Systemic Inflammation Associated With World Trade Center Dust Exposures and Airway Abnormalities in the Local Community

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Background: Destruction of the World Trade Center (WTC) towers on September 11, 2001, released massive dust, gas, and fumes with environmental exposures for community members. Many community members have lower respiratory symptoms (LRSs) that began after September 11, 2001, and remain persistent. We evaluated whether systemic inflammation measured by C-reactive protein was associated with WTC dust exposures, persistent LRS, and lung function. **Methods:** Community members self-referred for the treatment of symptoms related to September 11, 2001. C-reactive protein and lung function measurements, including spirometry and forced oscillation tests (impulse oscillation system), were included as routine analyses in patients (2007 to 2012). **Results:** Increased C-reactive protein levels were associated with the type of WTC dust exposure, LRS, reduced spirometry, and increased forced oscillation measurements ($n = 724$). **Conclusions:** Ongoing systemic inflammation measured years after the event was associated with WTC dust exposures, persistent LRS, and abnormal lung function in a community cohort. These findings have implications for treatment and surveillance.

T he destruction of the World Trade Center (WTC) towers on September 11, 2001, released massive amounts of dust, gas, and fumes with potential environmental and occupational exposures for thousands of individuals including community members who lived (residents) and worked (local workers) in the area, individuals who cleaned the area (cleanup workers), and for those involved in rescue and recovery (rescue/recovery workers). These groups had risk for acute dust and fume exposure via inhalation of the initial dust clouds created as the WTC buildings collapsed (WTC dust cloud), as well as chronic exposure from resuspended dust from incompletely cleaned indoor and outdoor areas, fumes from fires that burned for 4 months, and exposure from prolonged cleanup activities.[1,2] The dust was composed of highly alkaline materials (pH 11) with pulverized concrete, fiberglass, glass, plastics and contained polycyclic aromatic hydrocarbons, volatile organic compounds, lead, dioxin, and furans.[3,4] We and others have documented lower respiratory symptoms (LRSs) in community members and rescue and recovery workers with WTC dust exposures that began after September 11, 2001, and have persisted (persistent LRS).[5–11]

The Bellevue Hospital Center WTC Environmental Health Center (WTC EHC) is a program that provides medical and mental health treatment for community members and cleanup workers.[10] The program was launched through joint efforts of the local community, organized labor, and the medical community.[12] Similar to rescue and recovery workers, common physical symptoms in community members include persistent LRS that began after September 11, 2001.[9,10]

C-reactive protein (CRP) is a member of the "pentraxin" family of proteins secreted in plasma.[13] Although initially described as an acute-phase protein in response to inflammation, multiple cells, including macrophages, smooth-muscle-like cells, and respiratory epithelial cells, synthesize and secrete CRP, suggesting that it participates in both systemic and local immune responses.[13] Serum CRP levels are considered a biomarker of low-grade, subclinical, and smoldering inflammation and a marker of disease activity, although levels are also increased in the obese and among tobacco users.[13–15] Levels of CRP are a well-described marker of inflammation in cardiovascular disease,[16–18] with levels less than 1 mg/L, 1 to 3 mg/L, and more than 3 mg/L denoting low, intermediate, and higher vascular risk groups.[19] C-reactive protein may also exacerbate tissue damage,[13,20] suggesting an association of CRP with physiologic damage in organs other than the heart. Indeed, increased CRP levels are associated with reduced lung function in nonsmokers[21–24] and CRP levels are also associated with incident risk of chronic obstructive pulmonary disease and chronic obstructive pulmonary disease exacerbations and accelerated decline in spirometry measurements.[25–27]

Few studies have characterized inflammation in response to WTC dust and fumes. Animal models have been complicated by

Learning Objectives

- Become familiar with the persistent lower respiratory symptoms reported in people exposed to the World Trade Center (WTC) site, and with previous data on inflammation associated with this exposure.
- Describe the new findings linking C-reactive protein (CRP) with WTC exposure, lower respiratory symptoms, and lung function in exposed people.
- Discuss the study implications for understanding the pulmonary effects of WTC exposure and for patient treatment and surveillance.

