OPPT-2003-0012

AR226-3694

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June 1, 2006

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Re: PFOA Human Health Effects Study

Ladies and Gentlemen:

In response to previous requests by the United States Environmental Protection Agency and other governmental entities for information relating to environmental and/or human exposures to PFOA, and in recognition of a potential threat to human health and/or the environment, we are enclosing at Exhibit A for inclusion in AR-226, OPPT-HQ-2003-0012, and the IRIS database for PFOA, a copy of a human health study relating to PFOA that does not appear to have been previously produced.

We represent the Plaintiffs in a lawsuit currently pending against the 3M Company in Minnesota State Court relating to the contamination of private and public drinking water supplies with PFOA, PFOS, and other perfluorochemicals attributable to 3M's operations and activities. Among the documents recently produced by 3M in that case was a copy of the enclosed draft article and memo, both originally marked "confidential" by 3M.

Upon review of that article, we noticed that the authors' conclusion that "PFOA is associated with alterations in peripheral blood lymphocyte numbers in PFOA production workers, suggesting that cell-mediated immunity may be affected by PFOA" appeared inconsistent with 3M's public representations that there is no evidence of any human health effects associated with PFOA exposure among its workers.[1/] The article and its conclusions also

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1/ For example, 3M is currently claiming on its corporate website that:

> The extensive research to date shows no adverse human health effects resulting from exposure to PFOS or PFOA. This is supported by observational research involving thousands of 3M production employees. That research was conducted by 3M and by the University of Minnesota under grants from 3M.
>
> In all of our years of research we have not found any evidence of adverse health effects in our employees. Over 25 years of medical surveillance of exposed employees failed to identify any health

did not appear to be contained or referenced within any of documents produced to date by 3M in response to USEPA's requests for all available human health data relating to PFOA exposures. (*See* AR-226) Although it appeared that the enclosed article had been prepared for publication, we could not find any reference to the article or its conclusions in any of the published literature on PFOA after it apparently had been submitted to 3M for pre-publication review in 1993.

Because the enclosed article reveals important human health data that did not appear to have been included or referenced in any of the on-going reviews or discussions of human health effects related to PFOA (including proceedings before the USEPA Science Advisory Board PFOA Review Panel) or in any of the submissions 3M had made to the public dockets relating to PFOA, we asked 3M in a letter dated May 1, 2006, to withdraw its claim of confidentiality over the enclosed materials[2/] and to confirm whether the study and its conclusions had previously been disclosed by 3M or any other entity.

On May 23, 2006, we received the enclosed letter (Exhibit B) from 3M's counsel withdrawing 3M's claim of confidentiality over the enclosed documents. With respect to any prior disclosure of the enclosed study and its conclusions, 3M confirmed that a decision was made not to publish the enclosed article after it had been submitted to 3M for review. 3M claims, however, that "information" contained within the article had previously been disclosed when 3M submitted a copy of Dr. Frank Gilliland's 1992 Ph.D. dissertation to USEPA on May 26, 2000, and that other "data addressed by his [Dr. Gilliland's] dissertation" was provided to USEPA when 3M submitted a copy of a 1993 publication by Dr. Gilliland to USEPA on January 28, 2000.

> effects attributable to exposure to PFOA or PFOS. ... These studies and medical surveillance results have been published in peer-reviewed scientific journals and shared with the EPA and with regulatory agencies in other countries.

(Exhibit D)

[2/] Because 3M marked the documents "confidential" under the Minnesota Court's protective order, we were prohibited under the terms of that order from sharing or disclosing the documents with any regulatory entities without first either obtaining 3M's permission or obtaining 3M's agreement to withdraw its confidentiality claim.

The copy of Dr. Gilliland's 1992 dissertation submitted to USEPA in 2000[3/] does refer to some of the data upon which the enclosed article is based. But the dissertation abstract summarizing that data states that a "positive association between hemoglobin, mean cellular volume, and leucocyte counts with PFOA," was found, suggesting the *opposite* of the conclusion provided in the enclosed article that PFOA exposure is *negatively* associated with peripheral blood lymphocyte counts. In addition to clarifying and explaining the specific inverse association between PFOA exposure and peripheral blood lymphocyte counts, the enclosed article also appears to include additional, clarifying data that does not appear to have been included in the earlier 1992 dissertation. (*See, e.g.,* Exhibit A, at Table 4).

Moreover, when 3M submitted the 1992 dissertation to USEPA in 2000, the abstract it attached to the document to summarize the key findings of the dissertation includes no reference to any immune system effects that are the subject and focus of the attached, clarifying article. (*See* AR-226-0473). Thus, those relying upon 3M's summary of the key findings in the 1992 dissertation or the abstract would not necessarily have been alerted to the association between PFOA exposure and peripheral blood lymphocyte numbers in PFOA production workers that is clarified and highlighted in the enclosed article[4/].

