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Firefighters' Health and Health Effects of the World Trade Center Collapse

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Firefighters are exposed to extremely hazardous environments. In addition to inhalation of products of combustion and the risk of fatal burn injuries, firefighters suffer a high rate of traumatic injury and death. Over the 3-year period of 2000 to 2002, there were an estimated 1.1 million firefighters with 102 deaths reported to the Bureau of Labor Statistics, giving a death rate of 9.3 per 100,000 compared to the national average for the U.S. work force of 4.2 per 100,000 (1). These statistics do not include the special circumstances of the World Trade Center (WTC) collapse on September 11, 2001, in which an additional 343 firefighters from the Fire Department of the City of New York (FDNY) died. This chapter will review the general hazards of this occupation and describe in detail the medical consequences of the WTC collapse.

HAZARDS OF FIREFIGHTING

Products of Combustion and Chemical Exposures

Toxic chemical gasses and irritating particulates are the major components of smoke produced in fires exposing firefighters to inhalation injury and possible carcinogenic risk. The exact nature of the smoke is determined by the type of material burned, the temperature of the fire, and the presence or absence of oxygen in the combustion environment (2). The combustion products of several common materials are presented in Table 28.1 (3). Firefighting is often considered to have two stages: a knockdown stage, when the fire is brought under control; and an overhaul stage, when smoldering fires are extinguished. Potentially harmful materials have been detected during all phases of fire suppression (4). Studies of controlled fires under laboratory conditions, structural fires, and forest fires offer insight into combustion products commonly encountered by firefighters (2–12).

Particulates generated during fires are a complex mixture of partial and complete combustion products of the original substance with additional compounds adsorbed on the surface (10). Measurement and analysis of particulates found in firefighting environments are limited. Personal breathing-zone sampling of Boston firefighters revealed a median total particulate concentration of 21.5 mg per m^3, with 15% of samples exceeding 100 mg per m^3 (8). In residential fires, total particulate concentrations ranged from undetectable

Table 28.1
Products of Combustion

Source	Major Combustion Products
Wood	CO, CO_2, hydrogen, methane, acetic acid, alcohols, tars, aldehydes, ketones
Cotton	CO, hydrogen cyanide, nitrogen oxides, aldehydes, ketones
Wool	CO, CO_2, hydrogen cyanide, hydrogen sulfide, benzene, toluene, carbon disulfide, carbonyl sulfide
Polyester	CO, CO_2, methane, benzene, saturated and unsaturated hydrocarbons
Nylon	CO, CO_2, hydrogen cyanide, ammonia, nitrogen oxides, low–molecular weight alkanes and alkenes, assorted lactams and nitriles
Polyurethane foams	CO, hydrogen cyanide, CO_2, acetonitrile, acrylonitrile, pyridine, benzonitrile, low–molecular weight hydrocarbons
PVC	CO, CO_2, hydrogen chloride, benzene, saturated aromatic hydrocarbons, phosgene
Urea-formaldehyde	CO, CO_2, hydrogen cyanide, ammonia

CO, carbon monoxide; CO_2, carbon dioxide; PVC, polyvinyl chloride. Adapted from Orzel RA. Toxicological aspects of fire smoke: polymer pyrolysis and combustion. *Occup Med.* 1993;8:415–429 with permission.

to 560 mg per m^3 during knockdown and up to 45 mg per m^3 during overhaul (4). A furnished tenement fire with dense smoke generated particles with mass median diameters of 1 μm during knockdown and 10 μm during overhaul (4). The overhaul phase of firefighting is associated with a negative impact on lung function (13). Diesel exhaust from vehicles in fire stations has also been identified as a significant source of particulate exposure (14).

Among the most toxic products of combustion is carbon monoxide (CO), which displaces oxygen from hemoglobin and is a significant contributor to many fire-associated fatalities. CO is present in nearly all fire environments as a result of incomplete combustion. Personal sampling devices worn by firefighters at structural fires have demonstrated CO levels ranging from 0 to 27,000 ppm (15). In a study of 22 fires, CO was the most common contaminant measured during knockdown (4). Approximately 10% of CO samples exceeded 1,500 ppm, which is a concentration immediately hazardous to life. During overhaul, CO levels were lower, ranging from background to 82 ppm. After an active shift, nonsmoking firefighters were found to have mean blood carboxyhemoglobin levels 2% greater than unexposed nonsmoking controls (16). CO levels found in forest fires are lower than those in structural fires, largely due to superior ventilation and lack of confinement (17).

Hydrogen cyanide is formed during the combustion of wool, paper, silk, and nitrogen-containing synthetic polymers and is another important cause of smoke inhalation fatalities (18). Hydrogen cyanide interferes with cellular respiration and has been detected in up to 50% of residential fires, especially fires involving vehicles, mattresses, or upholstered furniture (4,8,9). Small elevations in serum thiocyanate have been demonstrated in firefighters wearing respiratory protection after active duty (19).

Carbon dioxide (CO_2) is produced during the combustion of all organic materials and can act as an asphyxiant by displacing oxygen from the environment. Personal sampling measurements at residential fires revealed CO_2 concentrations from 350 to 5,410 ppm during knockdown and 130 to 1,420 ppm during overhaul, although concentrations of up to 70,000 ppm have been described (4,20). Irritant gases such as hydrogen chloride can be generated by the decomposition of chlorine-containing plastics such as polyvinyl chloride (PVC), acrylics, and flame retardants. Acrolein, a highly toxic aldehyde and respiratory irritant, has repeatedly been measured at hazardous levels in over half of residential fires. Other potentially toxic agents measured in firefighting environments include nitrogen oxides; sulfur dioxide and sulfuric acid; hydrogen fluoride; formaldehyde; metals such as lead, chromium, and arsenic; and a broad spectrum of volatile organic chemicals (21). Increasingly, firefighters are participating in responses to hazardous materials spills, providing potential exposure to thousands of chemicals used in industry.

