Objective: Comparing pulmonary function since the 2001 World Trade Center disaster, with preexposure data, in a New York City Police Department Emergency Responder cohort, without history of repetitive respiratory exposures. Methods: A total of 206 New York City Police Department Emergency Services Unit members reported arrival time, exposure duration, smoking history, respirator mask usage, and respiratory symptoms, and underwent clinical evaluation and follow-up spirometry, in 2002 and 2007. Results: A mean decline in forced vital capacity of 190 mL (3.7%) was observed 1-year postexposure in 2002, and 330 mL (6.4%) in 2007, compared with baseline data. Forced expiratory volume in the first second was not significantly changed in 2002 but declined 160 mL (3.9%) after 5 further years of follow-up. Conclusions: Abnormal spirometry was observed in 5.3% of subjects, particularly individuals experiencing higher exposure intensity, duration, or respiratory symptoms. The small number of smokers and subjects failing to wear protective respiratory masks showed greater declines.

Individuals who were present in the vicinity of the World Trade Center (WTC) collapse on September 11, 2001, were exposed to debris, dust and, more than 400 identified airborne pollutants. Early and persistent reductions in pulmonary function, compared with baseline measurements, have been reported in a variety of responder groups. Members of the New York City Police Department (NYPD) Emergency Services Unit (ESU)—a cohort without chronic pulmonary exposures—were deployed at the WTC site and exposed, along with other first responders, local residents, employees, and visitors, from the first moments of the disaster, through the protracted course of rescue and cleanup operations, nearly 1 year later.

Pulmonary function parameters (forced vital capacity [FVC], forced expiratory volume in the first second [FEV1]) were compared with baseline preexposure data, in 206 emergency service unit members of the NYPD, at 1 year following the WTC disaster, and after 5 more years of follow-up, examining the observed data for possible associations with (1) varying exposure intensities, (2) protective respiratory mask use, (3) respiratory symptoms, and (4) smoking history (Table 1).
TABLE 1. Mean Spirometry Results: Emergency Services Unit Cohort

<table>
<thead>
<tr>
<th>Year</th>
<th>n</th>
<th>L</th>
<th>P</th>
<th>% of P</th>
<th>SD</th>
<th>L</th>
<th>P</th>
<th>% of P</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>1999</td>
<td>206</td>
<td>4.05</td>
<td>94.75</td>
<td></td>
<td>±12.76</td>
<td>5.16</td>
<td>99.40</td>
<td></td>
<td>±11.88</td>
</tr>
<tr>
<td>2002</td>
<td>204</td>
<td>3.99</td>
<td>NS</td>
<td>96.05</td>
<td>±11.25</td>
<td>4.97</td>
<td>.010</td>
<td>97.05</td>
<td>0.003</td>
</tr>
<tr>
<td>2007</td>
<td>139</td>
<td>3.89</td>
<td>0.02</td>
<td>96.02</td>
<td>±13.72</td>
<td>4.83 &lt;.001</td>
<td>95.94</td>
<td>0.004</td>
<td></td>
</tr>
</tbody>
</table>

FEV1, forced expiratory volume in the first second; FVC, forced vital capacity; NS, not significant.

RESULTS

General
One year following exposure to the 2001 WTC disaster, 206 members of the NYPD ESU participated in our study, 34% of whom experienced the debris cloud of the WTC collapse, whereas 66% arrived at the site at a later time, 46% were exposed for greater than 2000 hours, 41% were exposed between 1000 and 2000 hours, and 14% were exposed for less than 1000 hours. A protective respiratory mask of some type was worn by 94% of participants for most of their exposure time.

When questioned regarding the presence of upper or lower respiratory symptoms in 2002, 81% responded affirmatively, whereas...
TABLE 2. Mean Spirometry Results Respiratory Symptoms

