife International, Ltd.	4/26/00	Robust Summary, Final Report, Protocol
Multi-Phase Exposure/Recovery Algal Assay Test Method	3M Env. Lab	12/16/81	Brief Robust Summary, Final Report
The Effects of Continuous Aqueous Exposure to 14C-78.02 on Hatchability of Eggs and Growth and Survival of Fry of Fathead Minnow (*Pimephales promelas*) and Summary of histopathological Examinations of Fathead Minnow (Pimephales promelas) Exposed to 78.02 for 30 Days	EG&G Bionomics	August, 1978 December, 1978	Brief Robust Summary, Final Reports
Effect of Potassium Perfluorooctanesulfonate on Survival, etc. (Daphnid reproduction)	3M Env. Lab	2/13/84	Brief Robust Summary, Final Report
Pimephales promelas 96-hour Toxicity Test Data Summary. Sample FC-94-X (Li salt of PFOS)	3M Env. Lab	3/25/94	Robust Summary, Summary Page, copies of data
48-HR Acute Toxicity to Daphnia, *Daphnia magna*. FC-94-X (Li salt of PFOS)	3M Env. Lab	2/10/94	Robust Summary, copies of data
Microbics Microtox Toxicity Test. Sample: FC-94-X (Li salt of PFOS)	3M Env. Lab	2/7/94	Robust Summary, summary of results, copies of data.
Evaluation of FC-94-X by OECD Activated Sludge Respiration Inhibition Test #209, Review of OECD 209 and BOD/COD Test Results for FC-94-X, test data sheets (Li salt of PFOS)	3M Env. Lab	7/22/98	Robust Summary, Summary Page, copies of data
96-Hour Acute Toxicity Test on Bluegill Sunfish (FC-99, DEA salt of PFOS)	Analytical BioChemistry Laboratories, Inc.	7/13/79	Robust Summary and Final Report
Acute Toxicity to Activated Sludge (FC-99, DEA salt of PFOS)	3M Env. Lab	6/4/79	Robust Summary and copies of data
Microtox data for FM-3820 (28% PFOS)	3M Env. Lab	7/24/91	Robust Summary and data summary

Acute Toxicity to Daphnia magna for FM-3820 (28% PFOS)	EnviroSystems of Resource Analysts, Inc.)	3/26/91	Robust Summary and Final Report
Toxicity to Algae (Selenastrum capricornutum) for FM-3820 (28% PFOS)	EnviroSystems of Resource Analysts, Inc.)	June, 1991	Robust Summary and Final Report

Summary Reports

Title	Laboratory or Author	Completion Date	Type
Final Comprehensive Report: FC 95	3M Env. Lab	3/15/79	Robust Summary, comments from Stephen A. Boyd from MSU and Final Report

Environmental Monitoring

Part 1: Multi-City Study

Title	Laboratory or Author	Completion Date	Type
Design and Structure of Multi-City Study	Battelle Memorial Institute	5/1/00	Report
Low Level Drinking Water Analytical Method	3M Env. Lab	4/28/00	Method
Fluorochemical Characterization of Drinking Water Samples. Columbus, GA (W2336)	Centre Analytical Laboratories, Inc.	2/29/00	Final Report
Fluorochemical Characterization of Drinking Water Samples. Pensacola, FL (W2176)	Centre Analytical Laboratories, Inc.	2/28/00	Final Report
Fluorochemical Characterization of Drinking Water Samples. Port St. Lucie, FL (W2363)	Centre Analytical Laboratories, Inc.	2/28/00	Final Report
Fluorochemical Characterization of Drinking Water Samples. Decatur, Alabama (W1979)	Centre Analytical Laboratories, Inc.	2/28/00	Final Report
Fluorochemical Characterization of Drinking Water Samples. Mobile, Alabama (W2151)	Centre Analytical Laboratories, Inc.	2/28/00	Final Report
Fluorochemical Characterization of Drinking Water Samples. Cleveland, Tennessee (W1973)	Centre Analytical Laboratories, Inc.	2/28/00	Final Report
Draft Drinking Water Health Advisory (DWHA) - PFOS	3M Corporate Toxicology	7/7/99	Report
Battelle Field Sampling Procedures Review. Columbus Georgia City Survey regarding Empirical Human Exposure Assessment. Multi-City Study	3M Env. Lab	8/3/99	Final Report
Multi-City Study. Field Report for Cleveland Tennessee and Decatur Alabama - Battelle Duxbury Activities	Battelle Memorial Institute	7/9/99	Final Report
Multi-City Study. Field Report for Columbus Georgia and Port St. Lucie Florida - Battelle Duxbury Activities	Battelle Memorial Institute	10/26/99	Final Report
Final Multi-City Study. Field Report for Mobile Alabama and Pensacola Florida - Battelle Duxbury Activities	Battelle Memorial Institute	9/29/99	Final Report
Quality assurance Project Plan for Empirical Human Exposure Assessment. Multi-City Study Sampling Task	Battelle Memorial Institute	5/14/99	QAP
Amendment 1 to the Quality Assurance Project Plan and Associated SOP's	Battelle Memorial Institute	6/16/99	Ammendment

Part 2: Biosphere Studies

Title	Laboratory or Author	Completion Date	Type
LCMSMS Analysis of Extracts Reported in: "Preliminary Report Analysis of Perfluorinated Compounds in Environmental Samples"	Michigan State University (P. Jones and K. Kannan)	4/7/99	Report
Analysis of Fluorochemicals in Wild Bird Livers	3M Env. Lab	4/28/99	Final Report
Screening of PFOS levels in Eagle and Albatross	3M Env. Lab	5/8/98	Report

Attachments to Letter to C. Auer dated May 4, 2000
Perfluoroctane Sulfonate Studies

Acute Toxicity

1) An Acute Inhalation Toxicity Study of T-2306 CoC in the Rat, Bio/dynamics, Inc., Project No. 78-7185, December 31, 1979. (FC-95, Perfluorooctane Sulfonate potassium salt)

2) Acute Oral Toxicity (LD50) Study in Rats with Fluorad® Fluorochemical Surfactant FC-95, International Research and Development Corporation, Project No. 137-083, May 31, 1978. (Note: 3M files indicate samples taken during the study were not analyzed.)

3) Eye and Skin Irritation Studies Report on Sample T-1166 (FC-98, Potassium Perfluoroethylcyclohexyl Sulfonate, presumed 100 %), Warf Institute Inc., Project No. 5011023, January 28, 1975.

   a) Combined Eye and Skin Irritation Studies Report

   b) Eye Irritation Study (with washout procedure) Report

Attachments to Letter to C. Auer dated May 4, 2000
Perfluoroctane Sulfonate Studies

Genotoxicity

1) Mutagenicity Evaluation of T-2014 CoC in the Ames Salmonella/Microsome Plate Test Final Report, Litton Bionetics Project No. 20838, Protocol No. DMT-100, February 20, 1978.

2) Memorandum Report from S. R. Rohfing to A. N. Welter, dated March 31 1977, on Results of the Ames Spot Test for Mutagenicity screening of various FCs, including Sample 12-583 which is FC-95, Notebook Reference 45867-24, 25.

3) Mutagenicity Test on T-6295 in an *in vivo* Mouse Micronucleus Assay, Final Report, Corning Hazleton , Inc. (CHV), CHI Study No. 17403-0-455, May 23, 1996, and protocol and amended protocol.

4) Final Report, Chromosomal Aberrations in Human Whole Blood Lymphocytes with PFOS, Covance Laboratories, Inc., Covance Study No. 20784-0-449, 3M Reference No. T-6295.18, October 25, 1999.

5) Final Report, Unscheduled DNA Synthesis in Rat Liver Primary Cell Cultures with PFOS, Covance Laboratories, Inc., Covance Study No. 20784-0-447, 3M Reference No. T-6295.19, November 9, 1999, and protocol.

6) Final Report, *Salmonella-Escherichia coli*/Mammalian-Microsome Reverse Mutation Assay with PFOS, Covance Laboratories, Inc., Covance Study No. 20784-0-409, 3M Reference No. T-6295.17, November 5, 1999, and protocol.

7) Final Report, *In Vitro* Microbiological Mutagenicity Assays of 3M Company Compounds T-2247 CoC and T-2248 CoC, SRI International, SRI Project No. LSC-4442-016, 3M Refernece No. T-2247.1 (FC-99 Old Formula, L-4299 which is 50 % of the diethanolamine salt of perfluorooctanesulfonate in water), September 5, 1978.

8) Prof. Nicola Loprieno, "Evaluation of Mutagenicity Studies Developed on (PFOS) Perfluorooctane Sulfonate," prepared at the request of John L. Butenhoff, Ph.D., 3M Corporate Toxicology, January, 2000.

9) Final Report – Bacterial Reverse Mutation Assay of τ-1, Hita Research Laboratories, Chemical Biotesting Center, Study Code K01-1802, 3M Reference No. T-6667.1 (FC-98, Potassium Perfluoroethylcyclohexyl Sulfonate), September, 1996.

Attachments to Letter to C. Auer dated May 4, 2000
Perfluoroctane Sulfonate Studies

Repeated-Dose Toxicity

1) Ninety-Day Subacute Rhesus Monkey Toxicity Study, with Fluorad® Fluorochemical Surfactant FC-95, International Research and Development Corporation, Project No. 137-092, December 18, 1978.

   a) Study Report

   b) Aborted Study: Ninety-Day Subacute Rhesus Monkey Toxicity Study, with Fluorad® Fluorochemical Surfactant FC-95, International Research and Development Corporation, Project No. 137-087, January 2, 1979.

2) Ninety-Day Subacute Rat Toxicity Study, with Fluorad® Fluorochemical Surfactant FC-95, International Research and Development Corporation, Project No. 137-085, November, 1978.

3) 104-week Dietary Chronic Study and Carcinogenicity Study with Perfluorooctane Sulfonic Acid Potassium Salt (PFOS: T-6295) in Rats, Covance Laboratories Inc., Study Number 6329-183. In progress.

   a) Summary Report - Week 53 undated

   b) "Liver Slide Review," Marvin Case to John Butenhoff and Andrew Seacat dated April 5, 2000 relaying the results of an independent histopathologic review of liver slides from the study.

   c) Second Draft Cell Proliferation Report, Pathology Associates International, August 24, 1999. [final interim report, to be incorporated in final report]

   d) Study Report of Determination of Cyanide Insensitive Palmitoyl-CoA oxidation in samples from 3M Environmental Laboratory - Covance Studies 6329-183 and 6329-212, Centre For Xenobiotic Research, University of Dundee, Biomedical Research Center, Study Number XR0108, February 18, 1999.

4) Range-finder: 4-Week Capsule Toxicity Study with Perfluorooctane Sulfonic Acid Potassium Salt (PFOS; T-6295) in Cynomolgus Monkeys, Covance Laboratories Inc., Study Number 6329-222

   a) Unaudited Draft Final Report, 4-Week Capsule Toxicity Study with Perfluorooctane Sulfonic Acid Potassium Salt (PFOS; T-6295) in Cynomolgus Monkeys, Covance Laboratories Inc., Study Number 6329-222 (draft not complete).

b) Cell Proliferation Report, 4-Week Capsule Toxicity Study with Perfluorooctane Sulfonic Acid Potassium Salt (PFOS; T-6295) in Cynomolgus Monkeys, Covance Laboratories Inc., Study Number 6329-222 (draft to be incorporated in final report)

c) Protocol – Analytical Study, Quantitative Analysis of Perfluorooctane Sulfonic Acid Potassium Salt (PFOS; T-6295) in Cynomolgus Monkeys Following Administration of a 4-Week Capsule Toxicity Study, 3M Environmental Laboratory, AMDT-041598.1

d) Memorandum from Marvin Case, regarding histopathology review of liver tissue in Covance Study 6329-222, July 27, 1998

5) 26-Week Capsule Toxicity Study with Perfluorooctane Sulfonic Acid Potassium Salt (PFOS: T-6295) in Cynomolgus Monkeys, Covance Laboratories Inc., Study Number 6329-223. In progress.

a) Undated report covering the 26-week dosing phase and one year of recovery.

b) John Butenhoff, "Dose-Setting Rationale for Six-Month Chronic Oral Study in Cynomolgus Monkeys," dated July 29, 1998 and followup Aug. 3, 1998.

c) Fecal Urobilinogen Analysis, Mayo Clinic, Porphyrins and Nutritional Chemistry Group, Test Code: 8308.

i) Summary Report from Dr. Joseph P. McConnell, dated March 16, 1999.

ii) General information from Mayo Clinic, Porphyrins and Nutritional Chemistry Group on Urobilinogen Analysis, dated January 14, 1999.

iii) Individual animal urobilinogen lab reports (raw data) from Mayo Clinic, Porphyrins and Nutritional Chemistry Group.

d) Pathology Report (Ancillary Study), Electron Microscopic Evaluation of Liver in Cynomolgus Monkeys, Pathology Associates International, Study No. EM99.76, July 13, 1999.

e) Pathology Review, Marv Case to Andrew Seacat, dated July 22, 1999 relaying the results of a histopathology review of slides.

Attachments to Letter to C. Auer dated May 4, 2000
Perfluoroctane Sulfonate Studies

f) Laboratory Report, Interim Report of Preliminary Data for 26 Week Capsule Toxicology Study with PFOS in Cynomolgus Monkeys, 3M Environmental Laboratory, Report No. FACT-TOX-030, dated March 29, 1999.

6) Two Week Oral Rangefinding Toxicity Study of T-2509CoC in Rats, Safety Evaluation Laboratory, Riker Laboratories, Inc., Experiment No. 179RR023, 3M Reference no. T-2509.3 (FC-99 New Formula, L-4509, 25 % diethanolamine salt of perfluorooctanesulfonate in water), February 25, 1980.

7) [Submitted under claim of Confidentiality]

Attachments to Letter to C. Auer dated May 4, 2000
Perfluoroctane Sulfonate Studies

Pharmacokinetic Studies

1) Skin Absorption Studies on Surfactants (1983)

   a) Report from W. C. McCormick to D. R. Ricker, dated September 26, 1983 summarizing data

   b) 28 Day Percutaneous Absorption Study in Rabbits with FC-95, Safety Evaluation Laboratory, Riker Laboratories, Inc., Experiment No. 0979AB0632 (FC-95)

   c) 28 Day Percutaneous Absorption Study in Rabbits with FC-99, Safety Evaluation Laboratory, Riker Laboratories, Inc., Experiment No. 0979AB0633, 3M Reference No. T-3988.1 (FC-99, diethanolamine salt of perfluorooctanesulfonate, assumed to be 25 % in water)

2) Single-Dose Intravenous Pharmacokinetic Study of T-6053 in Rabbits, 3M Environmental Laboratory (FC-99, diethanolamine salt of perfluorooctanesulfonate in water Lot 130, Unit 177. 0.04 % FC solids in water), November 16, 1995. Final Report - Analytical Study, which includes copy of *in vivo* Study No. AMDT-010495.1, Hazleton Wisconsin, Inc., Project No. HWI 6329-136, 3M Reference No. T-6053.1

3) Single-Dose Dermal Absorption / Toxicity Study of T-6053 in Rabbits, 3M Environmental Laboratory, Study No. AMDT-022195.1 (FC-99, diethanolamine salt of perfluorooctanesulfonate in water Lot 130, Unit 177. 0.04 % FC solids in water), November 22, 1995. Final Report - Analytical Study, includes *in vivo* Study Hazleton Wisconsin, Inc., Project No. HWI 6329-137, 3M Reference No. T-6053.2

4) Fluorochemical (FC) Levels in Naïve Rats, 3M Medical Department, Toxciology Services, Study No. T-6316.9, DT21, Draft Report for Objective 3, May 14, 1999.

5) Analytical Data submitted to Dr. Jennifer Seed, USEPA, by letter dated May 3, 2000, including serum measurements from two in-life studies:

   a) Analytical data from Advanced Bioanalytical Services Study No. FACT-TOX-111, with respect to Oral (Gavage) Pharmacokinetic Recovery Study of Perfluorooctane Sulfonate in Rats, Argus Laboratories Protocol No. 418-015, 3M Reference T-6295.14.

b) Analytical data from Advanced Bioanalytical Services Study No. FACT-TOX-110., with respect to Oral (Gavage) Pharmacokinetic Study of Perfluorooctane Sulfonate in Rats, Argus Laboratories Protocol No. 418-013, 3M Reference T-6295.12.

6) In Vitro Comparative Metabolism Study in Rat and Human Hepatocytes with Various Fluorochemicals, 3M Reference T-6295.1, study of T-6292 (N-ethyl FOSE), T-6293 (N-ethyl FOSE monophosphate ester), T-6294 (N-ethyl perfluorooctane sulfonamide), and T-6295 (Perfluorooctane Sulfonate)

a) Range-finding Cytotocity Assay, SRI International Toxicology Laboratory, Study No. B010-95 - protocol and faxed results dated Oct. 26, 1995, Dec.12, 1995, and Jan. 16, 1996

b) Metabolism of T-6292, T-6293 , T-6294, T-6295 by Rat and Human Hepatocytes, SRI International Toxicology Laboratory, Study No. B011-95

c) Advanced Bioanalytical Services, Inc., Analytical Report, Additional Characterization of Metabolites of T-6292, T-6293 and T-6294 from Rat and Human Hepatocytes by TurboIonSpray LC/MS and LC/MS/MS. Semi-Quantitative Analysis of T-6295 in Rat and Human Hepatocytes Incubated with T-6292, T-6293 and T-6294 by LC/MS/MS, January 28, 1998, Report 98AGKP01.3M

d) Working Interpretation of Results, chart entitled Perfluorosulfonamide Metabolism in Rat vs. Human Hepatocytes, updated Feb. 5, 1998 based on ABS Jan. 1998 report

Attachments to Letter to C. Auer dated May 4, 2000
Perfluoroctane Sulfonate Studies

Mechanistic

1) Reports from University of Minnesota Duluth Research (Kendall Wallace):

   a) Kendall B. Wallace, Biochemical and Molecular Mechanistic Studies of N-Alkyl Perfluorosulfonamides, Research Proposal, April 8, 1997, and Updated Proposal May 7, 1998

   b) Kendall B. Wallace and Anatoli Starkov, The Effect of Perfluorinated Arylalkylsulfonamides on Bioenergetic s of Rat Liver Mitochondria, Feb. 4, 1998

   c) Report on Covance Studies, assessment of mitochondrial bioenergetics, undated

   d) Summary of the Effects of PFC's [Perfluorinated Compounds] on Mitochondrial Bioenergetics In Vitro, undated

   e) Report, Effects of Selected Perfluoro-compounds on Mitochondrial Beta-Oxidation, Dec. 20, 1999

   f) Report, Effect of Acute FC Administration on Catalase and acylCoA Oxidase Expression, January 27, 2000

2) Nabbefeld, et al., Displacement of a Fluorescently Labeled Fatty Acid Analogue fromFatty Acid Carrier Proteins by Wyeth – 14,643, Ammonium Perfluorooctanotate, Potassium Perfluorooctane Sulfonate and Other Known Peroxisome Proliferators, Abstract, Society of Toxicology, 1998 Annual Meeting

Attachments to Letter to C. Auer dated May 4, 2000
Perfluoroctane Sulfonate Studies

Teratology

Pilot Teratology Study in Rats, T-3551 Final Report, May 13, 1983, 3M Reference T-3551.12

Attachments to Letter to C. Auer dated May 4, 2000
Perfluoroctane Sulfonate Studies

Human Sera/Medical Surveillance/Epidemiology

Memorandum from D.E. Roach and S.D. Sorenson, 1983 Decatur Blood Fluoride Review, January 20, 1984

Antwerp Blood Testing Results from June 1995, by Jeffrey H. Mandel, M.D., M.P.H., and Jean Burris, R.N., O.H.N., M.P.H., November 6, 1995

Analysis of Serum Values in Decatur Workers, prepared by Michel Burlew for Larry Zobel, M.D. and Jeffrey Mandel, M.D., April 2, 1998

Laboratory Report, Analysis of FCs in Samples of Children's Sera, Laboratory Report No. FACT-GEN-011, 3M Environmental Laboratory, May 21, 1999

Laboratory Composite Report, Analytical Reports of Data for Fluorochemical Analysis in Human Sera, LIMS No. 1623, 3M Environmental Laboratory, April 28, 2000

ATTACHMENT TO LETTER TO C. AUER DATED MAY 4, 2000: ONGOING ENVIRONMENTAL STUDIES ON PERFLUOROOCTANESULFONATES

Physical/Chemical Properties

Potential Fluorochemical Combustion By-Products (involves review of results of literature search regarding potential for formation of florindated dioxins and furans), 3M Environmental Laboratory. Expected completion: Sept. 2000. Study paper in progress.

Fluorochemical Decomposition Process: Quantification and Assessment (involves computational chemistry calculations of bond-breaking strengths of sulfonated perfluorochemicals), Battelle Memorial Institute. Expected completion: Aug. 2000. Study paper in progress.

Environmental Fate and Transport

Abiotic Degradation Studies (hydrolysis and indirect photolysis), 3M Environmental Laboratory. Expected completion: June 2000 (hydrolysis); Aug. 2000 (indirect photolysis). (Summary study plan and screening results summary being provided to EPA)

Biodegradation Studies (aerobic acclimated closed bottle biodegradation, aerobic soil/sediment biodegradation, pure culture aerobic, and fluorochemical decomposition process, stability in water, photodegradation), Springborn Laboratories, Inc. Expected completion: Aug. 2000. (Summary study plan being provided to EPA)

Ecotoxicity Elements

PFOS: A 96-Hour Toxicity Test with the Freshwater Alga (*Anabaena flos-aquae*), Wildlife International, Ltd. Expected completion: July 2000. (Protocol being provided to EPA)

PFOS: A 96-Hour Toxicity Test with the Freshwater Diatom (*Navicula pelliculosa*), Wildlife International, Ltd. Expected completion: July 2000. (Protocol being provided to EPA)

PFOS: A 96-Hour Toxicity Test with the Marine Diatom (*Skeletonema costatum*), Wildlife International, Ltd. Expected completion: July 2000. (Protocol being provided to EPA)

PFOS: A 7-Day Toxicity Test with Duckweed (*Lemna gibba*), Wildlife International, Ltd. Expected completion: July 2000. (Protocol being provided to EPA)

Phytotoxicity – Seedling Emergence, Wildlife International, Ltd. Expected completion: July 2000. Protocol in progress.

Environmental Monitoring

Global Environmental Sampling Plan, Michigan State University. Expected completion: Dec. 2000. (Summary being provided to EPA)

Attachments to Letter to C. Auer dated May 4, 2000
Perfluoroctane Sulfonate Studies

Ongoing Research/Study Protocols

1) 104-week Dietary Chronic Study and Carcinogenicity Study with Perfluorooctane Sulfonic Acid Potassium Salt (PFOS: T-6295) in Rats, Covance Laboratories Inc., Study Number 6329-183. In progress. Interim data provided.

2) 26-Week Capsule Toxicity Study with Perfluorooctane Sulfonic Acid Potassium Salt (PFOS: T-6295) in Cynomolgus Monkeys, Covance Laboratories Inc., Study Number 6329-223. In progress. Interim data provided.

3) Protocol for Study: Low Level PFOS Dose versus Rat Serum and Liver PFOS, 3M Medical Department, Corporate Toxicology, Study No. T-6295.16 DT31, October 29, 1998. (Study in progress.)

4) Protocol for Study: Pharmacokinetic Study of POSF in Rats, 3M Medical Department, Corporate Toxicology, Protocol for Study No. T-7098.1, January 7, 1999. (Study in Progress).

5) Study Plan, ST-43: Standard Procedure for Liver Subellular Fractionation, undated, 3M Toxicology Laboratory

6) Plan for Study Nos. T-6295.23; ST-46, Exploratory In-Vitro Pervutaneous Absorption Study of Theophylline, Salicylic Acid, Perfluorooctylsulfonate, and Ammonium Perfluorooctanoate in SkinEthic Reconstituted Epidermis Model, May 4, 2000, 3M Toxicology Laboratory

7) Study Plan, Luebker, Perfluorooctane Sulfonic Acid Induced HMG-CoA Reductase Inhibition in Pregnant Rats and Rat Pups, January 21, 2000

ATTACHMENT TO LETTER TO C. AUER DATED MAY 4, 2000: PLANNED ENVIRONMENTAL STUDIES ON PERFLUOROOCTANESULFONATES

Environmental Fate and Transport

Soil Adsorption/Desorption, 3M Environmental Laboratory. Start date: May 2000. Protocol in progress.

Multi-Media Modeling of PFOS Distribution, Mackay, D. (Trent University) Start date: June 2000. Protocol in progress.

Bioconcentration Factor, Wildlife International, Ltd. Start date: Sept. 2000. Protocol in progress.

Ecotoxicity Elements

Acute Toxicity to Bluegill Sunfish, Wildlife International, Ltd. Start date: July 2000. Protocol in progress.

Acute Toxicity to Sheepshead Minnow, Wildlife International, Ltd., Start date: July 2000. Protocol in progress.

Phytotoxicity – Vegative Vigor and Plan Uptake, Wildlife International, Ltd. Start date: June 2000. Protocol in progress.

Acute Toxicity to *Eiseinia foetida* (Earthworms), Wildlife International, Ltd. Start date: June 2000. Protocol in progress.

FETAX (Frog Embryo Teratogenesis), Wildlife International, Ltd. Start date: June 2000. Protocol in progress.

Mallard Duck Reproduction (Dietary), Wildlife International, Ltd. Start date: July 2000. Protocol in progress.

Bobwhite Quail Reproduction (Dietary), Wildlife International, Ltd. Start date: July 2000. Protocol in progress.

Environmental Monitoring (Environmental Sampling & Release Estimation)

Estimation of PFOS in Life-Cycle Waste Streams, Battelle Memorial. Start date: May 2000. Protocol in progress.

Estimation of Life-Cycle Releases, Battelle Memorial Institute. Start date: January 2001. Protocol in progress.

Carpet Release Study, Battelle Memorial Institute. Start date: June 2000. Protocol in progress.

Multi-City Study, Centre Analytical Laboratories, Inc.; 3M Environmental Laboratory. Start date: June 2000. (Study plan being provided to EPA)

Multi-City Study – Analyses of Sediments. Start date: June 2000.

Multi-City Study – Analyses of Water Columns. Start Date: Sept. 2000.

Multi-City Study – Analyses of Surface Water Film. Start date: Sept. 2000.

Multi-City Study – Analyses of POTW Effluents. Start date: Sept. 2000.

Multi-City Study – Analyses of POTW Sludge. Start date: June 2000.

Multi-City Study – Analyses of Landfill Leachates. Start date: Sept. 2000.

Multi-City Study – Analyses of Fish. Start date: June 2000.

Multi-City Study – Analyses of "Market Baskets." Start date: June 2000.

Attachments to Letter to C. Auer dated May 4, 2000
Perfluoroctane Sulfonate Studies

Planned Studies

1) Preliminary Study Outline, One Generation Reproduction Study of PFOS in Rats, Pharmacokinetic Analysis, May 3, 2000

2) Preliminary Study Outline, One Generation Reproduction Study of PFOS in Rats, Mevalonic Acid/Cholesterol Challenge and NOEL Investigation in Rats, April 27, 2000

ATTACHMENT TO LETTER TO C. AUER DATED MAY 4, 2000:
PRE-1976 ENVIRONMENTAL STUDIES ON PERFLUOROOCTANESULFONATES

Data from Fathead Minnow Study on FC-93 (25% NH4 salt of PFOS in IPA and water), 3M Environmental Laboratory, Aug. 2, 1974. (Robust summary, MSDS and copies of data being provided to EPA)

Data from Fathead Minnow Study on FC-93 (25% NH4 salt of PFOS in IPA and water), 3M Environmental Laboratory, Oct. 19, 1974. (Robust summary, MSDS and copies of data being provided to EPA)

BOD/COD results for FC-93 (25% NH4 salt of PFOS in IPA and water), 3M Environmental Laboratory. Completion date: July 18, 1974. (Robust summary and copy of results being provided to EPA)

ATTACHMENT TO LETTER TO C. AUER DATED MAY 4, 2000:
PRE-1976 TOXICOLOGY STUDIES ON PERFLUOROOCTANESULFONATES

Eye and Skin Irritation Studies Report on Sample T-1117, Warf Institute Inc., Project No. 4102871, November 7, 1974, and explanatory correspondence indicating material is FC-95 (Perfluorooctane Sulfonate potassium salt)

Eye, Skin and Acute Dermal LD50 Study Report on Sample T-991 (FC-93, L-3356, Ammonium Salt of Perfluorooctane Sulfonate, 25% in 20% Isopropyl Alcohol and 55% Water), Warf Institute Inc., Project No. 4053862, June 25, 1974

Attachments to Letter to C. Auer dated May 4, 2000
Perfluoroctane Sulfonate Studies

Bibliography of Acute Toxicity Studies Not Submitted

1) Acute Oral Toxicity-Rats on Sample T-1389, Biosearch Inc., March 4, 1976 (FC-95, Perfluorooctane Sulfonate potassium salt).

2) Acute Oral Toxicity-Rats on Sample T-1390, Biosearch Inc., March 4, 1976. [FC-98]

3) Acute Oral Toxicity-Rats on Sample T-2297 CoC (Ammonium Perfluorooctane Sulfonate, FC-93 Solids), Biosearch, Inc. October 13, 1978.

4) Acute Oral Toxicity-Rats on Sample T-2275 CoC (FC-99 Old Formula, L-4299, NB 48490, which is 50 % of the Diethanolamine Salt of Perfluorooctane Sulfonate in water), Biosearch, Inc., September 25, 1978.

5) Primary Skin Irritation Test with T-2509CoC in Albino Rabbits, Safety Evaluation Laboratory, Riker Laboratories, Inc., Experiment No. 479EB0332, 3M Reference no. T-2509.4 (FC-99 New Formula, L-4509, 25 % Diethanolamine Salt of Perfluorooctane Sulfonate in water), June 28, 1979.

6) Acute Ocular Irritation Study with T-2509CoC in Albino Rabbits, Safety Evaluation Laboratory, Riker Laboratories, Inc., Experiment No. 479EB0333, 3M Reference no. T-2509.1 (FC-99 New Formula, L-4509, 25 % Diethanolamine Salt of Perfluorooctane Sulfonate in water), July 13, 1979.

7) Acute Inhalation Toxicity Study of T-2308 CoC in the Rat, Bio/dynamics, Inc., Project No. 78-7187, 3M Reference No. T-2308 (FC-98, Potassium Perfluoroethylcyclohexyl Sulfonate, presumed 100 %), April 12, 1979.

8) Acute Ocular Irritation Test with T-2960CoC in Albino Rabbits, Safety Evaluation Laboratory, Riker Laboratories, Inc., Experiment No. 0880EB0598, 3M Reference No. T-2960.2 (FC-90, L-4649, Diethanolamine Salt of Perfluoroethylcyclohexyl Sulfonate, 25 % in water), February 18, 1981.

9) Acute Dermal Toxicity Study with T-2960CoC in Albino Rabbits, Safety Evaluation Laboratory, Riker Laboratories, Inc., Experiment No. 0880AB0599, 3M Reference No. T-2960.1 (FC-90, L-4649, Diethanolamine Salt of Perfluoroethylcyclohexyl Sulfonate, 25 % in water), January 15, 1981.

10) Primary Skin Irritation Test with T-2960CoC in Albino Rabbits, Safety Evaluation Laboratory, Riker Laboratories, Inc., Experiment No. 0880EB0597, 3M Reference

No. T-2960.4 (FC-90, L-4649, Diethanolamine Salt of Perfluoroethylcyclohexyl Sulfonate, 25 % in water), January 15, 1981.

11) Acute Oral Toxicity Study with T-2960CoC in Albino Rabbits, Safety Evaluation Laboratory, Riker Laboratories, Inc., Experiment No. 0980AR0600, 3M Reference No. T-2960.3 (FC-90, L-4649, Diethanolamine Salt of Perfluoroethylcyclohexyl Sulfonate, 25 % in water), February 18, 1981.

12) Acute Oral Toxicity - Rats, Biosearch, Inc., 3M Reference No. T-2296 (FC-93, Ammonium Pefluorooctane Sulfonate, 25 % in 20 % Isopropyl Alcohol and 55 % Water), October 19, 1978.

ATTACHMENT TO LETTER TO C. AUER DATED MAY 4, 2000: PUBLISHED STUDIES ON PERFLUOROOCTANESULFONATES

Environmental Studies

Key, B.L.; Howell, R.D.; Criddle, C.S. "Fluorinated Organics in the Biosphere," *Environ. Sci. Technol*, 1997, 31: 2445-2454

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Toxicology Studies

Belisle J, Hagan DF (1978). Anal Biochem. 87, 545 (Note Error: In this report the blank was erroneously reported as 0.02 mg; it should be 0.02μg.)

Belisle, J (1981). Science 212, pp. 1509-1510.

Guy, WS (1972). Ph.D. Thesis, University of Rochester, Rochester, NY.

Guy, WS, Taves, DR, Brey, Jr., WS (1976). Organic fluorocompounds in human plasma: prevalence and characterization. (In) Biochemistry Involving Carbon-Fluorine Bond, pages 117-134.

Haughom, B. and Spydevold, O. 1992. The mechanism underlying the hypolipemic effect of perfluorooctanoic acid (PFOA), perfluorooctane sulphonic acid (PFOSA) and clofibric acid. *Biochim. Biophys. Acta.* 1128, 65-72.

Ikeda, T., Fukuda, K., Mori, I., Enomoto, M., Komai, T. and Suga; T. 1987. Induction of cytochrome P-450 and peroxisome proliferation in rat liver by perfluorinated octanesulfonic acid. In: *Peroxisomes in Biology and Medicine*, H.D. Fahimi and H. Sies, Eds. Springer Verlag, New York, 304-308.

Johnson, J.D., Gibson, S.J. and Ober, R.E. 1984. Cholestramine-enhanced fecal elimination of carbon-14 in rats after administration of ammonium [^{14}C]perfluorooctanoate or potassium [^{14}C]perfluorooctanesulfonate. *Fund. Appl. Toxicol.* 4, 972-976.

Nabbefeld D., Butenhoff J., Bass N. and Seacat A. 1998. Displacement of a fluorescently labeled fatty acid analogue from fatty acid carrier proteins by wyeth-14,643, ammonium perfluorooctanoate, potassium perfluorooctane sulfonate and other known peroxisome proliferators. (SOT Abstract. Accepted, *Toxicologist* 1998).

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Sohlenius, A-K., Eriksson, A.M., Högström, C., Kimland, M. and DePierre, J.W. 1993. Perfluorooctane sulfonic acid is a potent inducer of peroxisomal fatty acid B-oxidation and other activities known to be affected by peroxisome proliferators in mouse liver. *Pharmacol. Toxicol.* 72, 90-93.

Taves D (1968a). Evidence that there are two forms of fluoride in human serum. Nature 217;1050-1051.

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Taves D, Guy W, Brey W (1976) . Organic fluorocarbons in human plasma: Prevalence and characterization. In: Filler R, eds. Biochemistry Involving Carbon-Fluorine Bonds. Washington, DC:American Chemical Society, pages 117-134.