Firefighters are also exposed to a wide variety of potential carcinogens. Benzene, a recognized leukemia-causing agent, is detectable at nearly all fires. Polycyclic aromatic hydrocarbons (PAHs) in soots, tars, and diesel exhaust; arsenic in wood preservatives; formaldehyde in wood smoke; and asbestos in building insulation are other carcinogens commonly found in the fire atmosphere. Specific incidents can result in exposures to uncommon but potent carcinogens, including pesticides, polychlorinated biphenyls, and dioxins. Detailed assessment of firefighters' exposures to carcinogens is difficult to perform given the episodic, unpredictable, and complex nature of firefighter exposures. Attempts to measure the amounts of these carcinogens in serum or excreted in the urine have shown only moderate exposure, suggesting that personal protective equipment, when used properly, is effective (22).

As protection against a potentially lethal environment, the Occupational Safety and Health Administration (OSHA) requires firefighters to use self-contained

breathing apparatus (SCBA) respirators designed to provide protection factors of 10,000 when fighting structural fires (contaminants inside the face piece are reduced to 1/10,000 of ambient concentrations) (23). Actual protection factors provided by these respirators are not well-characterized. During actual firefighting work conditions, typical SCBA respirator air supply is not adequate and lasts less than 20 minutes. Furthermore, decreased face-piece fit, possible respirator overbreathing from extreme physical exertion, and patterns of respirator use may add to lower levels of protection. Most firefighters wear their respirators during knockdown of structural fires, but some may not wear the respirator during overhaul or for nonstructural fires, when potentially harmful exposures may still be present. Thus, subclinical smoke inhalation is an expected consequence of firefighting. Despite its limitations, the use of SCBA respirators has dramatically reduced firefighter exposures over the past 20 years (24). For this reason, studies from periods when SCBA use was limited may not be generalizable to situations where SCBA use is now more common.

Biologic Hazards

Firefighters are increasingly involved in rescue operations and the provision of emergency medical services. In this capacity, firefighters are at risk for communicable diseases transmitted by blood, respiratory secretions, or other infectious materials. A study of infectious disease exposures in 650 dual-trained firefighter/emergency medical technicians found an overall incidence rate of 4.4 infectious exposures per 1,000 emergency medical services calls (25). Many populations served by emergency medical service personnel have elevated rates of bloodborne communicable diseases such as hepatitis B, hepatitis C, and human immunodeficiency virus (HIV) (25–27). Although no studies have documented increased rates of transmission of these diseases to firefighters, the prevalence of hepatitis B among emergency service personnel in several major metropolitan areas is elevated, and significant correlations have been observed between hepatitis B infection and years of work in emergency medical service (28–30). Firefighters are included in the OSHA bloodborne pathogens standard and should be educated about the risks of bloodborne infection and means of prevention (31). Vaccination with hepatitis B is required by OSHA for firefighters who have a potential exposure to blood or other infectious materials. In addition, firefighters are at risk for diseases spread by airborne droplets such as tuberculosis. Firefighters should be knowledgeable about tuberculosis prevention and enrolled in purified protein derivative screening programs, in accordance with Centers for Disease Control and Prevention guidelines for health care workers (32).

Physical Hazards

Firefighters are exposed to extremes of heat. Between 1990 and 1995, over 25,000 firefighters sustained physical or chemical burns resulting in significant morbidity and mortality. In addition, over 20,000 firefighters experienced heat exhaustion or frostbite (33). Heavy turnout gear, SCBAs, and barrier clothing used for hazardous material incidents can increase the work of firefighting and impede body temperature control mechanisms, further contributing to thermal stress. The use of more modern personal protective equipment has reduced the severity and the number of burns suffered by FDNY firefighters (34,35). However, this protective benefit is not without cost; modern protective garments impose increased thermal stress, increased workloads, and significant ergonomic disadvantages (36). By modifying modern protective garments to include lighter weight, more flexible and breathable textiles (both within and under the thermal protective garment), the negative impacts of increased thermal stress and workload can be minimized (36). Further reductions in energy expenditure can be obtained by improving physical fitness, weight reduction, and altering firefighting attack strategies to provide assistance and relief time.

Firefighters are also subject to loud noise exposures from sirens, engines, air horns, and pumps. Exposures during emergency response operations can approach 115 dB for brief periods of time, although firefighters' 8-hour time-weighted average noise exposures are frequently beneath the OSHA permissible exposure level of 90 dB (37). Audiometric testing of firefighters has consistently demonstrated excess hearing loss compared to controls, with greater deficits among senior personnel (38,39). Firefighters should be educated about hearing protection and enrolled in hearing-conservation programs to minimize noise-induced hearing loss.

Stress and Shift Work

In the course of their work, firefighters are routinely placed in demanding situations and high-risk environments. As such, firefighting is recognized as one of the most stressful occupations. Although the stresses inherent to firefighting are clear, the physical and emotional consequences of those stresses are less obvious. Stress has been suggested as a cause of cardiovascular disease among firefighters. However, the mechanisms for stress-induced cardiovascular effects remain poorly defined, and there is still debate as to whether firefighters have elevated rates of cardiovascular mortality.

Recent interest has focused on the psychological sequelae of involvement in traumatic fire and rescue events. Following a severe PVC chemical fire, firefighters combating the blaze demonstrated higher measures of postincident psychological distress than unexposed

firefighter controls (40). Increased rates of self-reported psychological distress and alcohol use have also been reported in firefighter populations (41). After working at the WTC, FDNY firefighters have reported increased utilization of counseling services due to posttraumatic stress disorder and/or depression (42). Further studies will be needed before the relationship of traumatic events to firefighters' long-term psychological and emotional well-being can be determined (43).

Firefighters typically work long shifts and are subject to fatigue, alterations in mood, and sleepiness as a consequence of shift work (44). With the flexibility of their work schedule, firefighters may seek additional employment opportunities for their off hours, placing them at risk for further occupational exposures, injuries, and illnesses.

