FINAL REPORT

Epidemiology, 220-6W-08
Medical Department
3M Company
St. Paul, MN 55144

Date: August 20, 2010

Title: Biomonitoring Assessment of the 3M Cottage Grove Building 15 Demolition and Disposal Project: Phase I

Study
Start Date: August 26, 2009

Protocol Number N/A
IRB Approval N/A

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Summary

In 2008-2009, 3M and contract workers were involved with the removal of equipment and lines (referred to as Phase I) in Building 15 prior to its demolition and disposal (Phase II). A total of 66 workers participated in Phase I of the 3M Cottage Grove Building 15 Demolition and Disposal project. Fifty-six workers (84.8%) had both baseline and end-of-project serum perfluorooctanoate (PFOA) and perfluorooctanesulfonate (PFOS) concentrations determined. The other 10 workers (15.2%) only had a baseline assessment. Forty-five (80.3%) of the 56 workers had a higher end-of-project PFOA concentration than at baseline, indicating occupational exposure to PFOA during the project. For these 45 workers with occupational exposure, the mean number of days between their paired measurements was 96 days (range 23 - 251). The 45 subjects' mean baseline PFOA concentration was 22.7 ng/mL (95% CI 0 - 50.1) and their mean end-of-project PFOA concentration was 156.4 ng/mL (95 CI 103.4 - 209.4). The corresponding geometric means for PFOA were 6.0 ng/mL (95% CI 4.4 - 8.3) and 78.1 (95% CI 52.0 - 117.1). The mean matched-pair PFOA concentration difference for the 45 exposed individuals was +133.7 ng/mL ($p < 0.0001$). The 45 subjects' mean baseline and end-of-project PFOS concentrations were 25.9 ng/mL (95% CI 15.3 - 36.5) and 30.9 ng/mL (95% CI 19.1 - 42.6), respectively. The mean matched-pair difference for PFOS was +5.0 ng/mL ($p = 0.07$). The lack of any major changes for PFOS was due to the fact that, unlike PFOA, PFOS had not been manufactured in Building 15 for several decades.

There were no statistically significant adverse changes in this workforce's serum lipids (total cholesterol, non-HDL cholesterol, HDL cholesterol), renal function (BUN, creatinine), or liver clinical chemistries (total bilirubin, alkaline phosphatase, AST, ALT) related to the change (difference) in serum PFOA concentration from baseline to end-of-project. For example, using

multiple regression models, a 100 ng/mL increase in PFOA resulted in a non-statistically significant decrease of 0.8 mg/dL total cholesterol (95% CI (-6.6) - 4.9), a 2.2 mg/dL decrease in non-HDL cholesterol (95% CI (-8.0) - 3.6), and a 1.4 mg/dL increase in HDL (95% CI (-6.2) - 3.5). In the adjusted models for these three lipid-related variables, none of the 6 covariates considered in the models were statistically significant at $p < 0.10$ except for sex in the HDL model. The other covariates considered for these models were age, BMI, alcohol use, lipid lowering medication use, and the number of days between baseline and end-of-project measurement. For AST and ALT, a 100 ng/mL increase in PFOA resulted in an unadjusted 0.6 U/L increase in AST (95% CI (-1.7) - 0.5) and a statistically significant 1.8 U/L decrease in ALT (95% CI (-3.3) - 0.3). This statistically significant association likely has no clinical relevance because the ALT value decreased with increasing PFOA concentration.

Inferences from the present analysis are limited by the number of workers who participated and the study's inability to acquire fasting blood samples due to the logistical necessity of obtaining the samples from a predominantly contract workforce throughout their available work day. The lack of fasting, however, would only bias the serum triglyceride, blood glucose, and the indirectly calculated LDL measurements. In conclusion, this longitudinal assessment of 45 workers with demonstrated occupational exposure to PFOA did not observe changes in blood lipids, renal function, or liver enzymes related to their change in PFOA concentrations measured between baseline and end-of-project measurements. This longitudinal assessment does not support some cross-sectional epidemiologic studies that have reported positive associations between PFOA and non-HDL cholesterol. Unlike the present study, these cross-sectional investigations were unable to assess temporality because of their study design.

Introduction

Beginning in 2008, exposure remediation projects have been launched regarding legacy perfluorochemicals at the 3M Company. These included the 3M Woodbury landfill remediation project, the 3M Cottage Grove Building 25 re-roof project, the 3M Cottage Grove D1/D2 excavation project, the 3M Decatur Building 2/49 demolition and disposal project, and the 3M Cottage Grove Buildings 15 and 73 demolition and disposal project (hereafter referred to as only Building 15). For each of these projects baseline and end-of-project assessments were required of 3M employees and contractor workers who entered specified work zones where potential exposure to legacy perfluorochemicals (e.g., PFOA (perfluorooctanoate) and PFOS (perfluorooctanesulfonate) was possible, but due to the uniqueness of the work, the magnitude of potential exposure was unknown.

The purpose of this report is to provide an analysis of the PFOA and PFOS serum concentrations (ng/mL) measured at baseline and end-of-project time periods for those workers involved with equipment and line removal from Building 15 at 3M Cottage Grove. This is referred to as Phase I of this project. The second phase of this project (hereafter referred to as Phase II) involved the subsequent demolition and disposal of Building 15 and these results have already been reported elsewhere (Olsen et al. 2010). Trend analyses of the changes in PFOA related to serum clinical chemistries were conducted because of the higher PFOA concentrations observed among the majority of workers during Phase I. Despite several epidemiology reports describing positive associations between serum lipids and PFOA, none conducted longitudinal assessments.

Methods

1. Informed consent

The purpose of the project was explained in an informed consent. Subjects read and signed this informed consent at both baseline and end-of-project assessments. Subjects were informed they could not work on this specific project without such compliance.

2. Clinical chemistries

Clinical chemistries included a lipid panel profile, blood glucose, BUN, creatinine, serum electrolytes, and liver enzyme tests. Fasting was not a requirement because of the logistics of collecting blood samples during various times of the day workers would arrive to be tested. Clinical chemistries were analyzed by Quest Diagnostics. The specific tests measured were:

Total Cholesterol (mg/dL)
HDL Cholesterol (mg/dL)
LDL Cholesterol (mg/dL – indirect calculation)
Triglycerides (mg/dL)
Non-HDL Cholesterol (mg/dL – calculated)
Cholesterol/HDL Ratio (calculated)
BUN (mg/dL)
Creatinine (mg/dL)
Total Bilirubin (mg/dL)
Alkaline Phosphatase (U/L)
AST (U/L)
ALT (U/L)
Total Protein (g/dL)
Albumin (g/dL)
Globulin (g/dL – calculated)
Albumin/Globulin Ratio (calculated)
Sodium (mmol/L)
Potassium (mmol/L)
Chloride (mmol/L)
Carbon Dioxide (mmol/L)
Calcium (mg/dL)

Individual clinical chemistry analyses at baseline and at end-of-project were medically reviewed by Dr. Gibson. Subjects were informed in writing that this examination program was not a full medical 'check-up.' Values out of reference range were indicated with a notation for the subjects to follow up with their primary care physician if they had abnormal test results. However, questions could be directed to Dr. Gibson regarding their clinical chemistry test results should the subject so desire. At the baseline exam subjects also responded to a brief medical questionnaire. Obtained at the baseline assessment was a self-reported questionnaire that inquired about basic demographic data (age, height, and weight), a brief medical history, and current medication use (blood pressure, lipid lowering, and glucose lowering).

Statistical analyses included basic descriptive measures of central tendency, a matched-pair analysis examining the changes between baseline and end-of-project for PFOA, PFOS, and the clinical chemistries, and multiple regression analyses employing standard statistical methods. Analyses were performed using JMP-SAS (Cary, NC).

3. Analytical measurements of PFOA and PFOS

Serum samples were analyzed for PFOA and PFOS by state-of-the-art high performance liquid chromatography mass spectrometry methods by the 3M Medical Department's Toxicology Laboratory under the direction of Dave Ehresman. Medical Department personnel collected blood samples that were processed to provide serum samples for analysis. These samples were assigned unique identification numbers and randomized prior to the samples being delivered for analysis. The 3M Medical Department's Toxicology Laboratory was "blinded" to the identity of all samples received for analysis.