<table>
<thead>
<tr>
<th>Year</th>
<th>n</th>
<th>FVC (L) (%) nl</th>
<th>SD</th>
<th>FEV1 (L) (%) nl</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptomatic</td>
<td>1999</td>
<td>113</td>
<td>5.06 (99.33)</td>
<td>± 11.89</td>
<td>3.98 (93.33)</td>
</tr>
<tr>
<td></td>
<td>2002</td>
<td>113</td>
<td>4.90 (96.27)</td>
<td>± 11.55</td>
<td>3.90 (94.10)</td>
</tr>
<tr>
<td></td>
<td>2007</td>
<td>79</td>
<td>4.67 (95.03)</td>
<td>± 13.68</td>
<td>3.72 (94.33)</td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>1999</td>
<td>91</td>
<td>5.20 (99.58)</td>
<td>± 11.79</td>
<td>4.16 (96.46)</td>
</tr>
<tr>
<td></td>
<td>2002</td>
<td>89</td>
<td>5.08 (96.90)</td>
<td>± 9.96</td>
<td>4.13 (97.67)</td>
</tr>
<tr>
<td></td>
<td>2007</td>
<td>59</td>
<td>5.05 (96.08)</td>
<td>± 11.21</td>
<td>4.13 (97.42)</td>
</tr>
</tbody>
</table>

FEV1, forced expiratory volume in the first second; FVC, forced vital capacity.

Spirometry

Pulmonary function data prior to 9/11/01 and follow-up testing in 2002 and 2007 are summarized in Table 1. The baseline FVC of 206 subjects tested within 36 months prior to 9/11/01 averaged 5.16 L (95% CI, 5.05 to 5.26 L). FVC measurements decreased by 190 mL (3.68%) in 2002 and by 330 mL (6.4%) in 2007 (95% CI, 4.86 to 5.08 and 4.69 to 4.97; \( P < 0.01 \)), respectively.

FEV1 averaged 4.05 L pre-9/11/01, and 3.99 L in 2002, and fell 160 mL (3.95%) in 2007 (95% CI, 3.91 to 4.07 and 3.78 to 4.00) \( P = 0.02 \) (Figs. 1 and 2) (Table 1).

Pulmonary studies remained unchanged in functional classification from preexposure measurements (>80% predicted) in (95%) of the 206 subjects. In 5.3% of the subjects, pulmonary function parameters deteriorated from “Normal” to “Mild” reduction (60% to 79% of predicted). No subjects in the study population decreased to a “Moderate” (40% to 59% predicted) or “Severe” reduction in classification of pulmonary function (<40% predicted).
Respiratory Symptoms

Significant pulmonary function declines were seen in individuals who complained of respiratory symptoms (upper or lower) at 1-year postexposure in 2002 and in 2007, with FVC declining 240 mL (4.67%) in 2002 (95% CI, 4.76 to 5.05 L) \( P = 0.03 \) and 470 mL (9.14%) in 2007 (95% CI, 4.49 to 4.85 L) \( P = 0.01 \). In asymptomatic individuals, FVC declined 120 mL (2.31%) in 2002 (95% CI, 4.92 to 5.23 L) \( P = 0.23 \) and 150 mL (2.88%) in 2007 (95% CI, 4.84 to 5.26 L) \( P = 0.17 \). (Fig. 3)

In symptomatic individuals, FEV1 declined 80 mL (2.01%) in 2002 (95% CI, 3.79 to 4.01 L) \( P = 0.16 \) and remained unchanged in 2007 (95% CI, 3.97 to 4.29 L) \( P = 0.66 \). (Fig. 4) (Table 2)

Smoking History

Individuals with a positive smoking history showed reductions in FVC of 250 mL (4.84%) at 1-year postexposure in 2002 (95% CI, 4.63 to 5.19 L) \( P = 0.05 \) and 450 mL (8.72%) in 2007 (95% CI, 4.44 to 4.98 L) \( P = 0.02 \). In nonsmokers, FVC fell 160 mL (3.11%) in 2002 (95% CI, 4.87 to 5.10 L) \( P = 0.09 \) and 280 mL (5.4%) in 2007 (95% CI, 4.70 to 5.02 L) \( P < 0.01 \). (Fig. 5)

FEV1 in the smoking subgroup was less affected over the study period, than FVC, declining 70 mL (1.74%) in 2002 (95% CI, 3.74 to 4.16 L) \( P = 0.16 \) and 170 mL (4.23%) in 2007 (95% CI,
3.62 to 4.07 L) \( P = 0.07 \). Similarly, in non-smokers, FEV1 fell 70 mL (1.72%) in 2002 (95% CI, 3.92 to 4.09 L) \( P = 0.74 \) and 170 mL (4.18%) in 2007 (95% CI, 3.78 to 4.04 L) \( P = 0.05 \) (Fig. 6) (Table 3).