types of WTC dust used, size of particles, and delivery; however, these studies suggest an association between acute inhalation of WTC dust and regulation of genes related to lung inflammation and oxidative stress.[28,29] Elevated levels of systemic inflammatory biomarkers including CRP, measured within the first year after the destruction of the towers, are associated with subsequent decline in lung function in firefighters.[30,31] Increased neutrophils and eosinophils have also been described in patients with WTC-related LRS.[32,33] We documented persistent LRS many years after the event in the WTC EHC population with acute and chronic exposures to WTC dust and fumes.[10] We therefore evaluated whether systemic inflammation measured many years after the event (ongoing inflammation) was associated with persistent LRS, lung function, and type of WTC dust exposure in the WTC EHC.

METHODS

Subjects

Individuals were self-referred to the WTC EHC with health issues related to September 11, 2001, exposures as previously described.[10] The Institutional Review Board of New York University School of Medicine approved the research database (NCT00404898), and only data from patients who provided informed consent were used for analysis. Patients were included for analysis if they had CRP measurements, an acceptable baseline spirometry measurement, and valid forced oscillation as performed by impulse oscillometry system (IOS) measurements.

Procedures and Definitions

Upon enrollment in the WTC EHC, patients responded to a multidimensional interviewer-administered questionnaire that included demographic information, tobacco use, and characterizations of WTC-related exposures.[10] The presence and severity of respiratory symptoms was also obtained by questionnaires. Blood samples were obtained for peripheral CRP measurements in serum samples in newly enrolled patients as well as in those undergoing routine monitoring and treatment. Lower respiratory symptoms of cough, wheezing, chest tightness, and dyspnea were considered WTC-related if they had onset after September 11, 2001 (new onset), and persistent if they were present more than twice each week in the month preceding the interview.[10] Patients who reported more than 5 pack-year history of tobacco use were defined as smokers. Individuals were classified as having WTC dust cloud exposure if they reported being in the initial WTC dust cloud from the collapsing buildings. Patients were characterized as resident, local worker, cleanup worker, rescue/recovery worker, or other on the basis of their initial questionnaire responses about residence and workplace.[10]

Lung Function

All individuals were referred for screening spirometry at the time of enrollment. Spirometry was performed in accordance with American Thoracic Society/European Respiratory Society standards[34] on a Viasys Vmax spirometer (Yorba Linda, CA). Spirometry data were electronically downloaded along with an automated quality assurance code. All studies were performed in the Bellevue Hospital Center Pulmonary Function laboratory. Data for prebronchodilator forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV_1) are presented in liters or as percentages of predicted (percent predicted FVC and percent FEV_1) derived from the Third National Health and Nutrition Examination Survey.[35] Patients were categorized as having a normal, low FVC, obstructed, or low FVC/obstructed spirometry pattern at enrollment on the basis of the lower limits of normal as previously described.[35,36] Forced oscillation testing (FOT) was performed using a Jaeger Impulse Oscillation System in accordance with published recommendations and reported as follows: resistance measured at an oscillation frequency

of 5 Hz (R_5), frequency dependence of resistance calculated as the difference between resistance at 5 and 20 Hz (R_{5-20}), and the reactance area (Ax) defined as the area under the reactance curve from 5 Hz to the resonant frequency. A minimum of three trials, lasting 30 seconds, was performed. Only data from trials with constant tidal volume were analyzed. Because the impulse oscillation system analyzes 150 impulses over a 30-second measurement, coherence more than 0.70[37,38] and reproducibility between trials (variability <10%) were required.[11,39]

CRP Measurements

CRP measurements were obtained during an initial visit, treatment, or monitoring visit for patients with visits between August 2007 and December 2012. C-reactive protein was measured in the Bellevue Hospital Chemistry laboratory using a wide-range CRP assay (Siemen's Diagnostic Center, Tarrytown, NY), which is comparable to high-sensitivity CRP[40] and has a lower limit of detection of 0.12 mg/L. On the basis of cardiovascular risk studies, a value of more than 3 mg/L was considered a "high" value.[19]

