# Bronchial hyperreactivity and other inhalation lung injuries in rescue/recovery workers after the World Trade Center collapse

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***Background:*** **The collapse of the World Trade Center (WTC) on September 11, 2001 created a large-scale disaster site in a dense urban environment. In the days and months thereafter, thousands of rescue/recovery workers, volunteers, and residents were exposed to a complex mixture of airborne pollutants.**

***Methods:*** **We review current knowledge of aerodigestive inhalation lung injuries resulting from this complex exposure and present new data on the persistence of nonspecific bronchial hyperreactivity (methacholine PC20 ≤8 mg/mL) in a representative sample of 179 Fire Department of the City of New York (FDNY) rescue workers stratified by exposure intensity (according to arrival time) who underwent challenge testing at 1, 3, 6, and 12 months post-collapse.**

***Results:*** **Aerodigestive tract inflammatory injuries, such as declines in pulmonary function, reactive airways dysfunction syndrome (RADS), asthma, reactive upper airways dysfunction syndrome (RUDS), gastroesophageal reflux disease (GERD), and rare cases of inflammatory pulmonary parenchymal diseases, have been documented in WTC rescue/recovery workers and volunteers. In FDNY rescue workers, we found persistent hyperreactivity associated with exposure intensity, independent of airflow obstruction. One year post-collapse, 23% of highly exposed subjects were hyperreactive as compared with only 11% of moderately exposed and 4% of controls. At 1 yr, 16% met the criteria for RADS.**

***Conclusions:*** **While it is too early to ascertain all of the long-term effects of WTC exposures, continued medical monitoring and treatment is needed to help those exposed and to improve our prevention, diagnosis, and treatment protocols for future disasters. (Crit Care Med 2005; 33[Suppl.]:S102–S106)**

**KEY WORDS: World Trade Center; inhalation lung injury; nonspecific bronchial hyperreactivity; airborne particulates; firefighters; rescue workers**

On September 11, 2001, the attack on and subsequent collapse of the World Trade Center (WTC) released a complex mixture of airborne pollutants (particulates and chemicals of combustion/pyrolysis). Rescue/recovery workers, as well as volunteers and residents, were exposed to airborne pollutants, leading to aerodigestive inhalation injuries. This review presents new data on the persistence of bronchial hyperreactivity in firefighters and summarizes our current knowledge of aerodigestive inhalation lung injuries in WTC rescue/recovery workers.

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Over 400 different substances have been identified in airborne and settled samples of WTC dust (1, 2). It has been estimated that approximately 70% of the towers' structural components were pulverized during the collapse, producing small- (2.5–10 μm) and large- (>10 μm) sized respirable particulates (1–9). Along with the collapse and pulverization of the buildings, structural fires persisted until mid-December, releasing products of combustion/pyrolysis (4, 6, 10). The necessary use of gasoline- and diesel-powered heavy machinery to remove approximately 1,500 million kilograms of rubble reaerosolized dust and produced additional combustion products (4, 8, 9).

Respiratory exposure occurred for several reasons. Effective respiratory protection, an elastomeric P-100 half-face respirator, was for the most part unavailable for the first days to week postcollapse (11, 12), and adherence with proper use guidelines was suboptimal in the weeks and months that followed (11, 12). Although the upper respiratory tract normally filters out large particulate matter, the nasal filter in most WTC rescue/recovery workers was inadequate because the increased minute ventilation required for rescue/recovery activities strongly favors mouth breathing, and the large concentration of aerosolized dust along with its strong alkalinity overwhelms and impairs nasal clearance mechanisms (13). Ten months after the WTC collapse, induced sputum from firefighters of the Fire Department of the City of New York (FDNY) demonstrated particle size deposition patterns characteristic of WTC dust (14), a sign that nasal clearance was indeed overwhelmed in rescue/recovery workers with WTC dust exposure at Ground Zero. These investigators also found significantly more large particulates (>5 μm) in lower airway secretions of FDNY firefighters compared with Tel Aviv firefighters; because particle alkalinity increased with size (2, 15), this finding may explain the pronounced lower respiratory tract symptomatology, hyperreactivity, and dysfunction observed in WTC rescue/recovery workers.