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PHYSICAL – CHEMICAL PROPERTIES

A Robust Summary, Final Report, and Protocol on the physical-chemical properties of perfluorooctanesulfonate are included for each of the following parameters:

PARAMETER	DATE OF REPORT	RESULTS
Melting Point/Melting Point Range	2/24/99	$\geq$ 400°C
Vapor Pressure	5/5/99	3.31×10^{-4}P@20°C
n-Octanol/Water Partition Coefficient	2/11/00	Not calculable; three phases
Air-Water Partition Coefficient	3/19/00	0($<2 \times 10^{-6}$)
Solubility in pure water	5/3/99	570mg/l
Solubility Measurements on FC-95	2/6/81	1080mg/l

The data presented in the study "Solubility Measurements on FC-95," was determined by indirect measurement, not by actual analysis. Therefore, the data is not reliable.

Please note that the March 1, 2000 submittal to EPA entitled "Sulfonated Perfluorochemicals in the Environment Sources, Dispersion, Fate and Effects" included solubility data on water other than pure (i.e., fresh water; filtered sea water; unfiltered sea water). These data were developed, however, in support of other studies and not produced using GLP Standards. For this reason, Robust Summaries, Final Reports, or protocols for this specific data are not being provided.

ENVIRONMENTAL FATE AND TRANSPORT

This section presents information and test results from abiotic, and biotic degradation and soil adsorption studies. Degradation studies include hydrolysis, photolysis, and biodegradation. Much of this work is in progress with final reports scheduled for the June to August, 2000 timeframe.

As these studies progress, there are certain key findings that can be presented as preliminary results:

1. There has been no indication that perfluorooctanesulfonate undergoes any degradation from hydrolysis, photolysis, or biodegradation mechanisms.

2. In all hydrolysis and photolysis studies, perfluorooctanesulfonate has not been detected as a degradation product in any conclusive experiment. This preliminary finding calls into question the assumption of expected degradation of other fluorochemicals to perfluoroctanesulfonate.

3. In the studies focused on hydrolysis of fluorochemical polymers that form the structure of the specific industrial and consumer products, it has been determined that these materials are relatively stable in the environment. For example, the following half-lives are estimated for various polymers:

POLYMER	HALF-LIFE
Acrylate and ester	1-5 years
Polyethylene glycol based	3-50 years
Urethane	>500 years

For hydrolysis to occur, polymers must be subjected to an aqueous environment, which is not expected to occur in a municipal or industrial landfill.

4. Relative to photolysis, the current data suggests a hypothesis that these materials will photolyze to carboxylate structures. These structures have much different properties then sulfonates in that they are much less bioaccumulative in ecological species.

Additional discussion of these results and ongoing studies will be presented in subsequent submissions and reports.

ECOTOXICITY ELEMENTS

This section presents information and test results from a series of ecotoxicity studies on perfluorooctanesulfonates. The information is presented as Robust Summaries, Final Report and Protocol for each ecotoxicity element.

The studies performed during 1999 and in early 2000 were carried out using GLP Standards. In contrast, ecotoxicity studies performed during the period 1974 to 1998 were conducted using protocols and analytical methodologies available at the time of the study. In addition, in these older tests, the sulfonated perfluorochemical products were variable mixtures and contained more impurities. Several tests were hampered by the insolubility of the perfluorochemical and results are expressed as "greater than" the measured solubility. Therefore, the data presented in these historical reports may not be reliable.

ENVIRONMENTAL MONITORING:
PART ONE -- MULTI-CITY STUDY

The multi-city study was designed to obtain preliminary data about dispersion of fluorochemicals in the environment, uptake into foods and presence in drinking water to understand the potential sources of human and environmental exposures that might result from this type of dispersion. The multi-city study paired a city having manufacturing or commercial use of fluorochemical products based on customer sales with a city that does not. Initially six cities, (three pairs) are being examined. The study may be expanded depending on further results.

The multi-city study will yield environmental distribution data as well as data on potential sources of human exposure. The cities were selected to represent urban locations with various levels of fluorochemical releases and various types of municipal water supplies. The samples to be obtained, where possible, include: urban air, surface water column and surface microlayer, sediment, river fish, drinking water intake, treated drinking water, tap water, the influent to and effluent from publicly-owned waste treatment works, sludge, and municipal landfill leachate. Additionally, a "market basket" of several food products will be sampled. These include: beef, pork, chicken, hot dogs, catfish, eggs, milk, bread, green beans, apples from grocery stores and, if possible, produce from local farmers' markets.

The attached material data provides more detail on the design and structure of the study and represents the first results from the multi-city study. Included are reports on the quality assurance plan and field sampling procedures used and the results of the drinking water samples taken from the six cities. The results indicate that drinking water in four cities (Decatur, Alabama; Cleveland, Tennessee; Mobile, Alabama; and Port St. Lucie, Florida) did not contain detectable levels of fluorochemicals. Only two cities (Columbus, Georgia, and Pensacola, Florida) contained detectable levels of sulfonated fluorochemicals in the drinking water. The results show that the levels are in the range of 40-60 parts per trillion of perfluoroctane sulfonate. Only one city, Columbus, Georgia, showed very low detectable levels of perfluorooctanoate.

Also included is a copy of a draft "lifetime" drinking water health advisory developed for PFOS. This advisory reflects a very conservative approach based on application of "safety factors." The advisory level of 1 part per billion should not be misconstrued as threshold for danger or concern, but only a reference point based on application of conservative methods and the information available to date. A comparison of the drinking water data from the multi-city study indicates that there are two orders of magnitude of safety between the draft drinking water advisory and the results from these two cities in the multi-city study.

ENVIRONMENTAL MONITORING:
PART TWO – BIOSPHERE SAMPLING AND ANALYSIS

A plan to assess potential environmental exposure to perfluorooctanesulfonate and other fluorochemical substances has been developed by 3M and outside experts. One component of this plan involves characterization of the geographic distribution of fluorochemicals in biotic and abiotic receptors. Two studies are in progress, one focused in the vicinity of the 3M Decatur, Alabama manufacturing facility, and the other a much more comprehensive global biosphere monitoring program. The preliminary results obtained to date have been reported in the 3M Environmental White Paper entitled "Sulfonated Perfluorochemicals in the Environment: Sources, Dispersion, Fate and Effects."

The study in the Decatur, Alabama area is being designed to understand the impact, if any, of production operations in the local environment. Samples of the groundwater, surface water, sediments and fish and bird species will be collected in May and June, 2000 for analyses. This data will be used to evaluate the environmental presence of fluorochemicals and to assess the potential of any effects using ecotoxicological test results.

The Biosphere monitoring program was designed in consultation with Dr. John Geisy of Michigan State University. This plan is being viewed as an iterative process to assess global distribution of fluorochemicals. As results are obtained from the global environment, the plan is to concentrate on those areas where fluorochemicals are detected in samples and focus on additional sampling and analyses in those specific locations.

Initially, samples of tissues and blood plasma are being collected from archived specimens covering different species and locations. Areas of focus include North America (Great Lakes and coastal marine locations), the arctic region, and Europe. Species to be studied include lake trout, walleye, salmon, catfish, and brown trout; cormorants, eagles and albatross; mussels and shellfish; marine mammals; and other species. This sampling plan is in progress and as data is obtained and reports generated, additional submissions will be made to EPA.

Included in this section are the following documents:

1. **LCMSMS Analysis of Extracts reported in: "Preliminary Report Analysis of Perfluorinated Compounds in Environmental Samples" by P. Jones and K. Kannan - 4/7/99**

2. **Final Laboratory 3M Reports on Analysis of Fluorochemicals in Wild Bird Livers – 4/28/99**

3. **Screening of PFOS levels in Eagle and Albatross - 5/8/98**

EXHIBIT F

EPA United States Environmental Protection Agency

Headquarters Press Release

Washington, DC

Date Published: 05/16/2000

Title: EPA and 3M

FOR RELEASE: TUESDAY, MAY 16, 2000

Following negotiations between EPA and 3M, the company today announced that it will voluntarily phase out and find substitutes for perfluorooctanyl sulfonate (PFOS) chemistry used to produce a range of products, including some of their Scotchgard lines. 3M data supplied to EPA indicated that these chemicals are very persistent in the environment, have a strong tendency to accumulate in human and animal tissues and could potentially pose a risk to human health and the environment over the long term. EPA supports the company's plans to phase out and develop substitutes by year's end for the production of their involved products.

"Today's phaseout announcement by 3M will ensure that future exposure to these chemicals will be eliminated, and public health and the environment will be protected," said EPA Administrator Carol M. Browner. "EPA will work with the company on the development of substitutes to ensure that those chemicals are safe for the environment. 3M deserves great credit for identifying this problem and coming forward voluntarily."

PFOS chemicals are used to produce a range of products from fire fighting foams, coatings for fabrics, leather, and some paper products, to industrial uses such as mist suppressants in acid baths. The company is continuing a major research effort on these chemicals to enhance the understanding of any potential risks that may be associated with this class of chemicals. EPA will also be evaluating the chemicals to determine how individuals and the environment are exposed and what potential adverse effects may exist. If future regulatory actions are required, EPA will take them.

At present, 3M is the only US manufacturer of PFOS. EPA will be contacting foreign governments and other chemical manufacturers, both domestically and internationally, to

seek their support for a voluntary phaseout of PFOS and related chemicals.

R-69 ###

EXHIBIT G

2pp

3M News

AR226-0641

FOR IMMEDIATE RELEASE

3M Phasing Out Some of its Specialty Materials

ST. PAUL, Minnesota – May 16, 2000 – 3M today announced it is phasing out of the perfluorooctanyl chemistry used to produce certain repellents and surfactant products.

The affected product lines represent about two percent of 3M's nearly $16 billion in annual sales. These include many Scotchgard products, such as soil, oil and water repellent products; coatings used for oil and grease resistance on paper packaging; fire-fighting foams; and specialty components for other products. 3M said it plans to substantially phase out production by the end of the year and will work with customers to accomplish a smooth transition. "Our decision anticipates increasing attention to the appropriate use and management of persistent materials," said Dr. Charles Reich, executive vice president, Specialty Material Markets. "While this chemistry has been used effectively for more than 40 years and our products are safe, our decision to phase out production is based on our principles of responsible environmental management."

"We're reallocating resources to accelerate innovation in more sustainable opportunities and technologies. This decision is not only in the public interest, it's in the best interests of all our constituencies … our employees, customers, communities and investors," Reich said.

Sophisticated testing capabilities – some developed in only the last few years – show that this persistent compound, like other materials in the environment, can be detected broadly at extremely low levels in the environment and in people. All existing scientific knowledge indicates that the presence of these materials at these very low levels does not pose a human health or environmental risk. 3M expects to meet consensus earnings estimates for the rest of 2000. This excludes a one-time charge on the order of $200 million, that will be taken sometime this year.

"Our growth engines are more powerful than ever and we're confident in our ability to continue delivering on expectations," said L.D. DeSimone, chairman and CEO. "Many of our new technology platforms directly address the fastest-growing segments of the new economy such as electronics, telecommunications and flat-panel displays." "We expect the positive momentum in our financial performance to continue into 2001 with earnings somewhat above current analyst estimates," DeSimone said.

3M is a leading manufacturer of innovative products for industrial, consumer, trans-portation, safety, health care and other markets, with operations in more than 60 countries worldwide. The company is well known for its "Pollution Prevention Pays" environmental initiative, and its emission reduction programs including water-based replacement of solvents in manufacturing and replacements for ozone-depleting chlorofluorocarbons (CFCs).

Forward-Looking Statements Certain portions of this news release that do not relate to historical financial information constitute forward-looking statements. These forward-looking statements are subject to certain risks and uncertainties. Actual future results and trends may differ materially from historical results or those expected depending on a variety of factors, including: (1) worldwide economic conditions: (2) foreign exchange rates and fluctuations in those rates; (3) the timing and acceptance of new product offerings; (4) raw materials, including shortages and increases in the costs of key raw materials; and (5) legal proceedings.

Scotchgard is a trademark of 3M company.

Contain NO CBI

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Phone: 1-800-367-7683 or e-mail

Customer Contact Specialty Materials

3M Home Page News and Profile Press Box

[3M Home] [News and Profile] [Press Box]

EXHIBIT H

3M to Discontinue Some Scotchgard Repellent Products

By CAROLINE E. MAYER AND DAVID BROWN
MAY 17, 2000 | 12 AM

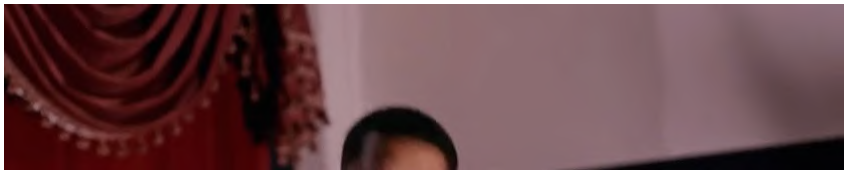
WASHINGTON POST

3M Co. announced Tuesday it would stop making many of its well-known Scotchgard stain-repellent products after finding that one of the chemical compounds used to make the products persists in the environment and is found in the bloodstream of people worldwide.

The substance, perfluorooctane sulfonate, is released in minute quantities by products as various as water-repellent coatings and fire-suppressing foams. It is made almost entirely by 3M, the huge St. Paul-based firm known formally as Minnesota Mining & Manufacturing Co.

Studies have not demonstrated any hazards to human health from the compound, known as PFOS. Like many synthetic compounds, however, it has proved toxic to laboratory animals at high doses.

"We have tested it pretty widely--not only in this country but in other countries, as well--and it's found in very low levels everywhere we test," said Bill Coyne, 3M's senior vice president for research and development. "It is persistent and pervasive, and that is the reason we don't want to continue to add it to the environment."



“Persistent and pervasive” man-made compounds have been among the biggest environmental headaches. . . . For some, such as the pesticide DDT and the insulating fluid PCB, the toxic effects are clear. For others, there is no clear hazard. However, any compound that doesn’t easily degrade is a source of worry.

“It’s not a rare occurrence that we do have persistent chemicals in the environment, but it’s an area that is very much a concern to the agency,” said Stephen Johnson of the Environmental Protection Agency’s office of prevention, pesticides and toxic substances. He said EPA supported 3M’s decision.

Gina Solomon, a physician and senior scientist at the Natural Resources Defense Council, an environmental group, praised the company for “removing the product before there is absolute scientific proof of harm. . . . If companies had taken the same kind of precautionary action with DDT and PCB, then we wouldn’t be in the same bad situation we’re in now.”

PFOS has been used since the 1950s, and 3M health officials have been measuring its concentrations in its workers since the 1970s, as well as monitoring their health.

“There have been no health effects in our employee population,” said Larry Zobel, a physician and the company’s corporate medical director. “People should know that these workers have no health effects related to these materials--that is the bottom line of 30 years of medical monitoring.”

Several years ago, however, company chemists gained the ability to measure PFOS in extremely small concentrations. In tests of stored blood from around the world, they found it in the bloodstream of people in the United States, Japan, Europe and China at levels of 10 to 100 parts per billion. When the ultra-sensitive test was done on numerous blood samples drawn in the 1980s, it was absent, suggesting the compound was beginning to accumulate in human tissue.

That finding led the company to do further toxicological studies on laboratory animals. In one, massive doses were given to rats, whose offspring subsequently showed high death rates soon after birth. Previous studies, at lower doses, had shown no birth defects or high death rates in the animals.

The company notified the EPA of the latest rat study in September 1998 and met with agency officials several months later, Zobel said. In March, the company and the EPA reviewed the data again, and the company decided to cease production of PFOS by the end of the year.

There are no immediate substitutes for the compound, although the company is searching for them, Coyne said. The company will also stop making a second, related compound, called perfluorooctanoic acid, which is used in industrial

processes and does not appear in consumer products. A small amount of PFOS may continue to be manufactured for use in fire-retardant foams, he added.

Innumerable consumer products contain PFOS in trace amounts. The compound is given off by coatings made by 3M and put on furniture fabric, carpets, car upholstery and food packaging to repel oil and water. These coatings can be applied by the manufacturer of the finished product or sometimes by consumers themselves.

"The surprise wasn't that it was in our workers--that's something we've known for some time," said Charles Reich, 3M's executive vice president of specialty material markets. "It was a complete surprise that it was in the blood bank supplies."

The company said the affected product lines account for $320 million in sales, about 2% of the company's annual sales of $16 billion. It said it would take a one-time charge of $200 million this year to reflect the product phase-out.

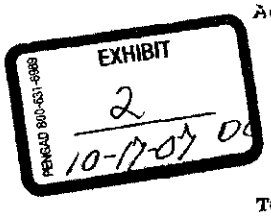
The stock market Tuesday applauded the move, with the stock closing at $90.06, up $4.13.

EXHIBIT I

Interoffice Correspondence 3M

Subject: Fluorocarbons in Human Blood Plasma

August 20, 1975



CONFIDENTIAL

TO: L. C. KROGH - COMMERICAL CHEMICAL DIVISION - 223-6SE
J. D. LAZERTE - COMMERCIAL CHEMICAL DIVISION - 236-1
R. A. NEWMARK - CENTRAL RESEARCH - 201-2W
J. A. PENDERGRASS - MEDICAL DEPARTMENT - 220-2E

FROM: G. H. CRAWFORD - PHOTOGRAPHIC PRODUCTS - 209-1S

Record of a Telephone Conversation - August 14, 1975

Person calling - Dr. William Guy
College of Medicine
University of Florida
Gainesville, Florida

Dr. Guy called again, following up on the subject (vide my earlier memo) to see if we had any further ideas as to possible sources of the fluorocarbon carboxylic acids found in human blood samples from Texas and New York. I got John Pendergrass on the line and Guy brought in a Dr. Tays (who apparently was involved in the original observation).

The original sampling involved plasma specimens from Albany, New York, Rochester, New York (low natural fluoride in the water) Hillsborough, Texas, Andrews, Texas, and Corpus Christi, Texas (high natural fluoride). There was no measurable difference by region (10^{-6} molar F^-). F^{19} NMR studies run by Prof. Wallace Brey (Dept. of Chem., U. of F.) indicate that the fluorine is organic and the suspected species is fluorocarbon carboxylic acid with a C_6 or C_7 fluoroalkyl group. Dr. Brey suspects a branched end on the chain, e.g. perfluoro t-butyl.

The discussion involved Dr. Guy's speculative questions as to where such a "universal" presence of such compounds in human blood could come from. (The compounds are not present in laboratory animals.) These included:

1. Biosynthesis from inorganic F^-.

2. Biosynthesis from aerosol freons (but they don't find chlorine).

3. Teflon cookware.

4. "Scotchgarded" fabrics.

Somewhere he got the information that 3M's fluorocarbon carboxylic acids are used as surfactants and wanted to know if they were present in "Scotchgard" or other items in general use by the public. We plead ignorance but advised him that "Scotchgard" was a polymeric material not a F.C. acid.

Apparently an earlier ('59-'60) study turned up similar quantities of F^- in human plasma (not necessarily FC derived); this would presumably antedate the increased use of either "Scotchgard" or "Teflon" cookware.

They have done experiments involving water boiled in Teflon cookware with negative results.

We suggested obtaining plasma specimens from uncivilized areas, e.g. New Guinea where they don't use too much "Teflon" cookware or "Scotchgard".

Of all the unlikely explanations above, the least unlikely is residual FC 143 (or whatever) we sell to DuPont to polymerize TFE in Teflon cookware. This is still pretty far-fetched. This was not (I hasten to say) suggested to Dr. Guy.

We adopted a position of scientific curiosity and desire to assist in any way possible and suggested that our own analytical people might be able to clarify Dr. Brey's NMR findings (I know Wallace Brey from way back. He is highly respected, conservative and not given to frivolous speculations).

After we hung up I called CRL Analytical, talked to John McBrady and Richard Newmark. It turns out that Newmark is acquainted with Brey and has, in fact, published in a NMR journal edited by Brey.

My recommendation (with J.P.'s concurrence) is to get Richard in touch with Brey, obtain spectra for his own interpretation perhaps samples to run on our equipment, etc. in other words, keep scientists talking to scientists in the spirit of cooperative scientific inquiry.

On the positive side - if it is confirmed to our satisfaction that everybody is going around with fluorocarbon surfactants in their bloodstreams with no apparent ill-effects, are there some medical possibilities that would bear looking into? We

know that fluorocarbons are good oxygen carriers (but this is straight FC-75, not dissolved FC 143). Can fluorocarbon surfactants improve the hemodynamics, wetting and capillary permeation of blood in cases of atherosclerosis, kidney blockage, senility and the like? Can hemolysis, platelet destruction and other blood damage during hemodialysis and cardiovascular surgical procedures be reduced by fluorocarbon surfactants? This is speculation (but not completely wild). I would like to suggest that we consider some animal experiments to see just how much of these materials can, in fact, be tolerated in the bloodstream - both from a defensive point of view and for the above (to me) intriguing reasons. What do you think, John?

GHC/lr

EXHIBIT J

Interoffice Correspondence

Subject: Telephone Call from Dr. Donald R. Taves, University of Rochester, N.Y. September 16, 1975 re Fluorochemicals in Blood

cc: J. E. ...ong -Toxicology-220-2E
J. A. Pendergrass-Medical-220-2E
R. A. Prokop -236-3B
R. J. Seffl -236-2A

September 22, 1975

TO: L. C. Krogh - Comml. Chem. - 223-6SE

FROM: J. D. LaZerte - Comml. Chem. - 236-1

Dr. Taves is one of the members of the research team that is working on the analysis of fluorochemicals in blood. He called to ask several questions and solicit our help. He claims that their findings to date indicate that the 20 ppb of fluorochemical th.. have found in human blood is either a derivative of a perfluorocarboxylic acid or a perfluorosulphonic acid. They are trying to determine specifically which compound or compounds are involved. He wanted to know if he could submit a sample to us for evaluation by our analytical people. I indicated that we would be willing to take a quick look at any such sample he would care to supply.

Dr. Taves and his group have been studying the various FDA petitions based, I presume, on the supposition that the fluorochemical found in the blood might be coming from paper or paperboard. He wanted to know if we would open up the content of our FDA petition to him. I gave him a very firm "no" in this regard, based on the fact that the petition contains our confidential method of manufacturing and yield information. He also asked about the structure of the DuPont material used by the packaging industry. I suggested that he direct this question to DuPont, not to us.

Taves indicated that their initial evaluations have not found fluorocarbons in test animals. One of the graduate students had recognized the fact that the animals whose blood had been used had been laboratory raised. They plan on repeating their work using household pets.

Details of the work that is being conducted by Taves at the University of Rochester and by Drs. Guy and Brey at the University of Florida were presented at the Chicago ACS meeting. The complete papers from this meeting are due to be submitted October 1st.

Wepner
EXHIBIT NO. 2
1-25-18

Taves did indicate that the 20 PPB might pose no toxicological hazard whatsoever but he left no doubt that they would pursue this subject until they have identified the source of the material and delved further into its toxicology.

JDL:ha

EXHIBIT K

J. HILLIS MILLER HEALTH CENTER • UNIVERSITY OF FLORIDA • GAINESVILLE
COLLEGE OF DENTISTRY
DEPARTMENT OF BASIC DENTAL SCIENCES
MSB/ BOX 232 Box J-424
area code 904 392-2661
zip 32610

October 16, 1975

R. A. Mitsch
3M Company
3M Center
Bldg. 235-1N
St. Paul, Minn. 55101

Dear Dr. Mitsch:

Enclosed is the copy of the paper I promised to send you at the ACS meeting. If you or other interested parties at 3M have any ideas on how we can better characterize these fluorocompounds please let either Dr. Taves or me know.

Yours truly,

Warren Guy

Warren S. Guy, D.D.S., Ph.D.

WG:jb

cc: Dr. Taves

Organic Fluorocompounds in Human Plasma: Prevalence and Characterization

Guy, W.S. (University of Florida, Department of Basic Dental Sciences); Taves, D.R. (University of Rochester, Department of Pharmacology and Toxicology); Brey, W.S., Jr. (University of Florida, Department of Chemistry).

Taves discovered that samples of his own blood serum contained two distinct forms of fluoride (1-4). Only one of these was exchangeable with radioactive fluoride. The other, non-exchangeable form was detectable as fluoride only when sample preparation included ashing. This paper is concerned with three aspects of this newly discovered, non-exchangeable form: 1) its prevalence in human plasma, 2) how its presence in human plasma affects the validity of certain earlier conclusions about the metabolic handling of the exchangeable form of fluoride, and 3) its chemical nature.

Preliminary work in this laboratory suggested that the non-exchangeable form was widespread in human plasma but did not exist in the plasma of other animals. Ashing increased the amount of fluoride an average of 1.6 ± 0.25 SD μM (range 0.4 - 3.0) in samples of plasma from 35 blood donors in Rochester, N.Y. (5). No such fluoride was detectable (above 0.3 μM) in blood serum from eleven different species of animal including horse, cow, guinea pig, chicken, rabbit, sheep, pig, turkey, mule and two types of monkey (6).

Standard methods for analysis of exchangeable fluoride in serum have in the past included ashing as a step in sample preparation (7). Taves showed that the amount of fluoride in serum that would mix with radioactive fluoride was only about one-tenth the amount generally thought to be present based on analyses using these older methods (4). When plasma samples from individuals living in cities having between 0.15 and 2.5 ppm fluoride in their water supply were analysed by these older methods, no differences were found between the averages for the different cities. This led to the conclusion that "homeostasis of body fluid fluoride content results with intake of fluoride up to and including that obtained through the use of water with a fluoride content of 2.5 ppm" (8). If the non-exchangeable form of fluoride predominated in these samples, differences in the exchangeable fluoride concentration would probably not have been apparent, and it would be unnecessary to postulate such rigorous

homeostatic control mechanisms for fluoride.

In this study plasma samples were collected from a total of 106 individuals living in five different cities with between 0.1 and 5.6 ppm fluoride in their public water supply. These were analyzed for both forms of fluoride. In this way the relationship between exchangeable fluoride concentration in the plasma and the consumption of fluoride through drinking water was re-evaluated, and the prevalence of the non-exchangeable form was further studied.

With respect to the chemical nature of the non-exchangeable form of fluoride several lines of evidence suggested that it was some sort of organic fluorocompound of intermediate polarity, tightly bound to plasma albumin in the blood. It migrated with albumin during electrophoresis of serum at pH nine (3) and was not ultrafilterable from serum (2). Attempts at direct extraction from plasma with solvents of low polarity like heptane, petroleum ether and ethyl ether were generally unsuccessful. Treatment of albumin solution (prepared by electrophoresis of plasma) with charcoal at pH three did remove the bound fluorine fraction. And finally, when plasma proteins were precipitated with methanol at low pH the fluorine fraction originally bound to albumin appeared in the methanol-water supernatant in a form which still required ashing to release fluorine as inorganic fluoride (5). Based on these considerations the non-exchangeable form of fluoride in human plasma is referred to as "organic fluorine" throughout the rest of this paper.

In order to further characterize the organic fluorine fraction, it was purified from 20 liters of pooled human plasma and characterized by fluorine nmr.

Materials and Methods

Analytical Methods. Values for organic fluorine were calculated by taking the difference between the amount of inorganic fluoride in ashed and unashed portions of the same material.

The following procedure was used to prepare ashed samples: 1) samples (sample size for plasma was 3 ml) were placed in platinum crucibles and mixed with 0.6 mmoles of low fluoride $MgCl_2$ and 0.1 mmoles of NaOH, 2) these were dried on a hotplate and then ashed (platinum lids in place) for 2-4 hr at 600° C in a muffle furnace which had been modified so that the chamber received a flow of air from outside the building (room air increased the blank and made it more variable), and 3) ashed samples were dissolved in 2 ml of 2.5 N H_2SO_4 and transferred to polystyrene diffusion dishes using 2 rinses with 1.5 ml of water.

The following procedure was used for separation of fluoride from both ashed and unashed samples: 1) samples (sample size for unashed plasma was 2 ml) were placed in diffusion dishes (Organ Culture Dishes, Falcon Plastics, Oxnard, Calif., absorbent

removed, rinsed with water), acidified with 2 ml of 2.5 N H_2SO_4, and agitated with a gentle swirling action on a laboratory shaker for 30 min to remove CO_2; 2) for each sample the trapping solution (0.5 ml, 0.01 N NaOH + phenolthalein-p-nitrophenol indicator) was placed in a small polystyrene cup in the center-well of the diffusion dish, 1 drop of 10% Triton-X 100 was added to the sample to decrease surface tension and make the diffusion rate more uniform between samples containing plasma and those not, the lid with a small hole made near its lateral margin was sealed into place with petroleum jelly, 0.02 ml of 4% hexamethyldisiloxane (Dow Corning, Fluid 200, 0.65 cs, Midland, Mich.) in ethanol was injected through the hole in the lid into the sample, and the hole was sealed immediately with petroleum jelly and a strip of paraffin film; and 3) samples were diffused with gentle swirling for at least 6 hr, diffusion was terminated by breaking the seal, and trapping solutions were removed (the indicator color was checked at this point to insure that they were still alkaline) and dried in a vacuum oven (60° C, 26 in-Hg vacuum, in the presence of a NaOH desiccant).

Fluoride was determined by potentiometry with the fluoride electrode. The system used consisted of a fluoride electrode oriented in an inverted position (model 9409A, Orion Research Inc. Cambridge, Mass.), a calomel reference electrode (fiber type), a plastic vapor shield which just fitted over the bodies of both electrodes forming an enclosed sample chamber in which water-saturated tissue paper was placed above the sample to prevent evaporization of the sample, and a high impedence voltmeter (model 401, Orion).

Samples were prepared and read in the following way: 10 µl of 1 M HAc was drawn into a polyethylene micropipette (Beckman Micro Sampling Kit, Spinco Div., Beckman Inst. Co., Palo Alto, Calif.) and deposited into the cup containing the residue from the trapping solution after drying; the flexible tip of the micropipette was used to wash down the walls of the cup; and the solution was then transferred to the surface of the fluoride electrode and the reference electrode brought into position. Surfaces of the two electrodes were blotted dry between samples.

Samples were read in order of increasing expected concentration and sets of samples were read between bracketing calibration standards. These standards were used in two different ways during a run. First, they were flooded onto the electrode surfaces to equilibrate them to concentrations expected for samples and to make them uniform. This procedure permitted the analyst to take reasonably stable readings for samples within one minute. Secondly, they were used in 10 µl volumes for readings used in preparing the standard curve.

Values for identical samples (usually triplicates) were averaged and the average blank was subtracted from sample means. These were then divided by the average fractional recovery of

fluoride (usually 90 to 95%) in standards treated the same way as the sample set.

Plasticware (Falcon Plastics) was used for all analytical procedures to avoid contamination by fluoride from glass. Liquid volume measurements were made with 1, 5 and 10 ml polystyrene pipettes and a polycarbonate volumetric flask (100 ml).

Reagents were purified to insure uniformly low blanks. Water was redistilled and deionized. Acetic acid and ammonia were redistilled. Fluoride contamination in $MgCl_2$ (analytical grade) was reduced by preparing a 1 M solution containing HCl to pH 1 and scrubbing with hexamethyldisiloxane vapor in a column through which the solution was continuously recycled. Following scrubbing the solution was boiled to one third volume to remove any residual volatile silicones and then made just basic with NH_4OH. Fluoride contamination in H_2SO_4 was reduced by repeated extractions of a 6.7 N solution with hexamethyldisiloxane and then boiling to one third volume to remove the residual silicone.

Buffered calibration standards were made from the same NaOH and HAc stock solutions as for samples.

The blanks for ashed samples ranged between 0.2 and 1.5 nmoles fluoride and were typically about 0.5 nmoles. The blanks were smaller for unashed samples; these ranged between 0.05 and 0.2 nmoles fluoride and were typically about 0.1 nmoles.

Factors affecting recovery of fluoride during diffusion were investigated with $^{18}F^-$ tracer. Recovery during diffusion was 97% after 80 min from 5 ml containing 2 ml of plasma. Increasing the acidity of the sample up to 5 N, the volume of the sample up to 7.5 ml, the amount of cold F^- up to 1 µmole, the amount of fluoride complexors up to 1 µmole of $Th(NO_3)_4$ had no material effect on the rate of fluoride diffusion. The absence of both plasma and detergent in the sample compartment markedly slowed the rate of diffusion. Not shaking the sample also slowed the rate of diffusion. Increasing the alkalinity of the trapping solution to 0.1 N increased the rate of diffusion but the lower concentration, 0.01 N, was required here to permit a lower ionic strength in the sample reading solution.

Overall recovery of added cold fluoride was measured. In samples containing neither plasma nor detergent the recovery after 6 hr diffusion averaged 93% and 95% for ashed and unashed samples, respectively. In samples containing plasma the recovery was 95% after 3 hr diffusion.

The degree to which fluorine from organic fluorocompounds could be fixed as inorganic fluoride by ashing varied from less than 1% for volatile compounds like p-aminobenzotrifluoride, m-hydroxybenzotrifluoride, benzyl fluoride and benzotrifluoride to over 80% for less volatile compounds like 5-fluorouracil, fluoroacetate and p-fluorophenylalanine.

Methods used here for separation of fluoride (diffusion at rm. temp.) (9) and its quantitation (fluoride electrode) (10) are considered to be quite specific for fluoride. One potentially

important interference, however, was codiffusable organic acids which might partially neutralize the trapping solution and thus lower the pH of the buffered reading solution. Indeed, it was found that samples containing relatively large concentrations of acetic acid (e.g., fractions 2, 3 and 4 from step 4 in the purification system) completely neutralized the trap within a few hours. The significance of this problem in the analysis of fluoride in blood plasma was investigated in two ways. First, four samples of human plasma were allowed to diffuse for three weeks, and no change in the color of the phenolphthalein indicator in the trapping solution was observed. Secondly, samples containing the same plasma were diffused for different periods up to 158 hr and the apparent fluoride was determined. No changes were observed between samples which correlated with diffusion time.

The sensitivity of the analytical method was limited by the blank rather than the sensitivity of the instruments used. Reproducibility varied with the amount of fluoride being measured. The coefficient of variation averaged 55% in the low range (samples containing 0.25 to 0.75 nmoles F^-) and 6.6% in the high range (10-12 nmoles F^-).

Blood Plasma. Human plasma was obtained from blood banks in five cities. According to public records these cities had not changed the fluoride concentration of their public water supply for at least six years prior to obtaining the samples. Samples were received in individual polyethylene bags which were part of the Fenwall ACD blood collection system. In blood collection using this system 450 ml of blood is drawn into a bag containing 67.5 ml of anticoagulant acid citrate dextrose (ACD) solution. When the cells are removed the ACD solution remains in the plasma. Because of this dilution of plasma a correction factor of 1.3 was applied to values obtained here for the concentration of fluoride. The potential error in this factor was ± 0.1 because of variation between standard limits for hematocrit and minimum volume of the blood donation. Bovine blood was obtained at slaughter and mixed immediately with ACD solution in 1 liter polyethylene bottles.

Electrophoresis. A continuous flow electrophoretic separator (model FF-3, Brinkman Inst., Inc., Westbury, N.Y.) was employed. Sample flow rate was 2.3 ml/hr, buffer flow rate was 72 ml/hr, voltage was 0.67 kv, and current was 140 mamp. Separation took 19 hr. Plate separation was 1 mm and operating temperature was between 2 and 4° C. The buffer was 0.12% $(NH_4)_2CO_3$, made by bubbling CO_2 from dry ice into redistilled NH_4OH until the pH reached 9.0.

Purification System. Steps in the purification system are summarized in table I. In the first step one liter of plasma (pooled from 5-6 individuals) was dialysed in seamless cellulose tubing (1 in. diameter) against 20 liters of water at 4° C. The dialysate was changed twice at 24 hr intervals. In the second step dialysed plasma was freeze dried.

