AR226-3931

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03/15/2010 02:08 PM

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March 15, 2010

FEDERAL EXPRESS

EPA Docket Center, MC 2822T
U.S. Environmental Protection Agency
EPA West, Room 3334
1301 Constitution Avenue, NW
Washington, D.C. 20004

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

To IRIS Database for PFOA/PFOS:

In letters dated April 21, 2004, June 2, 2004, January 7, 2005, and February 7, 2005, we submitted to USEPA preliminary data that had been made public during scientific seminars relating to a study of residents and workers exposed to PFOA. (Extra copies enclosed.) In our February 7, 2005, letter on this topic, we noted that an "article explaining the study and the cancer results in more detail has been peer-reviewed and accepted for publication" and that the article "is expected to be published" during the summer of 2005. As five years have now passed since that letter and the final article has not been made available in published, peer-reviewed literature, we felt it appropriate to clarify the record on the status of that article.

As indicated in our prior submissions, an article addressing the cancer results was peer-reviewed and accepted for publication in 2004. More specifically, the *Archives of Environmental Health* confirmed acceptance of the article for publication (following peer review) in October 2004 and confirmed in December of 2004 that the article was in-press and scheduled to appear in the sixth of the next six issues of that Journal.

Following a seminar in October 2005 during which one of the article's authors revealed which journal was publishing the article, a new Editor was appointed for the Journal and the article was removed from the on-going publication process. Thus, the article was not published, as anticipated.

CONTAINS NO CBI

Although we recognize that, since submission of the article for publication, an independent Science Panel was created, which will be studying in more depth (among other things) cancer among many of these same residents and workers exposed to PFOA, we wanted to make sure the public record was complete as to the scientific basis for the preliminary data and results included with our prior letters referenced above. We are, therefore, attaching a copy of the final, peer-reviewed version of the article that had been approved for publication and was in-press in 2005 from which that preliminary data was derived. We have been informed by the lead author of this article that there is no longer any claim of confidentiality over the document.

Very truly yours,

Robert A. Bilott

RAB:mdm
Enclosures
cc: Gloria Post (NJDEP)(w/ encl.) (via U.S. Mail)
Helen Goeden (MDH)(w/ encl.) (via U.S. Mail)
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Kaye H. Kilburn, Editor-in-Chief

October 8, 2004

James G. Dahlgren MD
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Santa Monica, CA 90403

Dear Jim,

The paper is now acceptable with your approval of the editorial changes that were made to simplify sentences and clarify meanings. Please go through these carefully and be sure they are all correct. Change then if necessary. Also, please make other changes you wish as you consider these.

Then two copies and the corrected disk should be forwarded to me.

Sincerely,

Kaye H Kilburn

Kaye H. Kilburn MD
Editor-in-Chief,
Archives of Environmental Health

PREVALENCE AND INCIDENCE OF CANCER IN POPULATIONS EXPOSED TO PERFLUOROOCTANOATE (PFOA)

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Short Title: CANCER PREVALENCE AND INCIDENCE GIVEN PFOA EXPOSURE

Abstract:

PFOA is a bio-persistent man-made chemical that is widely used and widely spread in the environment. Previous worker studies have suggested increased rates of prostate, kidney, bladder and other cancers. This study examines the cancer prevalence rates in a cross-sectional sample of residentially PFOA-exposed adults (578 subjects). There were 50/578 (8.65 %) cancers other than skin cancer in this population compared to 3.43% expected using age-adjusted US population prevalence rates for Whites. The all cancer age-standardized prevalence morbidity rate ratio (PR) ranged from small or none in the highest age categories and increased in each decreasing age category for both genders with the exception of 30 – 44 year old males where there were no reported cancers in the study population. Statistically significantly increased PRs for all cancer (2.58), uterine/cervical (33.12), multiple myeloma (15.71), lung (7.89), non-Hodgkin's lymphoma (6.67) and bladder (5.30) were found comparing the residentially PFOA-exposed population to national figures. Increased rates of colon/rectal and prostate cancer was also suggested with a PR of 2.65; CI = 0.99; 7.11) and 1.96; CI = (0.98; 3.92), respectively. Cancer incidence from an occupationally exposed population of 3394 adults is also considered; and the test for trend of increasing cancer with increasing exposure time was statistically significant with p-values all less than 0.05 (bladder, kidney and prostate). PFOA appears to increase cancer in humans.

Key words: perfluoroalkanes, C8, PFOA, cancer, environmental, questionnaire, incidence, prevalence

Introduction:

Perfluorooctanoic acid (PFOA or C8) is a man-made chemical with numerous industrial and consumer uses. Many stain resistant treatments of upholstery, carpet and textiles release PFOA into the environment.[1] Although PFOA is not an intended component of these treatments; fluorotelomer-based products like these degrade to PFOA and similar compounds. Fluorocarbon chemicals are also used in paper and plastic oil-resistant treatments, as well as in electrical insulation, fire foam, floor polish, insecticides and Teflon®.[2]

PFOA does not readily degrade in the environment[3, 4] but instead accumulates in air, soil and water.[5,6] Potential pathways for human exposure include food, water and leaching from commercial products such as food packaging, contact with products such as clothing, and inhalation of dust or vapor phase precursors coming off of household products, such as carpet.[7,8,9,10,11] PFOA is slowly eliminated from the human body.[11] Bioaccumulation heightens concern about adverse health effects.