## World Trade Center-Related Respiratory Health Concerns

At present, respiratory health concerns after exposure to WTC dust can be grouped into two major categories: current inflammation-related aerodigestive tract syndromes and late emerging diseases. Supporting toxicologic findings in animals and humans are now beginning to emerge. Before the WTC collapse, freshly fractured cement and gypsum-containing building wallboard, other components of concrete such as quartz, calcite, and halite (1–5), and highly concentrated synthetic vitreous fibers such as glass and mineral wool had already been implicated in irritative and inflammatory mucosal syndromes (16, 17). After the WTC attack, Gavett and coworkers compared inflammatory and physiological effects of WTC-derived small particulate matter (<2.5 μm) with nontoxic and toxic reference materials in mice (18). Although WTC small particulate matter (<2.5 μm) caused only mild pulmonary inflammation compared with the reference materials, a marked increase in airway reactivity to inhaled methacholine was observed. Corresponding findings in human WTC rescue workers have shown rising sputum neutrophil and eosinophil counts with increasing work duration at the WTC site, higher sputum MMP-9 levels in FDNY firefighters compared with Tel Aviv firefighters (14), and persistent nonspecific airway hyperreactivity in highly exposed FDNY rescue workers (19).

## Inhalation Aerodigestive Tract Injuries in World Trade Center Rescue/Recovery Workers

To date, clinical investigations have relied on self-reported arrival time and/or cumulative work activity at the WTC Ground Zero site. Correlations between measurable pulmonary function abnormalities and increasing WTC exposure have been demonstrated in several groups of rescue/recovery workers (Table 1). The vast majority of WTC-related respiratory syndromes in rescue/recovery workers can be attributed to aerodigestive tract inhalation injuries (Table 1), and studies appear to indicate an exposure intensity effect with inhalation injuries of greater severity in those who arrived in the first 48 hrs (11, 19, 20) or who had substantial cumulative exposure (21). Some investigators have shown significant associations between objective pulmonary function abnormalities and dust exposure intensity and/or new respiratory symptoms (11, 20, 19, 22, 23). However, associations between respiratory symptoms and dust exposure have been present even in studies in which associations between respiratory symptoms and pulmonary function abnormalities did not reach statistical significance (21–23). Although WTC rescue/recovery workers clearly experienced higher overall exposures than lower Manhattan residents, similar respiratory consequences have been noted in studies on community residents and office workers (24–27).

*Respiratory Symptoms and Pulmonary Function Abnormalities.* High rates of upper and lower respiratory irritant symptoms during the subacute postexposure time period have been described in four WTC rescue/recovery worker groups. In 362 previously healthy, exposure-stratified NYC firefighters, 78% had new upper and 73% had new lower respiratory symptoms 4 wks postcollapse (11). In 240 previously healthy NYC Emergency Service Unit (ESU) police officers, 77% had upper and/or lower respiratory symptoms during the first 5 mos postcollapse (3, 22). In 96 ironworkers, 77% had upper and/or lower respiratory symptoms 6 mos postcollapse (23). In 1,138 rescue/recovery workers and volunteers (various trades), 58% developed a new or worsening cough while working at the WTC site (21). Respiratory symptoms have been linked to WTC dust exposure intensity (mostly estimated by initial arrival time) in many of WTC rescue recovery groups (11, 19, 22, 23). Furthermore, respiratory symptom prevalence has been correlated with objective physiological pulmonary abnormalities with: 1) greater median declines in forced expiratory volume in 1 sec ($FEV_1$) and forced vital capacity (FVC) (last spirometry precollapse compared with first spirometry postcollapse) found among symptomatic as opposed to asymptomatic firefighters (11); 2) abnormal postcollapse spirometry ($FEV_1$ and/or FVC and/or $FEV_1$/FVC below lower limits of normal and/or bronchodilator response) occurring more often in NYC ESU police officers with dyspnea, chest discomfort, or wheezing (22); and abnormal spirometry ($FEV_1$/FVC or FVC less than lower limits of normal) in 33% of 1,138 WTC rescue/recovery workers and volunteers (various trades) with 11% of 1,138 having a bronchodilator response (21). However, it does not appear that abnormal spirometry correlated with pulmonary symptoms. Interestingly, Skloot and coworkers found large airway dysfunction on forced oscillation, significantly more common in those who had not worn a respirator with a canister in a group of 96 ironworkers (23). No such associations between respiratory protection and symptoms and/or pulmonary physiological abnormalities have been reported in FDNY rescue workers presumably because the FDNY group arrived