Statistical Methods

Serum CRP was analyzed either as a binary variable with two categories defined as "high" (>3 mg/L) or "Low" (≤3 mg/L) on the basis of cardiovascular risk studies, or as a continuous variable using the log transformation. Categorical variables were summarized using counts and proportions, and compared between high and low CRP groups using the chi-square test. Continuous variables were summarized using means and standard deviations (or median and inter-quartile range), and compared between the two groups using the nonparametric Wilcoxon test. Logistic regression was used to quantify the association between high CRP and the covariate of interest while adjusting for age, sex, body mass index (BMI), and smoking status. In subgroup analysis within subjects with normal spirometry pattern, linear regression was conducted to estimate the effects of IOS parameters on the log-transformed CRP levels.

RESULTS

Baseline Characteristics

The final study population consisted of 754 patients who had visits in the WTC EHC between August 2007 and December 2012, with CRP measurements and acceptable lung function studies (Fig. 1). The characteristics of the study population (Table 1) were similar to previous publications from the WTC EHC.[10,41] The study population was diverse with many female (50%, $n = 380$) and Hispanic (41%, $n = 309$) patients and a BMI of 28.4 ± 5.8 kg/m^2 (mean ± standard deviation). Our analysis showed that 66% of our population had a BMI less than 30 kg/m^2, 22 had a BMI between 30 and 35 kg/m^2, and 12% had a BMI more than 35 kg/m^2. Most individuals in the study population were local workers (47%, $n = 353$), with fewer residents and cleanup workers. Many reported being caught
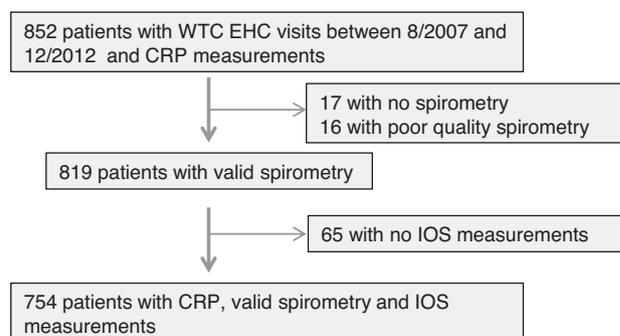

FIGURE 1. Development of study group from WTC EHC.

TABLE 1. Characteristics of the WTC EHC Study Population and Association With CRP Level

	Total $n = 754$	High CRP $n = 261$	Low CRP $n = 493$	P Value
Sex, n (%)				<0.002
Female	380 (50)	153 (59)	227 (46)	
Male	374 (50)	108 (41)	266 (54)	
Age at first visit, yrs, mean (SD)	49.3 (12.0)	51.9 (10.9)	48.0 (12.4)	<0.001
BMI, kg/m^2, mean (SD)	28.4 (5.8)	31.2 (6.6)	26.9 (4.8)	<0.001
Race and ethnicity, n (%)				0.037
Hispanic	309 (41)	114 (44)	195 (39)	
Non-Hispanic white	232 (31)	70 (27)	162 (33)	
Non-Hispanic black	133 (18)	56 (21)	77 (16)	
Asian or other	80 (10)	21 (8)	59 (12)	
Income, n (%)				0.22
≤15,000	335 (46)	127 (50)	208 (43)	
15,001–30,000	141 (19)	43 (17)	98 (20)	
>30,000	259 (35)	85 (33)	174 (36)	
Exposure category, n (%)				0.03
Resident	142 (19)	41 (16)	101 (20)	
Cleanup worker	170 (22)	48 (18)	122 (25)	
Local worker	353 (47)	140 (54)	213 (43)	
Other	89 (12)	32 (12)	57 (12)	
Caught in WTC dust cloud, n (%)				0.009
Yes	366 (49)	143 (56)	223 (46)	
No	377 (51)	112 (44)	265 (54)	
Smoking status, n (%)				<0.001
Yes	288 (39)	125 (49)	163 (34)	
No	453 (61)	131 (51)	322 (66)	