Sample extractions were performed using solid phase extraction (SPE) technique. The extraction and sample clean-up was based on a 100 uL sample size and utilized Waters (Milford, MA) Oasis® hydrophilic–lipophilic balance (HLB) 3.0mL cartridges (Ehresman et al. 2007).

The method used two stable labeled internal standards for quantitation. The internal standards used were a dual labeled PFOS where two ^{18}O molecules were included in the sulfonate group (internal standard, >99% purity, synthesized by Research Triangle Institute, Research Triangle Park, NC) and a dual labeled PFOA molecule, where the carboxyl and alpha carbons were labeled with ^{13}C stable isotope (greater than 97%, provided by DuPont, Wilmington, DE). All quantitations were based on matrix matched extracted standard curves.

A 5 uL injection of the sample eluate was introduced into the High Pressure Liquid Chromatograph (HPLC) which was directly interfaced into the triple quadrupole mass spectrometer (Applied Biosystems/MDS-Sciex Instrument Corporation, Forest City, CA). Standard curves covered the range from 1.0 - 150 ng/mL. Standard curves were evaluated using a quadratic regression model where the standards were weighted at 1/x, and each curve had an ''R'' value equal to or greater than 0.9998. Matrix spiked controls (QC samples) evaluated during this study all had acceptable results "with-in" their previously established ranges. Matrix-matched dilutions were used for samples requiring dilution to bring the samples into the linear range of the assay. Extracted serum and aqueous blanks remained below the lower limit of quantitation established at 1.0 ng/mL (lowest standard fitted on the standard curve used for this project).

4. Communication

After each blood collection, individual letters were sent to the participants describing their results. In the baseline assessments, two letters were sent. One referred to the clinical chemistries and the other letter provided baseline PFOA and PFOS concentrations. At end-of-project, two letters were again sent to each participant. The first letter provided the individual's clinical chemistry results. The second letter compared baseline to end-of-project serum PFOA and PFOS concentrations.

Results

A total of 66 individuals were tested at baseline for serum PFOA and PFOS concentrations and the aforementioned clinical chemistries. The majority were non-3M employees (86%). A subset of 56 were also tested at end-of-project of which 51 (91%) were non-3M employees.

Figure 1A is a scatterplot of the 56 individuals who had baseline and end-of-project PFOA concentrations (ng/mL). As shown on Figure 1, the identity line represents those values where the *x* axis (baseline) is equal to the *y* axis (end-of-project). Eleven (19.6%) individuals had serum PFOA concentrations at baseline that were greater than their end-of-project value. This is represented in Figure 1A by the 11 points to the right of the identity line. Forty-five (80.4%) individuals had their end-of-project serum PFOA concentrations greater than their baseline as represented by the points to the left of the identity line in Figure 1A. Figure 1B is restricted to these 45 individuals whose end-of-project values were greater than their baseline.

In a similar manner, Figure 2A is a scatterplot of the baseline and end-of-project PFOS concentrations (ng/mL) for the same 56 individuals. Unlike Figure 1A with PFOA, PFOS is

distributed around the identity line indicating there were minimal PFOS differences between baseline and end-of-project concentrations. Twenty-nine (52%) individuals had serum PFOS baseline concentrations greater than their end-of-project values. Figure 2B represents the PFOS baseline and end-of-project concentrations for those 45 individuals who had higher end-of-project serum PFOA concentrations than their baseline values. Again, the majority of points center around the identity line for PFOS in Figure 2B indicating minimal change in PFOS concentrations.

Based on the findings in Figures 1A through 2B, three groups were identified for further description and statistical analyses: the 10 individuals who had baseline measurements only; the 56 individuals who had baseline and end-of-project measurements; and the 45 individuals (subset of the 56) who had a higher end-of-project PFOA level than baseline. For each of these groups, their distributions of serum PFOA and PFOS concentrations are found in Tables 2 and 3, respectively. Among the 45 individuals whose PFOA concentration increased over baseline, their arithmetic mean PFOA concentration was 22.7 ng/mL at baseline. The PFOA mean increased to 156.4 ng/mL at end-of-project (see Table 2). The PFOA geometric mean increased to 78.1 ng/mL at end-of-project from a baseline of 6.0 ng/mL. However, there was less difference in these 45 subjects' mean PFOS concentrations: 25.9 ng/mL at baseline and 30.9 ng/mL at end-of-project (Table 3). The difference in PFOS geometric means was even less: 18.1 ng/mL at baseline compared to 21.2 ng/mL at end-of-project (Table 3).

The age, BMI, and number of days between baseline and end-of-project for the 3 groups are found in Table 4. Among the 45 individuals whose PFOA concentration increased, the mean number of days between baseline and end-of-project measurements was 96 days. Provided in Table 5 are additional demographic data for these three groups as well as the prevalence of self-

reported medical history, medication status, and alcohol usage. Most individuals were males and non-3M employees.

Because the data were paired samples, a matched-paired statistical analysis was performed where the mean difference is the average of the sum of the individual differences for the 56 or 45 subjects. See *Equation 1*.

Equation 1.

$$\text{Mean Difference} = \sum_{i=1}^{56 \text{ or } 45}(\text{end-of-project} - \text{baseline})/56 \text{ or } 45$$

Provided in Tables 6 and 7 are the mean baseline and end-of-project clinical chemistry values for the 56 and 45 subjects, respectively, along with their mean matched-pair difference between baseline and end-of-project. For the 45 subjects whose PFOA values increased over baseline, the mean matched-pair difference was 133.7 ng/mL ($p < 0.0001$) compared to 5.0 ng/mL for PFOS ($p = 0.07$) (Table 7). No statistically significant difference in the mean matched-pair was observed for total cholesterol, non-HDL cholesterol, HDL, or LDL. There was a 71 ng/mL reduction in the mean matched-pair triglyceride value ($p < 0.0003$). Other statistically significant ($p < 0.05$) mean matched-pair differences were observed for BUN, creatinine, total bilirubin, potassium, and carbon dioxide. The magnitude of these differences, however, was minimal and of no clinical significance. No statistically significant differences were observed for any liver enzyme mean matched-pair.

Tables 8 and 9 are the cross-sectional analyses for the clinical chemistry measurements performed at baseline and end-of-project, respectively, for the 56 subjects who participated at both times. At baseline, there were no statistically significant PFOA coefficients with either lipid, renal, or hepatic clinical chemistry measurements. The same was true with the end-of-

project cross-sectional analyses. There were statistically significant unadjusted PFOA (negative) coefficients for chloride at baseline and for calcium at end-of-project.

Of much greater inferential importance than the cross-sectional analyses are the longitudinal assessments of change (i.e., difference) in the clinical chemistries between end-of-project versus baseline versus the concurrent change in PFOA (and PFOS) concentrations. Presented in Table 10A are the unadjusted and adjusted models for the 56 subjects for these longitudinal assessments. The adjusted models are the full hierarchical model that included 6 independent variables, besides the change (difference) in PFOA concentration. These covariates included age, sex, calculated BMI from height and weight, alcohol use ($\geq$ 4 drinks per week, lipid lowering medication use (yes/no), and the number of days between baseline and end-of-project measurement. See Equation 2.

Equation 2. $\Delta Y = \alpha + \beta\Delta PFOA + \sum\gamma X_i$

Where
Δ = difference between end-of-project minus baseline value
Y = the dependent clinical chemistry variable
X_i = the i = 1 thru 6 covariates described above
α= intercept
β = regression coefficient of ΔPFOA
γ = regression coefficient for each covariate Xi

Through backward stepwise regression procedures with removal of any variable set at $p > 0.10$, except for PFOA which was forced ("locked") into all models, those models that did not revert back to the unadjusted model shown in Table 10A, are found in Table 10B. In a similar manner, Tables 11A and 11B present the same data restricted to the 45 subjects who only had PFOA values that increased between baseline and end-of-project.