In rodent studies PFOA and related compounds cause multiple adverse health effects, [12] including neoplasms of the liver, testes, pancreas, and mammary glands.[13] In humans PFOA increases cancer rates.[14] Human epidemiological studies of PFOA exposed workers have reported statistically significant elevations in prevalence of cancer in the prostate, [15] kidney, [16] bladder, [16,17] colon, [16] testes, [16] and other cancer types.[16] Also there have been excesses of lymphoma, multiple myeloma, breast cancer and melanoma reported.[18]

We report the cancer prevalence of a largely White residentially PFOA-exposed population using a self-administered questionnaire and compare that to the cancer prevalence in the U.S. population of Whites. Incidence data from an occupationally exposed population followed for over 50 years is also presented.

Methods

Residential data

We administered a questionnaire to volunteers from a population of approximately 23,900 households near a manufacturing plant located along the Ohio River in Wood County West Virginia (Table 1). PFOA has been used at this plant since the early 1950s. The Plant uses PFOA to manufacture Teflon® and other materials and has consistently released the substance in the local surrounding areas. According to the Environmental Working Group (EWG), in 1999 alone, the Plant released over 40 tons (86,806 pounds) of PFOA into the air and the Ohio River.[19] EWG also reports that the Plant reports it has reduced emissions to 10 tons (20,168 pounds) in 2002.[19] PFOA has been present in the Plant's drinking water at 1-5 parts per billion (ppb) (Figure 1). PFOA levels above 0.05 ppb have also been detected in public and private drinking water supplies in communities on both the Ohio and West Virginia sides of the Ohio River near the Plant, including the water supply of a neighboring manufacturing plant (Figure 1).

Study participants were recruited through public invitation via TV, radio, and newspaper advertisements inviting all users of the PFOA-contaminated public and private drinking water to participate in a study by filling out a questionnaire. Over a period of three days in August of

2003, five hundred and ninety-nine (599) volunteers (2.5%) from the targeted population participated in the study. Each person who filled out a questionnaire reported exposure to PFOA-contaminated water for at least a year. Although this could include subjects who used water with PFOA levels as low as 0.05 ppb, the majority (65%) who participated were exposed to water provided by the Lubeck Public Service District in West Virginia where PFOA ranged from 0.4-3.9 ppb and the Little Hocking Water Association in Ohio where PFOA ranged from 1.7-4.3 ppb (Table 1). Some study subjects worked in the Plant where PFOA was used and had residential and occupational exposures.

All participants between the ages of 20 and 80 years of age are included in this analysis. Cancer and U.S. population statistics are available in 5-year categories, with the "adult" category beginning at age 20. Only 4 persons older than 80 were surveyed. For these reasons, we restrict all analyses to those between the ages of 20 and 80.

Because a mandatory non-opt-out class action was certified against the Plant on behalf of all persons whose drinking water was contaminated with PFOA (>0.05 ppb), all of our study participants were mandatorily included in a law suit regardless of their individual choice to be included.

A sample question from the health questionnaire is set forth in Figure 1. Questionnaire responses were machine-readable,[20] scanned on-site and verified before the subjects left the test site. All subjects who reported cancer were contacted through a follow-up phone conversation to

verify the cancer information. Medical record confirmation is underway but not yet completed.

Occupational data

We also report on the incidence of cancer mortality among a historical cohort of workers at the Plant. The occupationally PFOA-exposed cancer data were obtained through discovery in the above-mentioned litigation. These data are publicly available through the Environmental Protection Agency.[16]

Cancer mortality is reported from 1959 through June 2003 among the cohort. Many of the older persons included did not have vital status data even though they would be well into their hundreds (years old). For this reason, we only include persons hired between 1950 and 1990 in our analyses. This maximizes the potential that, for those included in the analysis, some attempt was made to document their disease experience. Even in this restricted cohort, there are many subjects for whom no outcome data is recorded even though their current age if still living would be well over 100. We retain these subjects in our analysis with the understanding that the lack of follow-up data will result in measures of effect biased toward the null. This provides the reader with the most conservative measures of effect.

While working, all Plant employees had access to the same PFOA-contaminated drinking water; however, workers in the chemical division were frequently exposed to higher PFOA air levels while others at the plant were not.[21] For this reason, we compared the cancer incidence of those working in the chemical division to those working in another manufacturing department where

there was no direct work with PFOA performed. We also repeated the dose-response analysis including *all workers* hired between 1950 and 1990 who worked at the company for at least 6 months. The results from the restricted cohort were more conservative and are, therefore, presented in this paper.

Statistical Methods:

Residential data

Frequencies and percents for demographic, social, behavioral and physical characteristics are presented. For analyses where all cancers are grouped together, we counted only the *first primary* malignancy reported. For analyses where cancer types are reported separately, we included all *primary* malignancies.

Unadjusted odds ratios (OR) and confidence intervals were estimated using logistic regression for each variable using primary cancer other than skin as the outcome variable: (cancer: yes/no). Test for trends were estimated for all ordinal variables with greater than two categories.

Epidemiological cancer morbidity prevalence rates for the U.S. population of Whites were obtained from the SEER 2000 website.[18] SEER data provides an estimate of overall cancer prevalence and incidence of all types of cancer, other than skin cancer (except melanoma), by type, age and gender. Cancers represented in the SEER data are classified according to their primary site or origin regardless of where in the body they may eventually spread. For example, a lung cancer that spreads to the lymph nodes is still classified as lung cancer and not as a

lymphoma (cancer of the lymph nodes). The SEER Registries routinely collect data on cancer patient demographics, primary tumor site, and follow-up for survival status.

