

AR226-1963

MR 286371

Teri K.
Fick/US-Corporate/3M/US

05/12/2005 01:15 PM

To obryan.terry@epa.gov

cc Thomas J. DiPasquale/US-Corporate/3M/US@3M-Corporate

bcc Ephraim Young/US-Corporate/3M/US@3M-Corporate

Subject 8EHQ-1200-14837

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8EHQ-0505-14837

Hello Terry,

I am responding to a voice message that you left with Geary Olsen of 3M today regarding our April 29, 2005 supplemental submission to 8EHQ-1200-14837. In our April 29 letter we indicate that in December of 2000 we submitted a draft report of a mortality study, and that in September of 2001, we submitted the final report on that study.

Your voice message indicates that you do not have that final report in the TSCA 8(e) file.

Today I am sending you a copy of the September 5, 2001 letter to the TSCA Section 8(e) Coordinator where we did submit the final report. I will also include the final report. In that letter, we submitted 13 documents related to previous TSCA 8(e) notifications. All of the submitted documents had also been submitted to TSCA Docket AR-226. In that letter we did not cross reference the original 8EHQ# for the mortality study, which perhaps explains why it is not in your file.

I hope that this clarifies the situation. Please contact me if you have any further questions regarding this submission.

Teri Fick, MPH
220-2E-02
3M Medical Dept
Corporate Toxicology & Regulatory Services
3M IRB Administrator
3M TSCA 8(e) Coordinator
Tel: 651-575-3182
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April 29, 2005

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EPA East – Room 6428 Attn: Section 8(e)
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US EPA
1200 Pennsylvania Avenue NW
Washington DC 20460-0001

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RE: TSCA 8(E) SUPPLEMENTAL SUBMISSION:
Docket No. 8EHQ-1200-14837

Dear Sirs:

In December of 2000, 3M submitted a draft report of a mortality study of 3M employees at a perfluorooctanesulfonyl fluoride (POSF)-based fluorochemical manufacturing facility under the above-referenced docket number. The final report of that study was submitted in September of 2001.

We are now providing a final report on a follow-up incidence study that was conducted to investigate the findings from the prior study.

Please contact Geary Olsen at 651-737-8569 should you have any questions or if we can provide additional information.

Sincerely,

Katherine E. Reed
Staff Vice President
Environmental, Health and Safety Operations

Enclosure

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Document Processing Center (7407)
Office of Toxic Substances
U.S. Environmental Protection Agency
401 M Street, SW
Washington, DC 20460
Attn: TSCA Section 8(e) Coordinator

Dear Section 8(e) Docket Coordinator:

Re: TSCA 8(e) Supplemental Notice on Sulfonate-based Fluorochemicals

With this letter, 3M is providing final reports and other supplemental information related to previous TSCA Section 8(e) notifications. Many of the enclosed items are analytical reports providing blood serum and liver levels of test materials for which the in-life report referring to administered doses has already been submitted to the 8(e) docket. In other cases where the 8(e) notification consisted of preliminary data, we are submitting a final study report.

All of the enclosed items are already in EPA's possession and available in TSCA Docket AR-226. We believe, however, that placing these items in the 8(e) docket may allow for more convenient access to information directly related to previous 8(e) notifications by 3M.

The table below lists the enclosed items and references the study or data which already has been the subject of an 8(e) notification by 3M:

Attached Submission	Related Study/Data Already Filed Under 8(e)
1. Amended Analytical Study, 2(N-Ethylperfluorooctane sulfonamido)-ethanol in Two Generation Rat Reproduction, Determination of the Presence and Concentration of PFOS, M556, PFOSAA, and PFOSA in the Liver and PFOS, M556, PFOSAA, PFOSA and EtFOSE-OH in the Sera of Crl:CDBR VAF/Plus Rats Exposed to EtFOSE-OH, 3M Reference No. T-6316.5, Analytical Report TOX-013, LRN-U2095, June 11, 2001.	Combined Oral (Gavage) Fertility, Developmental and Perinatal/Postnatal Reproduction Toxicity Study of N-EtFOSE in Rats, 3M Reference No. T-6316.5, June 30, 1999, full report submitted February 15, 2000 to supplement earlier filing

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Attached Submission	Related Study/Data Already Filed Under 8(e)
✓ 2. Analytical Laboratory Report, Determination of the Presence and Concentration of Potassium Perfluorooctanesulfonate (CAS Number: 2759-39-3) in the Serum and Liver of Sprague-Dawley® Rats Exposed to PFOS via Gavage, Laboratory Report No. U2006, Requestor Project No. 3M TOX 6295.9, October 27, 1999. 3. Report Amendment 1, Combined Oral (Gavage) Fertility, Developmental and Perinatal/Postnatal Reproduction Toxicity Study of PFOS in Rats, Argus Research Laboratories, Inc., Protocol 418-008, Sponsor's Study No. 6295.9, April 13, 2000.	Combined Oral (Gavage) Fertility, Developmental and Perinatal/Postnatal Reproduction Toxicity Study of PFOS in Rats, Argus Research Laboratories, Inc., Sponsor's Study No. 6295.9, June 10, 1999, full report submitted February 15, 2000 supplementing earlier filing
4. Analytical Report, Determination of the Presence and Concentration of Perfluorooctanesulfonate, Perfluorooctanesulfonylamide, M556, and M570 in the Liver and Sera Samples, 3M Environmental Laboratory Ref. No. U2636, TOX-028, February 23, 2001	13-Week Dietary Study of N-Methyl Perfluorooctanesulfonamido Ethanol (N-MeFOSE) in Rats, 3M Ref. No. T-6314.1, Covance Study No. 6329-225, dated June 30, 2000, Section 8(e) filing July 24, 2000
5. Analytical Laboratory Report, Determination of the Concentration of PFOS, PFOSA, PFOSAA, and EtFOSE-OH in the Sera and Liver of Crl:CDBR VAF/Plus Rats Exposed to N-EtFOSE, 3M Environmental Laboratory Report No. TOX-098, Laboratory Request No. U2402, 3M Ref. No. T-6316.7, February 6, 2001.	Final Report, Oral (Gavage) Developmental Toxicity Study of 2(N-Ethylperfluorooctanesulfonamido)-ethanol in Rats, 3M Reference No. T-6316.7, December 17, 1998, submitted to Section 8(e) docket per letter of August 21, 2000
6. Analytical Laboratory Report on the Determination of the Presence and Concentration of Potassium Perfluorooctanesulfonate (PFOS) or another metabolite of 2(N-ethylperfluorooctanesulfonamido)-ethanol (N-EtFOSE) in Liver and Serum Specimens, 3M Environmental Laboratory Report No. TOX-097, Laboratory Request No. U2452, 3M Ref. No. T-6316.8, February 8, 2001	Final Report, Oral (Stomach Tube) Developmental Toxicity Study of N-EtFOSE in Rabbits, 3M Reference No. T-6316.8, January 11, 1999, submitted to Section 8(e) docket per letter of August 21, 2000
7. Final Report, Alexander, B., Mortality Studies of Workers Employed at the 3M Decatur Facility, University of Minnesota, April 26, 2001.	Preliminary data submitted to Section 8(e) docket in letter of December 15, 2000