**Exposure Intensity/Duration**

Declines in pulmonary function were also seen in responders who had greater exposure intensity, based on duration. In responders experiencing the highest intensity exposure (duration >2000 hours) FVC declined 170 mL (3.36%) at 1-year follow-up in 2002 (95% CI, 4.74 to 5.04 L) \( P = 0.016 \) and 340 mL (6.72%) at 5-year follow-up in 2007 (95% CI, 4.51 to 4.92 L) \( P < 0.001 \), whereas FEV1 declined 30 mL (0.75%) in 2002 (95% CI, 3.84 to 4.07 L) \( P = 0.32 \), and 120 mL (3.02%) in 2007 (95% CI, 3.7 to 4.03 L) \( P = 0.035 \). In the individuals with moderate exposure (1000 to 2000 hours), FVC fell 200 mL (3.81%) in the first year of study in 2002 (95% CI, 4.87 to 5.24 L) \( P = 0.81 \) and declined 270 mL (5.14%) in 2007 (95% CI, 4.276 to 5.19 L) \( P = 0.33 \), whereas FEV1 fell 80 mL (1.94%) in 2002 (95% CI, 3.9 to 4.17 L) \( P = 0.83 \) and 190 mL (4.61%) in 2007 (95% CI, 3.77 to 4.09 L) \( P = 0.28 \). In those with the lowest exposure duration (<1000 hours), FVC declined 120 mL (2.42%)
TABLE 5. Mean Spirometry Results: Exposure Intensity/Arrival Time

<table>
<thead>
<tr>
<th></th>
<th>Early arrival</th>
<th>Later arrival</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>FVC(L)</td>
</tr>
<tr>
<td>1999</td>
<td>68</td>
<td>5.06</td>
</tr>
<tr>
<td>2002</td>
<td>67</td>
<td>4.82</td>
</tr>
<tr>
<td>2007</td>
<td>66</td>
<td>3.94</td>
</tr>
<tr>
<td>1999</td>
<td>130</td>
<td>5.21</td>
</tr>
<tr>
<td>2002</td>
<td>130</td>
<td>5.04</td>
</tr>
<tr>
<td>2007</td>
<td>73</td>
<td>4.98</td>
</tr>
</tbody>
</table>

FEV1, forced expiratory volume in the first second; FVC, forced vital capacity.

in 2002 (95% CI, 4.5 to 5.12 L) \( P < 0.01 \) and 160 mL (3.23%) in 2007 (95% CI 4.3 to 5.28 L) \( P < 0.04 \), whereas FEV1 remained unchanged in 2002 with \( P = 0.09 \) and fell 100 mL (2.51%) in 2007 (95% CI, 3.49 to 4.27 L) \( P = 0.25 \) (Figs. 7 and 8) (Table 4).

### Exposure Intensity/Arrival Time

Responders who were exposed to the debris cloud at ground zero at the time of the WTC collapse or its immediate aftermath showed declines in FVC of 240 mL (4.74%) in 2002 (95% CI, 4.68 to 4.97 L) \( P = 0.001 \), and 410 mL (8.1%) in 2007 (95% CI, 4.46 to 4.85 L) \( P < 0.001 \). FEV1 fell 60 mL (1.5%) in 2002 (95% CI, 3.83 to 4.07 L) \( P = 0.02 \) and 200 mL (5%) in 2007 (95% CI, 3.65 to 3.96 L) \( P = 0.001 \). In individuals who were not present at the time of the WTC collapse and the resulting debris cloud, FVC fell 170 mL (3.26%) in 2002 (95% CI, 4.90 to 5.12 L) and 230 mL (4.41%) in 2007 (95% CI, 4.8 to 5.18 L) \( P = 0.001 \). FEV1 declined 50 mL (1.23%) in 2002 (95% CI, 3.9 to 4.13 L) \( P = 0.13 \) and 100 mL (2.46%) in 2007 (95% CI, 3.82 to 4.13 L) \( P = 0.001 \) (Figs. 9 and 10) (Table 5).

### Protective Respiratory Mask Usage

Most responders reported using fit-tested 3M N95 Respirator Masks (3M Corporation, St. Paul, MN), whereas few reported using standard construction dust masks. Individuals who made use of protective respiratory masks showed FVC reductions of 180 mL (3.48%) in 2002 (95% CI, 4.86 to 5.11 L) \( P = 0.001 \) and 300 mL (5.8%) in 2007 (95% CI, 4.7 to 5.04 L) \( P = 0.005 \). FEV1 fell 80 mL (1.97%) in 2002 (95% CI, 3.9 to 4.09 L) \( P = 0.45 \) and 150 mL (3.69%) in 2007 (95% CI, 3.78 to 4.05 L) \( P = 0.04 \). In individuals who did not use protective respiratory masks, FVC fell
FIGURE 11.