**Table 1.** Published reports of inhalation injuries by specific group of rescue/recovery workers

| Rescue/Recovery Worker Group | Aerodigestive Inhalation Injuries (Symptoms and Physiology) | Reference Nos. |
|---|---|---|
| FDNY firefighters | WTC cough, RUDS, GERD, and abnormal PFTS | 11, 20 |
| FDNY firefighters | Cough, wheeze, dyspnea, and reductions in PFTS compared with pre-WTC baselines | 11, 19, 20 |
| FDNY firefighters | NSBHR and RADS in WTC cough patients | 20 |
| FDNY firefighters | Persistent NSBHR and RADS in a stratified sample by exposure intensity | 19 |
| FDNY firefighter | Eosinophilic pneumonitis case report | 38 |
| Power engineer | Granulomatous pneumonitis case report | 39 |
| Ironworkers | Cough, wheeze, dyspnea, increased airways resistance and bronchodilator response | 23 |
| NYC ESU police | Cough, wheeze, dyspnea, abnormal PFTS and bronchodilator response | 22 |
| Rescue/recovery workers and volunteers (various trades) | Cough, wheeze, dyspnea, abnormal PFTS and bronchodilator response | 21 |

FDNY, Fire Department of New York; WTC, World Trade Center; RUDS, reactive upper airways dysfunction syndrome; GERD, gastroesophageal reflux disease; NSBHR, nonspecific bronchial hyperreactivity; RADS, reactive airways dysfunction syndrome.

during the collapse when few if any had proper respiratory protection during those first days (11, 12).

*Reactive Airways Dysfunction Syndrome.* Reactive airways dysfunction syndrome (RADS) is defined as acute and persistent respiratory symptoms and nonspecific bronchial hyperreactivity (NSBHR; methacholine PC20 <8 mg/mL) after acute exposure to an inhaled irritant in nonsmokers without an allergic history (28, 29). In an exposure-stratified sample of 179 FDNY rescue workers (nonsmokers, previously healthy without a history of allergies or respiratory illness/symptoms and now postcollapse with or without symptoms), we previously described the emergence of persistent RADS during serial challenge testing 1, 3, and 6 mos after the WTC attacks (19). Exposure intensity was defined according to arrival time with highly exposed FDNY rescue workers present during the collapse, moderately exposed FDNY rescue workers present after the collapse but within the first 2 days, and control FDNY rescue workers not present. NSBHR at 1, 3, and 6 mons was associated with exposure intensity independent of airflow obstruction (Fig. 1). Six months post-WTC, highly exposed were 6.8 times more likely than moderately exposed and controls to have NSBHR (95% confidence interval, 1.8—25.2; $p = .004$, chi-square) and NSBHR persisted in 55% of those who had NSBHR at 1 and/or 3 mos. Furthermore, NSBHR during the subacute postexposure period (1 and/or 3 mos) significantly predicted the development of RADS in 20% of highly exposed workers and in 8% of moderately exposed workers at 6 mos postcollapse ($p = .021$; McNemar's test).