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Attached Submission	Related Study/Data Already Filed Under 8(e)
8. Final Report, Acute Oral Toxicity Screen with T-3290CoC in Albino Rats, Safety Evaluation Laboratory, Riker Laboratories, Inc., Project No. 0882AR0362, 3M Reference No. T-3290 (40 % K ⁺ PFOSAA in 3 % EtOH, 17 % IPA and 40 % H ₂ O, L-6778, F-6873, Lot 501), November 5, 1982 <i>[Bibliography entry in Docket AR-226, final report was to be moved to TSCA 8(e) docket]</i>	Acute Oral Toxicity Screen with T-3290CoC in Albino Rats, Safety Evaluation Laboratory, Riker Laboratories, Inc., Project No. 0882AR0362, 3M Reference No. T-3290 (40 % K ⁺ PFOSAA in 3 % EtOH, 17 % IPA and 40 % H ₂ O, L-6778, F-6873, Lot 501), November 5, 1982, submitted to Section 8(e) docket in August 21, 2000 self-audit letter (which erroneously refers to rabbits rather than rats)
9. Giesy, J.P., and K. Kannan, Accumulation of Perfluorooctanesulfonate and Related Fluorochemicals in Fish Tissue, Michigan State University, June 20, 2001. 10. Giesy, J.P., and K. Kannan, Accumulation of Perfluorooctanesulfonate and Related Fluorochemicals in Mink and River Otters, Michigan State University, June 20, 2001. 11. Giesy, J.P., and K. Kannan, Perfluorooctanesulfonate and Related Fluorochemicals in Oyster, Crassostrea Virginica, From the Gulf of Mexico and Chesapeake Bay, Michigan State University, June 20, 2001. 12. Giesy, J.P. and K. Kannan, Perfluorooctanesulfonate and Related Fluorochemicals in Fish-Eating Water Birds, Michigan State University, June 20, 2001. 13. Giesy, J.P. and K. Kannan, Accumulation of Perfluorooctanesulfonate and Related Fluorochemicals in Marine Mammals, Michigan State University, June 20, 2001.	Preliminary data submitted to Section 8(e) docket May 26, 1999

If you have any questions about this submission, please contact me at (651)737-4795.

Sincerely,



Georgean Adams
Manager, 3M Corporate Product Responsibility

Enclosures

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Final Report

Mortality Study of Workers Employed at the 3M Decatur Facility

Submitted by:

Bruce H. Alexander, PhD

Division of Environmental and Occupational Health

School of Public Health

University of Minnesota

Mayo Mail Code 807

Mayo Building

420 Delaware Street S.E.

Minneapolis, MN 55455

April 26, 2001

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Summary

Objective

To determine whether occupational exposure to fluorochemicals exposure, particularly to perfluorooctanesulfonyl fluoride (POSF, $C_8F_{17}SO_2F$) based fluorochemicals, is related to mortality of employees of the 3M facility in Decatur, Alabama.

Methods

All workers with at least one year of cumulative employment at the Decatur facility were eligible for inclusion in the cohort. The cohort was followed through December 31, 1998 and death certificates of decedents were obtained and coded for analysis. The jobs held by cohort members were assigned to one of three exposure subgroups: high exposed, low exposed, and nonexposed, based on their exposure to POSF-based fluorochemicals. Standardized mortality ratios (SMR) with 95 percent confidence intervals were calculated to estimate the all cause and cause specific mortality risks associated with employment at the Decatur. The association between exposure to POSF-based fluorochemicals was evaluated among all exposed workers and workers with a minimum one year of exposure.

Results

A total of 145 deaths were identified in the 2083 cohort members. Sixty-five deaths occurred among workers ever employed in high exposed jobs. The overall mortality rates for the cohort and the exposure sub-cohorts were lower than expected in the general population. Two deaths from liver cancer were observed in the workers with at least one year of high or low exposure (SMR = 3.08, 95% CI=0.37-11.10). The risk of death from bladder cancer was elevated for the entire cohort (3 observed, SMR=4.81, 95% CI=0.99-14.06). All three bladder cancers occurred among workers who held a high exposure job (SMR=12.77, 95% CI=2.63-37.35). The employees who died from bladder cancer were maintenance workers (2) and incinerator and waste water treatment plant operator and process operator (1).

Conclusion

Workers employed in jobs with high exposure to POSF based fluorochemicals at Decatur have an increased risk of death from bladder cancer, however it is not clear whether these cases can be attributed to fluorochemical exposure or an unknown bladder carcinogen encountered during the course of maintenance work.

Introduction

Fluorinated organic compounds have been used for decades in a wide variety of industrial and commercial applications. Perfluorinated compounds are used in applications such as specialty lubricants, semiconductor manufacturing, protective barriers or coatings, surfactants, fire retardants, and non-conductive coolants.

The 3M Decatur (Alabama) manufacturing site began production in 1961. The site consists of two plants: Specialty Film (film plant) and Specialty Materials (chemical plant). The film plant is located approximately 300 yards west of the chemical plant. The major production buildings in the film plant are polyester and non-polyester films.