Among the 45 subjects who had an increase in PFOA concentration during Phase I, there were no statistically significant changes in total cholesterol, non-HDL cholesterol, HDL, LDL, or the cholesterol/HDL ratio associated with the higher PFOA concentrations (Table 11A). For example, a 100 ng/mL increase in PFOA was associated with an unadjusted non-statistically significant decrease of 0.8 mg/dL total cholesterol (95% CI (-6.6) - 4.9), a 2.2 mg/dL decrease in non-HDL cholesterol (95% CI (-8.0) - 3.6), and a 1.4 mg/dL increase in HDL (95% CI (-6.2) - 3.5) (Table 11A). In these models, none of the covariates were statistically significant at $p < 0.10$ except for sex in the HDL model (Tables 11A and 11B). Illustrated in Figures 3A – 3C are the non-significant linear trends and 95% confidence intervals for the unadjusted association between the changes in total cholesterol, non-HDL cholesterol, and HDL with the unadjusted change in PFOA concentration for the 45 subjects.

A decrease in serum triglycerides was statistically significantly associated with an increase in PFOA for the unadjusted and hierarchical models (Table 11A) as well as the backward regression model (Table1B). However, it is not possible to interpret this association since fasting was not a requirement.

There were no statistically significant changes in liver enzymes associated with increasing PFOA concentrations among the 45 subjects (Table 11A). Scatter plots for the unadjusted associations between changes in PFOA with alkaline phosphatase, AST, and ALT, are shown in Figures 4A – 4C. Adding the change in PFOS, as well as PFOA, into the models displayed in Tables 11A did not result in statistically significant associations for PFOS (data not shown).

Discussion

The collection of baseline and end-of-project samples for analysis of PFOA and PFOS provided an excellent method to assess each worker's exposure experience involved with the 3M Building 15 Demolition and Disposal Project. There was an increase in serum PFOA concentrations at end-of-project among the majority of workers involved in Phase I of the Building 15 Demolition and Disposal project, indicating exposure occurred during the removal of equipment and breaking of lines (pipes). Specific exposure tasks were not identified for each employee; thus it is not possible to ascertain which tasks yielded the highest potential for exposure. Increased exposure, determined by paired biomonitoring analyses, has not been observed at other 3M remediation sites that involved workers at the 3M Woodbury landfill remediation site, the 3M Cottage Grove Building 25 re-roof project, the Cottage Grove D1/D2 remediation site or Phase II of this Building 15 Demolition and Disposal project.

The higher end-of-project serum concentrations from Phase I provided an opportunity to conduct a longitudinal assessment of these 45 individuals to determine whether their serum lipids, renal function, and liver enzymes were affected. The data do not suggest that serum PFOA concentrations, that increased an average of 133 ng/mL over a 3+ month time period, modified total cholesterol, non-HDL cholesterol, or HDL measurement.

The baseline average of 22.6 ng/mL for the 45 workers is approximately 4 times the general population average (Nelson et al. 2010), but slightly less than the median PFOA concentration of 28 ng/mL reported for the affected mid-Ohio River valley community by Frisbee et al. (2009). It is 1 to 2 orders of magnitude lower than averages reported for occupationally exposed workers.

As reviewed by Steenland et al. (2010), positive associations have been reported between PFOA concentrations and non-HDL cholesterol levels in different populations including the general population (Nelson et al. 2010), a community affected with contaminated drinking water (Emmett et al. 2006; Steenland et al. 2009), and in some (Costa et al. 2009; Olsen et al. 2003; Sakr et al. 2007a; 2007b), but not all occupational investigations (Olsen et al. 1999; 2000; 2007). Most of these studies, however, have been cross-sectional investigations that can not fundamentally address the issue of causality due to their inability to assess temporal relationships. To add further to the confusion of these published studies, the strongest statistical associations reported have been in those populations with the lowest PFOA concentrations (Nelson et al. 2010), whereas the weakest associations have been reported in occupational populations whose serum PFOA concentrations are 2 to 3 orders of magnitude higher than the general population (Figure 5). Steenland et al. (2010) hypothesized that such findings could be the consequence of a biological pathway with non-HDL cholesterol that may be saturated at relatively low (i.e., general population level) PFOA concentrations. Data presented graphically by Nelson et al. (2010) based on the National Health and Nutrition Examination Survey (NHANES) general population, and by Steenland et al. (2009) from a community exposed through contaminated drinking water, do suggest that this saturation might exist between serum PFOA concentrations ranging between 20 and 50 ng/mL. Although no association was seen with non-HDL or HDL cholesterol in the present study, the average baseline concentration was near this threshold level.

The present study did not indicate perturbations in renal function or liver enzymes with increasing PFOA concentrations. Therefore, the present study's longitudinal assessment does not support the findings by Lin et al. (2009) who conducted a cross-sectional analysis of

NHANES data and reported a 1.86 unit increase in ALT with a 1 unit increase in log PFOA concentration. If this statistical association by Lin et al. was extrapolated to the present study, then the ALT concentrations among the 45 workers with increasing PFOA concentrations would have increased several orders of magnitude. This did not occur. Nor have there been clinically relevant increases in liver enzyme values observed in a series of cross-sectional or longitudinal occupational studies of ammonium perfluorooctanoate production workers (Costa et al. 2009; Olsen et al. 1999; 2000; 2003; 2007; Sakr et al. 2007a; 2007b; Steenland et al. 2010). In a longitudinal assessment of 454 DuPont workers whose medical records were abstracted for many years, Sakr et al. reported a 1,000 ng/mL increase in serum PFOA concentration was statistically significantly associated with a 0.008 mg/dL decline in total bilirubin (95% CI -0.014 to -0.002), a 0.35 U/L increase in AST (95% CI -10 to +0.60), and a non-significant 0.54 U/L increase in ALT (95% CI -0.46 to +1.54). These statistical associations would not have clinical relevance if extrapolated to the much lower concentrations of PFOA reported in the general population.

The present study is limited by the number of workers who participated and the inability to obtain fasting blood samples. This inability to request fasted blood samples was a logistical necessity because the predominantly contract workforce would participate throughout the normal work day. The lack of fasting, however, would only bias the serum triglyceride and glucose measurements. The lack of fasting could also affect the LDL cholesterol results due to its indirect calculation by the Friedewald formula. Consequently, these specific clinical chemistries are uninterpretable. Total cholesterol, non-HDL cholesterol, HDL cholesterol, renal function, and the liver enzymes would not have been biased by the lack of fasting. Also, it could be argued that the average assessment period of approximately 3 months between baseline and end-of-project may have been too short of follow-up time to observe an altered clinical chemistry

profile. However, increasing serum PFOA concentrations did not result in higher blood lipid values in male cynomolgus monkeys dosed daily for 6 months that resulted in steady serum concentrations ranging between 81,000 ng/mL and 156,000 ng/mL depending upon administered dose (range 3 to 20 mg/kg/day via oral capsule) (Butenhoff et al. 2002; 2004).

There was minimal change in PFOS concentrations in this study. The lack of any major changes was due to the fact that, unlike PFOA and its salts, PFOS had not been manufactured in Building 15 for several decades.

Conclusion

Based on biomonitoring data, serum PFOA concentrations increased over baseline for 45 (80.3%) of 56 workers during Phase I of the Building 15 Demolition and Disposal project. Phase I involved removal of equipment and lines. There was an arithmetic mean matched pair increase of 133.7 ng/mL PFOA (range 4.6 - 697 ng/mL) above the baseline average of 22.7 ng/mL. The geometric mean matched pair increase was 78.1 ng/mL PFOA. There was minimal change in PFOS owing to a lack of exposure in Building 15. There were no statistically significant changes in these 45 individuals' serum lipids (total cholesterol, non-HDL cholesterol, HDL cholesterol), renal function (BUN, creatinine), or liver clinical chemistries (total bilirubin, alkaline phosphatase, AST, ALT) associated with the increased PFOA concentrations that occurred over a mean of 96 days (range 23 - 251) between baseline and end-of-project measurements.