We calculated internally standardized morbidity prevalence ratios (PR) for all cancers.[22] These rates were compared with the rates in the U.S. population of Whites using the age distribution in the exposed population.[23,24] Similar estimates are constructed for each cancer type comparing the exposed population to the U.S. population. The internal standardization takes into consideration the differences in the age distribution between the exposed study population and the comparison population (in this case, the US population of Whites) where we derive our expected number of cancers. In effect, this measure is the number of cases that actually occurred in the exposed population (standard) divided by the number of cases that would have occurred in the absence of exposure. The latter number is estimated by assuming that the exposed group would have experienced, in the absence of exposure, the same stratum-specific risks experienced by the unexposed group.

Occupational data

Frequencies and percents for demographic and work characteristics are presented. For cancer-specific and all cancers analyses, we included all *primary* malignancies.

Proportional hazards models for selected cancers were used to compare survival times between workers directly using PFOA compared to those who did not directly work with PFOA. Hazard ratios, confidence intervals and p-values are presented.

PFOA dose-response relationships were assessed for each cancer type using logistic regression to adjust for age at hire while estimating odds ratios for increased rate of cancer comparing those who worked less than 21 years with those who worked between 21 – 29 years and those who worked greater than 29 years at the company. Test for trend is reported. The "years of exposure" variable was calculated by counting the years between hire date and cancer date, if cancer happened before termination. If cancer was after termination, we added years from date of hire to date of termination. If the person had no date of termination, but has a date of death, we add the years between date of hire and date of death. If date of termination is not missing and there is no cancer, we add from date of hire to date of termination. If there is no date of death and no date of termination we add from date of hire to July 1, 2003, the last documented date for follow-up in this cohort.

All statistical analysis was performed using SAS 8.0.[25]

Results

Residential data

The questionnaire was administered to 599 subjects. Fifteen (15) of the subjects were less than 20 years old and 4 were greater than 80, two (2) subjects were missing age and were therefore excluded from the analyses. Of the 578 subjects between the ages of 20 and 80, 77 subjects answered yes to the question "Have you ever been told by a doctor that you have cancer?" One of the 77 respondent's cancer diagnosis was not present on medical record verification. Of the

remaining 76 subjects, 41 reported one primary malignant cancer other than non-melanoma skin cancer, 8 individuals reported two primary malignancies that included at least one primary malignancy other than non-melanoma skin cancer, one person reported 3 malignancies that included one primary malignancy other than non-melanoma skin cancer and 23 reported skin cancer only. Four primary malignancies were diagnosed prior to reported exposure and were therefore excluded from the numerator and included in the denominator. In total, there were 50 primary malignancies other than skin cancer in the residentially PFOA-exposed population for an all cancer prevalence rate of 50/578 or 8.65 % compared to 3.43 % that were expected to occur in this residential population had they experienced the same age- and gender-specific rates as in the U.S. population of Whites.

Each subject's cancer status was confirmed with the respondent by telephone. Medical record confirmation of the 50 primary malignancies other than skin reports is in progress. To date, 26 (52%) requested medical records have been obtained, all confirmed the self-reported cancer diagnosis for primary malignancies other than skin.

Table 1 shows the water source distribution of the cross-sectional study population compared to the total population in the area. A greater percentage of study subjects consumed water from Little Hocking and Lubeck compared to the total population in the area. Those who consumed water from either or both of the two plants in the area also appear to be over-represented, however, the number of workers in the table represents current workers and the number of study participants is computed by counting all the participants who responded that they had ever

consumed water from either of the plants so this may be a fair representation of the proportion of people who have ever worked at one of the plants in the area.

Table 2 presents the demographic, social, behavioral and physical characteristics of the exposed population. The age distribution was about equal in one 15-year and three 10-year categories ranging from age 20 to 64. Fewer participants contributed to the oldest age category. The average age of the PFOA-exposed residents was 49 +/- 14.5 years. Gender was evenly split and nearly all of the participants were White. The majority of participants were high school graduates or had some college or vocational school. Nearly half of the study sample was overweight with a body mass index greater than 28 meters per kilograms squared. Sixty (60) percent of the sample never smoked and roughly 20 percent were represented in each of the two smoker categories: less than 15 years, greater than 15 years. Ten percent (10%) worked at plant 1 at some point in their life.

Table 3 presents the number and percent of all cancers in each demographic sub-category excluding skin cancers and including only the first reported primary malignant cancer for the 50 subjects who reported having cancer other than skin. Unadjusted odds ratios and 95% confidence intervals for each variable is presented comparing all cancer rates within each category (Table 3). The referent categories were chosen with the assumption of least risk. As expected, increasing age is associated with increased risk of cancer (test for trend: $p = 0.0002$). Increasing years smoking appears to be associated with increased risk of cancer (test for trend: $p = 0.16$). The lowest education level, less than 9th grade, is associated with the greatest risk of cancer. There is

no effect of body mass index on the all cancer rate in this group. The association between working in the Plant or the neighboring manufacturing facility and increased risk of all cancer did not reach the level of statistical significance, however, the Plant employees had a nearly 2-fold odds of cancer compared to those who never worked at either location, p-value = 0.13.

Next, Table 4 compares the all cancer prevalence in the PFOA-exposed residents to the cancer prevalence in the U.S. population of Whites by age and gender. The ratios in each age category for both males and females increase with decreasing age category culminating in the youngest age categories showing the largest effects with the exception of males aged 35 – 44 where there were no cancer cases reported in the exposed population.

