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**SELF-REPORTED HEALTH EFFECTS AMONG
COMMUNITY RESIDENTS EXPOSED TO
PERFLUOROOCTANOATE***

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ABSTRACT

Serious health effects due to perfluorooctanoate (PFOA) exposure are suspected. The aim of this study was to evaluate the health status of nearby residents, with prolonged exposure to PFOA in their drinking water. A population of 566 white residents who were plaintiffs or potential plaintiffs in a lawsuit was evaluated by questionnaire for health history and symptoms. Standardized Prevalence Ratios were estimated using National Health and Examination Survey (NHANES) data files for comparison rates. The exposed subjects reported statistically significant greater prevalence of angina, myocardial infarction, and stroke (SPR = 8.07, 95% C.I. = 6.54 – 9.95; SPR = 1.91, 95% C.I. = 1.40 – 2.62, and SPR = 2.17, 95% C.I. = 1.47 – 3.21, respectively), chronic bronchitis, shortness of breath on stairs, asthma (SPR = 3.60, 95% C.I. = 2.92 – 4.44; SPR = 2.05, 95% C.I. = 1.70 – 2.46; SPR = 1.82, 95% C.I. = 1.47 – 2.25, respectively), and other serious health problems. The increased prevalence of adverse health effects may be due to PFOA. Further study is needed.

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Organic fluorinated chemicals have been the focus of scientific and public health debate for some time. Most of the studies on these compounds have examined perfluorooctyl sulfonate (PFOS) and perfluorooctanoic acid (PFOA). In addition, PFOS and PFOA are commonly present and persist in the environment [1]. PFOA is produced as an emulsifier in the manufacturing process for fluoropolymers [2]. Since the 1980s, there has been a steady increase in the use of fluoropolymers due to their many useful properties [3]. Because of their specific chemical and physical properties, fluoropolymers are used in electrical insulation, fire foam, floor polish, insecticides, Teflon, stain-resistant treatments for upholstery, carpet, textiles, and oil-resistant treatments for paper and plastic [4].

Several sources of human exposure have been suggested, including environmental releases and some consumer products, but due to a dearth of research, specific pathways responsible for human exposure are not well understood [5]. Potential pathways for human exposure include food, water, leaching from commercial products such as food packaging, contact with products such as clothing, and inhalation of dust or vapor phase precursors coming off of household products such as carpet [6, 7-9, 10].

Perfluoroalkylated substances (PFAS) are eliminated slowly from the human body [11]. The biological half-life of PFOA is approximately 4½ years [12]. PFOA is persistent in the environment. It does not hydrolyse, photolyse or biodegrade under environmental conditions [13]. Bioaccumulation [14] and ubiquitous exposure to U.S. citizens heightens concern about adverse health effects [15, 16].

Although a number of studies have been conducted to determine the toxicity associated with PFAS, the relative toxicities of the different chemical structures like PFOA and PFOS are not clearly understood.

In rodent studies, PFAS cause multiple adverse health effects [17]. Endocrine disruption is observed with elevated estrogen and decreased testosterone in animal studies [18, 19]. Elevated Thyroid Stimulating Hormone (TSH) and elevated prolactin have been observed in humans [20, 21]. Increased liver weight has been documented in rats [22] and in monkeys [23]. Dose-dependent hepatic enzyme elevations have been seen in PFOA-exposed workers [24] and recently a small sample of PFOA workers in Trissino, Italy, was found to have slightly increased levels of non-HDL cholesterol and total cholesterol with increasing blood levels of PFOA [25]. In addition, a study in January 2005 reported statistically significant increases in cholesterol fractions (total and LDL) and increases in triglycerides in the highest serum PFOA exposure group [26]. Rat studies have shown liver and pancreatic neoplasms [19]. Studies of PFOA-exposed workers have shown increased rates of cancer, including prostate [27], kidney [28], bladder [28, 29], colon, testes, and other cancer types [28].

This article reports on the results of a cross-sectional comparison study of self-reported health history and symptoms. A separate manuscript reports an increase of cancer prevalence among this PFOA-exposed population [30].