In the third step the dried powder from electrophoresis was extracted with methanol in a soxhelet extraction apparatus (model 6810 G, Ace Glass, Inc., Vineland, N.J.). Cellulose extraction thimbles (model 6812 G, Ace Glass) were soaked overnight in methanol. Operating conditions were 25° to 30° C under a vacuum of 24 in-Hg. Coolant for the condenser was 80% ethanol; inlet temperature was -10° to -20° C and outlet temperature was -10° to 0°. Two liters of methanol were refluxed through the apparatus for a period of 4 hr and approximately 400 ml were lost to evaporation during that period. Glass beads were placed in the flask to prevent bumping.

In the fourth step the residue from the methanol extract was fractionated according to the method described by Siakotos and Rouser (11) for separating lipid and non-lipid components. The method is based on liquid-liquid partition in a column containing a dextran gel (Sephadex G-25, coarse, beaded, Pharmacia Fine Chemicals, Inc., N.Y.). Four eluents are used: 1) 500 ml chloroform/methanol, 19/1, saturated with water, 2) 1000 ml of a mixture of 5 parts of chloroform/methanol, 19/1, and 1 part of glacial acetic acid, saturated with water, 3) 500 ml of a mixture of 5 parts chloroform/methanol, 19/1, and 1 part glacial acetic acid, saturated with water, and 4) 1000 ml of methanol/water, 1/1. Their method was modified for use here by increasing the column length to that attained by using a full 100 grams of dextran beads. Sample size corresponded to that from 2.5 liters of the original plasma.

In the fifth step the residues from eluents 2 and 3 from two runs of step four were combined, applied to a silicic acid column, and eluted by reverse flow with an exponential gradient of increasing amounts of methanol in chloroform. The column (model SR 25/45, 2.5 cm i.d. x 45 cm, Pharmacia) was filled to a height of 30 cm with silicic acid (Unisil, 100-200 mesh, Clarkson Chem. Co., Inc., Williamsport, Pa., heat activated at 110° C for 2 days) and was washed with a complete set of elution solvents before use. The gradient maker (model 5858, set 4, Ace Glass Co.) was filled with 1 liter of methanol in the upper chamber and 2 liters of chloroform in the lower. The flow rate was adjusted by the height of the solvent reservoirs to an average of 3 ml/min for the first liter of eluent. The sample had to be transferred to the column by repeated washings with chloroform because of its low solubility in this solvent. This usually required about 30 ml of chloroform total. Dead volume for the system as 90 ml. Fractions of 15 ml volume were collected in carefully cleaned glass tubes.

Tubing and fittings to the columns were polytetrafluoroethylene (supplied largely by Chromatronix, Inc., Berkeley, Calif.).

All solvents were redistilled. Methanol and chloroform were ACS certified (Fisher Scientific Co.) and acetic acid was analytical reagent grade, U.S.P. (Mallinckrodt Chemical Works, St. Louis). Solvents were removed from samples in a flash evaporator.

NMR. The nmr spectrum was obtained on a Varian XL-100 spectrometer with Nicolet Technology Fourier Transform accessory. The sample was dissolved in an approximately 1/1 mixture of CH_3OH and $CDCl_3$ and spectra were run in a 5 mm tube. External referencing to $CFCl_3$ was used for the chemical shifts, and these are expressed with positive numbers to lower field (i.e., higher frequency). External lock was used. Typical conditions were a pulse length of 15 microseconds, a delay time between pulse cycles of 2.5 sec, and a time constant of -1 sec for exponential processing.

Results

Values for inorganic fluoride (F^-) and organic fluorine (R-F) in 106 plasma samples from humans living in five cities are shown in table II. These data show that the average fluoride concentration in plasma is directly related to the fluoride concentration in the water supply, and that the average organic fluorine concentration in plasma is not. No relationship between fluoride in plasma and organic fluorine in plasma was apparent by inspection of values for individual samples. The distribution of the values within cities are shown in figures 1 and 2. In both cases the distributions appear to be log normally distributed with only 3 or 4 individuals surprisingly deviant. In the cases of the two individuals with little or no apparent organic fluorine (figure 2, Andrews group, left margin), the inorganic fluoride levels were both in excess of 7 μM, making the difference measurement for organic fluorine difficult. The overall mean value for organic fluorine was 1.35 ± 0.85 SD μM.

Plasma was electrophoresed in an attempt to reproduce the findings of Taves (3) using plasma from another individual. Results shown in figure 3 closely match those found earlier in that a predominant form of organic fluorine appeared to migrate with albumin at pH 9, and in that organic and inorganic forms were clearly separated.

The recovery, mass balance and purification factors for steps in the purification system listed in table I are recorded in table III. These data show that about one-third of the original amount of organic fluorine in plasma is recovered in the major peak from silicic acid chromatography. Another third is accounted for in other fractions and the rest is not accounted for, presumably because of adsorption to surfaces of containers in

which samples were placed.

The blank for the purification process was obtained by using bovine rather than human plasma. No organic fluorine was detectable in the original bovine sample but as a further check the sample was dialysed to remove inorganic fluoride to facilitate making the measurement for organic fluorine by difference. Some organic fluorine was apparent in dialysed bovine plasma: 0.13 ± 0.11 SD μM (n=6), a statistically significant though small difference. This trace amount of organic fluorine clearly was not found in the same silicic fractions as the dominant peak from human plasma as shown in figure 4.

Human plasma had been stored in polyethylene bags with ACD solution. Analysis of ACD solution from unused blood bags and analysis of blood plasma before and after placing it in the bags showed that not more than 5% of the organic fluorine in human plasma could have come from this source.

The distribution of organic fluorine in fractions from silicic acid chromatography are shown in figure 4 for four batches corresponding to 5 liters of the original plasma each. There is clearly one dominant peak lying in approximately the same elution position for each batch (the exact position varied with column use and the degree of hydration of the silicic acid adsorbant). There were always some smaller secondary peaks, but they varied in size and position relative to the major peak.

The sample used for characterization by nmr was obtained by combining the fractions containing the major peaks in each of the four batches. Much of the material from batches one and two had been used for other purposes prior to this combination. The combined sample was rechromatographed on silicic acid and a single sharp peak obtained. The final sample was taken from the central portion of that peak and contained 3.3 μmoles of organic fluorine.

Four sample runs were made on the nmr spectrometer with 15,000 to 17,000 scans each and with a sweep width of 15,151 Hz in all but one run, where it was 7,576. The results of all runs were consistent with the spectrum shown in figure 5 and the chemical shifts shown in table IV. A blank run on the solvent mixture showed no instrumental artifacts which might have contributed to the spectrum. Chemical shifts determined for perfluoro-octanoic acid are also included in table IV. Comparison of the shifts in the unknown with that of perfluoro-octanoic acid show that there is a constant difference in shifts of about 2 ppm except for the $-CF_2-$ peak next to the functional group (peak E) where the shift is about 6 ppm. Only the latter is enough to be considered a significant deviation since external referencing was used for each. The difference in shift for peak E is consistent with the presence of amide or ester derivatives, or possibly with the presence of a sulfonic acid derivative as the functional group. One explanation for the additional peaks in the spectrum is the presence of branched isomers, peaks A and B

representing $-CF_3$ groups at branch points, peak C the $-CF_3$ groups two carbons removed from the branch points, and peak H representing $-CF_2-$ next to the branch points.

The sample was reanalyzed for organic fluorine following characterization by nmr to check for contamination; no additional fluorine was apparent. The degree to which fluorine from perfluoro-octanoic acid is fixed as inorganic fluoride during ashing was found to be 21 ± 3 SD % (n=3).

Discussion

These findings suggest that there is widespread contamination of human tissues with trace amounts of organic fluorocompounds derived from commercial products. All available information on this subject is in accordance with this interpretation. A series of compounds having a structure consistent with that found here for the predominant form of organic fluorine in human plasma is widely used commercially for their potent surfactant properties. For example, they are used as water and oil repellents in the treatment of fabrics and leather. Other uses include the production of waxed paper and the formulation of floor waxes (12). The findings presented here that the concentration of organic fluorine was not related to the concentration of inorganic fluoride either in blood or in the public water supply, and the earlier finding that there was little or no organic fluorine in the blood of animals other than human (6) are all in keeping with environmental sources such as these.

The prevalence of organic fluorine in human plasma is probably quite high since 104 of the 106 plasma samples tested here and all 35 in an earlier study (5) had measurable quantities. The prevalence of the particular compounds isolated and characterized here, i.e., perfluoro fatty acid (C_6-C_8) derivatives, is not known since the starting material for each batch shown in figure 4 was pooled from between 25 and 30 individuals and since only about one third of the original organic fluorine content was accounted for in the fractions containing these compounds (see table III).

Peaks other than the one characterized by nmr appear in the chromatograms shown in figure 4 suggesting that human plasma contains other forms of organic fluorocompounds. They are probably not volatile compounds like freons since it is doubtful that these would be detected by the analytical methods used in this study. They correspond in solubility to very polar lipids since they appear in fractions two and three in the fourth purification step. According to the authors of the method used in that step the first eluent contains most fats, the second and third eluents contain very polar fats like gangliosides and certain bile acids in addition to compounds like urea, phenylalanine and tyrosine. The last fraction contains water soluble non-lipid compounds (11). Components of these other peaks are

less polar than the compounds in the predominant peaks in accordance with the methanol-in-chloroform gradient used to elute them in the fifth purification step. Other forms not seen in silicic acid fractions may also exist since only about half the original organic fluorine was recovered in these fractions.

The actual amounts of the perfluorinated fatty acid derivatives in human plasma is not known both because individual plasma samples were not assayed for these particular compounds and because the degree to which organic fluorine from these compounds is converted to inorganic fluoride during ashing is not known. Metal salts of perfluorinated fatty acids have been reported to decompose at 175 to 250° C forming CO_2, volatile perfluorinated olefins one carbon shorter, and one atom of fluoride per molecule (13). About 3 fluorine atoms per molecule of perfluoro-octanoic acid were fixed as inorganic fluoride by ashing methods used here. Thus, values reported here for fluoride after ashing fractions from the major peaks in figure 4 probably represent somewhere between one-third and one times the molar amount.

Little has been published about the metabolic handling and toxicology of perfluorinated fatty acid derivatives. Computer assisted literature searches using Medline, Toxline and Chemcon developed no information on these subjects. This was surprising with respect to the widespread commercial use of such compounds. It would appear from information presented here that rapid excretion of such compounds into urine is unlikely since they are bound to albumin in the blood. On this topic it can also be stated that other chemicals are usually not toxic in blood concentrations similar to those found here for organic fluorine.

The concentration of organic fluorine in human plasma may be changing with time. In 1960 Singer and Armstrong reported that the plasma of 70 individuals residing in communities with 1 ppm or less fluoride in their public water supply had an average concentration of fluoride of 8.8 µM (8). They prepared their samples by ashing them and then distilling fluoride from the ash acidified with perchloric acid (7). Thus, it seems likely that their values for "fluoride" would have included organic fluorine had it been present. Assuming that inorganic fluoride concentrations at that time were similar to those found in this study (see table II), the organic fluorine component would exceed 7 µM. In 1969 the same investigators using the same method reported an average fluoride concentration of 4.5 µM for 6 plasma samples each pooled from at least 3 individuals supposedly living in fluoridated communities (14). This corresponds to an organic fluorine component of only about 4 µM. Organic fluorine concentration presented here averages only 1.35 µM. Therefore, there may have been a decrease in the concentration of organic fluorine in human plasma since the late 1950's. An alternant explanation might be that differences in

the analytical methods or differences in the sample populations caused these values to vary.

Organic fluorine is the predominant form of fluorine in human blood except where the concentration of fluoride in drinking water is high (in which case fluoride predominates, see table II). This together with the finding reported here that there is no apparent relationship between the concentrations of organic fluorine and inorganic fluoride in plasma helps explain why in earlier studies (8) no relationship was found between plasma fluoride determined in ashed samples and the fluoride content of the public water supply. The data in table II show that when methods specific for inorganic fluoride are applied, a clear relationship between fluoride in plasma and fluoride in the public water supply (between 0.1 and 5.6 ppm) can be demonstrated. Thus, there is no need to postulate the existence of such rigorous homeostatic control mechanisms for plasma fluoride as suggested earlier (8). Average plasma fluoride concentrations for individuals living in the same city as reported here reflect the balance established between fluoride in blood and that in bone mineral over periods of years. These findings do not contradict a passive homeostatic control mechanism in which bone mineral damps swings in blood fluoride concentration over relatively shorter periods of time.

The values presented here for the average inorganic fluoride concentration of plasma from individuals living in a community having about 1 ppm fluoride in the water supply are consistent with recent findings of others using similar methods (14, 15).

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Q. I wonder if you tried to correlate within individuals the level of organic fluorine with age.

A. It would certainly be interesting to have this information but unfortunately we cannot supply it at this time. An expeditious approach might be to analyze chord blood from infants of mothers who had not received fluorine-containing anesthetics at childbirth. It would also be of interest to know whether individuals living in isolated regions have organic fluorine in their blood plasma.

Q. Did you say the sample analyzed by nmr contained methyl alcohol?
A. Yes, I did.

Q. Methyl alcohol will react very rapidly with fluorinated acids. The nmr spectrum may, therefore, represent that of methyl ester derivatives.

A. Methanol was also used in the last three steps of the purification system. The nmr spectrum is consistent with the presence of methyl ester derivatives of perfluorinated fatty acids (C_6-C_8) and their branched isomers.

Table I

PROCEDURE FOR PURIFICATION
OF FLUOROCOMPOUNDS FROM BLOOD PLASMA

Fraction Treated	Treatment	Fraction Removed
blood plasma	step 1: exhaustive dialysis against distilled water	smaller, water-soluble components
plasma proteins & protein-bound substances in water solution	step 2: lyophilization	water
plasma proteins & protein-bound substances	step 3: methanol extraction--soxhelet, 25°C, 24 in-Hg vacuum	plasma proteins
plasma lipids	step 4: column chromatography--liquid-liquid partition on Sephadex	lipids of low polarity and residual polar contaminants
polar lipids	step 5: column chromatography--adsorption on silicic acid	unknown: several yellow fractions

Table II

CONCENTRATION OF FLUORIDE (F^-) AND ORGANIC FLUORINE (R-F) IN BLOOD PLASMA SAMPLES FROM FIVE CITIES HAVING DIFFERENT FLUORIDE CONCENTRATIONS IN THEIR WATER SUPPLY

	[F^-] in Plasma[a], μM			[R-F] in Plasma[a,b], μM		
City ([F^-] in Water, ppm)	Mean± SD(n)	Range	Diff.[c] P<.05	Mean± SD(n)	Range	Diff.[c,d] P<.05
Albany, N.Y. (<.1)	0.38± 0.21 (30)	0.14- 1.1		1.2± 0.6 (30)	0.3- 2.6	
			sig,			n.s.
Rochester, N.Y. (1.0)	0.89± 0.75 (30)	0.35- 4.2		1.6± 1.2 (30)	0.5- 6.8	
			n.s.			n.s.
Corpus Christi, Tex. (0.9)	1.0± 0.35 (12)	0.60- 1.7		1.3± 0.9 (12)	0.4- 3.9	
			sig.			n.s.
Hillsboro, Tex. (2.1)	1.9± 0.9 (4)	0.60- 2.6		2.3± 0.6 (4)	1.5- 2.8	
			sig.			sig.
Andrews, Tex. (5.6)	4.3± 1.8 (30)	1.4- 8.7		1.1± 0.5 (30)	0.1- 2.3	

[a] Each value used in the computation was the average of at least three replicate analyses and was corrected for dilution by ACD solution by multiplying it by 1.3.

[b] taken to be the difference between the amount of inorganic fluoride measured in ashed and unashed aliquots of the same sample

[c] by t-test assuming equal variance in each group

[d] The difference between Rochester and Andrews is statistically significant.

Table III

MASS BALANCE, RECOVERY AND PURIFICATION
FACTOR FOR STEPS IN THE PURIFICATION SYSTEM

Fraction	Dry Wt. grams	Amt. R-F[a] nmoles	Recovery[b] %	Purification
human plasma (ACD, 2.5 liter batch)	200	1725 ±273(6)		
Methanol Extraction				
extract	10.1	1476 ±60(6)	85.6 ±14.0	17 X
residue	--	105 ±37(4)	6.1 ±1.0	--
Sephadex Column				
Fraction I	--	125 ±18(4)	7.3 ±1.6	--
Fractions II + III	1.29	1195 ±129(6)	69.3 ±13.3	108 X
Fraction IV	--	118 ±29(4)	6.8 ±1.2	--
Silicic Acid Column				
major peak	.03[c]	630[d]	36.5	2,440 X
other peaks combined	--	240[d]	13.9	--

[a] mean ± SD(n)
[b] percent of the amount of R-F in the original plasma sample, mean ± SD
[c] estimate based on weighing the contents of two tubes in the center of the major peak
[d] estimate based on area under peaks from graph

Table IV

RESULTS OF NMR SPECTROSCOPIC ANALYSIS

Peak Designation	Chemical Shift[a], ppm: Sample	Chemical Shift[a], ppm: Perfluoro-octanoic Acid	Suggested Assignments
A	-70.7		$-CF_3$ groups at branch points
B	-71.9		terminal $-CF_3$ in branched isomers
C	-80.0		terminal $-CF_3$ in straight chain
D	-81.0	-82.6	$-CF_2-$ next to X[b]
E	-114.3	-120.2	$-CF_2-$ in $-CF_2-CF_2-CF_2-$
F	-120.3	-123.1	$-CF_2-$ next to branch points
G	-121.5	-124.2	$-CF_2-$ next to terminal $-CF_3$
H	-122.3		
I	-126.0	-127.6	

[a] External referencing to $CFCl_3$ was used for the chemical shifts, and these are expressed with positive numbers to lower field (i.e., higher frequency).

[b] where X is likely to be -CO-Y

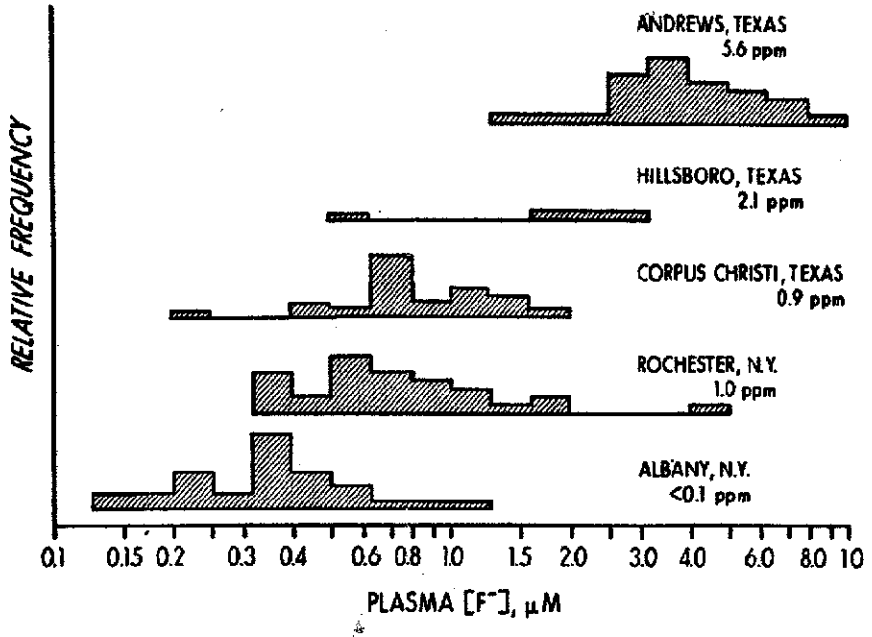


Figure 1. Relationship Between the Concentration of Fluoride in Human Plasma and the Concentration of Fluoride in the Drinking Water

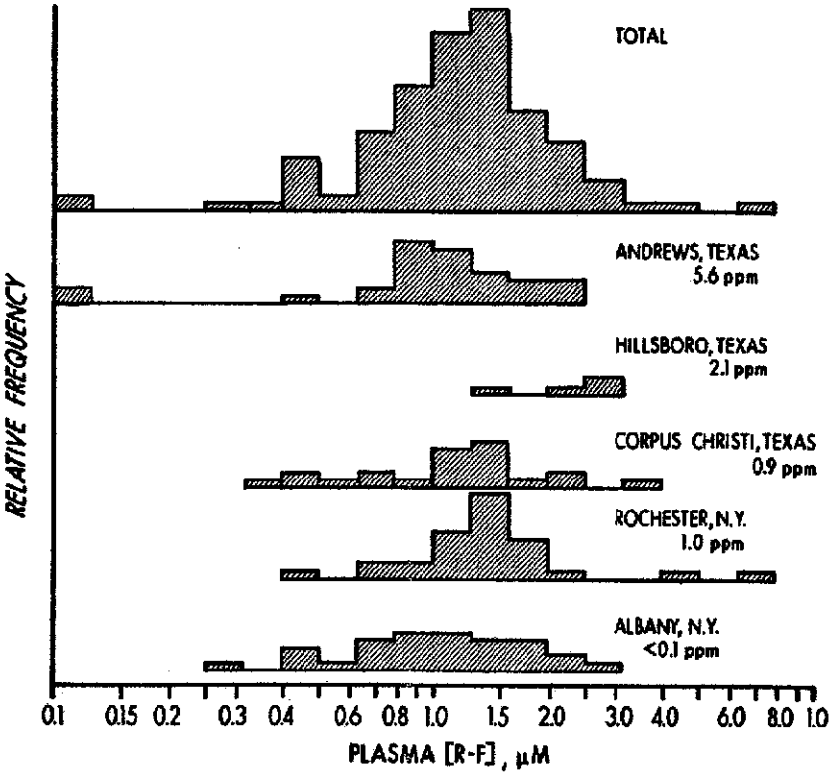


Figure 2. Relationship Between the Concentration of Organic Fluorine in Human Plasma and the Concentration of Fluoride in the Drinking Water

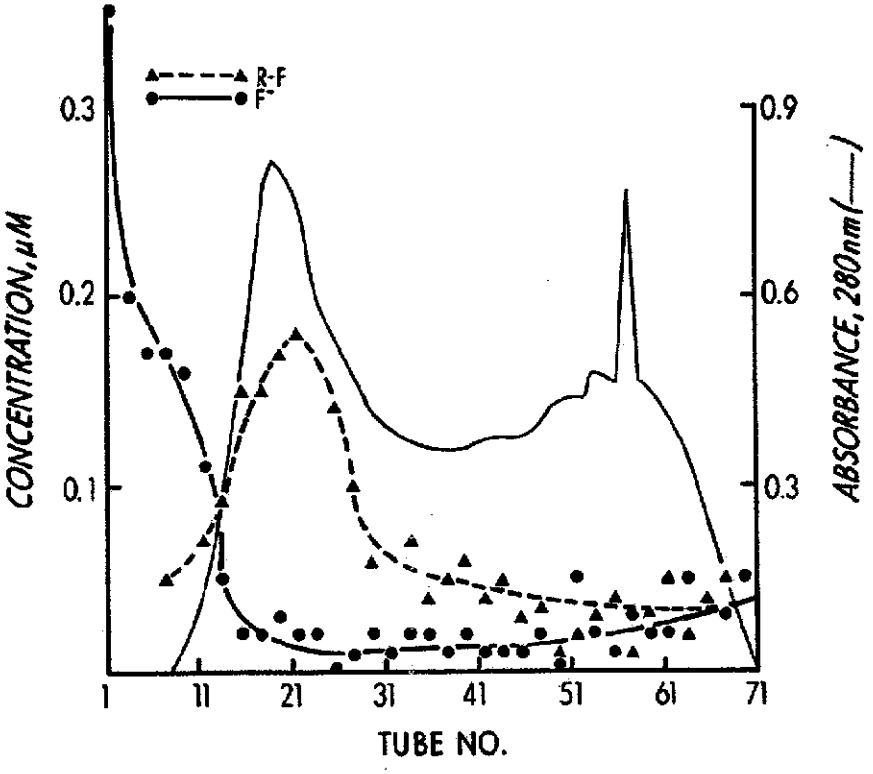


Figure 3. Separation of Fluoride and Organic Fluorine in Human Plasma by Electrophoresis

A sample (about 45 ml) of human plasma was electrophoresed in pH 9 buffer and fractions between the sampling port (near tube 72) and the positive pole (near tube 1) were analysed for the fluoride content of both ashed and unashed aliquots. Relative concentrations of proteins were estimated by absorbance at 280 nm.

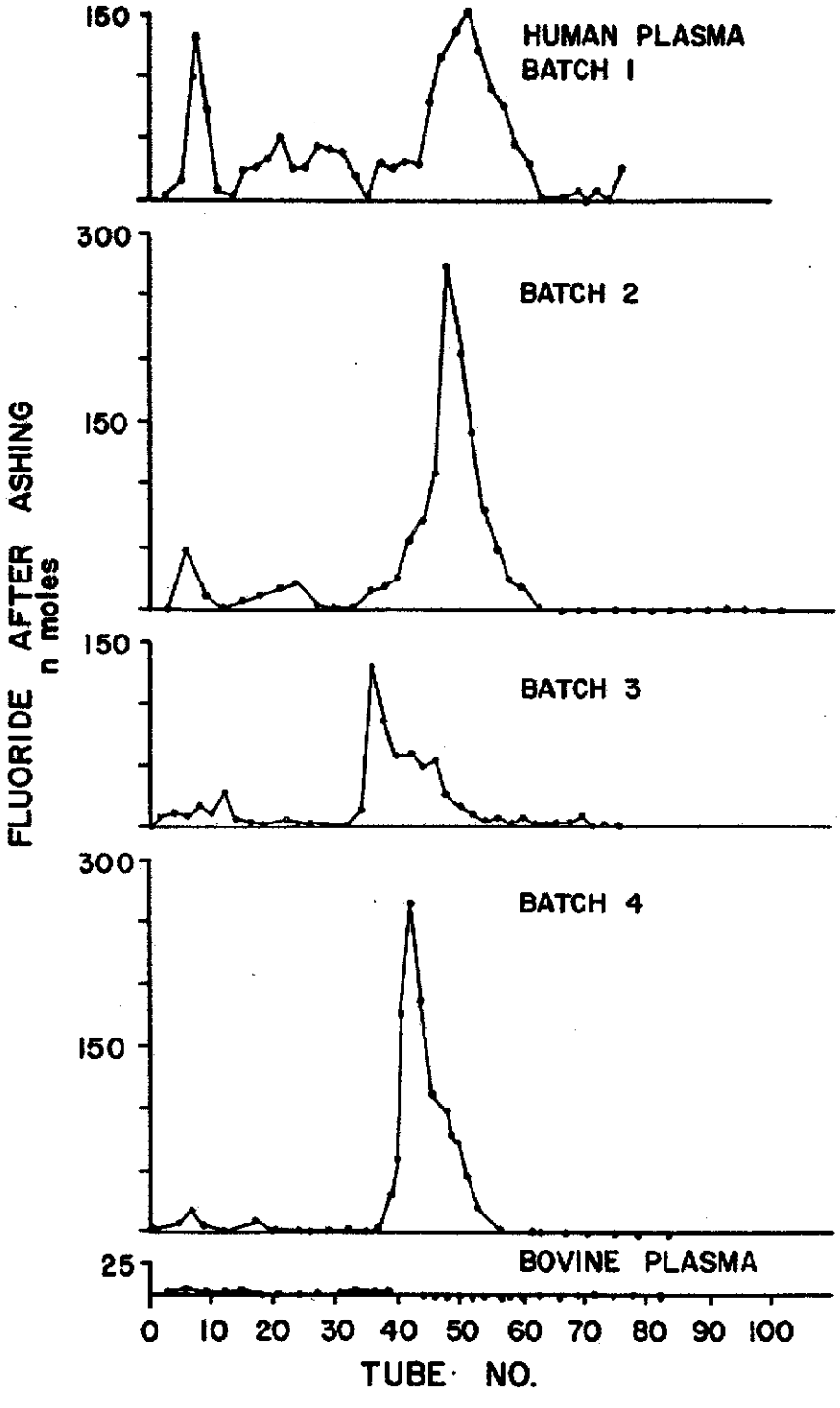


Figure 4. Distribution of Organic Fluorine from Human and Bovine Plasma in Fractions from Silicic Acid Chromatography

Figure 5. NMR Spectrum of Organic Fluorocompound(s) Isolated from Human Plasma

EXHIBIT L

J. A. Ubel, M.D.

3M "CONFIDENTIAL"

CHRONOLOGY - FLUOROCHEMICALS IN BLOOD

AUG 28 1977

August 22, 1975 - Initiating event. J.D. LaZerte receives phone call from W.S. Guy. W.S. Guy, D.R. Taves, and W.S. Brey Jr. are to present a paper at the Chicago A.C. & S. meeting entitled "Characteristics and Concentration of Organic Fluorocompounds Found in Human Tissues". W.S. Guy was attempting to locate the source of the organic fluorocompound and thought that SCOTCHGARD might be the source. J.D. LaZerte advises Guy not to speculate.

August 25, 1975 - At the request of Commercial Chemicals Division Control Research sends B.W. Nippolt to the Chicago ACS Meeting to hear the paper by Guy, Taves and Brey. A copy of the ^{19}F NMR spectrum of the fluorochemical isolated from human blood is shown.

September 17, 1975 - At a joint CRL-CCD meeting B.W. Nippolt presents data from the Chicago ASC paper of Guy, Taves and Brey. A copy of the ^{19}F spectrum of the fluorochemical isolated from human blood is shown.

September 21, 1975 - Commercial Chemicals Division Laboratory begins submitting ten samples of perfluorocarboxylic and perfluorosulfonic acid derivatives to Central Research Analytical for ^{19}F NMR analysis in an attempt to identify the material found by Guy and Taves in human blood.

September 22, 1975 - Taves calls J.D. LaZerte to see if 3M will further analyze sample of fluorochemical isolated from human blood and is given a qualified "yes". Further requests that we open contents of FDA (FC-807) petition to him and is given an unqualified no. Taves indicates "strong and continuing" interest in finding source of fluorochemical.

October 7, 1975 - Central Research Analytical submits research proposal to determine quantity and character of organic fluorine in human blood with an estimated project duration of 5 months and estimated cost of $12,000.

October 21, 1975 - Research proposal accepted by Commercial Chemicals Division.

EXHIBIT Stensm 3 Planet Depos, LLC 11-7-19

November 6, 1975 - Of the ten samples submitted on September 21, 1975, Central Research reports that the ^{19}F NMR analysis shows that the spectrum of $C_8F_{17}SO_3H^*$ or its salts matches that presented by Guy and Taves.

$^*C_8F_{17}SO_3H$ - LD_{50} (Oral)	Less than 630 mg/Kg - Toxic
$C_8F_{17}SO_3K$ - LD_{50} (Oral)	About 1250 mg/Kg - Moderately Toxic

December 16, 1975 - J.D. LaZerte, H.E. Freier and J.E. Long visit Guy and Taves at University of Rochester. Agreement is reached that 3M will attempt to isolate and identify fluorochemicals in blood.

February 17, 1976 - Central Research Analytical completes development of accurate analytical method for determining ppb quantities of organic fluorine in human blood. Method is tested on sample of pooled serum from American Red Cross.

April 14, 1976 - Central Reserach Analytical completes analysis of four blood samples from Commercial Chemical Division personnel. Laboratory personnel exposed to fluorochemicals have up to 100 times "normal" amounts of organically bound fluorine in their blood.

May 4, 1976 - Taves calls D.F. Hagen of CRL and requests help in developing a chromotographic method for analyzing perfluoro-octanoic acid. He requests that we analyze some of his perfluorocanoic acid.

May 13, 1976 - H.E. Freier calls Taves. Agrees to analyze their sample by gas chromotography.

June 29, 1976 - Central Research Analytical completes analysis of nine blood samples including three from Chemolite. Chemolite personnel exposed to fluorochemicals have up to 1000 times "normal" amounts of organically bound fluorine in their blood. Results from previously exposed laboratory personnel indicate that organically based fluorine remains in the blood for an indefinite period.

July 19, 1976 - 3M Medical Department initiates program to study blood chemistry of persons exposed to fluorochemicals.

August 23, 1976 - Central Research Analytical completes analysis of nine blood samples indluding eight from Cordova. Cordova personnel exposed to fluorochemicals have up to 50 times "normal" amounts of organically bound fluorine in their blood.

August 26, 1976 - Central Research Analytical isolates and characterizes fluorochemical from blood of Chemolite supervisor. The fluorochemical is identified as $C_7F_{15}CO_2H$ or one of its salts by G.C. and ^{19}F NMR.

September 9, 1976 - Central Research Analytical completes analysis of the blood of mice which were fed FC-807 at 1000 and 3000 ppm for 30 days. The mice which were fed FC-807 had roughly 4000 times as much organically bound fluorine in their blood as "nonexposed" mice.

September 17, 1976 - Central Research Analytical characterizes the fluorochemical metabolite from the mouse feeding studies as $C_8F_{17}SO_3H$ or one of its salts. Characterization by ^{19}F NMR.

September 20, 1976 - H.E. Freier calls Taves to keep him informed of our interest. Gave Taves results of CRL analysis of the $C_7F_{15}CO_2H$ which Taves sent. Taves is also told:

1. We are using a modified Wickbold method for fluorine analysis.
2. We have analyzed pooled Red Cross plasma and found organic fluorine levels comparable to those in the literature.
3. We have not yet begun to isolate fluorochemicals in pooled Red Cross plasma.

October 8, 1976 - Central Research Analytical completes analysis of thirteen blood samples including seven from Decatur. Decatur personnel exposed to fluorochemicals have up to 300 times "normal" levels of organically bound fluorine in their blood. Other samples show:

1. Rats exposed to FC-70 do not have FC-70 in their blood.
2. Individuals exposed to fluorochemicals over twenty years ago and not exposed since, have "normal" organically bound fluorine levels.

October 18, 1976 - Central Research Analytical isolates and characterizes fluorochemical from blood of Decatur cell operator. The fluorochemical is identified as $C_8F_{17}SO_3H$ or one of its salts by ^{19}F NMR.

October 20, 1976 - H. E. Freier calls Taves to report results on analysis of $C_7F_{15}CO_2H$ sample supplied by Taves.

October 28, 1976 - Dr. Leon Singer requests sample of $C_7F_{15}CO_2H$ from 3M. Singer believes he can improve on Taves's method of analysis.

November 8, 1976 - 3M sends 25 g $C_7F_{15}CO_2H$ to Dr. Leon Singer

November 17, 1976 - Central Research Analytical completes analysis of six blood samples from Chemolite personnel exposed to fluorochemicals and again finds up to 1000 times "normal" levels of organic fluorine. Further analysis of one individual's blood showed both $C_7F_{15}CO_2H$ and $C_8F_{17}SO_3H$ to be present.

Blood samples are sent to General Activation Analysis to see if Neutron Activation Analysis can be used for determining organically bound fluorine.

December 1, 1976 - Industrial Hygiene begins medical examination of Chemolite personnel including those exposed to fluorochemicals. Examination includes blood, urine and enzyme analysis as well as a partial physical examination.

January, 1977 - J. E. Long arranges to supply Central Research Analytical with blood and liver samples from rats exposed to FC-43 vapors.

January 14, 1977 - Central Research Analytical is unable to detect FC-43 in the blood of rats exposed to FC-43, but finds that organically bound fluorine is present in the blood of exposed rats at seven times the level of a control.

January 15, 1977 - Industrial Hygiene completes medical examinations of Chemolite personnel. Those exposed to fluorochemicals show no medical abnormalities which can be attributed to fluorochemical exposure.