Mean Spirometry Results
Problems

FIGURE 12.

TABLE 6. Mean Spirometry Results Protective Mask Usage

<table>
<thead>
<tr>
<th>Year</th>
<th>n</th>
<th>FVC(L) (%)</th>
<th>SD</th>
<th>FEV1(L) (%)</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Masks</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1999</td>
<td>150</td>
<td>5.17 (99.55)</td>
<td>± 12.09</td>
<td>4.07 (94.99)</td>
<td>± 12.72</td>
</tr>
<tr>
<td>2002</td>
<td>148</td>
<td>4.99 (96.77)</td>
<td>± 11.32</td>
<td>3.99 (95.52)</td>
<td>± 11.35</td>
</tr>
<tr>
<td>2007</td>
<td>104</td>
<td>4.87 (96.08)</td>
<td>± 14.01</td>
<td>3.92 (96.06)</td>
<td>± 14.24</td>
</tr>
<tr>
<td>No masks</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1999</td>
<td>10</td>
<td>4.68 (95.50)</td>
<td>± 13.37</td>
<td>3.78 (93.90)</td>
<td>± 11.27</td>
</tr>
<tr>
<td>2002</td>
<td>10</td>
<td>4.44 (92.70)</td>
<td>± 16.24</td>
<td>3.67 (93.20)</td>
<td>± 9.45</td>
</tr>
<tr>
<td>2007</td>
<td>7</td>
<td>4.02 (81.86)</td>
<td>± 12.75</td>
<td>3.36 (83.57)</td>
<td>± 10.18</td>
</tr>
</tbody>
</table>

FEV1, forced expiratory volume in the first second; FVC, forced vital capacity.

240 mL (5.13%) in 2002 (95% CI, 4.04 to 4.83 L) P = 0.026 and 660 mL (14.1%) in 2007 (95% CI, 3.48 to 4.57 L) P = 0.068. FEV1 in this group fell 110 mL (2.91%) in 2002 (95% CI, 3.38 to 3.96 L) P = 0.38 and 420 mL (11.11%) in 2007 (95% CI, 2.89 to 3.8 L) P = 0.21. The number of subjects in these subgroups, however, was very small. (Figs. 11 and 12) (Table 6).

DISCUSSION

The expected decline in FEV1 in smokers has been estimated at approximately 0.5% per year (20 to 30 mL).4 Previous studies of rescue workers at the WTC sites have demonstrated reductions in lung volumes which have exceeded the decreases expected because of age-related changes alone.5 In this study, FVC declined 190 mL (3.7%) in 2002, whereas FEV1 appeared to be unaffected at 1 year following the WTC exposure. Spirometry measurements continued to decline in 2007, with FVC falling 330 mL (6.4%) and FEV1 160 mL (4.0%), about twice the expected decline for an average smoker for the study period.

Occupational studies of first responders with histories of chronic and repetitive respiratory exposures, such as firefighters, have reported an excessive decline in function in the first year following exposure, with subsequent PFT recovery.6-8 These studies also report differences in pulmonary function declines between individuals, based on exposure intensities such as arrival time at ground zero, and other variables.9,10

Pulmonary functional classifications remained unchanged over the study period in 95% of our subjects, whereas 5.3% of
our cohort deteriorated from “normal” to “mild airway disease,” and none progressed to “moderate” or “severe disease” classifications, by ATS criteria.

In our study, significant differences were seen in pulmonary function in individuals who (1) experienced the debris cloud of the WTC collapse, (2) were exposed for longer periods of time, or (3) developed respiratory symptoms following exposure. Although some of the findings are consistent with observations noted in other responder groups studied,1,6,7,10–13 the type of pulmonary dysfunction observed in our cohort tended to be of a more restrictive or mixed pattern than obstructive in nature.