In addition, the 1993 publication referred to by 3M as addressing "data covered" in the 1992 dissertation addresses only the mortality study findings addressed in the dissertation and does not appear to reference the immune system findings. *See* Gilliland, et al., *Mortality Among Employees of a Perfluorooctanoic Acid Production Plant,* J. Occup. Med. 1993; 35:950-954 (finding that "ten years of employment in exposed jobs was associated with a 3.3-fold increase (95% CI, 1.02 to 10.6) in prostate cancer mortality compared to no employment in PFOA production" among 3M's employees). Although a second article also was published addressing "data covered" in Dr. Gilliland's 1992 dissertation, that article similarly appears to make no mention of the lymphocyte data at issue in the enclosed article. *See* Gilliland & Mandel, *Serum Perfluorooctanoic Acid and Hepatic Enzymes, Lipoproteins, and Cholesterol: A Study of Occupationally Exposed Men*, 29 Am. J. Indus. Med. 560-568 (1996).

3/ The copy of the 1992 dissertation available in AR-226 appears to be missing the following pages: 72, 126-127, 134, 175, 198-199, 235, and 280-283. 3M has since produced to us an additional copy of the dissertation with copies of the missing pages. Those pages are attached at Exhibit C.

4/ In that regard, we note that the lymphocyte findings addressed specifically in the enclosed article do not appear to be mentioned in USEPA's draft hazard or risk assessments for PFOA, although the 1992 dissertation is listed among the references.

Because the enclosed study appears to reveal an association between PFOA exposure and alteration of the human immune system that does not appear to have been considered in the reviews conducted to date of human health effects related to PFOA, we are providing the enclosed study so that this additional data can be considered and evaluated in connection with ongoing regulatory investigations related to PFOA.

Very truly yours,

Robert A. Bilott

RAB/mdm
Enclosures

bcc: J. Mark Englehart, Esq. (w/o encls.)
Gale D. Pearson, Esq. (w/o encls.)
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Internal Correspondence

3M

TO: Fluorochemical Steering Committee Members

FROM: Jeff Mandel, M.D., 333-8670 (220-3W-05)

SUBJECT: Papers/Dr. Gilliland

DATEL: December 7, 1993

These are two additional papers that are being prepared by Dr. Gilliland. They need our input/approval before being submitted for publication.

The papers are negative for the most part. We're working with him regarding some of the wording. We're operating under the premise that these sorts of topics need 3M support.

Please send your comments back to me by the end of the month so I can communicate with Dr. Gilliland.

JHM/vlk

Peripheral Blood Lymphocyte Count in Men Occupationally Exposed to Perfluorooctanoic Acid

Frank D. Gilliland, M.D., Ph.D.
Jack S. Mandel Ph.D.

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Reprint requests should be addressed to Frank D. Gilliland, University of New Mexico School of Medicine, New Mexico Tumor Registry, 900 Camino de Salud NE, Albuquerque, NM 87131. Telephone (505) 277-5541, Fax (505) 277-7041.

This study was supported in part by NIOSH Grant T150h07098-16 and 3M Medical Department

Running title: Lymphocyte count and PFOA

ABSTRACT

Studies in Rhesus monkeys suggest that perfluorooctanoic acid (PFOA) has immunotoxic effects on the cell-mediated immune system in primates. The predominant histopathological lesion in PFOA-treated monkeys was diffuse atrophy of lymph nodes and splenic germinal centers. Although PFOA accounts for the majority of fluorine present in the serum of the general population and in occupationally exposed workers, little information is available concerning human responses to PFOA exposure. To assess whether PFOA exposure is associated with effects on the human cell-mediated immune system, we examined the cross-sectional associations between peripheral blood lymphocyte counts and PFOA in 115 workers employed at a PFOA production plant. Total serum fluorine was used as a surrogate measure for serum PFOA levels. Peripheral blood lymphocyte count was significantly associated with total serum fluoride level; however, the magnitude and direction of the relationship was dependent on smoking, alcohol use, and obesity status. For example, for non-smokers and moderate drinkers, an increase of 10 ppm in total serum fluoride was associated with a decrease in lymphocyte count of 1640 cells in non-obese (BMI=25 kg/m^2) workers and by 925 cell in obese workers (BMI=35 kg/m^2). PFOA is associated with alterations in peripheral blood lymphocyte numbers in PFOA production workers, suggesting that cell-mediated immunity may be affected by PFOA.

KEY WORDS perfluorooctanoic acid, epidemiology, immune toxicity, lymphocyte count

INTRODUCTION

Perfluorooctanoic acid (PFOA) is widely used in industrial processes and consumer products as a result of its unique chemical properties and potent surface activity (Griffith, 1980). Because PFOA has a long biological half-life, small frequent doses can accumulate to appreciable levels (Ubel, 1980). As a result, PFOA has been found in the serum of all human population studied, and accounts for the majority of fluorine present in the serum of populations in industrialized countries[1-7].