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Table 1. Distribution of Workers by Company and Participation for Phase I of the Building 15 Demolition and Disposal Project

Company	Baseline	End-of-Project	Reason for No End-of-Project Sample
Non-3M employee	57	51	Unknown
3M employee	9	5	Unknown
Total	66	56	

Table 2. PFOA Baseline and End-of-Project Concentrations (ng/mL) for 10, 56, and 45 Subjects Involved with Phase I of the Building 15 Demolition and Disposal Project

Participated Only at Baseline (N = 10)

						Arithmetic		Geometric	
	Min	Q1	Median	Q3	Max	Mean	95% CI	Mean	95% CI
Baseline	2.9	7.6	33.3	196.3	345	102.9	5.4 – 198.3	33.4	9.6 – 116.0

Participated at Baseline and End-of-Project (N = 56)

						Arithmetic		Geometric	
	Min	Q1	Median	Q3	Max	Mean	95% CI	Mean	95% CI
Baseline	1.3	4.0	6.1	22.4	1,325	100.0	35.4 – 164.6	12.5	7.6 – 20.5
End-of-Project	4.6	37.6	113.8	279.5	1,026	194.1	135.8 – 252.4	94.1	65.0 – 136.4

Participated at Baseline and End-of-Project and Had Higher PFOA Concentrations at End-of-Project (N = 45)

						Arithmetic		Geometric	
	Min	Q1	Median	Q3	Max	Mean	95% CI	Mean	95% CI
Baseline	1.3	3.2	5.3	7.1	611	22.7	0 – 50.1	6.0	4.4 – 8.3
End-of-Project	4.6	37.6	93.9	206.5	697	156.4	103.4 – 209.4	78.1	52.0 – 117.1

Table 3. PFOS Baseline and End-of-Project Concentrations (ng/mL) for 10, 56, and 45 Subjects Involved with Phase I of the Building 15 Demolition and Disposal Project

Participated at Baseline Only

						Arithmetic		Geometric	
	Min	Q1	Median	Q3	Max	Mean	95% CI	Mean	95% CI
Baseline	14.6	24.7	36.5	310.8	872.5	181.8	0 – 386	64.1	22.5 – 182.8

Participated at Baseline and End-of-Project (N – 56)

						Arithmetic		Geometric	
	Min	Q1	Median	Q3	Max	Mean	95% CI	Mean	95% CI
Baseline	4.4	13.3	18.4	36.6	258	45.6	29.2 – 62.0	25.4	19.5 – 33.1
End-of-Project	5.9	16.0	24.2	44.6	229	46.8	31.7 – 62.0	28.1	21.7 – 36.4

Participated at Baseline and End-of-Project and Had Higher PFOA Concentrations at End-of-Project (N = 45)

						Arithmetic		Geometric	
	Min	Q1	Median	Q3	Max	Mean	95% CI	Mean	95% CI
Baseline	4.4	12.2	16.0	25.8	187	25.9	15.3 – 36.5	18.1	14.6 – 22.5
End-of-Project	5.9	12.6	19.7	30.4	218	30.9	19.1 – 42.6	21.2	16.8 – 26.8

Table 4. Age, BMI, and Number of Days for Different Subject Groups

	N = 10 Subjects			N = 56 Subjects			N = 45 Subjects		
	Mean	95% CI	Range	Mean	95% CI	Range	Mean	95% CI	Range
Age	46.9	39.9 – 53.9	26.8 – 55.0	43.4	40.7 – 46.1	22.6 – 64.7	42.5	39.3 – 45.8	22.6 – 64.7
BMI	27.6	24.3 – 30.9	23.6 – 38.5	30.0	28.5 – 31.5	14.0 – 43.7	30.1	28.6 – 31.7	21.9 – 43.7
Days Between Baseline and End-of-Project	N/A	N/A	N/A	110	92 – 128	23 – 251	96	76 – 116	23 - 251

N/A = not applicable

Table 5. Additional Demographic Characteristics of Study Population, Phase I of the Building 15 Demolition and Disposal Project

	N = 10 Subjects		N = 56 Subjects		N = 45 Subjects	
	Number	Percent	Number	Percent	Number	Percent
Sex						
Males	9	90.0	52	92.8	41	91.1
Females	1	10.0	4	7.2	4	8.9
Employment						
3M	4	40.0	5	8.9	1	2.2
Non-3M	6	60.0	51	91.1	44	97.8
Self Reported Medical History						
High Blood Pressure	2	20.0	11	20.7	7	15.9
Hepatitis	0	0.0	1	1.9	0	0.0
Cirrhosis	0	0.0	1	1.9	0	0.0
Other Liver Disease	0	0.0	1	1.9	0	0.0
Gall Bladder Disease	0	0.0	2	3.8	1	2.2
Diabetes	0	0.0	3	5.7	2	4.5
Self Reported Medications						
High Blood Pressure	2	20.0	9	17.3	7	16.3
High Cholesterol	1	10.0	8	15.4	5	11.6
Diabetes	0	0.0	2	3.8	2	4.5

Table 5. (continued)

	N = 10 Subjects		N = 56 Subjects		N = 45 Subjects	
	Number	Percent	Number	Percent	Number	Percent
Alcohol Usage						
< 1 per week	3	30.0	27	50.9	25	56.7
1 – 3 drinks/week	3	30.0	12	22.6	8	18.2
4 – 7 drinks/week	3	30.0	9	17.0	6	13.6
8 – 14 drinks/week	1	10.0	4	7.5	1	9.1
>14 drinks/week	0	0.0	1	1.9	1	2.3

Table 6. Baseline and End-of-Project Means of PFOA, PFOS, and Clinical Chemistries Along with Mean of Matched Pair Difference Among All Subjects (N = 56) with Both Measurements, Phase I of the Building 15 Demolition and Disposal Project

	Baseline		End-of-Project		Matched Pair Difference		
	Mean	95% CI	Mean	95% CI	Mean	95% CI	p value
PFOA	100.0	35.4 – 164.6	194.1	135.8 – 252.4	94.1	50.1 – 138.1	< 0.0001
PFOS	45.6	29.2 – 62.0	46.8	31.7 – 62.0	1.3	(-3.1) – 6.2	0.62
Total Cholesterol	202	192 – 212	201	191 – 211	(-0.4)	(-8) – 7	0.90
Non-HDL Cholesterol	156	145 – 167	153	143 – 164	(-2.3)	(-7) – 8	0.53
LDL	108	99 – 117	113	104 – 123	4.5	(-4) – 13	0.29
HDL	46	42 – 50	48	45 - 51	1.8	(-10) – 5	0.18
Triglycerides	261	210 – 311	202	174 – 231	(-59)	(-95) – (-22)	0.002
Glucose	100	93 – 107	103	94 – 112	2.6	(-5) – 11	0.51
BUN	16	14 - 17	17	16 – 18	1	0 – 2	0.08
Creatinine	0.98	0.94 – 1.01	1.01	0.97 – 1.05	0.03	0.01 – 0.06	0.02
Total Bilirubin	0.7	0.6 – 0.8	0.6	0.5 – 0.7	(-0.07)	(-0.14) – (-0.01)	0.02

Table 6. (continued)

	Baseline		End-of-Project		Matched Pair Difference		
	Mean	95% CI	Mean	95% CI	Mean	95% CI	p value
Alkaline Phosphatase	73	68 – 78	73	68 – 78	(-0.7)	(-3) – 2	0.62
AST	21	20 – 23	22	21 – 24	1.1	0 – 3	0.14
ALT	27	24 – 31	27	23 – 30	(-1)	(-3) – 1	0.41
Total Protein	7.4	7.3 – 7.5	7.4	7.3 – 7.5	0	(-0.1) – 0.1	0.73
Albumin	4.5	4.4 – 4.6	4.5	4.5 – 4.6	0	0 – 0.1	0.58
Globulin	2.8	2.7 – 2.9	2.8	2.7 – 2.9	0	(-0.1) – 0.1	0.96
Albumin/Globulin	1.6	1.6 – 1.7	1.6	1.6 – 1.7	0	(-0.1) – 0.0	0.51
Sodium	140.2	139.7 – 140.7	139.4	138.9 – 140.0	(-0.8)	(-1.4) – (-0.2)	0.01
Potassium	4.7	4.4 – 4.6	4.3	4.3 – 4.4	(-0.1)	(-0.3) – 0.0	0.04
Chloride	103.5	102.9 – 104.1	103.2	102.5 – 103.9	(-0.3)	(-1.0) – 0.3	0.28
Carbon Dioxide	25.0	24.6 – 25.4	23.8	23.2 – 24.5	(-1.1)	(-1.8) – (-0.5)	0.001
Calcium	9.7	9.6 – 9.8	9.7	9.6 – 9.8	0	(-0.1) – 0.1	0.88

Table 7. Baseline and End-of-Project Means of PFOA, PFOS, and Clinical Chemistries Along with Mean of Matched Pair Difference Among All Subjects (N = 45) with Increased PFOA Concentrations at End-of-Project, Phase I of the Building 15 Demolition and Disposal Project