Logan et al. provide a detailed description of the chemical plant.¹ The three major product groups (referred to as focus factories) in the chemical plant are protective chemicals, performance chemicals and fluoroelastomers. There are more than 700 different processes with more than 90 percent being batch processes. Raw materials and intermediates for each product group may flow through many different production buildings before packaging and shipment. Perfluorooctanesulfonyl fluoride (POSF, $C_8F_{17}SO_2F$) is the major sulfonate fluorochemical manufactured at Decatur and is used as the precursor to the production of a variety of perfluorinated amides, alcohols, acrylates and other fluorochemical polymers. POSF is produced via an electrochemical cell process. POSF-based fluorochemicals can be absorbed into the body and be metabolically transformed, to an undetermined degree, to perfluorooctanesulfonate (PFOS, $C_8F_{17}SO_3^-$). Biological monitoring (serum measurement) of PFOS has been used to assess the employees' exposure experience to POSF-based fluorochemicals at this plant.^{2,3}

Employees may be exposed by one or all routes (i.e., inhalation, skin contact/absorption, and ingestion) to these POSF-based fluorochemical materials. The primary route of exposure may be different for each employee and depends on several different factors such as: process conditions, job tasks, work location, personal hygiene, personal habits and general work practices. Although POSF-based chemicals are the primary fluorochemicals produced at this plant, exposure to other fluorochemicals, including perfluorohexanesulfonyl fluoride and perfluorooctanoate (PFOA), and exposure to non-fluorochemicals are likely

Regarding the toxicology of PFOS, several repeat dose studies have consistently demonstrated that the liver is the primary target organ.⁴ Liver tissue response to high doses of PFOS included the enlargement of liver and apparent alterations in metabolic processes with the reduction in serum cholesterol levels observed as the earliest clinical response to PFOS. These effects occurred in cynomolgus primates at serum PFOS levels at 100 ppm. Also at high doses, PFOS adversely affected survival of rat pups in the neonatal period of life as a result of maternal exposure during fetal development. Reduced weight gain, absorptions and resorptions were observed at the higher doses tested (1.6 mg/kg/day and 3.2 mg/kg/day). There were no effects on post-natal neurological development or on fertility and estrous cycling in offspring in multigeneration studies. Multiple genotoxicity assays suggested PFOS does not present a hazard from interaction with genetic material. Results from a two-year rat bioassay cancer study are scheduled to be reported in 2001.

Medical surveillance findings have not associated abnormal clinical chemistry results with Decatur employees' serum PFOS levels.² A previous mortality study of workers employed at the Decatur plant reported an overall lower mortality rate than expected.⁵ This report summarizes the results of an update of that cohort mortality study with specific emphasis on

those jobs which have the potential for fluorochemical exposure in employee that may result in elevated serum PFOS levels.

Methods

Cohort Enumeration

The cohort for this study was enumerated using employment records from the Decatur facility. Workers who accrued at least one year of cumulative employment at Decatur were eligible for inclusion in the cohort. Work history records were obtained from the plant and reviewed and abstracted into an electronic database. This effort was conducted by 3M staff under the supervision of the 3M Medical Department. The records of all employees were abstracted to record the workers name, Social Security Number, 3M identification number, date of birth, and the dates of any entry on the work history record, including layoffs and leaves of absence. Information about the particular jobs was abstracted wherever available, including the job department codes, and job title. The abstracted data were entered into a computer database and provided to University of Minnesota investigators.

To be eligible for the cohort a worker had to accrue at least 365 days of cumulative employment at the Decatur site by December 31, 1997. The eligibility of each cohort member was determined by summing their dates of employment. Periods of absence due to illness, military leave, maternity leave, or layoff did not contribute to eligibility. Currently employed workers were assigned December 31, 1997 as their last date of employment.

The newly enumerated cohort was linked to records from the original cohort to update employment information, and to verify names, social security numbers, dates of birth, and dates of death for previously identified decedents. Discrepancies identified in the records were resolved using TRW/Experian, a credit reporting agency, and the Social Security Administration

service for epidemiologic research studies. The latter reports the most recent account activity of an individual and whether they are recorded as deceased in the Social Security Death Index.

Follow-up and Determination of Vital Status

Eligible cohort members were followed from the day they accrued 365 days of cumulative employment till December 31, 1998 or their date of death. Vital records searches were performed for all cohort members who were not employed by 3M on December 31, 1997, or for whom a death certificate was not obtained in the original study. Determination of vital status was made by searching the National Death Index (NDI) for all workers in the original study and new workers included in the cohort. The Social Security Administration data and/or the Social Security Death Index (SSDI) was searched to verify the vital status of workers who terminated employment before 1979.

The records of cohort members identified as deceased through the NDI or SSDI were reviewed by hand to ensure a valid match, and a copy of the death certificate was requested from the state of record. The death certificates were coded by a licensed nosologist to the International Classification of Disease Version 8. A second licensed nosologist coded the death certificate using the rules for the ICD version in effect at the time of death. This second coding was used for verification and to enable the use of actual (unadjusted) mortality reference data.

Exposure Assessment

The relative differences in serum PFOS by job have been reported elsewhere.⁶ In this comprehensive assessment conducted in 1998, a total of 232 Decatur employees were randomly

selected for serum sampling with 80% participation (n = 126 chemical plant; 60 = film plant). Sera samples were extracted using an ion-pairing extraction procedure and were quantitatively analyzed for serum PFOS using high-pressure liquid chromatography/electrospray tandem mass spectrometry methods. The geometric mean serum PFOS level (95% confidence interval) for chemical plant employees was 0.94 ppm (0.79-1.13) and for film plant employees it was 0.14 ppm (0.11-0.16). The great majority of film plant jobs have no direct workplace exposure to fluorochemicals. Their serum levels are thought to be due, to a large extent, to environmental exposure in proximity to the chemical plant. Chemical plant jobs were categorized into eight categories: cell operators, chemical operators, maintenance workers (primarily mechanics and electricians), mill operators, waste treatment plant operators, engineers/laboratory workers, supervisors/managers and administrative assistants. The highest geometric mean level of serum PFOS was observed in cell operators (1.97 ppm) followed by the waste operators (1.50), chemical operators (1.48 ppm), and maintenance workers (1.30 ppm). Supervisors/managers (0.89 ppm), mill operators (0.59 ppm), engineer/lab workers (0.39 ppm) and administrative assistants (0.40 ppm) had lower geometric mean serum PFOS levels. Mill operators are entry level positions that usually progress to chemical operators within a few years of hire.

As mentioned previously, exposure to other fluorochemicals, including PFOA, are possible. Until 1998, PFOA was not manufactured at the Decatur facility. Rather, PFOA exposures were the result of it being produced as a by-product of the electrolytic cell production or in its use as an elastomer in fluoropolymer production. The primary route of PFOA exposure would be dermal contact. Although observed at slightly lower levels, the Decatur employees' serum PFOA levels correlated with their serum PFOS levels.³

Because production processes have remained constant over time, a straightforward exposure matrix was developed based on the work history records of the study cohort. With the knowledge of the major job-specific serum PFOS levels, a 3M industrial hygienist and epidemiologist assigned each unique job and department combination in the work history records to 1 of the following 3 major exposure categories:

1. no workplace exposure to POSF-based fluorochemicals (encompasses film plant jobs);
2. low potential workplace exposure to POSF-based fluorochemicals (includes such jobs as engineers, quality control technicians, environmental, health and safety workers, administrative assistants and managers)
3. high potential workplace exposure to POSF-based fluorochemicals (includes cell operators, chemical operators, maintenance workers, mill operators, waste operators and crew supervisors)

Hereafter these three categories will be referred to as the non-exposed, low exposed and high exposed subcohorts. In addition, for the purpose of cumulative exposure for this mortality analysis, these three categories were assigned a relative POSF-based job exposure value of 1, 3 and 10 respectively. All exposure assignments were made without knowledge of the mortality outcomes.