Table 5 presents the age-standardized morbidity prevalence ratio (PR) calculated from the observed and expected rates for each cancer type reported in the exposed population. The U.S. population rates for Whites are used to generate the expected age-adjusted rate per 100,000 using the age distribution in the exposed residents. The all cancer PR is significantly elevated at 2.58. Statistically significant increases in PRs were found for uterine/cervical cancer in women (33.12), lung (7.89), non-Hodgkins lymphoma (6.67) and bladder (5.30) cancers. We also found excess morbidity for several different cancer types among the residentially PFOA-exposed population although several of the estimates are based on small numbers including colon/rectal (4), multiple myeloma (2), melanoma (3) and kidney (1). Prostate in men was also nearly significant with a PR of 1.96 and confidence limits that just include 1.00 at the 0.05 significance level (CI: 0.98 – 3.92).

Occupational data

In Table 6, we introduce the occupational data from a cohort of PFOA-exposed workers who were hired between 1950 and 1990. There were 451 identification numbers in the data set with no other associated data. The remaining data set had 6105 workers with some occupational and health related data. From this total, we excluded 409 who were hired prior to January 1, 1950; 1442 who were hired after December 31, 1990; 160 who worked for company less than 6 months and 700 who were in divisions other than the two divisions that we know either did or did not work directly with PFOA. This left 3394 subjects who are represented in tables 7 and 8.

This cohort of workers was largely born between 1950 and 1969 (51.79%). The next largest birth cohort was between 1920 and 1959. Most were male (84.23%) and had direct exposure to PFOA (60.85%). The cohort was equally divided among the three "years of exposure" categories (less than 21 years, between 21 and 29 years and 30 – 50 years). Insufficient information on smoking status, body mass index, race or education level was available.

Table 7 uses proportional hazards models to control for age at hire while examining type-specific cancer rates among workers directly exposed to PFOA through their work compared to workers at the same plant who did not directly work with PFOA. In this survival analysis, pancreatic, respiratory, kidney, colon/rectal and prostate cancers were all significantly associated with direct PFOA exposure: hazard ratios ranging between 2.51 and 4.46. Breast, non-Hodgkin's

lymphoma, bladder and liver cancers did not show association with PFOA exposure in this analysis.

Table 8 examines these same data using logistic regression to explore a dose-response relationship between increased exposure and increased risk of cancer. Age at hire and division are included in the model to control for confounding. Prostate, kidney and respiratory (nearly) cancers have a dose response relationship with number of years exposed. Although bladder and pancreatic cancers show increasing odds ratios with years worked, the p-value exceeds 0.05. When we reran the analysis for table 8, including all who worked at least 6 months *in any department* hired between 1950 and 1990; every cancer-exposure relationship except for respiratory cancer was strengthened. We present the more conservative estimates.

Discussion:

Almost every person in the U.S. has measurable quantities of PFOA in his/her blood.[26, 27,28] Many studies in animals and humans suggest an increased risk of cancer with PFOA exposure. In particular, rodent studies indicated neoplasms in the liver,[29] testes, pancreas and mammary glands.[13] Human studies have suggested increased rates of prostate,[15] kidney,[16] bladder,[16,17] colon[16] and testes[16] cancers. The data presented in this paper are consistent with the prior findings.

Residential data

Similarly for both men and women, we found that the prevalence of cancer was elevated for

almost every age category compared to the U.S. population of Whites. Of particular interest and importance, the rate ratios were higher for the younger age categories suggesting that PFOA exposure not only caused more cancer but did so preferentially in younger populations.

Many of the cancers previously identified in the literature as suspected outcomes of PFOA exposure were also represented in this residential sample including uterine/cervical, bladder, prostate and colon/rectal cancers. We interpret these PRs as indicators of potential increased risk and not as effect measures describing the strength of the association. It is the consistency of these findings with previous animal and human studies that strengthens the conclusion that there is a measurable and important relationship between PFOA exposure and these cancer outcomes.

Lung cancer and non-Hodgkin's lymphoma also show large PRs (7.98; 6.67, respectively), each with confidence intervals that show significance at the 0.05 level. These two findings clearly call for additional research. We know that inhalation of dust or vapor is a potential pathway for human PFOA exposure.[10] Could it be that PFOA causes lung and other respiratory cancers? Given the dramatic rise in incidence of non-Hodgkin's lymphoma[18] over the past several decades any research question regarding causation is worth pursuing. Could ubiquitous bioaccumulation of PFOA be adding to the increased risk of non-Hodgkin's lymphoma?

Occupational data

The occupational data support the residential data, especially for males. Kidney, prostate and colon/rectal cancers were elevated in the proportional hazards models with significant p-values

but not bladder cancer. Respiratory cancer has a strong hazard ratio and very small p-value.

The dose-response analysis also indicates that the odds of prostate and kidney cancers increase with increasing years exposed at the company. Again, respiratory cancer approaches significance. The reported analysis is done with a restricted data set. When repeated including all workers in any department hired between 1950 and 1990 who worked at least 6 months all the odds ratios increase and the p-values decrease with the exception of respiratory. Conservatively, a causal association between PFOA and prostate and kidney cancers is supported with these data from an occupationally exposed cohort.

Despite varying methods for data collection and analysis, increased cancer rates (both prevalence and incidence) were similar for PFOA exposed residents and workers for prostate and colon/rectal cancers. Kidney cancer is also indicated in both data sets but based on only 1 case in the residential. Increased rates of respiratory cancer are a new finding in both data sets that demands further investigation.