MATERIALS AND METHODS

Study Subjects

We administered a questionnaire to volunteers from a population of approximately 23,900 households (approximately 70,010 individuals) near a Teflon manufacturing plant located along the Ohio River in Wood County, West Virginia. All of our study participants were mandatorily included in a law suit, regardless of their individual choice to be included, because a non-opt-out class action was certified against the local plant on behalf of all persons whose drinking water was contaminated with PFOA (>0.05 parts per billion (ppb)).

PFAS has been used at this plant since the early 1950s. The plant uses PFAS to manufacture Teflon and other materials and has consistently released perfluoroalkylated substances into the surrounding areas. According to the Environmental Working Group (EWG), in 1999 alone, the plant released more than 40 tons (86,806 pounds) of PFOA into the air and the Ohio River. The plant reports it reduced emissions to 10 tons (20,168 pounds) in 2002 (EWG). PFOA has been present in the plant's drinking water at 1-5 parts per billion (ppb). PFOA levels above 0.05 parts per billion have also been detected in public and private drinking water supplies in communities on both the Ohio and West Virginia sides of the Ohio River near the plant, including the water supply of the neighboring manufacturing plant.

Study participants were recruited through a public invitation via TV, radio, and newspaper advertisements inviting all users of the PFOA-contaminated public and private drinking water sources to participate in a study by filling out a questionnaire. Over a period of three days in August 2003, five hundred and ninety-nine (599) volunteers or 2.5% from the targeted population participated in the study. Most were white. Each person who filled out a questionnaire reported exposure to PFOA-contaminated water for at least a year. They included subjects who used water with PFOA levels as low as 0.05 parts per billion (ppb), such as from Pomeroy, Ohio, and Mason, West Virginia, which is similar to levels measured elsewhere in the greater region. However, the majority (65%) who participated were exposed to water provided by the Lubeck Public Service District in West Virginia where PFOA ranged from 0.4-3.9 ppb and the Little Hocking Water Association in Ohio where PFOA ranged from 1.7-4.3 ppb. Each water district in the area reported PFOA levels including well testing data, and company records have released information on PFOA in the company drinking water (see Table 1). Some study subjects worked in the plant where PFAS were used and therefore had both residential and occupational exposures.

Data Collection and Questionnaire

We collected data on 599 adults exposed to PFOA in their drinking water from a nearby manufacturing plant. Trained and experienced proctors administered

Table 1. PFOA Levels by Water District/Source and Percent of Study Subjects Reporting Water Consumption from Each Source

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

^aAll PFOA levels were reported by their respective water districts and are available via the Internet.

^bEstimated as 2.8 people per household.

^cCompany employment reports.

Note: N/A not applicable.

the questionnaire in groups of 30 to 40 persons or individually by telephone interview. In the group sessions, all subjects were given the same instruction and proctors were available to answer questions.

The questionnaire explores demographics, including age, gender, occupational and residential history as well as medical, social, and behavioral history. We asked about health symptoms, diseases, surgeries, medications, family history, income, chemical exposures, and lifestyle measures including smoking and drinking.

One unlikely symptom question is designed to test for the veracity of the responses provided. Questionnaire responses are machine-readable, scanned on-site, and verified before subjects leave. This basic questionnaire has been used in prior studies of exposed and unexposed groups [31].

Comparison Databases

The Centers for Disease Control and Prevention's (CDC) National Center for Health Statistics (NCHS), Division of Health Examination Statistics (DHES) has conducted health and nutrition surveys since the early 1960s and is designed to obtain a nationally representative sample of the health and nutritional status of the U.S. population. Details for each survey in addition to the public use data files are available online (CDC).

The most recent National Health and Nutrition Examination Survey (NHANES) began in 2001. Every year, approximately 7,000 civilian, non-institutionalized U.S. citizens of all ages are interviewed; of these citizens, approximately 5,000 complete the health examination component of the survey. NHANES 2001-2002 over-samples low-income persons, adolescents 12-19 years, persons 60+ years of age, African Americans and Mexican Americans.