Analysis

The mortality experience of the Decatur cohort was compared to that of the state of Alabama. An additional reference population using regional counties was also used to check the results and

rule out large variations within the state. The twenty three regional counties, Blount, Calhoun, Cherokee, Colbert, Cullman, De Kalb, Etowah, Fayette, Franklin, Jackson, Lamar, Lauderdale, Lawrence, Limestone, Marion, Marshall, Morgan, St. Clair, Shelby, Talladega, Tuscaloosa, Walker, and Winston, were selected for the original study by Mandel and Johnson.⁵ The reference data were obtained from the Mortality Population Data System (MPDS) center at the University of Pittsburgh. These data are derived from National Center for Health Statistics data and provide rates for all causes of death combined and deaths due to malignant neoplasms back to 1940, and non-malignant cause specific death rates from 1962 forward. These reference data are age (5 year), gender, race, and calendar period (5 year) specific, and are coded using the rules for the ICD version in effect for the calendar period.

Standardized mortality ratios (SMR) were computed for all cause and specific cause of death using the Alabama reference data. The SMRs and appropriate 95% confidence intervals were computed using the PC Life Table Analysis System (PCLTAS) software developed by the National Institutes of Occupational Safety and Health (NIOSH).⁷ This program computes age, gender, and race specific SMR using standard life table methods. The expected number of deaths are estimated by multiplying the age, gender, race, and calendar period tabulated person-years of follow-up to the corresponding cause specific reference mortality rates. Race of the workers was not available from the company records, but the population of workers is mostly Caucasian. Therefore, the reference rates for Caucasians were used for this analysis.

The all cause and cause specific SMRs were computed for the entire cohort and the subcohorts of ever high exposure, ever low exposure, and non-exposed. This initial summary analysis was

followed by an exposure specific analysis in which the exposed subcohorts were restricted to workers who had at least one year of cumulative employment in jobs with high or low exposure. A large number of the workers who ever worked in exposed jobs worked in those jobs for a short period before being permanently transferred to a nonexposed job. For this analysis the period of follow-up began when the individual reached one year of cumulative exposure, thereby counting person-time that occurred after the minimum exposure was accrued.

Causes of death potentially related to fluorochemical exposure were analyzed by duration of employment in the three fluorochemical exposure subgroups. The causes of death of a priori interest were cancers of the digestive system, in particular liver cancer, and liver cirrhosis, because, in animals, PFOS circulates through the enterohepatic circulation and concentrates in the liver.^{4,8} Other causes of death that appeared to be in excess in one or more of the fluorochemical exposed groups were also evaluated by duration of exposure.

Results

Of the 3512 workers identified a total of 2083 workers met the one-year enrollment criteria. Of these, 47 percent (982) worked at some time in jobs where exposure to POSF-based fluorochemicals was considered high (Table 1). Approximately 14% (289) worked in low exposure areas, but never held a job in the high exposure areas, and 812 (39%) were considered to have no or minimal workplace exposure to fluorochemicals, i.e. film plant employees. The high exposure group was modestly younger than the other workers, but had a longer average tenure at the plant than the rest of the cohort. Male workers made up 83 percent of the cohort and 84 percent of the high exposed sub-cohort. There were a total of 145 deaths identified in the cohort, 65 deaths in the high exposure group, 27 in the low exposure group, and 53 in the nonexposed group. Death certificates were obtained for 139 (96%) of the decedents. The 6 missing death certificates were in the high exposed group (N=3), the low exposure group (N=2), and the non-exposed group (N=1). A total of 50,970 person-years of follow up was accrued for the entire cohort.

There were 782 workers who worked a minimum of 1 year in high exposure jobs and 1065 workers who worked a minimum of 1 year in high or low exposure jobs. The latter subcohort included workers who held high and low exposure jobs for less than one year, but had a combined exposure period of one year (Table 2). Fifty-three deaths were among those working one year or more in high exposure jobs, and additional 29 deaths were in the combined high and low exposure group.

The all cause and cause specific mortality rates for the entire cohort were lower than expected compared to the general population of Alabama; 145 observed and 230 expected (SMR=0.63, 95% CI=0.53-0.74). (Table 3) A similar pattern was observed for all deaths from cancer; 39 observed, 54 expected (SMR=0.72, 95% CI = 0.51-0.98). The all cause and all cancer causes of death were fewer than expected for the high exposure (Table 4), low exposure (Table 5), and nonexposed (Table 6) subcohorts. When restricted to workers who accrued at least one year of employment in the high exposure (Table 7), and high or low exposure (Table 8) subgroups, the standardized mortality ratios for all causes of death and all malignant neoplasms were well below unity.

Five deaths from cirrhosis of liver were identified in the entire cohort, two of which occurred in the high exposure group; however this did not exceed the number expected. Two deaths from liver cancer were observed in the entire cohort, with 1.24 expected (SMR=1.61, 95% CI=0.20-5.82). One liver cancer was observed in the sub-cohort employed in a high exposure job for at least a year (0.50 expected, Table 7), and the other held a low exposure job for at least one year. The SMR for liver cancer among workers who held high or low exposure jobs for at least one year was 3.08 (95% CI 0.37-11.10) (Table 8).

The details of the two liver cancer cases are as follows.

- Case 1. Died at the age of 85. The cause of death listed on the death certificate was "respiratory arrest" due to "carcinoma of the liver with metastatic disease". The time from onset of the liver cancer to death was recorded as 4 months. The death certificate was coded by one nosologist to ICD8 155.0 (primary hepatocellular carcinoma), and to ICD9 155.2 (malignant neoplasm of the liver not specified as primary or secondary) by the second nosologist. There were no other contributing causes of death entered on the death certificate.

This individual worked as a yard clean-up man and general helper from the mid-1960s through the mid 1970s. Retired at age 65.