In this same group using the same methods as previously described (19), we now report our findings at the 1-yr postcollapse time point. Of the 179 original participants, 1-yr follow-up methacholine challenge testing was available for 63% and symptom questionnaires for 69%. NSBHR was again defined as a methacholine PC20 <8 mg/mL. At all time points, including the 1 yr post-WTC, there remained significantly more FDNY rescue workers with NSBHR in the highly exposed group as compared with the moderately exposed and control groups (Fig. 1; $p < .01$ at all time points; $p = .038$ at 1 yr; chi-square), independent of air flow obstruction. At 1 yr postcollapse, 23% of the highly exposed had NSBHR as compared with only 11% of the moderately exposed and 4% of the controls. Similarly at 1 yr postcollapse, RADS (NSBHR and symptoms) was present in 18% of the highly exposed as compared with 11% of the moderately exposed (Fig. 2). NSBHR during the subacute postexposure period (1–3 mos) argued for but no longer significantly predicted the persistence of NSBHR (Fig. 3) and RADS (Fig. 4) at 1 yr postcollapse. These trends failed to reach statistical significance, most likely as a result of longitudinal dropout reducing the number of participants. Although NSBHR and RADS was previously thought to resolve with removal from exposure (29–31), these findings in highly exposed FDNY rescue workers are consistent with several recent non-WTC-related reports, indicating persistence of respiratory symptoms, physiological abnormalities and RADS (30, 31).

*Reactive Upper Airways Dysfunction Syndrome.* Reactive upper airways dysfunction syndrome (RUDS) is defined as chronic rhinitis triggered by exposure to inhaled irritants (32, 33). In contrast to the objective physiological evidence of NSBHR required for a diagnosis of RADS, objective testing for diagnosing RUDS is not well defined. High rates of upper airways symptoms have been described in WTC rescue/recovery workers. Four weeks after the WTC collapse, 78% of FDNY firefighters reported at least one new or significantly worsened ear, nose and throat symptom in an exposure-stratified sample of 362 workers (11), with significantly higher prevalence ratios in more heavily exposed groups (exposure was categorized by arrival time).

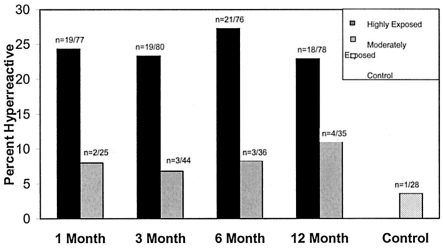


Figure 1. The incidence of bronchial hyperreactivity in Fire Department of New York rescue workers by exposure groups at 1 and 3, 6 and 12 mos post-World Trade Center collapse. Controls were tested only at 3 mos. Total numbers of participants within each exposure group are indicated. The proportion of hyperreactive participants in the highly exposed group was significantly higher than in moderately exposed and control groups at all time points: 1 mo ($p = .004$), 3 mos ($p = .002$), 6 mos ($p = .009$), and 12 mos ($p = .038$).

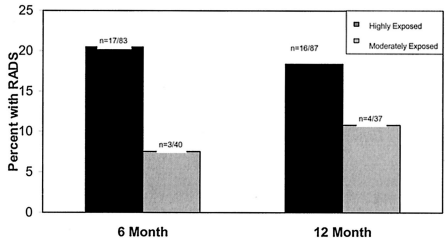


Figure 2. The incidence of reactive airways dysfunction syndrome (*RADS*) in Fire Department of New York rescue workers by exposure groups at 6 and 12 mos post-World Trade Center collapse. Total numbers of participants within each exposure group are indicated. The proportion with RADS (hyperreactive with symptoms and no history of prior pulmonary disease, allergies, or tobacco use) in the highly exposed group was significantly higher than in moderately exposed group at 6 mos ($p = .009$) and 12 mos ($p = .009$).