BMI, body mass index; CRP, C-reactive protein; SD, standard deviation; WTC, World Trade Center.

in the WTC dust cloud on September 11, 2001 (49%, $n = 366$). Most of the WTC EHC patients were nonsmokers. The prevalence of new onset and persistent LRS was similar between the study group (86.1%) and that reported for the WTC EHC group as a whole.[10]

CRP Values

The distribution of CRP levels is shown (Fig. 2). CRP levels were not normally distributed (median CRP value of 1.48, range 0.0 to 42.75), and thus were dichotomized as high (>3 mg/L) or low (≤3 mg/L) or log-transformed for analysis of continuous data. More than 35% ($n = 261$) had high CRP values. Patients dichotomized by high and low CRP measurements are shown in Table 1. Consistent with published data for other populations,[13] females, older individuals, and those with an elevated BMI were more likely, than males and younger individuals, to have high CRP (Table 1). Individuals with a history of tobacco use were also more likely to have high CRP compared with those without. Subsequent analyses were adjusted for these differences.

Association of CRP and WTC Dust and Fume Exposures

WTC dust and fume exposures were characterized by presence in the WTC dust cloud on September 11, 2001 (acute exposure), and as exposure category defined as the potential for chronic and acute exposures as a local worker, resident, or cleanup worker[2] (Table 1). Exposure to the WTC dust cloud was associated with an increased prevalence of high CRP (56% vs 46%; $P < 0.009$). Exposure categories were also associated with differences in the prevalence of high CRP, with local workers more likely to have high CRP compared with other groups ($P < 0.03$).
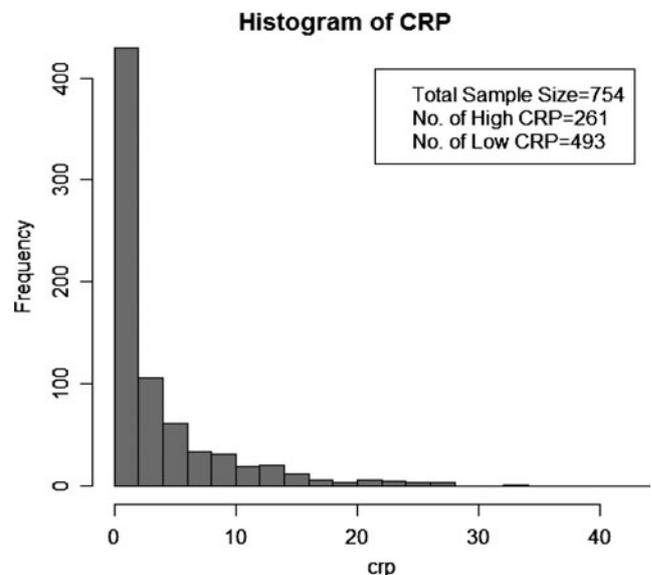
FIGURE 2. Histogram of CRP measurements in study group.

CRP and LRS

As shown in Table 2, high CRP was significantly associated with each individual's persistent LRS. In logistic regression analysis, the association between High CRP and individual LRS remained after adjustment for age, BMI, sex, smoking status, dust cloud, and exposure status (Table 3).

TABLE 2. New Onset and Persistent LRS and CRP Level

Symptom	Total n (%)	High CRP n (%)	Low CRP n (%)	P Value*
Cough				0.0003
No	335 (48)	94 (39)	245 (53)	
Yes	365 (52)	150 (61)	215 (47)	
Wheezing				0.001
No	519 (75)	160 (68)	359 (79)	
Yes	171 (25)	77 (32)	94 (21)	
Dyspnea at rest				0.0008
No	547 (75)	170 (67)	377 (79)	
Yes	182 (25)	82 (33)	100 (21)	
Chest tightness				0.01
No	454 (65)	144 (59)	310 (69)	
Yes	244 (35)	102 (41)	142 (31)	

*Pearson chi-square test.
CRP, C-reactive protein.