The limitations of the data from the residential study include small sample sizes for rare cancers, lack of a true unexposed comparison group and potential self-selection bias of study participants. We reduce the limitation of the small sample size by analyzing the risk of all cancers and limiting interpretation to common cancers. National statistics estimated using SEER data address the need for a comparison group however, it is not possible to control for potential confounding from variables other than age and gender using these data. Possible selection bias

was examined by evaluating the effect on all cancer prevalence of commonly known and well-understood risk factors: age, gender, smoking status and educational attainment. The all cancer rate increased with increasing age, increased smoking and for those in the lowest category of educational attainment. If these data reflected self-selection bias or reporting bias we would expect one or more of these variables to behave outside the norm.

Furthermore, the distribution of cancer types parallels that seen in PFOA exposed workers. Even if there were a selection bias with a disproportionate number of patients with cancers, there is no reason to assume that only patients with certain types of cancers would be more likely to volunteer for the study. Our study results are consistent with the literature on PFOA that suggests increased rates of uterine/cervical, prostate, bladder and colon/rectal cancers. The only unexpected result was for the respiratory that mirrored occupational data and is biologically plausible.

Some researchers question whether reliable data can be obtained from participants in a class action lawsuit. While it is claimed that litigants exaggerate their symptoms we know of no evidence that this is true, particularly when examining averaged group responses. A study by the Agency for Toxic Substance and Disease Registry (ATSDR) revealed no evidence of "litigation bias" among subjects being followed for health effects from trichloroethylene in their drinking water[30] with no statistically significant difference in the validity of the survey responses from litigant versus non-litigant populations. They also report "litigants are no more likely than non-litigant(s) to provide inaccurate or exaggerated responses."

Observational bias is unlikely to influence these results because the techniques used to collect the data do not require interpretation and all cancers were verified by telephone interview. Medical records are being pursued to compare to questionnaire responses. With nearly half of the medical records received all of the reported primary malignancies have been confirmed. The questionnaires were filled out in a neutral environment supervised by trained proctors. All subjects were given the same instructions. Moreover, the study participants are members of a mandatory, non-opt-out class action where their participation in the lawsuit is not voluntary. Recall bias is also unlikely to affect the results, particularly when it comes to cancer.

Increased prevalence rates of reproductive, kidney and bladder cancer are also seen in PFOA exposed workers. The excess of reproductive cancers (i.e. cervical/uterine and prostate) is also consistent with the observation of altered reproductive hormones among PFOA exposed workers.[15] Animal studies with ingested PFOA support the findings in this study.[16]

The increased risk of respiratory cancer in the residential data with a PR of 7.96 is similar to the occupational data with a hazard ratio of 4.65 for direct PFOA exposure. And there is a dose-response p-value of 0.08 for increasing odds of respiratory cancer with increasing categories of years exposed at the company. No published worker studies have reported increased risk of respiratory cancers. However, extensive animal data on PFOA toxicity through inhalation supports and strengthens this finding.[13] These data show an association between increased cancer prevalence in a human population and exposure to PFOA. Because of wide spread human

exposure to this compound these findings are important for public health

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Acknowledgements:

The Law Firm of Winter Johnson & Hill PLLC, Law Firm of Taft, Stettinius & Hollister LLP, Law Firm of Hill, Peterson, Carper, Bee & Deitzler, PLLC.

Human Subjects: The study was conducted in accordance with institutional guidelines for the protection of human subjects.

Conflict of Interest: The above-mentioned law firms funded this study; the first author of this manuscript is sometimes invited as an expert witness.

Table Legend:

Table 1. PFOA levels by Water District/source and percent of study subjects reporting water consumption from each source.

Table 2. Numbers and percents for demographic, social, behavioral and physical characteristics in a residentially PFOA-exposed population.

Table 3. Estimated unadjusted odds ratios (OR) of cancer (yes/no) and 95% confidence intervals (CI) for demographic, social, behavioral and physical characteristics in a residentially PFOA-exposed population.

Table 4. All cancer prevalence rates per 100,000 in PFOA-exposed residents by age and gender compared to U.S. population rates for Whites.

Table 5. Standardized Morbidity Prevalence Ratio (PR) comparing observed cancer rate per 100,000 among a residentially PFOA-exposed population to the expected cancer rate using the age-specific rates of the U.S. population for Whites and the age distribution of the exposed population.

Table 6. Numbers and percents for demographic and work characteristics in an occupationally PFOA-exposed population.

Table 7. Estimated test for trend of cancer incidence with increasing years worked at the company using adjusted odds ratios (OR) and 95% confidence intervals (CI) for selected cancers in an occupationally PFOA-exposed cohort hired between 1950 and 1990: results of a logistic regression analysis controlling for age at hire and department.

Table 8. Proportional Hazards models for selected cancers in an occupationally PFOA-exposed cohort hired between 1950 and 1990 adjusted for age-at-hire, gender comparing those workers in

a department with direct exposure versus those in a department with no direct exposure.

Figure Legend:

Figure 1: Example of the Questionnaire Section Pertaining to Cancer Prevalence.