January 20, 1977 - Attempted analysis for organically bound fluorine in blood by General Activation Analysis using photon activation is unsuccessful.

January 27, 1977 - Central Research Analytical completes method for determining organically bound fluorine in whole blood. Blood samples from American Red Cross donors have "normal" plasma levels of organic fluoride.

February 3, 1977 - Central Research Analytical completes wrok on livers of rats exposed to FC-43. Gas chromotography shows FC-43 to be present at approximately 2ppm. Total organic fluorine level is 8.7 ppm in exposed rats as compared to 7.8 ppm in the control.

January 4, 1977 - Central Research Analytical completes work on blood and livers of rats fed 3000 and 10,000 ppm FC-807 for 30 days. Rats fed at 3000 ppm show an organic fluorine level of 75 ppm in blood while those fed at 10,000 ppm show a level of 125 ppm. (Control = 0.03 ppm.)

Analysis of the livers of the rats fed at the 10,000 ppm level show an organic fluorine level of 500 ppm (Control = 1 ppm).

February 14, 1977 - Central Research Analytical begins a concentrated effort to characterize $C_8F_{15}SO_3H$ derivatives in the 10 ppb range using the Gas Chromotography.

April 12, 1977 - J. D. LaZerte reviews status of organic fluorochemicals in blood with J. V. Erwin and P. H. Schertler of Personal Care Products. Decision made to determine amount of organically bound fluorine in blood of individuals who use Skaid Brand Repellents.

May 5, 1977 - Central Research Analytical completes analysis of blood from 3 employees at High Point, North Carolina. Organically bound fluorine level is on the high side of "normal".

June 9, 1977 - Central Research Analytical completes analysis of blood from three employees who use Skaid Brand Repellents. All blood samples contain organically bound fluorine at higher than "normal" levels. One sample is ten times "normal".

June 15, 1977 - J. D. LaZerte reviews status of organic fluorochemicals in blood with J. A. Muhlenpoh and R.W.H. Chang of Home Health Care Products. Muhlenpoh and Chang review plans for use of fluorochemicals in plague and carrier prevention.

July 6, 1977 - J. E. Long submits tentative schedule for chronic toxicity/carcinogenity study on FC-807 metabolite, FC-143 and Ethyl FOSE Alcohol.

July 29, 1977 - July issue of "Fluoride" contains special report on AAAS Fluoride Symposium held on February 25, 1977. Guy and Taves again report finding $C_7F_{15}CO_2H$ in pooled plasma and attribute its presence to industrial products such as SCOTCHGARD and SEPEL.

August 3, 1977 - Toxicology proposes four studies to be carried out with SCOTCHGARD and FLUORAD type products. Purpose of studies is to determine if these materials can enter the blood in significant quantities.

EXHIBIT M

FORM 2643-C PWO

CENTRAL ANALYTICAL LABORATORY

Report No. 6448

Date November 6, 1975

2

Subject: Fluorine-19 NMR of ten samples

Requestor: R.A. Prokop Dept. Name Comm. Chem. Proj. No. 91501624

Request No. C43831 Dated 9/22/75

Report:	
C43832	9/22/75
C43833	9/22/75
C43835	9/22/75
C43836	9/22/75
C43847	9/23/75
C43848	9/23/75
C40004	10/21/75

The following samples were submitted for analysis by fluorine NMR spectroscopy: $C_7F_{15}CO_2H$, $C_7F_{15}CO_2CH_3$, $C_8F_{17}SO_2NH_2$, $C_8F_{17}SO_3H$, Methyl FOSE, Ethyl FOSE, FC 95, FC 807, FC 808, and FC 824. Of the compounds submitted $C_8F_{17}SO_3H$ resembled most closely the fluorine NMR spectrum given at the Chicago A.C.S. meeting on August 26, 1975, by W.S. Guy.

Richard A. Newmark

RICHARD A. NEWMARK

/KD

c: J.E. Long 220-2E

EXHIBIT N

(4)

1975 Using a preconcentration method and NMR, Guy and Taves report presence of organic fluorine compounds in blood bank blood from around the country (average concentration about 0.03 ppm OF, which corresponds to about 45 ppb PFOS). Work was first reported at a conference (ACS?) and subsequently published in Biochemistry Involving Carbon-Fluorine Bonds, "Organic Fluorocompounds in Human Plasma: Prevalence and Characterization" in 1977. Guy and Taves hypothesis that POAA is the OF compound. This is never satisfactorily verified (e.g. by MS or by NMR).

1975 - probably in September; According to Richard Newmark, Dallas Zimmerman (3Mer) obtained copy of the NMR spectra at the meeting and spoke with CAL about the possibility of a 3M-produced contaminant.

1976 - by October, CAL has the ability to measure PFOS in sera using NMR (report #AR7230)

According to Richard Newmark, CAL team lead by Don Hagan and Jon Belisle (Richard Newmank - NMR) confirm that Guy and Taves' spectra reflects the presence of PFOS - not POAA - as the major OF compound.

According to Richard Newmark, Newmark generates 6 reports to this affect. **Can we locate any of these reports?**

According to Richard Newmark, Newmark analyzes samples he receives from Hagan that he believes are blood bank samples but does not know for sure. **Can we locate the notebook that references the identity of the sample in order to match it with microfisched spectra?**

1977 Unspecified fluorochemical (called "B") is identified in sera samples from High Point, NC. No conclusions are made about the specific compound, but data is attached. Analysis was by GC.

1977 Elevated R-F values are found in 3 3M employees who use Ensure and Skaid skin care products. Report suggest that there's not enough samples for specific compound id, yet GC data is attached indicating presence of "B".

1979 Guy and Taves author a paper speculating that POAA is the main OF in human blood.

According to Richard Newmark, Guy and Taves send this paper to CAL for review.

According to Richard Newmark, 3M lawyers urge CAL not to release the true identity (PFOS) of the OF compound.

Belise, Hagan, and Bunnelle publish internal reports measuring POAA and PFOS in worker blood using GC/ECD. (report # A73629)

Belilse and Hagan publish a paper suggesting the accuracy of Guy and Taves' conclusions about the identity of the OF found in blood. They propose a new analytic method (derivitization followed by GC/ECD analysis) for the analysis of POAA extracted from tissues and fluids. Recoveries of POAA are determined by spiking human sera free of POAA. **Doesn't the ability of these researchers to verify blank (with respect to POAA) sera prior to spiking indicate that Guy and Taves conclusion was inaccurate?** Analytical Biochemistry; "A Method for the Determination of Perfluorooctanoic Acid in Blood and Other Biological Samples".

Need copies of any papers Guy and Taves published from 1975 on.

Concentration of branched isomer in metabolised material confirmed (5/77, report #C46956) and (5/6/77, report #A64037)

EXHIBIT O

Interoffice Correspondence 3M

Subject: FM-3422
Rat Toxicity Study
Request A67820
April 6, 1978

cc: H.E. FREIER 201-1S
J.D. LAZERTE 236-1
B.W. NIPPOLDT 201-1S
R.A. PROKOP 236-3B
F.A. UBEL 220-2E
D.G. WEIBLEN 201-1S

TO: J.E. LONG 220-2E

FROM: JON BELISLE 201-1S

Samples of serum from the rats surviving the IRDC 90 day sub-acute rat toxicity study (reference 137-086) were received for determination of 3422. The analytical results were reported to you in my letter of March 13. In that letter, I speculated the presence of metabolized 3422 and suggested characterization for FC - 95.

Having developed a new sample handling technique appropriate for the above type of sample, the serum samples were analyzed for total fluoride content.

RAT	DOSE (ppm)	3422 in serum (ppm)①	TOTAL FLUORIDE IN SERUM (ppm)
Male	0	0	0.6
Male	100	< 0.1	100
Male	300	< 0.1	285
Female	0	0	--
Female	100	< 0.1	120
Female	300	< 0.3	335

① reported in March 13, 1978 letter.

FLUORINE - - - N M R

The serum samples were further characterized for fluorine by NMR (Richard Newmark). To the male serum - 0 ppm 3422 dose level was added FC-95 ($C_8F_{17}SO_3K$) and the sample prepared for NMR. The male serum - 300 ppm 3422 was prepared for NMR.

Exhibit
1166
State of Minnesota v. 3M Co.,
Court File No. 27-CV-10-28862

3MA10035579

RESULTS

The F/NMR spectrum of the 2 samples were identical (slight difference seen in branching). This means that the serum contains $C_8F_{17}SO_3^-$ that the rat has metabolized from 3422.

DISCUSSION

I would suggest that this study feeding FM-3422 ($C_8F_{17}SO_2N(C_2H_5)CH_2CH_2OH$) and a previous study with mice feeding FC-807 ($C_8F_{17}SO_2N(C_2H_5)CH_2CH_2OP-$) in which both serums were found (F/NMR) to contain $C_8F_{17}SO_3^-$ is a significant finding. It implies that any 3M product bearing the $C_8F_{17}SO_2NCH_2-$ group upon exposure to rats or mice would generate $C_8F_{17}SO_3^-$ which accumulates in the animal's blood and tissue (see liver analysis to be reported later).

The next step would be to extrapolate these findings to man per Guy and Taves research. Thus, I have suggested before and will state again the significance of characterizing those previous samples from 3M employees exposed to 3 M's skin protectants and carpet treatment products. If $C_8F_{17}SO_3^-$ is found in these persons blood, then the public health issue becomes simply one of frequency and type of exposure to 3 M products.

Jon Belisle

JB/jb

EXHIBIT P

COMMERCIAL CHEMICALS PRODUCT LINES

"FLUORINERTS"	ELECTRONIC LIQUIDS
"FLUOREL/KEL-F"	ELASTOMERIC RUBBER/PLASTICS
"LIGHT WATER"	AQUEOUS FILM FORMING FOAMS FIRE FIGHTING LIQUIDS
"SCOTCHGARD, SCOTCHBAN"	TEXTILE/PAPER TREATMENTS
"FLUORAD"	SURFACTANTS

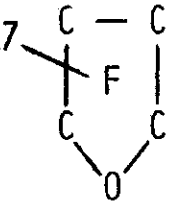
"FLUORINERT" ELECTRONIC LIQUIDS

PERFLUORINATED CARBON CHAINS

FC-88	PERFLUOROPENTANE	C_5F_{12}
FC-72	PERFLUOROHEXANE	C_6F_{14}

BLENDS OF PERFLUORINATED CYCLIC ETHERS AND PERFLUORINATED CARBON CHAIN

FC-75 $C_8F_{16}O$ (CYCLIC) + C_8F_{18}

FC-43 C_8F_{17}  + $C_{11}F_{24}$

USES; VAPOR PHASE SOLDERING, QUALITY CONTROL FOR ELECTRONIC PARTS, HEAT TRANSFER FLUIDS, COOLING OF ELECTRONIC COMPONENTS

TOXICITY OF "FLUORINERT" BRAND ELECTRONIC LIQUIDS

"FLUORINERT" LIQUIDS	ACUTE ORAL TOXICITY LD_{50} (Rat)	SKIN IRRITATION (Rabbit)	EYE IRRITATION (Rabbit)	ACUTE INHALATION LC_{50} (Rat)
FC-88	34.6 g/kg (oral)	Non-irritating	Non-irritating	No deaths when animals were exposed to 3300 mg/liter of air for 3 hours.
FC-78	10 g/kg (oral)	Non-irritating	Non-irritating	No deaths when animals were exposed to 340 mg/liter of air.
FC-72	34.6 g/kg (oral)	Minimally irritating	Non-irritating	No deaths when animals were exposed to near saturated vapors at room temperature for 2 hours.
FC-77	10 g/kg (oral)	Non-irritating	Non-irritating	No deaths when animals were exposed to 250 mg/liter for 1 hour at room temperature.
FC-104	23.1 g/kg (oral)	Non-irritating	Non-irritating	No Data
FC-75	34.6 g/kg (oral)	Non-irritating	Non-irritating	No deaths when animals were exposed to 750 mg/liter of air for 4 hours.
FC-40	34.6 g/kg (Intraperitoneal)	Non-irritating	Non-irritating	No deaths when animals were exposed to "near saturated" atmosphere at room temperature.
FC-43	10 g/kg (oral)	Non-irritating	Minimally irritating	No deaths when animals were exposed to 15 mg/liter for 4 hours at room temperature.
FC-48	34.6 g/kg (oral)	Non-irritating	Non-irritating	No deaths when animals were exposed to 90 mg/liter for 4 hours (300° F).
FC-70	10 g/kg (oral)	Non-irritating	Non-irritating	No deaths when animals were exposed to near saturated vapors at room temperature for 2 hours.

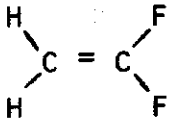
NOTE: All "FLUORINERT" Liquids, except FC-40, are classified as being practically non-toxic orally.
FC-40, is classified as being practically non-toxic intraperitoneally.

"FLUOREL/KEL-F" ELASTOMERS/THERMOPLASTIC

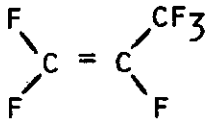
"FLUOREL" FLUOROELASTOMERS

COPOLYMERS OF

VINYLIDENE FLUORIDE



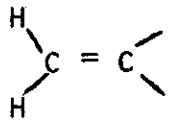
PERFLUOROPROPENE



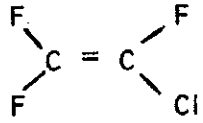
"KEL-F" THERMOPLASTICS

COPOLYMERS OF

VINYLIDENE FLUORIDE



CHLOROTRIFLUOROETHYLENE



USES: "FLUOREL": HEAT RESISTENT
O-RINGS, GASKETS, ETC.

"KEL-F": ACID/BASE RESISTANT THERMOPLASTIC LAQUERS, AND COATINGS FOR ALUMINUM, COPPER, STEEL & PLASTIC

TOXICITY SUMMARY OF

"FLUOREL" AND KEL-F" PRODUCTS

"FLUOREL" ELASTOMER

PRIMARY SKIN IRRITATION (RABBIT):	0.0/8.0	NON-IRRITATING
ACUTE INHALATION, THERMAL DECOMPOSITION:	10/10 DEATHS TOXIC PRODUCTS AT 260°C.	

"KEL-F" THERMOPLASTIC

ACUTE ORAL TOXICITY (RAT):	>5 GM/KG	PRACTICALLY NON-TOXIC
PRIMARY SKIN IRRITATION (RABBIT):	0.0/8.0	NON-IRRITATING
EYE IRRITATION (RABBIT):	15.5/110.0	MINIMALLY IRRITATING

"LIGHT WATER" AQUEOUS FILM FORMING FOAMS

TYPICAL FORMULATION: FC-203

2-3%	FLUOROCHEMICAL FOAMER*
1-2%	FLUOROCHEMICAL SURFACTANTS†
3%	HYDROCARBON SURFACTANTS
2%	SOAP
65%	WATER
25%	BUTYL CARBITOL

*FLUOROCHEMICAL FOAMER

$$\begin{array}{l} \quad\quad\quad\quad CH_2CHOHCH_2SO_3Na \\ \quad\quad\quad\quad | \\ C_6F_{13}SO_2NC_3H_6N(CH_3)_2C_2H_4OH \end{array}$$

†FLUOROCHEMICAL SURFACTANT

FC-95 - $C_8F_{17}SO'_3K$

USES: FIRE EXTINGUISHING LIQUIDS
ESPECIALLY GOOD FOR EXTINGUISHING SOLVENT AND FUEL FIRES

"LIGHT WATER" AFFF TOXICITY SUMMARY OF PRODUCTS

PRODUCT	PRIMARY SKIN IRRITATION	EYE IRRITATION	ACUTE ORAL TOXICITY
FC-201	0.0 NON-IRRITATING	12.7/110 MINIMALLY	LD_{50} 1.0-3.0 G/KG
FC-203A	0.0 NON-IRRITATING	11.0/110 MINIMALLY	LD_{50} >5 G/KG
FC-206	0.0 NON-IRRITATING	6.0*/110 MILDLY	LD_{50} >5 G/KG
FC-206A	0.1 MINIMALLY IRRITATING	17.1/110 MILDLY	LD_{50} > 5 G/KG
FC-206A DILUTED (6%)	0.0 NON-IRRITATING	0.0/110.0 NON-IRRITATING	
FC-600	.96 SLIGHTLY IRRITATING	9.3†/110.0 MODERATELY	LD_{50} >10 G/KG
FC-600 DILUTED (6%)	0.0 NON-IRRITATING	13.6/110.0 MINIMALLY	

*IRRITATING THROUGH 5 DAYS
†IRRITATING THROUGH 7 DAYS

"SCOTCHGARD/SCOTCHBAN" TEXTILE/PAPER TREATMENTS

"SCOTCHGARD" TEXTILE TREATMENTS

FLUOROCHEMICAL EMULSIONS

FC-234:

30% SOLIDS TERPOLYMER: METHYL FOSE ACRYLATE/
BUTYL ALCOHOL/
POLY MEG 2000 DIMETHYL ACRYLATE

IN: WATER
METHYL ISOBUTYL KETONE
ETHYLENE GLYCOL

FC-378:

30% SOLIDS 2 ET
FOSE†/TDI: URETHANE

IN: WATER
METHYL ISOBUTYL KETONE
ETHYLENE GLYCOL

*MEFOSE ACRYLATE: $C_8F_{17}SO_2N(CH_3)C_2H_4OC(=O)C(H)=CH_2$

†ETFOSE: $C_8F_{17}SO_2N(C_2H_3)C_2H_4OH$

USES: PROVIDES SOIL, STAIN AND WATER REPELLANCY TO A VARIETY OF FABRICS.

"SCOTCHBAN" PAPER TREATMENTS

FC-807: 33% SOLID SALT OF A FLUOROCHEMICAL PHOSPHATE ESTER

$$[C_8F_{17}SO_2N(C_2H_3)CH_2CH_2O]_2\overset{O}{\overset{\|}{P}}ONH_4$$

IN WATER AND ISOPROPYL ALCOHOL

USES: OIL AND STAIN RESISTANCE IN PAPER PRODUCTS. FC-807 IS CURRENTLY APPROVED BY THE U.S. FOOD AND DRUG ADMINISTRATION FOR USE IN FOOD PACKAGING.

"SCOTCHGARD/SCOTCHBAN" TOXICITY SUMMARY

SCOTCHGARD	PSI	GPS HSP*	EI	LD_{50} AOT	I.T.	AMES	SUB-CHRONIC DATA
FC-214	0.0	NEG.	0.0	>5G/KG	LOW HAZARD	N.K.	NO
FC-234	0.0	N.K.	47.31 MODERATELY	>5G/KG	N.K.	N.K.	NO
FC-380	0.0	NEG.	8.0 MINIMALLY	>10G/KG	LOW HAZARD	NEG.	NO
FC-388	0.0	NEG.	15.3 MILDLY	>5G/KG	LOW HAZARD	NEG.	NO
SCOTCHBAN							
FC-807	0.0	NEG.	<15.0 MINIMALLY	>15.4G/KG	N.K.	NEG.	YES
FC-808	<1.6 MINI-MALLY	N.K.	4.0 MINIMALLY	>15G/KG	N.K.	NEG.	YES

*GUINEA PIG SENSITIZATION HUMAN SKIN PATCH STUDY

"FLUORAD" SURFACTANTS

PERFLUOROOCTYL SULFONIC ACID DERIVATIVES

FC-95: $C_8F_{17}SO_3^-K^+$

FC-99: $C_8F_{17}SO_3^-H_2N^+(CH_2CH_2OH)_2$

FC-128: $C_8F_{17}SO_2NH(CH_2)_3N(CH_3)_3^+I'$

CARBOXYLIC ACID DERIVATIVE

FC-143: $C_7F_{13}COO'NH_4^+$

USES: REDUCE SURFACE TENSION OF AQUEOUS AND NON-AQUEOUS SYSTEMS: ETCHING BATHS, SPECIALTY INKS, FLOOR POLISH EMULSIONS AND PHOTOGRAPHIC SOLUTIONS, TEFLON EMULSIFIER.

FLUORAD SURFACTANT TOXICITY SUMMARY

	P.S.I.	E.I.	A.O.T.	I.T.	AMES	SUBCHRONIC
FC-95	0.0 NON-IRRITATING	9.3 MILDLY	LD_{50} 251MG/KG	LC_{50} 5.2MG/L	NEGATIVE	YES
FC-99	0.1 MINIMALLY	15.2 MILDLY	LD_{50} > 5G/KG	N.K.	N.K.	YES - 14 DAY SUBACUTE
FC-128	0.9 SLIGHTLY	5.8 MINIMALLY	LD_{50} 1250MG/KG SLIGHTLY	LC_{50} 22.22-6623 MG/L	N.K.	No
FC-134	0.5 MINIMALLY	7.0 MINIMALLY	LD_{50} 500MG/KG MODERATELY TOXIC	LD_{50} >5.1MG/L RESPIRATORY IRRITANT	N.K.	No
FC-143	0.0 NON-IRRITATING	14.0 MINIMALLY	LD_{50} 540MG/KG	LC_{50} >18.6MG/L RESPIRATORY IRRITANT	NEGATIVE	YES

MANUFACTURE OF FC-143
CARBOXYLIC ACID DERIVATIVE

100% PERFLUOROOCTANOIC ACID

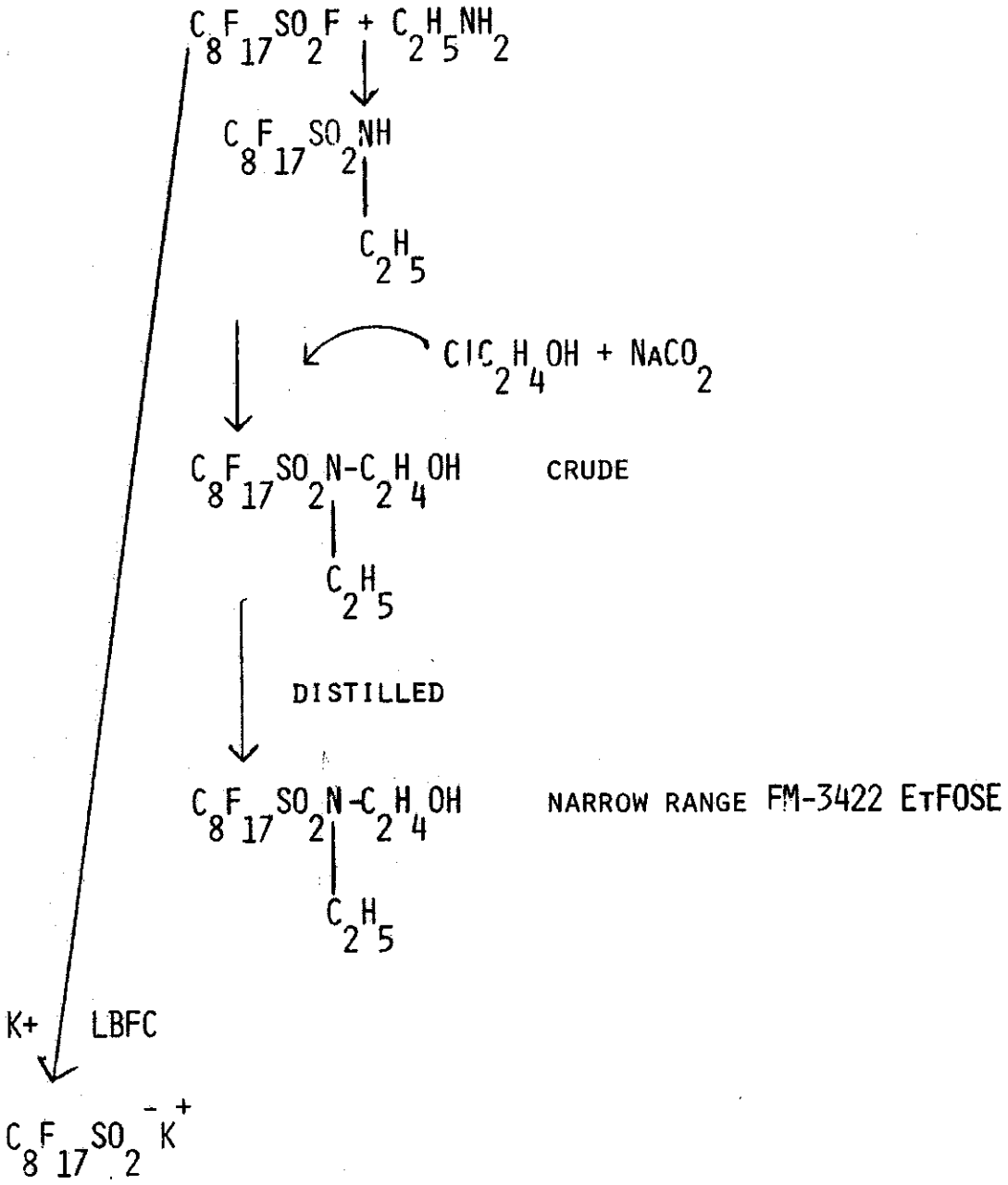
$C_7F_{15}CO_2H + NH_3$

↓

$C_7F_{15}CO_2NH_4$

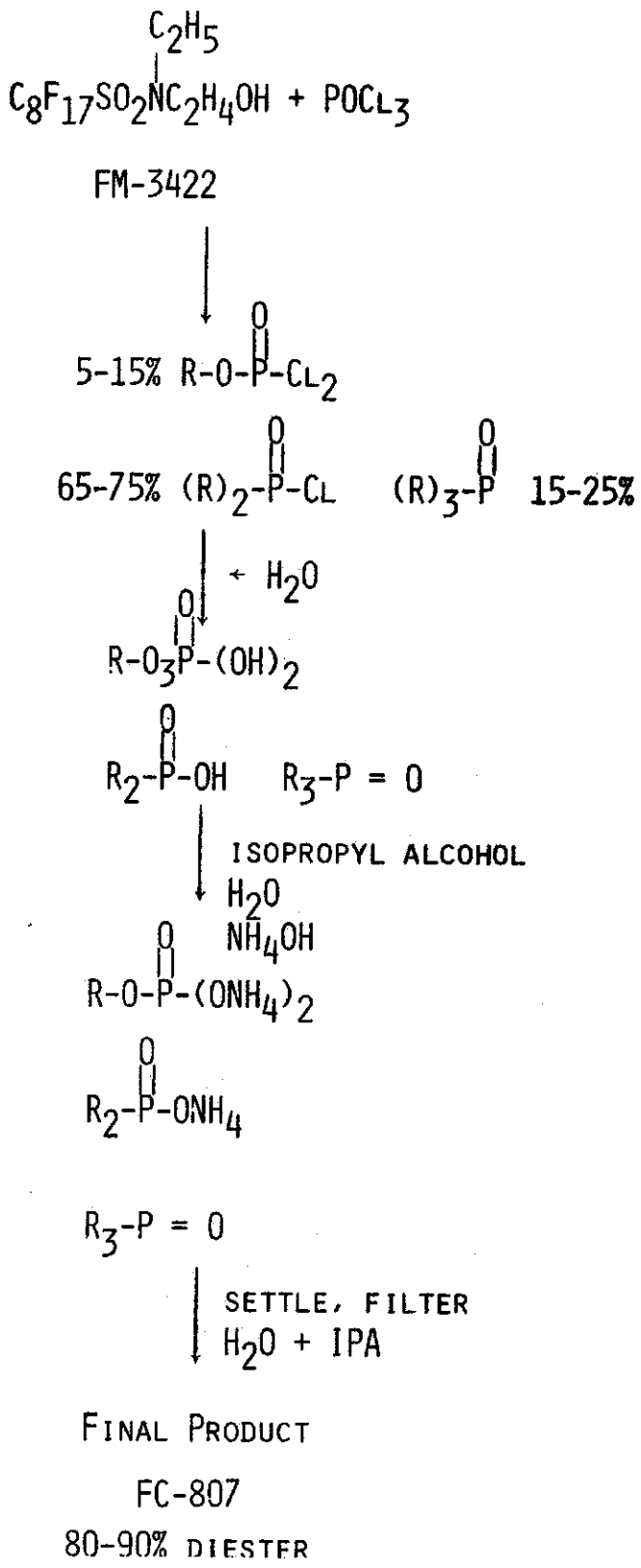
FC-143

MANUFACTURE OF EtFOSE ALCOHOL AND FC-95



FC-95

MANUFACTURE OF FC-807



PRODUCT LINES BASED ON

PERFLUOROOCTYL SULFONYL ACID DERIVATIVES

"FLUORAD" Surfactants	27
"SCOTCHGARD" Fabric and Textile Treatments	58
"SCOTCHBAN" Paper Treatments	10
"LIGHTWATER" Aqueous Film Forming Foams (C_6 Sulfonyl Acid Derivatives)	10

CHRONOLOGY OF EVENTS LEADING TO THE INITIATION OF 90 DAY STUDIES

1971 D. R. TAVES REPORTS ORGANIC AND INORGANIC FORMS OF FLUORINE IN HUMAN SERUM.

1975 TAVES PRESENTS ^{19}F NMR SPECTRA DATA TO 3M

CRL IDENTIFIES ^{19}F NMR SPECTRUM AS $C_8F_{17}SO_3H$ OR ITS SALTS

1976 ANALYTICAL METHOD FOR LOW LEVEL DETECTION OF R^+F' DEVELOPED

3M WORKERS SAMPLED

CARBOXYLIC ACID IDENTIFIED IN 3M EMPLOYEE $C_7F_{15}COO'H^+$

1977 ANALYSIS OF R^+F' LEVELS IN SHORT-TERM ANIMAL STUDIES BEGINS

90 DAY STUDIES ON FC-143, FC-95 AND FM-3422 INITIATED

90 DAY ORAL STUDY, FC-143, RAT

DOSE	DEATHS	PHARMACOTOXIC SIGNS AND PATHOLOGY
10PPM	0/10	NO REMARKABLE PATHOLOGY
30PPM	0/10	IN MALES: INCREASED LIVER AND KIDNEY WEIGHTS
100PPM	1/10*	IN MALES: INCREASED KIDNEY WEIGHTS
300PPM	1/10*	IN MALES: INCREASED LIVER AND KIDNEY WEIGHTS, SOME LIVER PATHOLOGY
1000PPM	0/10	IN MALES: LIVER DISCOLORATION WITH SLIGHT HYPERTROPHY OF THE HEPATOCYTES. BLOOD EFFECTS.

*RATS DIED AFTER BLOOD COLLECTION.

90 DAY ORAL STUDY, FC-143, RHESUS MONKEY

DOSE	DEATHS	PHARMACOTOXIC SIGNS, PATHOLOGY
3 MG/KG/DAY	0/4	SOFT STOOL, OCCASIONAL EMESIS. INCREASED PLATELET COUNT
10 MG/KG/DAY	0/4	ANOREXIA, PALE FACE & GUMS INCREASE IN ACTIVATED PARTIAL PROTHROMBIN TIME (APPT)
30 MG/KG/DAY	3/4	SAME AS ABOVE, SWOLLEN FACE AND EYES DECREASED ACTIVITY PROSTRATION DEATH 7-12 WEEKS HIGHLY INCREASED APPT. PATHOLOGY REVEALED HEMOPOETIC EFFECT
100 MG/KG/DAY	4/4	SAME AS ABOVE. DEATH 2-5 WEEKS.

90 DAY ORAL STUDIES FC-95, RAT

DOSE	DEATH	PHARMACOTOXIC SIGNS & PATHOLOGY
30PPM	0/10	NO SIGNIFICANT PHARMACOTOXIC SIGNS. PATHOLOGY REVEALED SOME MINOR LIVER EFFECTS.
100PPM	5/10	INCREASED SENSITIVITY TO EXTERNAL STIMULI. CONSULSIONS, CNS EFFECTS. LIVER NECROSIS, GI TRACT HEMORRHAGING. HEMATOPOETIC EFFECT: THYMUS, SPLEEN AND MESENTARY LYMPH NODES.
300PPM	10/10	INCREASED SENSITIVITY TO EXTERNAL STIMULI. EMACIATION, CONVULSIONS. HUNCHED BACK. PATHOLOGY SAME AS 100PPM
1000PPM	10/10	SAME AS ABOVE.
3000PPM	10/10	SAME AS ABOVE, REDUCED MOTOR ACTIVITY.

90 DAY ORAL RHESUS MONKEY STUDY FC-95 I.

DOSE	DEATH	PHARMACOTOXIC SIGNS & PATHOLOGY
10MG/KG/DAY	4/4 (11-20 DAY)	ANOREXIA, SLIGHT TO SEVERE DECREASES IN ACTIVITY, EMESIS. BODY TREMORS, TWITCHING, CONVULSIONS AND PROSTRATION. LIVER DISCOLORATION NOTED BUT NO HISTOPATHOLOGICAL EVIDENCE OF DAMAGE.
30MG/KG/DAY	4/4 (7-10 DAY)	SAME AS ABOVE.
100MG/KG/DAY	4/4 (3-5 DAY)	SAME AS ABOVE.
300MG/KG/DAY	4/4 (2-4 DAY)	SAME AS ABOVE.

II. 90 DAY ORAL RHESUS MONKEY TOXICITY STUDY

DOSE	DEATH	PHARMACOTOXIC SIGNS & PATHOLOGY
0.5MG/KG.DAY	0/4	GI TRACT TOXICITY. LIPID DEPLETION OF ADRENALS, ATROPHY OF PANCREATIC EXOCRINE CELLS AND SEROUS ALVEOLAR CELLS OF THE SALIVARY GLANDS.
1.5MG/KG/DAY	0/4	GI TRACT TOXICITY. SAME AS ABOVE.
4.5MG/KG/DAY	4/4 (5-7 WEEK)	GI TRACT TOXICITY. SEVERE RIGIDITY, CONVULSIONS, BODY TREMORS, PROSTRATION, AND WEIGHT LOSS.

90 DAY RAT FM 3422

DOSE	DEATH	PHARMACOTOXIC SIGNS & PATHOLOGY
30PPM	0/10	
100PPM	0/10	IN MALES: INCREASED LIVER AND KIDNEY WEIGHT.
300PPM	2/10*	INCREASED LIVER AND KIDNEY WEIGHTS. LIVER AND KIDNEY DISCOLORATION. LIVER: HYPERTROPHY AND NECROSIS. KIDNEY: TUBULAR NEPHROSIS.
1000PPM	10/10	INCREASED SENSITIVITY TO EXTERNAL STIMULI. EMACIATION, HUNCHED BACK, CONVULSIONS. SAME AS ABOVE.
3000PPM	10/10	SAME AS ABOVE.
10,000PPM	10/10	SAME AS ABOVE.

*DIED AFTER BLOOD COLLECTION.

90 DAY ORAL RHESUS MONKEY STUDY FM 3422

DOSE	DEATH	PHARMACOTOXIC SIGNS & PATHOLOGY
1MG/KG/DAY	0/4	DIARRHEA, NO REMARKABLE GROSS OR HISTOPATHOLOGY.
3MG/KG/DAY	0/4	DIARRHEA, NO REMARKABLE GROSS OR HISTOPATHOLOGY.
10MG/KG/DAY	0/4	DIARRHEA, IN MALES INCREASE LIVER WEIGHT. NO HISTOPATHOLOGY.
30MG/KG/DAY	1/4	BLOODY MUCOUS IN STOOL, EMESIS, DIARRHEA. IN MALES INCREASED LIVER WEIGHT. LIPID DEPLETION OF ADRENALS. MODERATE ATROPHY OF PANCREATIC EXOCRINE CELLS.