TABLE 3. Association of LRS With High CRP Level*

Symptom†	OR	P Value
Cough (n = 704)	1.83	0.001
Wheezing (n = 690)	1.73	0.008
Dyspnea at rest (n = 729)	1.67	0.008
Chest tightness (n = 698)	1.61	0.011

*Adjustment includes sex, Age, log (BMI), smoking status, caught in WTC dust cloud, and exposure category.
†The number varies for each symptom because of missing data.
OR, odds ratio.

CRP and Spirometry

Individuals with high CRP had reduced spirometry measurements compared with those with low CRP, including reduced percent predicted FVC (86.5% vs 96.1% predicted, respectively; $P < 0.001$) and FEV_1 (84.0% vs 93.0% predicted, respectively; $P < 0.001$) (Table 4) but not FEV_1/FVC. When analyzed as a continuous variable, the inverse association remained between CRP and percent predicted FVC ($P < 0.01$) and FEV_1 ($P < 0.01$) remained even after adjustment for covariates (sex, age, BMI, smoking status, WTC dust cloud, and exposure category). When analyzed according to spirometry pattern, high CRP was significantly associated with a low FVC pattern (odds ratio = 2.58; $P < 0.001$) but not with an obstructed pattern (Table 5).

CRP and FOT Measures

Despite the presence of LRS, most patients (70%) had normal spirometry, in accordance with prior publications.[10,41,42] We previously demonstrated elevated IOS measurements in symptomatic WTC-exposed populations with a normal spirometry pattern, suggesting the presence of small airway abnormalities.[11,39,43–46] We therefore examined the association between CRP and forced oscillation using the IOS technique. Values for R_5, $R_{5–20}$, and Ax are shown in Table 6. High CRP was associated with elevated values for all three parameters. To understand the association between CRP and forced oscillation measurements, we further quantified the association between CRP levels as a continuous variable and IOS measurements in the population with a normal spirometry pattern ($n = 528$, Table 7).

CRP levels remained positively associated with R_5, $R_{5–20}$, and Ax in this population, with a normal spirometry pattern even after adjustments for covariates.

DISCUSSION

Systemic inflammation, characterized by CRP, has been suggested in association with environmental exposures and reductions in lung function. CRP levels obtained within the year after WTC dust exposure have also been associated with a subsequent decline in lung function in WTC responders.[31] We now show an association between CRP measurements many years after the event, with a history of WTC dust/fume exposures, persistent LRS, and reductions in lung function in a community population. These data suggest ongoing systemic inflammation in patients in the WTC EHC and suggest an association with symptoms and small airway dysfunction.

Quantitative assessment of serum CRP levels is a sensitive marker of inflammation, and although barely detectable in healthy control subjects, levels are increased in a variety of disease states.[13] The CRP levels in our population were consistent with those described for other populations, and elevated CRP values were associated with obesity and tobacco as previously described.[13] In view of the nonnormality of the distribution of CRP, and the presence of known covariates, we used two approaches to analyze our data: we dichotomized CRP levels into high and low levels consistent with those described for cardiovascular risk[19] and used CRP as a continuous variable with linear regression analysis of log-transformed CRP values with adjustment for covariates. Both of these analyses showed an association between elevated CRP levels and LRSs and reductions in lung function.