Injuries

Firefighting has one of the highest rates of injuries of all occupations, with 41% of firefighters reporting work-related injuries in 1993 (45). This has not abated in more recent studies (46). Statistics from 1995 describe 94,500 firefighter injuries that required medical attention or resulted in at least 1 day of restricted activity (47). The most common injuries were musculoskeletal, including sprains, strains, and muscle pains, including back pain, that were produced from overexertion. Firefighter tasks, such as carrying hoses and victims, and climbing ladders, frequently require heavy lifting, twisting, stretching, and exerting efforts, activities that have been identified in ergonomic studies as risk factors for injury (48). Physical fitness programs for firefighters may be effective in reducing injury rates (49). Lacerations and burns (physical and chemical) are also frequently reported. These injuries result in lost work hours, significant medical expenses, and even premature disability.

Reproductive Hazards

Firefighters are regularly exposed to chemical and physical hazards that could influence reproductive health. Products of combustion such as acrolein, benzene, CO, and formaldehyde are associated with reproductive toxicity in animal models, although human data are lacking (50). Heat is a reproductive toxin, affecting spermatogenesis and fertility in heat-exposed cohorts; however, the effect of heat on the reproductive health of firefighters has not been studied (51). Lifting also poses a risk for pregnant women, especially in the later stages of pregnancy (52). Offspring of firefighters were found to have increased risk of ventricular septal defects and atrial septal defects in one case-control study, but additional data on the reproductive health of firefighters are needed (53).

LATE-EMERGING DISEASES

Firefighters are exposed to many agents that could potentially cause cardiovascular, respiratory, and malignant disease. Attempts to establish associations between firefighters and occupational diseases have yielded conflicting results, reflecting the challenges encountered in studying this population. Firefighters are selected for their abilities to perform strenuous tasks. This healthy worker population of firefighters may demonstrate lower rates of disease than a normal comparison population, masking exposure–response associations (54). To control for this, some studies rely on comparisons of firefighters to policemen, a group presumed to be similar in physical abilities and socioeconomic status. The occupational exposures experienced by firefighters may vary greatly and are influenced by the types of fires encountered, job responsibilities, and use of personal protective equipment. Data on nonoccupational risk factors such as cigarette smoking are rarely available. Firefighters who experience health problems related to their work may choose to leave their position, creating a survivor effect of individuals more resistant to the effects of firefighter exposures. Despite these difficulties, many important observations about the health of firefighters have been made. Overall, firefighters have repeatedly been shown to have all-cause mortality rates less than or equal to reference populations (55–65). However, several specific causes of morbidity and mortality warrant further discussion.

Cardiovascular Disease

Early U.S. Vital Statistics data suggested an increased cardiovascular mortality among firefighters (66). Recognition that firefighters were exposed to CO, smoke, and physical and psychological stress and the subsequent demonstration of electrocardiogram abnormalities in a small number of firefighters led to additional investigations of this association (67). The epidemiologic data are summarized in Table 28.2 (55–64, 68–70). The majority of studies show risks of cardiovascular disease less than or equal to comparison populations. Although this may be due to the healthy worker effect, studies using police officers for comparison populations similarly show no excess risk. Several studies found increasing risk of cardiovascular disease with increasing duration of employment. In one positive study, Bates (70) reported excess circulatory disease mortality in a cohort of 646 Toronto firefighters between the ages of 45 and 54 years; however, a subsequent study of 5,995 Toronto firefighters, including Bates's cohort, found no significant elevations in circulatory system standardized mortality ratios (SMRs) and no trend by duration of employment (60).

Because risk factors for coronary heart disease are so prevalent in both firefighter and general populations, it will be difficult to statistically prove the impact of

Table 28.2
Cardiovascular Risk Estimates Among Firefighters

Author	Population	Year	Risk Ratio	95% Confidence Interval (CI)
Musk et al. (55)	5,655 Boston firefighters	1978	0.86	NA
Dibbs et al. (68)	171 Boston firefighters	1982	0.5	0.2 to 1.4
Feuer et al. (56)	New Jersey (PMR)	1986	1.01 vs. police 1.02 vs. New Jersey population 1.09 vs. U.S. population	
Vena et al. (57)	1,867 Buffalo firefighters	1987	0.92	0.81 to 1.04
Demers et al. (59)	4,546 northwest firefighters	1992	0.79 vs. U.S. population 0.86 vs. police	0.72 to 0.87 0.74 to 1.00
Heyer et al. (58)	2,289 Seattle firefighters	1990	0.78	0.68 to 0.92
Sardinas et al. (69)	306 Connecticut firefighters	1986	1.07	0.91 to 1.23
Bates (70)	596 Toronto firefighters	1987	1.73	1.12 to 2.66
Guidotti (61)	3,328 Alberta firefighters	1993	1.03	0.88 to 1.21
Burnett et al. (62)	27 U.S. states (PMR)	1994	1.01	0.97 to 1.05
Tornling et al. (64)	1,116 Stockholm firefighters	1994	0.84	0.71 to 0.98
Beaumont et al. (63)	**3,066 San Francisco firefighters**	**1991**	**0.89**	**0.81 to 0.97**
Aronson et al. (60)	5,995 Toronto firefighters	1994	0.99	0.89 to 1.10

Risk ratio is expressed by authors as standardized mortality ratio, proportionate mortality ratio (PMR), standardized incidence ratio, mortality odds ratio, or risk ratio, with no excess risk equal to 1. Bold is statistically significant.

firefighting on coronary heart disease outcomes unless other factors are considered. In a recent case-controlled study by Kales et al. (71), 52 male firefighters with death due to coronary heart disease were compared to control populations. Although standard cardiovascular risk factors (i.e., obesity, smoking, hypertension, cholesterol, prior coronary heart disease) were significantly elevated, fire suppression activities at the time of death carried the highest relative risk for death due to coronary heart disease [odds ratio (OR) = 64.1; 95% confidence interval (CI), 7.4 to 556] when compared to non–fire suppression activities such as training (OR = 7.6; 95% CI, 1.8 to 31.3) and alarm response (OR = 5.6; 95% CI, 1.1 to 28.8). Furthermore, the circadian pattern of deaths due to coronary heart disease was associated with emergency response calls (77% occurring between noon and midnight); a pattern quite the opposite of that observed in general populations, where most events occur in the early morning hours. Thus, firefighters can sustain acute myocardial injuries resulting from acute CO poisoning and/or the acute stress of firefighting. More complete datasets with long-term follow-up are needed to determine whether chronic fire-related exposures result in coronary artery disease, cardiomyopathy, and increased cardiac mortality in this occupational cohort.