Little is known about the toxic potential of PFOA in humans; however, studies have suggested that the cell-mediated immune system may be a site of toxicity in primates. Rhesus monkeys treated with oral PFOA developed histologic changes in spleen and lymph nodes. The primary histopathologic lesion was atrophy in lymph node and splenic germinal centers[1]. The immune system of rodent species has not been reported to undergo similar histopathologic changes in subacute and chronic feeding studies [1]. No data are available concerning immunotoxicity in humans. Because PFOA is present in the serum of exposed workers and in the general population [3-7], it is a matter of concern whether the findings in monkeys indicate the potential for human immunotoxicity. To assess whether PFOA could affect the cell-mediated immune system in humans, we studied the association of PFOA, as measured by total serum fluorine, with peripheral blood lymphocyte count (PBL) in 115 occupationally exposed employees at a plant that produces PFOA.

MATERIALS AND METHODS

All current workers employed in PFOA production over the 5 previous years and a sample of workers in jobs with no apparent PFOA exposure for the previous 5 years were invited to participate. Participants had vital parameters measured in the plant medical department by an occupational health nurse, completed a medical history questionnaire, and underwent venipuncture. Blood was drawn for assays of peripheral lymphocyte count. A fluorine-free 15 ml vacutainer was used to collect blood for total serum fluorine determination. Total serum fluorine was used as the measure of PFOA in this occupational group. Total serum fluorine was determined using the sodium biphenyl extraction and atomic absorption spectrometry [8].

Pearson correlation coefficients were calculated to assess the univariate associations between PBL and age, body mass index (BMI), cigarette use, and alcohol use. Linear multivariate regression models were fit to estimate the associations of PBL count with PFOA, adjusted for age, BMI, cigarette use, and alcohol use. Two way interactions between total serum fluorine and the four covariates were evaluated using residual analysis, model fit, and tests of significance. Interaction terms were included in the final model if biologically plausible and if the parameter estimate for the interaction term was significant at the alpha =.10 level.

RESULTS

Participant characteristics are displayed in Table 1. Total serum fluorine values ranged from 0 and 26 ppm with a mean of 3.3 ppm. Twenty-three

(20.0%) participants had serum values less than 1 ppm, 6 (5.2%) had values between 10 and 15 ppm, and 5 (4.4%) had values greater than 15 ppm.

Table 2 presents the correlation coefficients between PBL and total serum fluorine, age, BMI, alcohol use, and cigarette consumption. Peripheral blood lymphocyte count was significantly correlated with total serum fluorine (r=.19, p=.04). The final regression model for PBL, which adjusted for age, BMI, cigarette use, and alcohol use, included data for 111 workers; 4 of the 115 workers had missing assay values and were excluded from the analysis (Table 3). Total serum fluorine was inversely associated with PBL; however, the relationship was complex, with significant interactions between total serum fluorine and BMI, cigarette use, and alcohol use. Table 4 illustrates the relationship for a 10 ppm change in total serum fluorine for combinations of smoking, alcohol consumption, and BMI status. In moderate drinkers (1-3oz alcohol per day), PBL decreased in the categories of smoking and of obesity; in light drinkers (<1oz per day), PBL decreased in non-obese smokers only.

Discussion

Serum PFOA level is associated with changes in the cell-mediate immune system, as evidenced by changes in PBL; however, the relationship depends upon smoking, alcohol use, and obesity. PFOA could modulate cell counts by altering the known effects of smoking, alcohol consumption, and adiposity on peripheral leukocyte counts (refs). No human studies of the effect of PFOA on the immune system are available for comparison; however, a study of this group of workers suggests that PFOA may modify the hepatic response to

alcohol and obesity. Modulation of endobiotic and xenobiotic metabolism could represent a common mechanism for the observed association of PFOA with alterations in hepatic and immune response.

The relationship between the changes in PBL in humans and the lymph node and splenic germinal center atrophy observed in monkeys is not clear. Changes in PBL may be unrelated to germinal center atrophy. Alternatively, small changes in PBL could reflect larger changes in T cell subsets. In addition, the modification of the relationship between smoking and PBL by PFOA could be the result of changes in the number of particular T cell subsets. Furthermore, PFOA may be associated with changes in immune function beyond simple changes in cell number. Cytokine signaling is important in immune function and could be altered by PFOA exposure [27]. The response to antigen binding depends upon rearrangement of membrane proteins. Changes in the membrane physical characteristics produced by the potent surfactant action of PFOA could alter immune responses.

Interpretation of the findings requires careful consideration of the study limitations. Given the occupational study setting, the voluntary participation, and the requirements for blood sample collection, the overall participation was unexpectedly high, and non-response bias is likely to be small. Workers not included may have had a different response pattern than those who were included. Migration out of the high exposure jobs is unlikely to be the result of subclinical changes in PBL counts. The vast majority of workers who had significant exposure over the previous 5 years would be included in the study sample as the turn-over rate in plant employees was low (3% per year) and the study included all current employees with appropriate

job histories. Selection bias is not a likely explanation for the findings in this study.