Table 1. PFOA levels by water district/source and percent of study subjects reporting water consumption from each source

PFOA Levels (ppb)	Location	Households	Total Population[a] (% total pop)	Study Sample (% study sample)
1.7-4.3	Little Hocking, Ohio	4200	11,760 (17%)	161 (27%)
0.4-3.9	Lubeck, WV	3700	10,360 (15%)	219 (37%)
0.25-0.37	Tuppers Plains, Ohio	4800	13,440 (19%)	19 (3%)
0.08-0.13	Belpre, Ohio	6000	16,800 (24%)	45 (8%)
0.06-0.1	Mason, WV	4200	11,760 (17%)	4 (0.7%)
0.06-0.07	Pomeroy, Ohio	1000	2,800 (4%)	1 (0.2%)
0.165	Blennerhassett	71	200 (0%)	6 (1%)
1.0-5.0	Dupont, Washington Works[b]	N/A	2,200 (3%)	90 (15%)
1.75-1.87	GE Plastics[b]	N/A	700 (1%)	43 (7%)
0.05-8.6	68 Private Wells WVA & Ohio	68	190 (0%)	11 (2%)
			70,010	599

[a] Estimated as 2.8 people per household
[b] Company employment reports
Abbreviations:
N/A Not Applicable

Table 2. Frequencies and percents for demographic, social, behavioral and physical characteristics in a residentially PFOA-exposed population.

	No.	%
Age Category		
20 – 34	105	18.17
35 – 44	104	17.99
45 – 54	135	23.36
55 – 64	154	26.64
65 – 80	80	13.84
Gender		
Male	284	49.13
Female	294	50.87
Race/Ethnicity		
White	558	97.38
African American	6	1.05
Others	9	1.57
Education Level		
Less than 9th grade	17	2.98
9 – 11th grade	59	10.33
12th/Vocational/Some College	430	75.31
College Graduate	65	11.38
Body Mass Index		
Underweight (<23)	106	18.53
Average (23 – 28)	190	33.22
Overweight (>28)	276	48.25
Smoking Status		
Never smoked	252	60.58
Smoked less than 15 years	72	17.31
Smoked more than 15 years	92	22.12
Work History		
Plant 1	54	9.42
Plant 2	19	3.32
No plant work	500	87.26

Table 3. Estimated unadjusted odds ratios (OR) of cancer (yes/no) and 95% confidence intervals (CI) for demographic, social, behavioral and physical characteristics in a residentially PFOA-exposed population.

	No. [a] (%)[b]	OR	95% CI	P-value[c]
Gender				
Male	25 (8.80%)	1.04	(0.58 - 1.86)	0.90
Female (ref.)	25 (8.50%)	1.00	-	-
Age Category				**0.0002**
20 – 34 (ref.)	5 (4.76%)	1.00	-	-
35 – 44	4 (3.88%)	0.80	(0.21 – 3.07)	0.74
45 – 54	9 (6.67%)	1.43	(0.46 – 4.40)	0.53
55 – 64	16 (10.39%)	2.32	(0.82 – 6.54)	0.11
65 – 80	16 (20.00%)	5.00	(1.75 – 14.32)	0.003
Education Level				
Less than 9th grade	8 (47.06%)	10.84	3.97-29.53	<0.0001
9th grade or higher	42 (7.58%)	1.00	-	-
Body Mass Index				**0.93**
Underweight (<23)	8 (7.55%)	0.78	(0.33 – 1.86)	0.58
Average (23 – 28) (ref.)	18 (9.47%)	1.00	-	-
Overweight (>28)	23 (8.33%)	0.87	(0.46 – 1.66)	0.67
Smoking Status				**0.16**
Never smoked (ref.)	20 (7.94%)	1.0	-	-
Smoked less than 15 years	7 (9.72%)	1.25	(0.51 – 3.08)	0.63
Smoked more than 15 years	12 (13.04%)	1.74	(0.81 – 3.72)	0.15
Work Site				
Plant 1	8 (14.81%)	1.87	(0.83 – 4.22)	0.13
Plant 2	1 (5.26%)	0.48	(0.06 – 3.62)	0.47
No plant employment (ref)	41 (8.20%)	1.0	-	-

[a] Number of cancer observations considered in the logistic procedure

[b] % with cancer in each category.

[c] Bolded p-value refers to the p-value for a test for trend

Table 4. All cancer prevalence rates per 100,000 in PFOA-exposed residents compared to the US population rates for Whites by age and gender.

	Males		Females		Prevalence Ratio			
Age Group	Age Specific Rates (US[a])	Age Specific Rates (EP[b])	Age Specific Rates (US)	Age Specific Rates (EP)	EP/US Males	P	EP/US females	P
20-34	338	1,923	451	7,547	5.69	0.16	16.75	0.0001
35-44	799	-	1,447	7,547	-	-	5.21	0.008
45-54	1,722	4,839	3,167	8,219	2.81	0.09	2.59	0.03
55-64	5,080	9,211	5,390	11,538	1.81	0.10	2.14	0.03
65+	15,661	32,558	9,173	5,405	2.08	0.009	0.59	0.85

[a]US = United States population (Whites only)
[b]EP = Residentially PFOA-exposed population

Table 5. Standardized Morbidity Prevalence Ratio (PR) comparing observed cancer rate per 100,000 among a residentially PFOA-exposed population to the expected cancer rate using the age-specific rates of the general U.S. population and the age distribution of the exposed population.

CANCER TYPE	Number of Cancers	Observed Rates[a] (per 100,000)	Expected Rates[b] (per 100,000)	PR	CI[c]
All Cancer	50	8,651	3,426	2.58	1.91 – 3.47*
Uterine/Cervical[d]	9	3,061	96	33.12	17.03 – 64.41*
M. Myeloma	2	346	22	15.71	3.91 – 63.14*
Lung	7	1,211	153	7.89	3.72 – 16.74*
Non-Hodgkins	5	865	130	6.67	2.76 – 16.13*
Bladder	5	865	163	5.30	2.19 – 12.87*
Colon/Rectal	4	692	261	2.65	0.99 – 7.11
Kidney	1	173	79	2.20	0.31 – 15.63
Prostate	9	3,169	1633	1.96	0.98 – 3.92
Melanoma	3	519	214	2.42	0.78 – 7.54
Breast	5	1,701	1,579	1.12	0.46 – 2.71

[a] Observed cancer rates per 100,000 in the exposed population

[b] Internally standardized rates per 100,000, age adjusted using the distribution in the PFOA exposed residents, SEER 2000.

