FINAL REPORT

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Study
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Summary

The purpose of this report is to provide a descriptive analysis of the perfluorooctanoate (PFOA) and perfluorooctanesulfonate (PFOS) serum concentrations (ng/mL) measured at baseline and end-of-project time periods for workers involved in the 3M Decatur 304 project. At the end of 2012, a remediation project was performed on process piping and vessels associated with the 304 reactor system in Building 3 at 3M Decatur. A total of 34 workers (33 contract workers and 1 3M employee) participated in the biomonitoring assessment for this remediation project. Overall, these workers had a statistically significant increase in the mean matched-pair serum concentrations for PFOA [20.1 ng/mL (95% CI 10.8 – 29.5)] and PFOS [67.7 ng/mL (95% CI 39.0 – 96.3)]. These increased PFOA and PFOS concentrations were not significantly associated with the changes measured in total cholesterol, non-HDL, HDL, and the total cholesterol/HDL ratio during the same time period.

Introduction

Several 3M remediation projects have been completed regarding legacy perfluoroalkyls. In Minnesota, these projects have included the 3M Cottage Grove Building 15/73 demolition and disposal project, the 3M Woodbury landfill remediation project, the 3M Oakdale landfill remediation project, the 3M Cottage Grove Building 25 re-roof project, the 3M Cottage Grove D1/D2 and D9 projects, and the East Cove remediation projects. Also finalized was the biomonitoring assessment of SKB Environmental employees who hauled waste material from these projects and subsequently deposited it in the SKB Environmental licensed industrial landfill operations located in Rosemount, Minnesota.

At the 3M Decatur (Alabama) facilities, remediation activities focused on the demolition and disposal of the Buildings 2/48/49 (Olsen et al. 2011). Recently completed was the work on

the Decatur 304 project within Building 3 at Decatur. For all of these 3M projects in Minnesota and Alabama, 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 (PFOA and PFOS) was possible but due to the uniqueness of the work the magnitude of exposure was unknown. Both baseline and end-of-project assessments required response to a medical questionnaire and blood (serum) measurements of PFOS, PFOA, and several clinical chemistries.

The purpose of this report is to provide a descriptive analysis of the PFOA and PFOS serum concentrations (ng/mL) at baseline and end-of-project time periods for the 3M Decatur 304 project. At the end of 2012, demolition was performed on process piping and vessels associated with the 304 reactor system in Building 3 at 3M Decatur. In addition, the flaker that was located in close proximity to the 304 Area, had to be temporarily relocated until the demolition in the 304 Area was completed. Once relocated, the flaker was repositioned in close proximity to its original location and new duct work was run from the main exhaust duct in the area back to the flaker. Historically, the flaker was utilized to flake and package some C8-based materials. It should be noted that when the section of duct work was removed from the flaker to the main exhaust duct tie-in in the area, there was approximately 0.75 inches of residual material which had accumulated over time in the bottom of the removed duct. Contractors involved in this remediation provided job safety analyses. As an example, a job safety analysis (JSA) for the performance of the 304 demolition work provided three levels of personal protective equipment (PPE) requirements based on the nature of the task being performed associated with the project. These levels were identified as being Level I PPE, Level II PPE, and Level III PPE. Level I PPE corresponded to most stringent of the PPE requirements and was utilized for tasks which were

expected to have the highest exposure potential to residual materials in the process equipment. An example of a task with Level I PPE requirements would be the sawing and removal of process piping. Other tasks which were performed as part of the project with lesser exposure potential have the corresponding task-specific level of PPE requirements.

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

Unlike previous biomonitoring assessments, clinical chemistries for the Decatur 304 project involved only a lipid panel profile. This was an inadvertent ordering mistake by the nurse who requested this panel. Not included, therefore, were blood glucose, BUN, creatinine, serum electrolytes, and liver enzyme tests (including alkaline phosphatase, AST, ALT, and total bilirubin). Fasting was not a requirement because of the logistics of collecting blood samples during various times of the day. The lipid clinical chemistries were analyzed by Quest Diagnostics. Lipid analyses at baseline and at end-of-project were medically reviewed by Dr. Buehrer. 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. Buehrer regarding their lipid test results should the subject so desire.