- Case 2: Died at the age of 36. The cause of death listed on the death certificate was "hepatocellular ca" and coded to ICD8/9 155.0 (primary hepatocellular carcinoma) by both nosologists. The time from disease onset till death was recorded as 2 months. There were no other contributing causes of death listed on the death certificate. This individual worked in the mid-1970's as a general helper for 2.5 months, a process helper for 7 months, and a material handler for 5 months. He terminated employment at age 20.

Medical validation by follow-back to the physician of record on the death certificate was conducted for the deaths from liver cancer to verify that liver was the primary cancer. Medical records were obtained for both liver cancers and they were confirmed as primary hepatocellular carcinomas.

Three deaths were attributed to malignant neoplasms of the bladder (0.62 expected in the entire cohort, SMR 4.81, 95% CI=0.99-14.05). The workers who died from bladder cancer were in the sub-cohort that worked in jobs with high exposure for at least one year (0.19 expected, SMR=16.12, 95% CI=3.32-47.41, Table 7). All three cases of bladder cancer were male and each had worked in high exposure jobs for at least 5 years (Table 9); the SMR for five or more years of employment in a high exposure job was 24.49 (3 observed, 0.12 expected). The results for bladder cancer in relation to fluorochemical exposure did not change when the reference population was the local counties rather than the entire state of Alabama.

The job classifications of the workers who died from bladder cancer were.

1. Maintenance mechanic, lead man, maintenance foreman, and crew supervisor. Twenty years employment at Decatur 5.3 years in high exposure jobs. First employed in 1960.

2. Maintenance mechanic, general maintenance mechanic; Twenty-three years employment at Decatur, 8.5 years in high exposure jobs, and 1.2 years in low exposure jobs. First employed in 1962.
3. General helper, packer, rewinder, process operator, material handler incinerator and utility operator, and incinerator and waste water operator. Twenty-three years of employment at Decatur. Thirteen years in high exposure jobs, 1.3 years in jobs with low exposure. First employed in 1966.

Discussion

This study evaluated the mortality experience of workers with at least one year of employment at the 3M facility in Decatur, Alabama. The overall mortality rates were below the expected for most causes. An excess of death from bladder cancer was detected and limited to workers who worked in jobs with high exposure to POSF-based fluorochemicals for at least five years. All other causes of death were below the expected or occurred too infrequently to evaluate with confidence.

Some limitations must be considered when interpreting the results of this mortality analysis. Although several methods of follow-up were employed to identify decedents in this cohort, there is the possibility that some deaths were not ascertained. A death certificate was not obtained for 6 of known decedents, thus they were not included in the cause specific death analysis. The extent to which these limitations would affect the results is unknown, but they would have their largest effect on the analysis if the unknown and uncoded causes of death were attributed to relatively rare causes of death, including liver, bladder, kidney, and prostate cancer. Another limitation in this study is the lack of employee specific exposure data for PFOS and other fluorochemicals. The employment records were used as surrogates of exposure. The relative exposure estimates were guided with data from a previous exposure assessment, which reduced misclassification, but did not eliminate it. Finally, the focus of this study was fluorochemical exposure, therefore exposure to other chemicals in the workplace was not assessed.

The current study is an update of a cohort mortality study by Mandel and Johnson⁵. The main difference in the methods used for this study is the more specific exposure assessment. The

earlier analysis divided the cohort solely into chemical and film processing workers. In this analysis an effort was made to separate the chemical workers in the high and low fluorochemical exposure jobs; thus the results are more specific to exposures. There are seven more years of follow-up in this study, which added 71 additional deaths to the analysis. Nevertheless, the cohort is relatively young and the low cumulative mortality experience limits the power of this study to evaluate associations between occupational exposures and employment at the Decatur facility.

The number of deaths from bladder cancer was more than expected based on the prevailing rates for Alabama and the regional counties. The report by Mandel and Johnson also showed an excess of bladder cancer, but was only based on one case, and therefore could have been a chance finding. In the current analysis only three cases were observed, however the fact that all three cases worked for at least five years in the high exposed jobs warrants further evaluation. There are four possible explanations for this occurrence; 1) the cancers are due to exposure to fluorochemicals, 2) the cancers are attributable to another, undetermined occupational exposure, 3) the cancers are related to non-occupational factors such as smoking or other personal habits, or 4) the finding is due to chance. With only three cases the possibility that the finding is due to chance cannot entirely be ruled out. However, given the magnitude of the risk estimate it would take many years of additional follow-up with no further deaths from bladder cancer for this excess to fully attenuate to null.

Preliminary examination of a two year feeding bioassay of with either N-ethylperfluorooctane sulfonamide alcohol or PFOS has not shown an increased risk of bladder tumors.⁴ The former

compound can metabolize to an undetermined, to PFOS. There are no lifetime inhalation bioassay studies for POSF.

A known risk factor for bladder cancer is tobacco smoking.⁹ However, in this cohort, the rates of other smoking related cancers, including lung cancer, were not elevated, suggesting that the cohort as a whole did not smoke appreciably more than the general population. Although conceivable, it is unlikely that smoking alone could account for a thirteen-fold increase in the rate of bladder cancer among employees who ever held a job with high exposure.

In addition to smoking, bladder cancer has been associated with several occupational exposures. Employment in the textile industry in particular has long been associated with risk of bladder cancer due to the use of aniline dyes and other agents. Several chemicals currently or historically used in the textile industry have been classified by the International Agency for Research on Cancer (IARC) as Class 1 (carcinogenic), Class 2A (probably carcinogenic) and Class 2B (possibly carcinogenic). The majority of the chemicals evaluated fall into the 2B category including plasticizers such as acetamide and di(2-ethyl hexyl)-phthalate, flame retardants such as thiourea, tris (2,3-dibromo propyl phosphate), and antimony trioxide.¹⁰ Occupational exposure to aromatic amines in the textile and other industries are possibly responsible for up to 25 percent of bladder cancer in some countries.¹¹ Exposure to polycyclic aromatic hydrocarbons (PAHs) has also been linked to bladder cancer.¹² These associations have been reported in the aluminum industry, and occupations where exposure to coal tar pitch is common. Other occupational exposures possibly related to bladder cancer that are less industry specific, include exposure to

metal cutting fluids, diesel exhaust, polybrominated biphenyls, and perchloroethylene and other solvents.¹³⁻¹⁹

Although the bladder cancer cases in this cohort occurred among workers who held high POSF-based exposed jobs, the specific jobs of the cases suggest that other exposures could have occurred. The bladder cancer cases worked mostly in maintenance or at the incinerator and waste water treatment plant. The exposures in these jobs are not limited to those encountered in a specific chemical production process. Maintenance workers may work in many areas of the plant. The waste water treatment and incinerator workers are on the receiving end of the waste stream, thus they too may have encountered a number of chemical exposures.