Total serum fluorine was used as a surrogate variable for PFOA exposure. The use of total serum fluorine has been validated in past biological monitoring in the plant and other plants using PFOA [2]. Approximately 90% of total serum fluorine in workers was reported to be in the form of PFOA [2, 8]. Total serum fluorine is likely to be highly correlated with serum PFOA in this cohort. The coefficient of variation for total serum fluorine was 66%. At the low end of the spectrum (< 1 ppm), where the assay is limited by sensitivity, the total serum fluorine values may overestimate the true value. The measurement errors are likely to lead to an underestimate of the effect of PFOA on the physiologic endpoints. The duration of exposure may be an important determinant of PFOA level and effect; however, information on the duration of employment in exposed jobs was not available, because plant records did not contain sufficient information to reconstruct exposures.

Inflammatory and infectious processes, which are major determinants of PBL, were not assessed in this study. Because there is no evidence that these processes are related to total serum fluorine or serum PFOA, they are unlikely to confound the estimated relationships.

The changes in PBL counts associated with PFOA exposure present a complex picture. Alcohol use, cigarette use, and BMI modified the association of cell count with PFOA. The magnitude of these associations is not clinically significant from an infectious disease perspective; however, judgment as to the clinical relevance of such changes must await further study. More

research is needed in the area of PFOA immunotoxicity. The findings of the present study need to be confirmed. Changes in T cell subsets could be confirmed by immunophenotyping lymphocytes using well established flow cytometry methods [28, 29]. In addition, the standard immunotoxicologic assessment defined by the National Toxicology Program [30] needs to be conducted for PFOA.

Table I Participants characteristics

	mean	range
total fluorine (ppm)	3.3	0-26
age (years)	39.2	24-59
BMI (kg/m2)	26.9	18.8-40.5
	number	percent
Alcohol use		
<1 ounce per day	87	75.6
1-3 onces per day	20	17.4
>3 onces per day	0	0.0
non-response	8	7.0
Cigarettes use		
non-smoker	85	73.9
current smoker	28	24.4
non-response	2	1.7

TABLE2 Pearson correlation coefficients between total serum fluoride, age, body mass index (BMI), daily alcohol use, daily tobacco consumption, and peripheral blood lymphocyte count

	Total fluorine (ppm)	Age (years)	BMI (kg/m^2)	Alcohol (oz/day)	Tobacco (cigs/day)
LYMPHOCYTES	.19 p=.04	-.05	.04	.15	.28 p=.002

TABLE 3 Linear multivariate regression model of factors predicting the lymphocyte count among 111 workers.

Variable	β	SE(β)	p-value
Intercept	2205.6	611.1	.0005
Total Fluorine (ppm)	-342.7	125.3	.007
Alcohol *			
low (<1oz/day)	-526.6	222.7	.02
nonresponse (NR)	-977.1	355.7	.007
low X Fluorine	189.0	52.3	.0005
NR X Fluorine	247.9	103.9	.02
Cigarettes/day	34.0	6.9	.0001
Cigs/day X Fluorine**	-3.3	1.45	.02
BMI (kg/m^2)	1.58	19.6	.94
BMI X Fluorine**	7.15	4.1	.08

R^2= .35

#Reference category is drinkers who consumed 1-3 oz ethanol/day.

*interaction terms alcohol category by total fluoride; cigarettes/day by total fluoride, BMI by total fluoride.

Table 4 Change in lymphocyte count for 10 ppm increase in total serum fluorine

< 1 oz. alcohol/day	Non-smoker	Smoker (20 cigs/day)
BMI 25 mg/Kg^2	+750	-406
BMI 35 mg/Kg^2	+965	+306

1-3 oz. of alcohol/day	Non-smoker	Smoker (20 cigs/day)
BMI 25 mg/Kg^2	-1640	-2300
BMI 35 mg/Kg^2	-924	-1585

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May 22, 2006

Via Federal Express

Robert A. Bilott
Taft, Stettinius & Hollister, LLP
425 Walnut Street, Suite 1800
Cincinnati, OH 45202-3957

Re: *Palmer, et al. v. 3M Company,* Court File No. C2-04-6309

Dear Mr. Bilott:

This letter responds to your multiple inquiries regarding document 3MA00323875-890, which is an undated draft manuscript by Gilliland & Mandel produced to Plaintiffs from 3M's files.

Counsel for 3M has now spoken with Dr. Frank Gilliland, who is the author of the document, as well as Dr. Jack Mandel, who is listed as a co-author. Because the document is Dr. Gilliland's intellectual property, 3M felt it was important to obtain Dr. Gilliland's permission before providing you with a non-confidential copy. I am enclosing a copy of the document bearing the Bates numbers but without the *Palmer* protective order legend.

Dr. Gilliland tells us that the document in question is an "early draft" of a potential manuscript, which he decided not to pursue. The decision not to pursue publication of the manuscript was his alone. It was 3M's understanding, based on a June 29, 1993, letter from Dr. Gilliland (3MA10017137), that he intended to submit three papers based on his student dissertation for publication. Two papers were published. Dr. Gilliland has informed us that he concluded that there was no finding that warranted publication of a third manuscript.