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 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 "within" 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. Data Analyses

Because the data are paired samples, a matched-paired statistical analysis was performed where the mean value was determined for the average of the sum of the individual differences for the participants. See Equation 1.

Equation 1.

$$\text{Mean Change} = \sum_{N=34\ \text{participants}}(\text{end-of-project} - \text{baseline})/34$$

The mean 'matched-pair' change was calculated for PFOA, PFOS, total cholesterol, non-HDL cholesterol, HDL cholesterol, and a total cholesterol/HDL ratio. Ninety-five percent confidence intervals were calculated for this mean 'matched-pair' change.

Linear regression analyses examined the change in the clinical parameter by the change in the perfluoroalkyl concentration. Crude and adjusted coefficients were calculated. Covariates

included in the adjusted analysis were 1) days between baseline and end-of-project measurements; and 2) the subject's body mass index at baseline (BMI).

5. Communication

After each blood collection, individual letters were sent to the participants describing their lipid results. In the baseline assessments, two letters were sent. One referred to the lipid chemistries and the other letter provided baseline PFOA and PFOS concentrations. At the end-of-project, two letters were again sent to each participant. The first letter provided the individual's lipid results. The second letter compared baseline to end-of-project serum PFOA and PFOS concentrations. Aggregate (non-identifying) results were communicated to supervisors representing the participating contractors identified immediately below as Contractor "A", Contractor "B", and Contractor " C."

Results

A total of 34 workers involved in the Decatur 304 project had baseline and end-of-project biomonitoring assessments. One of these workers was a 3M employee; the others were involved with 5 contractor companies (number of workers in parenthesis): Contractor "A" (19); Contractor "B" (4); Contractor "C" (6); Contractor "D" (2); and Contractor "E" (2). There were 11 contract workers who participated in only a baseline assessment. Reasons for the nonparticipation at the end-of-project included: 1) some individuals never participated in the Decatur 304 project once it began; 2)others had left the contractor prior to completion of the project; and 3) others did not participate.

Figure 1 and Figure 2 show the relationship between baseline and end-of project PFOA and PFOS concentrations, respectively, for the 34 workers who had paired measurements. Values above or below the diagonal line represent increased or decreased changes. The majority of individuals had their PFOA and PFOS concentrations increase while working on the Decatur 304 project (i.e., their values were above the diagonal line in these 2 figures).

Overall, the 34 workers had statistically significant mean matched-pair increases of 20.1 ng/mL for PFOA and 67.7 ng/mL for PFOS while working on the Decatur 304 project (Table 1). The mean amount of time between baseline and end-of-project blood collections was 103 days (95% CI 99 – 107; range 86 – 127). The median matched-pair changes for PFOA and PFOS were comparable to the mean matched-pair changes (Table 1). The largest changes occurred with Contractor A where the mean matched-pair changes were 29.9 ng/mL for PFOA and 106.0 ng/mL for PFOS (Table 2). Contractor A was directly involved with the demolition performed on process piping and vessels associated with the 304 reactor system in Building 3; thus workers had the greatest potential for exposure. Results for contractors (D and E), who only had 2 workers each involved with the project, are not displayed in Table 2 to protect the confidentiality of the individual data (i.e., the range of data would have identified the 2 individual values for the contractor). The concentration changes were comparable to those reported for the ranges seen with Contractors B and C in Table 2.

Table 3 provides the distribution of lipid clinical parameters at baseline and end of project for the 34 workers. There were no statistically significant mean matched-pair changes in total cholesterol, non-HDL, HDL, or total cholesterol/HDL ratio (Table 4). These nonsignificant mean matched-pair changes included lower total cholesterol, non-HDL, and HDL values.

Excluding 5 individuals who reported they did not take lipid lowering medications, the remaining 29 individuals' mean matched-pair changes for PFOA and PFOS (Table 5) were comparable to those reported for all workers (Table 1). These 29 individuals did not have significant changes in their lipid parameters (Tables 6 and 7).