COMPARISON OF THE SUBACUTE DATA

1. FC-95 IS THE MOST TOXIC OF THE THREE COMPOUNDS. FOLLOWED BY FM-3422 AND FC-143

2. IN GENERAL MALE RATS WERE MORE SENSITIVE TO THE COMPOUNDS THAN FEMALE RATS.

3. NO APPARENT SEX DIFFERENCES WERE NOTED WITH MONKEYS.

4. THE TARGET ORGANS IN RATS WERE THE LIVER, KIDNEY, CENTRAL NERVOUS SYSTEM (CNS), GI TRACT, AND RETICULOENDOTHELIAL SYSTEM. IN MONKEYS THE LIVER AND KIDNEY EFFECTS WERE ABSENT. GI TRACT DISTURBANCES, RETICULOENDOTHELIAL SYSTEM AND CNS TOXICITY WERE EVIDENT.

5. MONKEYS WERE GENERALLY MORE SENSITIVE TO THE FLUOROCHEMICAL TOXICITY THAN RATS. 10 PPM IN DIET ~/MG/KG/DAY

	RAT	MONKEY
FC-143	0/10 @ 100 MG/KG/DAY	3/4 30 MG/KG/DAY
FC-95	5/10 @ 10 MG/KG/DAY	4/4 4.5 MG/KG/DAY
FM-3422	10/10 @ 100 MG/KG/DAY	1/4 30 MG/KG/DAY

METABOLISM STUDIES

POSITION OF CARBON-14 LABEL

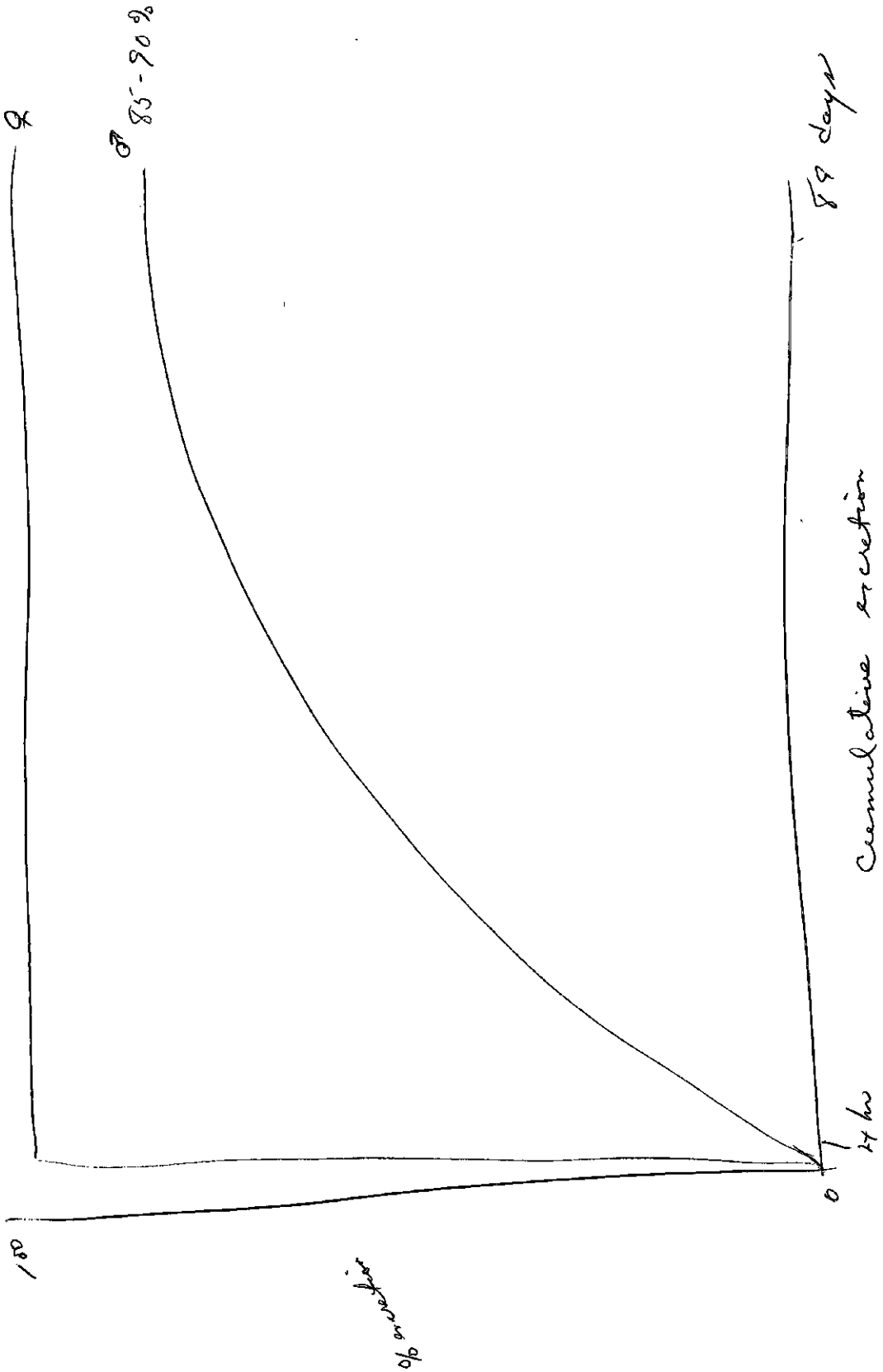
FC-143 $C_7F_{15}{}^*COO^-NH_4^+$

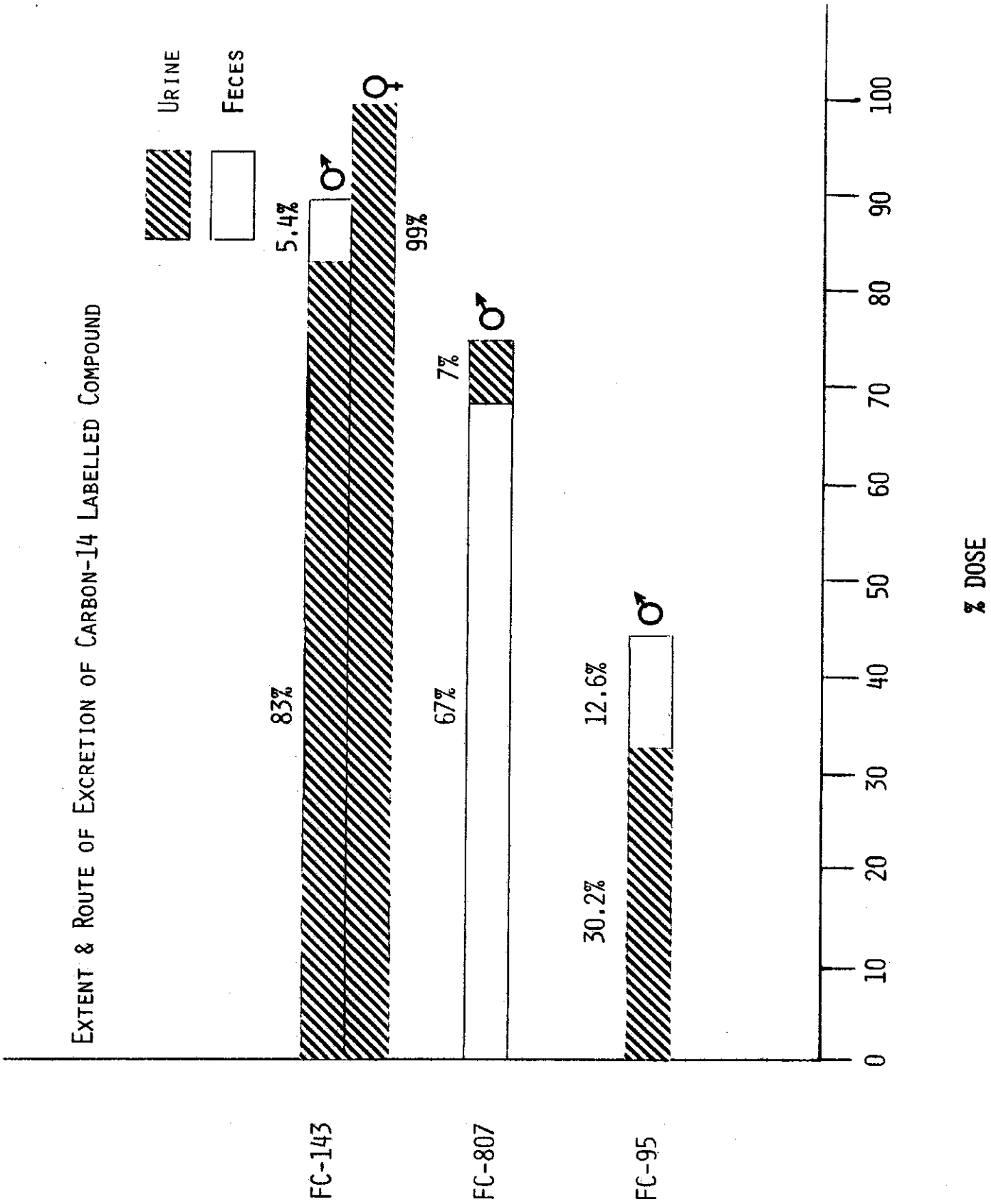
FC-95 $C_7F_{15}{}^*CF_2SO_3^-K^+$

FC-807 $(C_7F_{15}{}^*CF_2SO_2N(C_2H_5)CH_2CH_2O)_2PO_2^-NH_4^+$

ORAL ABSORPTION OF ^{14}C LABELLED FLUOROCHEMICALS

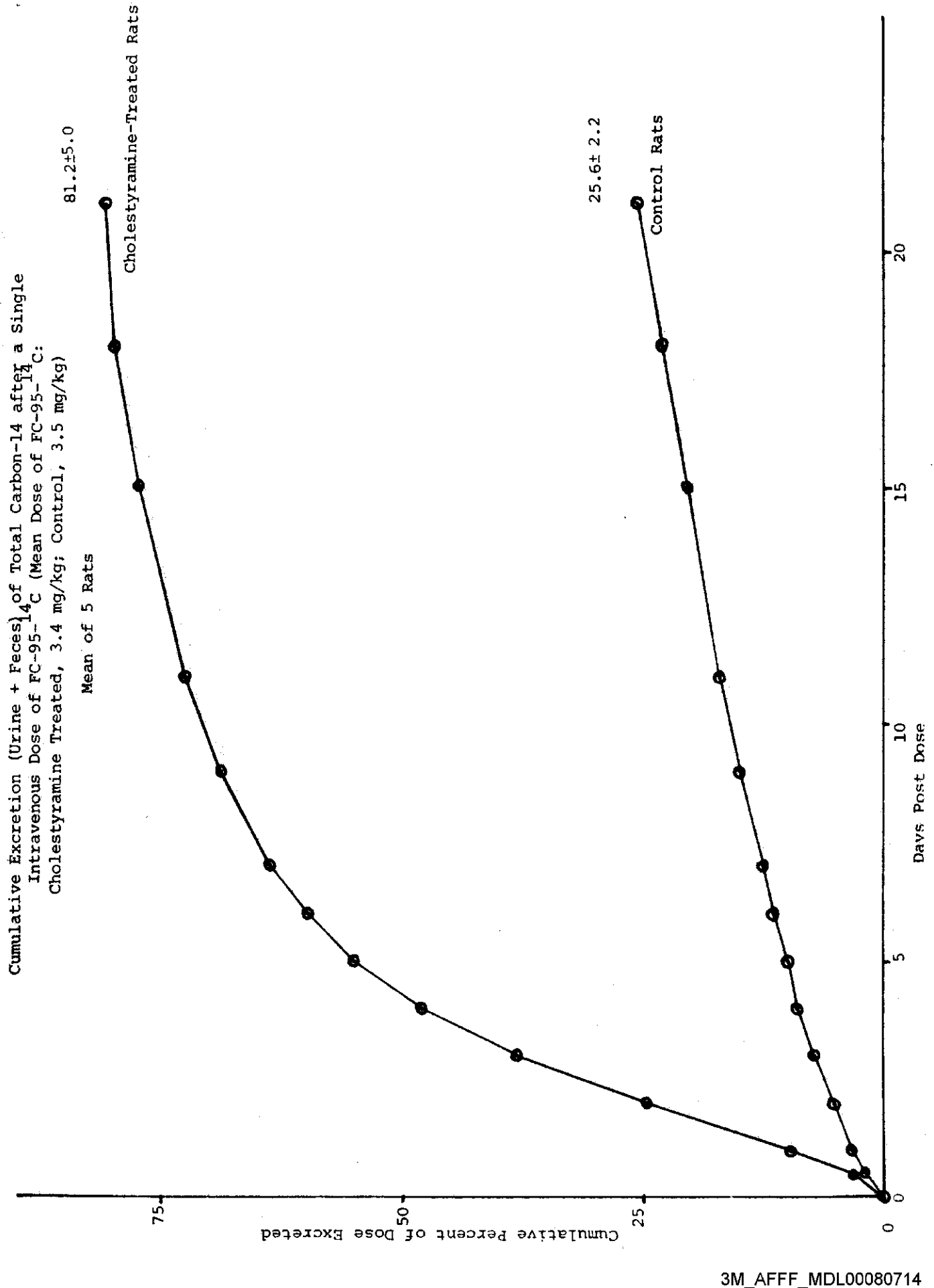
FC-807	≤ 5%
FC-95	~95%
FC-143	~93%


%
85-90 %
59 days
Cumulative excretion
24 hr
0
100
% excretion


EXTENT & ROUTE OF EXCRETION OF CARBON-14 LABELLED COMPOUND
URINE
FECES
♂
♀
5.4%
99%
83%
7%
67%
12.6%
30.2%
FC-143
FC-807
FC-95
0
10
20
30
40
50
60
70
80
90
100
% DOSE

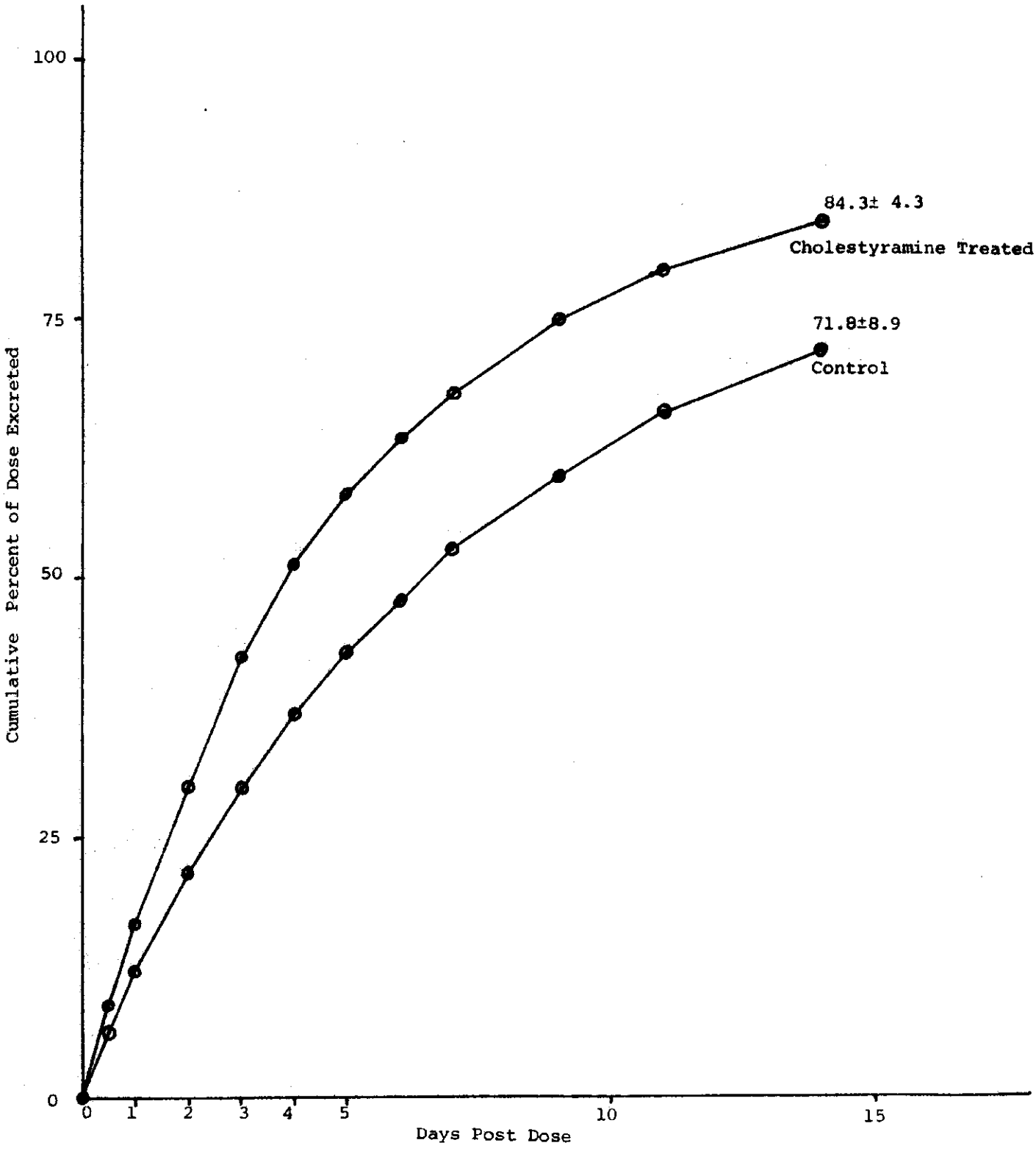
TISSUE DISTRIBUTION OF ^{14}C LABELLED FLUOROCHEMICALS

		FC-143 % OF DOSE	FC-95 µG/G TISSUE	FC-807 µG/G TISSUE
1.	LIVER	2.5%	20.6	31
2.	SPLEEN	<0.5%	0.5	277
3.	PLASMA	1.1%	2.2	1.5
4.	BONE MARROW	<0.5%	0.5	73
5.	KIDNEY	<0.5%	1.1	1.7
6.	ADRENALS	<0.5%	<0.5	3.9
7.	RBC	<0.5%	N.R.	1.2
8.	EYE	<0.5%	0.5	0.2
9.	LUNG	<0.5%	1.1	N.R.


Cumulative Excretion (Urine + Feces) of Total Carbon-14 after a Single Intravenous Dose of FC-95-^{14}C (Mean Dose of FC-95-^{14}C: Cholestyramine Treated, 3.4 mg/kg; Control, 3.5 mg/kg)
Mean of 5 Rats
Cholestyramine-Treated Rats
81.2±5.0
Control Rats
25.6± 2.2
Cumulative Percent of Dose Excreted
0
25
50
75
0
5
10
15
20
Days Post Dose

Cumulative Excretion (Urine + Feces) of Total Carbon-14 After a Single Intravenous Dose of FC-143-^{14}C (Mean Dose of FC-143-^{14}C: Cholestyramine Treated, 13.3 mg/kg; Control, 13.5 mg/kg)

Mean of 5 Rats



WHAT HAVE THE ANIMAL STUDIES ON FC-95 AND FC-143 SHOWN?

1. FC-95 APPEARS TO BE THE MOST TOXIC OF THE COMPOUNDS EXAMINED.
2. FC-143 APPEARS TO BE THE LEAST TOXIC.
3. BOTH ARE WELL ABSORBED FROM THE GI TRACT.
4. FC-143 APPEARS TO BE QUICKLY ELIMINATED.
5. FC-95 IS SLOWLY ELIMINATED.
6. BOTH COMPOUNDS APPEAR TO HAVE EFFECTS ON THE HEMOPOETIC SYSTEM AND GI TRACT. THE LIVER AND KIDNEY EFFECTS PRESENT IN RODENTS ARE ABSENT IN PRIMATES.
7. MALE RATS ARE MORE SENSITIVE THAN FEMALE RATS.
8. PRIMATES ARE MORE SENSITIVE THAN RATS.
9. CHOLESTYRAMINE ADMINISTRATION MAY BE A POSSIBLE WAY TO ELIMINATE FC IN THE BLOOD OF WORKERS.

Other Animal Toxicity Studies in Progress

1. Surfactant skin absorption study: contrasting solid and liquid forms of the surfactants

2. Teratology study on FC-95 and FM-3422

3. Further investigation platelet aggregation and APTT.

EXHIBIT Q

FC-95, FC-143 and FM-3422 - 90 Day Subacute Toxicity Studies Conducted at IRDC - Review of Final Reports and Summary

OVERALL SUMMARY AND RECOMMENDATIONS

FC-95 was the most toxic of the three compounds studied and certainly more toxic than anticipated. It produced mortalities in rats at a dietary dose of 100 ppm (≈10 mg/kg/day) and in monkeys at an oral dose of 4.5 mg/kg/day. The primary target organs in rats were the liver, hematopoietic tissues and upper gastrointestinal tract and in monkeys, the gastrointestinal tract although no pathological lesions were reported. FC-143 appeared to be the least toxic of the three compounds studied and produced no mortalities in rats at dietary doses as high as 1000 ppm (≈ 100 mg/kg/day). However, definite evidence of liver toxicity was seen at the high dose. In monkeys, FC-143 caused deaths at oral doses of 100 (4/4) and 30 (3/4) mg/kg/day and evidence of effects on hematopoietic tissue at these lethal doses. Like FC-95 and FM-3422, FC-143 also produced clinical evidence of gastrointestinal toxicity but no associated pathological lesions. FM-3422 caused deaths in rats at dietary doses of 1000, 3000 and 10,000 ppm (≈ 100, 300 and 1000 mg/kg/day respectively) and in monkeys (1/4) at an oral dose of 30 mg/kg/day. The primary target organ in rats appeared to be the liver although there was some gross evidence of kidney and upper gastrointestinal tract involvement as well. In monkeys, the gastrointestinal tract was affected clinically, but there were no pathological lesions reported at necropsy.

The goals of conducting these 90 day subacute toxicity studies of 1) defining doses for chronic experiments and 2) obtaining general toxicological information on the three compounds appear to have been met. However, several questions surfaced that deserve further clarification. The apparent effect of FC-95 on the liver and hematopoietic system of rats should be studied for reversibility. The question of clinical gastroinestinal signs in monkeys with all three compounds without any gross or microscopic pathology is certainly perplexing, but may not be worth further pursuit since the oral route is not a likely one for man. If another study with FC-143 is conducted to help define the gastrointestinal and hematopoietic effects, the dog should be considered. Since the most likely route of exposure in plant workers is by inhalation, an inhalation study, probably with FM-3422, could be useful in evaluating any effects via pulmonary exposure. Marv Case and Bill McCormick are preparing protocols for follow-up to the toxicity questions mentioned.

Because of the apparent persistence of these fluorochemicals in the body, the most important question remains possible long term effects. Although lifetime rodent studies have limitations in predicting chronic effects (carcinogenesis) for man, they are still considered the most reliable indicators available. Unless there are adequate data through human epidemiological evaluations that can reasonably assure relative safety of these compounds following long term exposure, lifetime rodent studies should be undertaken as soon as possible. It may be possible to limit the number of compounds evaluated in lifetime rodent studies to one or two if metabolic data can be used to establish a common linkage between compounds.

INDIVIDUAL SUMMARIES

FC-95

Study No. 137-085 - 90 Day Subacute Rat Toxicity Study

Dietary levels of FC-95 were administered to five male and five female rats/level at 30, 100, 300, 1000 and 3000 ppm which approximates 3, 10, 30, 100 and 300 mg/kg/day respectively. All rats at the three highest doses and 5/10 at 100 ppm died during the study. Predominant signs observed included emaciation, convulsions, altered posture, ocular, oral and anal discharges, hyperreactivity and reduced motor activity. Mortalities occurred in a sequence related to dose, with earlier deaths seen at the highest level. There was compound and dose related evidence of reduced body weight gain and food consumption with actual weight loss at higher lethal doses. At 30 ppm only slight body weight effects were present. The most notable clinical pathology effects were observed at 100 ppm (values not obtained at higher levels) and consisted of enzyme level increases suggestive of possible liver toxicity and decreased erythrocytic values (principally hemoglobin and hematocrit with slight lowering of red cell counts) indicating an anemia. Pathologically, the most consistent and apparent compound related effect involved liver, hematopoietic tissues (thymus, bone marrow, spleen, mesenteric lymph nodes), gastrointestinal tract, muscle and skin.

In summary, FC-95 was relatively toxic to rats causing mortalities at dietary doses as low as 100 ppm ($\approx$ 10 mg/kg/day). Primary target organs appeared to be liver, hematopoietic tissues, stomach and small intestine with some indication of a compound related effect in muscle and skin.

Study No. 137-087 - 90 Day Subacute Rhesus Monkey Toxicity Study

FC-95 was administered by gastric intubation as an aqueous suspension to two male and two female rhesus monkeys/level at doses of 10, 30, 100 and 300 mg/kg/day for up to 20 days. Because of unexpected early mortalities in all monkeys at all levels (days 2-4 at 300, 3-5 at 100, 7-10 at 30 and 11-20 at 10 mg/kg/day), the study was inconclusive. Prominent signs observed consisted of anorexia, decreased activity, emesis with some diarrhea, body stiffening, general body trembling and twitching, weakness, convulsions and prostration. No clinical pathology work was done because of the short study duration. The only pathological lesions reported consisted of gross yellowish-brown liver coloration at 100 and 300 mg/kg/day but no histopathologic basis for this finding was observed.

In summary, FC-95 proved to be considerably more toxic to monkeys than anticipated resulting in early deaths preceded by gastroinestinal and central nervous system signs. Although far from definitive, this study suggested the gastrointestinal tract and possibly liver as target organs.

Study No. 137-092 - 90 Day Subacute Rhesus Monkey Toxicity Study (Second Study)

Since all monkeys died in the first FC-95 study (137-087), a second experiment was conducted using oral gavage doses of 0.5, 1.5 and 4.5 mg/kg/day administered to two male and two female monkeys/dose. The controls were the same monkeys used in the first FC-95 experiment. All 4.5 mg/kg monkeys exhibited signs of gastrointestinal tract toxicity (anorexia, emesis, black stools, dehydration) starting on day 1 or 2 of the study, and all died or were sacrificed in extremis between weeks 5-7. Prior to death, these monkeys showed marked or severe rigidity, convulsions, general body tremors, prostration and loss of body weight. The monkeys at lower doses all survived, but evidence of gastrointestinal toxicity was observed both at 1.5 and 0.5 mg/kg/day. The only consistent clinical pathology observation reported was decreased alkaline phosphatase values at all three doses. No gross pathological lesions considered compound related were observed and the only microscopic pathology of apparent compound relationship consisted of lipid depletion in the adrenals, atrophy of pancreatic exocrine cells and atrophy of the serous alveolar cells of the submandibular salivary glands in high dose monkeys. These latter effects may be due to general debilitation of the animals.

In summary, FC-95 was relatively toxic to rhesus monkeys producing deaths at doses as low as 4.5 mg/kg/day in 5-7 weeks. The apparent target organ was the upper gastrointestinal tract although no pathological lesions were reported even at the high dose.

FC-143

Study No. 137-089 - 90 Day Subacute Rat Toxicity Study

Dietary levels of FC-143 administered to five male and five female rats/level were 10, 30, 100, 300 and 1000 ppm which approximates 1, 3, 10, 30 and 100 mg/kg/day respectively. Clinically, the only effect observed was slightly decreased body weight gains at 300 and 1000 ppm. Clinical pathology abnormalities reported in high dose male rats only included slightly lowered erythrocyte counts, and elevated BUN and alkaline phosphatase values. There were several other variations from control groups in the clinical pathology parameters including fairly consistent lowering of calcium levels at all doses, but these were not considered abnormal based on the contract laboratory's comparison to background control data. Pathological abnormalities were confined to the liver and included gross enlargement and discoloration at 1000 ppm, increased organ weights at 1000 and 300 ppm and several microscopic changes at 1000 ppm.

In summary, FC-143 was well tolerated in rats at doses up to and including 300 ppm (≈ 30 mg/kg/day). There was obvious liver toxicity at 1000 ppm (≈ 100 mg/kg/day), but no mortalities occurred.

Study No. 137-090 - 90 Day Subacute Rhesus Monkey Toxicity Study

FC-143, suspended in 0.5% methocel, was administered by gastric intubation to two male and two female rhesus monkeys/dose at 3, 10, 30 and 100 mg/kg/day. All high dose monkeys died during weeks 2-5 and 3/4 30 mg/kg monkeys died during the last half of the study. All monkeys that died showed clinical evidence of gastrointestinal toxicity (anorexia, emesis, dark stools), but there were no associated pathological lesions found at necropsy. No mortalities occurred and only occasional signs of gastrointestinal effects were reported at the two lower doses except for one 10 mg/kg monkey that had signs of gastrointestinal toxicity for several days late in the study. There were a few abnormalities reported in clinical pathology parameters, but no consistent pattern was observed. Gross and microscopic pathological lesions were restricted to the two highest dose levels and consisted of lipid depletion in adrenals, hypocellularity of bone marrow and atrophy of lymphoid follicles of the spleen and lymph nodes.

In summary, FC-143 was less toxic than FC-95 in rhesus monkeys but, at lethal doses (100 and 30 mg/kg/day), evidence of effects on hematopoietic tissue was seen. Like FC-95, the gastrointestinal tract also appeared to be a target organ although this was not confirmed on histopathological examination.

FM-3422

Study No. 137-086 - 90 Day Subacute Rat Toxicity Study

FM-3422 was administered in the diet to five male and five female rats/level at 30, 100, 300, 1000, 3000 and 10,000 ppm which corresponds to approximately 3, 10, 30, 100, 300 and 1000 mg/kg/day respectively. All rats at the 1000, 3000 and 10,000 ppm levels died between days 9 and 29. Prominent signs observed in these rats included emaciation, altered posture, convulsions, reduced motor activity and/or increased sensitivity. At 30 ppm, a slight decrease in body weight gain in females was the only clinical effect reported. There were also some slight abnormalities in serum enzyme levels, but no pronounced trends. Likewise, minimal effects were seen at 100 ppm. At 300 ppm there appeared to be increased compound related clinical signs, decreased body weight gain and food consumption, depressed hematological parameters and several alterations in clinical chemistry values. Pathologically, the liver was grossly enlarged with accentuated lobulation and discoloration with the 300 ppm group being more severely effected than the 1000 or 3000 ppm rats. This apparent reversed order of toxicity related to dose could be due to the early mortalities of the high dose rats and, therefore, a short dosing duration. The liver abnormalities seen grossly were associated with increased liver weights and microscopic lesions. Some kidney discoloration and evidence of stomach irritation were also observed grossly at 300 ppm.

In summary, FM-3422 was lethal at doses of 1000, 3000 and 10,000 ppm which is approximately 100, 300 and 1000 mg/kg/day respectively. The liver appeared to be the primary target organ, but there was gross pathological evidence of possible kidney and stomach involvement at the 300 ppm level also.

Study 137-088 - 90 Day Subacute Rhesus Monkey Toxicity Study

FM-3422, suspended in propylene glycol, was administered by gavage to two male and two female monkeys/level using doses of 1, 3, 10 or 30 mg/kg/day. The vehicle appeared to cause anorexia early in the study necessitating volume reduction from 5 to 2 ml/kg. The only mortality occurred in one high dose monkey the last week of dosing. Gastrointestinal signs consisting of emesis, diarrhea and black stools with mucus or bloody mucus were seen in most monkeys from most groups. There were no clinical pathology observations that appeared to be significant compound effects. Pathological lesions reported included lipid depletion of adrenals and atrophy of pancreatic exocrine glands at 30 mg/kg only.

In summary, FM-3422 caused mortality at 30 mg/kg in 1/4 monkeys and appeared to primarily effect the gastrointestinal tract although there was no supporting microscopic evidence.

R. N. Nolan 3/20/79
Date

RAN/lmr

EXHIBIT R

Interoffice Correspondence 3M

Subject:

May 26, 1978

RECEIVED

MAY 30 1978

T. J. SCHEUERMAN

CONFIDENTIAL

TO: L. W. LEHR
J. M. PITBLADO
F. A. UBEL
T. A. SCHEUERMANN

FROM: R. M. ADAMS

This will confirm arrangements made for a meeting at 9:30 a.m. July 12 in Mr. Lehr's conference room, 220-14W, on the subject of fluorochemicals in blood. An hour should be sufficient for this review.

posted

RMA:va

13 078552

EXHIBIT S

Interoffice Correspondence 3M COMPANY

Subject

June 1, 1978

irc: T. L. Kerley-Riker Res.-218-1
J. D. La Zerte-Comm'l.Chem.Div.-236-1L

CONFIDENTIAL

TO: R. J. DAVIS - GENERAL COUNSEL - 220-12E
J. E. LONG - MEDICAL DEPARTMENT - 220 - 2E
R. A. NELSON - RIKER RESEARCH - 21[illegible]
J. A. PENDERGRASS - MEDICAL D[illegible]
R. A. PROKOP - COMMERCIAL CH[illegible]
T. J. SCHEUERMAN - GENERAL
F. A. UBEL - MEDICAL DEPAJ

FROM: L. C. KROGH - COMMERCIAL CHE[illegible]ION - 223 - 6SE

This meeting is being called to consider the use of an outside consultant to review our results to date in the fluorochemicals in blood program.

Mr. Lehr has specifically requested that an outside consultant review our results and render an independent opinion as to whether we are correct in our assumption that we do not have a reportable situation under Section 8(e) of the Toxic Substances Act.

The meeting will begin at 1:00 p.m. and will be held in Conference Room 196-A, Building 223 - 6N, on Monday, June 5, 1978.

LCK:jmb

RECEIVED
JUN -5 1978
R. J. DAVIS

Dedicated to a better way

13 078091

3M_AFFF_MDL02342749

EXHIBIT T

3M COMPANY

GENERAL OFFICES • 3M CENTER • SAINT PAUL, MINNESOTA 55101 • TEL. (612) 733-1110

Commercial Chemicals Division

February 13, 1979

Dr. Harold C. Hodge
School of Medicine
Department of Pharmacology
University of California
San Francisco, CA 94143

Dear Dr. Hodge:

As we agreed in our telephone conversation of January 31, 1979, I am sending you the following:

(1) One Ninety Day Subacute Rat Toxicity Study using FLUORAD® Fluorochemical FC-143.

(2) One Ninety Day Subacute Rhesus Monkey Toxicity Study using FLUORAD® Fluorochemical FC-143.

(3) One Ninety Day Subacute Rat Toxicity Study using FLUORAD® Fluorochemical Surfactant FC-95.

(4) One Ninety Day Subacute Rhesus Monkey Toxicity Study using FLUORAD® Fluorochemical Surfactant FC-95.

(5) One Ninety Day Subacute Rat Toxicity Study using FM-3422.

(6) One Ninety Day Subacute Rhesus Monkey Toxicity Study using FM-3422.

(7) One Technical Report by Jon Belisle entitled IRDC 137-088: FM-3422/Monkey.

I would like to confirm Thursday, April 12, 1979, as our next meeting date with you. We will be making the trip by 3M Company plane and will be arriving at San Francisco Airport at about 8:30 a.m. From there we will go to the Hilton Inn, International where we have reserved a meeting room. (A sign with the location of the meeting room should be posted in the lobby.) The Hilton Inn is at the airport and we expect to be there at 9:00 a.m. We plan to leave the Hilton around 3:30 p.m. This is flexible and depends upon how long our discussions take.

02 010793

Our group will include the following persons:

M.T. Case, Pathologist, Riker Research

F. Griffith, Manager, Toxicology Services, Medical Dept.

L.C. Krogh, Division Vice President, Commercial Chemicals Div.