Mortality from bladder cancer is relatively low with annual incidence rates are five to ten times greater than mortality rates, depending on age and gender. Clearly this mortality study did not ascertain all cases of bladder cancer in this population. Based on rates of bladder cancer in the U.S. from the Surveillance Epidemiology and End Results (SEER) cancer registry data, 6.9 incident cases of bladder cancer in this population during the follow-up period. Because the population under study is not covered by a cancer registry ascertaining these cases would require direct surveys with living cohort members.

Death from liver cancer and cirrhosis of the liver were a priori outcomes of interest for this study because PFOS is concentrated in the liver.⁸ The occurrence of cirrhosis of the liver did not exceed the expected. There were two deaths from liver cancer in the fluorochemical-exposed subcohorts; approximately three times that expected. Both cases were verified as primary cancers

by medical record follow-back. Though more than expected, these results are difficult to interpret because they are based on only two cases. The case with the highest fluorochemical exposure held a high PFOS exposed job for only 14 months. The other case occurred in an older man who worked as a general helper in departments with low exposure for eleven years. There were no liver cancer cases among the workers with much longer duration of employment in high exposure jobs, thus a dose response relationship could not be evaluated.

Both PFOS and PFOA are hepatic peroxisome proliferators in the rat.^{15,20} A two-year bioassay of PFOA in the diet caused an increase of hepatic, pancreas, and Leydig cell adenomas.²¹ Two-year bioassays of feeding studies of PFOS in rats are near completion.⁴ It is unlikely that peroxisome proliferators are carcinogenic to humans under anticipated conditions and levels of human exposure.²²

Known causes of primary hepatocellular carcinoma include the hepatitis B and C viruses and aflatoxins produced by *Aspergillus flavus* and *Aspergillus parasiticus* primarily in foodstuffs.²³ Hepatitis B virus is thought to be responsible for up to 85% of liver cancers in endemic areas, however the prevalence of infection in the U.S. is relatively low. Other contributing causes may include cirrhosis due to excessive alcohol consumption. Exposure to high concentrations of vinyl chloride is associated with angiosarcoma of the liver; a very rare cancer. However, there are no clear occupational causes of primary hepatocellular carcinoma. While the possibility exists that this finding is not due to chance, the results should be interpreted with caution.

Recommendations

Due to the apparent excess risk of bladder cancer in this population a review of potential exposure to known occupational bladder carcinogens at the Decatur site is warranted.

The relatively young age and small size of the cohort currently precludes a detailed analysis by exposure, particularly for less common diseases. To further evaluate the health of this cohort in reference to the risk of liver and bladder cancer, and continue to develop the understanding of the toxicology of fluorochemicals, continued follow-up and monitoring of this cohort is recommended.

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Table 1. Characteristics of 3M employees with a minimum of 1 year of cumulative employment at the Decatur facility.

	High exposed ^a	Low exposed ^b	Non-exposed ^c	Total
Total	982	289	812	2083
Gender				
M	826 (84%)	204 (71%)	700 (86%)	1730 (83%)
F	156 (16%)	85 (29%)	112 (13%)	353 (17%)
Mean age at follow-up	49.9	53.6	51.5	51.1
Median age at follow-up	49.7	52.9	51.4	50.9
Mean year of birth	1948	1944	1946	1947
Median year at birth	1948	1945	1947	1947
Mean years at Decatur	16.4	13.3	13.7	14.9
Median years at Decatur	16.7	10.4	9.9	13.2
Person-years of follow-up	21867	6823	20304	50970
Deaths	65	27	53	145

a: Ever employed in high exposure jobs in the chemical plant.

b: Ever employed in low exposure jobs, but never a high exposure job in the chemical plant.

c: No or minimal workplace fluorochemical exposure, i.e. film plant employees.

Table 2. Characteristics of Decatur workers with a minimum of one year employment in high exposure and high or low exposure jobs.

	High exposed ^a	High or Low exposed ^b
Total	782	1065
Deaths	53	82
Person years	17108	24231
Gender		
M	668 (85%)	867 (81%)
F	114 (15%)	198 (19%)
Mean age at follow-up	50	51
Mean year of birth	1948	1947
Mean years at Decatur	16.6	15.8
<u>Mean years of exposure</u>	<u>10.5</u>	<u>11.8</u>

a: Employed in high exposure jobs in the chemical plant for at least a year.

b: Employed in high or low exposure jobs in the chemical plant for at least a year.

Table 3. Cause specific deaths and standardized mortality ratios for selected causes of death for all Decatur employees (chemical and film plants combined).

Cause	Observed	Expected	SMR	95% CI
All Deaths	145	230.09	0.63	0.53-0.74
<u>Cancers</u>				
All Malignant Neoplasms	39	54.26	0.72	0.51-0.98
Digestive Organs and Peritoneum	5	9.78	0.51	0.17-1.19
Esophagus	2	1.14	1.76	0.21-6.35
Stomach	0	1.07	0.00	0.00-3.46
Large Intestine	1	3.35	0.30	0.01-1.66
Rectum	0	0.63	0.00	0.00-5.84
Biliary Passages and Liver Primary	2	1.24	1.61	0.20-5.82
Pancreas	0	2.18	0.00	0.00-1.69
Respiratory System	15	21.01	0.71	0.40-1.18
Larynx	0	0.51	0.00	0.00-7.25
Bronchus, Trachea, Lung	15	20.33	0.74	0.41-1.22
Breast	2	1.28	1.57	0.19-5.66
Prostate	0	1.44	0.00	0.00-2.57
Testis and Other Male reproductive Organs	0	0.34	0.00	0.00-10.96
Urinary Organs	3	1.89	1.59	0.33-4.65
Kidney	0	1.26	0.00	0.00-2.92
Bladder and Other Urinary	3	0.62	4.81	0.99-14.06
Malignant Melanoma of Skin	3	1.80	1.67	0.34-4.88
Thyroid and Other Endocrine Glands	0	0.19	0.00	0.00-19.01
All Lymphatic and Hematopoietic Tissue	4	5.68	0.70	0.19-1.80
<u>Non-malignant causes</u>				
Cerebrovascular Disease	5	7.13	0.70	0.23-1.64
All Heart Disease	35	62.53	0.56	0.39-0.78
Nonmalignant Respiratory Disease	1	10.60	0.09	0.00-0.52
Cirrhosis of Liver	5	5.89	0.85	0.27-1.98
Accidents	27	36.65	0.74	0.49-1.07
Motor Vehicle Accidents	15	22.05	0.68	0.38-1.12
All Other Accidents	12	14.61	0.82	0.42-1.44
Violence	10	19.27	0.52	0.25-0.95
Suicides	5	11.42	0.44	0.14-1.02
Homicides	5	7.85	0.64	0.21-1.49

Table 4. Cause specific deaths and standardized mortality ratios for selected causes of death for Decatur employees ever employed in a high exposure job.