You asked whether the information in the document has been disclosed previously. The answer is yes. The document was based on Dr. Gilliland's Ph.D. dissertation, which was completed in 1992 and has been available through the University of Minnesota medical school library since 1993.[1] In addition, Dr. Gilliland authored a 1993 publication that addressed data

[1] The dissertation was cataloged by the University of Minnesota on March 10, 1993. See the catalog entry at http://saturn.oit.umn.edu/F/Y23UTYA6V2J1Q64F4FIPT7SK4B358FVAF4HHX2CFP1X62LR2-B829104?func=full-set-set&set_number=019979&set_entry=000026&format=999. The catalog entry notes that the dissertation is available for normal library loan: Availability TC Bio-Medical Library QC463.F55 G481f 1992 Regular Loan. In addition, as you or your experts are no doubt aware, dissertations are customarily lodged not only with the university library, but also with a dissertation service established by the University of Michigan known as University Microfilms, www.umi.com, and now operated by Proquest at www.proquest.com. Through this service, dissertations are widely available through any library or over the internet. Researchers often start by searching this database to see if others

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covered by his dissertation. *See* Gilliland, et al., Mortality Among Employees of a Perfluorooctanoic Acid Production Plant, J Occup Med 1993; 35:950-954. 3M submitted this publication along with several related publications on fluorochemical production worker medical surveillance to U.S. EPA on January 28, 2000, and also submitted Dr. Gilliland's dissertation to U.S. EPA on May 26, 2000. Both of these submissions are available in EPA's public dockets (the 8(e) docket and the AR-226 docket respectively). In response to your letter of this morning, asking for a copy of the dissertation, I am also enclosing a copy of that document, which has been produced to you at 3MA00749385-681, without a confidentiality legend.

In short, because the data discussed in the document have been public and widely available since at least 1993, Dr. Gilliland graciously consented to the release of the document without a confidentiality legend.

Very truly yours,

Michael C. McCarthy

MCM:mlt
cc: Dr. Frank Gilliland *(w/enc. – via U.S. Mail)*
Dr. Jack Mandel *(w/enc. – via U.S. Mail)*
Plaintiffs' counsel *(w/o enc. – via U.S. Mail)*
Defendant's counsel *(w/o enc. – via e-mail)*

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have already addressed a topic. Dr. Gilliland's dissertation has been available through this service, and can be located and ordered directly over the internet or through libraries world-over.

TABLE 4.1.9 TOTAL SERUM FLUORIDE DISTRIBUTION
3M CHEMOLITE PLANT, COTTAGE GROVE, MINNESOTA

TOTAL FLUORINE (PPM)	NUMBER	PERCENT
<1	23	20.0
1-3	65	56.5
>3-10	16	13.9
>10-15	6	5.2
>15-26	5	4.4
TOTAL	115	100.0
MEAN TF	3.3	
SD	4.7	
MEDIAN TF	2	
RANGE	0-26	

TABLE 4.1.64 LINEAR MULTIVARIATE REGRESSION MODEL OF FACTORS PREDICTING THE TRIGLYCERIDES AMONG 111 MALE WORKERS. 3M CHEMOLITE PLANT, COTTAGE GROVE, MINNESOTA

Variable	β	SE(β)	p-value
Intercept	-114.50	117.20	.33
Total Fluoride (ppm)	2.38	2.31	.15
Cigarettes/day	2.28	1.05	.03
BMI (kg/m^2)	6.07	3.39	.08
Age (years)	2.32	1.44	.11
Alcohol #			
low (<1oz/day)	-11.48	29.4	.70
nonresponse (NR)	-19.94	49.03	.69
Free Testosterone*	7.34	3.37	.03
Bound Testosterone*	-.21	.08	.009

R^2= .19
#Reference category is moderate drinkers who consume 1-3 oz ethanol/day.
*ng/dl

TABLE 4.1.65 PEARSON CORRELATION COEFFICIENTS BETWEEN TOTAL SERUM FLUORIDE, AGE, BODY MASS INDEX (BMI), DAILY ALCOHOL USE, DAILY TOBACCO CONSUMPTION, AND HEPATIC PARAMETERS
3M CHEMOLITE PLANT, COTTAGE GROVE, MINNESOTA

	TOTAL FLUORINE (ppm)	AGE (years)	BMI (kg/m^2)	ALCOHOL (oz/day)	TOBACCO (cigs/day)
SGOT*	.01	-.10	.09	.12	-.11
SGPT**	.01	.01	.20 p=.02	.03	-.11
GGT#	-.04	.12	.27 p=.004	.15	.03
AKPH##	-.03	.27 p=.004	.19 p=.04	-.19 p=.05	.26 p=.006

*SERUM GLUTAMIC OXALOACETIC TRANSAMINASE IU/dl
**SERUM GLUTAMIC PYRUVIC TRANSAMINASE IU/dl
#GAMMA GLUTAMYL TRANSFERASE IU/dl
##ALKALINE PHOSPHATASE IU/dl