Respiratory Disease

Toxic combustion products can have profound effects on the respiratory system, causing acute symptoms, physiologic changes, and chronic diseases. Respiratory irritants, such as hydrochloric acid, phosgene, ammonia, oxides of nitrogen, aldehydes, and sulfur dioxide, can cause direct damage to the proximal airways, distal airways, and alveolar-capillary membrane. The combustion products of synthetic materials in modern furniture and building materials may produce smoke that is more toxic than that produced in the past. Clinical manifestations of acute and/or chronic smoke inhalation can range from mild irritant symptoms of the upper and lower airways to life-threatening adult respiratory distress syndrome; irritant-induced asthma, bronchiolitis obliterans, bronchiectasis, chronic bronchitis, airway injuries, and pulmonary fibrosis have also been described (72). In FDNY firefighters, the incidence and prevalence of sarcoidosis has also been found to be increased compared to concurrent and historic control populations (73). Despite this varied list of smoke inhalation–induced respiratory diseases, for FDNY firefighters, asthma is by far the most common disease and is nearly always the cause of permanent respiratory disability.

The frequency, severity, and duration of smoke exposures appear to be important determinants of clinical outcomes as well as individual host susceptibility factors (72,74–76). Chemical composition and reactivity, water solubility, particle size, and temperature characteristics of the combustion products also influence the pulmonary effects. Although the complexities of exposure assessment and unpredictable nature of fires have permitted only limited evaluation of acute dose-response relationships and even less refined assessments of the long-term effects of smoke inhalation, many important observations have been made about the acute and chronic effects of smoke inhalation in firefighters.

Several studies have examined changes in firefighters' lung function in conjunction with measures of airway reactivity. Sheppard et al. (77) measured baseline airway reactivity to methacholine in 29 firefighters and then followed preshift, postshift, and postfire spirometry over an 8-week period. Significant declines in the forced expiratory volume in 1 second (FEV_1) and/or the forced vital capacity (FVC) were more frequent following work shifts with fires and occurred regardless of firefighters' baseline airway reactivity. Sherman et al. (78) performed spirometry and methacholine challenge testing before and after firefighting activities in 18 Seattle firefighters. Firefighting was associated with acute reductions in FEV_1 (3.4% ± 1.1%) and forced expiratory flow after 25% to 75% of vital capacity had been expelled (FEF_{25-75}) and an acute increase in airway responsiveness to methacholine. Increased airway responsiveness has been identified as a requirement for the diagnosis of reactive airways dysfunction syndrome (RADS; new-onset asthma in a nonallergic nonsmoker after acute exposure to fumes, gases, and possibly other pollutants) and as a risk factor for the development of chronic obstructive pulmonary disease. The finding of increased airway responsiveness in firefighters suggests that they may be at risk for accelerated loss of ventilatory function. Chia et al. (79) exposed 10 new firefighter recruits and 10 experienced firefighters with normal airway reactivity to smoke in a chamber without respiratory protection. Following exposure, the new recruits maintained normal airway reactivity. However, 80% of the experienced firefighters developed increased airway reactivity. The authors suggested smoke-induced chronic injury or inflammation of the pulmonary epithelium in experienced firefighters might lead to increased risk of airway reactivity. Evaluating 13 victims of smoke inhalation 3 days after the fire, Kinsella et al. (80) found 12 (92%) of 13 victims to have airway reactivity, which was strongly correlated with carboxyhemoglobin levels. Repeat assessment 3 months later showed most to have improvement in airway reactivity but not FEV_1 or specific conductance. The authors speculated that airway obstruction following smoke inhalation might be more common and persistent than generally recognized.

Recent studies of fire victims using bronchoalveolar lavage have provided insights into the cellular and biochemical effects of smoke inhalation. Following smoke inhalation, significant numbers of neutrophils are recruited to the airways (81). Neutrophils are capable of releasing proteolytic enzymes and inflammatory cytokines, which may contribute to injury of the airway epithelium and the development of bronchospasm and airway hyperreactivity. In patients with inhalation injury and cutaneous burns, increased numbers of both alveolar macrophages and neutrophils have been demonstrated in the airways; the alveolar macrophage may further contribute to the inflammatory response by elaborating additional cytokines such as tumor necrosis factor and interleukin-1, interleukin-6, and leukotriene B4 (72). Although preliminary, these findings suggest potential mechanisms for the decrements in lung function and increases in airway reactivity demonstrated in epidemiologic investigations.

Longitudinal studies of lung function in firefighters have provided conflicting results. Peters et al. (82) reported accelerated loss of FEV_1 and FVC over a 1-year follow-up of Boston firefighters. Rates of decline were more than twice the expected rate (77 mL per year versus 30 mL per year for FVC) and were significantly related to the frequency of fire exposure. However, in subsequent follow-up studies at 3-, 5-, and 6-year intervals, investigators found rates of decline comparable to the general population, which were unrelated to indices of occupational smoke exposure (83–86). There was evidence of survival bias in the cohort, as firefighters with respiratory difficulties were selectively moved to lesser-exposed jobs. The authors concluded that selection factors within the fire department and increased use of personal respiratory protective equipment were important in reducing the effects of smoke inhalation; significant attrition in follow-up cohorts may also have influenced the results. A 5-year study of firefighters participating in the Normative Aging Study found firefighters to have greater rates of decline in FEV_1 and FVC than nonfirefighters (18 mL per year and 12 mL per year, respectively). It is important to note that the participants in these studies were evaluated before routine use of respiratory protective equipment and may have sustained very significant smoke exposures. Two more recent studies of firefighters from the United Kingdom have not shown evidence for longitudinal decline in lung function (87,88). In FDNY firefighters, pulmonary function was followed over nearly 5 years (1997 to 2001) prior to the WTC collapse, and the mean adjusted decrease in FEV_1 was 30 mL per year (89). Overall, the evidence suggests that firefighter cohorts using appropriate respiratory protective equipment do not have accelerated loss of ventilatory function, although additional research is needed in this area. It is important to note that Wildland firefighters, who do not typically wear protective respiratory equipment, have been shown to have decrements in lung function and increased airway responsiveness after a season of fighting fires (90).