	Baseline		End-of-Project		Matched Pair Difference		
	Mean	95% CI	Mean	95% CI	Mean	95% CI	p value
PFOA	22.6	0.0 – 50.1	156.4	103.4 – 209.4	133.7	87.3 – 180.2	<0.0001
PFOS	25.9	15.3 – 36.5	30.9	19.1 – 42.6	5.0	(-0.3) – 10.2	0.07
Total Cholesterol	201	190 – 213	199	188 – 209	(-2.5)	(-11) – 6	0.56
Non-HDL Cholesterol	157	144 – 169	153	142 – 164	(-4.1)	(-13) – 5	0.36
LDL	107	97 – 117	113	103 – 124	5.2	(-5) – 15	0.36
HDL	45	40 – 49	46	42 – 50	2	(-2) – 5	0.34
Triglycerides	278	219 – 337	207	177 – 238	(-71)	(-115) – 27	0.002
Glucose	102	93 – 110	102	92 – 113	0.7	(-9) – 10	0.88
BUN	15	14 – 17	17	16 – 18	1.5	0 – 3	0.04
Creatinine	0.96	0.92 – 1.0	1.00	0.96 – 1.05	0.04	0.01 – 0.08	0.02
Total Bilirubin	0.6	0.6 – 0.7	0.6	0.5 – 0.7	(-0.07)	(-0.1) – 0.0	0.03
Alkaline Phosphatase	75	69 – 80	74	69 – 79	(-1.1)	(-4) – 2	0.45

Table 7. (continued)

	Baseline		End-of-Project		Matched Pair Difference		
	Mean	95% CI	Mean	95% CI	Mean	95% CI	p value
AST	20	19 – 22	21	19 – 23	0.7	(-1) – 2	0.41
ALT	26	23 – 30	24	22 – 27	(-2)	(-4) – 0	0.10
Total Protein	7.4	7.3 – 7.5	7.4	7.3 – 7.5	0	(-0.1) – 0.1	0.65
Albumin	4.5	4.4 – 4.6	4.6	4.5 – 4.6	0	0 – 0.1	0.45
Globulin	2.8	2.7 – 3.0	2.8	2.7 – 2.9	0	(-0.1) – 0.1	0.91
Albumin/Globulin	1.6	1.6 – 1.7	1.6	1.6 – 1.7	0	(-0.1) – 0.1	0.99
Sodium	140	139.5 – 140.5	140	139.1 – 140.2	(-0.4)	(-1.0) – 0.3	0.28
Potassium	4.5	4.3 – 4.6	4.3	4.2 – 4.4	(-0.2)	(-0.3) – 0.0	0.03
Chloride	103	102.6 – 103.9	103	102.6 – 103.8	0	(-0.7) – 0.6	0.90
Carbon Dioxide	25.0	24.6 – 25.5	24.1	23.4 – 24.7	(-1.0)	(-1.6) – (-0.3)	0.008
Calcium	9.7	9.6 – 9.8	9.7	9.6 – 9.8	0.0	(-0.1) – 0.1	0.74

Table 8. Unadjusted and Adjusted[a] PFOA Regression Coefficients for Cross-sectional Analysis at Baseline, N = 56 Subjects.
Phase I of Building 15 Demolition and Disposal Project

Variable	Unadjusted PFOA Coefficient	95% Confidence Interval Lower	Upper	p value	Adjusted PFOA Coefficient	95% Confidence Interval Lower	Upper	p value
Total Cholesterol	-0.00750	-0.05028	0.03529	0.73	0.02766	-0.05449	0.10981	0.50
Non-HDL Chol	-0.01439	-0.06094	0.03216	0.54	0.01003	-0.07680	0.09686	0.82
HDL	0.00689	-0.00974	0.02353	0.41	0.01763	-0.01268	0.04794	0.25
LDL	0.00237	-0.03230	0.03704	0.89	0.01241	-0.05262	0.07744	0.70
Chol/HDL	-0.00116	-0.00312	0.00079	0.24	-0.00133	-0.00499	0.00234	0.47
Triglycerides	-0.11917	-0.33017	0.09183	0.27	-0.11063	-0.52774	0.30648	0.60
Glucose	-0.00737	-0.03714	0.02240	0.62	-0.01978	-0.06614	0.02658	0.40
BUN	-0.00064	-0.00578	0.00449	0.80	-0.00161	-0.01126	0.00803	0.74
Creatinine	-0.000009	-0.00016	0.00014	0.90	-0.000004	-0.00026	0.00026	0.98
Total Bilirubin	0.00005	-0.00038	0.00047	0.83	0.000006	-0.00079	0.00090	0.89
Alkaline Phos	-0.01010	-0.03147	0.01127	0.35	-0.02068	-0.06401	0.02266	0.34
AST	0.004029	-0.00178	0.00984	0.17	0.00774	-0.00281	0.01830	0.15

Table 8. (continued)

Variable	Unadjusted PFOA Coefficient	95% Confidence Interval Lower	Upper	p value	Adjusted PFOA Coefficient	95% Confidence Interval Lower	Upper	p value
ALT	0.00565	-0.00830	0.01959	0.42	0.01250	-0.01400	0.03898	0.35
Total Protein	-0.00022	-0.00066	0.00022	0.32	-0.00037	-0.00012	0.00049	0.40
Albumin	-0.00020	-0.00050	0.00010	0.19	-0.00024	-0.00077	0.00029	0.37
Globulin	-0.00002	-0.00042	0.00038	0.92	-0.00013	-0.00087	0.00062	0.74
Albumin/Globulin	-0.00003	-0.00032	0.00026	0.85	0.00002	-0.00050	0.00054	0.93
Sodium	0.00057	-0.00159	0.00272	0.60	0.00091	-0.00301	0.00483	0.64
Potassium	-0.00016	-0.00059	0.00027	0.46	-0.0000002	-0.00084	0.00083	0.99
Chloride	0.00256	0.00012	0.00499	0.04	0.00149	-0.00322	0.00619	0.53
Carbon Dioxide	-0.0017	-0.00347	0.00006	0.06	0.00057	-0.00281	0.00395	0.73
Calcium	-0.00009	-0.00049	0.00031	0.66	0.00017	-0.00051	0.00085	0.61

a. Adjusted for self-reported age, BMI (height and weight), alcohol use ($\leq$ 4 drinks/week, and lipid lowering medication use (yes/no)

Table 9. Unadjusted and Adjusted[a] PFOA Regression Coefficients for Cross-sectional Analysis at End-of-Project, N = 56 Subjects, Phase I of Building 15 Demolition and Disposal Project

Dependent Variable	Unadjusted PFOA Coefficient	95% Confidence Interval Lower	Upper	p value	Adjusted PFOA Coefficient	95% Confidence Interval Lower	Upper	p value
Total Cholesterol	-0.00075	-0.04750	0.04599	0.97	0.00933	-0.05931	0.07797	0.79
Non-HDL Cholesterol	-0.00402	-0.05415	0.04611	0.87	0.01642	-0.05657	0.08940	0.65
HDL	0.00327	-0.01235	0.01888	0.68	-0.00708	-0.02973	0.01556	0.53
LDL	-0.00515	-0.04904	0.03874	0.81	0.01243	-0.05509	0.08006	0.71
Chol/HDL	-0.00014	-0.00190	0.00162	0.87	0.00119	-0.00140	0.00377	0.36
Triglycerides	-0.00584	-0.13923	0.12756	0.93	0.01824	-0.18454	0.22102	0.86
Glucose	0.00339	-0.03868	0.04547	0.87	-0.03758	-0.09563	0.02048	0.20
BUN	-0.00561	-0.01075	-0.00047	0.03	-0.00493	-0.01097	0.00111	0.11
Creatinine	-0.00015	-0.00034	0.00005	0.13	-0.00028	-0.00053	-0.00003	0.03
Total Bilirubin	-0.00005	-0.00041	0.00032	0.79	-0.00003	-0.00059	0.00053	0.91
Alkaline Phosphatase	-0.00775	-0.03153	0.01602	0.52	0.00450	-0.03232	0.04132	0.81
AST	0.00083	-0.00707	0.00874	0.83	-0.00123	-0.01306	0.01061	0.84

Table 9. (continued)