Cause	Observed	Expected	SMR	95% CI
All Deaths	65	93.56	0.69	0.54-0.89
<u>Cancers</u>				
All Malignant Neoplasms	18	21.54	0.84	0.50-1.32
Digestive Organs and Peritoneum	2	3.91	0.51	0.06-1.85
Esophagus	1	0.46	2.16	0.05-12.02
Stomach	0	0.42	0.00	0.00-8.75
Large Intestine	0	1.33	0.00	0.00-2.77
Rectum	0	0.26	0.00	0.00-14.32
Biliary Passages and Liver Primary	1	0.50	2.00	0.05-11.10
Pancreas	0	0.86	0.00	0.00-4.30
Respiratory System	7	8.24	0.85	0.34-1.75
Larynx	0	0.20	0.00	0.00-18.51
Bronchus, Trachea, Lung	7	7.97	0.88	0.35-1.81
Breast	0	0.48	0.00	0.00-7.71
Prostate	0	0.49	0.00	0.00-7.53
Testis and Other Male Reproductive	0	0.15	0.00	0.00-25.34
Urinary Organs	3	0.75	4.02	0.83-11.75
Kidney	0	0.51	0.00	0.00-7.21
Bladder and Other Urinary Organs	3	0.23	12.77	2.63-37.35
Malignant Melanoma of Skin	2	0.76	2.62	0.32-9.46
Central Nervous System	0	0.99	0.00	0.00-3.73
Thyroid and Other Endocrine Glands	0	0.08	0.00	0.00-46.23
All Lymphatic and Hematopoietic Tissue	1	2.31	0.43	0.01-2.40
<u>Non-malignant causes</u>				
Cerebrovascular Disease	2	2.76	0.72	0.09-2.62
All Heart Disease	14	24.78	0.56	0.31-0.95
Nonmalignant Respiratory Disease	0	4.00	0.00	0.00-0.92
Cirrhosis of Liver	2	2.46	0.81	0.10-2.94
Accidents	17	15.87	1.07	0.62-1.72
Motor Vehicle Accidents	9	9.60	0.94	0.43-1.78
All Other Accidents	8	6.26	1.28	0.55-2.52
Violence	3	8.38	0.36	0.07-1.05
Suicides	1	4.97	0.20	0.01-1.12
Homicides	2	3.42	0.59	0.07-2.11

Table 5. Cause specific deaths and standardized mortality ratios for selected causes of death for Decatur employees ever employed in a low exposure job, but never a high exposure job.

Cause	Observed	Expected	SMR	95% CI
All Deaths	27	42.43	0.64	0.42-0.93
<u>Cancers</u>				
All Malignant Neoplasms	6	11.47	0.52	0.19-1.14
Digestive Organs and Peritoneum	2	2.02	0.99	0.12-3.57
Esophagus	0	0.22	0.00	0.00-16.91
Stomach	0	0.22	0.00	0.00-16.64
Large Intestine	1	0.70	1.43	0.04-7.94
Rectum	0	0.12	0.00	0.00-30.04
Biliary Passages and Liver Primary	1	0.25	3.94	0.10-21.88
Pancreas	0	0.47	0.00	0.00-7.85
Respiratory System	4	4.60	0.87	0.24-2.22
Larynx	0	0.11	0.00	0.00-33.25
Bronchus, Trachea, Lung	4	4.46	0.90	0.24-2.29
Breast	0	0.40	0.00	0.00-9.33
Prostate	0	0.46	0.00	0.00-7.97
Testis and Other Male Reproductive	0	0.04	0.00	0.00-102.57
Urinary Organs	0	0.40	0.00	0.00-9.25
Kidney	0	0.24	0.00	0.00-15.13
Bladder and Other Urinary Organs	0	0.16	0.00	0.00-23.78
Malignant Melanoma of Skin	0	0.27	0.00	0.00-13.71
Thyroid and Other Endocrine Glands	0	0.03	0.00	0.00-109.79
All Lymphatic and Hematopoietic Tissue	0	1.05	0.00	0.00-3.52
<u>Non-malignant causes</u>				
Cerebrovascular Disease	2	1.61	1.25	0.15-4.50
All Heart Disease	7	12.85	0.54	0.22-1.12
Nonmalignant Respiratory Disease	0	2.64	0.00	0.00-1.40
Cirrhosis of Liver	1	0.93	1.07	0.03-5.95
Accidents	1	4.33	0.23	0.01-1.28
Motor Vehicle Accidents	0	2.50	0.00	0.00-1.47
All Other Accidents	1	1.82	0.55	0.01-3.05
Violence	3	2.34	1.28	0.26-3.74
Suicides	1	1.45	0.69	0.02-3.83
Homicides	2	0.89	2.24	0.27-8.08

Table 6. Cause specific deaths and standardized mortality ratios for selected causes of death for nonexposed Decatur employees, i.e. only worked in film plant.