J.D. LaZerte, Technical Director, Commercial Chemicals Div.

R.A. Nelson, Laboratory Manager, Riker Research

R.E. Ober, Laboratory Manager, Riker Research

J.A. Pendergrass, Associate Director, Medical Department

R.A. Prokop, Laboratory Manager, Commercial Chemicals Div.

F.A. Ubel, Medical Director, Medical Department

We look forward to seeing you on April 12. If you have any questions please call me.

Very truly yours,

R. A. Prokop
Manager, Research
Commercial Chemicals Division

RAP/ko

F.A. Ubel - 220-2

02 010795

EXHIBIT U

3M REQUEST FOR COMPANY AIRPLANE

FORM 9278-L

— Please type —

Submitted by (Name, Dept., Bldg., Flr.): Lester C. Krogh - Commercial Chemicals Div.-223-6SE

Extension: 2425

Aircraft desired: [X] Grumman Gulfstream II [ ] Fairchild F-27 [ ] Other:

ITINERARY (to change dates or itinerary, submit revised form)	Departure Date	Departure Hour	From (City/Airport)	To (City/Airport)	Hotel (if any)
1	7/21/78	6:15a.m	St. Paul (Holman)	Northeast Airmotive, Portland, Me. Jetport	
2	7/21/78	4:00p.m	Northeast Airmotive, Portland, Me. Jetport	St. Paul (Holman)	
3					
4					

REASON FOR TRIP: To meet with Dr. Harold C. Hodge

DEPT. TO BE CHARGED: If one dept. is to be charged for flight, show here. If more than one dept. or empl. no./project is to be charged, show below after each name. —Do not fill in both areas— ▶ Dept. No. | Employee No. or Project

GROUP LEADER: J. D. LaZerte

Aviation Dept. Credit: 0278 - 921 — | Amount: $

Passengers Name & Initials, and Affiliation if not 3M	3M	Dept. To Be Charged	Sub Acct.	Empl. No. or Project	AREA WITHIN BOLD LINE FOR AVIATION DEPT. USE From	To	Miles	Amount
1 LaZerte, J.D.	X	3058	711	44520 H				
2 Long, J. E.	X	0227	711	19557 H				
3 Krogh, L. C. *	X	3052	711	45042 H				
4 Prokop, R. A.	X	3058	711	42368 H				
5 Ubel, F. A.	X	0224	711	45202 H				
6			711	H				
7			711	H				
8			711	H				
9 PLEASE SERVE BREAKFAST ON THE PLANE			711	H				
10			711	H				
11			711	H				
12 *TO PORTLAND, ME. ONLY.			711	H				
13			711	H				
14			711	H				
15			711	H				
16			711	H				
17			711	H				
18			711	H				

02 011158

DIVISION LEVEL APPROVAL — Name and Position: L. C. Krogh, Lester C. Krogh, Division Vice President Commercial Chemicals Division — Date: 6/27/78

GROUP LEVEL APPROVAL — Name and Position: J. M. Pitblado, Group Vice President — Date:

Confirmed by — Date

Send original to Aviation, Dept. A copy will be sent back to the "Submitted By" name and address.

For extended trips — use one form for each day.

Recheck department numbers and employee numbers.

EXHIBIT V

Interoffice Correspondence 3M

DRAFT

Subject: MEETING MINUTES - MEETING WITH H.C. HODGE

3M "CONFIDENTIAL"

April 26, 1979

THOSE PRESENT:

M.T. Case - 218-2
F.D. Griffith - 220-2E
H.C. Hodge - U. of California
L.C. Krogh - 223-6SE
J.D. LaZerte - 236-1
R.A. Nelson - 218-3
R.E. Ober - 218-2
J.A. Pendergrass - 220-2E
R.A. Prokop - 236-2B
F.A. Ubel - 220-2E

Those present met on April 12, 1979 at the Hilton Hotel in San Francisco California to review recent results which are relevant to the Fluorochemicals in Blood program and to discuss future plans.

R.A. Prokop began the meeting by giving background on FC-807. FC-807 is used in combination with a hydrocarbon sizing agent to give oil and water repellency to paper and paperboard. One of its principle uses is as an indirect food additive, and a petition was granted in the late 1960's for its use as such. It is manufactured by reacting perfluorooctanesulfonyl fluoride with ethyl amine. Subsequent reaction of the sulfonamide with ethylene carbonate followed by sequential reaction with $POCl_3/H_2O$ and ammonia give FC-807. (See attached flowsheets) It is sold as a 35-40% solution in isopropyl alcohol.

F.A. Ubel reviewed recent developments in the areas of serum organic fluorine levels, human health and epidemiology as they relate to the fluorochemicals in blood program.

The serum organic fluorine level of the individual who was previously removed from fluorochemical exposure when his

Exhibit 1204
State of Minnesota v. 3M Co., Court File No. 27-CV-10-28862

serum organic fluorine level rose to 70 ppm has recently been measured. The level was found to be 45 ± 5 ppm. The amount of FC-143 in his urine was found to be 220 ug for a 24 hour period.

Serum organic fluorine levels have recently been measured on selected employees at Cordova, Decatur and Chemolite. In comparison with levels measured in 1976 and 1977, there has been little change.

The serum organic fluorine levels of 15 Chemolite employees were measured in 1978 prior to their being involved in the packaging of FC-143. Repeat measurements of their serum organic fluorine taken recently showed some unexpected elevations in level. Inorganic levels were also higher than expected. ? Contamination with inorganic fluoride is suspected to be the cause of the high inorganic levels in serum, but the entire set of results will be reviewed to see if any errors were made. A study will be done attempting to relate exposure to serum organic fluorine levels.

Eight serum samples from rural China were analyzed for organic and inorganic fluorine levels. Organic fluorine levels ranged from 0.004 ppm to 0.017 ppm and inorganic fluoride levels from 0.044 to 0.076 ppm.

The epidemiology study is still in progress. The study involves tracing about 3500 people and involves about 100 deaths. So far there does not appear to be what might be considered as "unusual causes of death".

H.C. Hodge commented that data from this epidemiology study is very important and that the study should be carried out carefully. Criteria set forth by the N.C.I. should be followed. Dr. Hodge further stated that he would rather accept data from this type of study for identifying human health effects than data from animal studies, but that the National Cancer Institute would not agree.

F.A. Ubel summarized a conversation with two Mayo Clinic physicians regarding the significance of slightly elevated liver enzyme studies in some of the Chemolite employees. No definite conclusions were reached, except that the values were very slightly elevated and in some instances were felt to be compatible with their history of alcohol ingestion. They could not make any statement unless they had a chance to examine the employees personally.

Dr. Hodge was shown a summary of the abnormal findings in those 3M employees who had known fluorochemical blood levels. He was given a copy.

The results of the physical examinations done on Decatur and Cordova employees look very good. There does not appear to be evidence of a work related problem. The Chemolite employees showed more abnormalities, but the majority of these appeared to be related to a known medical problem or medication. It was the conclusion of the physicians who supervised the examinations that there did not appear to be a problem which could be identified as work related. Additional analysis of the data will be done.

J.A. Pendergrass reviewed data on workplace concentrations of various fluorochemicals in Alabama and Minnesota plants. In most cases the workplace concentrations of fluorochemicals are low-lower than some time weighed averages recommended for known or suspected carcinogens. Fluorochemical salts are exceptions. Due to their dusty nature workplace concentrations are higher. Levels of one of these materials, FC-143, have been reduced to an acceptable value. Work is underway to reduce employee exposure to other fluorochemical salts.

R.A. Nelson reviewed results of 90 day subacute toxicity studies using FC-95, FC-143 and FM-3422. Of these compounds FC-95 was the most toxic. It produced deaths in the monkey at 4.5 mg/Kg and in the rat at 10 mg/Kg. Target organs in the rat were liver haemopoietic tissue, stomach and small intestine. In monkeys, the apparent target organ was the upper G.I. tract.

The liver and possibly the kidney and G.I. tract were target organs in the rats fed FM-3422. In the case of monkeys there was chemical evidence of G.I. toxicity.

FC-143 produced liver changes in rats at the highest dose. In monkeys hematopoietic tissue was effected at lethal doses and there was evidence of gastrointestinal toxicity.

H.C. Hodge also presented his summary of results of the 90 day studies on FC-143, FM-3422 and FC-95 (attached). These levels were 1.5 mg/Kg for FC-143 and FM-3422 in the rat, and 3 mg/Kg in the monkey. For FC-95 there was no no effect level in the rat and no data for estimating a no effect level in the monkey. There appears to be an indication of liver effects from FC-95, FM-3422 and FC-143 at all dose levels in the rat studies.

H.C. Hodge questioned whether some of the toxic effects observed in the animal studies might be due to low surface tension. Surface active agents are known to be capable of causing a problem in the gut. It was pointed out that some fluorochemicals are the most potent surfactants known.

F.A. Griffith pointed out that toxic effects such as those observed with FC-95, FM-3422 and FC-143 are common with surfactants.

R.A. Nelson pointed out that the liver effects are probably reversible.

H.C. Hodge pointed out that a buildup of fluoride ion can be detected most rapidly in the bone. Thus evidence of breakdown of fluorochemicals to fluoride ion could be detected in the bone. Bone should be collected at the end of a chronic study and analyzed for fluoride.

Basic diet important must preclude adsorption of these compounds by bone

H.C. Hodge was then asked for his recommendations on future work. After a brief review of customer exposure to 3M fluorochemicals Dr. Hodge recommended the following:

1. It does not appear that there is a toxicity problem outside 3M. However we do not know if there are toxicity problems due to employee exposure. Reduction in exposure should have top priority.

2. Metabolic instead of chronic studies should be done next. Sequestering kinetics and metabolites should be looked at.

3. Reproduction studies, including teratology, should be given high priority. Two generations should be used. Such studies should take less than one year.

4. Carcinogenicity of fluorochemicals should be looked at. Start with Ames testing and continue to more sophisticated mutagenicity tests. If any of these turn out positive it will be necessary to go on to a chronic study.

R.A. Nelson questioned the reliability of test other than the Ames. The transformation test may be on the way out. Different regulatory agencies are not getting results which agree.

M.T. Case outlined proposed studies on FC-807 and studies being considered for FC-95 and FC-143. (Slides attached) A question was raised on the FC-807 study as to whether the F.D.A. should be asked about the protocol. It was suggested that we should proceed without consulting the F.D.A. since the study is scientifically valid.

R.E. Ober outlined planned work in the metabolic area.(Slides attached) This proposed work involved metabolism of FC-807, persistence of FC-95 and FC-143, and skin absorption studies.

It was suggested that H.C. Hodge be given more time to consider the proposed slides on FC-807, FC-95, FC-143 and the metabolism work before being asked for an opinion.

ADDENDUM

I called Dr. Hodge on April 20, 1979 to give him the acute oral toxicity data on FC-95, FC-143 and FM-3422 which was generated at IRDC prior to the 90 day studies. He asked that the following be added to the meeting minutes:

The study of levels of FC-807 or its metabolites is of utmost importance in determining possible future problems. It should be determined if FC-807 or its metabolites are present in man, what level they are present, and the degree of persistence (half-life) of these materials. If the levels are high and widespread and the half-life is long, we could have a serious problem.

R.A. Prokop

RAP/ko
Attachments

EXHIBIT W

Com Chem file

Interoffice Correspondence 3M

Subject: MEETING MINUTES -
MEETING WITH H.C. HODGE

3M "CONFIDENTIAL"

JUNE 7, 1979

THOSE PRESENT:

M.T. CASE - 218-2
F.D. GRIFFITH - 220-2E
H.C. HODGE - U. OF CALIFORNIA
L.C. KROGH - 223-6SE
J.D. LAZERTE - 236-1
R.E. OBER - 218-2
J.A. PENDERGRASS - 220-2E
R.A. PROKOP - 236-2B
F.A. UBEL - 220-2E
R.A. Nelson - 218-3

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R.A. Prokop began the meeting by giving background on FC-807. FC-807 is used in combination with a hydrocarbon sizing agent to give oil and water repellency to paper and paperboard. One of its principle uses is as an indirect food additive, and a petition was granted in the late 1960's for its use as such. It is manufactured by reacting perfluorooctanesulfonyl fluoride with ethyl amine. Subsequent reaction of the sulfonamide with ethylene carbonate followed by sequential reaction with $POCl_3/H_2O$ and ammonia give FC-807. (See attached flowsheets) It is sold as a 35-40% solution in isopropyl alcohol.

F.A. Ubel reviewed recent developments in the areas of serum organic fluorine levels, human health and epidemiology as they relate to the fluorochemicals in blood program.

The serum organic fluorine level of the individual who was previously removed from fluorochemical exposure when his serum organic fluorine level rose to 70 ppm has recently been measured. The level was found to be 45 + 5 ppm. The amount of FC-143 in his urine was found to be 220 ug for a 24 hour period.

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Eight serum samples from rural China were analyzed for organic and inorganic fluorine levels. Organic fluorine levels ranged from 0.004 ppm to 0.017 ppm and inorganic fluoride levels from 0.044 to 0.076 ppm. In the U.S. serum organic fluorine levels range from 0.002 to 0.13 ppm and inorganic levels from 0.003 to 0.17 ppm.

The epidemiology study is still in progress. The study involves tracing about 3500 people and involves about 100 deaths. So far there does not appear to be what might be considered as "unusual causes of death".

H.C. Hodge commented that data from this epidemology study is very important and that the study should be carried out carefully. Criteria set forth by the N.C.I. should be followed. Dr. Hodge further stated that he would rather accept data from this type of study for identifying human health effects than data from animal studies, but that the National Cancer Institute would not agree.

F.A. Ubel summarized a conversation with two Mayo Clinic physicians regarding the significance of slightly elevated liver enzyme levels in some of the Chemolite employees. No definite conclusions were reached, except that the values were very slightly elevated and in some instances were felt to be compatible with their history of alcohol ingestion. They could not make any statement unless they had a chance to examine the employees personally.

Dr. Hodge was shown a summary of the abnormal findings in those 3M employees who had known fluorochemical blood levels. He was given a copy.

The results of the physical examinations done on Decatur and Cordova employees look very good. There does not appear to be evidence of a work related problem. The Chemolite employees showed more abnormalities, but the majority of these appeared to be related to a known medical problem or medication. It was the conclusion of the physicians who supervised the examinations that there did not appear to be a problem which could be identified as work related. Additional analysis of the data will be done.

J.A. Pendergrass reviewed data on workplace concentrations of various fluorochemicals in Alabama and Minnesota plants. In most cases the workplace concentrations of fluorochemicals are low-lower than some time weight averages recommended for known or suspected carcinogens. Fluorochemical salts are exceptions. Due to their dusty nature workplace concentrations are higher. Levels of one of these materials, FC-143, have been reduced to an acceptable value. Work is underway to reduce exposure to other fluorochemical salts.

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The liver and possibly the kidney and G.I. tract were target organs in the rats fed FM-3422. In the case of monkeys there was clinical evidence of G.I. toxicity.

FC-143 produced liver changes in rats at the highest dose. In monkeys hematopoietic tissue was affected at lethal doses and there was evidence of gastrointestinal toxicity. H.C. Hodge also presented his summary of results of the 90 day studies on FC-143, FM-3422 and FC-95 (attached). His projected no-effect levels were 1.5 mg/Kg for FC-143 and FM-3422 in the rat, and 3 mg/Kg in the monkey. For FC-95 there was no no-effect level in the rat and no data for estimating a no-effect level in the monkey. There appears to be an indication of liver effects from FC-95, FM-3422 and FC-143 at all dose levels in the rat studies.

H.C. Hodge questioned whether some of the toxic effects observed in the animal studies might be due to low surface tension. Surface active agents are known to be capable of causing a problem in the gut. It was pointed out that some fluorochemicals are the most potent surfactants known.

F.A. Griffith pointed out that toxic effect such as those observed with FC-95, FM-3422 and FC-143 are common with surfactants.

R.A. Nelson pointed out that the liver effects are probably reversible, but this would require further study to prove.

H.C. Hodge pointed out that a buildup of fluoride ion can be detected most rapidly in the bone. Thus evidence of breakdown of fluorochemicals to fluoride ion could be detected in the bone. Bone should be collected at the end of a chronic study and analyzed for fluoride.

H.C. Hodge was then asked for his recommendations on future work. After a brief review of customer exposure to 3M fluorochemicals Dr. Hodge recommended the following:

1. It does not appear that there is a toxicity problem outside 3M. However we do not know if there are toxicity problems due to employee exposure. Reduction in exposure should have top priority.

2. Metabolic instead of chronic studies should be done next. Sequestering kinetics and metabolites should be looked at.

3. Reproduction studies, including teratology, should be given high priority. Two generations should be used. Such studies should take less than one year.

4. Carcinogenicity of fluorochemicals should be looked at. Start with Ames testing and continue to more sophisticated mutagenicity tests. If any of these turn out positive it will be necessary to go on to a chronic study.

R.A. Nelson questioned the reliability of tests other than the Ames. The transformation test may be on the way out. Different regulatory agencies are not getting results which agree.

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R.E. Ober outlined planned work in the metabolic area. (Slides attached) This proposed work involved metabolism of FC-807, persistence of FC-95 and FC-143, and skin absorption studies.

It was suggested that H.C. Hodge be given more time to consider the proposed slides on FC-807, FC-95, FC-143 and the metabolism work before being asked for an opinion.

ADDENDUM

I called Dr. Hodge on April 20, 1979 to give him the acute oral toxicity data on FC-95, FC-143 and FM-3422 which was generated at IRDC prior to the 90 day studies. He asked that the following be added to the meeting minutes:

The study of levels of FC-807 or its metabolites is of utmost importance in determining possible future problems. It should be determined if FC-807 or its metabolites are present in man, what level they are present, and the degree of persistence (half-life) of these materials.

R.A. Bishop

RAP/ko
Attachments

EXHIBIT X

Interoffice Correspondence 3M

Subject: MEETING MINUTES -
MEETING WITH J.R. MITCHELL

3M "CONFIDENTIAL"

JUNE 7, 1979

THOSE PRESENT:

M.T. CASE - 218-2
F.D. GRIFFITH - 220-2E
L.C. KROGH - 223-6SE
J.D. LAZERTE - 236-1
J.R. MITCHELL - BAYLOR SCHOOL OF MEDICINE
R.A. NELSON - 218-3
R.E. OBER - 218-2
J.A. PENDERGRASS - 220-2E
R.A. PROKOP - 236-2B
F.A. UBEL - 220-2E

Those present met on April 13, 1979 at the Host International Hotel in Houston Texas to review recent results which are relevant to the Fluorochemicals in Blood Program and to discuss future plans.

R.A. Prokop began the meeting by giving background on preparation of 3M fluorochemicals. He reviewed the preparation of perfluoro-octanesulfonyl fluoride, perfluorooctanoyl fluoride, inert fluids, FC-95, FC-143 and FC-807. (Slides attached)

J.R. Mitchell expressed concern that residual $C_7F_{15}COF$ might be present in FC-143. He was concerned that $C_7F_{15}COF$ might be an excellent acylating agent and thus a potential carcinogen. He was told that since $C_7F_{15}COF$ is treated vigorously with excess base, it was highly unlikely that even trace amounts would be present in FC-143.

J.A. Pendergrass reviewed data on workplace concentrations of various fluorochemicals in Alabama and Minnesota plants. (See attached slides and Meeting Minutes of meeting with H.C. Hodge, 4/12/79).

02 010755

F.A. Ubel reviewed recent developments in the areas of serum organic fluorine levels, human health and epidemiology as they relate to the fluorochemicals in blood program. (See attached slides and Meeting Minutes of meeting with H.C. Hodge, 4/12/79).

J.R. Mitchell had the following questions and comments:

1. Wives of employees having high serum organic fluorine levels should be examined for the presence of organic fluorine in their serum.

2. Have you looked for target organ problems in your epidemiology study? (Answer-yes, we found nothing.)

3. Why are there so many (apparent) alcoholics in packaging? Is there a correlation between serum organic fluorine levels, alcohol and occupation? Is this being done in the epidemiology study? (Answer-no.)

4. You should get more information on length of employment and type of exposure to a specific chemical. Fat and liver biopsies are important. Indications of exposure can be obtained from serum organic fluorine levels.

5. You should determine the saturation level of human albumin with fluorochemicals. Human metabolism and distribution in the body are important. We must know the amount of organic fluorine in the fat and liver. It is possible that certain fluorochemicals are only in the blood. This information combined with analysis for serum organic fluorine levels gives better information than classical animal studies.

6. It would be medically acceptable to do a liver biopsy on employees who are exposed to fluorochemicals and are also alcoholics. Fat biopsy poses no problem. It would not be advisable to do liver biopsies on employees who are not alcoholics.

7. If there turns out to be health problems due to organic fluorochemicals in blood, the fluorochemicals could possibly be removed by hemoperfusion, plasma perfusion or plasma phoresis.

A comment was made that it would be beneficial to our understanding of fluorochemical distribution if tissue samples were available from a deceased individual who had worked with fluorochemicals. J.R. Mitchell agreed.

R.A. Nelson reviewed results of 90 day subacute toxicity studies using FC-95, FC-143 and FM-3422 (See attached slides and Meeting Minutes with H.C. Hodge of 4/12/79). J.R. Mitchell made the following comments:

1. The effects of fluoride ion on hematopoitic effects and liver toxicity should be investigated.

2. The slides from the I.R.D.C. study relating to liver toxicity should be obtained and reviewed to better define the liver changes.

M.T. Case agreed that we could get slides from 90 day studies at I.R.D.C. for possible re-examination.

M.T. Case summarized briefly the proposed study on FC-807 including the reasons for proceeding with the studies and the selected dose levels for the in-utero study. J.R. Mitchell made the following comments:

1. **At present you have no definite evidence that FC-807 has entered man. Animal studies may be irrelevant.**

2. **Results are obtainable on FC-807 in humans. Analytical work can be done on virgin persons. Exposure can be studied.**

3. **You should get more information (in man) to properly design animal studies. Dose levels must be set and the proper animal species chosen which has metabolic pathways comparable to man.**

J.R. Mitchell was asked if he recommended any mutagenicity tests other than the Ames. He replied that he did not.

R.E. Ober questioned whether the amount of FC-807 actually transferred to food should be looked at. J.R. Mitchell considered this to be a good approach.

R.E. Ober questioned whether distribution of FC-807 should be looked at in animals before man. J.R. Mitchell replied that animals should not be studied in place of man if man is available.

R.E. Ober questioned whether it would be desirable to try to remove fluorochemicals from man by means of resins. J.R. Mitchell was uncertain as to this approach. It would depend on how fast the resin would remove the fluorochemicals.

J.R. Mitchell then summarized the meeting in the form of a slide as follows:

SUMMARY BY J.R. MITCHELL

I. Potential Exposure

1. 3M Employees
2. Subcontractor Employees
3. Public Health
4. Environmental (fish, fowl, etc.)

II. Possible Legal Issues

1. TSCA Sec. 8(e)

? All criterial must be filled

2. Human Injury

02 010757

III. Clinical (Human) Studies

1. R_f disposition and distribution
2. Metabolites in tissue and urine
3. Intervention of Hemoperfusion of plasma phoresis (removal by) chelestramine type resins - others?
4. Epidemiology
   a. Of assay and family
   b. Other sources in USA
5. Epidemiology Review by Categories
   a. Compound
   b. Time Level
   c. Dose
   d. Organ System

IV. Animal Studies

1. Pharmocokinetic
2. Toxic Effects
   a. Acute
   b. Effect of F^-

V. Analytical

1. Distribution
   a. % binding and saturation
2. Metabolites
3. Contaminants from Manufacture
4. Route of exposure ⟶ disposition

During the summary Dr. Mitchell placed emphasis on epidemiology. Information on categories should be obtained immediately. This should be correlated with levels of organic fluorine in the blood. Deaths, hematopoetic effects, rare tumors should be investigated.

Dr. Mitchell also commented on capabilities at the Baylor School of Medicine for analyzing for trace amounts of chemicals in serum and tissue. By using modified GC/MS techniquies amounts at the parts per trillion level can probably be detected. Besides being a more rapid method of analysis than we are now using, this technique might be used to determine fluorochemicals in tissue from fat and liver biopsies. This would allow one to determine the distribution of fluorochemicals in humans.

It was agreed that J.R. Mitchell will investigate the possibility of using the above technique to analyze trace amounts of 3M fluorochemicals. After consulting with analytical personnel at the Baylor School of Medicine, he will contact R.E. Ober as soon as possible and a decision as to how to proceed will be agreed upon.

R.A. Prokop

RAP/ko
Attachments

EXHIBIT Y

Interoffice Correspondence 3M

DRAFT

Subject: MEETING MINUTES -
MEETING WITH J.R. MITCHELL

April 26, 1979

THOSE PRESENT:

M.T. Case - 218-2
F.D. Griffith - 220-2E
L.C. Krogh - 223-6SE
J.D. LaZerte - 236-1
J.R. Mitchell - Baylor School of Medicine
R.A. Nelson - 218-3
R.E. Ober - 218-2
J.A. Pendergrass - 220-2E
R.A. Prokop - 236-2B
F.A. Ubel - 220-2E

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J.R. Mitchell expressed concern that residual $C_7F_{15}COF$ might be present in FC-143. He was concerned that $C_7F_{15}COF$ might be excellent acylating agent and thus a potential carcinogen. He was told that since $C_7F_{15}COF$ is treated vigorously with excess base, it was highly unlikely that even trace amounts would be present in FC-143.

J.A. Pendergrass reviewed data on workplace concentrations of various fluorochemicals in Alabama and Minnesota plants. (See attached slides and Meeting Minutes of meeting with H.C. Hodge, 4/12/79).

Exhibit
2722
State of Minnesota v. 3M Co.,
Court File No. 27-CV-10-28862

3MA10034826

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J.R. Mitchell had the following questions and comments:

1. Wives of employees having high serum organic fluorine levels should be examined for the presence of organic fluorine in their serum.

2. Have you looked for target organ problems in your epidemiology study? (Answer-yes, we found nothing.)

3. Why are there so many (apparent) alcoholics in packaging? Is there a correlation between serum organic fluorine levels, alcohol and occupation? Is this being done in the epidemiology study? (Answer-no.)

4. You should get more information on length of employment and type of exposure to a specific chemical. Fat and liver biopsies are important. Indications of exposure can be obtained from serum organic fluorine levels.

5. You should determine the saturation level of human albumen with fluorochemicals. Human metabolism and distribution in the body are important. We must know the amount of organic fluorine in the fat and liver. It is possible that certain fluorochemicals are only in the blood. This information combined with analysis for serum organic fluorine levels gives better information than classical animal studies.

6. It would be medically acceptable to do a liver biopsy on employees who are exposed to fluorochemicals and are also alcoholics. Fat biopsy poses no problem. It would not be advisable to do liver biopsies on employees who are not alcoholics.

7. If there turns out to be health problems due to organic fluorochemicals in blood, the fluorochemicals could possibly be removed by haemoperfusion, plasma perfusion or plasma phoresis.

A comment was made that it would be beneficial to our understanding of fluorochemical distribution if tissue samples were available from a decreased individual who had worked with fluorochemicals. J.R. Mitchell agreed.

R.A. Nelson reviewed results of 90 day subacute toxicity studies using FC-95, FC-143 and FM-3422 (See attached slides and Meeting Minutes with H.C. Hodge of 4/12/79). J.R. Mitchell made the following comments:

1. The effects of fluoride ion on haematopoitic effects and liver toxicity should be investigated.

2. The slides from the I.R.D.C. study relating to liver toxicity should be obtained and reviewed to better define the liver changes.

3. Some of the symptoms in animals from these 90 day studies are similar to those observed with carcinogens.

M.T. Case agreed to get slides from all 90 day studies at I.R.D.C. and re-examine them.

M.T. Case summarized briefly the proposed study on FC-807 including the reasons for proceeding with the studies and the selected dose levels for the in-utero study. J.R. Mitchell made the following comments:

1. At present you have no definite evidence that FC-807 has entered man. Animal studies may be irrelevant.

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R.E. Ober questioned whether it would be desirable to try to remove fluorochemicals from man by means of resins. J.R. Mitchell was uncertain as to this approach. It would depend on how fast the resin would remove the fluorochemicals. There could be problems.

J.R. Mitchell then summarized the meeting in the form of a slide as follows:

SUMMARY BY J.R. MITCHELL

I. People at Risk

1. 3M Employees
2. Subcontractor Employees
3. Public Health
4. Environmental (fish, fowl, etc.)

II. Legal Issues

1. TSCA Sec. 8(e)

? All criterial must be filled

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   a. Of assay and family
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4. Route of exposure ⟶ disposition

During the summary Dr. Mitchell placed emphasis on epidemiology. Information on categories should be obtained immediately. This should be correlated with levels of organic fluorine in the blood. Deaths, haemopoetic effects rare tumors should be investigated.

Dr. Mitchell also commented on capabilities at the Baylor School of Medicine for analyzing for trace amounts of chemicals in serum and tissue. By combining negative ion plasma chromotography with a nuclear quadropole unit, amounts at the parts per trillion level can be detected. Besides being a more rapid method of analysis than we are now using, this technique might be used to determine fluorochemicals in tissue from fat and liver biopsies. This would allow one to determine the distribution of fluorochemicals in humans.

It was agreed that J.R. Mitchell will investigate the possibility of using the above technique to analyze trace amounts of 3M fluorochemicals. After consulting with analytical personnel at the Baylor School of Medicine, he will contact R.E. Ober as soon as possible and a decision as to how to proceed will be agreed upon.

R.A. Pierce

RAP/ko
Attachments

EXHIBIT Z

THURSDAY, MARCH 16, 1978
PART V

ENVIRONMENTAL PROTECTION AGENCY

TOXIC SUBSTANCES CONTROL ACT

Statement of Interpretation and Enforcement Policy; Notification of Substantial Risk

[6560-01]

ENVIRONMENTAL PROTECTION AGENCY

[FRL 849-2]

TOXIC SUBSTANCES CONTROL ACT

Notification of Substantial Risk Under Section 8(e)

AGENCY: Environmental Protection Agency.

ACTION: Statement of interpretation and enforcement policy.

SUMMARY: This action states EPA's interpretation of, and enforcement policy concerning, section 8(e) of the Toxic Substances Control Act (TSCA) (90 Stat. 2029, 15 U.S.C. 2607). The provisions of that section went into effect on January 1, 1977.

Section 8(e) states that "any person who manufactures, processes, or distributes in commerce a chemical substance or mixture and who obtains information which reasonably supports the conclusion that such substance or mixture presents a substantial risk of injury to health or the environment shall immediately inform the Administrator of such information unless such person has actual knowledge that the Administrator has been adequately informed of such information."

DATES: The policy expressed in this document is in effect as of the date of publication.

FOR FURTHER INFORMATION CONTACT:

Frank D. Kover, Assessment Division, Office of Toxic Substances (WH-557), Environmental Protection Agency, 401 M Street SW., Washington, D.C. 20460, 202-755-2110.

SUPPLEMENTARY INFORMATION: On September 9, 1977, the Agency proposed guidance (42 FR 45362) on its interpretation of and policy concerning the provisions of section 8(e). Although the proposed "guidance" was an interpretive rule and statement of policy exempt from the notice and public comment provisions of the Administrative Procedure Act (5 U.S.C. 553), the Agency solicited comments on several issues to make more informed decisions. On October 11, the comment period was extended from October 15 to October 31, 1977 (42 FR 54857). On November 4, 1977, a supplemental notice to the proposed guidance was published (42 FR 57744), deleting the November 15 date for reporting certain information obtained before 1977 and stating that a new date would be established in the final guidance.

In developing this policy statement, two meetings have been held (February 1, 1977, and October 26, 1977) with selected representatives of industry and environmental and other interested groups. Comments submitted pursuant to the February 1 meeting were addressed in the preamble to the September 9 proposal. Over 100 written comments have been submitted pursuant to the September 9 proposal from trade associations, businesses, environmental groups, labor unions, State and Federal agencies, and other interested parties. Appendix B describes significant issues raised in these comments and the Agency's response to them.

The major modifications to the September 9 proposal are summarized in points 1 through 7 below.

(1) Pursuant to some question over the definition and nature of "guidance," this document is now described more accurately as a "policy statement." It is exempt from the notice and public comment provisions of the Administrative Procedure Act, as well as provisions concerning delayed effective dates.

(2) Many commenters expressed the view that to apply these requirements to officers and employees of a business organization would result in ill-considered, premature reports and would unfairly subject employees to conflicting responsibilities as individual respondents and as corporate agents. Other commenters expressed support for the view that certain employees have a responsibility to report pertinent information, and felt that the phrase "capable of appreciating pertinent information" appropriately described those employees.

The September 9 proposal would have applied section 8(e) requirements to commercial establishments as well as to employees capable of appreciating pertinent information, but stipulated enforcement priorities intended to encourage corporate processing and centralized reporting of such information (42 FR 45363). The intent was to ensure that pertinent information obtained by employees is promptly and appropriately considered, while minimizing duplicative or ill-considered submissions.

The Agency now feels that these objectives would best be served by allowing commercial establishments—under certain conditions designed to ensure full disclosure—to assume exclusive responsibility for reporting to EPA any substantial-risk information obtained by individual officers or employees. Accordingly, this policy statement stipulates that individual officers and employees will have fully discharged their section 8(e) obligations once they have notified the designated responsible company supervisor or official of pertinent information, *provided*, that the employing company or firm has established, internally publicizes, and affirmatively implements procedures governing such notifications. These procedures, at a minimum, must: (1) Specify the information that must be reported; (2) indicate how the notifications are to be prepared and submitted; (3) note the Federal penalties for failing to report; and (4) provide a mechanism for promptly notifying officers and employees who have submitted reports of the company's disposition of those reports, including whether or not they were submitted to EPA (and if not, informing employees of their right to report to EPA, as protected by TSCA section 23). EPA believes these four criteria will ensure prompt and appropriate processing of pertinent information.

Establishment of such procedures notwithstanding, all officials responsible and having authority for the organization's execution of its section 8(e) obligations retain personal liability for ensuring that substantial-risk information is reported to EPA.

(3) The September 9 proposal stated, in Part III, that a person obtains information when he is aware that it "may suggest" substantial risk. Numerous commenters questioned the Administrator's authority to compel the reporting of information which "may suggest" substantial risk. The Administrator agrees that section 8(e) addresses information that "reasonably supports the conclusion" of substantial risk and has deleted the "may suggest" provision, but emphasizes that "reasonably supports the conclusion" of substantial risk is not identical to a conclusive demonstration of substantial risk. The former typically occurs, and must be reported, at an earlier stage. Part VI in this policy statement provides Agency interpretation of the types of information that "reasonably support" such a conclusion.

(4) Numerous commenters requested clarification of different aspects of Part V of the September 9 proposal ("Information Which Reasonably Supports a Conclusion of Substantial Risk"), particularly concerning environmental effects, and suggested different interpretations of what constitutes a "substantial risk". The Agency continues to focus in this policy statement on the effects set forth in the September 9 proposal, but clarifies that the substantiality of a risk is a function of both the seriousness of the effect and the probability of its occurrence (see Part V).

(5) Numerous commenters maintained that section 8(e) only applies prospectively to information obtained after January 1, 1977. The Agency disagrees, as explained in the preamble to the September 9 proposal. This policy statement continues to apply section 8(e) to information obtained before 1977 of which a person has

been aware since January 1, 1977. In response to requests for clarification, the statement defines what constitutes such awareness. In this manner, EPA intends to limit the need for searches of historical records and files.