Including only workers (n = 21) who did not self-report taking cholesterol lowering medications and had end-of-project PFOA and PFOS concentrations higher than baseline levels, the findings resulted in somewhat higher mean matched pair changes of PFOA (30.9 ng/mL) and PFOS (100.9 ng/mL) (Table 8). Mean lipid changes trended nonsignificantly lower (Tables 9 and 10).

Tables 11, 12, and 13 provide the linear regression results for the 3 groups of workers discussed above: all workers (n = 34); those not taking lipid lowering medications (n= 29); and those not taking lipid lowering medications with only increased PFOA and PFOS concentrations (n = 21), respectively. The changes in total cholesterol, non-HDL, and total cholesterol/HDL ratio were not significantly associated with the changes in PFOA or PFOS in any of these analyses. The change in HDL was negatively associated with the change in PFOA and PFOS for all subjects (Table 11) but became less so with the more restricted worker categorizations and analyzing for adjusted coefficients (Tables 12 and 13). Covariates in the adjusted regression models included BMI [mean BMI = 30.6 (95% CI 28.5 – 32.7; range 20.7 – 49.2) and days between perfluoroalkyl blood collections (mean = 103 days)].

Discussion

Comparison of baseline and end-of-project serum concentrations of PFOA and PFOS provided each worker a method to assess his/her exposure experience in the Decatur 304

remediation project located in Building 3. Given the fact that exposure potential during the course of the remediation project was unknown, the use of biomonitoring data allowed for an assessment of the actual exposure. These analyses indicated there were occupational exposures for the majority of workers involved with this project with average increases in serum PFOA and serum PFOS concentrations over baseline values of approximately 50%. Mean matched pair increases were approximately 20 ng/mL and 70 ng/mL, respectively.

Because the biomonitoring data indicated there were increases in serum PFOA or PFOS concentrations, the clinical chemistry data were analyzed in relation to these changes. Potential exposure to any other material would not be known since only PFOA and PFOS were analyzed in the workers' serum. Total cholesterol, non-HDL cholesterol, and the total cholesterol/HDL ratio were not associated with the increased concentrations of PFOA and PFOS. A modest, nonsignfiicant inverse association was observed with changes in HDL and PFOA or PFOS although interpretation is difficult because of the relatively few workers involved in this Decatur 304 project.

The increased PFOA and PFOS concentrations observed among the workers in the Decatur 304 project were lower than the PFOA increases reported for workers involved with the Cottage Grove Building 15 Phase I demolition project (Olsen et al. 2010). In this Cottage Grove project, 45 workers involved in the demolition of Building 15 had higher end-of-project PFOA concentrations than at baseline, indicating occupational exposure to PFOA. For these 45 workers, the mean number of days between their paired measurements was 96 days (range 23 - 251). The 45 subjects' mean matched-pair PFOA concentration increase was 133.7 ng/mL (95% CI 87.3 - 180.2; $p < 0.0001$). The mean matched-pair difference for PFOS was 5.0 ng/mL (95% CI (-0.3) - 10.2; $p = 0.07$). The lack of any substantial 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 among these 45 workers' serum lipids, renal function or liver clinical chemistries related to the change in serum PFOA concentration from baseline to end-of-project.

A similar analysis was conducted of 47 workers at 3M Decatur who were involved in the demolition of Buildings 2/48/49 (Olsen et al. 2011). Their end-of-project PFOA and PFOS concentrations increased (or remained the same) and had mean matched-pair concentration increases of 10.3 ng/mL for PFOA and 12.0 ng/mL for PFOS, respectively (both $p < 0.0001$). For these 47 subjects, there were no statistically significant mean changes for total cholesterol (-1.4 mg/dL, $p = 0.70$) or non-HDL (-4.2 mg/dL, $p = 0.22$). There was a statistically significant increase in the mean HDL (2.8 mg/dL, $p = 0.006$). Linear regression analyses did not result in statistically significant associations between changes total cholesterol, non-HDL, and HDL with the increased concentrations of PFOA and PFOS.