[c] Confidence Interval

[d] 7 women reported cervical and 6 reported uterine cancer, 3 women reported both

* Confidence interval excludes the null value

Table 6. Numbers and percents for demographic and work characteristics in an occupationally PFOA-exposed population.

	No.	%
Birth Cohort		
1900 – 1919	160	3.76
1920 – 1939	1209	28.42
1940 – 1959	2203	51.79
1960 – 1989	682	16.03
Gender		
Male	3583	84.23
Female	671	15.77
Years of Occupational Exposure		
<21 years	1266	30.92
21 – 29	1462	35.71
30 – 50	1366	33.37
Department		
No direct PFOA exposure	2157	60.85
Direct PFOA exposure	1388	39.15

Table 7. Proportional Hazards models for selected cancers in an occupationally PFOA-exposed cohort hired between 1950 and 1990 adjusted for age at hire comparing those working in a department with direct PFOA exposure compared to those in a department with no direct exposure.

	No. (%) of all those in category w/cancer	Hazard Ratio	CI	P-value
Pancreatic cancer				
No direct exposure	2(0.09%)	1.00		
Direct PFOA exposure	6(0.48%)	4.46	(0.87,22.91)	0.07
Respiratory cancer				
No direct exposure	11(0.51%)	1.00		
Direct PFOA exposure	26(2.10%)	4.41	(2.13,9.13)	<0.0001
Kidney cancer				
No direct exposure	6(0.28%)	1.00		
Direct PFOA exposure	11(0.89%)	3.14	(1.10,8.95)	0.03
Colon/rectal cancer				
No direct exposure	9(0.42%)	1.00		
Direct PFOA exposure	11(0.89%)	2.96	(1.15,7.64)	0.02
Prostate cancer[a]				
No direct exposure	14(0.65%)	1.00		
Direct PFOA exposure	23(1.86%)	2.51	(1.24,5.08)	0.01
Non-Hodgkin's Lymph				
No direct exposure	3(0.14%)	1.00		
Direct PFOA exposure	3(0.24%)	2.44	(0.47, 12.73)	0.29
Bladder cancer				
No direct exposure	10(0.46%)	1.00		
Direct PFOA exposure	10(0.81%)	1.46	(0.59,3.54)	0.41
Liver cancer				
No direct exposure	1(0.05%)	1.00		
Direct PFOA exposure	1(0.08%)	1.13	(0.06.23.07)	0.94

	No. (%) of all those in category w/cancer	Hazard Ratio	CI	P-value
Breast cancer				
No direct exposure	5(0.23%)	1.00		
Direct PFOA exposure	1(0.08%)	0.21	(0.02, 1.88)	0.16

[a] Women excluded from analyses

Table 8. Dose response for cancer incidence with increasing years of occupational exposure using adjusted odds ratios (OR) and 95% confidence intervals (CI) for selected cancers in an occupationally PFOA-exposed cohort hired between 1950 and 1990: results of a logistic regression analysis controlling for age at hire and department.

Cancer Type	OR	95% CI	P-Value [b]
Prostate			**0.0002**
<21 years[a]	1.0	-	-
21 – 29	2.68	0.82 – 8.79	0.10
30 – 50	8.71	2.63 – 28.83	0.0004
Kidney			**0.03**
<21 years	1.0	-	-
21 – 29	6.28	0.75 – 52.89	0.09
30 – 50	11.57	1.38 – 97.32	0.02
Respiratory			**0.07**
<21 years	1.0	-	-
21 – 29	1.42	0.61 – 3.30	0.42
30 – 50	1.47	0.63 – 3.43	0.37
Bladder			**0.17**
<21 years	1.0	-	-
21 – 29	1.30	0.40 – 4.24	0.66
30 – 50	2.09	0.59 – 7.40	0.29
Colon/Rectal			**0.24**
<21 years	1.0	-	-
21 – 29	0.38	0.10 – 1.50	0.17
30 – 50	1.41	0.50 – 4.00	0.52
Pancreatic			**0.35**
<21 years	1.0	-	-
21 – 29	1.71	0.28 – 10.49	0.56
30 – 50	1.92	0.28 – 13.20	0.51

[a] Years of occupational exposure

[b] Bolded p-value refers to the p-value for a test for trend

Figure 1: Questionnaire

Have you ever been told by a doctor that you have cancer? O Yes O No

If you were diagnosed with cancer, are you still receiving medical treatment? O Yes O No

If you answered yes to the previous cancer questions, please indicate which cancer(s) and when you were diagnosed.