Dependent Variable	Unadjusted PFOA Coefficient	95% Confidence Interval Lower	Upper	p value	Adjusted PFOA Coefficient	95% Confidence Interval Lower	Upper	p value
ALT	0.00373	-0.01079	0.01825	0.61	0.00156	-0.02069	0.02381	0.89
Total Protein	-0.00028	-0.00073	0.00017	0.21	-0.00047	-0.00112	0.00017	0.15
Albumin	0.00022	-0.000540	-0.00009	0.16	-0.00034	-0.000803	0.00012	0.15
Globulin	-0.00006	-0.00044	0.00033	0.76	-0.00013	-0.00071	0.00044	0.65
Albumin/Globulin	-0.00007	-0.00035	0.00022	0.64	-0.00003	-0.00046	0.00040	0.89
Sodium	-0.00103	-0.00356	0.00151	0.42	-0.00160	-0.00530	0.00209	0.39
Potassium	0.00014	-0.00026	0.00053	0.49	0.00034	-0.00025	0.00093	0.25
Chloride	-0.00029	-0.0033	0.00278	0.85	-0.00345	-0.00752	0.00062	0.09
Carbon Dioxide	-0.00100	-0.00411	0.00210	0.52	0.00130	-0.00317	0.00576	0.56
Calcium	-0.00041	-0.00083	0.000003	0.05	-0.00036	-0.00095	0.00023	0.23

a. Adjusted for self-reported age, BMI (height and weight), alcohol use (≤ 4 drinks/week, and lipid lowering medication use (yes/no)

Table 10A. Unadjusted and Adjusted PFOA Regression Coefficients for All (N = 56) Subjects with Baseline and End-of-Project Measurements, Phase I of the Building 15 Demolition and Disposal Project

	Unadjusted PFOA				Adjusted[a] PFOA			
Dependent Variable	PFOA Coefficient	95% Confidence Interval Lower	Upper	p value	PFOA Coefficient	95% Confidence Interval Lower	Upper	p value
Total cholesterol	0.01745	-0.06272	0.02784	0.44	-0.01051	-0.06978	0.04877	0.72
Non-HDL cholesterol	-0.02292	-0.06784	0.02201	0.31	-0.02110	-0.07894	0.03674	0.47
HDL	0.00546	-0.01140	0.02232	0.52	0.01059	-0.01046	0.03165	0.32
Chol/HDL	0.00207	0.00036	0.00038	0.02	0.00212	0.00085	0.00043	0.06
LDL	0.01904	-0.04617	0.08425	0.56	0.02621	-0.06143	0.11387	0.55
Triglycerides	-0.40696	-0.60357	-0.21035	0.0001	-0.42673	-0.67693	-0.17654	0.001
Glucose	-0.03405	-0.08276	0.01466	0.17	-0.04638	-0.10679	0.01402	0.13
BUN	0.00053	-0.00668	0.00774	0.88	-0.00299	-0.01233	0.00636	0.52
Creatinine	-0.00011	-0.00029	0.00007	0.24	-0.00022	-0.00046	0.00002	0.07
Total Bilirubin	0.00002	-0.00038	0.00041	0.93	0.00003	-0.00051	0.00057	0.91
Alkaline Phos	0.00009	-0.01728	0.01710	0.99	-0.00202	-0.02363	0.01959	0.85
AST	-0.00784	-0.01716	0.00148	0.10	-0.01033	-0.02233	0.00166	0.09

Table 10A. (continued)

	Unadjusted PFOA				Adjusted[a] PFOA			
Dependent Variable	PFOA Coefficient	95% Confidence Interval Lower	Upper	p value	PFOA Coefficient	95% Confidence Interval Lower	Upper	p value
ALT	-0.01973	-0.03294	-0.00652	0.004	-0.02299	-0.04090	-0.00509	0.01
Total Protein	0.00023	-0.00033	0.00080	0.41	0.000041	-0.00035	0.00117	0.28
Albumin	-0.000003	-0.00032	0.00031	0.99	-0.00004	-0.00046	0.00038	0.83
Globulin	0.00024	-0.00020	0.00067	0.28	0.00045	-0.00009	0.00100	0.10
Albumin/Globulin	0.00002	-0.00029	0.00025	0.87	-0.00015	-0.00049	0.00019	0.38
Sodium	0.00175	-0.00200	0.00549	0.35	0.00109	-0.00351	0.00552	0.65
Potassium	0.00004	-0.00073	0.00082	0.91	0.00048	-0.00054	0.00151	0.34
Chloride	-0.00230	-0.00614	0.00155	0.24	-0.00265	-0.00773	0.00243	0.30
Carbon Dioxide	0.00448	0.00047	0.00850	0.03	0.00437	-0.00083	0.00956	0.10
Calcium	-0.00024	-0.00081	0.00034	0.42	-0.00043	-0.00118	0.00033	0.26

a. Full hierarchical model as explained in text

Table 10B. Adjusted PFOA Regression Coefficients for All (N = 56) Subjects with Baseline and End-of-Project Measurements. Includes Only Covariates with $p < 0.10$ Coefficients, Phase I of the Building 15 Demolition and Disposal Project

Dependent Variable	Adjusted PFOA Coefficient	95% Confidence Interval Lower	Upper	p value	Adjusted for in Model
HDL	0.00321	-0.01265	0.01908	0.69	sex
Triglycerides	-0.45527	-0.66000	-0.25054	<0.0001	lipid lowering med status
Chol/HDL	0.00187	0.00023	0.00350	0.03	sex
Glucose	-0.02069	-0.07138	0.02999	0.42	lipid lowering med status
Albumin/Glob	0.000001	-0.00026	0.00027	0.99	sex
Sodium	0.00223	-0.00122	0.00568	0.20	age, sex
Potassium	0.00011	-0.00066	0.00087	0.78	sex
Chloride	-0.00215	-0.00588	0.00157	0.25	age
Carbon Dioxide	0.00476	0.00037	0.00914	0.03	BMI, sex

Table 11A. Unadjusted and Adjusted PFOA Regression Coefficients for All (N = 45) Subjects with Baseline and End-of-Project Measurements That Had Increased PFOA Concentrations, Phase I of the Building 15 Demolition and Disposal Project

	Unadjusted PFOA				Adjusted[a] PFOA			
Dependent Variable	PFOA Coefficient	95% Confidence Interval Lower	Upper	p value	PFOA Coefficient	95% Confidence Interval Lower	Upper	p value
Total Cholesterol	-0.00812	-0.06558	0.04935	0.78	-0.01537	-0.09663	0.06590	0.70
Non-HDL Cholesterol	-0.02221	-0.08022	0.03581	0.44	-0.02816	-0.10830	0.05197	0.48
HDL	0.01409	-0.0062	0.03490	0.18	0.01280	-0.01688	0.04247	0.38
LDL	0.02362	-0.06976	0.11699	0.61	0.05509	-0.11479	0.22497	0.50
Chol/HDL	0.00254	0.00029	0.00479	0.03	0.00273	-0.00046	0.00592	0.09
Triglycerides	-0.47837	-0.73253	-0.22421	0.0005	-0.57213	-0.89957	-0.24468	0.001
Glucose	-0.02623	-0.08877	0.03631	0.40	-0.07210	-0.14597	0.00177	0.06
BUN	-0.00422	-0.01326	0.00483	0.35	-0.00083	-0.01445	0.01280	0.90
Creatinine	-0.000214	-0.00044	0.00001	0.06	-0.00019	-0.00051	0.00013	0.23
Total Bilirubin	-0.00003	-0.00042	0.00036	0.87	-0.00002	-0.00047	0.00050	0.94
Alkaline Phosphatase	-0.00012	-0.02056	0.02032	0.99	-0.00738	-0.03244	0.01768	0.55
AST	-0.00605	-0.01706	0.00496	0.27	-0.00686	-0.02143	0.007727	0.34

Table 11A. (continued)