As reviewed by Steenland et al. (2010) and Olsen et al. (2012) positive associations have been reported between PFOA concentrations and non-HDL cholesterol levels in cross-sectional investigations of a random sample of the non-institutionalized United States general population (Nelson et al. 2010) via the National Health Nutrition Examination Survey (NHANES) and a mid-Ohio River community affected with contaminated drinking water (Steenland et al. 2009; Frisbee et al. 2010). Other large cross-sectional epidemiologic studies have (Eriksen et al. 2012) and have not (Patel et al. 2012;Fisher et al. 2013) observed such associations. The magnitude of the non-linear associations reported in cross-sectional studies has not been observed in cross-sectional studies of perfluoroalkyl manufacturing workers (Sakr et al. 2007a; Olsen and Zobel 2007) or observed in longitudinal (Sakr et al. 2007b; Olsen et al. 2012) investigations of workers

(Sakr et al. 2007b; Olsen et al. 2012). The positive epidemiologic associations from cross-sectional studies are contrary to well-established modes of action reported in toxicology studies that have demonstrated a hypolipidemic effect (Olsen et al. 2012). Nevertheless, Fletcher et al. (2013) recently reported several associations between serum PFOA and PFOS levels and changes in the expression of human genes involved in either cholesterol transport or mobilization. Fletcher et al. suggested this led to a "hyperchoesterolemic environment". However, their findings have minimum diagnostic or predictive value because they only evaluated transcript concentrations in peripheral lymphocytes without other functional measures such as protein concentrations, free cholesterol and esterified cholesterol content of the cells, or enzyme activity.

Conclusion

A total of 34 workers participated in a biomonitoring assessment of the Decatur 304 project. The majority of workers had occupational exposure to PFOA and PFOS during the course of the project. The mean change in concentration was 20.1 ng/mL and 67.7 ng/mL, respectively, that occurred over a mean duration of 103 days. The findings did not suggest a positive association between increasing cholesterol levels and PFOA/ PFOS concentrations. The study was limited because of its small sample size, the limited duration of follow-up, and the inadvertent omission to analyze for other clinical parameters.

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Table 1. PFOA and PFOS Concentrations (ng/mL) at Baseline and End-of-Project with Matched-Pair Changes, All Subjects (N = 34)

	Baseline		End-of-Project		Matched Pair Change	
	PFOA	PFOS	PFOA	PFOS	PFOA	PFOS
Mean	41.3	77.6	61.4	145.2	20.1*	67.7*
(95% CI)	(15.5 - 67.1)	(43.6 - 111.6)	(34.6 - 88.3)	(109.8 - 180.7)	(10.8 - 29.5)	(39.0 - 96.3)
Median	14.1	46.3	44.7	120.5	18.5	58.2
Range	1.8 – 381.0	5.6 – 513.0	4.3 - 442	8.6 – 451.0	(-26.0) - 95.3	(-62) - 325.3

*Matched-pair p value <0.0001

Table 2. PFOA and PFOS Concentrations (ng/mL) for 3 Contractors Working on Decatur 304 Project at Baseline and End-of-Project with Matched-Pair Changes

Table 2A (Contractor A) (N = 19)

	Baseline		End-of-Project		Matched-Pair Change	
	PFOA	PFOS	PFOA	PFOS	PFOA	PFOS
Mean	16.1	41.4	46.0	147.4	29.9*	106.0*
95% CI	(7.5 – 24.7)	(23.8 – 59.0)	(33.7 – 58.3)	(16.2 – 188.6)	(17.0 – 42.8)	(64.7 – 147.2)
Median	10.2	28.1	40.6	142.0	23.3	72.3
Range	1.8 – 73.6	5.6 – 135.0	10.8 – 99.5	27.7 – 337.0	(-15.5) – 95.3	(-0.4) – 325.3

*p < 0.0001

Table 2B (Contractor B) (N = 4)

	Basline		End-of-Project		Matched-Pair Change	
	PFOA	PFOS	PFOA	PFOS	PFOA	PFOS
Mean	29.8	82.7	49.1	127.6	19.3**	44.9**
SE[a]	10.4	19.7	12.2	35.3	8.3	19.8
Median	29.4	77.5	56.3	125.5	18.9	48.0
Range	9.4 – 51.0	44.6 – 131.0	14.9 – 68.8	43.3 – 216.0	0.1 – 39.1	(-1.3) – 85.0