In our cohort, the FVC—a measure that is arguably more commonly affected in restrictive defects—was more prominently impacted by the WTC exposure than FEV1, more often identified as a measure of obstructive processes. This is not surprising, given the complex mix of dust and debris particles inhaled at the WTC collapse,1,2,14 which may have elicited a response similar to the pulmonary effects observed in some historical environmental exposures.15–17 The changes we observed in our cohort were particularly true for smokers, who showed continued FVC declines in the follow-up study period, whereas FEV1 declines between smokers and nonsmokers remained flat. These observations may point to a mixed defect developing in WTC-exposed smokers, with early restrictive changes, possibly followed by later appearing obstructive changes.14,18–20

In addition, the similarity in degree of pulmonary declines between our cohort and others studied12,13,21 seems to further dispel any postulated distinctions in the severity of pulmonary effects expected between responders having histories of chronic pulmonary exposures and those who do not. This may bode well for the ESU cohort, and possibly thousands of other exposed non-ESU NYPD officers, who share similar training, physical attributes (mean height, weight, body mass index), as well as postexposure spirometry, and have a lower mean age and lower smoking rates (NYPD Personnel Bureau data). Nevertheless, exposure to the unprecedented environmental event that the WTC disaster represents makes it impossible to confirm data drawn upon historical events, or the relatively early reported findings, for predicting the nature of possible long-term sequelae, or for further prognostication, and may, in fact, require a new paradigm.14

LIMITATIONS

Cohort subjects were members of the Emergency Services Unit, a self-selected group of officers who undergo specialized training for specific technical and physical capabilities required in rescue and recovery (ie, confined space operations, rappelling), who traditionally underwent symptoms and tend to downplay physical complaints and may have exhibited a well-observed reticence on the part of law enforcement populations, toward disclosing symptoms, for fear of stigmatization or job loss (healthy worker effect). Nevertheless, there is no evidence that the nature of this physical training has any significant impact upon pulmonary function parameters.22

The NYS WTC Disability Law was passed by the NYS Legislature in 2005,23,24 creating a presumption of causal relationship for any disabilities arising from exposure to the WTC disaster, and awarding those entitled with higher, tax-free pensions than other individuals for any disabilities arising from exposure to the WTC disaster, and after more than 5 years of follow-up, in 2007. Significant declines in pulmonary function were observed in 5.3% of subjects, compared with their baseline preexposure measurements.

The reduced pulmonary function in this cohort was most significantly associated with (1) the presence of respiratory symptoms and (2) a history of high-intensity exposure, as defined by experiencing the debris cloud of the WTC towers collapse, or by the extended number of hours working at the WTC site. More severe pulmonary deficits were also observed in individuals who failed to don protective respiratory masks, and among smokers, as has been reported in other studies.6,7,9 Nevertheless, because of the small number of subjects in some of our subgroups (nonsmokers and non–mask wearers), wider conclusions cannot be drawn. Our findings do, however, mirror reports from other responder cohorts—both with and without histories of chronic, recurring pulmonary exposures—suggesting a similar pulmonary response across responder groups to the exposure of the WTC disaster debris cloud.11,12

The relatively mild pulmonary effects seen in most responder cohorts studied6,8,11–13 the absence of pulmonary disabilities resulting from the WTC exposure in our cohort, and the dearth of such disabilities in thousands of other exposed NYPD responders, thus far (NYPD Personnel Bureau data), may provide encouraging expectations regarding the long-term prognosis for these groups. Nevertheless, despite objectively mild spirometry declines,12,13,26 and the fact that our cohort has fared well clinically over the study period, persistent or worsening pulmonary function seen in a significant number of subjects remains a continued concern and may be a harbinger of further difficulties arising in the future.14,19 Some of the subgroups in this cohort (smokers, symptomatic, and highly exposed) merit especially close observation, to monitor whether their pulmonary functions stabilize, resolve, or worsen over time, as the greater pulmonary declines observed in this study, may signal increased risk.

Moreover, the observations made in this study underscore the need for a comprehensive approach to urban disaster planning and preparedness.20 Particular attention is necessary for establishing guidelines for the use of effective personal protective gear, and limitations of tours of duty for workers engaged in rescue and recovery in environments where the potential exists for toxic exposure. Support for monitoring programs and long-term follow-up for exposed individuals, and for innovative approaches to more effective smoking cessation programs, may also be crucial. Attention to these issues will help guide clinicians and urban planners as to the direction and focus of existing monitoring programs and will be critical in helping to protect first responders and ordinary citizens caught up in the midst of natural or man-made urban calamities.
REFERENCES