TABLE 4.1.72 ALKALINE PHOSPHATASE (AKPH) BY BODY MASS INDEX, AGE, SMOKING AND DRINKING STATUS
3M CHEMOLITE PLANT, COTTAGE GROVE, MINNESOTA

		AKPH (IU/dl)				
	N(%)	MEAN	SD	MEDIAN	RANGE	TEST#
BMI						
<25	41(35.7)	79	22.1	75	38-153	F=1.53
25-30	57(49.6)	84	21.9	81	41-153	p=.22
>30	17(14.8)	90	27.1	90	43-153	
AGE						
<30	21(18.3)	78	22.2	76	38-153	F=2.78
31-40	48(41.7)	80	20.3	76	50-15.	p=.45
41-50	27(23.5)	86	24.1	83	43-153	
51-60	19(16.5)	95	24.1	94	41-130	
Alcohol						
<1oz/d	87(81.3)	85	24.0	2	38-153	F=2.05
1-3oz/d	20(18.7)	77	16.9	75	51-124	p=.16
missing	8	82	22.5	70	60-115	
Tobacco						
smoker	28(24.8)	85	23.8	85	61-153	F=6.48
nonsmoker	85(75.2)	77	22.0	77	38-153	p=.012
missing	2	86	24.8	86	68-103	
TOTAL	115					

#univariate Anova

TABLE 4.2.20 NUMBERS OF DEATHS AND STANDARDIZED MORTALITY RATIOS (SMRs) BY LATENCY, BASED ON MINNESOTA WHITE MALE RATES, AMONG MALE EMPLOYEES EVER EMPLOYED IN THE CHEMICAL DIVISION, 1947-1989.

	LATENCY ≥ 15 YEARS			
Cause of Death	Obs	Exp	SMR	95% CI
All causes	105	128.4	.82	.67-.99
Cancer	34	29.95	1.14	.79-1.59
Gastrointestinal	9	8.30	1.08	.49-2.06
Colon	7	3.00	1.33	.36-3.42
Pancreas	4	1.75	2.28	.61-5.85
Respiratory	10	9.98	1.00	.48-1.94
Lung	9	9.50	.95	.43-1.80
Prostate	3	1.87	1.61	.32-4.70
Lymphopoietic	4	3.28	1.72	.33-3.12
Cardiovascular	44	64.67	.68	.49-.91
All Gastrointestinal	4	6.37	.63	.17-1.61
All respiratory	5	6.49	.77	.25-1.80
Diabetes	2	1.72	1.17	.43-4.21
Injuries	6	8.54	.70	.26-1.53

Abbreviations used are: Obs, observed; Exp, expected; CI, confidence interval; CHD, coronary and atherosclerotic heart disease; CD, Chemical Division.

5. DISCUSSION

5.1 Physiologic Effects Study

5.1.1 Introduction

This was a cross-sectional study of the relationship between selected physiologic parameters and PFOA exposure which was assessed using total serum fluorine. Participants were recruited from workers employed during November, 1990 in the Chemical Division of the 3M Chemolite Plant in Cottage Grove, Minnesota. All current workers who had worked in high exposure jobs at any time in the five previous years were invited to participate. A sample of workers employed in low exposure jobs was frequency matched to the age distribution of workers in high exposrue jobs.

Participants completed a corporate medical history questionnaire and had vital parameters measured by an occupational health nurse. Blood was drawn for assays of total serum fluorine, seven hormones involved in the hypothalamic-pituitary-gonadal axis, serum lipids, lipoproteins, hepatic function parameters, and hematology indices. Blood was drawn in the morning after workers were assigned to the day shift for at least three days.

In 93% of participants serum fluorine levels were at least 10 times the background levels in the general population and in 3M workers not employed at Chemolite. Many workers who lacked PFOA exposure by job history had elevated PFOA levels. The sources of the unexpected PFOA exposure are unknown.

5.1.2 Hormones

The findings from this study are consistent with the hypothesis that perfluorooctanoic acid (PFOA) affects the human hypothalamic-pituitary-gonadal axis. This study showed that relatively low levels of serum PFOA (20μM) depressed free testosterone and elevated estradiol but did no affect LH or FSH levels. The association between free testosterone and PFOA was different in

older men than in younger men. In older men, free testosterone (FT) was depressed below 10 ng/ml at serum fluoride levels below one part per million (estimated PFOA levels below 1 µM). In younger men, FT decreased toward 10 ng/ml at serum fluoride levels above 15 ppm (estimated PFOA levels below 15 µM). Increasing age may increase men's susceptibility to the testosterone lowering effects of PFOA. The associations between PFOA and the hormone levels may reflect a true causal relationship, or may be a result of chance, bias, or uncontrolled contounding. There are no human studies of PFOA associated reproductive toxicity available for comparison. Studies of the effects of PFOA in rodents have demonstrated a similar decrease in testosterone, increase in estradiol, and little change in LH [19].