(6) This policy statement now provides that any information published in scientific literature, in any language, is exempt if it is referred to in abstracts published by specified abstracting services.

(7) This policy statement describes in a new Part X how to submit claims of confidentiality.

Accordingly, the Administrator's interpretation of and policy towards section 8(e) is set forth below.

Dated: February 24, 1978.

DOUGLAS COSTLE
Administrator.

I. DEFINITIONS

The definitions set forth in TSCA section 3 apply to these requirements. In addition, the following definitions are provided for purposes of this policy statement:

The term "manufacture or process 'for commercial purposes' " means to manufacture or process: (1) For distribution in commerce, including for test marketing purposes, (2) for use as a catalyst or an intermediate, (3) for the exclusive use by the manufacturer or processor, or (4) for product research and development.

The term "person" includes any natural person, corporation, firm, company, joint-venture, partnership, sole proprietorship, association, or any other business entity, any State or political subdivision thereof, any municipality, any interstate body and any department, agency, or instrumentality of the Federal Government.

The term "substantial-risk information" means information which reasonably supports the conclusion that a chemical substance or mixture presents a substantial risk of injury to health or the environment.

II. PERSONS SUBJECT TO THE REQUIREMENT

Persons subject to section 8(e) requirements include both natural persons and business entities engaged in manufacturing, processing, or distributing in commerce a chemical substance or mixture. In the case of business entities, the president, chief executive officer, and any other officers responsible and having authority for the organization's execution of its section 8(e) obligations must ensure that the organization reports substantial-risk information to EPA. The business organization is considered to have obtained any information which any officer or employee capable of appreciating the significance of that information has obtained. It is therefore incumbent upon business organizations to establish procedures for expeditiously processing pertinent information in order to comply with the schedule set forth in Part IV.

Those officers and employees of business organizations who are capable of appreciating the significance of pertinent information are also subject to these reporting requirements. An employing organization may relieve its individual officers and employees of any responsibility for reporting substantial-risk information directly to EPA by establishing, internally publicizing, and affirmatively implementing procedures for employee submission and corporate processing of pertinent information. These procedures, at a minimum, must: (1) Specify the information that officers and employees must submit; (2) indicate how such submissions are to be prepared and the company official to whom they are to be submitted; (3) note the Federal penalties for failing to report; and (4) provide a mechanism for promptly advising officers and employees in writing of the company's disposition of the report, including whether or not the report was submitted to EPA (and if not informing employees of their right to report to EPA, as protected by TSCA section 23). An employee of any company that has established and publicized such procedures, who has internally submitted pertinent information in accordance with them, shall have discharged his section 8(e) obligation. Establishment of such procedures notwithstanding, all officials responsible and having authority for the organization's execution of its section 8(e) obligations retain personal liability for ensuring that the appropriate substantial-risk information is reported to EPA.

Business organizations that do not establish such procedures cannot relieve their individual officers and employees of the responsibility for ensuring that substantial-risk information they obtain is reported to EPA. While officers and employees of such organizations may also elect to submit substantial-risk information to their superiors for corporate processing and reporting, rather than to EPA directly, they have not discharged their individual section 8(e) obligation until EPA has received the information.

NOTE.—Irrespective of a business organization's decision to establish and publicize the procedures described above, it is responsible for becoming cognizant of any substantial-risk information obtained by its officers and employees, and for ensuring that such information is reported to EPA within 15 working days.

III. WHEN A PERSON WILL BE REGARDED AS HAVING OBTAINED INFORMATION

A person obtains substantial-risk information at the time he first comes into possession of or knows of such information.

NOTE.—This includes information of which a prudent person similarly situated could reasonably be expected to possess or have knowledge.

An establishment obtains information at the time any officer or employee capable of appreciating the significance of such information obtains it.

IV. REQUIREMENT THAT A PERSON "IMMEDIATELY INFORM" THE ADMINISTRATOR

With the exception of information on emergency incidents of environmental contamination [see Part V(c)] a person has "immediately informed" the Administrator if information is received by EPA not later than the 15th working day after the date the person obtained such information. Supplementary information generated after a section 8(e) notification should, if appropriate, be immediately reported. For emergency incidents of environmental contamination, a person shall report the incident to the Administrator by telephone as soon as he has knowledge of the incident (see Part IX for appropriate telephone contacts). The report should contain as much of the information required by Part IX as possible. A written report in accordance with Part IX (a) through (f) is to be submitted within 15 days.

Information currently in the possession of a person who is subject to reporting must be reported within 60 days of publication of this policy statement.

V. WHAT CONSTITUTES SUBSTANTIAL RISKS

A "substantial risk of injury to health or the environment" is a risk of considerable concern because of (a) the seriousness of the effect [see Subparts (a), (b), and (c) below for an illustrative list of effects of concern], and (b) the fact or probability of its occurrence. (Economic or social benefits of use, or costs of restricting use, are not to be considered in determining whether a risk is "substantial".) These two criteria are differentially weighted for different types of effects. The human health effects listed in Subpart (a) below, for example, are so serious that relatively little weight is given to exposure; the mere fact the implicated chemical is in commerce constitutes sufficient evidence of exposure. In contrast, the remaining effects listed in Subparts (b) and (c) below must involve, or be accompanied by the potential for, significant levels of exposure (because of general production levels, persistence, typical uses, common means of disposal, or other pertinent factors).

Note that: (i) The effects outlined below should not be reported if the re-

spondent has actual knowledge that the Administrator is already informed of them.

(ii) Information respecting these effects can be obtained either directly, by observation of their occurrence, or inferred from designed studies as discussed in Part VI.

The Agency considers effects for which substantial-risk information must be reported to include the following:

(a) *Human health effects*—(1) Any instance of cancer, birth defects, mutagenicity, death, or serious or prolonged incapacitation, including the loss of or inability to use a normal bodily function with a consequent relatively serious impairment of normal activities, if one (or a few) chemical(s) is strongly implicated.

(2) Any pattern of effects or evidence which reasonably supports the conclusion that the chemical substance or mixture can produce cancer, mutation, birth defects or toxic effects resulting in death, or serious or prolonged incapacitation.

(b) *Environmental effects*—(1) Widespread and previously unsuspected distribution in environmental media, as indicated in studies (excluding materials contained within appropriate disposal facilities).

(2) Pronounced bioaccumulation. Measurements and indicators of pronounced bioaccumulation heretofore unknown to the Administrator (including bioaccumulation in fish beyond 5,000 times water concentration in a 30-day exposure or having an n-octanol/water partition coefficient greater than 25,000) should be reported when coupled with potential for widespread exposure and any non-trivial adverse effect.

(3) Any non-trivial adverse effect, heretofore unknown to the Administrator, associated with a chemical known to have bioaccumulated to a pronounced degree or to be widespread in environmental media.

(4) Ecologically significant changes in species' interrelationships; that is, changes in population behavior, growth, survival, etc. that in turn affect other species' behavior, growth, or survival.

Examples include: (i) Excessive stimulation of primary producers (algae, macrophytes) in aquatic ecosystems, e.g., resulting in nutrient enrichment, or eutrophication, of aquatic ecosystems.

(ii) Interference with critical biogeochemical cycles, such as the nitrogen cycle.

(5) Facile transformation or degradation to a chemical having an unacceptable risk as defined above.

(c) *Emergency incidents of environmental contamination*—Any environmental contamination by a chemical substance or mixture to which any of the above adverse effects has been ascribed and which because of the pattern, extent, and amount of contamination (1) seriously threatens humans with cancer, birth defects, mutation, death, or serious or prolonged incapacitation, or (2) seriously threatens non-human organisms with large-scale or ecologically significant population destruction.

VI. Nature and Sources of Information Which "Reasonably Supports the Conclusion" of Substantial Risk

Information attributing any of the effects described in Part V above to a chemical substance or mixture is to be reported if it is one of the types listed below and if it is not exempt from the reporting requirement by reason of Part VII of this policy statement. A person is not to delay reporting until he obtains conclusive information that a substantial risk exists, but is to immediately report any evidence which "reasonably supports" that conclusion. Such evidence will generally not be conclusive as to the substantiality of the risk; it should, however, reliably ascribe the effect to the chemical.

Information from the following sources concerning the effects described in Part V will often "reasonably support" a conclusion of substantial risk. Consideration of corroborative information before reporting can only occur where it is indicated below.

(1) *Designed, controlled studies.* In assessing the quality of information, the respondent is to consider whether it contains reliable evidence ascribing the effect to the chemical. Not only should final results from such studies be reported, but also preliminary results from incomplete studies where appropriate. Designed, controlled studies include:

(i) In vivo experiments and tests.

(ii) In vitro experiments and tests. Consideration may be given to the existence of corroborative information, if necessary to reasonably support the conclusion that a chemical presents a substantial risk.

(iii) Epidemiological studies.

(iv) Environmental monitoring studies.

(2) *Reports concerning and studies of undesigned, uncontrolled circumstances.* It is anticipated here that reportable effects will generally occur in a pattern, where a significant common feature is exposure to the chemical. However, a single instance of cancer, birth defects, mutation, death, or serious incapacitation in a human would be reportable if one (or a few) chemical(s) was strongly implicated. In addition, it is possible that effects less serious than those described in Part V(a) may be preliminary manifestations of the more serious effects and, together with another triggering piece of information, constitute reportable information; an example would be a group of exposed workers experiencing dizziness together with preliminary experimental results demonstrating neurological dysfunctions.

Reports and studies of undesigned circumstances include:

(i) Medical and health surveys.

(ii) Clinical studies.

(iii) Reports concerning and evidence of effects in consumers, workers, or the environment.

VII. Information Which Need Not Be Reported

Information need not be reported if it:

(a) Has been published by EPA in reports;

(b) Has been submitted in writing to EPA pursuant to mandatory reporting requirements under TSCA or any other authority administered by EPA (including the Federal Insecticide, Fungicide and Rodenticide Act, the Clean Air Act, the Federal Water Pollution Control Act, the Marine Protection, Research, and Sanctuaries Act, the Safe Drinking Water Act, and the Resource Conservation and Recovery Act), provided that the information: (1) Encompasses that required by Part IX (c) through (f); and (2) is from now on submitted within the time constraints set forth in Part IV and identified as a section 8(e) notice in accordance with Part IX(b);

(c) Has been published in the scientific literature and referenced by the following abstract services: (1) Agricola, (2) Biological Abstracts, (3) Chemical Abstracts, (4) Dissertation Abstracts, (5) Index Medicus, (6) National Technical Information Service.

(d) Is corroborative of well-established adverse effects already documented in the scientific literature and referenced as described in (c) above, unless such information concerns emergency incidents of environmental contamination as described in Part V(c), or

(e) Is contained in notification of spills under section 311(b)(5) of the Federal Water Pollution Control Act.

VIII. Information First Received by a Person Prior to the Effective Date of TSCA

Any substantial risk information possessed by a person prior to January 1, 1977, of which he is aware after that date shall be reported within 60 days of publication of this policy statement. The Agency considers that a person is "aware" of:

(a) Any information reviewed after January 1, 1977, including not only written reports, memoranda and other documents examined after January 1, 1977, but also information referred to in discussions and conferences in which the person participated after January 1, 1977;

(b) Any information the contents of which a person has been alerted to by date received after January 1, 1977, including any information concerning a chemical for which the person is presently assessing health and environmental effects;

(c) Any other information of which the person has actual knowledge.

IX. Reporting Requirements

Notices shall be delivered to the Document Control Officer, Chemical Information Division, Office of Toxic Substances (WH-557), Environmental Protection Agency, 401 M Street SW., Washington, D.C. 20460.

A notice should:

(a) Be sent by certified mail, or in any other way permitting verification of its receipt by the Agency,

(b) State that it is being submitted in accordance with section 8(e),

(c) Contain the job title, name, address, telephone number, and signature of the person reporting and the name and address of the manufacturing, processing, or distributing establishment with which he is associated,

(d) Identify the chemical substance or mixture (including, if known, the CAS Registry Number),

(e) Summarize the adverse effects being reported, describing the nature and the extent of the risk involved, and

(f) Contain the specific source of the information together with a summary and the source of any available supporting technical data.

For emergency incidents of environmental contamination (see Part V(c)), a person shall report the incident to the Administrator by telephone as soon as he has knowledge of the incident (see below for appropriate telephone contacts). The report should contain as much of the information required by instructions (b) through (f) above as possible. A written report, in accordance with instructions (a) through (f) above, is to be submitted within 15 days. Twenty-four hour emergency telephone numbers are:

Region I (Maine, Rhode Island, Connecticut, Vermont, Massachusetts, New Hampshire), 617-223-7265.
Region II (New York, New Jersey, Puerto Rico, Virgin Islands), 201-548-8730.
Region III (Pennsylvania, West Virginia, Virginia, Maryland, Delaware, District of Columbia), 215-597-9898.
Region IV (Kentucky, Tennessee, North Carolina, South Carolina, Georgia, Alabama, Mississippi, Florida), 404-881-4062.
Region V (Wisconsin, Illinois, Indiana, Michigan, Ohio, Minnesota), 312-353-2318.
Region VI (New Mexico, Texas, Oklahoma, Arkansas, Louisiana), 214-749-3840.
Region VII (Nebraska, Iowa, Missouri, Kansas), 816-374-3778.
Region VIII (Colorado, Utah, Wyoming, Montana, North Dakota, South Dakota), 303-837-3880.
Region IX (California, Nevada, Arizona, Hawaii, Guam), 415-556-6254.
Region X (Washington, Oregon, Idaho, Alaska), 206-442-1200.

X. Confidentiality Claims

(a) Any person submitting a notice to EPA under section 8(e) of TSCA may assert a business confidentiality claim covering all or part of the information contained in the notice. Any information covered by a claim will be disclosed by EPA only to the extent, and by means of the procedures, set forth in 40 CFR Part 2 (41 FR 36902, September 1, 1976).

(b) If no claim accompanies the notice at the time it is submitted to EPA, the notice will be placed in an open file to be available to the public without further notice to the submitter.

(c) To assert a claim of confidentiality for information contained in a notice, the submitter must submit two copies of the notice.

(1) One copy must be complete. In that copy the submitter must indicate what information, if any, is claimed as confidential by marking the specified information on each page with a label such as "confidential," "proprietary," or "trade secret."

(2) If some information in the notice is claimed as confidential, the submitter must submit a second copy. The second copy must be complete except that all information claimed as confidential in the first copy must be deleted.

(3) The first copy of the notice will be disclosed by EPA only to the extent, and by means of the procedures, set forth in 40 CFR Part 2. The second copy will be placed in an open file to be available to the public.

(d) Any person submitting a notice containing information for which they are asserting a confidentiality claim should send the notice in a double envelope.

(1) The outside envelope should bear the same address outlined in section IX of this policy statement.

(2) The inside envelope should be clearly marked "To be opened only by the OTS Document Control Officer."

XI. Failure To Report Information

Section 15(3) of TSCA makes it unlawful for any person to fail or refuse to submit information required under section 8(e). Section 16 provides that a violation of section 15 renders a person liable to the United States for a civil penalty and possible criminal prosecution. Pursuant to section 17, the Government may seek judicial relief to compel submittal of section 8(e) information and to otherwise restrain any violation of section 8(e).

Appendix A.—Quick Reference Summary for Emergency Incidents of Environmental Contamination

A. What Should Be Reported as an Emergency Incident

An emergency incident of environmental contamination is "any environmental contamination by a chemical substance or mixture . . . which, because of the pattern, extent and amount of contamination, (1) Seriously threatens humans with cancer, birth defects, mutation, death, or serious or prolonged incapacitation, or (2) seriously threatens non-human organisms with large scale or ecologically significant population destruction". (See Part V(c) for complete description.)

B. What Need Not Be Reported as an Emergency Incident

Information contained in notification of spills under section 311(b)(5) of the Federal Water Pollution Control Act (FWPCA). (For a complete list of exemptions to reporting, see Part VII.)

C. When and Where To Report Emergency Incidents

Emergency incidents of environmental contamination are to be reported immediately by telephone to the appropriate EPA Regional 24-hour telephone emergency line listed below.

Region I (Maine, Rhode Island, Connecticut, Vermont, Massachusetts, New Hampshire), 617-223-7265.
Region II (New York, New Jersey, Puerto Rico, Virgin Islands), 201-548-8730.
Region III (Pennsylvania, West Virginia, Virginia, Maryland, Delaware, District of Columbia), 215-597-9898.
Region IV (Kentucky, Tennessee, North Carolina, South Carolina, Georgia, Alabama, Mississippi, Florida), 404-881-4062.
Region V (Wisconsin, Illinois, Indiana, Michigan, Ohio, Minnesota), 312-353-2318.
Region VI (New Mexico, Texas, Oklahoma, Arkansas, Louisiana), 214-749-3840.
Region VII (Nebraska, Iowa, Missouri, Kansas), 816-374-3778.
Region VIII (Colorado, Utah, Wyoming, Montana, North Dakota, South Dakota), 303-837-3880.
Region IX (California, Nevada, Arizona, Hawaii, Guam), 415-556-6254.
Region X (Washington, Oregon, Idaho, Alaska), 206-442-1200.

In addition, a written report, in accordance with instructions (a) through (f) of Part IX, is to be submitted within 15 days to the Document Control Officer, Chemical Information Division, Office of Toxic Substances (WH-557), 401 M Street SW., Washington, D.C. 20460.

Appendix B—Significant Comments and Responses

A. Persons Subject to These Requirements

Comment 1: Employees cannot be held subject to these requirements, since: (a) They only have a partial role in the manufacture, processing, or distribution of chemicals, (b) in other sections of TSCA, the term "person who manufactures, processes, or distributes" chemicals clearly refers to business organizations; "persons" should be consistently defined, and (c) the application of criminal penalties mandates a strict interpretation of this word.

Response: The Agency considers that different sections of TSCA, having different purposes, are appropriately directed to different respondents. In the case of section 8(e), officers and employees who are capable of appreciating the significance of information have a legitimate responsibility to be alert to and report substantial-risk information. The guidance has been modified so that natural persons and business entities can fulfill their section 8(e) obligations in different ways. Most officers and employees can discharge their section 8(e) obligations by submitting pertinent information to corporate superiors, provided that the company has established the risk-evaluation procedures characterized in Part II. In the case of a business organization, its president, chief executive officer, and other officials responsible and having authority for the business organization's execution of its section 8(e) obligations must ensure that the organization reports substantial-risk information to EPA.

Comment 2: Even if employees can be held subject to these requirements, they should not be. To do so would force employees and employers into conflicting positions, inviting internal corporate dissension and over- reporting. Further, individuals often do not have the overview necessary to reach considered, well-supported decisions. Corporate reporting by designated officials will provide EPA with more reliable data.

Response: The Agency considers that employees have a legitimate role in risk reporting; it is imperative that risk information obtained by employees be appropriately considered. Officers and employees can fulfill their role in the reporting of substantial-risk information, without the disadvantages described above, by reporting information to superiors for corporate consideration, and, having done so, will have discharged their obligation to EPA. This is contingent upon the establishment by the business organization of certain procedures for risk-evaluation, thereby assuring the appropriate consideration of such reports. Those officers responsible and having authority for the organization's execution of its section 8(e) obligations must ensure that the organization reports substantial-risk information to EPA.

Comment 3: Clarify which employees are covered, and the extent of their obligation. Are employees "capable of appreciating pertinent information" by virtue of rank, or knowledge? Are rank and file employees subject to these requirements, or just supervisory and managerial personnel, company toxicologists, etc.? Is an employee absolved of further responsibility if he reports to his supervisor?

Response: The Agency considers that the phrase "capable of appreciating the significance of pertinent information" appropriately describes those officers and employees who have a responsibility to be alert to and report substantial-risk information, including not only relatively senior corporate officers but also many corporate employees. The policy statement modifies the September 9 proposal, in response to the concerns expressed in Comments 2 and 3, to permit most officers and employees to discharge their obligation by submitting information to corporate superiors, subject to the conditions described in Part II.

Comment 4: Consultants and independent labs should not be subject to these requirements.

Response: Contractors and independent labs are not responsible for reporting information they have obtained directly to EPA; rather, their client manufacturers, processors and distributors are responsible for reporting such information.

B. THE "OBTAINING" OF INFORMATION

Comment 5: The "may suggest" criterion in Part III of the proposal serves to compel further examination of information that by itself is not subject to section 8(e) requirements. The statutory language calling for "reasonable support" does not support this. Further, risk assessment often requires anywhere from months to several years of study after preliminary results "suggest" risk, far exceeding the 15-day compliance period.

Response: The Agency does not intend to compel under section 8(e) examination of information that by itself is not subject to section 8(e) requirements and has deleted the "may suggest" provision, providing its interpretation of what constitutes evidence that "reasonably supports the conclusion" of substantial risk in a new Part VI.

Comment 6: Section 8(e) obligations are incurred upon obtaining conclusory substantial-risk information.

Response: The Agency disagrees, and considers that "reasonable support" of a conclusion of substantial risk is not identical to the conclusion itself. The former typically occurs, and must be reported, at an earlier stage.

Comment 7: The statement, in Part III of the proposal that a person has obtained information if he ". . . should know of the existence of such information not in his possession but which would be delivered to him on request," tends to compel an active search for substantial-risk information rather than the reporting of substantial-risk information a person "obtains." This is of particular concern to importers with limited access to information possessed by their suppliers.

Response: The Agency considers that section 8(e) applies to information which a person possesses or of which he knows. It is not intended to compel searches for information or extraordinary efforts to acquire information. The Agency further considers, however, that "known" information includes information which a prudent person similarly situated could reasonably be expected to know. Negligence or intentional avoidance of information does not absolve a person of his section 8(e) obligation. Part III has been modified to express these intentions.

Comment 8: Circumstances can exist when coming "into possession" of risk information does not correspond to an understanding of the implications of the information; "obtains" should be defined in terms of possession of information and awareness of its import.

Response: The "obtaining" of information occurs via persons who are "capable of appreciating the significance of pertinent information." There will likely be circumstances in which the evaluation of information clarifies its full import; the establishment of corporate procedures for processing risk-information prescribed in Part II will expedite this.

C. TIME ALLOWED FOR COMPLIANCE

Comment 9: Fifteen calendar days is insufficient to determine whether information which "may suggest" substantial risk should be reported; it is even insufficient to accommodate normal procedural time constraints (corporate processing, mailing, holidays, etc.).

Response: The Agency has changed the compliance period to 15 business days. It is imperative that procedures be established to expedite the reporting of substantial-risk information, not that reporting conform to existing procedures.

Comment 10: Allow from 30 to 90 days for the second phase of reporting; alternatively, do not prescribe a time limit for additional reporting.

Response: Having deleted the "may suggest" criterion, the Agency sees no need to provide a second phase to the reporting period. Supplemental information that is generated after a section 8(e)-notification should, if appropriate, be immediately reported.

Comment 11: Allow from 30 to 120 days to report pre-1977 information; this period should commence: (a) upon final publication, (b) January 1, 1978, (c) following the inventory reporting period since many of the same corporate personnel will be implementing both requirements.

Response: The policy statement prescribes a 60 day reporting period, commencing immediately upon publication. Section 8(e) has been in effect since January 1, 1977; postponement in reporting substantial-risk information is not warranted.

D. EFFECTS AND INFORMATION THAT MUST BE REPORTED

Comment 12: The reporting of "any instance" of cancer, birth defects, etc., in humans is too broad and such information will be of little use; chemical workers, like the general population, develop cancers and other ailments of uncertain etiology.

Response: This policy statement clarifies that the reporting of single occurrences of human cancer or other serious effects will depend upon evidence strongly implicating one (or a few) chemical(s).

Comment 13: Dermal ailments and nausea are poorly chosen examples of precursor symptoms. Deleting these examples will avoid unduly emphasizing them when other symptoms may be more important, yet will not eliminate the obligation to report them if they are suspected precursors.

Response: The Agency agrees.

Comment 14: How are reportable data distinguished from routine tests including range tests such as LD_{50}'s?

Response: This policy statement directs the reporting of specified effects when unknown to the Administrator. Many routine tests are based on a knowledge of toxicity associated with a chemical; unknown effects occurring during such a range test may have to be reported if they are those of concern to the Agency and if the information meets the criteria set forth in Parts V and VI.

Comment 15: The most widespread "in vitro" test is the Ames test, which is subject to considerable debate. Clarify the circumstances under which positive results of in vitro tests must be reported.

Response: Part VI clarifies that the reporting of in vitro tests will depend upon the existence of corroborative information if necessary to reasonably support the conclusion of substantial risk.

Comment 16: The description of "extreme persistence" as a substantial risk is an example of the need to redefine Part V(c) ("Environmental Effects"). Persistence and bioaccumulation should be considered risks only when coupled with toxicity and significant exposure.

Response: Part V now clarifies those effects for which reporting depends upon a significant exposure potential. Persistence by itself is no longer itemized as a reportable effect but rather is considered to be a component of exposure potential; it may also underlie the measurements described in Part V(b)(1). Laboratory indicators of pronounced bioaccumulation are to be reported when coupled with potential for widespread exposure and any non-trivial adverse effect.

Comment 17: The n-octanol/water partition coefficient addresses a physico-chemical property, not biological effects, and is not alone an indicator of substantial risk; further, the values stated for the coefficient and the bioaccumulation factor in fish do not correspond.

Response: The Agency acknowledges the numerical error and has amended the values to correspond. This policy statement now directs the reporting of an experimental measurement of bioaccumulation when coupled with an adverse effect and potential for widespread exposure.

Comment 18: The requirement that information which "links" an effect to a chemical be reported is too broad and contradicts the statutory language of "reasonably supports".

Response: The Agency has provided in a new Part VI its interpretation of "reasonably supports".

Comment 19: A determination that information "reasonably supports the conclusion" of substantial risk cannot be made independently of considerations of use since the method and manner of using a chemical may influence the occurrence of an effect; in particular, the criteria should reflect a distinction between normal and abnormal uses of chemicals.

Response: The Agency considers that the appropriate components of a "substantial risk" with respect to a chemical are (a) the seriousness of the effect, and (b) total exposure potential. The method and manner of using a chemical is one of several factors determining its exposure potential. As described in Part V, the importance of exposure potential as a component of "substantial risk" depends upon the kind of effect of concern. Thus, the effects described in Part V(a) are so serious that relatively little weight is given to exposure; the effects described in Parts V (b) and (c) involve a significant exposure or exposure potential.

The Agency further considers that a definition of "normal" use for a particular chemical will often depend upon a knowledge of the risks associated with the chemical.

E. INFORMATION THAT NEED NOT BE REPORTED

Comment 20: Information published in scientific literature in languages other than English should be exempted if published in summary form by abstracting services. Can the accuracy of English language abstracts and commercial translations of foreign literature be assumed?

Response: This policy statement now provides that information published in scientific literature, whether in English or another language, is exempt from reporting if published in summary form by certain specified abstract services.

Comment 21: Information exchange systems with other Federal agencies should be immediately established so that respondents need not report to EPA information already reported to other Agencies, and vice versa. Such duplicative reports are unduly burdensome.

Response: EPA is coordinating this program with other agencies now. When this coordination is successfully completed, the policy statement will be amended to exempt from the reporting requirement information that has been submitted to other specified agencies. In the meantime, substantial-risk information must be reported directly to EPA; such a report does not discharge any reporting obligation to other agencies.

F. INFORMATION FIRST RECEIVED PRIOR TO THE EFFECTIVE DATE OF TSCA

Comment 22: The tense of the verb "obtains" reveals that section 8(e) was intended to be applied prospectively to information newly acquired after January 1, 1977. Utilize section 8(d) or other rules to acquire information obtained before then.

Response: As discussed in the preamble to the September 9 proposal, the Agency considers section 8(e) to apply to risk information possessed by or known to a person before, on, or after January 1, 1977. Concerning information first obtained before 1977, this policy statement continues to require reporting of information received if a person has been aware of it since January 1, 1977, for the reasons discussed in the September 9 preamble.

Comment 23: The term "aware" is too vague to be of any help in responding to these requirements. Since many corporate employees are potentially subject to these requirements, and given uncertainty over the extent to which they ought to be aware of pre-1977 information, this provision tends to compel the very file search it was intended to avoid. The term "aware" should be further defined, possibly in terms of actual knowledge.

Response: The Agency in Part VIII of this policy statement now defines the pre-1977 information of which a person is considered to be aware.

G. CONFIDENTIAL INFORMATION

Comment 24: EPA should delay guidance until procedures are published governing the treatment of confidential submissions.

Comment 25: EPA should treat all submissions as confidential until the information is verified.

Comment 26: EPA should automatically publish section 8(e) notices.

Response to Comments 24 through 26: EPA has included a new Part X which describes how to submit a claim of confidentiality and states that any or all of the information submitted may be claimed as confidential. Such information will be disclosed by EPA only to the extent, and by means of the procedures, set forth in 40 CFR Part 2.

H. MISCELLANEOUS

Comment 27: What is the statutory basis or need for guidance? What is its exact status under the Administrative Procedure Act?

Response: This policy statement sets forth EPA's interpretation of and policy concerning TSCA section 8(e). As an interpretive rule and statement of policy it is not subject to the comment period and delayed effective date provisions of the Administrative Procedure Act (5 U.S.C. 553). Although TSCA does not mandate a policy statement, the Agency of necessity must develop the criteria which will govern enforcement activities. Trade associations and businesses were among those who previously expressed interest in such a statement to guide their compliance.

Comment 28: Clarify whether these requirements apply to chemicals previously but no longer manufactured, processed, or distributed in commerce by a person.

Response: Information obtained before 1977 must be reported if the person has been aware of it since January 1, 1977, as prescribed by Part VIII. Concerning chemicals which a person has discontinued manufacturing, processing, or distributing since January 1, 1977, information obtained before the time of discontinuation is subject to these requirements. It is expected that the acquisition of information after that time will be minimal; however, should additional information be acquired, it may trigger the reporting described in Part VIII.

Comment 29: Clarify the meaning of "substantial risk" relative to other risks addressed by TSCA.

Response: A substantial risk is defined in Part V(a) of this policy statement as a risk of considerable concern because of (a) the seriousness of the effect, and (b) the fact or probability of its occurrence. As opposed to other risks addressed by TSCA, economic or social benefits of use, or costs of restricting use, are not to be considered in determining whether a risk is "substantial".

Comment 30: To what extent are "users" of chemicals subject to these requirements?

Response: The Agency considers that many industrial uses of chemicals actually fall within the scope of "processing" chemicals. A manufacturer, processor, or distributor who obtains substantial-risk information concerning chemicals he handles should be alert to the possibility he may have to report it.

Comment 31: Are chemicals manufactured, processed and distributed in commerce in small quantities solely for purposes of research and development subject to these requirements?

Response: In general, the Agency considers that much manufacturing, processing, and distribution in commerce of chemicals in small quantities solely for purposes of research and development is conducted for "commercial purposes". Such purposes would include the sale and distribution of such materials, as well as their use by the manufacturer or processor in activities (for example, product research and development and studies assessing the feasibility and safety of using chemicals) preceding his or a client's commercial use of such materials or others on a larger scale.

As described in Part V, the Agency considers that "substantial risks" depend in part upon an exposure potential. Thus, the occurrence of the effects described in Part V(a) presuppose exposure to the chemical and must be reported; reporting of the other effects will depend upon a potential for significant levels of exposure.

Comment 32: Are raw materials, intermediates, and inert ingredients produced or used in the manufacture of a pesticide subject to TSCA?

Response: The Administrator considers that raw materials, intermediates and inert ingredients produced or used in the manufacture of a pesticide are substances or mixtures which can be regulated under TSCA.

In order to be considered a pesticide, a substance must be intended for use as a pesticide. Raw materials, intermediates, and inert ingredients produced or used in the manufacture of a pesticide are not themselves regulated under FIFRA (unless they happen to be pesticides themselves) and, therefore, are subject to TSCA. The pesti-

cide regulations at 40 CFR 162.4 are consistent with this view.

Comment 33: Are intermediates and catalysts intended solely for use in the production of a food, food additive, drug, cosmetic, or device subject to TSCA?

Response: The Administrator considers that intermediates and catalysts intended solely for use in the production of a food, food additive, drug, cosmetic, or device are excluded from regulation under TSCA. The definitions of the FFDCA provide that chemical substances which are intended for use as a component of a food, food additive, drug, cosmetic, or device are encompassed within the meaning of such terms, respectively. The FDA considers intermediates and catalysts to be such components. Therefore, they are subject to regulation under the FFDCA. Any such substance is excluded from regulation under TSCA insofar as it is actually manufactured, processed, or distributed in commerce solely for use in the production of a food, food additive, drug, cosmetic, or device.

Comment 34: Employees should have the option to submit reports anonymously.

Response: EPA considers that any person may report information to EPA under TSCA. Those who are required to do so under section 8(e) are persons who manufacture, process, or distribute in commerce chemical substances or mixtures, including not only business entities but also such employees as described in Part II. In order to establish that such persons have discharged their obligations, and in order to encourage responsible review of the quality of information and the substantiality of risks, EPA believes that notifiers should identify themselves. Section 23 will adequately protect employees from discrimination pursuant to notifications they have made under section 8(e).

[FR Doc. 78-7064 Filed 3-15-78; 8:45 am]

EXHIBIT AA

UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF SOUTH CAROLINA
CHARLESTON DIVISION

IN RE: AQUEOUS FILM-FORMING FOAMS (AFFF) PRODUCTS LIABILITY LITIGATION	)	MDL NO. 2:18-mn-2873-RMG
THIS DOCUMENT RELATES TO ALL CASES	)	

THURSDAY, AUGUST 19, 2021
CONFIDENTIAL - PURSUANT TO PROTECTIVE ORDER

– – –

Remote videotaped deposition of 3M Company 30(b)(6) designee Jon Gerber, held remotely at the location of the witness in Cottage Grove, Minnesota, commencing at 9:02 a.m. Eastern, on the above date, before Carrie A. Campbell, Registered Diplomate Reporter and Certified Realtime Reporter.

– – –

GOLKOW LITIGATION SERVICES
877.370.3377 ph | 917.591.5672 fax
deps@golkow.com

factors, and then the potential for widespread distribution and potential for widespread exposure.

Q. Okay. We'll get to that.

But death is a nontrivial adverse effect in Mr. Gerber's opinion, right?

A. Yes.

Q. Okay. So let's keep reading.

Under Pronounced Bioaccumulation it's written, "Measurements and indicators of pronounced bioaccumulation, heretofore unknown to the administrator, including bioaccumulation in fish beyond 5,000 times water concentration in a 30-day exposure or having an N-octanol/water partition coefficient greater than 25,000, should be reported when coupled with potential for widespread exposure and any nontrivial adverse effect."

Right?

A. That's correct.

Q. Okay. Let's try this. True or false: By 1980, 3M was in possession of information that PFOS was a bioaccumulative

compound, that it was widespread in the blood of the general population, and that it killed rhesus monkeys that were exposed to it.

True or false?

A. Based on my review of the documents, 3M had all of -- had those pieces of information, although it -- bioaccumulation, again, I think that's that -- maybe it was the slow elimination rate that was recognized at the time, but all of those informations need -- all of that information needs to be put together and judgment applied in making a TSCA 8(e) reporting decision.

Q. Right. And 3M did that.

3M had all of that information and decided not to disclose it at that time in 1980, right?

A. Yes. I've reviewed documents that -- you know, after the -- those studies were conducted, that information was reviewed against EPA's reporting criteria, and the company made the determination that the information was not substantial risk information under TSCA 8(e).