Yes	No		Year
O	O	Hodgkin's	
O	O	Non-Hodgkin's	
O	O	Other Lymphoma	
O	O	Leukemia	
O	O	Multiple Myeloma	
O	O	Thyroid	
O	O	Testicular	
O	O	Breast	
O	O	Uterine	
O	O	Cervical	
O	O	Ovarian	
O	O	Brain	
O	O	Mouth,Nose,Throat	

Yes	No		Year
O	O	Lung	
O	O	Bone	
O	O	Stomach	
O	O	Liver	
O	O	Kidney	
O	O	Pancreas	
O	O	Colon	
O	O	Rectal	
O	O	Prostate	
O	O	Skin	
O	O	Melanoma	
O	O	Bladder	
O	O	Gall Bladder	
O	O	Other	

Other Cancer Type

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April 21, 2004

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Re: PFOA Human Health Effects Study: Cancer Data

Dr. Charles M. Auer
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Richard H. Hefter. Chief
Jennifer Seed
Mary Ellen Weber
Mary Dominiak
April 21, 2004
Page 2

Ladies and Gentlemen:

In response to USEPA's prior requests for available information regarding the potential threat to human health or the environment from PFOA, we have attached a copy of an abstract presented this week during the annual Society of Environmental Toxicology and Chemistry (SETAC) meeting in Prague, Czechoslovakia referencing cancer rates among individuals exposed to PFOA-contaminated drinking water in communities near E.I. duPont de Nemours and Company's ("DuPont's") Washington Works Plant in Wood County, West Virginia. In view of the nature of the potential public health threat at issue and DuPont's failure to respond to our requests for confirmation that it has submitted the results of this study to USEPA under Section 8(e) of TSCA, we are forwarding this information to you directly to make sure that USEPA has access to the information. The abstract and related poster information also is available on-line through SETAC. Please include this information in AR-226, OPPT-2003-0012, and the appropriate IRIS database for PFOA.

Very truly yours,

Robert A. Bilott

RAB/mdm
Attachment
cc: IRIS Submission Desk (w/ attachment)
Mark Garvey, Esq. (USEPA) (w/ attachment)
R. Edison Hill, Esq. (w/ attachment)
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Re: PFOA Human Health Effects Study: Cancer Data

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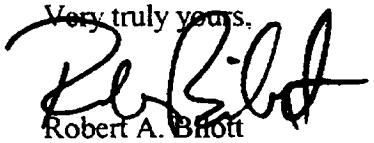
June 2, 2004
Page 2

Ladies and Gentlemen:

In response to USEPA's prior requests for available information regarding the potential threat to human health or the environment from PFOA, we have attached copies of two abstracts (LE5954-7) that have been submitted for presentation during the International Conference on Environmental and Public Health Management: Persistent Toxic Substances, which will be held in Hong Kong in November of 2004. These abstracts reference some of the age-adjusted data discussed more generally in the abstract that we forwarded to you on April 21, 2004. All of the abstracts refer to data generated in connection with the same study of cancer rates among individuals exposed to PFOA-contaminated drinking water in communities near E.I. duPont de Nemours and Company's Washington Works Plant in Wood County, West Virginia. As with the prior abstract information, we request that you include this information in AR-226, OPPT-2003-0012, and the appropriate IRIS database for PFOA.

Very truly yours,



Robert A. Bilott

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Re: PFOA Human Health Effects Study: Cancer Data

Ladies and Gentlemen:

In response to USEPA's request for available information regarding the potential threat to human health or the environment from PFOA, we previously forwarded to you preliminary abstracts/summaries of data generated in connection with a survey of adverse health effects among individuals exposed to PFOA-contaminated drinking water in communities near E.I. duPont de Nemours and Company's Washington Work Plant in Wood County, West Virginia (*see, e.g.*, OPPT-2003-0012-607, OPPT-2003-0012-677, and AR-226-1714-16). As a supplement to those previous submissions, we have enclosed a copy of several slides from a presentation that was made to the public in Hong Kong during the November 2004 International

Conference on Environmental and Public Health Management: Persistent Toxic Substances. The slides summarize some of the reproductive effects data generated from the survey of the community near DuPont's Washington Works Plant in West Virginia. As with the prior study data, we request that you include this information in AR-226, OPT-2003-0012, and the appropriate IRIS database for PFOA.

Very truly yours,

Robert A. Bilott

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February 7, 2005

FEDERAL EXPRESS

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Re: PFOA Human Health Effects Study: Cancer Data

Ladies and Gentlemen:

In response to USEPA's request for available information regarding the potential threat to human health or the environment from PFOA, we previously forwarded to you preliminary abstracts/summaries of data generated in connection with a survey of adverse health effects self-reported among individuals exposed to PFOA-contaminated drinking water in communities near E.I. duPont de Nemours and Company's Washington Works Plant in Wood County, West Virginia (*see, e.g.*, OPPT-2003-0012-607, OPPT-2003-0012-677, OPPT-2003-0012-836, AR-226-1714-16, and AR-226-1893-94). As a supplement to those previous submissions, we have enclosed a copy of several tables providing more detailed summaries of the age-adjusted, self-

reported cancer data from the PFOA community health study. (Exhibit 1) An article explaining the study and the cancer results in more detail has been peer reviewed and accepted for publication. The article is expected to be published this summer. Also enclosed are charts summarizing some of the other adverse health effects reported in the same community study. (Exhibit 2). An article explaining these results has recently been completed and is being submitted for peer review and publication. In addition, we have enclosed documents recently released by one of the public water suppliers to the community at issue, which discuss the increasing levels of PFOA being detected in that particular public water supply. (Exhibit 3) As with the prior PFOA community study data, we request that you include this information in AR-226, OPT-2003-0012, and the appropriate IRIS database for PFOA.

Very truly yours,

Robert A. Bilott

RAB/mdm
Enclosures
cc: IRIS Submission Desk (w/ encls.)
Mark J. Garvey, Esq. (USEPA) (w/ encls.)
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