Statistical Methods

Frequencies and percents for demographic, social, behavioral and physical characteristics are presented. Standardized Prevalence Ratios (SPRs) for certain disease endpoints were calculated using the NHANES 2001-2002 data files for comparison rates. SPRs are simply observed to expected ratios, (number of cases in exposed population/total number exposed) divided by (number of cases in the comparison population/total in the comparison population). A value of one represents the null or an equal value in both the observed population (survey data) and the expected population (NHANES). Values greater than one indicate higher prevalence in the observed population compared to expected and values less than one indicated lower prevalence in the observed compared to expected. Statistical significance is demonstrated with either *p*-values, confidence limits, or both. Other studies have used the NHANES data files for similar analyses [33].

NHANES data was restricted to non-Hispanic whites 18 and older to be comparable to the exposed population. Only whites were included in the analysis because the number of non-white participants was too small to yield robust results. Rates were calculated for each medical condition using sampling weights developed specifically for the NHANES data source, controlling for age and gender. The sampling weights account for the sampling methods as well as the non-response rates, and they render the data representative of the total U.S. population. The weights also provide for accurate assessment of the sampling error of statistics based on these survey data [34].

Exposed subjects were restricted to those who had lived in the area for a minimum of one year. The health effects of participants who were diagnosed prior to the time of residence were excluded from the analysis.

The 76 subjects who had worked at the Teflon plant were compared to the general population of the exposed area. They were subsequently included in the exposed group because no worker-exposed differences were found.

RESULTS

The questionnaire was administered to 599 subjects. Ten of the subjects were less than 18 years old and 21 were non-white, and two subjects were missing age and were therefore excluded from the analyses. The remaining group of 566 residentially PFOA-exposed adults are middle aged (49.9 years with a standard deviation (SD) of 14.43 years), a balanced proportion of males and females, with slightly more than high school attainment of education (12.76 years of school, SD 2.18). The average body mass index is 28.36 kg/m² with a large SD (+/- 6.41). Almost half (48.25%) of the sample can be classified as over-weight (BMI > 28), one-third (33.22%) as average (BMI = 23 – 28), and the remaining (18.53%) as underweight (BMI < 23).

Table 2 shows the Standardized Prevalence Ratios (SPRs) comparing the exposed group and NHANES 2001-2002 data while controlling for age and gender. The significantly elevated SPR for cardiovascular problems was 4.29 (95% C.I. = 3.47 – 5.29), and included is that of angina (SPR = 8.07; 95% C.I. = 6.54 – 9.95), myocardial infarction (SPR = 1.91; 95% C.I. = 1.40 – 2.62), and stroke (SPR = 2.17; 95% C.I. = 1.47 – 3.21). Respiratory health effects were elevated for chronic bronchitis (SPR = 3.60; 95% C.I. = 2.92 – 4.44) and asthma (SPR = 1.82; 95% C.I. = 1.47 – 3.21). The prevalence rate for shortness of breath was also elevated (SPR = 2.05; 95% C.I. = 1.70 – 2.46). Thyroid problems and diabetes were marginally elevated in this PFOA-exposed group (SPR = 1.56; 95% C.I. = 0.22 – 1.98 and SPR = 1.54; 95% C.I. = 1.16 – 2.05, respectively). The overall rate of liver disease was not significantly elevated. The prevalence of liver problems was 3.4% (18 persons) with liver disease diagnosed by their doctors and 10.6% (59 persons) reporting elevated liver enzymes.

Table 3 examines selected outcomes by age and gender. The effect of exposure appears to be exaggerated in young adulthood for several of the listed outcomes: high blood pressure, cardiovascular problems, and liver problems. By age 65 and over, the effect measure is either at the null (liver, kidney, and thyroid disease in males) or greatly reduced (cardiovascular problems). However, the exposed population (EP)/US ratios for the older population remain high for asthma and chronic bronchitis, and are slightly elevated for high blood pressure, shortness of breath, and cardiovascular problems.