The effects of frequent smoke exposure on mortality from nonmalignant chronic respiratory conditions have also been investigated. Studies comparing firefighters to U.S. population controls have demonstrated reduced chronic respiratory disease mortality rates in firefighters, despite evidence of acute and chronic pulmonary effects of smoke inhalation. However, these results may be due to the healthy worker effect, where selections of healthy workers results in mortality rates lower than a

general reference population. In a study of New Jersey firefighters, Feuer and Rosenman (56) found an excess of nonmalignant respiratory disease compared to police controls (proportionate mortality ratio = 1.98, $p < 0.05$). Rosenstock et al. (65) compared a cohort of firefighters from the Northwestern United States to police and found an increased SMR of 141 for nonmalignant respiratory disease, as opposed to a deficit when compared to U.S. rates. However, a subsequent study of the same cohort with a longer period of follow-up found the risks of nonmalignant respiratory disease to be of lower magnitude (incidence density ratio = 1.11; 95% CI, 0.71 to 1.73) (59). There is need for additional research on the chronic effects of smoke inhalation using appropriate control groups, especially in the context of changing firefighter exposures.

Malignancies

Firefighters are also exposed to a wide variety of potential carcinogens. Detailed assessment of firefighters' exposures to carcinogens must account for the varied, unpredictable, acute, chronic, and complex nature of firefighter exposures and is beyond the scope of this review. Components of fire smoke depend on what is being burned and may include hundreds to thousands of chemicals and byproducts of combustion and pyrolysis including PAHs, benzene, PVC, acrylonitriles, creosote, polychlorinated biphenyls, dioxins, petrochemicals, formaldehyde, pesticides, herbicides, and heavy metals and asbestos (91). When reviewing occupational cancer studies of firefighters, it is important to note whether the healthy worker effect, longitudinal dropout (due to job change, retirement, and death), and the inaccuracies of death certificates have been accounted for, and rarely, if ever, have studies been designed well enough or with adequate numbers to accomplish this. However, despite these limitations, biologic plausibility exists and/or epidemiologic evidence suggests that firefighters, when compared to the general population, may have an increased incidence of or mortality from brain cancer, leukemia, non-Hodgkin lymphoma, multiple myeloma, bladder cancer, testicular cancer, gastric cancer, esophageal cancer, colorectal cancer, liver cancer, skin cancer, and lung cancer (92).

WORLD TRADE CENTER STUDIES

Two commercial jets filled with more than 90,000 liters of fuel struck the twin towers of the WTC on September 11, 2001 (Fig. 28.1). The resulting fire caused the collapse of two 100-story towers, killing 2,726 people (including 343 FDNY firefighters) and creating a rubble pile rising six stories above ground. This event released an unprecedented amount of dust and products of

Figure 28.1 World Trade Center collapse. Painting by Holly Meeker Rom.

combustion into the surrounding area, leading to an acute intense exposure of thousands of rescue workers and bystanders. In addition to this early exposure, the fire in the rubble pile burned for 3 months, and the excavation of debris proceeded for months thereafter, producing additional lower level exposures in rescue and recovery workers. The magnitude of the atrocity focused intense medical, scientific, and political interest on the exposed populations, leading to a wealth of information on the health effects of the WTC collapse. The application of significant resources to this discreet event has yielded valuable insights in many fields relevant to environmental and occupational medicine. This section will summarize findings of the first 4 years in this ongoing effort in medical monitoring of the health effects of the WTC collapse.

The initial collapse produced a cloud so thick that survivors reported total darkness at ground level (93,94). This cloud extended for many blocks from the WTC site, enveloping thousands of rescue workers and civilian bystanders. Their mouths filled with material, leading to inhalation and swallowing of large amounts of dust. The characteristics of the dust are essential in understanding the consequences of this immediate high-intensity exposure.

The energy of the collapse broke construction material into small particles of various sizes that were suspended in the air for a significant period (89). For many, especially those caught in the debris cloud following the

collapse or exposed to high concentrations of dust during other activities, this dust was swallowed, affecting the digestive system with new-onset or exacerbations of gastroesophageal dysfunction (GERD). The large particles (>3 μm in diameter) suspended in the cloud lodged in the sinuses and trachea, leading to upper respiratory tract irritation. The dust also contained small respirable particles that affected the lower airways. In addition, the volume and density of aerosolized particles was so intense on September 11, 2001 that the upper airway filter was overwhelmed, and large particles penetrated into the smaller airways (96). Concrete was a major contributor to the dust, leading to a pH ranging from 9 to 11 that produced marked alkaline "burn" of mucosal surfaces. The high pH material was concentrated in the larger size fractions, which would be most irritating to the sinuses, esophagus, and large airways. Inorganic material was concentrated in the large particles as well (97). Equivalent concentrations of smaller sized particles were shown to actually elicit more rather than less release of inflammatory mediators from human lung cells in vitro (98).

Chlorine was detectable in the particulate matter $(PM)_{2.5}$ fraction, while antinomy, aluminum, titanium, and magnesium were present principally in the PM_{10-53} fraction, and iron, zinc, and calcium were most highly abundant in the $PM_{2.5-10}$ and PM_{10-53} fractions. Continued exposure to pollutants occurred due to subterranean fires (lasting until the end of December 2001) and demolition work for many months thereafter. Despite significant concentrations of lead and mercury in environmental samples, measurements of serum lead and urine mercury in over 11,000 FDNY firefighters failed to find significant elevations.