Environmental exposures have been associated with systemic inflammation including particulate and gaseous pollutants in Beijing, China, and traffic-related pollution.[47–49] In children, CRP levels are associated with pollutant exposure and wheezing.[50] In our cohort, elevated CRP values were associated with WTC dust/fume exposures. A history of acute WTC dust exposure from exposure to the WTC dust cloud created as the buildings collapsed was associated with high CRP. In addition, risk for high CRP also varied with exposure category, specifically the local worker category; these patients had potential for both chronic and acute exposures.[2] Measurements of CRP in our cohort were obtained many years after September 11, 2001, and thus we cannot determine whether levels were a direct result of WTC dust exposures or a result of a disease process that resulted from these exposures. Because CRP is an acute-phase protein with a short half-life,[51] it is more likely that the increase in CRP levels many years after the event was associated with an ongoing process induced by the initial exposure. We and others have described the relationship between WTC dust exposures and LRS in a community population.[2,9,10] In this study, elevated CRP levels were associated with LRS even after adjusting for covariates including obesity, tobacco use, and WTC dust exposures. This finding further suggests that CRP levels were associated with an ongoing disease process. The association between CRP and LRS is consistent with previous studies of asthma or asthma-like symptoms, and asthma severity.[13,52–54]

An association between CRP and reduced spirometry measurements has been shown in healthy populations and in those with chronic obstructive lung disease.[21,22,55–60] We found an association between a decrease in FEV_1, FVC, but not FEV_1/FVC with increased CRP levels, even after adjusting potential confounders of age, BMI, smoking status, and WTC related exposures. Indeed, the strongest association we noted was between CRP and a reduced FVC. Previous studies in WTC populations show a reduction in lung volumes rather than measures of airflow obstruction,[10,11,21,27,42,61,62] a finding that has led to the suggestion that many of the symptoms in the WTC-exposed patients are due to small airways abnormalities, because severe small airway abnormalities can lead to a reduction in

TABLE 4. Spirometry Measurements in WTC EHC Study Population and CRP Level

Variable	Total Mean (SD)	High CRP Mean (SD)	Low CRP Mean (SD)	P Value*
Spirometry				
Percent predicted FVC	92.8 (17.4)	86.5 (17.1)	96.1 (16.6)	<0.001
Percent predicted FEV_1	89.9 (17.9)	84.0 (18.2)	93.0 (16.9)	<0.001
FEV_1/FVC	76.9 (8.0)	76.7 (9.1)	77.0 (7.3)	0.685

*Wilcoxon test.
CRP, C-reactive protein; FEV_1, forced expiratory volume in 1 second; FVC, forced vital capacity; SD, standard deviation.

TABLE 5. Spirometry Pattern in WTC EHC Study Population and CRP Level

Spirometry Pattern	Total n (%)	High CRP n (%)	Low CRP n (%)	OR*	P Value
Normal	528 (70)	161 (30)	367 (70)	1	
Obstructed†	76 (10)	22 (29)	54 (71)	1.39	0.28
Low FVC‡	124 (16)	65 (52)	59 (48)	2.58	<0.001
Low FVC/obstructed§	26 (3)	13 (50)	13 (50)	1.99	0.13

*Adjusted odds ratio.
†FVC > LLN FVC and FEV_1/FVC < LLN FEV_1/FVC.
‡FVC < LLN FVC and FEV_1/FVC > LLN FEV_1/FVC.
§FVC < LLN FVC and FEV_1/FVC < LLN FEV_1/FVC.
CRP, C-reactive protein; FVC, forced vital capacity; OR, odds ratio.

TABLE 6. IOS Measurements in WTC EHC Study Population and CRP Level

Variable	Total Median (IQR)	High CRP Median (IQR)	Low CRP Median (IQR)	P Value
IOS				
R_5	4.69 (2.26)	5.38 (2.83)	4.49 (2.02)	<0.001
R_{5-20}	0.93 (1.15)	1.30 (1.35)	0.79 (0.98)	<0.001
A_X	6.26 (10.12)	9.65 (13.18)	4.75 (6.96)	<0.001

CRP, C-reactive protein; IOS, impulse oscillometry system; IQR, inter-quartile range.

vital capacity rather than abnormal airflow measurements.[44–46] This suggestion has been supported by elevations in forced oscillation measurements in WTC populations even in the setting of normal spirometry.[63–65] Small airway injury in WTC dust-exposed subjects with reduced vital capacity has also been demonstrated by pathologic studies[66] and imaging studies.[67] We now also show an association between CRP and FVC and CRP and increased FOT measurements even in the setting of normal spirometry. Taken together, these findings suggest the association of ongoing inflammation with small airways abnormalities.