DISCUSSION

The U.S. EPA pressured perfluorooctane sulfonic acid (PFOS) off the market in 2000 because of its persistence in the environment and human body, concerns

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Table 2. Standardized Prevalence Ratio (SPR) Comparing Observed Disease Rate Per 100,000 among a Residentially PFOA-Exposed Population to the Expected Disease Rate of the General U.S. Population Controlling for Age and Gender

Disease or symptom type	Number diseased in exposed	Observed rates (per 100,000)	Expected rates ^a (per 100,000)	SPR	CI ^b
Cardiovascular problems ^c	170	30,088	7,019	4.29	3.47–5.29*
Chronic bronchitis	113	22,114	6,145	3.60	2.92–4.44*
Kidney disease	21	3,757	1,665	2.26	1.45–3.51*
Shortness of breath on stairs	323	57,270	27,994	2.05	1.70–2.46*
Asthma	105	20,669	11,369	1.82	1.47–2.25*
Thyroid problems	82	15,589	10,019	1.56	1.22–1.98*
Diabetes	56	9,947	6,457	1.54	1.16–2.05*
High blood pressure	186	33,096	28,077	1.18	0.97–1.43
Liver problems	19	3,754	3,728	1.01	0.64–1.59

^aExpected rates are from NHANES 2001-2002 using sampling weights to calculate an unbiased estimate of national rates while adjusting for non-response, survey design, and sampling technique while giving an accurate estimate of sampling error.

^bConfidence Interval

^cIncludes MI, Stroke, Angina

*Statistically significant ($p \leq 0.05$)

about its toxicity, and the fact that it is widely found in human populations and wildlife [3, 11, 14]. A short time later, one main producer, 3M, discontinued the perfluorooctanesulfonyl fluoride chemistry used to manufacture PFOS-based chemicals. Animal studies have confirmed that PFOA and PFOS are toxic [5, 35, 36] and worker studies indicate human health effects [29, 37]. To our knowledge, this is the first study evaluating the health status of residents living near a Teflon production facility.

The reported emissions from the production facility have contaminated the local water supply. Public drinking water delivered to those living near the plant contains more than 0.05 ppb PFOA as reported by the water districts in the area. Neighbors are exposed to much higher than background levels of PFOA in drinking water and air [37, 38]. A recent study conducted by Edward

Table 3. Prevalence Ratios (PR) Comparing Observed Disease Rate per 100,000 among a
Residentially PFOA-Exposed Population to the Expected Disease Rate of the General U.S. Population
by Age Group and Gender for Various Disease Outcomes

Age group	Males		Females		Prevalence ratio	
	Age specific rates (US ^a)	Age specific rates (EP ^b)	Age specific rates (US)	Age specific rates (EP)	EP/US males	EP/US females
Asthma						
18-34	12543.87	37209.30	15209.92	30000	2.97	1.97
35-49	7895.13	14705.88	15149.32	21052.63	1.86	1.39
50-64	9363.58	12903.23	13065.51	21568.63	1.38	1.65
65+	5694.06	19047.62	10790.07	18181.82	3.35	1.69
Chronic Bronchitis						
18-34	4136.27	23255.91	5867.84	18000	5.62	3.07
35-49	4716.72	20000	8192.81	25333.33	4.24	3.09
50-64	2870.57	18750	8022.41	27884.62	6.53	3.48
65+	5000.83	15000	11843.53	25000	2.99	2.11
High Blood Pressure						
18-34	9799.81	22000	7359.86	9090.91	2.24	1.24
35-49	18366.59	21250	17218.61	13414.63	1.16	0.78
50-64	32115.15	37623.76	38440.91	50877.19	1.17	1.32
65+	48057.77	59090.91	60185.45	57142.86	1.23	0.95
Shortness of Breath Climbing Stairs						
18-34	—	45098.04	—	58181.82	—	—
35-49	18804.02	44444.44	32506.66	58790.12	2.36	1.75
50-64	33173.62	51960.78	42327.80	73684.21	1.57	1.74
65+	37010.25	54545.45	49553.36	71428.57	1.47	1.44
					P	P
Asthma					<0.0001	<0.0001
Chronic Bronchitis					0.0005	0.0003
High Blood Pressure					0.002	<0.0001
Shortness of Breath Climbing Stairs					<0.0001	0.01
					<0.0001	0.0008
					<0.0001	0.05
					0.002	0.10
					<0.0001	<0.0001
					<0.0001	0.006
					—	—
					<0.0001	<0.0001
					<0.0001	<0.0001
					<0.0001	<0.0001