**p < 0.05

[a] Standard Error

Table 2C (Contractor C) (N = 6)

	Baseline		End-of-Project		Matched-Pair Change	
	PFOA	PFOS	PFOA	PFOS	PFOA	PFOS
Mean	32.9	58.2	36.6	73.2	3.8***	15.0****
SE[a]	17.8	21.9	16.5	43.2	11.1	40.2
Median	19.4	42.1	22.7	82.2	0.8	4.9
Range	2.6 – 118.0	9.5 - 132.0	4.3 - 115.0	8.6 - 116.0	(-9.7) - 20.1	(-30.4) - 70.9

***p = 0.22; ****p = 0.20

[a] Standard Error

Table 3 Distribution of Lipid Clinical Parameters at Baseline and End-of-Project, All Subjects (N = 34)

	Baseline				End-of-Project			
	Total Chol	Non-HDL	HDL	T Chol/HDL	Total Chol	Non-HDL	HDL	T Chol/HDL
Mean	183	141	42	4.6	177	136	41	4.5
(95% CI)	(169 - 197)	(127-155)	(39 - 45)	(4.1 - 5.1)	(162 - 191)	(122 - 149)	(37 - 44)	(4.0 - 4.9)
Median	189	151	41	4.5	182	145	40	4.4
Range	95 - 250	59 - 204	19 - 61	2.4 - 8.4	100 - 255	60 - 224	24 - 74	2.2 - 8.2

Table 4. Matched-Pair Changes of Lipid Parameters, All Subjects (N = 34)

	Total Chol	Non-HDL	HDL	Total Chol/HDL
Mean	-6.7	-5.4	-1.4	-0.1
(95% CI)	((-14.7) - 1.3)	((-13.0 – 2.3)	((-3.9) - 1.2)	((-0.4 - 0.2)
Median	-5.0	-3.0	0.0	0.0
Range	(-61) - 37	(-56) - 31	(-20) - 13	-2.5 - 1.2

Table 5. Matched-Pair Changes of PFOA and PFOS Concentrations (ng/mL) for Subjects Who Self-Reported Not Taking Lipid Lowering Medications (N = 29)

	Baseline		End-of-Project		Matched- Pair Change	
	PFOA	PFOS	PFOA	PFOS	PFOA	PFOS
Mean	46.7	84.9	66.6	154.9	19.8*	70.0**
(95% CI)	(16.7 – 76.8)	(45.4 – 124.4)	(35.4 – 97.8)	(115.3 – 194.5)	(9.2 - 30.5)	(37.1 – 102.8)
Median	19.6	58.6	45.5	124.0	17.7	55.6
Range	1.8 - 381	5.6 - 513	10.8 - 442	35.4 - 451	(-26.0) - 95.3	(-62) - 325.3

*$p < 0.001$

** $p < 0.0001$

Table 6. Distribution of Lipid Clinical Parameters at Baseline and End-of-Project, Subjects Not Taking Lipid Lowering Medications (N = 29)

	Baseline				End-of-Project			
	Total Chol	Non-HDL	HDL	T Chol/HDL	Total Chol	Non-HDL	HDL	T Chol/HDL
Mean	178	137	41	4.5	172	133	40	4.4
(95% CI)	(164 - 193)	(122 - 152)	(38 - 45)	(4.0 - 5.1)	(157 - 187)	(128 - 146)	(37 - 43)	(4.0 - 4.8)
Median	188	148	41	4.5	177	142	40	4.4
Range	95 - 233	59 - 202	19 - 61	2.4 - 8.4	100 - 235	60 - 189	24 - 56	2.2 – 6.9

Table 7. Matched-Pair Change of Lipid Parameters, Subjects Self-Reported Not Taking Lipid Lowering Medications (N = 29)

	Total Chol	Non-HDL	HDL	Total Chol/HDL
Mean	-6.4	-5.1	-1.3	-0.1
(95% CI)	((-14.5) - 1.7)	((-12.6 – 2.4)	((-4.0) - 1.5)	((-0.4) - 0.2)
Median	-3.0	-1.0	0.0	0.0
Range	(-51) - 37	(-53) - 30	(-20) - 8	(-2.5) - 1.2