	Unadjusted PFOA				Adjusted[a] PFOA			
Dependent Variable	PFOA Coefficient	95% Confidence Interval Lower	Upper	p value	PFOA Coefficient	95% Confidence Interval Lower	Upper	p value
ALT	-0.01844	-0.03328	-0.00361	0.02	-0.01503	-0.03539	0.00533	0.14
Total Protein	0.00024	-0.00048	0.00095	0.66	0.00033	-0.00068	0.00134	0.51
Albumin	-0.00008	-0.00047	0.00032	0.71	-0.00007	-0.00065	0.00050	0.80
Globulin	0.00031	-0.00022	0.00084	0.24	0.00040	-0.00037	0.00117	0.29
Albumin/Globulin	-0.00011	-0.00043	0.00021	0.48	-0.00007	-0.00056	0.00041	0.76
Sodium	-0.00123	-0.00556	0.00311	0.57	-0.00041	-0.00579	0.00497	0.88
Potassium	0.00045	-0.00046	0.00139	0.34	0.00100	-0.00018	0.00219	0.09
Chloride	-0.00559	-0.00982	-0.00135	0.01	-0.00415	-0.01029	0.00199	0.18
Carbon Dioxide	0.00493	0.00063	0.00925	0.03	0.00419	-0.00167	0.01004	0.15
Calcium	-0.00055	-0.00123	0.00013	0.11	-0.00070	-0.00169	0.00029	0.16

a. Hierarchical model as explained in text

Table 11B. Adjusted PFOA Regression Coefficients for All (N = 45) Subjects with Baseline and End-of-Project Measurements That Had Increased PFOA Concentrations. Models Include Only Those Covariates Where Coefficient(s) $p < 0.0001$, Phase I of the Building 15 Demolition and Disposal Project

Dependent Variable	Adjusted PFOA Coefficient	95% Confidence Interval Lower	Upper	p value	Adjusted for in Model
HDL	0.00899	-0.01093	0.028913	0.37	sex
Chol/HDL	0.00204	-0.00015	0.00423	0.07	sex
Creatinine	-0.00024	-0.00047	-0.00001	0.04	age
Total Bilirubin	0.00002	-0.00004	0.00004	0.92	alcohol, days between tests
Alk Phos	0.00465	-0.01536	0.02466	0.64	sex, lipid lowering med status
Sodium	-0.00089	-0.00498	0.00321	0.66	age, BMI, sex, alcohol
Potassium	0.00064	-0.00028	0.00156	0.17	sex
Carbon Dioxide	0.00385	-0.00046	0.00816	0.08	sex, alcohol

Figure 1A. 3M Cottage Grove Building 15 Demolition and Disposal Project. Phase I Baseline and End-of-Project PFOA Concentrations (ng/mL), N = 56 Subjects.

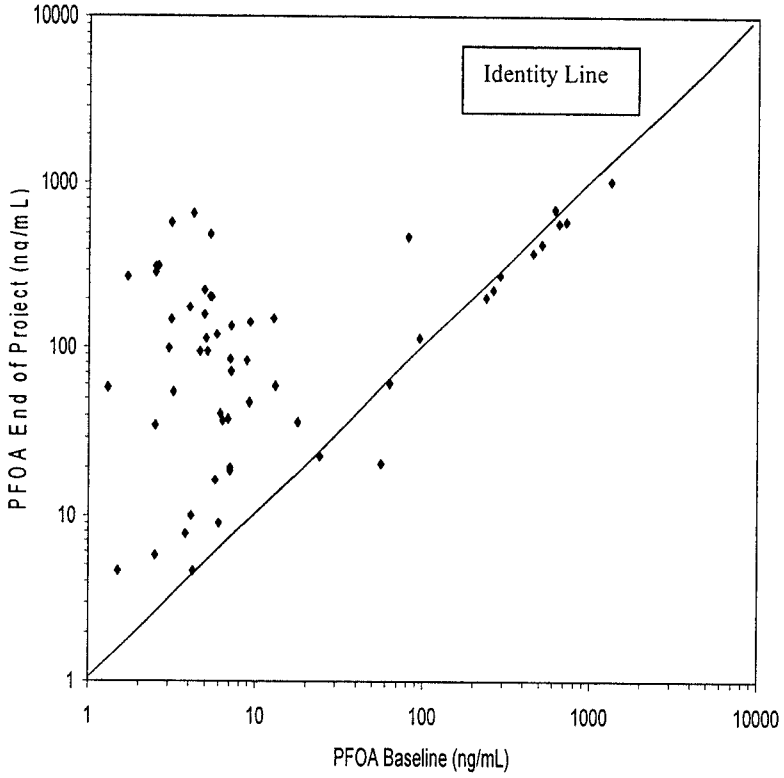

Figure 1B. 3M Cottage Grove Building 15 Demolition and Disposal Project. Phase I for N = 45 Subjects with Increased End-of-Project PFOA Concentrations (ng/mL) over Baseline.

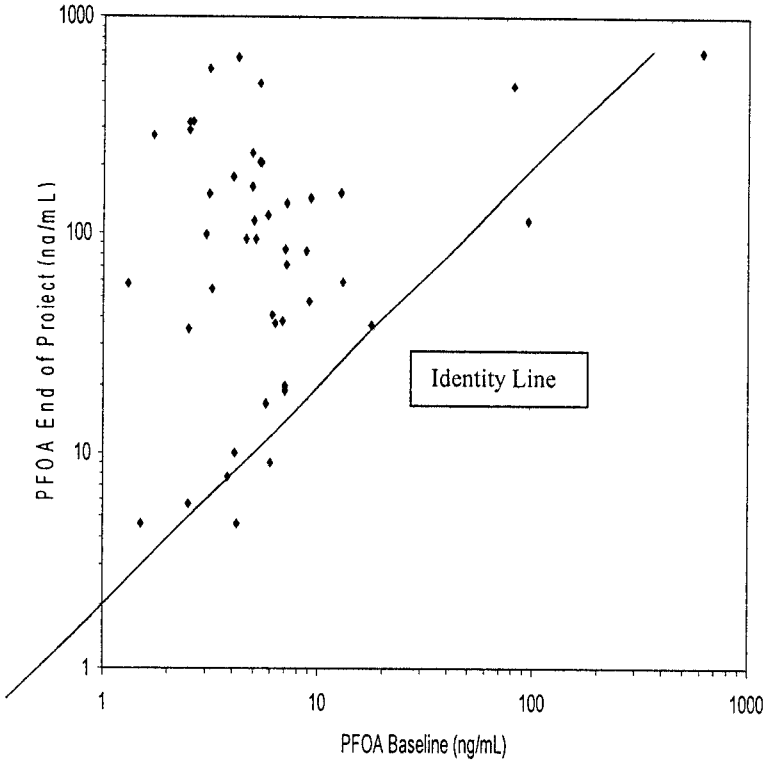

Figure 2A. 3M Cottage Grove Building 15 Demolition and Disposal Project. Phase I. Baseline and End-of-Project PFOS Concentrations (ng/mL) for N = 56 Subjects

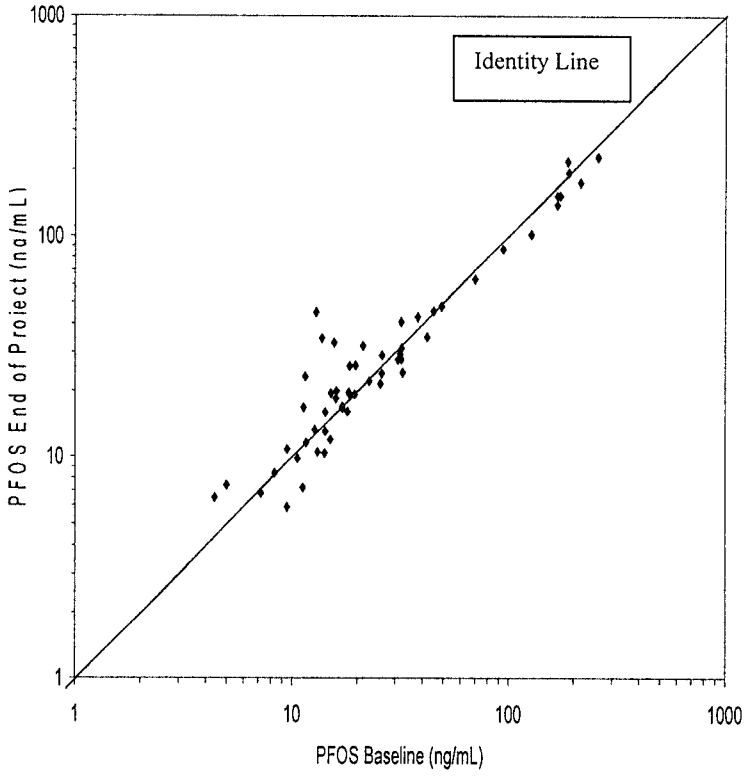

Figure 2B. 3M Cottage Grove Building 15 Demolition and Disposal Project. Phase I for PFOS Concentrations (ng/mL) for N = 45 Subjects with Increased End-of-Project PFOA Concentrations (ng/mL) over Baseline.