EXHIBIT BB

Interoffice Correspondence 3M

dc: L.C. Krogh - 223-6SE
J.D. LaZerte - 236-1

Subject: Minutes of Meeting With Dr. G. H. Patterson and Dr. J. H. McClure of duPont

August 3, 1978

THOSE PRESENT:

J. W. BELISLE	201-1S
V. A. BUNNELLE	201-1S
H. E. FREIER	201-1S
D. F. HAGEN	201-1W
J. H. McCLURE	duPont
G. H. PATTERSON	duPONT
R. A. PROKOP	236-3B
S. D. SORENSON	220-2E

A meeting was held on July 31, 1978 with Dr. G. H. Patterson and Dr. J. H. McClure of the Jackson Research Laboratories, Chemical, Dyes and Pigments Department of duPont. Dr. McClure and Dr. Patterson are part of the physical and analytical department of the laboratory and had come to 3M to ask technical questions on the Belisle-Hagen method for determining organically bound fluorine in blood and serum. Their prime concern was over organic fluorine levels in blood of duPont employees in the Chemical, Dyes and Pigment Department.

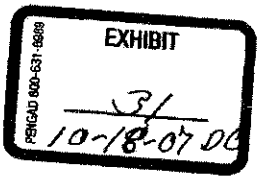
These employees are exposed to duPont fluorochemicals not those from 3M. Possible employee exposure to 3M fluorochemicals at duPont is in the Plastics Department or in the Textiles and Fiber Department. The Textiles and Fiber Department had asked Dr. Patterson and Dr. McClure to obtain a method for analyzing FC-143 in air.

Dr. Patterson and Dr. McClure asked a variety of technical questions concerning the method for analyzing organically bound fluorine in blood. All questions were answered and it was agreed that they could contact J. A. Belisle if further questions arose.

Drs. Patterson and McClure stated that their analytical staff, with the concurrence of Dr. W. A. Sheppard, had confirmed that the F^{19} NMR spectra appearing in the paper by Guy, Taves and Brey was that of a perfluorocarboxylic acid and asked if we had also confirmed this. They were told that we disagreed, but were given no further clarification.

R.A. Prokop 8/4

Submitted by:
R.A. Prokop
/df



3MA00967733

EXHIBIT CC

abundant rainfall. Our results indicate that in the presence of water containing strongly complexing organic species, plutonium can be mobilized to a significant degree as a soluble organic complex that can withstand submicrometer filtration, $Fe(OH)_3$ carrier precipitation, and surface adsorption on sediments. [Means *et al.* (*9*) have described the drastic decrease in K_d values for trace metals in the presence of even very small concentrations of strong complexing agents.] Whether these complexes existed in the waste at the time of disposal or were formed in the trenches after disposal is not known, but it is likely that there are some complexes from both sources. Hence it is important that all organic matter in transuranium wastes be destroyed in order to prevent the formation of stable, potentially mobile complexes of plutonium. Moreover, ground water in the area should be free of strongly complexing ligands. For this reason, it is highly inadvisable to locate a chemical waste disposal site adjacent to a radioactive waste disposal site. Naturally occurring organic substances in ground water appear less likely to mobilize plutonium than organic matter in the wastes. Results of an earlier study (*10*) indicate that plutonium is not appreciably solubilized by fulvic compounds in natural waters.

Although the results of this study indicate that the plutonium is in true solution, a previous investigation (*11*) of plutonium in a pond water indicated that the solubilized plutonium was predominantly colloidal. Hence it is clear that the chemical and physical form of plutonium, and therefore its migration behavior, varies widely with the composition of the water. Subsequent studies with different types of ground water should establish a more precise relation between plutonium speciation and ground water composition.

JESS M. CLEVELAND
TERRY F. REES
U.S. Geological Survey, Denver, Colorado 80225

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12. We thank H. H. Zehner and D. D. Zettwoch of the U.S. Geological Survey for their invaluable assistance in sampling the trenches at Maxey Flats. Special thanks are due to J. L. Means and D. H. Hastings of Battelle Columbus Laboratories for their EDTA analyses, gel filtration experiements, and expert interpretation of the resultant data obtained. The analytical support of V. C. Marti and P. K. Roscio and the sampling assistance of R. H. Fish are also gratefully acknowledged.

3 December 1980; revised 9 Feburary 1981

Organic Fluorine in Human Serum: Natural Versus Industrial Sources

Abstract. *The concentration of organic fluorine in human serum has been reported to vary from 0.0 to 0.13 part per million in persons not exposed to industrial fluorochemicals. To help ascertain whether the natural environment is a source of organic fluorine in human serum, samples from a group of rural Chinese were analyzed. The samples contained low levels of organic fluorine as well as the expected inorganic fluoride.*

It has been observed (*1*) that there are two forms of fluorine in human serum: exchangeable and nonexchangeable with $^{18}F^-$. Research (*2*) has been directed toward identifying the nonexchangeable or organic form. Organic fluorine has been detected in human plasma (*3*); in one study it was not detected in the serum of a variety of animals (*4*), although in other studies it was found in bovine (*5*) and rat (*6*) serum. Since it has been suggested that organic fluorocompounds in human blood are derived from commercial sources (*2*), we analyzed serum from humans who live in a rural area in the People's Republic of China and compared the results with reported values for people in urban areas of the United States.

Several methods have been used to measure total fluorine in serum and plasma. Most samples have been analyzed by open ashing, which causes a variable loss of fluorine (*5*). For example, only 21 percent of the fluorine in perfluorooctanoic acid is recovered as inorganic fluoride by this method (*2*). The use of the closed oxygen bomb technique (*5–7*) avoids most of these losses. With this technique, recovery of fluorine from perfluorooctanoic acid is > 90 percent (*7*). The method yields a mean blank of 0.02 μg, which corresponds to 0.002 part per million (ppm) in a 10-ml serum sample.

Eight samples of human serum were obtained from Chinese donors who live in a rural commune, with little chance for exposure to industrial fluorochemicals. The samples were analyzed for organic fluorine and inorganic fluoride (F^-) by the oxygen bomb method. As shown in Table 1, all the samples from the Chinese contained detectable concentrations of organic fluorine. These concentrations are at the low end of the range compared to those in groups representing a more urban environment. Ash analysis of 65 plasma samples from residents of New York State gave an average value for organic fluorine of 0.03 ppm (lowest value, 0.005 ppm) (*3*). In plasma samples from 106 individuals living in five cities in two states, a mean organic fluorine concentration of 0.025 ppm (ashing) was observed, with two samples estimated to contain < 0.005 ppm (*2*). In plasma samples from 264 people in one Minnesota community, the average concentration of organic fluorine was 0.045 ppm (ashing); one sample contained no detectable organic fluorine (0.00 ppm) (*8*). Ash analysis of a pooled serum sample from Argentinians showed an organic fluorine concentration of 0.085 ppm (*9*). Oxygen bomb analysis of serum samples from nine Minnesota residents gave an average value of 0.02 ppm (lowest value, 0.01 ppm) (*7*).

The concentrations of F^- in the Chinese were slightly higher than the 0.02 ppm reported by Belisle and Hagen (*7*)

Table 1. Concentrations of organic fluorine and inorganic fluoride in eight rural Chinese.

Person	Organic fluorine (ppm)	Inorganic fluoride (ppm)
1	0.008	0.051
2	0.013	0.054
3	0.011	0.046
4	0.014	0.046
5	0.009	0.044
6	0.009	0.049
7	0.004	0.046
8	0.017	0.076

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for a group of Minnesotans. However, in another study (*8*), an average value of 0.058 ppm was reported for Minnesotans. Guy (*3*) reported a mean of 0.015 ppm F^- in inhabitants of New York State, and later showed the concentration of F^- in plasma to be dependent on the level of F^- in the drinking water. Therefore, the slightly higher concentrations of F^- in the Chinese may be due to fluoride in their food and drinking water.

It is difficult to compare reported fluorine values due to the variety of analytical procedures used. Negative factors (such as volatility and incomplete sample decomposition), positive factors [such as contamination with F^- and Freons (*3*)], and the method itself (*10, 11*) influence the reported values. Due to the paucity of values determined with the oxygen bomb method, it is necessary to use results obtained by ashing for comparing levels of organic fluorine in serum.

Many compounds containing organic fluorine have useful industrial and medical applications (*12*); the wide use of these compounds implies widespread exposure to them. Reviews have been written on the role of organic fluorine in biochemistry (*13*), psychiatry (*14*), and toxicology (*15, 16*). The fluoroorganic compounds methoxyflurane and halothane are anesthetics (*17*), and artificial blood contains perfluorocarbons (*18, 19*). Several natural sources have also been suggested (*3*).

In a recent study on the exposure of industrial workers to fluorochemicals (*20*), elevated concentrations of organic fluorine (1 to 71 ppm) were found in the serum of chemical employees handling a specific fluorochemical (ammonium salt of perfluorooctanoic acid, $C_7F_{15}CO_2^-NH_4^+$). It was also found that this fluorochemical is slowly eliminated from the body. Therefore, it appears that blood levels of organic fluorine are dependent on the frequency of exposure to specific fluorochemicals.

If man (*20*), rat, or monkey (*21*) is exposed to ammonium perfluorooctanoate, the compound is subsequently found in the blood serum. This is not surprising when one considers the results of a study on the binding of perfluorooctanoic acid to human serum (*6*): more than 99 percent of this added organic fluorine was bound to serum constituents.

It is clear that nearly everyone (> 98 percent) has both forms of fluorine in his blood and that the reported values are somewhat dependent on the method of analysis. The value for F^- depends on diet and drinking water while the value for organic fluorine could be influenced by exposure to certain fluorine-containing compounds from both natural and synthetic sources.

While it was originally suggested (*2*) that the prevalence of organic fluorine in human plasma is due to commercial sources, there now is evidence that the concentrations have been decreasing over the past 15 years (*2*)—although the trend may be due to the methods used to analyze the blood samples. As yet, we find no conclusive evidence to indicate that the prevalence of trace amounts of organic fluorine in human blood is primarily the result of industrial fluorochemicals. Rather, the main source may be some naturally occurring organic fluorine.

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Central Research Laboratories,
3M Company,
St. Paul, Minnesota 55133

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5 November 1980; revised 19 January 1981

Niacin Reduces Paraquat Toxicity in Rats

Abstract. *Rats poisoned with paraquat benefited from daily niacin therapy. Niacin-treated rats showed delayed and reduced dyspnea. Deaths began approximately 30 hours later. The time required for niacin-treated rats to reach 50 percent mortality increased from 60 to 120 hours, and the death rate was reduced from 75 to 55 percent. The benefit by niacin is consistent with the demonstrated role of niacin in preventing cellular decreases of nicotinamide adenine dinucleotide during poisoning of bacteria by paraquat and by hyperbaric oxygen.*

Paraquat [1,1′-dimethyl-4,4′-bipyridinium (cation) dichloride] is a nonselective postemergence herbicide and defoliant used on a wide variety of crops. Although paraquat is one of the safer herbicides as applied agriculturally, it has caused over 400 human deaths from accidental and suicidal ingestions (*1*). Paraquat also has allegedly caused lung damage to drug users who smoked marijuana obtained from Mexico (*2*). Human fatalities generally are caused by pulmonary impairment, regardless of the method of contact. We have found that niacin is beneficial to rats poisoned by paraquat.

This finding developed from earlier research which had disclosed common sites of damage at the enzyme level in bacteria poisoned by paraquat and by hyperbaric oxygen (*3–8*). The evidence included the discovery that niacin and thiamine were beneficial for the growth of *Escherichia coli* poisoned by hyperbaric oxygen (*5*) or by paraquat (*8*). The mechanism of thiamine protection remains unknown, but there is evidence (*6–8*) that niacin protects *E. coli* because it circumvents the consequences of the poisoning of quinolinate phosphoribosyltransferase. This enzyme is universally required for the de novo synthesis of nicotinamide adenine dinucleotide (NAD); therefore, there is reason to believe that the results may apply to higher life forms.

Consequently, we studied the effects of niacin on paraquat-poisoned rats. The paraquat was given intraperitoneally in two doses of 30 mg per kilogram of body weight, 24 hours apart. Rats that received only paraquat began to die after approximately 30 hours, and 50 percent of the animals were dead by 60 hours (Fig. 1). The group of rats that also received intraperitoneal injections of 500 mg of niacin per kilogram of body weight every 24 hours for 5 days, beginning with

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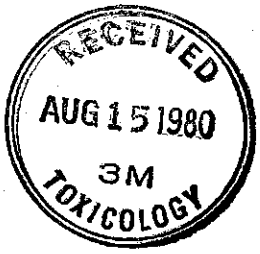
EXHIBIT DD

SOME PROBABLE QUESTIONS ON 3M FLUOROCHEMICALS WITH SUGGESTED ANSWERS



A. ORGANIC FLUORINE IN HUMAN BLOOD

A-1. What is "organic" fluorine in blood?

Fluorine in blood exists in two forms. One form is inorganic fluoride ion. The other form of fluorine is non-ionic in nature and is covalently bound in an organic molecule.

A-2. What is the normal level of organic F in blood?

Reported range is 0.01 to 0.1 ppm.

A-3. What is an elevated level of organic F in blood?

It is not easy to define precisely an "elevated" level. We have adopted 1.0 ppm as an elevated level.

A-4. What fluorochemicals have so far been detected in blood?

Perfluorooctane sulfonic acid, Perfluorohexane suflonic acid, Perfluorooctanoic acid.

A-5. Do you believe other fluorochemicals could be present in blood?

Yes, such fluorochemicals could be derived not only from exposure to synthetic/industrial fluorochemicals (manufactured by several companies) but also from natural sources particularly through the food chain.

Dichapetalem cymosum (Gifblaar), a plant indigenous to South Africa, produces or biosynthesizes fluoroacetic acid, so also do two leagumes growing in Australia, Acacia georginae and Bastrolobium grandiflorum and another plant from South America, Palicourea marcgravii (rat weed). More recently, certain long chain fatty acids containing an -fluoro substituents such as 18-Fluoro-cis-, 9-octandecenoic acid (fluoro oleic acid), 16-fluoro palmitic, 6-fluoro capric acid, and 14-fluoro myristic acid have been identified in plant sources.

Further salad and forage crops, soybean, crested wheat grass, when exposed to high levels of inorganic fluoride in field and laboratory studies, were reported to biosynthesize fluoroacetate and fluorocitrate.

A-6. What relationship do the known fluorochemicals in blood have to the products exposed to or used by the individuals?

In our experience, at 3M plants, fluorochemicals in the blood of workers are related to the fluorochemicals they are exposed to - mostly FC-143 in Chemolite workers and mostly FC-95 in Decatur workers.

A-7. Do other companies' employees have fluorochemicals in their blood? If so are they 3M fluorochemicals or some other companys' fluorochemicals?

We believe that every one has some amount of organic fluorine in his/her blood, but the fluorochemical is not necessarily of industrial origin.

We do not know whether employees of other companies have industrial fluorochemicals in their blood. There are no published reports. To the best of our knowledge, 3M is the first to publish such results on FC levels in blood of the employees. We believe there may be some instances of occurrence of FCs in the blood of employees of other companies also.

A-8. What work have other fluorochemical companies done? What are their findings?

As yet, there is no published information about what other companies have done.

A-9. If 3M fluorochemicals are structurally unique, might the problem involve only 3M fluorochemicals?

We do not know.

A-10. Is there any reason to believe that anyone else's fluorochemicals (i.e., other than 3M's) can be found in blood?

We do not know for sure, but may be.

A-11. Do other companies make fluorochemicals structurally similar to 3M fluorochemicals?

That might be the case, but ----

A-12. How long do fluorochemicals stay in a person's blood?

We have definite information on perfluorooctanoic acid. It may stay for years and excrete slowly. We do not know for certain about other fluorochemicals.

A-13. Does organic fluorine concentrate in any part of the body other than blood? If so, what does it mean?

Blood is the only human tissue/organ studied thus far.

A-14. What is the relationship of fluorochemicals in blood to fluorochemical-containing blood substitutes?

There is some similarity. Both are perfluoro compounds.

B. 3M INDUSTRIAL HYGIENE PROGRAM

B-1. Has 3M told its affected employees what the level of organic F is in their blood? If not, why not?

Yes. They were all informed. Further the results of laboratory tests and physical examinations which formed a part of special health screening program were always sent to a physician of their choice.

B-2. How do the employees feel about having elevated levels of organic F in blood?

We are unable to speak for them, but we have discussed the subject with many of them. They are, of course, interested and some are concerned and we share that concern with them. We have made a concerted effort to keep the employees informed by the use of videotapes, crew meetings and health evaluations.

B-3. How many employees have elevated levels of organic F in blood? How high are the levels elevated?

Sampling is not yet complete. We anticipate elevation in those employees involved with manufacturing and laboratory use of these materials. I would estimate that at Chemolite we are talking about 200 or 300 people.

B-4. Have the F levels in blood of employees dropped with decrease in exposure levels?

In some instances, yes. Removal from exposure seems to result in a decline. We are unable to speak of a direct relationship yet.

B-5. What about the employee's wives and children getting exposure to fluorochemical dust carried home by the employees?

We have no data, but it may be possible. With the current protectional clothing and with reasonable personal hygiene (like after-work showers), this should be minimal.

B-6. How soon can you arrange testing the blood of family members, if the employee so wishes?

Only if there is a good reason for doing it.

B-7. Is there a 3M guideline for exposure to fluorochemicals? What is the recommended safe exposure level?

3M guidelines are no more than 0.1 mg/m^3 in air and no skin contact. It generally takes good engineering controls to obtain these objectives. These are 3M general guidelines, and <u>not</u> based on toxicological or any other data.

B-8. What steps is 3M practicing to minimize or eliminate exposure of the employees to fluorochemicals?

a. Further engineering for complete enclosure of all chemical processes.
b. Personal protective clothing and respirator until the complete implementation of the new engineering steps.
c. Good personal hygiene, e.g., promptly washing off spills or contamination, showers at the end of work shift.

B-9. What steps is 3M recommending to the customers to reduce exposure of customers' employees to fluorochemicals?

Recommendations are contained on the Material Safety Data Sheets, furnished with the product.

B-10. Has the company been implementing its own recommendations in a satisfactory manner?

Yes, progress is being made.

B-11. What is the risk of a new employee coming to work in a fluorochemical area of having an elevated blood level of organic F, since the present implementation of 3M recommendations?

There is a small risk. The risk is less now than before. We have taken precautions to minimize exposure for all employees. This includes proper handling procedures and a continuous tightening of industrial hygiene controls.

B-12. Have we tested the fluorochemical levels in the blood of any new employees since the stricter exposure limits have been set?

We wish to obtain such information, but most of our workers (including our "new"* employees) have some time or other worked in fluorochemical production areas.

*Worked some years ago at Chemolite Plant, were laid off for some time and were reemployed recently by 3M.

B-13. Does exposure to fluorochemicals worsen the health status of employees who are already in a less than good health (i.e., asthma, heart condition, drinkers/smokers, frequent head aches, etc.)?

We have allowed such persons to work in fluorochemical areas. We do not know of any health effect on our employees that would require work restrictions related to the "less than good health" situations. We have been unable to identify any illness, disease or adverse effect that could be associated with the levels of fluorochemicals that we encountered among our employees. Further epidemiology study at Chemolite has revealed no unfavorable trends.

B-14. If fluorochemicals pose a possible health hazard, why don't we stop manufacturing fluorochemicals?

When the presence of organic fluorine in blood was recognized for the first time, the manufacture of fluorochemicals was already approximately 25-30 years old. We were unaware of any health problems at the plant where these fluorochemicals have been produced for all these 30 years. We have not been able to identify any adverse health related problems associated with the presence of fluorochemicals in blood. As with most substances, we feel that they can be handled with proper industrial hygiene controls.

C. PRODUCT INFORMATION

C-1. What 3M products contain perfluorooctanoic acid or its salts?

This is proprietary information. The answer depends on the person seeking the information and 3M's relationship with the person.

C-2. What 3M products contain fluorine?

A relatively small percent (less than 1%). We cannot tell which ones (proprietary information).

C-3. Can you provide a list of all 3M fluorochemical products which we should be concerned about?

The answer is to be tailored to the "need to know". We provide toxicity data on most of our products (MSDS).

C-4. Can you list the products for which there should be no concern?

Proper handling is the key. Such information is provided with the products.

C-5. I am with OSHA. I read about your fluorochemicals in blood studies in a recent issue of AIHA Journal. I recently inspected a carpet mill where they spray FC-XXX. I am making a return visit to do some air monitoring and I would like to know the full composition of FC-XXX including residual monomers. Can you please provide me the information?

Perfluorooctanoic acid is not used in carpet treatment.

C-6. Two days ago I had someone come in and SCOTCHGARD my carpet. My one year old child played on the wet carpet. The next day he did not feel well and I took him to our doctor. The doctor wanted to know what was in the product. The carpet cleaning company said that something called Butyl Cellosolve was present, but had no further information. Can you call the doctor and provide him with the necessary information?

Yes.

D. CUSTOMER SERVICE (QUESTIONS FROM OUR CUSTOMERS)

D-1. Will the use of 3M fluorochemicals result in elevation of organic fluorine in blood of our employees?

We do not know. It depends on which fluorochemical it is and the degree of employee exposure to it.

D-2. Is it safe for our employees to work with fluorochemicals? Is there any danger from inhaling spray or fumes, having skin contact (SCOTCHGARD, SCOTCHBAN, FLUORINERTS, LIGHT WATER). Please answer for each one of the products.

Yes, with proper handling procedures (MSDS) and industrial hygiene controls. Although we have not identified any associated adverse health effects due to exposure to our fluorochemicals, we have continued to lower substantially the potential for exposure. The fluorochemicals can be handled safely. In any chemical industry, general good practices should include avoidance of skin contact, ingestion or inhalation. Questions on toxicology specific to any of 3M products could be addressed to our Toxicology services in the 3M Medical Department.

D-3. What are the maximum permissible (non-hazardous) levels in air of each of the fluorochemicals supplied to us?

Such established threshold limit values (TLVS) or OSHA standards for any of our fluorochemicals have not been defined. 3M has established arbitrarily a 0.1 mg/m^3 limit in air and no skin contact for certain of its materials in 3M plants. With the exception of the FLUORAD Surfactants, most of the other materials these limits apply to are primarily in-house intermediates. Our recommendation of 0.1 mg/m^3 limit is not based on toxicological effects, but it is a rather conservative low level minimal exposure.

D-4. What personal safety precautions do you recommend (respirator type, glove material, clothing material) for our employees? Do you recommend any extra precautions?

These would be highly dependent on the specific material and the mode of use. For the dry surfactants, personal protection precautions should include:

a. Dust-respirator, 3M 9900
b. Impervious gloves. These must be worn and maintained so that dust does not collect inside the glove; otherwise skin contact would be possible.
c. Impervious clothing. Disposable impervious clothing such as Tyvels should be suitable.
d. Rubber or plastic toats to keep the material from contaminating the shoes.
e. Effective local exhaust ventilation to capture and remove air borne contaminants.
f. Enclosed handling system at all possible process steps.
g. Implementation of good personal hygiene habits - prompt washing of contaminated skin areas, washing of hands, arms and face before eating, drinking or smoking; showering at the end of the work shift.

D-5. What control measures to reduce the ambient fluorochemical level would you recommend, which is appropriate to our use and application? (1000 lbs/month inerts evaporated into the atmosphere in a company manufacturing electronic components.)

See answers to question D-4, particularly recommendations e. and f.

D-6. Should we have our employees' blood tested?

We have no basis for making any such recommendation.

D-7. How do we monitor the fluorochemical levels in air and in blood of our employees? Is 3M going to help us?

3M could provide the knowhow (analytical techniques). The extent of 3M help is to be decided on individual basis.

D-8. What precautions should we convey to our customers using SCOTCHGARD treated carpet/fabric/garments?

None.

D-9. Rat studies indicate compounds are more toxic in males. Should I preferentially restrict males to fluorochemical exposure?

This observation pertains to only one fluorochemical and one species and does not extend to other fluorochemicals or other species, including primates. There is not the right-type of information to take the above measures stated in the question.

D-10. We have women working in our production area. Do fluorochemicals have any effects on reproduction (teralogenic or feto-toxic effects)?

Not to the best of our knowledge. Further studies are in progress.

D-11. Are there any other special effects of fluorochemicals on women?

No, not to the best of our knowledge.

D-12. My union syas that they want physical examinations. Do you think that such examinations are necessary?

We have not identified any health hazards in our employees.

D-13. What should we do if one of our workers has a high organic fluorine level in his blood?

We have found no reason to remove the person from his job. Our recommendation would be "reduce exposure" (thoroughly review the process and engineering controls, procedures, work habits and personal protective equipment used).

D-14. Do you think that your products - SCOTCHGARD, SCOTCHBAN, AFFF and FLUORINERTS - would continue to be available? Or, are you planning to discontinue their production.

The products will continue to be available. We see no need for discontinuing any of our products.

D-15. Do you have analytical methods so that we can sample and evaluate exposure levels in our operating areas?

Yes, we have some analytical methods, two of which have been published. We will try to supply the methods.

D-16. Are you going to develop analytical methods for other fluorochemicals?

Yes, method development is an ongoing program in our laboratories.

E. PUBLIC HEALTH ISSUES

E-1. Does the presence of fluorochemicals in blood involve the general public?

Organic fluorine was found for the first time in the blood of the general public. While part of it could be from natural sources (see A-4), a portion of it could be from synthetic fluorochemicals. Guy and Taves suggested the presence of perfluorooctanoic acid in a pooled sample of blood from the general public. It could have been present in the blood of just one person or few persons or more. It is difficult to say how many may have fluorochemicals of industrial origin in their blood. Keeping in mind the possibility that some of the organic fluorine in blood could be derived from natural sources (i.e., non-industrial fluorochemicals) we have obtained and analyzed blood from people in China far removed from any industrial exposure. The blood for these persons also contained traces of organic fluorine. So it would seem safe to say organic fluorine could be present in the blood of all general public. How much of it is from natural sources and how much from exposure to industrial fluorochemicals is not easy to determine, because of the smallness of the amount present and the difficulties in characterizing such small trace amounts.

E-2. What is the relationship between blood fluorochemical levels in the general public and the products used?

We don't know. Because of the low levels of organic fluorine in the blood of the general public it is not possible to characterize or identify the fluorochemical so present. A great deal of progress in analytical methodology is required before data necessary to answer this question can be collected.

E-3. Have studies relating to the general public been planned?

Yes, we are primarily developing analytical methodology required to undertake such studies.

E-4. What are the exposure levels of people residing near 3M fluorochemical plants?

We do not know.

E-5. Is there any danger associated with handling, wearing, sitting on or crawling on fabrics/carpets treated with SCOTCHGARD?

Consumers do not face any health threat, on the basis of information known at this time.

E-6. I am a physician. I have a patient in our emergency ward who had just swallowed LIGHT WATER FC-XXX Concentrate. Should I induce vomiting?

No.

E-7. My two year old son was in the room when I used 3 cans of SCOTCHGARD aerosol to spray my couch. I heard about fluorochemicals in blood and in your product. Must I be concerned that my child may now have fluorochemicals in his blood? What should I do?

Should not be concerned.

F. HEALTH EFFECTS

F-1. Does the presence of fluorochemicals in blood cause cancer?

There is no such evidence. An extensive epidemiology study was carried out, which involved all past employees at one of our large chemical plants covering approximately 30 years. There were no increased deaths in any disease category (including cancer) among the workers engaged in fluorochemical production over the death rates in a control population. Additionally 3M has tested specific fluorochemicals for mutagenecity and recombenogenic effects. The results were negative, suggesting that 3M fluorochemicals have no carcinogenic potential.

F-2. What tests has 3M conducted to determine whether fluorochemicals may cause cancer?

a. Ames Salmonella typhimurium assay for mutagenecity. 5 strains of S. typhimurium were used in these tests.
b. Yeast, Saccharomyces cerevisiae recombinant bioassay.

F-3. Have you conducted lifetime feeding studies in animals to determine if fluorochemicals can cause cancer? If so what are the results? If you have not, why not?

We are considering such studies. We would rather depend on information on humans than on observations on experimental animals. Our epidemiology studies on our plant workers has not revealed any information pointing in the direction of cancer potential of our fluorochemicals.

F-4. Can any of the following ill effects be caused by fluorochemicals? a. blood disease, b. impotency, c. birth defects, d. chromosome damage, e. reproductive effects, f. immonoresponsive effects.

No, not to our knowledge.

G. TOXICOLOGY STUDIES

G-1. Have you studied the toxicological effects via inhalation route?

Some acute inhalation studies have been carried out on some of our fluorochemicals.

G-2. The 28-day mice and rat studies were conducted at Industrial Bio-Test Labs. Have you validated these studies?

Yes, the results are consistent with the 90-day study results (GLP, no).

H. ENVIRONMENTAL ISSUES

H-1. I have heard that fluorochemicals are persistent. Does this mean that they are like PCBs and DDT?

The answer is "NO". PCBs and DDT are environmentally hazardous, because they combine 3 characteristics - (1) they are persistent, (2) they are concentrated in the living organisms and (3) they cause serious toxic effects. As a general rule, highly fluorinated organic compounds, particularly those with completely fluorinated portions are persistent. Like DDT and PCBs these molecules, or at least their highly fluorinated portions, will persist unchanged for long times under typical environmental conditions, but persistence alone does not mean that a compound is an environmental hazard. To be hazardous, the chemical has also to cause some adverse effects. Persistence, however, is a reason to carefully study fluorinated products to demonstrate that they are unlikely to cause undesirable effects at anticipated environmental concentrations. 3M has and is continuing to conduct such studies on their fluorochemical compounds. To date, no evidence exits that a 3M fluorochemical presents an unreasonable environmental risk.

H-2. Do fluorochemicals bioconcentrate?

Not all fluorochemicals bioconcentrate. Laboratory studies using fish have shown that some lipophilic fluorochemicals do concentrate from water into fish. The mechanism appears to be simple partition of these lipophilic compounds into the fatty tissues of the fish. Compounds with high octanol-water partition coefficients are most likely to bioconcentrate in this manner. The 3M fluorochemical having the highest octanol-water partition coefficient studied to date was found to concentrate in 14 days to levels about 200 and 300 times the concentration in water in the case of Channel Cat Fish and Bluegill Sunfish. On return to fluorochemical free water, approximately 95% of this compound was cleared from the storage tissues. Inspite of such a degree of bioconcentration, none of the 3M fluorochemicals were found to exert any toxic effects on fish, fish egges or fish fry (exposed to saturated solutions of the fluorochemicals). In addition, their low production levels make high environmental concentrations unlikely. Thus, although some fluorochemicals bioconcentrate, the data we have indicates that these fluorochemicals do not present any unreasonable environmental risk.

H-3. Are there any special concerns about disposing of process wastes. What procedures do you recommend for waste disposal?

Fluorochemical waste disposal does present some concerns. Waterborne process wastes are likely to resist biodegration, but since it is impractical to remove them from dilute water solutions, 3M normally recommends discharging these solutions to waste water treatment systems. Fluorochemicals so disposed may be removed to some extent by being adsorbed in the sewage sludge, particularly if they have low water solubility. But, water soluble fluorochemicals may pass through the treatment system undegraded. For most fluorochemicals, such a passage is of negligible concern because of their low toxicity. Large discharges of moderately toxic or foam producing fluorochemical products, however, may require monitoring (metering) to avoid adverse effects on both the treatment system and the receiving aquatic environment.

Solid fluorochemical wastes may be either landfilled or incinerated. 3M recommends burying most solid fluorochemicals in a chemical waste landfill. Although many fluorochemicals are nontoxic and it appears that they could be safely buried in a general sanitary landfill, because of their persistence, 3M choses to err on the side of caution. Burying in a chemical waste landfill will prevent problems due to as yet unknown adverse effects.

Incineration of fluorochemical waste requires special care because their combustion products include corrosive and toxic materials such as hydrogen fluoride (HF) and perfluoroisobutylene. Exposure to these combustion products can cause toxic effects to people, animals or vegetation. Being very reactive, they could also speed the deterioration of the incineration equipment. For this reason, incineration of fluorochemical wastes should be avoided except when special facilities designed to handle safely halogen containing chemical wastes are available.

H-4. Do fluorochemicals cause ozone depletion?

Unlike the fully halogenated chloro-fluoroalkanes such as F-11 (CCl_3F), F-12 (CCl_2F_2) and F-113 ($C_2Cl_3F_3$), 3M perfluorochemicals do not contain chlorine. Chlorine, not fluorine, is the element involved in ozone depletion reactions in the stratosphere. Although some of 3M volatile fluorochemicals may be stable enough to reach the stratosphere, they will not contribute to ozone depletion by the known chemical mechanisms of ozone depletion.

J. REGULATORY ISSUES

J-1. If you have known about the condition of elevated plasma organic F levels in your employees four years ago, why have you not notified the government during that time?

We gathered data; we have no legal obligation; there were no untoward health effects.

When we first learned about the elevated plasma levels, the number of persons examined were few, the analytical methods were more involved and time consuming. The total information was meager. There were no health problems. At this point, primarily 3M had the responsibility to generate more data and to investigate the significance of the findings. Toxicology studies in experimental animals were undertaken. An independent epidemiology study was instituted. Results of these studies have been submitted for publication and in the meantime also discussed with representatives of government agencies - OSHA.

J-2. Prior to the publication, has 3M informed the Government (EPA, OSHA, NIOSH, CPSC, FDA, etc.) about the fluorochemical contamination in blood of the general public?

Such findings were already published in scientific journals by the investigators themselves. The information is a matter of public record.

J-3. What was the reaction or comment of the Government on the occurrence of fluorochemicals in the blood of your employees or the general public?

As a representative of OSHA, Dr. Vincent F. Garry, Director, Environmental Pathology Laboratory, University of Minnesota had been in contact with us. He was acquainted with our findings and he made the following comments on long-term human effects. "Indicated in your recent clinical study, there are a number of workers with elevated total fluoride levels with no perceivable noxious health effects. The epidemiologic evidence seems to confirm this notion. In the body of the data it is noted that cardiovascular problems are well below expected." Dr. Garry suggests that we should explore the role of fluorochemicals in minimizing platelet aggregation, etc. "...In a clinical pharmacological sense, then, they could be of some therapeutic value".

J-4. Are the fluorochemical levels in blood currently regulated by any government agency?

No.

J-5. Have you any other information that you have not published? If so, what information do you have? When do you publish such information?

We will publish when we have enough information to justify publication.

J-6. Will you give additional unpublished information in your possession to any government agency, if so requested?

Yes, to an appropriate agency.

J-7. We have been using paper treated with fluorochemicals in direct contact application for food packaging for ten years. I am surprised that an FDA approval was obtained on a product that shows the "effects" on human blood described in your recent publication. Are these findings going to affect the FDA approval?

The publication does not deal with the fluorochemical you now use to treat the food packaging wrapper. The chemical in question is only an "indirect" food additive. This fluorochemical is absorbed very little, even if it is extracted from the wrapper into the food. The information published has no bearing on the FDA approval.

J-8. Did 3M file any information on fluorochemicals under EPA-TSCA 8 (e)?

No, it was not found to be necessary on the basis of inhouse evaluation of the various criteria for reporting under EPA-TSCA 8 (e).

K. LEGAL ISSUES

K-1. What liability is 3M willing to accept for any ill effect that we, the customers, determine now or in the future?

K-2. Does 3M face any law suits as a result of causing elevated levels of fluorine in anybody's blood.