Table 8. PFOA and PFOS Concentrations (ng/mL) for Subjects Not Taking Lipid Lowering Medications and Whose End-of-Project PFOA and PFOS Concentrations Were Higher than Baseline (N = 21)

	Baseline		End-of-Project		Matched-Pair Change	
	PFOA	PFOS	PFOA	PFOS	PFOA	PFOS
Mean	34.0	58.8	64.9	169.7	30.9*	100.9*
(95% CI)	(0 – 70.9)	(28.7 - 88.9)	(23.9 - 105.9)	(126.9 – 303.6)	(19.6 - 42.1)	(64.0 - 137.8)
Median	9.9	24.7	42.5	142.0	22.0	72.3
Range	1.8 - 381	5.6 - 281	10.8 - 442	35.4 - 365	(2.8) - 95.3	(10.7) - 325.3

*$p < 0.0001$

Table 9. Distribution of Lipid Clinical Parameters of Subjects (N = 21) Not Taking Lipid Lowering Medications and Whose End-of-Project PFOA and PFOS Concentrations Were Higher than the Baseline

	Baseline				End-of-Project			
	Total Chol	Non-HDL	HDL	T Chol/HDL	Total Chol	Non-HDL	HDL	T Chol/HDL
Mean	179	138	42	4.4	171	130	40	4.3
(95% CI)	(161 - 197)	(119-156)	(38 - 45)	(3.9 - 5.0)	(154 - 188)	(113 - 148)	(38 - 43)	(3.8 - 4.8)
Median	183	148	40	4.5	177	142	40	4.2
Range	95 - 233	59 - 202	31 - 61	2.4 – 7.5	100 – 235	60 - 189	28 - 55	2.2 – 6.3

Table 10. Matched-Pair Change of Lipid Parameters for Subjects Self-Reported Not Taking Lipid Lowering Medications and Whose End-of-Project PFOA and PFOS Concentrations Were Higher than Baseline Levels (N = 21)

	Total Chol	Non-HDL	HDL	Total Chol/HDL
Mean	-8.3	-7.1	-1.1	-0.1
(95% CI)	((-18.1) - 1.6)	((-16.0) - 2.0)	((-4.6) - 2.3)	((-0.4 - 0.2)
Median	-7.0	-5.0	1.0	-0.1
Range	(-51) - 37	(-53) - 30	(-20) - 8	(-1.6) - 1.2

Table 11. Regression Coefficients of Change (Δ) in Lipid Parameter (y) by Change (Δ) in Perfluoroalkyl (PFOA or PFOS) (x), All Subjects (N = 34)

	Δ PFOA				Δ PFOS			
	Crude Coefficient	p value	Adjusted* Coefficient	p value	Crude Coefficient	p value	Adjusted Coef ficient	p value
Δ Total Cholesterol	-0.1753	0.25	-0.1768	0.26	-0.0748	0.13	-0.0785	0.13
Δ Non-HDL	-0.0868	0.55	-0.0873	0.56	-0.0455	0.33	-0.0478	0.33
Δ_HDL	-0.0885	0.06	-0.0895	0.07	-0.0294	0.06	-0.0307	0.06
Δ Total Chol/HDL Ratio	0.0049	0.36	0.0053	0.34	0.0006	0.74	0.0007	0.69

*Adjusted regression coefficient for BMI and number of days between baseline and end-of-project assessments.

Table 12. Regression Coefficients of Change (Δ) in Lipid Parameter (y) by Change (Δ) in Perfluoroalkyl (PFOA or PFOS) (x), Subjects Not Taking Lipid Lowering Medications (N = 29)

	Δ PFOA				Δ PFOS			
	Crude Coefficient	p value	Adjusted* Coefficient	p value	Crude Coefficient	p value	Adjusted Coef ficient	p value
Change in Total Cholesterol	-0.1503	0.30	-0.1391	0.35	-0.0665	0.16	-0.0621	0.20
Change in Non-HDL	-0.0786	0.57	-0.0713	0.61	-0.0423	0.34	-0.0393	0.40
Change in HDL	-0.0717	0.15	-0.0678	0.20	-0.0241	0.13	-0.0228	0.19
Change in Total Chol/HDL Ratio	0.0039	0.46	0.0041	0.47	0.0005	0.79	0.0005	0.79

*Adjusted regression coefficient for BMI and number of days between baseline and end-of-project assessments.