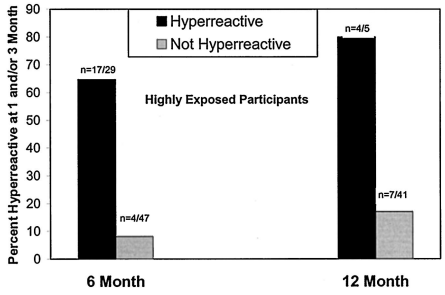


Figure 3. The incidence of persistent bronchial hyperreactivity in Fire Department of New York rescue workers by exposure groups at 6 and 12 mos post-World Trade Center collapse. Highly exposed rescue workers who were hyperreactive early on at 1 and/or 3 mos were more often hyperreactive at 6 mos and at 12 mos than exposed workers who were not hyperreactive at 1 and/or 3 mos. However, this trend did not reach statistical significance, probably as a result of loss of power from longitudinal dropout.

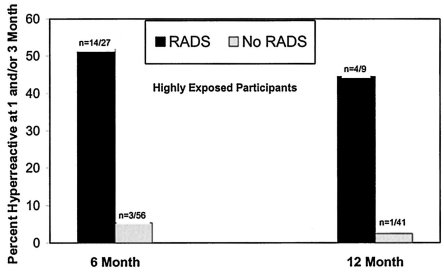


Figure 4. The incidence of reactive airways dysfunction syndrome (*RADS*) in Fire Department of New York rescue workers by exposure groups at 6 and 12 mos post-World Trade Center collapse. Highly exposed rescue workers who were hyperreactive early on at 1 and/or 3 mos, qualified for the diagnosis of RADS at 6 mos, significantly more often ($p = .021$) than highly exposed workers who were not hyperreactive at 1 and/or 3 mos. At 12 mos, this trend no longer reached statistical significance, probably as a result of loss of power from longitudinal dropout.

While working at the WTC site, 92% of 1,138 rescue/recovery workers and volunteers (various trades) at least one new or significantly worsened ear, nose and throat symptom (21). Five months after the collapse, 52% of 96 ironworkers had persistent sinus complaints (23). Persistent nasal and/or throat symptoms were also found in 41% of 240 ESU police officers (74% of the NYC police ESU) between 1 and 5 mos after the collapse (22).

*Gastroesophageal Reflux Disorder.* Unrelated to WTC, we found no published studies linking the emergence of gastroesophageal reflux disease (GERD) with exposures to respirable irritants, although persistent upper gastrointestinal complaints under various diagnostic labels have previously been described in five of six case reports of longstanding RADS (32) and in a case series of persistent gastrointestinal complaints after exposure to chemical irritants (34). Among FDNY rescue workers, who as a group sustained the most intense exposure to airborne irritants at Ground Zero, two different studies have now described high rates of reflux disease (87% of 332 firefighters with WTC cough (20), and 28% to 45% of 179 exposure-stratified FDNY rescue workers between 1 and 6 mos postcollapse, with the higher rates in hyperreactive rescue workers (19)). Although no clear mechanism for the development of GERD has been described in this setting, ingestion of airborne or expectorated respirable materials are presumed etiologies (20). Whether GERD is unique to the WTC exposure or represents a previously unrecognized aspect of inhalation injury in general, whether it marks more severe total dust exposure as opposed to/in conjunction with more severe host inflammatory reaction, as well as whether this gastrointestinal syndrome will persist or resolve, are all questions that remain unanswered at present. What is clear is that when GERD is present in this setting, treatment should be initiated because of the known causal or exacerbating relationship between GERD and airway diseases such as sinusitis, asthma, and chronic cough (35–37).