The association between PFOA and free testosterone may have been mediated by elevated estradiol and prolactin. Elevated estradiol decreases testosterone and other steroid hormone synthesis in Leydig cells. LH response to low testosterone is attenuated by estradiol through negative feedback mechanisms at the pituitary and hypothalamic levels [112, 113]. Elevated prolactin sensitizes the hypothalamus and pituitary to estrogens feedback. The combined effect of elevated estradiol and prolactin could have reduced the secretion of LH and the subsequent Leydig cell response. Estradiol has direct effects on Leydig cell testosterone synthesis. In rats, PFOA decreased androstenedione and testosterone, but not 17 alpha-hydroxyprogesterone [19]. The metabolism of 17 alpha-hydroxyprogesterone to androstenedione was inhibited at the step of the C-17,20 lyase. The activity of the rate limiting C-17,20 lyase has been reported to be under estradiol regulation in rat Leydig cells [114, 115]. Thus, in PFOA treated rats, elevated levels of estradiol may inhibit the C-17,20 lyase and thereby reduce testosterone synthesis. The increase in the estradiol-testosterone ratio observed in workers is compatible with this mechanism for decreased free testosterone.

The primary source of estradiol in males is the P450 (P450 19) mediated aromatization of testosterone [116, 117]. Additional estradiol is secreted directly from Leydig cells. The observed increase in estradiol may be the result of increased production from one of these two sources or may be the result of inhibition of P450 mediated estradiol metabolism [118]. Perfluorooctanoic acid, a

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TABLE A4.1.21 FOLLICLE STIMULATING HORMONE TO PROLACTIN RATIO (FSH/P) BY BODY MASS INDEX, AGE, SMOKING AND DRINKING STATUS 1990 PERFLUOROCHEMICAL EFFECTS STUDY, 3M CHEMOLITE PLANT, COTTAGE GROVE, MINNESOTA

		FSH/P				
	N	MEAN	SD	MEDIAN	RANGE	TEST#
BMI						
<25	40	0.72	0.51	0.57	0.2-2.1	F=.22
25-30	56	0.79	0.52	0.65	0.1-2.6	.80
>30	17	0.74	0.58	0.57	0.2-2.6	
AGE						
<31	20	0.46	0.23	0.45	0.2-1.0	F=5.41
31-40	48	0.71	0.51	0.54	0.1-2.6	p=.002
41-50	26	0.88	0.50	0.73	0.2-2.1	
51-60	19	1.05	0.62	0.81	0.2-2.6	
Alcohol						
<1oz/d	86	0.81	0.57	0.66	0.2-2.1	F=3.18
1-3oz/d	19	0.57	0.32	0.50	0.1-2.6	p=.08
missing	8	0.66	0.31	0.60	0.2-2.6	
Tobacco						
smoker	27	1.00	0.57	0.79	0.3-2.6	F=7.90
nonsmoker	84	0.68	0.49	0.51	0.1-2.6	p=.006
missing	2	0.75	0.57	0.62	0.3-1.2	
TOTAL	113					

#univariate Anova

APPENDIX 3

TABLES OF HORMONE RATIOS BY TOTAL SERUM FLUORIDE

TABLE A4.2.1 HORMONE RATIOS BY TOTAL SERUM FLUORIDE:
ESTRADIOL/FREE TESTOSTERONE (E/TF)
ESTRADIOL/BOUND TESTOSTRONE (E/TB)
ESTRADIOL/THYROID STIMULATING HORMONE (E/TSH)
1990 PERFLUOROCHEMICAL EFFECTS STUDY,
3M CHEMOLITE PLANT, COTTAGE GROVE, MINNESOTA

TOTAL FLUORIDE ppm	N	MEAN	SD	MEDIAN	RANGE	TEST#
			E/TF			
<1	23	2.5	1.2	2.2	0.7-5.3	F=1.65
>=1-3	64	2.1	0.8	1.9	0.8-5.4	p=.16
>3-10	15	2.2	0.6	2.2	0.8-3.2	
>10-15	6	2.6	1.1	2.1	1.6-4.0	
>15-26	5	2.7	0.7	3	1.9-3.3	
TOTAL	113	2.25	.92	2.1	0.7-5.4	
			E/TB (X100)			
<1	23	7.3	3.5	6.3	1.1-14.4	F=1.17
>=1-3	64	5.9	2.5	5.5	1.7-13.5	p=.33
>3-10	15	6.7	2.8	5.9	2.9-12.3	
>10-15	6	6.9	2.8	5.6	4.3-11.7	
>15-26	5	6.3	1.8	5.3	4.8-8.4	
TOTAL	113	6.4	2.8	5.8	1.2-14.4	
			E/TSH			
<1	23	29.2	21.8	21.0	10.4-108.1	F=0.75
>=1-3	64	24.9	13.2	23.6	1.8-52.2	p=.56
>3-10	15	26.7	16.7	21.1	3.9-59.0	
>10-15	6	19.3	1.9	15.8	7.8-45.8	
>15-26	5	19.8	6.8	16.9	15.2-31.5	
TOTAL	113	25.5	15.6	21.3	1.76-108.1	