Cause	Observed	Expected	SMR	95% CI
All Deaths	53	88.31	0.60	0.45-0.79
<u>Cancers</u>				
All Malignant Neoplasms	15	20.45	0.73	0.41-1.21
Digestive Organs and Peritoneum	1	3.72	0.27	0.01-1.49
Esophagus	1	0.44	2.25	0.06-12.51
Stomach	0	0.40	0.00	0.00-9.17
Large Intestine	0	1.27	0.00	0.00-2.92
Rectum	0	0.24	0.00	0.00-15.24
Biliary Passages and Liver Primary	0	0.47	0.00	0.00-7.79
Pancreas	0	0.82	0.00	0.00-4.50
Respiratory System	4	7.90	0.51	0.14-1.30
Larynx	0	0.19	0.00	0.00-19.21
Bronchus, Trachea, Lung	4	7.64	0.52	0.14-1.34
Breast	2	0.39	5.11	0.62-18.45
Prostate	0	0.48	0.00	0.00-7.74
Testis and Other Male Reproductive	0	0.14	0.00	0.00-27.23
Urinary Organs	0	0.72	0.00	0.00-5.13
Kidney	0	0.49	0.00	0.00-7.49
Bladder and Other Urinary Organs	0	0.23	0.00	0.00-16.29
Malignant Melanoma of Skin	1	0.72	1.38	0.03-7.67
Thyroid and Other Endocrine Glands	0	0.08	0.00	0.00-48.41
All Lymphatic and Hematopoietic Tissue	3	2.19	1.37	0.28-4.00
<u>Non-malignant causes</u>				
Cerebrovascular Disease	1	2.62	0.38	0.01-2.12
All Heart Disease	14	23.72	0.59	0.32-0.99
Nonmalignant Respiratory Disease	1	3.81	0.26	0.01-1.46
Ulcer of Stomach and Duodenum	0	0.16	0.00	0.00-22.90
Cirrhosis of Liver	2	2.37	0.84	0.10-3.05
Accidents	9	14.67	0.61	0.28-1.17
Motor Vehicle Accidents	6	8.82	0.68	0.25-1.48
All Other Accidents	3	5.84	0.51	0.11-1.50
Violence	4	7.79	0.51	0.14-1.31
Suicides	3	4.59	0.65	0.13-1.91
Homicides	1	3.20	0.31	0.01-1.74

Table 7. Cause specific standardized mortality ratios for workers employed for at least one year in a high exposure job.

Cause	Observed	Expected	SMR	95% CI
All Deaths	53	73.26	0.72	0.54-0.95
<u>Cancers</u>				
All Malignant Neoplasms	14	16.67	0.84	0.46-1.41
Digestive Organs and Peritoneum	2	3.05	0.66	0.08-2.37
Esophagus	1	0.37	2.73	0.07-15.16
Stomach	0	0.33	0.00	0.00-11.15
Large Intestine	0	1.03	0.00	0.00-3.57
Rectum	0	0.20	0.00	0.00-18.24
Biliary Passages and Liver Primary	1	0.39	2.57	0.06-14.26
Pancreas	0	0.67	0.00	0.00-5.52
All Other Digestive	0	0.06	0.00	0.00-66.45
Respiratory System	6	6.45	0.93	0.34-2.03
Bronchus, Trachea, Lung	6	6.24	0.96	0.35-2.09
Breast	0	0.27	0.00	0.00-13.67
Prostate	0	0.40	0.00	0.00-9.26
Urinary Organs	3	0.59	5.11	1.05-14.93
Kidney	0	0.40	0.00	0.00-9.20
Bladder and Other Urinary Organs	3	0.19	16.12	3.32-47.14
Malignant Melanoma of Skin	1	0.60	1.67	0.04-9.25
Thyroid and Other Endocrine Glands	0	0.06	0.00	0.00-59.68
All Lymphatic and Hematopoietic Tissue	1	1.80	0.56	0.01-3.08
<u>Non-malignant causes</u>				
Cerebrovascular Disease	2	2.14	0.93	0.11-3.37
All Heart Disease	12	19.52	0.61	0.32-1.07
Nonmalignant Respiratory Disease	0	3.12	0.00	0.00-1.18
Cirrhosis of Liver	2	1.93	1.03	0.13-3.73
Nephritis and Nephrosis	0	0.39	0.00	0.00-9.38
Accidents	13	12.46	1.04	0.56-1.78
Motor Vehicle Accidents	6	7.53	0.80	0.29-1.74
All Other Accidents	7	4.93	1.42	0.57-2.92
Violence	3	6.63	0.45	0.09-1.32
Suicides	1	3.93	0.25	0.01-1.41
Homicides	2	2.71	0.74	0.09-2.67

Table 8. Cause specific standardized mortality ratios for workers employed for a minimum of one year in high or low exposure jobs, i.e. chemical plant employees.

Cause	Observed	Expected	SMR	95% CI
All Deaths	82	117.08	0.70	0.56-0.87
<u>Cancers</u>				
All Malignant Neoplasms	19	28.45	0.67	0.40-1.04
Digestive Organs and Peritoneum	3	5.13	0.59	0.12-1.71
Esophagus	1	0.59	1.69	0.04-9.41
Stomach	0	0.56	0.00	0.00-6.60
Large Intestine	0	1.75	0.00	0.00-2.10
Rectum	0	0.33	0.00	0.00-11.24
Biliary Passages and Liver Primary	2	0.65	3.08	0.37-11.10
Pancreas	0	1.15	0.00	0.00-3.21
All Other Digestive	0	0.10	0.00	0.00-38.81
Respiratory System	10	11.20	0.89	0.43-1.64
Bronchus, Trachea, Lung	10	10.84	0.92	0.44-1.70
Breast	0	0.66	0.00	0.00-5.63
Prostate	0	0.88	0.00	0.00-4.21
Urinary Organs	3	1.00	3.01	0.62-8.79
Kidney	0	0.65	0.00	0.00-5.66
Bladder and Other Urinary Organs	3	0.35	8.68	1.79-25.39
Malignant Melanoma of Skin	1	0.88	1.14	0.03-6.32
Thyroid and Other Endocrine Glands	0	0.10	0.00	0.00-37.95
All Lymphatic and Hematopoietic Tissue	1	2.88	0.35	0.01-1.93
<u>Non-malignant causes</u>				
Cerebrovascular Disease	4	3.79	1.05	0.29-2.70
All Heart Disease	19	32.73	0.58	0.35-0.91
Nonmalignant Respiratory Disease	0	5.83	0.00	0.00-0.63
Cirrhosis of Liver	3	2.89	1.04	0.21-3.03
Accidents	15	17.07	0.88	0.49-1.45
Motor Vehicle Accidents	7	10.20	0.69	0.27-1.41
All Other Accidents	8	6.87	1.16	0.50-2.29
Violence	6	9.09	0.66	0.24-1.44
Suicides	2	5.44	0.37	0.04-1.33
Homicides	4	3.65	1.10	0.30-2.80

Table 9. Bladder cancers (observed and expected) with SMRs and 95% CI for Decatur workers ever exposed to PFOS by years of exposure and gender.

Years in High PFOS Exposed jobs	Observed	Expected	SMR	95% CI
< 1	0	0.05	0.00	0-75.56
1-<5	0	0.06	0.00	0-58.13
5-<10	2	0.05	41.46	4.66-149.81
• 10	1	0.07	13.45	0.18-74.88
Total	3	0.23	12.77	2.63-37.35