Given the well-documented toxicity of asbestos, rapid and careful attention focused on measuring this component of the dust produced by the WTC collapse. From 1971 until 1973 (when it was banned), chrysotile asbestos was used for fire insulation in the lower floors of the WTC (99,100). The U.S. Environmental Protection Agency (EPA) tested more than 10,000 ambient air samples from the area surrounding the WTC site. While asbestos comprised 0.8% to 3.0% of the settled dust, only two of the air samples analyzed by EPA were found to contain asbestos at levels above the clearance standard of 70 fibers per square millimeter. The sampling was started several days after the collapse and therefore may not have reflected the intensity of exposure to asbestos suffered by those who were present during the first several days. In one severely ill firefighter who was caught in the collapse, bronchoalveolar lavage (BAL) fluid obtained 2 weeks later demonstrated nonferruginous asbestos, suggesting the fibers seen in BAL were recently inhaled (Fig. 28.2) (101). BAL from this patient also contained fly ash and fibrous glass, which confirmed recent exposure to dust similar to that found at the WTC site.

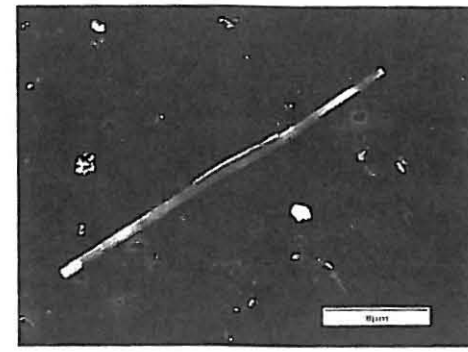

Figure 28.2 Amosite asbestos fiber (uncoated) 33 μm in length by 1 μm in width obtained from BAL of a firefighter 2 weeks after the WTC collapse. (From: Rom WN, Weiden M, Garcia R, et al. Acute eosinophilic pneumonia in a New York City firefighter exposed to World Trade Center dust. *Am J Respir Crit Care Med.* 2002;166:797–800 with permission.)

Respirable particles with an aerodynamic diameter less than 2.5 μm represented 0.88% to 1.98% of the total settled dust mass. Fibrous glass and other inorganic particles produced by destruction of the buildings and their contents comprised a majority of the dust (95,97). The products of combustion yielded organic components of the dust, including PAHs at levels of 5 μg per g to hundreds of micrograms per gram (102). Investigation of expectorated sputum obtained from rescue workers 10 months after the collapse documented high levels of dust particles in the alveolar samples. In addition, rescue workers who were at the site for more than 10 days had higher levels of neutrophils and eosinophils in their induced sputum, suggesting that the lower level exposures produced during the recovery effort produced long-term inflammation in the lung (Table 28.3) (96). It is too early to determine if increased cancer rates will result from these exposures, but the oncogenic potential of these exposures merits long-term follow-up of those with significant exposure.

Rescue workers who were members of the FDNY were intensively studied. This group of workers provided a unique opportunity to define the effects of the exposures to WTC dust and products of combustion because preexposure health evaluations, including nearly 5 years of pulmonary function tests, were available on the entire cohort. Medical interviews and FDNY work logs documented exposure intensity. The exposure variables that correlated with the intensity of exposure were work assignment and the time of arrival at the WTC site. Cumulative exposure has not yet been analyzed. An additional advantage of studying this cohort is that dropout is minimized because active FDNY employees undergo mandated routine health evaluations and retired FDNY members are actively recruited to continue in this long-term medical monitoring program now funded by the National Institute of Occupational Safety and Health.

Table 28.3
Differential Counts, Metalloproteinase 9 Levels, and Particle Size Distribution in Fire Department of New York City Firefighters Analyzed According to Cumulative Exposure (<10 vs. ≥10 days of exposure)

	<10 Days	≥10 Days	p
Duty (years)	15.6 ± 8.3	17.6 ± 8.6	0.78
Macrophages (%)	31.1 ± 13.7	38.4 ± 16.5	0.18
Neutrophils (%)	44.2 ± 16.5	55.7 ± 15.2	0.05
Lymphocytes (%)	11.6 ± 7.0	12.9 ± 6.8	0.59
Eosinophils (%)	1.5 ± 1.9	4.4 ± 5.2	0.04
Particles >2 μm (%)	32.3 ± 13.7	38.6 ± 17	0.48
Particles >5 μm (%)	7.8 ± 3.3	9.3 ± 7.1	0.28
Ln MMP-9 (pg/mL)	0.37 ± 0.67	0.44 ± 1	0.84

Differential cell counts are expressed as percent of 200 cells counted in Giemsa cytospin preparation. MMP-9, metalloproteinase 9. (From Fireman EM, Lerman Y, Ganor E, et al. Induced sputum assessment in New York City firefighters exposed to World Trade Center dust. *Environ Health Perspect.* 2004;112: 1564–1569 with permission.)

Pre-WTC pulmonary function tests served as a baseline for a vast majority of the cohort and showed that vital capacity and FEV_1 for FDNY firefighters averaged 95% of predicted normal (age and height adjusted), with a normal distribution around the mean (Fig. 28.3). These data support the conclusion that a health worker effect existed in this cohort before the exposure to WTC dust and products of combustion. Typically, average annual loss in lung capacity due to aging alone is about 30 mL. Pulmonary function testing performed on the entire cohort of exposed workers in the 6 months after September 11 found an average loss in FEV_1 of 372 mL (89). A similar loss in FVC was also observed. The decline in lung function occurred in all members of the cohort,