#univariate Anova

TABLE A4.2.2 HORMONE RATIOS BY TOTAL SERUM FLUORIDE:
BOUND TESTOSTERONE/FREE TESTOSTRONE (TB/TF)
BOUND TESTOSTRONE/FOLLICLE STIMULATING HORMONE (TB/FSH)
BOUND TESTOSTERONE/PROLACTIN (TB/P)
FREE TESTOSTERONE/PROLACTIN (TF/P)
1990 PERFLUOROCHEMICAL EFFECTS STUDY,
3M CHEMOLITE PLANT, COTTAGE GROVE, MINNESOTA

	N	MEAN	SD	MEDIAN	RANGE	TEST#
TOTAL FLUORIDE ppm			TB/TF			
<1	23	36.7	9.6	37.1	16.7-62.6	F=.94
>=1-3	64	36.8	9.6	35.6	19.2-58.8	P=.45
>3-10	15	34.5	7.0	33.3	25.0-43.6	
>10-15	6	38.3	7.6	38.9	29.3-47.6	
>15-26	5	43.6	10.6	39.9	36.9-62.4	<=10vs >10
TOTAL	113	36.8	9.2	37.0	16.7-62.6	T=2.10
						P=.15
			TB/FSH			
<1	23	148.3	85.6	120.4	56.7-411.0	F=.40
>=1-3	64	130.9	76.3	114.2	23.1-347.7	P=.81
>3-10	15	135.3	81.4	86.9	49.0-303.3	
>10-15	6	122.7	48.3	135.8	34.4-169.8	
>15-26	5	162.9	76.4	143.5	67.1-253.5	
TOTAL	113	136.1	77.1	120.0	23.1-411.0	
			TB/P			
<1	23	82.1	43.2	67.5	19.8-205.5	F=.40
>=1-3	64	87.5	81.2	63.3	23.0-624.2	P=.81
>3-10	15	86.4	51.0	78.0	28.1-224.2	
>10-15	6	51.5	27.9	52.1	22.2-89.3	
>15-26	5	90.3	30.6	83.4	58.3-129.7	
TOTAL	113	84.5	67.3	70.7	19.8-624.2	
			TF/P			
<1	23	2.34	1.46	2.01	.7-7.8	F=.52
>=1-3	64	2.38	1.97	1.88	.6-15.0	P=.72
>3-10	15	2.64	1.84	2.40	.8-8.1	
>10-15	6	1.40	0.82	1.35	.5-2.3	
>15-26	5	2.20	0.91	2.16	.9-3.5	
TOTAL	113	2.35	1.78	1.95	.5-15.0	

#univariate Anova

PFOS-PFOA Information

- What is 3M Doing?
- Human Health and the Environment
- Report on Drinking Water Near 3M Plants

Human Health and the Environment

Human Health

PFOS and PFOA are extremely well-researched materials. This research has been conducted by 3M and other scientists around the world.

The extensive research to date shows no adverse human health effects resulting from exposure to PFOS or PFOA. This is supported by observational research involving thousands of 3M production employees. That research was conducted by 3M and by the University of Minnesota under grants from 3M.

In all of our years of research we have not found any evidence of adverse health effects in our employees. Over 25 years of medical surveillance of exposed employees failed to identify any health effects attributable to exposure to PFOA or PFOS. It is important to note that employees working directly with these materials in a manufacturing facility have much higher exposure than that found in the general population. These studies and medical surveillance results have been published in peer-reviewed scientific journals and shared with the EPA and with regulatory agencies in other countries.

A bibliography of these studies can be downloaded below.

Reference: 3M Employee Medical Studies (PDF, 26 KB)

In addition to research of the exposure of 3M's production employees who worked closely with these materials, the company has studied the low level presence of these materials in the general population. Along with our employee research, these studies have also been published in scientific literature.

A bibliography of these studies can be downloaded below.

Reference: 3M General Population Exposure (PDF, 13 KB)

Interim results of a study being conducted by the University of Pennsylvania are consistent with the results of 3M's own studies of its production employees. The University of Pennsylvania study, funded by a National Institute of Health Environmental Justice grant, is evaluating the health of residents of Little Hocking, Ohio who have been exposed to elevated levels of PFOA in their drinking water as the result of releases from a different company's manufacturing plant. The study's principal investigator is University of Pennsylvania School of Medicine Professor Dr. Edward Emmett, who is board certified in occupational medicine and

Related Links

City of Oakdale: www.ci.oakdale.

City of Lake Elm www.lakeelmo.c Lake Elmo New: Water Report (PDF, 2294 KB)

MDH: www.health.stat Perfluorochemic (PDF, 32 KB)

MPCA: www.pca.state.r Perfluorochemic Present and Fut (PDF, 64 KB)

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