Table 13. Regression Coefficients of Change (Δ) in Lipid Parameter (y) by Change (Δ) in Perfluoroalkyl (PFOA or PFOS) (x) for Subjects Not Taking Lipid Lowering Medications and Whose End-of-Project PFOA and PFOS Levels Were Higher than Beginning (N = 21)

	Δ PFOA				Δ PFOS			
	Crude Coefficient	p value	Adjusted* Coefficient	p value	Crude Coefficient	p value	Adjusted Coef ficient	p value
Δ Total Cholesterol	-0.1235	0.54	-0.0596	0.76	-0.0519	0.40	-0.0308	0.62
Δ Non-HDL	0.0039	0.98	0.0552	0.76	-0.0121	0.83	-0.0046	0.94
Δ HDL	-0.1274	0.06	-0.1148	0.11	-0.0396	0.05	-0.0354	0.12
Δ Total Chol/HDL Ratio	0.0058	0.34	0.0065	0.29	0.0015	0.42	0.0017	0.38

*Adjusted regression coefficient for BMI and number of days between baseline and end-of-project assessments.

Figure 1. Scatterplot of Baseline and End-of-Project PFOA Concentrations (ng/mL) for the 3M Decatur 304 Project, N = 34 Subjects. Diagonal is the 'identity line'.

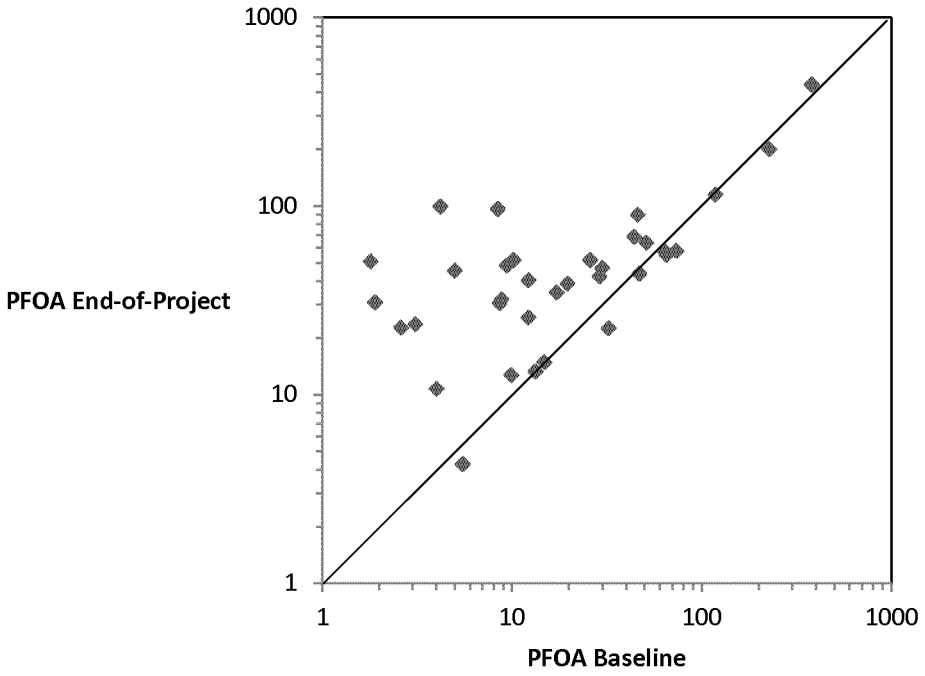

Figure 2. Scatterplot of Baseline and End-of-Project PFOS Concentrations (ng/mL) for the 3M Decatur 304 Project, N = 34 Subjects. Diagonal is the 'identity line'.