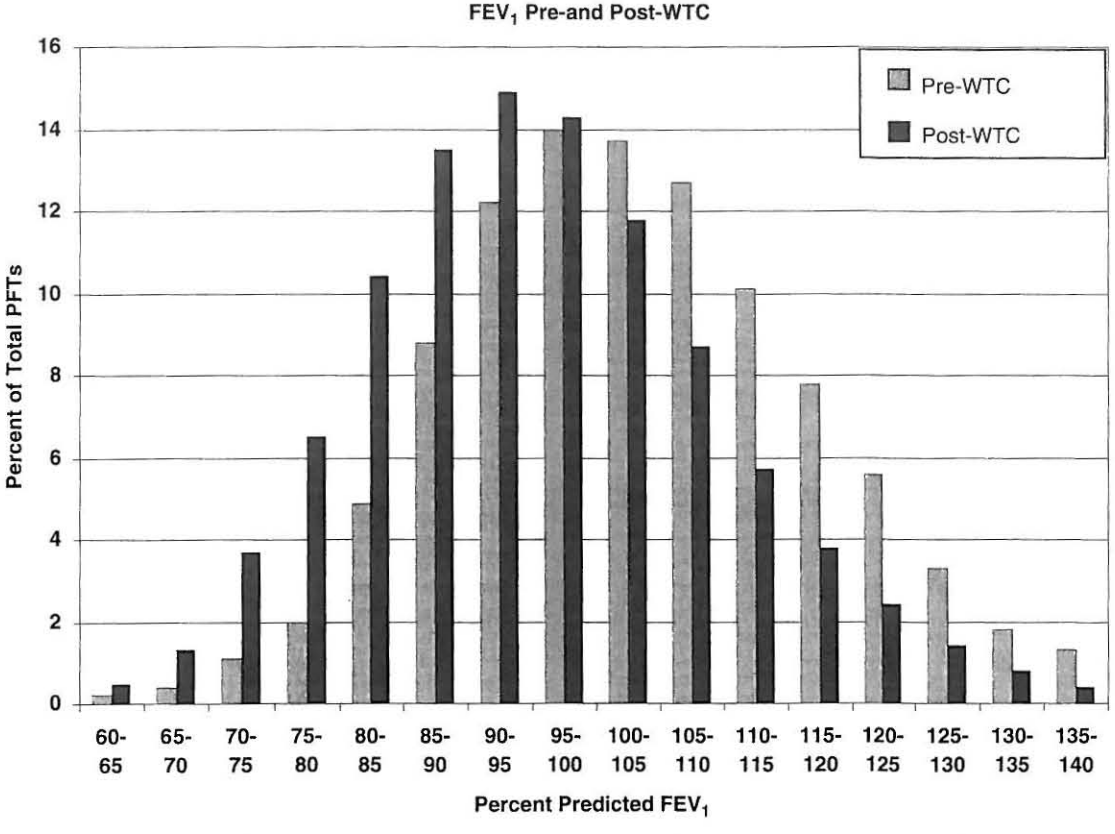

Figure 28.3 Distribution of FEV_1 in approximately 12,000 firefighters before and after the WTC collapse. $p < 0.01$.

with a shift in the entire normal distribution to the left (Fig. 28.3). In addition, those FDNY rescue workers who were at the site during the morning of the collapse had the greatest loss of pulmonary function (89). Future studies are ongoing to determine the effects of time and treatment on lung function. Our preliminary data suggest that early diagnosis and aggressive treatment may result in clinically significant improvements.

In addition to spirometry, which was available on a vast majority of the FDNY cohort, a random cross-section of 150 rescue workers agreed to undergo serial spirometry and methacholine challenge testing at 1, 3, and 6 months after exposure (103). Similar to the entire FDNY cohort (89), there was a progressive loss of FEV_1 and FVC over the 6-month study period, with preservation of the FEV_1/FVC ratio in the normal range. Methacholine challenge testing confirmed a high incidence of airway inflammation in this randomized stratified sample of FDNY firefighters. There was a strong relationship between exposure intensity and the development of airway reactivity (Fig. 28.4) (103). The highest exposed FDNY firefighters present at the site during the collapse had a 23% prevalence of airway hyperreactivity 6 months after exposure as compared to a 6% prevalence of airway hyperreactivity in nonexposed FDNY firefighters. Highly exposed workers were 8.9 times more likely than moderately exposed workers and controls to have airway hyperreactivity, an association that remained significant after adjusting for ex-smoking, airflow obstruction, age, and respiratory symptoms. Furthermore, airway hyperreactivity at 1 month was the best predictor of airway hyperreactivity at 6 months, and the presence of airway hyperreactivity was a strong predictor of symptoms, medical leave, and RADS.

Because cough was so prevalent after the collapse, we found it useful to define "WTC Cough" as a new, persistent cough with respiratory symptoms severe enough to require medical leave for 4 weeks or longer (93). In the first 6 months after the collapse, 332 FDNY firefighters met criteria required for "WTC Cough," and once again, an exposure intensity gradient was displayed, with 8% of highly exposed, 3% of moderately exposed, and 1% of less-exposed firefighters having "WTC Cough." Additional symptoms were nasal congestion (54%), dyspnea (95%), chest discomfort (85%), and GERD (87%). There were near-equal reductions in FVC and FEV_1, with greater than 500-mL declines observed in 54%. The ratio of FEV_1/FVC was unchanged, but residual volumes were increased, and there were reductions in flow rates at low lung volumes, suggesting small airway dysfunction had reduced both FEV_1 and FVC in equal proportion. Although the routine chest radiographs showed few abnormalities, chest computed tomography scans did document peribronchial inflammation, and expiratory images showed a mosaic attenuation pattern supporting the conclusion that a significant subset of the cohort suffered from small-airway inflammation. In those tested prior to treatment, 63% had a bronchodilator response, and 24% had airway hyperreactivity consistent with the definition of irritant-induced asthma or RADS. With completion of the WTC rescue/recovery effort, additional FDNY firefighters have presented for treatment. Treatment has improved symptoms, but as of May 15, 2006, over 600 FDNY firefighters (5% of the workforce) have qualified for permanent respiratory disability benefits based on abnormal pulmonary function and/or airway hyperreactivity that prevents the safe performance of their job duties in smoke-filled noxious environments. Nearly all of these firefighters arrived at WTC during the first 48 hours after the attack, and most were present during the morning of the collapse.