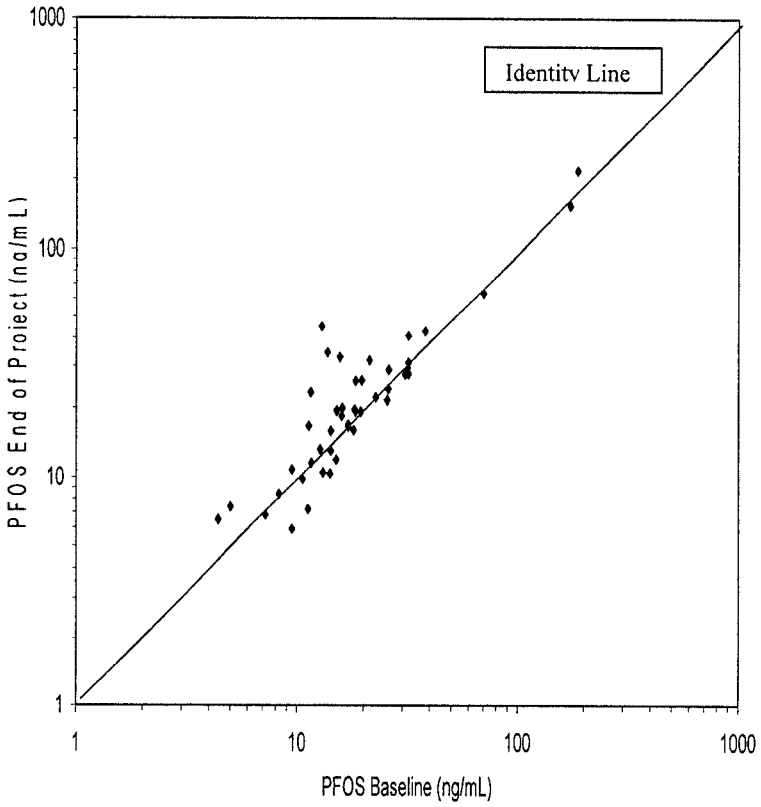

Figure 3a Total Cholesterol Difference by Change in PFOA

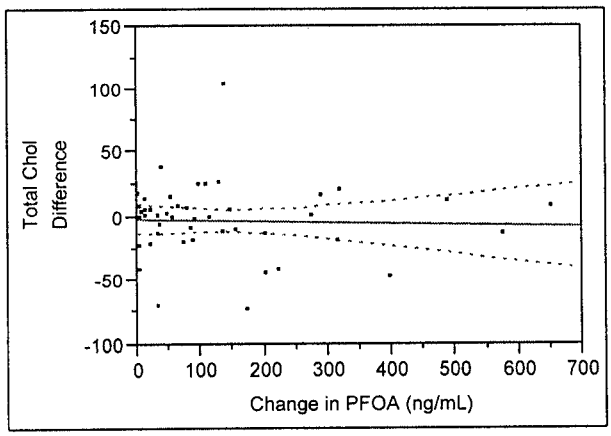

Figure 3b. Non-HDL Cholesterol Difference by Change in PFOA

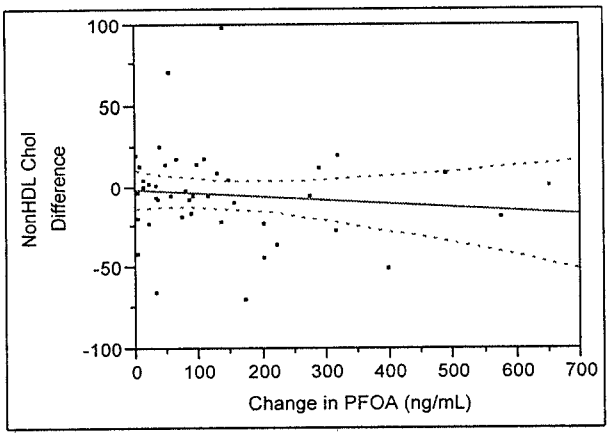

Figure 3c. HDL Cholesterol Difference by Change in PFOA

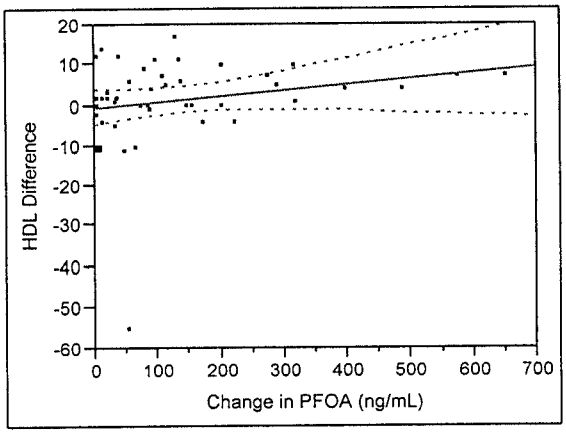

Figure 4a. Difference in Alkaline Phosphatase by Change in PFOA

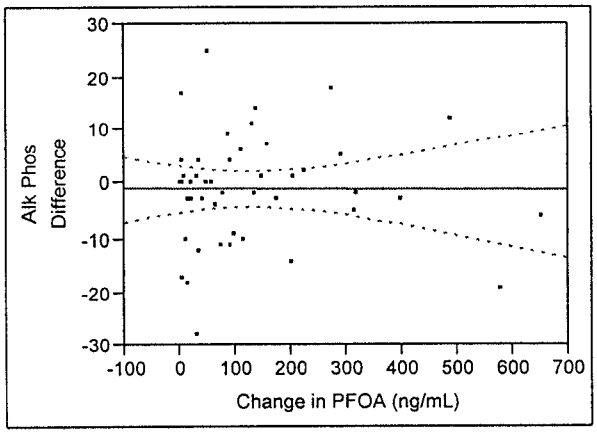

Figure 4b. Difference in AST by Change in PFOA

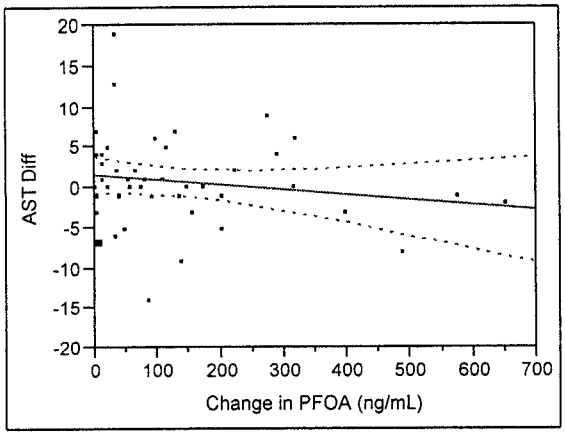

Figure 4c. Difference in ALT by Change in PFOA

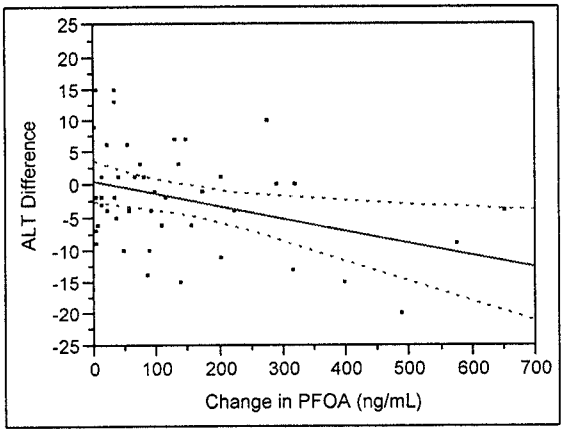

Figure 5. Mean PFOA Concentration (ng/mL) and Slope of Linear Relationship with Cholesterol in Epidemiology Research Studies. Adapted from Steenland et al. (2010)