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Cardiovascular Problems									
18-34	647.54	21568.63	746.23	21818.18	33.31	<0.0001	29.24	<0.0001	
35-49	3273.62	28395.06	1775.02	21951.22	8.67	<0.0001	12.37	<0.0001	
50-64	8524.01	41176.47	7616.51	32456.14	4.83	<0.0001	4.26	<0.0001	
65+	26458.91	40909.09	18080.36	25714.29	1.55	<0.0001	1.42	0.005	
Liver									
18-34	424.68	2325.58	1696.30	6122.45	5.48	0.09	3.61	0.009	
35-49	6240.89	2898.55	2642.29	4000	0.46	0.63	1.51	0.08	
50-64	5221.11	5376.34	3983.46	3921.57	1.03	0.10	0.98	0.15	
65+	3400.71	2439.02	3026.29	—	0.72	0.50	—	—	
Kidney Disease									
18-34	342.84	2000.00	—	3636.36	5.83	0.08	—	—	
35-49	985.12	2500.00	267.94	1234.57	2.59	0.06	4.61	0.10	
50-64	1497.24	6930.69	2369.68	1785.71	4.63	<0.0001	0.75	0.38	
65+	6177.16	4545.45	4083.52	11428.57	0.74	0.39	2.80	0.006	
Thyroid Disease									
18-34	—	—	5761.79	13725.49	—	—	2.38	0.0008	
35-49	3551.87	5555.56	10420.19	20512.82	1.56	0.04	1.97	<0.0001	
50-64	4169.26	7216.49	18424.43	30188.68	1.73	0.005	1.64	<0.0001	
65+	12164.48	11904.76	28167.66	32352.94	0.98	0.11	1.15	0.1	

*Expected rates are from NHANES 2001-2002 using sampling weights to calculate an unbiased estimate of national rates while adjusting for non-response, survey design, and sampling technique while giving an accurate estimate of sampling error.

^bPFAS-exposed population (EP)

^cMI, Stroke, Angina

Emmett at the University of Pennsylvania among community residents served by Little Hocking Water Association (LHWA) showed high levels of serum perfluorooctanoate [40]. Dr. Emmett reported *median* residential blood levels of 327 ng/ml (interquartile range: 187–572) from a random sample of 74 residents served by LHWA. Levels were high for all members of the sampled group including children, adults, and the aged. Occupational exposure was restricted by limiting the study participants to those who had worked one year or less at the nearby chemical plant during the previous 10 years. Even so, the C8 blood levels approached and sometimes overlapped that of reported worker levels. Dr. Emmett contends that the exposure source is the drinking water and not the air. Twenty-seven percent (27%) of our study sample obtained their water from LHWA; our remaining subjects were served by other nearby water districts (see Table 1).

This questionnaire study of the PFOA-exposed population in the Ohio and West Virginia communities near the plant suggests an increased prevalence of several serious health problems. The interpretation of these results depends, however, on several unknown factors. The assumption is that the Parkersburg sample is equivalent to the NHANES sample with regard to all factors that influence the health measures in this study, for example: smoking status, alcohol consumption, and genetic predisposition. If this is the case, the prevalence of these problems is significantly increased compared to National rates.

This study showed an increase in the reported prevalence of respiratory and cardiovascular problems among the PFOA-exposed subjects. These findings are consistent with previous occupational studies of exposed workers [25, 28, 41]. In addition, Gilliland found PFOA alteration in high-density lipoproteins (HDL) in a dose-dependent fashion among PFOA-exposed workers [27]. The alteration was observed only in moderate drinkers, and was considered non-significant after adjusting for alcohol use, age, BMI, and cigarette use. Olsen reported a positive association between measured serum PFOS and total cholesterol although he claims that the finding is spurious [2]. He also reports a positive association between PFOA and triglycerides, but discounts the finding because it is inconsistent with previous research on rats. Costa reports a slight increase of total cholesterol in workers exposed, which also showed an increasing trend associated with the highest blood PFOA levels [25]. These findings are significant because now both PFOS and PFOA have been associated with an increase in cholesterol. Most recently, another report describes an inverse relationship between PFOA and HDL cholesterol coupled with a positive relationship between PFOA and non-HDL cholesterol [26]. These studies suggest a biological mechanism for increased cardiovascular disease in PFAS-exposed populations [25].