Longitudinal analyses from studies of young adults suggest that systemic inflammation predates the development of chronic lung disease and the decline in lung function.[21] This finding is consistent with a study of firefighters with WTC dust exposure, in which measurements of CRP predicted the decline in lung function.[31] Nevertheless, in some studies, the decline in FEV_1 was itself a strong and significant predictor of CRP in young adults,[21,55,56] raising the possibility that systemic inflammation may be a consequence of a decline in lung function rather than the cause of the decline.[68] Our data support an association between systemic inflammation and reduced lung function, but because our data were obtained at one point in time we cannot determine cause and effect.

TABLE 7. Linear Regression Analysis of CRP and IOS Measurements in Patients with a Normal Spirometry Pattern (n = 528)*

IOS Parameter	Slope	P Value
R_5	0.62	0.002
R_{5-20}	0.54	0.003
A_X	0.27	<0.001

*Adjustment includes sex, age, log (BMI), smoking status, WTC dust cloud, and exposure category.

There are several potential limitations to this study. The use of statins has been shown to reduce the association between CRP and pollutants.[69,70] We do not have information on the use of statins in our population and cannot account for any possible effect of these or other medications. We used serum measurements of CRP using a wide-range CRP assay rather than the high-sensitivity method usually reported. Measurements using the wide-range CRP assay have been reported to correlate with values obtained with a high-sensitivity method, and levels are considered interchangeable.[40] Our

measurements of CRP were not always simultaneous with our lung function measurements; however, the association between CRP and spirometry and FOT measurements remained even when we analyzed a subgroup ($n = 544$) of patients with measurements within 6 months of each other (data in Appendixes A1 and A2).

In summary, our data suggest ongoing systemic inflammation in a community population seeking treatment for symptoms that occurred after exposures to WTC dust and fumes. Systemic inflammation was noted many years after WTC exposures and was associated with different types of WTC dust exposures. Inflammation was also associated with LRSs as well as reduced spirometry measurements. Importantly, elevated CRP levels were also associated with isolated elevated IOS measurements, suggesting an association with small airway abnormalities. The finding of ongoing systemic inflammation has implications for future surveillance of this population. The association of systemic inflammation with a reduction in lung function, particularly small airway abnormalities, is important for future treatment in this population.

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APPENDIX

TABLE A1. Spirometry Pattern in WTC EHC Study Population and CRP Level in Patients With Values Within 6 Months of Each Other (*n* = 544)

Spirometry Pattern	Total *n* = 544	High CRP *n* = 171	Low CRP *n* = 373	OR[a]	*P* Value*
Normal	413 (76)	117 (68)	296 (79)	1	
Obstructed[b]	56 (10)	18 (11)	38 (10)	1.197878	0.66
Low FVC[c]	55 (10)	25 (15)	30 (8)	2.104593	0.015
Low FVC and Obstructed[d]	20 (4)	11 (6)	9 (3)	3.82705	0.021

*P values are for chi-square tests or Fisher tests, compared with "normal."
[a]Unadjusted odds ratio.
[b]FVC > LLN FVC and FEV_1/FVC < LLN FEV_1/FVC.
[c]FVC < LLN FVC and FEV_1/FVC > LLN FEV_1/FVC.
[d]FVC < LLN FVC and FEV_1/FVC < LLN FEV_1/FVC.
CRP, C-reactive protein; FVC, forced vital capacity; OR, odds ratio.

TABLE A2. Linear Regression Analysis of CRP and IOS Measurements in Patients With a Normal Spirometry Pattern (*n* = 413)*

IOS Parameter	Slope	*P* Value
R_5	0.029	0.01
R_{5-20}	0.015	0.02
Ax	0.107	0.001

*Adjustment includes sex, age, log (BMI), smoking status, WTC dust cloud, and exposure category.
IOS, impulse oscillometry system.