The high incidence of other aerodigestive symptoms in those with "WTC Cough" prompted further investigation in the FDNY cohort. Reactive upper airways dysfunction syndrome (RUDS) is defined as chronic rhinosinusitis and/or throat inflammation triggered by exposure to inhaled irritants (104,105). In contrast to the objective physiologic evidence of nonspecific airway or bronchial hyperreactivity (usually tested with methacholine) required for a diagnosis of RADS, objective testing for diagnosing RUDS is not well-defined. Four weeks after the WTC collapse, 78% of FDNY firefighters reported at least one new or significantly worsened symptom consistent with RUDS (nasal/sinus congestion, drip, swelling, tenderness and/or throat irritation, hoarseness, or spasms) in an exposure-stratified sample of 362 FDNY firefighters (106). In an unselected cross-section of 179 exposure-stratified FDNY rescue workers, 28% to 45% had significant GERD (103). In all studies, significantly higher symptom rates were reported in the more heavily exposed groups (exposure was again categorized by arrival time). Similar findings have been found in the entire FDNY cohort. Although no clear

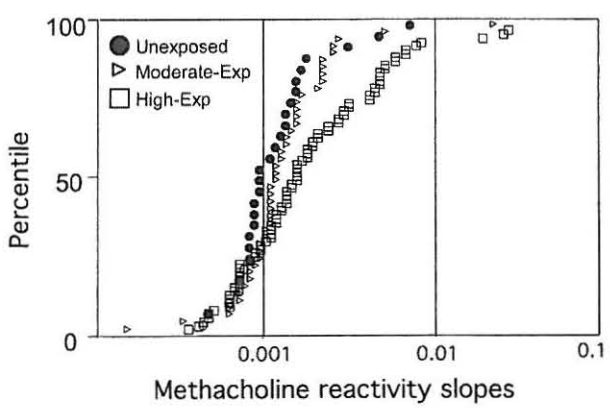

Figure 28.4 Distribution of methacholine responsiveness in a cross-section of firefighters with different intensities of exposure to dust at the WTC site. This is calculated by the slope method rather than by PC_{20}. Data points and curves further to the right on the x-axis indicate hyperreactivity at a lower dose of methacholine.

mechanism for the development of GERD has been described in this setting, ingestion of airborne or expectorated respirable materials are presumed etiologies, possibly exacerbated by increased psychosocial stress and shift work–related dietary discretions. Whether GERD is unique to the WTC exposure or represents a previously unrecognized aspect of inhalation injury in general is a question that needs further investigation. What is clear is that, when GERD is present in this setting, treatment of GERD reduces the severity of airway diseases such as sinusitis, asthma, and chronic cough (107–109).

The clinical presentation of symptoms in other non-FDNY WTC-exposed populations has been similar to those observed in the FDNY cohort. In 240 previously healthy New York City (NYC) Emergency Service Unit (ESU) police officers, 77% had upper and/or lower respiratory symptoms during the first 5 months after collapse (110). In 96 ironworkers, 77% had upper and/or lower respiratory symptoms 6 months after collapse (111). In 1,138 rescue/recovery workers and volunteers of various trades, 58% developed a new or worsening cough while working at the WTC site. Thirty-three percent of these workers had abnormal spirometry, and 11% had a bronchodilator response (112). Respiratory symptoms have been linked to WTC dust exposure intensity (mostly estimated by initial arrival time) in many WTC rescue recovery groups. Furthermore, respiratory symptom prevalence has been correlated with objective physiologic pulmonary abnormalities. Abnormal postcollapse spirometry occurred more often in NYC ESU police officers with dyspnea, chest discomfort, or wheeze (110). Surprisingly, pulmonary symptoms correlated poorly with abnormal spirometry. More specialized testing, such as forced oscillation, did find abnormalities, which were significantly more common in those who had not worn a respirator with a canister in a group of 96 ironworkers (111). No such associations between respiratory protection and symptoms and/or pulmonary physiologic abnormalities have been reported in FDNY rescue workers presumably because the FDNY group arrived during the collapse when few, if any, had proper respiratory protection during those first days (106,113).

The similarity between FDNY and non-FDNY worker/volunteer WTC cohorts extends to other aerodigestive symptoms as well. While working at the WTC site, 92% of 1,138 rescue/recovery workers and volunteers (various trades) experienced at least one new or significantly worsened nasal/sinus or throat symptom (112). Five months after the collapse, 52% of 96 ironworkers had persistent sinus complaints (111). 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 months after the collapse (112). Previously normal residents surveyed 12 ± 4 months after the collapse had threefold increased new-onset respiratory symptoms compared to unexposed controls in New York; importantly, 26% of exposed residents versus 7.5% of controls had persistence of these new-onset respiratory symptoms (114).

CONCLUSION

Despite the longstanding recognition of the dangers of fighting fires, firefighters continue to sustain elevated rates of injury and illness. Although substantial progress has been made in protecting respiratory health through the use of personal protective equipment, firefighters continue to demonstrate increased rates of fatal and nonfatal injuries, hearing loss, respiratory disease, and certain malignancies. Newly appreciated risks of communicable diseases, cardiac diseases, and reproductive toxicity should stimulate the need for further investigations. For WTC-exposed firefighters, the major impact has been on respiratory (aerodigestive: irritant asthma, RUDS, and/or GERD) and mental (posttraumatic stress disorder and depression) health. The long-term course of WTC-related exposures and chronic inflammation will require continued diligent observation of this cohort. However, in all firefighter studies, the healthy worker and longitudinal dropout effects must be considered because, otherwise, health risks may be underestimated or ignored. Given the firefighters' important role in protecting society, their health should receive special attention. Respiratory, communicable, cardiac, and malignant disease may be reduced by conscientious use of respiratory protective equipment through all stages of firefighting activities, but until improved technology makes the wearing of such equipment practical for actual work-related time periods, we should not be surprised that compliance with such equipment will be less than adequate. As was done with thermal protection to reduce burn injuries, future efforts should be targeted towards a multidisciplinary approach to preventing injury and illness and protecting firefighters' health and well-being.

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Environmental and Occupational Medicine

FOURTH EDITION

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