The increased prevalence of diabetes in the exposed subjects suggests an interference with insulin function. Studies of monkeys and rats exposed to PFOA revealed increased serum glucose levels [42, 43].

Many studies of PFAS-exposed populations found elevated liver enzymes [6, 20, 24, 27, 44, 45], although most authors discount their findings. The current study was limited in its ability to evaluate the occurrence of liver problems in this population. The reason is two-fold:

1. small sample size; and
2. the question asked of the exposed residents is similar, but not precisely the same as that asked in NHANES.

The question asked in NHANES is "Have you ever had liver problems?" and the question asked in the resident's survey was "Have you ever been told by a doctor that you had a liver disease?" Our question was more restrictive than the comparison question, introducing a definite bias toward the null value making it harder to detect an effect. Nevertheless, we still see large prevalence ratios in the youngest age categories for both males and females demonstrating a potential effect of PFAS exposure on liver function in this population.

The limitations of the data from the residential study include small numbers of cases of rare diseases, lack of a true unexposed comparison group, and potential self-selection bias of study participants. We reduce the limitation of the small sample size by analyzing disease outcomes with relatively large numbers in our data set. National statistics estimated using NHANES data address the need for a comparison group, however, it is not possible to control for potential confounding from variables other than age and gender using these data, and some questions like liver problems or diseases were not strictly comparable to one another. Possible selection bias was examined by evaluating the effect of all cancer prevalence in this group on commonly known and well-understood risk factors: age, gender, smoking status, and educational attainment, which are also reported in a companion paper examining cancer incidence and prevalence in this residential population [30]. The all-cancer rate increased with increasing age, increased smoking, and for those in the lowest category of educational attainment. If one or more of these variables behave outside the norm, we would surmise that this data reflected self-selection bias or reporting bias. However, each category we examined behaved as expected.

Furthermore, the diseases and symptoms identified in this analysis parallel those seen in PFAS-exposed workers. Even if there were selection bias with a disproportionate number of patients with various health problems, there is no reason to assume that only patients with certain types of diseases would be more likely to volunteer for the study. Our study results are consistent with the literature on PFOA and simply support previously reported findings.

The plant workers in this study were presumed to have larger exposures compared to those who did not work at the plant. Unexpectedly, we did not see a difference in health status. Although puzzling, we did not dismiss our findings in residents because of the lack of differential. Subsequently, we learn from Dr. Emmett's timely study that the residential blood levels in LHWA are

remarkably similar to those of workers, 60-80 times higher than expected, given commonly measured background levels of PFAS. Although these blood levels were not measured among our study participants, the measurements did come from residents in the same water district serving 27% of our participants. We feel this adequately explains the lack of difference between worker and non-worker health effects in this study.

Some observers have suggested that plaintiffs in a law suit create an inherent selection bias. The basis for this idea is that the subjects who volunteer to be plaintiffs exaggerate or lie about their symptoms. In addition, a study on the question of selection bias reports that plaintiffs are not more likely to enhance their symptoms than is a normal population [46].

CONCLUSION

In conclusion, living near this Teflon production facility appears to be associated with serious adverse health effects. Additional studies are needed to further delineate the extent and type of health problems associated with PFAS exposure. Such studies are also necessary to define safe levels for these environmental exposures so that governmental agencies can make recommendations and rules to ensure the public's health. The subjects of this study are still living in the same conditions. The water is still contaminated and the plant still continues to dump tons of PFOA every year into the environment. Our findings indicate that chemical companies should reduce their emissions and discharges, and governmental agencies should ensure the public's health by limiting this exposure.

AUTHORS' CONTRIBUTIONS

PA and HT assisted in writing the manuscript, JK completed the analysis, DG reviewed drafts of the manuscript, and JD conceived of the study, supervised all aspects of its implementation, and reviewed drafts of the manuscript.

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