*World Trade Center Cough.* During the year after the WTC attack, we described a syndrome of clinical, physiological, and radiographic abnormalities resulting from significant unrelenting airway inflammation in 332 New York City firefighters (20). The case definition "WTC cough" specified a persistent cough severe enough to require at least 4 wks of medical leave with onset during the 6 mos after the WTC collapse. Clinical symptoms were consistent with rhinosinusitis and bronchitis, with a surprisingly high rate of symptoms consistent with GERD (87%). Physiological (bronchodilator response [63%] and NSBHR [24%]) and radiographic (computed tomography scan evidence of air trapping [51%] and bronchial wall thickening [24%]) correlates of airway inflammation were found. The incidence of this syndrome was correlated with WTC dust exposure intensity (estimated by initial arrival time at the WTC site). With institution of early, aggressive antiinflammatory therapy, approximately half of the 332 firefighters with this condition had improved to the point at which they could resume active firefighting activities at the end of the 6-mo study period. Over the next 18 mos, at least twice as many FDNY firefighters have met this case definition of "WTC cough" (Prezant, unpublished data).

*Inflammatory Pulmonary Parenchymal Syndromes.* To date, one case of eosinophilic pneumonitis in an FDNY rescue worker (38) and one case of granulomatous pneumonitis in a construction rescue worker have been identified in persons who worked at the WTC site (39). Both patients presented during the immediate postexposure period (i.e., $<$6 wks postcollapse), demonstrated WTC-derived particulate material on pulmonary parenchymal sampling, and promptly improved on systemic antiinflammatory therapy (radiographic parenchymal abnormalities persisted in the patient with granulomatous pneumonitis). Before WTC, we have reported a higher-than-expected rate of biopsy proven sarcoidosis in FDNY firefighters (40), raising the possibility that combustion/pyrolysis products play a role in the etiology. For FDNY firefighters in the 2 yrs post-WTC, the incidence of biopsy proven sarcoidosis increased by two- to threefold pre-WTC rates, and in the third year post-WTC has now returned to baseline. In addition, since WTC there have been two cases of biopsy-proven idiopathic fibrosis, whereas there were no cases during the 15 yrs pre-WTC (FDNY computerized medical database started in 1985; Prezant, unpublished data).

*Late Emerging Diseases—Potential Concern for Malignancies.* It is too early for any of the WTC medical screening, monitoring, or treatment programs to provide clinical prevalence or incidence data on malignancies. However, among those exposed, concern exists because multiple combustion/pyrolysis products are known or potential carcinogens. Substances identified in WTC dust samples include phthalate, numerous different polycyclic aromatic hydrocarbons such as dioxins and brominated diphenyl ethers, and also polychlorinated biphenyls, polychlorinated dibenzodioxins, and polychlorinated furans (1–3). Furthermore, asbestos was used in the WTC construction, and settled WTC dust samples may contain as much as 3% by mass of asbestos (3). Based on serial air sampling, Rappaport and coworkers modeled airborne PAH levels at the WTC site after September 14, 2001. Although initial elevated levels declined rapidly with the dissipation of fires during the 3 mos, concentrations remained two- to eightfold greater than normal NYC background levels, probably as a result of the necessary use of diesel-powered heavy machinery during rescue/recovery efforts (4). When compared with FDNY firefighters who were not exposed, biomonitoring in 321 heavily exposed FDNY firefighters, 4 wks postcollapse, revealed statistically significant elevations of the urinary 1-hydroxypyrene (a PAH metabolite), urinary antimony (a heavy metal commonly found in plastics and fire retardants), and serum heptachlorodibenzodioxin and heptachlorodibenzofuran (dioxin congeners) (41). Although statistically significant elevations were noted, none were elevated to anywhere near clinical significance or current occupational guidelines. Clearly, long-term monitoring is indicated to determine if WTC-related malignancies occur.

## CONCLUSION

Intense and/or prolonged exposure to WTC dust and combustion products has caused aerodigestive inhalation injuries that include persistent RADS, RUDS, and GERD, with an exposure dose-response relationship evident in WTC rescue/recovery workers. Continued medical monitoring and treatment protocols are needed to reduce the health impact and to determine the long-term consequences of this unique exposure. The increased incidence of aerodigestive inhalation injuries emphasizes the need for adequate respiratory protection and improved monitoring/treatment protocols at future disasters. The success of such

plans will depend on solving immediate supply, comfort, compliance, and supervision problems under extreme disaster conditions.

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